EFFECT OF WINTER GRAZING PROGRAMS ON FEEDLOT PERFORMANCE, VISCERAL ORGAN MASS, BODY COMPOSITION, AND SPLANCHNIC METABOLISM OF STEERS

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Introduction

Extensive animal production systems often integrate several management practices before the final end product, and beef cattle production is no exception (Drouillard and Kuhl, 1999). Beef cattle production systems often involve cow-calf, growing and (or) backgrounding, and finishing phases of production. At each phase of production, decisions are made about the level of nutrition that the animal will receive. Nutritional decisions can be immediately beneficial, neutral, or negative with respect to animal growth. Additionally, nutritional decisions can have long-term effects on future animal growth.

A period of grazing is often incorporated into production systems for beef cattle. However, season and weather have large effects on quantity and quality of available forage (Lawerence and Pearce, 1964). Restrictions include dry matter intake (**DMI**), energy, and (or) protein intake, which will reduce animal performance. However, when previously restricted cattle are refed they exhibit compensatory growth (Fox et al., 1972; Ferrell et al., 1986; Sainz and Bentley, 1997). Compensatory growth has been defined as the more rapid or efficient growth of cattle following a period of nutritional restriction or environmental stress (NRC, 1996). Many reports document the occurrence of compensatory growth in cattle. Generally, compensatory growth by animals is defined as

1) increased rate of live body weight gain; 2) more efficient rate of body weight gain (Fox et al., 1972; Ferrell et al., 1986; Sainz et al., 1995); and 3) reduced maintenance energy requirements (Fox et al., 1972; Shetty, 1990). The occurrence of compensatory growth and the overall response to restriction is highly variable (Coleman and Evans, 1986; Drouillard et al., 1991a, NRC, 1996).

The following discussion will examine the effects of previous nutrition on subsequent performance and the occurrence of compensatory growth. Following that discussion is an examination of some of the possible underlying mechanisms of compensatory growth: changes in visceral organ mass, blood metabolites, and net flux of metabolites across splanchnic tissues.

Effect of Previous Nutrition on Subsequent Growth Performance

High Forage Diets

Performance. Seasonal patterns of forage growth result in variations in forage availability and forage nutritive value, and this greatly influences cattle performance. Several researchers have reported decreased animal body weight (BW) gains when animals consume low-quality forage-based diets (White et al., 1987; Lewis et al., 1990). In an experiment conducted by White et al. (1987), steers grazed either wheat pasture (ADG = 0.71 kg/d) or consumed poor quality bermudagrass hay with decreasing supplementation levels and ADG (0.16, -0.07, and –0.23 kg/d). After 98 d, steers were placed on high quality pastures for a 112-d grazing period. Grazing ADG (112 d) was inversely related to previous ADG (0.37, 0.46, 0.51, and 0.54 kg/d for wheat pasture, bermudagrass hay with decreasing level of supplementation, respectively). Steers that

had previously been restricted in BW gain by consuming low quality forage exhibited compensatory growth during the summer grazing period. In a similar study, Lewis et al. (1990) utilized corn residue silage with differing levels of supplementation to create low, medium, and high levels of steer BW gain during a 106-d winter feeding period (0.28, 0.38, and 0.50 kg/d, respectively). Steers then grazed fertilized pastures during the summer. Daily gain of steers during the summer decreased linearly with increasing level of winter-feeding. The reduction in growth performance during the summer was 81 g for every 100 g of winter gain. Steers that gained the least during the winter exhibited compensatory growth by gaining 27% faster during the summer grazing period. Lawrence and Pearce (1964) observed a similar effect of decreasing winter BW gain of animals resulting in greater summer grazing BW gains. Baker et al. (1992) examined the effect of restricted energy intake from silage diets prior to summer grazing. Steers that had been restricted during silage feeding had 17% greater live, shrunk, and empty body weight (EBW) daily gains during grazing compared with steers that had ad libitum silage consumption. In the experiments of White et al. (1987) and Lewis et al. (1990), when steers that exhibited compensatory growth during the summer grazing period were placed into a feedlot, no differences in ADG, intake, or gain efficiency were reported among different winter gain treatments.

Body composition. Empty body weight (EBW) is often used to relate body composition and organ mass. Empty body weight is defined as live BW minus digesta mass. Restricted steers, in the experiment of Baker et al. (1992), after the silage-feeding period had reduced live, EBW, and carcass weights compared to ad libitum fed steers. Empty body of restricted steers contained 34% less fat, 6% more protein, and 39% less

total energy than ad libitum fed steers. After steers were realimented by grazing perennial ryegrass pastures for 172 d, previously restricted steers had 18% greater empty body fat, 6% lower protein, and similar total energy accretion compared with previously unrestricted steers.

The compensatory growth response of cattle consuming roughage diets appears to depend on the genetic gain potential of the cattle. During realimentation, the growth potential of a steer will result in an increased ADG, such that BW of normal and realimented steers will be similar given adequate opportunity. Body composition is dictated by the level of available nutrients and priority of tissue accretion; bone > lean >> fat.

High Grain Diets

Performance. In many studies reporting compensatory growth, high-grain or finishing diets were utilized during realimentation after restriction of DMI. Fox et al. (1972) utilized Hereford steers to examine the effect of 190 or 145 d of energy and protein restriction (maintenance level of feeding) on subsequent compensatory growth when steers were fed corn or soybean flake diets. Compensating steers had 24% greater ADG and 20% greater gain efficiency compared to ad libitum controls up to 364 kg of BW and 35% greater ADG and 25% greater gain efficiency up to 454 kg of BW. Dry matter intake was similar (6.5 kg) between compensating steers and ad libitum controls up to 364 kg; however, DMI by compensating steers was 1.1 kg greater compared with ad libitum controls up to 454 kg. Carstens et al. (1991) restricted the growth of Angus x Hereford steers to 0.4 kg/d for 189 d. During realimentation, compensating steers had

72% greater ADG from 325 to 420 kg BW compared with ad libitum-fed control steers. From 325 to 420 kg BW, compensating steers had 0.92 kg lower DMI and about a two-fold greater gain efficiency. During the entire re-feeding period (325 to 500 kg BW), compensating steers had greater ADG (1.59 vs. 1.16 kg/d), similar DMI, and 50 g/kg ADG:DMI improvement compared with control steers. Yambayamba et al. (1991) demonstrated the effect of length of restriction on BW gain of compensating heifers. Heifers were either fed ad libitum to 400 kg BW, intake restricted to gain 0.5 kg/d for two months and then fed ad libitum to 400 kg BW, or intake restricted to gain 0.5 kg/d for two months then intake restricted to maintain BW for two months and then fed ad libitum to 400 kg BW. Heifers that had been restricted to 0.5 kg/d for two months had 14% greater daily BW gains when fed ad libitum for 58 d compared with ad libitum fed controls. Heifers that had been restricted to 0.5 kg/d for two months followed by 0.0 kg/d for two months had 40% greater ADG during the early ad libitum feeding period compared with ad libitum fed controls and 31% greater ADG compared to two month restricted heifers. Heifers restricted for two months had DMI that was 1 kg/d less than ad libitum fed heifers and 26% greater gain efficiency. Heifers restricted for four months had 1.5 kg/d greater DMI, and 30% greater gain efficiency than ad libitum fed heifers during the early feeding period.

Conflicting data of increased BW gains during re-feeding of previously restricted steers has been reported (Rompala et al., 1985; Hayden et al., 1993). Rompala et al. (1985) reported that steers fed grass hay for 70 d to maintain BW and then refed a high-moisture corn, corn silage diet had similar live and EBW ADG compared with normally fed steers that gained 1.15 kg/d live weight and 1.05 kg/d EBW. Previously restricted

steers also had similar DMI and EBW gain efficiency compared with normally fed steers. Similarly, Hayden et al. (1993) reported similar EBW gain between steers previously energy-restricted for 92 d and energy-adequate steers finished for 88 d. However, Hayden et al. (1993) did report 1.09 kg/d greater DMI which resulted in 11% lower gain efficiency by compensating steers.

Drouillard et al. (1991a) examined the effects of long and short duration (154 or 77 d), sever or mild, and metabolizable protein (54 or 62 g/kg DM metabolizable protein) or energy restriction (0.62 or 0.75 Mcal/kg net energy for gain) of steers compared with finishing control steers (90 g/kg DM metabolizable protein, 1.61 Mcal/kg net energy for gain). Steers that had been restricted in either metabolizable protein or energy for long or short periods exhibited compensatory growth (except short, mild energy restriction). Compensating steers during realimentation exhibited increased ADG (1.53 and 1.43 vs. 1.28 kg/d for energy and protein restriction vs. finishing control, respectively) and increased DMI (10.5 and 10.1 vs. 9 kg/d for energy and protein restriction vs. finishing control, respectively). However, gain efficiency was similar between compensating steers and finishing controls. An inverse relationship was observed for severity of energy restriction and finishing ADG and gain efficiency. Also the duration of energy restriction did affect gain or efficiency of steers (short, 77 d < long, 154 d). The authors reported that protein restriction had a larger effect on animals during the restriction period than energy restriction. Additionally, the restriction of protein during the growing phase may have necessitated the increase of crude protein levels during finishing to replace body nitrogen required for full growth. Additional studies with lambs by Drouillard et al. (1991b) re-examined the effects of metabolizable protein or energy restriction on lamb

performance. In the lamb trial, the restriction period was for 35 d. During the refeeding period after a two-week adjustment to the finishing diet, protein and energy-restricted lambs did not differ and had 20% greater ADG, 0.2 kg/d greater DMI, and 8% greater EBW gain efficiency than unrestricted lambs. Wester et al. (1995) conducted a similar experiment to examine the effect of protein or energy restriction on lamb growth. Lambs were limit fed their respective diets during a 7-week restriction period that resulted in ADG of 0.25, 0.03, and 0.02 for ad libitum control, energy and protein restricted lambs, respectively. All lambs were then allowed to consume feed to appetite during a 2-week repletion period. Average daily gain was 29 and 21% greater in energy and protein-restricted lambs, respectively, compared with control lambs. Dry matter intake (kg/d) was greater in energy-restricted compared with protein-restricted lambs. Dry matter intake, as % of final BW, was greater in both energy and protein-restricted lambs compared with control lambs. Similarly, gain efficiency in energy and protein-restricted lambs was 27% greater compared with control lambs. In contrast, Abdalla et al. (1988) did not observe any compensatory growth in Holstein steer calves that had previously been restricted in energy intake. However in the same experiment, steers that had been protein restricted did exhibit compensatory growth (increased EBW ADG, intake relative to metabolic body weight, and gain efficiency). In two experiments reported by Rossi et al. (2001), steers that had previously been restricted in crude protein and then refed diets with adequate crude protein had 14 and 27% greater ADG and 13 and 27% greater gain efficiency than steers fed diets adequate in crude protein.

Sainz et al. (1995) utilized a 75%- high-concentrate diet fed ad libitum or limit-fed and a 96% hay-roughage diet (restriction of energy intake) fed ad libitum to create

three treatments to examine compensatory growth. During the growing phase, EBW ADG was 1.96, 0.69 and 0.77 kg/d for concentrate-ad libitum, concentrate-limit fed and roughage-fed steers, respectively. During the subsequent finishing phase all steers were fed the same concentrate diet ad libitum. Steers that were previously limit-fed had the greatest EBW ADG followed by the roughage-fed steers and then ad libitum-fed steers (1.92 > 1.74 > 1.22 kg/d, respectively). Dry matter intake of steers of both restricted treatments was greater than DMI intake by the ad libitum steers (11.36 vs. 9.04 kg/d). Gain efficiency of limit fed steers was 23% greater and 9% greater in roughage fed steers compared with the ad libitum steers. Steers were fed to similar final BW, but final EBW were 30 kg greater for intake-limited and roughage-fed steers compared with ad libitum-fed steers. Subsequent analysis determined that previously limit-fed steers had the lowest estimated maintenance energy requirements, whereas the roughage-fed steers had increased maintenance energy requirements compared with steers fed the concentrate diet ad libitum.

The response of steers and lambs to restrictions of intake, energy, and protein has varied in magnitude and timing during realimentation. In general most studies reported an increase in ADG, DMI, and ADG:DMI during the early compensatory period and a number of studies reported increased ADG of compensating animals during the entire realimentation period. The most variable response was DMI, in that some studies did not report significant differences in DMI between normal and compensating animals. One general observation was that the degree of compensation during realimentation was inversely related to severity of the previous nutrient restriction.

Digestibility. Changes in diet digestibility may be associated with increased DMI of compensating animals. Interestingly, Thomson et al. (1982) demonstrated species differences between lambs and steers in diet digestibility during restriction. Diet DM and gross energy digestibility were numerically greater and crude protein significantly greater in lambs undergoing energy restriction compared with control lambs. Diet DM and gross energy digestibility were numerically lower and crude protein digestibility was not different for steers undergoing energy restriction compared with control steers. During realimentation, when metabolizable energy intake was similar for control and previously restricted lambs or steers, lambs had similar DM, gross energy, and crude protein digestibility. Previously restricted steers consuming similar metabolizable energy had DM and gross energy digestibility lower than control steers but similar crude protein digestibility. Thomson et al. (1982) also reported overall diet digestibility of steers was lower in compensating steers from 300 to 405 kg of BW, thereafter diet digestibility was similar between control and previously energy-restricted steers. A 5% lower total tract DM digestibility, 5.3% lower energy digestibility, and a 4.7% lower crude protein digestibility was reported by Hayden et al. (1993) for previously energy-restricted steers compared with non-restricted steers during d 42 to 45 of the refeeding period. In contrast to the reports of Thomson et al. (1982) and Hayden et al. (1993), Hornick et al. (1998) reported increased nitrogen digestibility for Belgium Blue bulls that were previously restricted in energy and protein intake for 115 d compared with non-restricted steers.

Body Composition. Different rates and patterns of live BW gain caused by restrictions in DMI or restrictions of energy or protein, as previously discussed, also result in important differences in body composition. Baker et al. (1992) reported 43%

greater empty body fat, 15% lower empty body protein, and 36% lower empty body water concentration in steers fed silage ad libitum steers compared with restricted steers. Differences in fat and protein contents resulted in ad libitum steers having 1.7 MJ/ kg EBW more body energy than restricted silage fed steers. When these steers subsequently grazed N-fertilized ryegrass pastures, and restricted steers demonstrated compensatory growth; final fat, protein, and water content of the empty body were similar after the 172 d grazing period. The similar final body composition implies differential accretion of fat and protein between normal and compensating steers. Predictive allometric growth equations of Carstens et al. (1991) showed that hot carcass weight gain occurred faster than gain of EBW in control steers, whereas the rate of hot carcass weight gain was similar to EBW gain in compensating steers. This would imply that compensating steers were increasing non-carcass tissues faster than control steers during the refeeding period. During the refeeding period, accretion rates of protein in the empty body were 28% greater in compensating steers compared with control steers; whereas fat, water, and ash accretion rates were not different. Carcass accretion rates for protein, fat, water, and ash did not differ between treatments. Non-carcass protein and water accretion rates were greater in compensating steers compared with control steers. Fat accretion rate was 24% greater in control steers compared with compensating steers. Therefore, the majority of the increase in rate of protein accretion in the empty body of compensating steers was due to the increase in protein in non-carcass tissues rather than carcass tissues.

Wright and Russel (1991) examined body composition of compensating and normal fed steers at three BW. At 350 kg of EBW, fat content was 3% greater, energy content 1.13 MJ/kg greater, and protein 1% lower in adequately fed steers compared with

steers undergoing compensatory growth. Carcass characteristics were similar with the exception that compensating steers had 11% greater protein than normal steers. Non-carcass protein was similar, but normal steers had 26% greater fat content than compensating steers. At 400 kg, the compensating steers still had lower concentrations of fat and energy and greater concentrations of protein and water than adequately fed steers (Wright and Russel, 1991). When steers reached 450 kg of empty body, carcass fat, protein, and energy contents were similar; non-carcass fat and energy continued to remain greater in adequately fed steers than compensating steers. A similar effect of previous restriction on compensating steer fat and protein mass was observed by Rompala et al. (1985). Compensating steers had greater empty body fat free mass and lower fat mass from 250 to 400 kg of EBW; whereas at 450 and 500 kg EBW, fat free mass and fat mass were similar between compensating and normal steers. The rates of protein deposition were greater and fat deposition lower in the empty body of compensating steers compared with normal steers and inversely related from 200 to 300 kg of EBW (Rompala et al., 1991). Hayden et al. (1993) repoted an interesting contrast to other studies that had restricted energy intake to elicit compensatory growth. After the restriction period, energy-restricted steers had 14% less empty body protein and 22% less empty body fat. During the 88-d refeeding period empty body protein and fat gain were similar between compensating and normal steers, but initial differences in protein and fat content remained.

In work reported by Drouillard et al. (1991b), using energy or protein restricted lambs for 35 d, protein and water content remained unchanged, and fat content of the empty body increased by 20 g/d compared with unrestricted lambs. Protein-restricted

lambs decreased empty body protein and water mass and deposited 30 g/d of fat during the restriction period. During the first two weeks of the refeeding period, protein-restricted lambs deposited greater amounts of protein and fat in carcass and non-carcass tissues compared with energy-restricted lambs. After the initial two weeks of the refeeding period, composition of empty body gain of energy and protein restricted lambs was similar. Sainz et al. (1995) also conducted an experiment that examined energy or protein restriction. Sainz et al. (1995) reported increased fat mass at 327 kg of BW (back fat, kidney-pelvic-heart, marbling, abdominal, and total empty body) in ad libitum concentrate fed steers compared with energy-restricted limit-fed concentrate steers or ad libitum roughage-restricted steers. Empty body protein was greater in the restricted steers compared with the ad libitum-fed steers. At the final EBW, after ad libitum intake of the high-concentrate diet, all treatments had similar carcass fat and protein measurements.

In general, previous restriction of either DM, energy, or protein decreased fat content while increasing protein content in the empty body compared with non-restricted animals. However, protein restricted animals tend to deposit more fat than lean tissue because of a lack of anabolic precursors. Accretion rates of fat or protein during the refeeding period are generally inversely related to accretion during the restriction period. Compensating animals continue to have similar protein accretion rates as control animals, but fat accretion rates tend to be higher. Differences in the accretion rate of protein especially are evident between carcass and non-carcass tissues. The differential rates of accretion coupled with the differences in initial body composition before the refeeding

period often results in similar final empty body composition between normal and compensating steers when fed to similar final BW.

Effect of Previous Nutrition on Splanchnic Organ Mass and Energy Use

Nutrient Restriction

Aside from the effects that any type of nutrient restriction might have on growth performance, effects of nutrient restriction on splanchnic organ mass and energy use may be part of the mechanism of compensatory growth. The gastrointestinal tract (GIT) of the ruminant makes up a relatively small proportion of BW (6 - 10%, Burrin et al., 1990). In contrast, the GIT can account for up to 16 to 28% of whole body oxygen consumption, and the liver can account for 12 to 24% of whole body oxygen consumption (McBride and Kelly, 1990). Together the GIT and liver, which make up less than 15% of BW, can consume as much as 40% of the energy required for maintenance.

Reticulo-rumen. Changes in the mass of the reticulo-rumen as a result of nutrition can have large effects on subsequent performance because alterations in reticulo-rumen size can affect DMI. Kouakou et al. (1997) reported an increase in mass of the reticulo-rumen in response to forage maturity in wether lambs consuming bermudagrass or orchardgrass hay ad libitum. McLeod and Baldwin (2000) divided the rumen and reticulum in lambs fed at two different levels of intake of diets that were 75% forage or 75% concentrate. Mass of the rumen (% of EBW) was greater in steers consuming feed at the high level of intake compared with steers at the low level of intake. Mass of the reticulum (% of EBW) was greater in lambs from the high forage diet and greater for high intake lambs. However, Sun et al. (1994) and Kouakou et al. (1997) did not observe

any difference in reticulo-rumen mass in lambs consuming different forages with different supplementation levels.

Overall DMI has been reported to affect reticulo-rumen mass. A decrease in DMI to 85% of ad libitum reduced reticulo-rumen mass by 3% in lambs (Fluharty and McClure et al., 1997). Ferrell et al. (1986) reported a 26 and 41% decrease in stomach mass of lambs fed the same diet but limited in intake so that lambs gained 0.38, 0.12 or – 0.14 kg/d. Similarly Burrin et al. (1990) observed a 29% increase in stomach mass in ad libitum-fed lambs compared with maintenance-fed lambs, and a 13% decrease in stomach mass of lambs fed at maintenance compared to ad libitum fed lambs across a 21 d feeding period. Nozière et al. (1999) also observed a 38% decrease in reticulo-rumen mass of ewes underfed energy and protein for 78 d compared with ewes fed at maintenance. However, Rompala and Hoagland (1987) reported no difference in mass of stomach complex between lambs fed ad libitum or 50% ad libitum for 21 d. Restrictions of energy and protein intake have been reported to decrease the absolute weight of the stomach complex in lambs (Drouillard et al., 1991b), but increase stomach mass (% of EBW) (Drouillard et al., 1991b and Wester et al., 1995). Restriction of energy intake in cattle did not affect stomach mass, however restriction of energy by feeding a high-roughage diet did increase stomach mass compared to concentrate fed steers (Sainz and Bentley, 1997).

Concentrations of nucleic acids and protein are indicative of cell number and cellular activity. McLeod and Baldwin (2000) reported increased cell number (DNA content), increased protein synthetic capacity or cell activity through increases in (RNA content). However, cell size (protein:DNA) in the ruminal epithelium of lambs on high

levels (2X maintenance) vs. low levels (maintenance) of intake was not different regardless of diet type (75:25 roughage:concentrate and vise versa). No difference in ruminal mucosa DNA, RNA or protein:DNA were observed between underfed (41 and 47% of energy and protein requirement) and maintenance-fed ewes (Nozière et al., 1999) or ad libitum and maintenance fed lambs (Burrin et al., 1992). This could be because the difference between levels of intake in the experiment of Nozière al. (1999) was insufficient to stimulate a cellular response in the ewes. In the experiment of Burrin et al. (1992) using lambs, a response was evident (ad libitum > maintenance) in all measurements when expressed on an EBW basis.

Growing steers fed 75% concentrate diets had lower DNA content compared with steers fed 96% roughage (Sainz and Bentley, 1997). In those same steers RNA content was greater in steers fed ad libitum (concentrate or roughage) compared with limit-fed steers. Protein:DNA was greater in steers fed concentrate ad libitum compared with limit fed or roughage fed steers.

Reticulo-rumen mass is sensitive to both diet type and intake level. Increasing dietary roughage or level of intake increased reticulo-rumen mass in several studies. Reticulo-rumen mass (% of EBW) was also increased in lambs and steers that were energy or protein intake restricted. The increase in mass (% of EBW) would also be a function of decreased EBW in nutrient restricted lambs or steers compared to control lambs or steers. Cellularity of the reticulo-rumen (cell number, cell activity, and cell size) increased in animals on higher levels of intake. Presumably ruminants on higher planes of nutrition have increased quantities of fermentation products to absorb and

metabolize in the rumen. Concentrate diets also stimulate cell activity but not cell number compared with roughage diets.

Intestines. Varied responses of the small intestine to diet type and intake have been reported. Maturity of grass hay has been reported to increase small intestinal mass in lambs (Kouakou et al., 1997b), as has type of forage (alfalfa > bermudagrass; Kouakou et al., 1997a). McCleod and Baldwin (2000) reported increased small intestine mass (% EBW) with increased forage inclusion in the diet and increased level of intake in high forage and high-concentrate diets. Ferrell et al. (1986) reported linear decreases in small intestine mass from high to low levels of intake. Burrin et al. (1990) observed a linear increase in small intestine mass of ad libitum fed lambs and a linear decrease of intestinal mass in maintenance-fed lambs during a 21-d feeding period. Nozière et al. (1999) reported a 23% decrease in small intestine wet mass in ewes that were underfed energy and protein compared with ewes fed at maintenance. In energy and protein restricted lambs, (Drouillard et al., 1991b) observed decreased small intestine (% of EBW) after a 35-d restriction period. Sainz and Bentley (1997) observed similar small intestine mass among steers fed concentrate ad libitum or in limited amounts; however, ad libitum roughage fed steers had greater small intestine mass than concentrate fed steers. Contrary to the preceding reports, Rompala and Hoagland (1986), Wester et al. (1995), and Fluharty and McClure (1997) did not observe any effect of intake, energy or protein restriction, or protein level on small intestinal mass.

Variable effects of intake on cellularity of the small intestine have been observed. Nozière et al. (1999) using jejunum and McLeod and Baldwin (2000) analyzing duodenum, jejunal, and ileal tissue observed no difference in DNA, RNA, or

protein:DNA in sheep underfed (41 and 47% of energy and protein, respectively) or maintenance fed and sheep with low or high intakes of predominately roughage or predominately concentrate diets. Ten-fold differences in DNA, RNA and protein:DNA concentrations reported by Nozière et al. (1999) and McLeod and Baldwin (2000) demonstrates the large amount of variation that exists between reported values. Burrin et al. (1992) reported an increase in duodenal DNA concentration between ad libitum and maintenance-fed lambs after a 21-d feeding period. Concentrations of RNA exhibited an intake level x day interaction; RNA concentration increased in ad libitum-fed lambs and decreased in maintenance-fed lambs during the feeding period. Protein concentration was similar between treatments, however when protein was expressed as mg/kg of EBW, ad libitum lambs had greater protein concentrations compared with maintenance-fed lambs. Small intestinal cellularity was affected by intake level and diet type (Sainz and Bentley, 1997). Limit-fed steers had 34% greater small intestinal DNA concentration compared with ad libitum-fed steers consuming high concentrate or high-roughage diets; whereas, roughage-fed steers had 34% lower RNA concentration compared with concentrate-fed steers regardless of level of intake.

The small intestine has a variable response to differences in diet and nutrition. However, some generalizations can be made. Increasing bulk of diets through differences in forages or inclusion of roughage increased small intestinal mass. Additionally, increased DMI of any diet increased small intestinal mass. Restriction of energy or protein intake had variable effects in small intestinal mass. When differences in cellularity of the small intestine are evident, dietary intake appears to be the largest driving force. In the work of Burrin et al. (1992) overall feed intake influenced

cellularity, but differences in diet type independent of intake were reported by Sainz and Bentley (1997).

Liver. The liver, which plays a central role in nutrient metabolism is influenced both by inputs from the stomach complex and intestines in ruminants. Kouakou et al. (1997a, b) reported forage type and maturity affected liver mass. High-quality, early-maturity bermudagrass and orchardgrass hay increased liver mass in lambs compared with lambs consuming lower-quality, later-maturity hays. Additionally, alfalfa hay supplemented with cereal grains resulted in greater liver mass in lambs compared with ryegrass-wheat hay with supplemental cereal grain. Lambs fed ryegrass-wheat hay with supplemental cereal grain had greater liver mass than lambs fed bermudagrass hay with grain supplementation (Kouakoa et al., 1997; Sun et al., 1994).

In addition to forage type, diet type has been implicated in affecting liver mass. Sainz and Bently (1997) and McLeod and Baldwin (2000) observed increased liver mass in concentrate-fed steers and sheep, respectively, compared with forage-fed animals. Additionally, animals that had greater intake of concentrate diets or predominately roughage diets had greater liver mass (Rompala et al., 1986; Sainz and Bentley, 1997; McLeod and Baldwin, 2000). Ferrell et al. (1986) demonstrated the relative responsiveness of the liver to intake of nutrients that affected rate of BW gain. Lambs fed to gain 0.38 kg/d had liver masses that were 393 g greater than lambs fed to gain 0.12 kg/d. Lambs fed to lose 0.14 kg/d had liver masses that were 159 g less than low BW gain lambs. Wright and Russel (1991) reported that liver mass in steers previously restricted in DMI was 0.76 kg less than full-fed steers at 350 kg of BW. Similarly, Burrin et al. (1990) demonstrated the response of the liver to maintenance feeding. Liver mass

(g/kg of EBW) was decreased by 5 g/kg of EBW in lambs fed at maintenance for 21 d in comparison with lambs fed ad libitum which exhibited increased liver size of 3.6 g/kg of EBW. However, Nozière et al. (1999) demonstrated that the liver does have a minimum threshold for proportional size. In their experiment, with ewes underfed energy and protein for 78 d, liver mass (g/kg of EBW) was not different from liver mass of maintenance-fed ewes. Drouillard et al. (1991b) and Wester et al. (1995) reported that lambs, which were energy or protein restricted, had decreased liver mass (% of EBW) compared with control lambs fed adequate energy and protein. However, Drouillard et al. (1991b) reported a similar reduction in liver mass (% of EBW) between energy and protein-restricted lambs fed the restriction diet for 35 d; whereas, Wester et al. (1995) reported greater (0.34%) liver mass (% of EBW) in protein restricted lambs compared with energy-restricted lambs fed the restriction diet for 49 d.

Cellularity of the liver is an important response to level of nutrition in previously restricted animals. Nozière et al. (1999) noted a 20% increase in DNA (mg/g of tissue) in ewes that had been underfed for 78 d compared with maintenance-fed ewes. Burrin et al. (1992) reported a similar effect in maintenance-fed lambs compared with ad libitum-fed lambs. During a 21-d feeding period, maintenance-fed lambs linearly increased liver DNA by 2.5 mg/g tissue; whereas, ad libitum-fed lambs had DNA concentrations that were 0.6 mg/g of tissue lower during the feeding period. Sainz and Bentley (1997) did not report any differences in liver DNA content in steers fed concentrate diets at two levels of intake or roughage fed ad libitum. Liver RNA concentration was greater in ad libitum concentrate-fed steers compared with ad libitum roughage-fed steers, that had greater RNA concentration than limited concentrate-fed steers (Sainz and Bentley, 1997).

Nozière et al. (1999) and Burrin et al. (1992) reported values that were not different between underfed and maintenance-fed ewes and maintenance and ad libitum-fed lambs, respectively. Protein:DNA was increased by level of intake (ad libitum > maintenance and ad libitum > limit-fed) and diet type (concentrate > roughage) in studies of Burrin et al. (1992) and Sainz and Bentley (1997).

The liver can be characterized as being responsive to nutrient intake. Liver mass was increased in animals fed concentrate diets and increased level of intake of concentrate diets. Restriction of energy or protein intake decreases liver mass compared with livers of control animals. However, the liver of intake-restricted animals does have a greater number of cells compared with control animals. The RNA content is increased by concentrate diets but is not particularly sensitive to level of intake. Whereas, the size of hepatocytes (protein:DNA) is increased with concentrate diets and greater levels of intake.

Realimentation After Nutrient Restriction

Changes in GIT and liver mass and cellularity during realimentation can significantly affect growth performance of animals. Previous reductions in GIT and liver mass that decreased maintenance energy expenditure during the restriction period can carry over into the realimentation period. The carry-over effect may be one of the mechanisms responsible for compensatory growth in previously restricted animals.

Reticulo-rumen. Rompala and Hoagland (1987) reported that increased intake of lambs from 21 d of 50% of ad libitum intake to ad libitum intake resulted in stomach complex mass that was not different from the stomach mass of lambs continually fed ad

libitum intakes after only 5 d. Ferrell et al. (1986) demonstrated the effect that BW gain and implicit nutrient intake had on stomach mass in lambs. Previously restricted lambs had 7 to 10% greater stomach mass compared with lambs that had been fed to gain greater BW. Overfeeding (236 and 271% of requirements of energy and nitrogen, respectively) of previously underfed or maintenance fed ewes resulted in similar reticulo-rumen wet mass after 26 d of feeding (Nozière et al., 1999). The response to refeeding resulted in greater reticulo-rumen mass (% of EBW) in compensating lambs that had been previously restricted in energy or protein intake compared with control lambs (2.57 vs. 1.91% of EBW, respectively) after only 14 d of refeeding (Wester et al., 1995). However, compensating lambs that had been energy or protein restricted in the experiment of Drouillard et al. (1991b) had lower stomach mass (% of EBW) after 14 d of finishing compared with unrestricted lambs, but were not different after 50 d of finishing. The delay in increase of reticulo-rumen mass was attributed to feeding practices that limited intake during the first 14 d to avoid acidosis concerns in the compensating lambs. In the work by Sainz and Bentley (1997), limit-fed and roughage-fed steers were fed a concentrate diet ad libitum from 327 to 481 kg EBW. Steers limit-fed for 112 d and then realimentated for 89 d had stomach mass that was not different from ad libitum-concentrate fed steers. Steers roughage-fed for 112 d and then realimented for 111 d had stomach mass that was 9% lower than steers previously fed concentrate.

After finishing, concentration of DNA was greater in steers that had ad libitum access to either concentrate or roughage compared with steers that were limit fed the concentrate diet during the 112 d growing period. Concentrations of RNA and

protein:DNA were greater in 112 d limit fed steers compared with steers that had been fed ad libitum during the growing period. Nozière et al. (1999) reported DNA and RNA concentrations, and protein:DNA that were not different between underfed or maintenance-fed ewes that were subsequently overfed. The similarity in cellularity of ewes could be attributable to massive overfeeding in both treatments, which would have abolished any previous differences.

Intestines. Similar to the stomach mass, intestinal mass was not different between lambs offered feed ad libitum vs. 50% ad libitum after 5 d of ad libitum feeding (Rompala and Hoagland, 1987). Ferrell et al. (1986) reported a 23 and 26% increase in small intestine mass in lambs fed to gain 0.64 or 0.38 kg/d, respectively, after a previous 42-d restriction period in which the lambs lost 0.14 kg/d. Small intestinal mass (% of EBW) in previously protein-restricted, compensating lambs was greater than control or previously energy-restricted, compensating lambs after 14 d of refeeding (Wester et al., 1995). In contrast, previously restricted, compensating lambs small intestinal mass (% of EBW) remained less than control lambs after 14 d of finishing (Drouillard et al., 1991b); however, after 50 d of finishing small intestinal mass (% of EBW) was not different among treatments. Steers that had been restricted by feed intake or by consuming roughage for 112 d, had 9% greater intestinal mass compared with ad libitum concentrate-fed steers (Sainz and Bentley, 1997). The high level of overfeeding used by Nozière et al. (1999), probably resulted in no observed differences in small intestine mass between previously underfed and maintenance-fed ewes.

Cellularity response of small intestine in previously restricted animals is limited. Nozière et al. (1999) reported no differences in DNA, RNA, or protein:DNA in

previously underfed ewes compared with maintenance-fed ewes when both treatments were overfed for 26 d. Small intestine from steers (Sainz and Bentley, 1997) had similar RNA concentrations and protein:DNA ratios between previously limit fed or roughage-fed steers compared with ad libitum concentrate-fed steers. Concentrations of DNA were greater in steers that had been fed roughage diets previous to the finishing period; however, DNA concentrations were not different between previously limit-fed and ad libitum concentrate-fed steers (Sainz and Bentley, 1997).

Similar to the reticulo-rumen, the response of the small intestine after realimentation was variable. In general small intestinal mass was increased or similar compared with non-restricted control animals in energy, protein, or intake restricted animals.

Liver. The liver is very responsive to the level of nutrition. Rompala and Hoagland (1987) reported that greater than 5 d was required for liver mass of lambs restricted to 50% of ad libitum intake for 21 d to reach liver mass of ad libitum fed lambs (431 vs. 550 g). However, Wright and Russel (1991) reported that liver mass in steers could be equalized within a 100 kg of BW gain period. Ferrell et al. (1986) demonstrated the importance of ADG before harvest on liver mass. All lambs had similar BW gain over a 84 d period, however lambs that had ADG of 0.64 and 0.38 kg/d during the last 42 d had increased liver mass compared with lambs that had ADG of 0.12 kg/d during the last 42 d. The effect of similar level of intake following a 98 d period of restriction on liver mass was demonstrated by Nozière et al. (1999), where ewes that were overfed energy and protein had similar liver mass after 26 d regardless of the prior 152 g difference in liver mass. Previously energy or protein-restricted lambs (Drouillard et al.,

1991b and Wester et al., 1995) had liver mass (% of EBW) that was not different by d 50 and 14, respectively. Sainz and Bentley (1997) reported 10% increase in liver mass after steers that had been limit fed or roughage fed for 112 d were finished.

Nozière et al. (1999) reported no difference in liver DNA, RNA, or protein:DNA of overfed ewes. Previously limit-fed or roughage-fed, compensating steers had DNA concentrations that were not different after finishing and greater RNA concentrations compared with previously ad libitum concentrate-fed steers. Previously limit fed steers had greater protein:DNA compared with previously ad libitum-fed steers after the finishing period.

The liver is very responsive to level of intake that affects ADG. The liver mass increases to a point dictated by the level of realimentation, independent of previous nutrition, provided adequate time for growth is allowed. The liver of previously restricted steers increased in activity (RNA content) most likely in response to the increased nutrient intake and metabolic demands for body growth. However, the response in compensating animals did not included increases in liver cell number (DNA content) or size (protein:DNA).

Oxygen Consumption

Concurrent with changes in visceral organ mass and cellularity associated with intake and level of nutrition are changes in energy use. Energy use by organs can be estimated by in vitro tissue oxygen consumption. Because different diets have been used to affect nutrient restrictions, this discussion will initially examine differences in effects of diet type on oxygen consumption.

Ruminal papillae are often used to estimate oxygen consumption by ruminal tissues. Studies of diets with greater protein content (alfalfa vs. bromegrass hay; Kelly et al., 1993) or greater metabolizable energy (timothy hay with soybean meal, and corn vs. timothy hay with soybean meal; Kelly et al., 1995) resulted in greater oxygen consumption by ruminal papillae. Harmon et al. (1991) examined the effect of level of intake (maintenance or 2 x maintenance) using 90% forage vs. 90% concentrate diets. Oxygen consumption by ruminal papillae was not effected by diet type, however increasing intake resulted in 27 and 12% increases in oxygen consumption by ruminal papillae from steers fed the forage and concentrate diets, respectively. In contrast, 75% forage diets vs. 75% concentrate diets, or high vs. low intake of diets did not result in any difference in oxygen consumption by ruminal or small intestinal tissues (McLeod and Baldwin, 2000). In the diets of McLeod and Baldwin (2000), gross energy and crude protein:metabolizable energy ratio were similar between diet types suggesting that the oxidative capacity of tissues was not influenced by diet. Burrin et al. (1990) reported similar ruminal epithelium oxygen consumption between ad libitum and maintenance fed lambs during a 21 d feeding period.

Small intestinal oxygen consumption rate has been reported to be affected by level of intake (McBride and Milligan, 1985a; Burrin et al., 1990; McLeod and Baldwin, 2000). McBride and Kelly (1985) reported that increasing level of digestible energy intake (0, 7.6, and 14.8 MJ/d) resulted in a linear increase in the percent inhibition by ouabain, a Na^+, K^+ ATPase blocker, of duodenal oxygen consumption. Additionally, duodenal Na^+, K^+ ATPase dependent respiration increased with increasing level of digestible energy intake, whereas Na^+, K^+ ATPase independent respiration decreased.

The results of McBride and Milligan (1985a) suggest differential cellular metabolism in the duodenum that is influenced by level of energy intake. Oxygen consumption by previously energy or protein restricted lambs was not different compared to control lambs after restriction, but increased relative to small intestinal oxygen consumption before the restriction period in control and restricted lambs (Drouillard et al., 1991b; Wester et al., 1995). After refeeding, oxygen consumption by small intestinal tissue was not different between control and previously restricted lambs (Drouillard et al., 1991b; Wester et al., 1995).

Rate of liver oxygen consumption per unit weigh of tissue has been reported not to be different between fed and fasted rats (Burrin et al., 1988) and fed and starved sheep (McBride and Milligan, 1985b). However, in the rat data of Burrin et al. (1988), whole liver oxygen consumption was 41% greater in fed rats compared with fasted rats. Differences in whole liver oxygen consumption were due to greater liver mass in fed rats. McBride and Milligan (1985b) reported increased inhibition of liver oxygen consumption by the Na^+, K^+ ATPase inhibitor ouabain in fed sheep. Increased inhibition of liver oxygen consumption by ouabain implies that livers from fed sheep expend more energy in transport and other activities associated with Na^+, K^+ ATPase. Increases in the proportion of energy attributable to Na^+, K^+ ATPase would also imply a greater maintenance energy requirement by those tissues. Drouillard et al. (1991b) reported decreased liver oxygen consumption rate in energy and protein restricted lambs after the 35-d restriction period. Wester et al. (1995) did not observe a similar reduction in liver oxygen consumption rate by energy or protein restricted lambs compared to control lambs; however, whole liver oxygen consumption was decreased in restricted lambs

compared with control lambs. After 14 d of finishing, previously energy or protein restricted lambs continued to have 15% lower liver oxygen consumption rates compared with unrestricted lambs (Drouillard et al., 1991b). Wester et al. (1995) reported previously energy or protein-restricted lambs had liver oxygen consumption rate and whole liver oxygen consumption that was not different from unrestricted lambs after 14 d of refeeding.

Increases in dietary crude protein and metabolizable energy increased ruminal papillae oxygen consumption. However, differences in diet type (75% concentrate vs. 75% roughage) were also reported not to affect ruminal papillae oxygen consumption. Additionally, conflicting results are reported for ruminal papillae oxygen consumption in response to increases in intake level. Intake level or energy or protein restriction does not affect small intestinal oxygen consumption. However, differences in intake do cause differences in cellular metabolism that required oxygen. Like the small intestine, liver oxygen consumption was not affected by level of intake. However, differences in intake level did elicit differences in cellular metabolism. Energy and protein restriction decreased the rate and whole organ oxygen consumption in lambs. However, when energy and protein restricted lambs were realimented, oxygen consumption compared to control lambs was variable between studies.

Effect of Previous Nutrition on Blood Metabolites and Endocrine Hormones

Metabolites

Glucose. Concentration of plasma glucose is very responsive to level of feed intake. Plasma glucose concentration of DMI-restricted steers decreased as the length of

the restriction period increased (Blum et al., 1985; Ellenberger et al., 1989; Yambayamba et al., 1996; Hornick et al., 1998). In steers that were subjected to long-term restriction, plasma glucose concentration reached minimal values after 200 d in steers fed to gain 0.13 kg/d (Blum et al., 1985) and 300 d in Belgian Blue bulls fed to gain 0.5 kg/d (Hornick et al., 1998). The majority of glucose production in ruminants comes from hepatic gluconeogenesis (Brockman and Laarveld, 1986); therefore glucose concentration is reflective of the availability of gluconeogenic precursors, and thus DMI. When previously restricted steers were realimented, plasma glucose concentrations increased rapidly. Blum et al. (1985) reported plasma glucose concentrations greater than pre-restriction levels 2 d after the initiation of refeeding. Concentrations of plasma glucose in steers restricted in intake were not different than non-restricted steers by d 50 (Ellenberger et al., 1989), 30 (Hayden et al., 1993), and 10 (Yambayamba et al., 1996). The increase in glucose concentration in compensating animals corresponded with increases in DMI and ADG (Blum et al., 1985; Ellenberger et al., 1989; Yambayamba et al., 1996).

Nonesterified fatty acids. Nonesterified fatty acids (NEFA) concentrations are indicative of the mobilization of fat as an energy source in animals during periods of low energy intake (Yambayamba et al., 1996). Concurrent with the reduction in glucose concentration during restriction is an increase in NEFA concentration. Nonesterified fatty acids are the result of oxidation in adipose tissue to provide NEFA and glycerol for hepatic metabolism (Brockman and Laarveld, 1986). Concentrations of NEFA in feed restricted heifers and steers were greater than ad libitum fed controls by d 20 and 15 of restriction (Yambayamba et al., 1996 and Blum et al., 1986, respectively). Increasing the

length of time of restriction increased NEFA concentrations in steers (Blum et al., 1986) and heifers (Yambayamba et al., 1996). However, feed restriction that resulted in 0.35 kg/d gain in steers (Ellenberger et al., 1989) did not elicit any difference in NEFA concentrations during the restriction period. Concentrations of NEFA were decreased by 30% in the early refeeding period and 60% in the late refeeding period in previously restricted compensating steers compared with control (normal growth) steers (Ellenberger et al., 1989). Rapid deceases in NEFA concentrations in compensating steers have been reported. Similar concentrations of NEFA were measured between normal and compensating steers and heifers by d 25 and 10 of the refeeding period (Hayden et al., 1993 and Yambayamba et al., 1996, respectively). Blum et al. (1985) reported NEFA concentrations lower than pre-restriction values on d 1 after initiation of the refeeding period.

Urea Nitrogen. The concentration of urea-N in blood is often affected by dietary crude protein concentration with variable effects of feed restriction. A decrease in urea-N concentration in intake-restricted animals did not occur until d 48 of the restriction period in heifers fed to achieve zero BW gain (Yambayamba et al., 1996) and d 140 of the restriction period in steers fed to gain 0.13 kg/d (Blum et al., 1985). The variability of the urea-N response is evident in the study of Hayden et al. (1993) in which restricted steers that had 0.53 kg/d EBW gain had greater urea-N concentrations than non-restricted steers at the end of the restriction period. During realimentation, previously restricted, compensating steers had declining urea-N concentrations up to d 30, which were lower than non-restricted steers and continued to be lower on d 60 of refeeding (Hayden et al., 1993). In contrast, urea-N concentrations during the realimentation period were not

different between compensating animals and normally fed control animals (Blum et al., 1985; Ellenberger et al., 1989; Yambayamba et al., 1996). Hayden et al. (1993) reported a correlation between deceased urea-N concentrations and increased empty body protein accretion. Other reports of compensating animals demonstrate increased BW gains with increasing urea-N concentration (Ellenberger et al., 1989; Yambayamba et al., 1996; Lammers et al., 1999). Blum et al. (1985) examined the effect of restriction on α-amino N (AAN) and albumin. Concentrations of AAN were lower in energy-restricted steers beginning shortly after the initiation of the restriction period. Concentrations of AAN were not different between compensating and control steers within 20 d after the initiation of the refeeding period. Concentrations of AAN in compensating steers were not different from pre-restriction concentrations by d 2 of the refeeding period. A similar rapid resumption of basal values by d 2 was observed for albumin concentration in compensating steers (Blum et al., 1985).

These data suggest that in compensating cattle, blood metabolite concentrations are dependent upon intake. Concentrations of glucose, urea-N, and AAN decrease and NEFA increase with restriction of intake and implicitly nutrients. However, when cattle are realimented metabolite concentrations respond in a fairly rapid time frame. The rapid response of circulating metabolites provides compensating cattle with adequate nutrients for deposition of tissue and subsequent BW gain.

Endocrine Hormones

Insulin. Plasma concentrations of insulin are positively related to DM or energy intake (Brockman and Laarveld, 1986). Barash et al. (1998) demonstrated a dose-

dependent response of insulin concentration to metabolizable energy (ME) intake after 23 d of reduced energy feeding in steers. At the end of the 77-d restriction period, steers fed diets with 2.43 Mcal of ME/kg and 11.7% crude protein had lower insulin concentrations than steers fed diets with 2.61 Mcal of ME/kg and 12.8% crude protein. Blum et al. (1985) and Yambayamba et al. (1996) reported similar decreases in plasma insulin concentration by d 20 of restriction in energy-restricted steers and heifers, respectively. In all instances (Blum et al., 1985; Hayden et al., 1993; Yambayamba et al., 1996, Barash et al., 1998), plasma insulin concentration was significantly lower in energy-restricted animals compared with normally-fed control animals regardless of the length of the restriction period (48 to 120 d). During the restriction period lower plasma insulin concentration allows energy-restricted animals to increase hepatic gluconeogenesis, lipolysis, and ketogenesis. The up-regulation of these mechanisms is a directed response to the reduction in DM or energy intake (Brockman and Laarveld, 1986).

When previously energy-restricted animals are refed high-energy diets, plasma insulin concentrations rapidly increased to concentrations similar to control animals (Blum et al., 1985; Hayden et al., 1993; Yambayamba et al., 1996, Barash et al., 1998). Blum et al. (1985) reported increases in plasma insulin concentration above pre-restriction basal levels by d 1 of refeeding. The increased plasma insulin effect of refeeding high-energy diets has been implicated in the initiation of the compensatory growth mechanism (Blum et al., 1985). The initiation of compensatory growth in previously energy-restricted animals could be a result of the anabolic stimulation provided by insulin (Brockman and Laarveld, 1986). Additionally, Eisemann et al. (1997) reported that sensitivity of peripheral tissue to insulin was decreased by age, BW,

and percent empty body fat. Compensating animals generally have decreased BW and empty body fat content; and, therefore would retain a greater peripheral and hepatic sensitivity to the anabolic effects of insulin.

Insulin Like Growth Factor I. Breier et al. (1988a) suggested that regulation of circulating plasma insulin-like growth factor I (IGF-I) may be mediated by hepatic high-affinity growth hormone (GH) receptors that are subject to nutritional manipulation. The mediation of circulating IGF-I concentrations through hepatic receptors is vital because the liver is the largest source of circulating IGF-I (Le Roith et al., 2001). Most tissues produce IGF-I, and therefore the local autocrine or paracrine effects of IGF-I could be of equal importance in stimulating growth as measurable-circulating IGF-I (Le Roith et al., 2001). Restriction of either energy and (or) protein has been reported to lower circulating IGF-I concentrations by d 20 of energy restriction in cattle (Barash et al., 1998; Ellenberger et al., 1989; Yambayamba et al., 1996) and by d 7 and 14 of protein and energy-restricted lambs (Wester et al., 1995). Thissen et al. (1990) reported decreased IGF-I concentrations in protein-restricted rats through GH dependent post-receptor events. Growth hormone and IGF-I act synergistically (Hua et al., 1993; Le Roith et al., 2001). Un-coupling of the GH-IGF-I axis has been demonstrated by Breier et al. (1988b), who reported steers receiving 3% of their BW in feed responded to boluses of GH by increasing IGF-I concentration compared with steers receiving 1% of their BW in feed, and Hua et al. (1993), who reported that fed sheep increased tissue IGF-I concentration in response to GH administration whereas starved sheep did not respond to GH.

When previously restricted animals are realimented, IGF-I concentrations quickly increase. Breier et al. (1986) and Yambayamba et al. (1996), using intake-restricted steers and heifers, respectively, reported resumption of IGF-I concentrations similar to ad libitum fed animals after d 10 of refeeding. Hayden et al. (1993) and Barash et al. (1998) reported that previously restricted compensating steers required 30 d to achieve IGF-I concentrations not different from adequate energy and protein intake control steers. Previously energy or protein restricted lambs achieved similar IGF-I concentrations as control lambs after only d 6 of the refeeding period (Wester et al., 1995).

Growth Hormone. Growth hormone concentrations increase during energy restriction (Blum et al., 1985; Ellenberger et al., 1989; Hayden et al., 1993; Yambayamba et al., 1996). The increase in GH concentration in energy-restricted steers required 48 and 60 d of restriction (Yambayamba et al., 1996 and Blum et al., 1985, respectively). Wester et al. (1995) reported no rise in GH concentration in energy-restricted lambs compared to adequate-energy control lambs, however protein-restricted lambs did exhibit an increase in GH concentration seven days after the initiation of the restriction period. It was speculated that the absence of the GH response in energy-restricted lambs was a result of the need to slowly release lipid stores for energy.

Realimentation of animals results in resumption of basal concentrations of GH. However, it can take up to 30 d (Hayden et al., 1993; Yambayamba et al., 1996) for resumption of GH concentrations similar to control animals. Blum et al. (1985) reported daily mean GH concentrations in compensating steers that were less than basal values on d 4 of the refeeding period, whereas Wester et al. (1995) reported resumption of GH concentration that were not different from control steers on d 4 of the refeeding period.

The decline in GH concentrations occurs with concurrent increases in IGF-I concentration. Le Roith et al. (2001) speculated that the need for high circulating IGF-I concentrations produced by the liver may not be for stimulation of growth, but rather to effect feedback on the hypothalamus to regulate GH secretion and modulate the metabolic effects elicited by GH.

Thyroid Hormones. The thyroid hormones, while not directly anabolic, do function with insulin to stimulate anabolic growth through protein synthesis and glucose utilization (Griffin and Ojeda, 1992). It has also been suggested that thyroid hormones function with IGF-I to stimulate bone and cartilage growth (Ellenberger et al., 1989). Additionally, Hayden et al. (1993) implicated triiodothryronine (T_3) as a metabolic signal indicative of energy status in animals and thyroxine (T_4) as a metabolic signal indicative of energy consumption.

Restriction of energy and protein intake that resulted in decreased BW gains reduced T_4 concentrations in the restricted animals compared with adequately-fed control animals (Blum et al., 1985; Ellenberger et al., 1989; Hayden et al., 1993; Wester et al., 1995; Yambayamba et al., 1996; Barash et al., 1998). However, the temporal responsiveness of T_4 varies. Blum et al. (1985) observed almost immediate reduction in T_4 concentration in steers fed to gain 0.13 kg/d. Wester et al. (1995) observed reduced T_4 concentration in lambs that were energy or protein restricted that gained 0.03 kg/d within 7 d of the initiation of the restriction. Barash et al. (1998) observed reduction of T_4 concentrations within 20 d of initiation of restriction in steers fed to gain 0.66 to 1.2 kg/d. Yambyamba et al. (1996) did not observe a decrease in T_4 concentration of heifers gaining 0.7 kg/d relative to ad libitum-fed heifers until d 48 of restriction. Concentrations

of T_3, similar to T_4, decreased in energy or protein restricted animals (Blum et al., 1985; Hayden et al., 1993; Wester et al., 1995; Yambayamba et al., 1996). Similarly to T_4, the time course for the decrease in T_3 varied. In steers, T_3 decreased almost immediately when steers were placed on restricted-energy intake compared with control steers (Blum et al., 1985). In heifers fed to gain greater BW than steers of Blum et al. (1995), T_3 tended to decrease by d 20 of restriction, and was significantly decreased by d 48 (Yambyamba et al., 1996). A similar time frame was required by energy or protein restricted lambs (Wester et al., 1995). Concentrations of T_3 in restricted lambs did not differ from control lambs until d 21 of restriction (Wester et al., 1995). Contrary to the preceding findings, Ellenberger et al. (1989) observed no difference in T_3 concentrations between normally fed steers fed to gain 1.29 kg/d and steers fed to gain 0.35 kg/d.

The response of thyroid hormones in compensating animals during the refeeding period is variable in magnitude and timing. Blum et al. (1985) reported greater T_4 concentrations in compensating, previously energy restricted steers during the first 15 d of the refeeding period and lower T_3 concentrations during the first 30 d compared with control steers. Hayden et al. (1993) reported that T_4 concentration in compensating steers remained lower than control steers through d 60 of the refeeding period, but T_3 concentration was not different from control steers after d 30 of the refeeding period. Wester et al. (1995) reported a similar pattern in which T_4 concentration of compensating lambs remained lower than control lambs, but T_3 concentration was not different from control on d 14 of refeeding. Yambayamba et al. (1996) reported similar T_4 and T_3 concentrations between compensating and normal heifers on d 30 of the refeeding period.

Leptin. Leptin is a 16 k-Da protein that is produced and secreted by white adipocytes (Houseknecht et al., 1998; Barb 1999). Since the discovery of leptin in 1994, leptin has been implicated in affecting feed intake, energy expenditure, energy balance, and immune function (Houseknecht et al., 1998; Barb, 1999; Spicer, 2001; Delavaud et al., 2002). In a review of the biology of leptin, Houseknecht et al. (1998) illustrated the regulatory effects of leptin on peripheral tissue metabolism. Leptin has been reported to affect peripheral insulin resistance by decreasing insulin action. Additionally, leptin has been reported to decrease insulin secretion from the pancreas. Leptin production is regulated by triglyceride content in adipose tissue (Barb, 1999). Increasing triglyceride concentrations resulted in increased leptin production (Barb, 1999). Increasing leptin concentrations decreases food intake, increases energy expenditure and increases activity by the animal (Houseknetch et al., 1998; Barb, 1999). Leptin has also been implicated in affecting GH secretion in pigs; infusion of leptin into the brain of normally fed pigs resulted in increased GH secretion (Barb, 1999). Houseknecht et al. (1998) suggested the best description of the role of leptin maybe as a "metabolism modifier".

Relationships between leptin and body fatness, intake and overall nutrition have been examined. Delavaud et al. (2000) reported highly significant correlations between ewe body condition score and leptin ($r = 0.72$) and body lipid content (% of BW) and leptin ($r = 0.68$) prior to imposing different dietary regimes. When ewes were fed 39% of maintenance requirements for 65 d, plasma leptin concentration was decreased by 56% compared with no change in ewes fed 90% of maintenance for the same period. Delavaud et al. (2002) reported that a strong ($r = 0.95$) curvilinear relationship exists between adipose cell volume or size and plasma leptin concentration. This relationship

implies that leptin concentration is related to adipose cell hypertrophy. Additionally, Delavaud et al. (2002) reported postprandial decreases in plasma leptin concentrations in well-fed and refed-previously restricted cows. The decrease in plasma leptin in those cows would indicate that leptin is not associated with satiety in ruminants. Daniel et al. (2002) also observed a high correlation ($r = 0.77$) between leptin concentration and body fat (ultrasound fat thickness) in sheep. Moreover, Daniel et al. (2002) demonstrated the episodic nature of leptin secretion in ewes with continual access to food. Leptin secretion characteristics as measured by area under the curve, peak number, and peak height were greater for fed than fasted ewes and greater for fat than thin ewes. Intervals between peak leptin concentrations were shorter for fed and fat ewes compared with fasted and thin ewes (Daniel et al., 2002).

The response of endocrine hormones during nutrient intake restriction has important implications for growth. Decreases in the anabolic hormones (insulin and IGF-I) in restricted cattle reduce their ability to increase in BW at rates comparable to control cattle. The uncoupling of the IGF-I- GH axis is also critical in the decreased growth response in intake or nutrient restricted cattle. Decreases in thyroid hormones as a result of intake and nutrient restriction might help to lower basal metabolism and conserve energy. Metabolic signal hormones like leptin are also affected by altered nutritional status. Realimentation of cattle increases the concentrations of endocrine hormones that had previously been decreased. However, the response time to realimentation is hormone dependent. It has been suggested that the lowered concentration of circulating hormones during the restriction period is advantageous because it increases the responsiveness of tissues to hormones and likely increases hormone production during realimentation.

Effect of Diet on Blood Flow and Net Flux Through Splanchnic Tissues

The effect of diet on blood flow through the organs of the GIT and across the liver is of great importance. Blood flow from the GIT assimilates absorbed nutrients for delivery to the liver. Metabolism of nutrients by the liver and flux out of the liver dictates the nutrients and their concentrations available for use by peripheral tissues.

Roughage Diets

Blood Flow. Blood flow across the portal-drained viscera (PDV), the liver, and arterial blood flow was not affected by forage type (Patil et al., 1996; Park et al., 1997). Goetsch et al. (1997a) reported that arterial, portal, or hepatic blood flow was not affected by forage type or chop lengths even though DMI was influenced. Additionally, inclusion of corn or alfalfa hay in hay diets increased total DMI but did not affect blood flow (Goetsch et al., 1997b). In contrast, increases of 400 g in daily DMI of alfalfa pellets increased PDV blood flow by 31% in sheep (MacRae et al., 1997) and an increase of 250 g/d of hay increased PDV blood flow by 14% in ewes (Han et al., 2002). Similarly, Reynolds et al. (1991b) reported a near equal increase in blood flow with increased DMI. A 39% increase in DMI resulted in a 46 and 44% increase in portal and hepatic blood flow, respectively (Reynolds et al., 1991b). However, Huntington et al. (1996) reported decreased portal and hepatic blood flow in steers when dietary concentrate was increased from 27 to 63% with a 1 kg difference in DMI between the two diets. Seal et al. (1992) also reported decrease mesenteric and portal blood flow between all forage and 50:50

forage concentrate diets. Ruminal blood flow, which is a portion of PDV blood flow, was increased by 5.2 L/h when nutrient supply was increased by infusion of nutrients into the rumen (Han et al., 2002).

Blood flow responds to changes in DMI and energy content of diet. Differences in forage type, processing, or supplementation did not affect blood flow. However, when the roughage content of the diet was replaced by concentrates blood flow decreased. Apparently differences in fermentation in the rumen and potential post-ruminal digestion can have significant effects on blood flow.

Oxygen Consumption. Oxygen consumption or the energy expenditure by splanchnic tissues constitutes a major portion of the maintenance energy requirements of animals (Crooker et al., 1991). Oxygen consumption by the PDV was similar for lambs consuming alfalfa, ryegrass-wheat, or bermudagrass hay (Park et al., 1997), lambs consuming warm- or cool-season grass hay with or without alfalfa hay (Patil et al., 1996), and lambs consuming ryegrass-wheat hay with supplemental corn or alfalfa (Goetsch et al., 1997b). An increase in PDV oxygen consumption was observed in lambs consuming ground and pelleted bermudagrass or ryegrass-wheat hay (Goetsch et al., 1997a). Han et al. (2002) reported increased oxygen consumption by the PDV of ewes consuming hay with additional infused urea or casein, and a linear increase in PDV oxygen consumption with increasing dietary bulk. Ruminal oxygen consumption was not affected by ruminal nutrient infusion or dietary bulk. Because metabolism of the splanchnic tissues (PDV and liver) is driven by DMI, they make up a substantial proportion of whole body oxygen consumption (Reynolds 2001). Increasing DMI of a 75% alfalfa diet increased splanchnic tissue oxygen consumption, and accounted for 72% of the increase in whole

body oxygen consumption (Reynolds et al., 1991a). Splanchnic tissues oxygen consumption was similar for lambs consuming ryegrass-wheat hay alone or supplemented either with corn and/or alfalfa hay (Goetsch et al., 1997b). The inclusion of 20% alfalfa hay in the diet of lambs consuming either warm- or cool-season hay increased splanchnic tissue oxygen consumption by 16%. Grinding and pelleting alfalfa hay increased splanchnic tissue oxygen consumption by 17% in lambs consuming either bermudagrass or ryegrass-wheat hay (Goetsch et al., 1997a).

Oxygen consumption by the PDV and splanchnic tissues generally increased with increasing DMI. However, increased DMI was not solely responsible for increased oxygen consumption by the splanchnic tissues because increased dietary bulk also increased oxygen consumption. Increasing the amount of nutrients available for digestion, absorption, and metabolism increases the workload of the splanchnic tissues, and thereby increases the energy consumption by those tissues.

Flux of Nitrogenous Metabolites. Release of ammonia-N by the PDV was increased by the inclusion of alfalfa in ryegrass-wheat hay diets and warm-season grass hay diets (Goetsch et al., 1997b; Patil et al., 1996, respectively). Inclusion of alfalfa in the diet provided additional ruminally fermentable nitrogen that was absorbed across the rumen. Increasing DMI of a 75% alfalfa diet by 39% increased ammonia-N release by 45% from the PDV of heifers (Reynolds et al., 1991b). Grinding and pelleting grass hays resulted in a decreased release of ammonia-N from the PDV (Goetsch et al., 1997a). Infusion of 8.5 g/d of urea and 33 g/d of casein into the rumen increased PDV ammonia-N flux by 10.39 mmol/h in ewes compared with ewes only infused with urea (Han et al., 2002). In contrast, Huntington et al. (1996) reported no difference in PDV release of

ammonia-N in steers consuming 73 or 47% roughage diets. Release of ammonia-N by the PDV was offset by a hepatic removal that resulted in a net utilization of ammonia-N by the splanchnic tissues (Patil et al., 1996; Goetsch et al., 1997a; Goetsch et al., 1997b). Uptake ammonia-N by the splanchnic tissues increased with increasing DMI (Reynolds et al., 1991b).

Unlike ammonia-N PDV flux, urea-N flux is generally removal and not affected by forage type (Goetsch et al., 1997a; Park et al., 1997), form of the diet (Goetsch et al., 1997a), or addition of corn or alfalfa to ryegrass-wheat hay diets (Goetsch et al., 1997b). In contrast, Huntington et al. (1996) reported decreased urea-N uptake by the PDV when concentrate level was increased. However the liver releases urea-N and this results in a positive flux of hepatic urea-N (Patil et al., 1996; Goetsch et al., 1997a; Goetsch et al., 1997b). Alfalfa hay or the inclusion of alfalfa in ryegrass-wheat hay increased hepatic urea-N flux (Park et al., 1997, Goetsch et al., 1997b, respectively). Increased liver urea-N release was reported to account for 16% of the increase in oxygen consumption by the liver with increased intake of 75% alfalfa diets (Reynolds et al., 1991a). Release of urea-N by the liver, which is greater than the removal by the PDV, resulted in the splanchnic release of urea-N (Patil et al., 1996; Goetsch et al., 1997a; Goetsch et al., 1997b). Release of urea-N from splanchnic tissues would supply N for recycling (Huntington et al., 1996).

Release of AAN from PDV was 56% greater in alfalfa hay diets compared with ryegrass-wheat or bermudagrass hay diets (Park et al., 1997). Ryegrass-wheat hay had 11% greater AAN PDV flux than bermudagrass hay diets (Goetsch et al., 1997a). Inclusion of alfalfa into warm- or cool-season grass hay diets increased AAN PDV flux

compared to grass hay diets only (Patil et al., 1996). However, the inclusion of corn or alfalfa in ryegrass-wheat hay diets resulted in similar AAN PDV flux in wethers. Similarly, Reynolds et al. (1991b) and Huntington et al. (1996) reported no differences in PDV AAN flux in cattle consuming diets that differed in forage:concentrate ratio. Interestingly, increasing diet bulk by feeding increasing amounts if hay coupled with infusion of urea and casein resulted in a linear increase of amino acid PDV flux in ewes (-3.03, 6.45, 12.21 mmol/h; low, medium, high bulk, respectively; Han et al., 2002). Amino acid PDV flux exhibited a linear increase in amino acid release in response to increasing diet bulk (Han et al., 2002). Hepatic uptake of AAN could be used as possible gluconeogenic precursors or urea cycle intermediates (Reynolds et al., 1991).

Flux of nitrogenous nutrients across the PDV appears to be primarily driven by crude protein content of the diet. Increasing crude protein in the diet by increasing intake or supplementation of high protein sources increased PDV release of ammonia-N and AAN. The source of the increase in ammonia-N is likely the rumen when ruminal digestible protein is supplied. Likewise the increase in AAN might be from increased microbial flow to the small intestine or increased flow of ruminal undegradable protein. The carbon skeletons provided by the sources of AAN can be utilized as gluconeogenic precursors. Urea-N uptake by the PDV is primarily a result of recycling urea N to the rumen. However, the increased release of urea-N from the liver for recycling is a source of energy expenditure incurred by the splanchnic tissues. Adequate ruminal degradable protein in the diet would reduce urea-N recycling to the rumen and decrease energy expenditure by the liver.

Flux of Energy Yielding Nutrients. Little starch reaches the lower GIT for digestion and absorption because of ruminal fermentation. What starch is digested in the lower GIT is utilized by the tissues of the GIT. Therefore, the PDV is generally a net utilizer of glucose, and removal of glucose across the PDV occurs in roughage-based diets (Patil et al., 1996; Goetsch et al., 1997a; Han et al., 2002). In the preceding reports, PDV glucose flux was not affected by grass hay type or the inclusion of alfalfa hay, level of bulk in the diet, or ruminal infusion of urea and casein. In contrast, Huntington et al. (1996) reported decreased glucose uptake by PDV when concentrate level was increased from 27 to 63%. Reynolds et al. (1991b) reported that increasing DMI of an alfalfa diet increased PDV uptake of glucose. Because PDV glucose flux is negative, hepatic glucose flux and the subsequent splanchnic tissue flux must be positive to maintain adequate glucose concentrations for metabolic needs of peripheral tissue. In the studies of Patil et al. (1996) and Goetsch et al. (1997a), which utilized animals that had similar requirements, hepatic and splanchnic glucose flux were similar regardless of diet type, supplementation, or diet form. However, Reynolds et al. (1991b) reported a 55% increase in hepatic and a 32% increase in splanchnic release of glucose when alfalfa intake increased by 39%. Release of glucose from the liver and splanchnic tissues in light of extraction of glucose by the PDV indicates substantial gluconeogenesis.

The primary gluconeogenic precursors that arise from ruminal fermentation are the volatile fatty acids (VFA) acetate and propionate. Because VFA are a product of ruminal fermentation, PDV releases VFA (Huntington et al., 1996; Patil et al., 1996; Goetsch et al., 1997a; Han et al., 2002). Level of nutrition does affect acetate and propionate PDV flux. Inclusion of alfalfa hay in warm- or cool-season grass hay diets

increased propionate PDV flux (Goetsch et al., 1997a). Likewise the addition of urea or casein to grass hay diets increased propionate PDV flux (Han et al., 2002). Acetate release by the PDV was reduced when roughage level was reduced from 73 to 37%, but propionate was not affected (Huntington et al., 1996). Because propionate is a gluconeogenic precursor, it is taken up by the liver. Additionally, acetate is utilized for lipid synthesis. Extraction of propionate was not effected by hay type, supplementation, (Patil et al., 1996), or form of the diet (Goetsch et al., 1997a). Small splanchnic release of propionate resulted from roughage diets (Huntington et al., 1996; Patil et al., 1996; Goetsch et al., 1997a).

Lactate in portal blood can come from two sources, ruminal absorption and glycolysis in the post-ruminal digestive tract (Reynolds and Huntington 1988a; Eisemann et al., 1997). Contribution of lactate from either ruminal or adipose sources is not well defined. Additionally, the ruminal contribution of lactate from ruminants consuming roughage-based diets may be minimal. However, Reynolds et al. (1991b) reported increased PDV lactate release with increased intake of 75% alfalfa diets by heifers. Interestingly, PDV lactate release was similar between 75% alfalfa and 75% concentrate diets. Han et al. (2002) reported similar PDV lactate flux between diets differing in bulk from hay intake and nutrients supplied by infusion. Ruminal contribution to PDV lactate flux varied from 21% in low bulk, low nutrient supplementation to 40% on medium bulk, high nutrient supplementation. Goetsch et al. (1997a) reported a 50% increase in PDV flux of lactate when bermudagrass and ryegrass-wheat hay was pelleted as opposed to coarsely chopped. The liver extracts nearly all lactate resulting in hepatic removal that is nearly equal to PDV release and therefore splanchnic flux is near zero (Goetsch et al.,

1997a). Removal by the liver is an important factor in gluconeogensis and transamination in the Cori cycle.

Flux of energy yielding nutrients across the PDV appears to be entirely diet dependent. Roughage diets increase the uptake of glucose by the PDV with some exceptions. Addition of concentrate sources into the diet decreases the uptake of glucose from arterial blood by the PDV. Because of the large uptake of glucose by the PDV, the liver synthesizes most of the glucose for peripheral use, although the kidney synthesizes some. Flux VFA and lactate are diet dependent. Release of VFA and lactate by the PDV is counterbalanced by uptake by the liver for metabolism into substrates that are utilized by the peripheral tissues. Acetate is an exception because it can be utilized directly for fatty acid synthesis.

Concentrate Diets

Blood Flow. Eisemann et al. (1996) examined the pattern of blood flow change in growing steers as they aged and gained BW. Portal vein and liver blood flow increased concomitant with DMI, age, and BW. Blood flow reached a plateau at 400 kg of BW and 400 days of age. Increased DMI of 75% concentrate diet increased portal and hepatic blood flow by 41 and 39% in heifers and 33 and 29% (Reynolds et al., 1991b, 1992). Burrin et al. (1989) fed pelleted diets that were 67% corn, 20% alfalfa at either ad libitum or at maintenance levels to growing lambs and measured blood flow. Arterial, portal, and hepatic blood flows increased 44, 5, and 21%, respectively, in ad libitum fed lambs during the 21 d feeding period; whereas, blood flows in maintenance fed lambs decreased 32, 7.5, 16% from d 0 to 21. Increases from low to medium (39%) and medium to high

(33%) DMI resulted in 40 and 47% increase in PDV blood flow and 79 and 30% increase in splanchnic blood flow in steers (Lapierre et al., 2000).

Differences in corn and sorghum processing (dry-rolled vs. steam-flaked) resulted in similar mesenteric, ruminal, portal, and hepatic blood flows (Alio et al., 2000; Theurer et al., 2002). Different underagadable intake protein (UIP) supplements to corn, pelleted corn cob diets had no effect on arterial blood flow (Bonhert et al., 1999). Supplementation with UIP supplements decreased portal blood flow by 14% and hepatic blood flow by 17% compared with soybean meal (Bonhert et al., 1999). Intraruminal infusion of VFA regardless of concentration increased portal blood flow in sheep by 23% compared with non-infused sheep (Kristensen et al., 2000). However, Krehbiel et al. (1992) observed no increase in portal or hepatic blood flow with increasing intraruminal infusion of butyrate. Lobley et al. (1998) reported that infusion into the mesenteric vein of amino acids increased arterial blood flow by 44% and decreased portal and hepatic blood flow by 11 and 7%, respectively, compared with pre-infusion blood flow.

Oxygen Consumption. Oxygen consumption by the PDV and liver in growing steers increased as steers aged, DMI increased, and gained BW (Eisemann et al., 1996). Liver oxygen consumption was greater than PDV oxygen consumption and splanchnic tissue oxygen consumption was 58% of whole body oxygen consumption at 236 kg of BW and 66% of whole body oxygen consumption at 522 kg of BW (Eisemann et al., 1996). Differences in DMI of concentrate has been reported to affect oxygen consumption by the PDV, liver, and splanchnic tissues. Maintenance level of intake resulted in a 30% reduction in PDV oxygen consumption in lambs after 21 d and no change in PDV oxygen consumption in ad libitum-fed lambs during a 21-d feeding

period (Burrin et al., 1989). Liver oxygen consumption increased by 29% in ad libitum fed lambs and decreased by 29% in maintenance-fed lambs after 21 d. Splanchnic tissue oxygen consumption was unchanged in ad libitum fed lambs, but was decreased by 30% from d 0 values in maintenance fed lambs after 21 d (Burrin et al., 1989). Reynolds et al., 1992) reported that a 44 % increase in DMI (g/kg of $BW^{0.75}$) by growing steers resulted in 30, 39, and 34% increase in PDV, hepatic, and splanchnic tissue oxygen consumption, respectively. Lapierre et al. (1999) reported a linear increase in CO_2 production with increasing intake of a 64% corn diet. Infusion of an amino acid solution into the mesenteric vein resulted in a 35% increase in oxygen consumption by the PDV and a 29% increase in liver oxygen consumption (Lobley et al., 1998). Reynolds et al. (1988a) reported that steers meal-fed concentrate diets had mesenteric, stomach, and PDV oxygen consumption that was not different from alfalfa fed steers.

Blood flow responds positively to increases in DMI. In each of the preceding studies, increasing DMI increased blood flow by substantial amounts. In addition, supplying additional nutrients by supplementation or infusion that increased the digestive and absorptive load for the PDV increased blood flow. These same trends were also observed for oxygen consumption by the PDV, liver, and splanchnic tissues. Many of the same reports that examined blood flow also examined oxygen consumption.

Flux of Nitrogenous Metabolites. Ammonia-N flux across PDV in growing steers varied curvilinearly as steers aged, increased DMI, and gained BW (Eisemann et al., 1996). Peak ammonia-N release from the PDV appeared to be at a metabolic BW of 87 kg (Eisemann et al., 1996). Hepatic uptake of ammonia-N by the liver exhibited a nearly inverse response to that of the PDV. The inverse relationship of PDV and hepatic flux

resulted in a small release of ammonia-N release by splanchnic tissues (Eisemann et al., 1996). Whitt et al. (1996) examined PDV and hepatic ammonia N flux during a 24-h period in steers fed twice daily. Ammonia N PDV flux peaked 1.5 h after feeding and decreased to daily average (131 mmol/h) by 5 h after feeding. Hepatic ammonia-N flux was a mirror image of PDV flux in steers. The removal of ammonia-N by the liver is essential because of ammonia's potential toxicity. Reynolds et al. (1991b, 1992) reported that a 45% increase in DMI of high concentrate diets resulted in 31 and 43% increased PDV release and hepatic uptake of ammonia-N. Similarly, differences in low and high DMI resulted in a 33% increase in ammonia N PDV release and a 35% increase in hepatic removal, whereas increasing DMI from medium to high increased ammonia-N PDV release by 19%, and increased hepatic removal by 16% (Lapierre et al., 2000). Splanchnic tissue flux of ammonia-N was unaffected by DMI (Reynolds et al., 1992; Lapierre et al., 2000).

Differences in grain processing did not affect PDV, hepatic, or splanchnic ammonia-N flux (Alio et al., 2000, Theurer et al., 2002). Mesenteric infusion of amino acids or intraruminal infusion of butyrate did not affect PDV or hepatic flux of ammonia-N in sheep (Lobley et al., 1998; Krehbiel et al, 1992, respectively). Splanchnic ammonia N flux was increased with 100 mmol/h of butyrate infusion but was decreased below control values at 200 mmol/h (Krehbiel et al., 1992). Addition of energy in the form of glucose or starch through abomasal infusion to a high concentrate diet did not alter PDV release of ammonia-N in beef heifers (Huntington and Reynolds, 1986). Bonhert et al. (1999) reported a decrease in ammonia-N PDV flux with increasing UIP supplementation

in lambs. The decrease in PDV ammonia-N release was coupled with a decreased hepatic removal of ammonia-N, but similar splanchnic flux.

Urea-N removal by PDV increased in growing steers as they increased DMI, age, and $BW^{0.75}$ (Eisemann et al., 1996). Hepatic flux exhibited a more dramatic increase in the rate of release, and splanchnic flux increased in overall rate of release as steers aged, increased DMI, and gained BW (Eisemann et al., 1996). In steers fed twice daily, PDV urea-N flux resulted in a net uptake by the PDV after the initial feeding of the day and release of urea-N after the second feeding, resulting in a mean daily uptake of urea-N by the PDV (Whitt et al., 1996). Hepatic urea-N flux changed from release to uptake 7 h after the initial morning feeding and uptake continued until approximately 4 h before initial morning feeding the next day (Whitt et al., 1996). Differences in DMI did not affect PDV uptake of urea N, however hepatic release of urea-N increased by 31% between low and medium DMI and 15% between steers on medium and high DMI (Lapierre et al., 2000). Because of the similarity in PDV flux, splanchnic urea-N flux was not affected by level of DMI. In contrast, PDV uptake of urea-N increased by 53 and 70% with high intake of concentrate diets compared with low intakes in two studies of Reynolds et al. (1991b and 1992). Release of urea-N from the liver was also significantly increased in cattle consuming greater amounts of high concentrate diets (Reynolds et al., 1991b and 1992); subsequent release of urea-N was also increased with increased DMI.

Increasing steam-flaked corn bulk density increased PDV urea-N uptake (Ailo et al., 2000), but grain processing (dry-rolled vs. steam-flaked) did not affect hepatic urea-N flux, and subsequent splanchnic urea-N flux did not differ between grain processing

methods in growing steers (Alio et al., 2000; Thuerer et al., 2002). Intraruminal infusion of butyrate did not affect urea-N PDV, hepatic, or splanchnic flux (Krehbiel et al., 1992). Likewise abomasal infusion of glucose or starch did not affect urea-N PDV uptake (Huntington and Reynolds, 1986). Increases in amino acid infusion or UIP feeding increased urea-N PDV uptake in lambs (Lobley et al., 1998; Bonhert et al., 1999, respectively). Hepatic urea-N flux increased with infusion of amino acids by increasing the release rate of urea-N (Lobley et al., 1998). Feeding UIP vs. ruminally degradable protein decreased urea-N release in lambs (Bonhert et al., 1999).

As steers aged, increased DMI, and BW, PDV flux of AAN increased and greater amounts of AAN were released to the liver (Eisemann et al., 1996). Hepatic flux of AAN resulted in uptake of AAN at a lower rate than PDV release but hepatic uptake increased as steers aged and increased DMI (Eisemann et al., 1996). The resulting splanchnic flux indicated a release of AAN in steers at 60 kg $BW^{0.75}$, but splanchnic flux was near zero at 114 kg $BW^{0.75}$ (Eisemann et al., 1996). Whitt et al. (1996) reported that AAN PDV, hepatic, and splanchnic flux was not sensitive to time of feed intake or time of day. Lapierre et al. (2000) reported a linear increase in AAN PDV flux between low, medium, and high levels of DMI in steers. Similarly, Reynolds et al. (1991b and 1992) reported 56 and 65% increased PDV release of AAN in cattle consuming 45% more of concentrate diets. Hepatic flux was not different between low, medium, and high levels of intake despite differences in PDV flux (Lapierre et al., 2000). However, two studies of Reynolds et al. (1991b and 1992) reported increased hepatic uptake of AAN when cattle consumed greater amounts concentrate diets. Differences in PDV flux between DMI levels resulted in an increased splanchnic tissue release of AAN on high levels of intake

(Reynolds et al., 1991b and 1992; Lapierre et al., 2000). Dry-rolled and steam-flaked corn diets resulted in similar PDV, hepatic, and splanchnic flux (Alio et al., 2000). Decreasing flake density increased PDV release of AAN (Ailo et al., 2000). Additional energy supplied by butyrate infusion did not alter PDV flux of AAN but did decrease hepatic flux with increasing concentration of butyrate infusion (Krehbiel et al., 1992). Splanchnic tissue flux was similar between control and butyrate infused steers; the splanchnic tissues had a net release of 43.5 mmol/h (Krehbiel et al., 1992). Infusion of additional amino acids into the mesenteric vein increased PDV release of AAN and increased removal hepatic flux of AAN above basal levels (Lobley et al., 1998). A second infusion of amino acids following the first infusion increased AAN PDV and hepatic flux above basal levels but not to the extent that the initial amino acid infusion produced (Lobley et al., 1998).

The high rate of metabolism of the liver can be attributed to its' high rate of protein turnover (Reynolds 2001; Lobley 2002). The high rate of protein turnover in the liver is a result of the synthesis of constitutive and exports proteins (Connell et al., 1997; Reynolds 2001; Lobley 2002). Export proteins synthesized by the liver such as albumin can be used as anabolic sources for peripheral tissues (Lobley 2002). In addition export and other constitutive proteins synthesized by the liver can act as temporary repositories of amino acids to mediate variation in amino acid supply (Lobely et al., 1998).

In cattle consuming concentrate based diets the flux of nitrogenous metabolites appears to be influenced by DMI. Increasing DMI increases the PDV release of ammonia-N and AAN. Increased PDV release of ammonia-N and AAN are probably a result of increased availability of nutrients for absorption. Ammonia-N uptake by the

liver counterbalances PDV release; however, AAN uptake by the liver is not always as great as PDV release and release of AAN from splanchnic tissues can be observed. Uptake of urea-N by the PDV, most like by the rumen, increased with increases in DMI. Increased fermentable carbohydrates of high concentrate diets increases the need of N by the ruminal microbes for microbial protein synthesis. A good example of this is the increased urea-N uptake by the PDV of steers consuming diets of lower flake density on steam flaked sorghum diets.

Flux of Energy Yielding Nutrients. Volatile fatty acid PDV, hepatic, and splanchnic flux were all affected by increases of DMI in growing steers (Eisemann et al., 1996). Increasing acetate release by the PDV and liver resulted in increasing release splanchnic flux of acetate for peripheral use. Because steers deposit more fat as they age when consuming high-energy diets the supply of acetate required by the peripheral tissues increases. Likewise, PDV flux release of propionate increased as steers aged and increased DMI (Eisemann et al., 1996). Hepatic flux increased in a similar fashion, but the liver utilized propionate resulting in a near zero splanchnic flux of propionate (Eisemann et al., 1996). Butyrate PDV, hepatic and subsequent splanchnic flux varied by less than 50 mmol/h as steers increased age and $BW^{0.75}$ (Eisemann et al., 1996).

Glucose flux across the PDV in growing steers remained relatively stable as steers gained BW, increased DMI, and aged (Eisemann et al., 1996); but did increase slightly at approximately 92 kg $BW^{0.75}$. However because of the increasing DMI and flux of gluconeogenic precursors, hepatic release of glucose increased as steers gained $BW^{0.75}$ (Eisemann et al., 1996). Subsequent splanchnic release of glucose also increased with body weight but leveled off at 92 kg $BW^{0.75}$. Reynolds et al. (1991b and 1992) reported

PDV release by cattle on low intake of concentrate diets and PDV uptake of glucose by cattle on high intake. Similarly, Whitt et al. (1996) reported removal of glucose by PDV in steers consuming a 64% concentrate diet. Increased PDV removal of glucose implies utilization of glucose exceeded absorption of glucose by intestinal tissues. In contrast, increasing level of DMI linearly increased release PDV flux in growing steers (Lapierre et al., 2000). Examination of within day variation showed that PDV glucose flux was release for only 3 h after the second feeding of the day (Whitt et al., 1996). Mean hepatic glucose flux indicated a release of glucose by the liver (Whitt et al., 1996), but removal occurred for 10 h during the day after the first of two feedings of steers. Therefore, a positive daily splanchnic flux was observed in steers on high-energy diets (Whitt et al., 1996). Similarly, the increase in DMI and gluconeogenic precursors resulted in an increase in release hepatic flux and splanchnic glucose flux (Reynolds et al., 1991b and 1992; Lapierre et al., 2000). Addition of intraruminal butyrate or increases in UIP did not affect PDV, hepatic, or splanchnic flux of glucose in steers or sheep, respectively (Krehbiel et al., 1992; Bonhert et al., 1999). However, Huntington and Reynolds (1986) reported increased PDV glucose release with abomasal glucose infusion compared with infusion of starch. Lower PDV glucose release associated with starch infusion implies digestion of starch to glucose is the rate-limiting step in glucose absorption (Reynolds et al., 1991b).

Release of lactate by the PDV was similar in growing steers as they increased DMI, BW, and age (Eisemann et al., 1996). Removal lactate flux indicated utilization of lactate by the liver in growing steers but remained stable with increases in DMI and BW (Eisemann et al., 1996). The subsequent splanchnic flux indicated removal of lactate,

and was constant across the growth period. In contrast Reynolds et al. (1991b and 1992) reported increased release of lactate by the PDV with increasing DMI of concentrate diets. However, cattle on lower DMI level of concentrate diet had hepatic removal greater than cattle on high DMI level (Reynolds et al., 1991b and 1992); but because of greater PDV release, splanchnic release of lactate was greater for cattle consuming greater amounts of concentrate diets (Reynolds et al., 1991b and 1992). Reynolds et al. (1998b) reported steers consuming alfalfa diets had 21% greater release of lactate from the PDV compared with steers consuming a concentrate diet. The majority of lactate in both alfalfa (77%) and concentrate (65%) diets originated from stomach tissues (Reynolds et al., 1988b).

Flux of NEFA across the PDV, liver, and splanchnic tissues have been reported to be DMI dependent (Reynolds et al., 1992; Lapierre et al., 2000). Steers consuming a medium level of DMI had greater release of NEFA by the PDV compared with low or high DMI steers. However, removal hepatic flux was greatest in low DMI steers followed by medium and then high-DMI steers (Lapierre et al., 2000); splanchnic flux was indicative of the extent of uptake of NEFA by the liver. In contrast, Reynolds et al. (1992) reported greater release of NEFA by the PDV and increased uptake by the liver for steers on a high level of intake, but splanchnic uptake was not different between steers on low and high levels of intake.

Flux of energy yielding nutrients is significantly affected by differences in intake. Volatile fatty acids released by the PDV increases with increasing DMI. Hepatic and splanchnic flux is mostly dependent on individual VFA. The liver metabolizes butyrate and propionate whereas acetate is released. The PDV even on high concentrate diets

takes up glucose. However, on high concentrate diets greater release of glucose from the liver is possible because of greater concentrations of gluconeogenic precursors. The effect of diet on lactate and NEFA metabolism is more variable than glucose. Release of lactate by the PDV is variable, but liver utilization remains nearly complete.

Summary and Conclusions

The occurrence and magnitude of compensatory growth that is exhibited in animals depends on many factors. This review focused on the effect of plane of nutrition on the occurrence of compensatory growth. Additionally, the review examined the effect of nutrition on some of the possible mechanisms that contribute to compensatory growth: visceral organ mass, cellularity and energy expenditure, circulating nutrients and hormones, and the flux of nutrients across splanchnic tissues.

Compensatory growth is generally characterized by increased BW gain, increased DMI, and increased gain efficiency. Compensating animals prior to the compensation period have a leaner body composition than contemporaries. Differential accretion of body protein and fat occurs during the realimentation period between compensating and normal steers. However, final body composition is often similar when all animals are fed to common final body weights.

Mass of critical visceral organs such as the GIT and liver are often changed in compensating cattle. In some cases the response of specific organs or tissues to nutrient restriction depends on the nature of the restriction (i.e., energy, protein, or intake). Generally, reduced visceral organ mass, cellularity, and oxygen consumption are

characteristic of animals that have undergone energy, protein, or intake restriction. The splanchnic tissues consume a large portion of an animal's maintenance energy requirements. The decrease in mass and energy expenditure that occurs in growing ruminants during energy, protein, and intake restriction decreases maintenance energy requirements. At the initiation of the realimentation period, the decreased maintenance energy requirements allow greater amounts of energy to be directed to growth.

Dietary energy, protein, or intake restrictions that elicit compensatory growth in animals alter circulating nutrients, metabolites, and hormones in those animals. Decreased nutrient intake will in turn decrease circulating nutrients available to the animal. Decreased nutrient intake also alters hormone concentrations in energy, protein, and intake restricted animals. Alteration of hormone concentration reconciles the animal's homeorhetic controls with nutrient intake. When dietary nutrient supply changes during realimentation circulating nutrient concentrations are quick to respond. However, hormonal balance and increases in hormone concentration in some instances require a longer time-course for change.

Level of intake and type of diet affects the nutrient profile and concentration that is ultimately absorbed by the GIT. The GIT requires nutrients and release of nutrients from the GIT varies with feed intake and diet type. The liver is an important thoroughfare for nutrients between the PDV and peripheral tissues. Depending on PDV release, peripheral needs, and nature of the nutrient, the liver can alter the concentration of nutrients that exit relative to the concentration that enters the liver. Ultimately the concentrations of nutrients that leave the liver dictate expression of compensatory growth in cattle.

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Chapter II

EFFECT OF LIVE WEIGHT GAIN DURING WINTER GRAZING ON SUBSEQUENT FEEDLOT PERFORMANCE, CARCASS CHARACTERISTICS AND BODY COMPOSITION OF BEEF STEERS [1,2,3,4]

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ABSTRACT

We compared feedlot performance, carcass characteristics, and body chemical composition to determine the effect of previous winter grazing BW gain in steers. Two experiments were conducted utilizing 48 Angus x Angus-Hereford steers in each experiment. The experimental designs were completely random. In each experiment, steers were randomly assigned to one of three treatments: high rate of BW gain grazing winter wheat (HGW); low rate of BW gain grazing winter wheat (LGW); or grazing dormant tallgrass native range (NR) with 0.91 kg/d of a 41% CP supplement. Steers grazed for 120 or 144 d in Exp. 1 and 2, respectively. Prior to placement into a feedlot in each experiment, four steers from each treatment were randomly selected for initial harvest to measure carcass characteristics, and carcass, offal, and whole body chemical composition. All remaining steers were placed into a feedlot and fed to the same backfat endpoint (1.27 cm). Six steers were randomly selected from each treatment for final chemical composition; carcass traits were measured on all steers. Initial carcass characteristics of steers prior to finishing were different ($P < 0.05$) between the three winter grazing treatments in Exp. 1 and 2. Feedlot live and empty BW ADG and gain efficiency was not different ($P > 0.10$) among treatments, but DMI (% of mean BW) of NR and LGW steers was greater ($P < 0.05$) compared with HGW steers in Exp. 1 and 2. Final carcass compositions were generally not different among treatments, except final carcass fat of HGW and NR was greater ($P = 0.03$) compared with LGW in Exp. 1 and greater ($P = 0.07$) in NR compared with HGW in Exp. 2. Final whole body composition

was similar ($P > 0.10$) among treatments in both Exp. 1 and 2. In Exp. 1, LGW carcass FFOM (fat free organic matter) accretion was greater ($P = 0.002$) and LGW and NR offal FFOM accretion was less ($P < 0.001$) compared with HGW. However in Exp. 2, NR and LGW fat accretion rate in the carcass and whole body was greater ($P < 0.01$) compared with HGW. In conclusion, differences in winter grazing ADG and initial body fat did not result in differences in feedlot gain or efficiency among treatments. Initial differences in body fat and FFOM content were nullified by feeding animals to a similar compositional endpoint. However, differences in accretive rates of fat and lean exist which could allow for different compositional endpoints depending on the length of the finishing period.

Key words: Cattle, Grazing, Feedlot, Body composition

Introduction

Beef production systems that incorporate grazing programs can have profound effects on body composition (Carstens et al., 1991), nutrient metabolism (Thomson et al., 1982), and subsequent feedlot performance (Drouillard et al., 1991a) of cattle. Sainz et al. (1995) reported that alterations in body composition through previous nutrition altered net energy maintenance requirements of growing cattle. Generally, as body fat content of cattle increases at a particular BW, metabolizable energy allowable daily gain decreases (NRC, 1996). Therefore, it is generally assumed that body fat content of cattle entering the feedlot can have important effects on cattle performance.

Previous nutrition that restricts cattle growth and limits body fat deposition can positively affect cattle performance in the feedlot through increased growth. Altering

previous nutrition has also been reported to affect composition of BW gain in the feedlot (Fox et al., 1972; Rompalla et al., 1985; Drouillard et al., 1991b). Much of the previous work examining feedlot growth has not utilized grazing systems, rather different levels of feed intake, restrictions in nutrient intake, and (or) differences between concentrate and roughage diets. In this experiment we wanted to compare winter grazing programs common to the Southern Great Plains. We hypothesized that steers of similar genetics with different BW gains and body fat resulting from winter grazing programs would exhibit different growth rates in the feedlot and have different body chemical composition changes during finishing. Therefore our objectives were to examine effect of previous BW gain during winter grazing on subsequent feedlot performance, carcass characteristics, and feedlot body chemical composition change.

Materials and Methods

Animals and Management

In each of two experiments, 48 fall-weaned Angus x Angus-Hereford steers (244 ± 23 kg, Exp. 1; 231 ± 25 kg, Exp. 2) from the same cowherd were randomly allotted to one of three winter grazing treatments. Treatments were: grazing winter wheat pasture to achieve a high rate of BW gain (**HGW**; mean herbage allowance = 1,665 ± 361 kg·steer^{-1}, Exp. 1; 2,093 ± 1,776 kg·steer^{-1}, Exp. 2); grazing winter wheat pasture and adjusting stocking density to maintain a low rate of BW gain (**LGW**; mean herbage allowance = 234 ± 53 kg·steer^{-1}, Exp. 1; 186 ± 93 kg·steer^{-1}, Exp. 2), or grazing dormant tallgrass native range (**NR**, very low stocking density). Steers grazing NR were offered 0.91 kg·steer^{-1}·d^{-1} of a cottonseed-meal based supplement (41% CP). No implants were

utilized during winter grazing. In Exp. 1, grazing was initiated on December 7, 1999 and terminated on April 6, 2000 after 120 d. In Exp. 2, grazing was initiated on December 18, 2000 and was terminated on May 10, 2001 after 144 d. Body weights of steers were obtained after a five to six hour removal from forage and water at the initiation of grazing, monthly during grazing, and at the termination of grazing.

Following grazing, steers were sorted by weight within winter grazing treatment and randomly assigned to feedlot pens. Prior to entering the feedlot, steers were co-mingled and allowed access to hay for 3 d to equalize gut fill. Steers were then shrunk without water for 5 to 6 h (Exp 1), or transported to the USDA, ARS Grazinglands Research Laboratory, El Reno, OK (Exp. 2) and weights were taken prior to placement into feedlot pens. At this time all steers were implanted with Revelor-S (Intervet; Millsboro, DE) and vaccinated for infectious bovine rhinotracheitis, bovine virus diarrhea, parainfluenza, and respiratory syncytial virus (Titanium 5, Diamond Animal Health; Des Moines, IA). In Exp. 1, steers were fed in 12.2 x 30.5 m open pens at the Willard Sparks Beef Research Center, Stillwater, OK, (3 pens/treatment, 4 steers/pen). In Exp. 2, steers were individually fed by use of Calan Broadbent Feeding System (American Calan; Northwood, NH) in 4.57 m^2 pens in an open-fronted building at the USDA, ARS Grazinglands Research Laboratory, El Reno, OK. During both experiments steers were adapted over four weeks to the final feedlot diet (Table 2.1) by replacing cottonseed hulls in Exp. 1 and ground alfalfa hay in Exp, 2 with corn. After adaptation to the final diet, in Exp. 1 steers were offered sufficient feed twice daily at 0800 and 1300 for the bunks to have approximately 2.5 kg at 2100 and no feed at 0700; in Exp. 2 all steers were offered ad libitum access to feed and were fed once daily at 0800. In Exp. 2,

one steer was removed from the HGW treatment because of refusal to eat from the Calan headgate. During the feedlot phase, steers were weighed unshrunk at monthly intervals. Steers were fed to a common endpoint of 1.27 cm of backfat as determined by ultrasound (Aloka, model 210, Aloka probe, model UST-5021; Aloka Co. Ltd., Wallingford, CT) between the 12th and 13th rib on the right side. When the backfat threshold was achieved, all steers in each treatment were harvested within nine days. The Oklahoma State University Institutional Animal Care and Use Committee approved the use of animals and research protocols.

Slaughter and Body Composition

Prior to placement in the feedlot for each experiment, four animals from each treatment were harvested for the determination of initial carcass characteristics and body composition. Steers were removed from their respective pastures at 0700 the morning of harvest and transported no more than 10 km to the Oklahoma Food and Agricultural Products Research and Technology Center (FAPRTC) abattoir. At harvest, steers were stunned with a captive bolt gun and killed by exsanguination. Weights of the noncarcass tissues and blood (offal), digesta weights, and hot carcass weights (**HCW**) were recorded. Offal tissues (minus digesta) were composited and ground twice using an Autio grinder (Astoria, OR) through a 10-mm aperture plate, mixed and sub-sampled in triplicate. After a 48-h chill, carcasses were re-weighed and carcass characteristics that included maturity, fat thickness at the 12th rib, 12th rib longissimus muscle area, kidney, pelvic, heart (KPH) fat, marbling score, quality grade, and yield grade were evaluated by faculty of the meats section in the Department of Animal Science. The right side of each cold

carcass was subsequently ground through a 10-mm followed by a 5-mm aperture plate, mixed and sub-sampled in triplicate. At final harvest for each experiment, six steers from each treatment were selected for composition measurements (two steers/pen in Exp. 1). Final body composition measurements were the same as for the initial harvest group. The remaining steers from each treatment were harvested and carcass characteristics measured as described for the initial harvest.

Triplicate samples of carcass and offal were analyzed for water by lyophilization to a constant weight. Lyophilized carcass and offal samples were further processed to reduce particle size by submersion in liquid nitrogen and ground using a Waring blender (Waring Products Co., Winsted, CT). Carcass and offal tissues were then subsequently analyzed for fat (extraction with diethyl ether for 48 h in Soxhlet apparatus), fat free organic matter (combustion of ether extraction residue, 500°C for 5 h), nitrogen (LECO, St. Joseph, MI), and phosphorus (AOAC, 1990). Energy content of carcass and offal tissues was calculated as weight of ether extracted material x 9.4 kcal/g and weight of fat-free organic matter (FFOM) x 5.55 kcal/g (Ferrell and Jenkins, 1998).

Statistical Analysis

All data for feedlot performance, carcass characteristics, chemical composition, and chemical accretion were analyzed as a completely random design using the Mixed procedure of SAS (SAS Inst. Inc., Cary NC). Gains of carcass, offal, and whole body composition were calculated as the difference between the final individual weights and predicted initial weights of steers measured at final harvest. The statistical model for feedlot performance in Exp. 1 included the term for treatment. The statistical model for

feedlot performance in Exp. 2 and all carcass characteristics, chemical composition, and chemical accretion data included the fixed effect of treatment and steer within treatment as the random effect. Treatment least squares means were calculated and means compared using LSD when protected by a ($P < 0.10$) *F*-value. Pen was the experimental unit for feedlot performance and feed intake in Exp. 1, whereas steer was the experimental unit in Exp. 2. For all other measurements, steer was the experimental unit.

Results

Winter Grazing

Live BW of steers during winter grazing and the feedlot period are shown in Figures 2.1 and 2.2, respectively. In Exp. 1, both HGW and LGW steers gained BW at similar rates during the first 45 d, whereas the NR steers gained no BW during this same period. After the first 45 d of winter grazing, LGW steers gained 39 kg and NR steers gained 20 kg during the remaining 74 d of the winter grazing period. In contrast, HGW steers gained 105 kg during the last 74 d of winter grazing. During Exp. 2, LGW and NR steers lost BW during the first 44 d, whereas HGW steers gained 21 kg. After d 45 of winter grazing in Exp. 2 HGW, LGW, and NR steers gained 1.49, 1.04, and 0.53 kg/d, respectively. Final grazing weights were greater for HGW steers than LGW steers, and both were heavier than NR steers in both Exp. 1 and 2. Winter grazing ADG were 1.31, 0.54, 0.16, and 1.10, 0.68, 0.15 kg/d for HGW, LGW, and NR steers in Exp. 1 and 2, respectively. Total gut fill at the end of winter grazing was not different ($P = 0.60$, average = 37.97 kg, Exp. 1; $P = 0.26$, average = 41.95 kg) between treatments.

Feedlot Performance

Experiment 1. Initial feedlot BW of HGW steers was 30% greater ($P < 0.001$) than LGW, which was 22% greater ($P < 0.001$) than NR steers (Table 2.2). Final BW of HGW and NR steers (average = 559 kg) were 7% greater ($P = 0.02$) than LGW steers. Mean DMI during the feedlot period did not differ ($P = 0.17$) among treatments (average = 10.42 kg/d). However, DMI (% of mean BW) of LGW and NR steers (average = 2.45%) was 11% greater ($P = 0.003$) compared with HGW steers. Throughout the entire feeding period, live BW gains were not different ($P = 0.95$) among treatments (average = 1.79 kg/d). Likewise, empty BW gains were not different ($P = 0.44$) among treatments (average = 1.69 kg/d). Across the entire feeding period, ADG:DMI was not different ($P = 0.41$) between treatments (average = 0.173 kg/kg).

Experiment 2. Initial feedlot BW of HGW steers was 19% greater ($P < 0.001$) than LGW steers, which was 30% greater compared with NR steers (Table 2.2). Final feedlot BW was not different ($P = 0.08$) among the treatments (average = 527 kg). Mean DMI during the feedlot period was not different ($P = 0.40$) among treatments (average = 9.92 kg/d). During the feedlot period, DMI (% of mean BW) of NR steers was 14% greater ($P = 0.01$) compared with HGW steers, LGW steers were intermediate. Across the entire feedlot period daily live and empty BW gains did not differ ($P = 0.24$ and 0.07; 1.68 and 1.77 kg/d, respectively) among treatments. Across the entire feedlot period, mean ADG:DMI among treatments was 0.177 ($P = 0.58$).

Carcass Characteristics

Experiment 1. Initial HCW were 64 and 100 kg greater for HGW steers compared with LGW and NR steers, respectively (Table 2.4). Greater initial HCW in HGW steers resulted in 3.5 and 6.7% greater ($P < 0.001$) dressing percent than LGW and NR steers. All initial measures of fat deposition: kidney, pelvic and heart fat, 12th-rib fat thickness, and marbling score were greatest ($P < 0.001$) for HGW carcasses followed by LGW and then NR carcasses. Final HCW were greater ($P = 0.03$) for HGW than LGW steers; HCW of NR steers were intermediate. Kidney, pelvic, and heart fat were greater ($P = 0.02$) for HGW carcasses compared with LGW and NR carcasses. All other final carcass characteristic measurements were not different ($P > 0.10$) among treatments. These results were expected because our goal was to harvest all steers at a similar 12th-rib fat thickness endpoint.

Experiment 2. Similar to Exp. 1, initial HCW from HGW steers were 27 and 70% greater ($P < 0.001$) compared with LGW and NR steers, respectively, in Exp. 2 (Table 2.5). Dressing percent of HGW steers was 2.9 and 7.4% greater ($P < 0.001$) than LGW and NR steers. Similar to Exp. 1, all initial estimations of fat deposition: kidney, pelvic and heart fat, 12th-rib fat thickness, and marbling score were greater ($P < 0.005$) in HGW carcasses compared with LGW and NR carcasses. All final carcass measurements did not differ ($P > 0.10$) among treatments.

Carcass Chemical Composition

Experiment 1. Initial carcass mass of HGW was greater ($P < 0.001$) compared with LGW, which was greater compared with NR (Table 2.6). Because of the large differences in carcass mass between treatments, all measured chemical components (water, FFOM, fat, ash, phosphorus, and energy) were greater ($P < 0.001$) for HGW compared with LGW and chemical components of LGW were greater ($P < 0.001$) compared with NR. Initial offal mass of HGW steers was 32.8 kg greater ($P = 0.05$) than NR steers. Total water and FFOM content of offal tissue was not different ($P > 0.10$; average = 59.6 and19.0 kg, respectively) among treatments. Total fat content and calculated energy content of offal followed a similar pattern as carcass fat and energy, being greatest ($P < 0.001$) in HGW followed by LGW and then NR steers. Amount of ash and phosphorus of offal was not different ($P > 0.10$) among treatments (1.47 and 0.5 kg, respectively). Initial whole empty body mass was greater ($P < 0.001$) in HGW steers compared with LGW steers, that had greater ($P < 0.001$) initial empty body mass than NR steers. Whole empty body water of HGW and LGW was 27.7 kg more than NR whole empty water but was not significantly different ($P = 0.36$). Whole empty body FFOM, fat and energy content was 21, 177, and 57% greater ($P < 0.001$) for HGW compared with LGW, which had 23, 154, and 28% greater FFOM, fat, and energy content compared with NR. Ash ($P = 0.005$) and phosphorus ($P = 0.02$) content in the whole empty body of HGW steers was greater compared with LGW and NR steers.

At final harvest, HGW and NR steers had carcasses that were heavier ($P = 0.05$, average = 338.3 kg), and contained more fat ($P = 0.03$, average = 99.0 kg) compared to LGW steers. Final carcass water, FFOM, and P were not different ($P > 0.10$; average =

167.3, 64.5, and 2.5 kg) among treatments. Final ash content of HGW was 2.0 kg greater ($P = 0.10$) than LGW; NR was intermediate. Final carcass energy content was not different ($P = 0.13$) among treatments, however HGW and NR carcasses did have 297 and 237 kcal/kg more energy, respectively, than LGW. Final offal mass was not different ($P = 0.11$) among treatments (average = 158.3 kg), as was final FFOM, fat, and ash content of offal ($P > 0.10$; 32.6, 34.1, and 2.2 kg, respectively). At final harvest, water content of NR offal was greater ($P = 0.10$) compared with LGW, HGW was intermediate. Final offal energy content did not differ ($P > 0.10$; average = 3173 kcal/kg) among treatments, because neither FFOM nor fat content of offal was different among treatments. Final phosphorus content of HGW offal was 0.25 kg greater ($P = 0.02$) compared with LGW and NR offal (average = 0.45 kg). Final whole empty body mass of HGW and NR (average = 500 kg) was greater ($P = 0.03$) compared with LGW (468 kg). Because of the relatively small differences between treatments for carcass and offal weight and chemical content, whole empty body chemical composition was similar ($P > 0.10$) among treatments.

The rate of carcass mass (average = 1.28 kg/d) and water (average = 509 g/d) accretion was not different ($P > 0.14$) among treatments during the finishing period (Table 2.5). Carcass FFOM accretion rate was 16% greater ($P = 0.004$) for LGW (220 g/d) compared with HGW and NR (average = 189 g/d). The carcass fat accretion rate did not differ ($P = 0.28$; average = 544 g/d), while carcass energy accretion was 30% greater ($P = 0.11$) in NR carcasses compared with HGW carcasses, LGW carcasses were intermediate. Ash accretion was not different ($P = 0.15$, average = 15 g/d) among treatments. Phosphorus accretion was not different ($P = 0.67$) among treatments (average = 9.4 g/d).

Offal mass accretion rate was 0.14 kg/d greater (P = 0.006) for HGW compared with LGW, 0.12 kg/d greater than NR. Water accretion rate was greater (P = 0.03) for HGW (311 g/d) compared with LGW and NR (244 g/d). Offal FFOM accretion rate was greater (P < 0.001) for HGW compared with LGW and NR. Fat accretion rate in offal tissue did not differ (P = 0.58) among treatments, but was numerically greater for LGW and NR (17%). Offal ash accretion rate was greatest (P = 0.04) for HGW, intermediate for NR, and least for LGW; phosphorus accretion rate was greater (P = 0.004) for HGW compared with LGW and NR offal. Offal energy accretion rate was 71% greater (P = 0.04, average = 8.33 kcal·kg^{-1}·d^{-1}) for LGW and NR compared with HGW (4.87 kcal·kg^{-1}·d^{-1}). Empty body mass accretion rate was not different (P = 0.51) among treatments (average = 1.85 kg/d). Water accretion rate was greater (P = 0.05) for HGW (882 g/d) compared with LGW and NR (average = 665 g/d). Empty body FFOM and fat accretion rate did not differ (P > 0.10) among treatments (average = 334 and 750 g/d, respectively). Even though empty body FFOM and fat accretion were not different, empty body energy accretion was greater (P <0.001) for LGW and NR (average = 12.20 kcal·kg^{-1}·d^{-1}) compared with HGW (4.66 kcal·kg^{-1}·d^{-1}). Empty body ash accretion rate was not different (P = 0.13, average = 22 g/d) nor was phosphorus accretion rate different (P = 0.67) among treatments (average = 10.3 g/d).

Experiment 2. Initial carcass mass of HGW was 48 kg greater (P < 0.001) compared with LGW carcasses which were 45.5 kg greater (P < 0.001) than NR carcasses (Table 2.6). A similar pattern was exhibited in carcass water, FFOM, and fat content; HGW carcasses had greater (P < 0.001) amounts compared with LGW, and LGW had

greater ($P < 0.001$) amounts of water, FFOM, and fat compared with NR. Carcass ash content of HGW was greater ($P = 0.006$) compared with LGW and NR. Initial carcass phosphorus content of HGW and LGW was greater ($P = 0.002$; average = 1.61 kg) than NR. Energy content of carcasses was affected by the content of FFOM and fat, and therefore followed a similar pattern; HGW carcasses had 583 kcal/kg greater energy ($P < 0.001$) compared with LGW carcasses, which had 723 kcal/kg greater energy ($P < 0.001$) compared with NR. Similar to initial carcass mass, initial offal mass of HGW was greater ($P < 0.001$) than LGW, which was greater ($P < 0.001$) compared with NR. Offal water content was 7.5 kg greater ($P = 0.002$) for HGW compared with LGW which was 9.7 kg greater compared with NR offal water content. Offal FFOM of HGW and LGW was 35% greater ($P = 0.008$; average = 21.6 kg) compared with NR. Initial offal fat content of HGW was 46% greater ($P < 0.001$) than LGW, and five-fold greater than NR offal. Because of the large differences in offal FFOM and fat content, energy content of HGW offal was 11% greater ($P < 0.001$) compared with LGW, which was 61% greater ($P < 0.001$) than NR. Offal ash content was not different ($P = 0.42$; average = 1.48 kg) among treatments. Initial empty body mass of HGW was greater ($P < 0.001$) compared with LGW, which was greater compared with NR. Because of the large differences in whole empty body mass: water, FFOM, fat, ash, phosphorus, and energy content of HGW were greater ($P < 0.002$) compared with LGW, and LGW chemical components were all greater ($P < 0.002$) compared with NR.

Final carcass mass did not differ ($P = 0.33$; average = 322.4 kg) among treatments. Final water and FFOM content were also not different ($P > 0.10$; average = 164.4 and 62.7 kg, respectively) among treatments. Final fat content of NR carcasses

was 12% greater (P = 0.07) compared with HGW, whereas LGW final fat content was intermediate. Final carcass energy content of LGW and NR (average = 3712 kcal/kg) was 9% greater (P = 0.04) compared with HGW because of the increased fat content and slightly more FFOM content. Final offal mass, water, FFOM, ash, and phosphorus were not different (P > 0.10; average = 156.5, 87.5, 29.7, 8.2, 2.03 kg, respectively) among treatments. Final offal fat content displayed a similar pattern as final carcass fat content; NR offal fat content was 4.3 kg greater (P = 0.07) compared with HGW and LGW offal fat content was intermediate. Ash content of the final offal tissue in HGW and LGW (average = 3.3 kg) was greater (P = 0.03) compared with NR offal ash content. Final offal phosphorus content of NR tended to be greater than HGW and LGW (P = 0.08; 0.62 vs. 0.52 kg). Final offal energy content was affected by fat content; LGW and NR final offal energy (average = 3328 kcal/kg) was greater (P = 0.02) compared with HGW (3048 kcal/kg). Final empty body mass was not different (P = 0.43; average = 478.8 kg) among treatments. Because of few differences in final carcass and offal chemical composition, final whole empty body composition did not differ (P > 0.10) among treatments. Numerical increases in empty body fat and energy content can be discerned between HGW and NR.

Carcass mass accretion rate of NR was 0.17 kg/d greater (P = 0.009) compared with HGW and LGW (average = 1.17 kg/d). Carcass FFOM accretion rate did not differ (P > 0.10) among treatments in Exp. 2 (average = 185 g/d). Carcass water accretion of NR was 32% greater (P = 0.02) compared with LGW, HGW was intermediate. Because LGW and NR steers initial carcass fat content was low, fat accretion in LGW and NR carcasses was 39% greater (P < 0.001, average = 583 g/d) than HGW carcass fat

accretion. Because LGW and NR carcasses had greater fat accretion rate, carcass energy accretion rate of NR was over two-fold greater ($P < 0.001$) compared with HGW carcasses and LGW was nearly two-fold greater ($P < 0.001$) compared with HGW carcasses. Carcass ash and phosphorus accretion rates were similar ($P > 0.10$) among treatments (average = 47 and 5.6 g/d). Offal mass accretion of NR was 18% greater ($P = 0.04$) compared with HGW and LGW. Offal water and FFOM accretion rates were not different ($P > 0.10$) among treatments (average = 219, and 86 g/d, respectively). Offal fat accretion of LGW and NR was greater ($P = 0.02$, average = 240 g/d) compared with HGW. Ash accretion rates in offal were greater ($P = 0.05$) for HGW compared with NR and LGW was intermediate. Phosphorus accretion rate was greater in NR ($P < 0.001$) than HGW or LGW. Like carcass and offal mass accretion, empty body mass accretion of NR was 19% greater ($P = 0.001$) compared with HGW and LGW (average = 1.67 kg/d). Because carcass and offal accretion rates of FFOM were not different, empty body accretion rate did not differ ($P = 0.19$; average = 271 g/d). Empty body water accretion was 31 greater ($P = 0.007$) for NR compared with LGW and HGW was intermediate. Empty body fat accretion rate was 36% greater ($P = 0.001$) for LGW and NR (average = 823 g/d) compared with HGW. Whole body ash and phosphorus accretion rates were similar ($P > 0.10$) among treatments. Energy accretion within the empty body was two-fold greater ($P < 0.001$) for NR compared with HGW and 123% greater for LGW compared with HGW.

Discussion

Steers that had lower BW gains and lower body fat (LGW and NR) at the end of winter grazing had similar feedlot performance, carcass characteristics, and body composition when fed to a similar end-point. Phillips et al. (1991 and 2001) compared feedlot performance of steers that had previously grazed dormant native prairie during the winter followed by spring wheat-cool season grass grazing or steers that had grazed winter wheat followed by spring wheat-cool season grass grazing. Steers that had grazed native prairie had lower grazing ADG (0.42 and 0.71 kg/d) compared with steers that had grazed wheat (0.55 and 0.71 kg/d; Phillips et al., 1991 and 2001, respectively). In the feedlot steers that had grazed native prairie had greater ADG, similar DMI and were 10% more efficient (Phillips et al., 1991). In contrast, in the present study steers with decreased winter ADG grazing wheat or native range had similar feedlot gains and efficiency but increased DMI relative to mean feeding weight. In addition, previous restriction of energy or protein intake by steers resulted in increased ADG and DMI during realimentation (Drouillard et al., 1991a). Other work by Wester et al. (1995), who utilized energy restricted lambs or metabolizable protein restricted lambs, reported restricted lambs had greater ADG, greater feed DMI (% BW) and were 27% more efficient compared with control lambs. Steers that were limit fed a concentrate diet or low energy forage diet prior to feedlot finishing exhibited increased DMI, empty body weight (EBW) gain, and increased gain efficiency compared with ad libitum-fed control steers (Sainz et al., 1995).

While there was no difference in feedlot performance among treatments in the current study, an increase in feedlot performance of LGW and NR steers might have been expected relative to HGW steers. Carstens et al. (1991) reported that steers that exhibited compensatory growth due to a 189-d restriction period in which they gained 0.4 kg/d, had ADG that was 37% greater than ad libitum-fed control steers. In an experiment by Wright and Russel (1991), Charolais-cross steers that had been restricted to 58% of the daily gain of control steers from 259 to 350 kg of BW, gained 38% faster from 350 to 400 kg while consuming similar amounts of feed compared with control steers, resulting in compensating steers having a 39% increase in gain efficiency.

Interestingly for both experiments, live BW ADG by HGW during the feedlot period was 1.79 and 1.72 kg/d for Exp. 1 and 2, respectively, and the LGW and NR live BW ADG was not greater than HGW. The greater ADG of HGW compared with LGW or NR steers in Exp. 2 also conflicts with the 1996 Beef Cattle NRC Level 1 Model. The Level 1 model predicts a negative relationship between ME allowable ADG and estimated body fat content, regardless of initial BW. Hayden et al. (1993) reported a reduced initial performance during the finishing period by previously restricted steers. These authors observed that previously forage fed, adequate energy intake steers gained 51% faster compared with previously forage fed, energy intake restricted steers during the first 34 d of a high-grain finishing diet. Similar to our results, White et al. (1987) reported that steers, which had the highest BW gains on winter wheat pasture, also had the greatest BW gains during the first 28 d of either a subsequent summer grazing period or feedlot finishing, but entire feedlot gains did not differ. Additionally, in Exp. 1 and Exp. 2, DMI (% mean BW) of all treatments was greater than what would be expected.

Rakestraw (1995) summarized DMI (% of mean BW) of feedlot steers during two years. Dry matter intake had a range of 2.10 to 1.95% with a mean of 2.03% of mean BW. In the current experiments DMI (% of mean BW) was 16% greater than that reported by Rakestraw (1995). The exceptional feedlot performance of high gaining wheat cattle has yet to be explained. The feedlot performance of the HGW steers would contradict traditional thinking, in that animals that enter the feedlot with a higher degree of body fat will not perform well and will be less efficient compared with animals of similar age and genetic background but having less body fat.

In Exp. 1, LGW and NR steers gained little BW during the 72 d prior to placement into the feedlot. Even though overall winter gain of LGW steers was 0.54 kg/d, the majority of this weight gain occurred during the first 50 d of the winter grazing period. Steers in LGW and NR treatments had been held at maintenance for some time and had apparently adjusted to that level of energy intake. Burrin et al. (1989) reported that in sheep fed at maintenance level of intake over 21 d, whole body oxygen consumption decreased by 10%, thus lowering the maintenance energy requirements of those lambs. These results agree with those of Sainz et al. (1995) and Wright and Russell (1991), where growth restricted steers had greater daily EBW gain and greater protein gain than non-restricted steers.

In contrast during the grazing period of Exp. 2, the majority of BW gained in LGW and NR steers occurred during the last 100 d. Interestingly, LGW and NR steers were gaining weight while grazing prior to placement in the feedlot in Exp. 2. Steers from the LGW and NR treatments were in a positive energy balance and gaining weight for 109 d, thus reducing the efficiency with which they might utilize the additional

energy provided in the feedlot diet compared with steers fed at maintenance. Ferrell et al. (1986) observed that lambs with similar ADG had similar fasting heat production regardless of previous rate of gain. In our case, the fact that LGW and NR steers were gaining BW prior to entering the feedlot might have negated any potential reduction in fasting heat production they would have otherwise had when they entered the feedlot.

At the initiation of the feedlot period, carcass characteristics and body composition were by design very different in both experiments. All measures of carcass fat content and actual carcass, offal, and body fat analysis indicated that HGW steers entered the feedlot with a large amount of body fat relative to LGW steers that had more body fat than NR steers. Other work supports the observed differences in body composition by our winter grazing treatments. Baker et al. (1992), using different forage levels in restricted and ad libitum fed steers, reported that restricted steers had 76% less empty body fat compared with ad libitum fed steers, and 0.33 Mcal/kg less body energy. In contrast restricted steers had 8% more protein and 6% more empty body water compared with ad libitum fed steers. This is similar to our data from both experiments, in that HGW steers initially had increased fat and energy contents but decreased proportion of water content compared with LGW and NR steers. While we did not initially harvest our animals at a similar EBW, Sainz et al. (1995) adjusted intermediate harvest date steers to a common 327 kg of EBW. In their study, ad libitum fed steers had greater measures of fat mass (back fat, KPH, marbling and abdominal fat). Carcass fat percent and empty body fat were also greater in ad libitum fed steers compared with limit-fed or forage-fed steers, but empty body protein mass was greater in the limit-fed or forage-fed steers. The intermediate harvest date of Sainz et al. (1995) was at a greater EBW than

our LGW and NR steers would have achieved before entering the feedlot, but our data are comparable. Baker et al. (1992) and Sainz et al. (1995) both reported increases in the percent protein in restricted steers. We did observe an increase in carcass protein in Exp. 1, but not offal or empty body, and no increases in protein by LGW or NR steers above that of HGW steers in Exp. 2. Fox et al. (1972) reported steers that had maintained BW for 190 or 154 d did not increase protein content compared with control steers.

In Exp. 1, LGW and NR steers that had decreased winter BW gains had lighter carcasses, but equal carcass and 23% greater FFOM accretion rates compared with HGW steers in spite of increased days on feed. These same steers had similar offal composition and 25% lower offal accretion rate compared with HGW steers, which resulted in no differences in empty body composition. These findings agree with those of Carstens et al. (1991), where at 450 kg of empty BW, which would be less than our estimated empty BW, previously restricted steers had greater carcass protein and water, and less carcass fat compared with control steers. The similarity among treatments in final empty body composition also agrees with the work of others (Wright and Russel, 1991; Coleman et al., 1993; Hayden et al., 1993; and Sainz et al., 1995) who reported no differences between control and previously restricted steers for final empty body composition. In Exp. 2, steers that entered the feedlot with reduced carcass, offal, and body fat (LGW and NR), had greater final percent fat in both the carcass (28%) and offal (24%) compared with HGW steers (25 and 21%, respectively). The amount of carcass and final empty body FFOM did not differ. This may suggest that LGW and NR steers deposited more fat than lean mass early in the feedlot period, which might explain the numerically lower BW gains compared with HGW steers. Increased offal mass accretion and increased

offal fat accretion in LGW and NR steers also support the idea of early deposition of fat early in the feedlot period. Energy restricted lambs have been shown to deposit more fat during the early finishing period (Drouillard et al., 1991b). The early deposition of fat rather than increased protein deposition in previously restricted lambs led those authors to speculate that the growth potential of the lambs had been compromised. Blum et al. (1985) utilizing two-year old energy-restricted and control, energy-adequate steers examined changes in heat production during and immediately after restriction. At the end of the restriction phase heat production by restricted steers was reduced by 100 $kcal \cdot kg^{0.75-1} \cdot d^{-1}$. However, by the fifth day of re-feeding, heat production had increased by 200 $kcal \cdot kg^{0.75-1} \cdot d^{-1}$ and remained constant. If LGW and NR steers had a similar pattern of heat production and fat deposition rate was increased during the early feedlot period, this might explain the absence of increased growth. Old and Garrett (1987) reported that as ME intake increases, the proportion of energy that is partioned to fat synthesis increases.

Implications

Historically, cattle that have had high body condition resulting from grazing or growing programs have been discounted in price when they entered feedlots because of their anticipated reduced performance. Our limited data over two years would indicate that anticipating reduced performance by high gaining wheat pasture steers may not always be appropriate. Differences in initial body composition of cattle can be effectively minimized at the end of the feeding period if all cattle are fed to the same backfat endpoint. Timing and degree of restriction might influence rate and composition

of tissue accretion. However, differences in growth rate and accretion rates of fat and lean exist which could allow for different compositional endpoints depending on the length of the finishing period.

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Table 2.1. Composition of the final feedlot diet (% of DM or Mcal/kg DM)

Item	Experiment 1	Experiment 2
Ingredient		
Dry whole shelled corn	79.0	-
Dry rolled corn	-	83.0
Cottonseed hulls	9.0	-
Ground alfalfa hay	-	8.0
Blended fat	3.0	-
Soybean meal	5.17	-
Cottonseed meal	-	4.0
Wheat middlings	1.04	-
Cane molasses	-	4.0
Urea	0.80	0.65
Limestone	0.93	0.70
Dicalcium phosphate	0.33	-
Salt	0.24	-
Rumensin, 176 g/kg	0.02	0.02
Tylan, 220 g/kg	0.01	0.01
Vitamin A, 30,000 IU/g	0.01	0.01
Vitamin E, 3,000 IU/g	0.001	-
Trace mineral premix[a]	0.04	0.03
Calculated nutrient composition		
Crude protein, %	13.40	13.48
NE_m, Mcal/kg	2.15	2.11
NE_g, Mcal/kg	1.38	1.37

[a] Guaranteed Analysis: Zn (13.5%), Mn (6.0%), Cu (3.6%), Fe (1.43%), Co (800 ppm), I (6,000 ppm), Se (100 ppm).

Table 2.2. Feedlot performance of steers from different winter grazing programs

Item	Treatment			SEM[a]
	HGW	LGW	NR	
	----------Experiment 1----------			
Initial liveBW, kg	404[b]	311[c]	255[d]	2.4
Final live BW, kg	563[b]	524[c]	555[b]	7.1
Days on feed	89	116	163	0.0
Feed DMI				
kg/d	10.68	10.43	10.15	0.17
% of mean BW	2.21[b]	2.50[c]	2.40[c]	0.02
Gain, kg/d				
Live BW	1.79	1.80	1.80	0.06
Empty BW	1.64	1.67	1.75	0.06
Gain:feed, kg/kg				
Live	0.17	0.17	0.18	0.006
Empty	0.15	0.16	0.17	0.005
	----------Experiment 2----------			
Initial live BW, kg	395[b]	333[c]	257[d]	5.6
Final live BW, kg	542	511	528	9.9
Days on feed	88	111	145	0.0
Feed DMI				
kg/d	10.25	9.73	9.79	0.30
% of mean BW	2.19[b]	2.31[bc]	2.50[c]	0.07
Gain, kg/d				
Live BW	1.72	1.60	1.71	0.05
Empty BW	1.77	1.69	1.86	0.05
Gain:feed, kg/kg				
Live	0.18	0.17	0.18	0.006
Empty	0.18	0.18	0.19	0.006

[a] Standard error of mean, Exp. 1, n = 3, Exp. 2, n= 12.

[b,c,d] Within a row, means without a common superscript letter differ ($P < 0.05$).

Table 2.3. Effect of winter grazing on carcass characteristics of steers entering the feedlot and at final harvest in Exp. 1

Item	Treatment			SEM[a]
	HGW	LGW	NR	
Initial harvest				
Hot carcass wt, kg	237[b]	173[c]	137[d]	4.9
Dressing, %	60.4[b]	56.9[c]	53.7[d]	0.64
12th Rib fat thickness, cm	1.17[a]	0.25[c]	0.0[c]	0.10
Kidney, pelvic, heart fat, %	2.63[b]	0.50[c]	0.14[c]	0.22
Longissimus area, cm^2	70.45[b]	54.99[c]	47.25[c]	3.62
Marbling score[e]	357[b]	260[c]	155[d]	24.0
Yield grade	2.54[b]	1.59[c]	1.36[c]	0.26
Final harvest				
Hot carcass wt, kg	342[b]	318[c]	332[bc]	6.3
Dressing, %	60.8	59.2	60.5	0.61
12th Rib fat thickness, cm	1.63	1.59	1.55	0.12
Kidney, pelvic, heart fat, %	2.19[b]	1.72[c]	1.68[c]	0.14
Longissimus area, cm^2	77.19	76.51	80.28	0.44
Marbling score	448	392	407	22.9
Yield grade	3.49	3.25	3.17	0.16

[a] Standard error of mean, n = 4 for initial harvest, n = 12 for final harvest.
[b,c,d] Within a row, means without a common superscript letter differ ($P < 0.05$).
[e] 100 = Practically devoid, 200 = Trace, 300 = Slight, 400 = Small.

Table 2.4. Effect of winter grazing on carcass characteristics of steers entering the feedlot and at final harvest in Exp. 2

	Treatment			
Item	HGW	LGW	NR	SEM[a]
Initial harvest				
Hot carcass wt, kg	225[b]	177[c]	132[d]	5.89
Dressing, %	59.7[b]	56.8[c]	52.3[d]	0.67
12th Rib fat thickness, cm	0.69[b]	0.08[c]	0.0[c]	0.06
Kidney, pelvic, heart fat, %	2.25[b]	1.31[c]	0.88[c]	0.21
Longissimus area, cm^2	56.70	55.60	47.40	2.94
Marbling score[e]	275[b]	75[c]	0.0[d]	17.23
Yield grade	2.76[b]	1.58[c]	1.43[c]	0.16
Final harvest				
Hot carcass wt, kg	328	310	320	6.9
Dressing, %	60.4	60.9	60.7	0.60
12th Rib fat thickness, cm	1.37	1.16	1.55	0.13
Kidney, pelvic, heart fat, %	1.82	1.83	1.67	0.09
Longissimus area, cm^2	79.34	74.61	74.18	2.28
Marbling score	405	387	426	16.6
Yield grade	3.01	3.06	3.38	0.17

[a] Standard error of mean, n = 4 for initial harvest, n = 12 for final harvest.
[b,c,d] Within a row, means without a common superscript letter differ ($P < 0.05$).
[e] 100 = Practically devoid, 200 = Trace, 300 = Slight, 400 = Small.

Table 2.5. Effect of winter grazing program on steer chemical composition in Exp. 1

	Carcass				Offal				Empty body			
	Treatment				Treatment				Treatment			
	HGW	LGW	NR	SEM[a]	HGW	LGW	NR	SEM	HGW	LGW	NR	SEM
Initial												
Mass, kg[b]	237.2[c]	173.7[d]	137.0[e]	5.15	107.0[c]	97.6[cd]	74.2[d]	8.24	344.2[c]	271.3[d]	211.2[e]	9.64
Water, kg	127.7[c]	107.8[e]	95.0[e]	2.97	64.4	62.8	51.6	5.53	176.5	177.7	149.4	14.98
FFOM[e], kg	53.6[c]	37.5[d]	29.6[e]	1.86	18.8	20.7	17.6	1.48	68.86[c]	56.9[d]	46.1[e]	1.94
Fat , kg	49.2[c]	24.8[d]	10.2[e]	1.93	22.3[c]	12.3[d]	3.9[e]	2.36	88.8[c]	32.0[d]	12.6[d]	9.04
Ash, kg	6.8[c]	3.7[d]	2.2[e]	0.46	1.5	1.7	1.2	0.20	10.1[c]	4.8[d]	3.1[d]	1.13
P, kg	1.7[c]	1.4[d]	0.9[e]	0.10	0.5	0.6	0.4	0.05	2.8[c]	1.8[d]	1.3[d]	0.30
Energy[f], kcal/kg	3201[c]	2542[d]	1900[e]	77	2936[c]	2357[d]	1787[e]	74	3570[c]	2275[d]	1773[d]	291
Final												
Mass, kg[b]	339.1[c]	317.2[d]	337.4[c]	6.27	161.2	151.2	162.5	3.86	500.2[c]	468.4[d]	500.0[c]	8.53
Water, kg	166.5	166.2	169.3	4.14	89.3	85.1	93.4	2.51	247.3	243.4	246.4	7.41
FFOM, kg	66.3	63.9	63.3	1.52	33.9	30.9	33.1	1.35	99.2	93.2	95.7	3.00
Fat, kg	99.2[c]	82.0[d]	98.7[c]	4.69	35.3	33.3	33.8	2.30	143.5	124.6	139.0	8.59
Ash, kg	7.0[c]	5.0[d]	6.0[cd]	0.61	2.6	1.8	2.2	0.33	10.3	7.2	8.8	1.00
P, kg	2.6	2.3	2.5	0.17	0.7[c]	0.5[d]	0.4[d]	0.06	3.5	2.9	3.0	0.21
Energy, kcal/kg	3846	3549	3786	100	3231	3206	3082	97	3798	3611	3669	135
Accretion[g]												
Mass, kg/d[b]	1.27	1.33	1.23	0.04	0.66[c]	0.52[d]	0.54[d]	0.03	1.94	1.85	1.77	0.05
Water, g/d	505	568	454	38	311[c]	231[d]	256[d]	20	882[c]	676[d]	654[d]	66
FFOM, g/d	173[c]	220[d]	206[c]	14	180[c]	100[d]	95[d]	12	353	350	300	22
Fat, g/d	589	501	543	37	158	187	184	21	668	807	776	71
Ash, g/d	7	14	24	6	12[c]	2[d]	7[cd]	3	20	16	31	9
P, g/d	10.3	8.3	9.5	1.62	2.0[c]	-0.6[d]	-0.2[d]	0.47	9.5	10.7	10.7	1.89

Energy, $kcal \cdot kg^{-1} \cdot d^{-1}$	8.84	10.21	11.51	0.88	4.87[c]	8.76[d]	7.89[d]	1.01	4.66[c]	12.84[d]	11.56[d]	1.19

[a] Standard error of measure, n = 4 for initial harvest, n = 6 for final harvest and accretion.
[b] Hot carcass.
[c,d,e] Within a row and tissue, means without a common superscript letter differ ($P < 0.05$).
[e] Fat free organic matter.
[f] Ether extract material x 9.4 kcal/g + fat-free organic matter x 5.55 kcal/g.
[g] Final kg – initial kg / days on feed.

Table 2.6. Effect of winter grazing program on steer chemical composition in Exp.2

	Carcass				Offal				Empty body			
	Treatment				Treatment				Treatment			
	HGW	LGW	NR	SEM[a]	HGW	LGW	NR	SEM	HGW	LGW	NR	SEM
Initial												
Mass, kg[b]	225.5[c]	177.5[d]	132.0[e]	5.90	111.7[c]	96.3[d]	73.0[e]	3.46	337.2[c]	273.8[d]	205.0[e]	8.45
Water, kg	126.8[c]	109.5[d]	91.3[e]	4.52	70.3[c]	62.8[d]	53.1[e]	2.43	197.1[c]	172.2[d]	144.4[e]	5.65
FFOM[e], kg	48.1[c]	42.0[de]	32.1[e]	2.06	22.9[c]	20.3[c]	16.0[d]	1.18	71.0[c]	62.3[d]	48.1[e]	2.72
Fat, kg	45.8[c]	22.8[d]	6.4[e]	2.50	16.8[c]	11.5[d]	2.8[e]	0.97	62.6[c]	34.3[d]	9.2[e]	3.11
Ash, kg	4.82[c]	3.26[d]	2.22[d]	0.41	1.60	1.74	1.11	0.34	6.4[c]	5.0[c]	3.3[d]	0.52
P, kg	1.77[c]	1.45[c]	1.00[d]	0.13	0.47	0.50	0.36	0.10	2.24[c]	1.95[c]	1.37[d]	0.14
Energy[f], kcal/kg	3103[c]	2520[d]	1797[e]	121	2552[c]	2289[d]	1583[e]	63	2917[c]	2437[d]	1720[e]	88
Final												
Mass, kg[b]	323.8	313.8	329.5	7.77	155.0	156.5	158.0	3.16	478.8	470.3	487.4	9.13
Water, kg	168.7	157.5	167.1	4.30	89.2	85.9	87.5	1.82	258.0	243.4	254.6	4.97
FFOM, kg	60.9	63.1	64.2	4.13	29.7	29.8	29.7	1.51	90.6	92.9	93.9	4.28
Fat, kg	81.8[c]	86.9[cd]	91.9[d]	2.89	32.6[c]	37.5[cd]	38.9[d]	1.80	114.5	124.4	130.8	4.31
Ash, kg	12.3	6.4	6.2	3.22	3.4[c]	3.3[c]	1.9[d]	0.40	15.7	9.7	8.1	3.42
P, kg	2.12	2.12	1.98	0.14	0.51	0.53	0.62	0.03	2.63	2.65	2.60	0.13
Energy, kcal/kg	3414[c]	3722[d]	3702[d]	88	3048[c]	3305[d]	3351[d]	74	3296	3581	3590	74
Accretion[g]												
Mass, kg/d[b]	1.14[c]	1.19[c]	1.39[d]	0.06	0.50[c]	0.52[c]	0.60[d]	0.03	1.63[c]	1.71[c]	1.98[d]	0.06
Water, g/d	481[cd]	407[c]	539[d]	29	216	195	247	20	668[cd]	602[c]	786[d]	34
FFOM, g/d	147	181	227	41	79	82	97	14	226	263	324	37
Fat, g/d	420[c]	573[d]	592[d]	27	184[c]	231[d]	249[d]	15	604[c]	804[d]	841[d]	38
Ash, g/d	87	27	28	38	21[c]	14[cd]	6[d]	4	108	41	34	40
P, g/d	4.1	5.8	6.9	1.2	0.5[c]	0.2[c]	1.8[d]	0.2	4.5	5.9	8.7	1.2

Energy, $kcal \cdot kg^{-1} \cdot d^{-1}$	3.57^{c}	10.26^{d}	13.43^{e}	0.93	5.73^{c}	8.61^{d}	12.39^{e}	0.65	4.37^{c}	9.75^{d}	13.15^{e}	0.85

[a] Standard error of measure, n = 4 for initial harvest, n = 6 for final harvest and accretion.
[b] Hot carcass.
[c,d,e] Within a row and tissue, means without a common superscript letter differ ($P < 0.05$).
[e] Fat free organic matter.
[f] Ether extract material x 9.4 kcal/g + fat-free organic matter x 5.55 kcal/g.
[g] Final kg – initial kg / days on feed.

Figure 2.1. Steer live BW during winter grazing and the feedlot period in Exp. 1

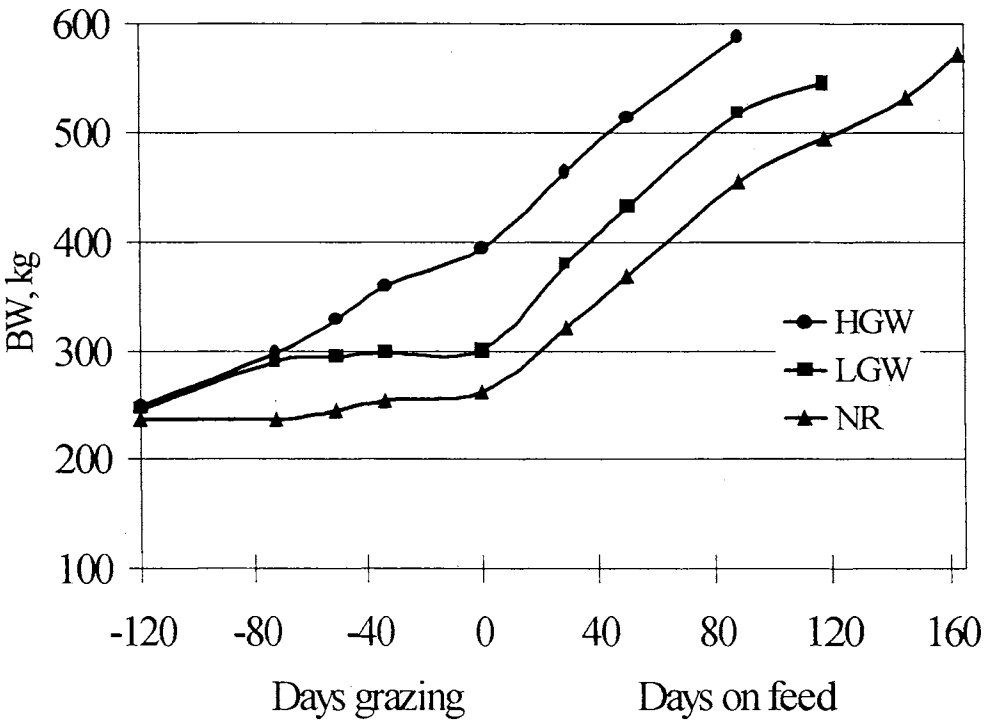
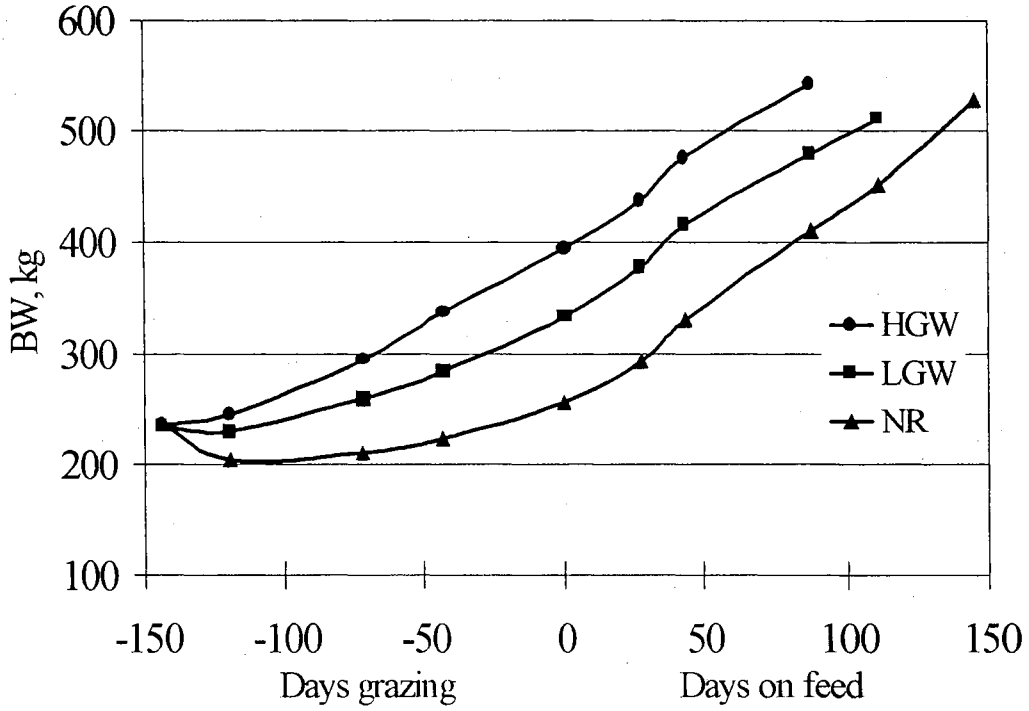


Figure 2.2. Steer live BW during winter grazing and the feedlot period in Exp. 2



Chapter III

EFFECT OF PREVIOUS LIVE WEIGHT GAIN DURING WINTER GRAZING ON VISCERAL ORGAN MASS AND OXYGEN CONSUMPTION IN CATTLE DURING HIGH-GRAIN FEEDING [1,2,3]

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ABSTRACT

We compared organ mass and oxygen consumption by tissues in steers with different previous winter grazing BW gain and organ mass change during finishing. Two experiments were conducted utilizing 48 Angus x Angus-Hereford steers in each experiment. The experimental designs were completely random. In each experiment, steers were randomly assigned to one of three treatments: high rate of BW gain grazing winter wheat (HGW); low rate of BW gain grazing winter wheat (LGW); or grazing dormant tallgrass native range (NR) with 0.91 kg/d of a 41% CP supplement. Steers grazed for 120 or 144 d in Exp. 1 and 2, respectively. Prior to placement into a feedlot in each experiment, four steers from each treatment were randomly selected for initial harvest to measure organ mass and oxygen consumption. All remaining steers were placed into a feedlot and fed to the same backfat endpoint (1.27 cm). Six steers were randomly selected from each treatment for final organ mass and oxygen consumption determination. Initial empty body weight (EBW) was greatest ($P < 0.001$) in HGW steers followed by LGW, and then NR steers in both Exp. 1 and 2 (370 > 280> 226 k g and 345 > 280 > 197 kg, respectively). Initial total gastro-intestinal tract (GIT) (g/kg of EBW) was greater ($P < 0.05$) in NR steers compared with HGW and LGW steers in both exp (73.9 > 65.7 and 80.3 > 63.3 g/kg EBW, Exp. 1 and 2 respectively). However in Exp. 1, total splanchnic tissue (TST) (g/kg of EBW) tended to be greater ($P = 0.06$) in HGW compared with NR steers. Initial liver oxygen consumption was greater in HGW and LGW compared with NR steers (34.5 > 16.9 mL/min), whereas initial small intestine oxygen consumption was greater in LGW compared to HGW and NR steers (12.1 > 5.2 mL/min). Reticulo-rumen oxygen consumption was similar ($P > 0.10$) among treatments.

The decrease of GIT ($g \cdot g\ EBW^{-1} \cdot d^{-1}$) during finishing was greater in NR compared with HGW an LGW steers in both Exp. 1 and 2, but the increases in TST was similar among treatments in each experiment. Differences in the timing of organ mass and lower initial oxygen consumption may be part of the mechanism that affects feedlot growth in steers from different previous grazing programs.

Key words: Cattle, Visceral Organs, Oxygen Consumption

Introduction

A strong positive correlation exists between fasting heat production (maintenance energy requirements) and visceral organ weight in response to plane of nutrition in sheep, rats, and cattle (Huntington and Reynolds, 1987; Burrin et al., 1992; Sainz and Bentley, 1997). This high correlation occurs because while the gastro-intestinal tract tissues (GIT) and liver make up only 8-14% of an animal's live BW (Burrin et al., 1990; Kelly et al., 1993), the GIT and liver is responsible for as much as 40% of the total body oxygen consumption (McBride and Kelly, 1990). This makes the GIT and liver (splanchnic tissues) inproportionally metabolically active compared to their relative contribution to live-weight mass. McBride and Kelly (1990) demonstrated this greater energy consumption to mass ratio when they showed that liver oxygen consumption was 1.2 to 1.6 times greater than that of skeletal muscle. Whereas energy use by the tissues that livestock production favors (i.e. lean skeletal muscle) are necessary, energy use by the splanchnic tissues to an extent is considered a waste of energy, or tax on production

(Reynolds, 2001). Therefore, any changes in visceral organ mass, and by convention energy use, may change the amount of energy and protein available to the animal for growth (Fluharty and McClure, 1997).

We hypothesized that differences in growing cattle with respect to growth and gain efficiency in the feedlot are partially due to visceral organ mass and associated energy expenditure. It was our objective as part of a larger experiment examining feedlot growth in cattle, to determine the effect of different winter grazing programs for growing cattle on organ mass and oxygen consumption by tissues in relation to subsequent feedlot performance, and discern if these were factors in dictating whether differences in growth would occur in cattle.

Materials and Methods

Animals and Management

A complete description of the procedures utilized for the animals is found in Hersom et al., 2002. Briefly, in each of two experiments we randomly allotted 48 fall-weaned Angus x Angus-Hereford steers (244 $\pm$ 23 kg, Exp. 1; 231 $\pm$ 25 kg, Exp. 2) from the same cowherd to one of three winter grazing treatments. Treatments were: grazing winter wheat pasture to achieve a high rate of BW gain (**HGW**; stocking density = 0.43 to 0.55 ha·steer^{-1}); grazing winter wheat pasture and adjusting stocking density to maintain a low rate of BW gain (**LGW**; stocking density = 0.16 to 0.52 ha·steer^{-1}), or grazing dormant tallgrass native range (**NR**; 0.63 ha·steer^{-1}). Steers grazing NR were offered 0.91·kg·steer^{-1}·d^{-1} of a cottonseed-meal based supplement (41% CP). No implants were utilized during winter grazing. At the end of the grazing phase steers were

placed in a feedlot and fed a high-grain finishing diet. Steers were fed to a common endpoint of 1.27 cm of backfat as determined by ultrasound (Aloka, model 210, Aloka probe, model UST-5021; Aloka Co. Ltd., Wallingford, CT) between the 12th and 13th rib on the right side. When the backfat threshold was achieved, all steers in each treatment were harvested within nine days. The Oklahoma State University Institutional Animal Care and Use Committee approved the use of animals and research protocols.

Organ Mass and Tissue Collection

In each experiment, we randomly selected four steers from each treatment to estimate empty body weight (**EBW**), organ mass and oxygen consumption prior to placement in the feedlot. We removed the steers from their respective treatments the morning of harvest. The steers were transported to the Oklahoma Food and Agricultural Products Research and Technology Center (FAPRTC) abattoir. Steers were stunned with a captive bolt and exsanguinated. Weights of blood, hide, head, internal organs, gastro-intestinal tract (**GIT**) organs, mesenteric fat trimmed from GIT organs, GIT contents, trim, and hot carcass were recorded. Contents of the reticulo-rumen and omasum were removed by opening the organ and removing contents by hand, the organs were then washed free of remaining feed particles. Abomasum and intestinal contents were removed by squeezing contents the length of the organ. Empty body weight was calculated as live BW minus weight of GIT contents, and total splanchnic tissue (**TST**) was calculated as the GIT plus liver, spleen, pancreas, and mesenteric fat. In addition, samples of tissues from the center lobe of the liver and duodenum 15 cm distal to the pylorus were collected. The samples were weighed snap frozen in liquid nitrogen, and

subsequently stored at $^{-}80^{\circ}C$ for further analysis. At final harvest, we randomly selected six steers per treatment to estimate final EBW, organ mass, and tissue oxygen consumption. Feedlot personnel removed steers from their pens early in the morning they were harvested, prior to feeding, for transport to the FAPRTC abattoir. The harvest procedure was same as the initial harvest procedure.

Tissue Oxygen Consumption

In Exp 2., during initial harvest we collected additional tissue samples from the liver, ventral sac of the rumen, and duodenum to estimate oxygen consumption. Abattoir personnel collected samples used for oxygen consumption as soon as possible after evisceration; organ contents were removed and organ mass recorded. Timely collection of tissues from some animals after stunning and exsanguinations was precluded by abattoir procedures (up to 50 min after exsanguination). Collected tissues were placed in ice-cold Krebs-Hensleit saline with glucose (**KHS**; Kelly et al., 1993) and transported to the laboratory. The transport media for ruminal epithelial tissues contained 25 mM of HEPES (Harmon et al., 1991). When tissue samples reached the laboratory, laboratory personnel transferred the tissues to fresh KHS at $37^{\circ}C$ bubbled with 95% oxygen gas (Kelly et al., 1993). Ruminal KHS contained approximately 90 mM acetate, 60 mM propionate, and 30 mM butyrate (Harmon et al., 1991). A 50-mg sub-sample of liver tissue was excised using a scalpel and lightly scored (Burrin et al., 1990). Individual ruminal papillae were removed from the rumen epithelial sample to accumulate 50 mg for analysis (Burrin et al., 1990; Harmon et al., 1991) and scored using a scalpel. All visible adipose tissue was removed from the small intestine, which was then cut

longitudinally to open the lumen. Small cross-sections were excised to accumulate 50 mg for analysis (Burrin et al., 1990). Rates of O_2 consumption were measured polargaphically using a Clark-style electrode (YSI model 5300, Yellow Springs Instruments, Yellow Springs, OH) positioned within a thermostatically controlled (37°C) cell chamber (Yellow Springs Instruments, Yellow Springs, OH). Triplicate tissue samples were placed in unoxygenated KHS solution in the O_2 electrode chamber and allowed to acclimate to the chamber for 1 min, after which O_2 consumption was measured over 5 min (Kelly et al., 1993). Triplicate tissue samples were also used to estimate ouabain-sensitive O_2 consumption and cyclohexamide-sensitive O_2 consumption (Kelly et al., 1993). Samples were placed in unoxygenated KHS solution containing 1 x 10^{-4} *M* of either ouabain or cyclohexamide (Kelly et al., 1993) in the O_2 electrode chamber and allowed to acclimate to the chamber for 1 min, after which O_2 consumption was measured over 5 min.

Calculations and Statistical Analysis

We calculated accretion data by subtracting the treatment mean of initial organ mass from the final organ mass of individual steers and dividing by days on feed in the feedlot. Initial, final, and rates of accretion of organ mass and oxygen consumption data were analyzed using the Mixed procedure of SAS (SAS Inst. Inc., Cary NC). The model included the fixed effect of previous winter grazing treatment and the random effect of steer with in treatment, experimental unit was steer. Least squares means were calculated and tested against the error term of steer within treatment. Treatment least squares means

were calculated and means compared using LSD when protected by a ($P < 0.10$) *F*-value. Results were considered significant if $P < 0.05$ and trends if $P > 0.05$ and $P < 0.10$.

Results

Animal Performance

Winter grazing daily BW gains were 1.31, 0.54, and 0.16 kg/d for HGW, LGW, and NR steers, respectively in Exp. 1. During Exp. 2, winter grazing daily BW gains were 1.10, 0.68, and 0.15 kg/d for HGW, LGW, and NR steers. Feedlot performance in Exp. 1 was not different ($P > 0.10$) among treatments (average ADG = 1.80 kg/d, gain:feed = 0.17 kg/kg), however DMI (% of mean BW) of LGW and NR steers was greater ($P = 0.003$) compared with HGW steers. Similarly, in Exp. 2 feedlot performance was not different ($P > 0.10$) among treatments (average ADG = 1.68 kg/d, gain:feed = 0.18 kg/kg), but DMI (% of mean BW) of NR was greater ($P = 0.01$) compared with HGW steers, LGW steers were intermediate.

Initial Harvest

Exp. 1. Initial live BW and the subsequent EBW were 83 and 90 kg greater ($P < 0.001$) for HGW compared with LGW steers, which were 55 and 54 kg greater than NR steers, respectively (Table 3.1). Total offal was similar ($P = 0.27$) among treatments (average = 318 g/kg EBW). However, within the total offal there were several organs that differed as a proportion of EBW between treatments. The mass (g/kg EBW) of HGW steers liver was 16% greater ($P < 0.001$) than livers of LGW and NR steers (18.6 vs. 15.6 g/kg of EBW). In contrast, steers that had been restricted in live BW gain (LGW and NR) tended ($P = 0.06$) to have 7% larger reticulo-rumens (24.3 g/kg of EBW)

compared with HGW steers (22.7 g/kg of EBW). Interestingly, the omasum of NR steers was 43% larger compared with omasums of steers that grazed wheat forage. High gain wheat steers had 11.7 g/kg of EBW greater ($P < 0.001$) mesenteric fat compared with LGW steers, which had 8.3 g/kg of EBW greater mesenteric fat than NR steers. Because of the greater proportions of stomach and small intestinal tissues, NR steers had 11% greater ($P = 0.02$) GIT (g/kg) compared with HGW and LGW steers (73.9 vs. 65.6 g/kg of EBW). Total splanchnic tissue tended to be greater ($P = 0.06$) in HGW steers compared with NR steers, while LGW steers were intermediate.

Exp. 2. Similar to Exp. 1, live BW and EBW of steers entering the feedlot were 62 and 65 kg greater ($P = 0.001$) for HGW compared with LGW, which were 80 and 83 kg heavier than NR steers (Table 3.2). Total offal was 38 g/kg of EBW greater ($P = 0.009$) in NR steers compared with steers that grazed wheat forage (average = 334 g/kg of EBW). However, unlike Exp. 1, the proportion of EBW made up by the liver was similar ($P = 0.26$; average = 16.9 g/kg ofEBW) among treatments. The entire stomach complex (reticulo-rumen, omasum, abomasum) of NR steers was 25% greater ($P < 0.05$) compared with HGW and LGW steers (average = 38.0 g/kg of EBW), whereas the proportion of EBW comprised of small intestine in previously restricted steers (LGW and NR; average = 17.5 g/kg of EBW) was 26% greater ($P = 0.01$) compared with the small intestine of HGW steers. Because of the greater proportions of the stomach complex and small intestine, total GIT in NR steers was 16% greater ($P < 0.001$) compared with LGW steers, which had 12% greater total GIT compared with HGW steers. Initial mesenteric fat in Exp. 2 followed a similar pattern as Exp. 1; mesenteric fat in HGW steers was 7.3 g/kg of EBW greater ($P < 0.001$) than LGW steers, which had 9.8 g/kg of EBW greater ($P <$

0.001) mesenteric fat than NR steers. However, because of divergent patterns in total GIT and mesenteric fat, initial TST was similar (P = 0.50; average = 105 g/kg EBW) among treatments.

Final Harvest

Exp. 1. Even though steers were harvested at similar backfat endpoints (Hersom et al., 2002), live BW of all steers and EBW of steers selected for organ mass measurements were greater (P = 0.02) for HGW and NR steers compared with LGW steers (Table 3.3). Similar to the initial harvest date, total offal was similar (P = 0.78; average 304 g/kg of EBW) among treatments. In addition, organ masses were similar (P > 0.10) among treatments with few exceptions. The exceptions included kidney and mesenteric fat mass. Kidney mass of HGW steers were greater (P = 0.02) than NR steers (2.1 vs 1.9 g/kg of EBW), LGW steers were intermediate. Mesenteric fat mass of HGW steers was greater 23% greater (P = 0.04) than LGW steers (37.3 vs. 28.8 g/kg of EBW), NR steers had intermediate mesenteric fat mass.

Exp. 2. Final live BW of LGW steers tended (P = 0.08) to be lower compared with HGW and NR steers, but EBW mass of steers selected for organ mass measurements was similar (P = 0.44; average = 509 kg) among treatments (Table 3.4). Total offal was also similar (P = 0.34; average 309 g/kg of EBW) among treatments. All components of the offal were similar (P > 0.10) among treatments with the exception of the omasum, which was greater (P = 0.05) in NR steers compared with HGW and LGW steers. The proportion of EBW comprised by mesenteric fat was 17% greater (P = 0.02) in LGW and NR steers (average = 37.8 g/kg of EBW) compared to HGW steers (31.3

g/kg of EBW). Because of the difference in mesenteric fat and similar liver and total GIT, TST was 8% greater (P = 0.01) in LGW and NR (average = 119.5 g/kg EBW) compared with HGW steers (110.2 g/kg of EBW).

Changes in Mass

Exp. 1. Changes in live BW of all feedlot steers and EBW of steers selected for organ mass measurement during the finishing period were similar (P > 0.10; average = 1.79 and 1.76 kg/d) between treatments (Table 3.5). Total offal became a smaller proportion of EBW during the finishing period in all treatments. The decline in total offal of LGW and HGW steers was similar (average = -168 g·g $EBW^{-1}\cdot d^{-1}$) and tended to be 88% greater (P = 0.10) than NR steers. This trend can be attributed to the increased number of days on fed that NR steers spent compared with HGW and LGW steers, because the proportion of total offal at the initiation of the finishing phase and at final harvest were similar among treatments. The liver in HGW steers exhibited a decline that was 85% greater (P < 0.001) compared with LGW and NR steers (-5.7 g·g $EBW^{-1}\cdot d^{-1}$). This greater rate of decline must be attributed to the relative adaptivity of the liver, because HGW steers entered the finishing phase with larger livers, but at final harvest the livers of all treatments were similar, and HGW steers spent the fewest days on feed. Conversely, the reticulo-rumen of HGW steers had a greater (P = 0.05) increase compared with NR steers, whose reticulo-rumen decreased as a proportion of EBW. The total GIT became a smaller proportion of the EBW in all treatments; GIT decline in NR was 55% greater (P = 0.06) compared with steers that grazed wheat (average = -39.5 g·g $EBW^{-1}\cdot d^{-1}$). Mesenteric fat deposition increased in all treatments. The increase in

mesenteric fat was 38% greater (P = 0.05) in NR steers compared with HGW and LGW steers (average = 102.3 g·g $EBW^{-1}\cdot d^{-1}$) despite the greater number of days on feed for NR steers. Coupling declines in total GIT with increases in mesenteric fat resulted in similar (P = 0.54; average = 48.2 g·g $EBW^{-1}\cdot d^{-1}$) increases of TST among treatments. However, when one examines the differences in the change of various organ masses previously described, one can see that different responses occurred to achieve similar TST change.

Experiment 2. Gains of live BW of all steers and EBW of steers selected for organ mass measurements were similar (P > 0.10; average = 1.68 and 1.93 kg/d respectively; Table 3.6). The change in total offal proportion was similar (P = 0.17; average = -292 g·g $EBW^{-1}\cdot d^{-1}$) among treatments. Increases in reticulo-rumen and omasum proportions in wheat steers and relatively small decreases in abomasum proportion resulted in increases (P < 0.05) in the stomach complex of HGW and LGW steers (average = 47.5 g·g $EBW^{-1}\cdot d^{-1}$) whereas the stomach complex of NR steers decreased (-23.4 g·g $EBW^{-1}\cdot d^{-1}$). The change exhibited by small intestine in NR and LGW steers was 80% greater (P < 0.001; average = -41.1 g·g $EBW^{-1}\cdot d^{-1}$) compared with HGW small intestine (-8.0 g·g $EBW^{-1}\cdot d^{-1}$). Differential rates of change in the stomach complex and intestines resulted in varied responses in the total GIT and differences were observed among all treatments (P < 0.001). High gain wheat total GIT increased, LGW total GIT decreased and NR total GIT decreased at a greater rate than LGW (P < 0.001). Similar to Exp. 1, mesenteric fat in LGW and NR steers (average = 163.4 g·g $EBW^{-1}\cdot d^{-1}$) increased 64% greater (P = 0.001) than HGW steers. Total splanchnic tissue proportion of EBW increased similarly (P = 0.45; average = 90.4 g·g $EBW^{-1}\cdot d^{-1}$) in all treatments.

Oxygen Consumption

Prior to placement in the feedlot, rumen papillae oxygen consumption was similar (P= 0.55; average = 5.99 $\mu L \cdot min^{-1} \cdot g^{-1}$ of wet tissue) among treatments (Table 3.7). Despite differences in gross wet weight of reticulo-rumens among treatments, oxygen consumption on a whole organ basis by the reticulo-rumen was similar (P = 0.16; average = 42.63 mL/min), however the small number of samples and large variation among animals could have affected our ability to discern significant differences between the HGW and previously restricted steers. In addition, inhibition of oxygen consumption by either ouabain or cyclohexamide was similar (P > 0.90; average = 34.46 and 21.02%, respectively) among treatments.

Initial small intestine oxygen consumption was 48% greater (P = 0.01) in LGW compared to NR, which was 7% greater (P = 0.01) compared to HGW. Similarly, small intestine whole organ oxygen consumption was 57% greater (P = 0.01) compared with HGW and NR (average = 5.19 mL/min). Inhibition of duodenal oxygen consumption was similar (P > 0.10) among treatments for both ouabain and cyclohexamide (54.1 and 47.1%, respectively).

Oxygen consumption by liver tissue was similar (P = 0.33; average = 6.02 $\mu L \cdot min^{-1} \cdot g^{-1}$ of wet tissue) among treatments. However because of the large differences in gross liver mass between steers that grazed wheat forage and steers that grazed native range forage, oxygen consumption by the whole liver of HGW and LGW steers (average = 34.5 mL/min) tended to be 51% greater (P = 0.09) compared to NR steers. Similar to the other tissues, inhibition by ouabain and cyclochexamide was similar (P > 0.10; average = 23.67 and 39.88%, respectively) among treatments.

At final harvest, the rate of oxygen consumption by ruminal epithelium, small intestine, or liver tissues were similar ($P > 0.10$; average = 5.4, 1.7, 3.2 $\mu L \cdot min^{-1} \cdot g^{-1}$ of wet tissue, respectively). Similarly, final whole organ oxygen consumption by the reticulo-rumen, small intestine and liver were similar ($P > 0.10$; 49.2, 43.1, 10.2 mL/min, respectively).

Discussion

In response to winter grazing treatments, steers entered the feedlot with different EBW. Controlling the amount of forage or grazing low- quality forage affected nutrient supply and ultimately final grazing EBW. Other research (Burrin et al. 1990; Sainz et al. 1995; Nozière et al. 1999) has demonstrated that limitations of DMI or protein will decrease EBW relative to adequately feed animals. The decrease in EBW exhibited by LGW and NR steers relative to HGW steers also caused differences in the proportion of EBW made up by the total offal and by several metabolically important visceral organs. Total offal in Exp. 1 was similar among treatments. However, during Exp. 2, the proportion of EBW comprised by offal tissues was increased in NR steers.

In Exp. 1, HGW steers had unlimited access to high-quality wheat forage and had increased liver size relative to their overall EBW compared with LGW and NR steers which had limited DMI or limited forage quality, respectively. The reduction in proportion of liver through control of DMI has previously been demonstrated in sheep (Burrin et al. 1990; Nozière et al. 1999) and steers (Sainz and Bentley, 1997). Other differences in diet quality have also been reported to affect liver size: increases in dietary protein concentration increased (Fluharty and McClure, 1997) whereas restrictions in

dietary protein concentration decreased (Drouillard et al. 1991; Wester et al. 1995) liver as a percent of EBW. However, a similar pattern in liver proportion of EBW was not observed in the current Exp. 2, which may be explained by the 63 g/kg of EBW increase of total offal in NR steers and the nearly 20 g/kg of EBW greater total offal in LGW steers. Interestingly, McCleod and Baldwin (2000), using different diets with different forage:concentrate ratios but with similar CP:ME ratios, reported sheep had similar liver mass as a percent of EBW. Our data seem to agree with the conclusions of Drouillard et al. (1991) and Sainz and Bentley (1997) in that the liver appears to respond to changes in protein and energy supply.

Conversely, restricted steers in Exp. 1 (LGW and NR) and NR steers in Exp. 2, exhibited increased reticulo-rumen proportion prior to the feedlot phase. Similar results have been observed when DMI and metabolizable energy intake were restricted (Wester et al. 1995; Fluharty and McClure 1997; Nozière et al. 1999). Sainz and Bentley (1997) demonstrated that steers limit-fed concentrate diets had similar stomach weights as steers ad libitum-fed concentrate diets. In the same experiment, steers fed high-roughage-diet had greater stomach weights compared with steers fed concentrate diets at either level. It appears that the stomach complex, particularly the reticulo-rumen responds to dietary energy density. The greatest difference in stomach complex proportions occurred between HGW and NR treatments, which corresponded to abundant high-quality wheat forage and low-quality native range forage.

Differences in the initial proportion of EBW comprised by the small intestine varied between experiments. Small intestine mass in Exp. 1 was similar among treatments, similar to Wester et al. (1995) who demonstrated no differences between

lambs fed diets adequate in energy and protein and protein or energy restricted lambs, and Fluharty and McClure (1997) who reported no differences in small intestine weight between steers fed 100 or 85% of ad libitum. However, work by Drouillard et al (1991) with energy and protein restricted lambs, and Burrin et al. (1990) and Nozière et al. (1999) using different levels of DMI showed reductions in small intestine mass as a proportion of EBW with restriction. Synergistic interactions between DMI and absorbable nutrients presented to the small intestine may ultimately dictate the proportion of the EBW contributed by the small intestine.

The effects of previous live BW gain and differences in EBW on total GIT and TST prior to placement into the feedlot depended on the various organ and tissue complexes. Increases in diet energy density appear to decrease the proportion of GIT, whereas diets of lower energy density increase the GIT (McLeod and Baldwin, 2000). Greater availability of energy and protein to the liver increase the proportion of liver and mesenteric fat (Wester et al. 1995; Fluharty and McClure 1997). Therefore, if sufficient differences in liver, total GIT, and mesenteric fat occur, differences in TST proportion are evident, as observed in Exp. 1. In Exp. 2, similar proportions of liver and divergent proportions of GIT and mesenteric fat resulted in similar TST. In addition, differences in levels of intake have been reported to affect TST mass (Fluharty and McCLure, 1997; and Nozière et al. 1999).

After finishing steers at the same estimated backfat, the proportions of EBW comprised by various organs was remarkably similar among treatments and between experiments. Our similar organ proportions between treatments agree with Drouillard et al. (1991) who ad libitium fed, and re-fed energy and protein restricted lambs to 50 kg

live BW, and Nozière et al. (1999) who re-fed ewes that had been under-fed energy and proteitn and maintenance-fed ewes for 26 d. However, work by Sainz and Bently (1997), in which steers were harvested at 481 kg EBW, demonstrated continued differences in liver, stomach, and intestines of steers with different BW gain prior to finishing. The important exception within our data was mesenteric fat. In Exp. 1 final mesenteric fat was greater in HGW steers compared with LGW steers and NR steers were intermediate. Conversely, during Exp. 2 LGW and NR steers finished with a greater proportion of mesenteric fat compared with HGW steers. The difference in mesenteric fat deposition could be one facet in the mechanism of compensatory growth. With less energy being deposited into fat, more would be available for synthesis of lean tissue. Total mesenteric fat deposition (initial + final mesenteric g/kg of EBW) shows that in Exp.1, HGW steers deposited a greater amount of total mesenteric fat compared with LGW and NR steers (65.1 vs. 44.9 and 42.8 g/kg of EBW). In contrast in Exp. 2, total mesenteric fat deposition by HGW, LGW and NR steers was similar (57.6, 56.9 and 46.9 g/kg of EBW, respectively). McLeod et al. (2002), reported that total energy intake did not effect EBW fat accretion rates. Those authors did report differences in visceral fat accretion due to differences in energy source infused into the abomasum (glucose > hydrolyzed corn starch). Therefore differences in digestibility and carbohydrate escape from the rumen could alter visceral fat accretion. Total GIT and TST proportions were similar among treatments, which indicates that within our experimental populations, organ mass is similar at similar EBW.

Even though final organ proportions were similar among treatments, the rate of change of several metabolically important organs was different among treatments. In

both experiments, total offal became a smaller proportion of the EBW during finishing. In Exp. 1, the HGW and LGW steers decreased total offal by at 8.5-fold greater rate compared with NR steers, whereas in Exp. 2, total offal of NR steers decreased at a 1.7-fold greater rate than HGW and LGW steers. Decreases in the proportion of total offal should not be surprising, especially in the NR steers, considering that when steers entered the feedlot they had low carcass weights relative to their total offal mass. Therefore, at the initiation of the feedlot phase, NR steers possessed adequate organ mass and could accrete carcass weight preferentially to visceral organ weight.

In Exp. 1, decrease in liver proportion during the finishing period was greater in HGW compared with restricted steers. High-gain wheat steers entered the feedlot with large initial liver proportions most likely because of the metabolic demand placed on the liver by grazing wheat pasture. Conversely, the slower decrease in liver proportion in LGW and NR steers in Exp. 1 may have been physiologically required because these steers had to adapt to a greater nutrient load presented to the liver in the early finishing period, compared to the nutrient load the liver was receiving during the winter grazing period. This presentation of nutrients to the liver could also explain the similarities in liver change in Exp. 2, where LGW and NR steers were gaining BW prior to placement into the feedlot (Hersom et al., 2003). Several experiments (Drouillard et al. 1991; Wester et al. 1995; Nozière et al. 1999) have demonstrated increases in liver proportion during realimentation after restrictions of energy, protein, or level of intake. In Exp. 1, the change in small intestine proportion was similar among treatments, whereas in Exp. 2, previously restricted steers had greater decreases in the proportion of small intestine compared with HGW steers. Our data agree with that of Drouillard et al. (1991) who

reported decreases in small intestine (% of EBW) during finishing in control and previously energy or protein restricted lambs. However, other work in sheep (Wester et al., 1995; Nozière et al., 1999) and steers (Sainz and Bentley, 1997) demonstrated increases in the proportion of small intestine in animals that had previously been restricted in intake of energy, protein or DM. In sheep data (Wester et al., 1995 and Nozière et al., 1999) realimentation periods were 14 and 26 d, whereas the steer data (Sainz and Bentley, 1997) is confounded by different previous diet type. In our experiments, an interesting difference among treatments was the increased change in the proportion of reticluo-rumen in HGW steers in Exp. 1 and HGW and LGW steers in Exp. 2 compared with NR steers across the finishing period. Our data suggest that steers with decreased grazing ADG do not have larger GIT, rather a greater proportion of their EBW upon entering the feedlot is comprised of GIT in particular the stomach complex. In Exp. 1, the total GIT in NR steers decreased at a two-fold faster rate compared with LGW and HGW steers during the finishing phase. In Exp. 2, NR steers decreased total GIT at over five –fold the rate compared with LGW and HGW steers, which were increasing total GIT proportion during finishing. Our observation of decreases in total GIT proportion agrees with those observed in control, and energy or protein restricted sheep by Drouillard et al. (1991) and sheep fed at maintenance or 45% maintenance then realimented at over 200% of maintenance by Nozière et al. (1999). Our observation do differ from Wester et al. (1995) and Sainz and Bentley (1997) who observed increases in GIT in previously energy restricted sheep or steers, respectively. Differences among studies may result from timing and type of restriction prior to the realimentation or

finishing phase, length of the re-feeding or finishing phase, and final chosen endpoint for harvest.

During both experiments the proportion of TST increased at similar rates in all treatments. Nozière et al. (1999) reported no increase or a small decrease in TST of ewes realimented after restriction, however the realimentation period was only 26 d and adult animals were used. As previously stated the way in which the change in TST occurred varied between treatments with the exception of the increase in mesenteric fat, yet differences in fat accretion was the moderating factor causing the increase in TST despite decreases in other organ proportions. Increases in TST proportion would indicate an overall increase in the maintenance energy requirements of the cattle during the finishing period (Huntington and Reynolds, 1987). Whereas this is physiologically unavoidable, our data suggests that the timing of the increase in TST and relative proportions of different organs and tissue changes are important and potentially open for manipulation through nutritional and management practices.

The rate of oxygen consumption of ruminal papillae from HGW steers was 30 and 14% greater compared with LGW and NR steers. A 30% difference in ruminal epithelial tissue oxygen consumption between steers fed forage diets at maintenance or two-times maintenance has been reported by Harmon et al. (1991). Similarly, Kelly et al (1993) observed an 18% increase in rate of oxygen consumption 12 h after feeding by ruminal epithelium from steers fed alfalfa hay or bromegrass hay. However, Drouillard et al. (1991) observed similar ruminal papillae oxygen consumption between control and previously energy or protein restricted lambs. Duodenal tissues in LGW and NR steers had increased oxygen consumption relative to duodenal tissue of HGW steers. Our data

are similar to Drouillard et al. (1991) who reported 14 and 12% increase above control lambs in duodenal epithelium oxygen consumption from lambs previously restricted in protein or energy, respectively. McLeod and Baldwin (2000) reported similar duodenal tissue rate of oxygen consumption from sheep fed either maintenance or two-time maintenance of either high-forage or high-concentrate diets. McBride and Milligan (1985) have reported similar results with sheep fed either low or high levels of dietary DE. Our ouabain inhibition data, while not significant, showed a 32% increase in inhibition in HGW compared with LGW and NR steers. This compares favorably with a 13% increase in ouabain inhibition in sheep fed high-DE compared with low-DE diets (McBride and Milligan 1985). The rate of liver oxygen consumption was similar among treatments, however when liver oxygen consumption is viewed on an organ basis, steers that consumed wheat forage had livers that consumed greater amounts of oxygen than did the livers of NR steers. Burrin et al. (1990) reported that in sheep the increase in liver oxygen consumption was a result of an increase in organ size rather than metabolic activity, which agrees with our data.

Final rates and total organ oxygen consumption in ruminal epithelium, small intestine, and liver tissues were not different among treatments at final harvest. Drouillard et al. (1991) and Wester et al. (1995) reported similar oxygen consumption by ruminal papillae, small intestine and liver tissues between unrestricted and energy or protein restricted lambs after re-feeding.

Implications

Winter grazing or growing programs can cause important differences in visceral organ mass. Lower visceral organ mass can lower energy expenditure of metabolically important tissues in steers before placement into feedlots. Differential changes in organ mass, similarities in final total splanchnic tissue mass, and lower initial energy expenditure by visceral organs of previously restricted steers may cause steers to exhibit increased growth during finishing.

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Table 3.1. Mass of visceral organs of steers from different winter grazing programs before placement into the feedlot, Exp. 1

	Treatment[a]				
Item	HGW	LGW	NR	SEM [b]	*P*-value
Live BW, kg	404	311	256	2.4	< 0.001
EBW [c], kg	370[f]	280[g]	226[h]	7.3	< 0.001
	----------g / kg EBW----------				
Total offal	322	324	308	7.0	0.27
Trim	17.0[f]	12.1[g]	10.7[g]	1.28	0.02
Feet and ears	25.9[f]	32.3[g]	33.5[g]	0.59	< 0.001
Hide	68.9[f]	76.7[g]	69.9[f]	1.91	0.05
Blood	37.8	37.5	38.9	1.91	0.84
Head	34.3	40.7	35.1	6.40	0.75
Heart	4.9	4.7	4.9	0.12	0.37
Lung	11.7	14.2	14.5	1.03	0.16
Liver	18.6[f]	15.6[g]	15.5[g]	0.44	0.001
Pancreas	1.3	1.1	1.2	0.08	0.18
Spleen	2.2	2.4	2.5	0.29	0.79
Kidney	3.1	2.8	3.1	0.22	0.55
Reticulo-rumen	22.7[f]	24.2[g]	24.4[g]	0.48	0.06
Omasum	7.6[f]	8.4[f]	14.1[g]	1.4	0.02
Abomasum	3.9	4.5	4.8	0.29	0.14
Small intestine	16.9	17.6	18.3	0.97	0.65
Large intestine	11.0	11.5	11.0	0.68	0.82
Cecum	1.1	1.9	1.3	0.27	0.13
Mesenteric fat	27.8[f]	16.1[g]	7.8[h]	2.06	< 0.001
Total GIT [d]	63.2[f]	68.1[f]	73.9[g]	2.20	0.02
TST [e]	110.6[f]	100.8[fg]	98.4[g]	3.40	0.06

[a] HGW = High gain wheat; LGW = Low gain wheat; NR = Native range.
[b] Standard error of mean, n = 4.
[c] Empty body weight
[d] Gastro-intestinal tract; includes reticulo-rumen, omasum, abomasum, small intestine, large intestine and cecum.
[e] Total splanchnic tissues; includes GIT, liver, pancreas, spleen, and mesenteric fat.
[f,g,h] Within a row, means without a common superscript letter differ (P < 0.05).

Table 3.2. Mass of visceral organs of steers from different winter grazing programs before placement into the feedlot, Exp. 2

Item	Treatment[a]			SEM[b]	*P*- value
	HGW	LGW	NR		
Live BW, kg	395	333	257	5.6	< 0.001
EBW[c], kg	345[e]	280[f]	197[g]	10.1	< 0.001
	----------g / kg EBW----------				
Total offal	324[f]	343[f]	371[g]	8.3	0.01
Trim	16.4	11.7	11.1	3.03	0.43
Feet and ears	29.4[f]	33.6[g]	38.5[g]	1.13	0.001
Hide	77.8	79.5	85.0	3.17	0.29
Blood	34.9	43.5	45.8	2.86	0.05
Head	38.0[f]	44.6[g]	52.1[h]	1.13	< 0.001
Heart	5.0	5.4	5.4	0.21	0.30
Lung	15.0[f]	14.7[f]	18.2[g]	0.93	0.05
Liver	16.2	17.2	17.5	0.57	0.26
Pancreas	1.1	1.2	1.3	0.13	0.32
Spleen	2.0	2.6	2.9	0.48	0.48
Kidney	2.7[f]	3.0[f]	4.0[g]	0.21	0.003
Reticulo-rumen	24.4[f]	25.6[f]	29.3[g]	1.09	0.03
Omasum	7.9[f]	9.9[g]	15.7[h]	0.64	< 0.001
Abomasum	3.8[f]	4.4[f]	5.4[g]	0.24	0.004
Small intestine	13.0[f]	16.6[g]	18.3[g]	1.01	0.01
Large intestine	8.6	9.3	9.6	0.67	0.57
Cecum	1.5	1.5	2.0	0.24	0.28
Mesenteric fat	26.3[f]	19.0[g]	9.2[h]	1.80	< 0.001
Total GIT[d]	59.1[f]	67.4[g]	80.3[h]	2.20	< 0.001
TST[e]	102.6	104.8	108.3	3.32	0.50

[a] HGW = High gain wheat; LGW = Low gain wheat; NR = Native range.
[b] Standard error of mean, n = 4.
[c] Empty body weight
[d] Gastro-intestinal tract; includes reticulo-rumen, omasum, abomasum, small intestine, large intestine and cecum.
[e] Total splanchnic tissues; includes GIT, liver, pancreas, spleen, and mesenteric fat.
[f,g,h] Within a row, means without a common superscript letter differ (P < 0.05).

Table 3.3. Mass of visceral organs of steers from different winter grazing programs at the final harvest date, Exp. 1

	Treatment[a]				
Item	HGW	LGW	NR	SEM[b]	*P*-value
Live BW, kg	563	524	555	7.1	0.02
EBW[c], kg	532[e]	501[f]	528[e]	7.3	0.02
	----------g / kg EBW----------				
Total offal	303	302	308	6.5	0.78
Trim	13.8	16.0	16.9	1.74	0.44
Feet and ears	22.6	23.7	22.4	0.63	0.34
Hide	71.2	70.2	75.9	3.60	0.51
Blood	30.9	29.8	29.7	1.54	0.82
Head	29.6	31.3	30.4	0.39	0.25
Heart	4.3	4.5	4.3	0.20	0.77
Lung	13.6	14.9	14.3	0.75	0.49
Liver	15.1	14.6	14.9	0.05	0.77
Pancreas	0.9	0.9	1.0	0.06	0.27
Spleen	1.9	2.5	2.1	0.26	0.38
Kidney	2.1[f]	2.0[fg]	1.9[g]	0.05	0.02
Reticulo-rumen	23.3	23.9	21.6	0.74	0.12
Omasum	9.1	9.7	10.7	0.89	0.45
Abomasum	3.1	3.8	2.7	0.39	0.14
Small intestine	13.3	12.9	12.9	0.48	0.84
Large intestine	9.8	10.8	9.5	0.49	0.19
Cecum	1.4	1.6	1.8	0.13	0.20
Mesenteric fat	37.3[f]	28.8[g]	35.0[fg]	2.19	0.04
Total GIT[d]	59.9	62.7	59.2	1.78	0.35
TST[e]	113.1	107.1	110.1	3.10	0.41

[a] HGW = High gain wheat; LGW = Low gain wheat; NR = Native range.
[b] Standard error of mean, n = 6.
[c] Empty body weight
[d] Gastro-intestinal tract; includes reticulo-rumen, omasum, abomasum, small intestine, large intestine and cecum.
[e] Total splanchnic tissues; includes GIT, liver, pancreas, spleen, and mesenteric fat.
[f,g,h] Within a row, means without a common superscript letter differ ($P < 0.05$).

Table 3.4. Mass of visceral organs of steers from different winter grazing programs at the final harvest date, Exp. 2

	Treatment[a]				
Item	HGW	LGW	NR	SEM[b]	*P*-value
Live BW, kg	542	511	560	9.9	0.08
EBW[c], kg	512	497	518	11.4	0.44
	----------g / kg EBW----------				
Total offal, kg	303	318	306	7.8	0.34
Trim	15.1	12.2	13.7	1.25	0.29
Feet and ears	23.5	24.1	23.6	0.41	0.56
Hide	73.9	73.5	71.2	1.98	0.31
Blood	27.3	29.6	22.1	3.30	0.29
Head	31.2	31.6	31.4	0.61	0.87
Heart	4.7	5.4	4.9	0.55	0.69
Lung	12.9	15.5	14.3	1.61	0.54
Liver	14.9	15.1	15.7	0.47	0.43
Pancreas	1.0	0.9	1.1	0.07	0.09
Spleen	1.9	2.2	2.1	0.28	0.78
Kidney	2.2	2.5	2.3	0.27	0.75
Reticulo-rumen	28.2	30.4	28.1	1.07	0.25
Omasum	9.5[f]	9.9[f]	11.6[g]	0.57	0.05
Abomasum	3.5	3.6	4.7	0.53	0.25
Small intestine	12.3	11.2	12.1	0.51	0.27
Large intestine	7.9	8.4	7.1	0.60	0.33
Cecum	1.6	1.4	1.9	0.25	0.39
Mesenteric fat	31.3[f]	37.9[g]	37.7[g]	1.65	0.02
Total GIT[d]	62.9	64.9	65.4	1.54	0.50
TST[e]	110.2[f]	118.9[g]	120.0[g]	2.18	0.01

[a] HGW = High gain wheat; LGW = Low gain wheat; NR = Native range.

[b] Standard error of mean, n = 6.

[c] Empty body weight

[d] Gastro-intestinal tract; includes reticulo-rumen, omasum, abomasum, small intestine, large intestine and cecum.

[e] Total splanchnic tissues; includes GIT, liver, pancreas, spleen, and mesenteric fat.

[f,g,h] Within a row, means without a common superscript letter differ ($P < 0.05$).

Table 3.5. Change in mass of visceral organs of steers from different winter grazing programs during finishing, Exp. 1

	Treatment[a]				
Item	HGW	LGW	NR	SEM[b]	*P*-value
Live BW, kg/d	1.79	1.80	1.78	0.06	0.95
EBW[c], kg/d	1.77	1.75	1.81	0.05	0.73
	----------g·g EBW^{-1}·d^{-1}----------				
Total offal	-159^{ef}	-177^{e}	-210^{f}	52	0.10
Trim	-35.1^{e}	31.0^{f}	37.8^{f}	14.3	0.004
Feet and ears	-35.8^{e}	-68.5^{f}	-66.5^{f}	4.8	< 0.001
Hide	13.9	-51.4	35.8	29.6	0.13
Blood	-75.4	-60.6	-55.6	13.9	0.59
Head	-52.0^{e}	-74.9^{f}	-28.3^{g}	5.4	< 0.001
Heart	-6.2	-1.4	3.4	1.7	0.16
Lung	20.9^{e}	5.1^{ef}	1.1^{f}	6.5	0.08
Liver	-38.3^{e}	-7.7^{f}	-3.7^{f}	3.9	< 0.001
Pancreas	-4.6^{e}	-0.9^{f}	-0.9^{f}	0.5	< 0.001
Spleen	-2.9	0.3	-2.2	1.8	0.44
Kidney	-10.7^{e}	-6.6^{f}	-7.6^{f}	0.4	< 0.001
Reticulo-rumen	6.8^{e}	-2.7^{ef}	-16.4^{f}	6.2	0.05
Omasum	16.0^{e}	11.0^{e}	20.8^{f}	7.3	0.006
Abomasum	-9.1	-5.4	-12.7	3.1	0.28
Small intestine	-40.3	-36.5	-32.0	3.7	0.30
Large intestine	-13.7	-6.1	-9.1	4.4	0.48
Cecum	4.1^{e}	-2.8^{f}	2.5^{e}	1.1	0.002
Mesenteric fat	103.6^{e}	101.0^{e}	163.1^{f}	18.7	0.05
Total GIT[d]	-36.3^{e}	-42.6^{e}	-88.4^{f}	15.4	0.06
TST[e]	24.5	49.9	70.1	28.4	0.54

[a] HGW = High gain wheat; LGW = Low gain wheat; NR = Native range.
[b] Standard error of mean, n = 6.
[c] Empty body weight
[d] Gastro-intestinal tract; includes reticulo-rumen, omasum, abomasum, small intestine, large intestine and cecum.
[e] Total splanchnic tissues; includes GIT, liver, pancreas, spleen, and mesenteric fat.
[f,g,h] Within a row, means without a common superscript letter differ (P < 0.05).

Table 3.6. Change in mass of visceral organs of steers from different winter grazing programs during finishing, Exp. 2

	Treatment[a]				
Item	HGW	LGW	NR	SEM[b]	*P*-value
Live BW, kg/d	1.72	1.60	1.71	0.05	0.24
EBW[c], kg/d	1.94	1.86	1.98	0.10	0.69
	----------g·g EBW^{-1}·d^{-1}----------				
Total offal	-245	-226	-406	69	0.17
Trim	-15.7	-17.0	16.0	17.7	0.31
Feet and ears	-68.2[e]	-79.3[e]	-92.2[f]	4.4	0.006
Hide	-45.8	-62.5	-84.8	16.0	0.25
Blood	-88.7	-99.8	-146.3	31.6	0.41
Head	-79.5[e]	-104.5[f]	-127.8[g]	7.0	< 0.001
Heart	-2.7	-0.67	-2.83	4.9	0.94
Lung	-24.8	6.7	-23.8	14.7	0.26
Liver	-15.2	-20.3	-11.3	4.1	0.32
Pancreas	-0.7	-2.7	-1.2	0. 6	0.07
Spleen	-1.7	-3.7	-5.0	2.5	0.64
Kidney	-5.3	-4.8	-11.0	2.4	0.16
Reticulo-rumen	44.2[e]	40.7[e]	7.3[f]	10.8	0.007
Omasum	18.3[e]	3.0[f]	-26.0[g]	5.2	< 0.001
Abomasum	-4.0[e]	-7.3[e]	-4.7	3.5	0.78
Small intestine	-8.0[e]	-44.0[f]	-38.2[f]	5.4	< 0.001
Large intestine	-8.2	-6.0	-15.2	4.6	0.37
Cecum	1.3	-2.2	-0.7	1.6	0.34
Mesenteric fat	59.0[e]	150.7[f]	176.0[f]	18.7	0.001
Total GIT[d]	44.3[e]	-16.0[f]	-91.8[g]	13.6	< 0.001
TST[e]	87.5	111.8	72.0	21.9	0.45

[a] HGW = High gain wheat; LGW = Low gain wheat; NR = Native range.
[b] Standard error of mean, n = 6.
[c] Empty body weight
[d] Gastro-intestinal tract; includes reticulo-rumen, omasum, abomasum, small intestine, large intestine and cecum.
[e] Total splanchnic tissues; includes GIT, liver, pancreas, spleen, and mesenteric fat.
[f,g,h] Within a row, means without a common superscript letter differ (P < 0.05).

Table 3.7. Oxygen consumption by tissues from steers before placement into the feedlot, Exp. 2

Item	Treatment[a]			SEM[b]	*P*-value
	HGW	LGW	NR		
Rumen papillae					
$\mu L \cdot min^{-1} \cdot g^{-1}$	7.02	4.91	6.03	1.51	0.55
whole organ, mL/min	58.66	36.43	32.79	10.33	0.16
ouabain inhibition, %	27.37	39.49	36.52	19.86	0.90
cyclohexamide inhibition, %	25.38	25.58	12.09	29.63	0.91
Duodenum					
$\mu L \cdot min^{-1} \cdot g^{-1}$	1.27[c]	2.63[d]	1.37[c]	0.28	0.01
whole organ, mL/min	5.77[c]	12.06[d]	4.61[c]	1.47	0.01
ouabain inhibition, %	68.78	44.65	48.94	12.71	0.27
cyclohexamide inhibition, %	51.80	52.26	37.25	12.89	0.62
Liver					
$\mu L \cdot min^{-1} \cdot g^{-1}$	6.17	7.00	4.88	0.95	0.33
whole organ, mL/min	35.24	33.77	16.87	5.80	0.09
ouabain inhibition, %	34.22	39.61	24.19	10.83	0.57
cyclohexamide inhibition, %	37.48	36.59	45.57	12.73	0.84

[a] HGW = High gain wheat; LGW = Low gain wheat; NR = Native range.
[b] Standard error of mean, n = 4.
[c, d] Within a row, means without a common superscript letter differ ($P < 0.05$).

Chapter IV

EFFECT OF LIVE WEIGHT GAIN DURING WINTER GRAZING ON BLOOD METABOLITES AND HORMONES DURING FEEDLOT FINISHING OF BEEF STEERS [1,2,3]

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ABSTRACT

Two experiments were conducted utilizing 48 Angus x Angus-Hereford steers in each experiment to determine the effect of previous winter grazing BW gain on concentrations of blood metabolites and hormones during feedlot finishing. In each experiment, steers were randomly assigned to one of three treatments: high rate of BW gain grazing winter wheat (HGW); low rate of BW gain grazing winter wheat (LGW); or grazing dormant tallgrass native range (NR) with 0.91 kg/d of a 41% CP supplement. Steers grazed for 120 or 144 d in Exp. 1 and 2, respectively. Plasma and serum were collected from all steers prior to placement into a feedlot in Exp. 1 and 2, and at six or seven times during feeding in Exp. 1 and 2 respectively. In Exp. 1, initial concentrations of insulin, triiodothyronine (T_3), and thyroxine (T_4) were greater ($P < 0.05$) in HGW steers compared with LGW and NR steers. Concentrations of IGF-I, glucose and plasma urea nitrogen were greater ($P < 0.05$) in steers that grazed wheat pasture compared with NR steers. In Exp. 2, initial concentrations of glucose, T_3, T_4, IGF-I, and leptin were greater ($P < 0.05$) in steers that grazed wheat pasture than NR steers. Concentrations of glucose, insulin, T_3, T_4, and IGF-I increased during the first 49 d in Exp. 1 and the entire finishing period in Exp. 2. Concentrations of glucose were greater ($P < 0.05$, average = 95.01 mg/dL) and insulin less ($P < 0.001$, average = 4.50 ng/mL) in LGW and NR steers compared with HGW steers (82.6 mg/dL, 6.54 ng/mL) on d 49 in Exp. 1, but similar ($P > 0.10$) among treatments in Exp. 2 on d 46. Final IGF-I and leptin concentrations (Exp. 2) were similar ($P > 0.10$, average = 453.67 and 15.56 ng/mL) among treatments. Previous BW gain can affect blood metabolites and hormones in steers entering the feedlot. Lower

concentrations of T_3, T_4, and IGF-I at the initiation of the feedlot period may be associated with growth in steers.

Keywords: Cattle, Grazing, Feedlot, Nutrients, Hormones

Introduction

Occurrence of differences in feedlot growth in cattle may be associated with altered endocrine function. Ellenberger et al. (1989) postulated that alterations in anabolic hormones might induce growth and, Hayden et al. (1993) found a strong correlation between protein anabolic hormones (IGF-I and insulin) and body growth in steers exhibiting increased rate of growth. Nutritional restriction reduces plasma concentrations of triiodothyronine (T_3), thyroxine (T_4), and IGF-I (Ellenburger et al., 1989; Yambayamba et al., 1996) and insulin (Yelich et al., 1995). Decreased concentrations of thyroid hormone have been reported to reduce maintenance energy requirements (Murphy and Loerch, 1994) and decrease protein degradation (Ellenburger et al., 1989). Van den Brande (1986) speculated that animals become more sensitive to anabolic hormones, especially IGF-I, after a period of nutritional restriction. Additionally, Yambayamba et al. (1996) speculated that changes in blood metabolites are not abrupt when animals are re-fed after restriction. The lag in response of blood metabolites might allow increased efficiency of energy use by decreasing the animal's maintenance energy requirements. Decreased maintenance energy requirements through

reduced thyroid function and increased sensitivity and/or responsiveness to anabolic hormones may be mechanisms that cause increased growth in cattle.

In this experiment we examined endocrine and metabolite responses of steers during feedlot finishing. Steers had similar genetics but different BW gains and body fat, resulting from different winter grazing programs prior to placement into the feedlot.

Materials and Methods

Animals and Management

A complete description of the procedures utilized for the animals are in a companion paper (Hersom et al., 2003). Briefly, in each of two experiments, 48 fall-weaned Angus x Angus-Hereford steers (244 $\pm$ 23 kg, Exp. 1; 231 $\pm$ 25 kg, Exp. 2) from the same cowherd were randomly allotted to one of three winter grazing treatments. Treatments were: grazing winter wheat pasture to achieve a high BW gain (**HGW**; 1.31 kg/d Exp. 1 and 1.10 kg/d Exp. 2, stocking density = 0.43 to 0.55 ha·steer^{-1}); grazing winter wheat pasture and adjusting stocking density to maintain a low BW gain (**LGW**; 0.54 kg/d Exp. 1 and 0.68 kg/d Exp. 2, stocking density = 0.16 to 0.52 ha·steer^{-1}), or grazing dormant tallgrass native range (**NR**; 0.16 kg/d Exp. 1 and 0.15 kg/d Exp. 2, stocking density = 0.63 ha·steer^{-1}). Steers grazing NR were offered 0.91 kg·steer^{-1}·d^{-1} of a cottonseed meal-based supplement (41 % CP). Implants were not utilized during winter grazing. At the end of the grazing phase, steers where placed in a feedlot and adapted to a high-grain finishing diet over four weeks. Steers from all treatments were harvested at approximately the same backfat endpoint (1.27 cm). The Oklahoma State University

Institutional Animal Care and Use Committee approved the use of animals and research protocols.

Blood Collection

Three days (d -3) prior to placement into the feedlot in Exp 1 and Exp 2, all steers were removed from pastures and after 5 to 6 h without feed and water blood was collected via jugular venipuncture into two 10 mL Vaccutainer tubes and placed on ice. Blood for plasma was collected into tubes containing sodium heparin and centrifuged (3,000xg for 20 min) within 1 h of collection. Blood for serum was collected into empty tubes and allowed to clot for 16 h at 2 to 8°C before centrifugation. Plasma and serum were stored in polyethylene tubes at –40°C until analysis.

In Exp 1, blood samples for plasma and serum were also collected 3 to 4 h after the steers received one-half of their daily feed allotment on d 14, 21, 28, 35, 42, and 49 of the feedlot period. In Exp 2, blood samples were collected 2 to 3 h after steers received their entire feed allotment for the day on d 26, 46, 67, 86 (HGW, LGW, and NR), 111 (LGW and NR), 132, and 145 (NR) from steers on feed.

Metabolite and hormone assays

Plasma glucose and urea nitrogen (**PUN**) concentrations were determined using a Cobas Mira analyzer (Roche Diagnostic Corporation, Indianapolis, IN). Intra- and interassay CV (n = 6) were 2 and 4% respectively. Serum NEFA concentration was determined by enzymatic colorimetric procedure (Wako-NEFA C, WAKO Chemicals USA, Dallas, TX) with modifications described by McCutheon and Banman (1986).

Intra- and interassay CV (n = 6) were 9 and 18% respectively. Serum T_3 and T_4 was quantified with a solid phase RIA for human T_3 and T_4 (Coat-A-Count, Diagnostic Products Corporation, Los Angles, CA). Assay sensitivity was 10 ng/mL of serum and the addition of 48 ng of T_4 to 25 μL resulted in 96% recovery. When 5, 10, 15, 20, and 25 μL of bovine serum were assayed, concentrations of T_4 were parallel to the standard curve. Intra- and interassay CV (n = 6) were 12 and 18% respectively. Assay sensitivity of T_3 was 0.4 ng/mL of serum and the addition of 15 ng of T_3 to 100 μL resulted in 91% recovery. When 4, 6, 8, and 10 μL of bovine serum were assayed, concentrations of T_3 were parallel to the standard curve. Intra- and interassay CV (n = 6) were 11 and 16% respectively. Concentrations of insulin in serum were quantified by solid phase RIA as described by Bossis et al. (1999). Serum IGF-I concentrations were determined using RIA with acid-ethanol extraction (Echternkamp et al., 1990). Recombinant human IGF-I (R&D Systems, Minneapolis, MN) was used for standards. Intra- and interassay CV (n = 6) were 19 and 18% respectively. Plasma concentrations of leptin were quantified with RIA (Delavaud et al., 2000) using purified recombinant ovine leptin produced as described by Gertler et al. (1998) for standards. Intrassay CV was 5%.

Statistical Analysis

For both Exp., data from d -3 were analyzed as a completely random design using the Mixed procedure of SAS (SAS Inst. Inc., Cary, NC). The statistical model included the treatment as the fixed effect and steer within treatment as a random effect. Treatment least squares means were calculated and means were compared using LSD when protected by a ($P < 0.10$) F-value. Data after placement into the feedlot were analyzed as

a completely random design with the Mixed procedure using a model that included the terms for treatment, day, and the interaction and days were repeated in the analysis. The covariance structure used was autoregressive of order 1 (Littell et al., 1996). In Exp. 1, all sampling dates were included, however in Exp. 2, because treatments were harvested at different dates, only through d 86 was used. If metabolites or hormones had significant treatment x day interactions best fit polynomial response curves were determined and tested for heterogeneity of regression were used to determine differences among HGW, LGW, and NR treatments (Yelich et al., 1995). Treatments response curves were then compared using LSD pair-wise comparisons; HGW vs. LGW, HGW vs. NR, LGW vs. NR. In Exp. 2, the final collection date was analyzed in a similar manner as d –3, to examine differences among treatment at final harvest. Simple Pearson correlations (SAS Inst. Inc., Cary, NC) were determined between leptin concentration and BW, and final leptin concentration and final carcass fat (kg).

Results

Experiment 1

Metabolites. Prior to placement into the feedlot, plasma glucose concentrations of HGW and LGW (average = 88.7 mg/dL) were greater (P = 0.04) than NR (Table 4.1). During d 14 through 42 in the feedlot plasma glucose concentrations were not different among treatments (P = 0.43) with no discernable pattern (treatment x day, P = 0.22).

Plasma urea nitrogen concentrations (Table 4.1) in HGW and LGW (average = 21.3 mg/dL) were greater (P < 0.006) than NR steers (16.31 mg/dL) prior to placement into the feedlot. After steers were placed in the feedlot and on a similar diet PUN

concentrations were similar (P > 0.10) among treatments and remained constant (treatment x day, P = 0.06).

Prior to placement into the feedlot serum NEFA concentrations were greater (P < 0.001) in LGW and NR steers (average = 515 mEq/L) compared with HGW steers (286 mEq/L, Figure 4.1). There was a treatment x day (P < 0.001) effect on concentrations of NEFA in serum during d 14 through 49. Concentrations of NEFA in steers were best described by a quartric regression equation. Analyses of heterogeneity of the response curves of NEFA concentration were different (P < 0.0001) between HGW and NR, and LGW and NR, but were not different (P = 0.29) between HGW and LGW.

Hormones. Prior to placement into the feedlot, serum insulin concentration in HGW steers was greater (2.56 ng/mL, P= 0.002) than LGW steers (1.43 ng/mL); insulin concentration of NR steers was intermediate (1.59 ng/mL; Table 4.2). After d 14 serum insulin concentration of HGW steers (4.74 ng/mL) was greater (P < 0.001) compared with LGW or NR steers (average = 3.26 ng/mL). Additionally, serum insulin concentration increased from day 14 through 49 (P < 0.001). No treatment x day effect was observed (P = 0.11). The Pearson correlation between glucose and insulin was negative for HGW steers (r = -0.006, P = 0.95), positive for NR steers (r = 0.20, P = 0.09), and for LGW steers (r = 0.22, P = 0.04). All treatments exhibited positive Pearson correlations (P < 0.001) between insulin and days on feed (r = 0.58, 0.45, 0.38; HGW, LGW, NR respectively), and only NR steers exhibited a correlation between days on feed and glucose concentration (Exp. 1, r = 0.35, P < 0.002).

Serum T_3 concentrations prior to placement into the feedlot were greatest (P = 0.003) in HGW steers followed by LGW steers which had greater serum T_3

concentrations than NR steers (1.63 > 1.38 > 1.17 ng/mL, respectively; Table 4.2). During the early sampling period, serum T_3 concentrations of HGW steers were greater ($P < 0.005$) than NR steers. At the end of the sampling period, serum T_3 concentrations were not different ($P > 0.12$) among treatments (average = 1.60 ng/mL).

Prior to placement into the feedlot, serum concentrations of T_4 (Table 4.2) in HGW steers (71.7 ng/mL) were greater ($P < 0.001$) compared with LGW and NR (average = 51.9 ng/mL). Early in the feedlot-sampling period, serum T_4 concentrations in HGW steers remained greater ($P < 0.02$) compared with LGW and NR steers. At the end of the feedlot-sampling period HGW and LGW serum T_4 concentrations were greater ($P < 0.02$) compared with NR steers.

Prior to placement into the feedlot, serum IGF-I concentrations of HGW and LGW (average = 469 ng/mL) were two-fold greater ($P < 0.01$) than NR (239 ng/mL). Concentration of serum IGF-I exhibited a treatment x day interaction ($P < 0.001$, Figure 4.2). Concentrations of IGF-I in steers were best described by a quadratic regression equation. The analysis of heterogeneity of response curves of serum IGF-I concentration was different ($P < 0.001$) between HGW and NR, and LGW and NR but was similar ($P = 0.27$) between HGW and LGW. After d 14 the feedlot period the serum IGF-I concentrations of HGW and LGW steers were greater ($P < 0.01$) compared with NR steers. On d 49 of the feedlot period, serum IGF-I concentrations in NR and HGW steers was 443 ng/mL, LGW steers was 285 ng/mL.

Experiment 2

Metabolites. Prior to placement into the feedlot, plasma glucose concentrations (Table 4.3) of HGW and LGW steers were greater ($P < 0.04$) compared with NR steers. During the first 86 d that steers were on feed, plasma glucose concentrations did not differ ($P = 0.86$) among treatments. Plasma glucose at final harvest in NR steers (d 145) was greater ($P < 0.03$) compared with HGW (d 86) and LGW steers (d 111).

Concentrations of NEFA were not different ($P = 0.15$, average = 265 mEq/L) among treatments when steers entered the feedlot. During the entire feedlot period, NEFA concentrations did not differ ($P = 0.55$) among treatments declining to their lowest concentration on d 86.

Prior to placement into the feedlot, PUN concentrations were greater ($P < 0.001$) in LGW (21.3 mg/dL) compared with HGW and NR steers (average = 14.4 mg/dL). Concentrations of PUN (Figure 4.3) exhibited a treatment x day interaction ($P = 0.07$). Plasma urea N concentrations were best described by a cubic regression equation. The analysis of heterogeneity for response curves of PUN was different ($P < 0.02$) between HGW, LGW, and NR treatments. Plasma urea nitrogen concentrations increased in all treatments after d 26. Plasma urea nitrogen concentrations were not different ($P > 0.14$) among treatments for the remainder of the feeding period.

Hormones. Plasma leptin concentrations (Table 4.3) prior to placement into the feedlot in HGW steers were greater ($P = 0.04$) compared with LGW and NR steers (4.55 vs. 2.83 ng/mL). Once steers were in the feedlot, leptin concentrations were not different ($P > 0.10$) between HGW and LGW steers except on d 46 when HGW steers had leptin concentrations that were 2.9 ng/mL greater ($P < 0.001$) than LGW steers. Native range

steers during the feedlot period had leptin concentrations that were 56 to 33% lower ($P < 0.05$) compared with HGW steers through 86 days on feed and 35 to 23% lower ($P < 0.05$) than and LGW steers through 86 days on feed. Serum leptin concentrations were not different ($P > 0.10$) among treatments at final harvest (average = 15.56 ng/mL). We observed a significant correlation between leptin and shrunk BW (Figure 4.6) during the feedlot period ($r^2 = 0.69$, $P < 0.001$). We also observed a significant correlation ($r^2 = 0.53$, $P = 0.02$) between carcass fat (kg) and leptin concentration in steers (Figure 4.7).

Serum insulin concentrations (Table 4.4) were not different ($P = 0.18$, average = 2.47 ng/mL) among treatments before steers entered the feedlot. In the feedlot, insulin concentrations did not differ ($P = 0.24$) among treatments. However, final serum insulin concentrations at harvest were greater ($P = 0.04$) in NR steers compared with LGW steers, HGW steers were intermediate (12.44, 7.78, 9.54 ng/mL, respectively). The Pearson correlation between glucose and insulin was negative for HGW steers ($r = -0.20$, $P = 0.15$), positive for NR steers ($r = 0.45$, $P < 0.001$), and for LGW steers ($r = 0.05$, $P = 0.68$). All treatments exhibited positive Pearson correlations ($P < 0.001$) between insulin and days on feed ($r = 0.48, 0.53, 0.67$; HGW, LGW, NR respectively), and only NR steers exhibited a correlation between days on feed and glucose concentration ($r = 0.51$, $P < 0.001$).

Prior to placement into the feedlot serum T_3 concentrations of HGW and LGW (average = 1.69 ng/mL) were nearly two-times greater ($P < 0.001$) than NR (0.93 ng/mL). Concentration of serum T_3 exhibited a treatment x day interaction ($P < 0.003$, Figure 4.4). Serum T_3concentrations were best described by a linear regression equation. The

analysis of heterogeneity for response curves of T_3 was not different ($P > 0.10$) between HGW, LGW, and NR treatments. During the early feedlot period, serum T_3 concentrations of HGW and LGW steers were greater ($P = 0.03$, average = 1.52 ng/mL) compared with NR steers (1.18 ng/mL). During the remaining feedlot period T_3 concentrations were not different ($P > 0.97$) among treatments. Concentrations of serum T_3 prior to final harvest were greater ($P = 0.004$) in NR compared with HGW steers; LGW steers were intermediate (1.76, 1.39, and 1.60 ng/mL, respectively).

Serum concentrations of T_4 (Table 4.4) prior to placement into the feedlot were greater ($P < 0.001$) in HGW and LGW steers (average = 60.6 ng/mL) compared with NR steers (34.1 ng/mL). During the early feedlot period, serum T_4 concentrations in HGW and LGW steers were greater ($P < 0.006$) than NR steers (average = 65.5 > 50.9 and average = 79.9 > 68.3 ng/mL). Final serum T_4 concentrations prior to harvest were greater ($P = 0.004$) in LGW and NR steers (average = 95.6 ng/mL) compared with HGW steers (77.8 ng/mL).

Prior to placement into the feedlot serum IGF-I concentrations of HGW and LGW (average = 212.8 ng/mL) were 2.5 fold greater ($P < 0.001$) than NR (84.2 ng/mL). Concentration of serum IGF-I exhibited a treatment x day interaction ($P < 0.007$, Figure 4.5). Serum IGF-I concentrations of steers were best described by a quadratic regression equation. Analysis of the response curves of serum IGF-I concentration was different ($P = 0.03$) between HGW and LGW, HGW and NR ($P = 0.001$) but was not different ($P = 0.21$) between LGW and NR. Serum IGF-I concentrations increased in all treatments ($P < 0.001$), apparently more rapidly in HGW and LGW steers compared with NR steers.

After d 46, serum IGF-I concentrations plateaued in HGW and LGW steers, whereas IGF-I concentration in NR steers did not plateau until d 111. Final pre-harvest IGF-I concentrations were similar (P = 0.87, average 455.1 ng/mL) among treatments.

Discussion

In a companion paper we reported that BW gains during winter grazing were 60 and 88% less in LGW and NR than HGW steers in Exp. 1 and 40 and 86% less in Exp. 2. Accordingly, empty body protein and fat masses were reduced in LGW and NR steers compared to HGW steers. The reduced BW gains during winter grazing, especially in NR steers, had the effect of reducing serum hormone, plasma glucose and urea nitrogen and increasing NEFA concentrations relative to HGW steers. Other workers (Ellenberger et al. 1989; Hayden et al. 1993; Yambayamba et al. 1996) have reported a similar response to reduced nutrient intake over an extended period.

Initial plasma glucose concentrations were lower in steers that grazed native range forage compared with steers that grazed wheat pasture. Plasma portal and liver glucose flux has been reported to be stable in beef steers fed 90% ad libitum intake of alfalfa hay or 64% concentrate diets twice daily (Whitt et al., 1996). Therefore, differences in initial plasma glucose concentrations can be ascribed to treatment differences and not a response to post-prandial time interval. Reduced plasma glucose concentrations corresponded with reduced concentration of insulin, which at low concentration is lipolytic (Hornick et al. 1993). The reduction in plasma glucose mostly likely elicited the increased serum NEFA concentrations that served as an alternative energy source.

The elevated concentration of PUN in HGW steers is an effect of the wheat forage diet; wheat forage has a large amount of soluble nitrogen (Horn, 1983) that is quickly absorbed into blood. Low gain wheat steers had elevated PUN concentration prior to placement into the feedlot, which may be a combination of the highly soluble N in wheat forage and mobilization of body tissues to meet energetic demands. Ellenberger et al. (1989) reported elevated blood urea nitrogen concentrations of steers after a 189-d restriction period characterized by gains of 0.4 kg/d. Hayden et al. (1993) observed an increase in glucose and a decline in PUN concentrations during the first 60 d of re-alimentation when steers that were previously energy-restricted for 92 d were adapted to a high-energy diet. In Exp. 1 plasma concentrations of glucose and PUN of LGW and NR steers followed similar trends as described by Hayden et al. (1993). However in Exp. 2, plasma glucose concentrations in LGW steers remained constant, and NR steers exhibiting increasing glucose concentrations. The decrease in NEFA concentrations in both LGW and NR steers in both Exp. 1 and Exp. 2 indicates that steers upon entering the feedlot were returning to a more positive energy balance. Indeed, NEFA concentrations in LGW and NR steers were less than HGW steers that were rapidly gaining BW when they entered the feedlot. The response observed in NEFA concentrations may imply that LGW and NR steers were utilizing dietary energy more efficiently to meet body energy demand rather than mobilizing body energy reserves. Composition data from serial harvest of these steers showed a numerical increase in accretion rate of whole body energy in both LGW and NR steers (Hersom et al., 2002).

Serum insulin concentrations increased in steers from all treatments when steers entered the feedlot. Insulin concentration steadily increased in HGW steers in Exp. 1

through d 49 on feed. In Exp. 2, insulin concentrations in HGW steer were maximal just prior to harvest. Insulin concentrations in LGW and NR steers exhibited steady increases in concentration, but were never greater than HGW steer insulin concentration during the first 49 d in Exp 1 and 132 d in Exp. 2. Hayden et al. (1993) reported that previously restricted steers exhibited linear increases in insulin concentration during realimentation and had greater insulin concentrations compared with normally fed animals after 60 d of realimentation. Perhaps an increased sensitivity to insulin or up regulation of receptors affinity occurred in both LGW and NR steers reducing the amount of circulating insulin required for homeostasis. Insulin resistance by peripheral tissues of steers has been reported (Eisemann et al., 1997). These authors examined insulin responsiveness and sensitivity in beef steers of different age and BW (275 vs. 490 kg) and determined that the metabolism of glucose decreases in sensitivity and responsiveness at heavier BW that also corresponded with increased age and body fat content. Our steers started the feedlot period at the same age however; HGW steers had greater initial body fat content (Hersom et al., 2002) compared with LGW and NR. High gain wheat steers would have maintained increased fat content relative to time in the feedlot compared with LGW and NR steers. Our results agree with Eisemann et al. (1997) in that body fat content could be a factor affecting the insulin response in finishing cattle.

Serum IGF-I concentrations were less in NR steers compared with steers that grazed wheat forage in both Exp. 1 and 2. Breier et al. (1988a) suggested that regulation of circulating IGF-I might be mediated through high-affinity hepatic growth hormone (GH) receptors that are subject to nutritional manipulation. Hayden et al. (1993) reported that after a 92-d energy restriction, GH concentration in restricted steers had increased

45% whereas IGF-I concentration was reduced by 43% compared with steers offered adequate energy for feedlot growth. In addition, a restriction in consumed nutrients has been shown to reduce liver mass in cattle (Drouillard et al., 1991b) and sheep in which IGF-I concentrations were reduced by dietary energy or protein restriction (Wester et al., 1995). Reduced liver mass would then result in a reduced total number of hepatocytes and therefore GH receptors for with which to stimulate hepatic IGF-I synthesis. In addition to the reduction in hepatocytes, protein restriction has been shown to decrease IGF-I concentration through GH dependent post-receptor events (Thissen et al., 1990). The un-coupling of the GH-IGF-I axis has been demonstrated by Breier et al. (1988b) who reported only steers on a high plain of nutrition responded to boluses of GH with increased IGF-I concentration.

Once steers were in the feedlot, IGF-I concentrations in NR steers were less than steers that grazed wheat forage until d 49 in Exp. 1 and d 67 in Exp.2. Breier et al. (1986) and Yambayamba et al. (1996) using intake-restricted steers and heifers, respectively, found that IGF-I concentration in previously energy restricted animals were similar to that of ad libitum fed animals after 10 d of refeeding. Ellenberger et al. (1989) also found a rapid resumption of IGF-I concentration, similar to ad libitum fed steers, in steers that had been restricted to 0.35 kg/d from 242 to 310 kg BW. Ellenberger et al. (1989) observed that previously energy-restricted steers also tended to have greater IGF-I concentrations compared with adequate energy, ad libitum fed steers during the later finishing period, whereas Yambayamba et al. (1996) showed that refed restricted heifers had similar IGF-I concentrations as ad libitum fed heifers. In Exp. 2, IGF-I concentrations of NR steers were similar to HGW steers after d 67, corresponding to

increased growth rate during that period (Hersom et al., 2002). Our results from both experiments agree with that of Hayden et al. (1993), in that IGF-I concentration of previously energy restricted steers was nearly equal to that of adequate energy-fed steers after 60 d of realimentation. One issue controlling the slow increases in IGF-I concentration in both the present experiments and that of Hayden et al. (1993) might be a deficiency in metabolizable protein (**MP**) during the early feedlot or re-feeding period. Drouillard et al. (1991a) speculated that not increasing the CP level of the finishing or re-feeding diet might limit steers from exhibiting their full compensatory growth potential, because MP restricted steers must first replace body N before they can begin protein accretion. Therefore, a restriction in either amino acid supply or energy could limit IGF-I production (Clemmons and Van Wyk, 1981).

During the first 49 d (Exp. 1) and 46 d (Exp. 2) of the feedlot period, LGW and NR steers had consistently lower insulin concentration and NR steers had lower IGF-I concentration compared with HGW steers. During the same period in Exp. 1, LGW and NR steers ADG and gain efficiency were not different compared with HGW steers performance (Hersom et al., 2002). The increased growth rate and efficiency would appear to support the hypothesis by Van der Brande (1986) that nutritional restriction increases tissue responsiveness to IGF-I during refeeding. Stick et al. (1998) found that an increase of 1 ng/mL of serum IGF-I was associated with an increase of 0.00135 kg/d and an 0.0001 kg gain/kg feed increase in efficiency across three levels of feed intake. Particularly in the NR steers, even though circulating IGF-I concentrations were not as great as HGW or LGW steers, the fact that concentrations were increasing may have been adequate to stimulate increased growth rate and efficiency.

The level of energy intake and subsequent BW gains of the grazing steers effected leptin concentrations prior to placement into the feedlot. Leptin concentrations are responsive to energy intake (Daniel et al., 2002; Delavaud et al., 2002). Prior to placement into the feedlot HGW steers (ADG = 1.10 kg/d) had 38% greater leptin concentrations compared with LGW and NR steers ADG = 0.68 and 0.15 kg/d, respectively). Delavaud et al. (2000) reported a 56% decrease in plasma leptin concentrations in ewes that were restricted to 39% of their estimated energy requirement for 65 d and incurred a 3.5% reduction in body fat and –0.21 kg/d BW change. Our steers did not lose BW however, the relative differences in concentrations of leptin in our restricted steers (LGW and NR) and adequate feed steers (HGW) do compare well with leptin concentrations of restricted and well fed ewes in Delavaud et al. (2000). Additionally, a positive relationship between leptin concentration and body composition and fat content has been reported (Houseknecht et al., 1998; Delavaud et. al., 2000). In Exp. 2, the differences in carcass and offal fat content of steers (Hersom et al., 2002) were also reflected in leptin concentrations, i.e. steers with greater fat contents (HGW) had greater leptin concentrations. All treatments increased leptin concentrations with increasing days in the feedlot. Our objective was to harvest all steers at a similar back fat end point and BW. The similarity in final back fat and BW resulted in concentrations of leptin at final harvest that were similar among treatments. Whereas leptin showed steady increases in concentrations, IGF-1 concentrations appeared to plateau in all treatments. Interestingly, the plateau in IGF-1 and steady increases in leptin concentrations could correspond to the decreasing accretion of body protein and continued increased accretion of body fat in maturing animals. The significant correlation between carcass fat and

leptin concentration that we report is similar to the positive relationships between leptin and beef carcass fat (McFadin et al., 2002) and sheep back fat thickness (Daniel et al., 2002) that has been previously found. To date very little research has examined the change in leptin concentrations during the entire feedlot period in cattle. Much of the previous leptin work was concerned with the effects of severe feed restrictions and differences in body composition at single points in time. Our data offer additional insight into the factors related to leptin expression such as initial BW and BW gain, body composition and the change in leptin concentration in beef cattle management systems.

The concentrations of the thyroid hormones prior to placement into the feedlot provide insight into the energy status and gain patterns leading up to the feedlot period. Concentrations of T_3 followed the same pattern as daily BW gains before entering the feedlot. In Exp. 1, concentration of T_3 in HGW steers were greatest, followed by LGW, and lowest concentrations in NR steers that had the lowest BW gains. However, in Exp. 2 when LGW and NR steers prior to entering the feedlot, T_3 levels were similar between HGW and LGW steers. Our data are in agreement with Hayden et al. (1993) who reported that T_3 concentrations are indicative of energy balance, and Murphy and Loerch (1994) who related T_3 concentration to level of intake of a finishing diet. Concentrations of T_4 prior to placement into the feedlot follow the same trend as T_3. Hayden et al. (1993) reported that T_4 appears to be positively associated with energy consumption. A positive relationship between energy intake and T_4 concentration is supported by our data. In Exp. 1, HGW steers had access to abundant forage and had the greatest T_4 concentrations entering the feedlot, whereas NR steers were consuming low quality, dry

native range forage and had the lowest concentration of T_4. Low gain wheat steers' intake of wheat forage was limited, and therefore LGW had intermediate T_4 concentrations relative to HGW and NR steers. Once steers were in the feedlot, T_4 concentrations of HGW steers only increased by 5 ng/mL compared with increases in T_4 concentrations by LGW and NR steers of 21 and 16 ng/mL, respectively. The shifts in T4 concentration in LGW and NR steers demonstrates the increased access to energy that these steers had when allowed to consume ad libitum quantities of feed.

Thyroid hormones synergize with insulin to stimulate protein synthesis, glycogen synthesis and glucose utilization (Griffin and Ojeda, 1992). Additionally, thyroid hormones act permissively with IGF-I to stimulate long bone and cartilage growth (Ellenberger et al. 1993). The initially reduced concentrations of T_3 and T_4 in LGW and NR steers may have optimized the anabolic nature of the thyroid hormones to enhance the actions of insulin and IGF-I, especially with an increased sensitivity to these hormones.

Implications

Growth by previously restricted cattle during feedlot realimentation is dependent upon many factors. In these experiments, the potential decreases in metabolic rate as measured by thyroid hormone concentrations and adequate stimulation of IGF-I production may have occurred to achieve increased body weight gains by the restricted steers. The complex interaction of previous nutrition, re-feeding strategies, and hormonal regulation all influence the extent of growth during realimentation. Interactions of body

composition and hormone concentrations may also signal a decrease in responsiveness to anabolic hormones as cattle reach approach final harvest.

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Table 4.1. Plasma concentrations of glucose and urea nitrogen (PUN) for steers from different winter grazing programs, Exp. 1 [a]

Item	Days on feed							SEM[b]
	-3	14	21	28	35	42	49	
Glucose mg/dL								
HGW	88.5[c]	88.4	85.8	88.1	98.1	91.0	82.6	4.15
LGW	88.9[c]	97.2	87.5	91.7	90.9	92.7	96.0	3.98
NR	73.7[d]	91.9	83.1	87.6	87.4	89.4	94.1	4.33
PUN, mg/dL								
HGW	21.24[c]	7.62	9.11	7.34	8.07	8.42	8.85	0.75
LGW	21.34[c]	5.39	8.14	7.93	9.83	9.14	8.09	0.72
NR	16.31[d]	4.71	7.84	7.75	8.82	8.54	8.50	0.77

[a] Treatment x day ($P > 0.10$).
[b] Standard error of mean, n = 12.
[c, d] Within a column and item, means without a common superscript letter differ ($P < 0.05$).

Table 4.2. Serum concentrations of insulin and thyroid hormones for steers from different winter grazing programs, Exp. 1 [a]

Item	Days on feed							SEM[b]
	-3	14	21	28	35	42	49	
Insulin, ng/mL								
HGW	2.56[c]	3.46	3.78	4.65	4.79	5.22	6.54	0.60
LGW	1.43[d]	3.53	3.40	3.00	2.87	3.79	4.74	0.59
NR	1.59[cd]	3.00	2.59	2.95	2.02	3.04	4.25	0.61
T_3, ng/mL								
HGW	1.63[c]	1.65	1.48	1.46	1.62	1.69	1.62	.080
LGW	1.38[d]	1.37	1.29	1.39	1.44	1.64	1.54	.078
NR	1.17[e]	1.23	1.03	1.18	1.22	1.53	1.57	.083
T_4, ng/mL								
HGW	71.7[c]	67.0	65.0	68.1	66.5	70.7	76.5	4.12
LGW	54.0[d]	54.2	51.7	60.7	61.0	65.7	75.5	3.99
NR	49.7[d]	48.1	39.2	47.2	50.0	56.1	65.9	4.28

[a] Treatment x day ($P > 0.10$).
[b] Standard error of mean, n = 12.
[c, d, e] Within a column and item, means without a common superscript letter differ ($P < 0.05$).

Table 4.3. Plasma glucose, NEFA, and leptin concentrations for steers from different winter grazing programs, Exp. 2 [a]

		Days on feed							
Item	-3	26	46	67	86	111	132	145	SEM[b]
Glucose, mg/dL									
HGW	83.3[c]	67.4	78.1	79.2	77.9[x]	-	-	-	2.2
LGW	78.8[c]	67.0	75.5	82.6	74.0	75.9[x]	-	-	2.1
NR	69.4[d]	64.0	80.3	82.4	79.1	77.1	86.5	86.4[y]	2.1
NEFA, mEq/L									
HGW	240	202	174	166	136	-	-	-	59.9
LGW	248	165	222	202	158	157	-	-	57.4
NR	314	195	194	173	141	176	165	185	57.1
Leptin, ng/mL									
HGW	4.55[c]	7.37	8.86	12.32	16.93				0.80
LGW	2.95[d]	4.97	5.96	9.93	14.61	15.12			0.77
NR	2.71[d]	3.21	3.89	6.59	11.28	10.85	12.47	14.62	0.77

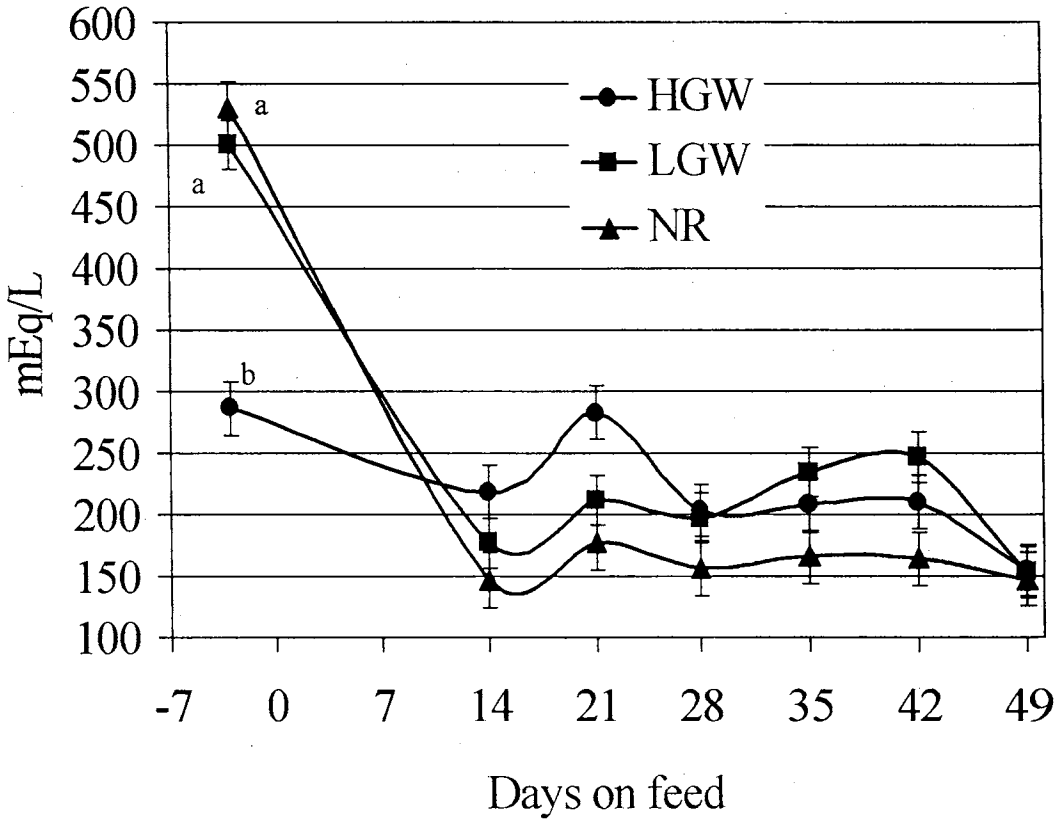
[a] Treatment x day ($P > 0.10$).
[b] Standard error of mean, n = 12.
[c, d] Within a column and item, means without a common superscript letter differ ($P < 0.05$).
[x, y] Final collection date, means without a common superscript differ ($P < 0.05$).

Table 4.4. Serum insulin and T_4 concentrations for steers from different winter grazing programs, Exp. 2 [a]

		Days on feed							
Item	-3	26	46	67	86	111	132	145	SEM[b]
Insulin, ng/mL									
HGW	2.73	6.07	5.94	5.08	9.54[xy]	-	-	-	0.55
LGW	2.58	4.49	4.87	4.97	7.76	7.58[x]	-	-	0.53
NR	2.11	3.53	4.34	4.95	7.69	6.40	8.05	12.44[y]	0.52
T_4, ng/mL									
HGW	60.7[c]	65.6	79.5	77.5	77.8[x]	-	-	-	2.36
LGW	60.6[c]	65.5	80.3	85.9	88.4	100.7[y]	-	-	2.26
NR	34.1[d]	50.9	68.3	72.2	80.6	86.37	85.8	90.6[y]	2.26

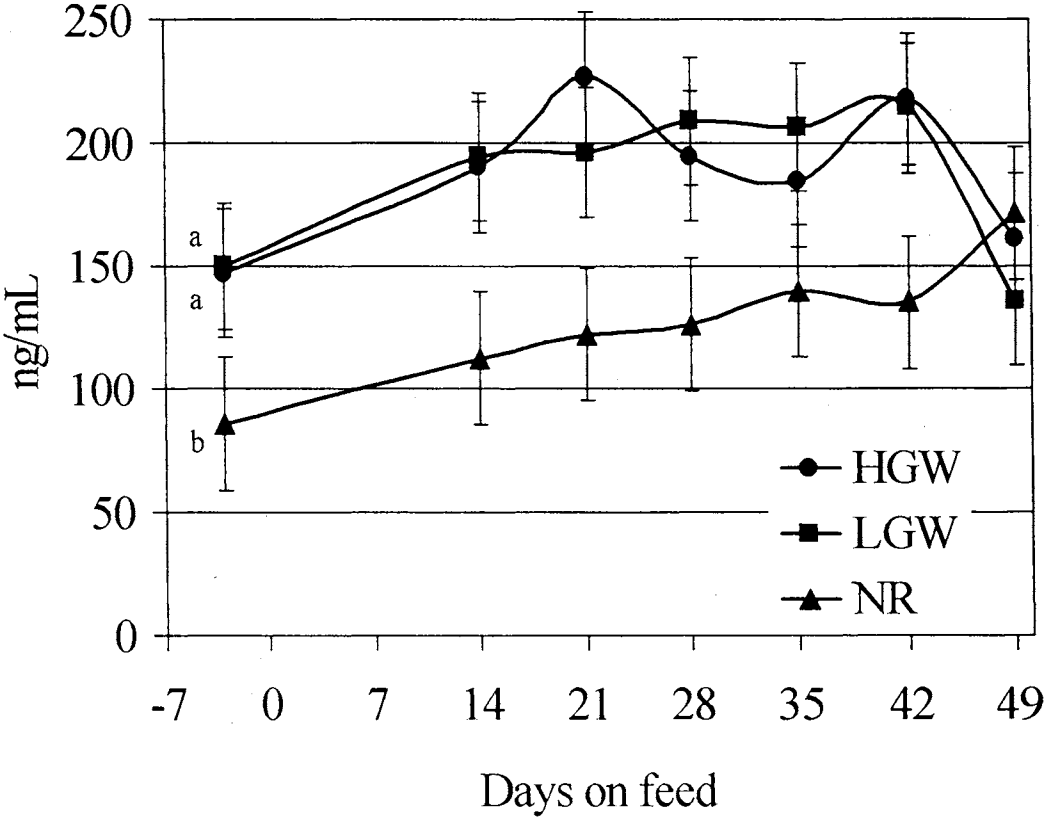
[a] Treatment x day ($P > 0.10$).
[b] Standard error of men, n = 12.
[c, d] Within a column and item, means without a common superscript letter differ ($P < 0.05$).
[x, y] Final collection date, means without a common superscript differ ($P < 0.05$).

Figure 4.1. Serum NEFA concentration of steers from different winter grazing programs, Exp. 1



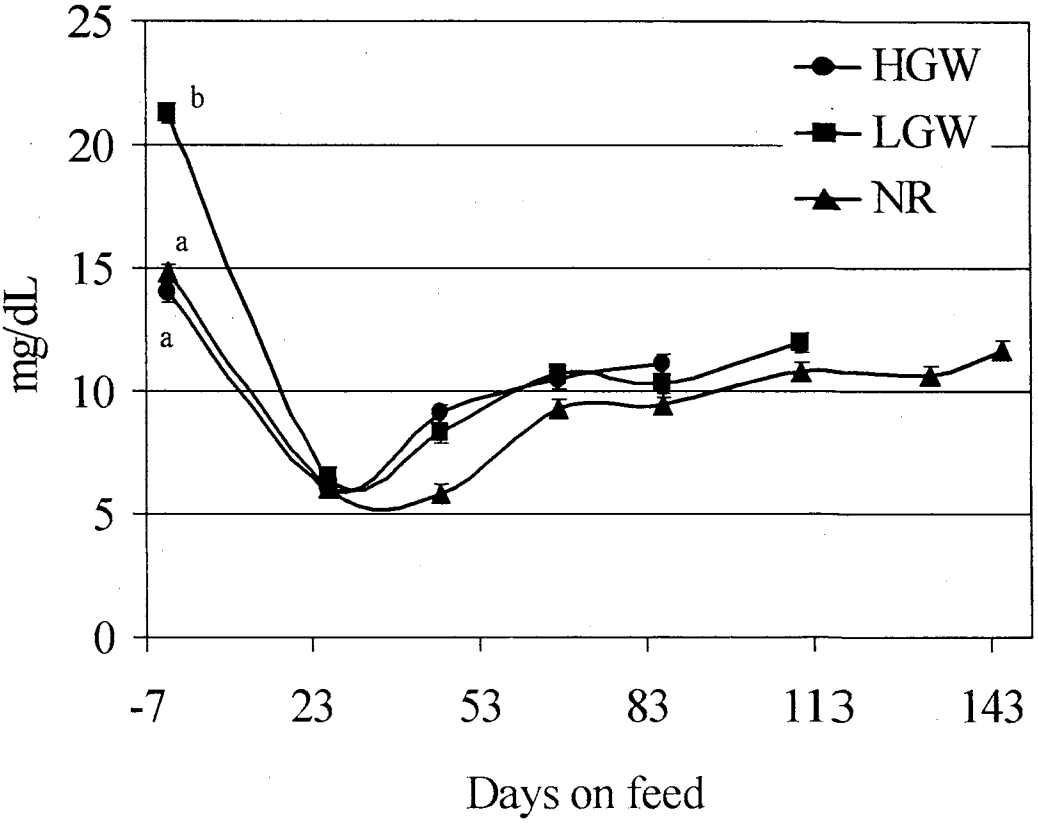
[a,b] Means within a day with different letters differ ($P < 0.05$)
Treatment x day (P , 0.001, quadratic)
HGW and LGW vs. NR, ($P < 0.001$); HGW vs. LGW, ($P = 0.29$)

Figure 4.2. Serum IGF-I concentration of steers from different winter grazing programs, Exp. 1



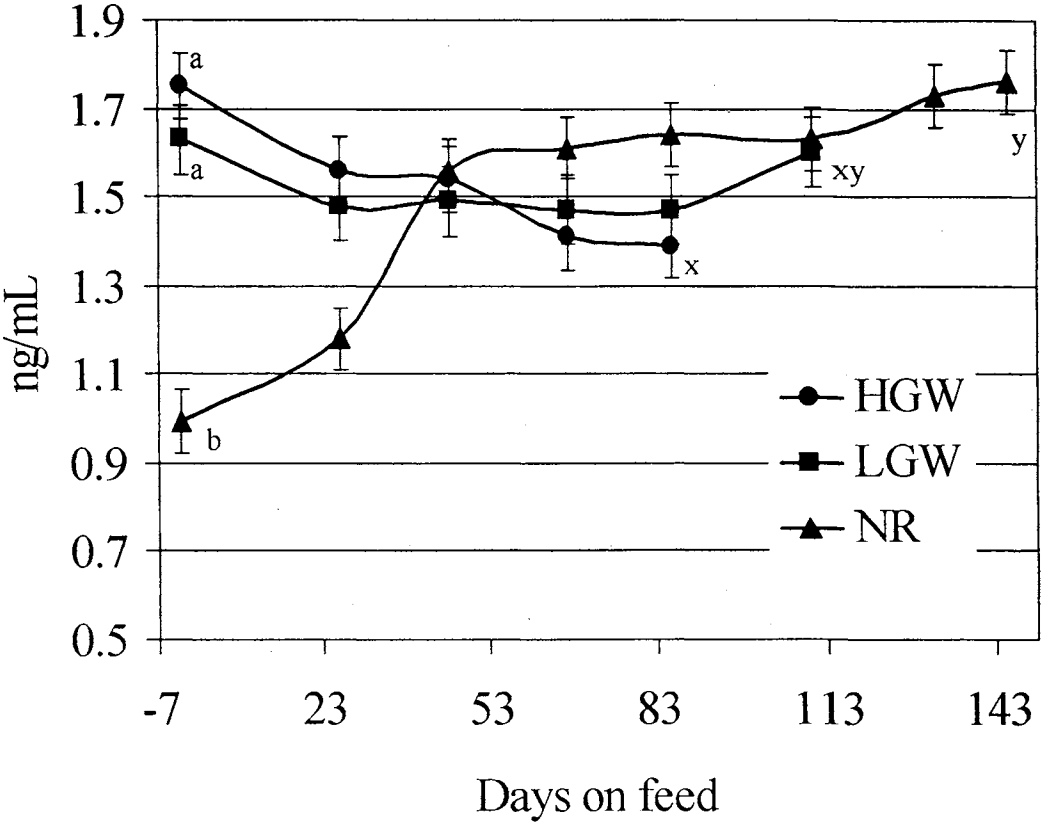
[a,b] Means within a day with different letters differ ($P < 0.05$)
Treatment x day ($P < 0.001$, quadratic)
HGW and LGW vs. NR, ($P < 0.001$); HGW vs. LGW ($P = 0.27$)

Figure 4.3. Plasma urea-N concentration of steers from different winter grazing programs, Exp. 2



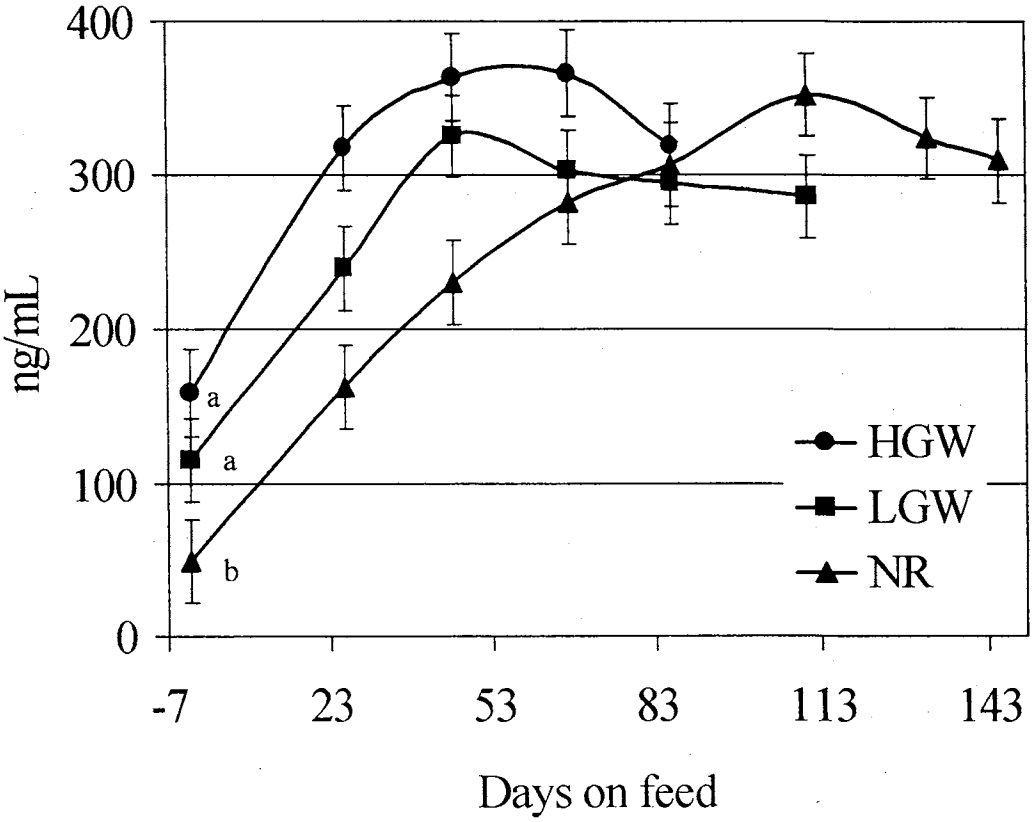
[a,b] Means within a day with different letters differ ($P < 0.05$)
Treatment x days, ($P = 0.07$, cubic)
HGW vs. LGW, ($P = 0.02$); LGW vs. NR, ($P < 0.001$)

Figure 4.4. Serum T_3 concentration of steers from different winter grazing programs, Exp. 2



[a,b] Means within a day with different letters differ ($P < 0.05$)
[x, y] Means of final day with different letters differ ($P = 0.01$)
Treatment x day ($P = 0.003$)

Figure 4.5. Serum IGF-I concentration of steers from different winter grazing programs, Exp. 2



[a,b] Means within a day with different letters differ (P < 0.05)
Treatment x day (P = 0.007, quadratic)
HGW vs. LGW, (P = 0.03); HGW vs. NR, (P = 0.001); LGW vs. NR, (P = 0.21)

Figure 4.6. Relationship of plasma leptin to BW in feedlot steers, Exp. 2

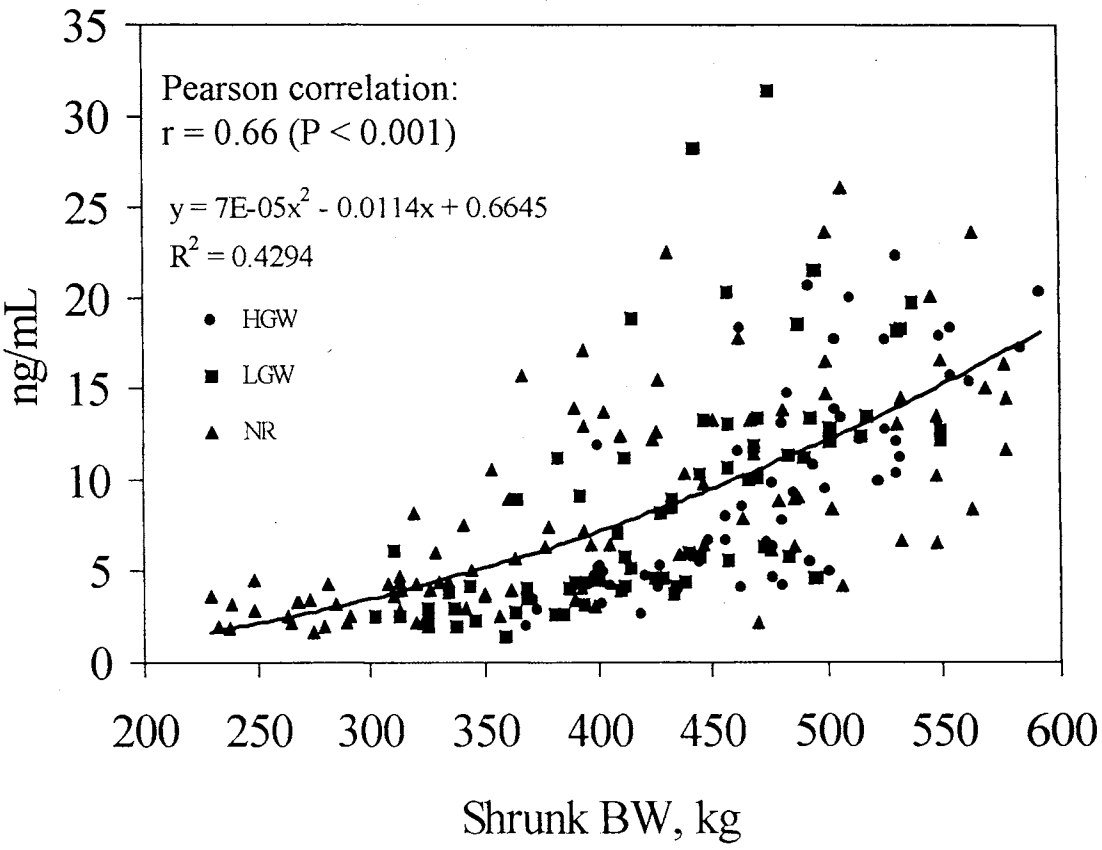


Figure 4.7. Relationship of final plasma leptin to final carcass fat (kg) in feedlot steers, Exp. 2

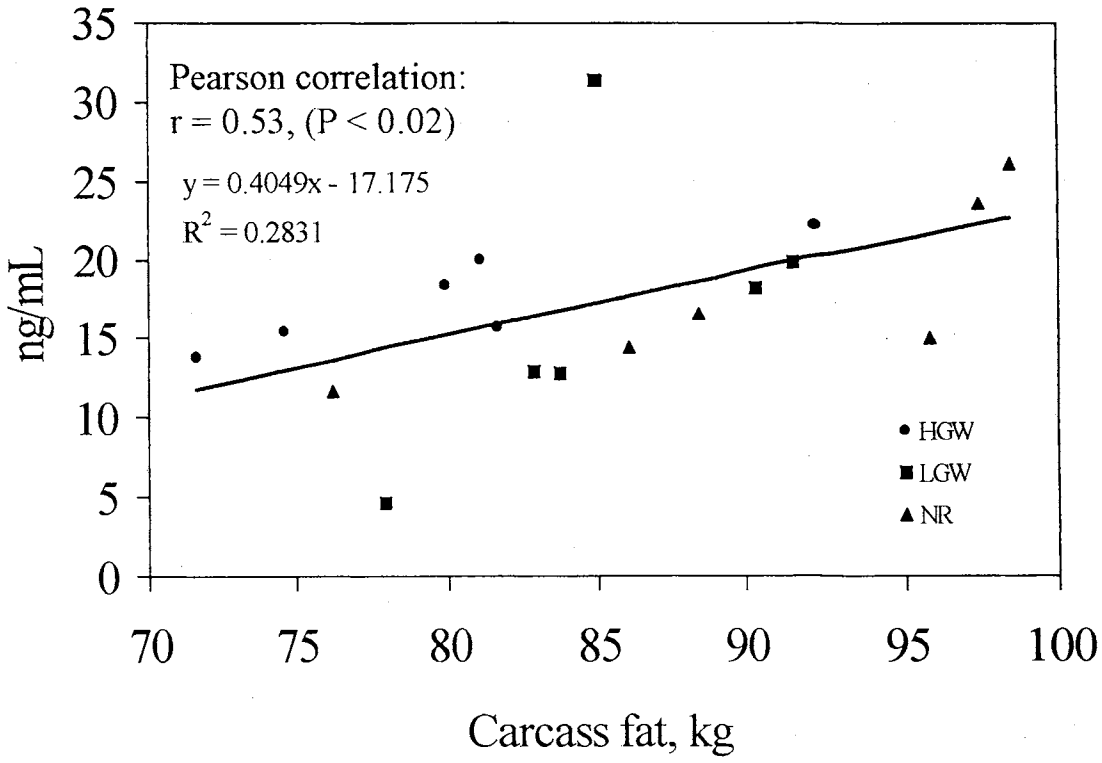
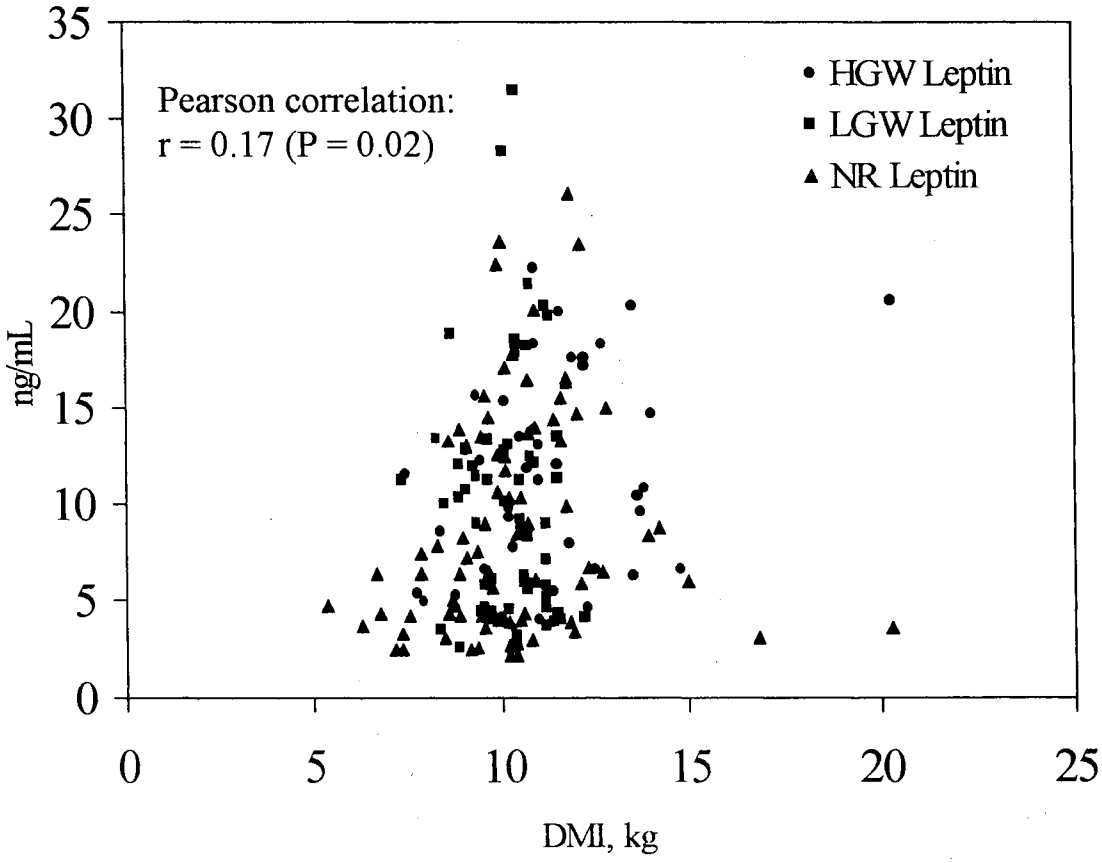


Figure 4.8. Relationship of plasma leptin to DMI in feedlot steers, Exp. 2



Chapter V

EFFECT OF PREVIOUS LIVE BODY WEIGHT GAIN ON ACID/BASE BALANCE, BLOOD FLOW, OXYGEN CONSUMPTION AND NET NUTRIENT AND HORMONE FLUX ACROSS SPLANCHNIC TISSUES DURING ADAPTATION TO A HIGH-GRAIN DIET IN BEEF STEERS[1,2,3]

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ABSTRACT

Ten multicatherized steers (average initial BW = 324 ± 45 kg) were used in a completely random design to determine the effect of previous BW gain on blood flow, oxygen consumption and hormone and metabolite flux across portal-drained viscera and liver of growing beef steers fed a high-grain diet. Treatments were high (**HG**; 1.25 ± 0.14 kg/d) or low (**LG**; 0.73 ± 0.13 kg/d) daily BW gain while grazing winter wheat pasture. Blood flow and net nutrient and hormone flux were determined on d 0, 14, 28, 42, 64, and 92 of the high-grain finishing period. Compensatory growth was evident in LG steers (30% by d 28); ADG and ADG:DMI were greater (P = 0.01; 2.13 vs. 1.31 kg/d; P = 0.005; 0.221 vs. 0.103) from d 0 through 28. Across the 92 d experiment, mean OM digestibility in HG steers was greater (P = 0.01; 80.0 vs. 79.3%) than LG steers. Portal blood flow increased with days on feed (P < 0.001), but was similar between treatments (P = 0.51; 664 L/h). Hepatic blood flow in LG steers was greater (P = 0.06) than HG steers (756 > 654 L/h) and increased (P < 0.001) with days on feed. Similarly, across the feeding period, total splanchnic tissue (**TST**) oxygen consumption was greater (P = 0.002) in LG than HG steers (805 vs. 597 mmol/h). Ammonia, urea-N, and α-amino N flux across TST were similar (P > 0.30) among treatments. Release of glucose from the TST was similar (P = 0.47) between treatments but increased with days on feed (P< 0.001). Insulin PDV release increased (P < 0.001) and hepatic removal of insulin decreased (P = 0.08) in both HG and LG with days on feed. Net insulin flux across TST increased (P= 0.06) with days on feed in both treatments. Leptin (P > 0.39) and IGF-I (P > 0.29) flux across TST was similar. Steers that had moderate BW gains prior to high

grain feeding had increased finishing performance despite lower OM digestibility and increased TST oxygen consumption. Numerically greater TST flux of α-amino N and glucose and numerically greater hepatic removal of lactate might have provided more anabolic precursors and energy for BW gain.

Key words: Cattle, Blood Flow, Nutrient Flux, Compensatory Growth

Introduction

Little information is available regarding the effect of previous live BW gain and compensatory growth on splanchnic tissue metabolism in beef steers. Compensatory growth is observed as increased BW gain resulting from increased DMI and lower maintenance energy requirements (Fox et al., 1972; Carstens et al., 1991). However, these characteristics must have metabolic controls; integration of metabolism into growth characteristics implicates the importance that tissues such as the gastro-intestinal tract and liver have on animal production (Eisemann et al., 1996).

Previous level of nutrition has been shown to affect the mass of splanchnic tissues (Wester et al., 1995; Sainz and Bentley, 1997). Lower dietary intake, energy, and protein restriction has been reported to lower liver and small intestinal mass but realimentation resulted in similar liver mass (Burrin et al., 1990; Wright and Russel, 1991; Sainz and Bentley, 1997). Restriction of intake, energy, or protein has been shown to increase reticulo-rumen mass (% of EBW), however upon realimenation, reticulo-rumen mass did

not differ between adequately fed and previously restricted animals (Drouillard et al., 1991; Sainz and Bentley, 1997).

Similar changes in blood flow and energy expenditure occur in response to changes in splanchnic tissue mass and DMI (Burrin et al., 1989). Splanchnic tissues are key to the absorption, transport, and recycling of nutrients required for growth. Conceivably, compensatory growth in cattle occurs because of changes in blood flow and associated oxygen consumption and nutrient flux across splanchnic tissues.

The incidence of sub acute acidosis is an important consideration in feeding high-grain diets (Elam, 1976). Acidosis occurs in conjunction with excessive consumption of fermentable carbohydrates (Slyter, 1976) that increase the acid load absorbed from the gastro-intestinal tract. Base excess in body fluids work to buffer the increased absorbed acid load (Owens et al., 1998) to maintain blood pH. Occurrence of acidosis depresses animal performance and nutrient absorption (Owens et al., 1998). Increased DMI by compensating steers might possibly cause incidence of sub-acute acidosis compromising animal performance.

The objective of this experiment was to determine the effect of different previous BW gains of steers on blood acid/base balance and blood flow, oxygen consumption, and net nutrient and hormone flux across splanchnic tissues during finishing on a high-grain diet.

Materials and Methods

Animal Management

Ten Angus x Angus-Hereford steers (average initial BW = 324 ± 45 kg, age = 395 ± 11 d) were randomly assigned to one of two treatments. Treatments were high (**HG**; 1.25 ± 0.14 kg/d) or low (**LG**; 0.73 ± 0.13 kg/d) daily BW gain while grazing winter wheat pasture. Stocking density was altered in the LG treatment to maintain the desired BW gain (Hersom et al., 2003). The grazing phase lasted 69 d from February 2 until April 12, 2001.

Prior to the surgery for placement of chronic indwelling catheters, steers were housed in individual, indoor pens (3.5 x 3.5 m) and fed a transition diet (Table 1) to maintain their respective BW gains. Water was provided ad libitum. Catheters were surgically placed in the portal vein, a hepatic vein, a mesenteric vein, and an adjacent mesenteric artery as described by Ferrell et al. (1991). Steers were allowed a minimum of 21 d to recover from surgery prior to the beginning of the initial collection period.

Steers were fed a constant amount of feed to maintain their respective BW gains through the initial blood collection date (d 0). After d 0, steers were adapted to the final diet in three steps by gradually replacing the transition diet with increasing amounts of the final diet (Table 1). Each step lasted six d; steers were fed ad libitum after the d-0 collection with the daily ration split between two feedings at 0800 and 1600. Body weights of steers were taken at the beginning and end of each sampling period. Steers were fed for a total of 92 d. The Oklahoma State University Institutional Animal Care and Use Committee approved all experimental procedures.

Sample Collection

The steers were placed into stanchions in a climate-controlled room 6-d prior to the initiation of blood collection. Total fecal collections were weighed daily for three d prior to blood collection. A sub-sample of the daily fecal collection was taken to form a composite for the 96 h fecal collection period. Simultaneous diet samples were also taken. Blood was collected on d 0, 14, 28, 42, 64, and 92 of the experiment. Five steers were sampled on one day, 3-HG steers and 2-LG steers; the other five steers were sampled on the next day, 2-HG steers and 3 LG steers.

A priming dose of 25 mL of 7% (d 0, 14, 28) or 10% (d 42, 64, 92) para-aminohippuric acid (PAH, pH = 7.4) was administered through a 0.45 μm filter (Millipore, Bedford, MA) into the mesenteric vein catheter at 0700. Para-aminohippuric acid was continuously infused at 0.8 mL/min for eight h (PHD 2000 Syringe pump, Harvard Apparatus Inc., Hollisoton, MA) following the priming dose. Blood was collected hourly from 0800 until 1600. Blood was drawn simultaneously from the portal vein, hepatic vein, and mesenteric artery catheters into syringes and the blood was placed into tubes (BD Vacutainer) treated with sodium heparin (2) and potassium oxalate and sodium fluoride (1). Hourly blood samples were placed on ice for transport to the laboratory. In the laboratory a sub-sample of the hourly whole blood from each site was taken for blood gas analysis (1304 pH/Blood Gas Analyzer, Instrumentation Laboratory, Lexington, MA). Additional hourly sub-samples from each site were used to determine packed cell volume and to form a daily whole blood composite pool. The remaining blood was used for plasma collection by centrifugation (450 x *g*, 4°C, 20 min); plasma was collected and frozen (-40°C) for further analysis.

Sample Analysis

Hourly plasma samples from each site were used to determine PAH, (Harvey and Brothers, 1962) with standards prepared from the infusion solution from each sampling day, α-amino N (**AAN**; Lorentz and Flatter, 1974), and ammonia N concentrations (Sigma chemical Co., St. Louis, Mo; 171-B). Glucose, urea N, total protein, and albumin concentrations were determined from hourly plasma samples using a Cobas Mira analyzer (Roche Diagnostic Corporation, Indianapolis, IN). Lactate (Sigma Chemical Co., St. Louis, Mo; 826-B) and NEFA concentrations (WAKO Chemicals USA, Dallas, TX) with modifications described by Yambayamba et al. (1996) were determined using daily plasma composite samples from each site. Insulin concentrations were determined on daily plasma composites using solid phase RIA (Coat-A-Count, Diagnostic Products Corporation, Los Angles, CA). Bovine pancreatic insulin (Sigma Chem. Co., St Louis, MO) was used for standards (Bossis et al., 1999). Daily composite plasma IGF-I concentrations were determined using RIA with acid-ethanol extraction (Echternkamp et al., 1990). Recombinant human IGF-I (R&D Systems, Minneapolis, MN) was used for standards. Plasma concentrations of leptin were quantified on daily composite samples with RIA (Delavaud et al., 2000) using purified recombinant ovine leptin produced as described by Gertler et al. (1998) for standards. Intra-assay coefficients of variation were as follows: α-amino N, 8.01%; ammonia N, 7.43%; glucose, 3.99%; urea N, 4.17%; total protein, 3.20%; albumin. 1.60%; lactate, 9.42%; NEFA, 26.04%; insulin, 9.09%; IGF-I, 19.27%; leptin, 5.49%. Inter-assay coefficients of variation were as follows: α-amino N, 9.71%; ammonia N, 4.35%; glucose, 1.48%; urea N, 1.40%; total protein, 3.24%;

albumin. 1.23%; lactate, 12.31%; NEFA, 9.11%; insulin, 40.30%; IGF-I, 11.41%; leptin, 6.94%.

Feed and fecal samples were dried at 55°C and ground with a Willey mill to pass a 2 mm screen. Dry matter and ash (AOAC 1990) and N concentration (LECO, St. Joseph, MI) were determined on feed and fecal samples from each sampling period.

Calculations and Statistical Analysis

Plasma flow through the portal drained viscera (**PDV**) and liver was calculated using the Fick principle as outlined by Katz and Bergman (1962): blood flow (**BF**) = $IR^{PAH} / (C_V{}^{PAH} - C_A{}^{PAH})$ in which BF represents plasma flow through the PDV or liver (mL/min), IR^{PAH} is the infusion rate (mg/min) of PAH and $C_V{}^{PAH}$ and $C_A{}^{PAH}$ are the PAH concentration (mg/mL) in venous and arterial plasma, respectively. Portal and hepatic blood flow were calculated directly, arterial blood flow was calculated as hepatic BF – portal BF. Individual plasma flows from any site deviating more than two SD from the mean were deleted and means recalculated (Bohnert et al., 1999). Oxygen concentrations were calculated (Burrin et al., 1989) and net flux of nutrients and hormones (Huntington et al., 1989) across the PDV, liver, and total splanchnic tissues (**TST**) were calculated using the following equations: PDV flux = portal BF x ($portal_{concentration}$ – $arterial_{concentration}$); hepatic flux = portal BF x ($hepatic_{concentration}$ – $portal_{concentration}$) + arterial BF x ($hepatic_{concentration}$ – $arterial_{concentration}$); and TST flux = PDV flux + hepatic flux. A positive flux number indicates a net release, whereas a negative flux number indicates a net uptake of a metabolite or hormone. Hepatic metabolite ratios (**HMR**) were calculated as (hepatic output / hepatic input) – 1 (Bonhert et al., 1999). A negative HMR indicates

the fractional extraction rate of a metabolite by the liver, whereas a positive HMR indicates the fractional increase of a metabolite from what entered the liver.

All data were analyzed as a completely random design using the Mixed procedure of SAS (SAS Inst. Inc., Cary, NC). The statistical model included treatment, day, and the interaction as fixed effects. The experimental unit and random term was steer within treatment. Data was collected repeatedly across experimental days. The spatial power law covariance structure was used (Littell et al., 1996) because of unequal spacing of collection days. Treatment least squares means were calculated and means were compared using LSD when protected by a ($P < 0.10$) F-value. Results were considered significant if $P < 0.05$ and trends if $P > 0.05$ and $P < 0.10$.

Results

Animal Performance

The number of steers sampled during each period and number of catheters patent at each site are listed in Table 2.

High gain steers were 75 kg heavier ($P = 0.002$) than LG steers on d 0 (Table 3). Bodyweight of the HG steers was greater ($P < 0.02$) than LG steers during the entire experiment. Body weight gains by the LG steers tended ($P = 0.10$) to be 28% greater than HG steers during the first 14 d. Similarly, LG steers ADG during the next 14 d (d 14- 28) was 1.05 kg/d greater ($P = 0.006$) than HG steers, and therefore through the first 28 d of the feeding period, ADG of LG steers was 63% greater ($P = 0.01$; 2.13 vs. 1.31 kg/d) than HG steers. From d 28 to d 92, ADG by LG steers was greater ($P = 0.03$; 1.23 vs. 0.94 kg/d). Overall, 92-d ADG tended ($P = 0.06$) to be greater for LG steers than HG

steers (1.28 vs. 1.06 kg/d). Dry matter intake up to and including d 0 was held constant to maintain pre-sampling ADG in both HG and LG steers. On d 14, HG steers tended (P = 0.09) to have greater DMI (% of BW) compared with LG steers (Figure 5.1), on d 28 DMI (% of BW) of LG steers was 16% greater (P = 0.003) than HG steers. After d 28, DMI (% of BW) was similar (P > 0.20) between treatments. Low gain steers had gain efficiencies that were 84% and two-fold greater (P = 0.01 and 0.02, respectively) during the first 14 and 28 d of the feeding period compared with HG steers. From d 42 through 92 gain efficiency was similar (P > 0.50) between treatments. However, 92-d gain efficiency was 37% greater (P = 0.005) for LG compared with HG steers (average = 0.185 vs. 0.135).

During the feeding period OM digestibility (**OMD**) was similar (P > 0.10) between treatments, but overall mean OMD by HG steers was greater (P = 0.01; 80.0% ± 0.17, 79.34% ± 0.09) than LG steers. In both treatments OMD increased from d 0 and peaked on d 28. Digestible OM intake (**DOMI**) on d 0 was greater (P = 0.01) by HG steers compared with LG steers. After d 0, DOMI was similar (P > 0.10) between treatments except on d 42 when DOMI was 51% greater in HG steers than LG steers. Mean DOMI by HG steers was 18% greater (P = 0.003) than LG steers (average = 13.6 vs. 11.5 kg/d).

Blood Gases

Arterial blood pH was similar between treatments (P = 0.47) and across days (P = 0.58); mean arterial pH was 7.42 during the 92-d experiment (Table 4). Similarly, portal blood pH was similar between treatments (P = 0.31) and across days (P = 0.11). Hepatic

blood pH was similar between treatments (P = 0.89) but increased (P = 0.04) as days on feed increased. Partial pressure of carbon dioxide ($\mathbf{pCO_2}$) was similar (P > 0.10) between treatments in arterial, portal, and hepatic blood. All three sites exhibited day effects (P = 0.06, 0.01, and 0.002 for arterial, portal, and hepatic, respectively) whereas pCO_2 decreased with increasing days on feed. Portal (P = 0.01) and hepatic (P = 0.07) blood exhibited treatment x day effects; HG steers had greater decreases in pCO_2 from d 0 to 92 compared with LG steers. Calculated bicarbonate levels in arterial, portal, and hepatic blood did not differ (P > 0.10) between treatments. Portal blood bicarbonate decreased (day effect P = 0.05) as days on feed increased. Arterial blood base excess in LG steers tended (P = 0.08) to be greater than HG steers until after d 42; on d 64 and 92 they were similar. Portal blood base excess exhibited a sharp decline in HG and LG steers until d 28 and 42, respectively (day effect; P = 0.02). Although not significant (P = 0.17), hepatic base excess followed a similar pattern. Oxygen saturation of arterial, portal, and hepatic blood was similar (P > 0.10; average = 98.6, 78.9, and 66.8%, respectively) between treatments. Oxygen saturation in both portal (P = 0.02) and hepatic (P 0.05) blood increased with days on feed in both treatments, peaking on day 42. Hemoglobin concentration in arterial, portal, and hepatic blood was similar (P > 0.10) between treatments during the feeding period (average = 9.76, 9.38, 9.47 g/dL). However, hemoglobin concentrations in arterial (P = 0.001), portal (P = 0.001), and hepatic (P = 0.06) blood did increase by 24, 26, and 27% with days on feed. Packed cell volume (**PCV**) was similar and remained constant in arterial blood in both treatments (treatment effect; P = 0.46; day effect; P = 0.43). Portal PCV increased (P = 0.02) with increasing

days on feed and the increase in HG steers tended to be greater (treatment x day effect, P = 0.07) than in LG steers.

Blood Flow and Oxygen Consumption

Arterial BF (L/h) was not different (P = 0.22; average = 83.5 ± 17.8 and 115.3 ± 16.0 L/h, HG and LG, respectively) between treatments during the 92-d feeding period (Table 5). Portal BF was similar (P = 0.51) between treatments, however portal BF increased (day effect, P < 0.001) after d 0, peaking on d 64 in both treatments. Hepatic BF in LG steers tended (P = 0.06) to be greater than HG steers, but both treatments had increased (P < 0.001) hepatic BF with increasing days on feed. Blood oxygen concentrations (mmol/L) of arterial (P = 0.86), portal (P = 0.81), and hepatic (P= 0.81) blood were similar between treatments. However, oxygen concentration increased (P = 0.001, 0.001, and 0.02; for arterial, portal, and hepatic, respectively) with increasing days on feed. Peak oxygen concentration in HG steers occurred on d 42 in arterial and portal blood, and on d 64 in hepatic blood. Low-gain steer peak oxygen concentration occurred on d 70 in all three sites. Oxygen consumption (mmol/h) by the PDV was similar (P = 0.47) between treatments and across days (P = 0.69). Mean PDV oxygen consumption was 367 mmol/h. Hepatic oxygen consumption by LG steers was 22% greater (P < 0.001, average = 441 mmol/h) than HG steers during the finishing period. Because of the difference in hepatic oxygen consumption, TST oxygen consumption in LG steers was 26% greater (P = 0.002, average = 805 mmol/h) than HG steers. Oxygen consumption by TST tended (P = 0.06) to increase with days on feed. High gain steers exhibited a steady

increase in TST oxygen consumption from d 0 to 64, whereas LG steers increased TST oxygen consumption from d 0 to 28 and then from d 42 to 92.

Metabolite Concentrations

Arterial and hepatic ammonia plasma concentrations were similar ($P > 0.10$) between treatments and across the finishing period. Portal ammonia concentration in HG steers was numerically ($P = 0.11$) greater than LG steers. Portal plasma ammonia concentration decreased in both treatments after d 0 and continued to decrease in HG steers until d 42 and then increased, whereas in LG steers after d 14 portal plasma ammonia concentrations generally increased until d 92 (treatment x day effect, $P = 0.06$). Urea-N concentration in HG steers was 19, 19, and 14% greater ($P < 0.05$) in arterial, portal, and hepatic plasma, respectively, than LG steers. Hepatic urea-N concentrations numerically decreased ($P = 0.12$) across days. Arterial AAN concentration was similar ($P = 0.25$, average = 3.52 mM) between treatments. Portal AAN concentrations tended ($P = 0.08$) to be greater in LG compared with HG steers (average = 3.69 vs. 3.56 mM). Concentrations of AAN increased from d 0 to d 28 in arterial, portal, and hepatic plasma in both treatments ($P = 0.02$, 0.0003, 0.002, respectively), thereafter concentrations declined until d 92. Albumin and total protein concentrations in arterial, portal, and hepatic plasma were similar ($P > 0.10$; average albumin = 516, 514, and 513 mM; average total protein = 66.8, 66.6, and 66.1 g/L) in both treatments across the finishing period.

Glucose concentrations in arterial, portal and hepatic plasma were similar ($P > 0.50$) between treatments. However, plasma glucose concentrations increased ($P \leq 0.02$)

from d 0 to 92 by 9, 9, and 14% in arterial, portal, and hepatic plasma, respectively. Similar to glucose concentrations, arterial, portal, and hepatic plasma lactate concentrations were similar ($P > 0.26$) between treatments. Lactate concentrations decreased by 22 and 25% in arterial and portal plasma from d 0 to 92 ($P \leq 0.004$), and by 31% in hepatic plasma ($P = 0.10$). Non-esterified fatty acid concentrations, like other energy metabolites, were similar ($P \geq 0.19$) between treatments in arterial, portal, and hepatic plasma. The arterial and portal blood NEFA concentrations decreased ($P \leq 0.05$) by 22 and 8 mM, respectively, from d 0 to 92. Hepatic plasma NEFA concentrations numerically ($P = 0.12$) decreased with increasing days on feed.

Hormone Concentrations

The concentration of arterial and portal plasma insulin was similar ($P \geq 0.14$, average = 0.315 and 0.383 ng/mL, respectively) between treatments. However d 0 insulin concentrations in arterial and portal plasma of HG steers were greater ($P < 0.05$) than LG steers. Hepatic plasma insulin concentration in HG steers was greater ($P < 0.001$, average = 0.450 vs. 0.323 ng/mL) than LG steers. Insulin concentration, similar to glucose concentration, increased ($P < 0.001$) with increasing days on feed in arterial, portal, and hepatic plasma in both treatments. Insulin concentration in hepatic plasma of HG steers increased up to d 64, whereas hepatic plasma insulin concentrations in LG steers increased up to d 92. Insulin-like growth factor-1 in arterial, portal, and hepatic plasma was similar ($P > 0.12$, average = 131.4, 134.6, and 136.3 ng/mL, respectively) between treatments and was similar ($P > 0.20$) across the finishing period. Leptin concentrations in arterial, portal, and hepatic plasma were similar ($P > 0.24$, average =

5.32, 5.36, and 5.48 ng/mL, respectively) between treatments. Plasma leptin concentrations in arterial, portal, and hepatic plasma increased ($P < 0.02$) in both treatments as days on feed increased. The decrease in leptin concentration in LG steers on d 42 occurred simultaneous to a decrease in OMI.

Metabolite Flux

Ammonia release across the PDV (Table 8) was similar ($P = 0.92$, average = 104 mmol/h) between treatments and across the finishing period ($P = 0.71$). Uptake of ammonia by the liver was similar ($P = 0.75$; average = -95.1 mmol/h) between HG and LG steers and was similar ($P = 0.55$) across the 92-d experiment. The liver was extracting 89.8% of the ammonia that was presented to it by the PDV. A greater mean PDV flux compared to hepatic flux resulted in a positive TST flux that was similar ($P = 0.44$, average = 14.6 mmol/h) in HG and LG steers and across the finishing period ($P = 0.79$). The HMR was not different ($P = 0.44$, average = 0.339) between HG and LG steers. The HMR exhibited a day effect ($P = 0.03$) decreasing 34% from d 0 to d 70.

Mean plasma urea-N PDV flux in HG steers tended ($P = 0.09$) to be greater than LG steers. Hepatic flux of urea-N was variable across the feeding period ($P = 0.63$) and between treatments ($P = 0.98$, average = 35.6 mmol/h). During the early feeding period the liver of HG steers was taking up urea-N from the GIT, whereas the liver of LG steers was releasing urea-N for recycling. Urea-N TST flux was positive and similar between treatments ($P = 0.49$, average = 142.0 mmol/h) and across the finishing period ($P = 0.94$). The urea-N HMR did not differ ($P = 0.96$, average = 0.011) between HG and LG steers and similar across the finishing period ($P = 0.82$).

Net release of AAN across the PDV (P = 0.60, average = 83.3 mmol/h) and liver (P = 0.20, average = -35.5 mmol/h) did not differ observed between HG and LG steers during the entire finishing period. There was a tendency (P = 0.10) for a day effect in hepatic AAN flux; LG steers had AAN positive flux on d 28, 42, and 92, whereas HG steers had positive hepatic flux on d 42. Total splanchnic tissue AAN was similar (P = 0.33) between treatments but tended to have a day effect (P = 0.11). Mean AAN HMR was similar (P = 0.12) even though HG steers had a negative HER (-0.044) whereas LG steers had a positive HMR (0.012) across the 92 d experiment. The positive HMR in LG steers was attributable to the 9 and 20% increase in AAN releases on d 28 and 42.

Albumin flux across the PDV (P = 0.98), liver (P = 0.54), and TST (P = 0.49) was similar (average = 603.1, -1,547.4, and –1,179.8 mmol/h, respectively) between HG and LG steers. Albumin HMR did not differ (P = 0.61; HMR > 2%) between HG and LG steers. Total protein PDV flux (P = 0.65, average = -107 g/h), hepatic flux (P = 0.99, average = -328 g/h), TST flux (P = 0.65, average = -568 g/h) were similar between treatments. Hepatic flux did exhibit a treatment x day trend (P = 0.11); hepatic uptake by the liver in HG steers tended to decrease with days on feed, whereas LG removal tended to increase with day on feed. Total protein HMR were similar (P = 0.81) between treatments and across the finishing period (P = 0.43).

Removal of glucose by the PDV was similar (P = 0.51, average = -43.1 mmol/h) between HG and LG steers. Portal-drained viscera glucose flux tended (P = 0.11) to increase with days on feed in both HG and LG steers. Peak PDV glucose removal occurred on d 64 and was 82% greater than on d 0. Hepatic glucose flux was similar (P = 0.99) between treatments. However glucose release by the liver increased with days on

feed ($P < 0.001$). Peak hepatic glucose flux occurred on d 64 for HG steers, which was 85% greater than d 0; peak hepatic glucose flux occurred on d 28 for LG steers and was 71% greater than d 0. Because of the large hepatic glucose flux, TST glucose flux resulted in a similar ($P = 0.47$) mean net release of glucose of 232 mmol/h between treatments. Peak TST glucose release occurred on similar days (day effect; $P < 0.001$) as peak hepatic glucose flux (HG = d 64, LG = d 28). Similar to hepatic and TST glucose flux, glucose HMR was similar ($P = 0.84$, average = 0.088) between treatments, but increased with days on feed ($P < 0.001$).

Lactate PDV flux did not differ ($P = 0.37$, average = 76.4 mmol/h) between treatments. However release of lactate by the PDV tended ($P = 0.09$) to exhibit a day effect; lactate flux increased from d 0 to 14 in both HG and LG steers, whereas after d 14 lactate PDV flux declined. Hepatic lactate removal was similar ($P = 0.67$, average = 28.2 mmol/h) between treatments and across the finishing period ($P = 0.99$). Low gain steers did exhibit a larger hepatic lactate removal of flux on d 14. Total splanchnic tissue flux did not differ ($P = 0.53$, average = 41.2 mmol/h) between HG and LG steers or across the finishing period ($P = 0.54$). Mean lactate HMR was similar ($P = 0.93$).

Flux of NEFA across the PDV was similar ($P = 0.49$, average = 0.033 mol/h) between treatments, but PDV in both treatments did not differ from zero. Portal-drained viscera flux of NEFA tended ($P = 0.08$) to exhibit a day effect. Portal drained viscera in LG steers was utilizing NEFA while PDV of HG steers released NEFA on d 0; thereafter PDV released NEFA in both HG and LG steers. Hepatic ($P = 0.95$) and TST ($P = 0.83$) flux were similar (average = -0.006 and 0.004 mol/h, respectively) between HG and LG steers. The HMR of NEFA was similar ($P = 0.93$; average = -0.019) between HG and

LG steers. From d 0 to 28 the liver was releasing more NEFA than was presented, whereas on d 42 to 92 the liver was extracting 2 to 18% of the NEFA that it was presented.

Hormone Flux

Insulin flux across the PDV was similar (P = 0.46, average = 45.3 μg/h) between treatments. Insulin PDV flux responded with a day (P < 0.001) effect, increasing in HG steers to a peak flux on d 64 which was 86% greater than on d 0, peak flux in LG steers occurred on d 92 and was 95% greater than d 0. Hepatic flux of insulin was similar (P = 0.12, average = 10.8 μg/h) in HG and LG steers. Removal of insulin by the liver tended (day effect; P = 0.08) to increase with days on feed. Hepatic insulin flux was positive on d 14 and 28, indicating release of insulin, thereafter, hepatic insulin flux was negative indicating removal of insulin by the liver. Total splanchnic tissue flux was similar (P = 0.62; average = 34.6 μg/h) and positive, indicating a release of insulin from the TST. Insulin TST flux tended (P 0.06) to be affected by day, with peak TST insulin flux occurring on d 70 in HG and d 42 in LG steers. Insulin HMR was similar (P = 0.64, average = 0.0004).

Portal drained viscera IGF-I flux was similar (P = 0.51, average = 189.3 μg/h) between HG and LG and similar (P = 0.88) across the finishing period. Hepatic IGF-I removal in LG steers was greater (P = 0.002) 249 μg/h compared with 212 μg/h in HG steers. This difference is mostly likely because of the large values for HG steers on d 28 and 70 and on d 92 in LG steers. Flux of IGF-I across the TST was similar (P = 0.29, average = 228.6 μg/h) despite the difference in hepatic flux. Hepatic metabolite ratios were different (P = 0.05) between HG and LG steers. Mean HMR for HG steers was

0.036, indicating a 4% release of IGF-I from the liver, whereas LG steers mean HMR was –0.016, indicating a extraction of 2% of the IGF-I presented to the liver.

Leptin flux across the PDV did not differ (P = 0.99, average = 7.74 μg/h) between treatments or across the finishing period (P = 0.88). The large variation in leptin PDV flux between steers may have precluded determining any differences. Similarly hepatic leptin flux did not differ (P= 0.61, average = 25.7 μg/h) between HG and LG steers or with increasing days on feed (P = 0.78). However, different patterns of hepatic leptin flux were evident between HG and LG steers. Because PDV and hepatic flux were not different, TST flux did not differ (P = 0.39, average = 9.1 μg/h) between HG and LG steers or among steers across the finishing phase (P = 0.20). The mean HMR of leptin was different (P = 0.01) between treatments. In HG steers the HMR was –0.01, indicating the liver was removing 1% of the leptin. The mean HMR in LG steers was 0.011, but was not different from zero.

Discussion

Compensatory growth has been defined as more rapid and efficient growth of an animal (Ferrell et al., 1986; Sainz and Bentley, 1995) following a period of nutritional restriction. Generally, compensatory growth is characterized by increased rate of BW gain (Fox et al., 1972; Sainz et al., 1995; Hersom et al., 2003), more efficient rate of BW gain (Ferrell et al., 1986; Sainz et al., 1995; Hersom et al., 2003), and reduced maintenance energy requirements (Fox et al., 1972; NRC 1996). Low-gain steers in the current study exhibited compensatory gain during the first 28 d of the finishing period. The LG steers exhibited the criteria of compensatory growth: greater ADG, increased

intake, and increased gain efficiency (Fox et al., 1972). To our knowledge no one has examined the effect of compensatory growth in steers on net nutrient flux.

Mean OM digestibility was greater for HG steer than LG steers. However, on d 0 and 14 LG steers had numerically greater diet digestibility, and digestibility in both treatments peaked on d 28. Lower diet DM digestibility has been previously reported (Thomson et al., 1982, Hayden et al., 1993) in re-fed cattle that had been previously energy restricted for 154 or 92 d. Thomson et al. (1982) proposed that decreased DM digestibility might result from an increased rate of passage caused by increased DMI by compensating steers. Digestible OM intake (DOMI) across the finishing period was greater in HG than LG steers. The difference in mean DOMI was due to differences on d 0 and 42. On d 0, the difference in DOMI occurred because LG steers were being limit fed to maintain their respective BW gain before the start of the finishing phase. The difference in DOMI on d 42 occurred due to low DOMI during the fecal collection period. The reduction in DOMI by LG steers might have had subsequent effects on oxygen consumption and blood metabolite flux.

One objective was to measure the effect of previous BW gain on blood acid/base balance. Because we expected LG steers to undergo of compensatory growth and the associated increase in DMI, we hypothesized that LG cattle might have been more susceptible to metabolic acidosis during adaptation to the high-grain diet. Therefore measures of blood acid/base balance were of interest. There were no differences in blood pH; mean arterial pH was 7.43, portal 7.32, and hepatic 7.33. However as stated by Owens et al. (1996) blood pH may not be indicative of acidosis in all circumstances. Steers in both treatments were fed to achieve ad libitum intake after the d 0 sampling.

Partial pressure of CO_2 did not differ between treatments, but in both treatments the pCO_2 decreased as DOMI increased. A similar trend was observed in portal blood bicarbonate levels. The decrease in bicarbonate suggests an increased need for buffering in the blood, however the decrease in pCO_2 indicates that the respiratory system was able to compensate and maintain blood pCO_2. In addition, arterial blood base excess tended to be lower in HG steers than LG steers. Differences in PDV tissues ability to disperse the metabolic acid load into blood could be important in controlling cellular metabolism and absorption of nutrients. Regression analysis demonstrated that portal blood base excess was negatively related to DOMI ($P = 0.03$, portal base excess = 3.04 + -0.289*DMI). The lowest portal blood base excess in HG steers coincided with the greatest DOMI. The lowest portal blood base excess of LG steers occurred on d 42 when DOMI was the lowest, however the next lowest value occurred on d 28 which was also the day with the highest DOMI. Regression analysis demonstrated hepatic blood base excess also was negatively related to DMI ($P = 0.04$, hepatic base excess = 3.85 + -2.72*DMI). Decreases in blood base excess coincided with both HG and LG steers adaptation to a finishing diet with greater concentrate levels, and the absorbed acid load from the diet most likely required more buffering. We concluded that compensating steers are at no more risk of experiencing metabolic acidosis than non-compensating steers.

Key to the dispersion and metabolism of absorbed nutrients is blood flow through the splanchnic tissues. Arterial, portal, and hepatic blood flow were similar among treatments. Portal and hepatic blood flows are similar to those previously reported (Reynolds and Huntington, 1988; Krehbiel et al., 1992; Eisemann et al., 1996; Lapierre et al. 2000) for growing steers of similar BW, age, and diets. Similar to other reported data,

portal and hepatic blood flow in both HG and LG steers increased concomitant with increased DOMI (Reynolds et al., 1992; Eisemann et al., 1996) and BW (Eisemann et al., 1996). However, Huntington et al. (1996) reported decreased portal and hepatic blood flow in steers fed 27 or 63% concentrate diets with 1 kg difference in DMI Variation in blood flow has been shown to occur throughout the day. Whitt et al. (1996) reported portal and hepatic plasma flows increased after feeding and varied by as much as 8 to 9% across the day. Many experiments examining nutrient flux utilize equally spaced feed delivery during the day (Eisemann et al., 1996; Huntington et al.1996; Goetsch et al., 1997; Lapierre et al., 2000). Even spaced feeding may decrease variation in blood flow because adherence to the Fick principle is more likely (Huntington, 1999). Our objective in the feeding management of our steers was to more closely replicate our larger feedlot feeding experiments (Hersom et al., 2002), and more realistic feed delivery patterns (i.e., twice daily). Therefore more variation in blood flow is to be expected when steady state conditions are not met. Additionally, Huntington (1999) reported that the greatest variation in blood flow is attributable to steer.

In vitro oxygen consumption has previously been reported to be lower for small intestinal and liver tissues in lambs prior to undergoing compensatory growth (Drouillard et al., 1991; Wester et al., 1995). Our values for PDV oxygen consumption are lower than those summarized by Huntington and Reynolds (1987) and lower than those reported by Reynolds and Huntington (1988a) and Reynolds et al. (1992) for beef steers. Oxygen consumption by the PDV in our experiment was similar between treatments and across the feeding period. In contrast, Eisemann et al. (1996) demonstrated increased PDV oxygen consumption as steers grew heavier and consumed more DM. In both HG

and LG steers, PDV oxygen consumption increased during the first 28 d, thereafter PDV oxygen consumption remained stable. The reduction in DOMI on d 0 by LG steers is evident by a 100 mmol/h reduction in PDV oxygen consumption. Similar and steady PDV oxygen consumption from d 28 to 92 probably is a reflection of the steady DOMI exhibited by steers on both treatments. Hepatic oxygen consumption was greater in LG than HG steers during the feeding period. Low gain steers increased hepatic oxygen consumption with the increase in DOMI starting after d 0, unlike HG steers that already had a higher level of intake. Total splanchnic tissue oxygen consumption increased in both HG and LG steers. The decrease in DOMI by LG steers on d 42 is evident in TST oxygen consumption, producing peak TST oxygen consumption on d 28 that falls on d 42 and then increases to d 92. Oxygen consumption by TST in HG steers increased up to d 64, a point at which they might have reached maturity and maximal PDV mass. Differences in energy intake have not affected blood flow and presumably oxygen consumption by PDV, hepatic, and splanchnic tissues (Goetsch et al., 1997; Krehbiel et al., 1998).

Changes in portal blood ammonia concentration reflect potential changes in ruminal fermentation characteristics (Eismann et al., 1996) and changes in DOMI over the course of the finishing period. However, venous-arterial differences in ammonia concentration coupled with changes in blood flow resulted in little change in PDV ammonia flux. Similarly, Huntington et al. (1996) reported no difference in PDV release of ammonia N in steers consuming 27 or 63% concentrate diets. In contrast, Reynolds et al. (1991) reported decreased PDV ammonia N release in 75% concentrate diets compared with 75% alfalfa diets. Ammonia N release by the PDV was also increased

with increased DMI (Reynolds et al., 1991, 1992). In light of differences in portal ammonia concentration, hepatic flux and the resulting TST flux reflect the liver's ability to maintain systemic ammonia concentration by changing the extraction ratio. Decreased release of ammonia by the TST in LG steers might also reduce the energy required to excrete ammonia. Release of urea-N by the PDV is of some concern. However, PDV urea-N flux was different from zero only on d 28 in HG and d 14 in LG steers. Generally, the PDV has demonstrated a utilization of urea-N (Reynolds and Huntington, 1988; Krehbiel et al., 1998, Lapierre et al., 2000). Differences in urea-N concentrations possibly reflect differences in ruminal fermentation and intake patterns between HG and LG steers. Additionally we report low HMR compared with values reported by Bonhert et al. (1999) for urea-N. Also unexpected was the extraction of urea-N to the extent that was observed on d 14 to 42 in HG steers. However, TST flux values fall within the range of those reported previously (Eisemann et al., 1996, Whitt et al., 1996, and Lapierre et al., 2000). Release of urea-N from TST would supply N for recycling (Huntington et al., 1996) and greater concentrate levels increase TST release of urea-N. Numerically lower TST flux in LG steers might either limit N recycling or decrease energy expenditure associated with N metabolism in the liver. Increased liver urea-N release was reported to account for 16% of the increase in oxygen consumption by the liver with increased intake of 75% concentrates diets (Reynolds et al., 1991). Because of the unusual results for urea-N, the relationship of ammonia and urea-N flux are not consistent.

Concentrations of AAN decreased with increasing days on feed, however release of AAN from the PDV varied because of changes in blood flow. Goetsch et al. (1997) reported a 36% decrease in AAN PDV flux with increasing energy supplied through the

substitution of 25% corn in grass hay diets. Reynolds et al (1991) and Huntington et al. (1996) reported no differences in PDV AAN flux in steers consuming diets differing in forage:concentrate ratio. In contrast, Lapierre et al. (2000) reported increased release of AAN by the PDV with increasing intake and an increase in grain processing. Release of AAN by the PDV could be a result of direct absorption of amino acids, deamination, and decarboxylation of amino acids by the GIT tissues (Reynolds et al., 1991). Extraction of AAN has been implicated as a key regulatory point in the control of nutrient distribution (Eisemann et al., 1996). Hepatic uptake of AAN in HG steers indicates the use of AAN as a possible gluconeogenic precursor or urea cycle intermediates (Reynolds et al., 1991). Release of AAN on d 28 and 42 from the liver and greater TST AAN release would indicate the synthesis of AAN and thus amino acids for peripheral use in LG steers.

Albumin and total protein concentrations in arterial, portal, and hepatic blood were not effected by treatment or days on feed. Additionally, flux of albumin and total protein were not effected by previous BW gain during finishing. Work of Connell et al., (1997) reported that fasting did not decrease total protein synthesis rate relative to fed sheep, however, fasted sheep had an albumin synthesis rate that was depressed by one-half. In the present experiment even when DOMI was reduced in LG steers, concentration and net flux of albumin and total protein remained unchanged, indicating the constitutive nature of the total protein in blood to maintain homeostasis. In addition to maintenance of blood homeostasis, albumin and total protein can be a repository for short-term amino acid storage and recycling. Release of albumin by the liver can be used as anabolic precursors (Lobley, 2002), and release of protein from the GIT can be processed in the liver to supply amino acids.

Glucose concentration increased with days on feed. The level of corn inclusion increased from d 0 until d 18 while steers were adapting to the final diet, thereafter increases in glucose in the diet were due to increased intake. As steers grew, the utilization of glucose by the PDV increased. Peak glucose use by the PDV was nearly five fold greater on d 64 compared with d 0 in HG steers and six-fold greater in LG steers. Reynolds et al. (1991) reported greater PDV glucose use by heifers fed a high ME diet compared with low ME diets. Huntington et al. (1996) also showed that increasing dietary concentration level from 27 to 63% increased PDV glucose use by 63%. Increased PDV removal of glucose implies utilization of glucose exceeded absorption of glucose by the intestinal tissues. Even though the diet provided increasing starch concentration during the adaptation diets, increases in glucose absorption lagged behind utilization. Huntington and Reynolds (1986) reported increased PDV glucose release with abomasal glucose infusion compared with infusion of starch. Lower PDV glucose release associated with starch infusion implies digestion of starch to glucose is the limiting step in glucose absorption (Reynolds et al., 1991). Eisemann et al. (1996) reported 20% lower PDV glucose utilization values as steers matured. Hepatic flux of glucose was positive indicating a release of glucose from the liver. Release of glucose from the liver in light of extraction of glucose by the PDV indicates substantial gluconeogenesis. Hepatic extraction ratios show addition of 4 to 13% glucose from the liver. Total splanchnic tissue release of glucose peaked on d 28 in LG and d 64 in HG steers. Even though treatments were statistically similar in TST glucose flux, LG steers had 13% greater TST flux than HG steers except on d 64. Slightly greater supplies of anabolic precursors could have stimulated the additional growth observed in LG steers.

Coupling slightly greater TST glucose and AAN flux in LG than HG steers might be part of the mechanism of compensatory growth in LG steers.

Blood lactate concentrations decreased with increasing days on feed. Greatest arterial blood lactate concentrations were observed on d 0, before high-grain finishing was initiated. Portal and hepatic blood lactate concentrations varied with day and would be dependent on DOMI and rumen fermentation patterns on sampling days. Portal drained viscera flux of lactate tended to be effected by days on feed. Both treatments exhibited peak PDV lactate flux on d 14, during the adaptation period to the final diet. Reynolds et al. (1991) and Huntington et al. (1996) reported that PDV lactate release was not affected by dietary concentrate level. Therefore, lactate PDV flux change during the adaptation diets in the current experiment most likely comes from other sources Lactate flux in the PDV comes from two sources; absorption by the ruminal epithelium and glycolysis in the post-ruminal digestive tract (Reynolds and Huntington 1988a; Eiseman et al., 1997). The change in lactate PDV flux could indicate increased use of lactate for visceral organ fat synthesis (Eisemann et al., 1996). Hepatic lactate flux generally indicated removal of lactate by the liver. Removal of lactate by the liver is an important factor in gluconeogenesis and transamination in the Cori cycle. The variable TST flux observed in both HG and LG steers reflects the variable extraction of lactate for further metabolism by the liver.

Blood NEFA concentrations were effected by day. With no discernable pattern to NEFA concentration, factors other than treatment or diet were affecting NEFA concentrations. Because the LG steers were gaining BW up to the d 0 sampling date, no differences in NEFA were expected. Plasma NEFA concentrations in contemporary HG

and LG steers utilized in the feedlot trial were also similar between treatments (Hersom et al., 2002 b). The trend of PDV NEFA release would indicate variation in the use of fat as an energy substrate by gut tissues. The variable uptake or release by the TST reflects the lipid metabolism by the mesenteric fat, not dietary lipid content (Freetly and Ferrell, 2000), and limited extraction of NEFA by the liver.

Blood insulin concentrations exhibited significant increases with increasing days on feed at all three sites. Insulin concentration was higher in HG steers than LG steers, this was especially evident in hepatic blood insulin concentrations. Release of insulin by the PDV increased with days on feed. In HG steers insulin PDV flux nearly doubled from d 0 to 28. It was not until d 42 that LG and HG steers had similar PDV insulin flux. Similarly, Lapierre et al. (1992) reported that increasing intake by 45% resulted in a 47% increase in PDV insulin release in growing steers. Increasing DMI did not affect hepatic uptake. In the current experiment, hepatic extraction of insulin did not occur until d 42 of the finishing period. After d 42 the liver in HG and LG steers began extracting insulin. Release of insulin by the TST increased during the finishing period. Similar TST insulin flux is a result of greater hepatic blood flow in LG steers and greater hepatic insulin concentration in HG steers. Eisemann et al. (1997) demonstrated a reduced sensitivity to insulin as steers increased in age, BW, and percentage of empty body weight as fat. In the present experiment all steers were the same age, however, HG steers started the finishing period with greater BW and whole body fat percent estimated by harvest of contemporary steers (Hersom et al., 2002a). The higher sensitivity to insulin because of reduced fat content in LG steers compared with HG steers could associated with part of

the mechanism of compensatory growth, in which compensating animals are more sensitive to the anabolic effects of insulin that in turn increase BW gain.

Blood concentrations of IGF-I were similar between treatments. Similar results for IGF-I concentration in contemporary HG and LG steers have been reported previously (Hersom et al., 2002 b). Because blood concentrations of IGF-I were similar, differences in flux of IGF-I are due to differences in blood flow. Increasing DMI by 45% has no effect on blood IGF-I concentration (Lapierre et al., 1992), but PDV flux indicated IGF-I release at low DMI and uptake at high DMI by steers. As a result of differences in PDV flux, TST uptake of IGF-I was greater at high DMI compared with low DMI intake (Lapierre et al., 1992). In contrast to the current study and previous work, Lapierre et al. (2000) reported a lack of detectable differences in IGF-I flux among steers with different feed intakes.

To our knowledge we are the first to report flux of leptin by steers consuming high-grain diets. Leptin concentrations increased with days on feed in both treatments. Leptin concentrations have previously been reported to be sensitive to energy intake (Daniel et. al., 2002; Delavaud et al., 2002). Positive correlations have also been reported between leptin concentration and body fat content (Houseknecht et al., 1998; Delavaud et al., 2002; Hersom et al., 2002 b). Leptin PDV flux would be more indicative of mesenteric fat status, and would act as a signal to the hypothalamic-pituitary axis (Barb, 1999). Hepatic uptake of leptin, though not affected by treatment or days on feed, is likely. Leptin is a protein hormone and thus would be degraded in the liver. Additionally Houseknecht et al. (1998) stated the existence of hepatic leptin receptors. Interestingly,

the TST flux of leptin and insulin were both positive. Barb (1999) indicated that leptin concentration can be positively influenced by insulin.

Implications

Steers that had previously been restricted in live BW gain on wheat pasture prior to placement on high-grain diets were able to compensate in BW compared with unrestricted steers. Compensating steers were more efficient and had greater diet digestibility during the early feeding period. Compensating steers also had livers that consumed more oxygen and thus expended more energy. This increase in energy consumption was not detrimental to efficient BW gain. Compensating steers had greater blood flow through the liver that contained greater concentrations of anabolic precursors such as glucose and α-amino N. The increased hepatic blood flow and greater concentrations of metabolites may be part of the underlying mechanism of compensatory growth in cattle.

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Table 5.1. Composition of the transition, adaptation, and final diet fed to steers

Ingredient	Transition	Step 1[a]	Step 2[b]	Step 3[c]	Final[c]
	---------- %, DM ----------				
Whole corn	40.0	54.0	62.4	70.8	80.0
Alfalfa hay	35.0	23.0	15.4	8.0	-
Cottonseed hulls	24.9	19.1	15.4	11.9	8.0
Soybean meal	0.05	5.9	6.1	6.2	6.0
Cane molasses	0.03	1.12	2.21	3.09	4.0
Salt	0.003	0.09	0.14	0.19	0.25
Limestone	-	0.35	0.56	0.77	1.0
Urea	-	0.23	0.36	0.5	0.65
Premix[e]	-	0.02	0.04	0.05	0.07
NE_m, Mcal/ kg DM	1.63	1.81	1.91	2.00	2.10
NE_g,Mcal/kg DM	0.91	1.07	1.16	1.24	1.34
CP, %	13.80	13.88	13.83	13.77	13.45

[a] Day 0 – 6 of feeding period.
[b] Day 7 – 13 of feeding period.
[c] Day 14 – 20 of feeding period.
[d] Day 21 – 98 of feeding period.
[e] 40% trace mineral mix (13.0% Zn, 6.0% Mn, 3.6% Cu, 1.43% Fe, 800 ppm Co, 6,000 ppm I, 100 ppm Se), 27% Rumensin-80 (Elanco, Indianapolis, IN), 17% Tylan-40 (Indianapolis, IN), 15% vitamin A 30,000 IU/g, 1% vitamin E 500 IU/g.

Table 5.2. Number of steers and patent catheters from each site during the experiment

	Days on feed					
Item	0	14	28	42	64	92
Arterial site						
HG[a]	5	5	5	5	5	4
LG[b]	5	5	5	4	3	3
Portal site						
HG	5	5	5	5	5	4
LG	5	5	5	4	3	3
Hepatic site						
HG	4	3	3	3	3	2
LG	5	5	5	4	3	2
Total steers						
HG	5	5	5	5	5	4[d]
LG	5	5	5	4[b]	3[c]	3

[a] HG = High gain (1.25 kg/d prior to high grain feeding), LG = Low gain (0.73 kg/d prior to high grain feeding).

[b] One LG steer dropped from blood collection because arterial catheter was not patent.

[c] One LG steer dropped from blood collection because mesenteric vein catheter was infected and prevented infusion.

[d] One HG steer removed from experiment because of problems with it's feet and legs.

Table 5.3. Incremental finishing performance and nutrient digestibility of steers with different previous BW gains

	Days on feed							*P*-value		
Item	0	14	28	42	64	92	SEM[a]	Treatment	Day	T x D[b]
BW, kg										
HG[c]	362	384	398	408	429	454	12.2	0.009	< 0.001	0.19
LG	287	317	344	353	375	403	12.2			
ADG, kg/d								0.04	< 0.001	0.18
HG	--	1.55	1.05	0.78	1.06	0.88	0.12			
LG	--	2.16	2.10	0.67	1.10	1.05	0.11			
DMI, kg/d								0.001	<0.001	0.03
HG	7.65	8.17	8.72	6.61	6.87	7.07	0.22			
LG	4.86	6.85	8.50	4.47	6.78	6.48	0.21			
DMI, % of BW								0.20	< 0.001	0.01
HG	2.32	2.47	2.24	1.70	1.60	1.48	0.05			
LG	1.99	2.28	2.60	1.78	1.72	1.61	0.50			
ADG:DMI, kg/kg								0.005	0.007	0.16
HG	--	0.161	0.109	0.103	0.143	0.120	0.014			
LG	--	0.296	0.221	0.099	0.164	0.156	0.014			
OM digestibility, %								0.01	<0.001	0.12
HG	70.4	74.9	84.02	82.4	83.7	84.6	0.17			
LG	74.6	77.3	83.5	78.9	81.3	80.5	0.10			
Digestible OM intake, kg/d								0.003	< 0.001	0.25
HG	5.5	6.2	7.4	5.44	6.2	6.2	0.20			
LG	3.7	5.4	7.2	3.6	5.9	5.5	0.19			

[a] Standard error of measure, n = see Table 2.

[b] Treatment x day.

[c] HG = High gain (1.25 kg/d prior to high grain feeding), LG = Low gain (0.73 kg/d prior to high grain feeding).

Table 5.4. Blood gas components from steers with different previous BW gains

	Days on feed							*P*-value		
Item	0	14	28	42	64	92	SEM[a]	Treatment	Day	T x D[b]
pH										
Arterial								0.47	0.58	0.53
HG[c]	7.42	7.43	7.43	7.42	7.44	7.36	0.02			
LG	7.43	7.43	7.44	7.42	7.44	7.44	0.02			
Portal								0.31	0.11	0.21
HG	7.31	7.34	7.30	7.31	7.33	7.33	0.004			
LG	7.32	7.31	7.31	7.31	7.33	7.31	0.004			
Hepatic								0.89	0.04	0.21
HG	7.30	7.34	7.32	7.32	7.35	7.35	0.004			
LG	7.32	7.33	7.34	7.33	7.34	7.34	0.004			
pCO_2										
Arterial								0.11	0.06	0.81
HG	41.42	40.88	40.36	40.94	39.13	39.57	0.39			
LG	42.33	42.25	39.93	42.53	40.31	41.09	0.39			
Portal								0.15	0.01	0.01
HG	55.03	53.03	55.42	54.87	52.02	52.70	0.78			
LG	57.02	57.30	54.84	55.42	52.12	56.68	0.77			
Hepatic								0.70	0.002	0.07
HG	59.53	51.43	54.35	55.69	53.24	50.86	0.75			
LG	57.38	56.18	52.95	54.07	52.14	54.73	0.62			
Bicarbonate, mmol/L										
Arterial								0.17	0.78	0.68
HG	27.21	27.3	26.79	26.80	26.86	27.21	0.83			
LG	28.49	28.13	32.30	28.13	27.66	28.64	0.84			
Portal								0.15	0.05	0.67
HG	28.56	28.04	27.76	27.66	27.83	27.94	0.37			
LG	30.30	29.19	28.13	27.94	28.12	29.14	0.37			

Hepatic								0.35	0.28	0.55
HG	29.70	28.09	28.37	28.91	29.31	28.64	0.45			
LG	30.65	29.38	29.26	28.73	28.55	29.61	0.37			
Blood base excess, mmol/L										
Arterial								0.08	0.25	0.49
HG	3.06	3.06	2.55	2.51	3.08	3.38	0.32			
LG	4.63	4.50	3.62	3.13	3.16	3.40	0.32			
Portal								0.57	0.02	0.63
HG	1.90	1.12	0.01[d]	0.90[d]	0.84[d]	0.72[d]	0.32			
LG	3.38	1.09	0.45[d]	0.21[d]	0.90[d]	1.12[d]	0.33			
Hepatic								0.51	0.17	0.61
HG	2.20	1.39	0.78[d]	1.94	2.23	1.87	0.40			
LG	3.76	1.97	1.56	1.36	1.69	2.26	0.33			
Calculated O_2 saturation, %										
Arterial								0.67	0.49	0.17
HG	98.67	98.65	98.71	98.86	96.9.	98.18	0.26			
LG	98.50	98.79	98.77	97.78	98.67	98.33	0.23			
Portal								0.38	0.02	0.45
HG	78.14	84.15	85.16	86.03	83.40	82.45	0.75			
LG	79.56	81.63	81.29	83.35	83.35	84.02	0.77			
Hepatic								0.25	0.05	0.79
HG	67.31	71.98	74.42	74.17	72.92	71.11	2.26			
LG	66.36	67.87	69.21	69.08	70.71	66.21	1.87			
Hemoglobin, g/dL										
Arterial								0.88	0.001	.065
HG	8.12	9.19	10.35	10.88	10.17	9.47	0.62			
LG	8.37	8.45	10.72	10.34	10.84	10.21	0.57			
Portal								0.75	0.001	0.53
HG	7.93	8.50	9.50	10.51	10.08	8.82	0.68			
LG	7.95	7.57	10.14	10.42	11.01	10.10	0.65			
Hepatic								0.74	0.06	0.92

HG	7.66	8.92	9.76	9.84	11.01	8.99	0.48			
LG	8.91	8.02	9.68	10.42	11.74	8.71	0.40			
Packed cell volume, %										
Arterial								0.46	0.43	0.88
HG	26.17	27.33	26.29	28.49	26.06	27.98	1.21			
LG	28.52	28.88	26.49	29.63	28.17	28.35	1.12			
Portal								0.48	0.02	0.07
HG	26.05	27.53	26.22	28.65	26.24	28.57	0.79			
LG	27.61	25.11	27.64	28.20	28.07	28.22	0.76			
Hepatic								0.81	0.88	0.09
HG	26.25	28.19	26.44	28.88	27.08	29.33	1.06			
LG	27.16	24.68	24.99	28.10	27.20	27.89	0.87			

[a] Standard error of measure, n = see Table 2.
[b] Treatment x day.
[c] HG = High gain (1.25 kg/d prior to high grain feeding), LG = Low gain (0.73 kg/d prior to high grain feeding).
[d] Means not different from zero ($P > 0.10$).

Table 5.5. Blood flow, oxygen concentration, and oxygen consumption in steers with different previous BW gains

	Days on feed							*P*-value		
Item	0	14	28	42	64	92	SEM[a]	Treatment	Day	T x D[b]
Blood flow, L/h										
Arterial								0.22	0.55	0.23
HG[c]	76.7	103.45	114.1	65.1	90.9	50.6[d]	17.81			
LG	74.7	105.6	121.8	120.0	72.6	197.2	15.95			
Portal								0.51	< 0.001	0.33
HG	432.7	589.5	758.1	669.6	904.4	627.9	53.55			
LG	435.7	557.1	579.2	722.4	962.9	586.2	56.59			
Hepatic								0.06	< 0.001	0.63
HG	485.8	514.7	737.8	709.1	930.9	518.3	34.43			
LG	504.4	727.6	758.8	749.9	942.2	853.0	32.05			
Blood O_2, mmol/L										
Arterial								0.86	0.001	0.27
HG	2.48	2.81	3.15	3.32	3.04	2.87	0.17			
LG	2.56	2.60	3.27	3.11	3.30	3.09	0.16			
Portal								0.81	0.001	0.43
HG	1.90	2.18	2.47	2.75	2.57	2.21	0.16			
LG	1.94	1.91	2.54	2.63	2.80	2.59	0.16			
Hepatic								0.81	0.02	0.85
HG	1.59	1.96	2.17	2.19	2.37	1.92	0.13			
LG	1.80	1.66	2.05	2.18	2.50	1.76	0.10			
Oxygen consumption, mmol/h										
PDV[e]								0.47	0.69	0.92
HG	-268.4	-333.0	-383.7	-391.3	-329.3	-384.7	35.47			
LG	-269.9	-431.2	-452.1	-353.7	-481.2	-331.6	38.16			
Hepatic								< 0.001	0.34	0.95
HG	-189.6[d]	-288.2[d]	-299.9[d]	-372.1	-547.4	-355.3[d]	71.75			
LG	-193.5[d]	-400.4	-515.0	-412.5	-392.2	-730.5	35.53			

TST[f]								0.002	0.06	0.85
HG	-424.3	-537.7	-640.6	-698.6	-753.1	-525.3	41.63			
LG	-414.9	-733.1	-967.1	-784.5	-880.2	-1052.7	43.04			

[a] Standard error of measure, n = see Table 2.
[b] Treatment x day.
[c] HG = High gain (1.25 kg/d prior to high grain feeding), LG = Low gain (0.73 kg/d prior to high grain feeding).
[d] Means not different from zero ($P > 0.10$).
[e] PDV = Portal drained viscera.
[f] TST = Total splanchnic tissue.

Table 5.6. Arterial, portal, and hepatic metabolite concentrations from steers with different previous BW gains

	Days on feed							P-value		
Item	0	14	28	42	64	92	SEM[a]	Treatment	Day	T x D[b]
Ammonia, μM										
Arterial								0.14	0.45	0.84
HG[c]	320	260	218	254	272	273	13.6			
LG	244	247	226	195	240	264	15.0			
Portal								0.11	0.002	0.06
HG	568	459	380	397	383	393	13.3			
LG	439	377	399	310	378	481	14.8			
Hepatic								0.28	0.42	0.26
HG	413	291	264	295	321	237	43.99			
LG	214	255	238	196	249	232	39.52			
Urea-N, mM										
Arterial								0.003	0.47	0.42
HG	7.55	7.79	7.34	6.87	6.48	7.40	0.22			
LG	6.31	4.42	5.78	5.78	5.48	7.14	0.25			
Portal								< 0.001	0.56	0.40
HG	7.80	7.98	7.64	7.03	6.64	7.05	0.12			
LG	6.50	4.68	5.87	5.83	5.53	7.38	0.17			
Hepatic								0.05	0.12	0.40
HG	7.83	7.60	7.22	6.83	5.28	7.54	0.33			
LG	6.45	4.73	5.93	6.06	5.65	7.53	0.28			
α-Amino N, mM										
Arterial								0.25	0.02	0.88
HG	3.47	3.66	4.06	3.50	3.23	2.66	0.10			
LG	3.69	3.63	3.97	3.67	3.46	3.23	0.11			
Portal								0.08	0.003	0.94
HG	3.42	3.75	3.97	3.71	3.36	3.15	0.04			

LG	3.57	4.13	3.91	3.71	3.53	3.31	0.06			
Hepatic								0.10	0.002	0.90
HG	3.44	3.93	4.01	3.89	2.94	2.57	0.08			
LG	3.47	3.78	4.34	4.09	3.22	3.13	0.08			
Albumin, mM										
Arterial								0.61	0.69	0.40
HG	519	529	517	505	515	527	8.0			
LG	517	486	495	508	533	534	8.9			
Portal								0.60	0.33	0.45
HG	511	522	521	506	519	528	8.8			
LG	507	491	494	515	524	532	9.2			
Hepatic								0.97	0.44	0.49
HG	502	536	511	504	505	516	14.4			
LG	505	500	489	500	540	544	12.5			
Total protein, g/L										
Arterial								0.68	0.12	0.17
HG	66.3	68.2	66.0	67.2	68.2	67.5	1.53			
LG	67.6	63.2	64.0	66.1	67.9	69.1	1.56			
Portal								0.68	0.35	0.10
HG	65.2	67.6	68.1	66.5	67.3	67.2	1.52			
LG	67.0	63.9	63.6	65.2	67.2	69.5	1.55			
Hepatic								0.67	0.47	0.26
HG	63.4	67.1	66.7	67.6	67.3	66.6	1.49			
LG	66.7	63.6	63.4	65.2	66.2	68.4	1.25			
Glucose, mM										
Arterial								0.77	0.002	0.76
HG	4.01	4.31	4.37	4.51	4.03	4.42	0.09			
LG	3.90	4.22	4.47	4.73	4.24	4.34	0.10			
Portal								0.50	0.02	0.72
HG	3.96	4.23	4.43	4.38	4.87	4.36	0.06			
LG	3.87	4.24	4.41	4.67	4.16	4.26	0.07			

LG	3.57	4.13	3.91	3.71	3.53	3.31	0.06			
Hepatic								0.10	0.002	0.90
HG	3.44	3.93	4.01	3.89	2.94	2.57	0.08			
LG	3.47	3.78	4.34	4.09	3.22	3.13	0.08			
Albumin, mM										
Arterial								0.61	0.69	0.40
HG	519	529	517	505	515	527	8.0			
LG	517	486	495	508	533	534	8.9			
Portal								0.60	0.33	0.45
HG	511	522	521	506	519	528	8.8			
LG	507	491	494	515	524	532	9.2			
Hepatic								0.97	0.44	0.49
HG	502	536	511	504	505	516	14.4			
LG	505	500	489	500	540	544	12.5			
Total protein, g/L										
Arterial								0.68	0.12	0.17
HG	66.3	68.2	66.0	67.2	68.2	67.5	1.53			
LG	67.6	63.2	64.0	66.1	67.9	69.1	1.56			
Portal								0.68	0.35	0.10
HG	65.2	67.6	68.1	66.5	67.3	67.2	1.52			
LG	67.0	63.9	63.6	65.2	67.2	69.5	1.55			
Hepatic								0.67	0.47	0.26
HG	63.4	67.1	66.7	67.6	67.3	66.6	1.49			
LG	66.7	63.6	63.4	65.2	66.2	68.4	1.25			
Glucose, mM										
Arterial								0.77	0.002	0.76
HG	4.01	4.31	4.37	4.51	4.03	4.42	0.09			
LG	3.90	4.22	4.47	4.73	4.24	4.34	0.10			
Portal								0.50	0.02	0.72
HG	3.96	4.23	4.43	4.38	4.87	4.36	0.06			
LG	3.87	4.24	4.41	4.67	4.16	4.26	0.07			

Hepatic								0.62	< 0.001	0.93
HG	4.09	4.79	4.85	5.08	4.54	4.92	0.10			
LG	4.08	4.61	4.90	5.15	4.52	4.61	0.09			
Lactate, mM										
Arterial								0.26	< 0.001	0.29
HG	0.86	0.32	0.81	0.54	0.47	0.64	0.022			
LG	0.80	0.63	0.75	0.60	0.44	0.66	0.26			
Portal								0.82	0.004	0.77
HG	0.91	0.64	0.92	0.78	0.57	0.63	0.035			
LG	0.84	0.83	0.95	0.74	0.49	0.67	0.040			
Hepatic								0.61	0.10	0.97
HG	0.87	0.74	0.92	0.64	0.62	0.59	0.058			
LG	0.81	0.58	0.89	0.69	0.51	0.63	0.053			
NEFA, mM										
Arterial								0.19	0.03	0.39
HG	211.2	190.2	241.6	150.0	156.3	189.4	7.78			
LG	278.7	179.1	198.7	176.3	194.3	211.5	8.81			
Portal								0.92	0.05	0.45
HG	252.4	202.4	265.2	155.2	218.7	208.1	13.30			
LG	219.3	225.0	222.3	182.1	218.0	247.3	14.23			
Hepatic								0.86	0.12	0.94
HG	254.2	221.3	268.3	149.8	168.8	212.0	18.57			
LG	266.6	240.2	237.8	175.9	181.1	146.5	16.57			

[a] Standard error of measure, n = see Table 2.

[b] Treatment x day.

[c] HG = High gain (1.25 kg/d prior to high grain feeding), LG = Low gain (0.73 kg/d prior to high grain feeding).

Table 5.7. Arterial, portal, and hepatic hormone concentrations from steers with different previous BW gains

	Days on feed							P-value		
Item	0	14	28	42	64	92	SEM[a]	Treatment	Day	T x D[b]
Insulin, ng/mL										
Arterial								0.14	< 0.001	0.32
HG[c]	0.176	0.188	0.248	0.500	0.493	0.451	0.022			
LG	0.137	0.161	0.241	0.512	0.281	0.397	0.024			
Portal								0.16	< 0.001	0.46
HG	0.209	0.177	0.293	0.608	0.624	0.596	0.030			
LG	0.147	0.166	0.257	0.633	0.374	0.514	0.033			
Hepatic								< 0.001	< 0.001	0.005
HG	0.233	0.203	0.349	0.624	0.710	0.584	0.018			
LG	0.140	0.176	0.271	0.295	0.335	0.425	0.017			
IGF-I, ng/mL										
Arterial								0.61	0.20	0.29
HG	148.11	117.76	146.76	138.87	114.94	141.45	8.48			
LG	118.82	139.08	133.24	145.71	112.31	119.88	8.86			
Portal								0.69	0.42	0.42
HG	150.49	127.92	139.19	146.46	120.49	136.43	7.52			
LG	119.06	145.70	143.97	141.99	118.64	124.58	7.99			
Hepatic								0.12	0.21	0.12
HG	153.38	109.78	170.45	161.96	122.69	151.73	7.23			
LG	121.97	146.35	134.83	145.44	120.66	96.39	6.44			
Leptin, ng/mL										
Arterial								0.24	0.001	0.37
HG	3.55	5.71	6.93	7.12	6.14	7.39	0.91			
LG	2.86	4.05	6.33	4.43	4.77	4.56	0.93			
Portal								0.26	0.001	0.40
HG	3.94	5.61	6.99	7.25	5.71	7.51	0.93			
LG	2.78	3.89	6.24	4.58	5.12	4.61	0.95			

Hepatic								0.24	0.02	0.42
HG	4.00	5.70	6.85	8.24	6.98	6.66	1.02			
LG	2.77	3.93	6.24	4.62	4.88	5.52	0.91			

[a] Standard error of measure, n = see Table 2.
[b] Treatment x day.
[c] HG = High gain (1.25 kg/d prior to high grain feeding), LG = Low gain (0.73 kg/d prior to high grain feeding).

Table 5.8. Nutrient flux across portal drained viscera, liver, and total splanchnic tissues and hepatic metabolism ratio of steers with different previous BW gains

	Days on feed							P-value		
Item	0	14	28	42	64	92	SEM[a]	Treatment	Day	T x D[b]
Ammonia, mmol/h										
PDV flux[c]								0.92	0.71	0.12
HG[d]	107.3	136.5	137.9	101.5	104.0	34.0[e]	11.10			
LG	102.1	80.4	112.3	66.8[e]	120.5	148.7	12.15			
Hepatic flux								0.55	0.75	0.24
HG	-78.0	-108.7	-73.0	-69.3	-118.1	-138.9	21.68			
LG	-121.3	-74.7	-104.9	-69.3	-118.1	-138.9	19.44			
TST flux[f]								0.37	0.79	0.63
HG	28.1[e]	21.3[e]	30.4[e]	19.2[e]	21.1[e]	25.8[e]	18.29			
LG	-17.9[e]	5.7[e]	7.3[e]	2.6[e]	6.8[e]	3.6[e]	16.39			
HMR[g]								0.44	0.03	0.18
HG	-0.294	-0.296	-0.284	-0.266	-0.196	-0.415	0.084			
LG	-0.548	-0.312	-0.356	-0.314	-0.355	-0.432	0.076			
Urea-N, mmol/h										
PDV flux								0.09	0.70	0.57
HG	102.2[e]	171.0[e]	287.9	100.3[e]	136.9	87.5[e]	27.00			
LG	93.2[e]	166.4	59.2[e]	4.7[e]	21.2[e]	148.5[e]	27.80			
Hepatic flux								0.98	0.63	0.12
HG	79.7[e]	-8.4[e]	-38.7[e]	-9.56[e]	90.1[e]	97.5[e]	29.49			
LG	-15.2[e]	60.4[e]	48.7[e]	159.4	110.5[e]	146.8[e]	25.75			
TST flux								0.49	0.94	0.95
HG	160.6	156.8[e]	142.2[e]	146.7[e]	213.2	143.1	36.63			
LG	78.1[e]	226.7	107.9[e]	164.1	131.6[e]	33.5[e]	34.67			
HMR								0.96	0.82	0.14
HG	0.022[e]	0.007[e]	-0.006[e]	0.001[e]	0.019[e]	0.026[e]	0.007			
LG	-0.004[e]	0.021[e]	0.010[e]	0.040	0.019[e]	-0.022[e]	0.006			

α-Amino N, mmol/h										
PDV flux								0.60	0.50	0.42
HG	-65.7[e]	25.7[e]	118.9[e]	133.8[e]	106.7[e]	341.3	67.12			
LG	-19.7[e]	311.6	-45.3[e]	12.0[e]	41.5[e]	38.2[e]	72.7			
Hepatic flux								0.20	0.10	0.39
HG	-25.6[e]	-46.1[e]	-152.8[e]	73.1[e]	-285.5	-182.4[e]	75.8			
LG	-41.3[e]	-184.7[e]	319.0	300.0	-287.7	88.0[e]	64.7			
TST flux								0.33	0.11	0.64
HG	-28.0[e]	44.3[e]	-13.6[e]	65.8[e]	-123.4[e]	-108.5[e]	73.3			
LG	-61.0[e]	126.9[e]	273.7	309.8	-267.5	57.5[e]	63.2			
HMR								0.12	0.17	0.58
HG	-0.019[e]	-0.026[e]	-0.040[e]	0.019[e]	-0.096[e]	-0.103[e]	0.026			
LG	-0.029[e]	-0.061[e]	0.109	0.120	-0.097[e]	0.028[e]	0.02			
Albumin, mmol/h										
PDV flux								0.98	0.94	0.79
HG	-4702[e]	-4393	-1262[e]	270[e]	4716[e]	2009[e]	2075			
LG	-3472[e]	3084[e]	-1028[e]	2319[e]	-5089[e]	309[e]	2326			
Hepatic flux								0.54	0.30	0.67
HG	-1447[e]	11714[e]	-10200[e]	-3270[e]	-13083[e]	-3625[e]	4149			
LG	-1365[e]	7485[e]	-4104[e]	-8662[e]	5890[e]	2100[e]	3676			
TST flux								0.49	0.17	0.95
HG	-8655[e]	3944[e]	-2704[e]	-5985[e]	904[e]	-2853[e]	2743			
LG	-4837[e]	10569[e]	-5132[e]	-6284[e]	1135[e]	5742[e]	2483			
HMR								0.61	0.31	0.76
HG	-0.004[e]	0.024[e]	-0.025[e]	-0.007[e]	-0.021[e]	-0.014[e]	0.009			
LG	-0.006[e]	0.016[e]	-0.010[e]	-0.022[e]	0.011[e]	0.005[e]	0.008			
Total protein, g/h										
PDV flux								0.65	0.22	0.22
HG	-673	-436	2417	-562	-815	3	277			
LG	-211	440	-306	-601	-704	165	309			
Hepatic flux								0.99	0.61	0.11

HG	-715	354	-1290	-467	260	-125	143			
LG	-169	-146	-172	-122	-853	-505	134			
TST flux								0.65	0.42	0.26
HG	-1820	401	-463	-861	-194	-125	243			
LG	-380	294	-478	-726	-1558	-100	221			
HMR								0.81	0.43	0.21
HG	-0.021	0.008	-0.026	-0.009	0.004	-0.003	0.003			
LG	-0.007	-0.003	-0.004	-0.003	-0.014	-0.009	0.003			
Glucose, mmol/h										
PDV flux								0.51	0.11	0.31
HG	-32.81[e]	-43.13[e]	60.64[e]	-98.78	-158.73	-37.26[e]	16.85			
LG	-12.47[e]	15.01[e]	-39.80[e]	-31.58[e]	-78.04[e]	-59.57[e]	18.93			
Hepatic flux								0.99	< 0.001	0.13
HG	73.72[e]	317.81	237.33	341.67	492.03	229.99	30.27			
LG	105.61	269.45	369.41	317.28	321.05	306.7	26.3			
TST flux								0.47	< 0.001	0.89
HG	15.30[e]	269.82	275.35	251.78	293.18	188.05	32.49			
LG	93.14	284.46	329.61	286.07	244.50	253.19	28.28			
HMR								0.84	< 0.001	0.05
HG	0.040	0.100	0.068	0.104	0.129	0.096	0.010			
LG	0.052	0.087	0.110	0.097	0.086	0.088	0.008			
Lactate, mmol/h										
PDV flux								0.37	0.09	0.85
HG	17.12[e]	203.68	69.33[e]	162.21	98.41	-11.33[e]	16.63			
LG	21.32[e]	120.18	120.02	70.95[e]	39.43[e]	5.13[e]	22.27			
Hepatic flux								0.67	0.99	0.27
HG	-30.50[e]	75.27[e]	-35.25[e]	-48.71[e]	-49.77[e]	-32.11[e]	27.72			
LG	-20.98[e]	-151.26	-13.10[e]	-41.99[e]	31.21[e]	21.76[e]	25.01			
TST flux								0.53	0.54	0.56
HG	-22.85[e]	139.58	52.93[e]	114.67[e]	73.08[e]	-35.53[e]	29.45			
LG	0.35[e]	-31.08[e]	106.92	29.12[e]	70.60[e]	-3.91[e]	26.52			

HMR								0.68	0.98	0.06
HG	-0.043[e]	0.256	-0.002[e]	-0.091[e]	-0.036[e]	-0.057[e]	0.064			
LG	-0.013[e]	-0.278	-0.015[e]	-0.027[e]	0.103[e]	0.028[e]	0.057			
NEFA, mol/h										
PDV flux								0.49	0.08	0.28
HG	0.016[e]	0.004[e]	0.026[e]	0.004[e]	0.067	0.012[e]	0.009			
LG	-0.030[e]	0.028[e]	0.015[e]	0.0[e]	0.025[e]	0.029[e]	0.010			
Hepatic flux								0.95	0.36	0.94
HG	0.009[e]	0.002[e]	-0.003[e]	-0.007[e]	-0.022[e]	-0.013[e]	0.011			
LG	0.022[e]	0.010[e]	0.015[e]	-0.004[e]	-0.036[e]	-0.046[e]	0.009			
TST flux								0.83	0.58	0.36
HG	0.022[e]	0.012[e]	-0.008[e]	0.001[e]	0.008[e]	0.0003[e]	0.009			
LG	-0.008[e]	0.039[e]	0.030	0.002[e]	-0.013[e]	-0.030[e]	0.009			
HMR								0.93	0.52	0.97
HG	0.059[e]	0.002[e]	0.068[e]	-0.061[e]	-0.129[e]	-0.082[e]	0.076			
LG	0.103[e]	0.162[e]	0.117[e]	-0.022[e]	-0.179[e]	-0.268[e]	0.068			

[a] Standard error of measure, reported in Table 2.
[b] Treatment x day.
[c] PDV = Portal drained viscera.
[d] HG = High gain (1.25 kg/d prior to high grain feeding), LG = Low gain (0.73 kg/d prior to high grain feeding).
[e] Means not different from zero ($P > 0.10$).
[f] TST = Total splanchnic tissue.
[g] HMR = Hepatic extraction ratio.

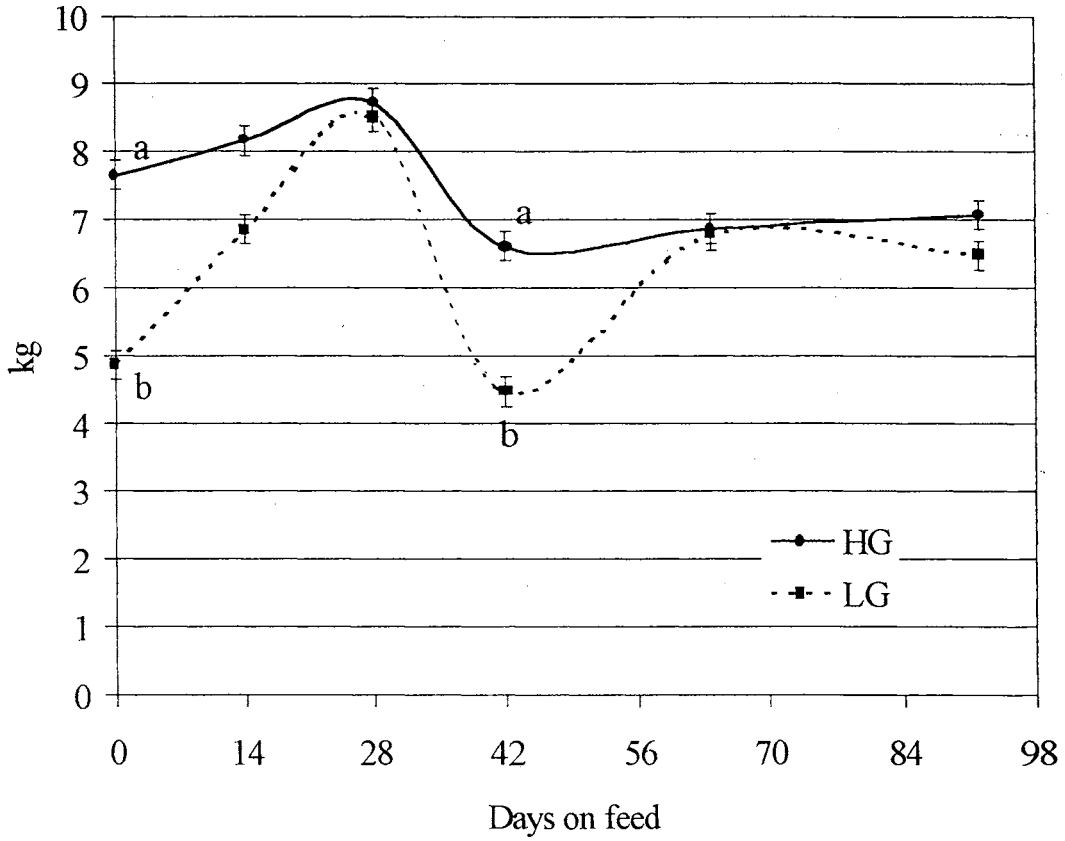
Table 5.9. Hormone flux across portal drained viscera, liver, and total splanchnic tissues and hepatic metabolism ratio of steers with different previous BW gains

	Days on feed							P-value		
Item	0	14	28	42	64	92	SEM[a]	Treatment	Day	T x D[b]
Insulin, μg/h										
PDV flux[c]								0.46	< 0.001	0.63
HG[d]	16.7[e]	-9.9[e]	31.3	69.2	120.2	77.2	7.75			
LG	4.0[e]	0.7[e]	6.5[e]	78.8	77.4	83.8	8.55			
Hepatic flux								0.12	0.08	0.72
HG	4.6[e]	3.1[e]	0.8[e]	-23.1[e]	-72.6[e]	-23.7[e]	7.1			
LG	-2.3[e]	8.5[e]	9.7[e]	-12.2[e]	-20.8[e]	-1.6[e]	6.4			
TST flux[f]								0.62	0.06	0.99
HG	20.1[e]	9.8[e]	35.9[e]	53.2	60.8	48.2[e]	9.68			
LG	1.7[e]	9.2[e]	16.2[e]	65.8	54.0	39.9[e]	8.76			
HMR[g]								0.64	0.42	0.79
HG	0.074[e]	0.078[e]	-0.002[e]	-0.051[e]	-0.097[e]	-0.061[e]	0.032			
LG	-0.033[e]	0.075[e]	0.071[e]	0.005[e]	-0.052[e]	0.003[e]	0.028			
IGF-I, μg/h										
PDV flux								0.51	0.88	0.43
HG	175.3[e]	420.6[e]	-474.0[e]	658.8[e]	302.2[e]	-496.9[e]	185.09			
LG	-194.4[e]	385.2[e]	647.5[e]	-71.7[e]	555.3[e]	363.7[e]	205.00			
Hepatic flux								0.002	0.24	0.15
HG	-180.0[e]	-209.6[e]	1812.0	753.2[e]	-1412.3	511.0[e]	94.53			
LG	162.8[e]	198.8[e]	-484.5[e]	438.4[e]	554.1[e]	-2390.3	87.53			
TST flux								0.29	0.37	0.29
HG	-110.9[e]	481.8[e]	1135.4[e]	1915.5	-689.2[e]	272.6[e]	375.4			
LG	-31.6[e]	581.0[e]	192.9[e]	242.3[e]	1066.0[e]	-2312.3	335.9			
HMR								0.05	0.62	0.31
HG	0.015[e]	-0.037[e]	0.169	0.076[e]	-0.074[e]	0.068[e]	0.018			
LG	0.016[e]	0.015[e]	-0.039[e]	0.033[e]	0.039[e]	-0.162	0.016			

Leptin, μg/h										
PDV flux								0.99	0.88	0.21
HG	120.3[e]	-42.5[e]	190.3[e]	74.4[e]	-395.5	98.6[e]	70.09			
LG	-31.7[e]	-108.4[e]	-87.2[e]	93.2[e]	226.2[e]	-44.9[e]	76.58			
Hepatic flux								0.61	0.78	0.39
HG	-8.0[e]	-11.5[e]	-289.6[e]	89.7[e]	484.7	-279.7[e]	80.75			
LG	-25.7[e]	19.3[e]	-27.4[e]	22.9[e]	-105.2[e]	438.9[e]	72.01			
TST flux								0.39	0.20	0.37
HG	164.0[e]	-99.4[e]	-500.7	195.6[e]	196.9[e]	-276.2[e]	103.8			
LG	-57.4[e]	-89.1[e]	-114.6[e]	130.8[e]	106.7[e]	452.5[e]	93.07			
HMR								0.01	0.90	0.34
HG	0.004	0.021	-0.020	0.007	0.058	-0.058	0.004			
LG	0.005	-0.002	-0.0001	-0.0002	-0.051	0.181	0.005			

[a] Standard error of measure, reported in Table 2.
[b] Treatment x day.
[c] PDV = Portal drained viscera.
[d] HG = High gain (1.25 kg/d prior to high grain feeding), LG = Low gain (0.73 kg/d prior to high grain feeding).
[e] Means not different from zero ($P > 0.10$).
[f] TST = Total splanchnic tissue.
[g] HMR = Hepatic extraction ratio

Figure 5.1. Dry matter intake of steers with different previous BW gains during high-grain finishing



[a,b] Means within a day with different letters differ ($P < 0.05$).

APPENDIX

Table A.1. Steer organ and digesta mass at intital harvest, Exp. 1

Item	High Gain Wheat				Low Gai	
Steer ID	G74	G50	G46	G75	Y96	Y100
Harvest Date	4/3/00	4/3/00	4/5/00	4/5/00	4/3/00	4/3/00
Live BW, kg	393.62	394.53	422.22	424.49	331.87	315.08
Hot carcass, kg	225.87	225.87	251.52	245.61	182.28	170.48
Hide , kg	25.85	24.70	26.00	27.00	23.65	20.40
Blood, kg	12.85	15.05	14.80	13.30	12.10	11.15
Head, kg	11.60	12.00	13.30	14.00	11.85	11.50
Heart, kg	1.68	1.74	1.78	2.00	1.40	1.22
Lung, kg	2.65	4.35	4.65	5.80	4.60	4.15
Liver, kg	6.48	7.08	7.06	6.90	4.10	4.56
Pancreas, kg	0.44	0.38	0.58	0.52	0.26	0.28
Spleen, kg	0.70	0.72	0.88	1.00	1.05	0.62
Kidney, kg	1.18	1.02	1.34	1.00	0.95	0.84
Reticulo-rumen, kg	8.40	8.40	8.35	8.40	6.80	6.95
Omasum, kg	2.88	2.45	2.46	3.50	2.40	2.64
Abomasum, kg	1.64	1.25	1.40	1.50	1.52	1.38
Small intestine, kg	5.30	7.50	5.90	6.45	4.30	5.10
Large intestine, kg	3.80	3.80	3.95	4.80	4.05	3.00
Cecum, kg	0.60	0.01	0.45	0.50	0.65	0.55
Mesenteric fat, kg	11.70	9.85	8.05	11.55	3.15	5.15
Feet and ears, kg	9.20	9.50	9.75	9.90	9.15	9.30
Trim, kg	6.55	6.25	6.35	5.99	3.65	2.95
Total Offal, kg	113.50	116.05	117.05	124.11	95.63	91.74
Empty Body, kg	352.02	362.68	377.12	390.84	292.07	281.93
Digesta Mass, kg (As is)						
Reticulo-rumen	31.55	26.75	35.90	26.10	28.80	24.45
Reticulo-rumen (DM)	3.88	4.31	4.85	3.38	2.91	2.52
Omasum	2.75	0.35	0.45	0.70	0.45	0.45
Abomasum	2.00	1.45	2.25	1.30	1.50	1.15
Small intestine	4.45	2.30	3.55	4.15	5.40	3.20
Large intestine	0.10	0.50	1.70	0.70	2.40	2.25
Cecum	0.75	0.50	1.25	0.70	1.25	1.65
Total Digesta	41.60	31.85	45.10	33.65	39.80	33.15

Table A.1. Continued

Item	Wheat		Native Range			
Steer ID	Y74	Y 65	Or97	Or99	Or95	Or98
Harvest Date	4/5/00	4/5/00	4/3/00	4/3/00	4/5/00	4/5/00
Live BW, kg	305.54	311.44	238.80	271.04	278.30	275.58
Hot carcass, kg	166.39	171.16	126.89	141.65	144.37	135.07
Hide , kg	21.05	20.80	12.95	15.80	18.15	16.55
Blood, kg	9.05	9.75	8.35	9.40	7.80	9.60
Head, kg	11.65	10.55	9.80	0.60	9.75	11.25
Heart, kg	1.25	1.34	1.06	1.08	1.18	1.08
Lung, kg	3.65	3.55	3.15	3.05	3.25	3.60
Liver, kg	4.22	4.54	3.22	3.72	3.72	3.32
Pancreas, kg	0.32	0.32	0.26	0.24	0.26	0.30
Spleen, kg	0.50	0.56	0.46	0.74	0.62	0.44
Kidney, kg	0.66	0.70	0.62	0.68	0.66	0.88
Reticulo-rumen, kg	6.45	6.90	5.20	5.45	5.85	5.50
Omasum, kg	1.68	2.65	4.20	2.45	3.04	2.90
Abomasum, kg	1.08	1.10	1.05	1.00	1.04	1.25
Small intestine, kg	5.15	5.05	3.55	4.55	4.15	4.30
Large intestine, kg	2.60	3.30	2.55	2.60	2.40	2.35
Cecum, kg	0.30	0.65	0.30	0.30	0.25	0.35
Mesenteric fat, kg	3.95	5.65	1.05	2.05	2.65	1.40
Feet and ears, kg	8.75	8.95	7.10	7.95	7.40	7.80
Trim, kg	3.60	3.36	1.25	2.90	2.10	3.50
Total Offal, kg	85.91	89.72	66.12	64.56	74.27	76.37
Empty Body, kg	272.79	272.79	202.95	232.19	240.50	228.98
Digesta Mass, kg (As is)						
Reticulo-rumen	25.70	28.60	27.55	29.80	29.15	33.90
Reticulo-rumen (DM)	3.57	3.13	4.19	4.19	4.21	5.04
Omasum	0.20	0.35	0.70	0.40	1.85	1.25
Abomasum	1.95	3.25	2.05	1.55	1.20	*1.60*
Small intestine	3.20	4.80	2.80	3.55	3.75	5.30
Large intestine	1.20	1.20	1.90	2.45	1.20	3.15
Cecum	0.50	0.45	0.85	1.10	0.65	1.40
Total Digesta	32.75	38.65	35.85	38.85	37.80	46.60

Table A.2. Steer organ and digesta mass at intital harvest, Exp. 2

Item	High Gain Wheat				Low Gai	
Steer ID	G31	G34	G27	G11	Y38	Y42
Harvest Date	5/16/01	5/16/01	5/16/01	5/16/01	5/17/01	5/17/01
Live BW, kg	413.14	395.89	365.92	399.52	296.92	325.06
Hot carcass, kg	241.30	219.74	210.20	230.86	167.98	171.61
Hide , kg	27.30	29.46	24.36	26.18	20.26	21.08
Blood, kg	14.24	13.54	7.78	12.90	10.54	12.96
Head, kg	13.98	13.58	12.10	12.80	11.40	12.30
Heart, kg	1.59	1.58	1.83	1.81	1.39	1.42
Lung, kg	4.89	5.44	4.93	5.46	3.67	4.75
Liver, kg	6.19	4.96	5.73	5.44	4.22	4.98
Pancreas, kg	0.45	0.28	0.40	0.34	0.29	0.34
Spleen, kg	0.65	0.68	0.82	0.64	0.68	0.63
Kidney, kg	0.99	0.89	0.87	0.93	0.70	0.88
Reticulo-rumen, kg	8.08	8.90	8.38	8.18	6.04	6.84
Omasum, kg	2.62	3.08	2.34	2.90	2.38	2.72
Abomasum, kg	1.30	1.18	1.38	1.38	1.14	1.32
Small intestine, kg	4.83	4.24	4.65	4.14	4.16	5.25
Large intestine, kg	3.04	2.56	3.26	2.96	2.10	2.72
Cecum, kg	0.30	0.50	0.70	0.50	0.38	0.56
Mesenteric fat, kg	11.08	6.84	8.46	10.04	3.30	6.06
Feet and ears, kg	10.30	10.62	9.34	10.30	9.00	9.08
Trim, kg	3.56	3.52	9.96	5.22	2.92	3.40
Total Offal, kg	115.39	111.85	107.29	112.12	84.57	97.29
Empty Body, kg	366.64	338.29	322.08	353.72	254.56	281.80
Digesta Mass, kg (As is)						
Reticulo-rumen	34.88	45.10	34.96	35.58	30.10	30.94
Reticulo-rumen (DM)	3.26	4.97	4.15	4.60	3.95	4.12
Omasum	3.66	3.40	2.22	3.30	4.20	3.82
Abomasum	2.04	1.42	0.42	1.30	1.64	2.42
Small intestine	3.86	4.60	2.94	3.12	3.78	3.44
Large intestine	1.04	1.68	1.28	1.26	1.28	0.86
Cecum	1.02	1.40	2.02	1.24	1.36	1.78
Total Digesta	46.50	57.60	43.84	45.80	42.36	43.26

Table A.2. Continued

Item	Wheat		Native Range			
Steer ID	Y40	Y30	Or288	Or537	Or178	Or277
Harvest Date	5/17/01	5/17/01	5/15/01	5/15/01	5/15/01	5/15/01
Live BW, kg	342.32	338.68	243.34	236.08	300.55	273.31
Hot carcass, kg	184.78	185.69	124.40	120.31	148.00	135.29
Hide , kg	26.32	21.56	15.22	16.32	17.30	17.84
Blood, kg	12.06	13.16	6.90	8.78	10.22	10.30
Head, kg	12.80	13.18	9.78	9.82	11.18	10.10
Heart, kg	1.57	1.65	0.91	0.99	1.22	1.13
Lung, kg	3.76	4.29	2.74	3.33	4.64	3.71
Liver, kg	5.42	4.74	2.92	3.27	3.97	3.68
Pancreas, kg	0.44	0.32	0.29	0.15	0.37	0.27
Spleen, kg	1.11	0.56	0.34	0.87	0.46	0.53
Kidney, kg	0.90	0.94	0.70	0.88	0.88	0.70
Reticulo-rumen, kg	7.65	8.18	4.98	5.51	6.24	6.32
Omasum, kg	3.46	2.62	2.52	2.82	4.00	3.14
Abomasum, kg	1.32	1.20	0.98	0.84	1.12	1.32
Small intestine, kg	5.18	4.07	3.28	3.63	3.28	4.12
Large intestine, kg	3.04	2.62	1.84	2.08	1.66	1.86
Cecum, kg	0.24	0.44	0.30	0.30	0.56	0.42
Mesenteric fat, kg	6.66	5.50	1.42	1.40	2.34	2.16
Feet and ears, kg	10.04	9.54	6.36	7.50	8.42	8.04
Trim, kg	4.42	2.40	2.42	2.24	1.90	2.06
Total Offal, kg	106.39	96.97	63.90	70.73	79.76	77.70
Empty Body, kg	299.74	284.68	183.58	175.12	224.87	205.13
Digesta Mass, kg (As is)						
Reticulo-rumen	30.32	41.40	47.60	48.78	60.04	53.64
Reticulo-rumen (DM)	3.89	5.00	5.46	4.93	8.27	4.54
Omasum	4.84	3.68	4.88	4.92	7.72	5.34
Abomasum	1.80	1.08	1.24	1.32	1.96	2.52
Small intestine	4.60	4.24	3.82	3.04	4.30	3.14
Large intestine	0.78	2.10	1.30	1.76	0.32	2.40
Cecum	0.24	1.50	0.92	1.14	1.34	1.14
Total Digesta	42.58	54.00	59.76	60.96	75.68	68.18

Table A.3. Steer organ and digesta mass at final harvest, Exp. 1

Item	High Gain Wheat					
Steer ID	G36	G39	G48	G49	G72	G73
Harvest Date	7/5/00	7/5/00	7/5/00	7/5/00	7/5/00	7/5/00
Live BW, kg	580.21	577.49	603.37	555.24	584.75	572.49
Hide , kg	33.05	38.30	38.15	34.35	40.60	42.55
Hot carcass, kg	333.69	345.95	348.22	341.41	326.43	338.68
Blood, kg	15.10	19.15	15.35	18.55	13.85	16.70
Head, kg	16.25	16.00	15.95	15.20	15.50	15.35
Heart, kg	2.04	2.50	2.44	2.52	2.02	2.16
Lung, kg	7.95	8.40	7.30	7.20	7.30	5.15
Liver, kg	8.15	8.95	8.04	8.40	7.35	7.25
Pancreas, kg	0.32	0.50	0.52	0.46	0.50	0.48
Spleen, kg	1.02	0.84	1.08	1.10	1.02	1.14
Kidney, kg	1.08	1.12	1.22	1.16	1.08	1.04
Reticulo-rumen, kg	13.25	11.80	12.95	11.85	12.70	11.70
Omasum, kg	5.60	6.05	3.42	4.44	5.00	4.35
Abomasum, kg	1.55	1.76	1.56	1.86	1.60	1.50
Small intestine, kg	6.85	7.55	6.65	7.15	7.40	6.80
Large intestine, kg	5.30	5.65	4.95	6.10	5.05	4.05
Cecum, kg	0.45	0.90	0.85	0.90	0.85	0.60
Mesenteric fat, kg	19.70	20.95	22.80	21.30	18.95	15.15
Feet and ears, kg	11.20	12.00	12.95	12.40	12.15	11.45
Trim, kg	7.20	7.45	11.45	4.65	5.70	7.75
Total Offal, kg	156.06	169.87	167.63	159.59	158.62	155.17
Empty Body, kg	531.61	530.79	534.57	518.79	525.55	512.29
Digesta Mass, kg (As is)						
Reticulo-rumen	43.80	36.40	60.05	27.50	50.20	54.15
Reticulo-rumen (DM)	15.26	13.64	24.29	6.69	18.02	20.29
Omasum	0.20	1.60	0.50	0.95	0.55	0.15
Abomasum	1.35	1.90	2.55	2.15	0.95	2.75
Small intestine	2.65	4.35	4.10	3.70	5.05	2.05
Large intestine	0.35	0.75	0.20	1.15	1.20	0.00
Cecum	0.25	1.70	1.40	1.00	1.25	1.10
Total Digesta	48.60	46.70	68.80	36.45	59.20	60.20

Table A.3. Continued

Item	Low Gain Wheat					
Steer ID	Y60	Y64	Y66	Y73	Y95	Y98
Harvest Date	8/9/00	8/9/00	8/9/00	8/9/00	8/9/00	8/9/00
Live BW, kg	551.16	569.32	542.98	552.97	551.61	564.78
Hide , kg	34.28	36.76	33.74	42.06	39.50	24.70
Hot carcass, kg	317.57	330.51	304.18	320.98	306.45	323.70
Blood, kg	16.70	15.36	11.38	18.02	13.70	14.56
Head, kg	15.12	15.92	15.88	18.06	14.44	14.50
Heart, kg	2.44	1.92	2.13	2.64	2.10	2.22
Lung, kg	8.02	8.02	6.98	7.06	7.32	7.24
Liver, kg	7.16	7.29	6.90	8.06	7.17	7.35
Pancreas, kg	0.58	0.51	0.46	0.39	0.37	0.49
Spleen, kg	0.87	1.94	1.32	1.23	1.07	0.96
Kidney, kg	0.95	0.93	0.93	1.03	1.06	1.03
Reticulo-rumen, kg	13.72	12.42	12.68	11.14	11.24	10.46
Omasum, kg	5.70	3.24	5.06	6.74	4.42	4.10
Abomasum, kg	3.46	1.78	1.66	1.66	1.48	1.46
Small intestine, kg	6.80	6.26	5.38	7.20	6.86	6.42
Large intestine, kg	5.92	6.12	5.02	5.12	5.12	5.04
Cecum, kg	0.50	0.92	0.68	0.86	0.80	0.94
Mesenteric fat, kg	15.92	19.36	12.58	10.72	16.51	11.42
Feet and ears, kg	11.76	11.00	12.52	12.46	12.36	10.98
Trim, kg	8.54	5.32	7.52	10.16	8.26	8.34
Total Offal, kg	158.44	155.07	142.82	164.61	153.78	132.21
Empty Body, kg	490.46	507.84	491.58	507.55	503.47	503.78
Digesta Mass, kg (As is)						
Reticulo-rumen	52.22	52.38	44.00	39.16	42.08	52.86
Reticulo-rumen (DM)	20.19	15.15	15.49	14.68	14.65	21.97
Omasum	1.24	0.36	1.06	0.52	0.56	0.86
Abomasum	2.86	2.78	2.82	0.98	0.86	0.54
Small intestine	2.74	3.90	2.90	3.20	1.88	4.16
Large intestine	0.76	1.56	0.04	0.06	1.34	0.38
Cecum	0.88	0.50	0.58	1.50	1.42	2.20
Total Digesta	60.70	61.48	51.40	45.42	48.14	61.00

Table A.3. Continued

Item	Native Range					
Steer ID	Or84	Or85	Or89	Or90	Or93	Or96
Harvest Date	9/19/00	9/19/00	9/19/00	9/19/00	9/19/00	9/19/00
Live BW, kg	582.03	572.95	546.16	559.78	581.12	643.32
Hide , kg	45.04	34.22	39.20	37.28	38.32	46.34
Hot carcass, kg	320.98	335.05	309.17	331.65	354.12	373.64
Blood, kg	16.02	16.62	14.12	12.96	16.38	18.18
Head, kg	16.04	16.84	14.52	15.24	15.96	17.74
Heart, kg	2.04	2.09	2.15	2.58	2.60	2.14
Lung, kg	8.10	6.84	8.74	6.74	8.02	6.58
Liver, kg	7.09	7.13	6.86	8.76	8.26	8.99
Pancreas, kg	0.44	0.56	0.63	0.53	0.52	0.52
Spleen, kg	0.99	0.97	1.01	1.85	0.84	1.01
Kidney, kg	0.97	0.98	1.00	1.05	0.90	1.02
Reticulo-rumen, kg	11.54	11.82	10.96	10.40	11.10	12.64
Omasum, kg	7.64	4.50	5.16	4.98	5.06	6.48
Abomasum, kg	1.32	1.50	1.48	1.42	1.46	1.34
Small intestine, kg	6.06	7.82	7.12	6.54	6.58	6.74
Large intestine, kg	4.08	4.74	5.70	4.74	5.48	5.22
Cecum, kg	0.84	0.92	1.04	1.02	0.80	0.92
Mesenteric fat, kg	19.82	15.46	16.48	16.14	22.48	20.82
Feet and ears, kg	12.94	10.92	11.26	12.40	11.44	11.82
Trim, kg	7.10	8.30	13.72	7.90	7.30	9.12
Total Offal, kg	168.07	152.23	161.15	152.53	163.50	177.62
Empty Body, kg	519.61	516.61	499.22	509.90	542.22	579.28
Digesta Mass, kg (As is)						
Reticulo-rumen	52.62	48.66	38.34	42.96	34.16	57.62
Reticulo-rumen (DM)	18.14	13.58	13.43	15.40	11.47	20.16
Omasum	1.48	1.52	1.18	0.46	0.48	1.20
Abomasum	1.86	2.30	0.98	1.80	1.18	2.42
Small intestine	3.92	3.46	3.36	3.36	2.00	2.06
Large intestine	0.68	0.22	1.32	0.52	0.60	0.12
Cecum	1.86	0.18	1.76	0.78	0.48	0.62
Total Digesta	62.42	56.34	46.94	49.88	38.90	64.04

Table A.4. Steer organ and digesta mass at final harvest, Exp. 2

Item	High Gain Wheat					
Steer ID	G29	G25	G28	G32	G30	G35
Harvest Date	8/7/01	8/7/01	8/7/01	8/7/01	8/7/01	8/7/01
Live BW, kg	551.61	531.18	585.66	524.37	576.58	576.58
Hot Carcass, kg	336.41	308.27	332.78	304.18	330.06	330.97
Hide , kg	36.15	33.10	41.30	37.15	38.90	40.65
Blood, kg	13.05	15.30	7.85	19.15	14.15	13.55
Head, kg	15.95	15.80	16.80	15.35	15.50	16.35
Heart, kg	2.42	2.04	2.87	2.21	2.40	2.60
Lung, kg	6.20	6.50	5.99	6.89	7.83	6.20
Liver, kg	7.98	7.16	7.82	7.50	8.06	7.18
Pancreas, kg	0.43	0.40	0.53	0.48	0.72	0.54
Spleen, kg	0.85	0.86	1.12	1.08	0.88	0.98
Kidney, kg	1.12	1.22	1.08	1.08	1.12	1.15
Reticulo-rumen, kg	12.05	13.40	15.15	14.40	18.00	13.65
Omasum, kg	4.50	4.60	5.60	4.55	4.30	5.75
Abomasum, kg	1.85	1.60	2.05	1.85	1.65	1.70
Small intestine, kg	5.91	6.69	6.15	6.64	6.86	5.40
Large intestine, kg	4.60	3.95	4.30	4.10	3.95	3.30
Cecum, kg	0.95	0.80	0.85	0.80	0.80	0.65
Mesenteric fat, kg	18.80	10.80	16.65	16.70	16.60	16.95
Feet and ears, kg	12.00	11.60	13.35	11.50	12.10	11.75
Trim, kg	6.15	8.00	9.53	7.11	8.55	7.10
Total Offal, kg	150.96	143.82	158.99	158.54	162.37	155.45
Empty Body, kg	503.81	483.78	537.56	492.02	528.17	528.13
Digesta Mass, kg (As is)						
Reticulo-rumen	37.00	39.85	39.50	26.85	40.26	39.60
Reticulo-rumen (DM)	7.65	7.75	6.85	5.03	8.16	8.07
Omasum	2.95	2.05	2.25	1.00	2.60	2.20
Abomasum	1.80	lost	1.80	0.55	1.00	0.90
Small intestine	4.25	3.35	3.10	2.75	2.90	3.50
Large intestine	0.75	0.75	0.70	0.75	0.70	1.20
Cecum	1.05	1.40	0.75	0.45	0.95	1.05
Total Digesta	47.80	47.40	48.10	32.35	48.41	48.45

Table A.4. Continued

Item	Low Gain Wheat					
Steer ID	Y39	Y41	Y37	Y34	Y35	Y36
Harvest Date	9/4/01	9/4/01	9/4/01	9/4/01	9/4/01	9/4/01
Live BW, kg	558.42	515.29	572.04	494.86	522.10	551.61
Hot Carcass, kg	322.79	293.74	332.78	291.47	309.17	332.78
Hide , kg	39.70	35.86	36.30	36.98	33.40	36.54
Blood, kg	14.50	18.76	15.72	13.48	13.42	11.94
Head, kg	15.80	15.92	16.18	15.60	15.52	15.12
Heart, kg	2.59	2.16	2.42	4.54	2.14	1.97
Lung, kg	6.68	6.76	6.60	13.30	6.24	5.94
Liver, kg	8.32	7.39	7.35	7.48	6.19	8.38
Pancreas, kg	0.50	0.44	0.39	0.50	0.40	0.54
Spleen, kg	0.88	0.86	0.89	2.04	0.83	0.81
Kidney, kg	1.09	1.00	1.10	2.18	1.01	0.94
Reticulo-rumen, kg	15.64	13.94	16.48	14.60	15.54	14.48
Omasum, kg	4.88	4.86	4.46	6.16	4.00	5.04
Abomasum, kg	1.76	1.60	2.50	1.42	1.74	1.68
Small intestine, kg	5.55	5.50	6.19	5.53	4.68	5.82
Large intestine, kg	4.10	4.30	4.76	3.20	3.76	5.14
Cecum, kg	0.98	0.48	0.84	0.60	0.58	0.66
Mesenteric fat, kg	20.72	17.40	22.50	16.80	16.38	19.78
Feet and ears, kg	11.96	12.00	13.04	11.20	11.54	12.14
Trim, kg	6.60	1.54	6.82	7.68	7.16	6.68
Total Offal, kg	162.25	150.77	164.54	163.29	144.53	153.60
Empty Body, kg	517.60	473.63	526.18	460.54	490.25	514.97
Digesta Mass, kg (As is)						
Reticulo-rumen	33.72	36.62	40.86	30.52	27.35	30.56
Reticulo-rumen (DM)	6.32	7.49	5.01	5.14	4.86	5.40
Omasum	0.78	0.22	0.40	0.40	0.98	1.42
Abomasum	0.46	0.12	0.96	0.42	0.38	1.14
Small intestine	4.96	3.52	2.18	1.74	2.20	2.80
Large intestine	0.00	0.54	0.22	0.44	0.36	0.28
Cecum	0.90	0.64	1.24	0.80	0.58	0.44
Total Digesta	40.82	41.66	45.86	34.32	31.85	36.64

Table A.4. Continued

Item	Native Range					
Steer ID	Or12	Or9	Or4	Or11	Or8	Or3
Harvest Date	10/23/01	10/23/01	10/23/01	10/23/01	10/23/01	10/23/01
Live BW, kg	601.55	592.47	501.67	519.83	572.04	569.77
Hot Carcass, kg	355.48	340.50	296.46	304.18	339.59	340.50
Hide , kg	38.64	39.52	33.98	38.52	32.06	38.06
Blood, kg	6.26	6.74	16.28	14.54	8.40	15.06
Head, kg	17.00	16.38	14.78	14.52	16.60	18.26
Heart, kg	2.97	2.94	2.02	2.30	2.65	2.41
Lung, kg	8.07	8.14	7.07	6.48	7.68	6.97
Liver, kg	9.10	7.77	7.65	8.02	8.60	7.69
Pancreas, kg	0.78	0.62	0.45	0.55	0.60	0.58
Spleen, kg	0.98	0.99	1.02	1.04	1.04	1.33
Kidney, kg	1.19	1.11	1.22	1.13	1.35	1.03
Reticulo-rumen, kg	15.22	14.30	12.54	12.77	14.94	17.64
Omasum, kg	7.38	5.84	5.68	5.10	6.04	5.88
Abomasum, kg	4.88	1.78	1.56	2.14	2.28	1.96
Small intestine, kg	5.63	6.47	5.31	6.02	7.69	6.52
Large intestine, kg	3.78	2.92	2.12	4.84	3.68	4.86
Cecum, kg	0.64	1.04	0.63	0.60	2.00	0.98
Mesenteric fat, kg	19.04	23.10	15.82	20.52	19.10	19.46
Feet and ears, kg	11.84	12.70	10.54	11.98	12.92	13.26
Trim, kg	9.06	6.68	5.74	6.88	6.94	7.38
Total Offal, kg	162.46	159.04	144.41	157.95	154.57	169.33
Empty Body, kg	547.85	541.53	464.47	487.77	529.74	534.63
Digesta Mass, kg (As is)						
Reticulo-rumen	43.04	41.16	30.38	27.52	35.02	30.78
Reticulo-rumen (DM)	6.99	6.84	5.66	5.15	6.69	5.59
Omasum	1.02	2.12	0.78	1.36	2.92	0.78
Abomasum	5.24	1.04	1.12	1.08	0.42	1.44
Small intestine	2.42	4.98	4.20	1.72	3.94	1.28
Large intestine	0.34	0.32	0.04	0.00	0.00	0.10
Cecum	1.64	1.32	0.68	0.38	0.00	0.76
Total Digesta	53.70	50.94	37.20	32.06	42.30	35.14

VITA 2

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Candidate for the degree of

Doctor of Philosophy

Thesis: EFFECT OF WINTER GRAZING PROGRAMS ON FEEDLOT PERFORMANCE, VISCERAL ORGAN MASS, BODY COMPOSITION, AND SPLANCHNIC METABOLISM OF STEERS

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