EFFECT OF GROWTH RATE ON CARCASS COMPOSITION, HORMONES, AND METABOLITES AT PUBERTY IN BEEF HEIFERS

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> One of life's quiet excitements is to stand somewhat apart from yourself and watch yourself softly becoming the author of something special .....
>
> Norman Maclean
>
> "A River Runs Through It"

TABLE OF CONTENTS

Chapter Page

LIST OF TABLES

Chapter III

Chapter IV

Table Page

Chapter V

Table Page

Appendix

LIST OF FIGURES

Chapter III

Figure Page

Figure Page

Chapter V

Figure Page

FORMAT OF THESIS

This thesis is presented in the Journal of Animal Science style and format, as outlined by the Oklahoma State University graduate college style manual. The use of this format allows for the independent chapters to be suitable for submission to scientific journals. Three papers have been prepared from the data collected for research to partly fulfill the requirements for the PhD. degree. Each paper is complete in itself with an abstract, introduction, materials and methods, results and discussion, implications and literature cited section.

CHAPTER I

INTRODUCTION

The beef cattle industry is segmented into several different production phases (e.g., cow-calf, stocker, feeder, packer, and purveyor). Each segment acts independently from the other, and production efficiency is determined differently for each segment. Today, the beef cattle industry must face issues like diet health, food safety, environmental activism, animal rights, grazing fees, water rights, and market share competition with poultry and swine. All of these issues can eventually affect profitability. Consequently, the beef cattle industry is coming to the realization that all segments much cooperate to overcome our liabilities and shortcomings. The *Strategic Alliances Field Study* recently conducted by the *National Cattlemen's Association* (NCA, 1993) is an excellent example of how communication and integrated management between industry segments enhances the individuals knowledge and understanding of the industry and also gives exposure exposure to important factors that affect profitability, quality, and consistency of the final product.

The importance of reproduction in relative economic terms is 10 times as important as production and 20 times as important as the product (Wilham, 1973). Hence, in reality, reproduction is the first link in the food chain which begins at least 22 months before the consumer buys a meat product. In short, the beef cow must become pregnant, complete a 9 mo gestation period, calve unassisted, and wean a healthy calf. A process that many times gets left out of the " What's for Dinner?" beef advertising

campaign supervised by the National Cattlemen's Association. Once weaned, the calf continues through the feeding and processing phases before the end product appears in retail case at the super market. For cow-calf, producers production efficiency is measured in terms of reproductive efficiency. Replacement heifer development is one of the most critical areas of production for the cow-calf operator phase because it plays an important role in future productivity of that animal in the cowherd (Lesmeister et al., 1973).

Historically, replacement heifers were bred to calve at 3 yr of age. However, beef production systems have become more intensive over the last few decades as more producers breed replacement heifers as yearlings to calve at two years of age (Short et al., 1994). Consequently, heifers must reach puberty at 12 to 14 mo of age and before the start of the breeding season to avoid decreased fertility associated with the pubertal estrus (Byerly et al., 1987). Heifers bred earlier in the breeding season have greater lifetime production potential (Lesmeister et al., 1973) and calving at 24 mo of age is biologically more efficient (increased pounds of calf weaned per cow at weaning) compared with calving at 36 mo of age (Short et al., 1994). Increased biological efficiency often results in increased economic efficiency (profits exceeds cost) through increased pounds of calf sold. Maximum production is not always biological and/or economically efficient so both biological and economic efficiency must be balanced (optimization) to fit the genetics, feed resources, environment, and the management system associated with the operation. Feeding programs that could enhance adequate growth and development of replacement heifers while increasing the percentage of heifers with normal estrous cycles at the start of the breeding season, without compromising biological efficiency, and operate within economic constraints of the operation could greatly increase profitability of cow calf operations. Increased reproductive efficiency will result in calves that are more uniform in age and weight at weaning.

The objectives of our experiments were:

1) to evaluate the effect of rate of gain on carcass composition, lipid partitioning, age, and weight at puberty.
2) to characterize concentrations of hormones and metabolites of beef heifers fed to attain different percentages of body fat at puberty.
3) to characterize the effects of rate of gain on secretory patterns of LH and growth hormone in prepubertal heifers fed to gain at different rates.

CHAPTER II

REVIEW OF LITERATURE

Introduction

Puberty is frequently defined as the time at which an animal is capable of reproducing itself (Robinson, 1977). From an endocrine standpoint, puberty is the observation of the first behavioral estrus followed by ovulation and development of a functional corpus luteum for a period characteristic of a particular species (Kinder et al., 1987). Attainment of puberty is a dynamic physiological event that characteristically occurs gradually over time. Factors that affect attainment of puberty include: age, weight, nutrition, genotype, season of birth, photoperiod and environment.

Attainment of Puberty: Historical and Current Hypotheses

Critical Body Weight

Kennedy and Mitra (1963) postulated that attainment of puberty in rats depended on reaching a "critical body weight". Frisch and Revelle (1970) observed that body weight was more constant than height at menarche in young girls, thus the "critical body weight" hypothesis was also postulated for humans. This concept is currently not accepted for several species since diet has been shown to effect age as well as weight at puberty in dairy heifers (Sorenson et al., 1959), sheep (Allen and Lamming, 1961), pigs

(Friend, 1976), rodents (Glass et al., 1979), and beef heifers (Arjie and Wiltbank, 1971; Grass et al., 1982). Body weight at menarche also varies with age in young girls (Frisch et al., 1971).

Critical Body Fat

Soon after the "critical body weight" hypothesis had been set forth, Kennedy, (1969) postulated a "critical amount of body fat" was required for attainment of puberty in the rat. It was later observed in young girls (Frisch et al., 1973) that percentage body fat and water did not vary with age at menarche and was then theorized that menarche was due to changes in body fat (Frisch and McArthur, 1974). The validity of this theory has been questioned in many species as body fat is not constant at puberty in nutritionally manipulated rats (Glass, et al., 1979), mice (Hansen et al., 1983), and beef heifers (Grass et al., 1982; Brooks et al., 1985). Bronson and Manning (1991) also questioned whether the "critical body fat" hypothesis actually applied to menarche in humans. Most reports in the literature would agree that body fat does not play a direct role in attainment of puberty but is an important component of total body energy balance.

Body Energy Balance

The current hypothesis of "whole-body energy balance" suggests that availability of total body energy, of which energy stored in adipose tissue is an important component, rather than specific substrate, metabolite, and/or hormone, modulates the activity of the GnRH pulse generator in regulating ovulation in both the prepubertal and adult female (Bronson and Manning, 1991; Schillo et al., 1992). This theory is supported by research linking the importance of metabolic fuels, glucose and fatty acids, to estrous cycle termination in rodents. An acute fast blocked estrous cycles immediately in lean hamsters

but not until several estrous cycles in obese hamsters (Schneider and Wade, 1989). Pharmacologic blocking of glycolysis and fatty acid oxidation blocked estrous cycles in ad lib fed hamsters (Schneider and Wade, 1989) and inhibited pulsatile LH secretion in ovariectomized lambs (Hileman et al., 1991). In beef heifers, factors that alter glucose availability like the VFA, propionate, (Trenkle, 1981) enhance LH release (Randel and Rhodes, 1980; Rutter et al., 1983) and decrease age at puberty (Mosely et al., 1977, 1982; McCartor et al., 1979). Pharmacologic blocking of glycolysis and fatty acid oxidation inhibited pulsatile LH secretion in ovariectomized lambs (Hileman et al., 1991).

Endocrine Events at Puberty

Gonadotropins

Luteinizing hormone (LH) and follicle stimulating hormone (FSH) are the primary gonadotropins controlling reproduction in the bovine (Hansel and Siefart, 1967). Both LH and FSH are synthesized by gonadotrophs (Childs et al., 1987) in the anterior pituitary and their secretion is controlled by gonadotropin releasing hormone (GnRH). GnRH is synthesized by neurosecretory cells in the hypothalamus (McCann, 1974), released into the hypothalamic-hypophyseal portal vessels, binds to plasma membrane receptors in the anterior pituitary (Wise et al., 1984), and stimulates the synthesis and secretion of LH and FSH (Noah and Childs, 1986).

Luteinizing hormone (LH) is released from the anterior pituitary in a pulsatile fashion (Rahe et al., 1980). Pulses of LH have been identified in the circulation of heifers as early as 1 mo of age (Schams et al., 1981; Schillo et al., 1982a) and exogenous GnRH induces LH release shortly after birth with the response increasing with age (Schams et al., 1981). Therefore, the hypothalamic-pituitary axis appears to be functional prior to puberty. Ovariectomy of calves as young as 1 mo of age resulted in an increase in

circulating concentrations of LH (Odell et al., 1970) with similar responses in 3, 6, and 9 mo old heifers (Anderson et al., 1981). Exogenous estradiol suppressed pulsatile LH release in ovariectomized heifers and the magnitude of depression decreased as heifer age increased (Schillo et al., 1982b). It appeared that estradiol has a negative feedback effect on LH secretion during the prepubertal period.

Day et al. (1984) conducted a series of experiments to characterize the estradiol negative feedback effect on LH release. Ovariectomy during the prepubertal period resulted in an immediate increase in frequency of LH pulses (Day et al., 1984) and the postovariectomy increase in pulse frequency could be blocked by exogenous estradiol. The ability of estradiol to suppress pulse frequency in ovariectomized heifers decreased with age and coincided with attainment of puberty in intact heifers. The number of estradiol receptors in the medial basal hypothalamus and pituitary decline before puberty (Day et al., 1987) in association with decreased responsiveness to negative feedback. It was concluded that small concentrations of estradiol from the ovaries of the prepubertal heifer inhibited pulsatile release of LH via negative feedback on the hypothalamic-pituitary axis to delay puberty.

Serum concentrations of LH, in monthly samples, gradually increased during the 110 d prior to puberty in heifers (Swanson et al., 1972). Gonzalez-Padilla et al. (1975) found large fluctuations in LH during the 60 d prior to the first estrus, in daily samples taken every 6 hr, but mean concentrations of LH did not increase during this period. Both studies indicated that maximal LH or surges were associated with behavioral estrus.

Kinder et al. (1987) summarized an extensive series of experiments conducted in his lab to characterize the secretory patterns of LH of heifers during the prepubertal period. They demonstrated that mean serum concentrations of LH tended to increase in a gradual linear fashion, pulse amplitude decreased in a cubic fashion, and pulse frequency increased in cubic manner during the 140 d period to puberty. Pulse frequency was the best indicator of the onset of puberty, and the greatest change in pulse frequencies

occurred during the 50 d prior to puberty. Acute administration of GnRH induces an LH surge and results in ovulations in prepubertal lambs (Foster et al., 1984; Keisler et al., 1985) and 12 mo old heifers (Tortonese et al., 1990) while GnRH administration in 4 to 5 mo old prepubertal heifers (McLeod et al., 1985; Skaggs et al., 1986) resulted in LH-like surges and ovulation in some heifers (Skaggs et al., 1986).

The critical event necessary for ovulation in the prepubertal heifer appears to be increased pulsatile release of LH. Restricted nutrient intake decreased pulsatile LH release in rats (Bronson, 1986), monkeys (Schreihofer et al., 1993a), and cattle (Imakawa et al., 1986; Richards et al., 1989a) that were exhibiting normal cycles. Nutritional anestrus in cattle (Imakawa et al., 1986; Richards et al., 1989a) was associated with decreased LH pulse frequency. Undernutrition impairs pulsatile release of LH in prepubertal rats (Bronson, 1986), lambs (Foster and Olster, 1985) and beef heifers (Day et al., 1986: Kurz et al., 1990; Hall et al., 1994) resulting in delayed puberty. Reduced pulsatile LH secretion can be reversed with increased nutrient intake in the rat (Bronson, 1986), lamb (Foster et al., 1986), beef heifer (Day et al., 1986; Kurz et al., 1990; Hall et al., 1994) and the nutritional anestrous beef cow (Richards et al., 1989a). Evidence in growth restricted lambs suggest that anterior pituitary function was not compromised by undernutrition (Foster et el., 1989; McShane and Keisler, 1991) but reduced pulsatile LH secretion arises from a deficiency of endogenous GnRH release (Foster et al., 1989; Ebling et al., 1990). Recent work in the lamb (Ebling et al., 1990) and monkey (Schreihofer et al., 1993a,b) suggest that stress signals, resulting from restricted nutrient intake, do not appear to be major inhibitory mechanisms limiting GnRH release. The nutritional/metabolic signals rather than relief of stress, lead to resumption of pulsatile LH release.

The role of FSH in the puberal process is unclear and has received little attention. Reports are conflicting as to secretory pattern of FSH. Schams et al. (1981) reported that FSH was released at a frequency of 1 to 2 pulses per 8 h in heifers at 1, 2, 5, and 10 mo of age with no evidence for increased pulse frequency associated with age. Page et al.

(1987) observed no evidence of episodic release of FSH in 10 mo old prepubertal heifers while McLeod et al. (1985) were unable to induce pulsatile secretion of FSH by GnRH treatment. However, when the ovary containing the largest follicle was removed via ovariectomy in prepubertal heifers, there was a transient increase in FSH secretion (Johnson et al., 1985; Tortonese et al., 1990) which resulted in compensatory hypertrophy of the remaining ovary (Johnson et al., 1985). Hypertrophy of the remaining ovary was blocked by exogenous charcoal-extracted follicular fluid (remove steroid components). Therefore, the ovary produces both steroidal and nonsteroidal components that stimulate and inhibit secretion of FSH, which is important in follicle recruitment (Draincourt, 1991).

Steroids

Ovarian steroids exert facilatory and inhibitory effects on the release of gonadotropins. Increased estrogen during the follicular phase of the estrous cycle in the cow initiates the LH surge that induces ovulation (Hansel and Convey, 1983). The facilatory mechanism of estradiol on LH secretion in prepubertal heifers appears to develop between 3 to 5 mo of age (Staigmiller et al., 1979; Schillo et al., 1983a). However, minimal concentrations of estradiol in prepubertal heifers exhibit an inhibitory effect on LH secretion (Day et al., 1984; Kinder et al., 1987) which decreases as the heifer becomes older (Schillo et al., 1982a; Day et al., 1984).

Early reports postulated that pulsatile secretion of LH, on the basis of LH measurements, was due to differential suppression by ovarian steroids (Goodman and Karsch, 1980). It was hypothesized that estradiol decreased pulse amplitude but not pulse frequency by suppressing the response of the pituitary to GnRH secretion. In contrast, progesterone decreased pulse frequency without reducing pulse amplitude by suppressing tonic LH secretion by acting on the brain to decrease frequency of GnRH pulses. Direct measurement of GnRH in the hypophyseal-portal blood pathway in the ewe provide

evidence that steroids, estradiol and progesterone, elicit their negative feedback effects by acting on the brain to inhibit pulsatile GnRH secretion (Karsch et al, 1987). The authors concluded that progesterone slows the pulse generator and results in low frequency pulses of LH during the luteal phase of the estrous cycle while estradiol suppresses pulsatile GnRH during times of anestrus.

Estrogen also has a facilatory role at the anterior pituitary to enhance LH secretion. Exogenous estradiol increases the capacity of the anterior pituitary to release LH in response to GnRH in ewes (Reeves et al., 1971) and cows (Kesner et al., 1981). Administration of estradiol increased receptors for GnRH in the pituitary of ovariectomized cows prior to the ovulatory surge of LH (Schoenemann et al., 1985). Increased exposure of the estrogen stimulated anterior pituitary to pulsatile releases of GnRH progressively increased the responsiveness of LH release, thereby creating the LH surge (Hansel and Convey, 1983). Therefore, increased GnRH secretion and increased pituitary responsiveness are necessary for the preovulatory surge of LH in cattle (Kesner and Convey, 1982). Nett et al. (1987) theorized that estradiol receptors increased in the anterior pituitary prior to the preovulatory surge of LH and were associated with the increased responsiveness to GnRH. In an elegant study utilizing ovariectomized ewes in which the pituitary gland was surgically disconnected from the hypothalamus, estradiol induced an increase in pituitary GnRH-receptors (Gregg and Nett, 1989) via classic gene expression (Gregg et al., 1989).

Increased estrogen during the follicular phase of the estrous cycle in the cow has long been associated with the surge of LH that induces ovulation (Hansel and Convey, 1983). Results of a recent study indicate that estradiol acts centrally upon the GnRH neurosecretory system to increase GnRH secretion, and thereby induces the ovulatory surge in the ewe (Moenter et al., 1990). The GnRH and LH surges appear to begin together, but the GnRH surge continued well beyond the LH surge (Moenter et al., 1990). The authors stressed that their finding in no way eliminated the importance of the pituitary

as a possible site for estradiol to exert either positive or negative effects on gonadotropin secretion.

Progesterone decreases serum concentrations of LH by reducing the frequency of GnRH pulses that are released from the hypothalamus (Goodman and Karsch, 1980; Karsch et al., 1987). Increased concentrations of progesterone during gestation (Little et al. 1982), the luteal phase of the estrous cycle (Rahe et al., 1982; Hansel and Convey 1983), and during exogenous administration (Hansel and Convey, 1983) decrease concentrations of LH. Progesterone increased prior to first ovulation in beef heifers (Gonzalez-Padilla et al., 1975) but at lesser concentrations and duration than those characteristic of the luteal phase of the bovine estrous cycle (Hansel and Convey, 1983). Gonzalez-Padilla et al. (1975) theorized that progesterone may have a facilatory role to establish competency of the hypothalamic-pituitary-ovarian axis to stimulate the LH surge in the prepuberal heifer similar to that in the cyclic cow. However, not all heifers exhibit a transient increase in progesterone before the first ovulation and estrus (Rutter and Randel, 1986). In contrast, progesterone is required for induction of estrus in ovariectomized anestrous ewes treated with estrogen (Robinson, 1954). This sequence of hormonal events may help to explain why the first, and possibly the second, ovulation is not accompanied by behavioral estrus in prepuberal lambs (Foster et al., 1986). Sources of progesterone in the bovine can be ovarian in nature (Berardinelli et al., 1979; Spicer et al., 1987) and from the adrenal gland (Wagner et al., 1979).

Growth Hormone (GH) / Insulin-like Growth Factor-I (IGF-I)

Growth hormone is secreted from the anterior pituitary in a pulsatile fashion and the liver and chondrocytes in the epiphysial plates are its primary targets (Malven, 1993). Growth hormone expresses a variety of metabolic effects that include insulin-like and diabetogenic actions on carbohydrate metabolism and the ability to mobilize lipids (Wallis,

1988). The kidney, mammary gland, and anterior pituitary gland also synthesize GH receptors (Hauser et al., 1990). The major receptor mediated event of GH appears to be secretion of IGF-I and the liver is the primary source of circulating IGF (Schwander et al., 1983). Receptors for IGF are located in fibroblast, chondrocytes, and osteoblast (Rechler and Nissley, 1985). Circulating IGF-I completes a feedback loop, similar to gonadal steroids on pituitary function, to decrease further release of GH in well nourished animals (Malven, 1993). Expression of IGF-I in GH deficient transgenic mice resulted in normal somatic and bone growth indicating the importance of IGF-I in postnatal growth (Behringer et al., 1990).

Restricted nutrient intake increases GH concentrations in rams (Clarke et al., 1993), steers (Brier et al., 1986; Mosely et al., 1988; McKinnon et al., 1993), and heifers (Houseknecht et al., 1988; Granger et al., 1989; Simpson et al., 1991; Armstrong et al., 1993), and decrease IGF-I concentrations in ram lambs (Clarke et al., 1993) steers (Brier et al., 1986; McKinnon et al., 1993) cows (Richards et al., 1991; Nugent et al., 1993) and heifers (Houseknecht et al., 1988; Granger et al., 1989; Armstrong et al., 1993). The positive relationship that exists between GH and IGF-I is uncoupled in feed restricted ruminants (Elsassar et al., 1989: Granger et al., 1989; Armstrong et al., 1993) and humans (Phillips, 1986; Ho et al., 1988). In contrast, decreased nutrient intake results in decreased GH concentrations in rats (Tannenbaum et al., 1979). Decreased nutrition increases pulse amplitude but does not influence pulse frequency which probably causes mean concentrations of GH to increase (Brier et al., 1986; Houseknecht et al., 1988; Thomas at al., 1991; Clarke et al., 1993). Others have attributed the differences in GH concentrations induced by dietary restriction only to differences in pulse frequency (Villa-Goodoy et al., 1990; Armstrong et al., 1993).

Increased GH concentrations during nutrient restriction are associated with increased NEFA concentrations, indicative of negative energy balance (Brier et al., 1986; Eisemann et al., 1986; Peters, 1986; Richards et al., 1989b). Increased circulating GH

concentrations increase lipolysis and alter lipogenesis (Di Marco et al., 1981; Eisemann et al., 1986; Peters, 1986) while mobilization of fatty acids from the adipocyte (Bines and Hart, 1982) provides energy for many cellular processes. Growth hormone may act as a homeostatic regulator in near maintenance diets to support lean tissue accretion (Eisemann et al., 1986; Peters, 1986) and to supply metabolic fuels for reproductive function (Schneider and Wade, 1989; Hileman et al., 1991) by altering metabolism of body tissues. The role of intermediary metabolism on reproductive function will be discussed later in this review.

Studies attempting to link GH with attainment of puberty have been inconclusive. Bucholtz et al. (1990) theorized that increased LH secretion near puberty is associated with decreased GH secretion in the ewe lamb, and increased GH during nutrient restriction inhibits LH secretion. They concluded that neuroendocrine sexual maturity was not compromised in ovariectomized estrogen treated prepubertal lambs infused with pituitary derived bovine GH compared with untreated lambs. Chronic treatment with GH did not influence age at puberty in beef (McShane et al., 1989; Hall et al., 1994) and dairy heifers (Murphy et al., 1991) nor did it influence LH pulse frequency and pulse amplitude (Suttie et al., 1991; Hall et al., 1994). In contrast, male rats (Arsenijevic et al., 1989) and beef heifers (Simpson et al., 1991) immunized against growth hormone-releasing factor had delayed puberty and IGF-I production was significantly decreased. Growth hormone treatment stimulates follicular growth in pigs (Spicer et al., 1990) and cows (De La Sota et al., 1991) and growth hormone-releasing factor increased the size of large follicles (Spicer & Enright, 1991) in heifers. Growth hormone influences granulosa cell function in vitro in the bovine (Langhout et al., 1991), rat (Advis et al., 1981; Jia et al., 1986), and pig (Hsu and Hammond, 1987).

The importance of GH in the prepubertal growth spurt is evident by decreased prepubertal growth patterns in GH-deficient children (Tanner et al., 1976; Aynsley-Green et al., 1976) which can be augmented by administration of GH and gonadal steroids

(Tanner et al., 1976; Aynsley-Green et al., 1976). The importance of sex steroids in promoting prepubertal growth has been demonstrated in children with precocious puberty and GH deficiency (Rappaport et al., 1987; Attie et al., 1990). Increased prepubertal levels of gonadal steroids have dramatic effects on circulating GH concentrations which are associated with increased GH pulse amplitude and unaltered GH pulse frequency in middle to late prepubertal boys and girls (Ross et al., 1987; Rose et al., 1991; Martha et al., 1992).

Concentrations of circulating IGF-I increase prior to puberty in female rodents (Handelsman et al., 1987), primates (Copeland et al., 1982), humans (Hiney et al., 1991) and beef heifers (Jones et al., 1991). Decreased IGF-I concentrations were positively associated with delayed puberty in heifers fed low quality hay compared to heifers fed hay plus supplement (Granger et al., 1989). In postpartum cows (Rutter et al., 1989; Nugent et al., 1993), IGF-I was negatively correlated to the postpartum anestrous interval when adequate nutrients were available. Houseknecht et al. (1988) concluded that dietary energy had a greater influence than energy source on IGF-I concentrations. Ovarian follicular fluid and serum concentrations of IGF-I are positively correlated (Echternkamp et al., 1990; Lucy et al., 1992a) and IGF-I may have direct local effects on granulosa cell mitosis and production of estradiol (Spicer et al., 1993) to stimulate follicular growth and eventual ovulation. Results of most studies in ruminants would agree that there is a positive association between circulating concentrations of IGF-I and increased nutrient intake.

The roles of GH and IGF-I in attainment of puberty are inconclusive. Both GH and IGF-I are modulated by availability of nutrition, however, the manner in which GH and IGF-I affect reproductive function has not been elucidated. GH appears to exert its effect through intermediary metabolism and by stimulating IGF-I secretion, and possibly by a direct effect on ovarian function. Whereas, IGF-I which exhibits autocrine and

paracrine actions at the ovary, appears to have a functional role in mediating cellular function and/or a metabolic effect.

Insulin

The role of insulin in intermediary metabolism and its role in the regulation of blood glucose concentrations has been well established. However insulin's role as a possible mediator in reproduction and more specifically hypothalamic-pituitary function is less clear. Concentrations of insulin and receptor numbers are increased in the hypothalamus and particularly the arcuate nucleus (Schwartz et al., 1992), the location of the GnRH pulse generator (Knobil, 1989) in the monkey, compared with the dorsomedial nucleus of the hypothalamus. The arcuate nucleus is also the site of synthesis of neuropeptide Y (Schwartz et al., 1992) which has been implicated in the regulation of GnRH secretion (see discussion below). The hypothalamus is also involved in food intake and body weight regulation (Schwartz et al., 1992).

Nutrient restriction decreases pulsatile release of LH (Day et al., 1986; Richards et al., 1989a; Kurz et al., 1990) and alters reproductive function. Induced nutritional anestrus in nonlactating cows is associated with decreased insulin and glucose concentration (Richards et al., 1989b) and decreased LH pulse frequency (Richards et al., 1989a). The glucoregulatory effect of insulin was compromised in nutritional anestrus cows infused with glucose. Glucose disappearance was retarded and insulin remained increased for a longer time compared with controls. Systemic insulin infusion in ovariectomized ewes resulted in decreased blood sugar and decreased LH pulse frequency (Clarke et al., 1990) an event that was prevented by glucose infusion. The authors suggested that reduced blood sugar in the central nervous system and not the direct effect of insulin caused the reduced LH secretion. In contrast, systemic infusion of insulin during the estrous cycle of beef cows (Harrison and Randel, 1986) and short term

intracerebroventricular infusion in growth restricted ovariectomized ewes did not alter LH secretion (Hileman et al., 1993). In the later study, upon refeeding of the previously growth restricted ewes, insulin infusion decreased LH pulse frequency and mean LH.

In the human, insulin resistance, with compensating hyperinsulinemia, is a normal phenomenon of puberty (Hindmarsh et al., 1988; Caprio et al., 1989) and the rise in GH during puberty maybe responsible for defective insulin action (Bartush-Marriam et al., 1982). By administering GH to adults, Bartush-Marriam et al.(1982) were able to mimic the prepubertal insulin-resistance state by modifying peripheral glucose uptake with out effecting the anabolic actions of insulin. Insulin resistance appears to be restricted to glucose metabolism and does not effect amino acid metabolism. Insulin may also have a regulatory function by influencing insulin-like growth factor binding proteins and sex hormone-binding globulin in humans. Insulin has been implicated in mediating insulin-like growth factor binding protein-1 concentrations in plasma (Nobels and Dewailly, 1992). insulin-like growth factor binding protein-1 may function to regulate IGF-1 activity by acting as a competitive binding site and decreasing IGF-1 action with receptors (DeVroede et al., 1986). Sex hormone-binding globulin declines throughout puberty (Apter et al., 1984; Holly et al., 1989) and the decrease may be caused by insulin (Nobels and Dewailly, 1992). Nobels and Dewailly, (1992) have suggested the following endocrine interactions that partially regulate pubertal development in humans. Insulin, GH, and IGF-I, as well as the gonadotrophs, act together to control ovarian function. Secretion of GH stimulates IGF-I production which in turn stimulates the ovary via its mitogenic activity and ability to potentiate the effects of gonadotropins. Insulin acts to decrease insulin-like growth factor binding protein-1 to increase unbound circulating IGF-1, and it decreases sex hormone-binding globulin concentrations to influence steroid concentrations. This process could be functional in the ruminant because GH, insulin, and IGF-I appear to have similar endocrine and metabolic functions in humans and ruminants.

Metabolic Signals

Metabolic fuels in the ruminant can be obtained from exogenous substrates, metabolic processes that occur within the animal such as gluconeogenesis, and from catabolism of body tissue such as lipolysis (Trenkle, 1981). As discussed in the previous section, LH appears to be the primary factor controlling the initiation of puberty and its secretory activity is severely altered by nutritional restriction. As a result, this section will deal with the mechanism in which nutrition influences metabolism and how availability of metabolic fuels influences LH secretion.

Food deprivation in Syrian hamsters blocked estrous cycles which was dependent on body weight before starvation (Schneider and Wade, 1989). Acute starvation blocked estrous cycles in the Syrian hamster, an effect that was countered by adding glucose to the drinking water (Morin, 1986). Acute fasting in monkeys decreased pulsatile release of LH an effect that was reversed by intragastric infusion of nutrients (Schreihofer et al., 1993a,b). Brief periods of fasting in men (Cameron et al., 1991), and male monkeys (Parfitt et al., 1991) alter the hypothalamic-pituitary-testicular axis, a response that is reversible with increased nutrient intake. Reduced concentrations of glucose were associated with cessation of estrous cycles after prolonged nutrient intake in the gilt (Armstrong and Britt, 1987) and nonlactating beef cow (Richards et al., 1989b). Glucose supply in the ruminant is either of exogenous origin from feedstuff or endogenous origin via gluconeogenesis with propionate being the major gluconeogenic substrate (Trenkle, 1981). Anestrus was associated with decreased weekly LH concentrations and pulse frequency in the beef cow (Richards et al., 1989a). Infusion of glucose did not influence LH secretory patterns in postpartum ewes (Rutter and Manns, 1986) and cows (McCaughey et al., 1988) on adequate planes of nutrition. In contrast, Garmendia (1986) reported that glucose infusion increased LH pulses and mean concentrations of LH during treatment with GnRH in lactating anestrous beef cows.

An inhibitor of glycolysis, 2-deoxy-D-glucose (2DG), and phlorizin, which causes glucosuria, have been used to evaluate the effect of altered glucose metabolism on LH secretion in ruminants. Infusions of 2DG had no effect on LH secretion in good condition ewes (Hileman et al., 1991). Conversely, 2DG infusion reduced the magnitude of estrogen induced LH surges with no effect on GnRH-induced release of LH in ewes (Crump et al., 1982). Estrus and ovulation were blocked in cows infused with 2DG (McClure et al., 1978). Others have induced hypoglycemia with phlorizin and reduced pulse amplitude of LH in moderate condition cows during the follicular phase of the estrous cycle (Rutter and Manns, 1988) and postpartum anestrous cows (Rutter and Manns, 1987).

Increased propionate:acetate ratios can be obtained by increasing the quantity of grain in the diet or by adding ionophores to the diet. Increased propionate obtained by feeding monensin (Randel and Rhodes, 1980) or abomasal infusion (Rutter et al., 1983) resulted in an increase in LH secretion in response to GnRH injection in prepubertal heifers. Heifers fed monensin had increased LH released in an estrogen induced LH surge in prepubertal heifers (Randel et al., 1982). A direct effect of monensin on the hypothalamic-pituitary axis and ovary has not been ruled out.

Total availability of metabolic fuels rather than the deficiency or presence of a particular metabolic fuel can alter the hypothalamic-pituitary-gonadal axis. Hamsters on adequate nutrient intake and treated separately with 2DG or methyl palmoxirate (MP, fatty acid inhibitor) did not have altered estrous cycles, however, administration of both MP and 2DG together decreased the number of hamsters showing estrous cycles (Schneider and Wade, 1989). Treatment of ovariectomized lambs concurrently with 2DG and MP completely blocked pulsatile release of LH (Hileman et al., 1991). Castrated , growth retarded lambs also had decreased circulating LH concentrations when 2DG and MP were given together (Bucholtz et al., 1992).

The effects of nutrition on FSH secretion are inconclusive. No difference in FSH secretory activity was observed in restricted or overfed pre- and postpubertal dairy heifers (Spicer et al., 1984). Decreased nutrient intake had no effect on FSH secretion in ewes (Rhind et al., 1985), and gilts (Cox et al., 1987; Armstrong and Britt, 1987). In contrast, feed restricted lambs had decreased FSH concentrations (Foster et al., 1989) while FSH increased after unilateral ovariectomy in fed rats but not in feed restricted rats (Meredith and Butcher, 1985). Gilts on high energy diets and given an insulin injection had increased FSH during the first 24 hr after injection compared to insulin injected gilts on low energy diets (Cox et al., 1987).

Nonesterified Fatty Acids (NEFA)

Increased NEFA concentrations in plasma are indicative of negative energy balance (Brier et al., 1986; Eisemann et al., 1986; Peters, 1986; Richards et al., 1989b) and are associated with increased circulating GH concentrations (Brier et al., 1986; Houseknecht et al., 1988; Granger et al., 1989 McKinnon et al., 1993; Armstrong et al., 1993), enhanced lipolysis, altered lipogenesis (DiMarco et al., 1981; Eisemann et al., 1986; Peters, 1986), and fatty acid release from the adipocyte (Bines and Hart, 1982) in ruminants. Decreased reproductive function of dairy cows in negative energy balance was associated with increased NEFA concentrations (Canfield and Butler, 1991). When nutritional anestrus was induced in cows (Richards et al., 1989a,b), and gilts (Armstrong and Britt, 1987) plasma concentrations of NEFA increased and LH pulse frequency decreased. Short term infusion of free fatty acids in ovariectomized mature ewes (Estienne et al., 1989) and ovariectomized ewe lambs (Estienne et al., 1990b) in a positive energy balance suppressed pulsatile GH release but did not alter pulsatile LH secretion. Because ewes were in a positive energy balance other metabolic fuels could stimulate the hypothalamus to alter LH secretion and any inhibitory effect of free fatty acids may have

been masked by metabolic fuels already present. Exogenous free fatty acid infusions decreased GH concentrations in rats (Imaki et al., 1986), humans (Imaki et al., 1985; Casanueva et al., 1987) , and sheep (Heretelendy and Kipnis, 1973; Sartin et al., 1988).

Excitatory Amino Acids

Amino acids have also been implicated as having a role in LH secretion. Intravaneous infusion of ovariectomized growth restricted lambs with a 5% amino acid solution increased LH pulse frequency compared with noninfused growth restricted controls (Bucholtz et al., 1988). However, it was not determined if a specific amino acid influenced LH secretory activity more than another. Long term abomasal infusion (22 d) of tyrosine to ovariectomized feed restricted lambs resulted in a small but significant increase in LH pulse frequency compared with controls infused with water (Hall et al., 1990).

Asparatate is an amino acid that has been implicated in mediating LH secretion. Aspartate appears to be a natural ligand and receptors are named for the analogue which binds to them. Aspartate's receptor is known as *N*-methyl-D aspartic acid receptor (Cowell, 1993). Infusion of *N*-methyl-D,L-aspartate (NMA), an analogue to aspartate, induces LH secretion in prepubertal rats (Urbanski and Ojeda, 1987), monkeys (Gay et al., 1987; Plant and Medhamurthy, 1989), gilts (Estienne et al., 1993), and sheep (Estienne et al., 1990a; I'Anson et al., 1990). Daily administration of NMA accelerates vaginal opening and first ovulation in rats (Urbanski and Ojeda, 1987) and results in initiation of spermatogenesis in male monkeys (Plant et al., 1989). *In vitro*, NMA is effective in directly stimulating GnRH release from hypothalamic tissue (Bourguignon et al., 1989) but not from cultured pituitary cells (Tal et al., 1983). The NMA-induced LH release was blocked in the presence of a GnRH antagonist in prepubertal male monkeys (Plant et al., 1989) and castrated male rats (Strobl et al., 1993) while administration of a GnRH

antagonist failed to suppress NMA stimulated FSH secretion to the same degree as LH secretion (Strobl et al., 1993). The later study and others suggest that FSH secretion is not directly stimulated by GnRH pulses in female (Grady et al., 1985) and male rats (Culler and Negro-Villar, 1986) but is directly influenced by other neuronal and/or hormonal inputs. Injections of GnRH antagonist in wethers effectively inhibited LH secretion but not FSH secretion (Grotjan et al., 1992). Cowell (1993) suggested that excitatory amino acids not only effect the secretion of GnRH from releasable pools but also appear to act at the genomic level. Acute stimulation of rats with NMA increased GnRH mRNA levels (Petersen et al., 1991), affects that are facilitated by pretreatment with ovarian steroids (Carbone et al., 1992). Whether GnRH neurons have receptors for NMA or the close proximity of NMA receptors to GnRH neurons causes stimulation of GnRH neurons is unknown. It has been theorized in monkeys (Plant and Medhamurthy, 1989) that during the prepubertal period there is an absence of an effective stimulus to the gonadotrophs resulting in a random, unsynchronized firing of GnRH neurons that lead to constant, low level delivery of GnRH to the pituitary to stimulate LH release. As a result, adequate secretion of LH may not occur until the central nervous system is developed so that a synchronous firing of GnRH neurons occurs.

Neuropeptide Y

Neuropeptide Y (NPY) is a 36 amino acid peptide of neural origin composed of amino- and C-terminal tyrosine residues (Tatemoto, 1982). There is immunreactivity to NPY in several areas of the sheep brain including the hypothalamus (Pfaff et al., 1989) and the activity is closely associated with GnRH cells in the hypothalamus (Pfaff et al., 1989). Neuropeptide Y modulates feed consumption in the rat (Clark et al., 1984), sheep (Miner et al., 1989), and pig (Parrott et al;., 1986) when injected intracerebroventricularly. Long term nutrient restriction in rats increased mRNA for NPY in the arcuate nucleus (Brady et

al., 1990; O'Shea and Gundlach, 1991) and long term feed restriction in ovariectomized estrogen treated ewes increased NPY concentrations in laterocerebro-spinal fluid (McShane et al., 1992). Neuropeptide Y has been linked to gonadotropin secretion, mediating feedback effects of gonadal steroids, and the onset of puberty. Intracerebroventricular administration of porcine NPY in ovariectomized and ovariectomized + estrogen treated ewes altered LH secretory activity in a dose dependent manner but had no effect on GnRH induced secretion of LH (McShane et al., 1992). Immunoreactivity of NPY in the hypothalamus of rats increased from birth until vaginal opening (Sutton et al., 1988). Administration of antibodies to NPY prevented the preovulatory LH surge prior to first ovulation in the rat (Minami et al., 1990) and the steroid-induced LH surge was blocked when NPY antibodies were injected intracerebroventricularly (Wehrenberg et al., 1989). *In vitro*, NPY increased the release of GnRH from medial basal hypothalamic and median eminence fragments of rats treated with ovarian steroids compared to untreated rats (Crowley and Karla, 1987; Sabatino et al., 1989). In a series of experiments conducted in prepubertal monkeys (Gore et al., 1993), increases in NPY release occurred during puberty, in parallel to GnRH release, infusion of NPY in the early prepubertal period did not effect GnRH release, but it did cause significant release in midpubertal monkeys, infusion of antibodies to NPY suppressed pulsatile GnRH release in midpubertal but not prepubertal monkeys. The authors concluded that in the female monkey the maturation of the NPY neuronal system contributes to the pubertal increase in GnRH release and NPY may be involved in the mechanism controlling the onset of puberty.

Ovarian Function Preceding Puberty

The ovaries of prepubertal heifers are responsive to gonadotropins and are capable of ovulation (Casida et al., 1943; Howe et al., 1962; Seidel et al., 1971) virtually from

birth, although results are quite variable. An increased number of induced ovulations occurred at 5 mo compared with 1 mo of age (Seidel et al., 1971).

The manner in which follicles grow and develop was initially difficult to determine because the reproductive tract could only be examined by sacrificing the animal or surgical removal of the reproductive tract via laparotomy. Through the process of serial slaughter of prepubertal heifers, Rajakoski (1960) hypothesized that follicular growth occurred in a wave-like fashion and two dominant follicles developed during the estrous cycle with the second being the ovulatory follicle. Ovaries of pre- and postpubertal heifers were palpated twice weekly in a study conducted by Swanson et al. (1972). Large numbers of follicles greater than .5 cm were recorded in prepubertal heifers and follicle growth and atresia was reported during the estrous cycle in agreement with Rajakoski (1960). Rajakoski's (1960) follicular growth hypothesis was confirmed utilizing ultrasonography (Pierson and Ginther, 1987) in cattle with normal estrous cycles. It was later determined that one to three follicular waves can occur during the estrous cycle (Sirois and Fortune, 1988). Turnover of dominant follicles, ranging from 8 to 12 mm, occurs before the onset of puberty in heifers (Roche and Boland, 1991; Hooper et al., 1993; Evans et al., 1993) apparently in a wave like fashion similar to those observed in cattle with estrous cycles (Pierson and Ginther, 1987; Sirois and Fortune, 1988).

Continual growth of antral follicles to development into preovulatory follicle is known as "follicular dynamics", a process that consist of recruitment, selection, and dominance as defined by Hodegn (1982) in primates. Recruitment is the process where groups of follicles mature under the proper gonadotropic support that permits progress towards ovulation. Selection is the process by which a single follicle avoids atresia and obtains the competence to become an ovulatory follicle. Dominance is when the selected follicle dominates through inhibition of recruitment of a new group of follicles. Follicular growth, development, and turnover has also been described using similar definitions in beef cattle (Ginther et al., 1989; Sirois and Fortune, 1988, 1990; Lucy et al., 1992b).

Species differences exist in selection of ovulatory size dominant follicles. In rats, primates and pigs, dominant follicle development is confined to the follicular phase of the estrous cycle and is directed toward ovulation. Whereas in cattle, sheep, and horses, dominant ovulatory size follicles occur throughout the estrous cycle with the dominant follicle present during the follicular phase being destined for ovulation (Fortune, 1994). Follicular dynamics and factors that modulate the process in the prepubertal heifer have yet to be researched.

Behavioral estrus in the absence of ovulation and corpora lutea development has been coined "nonovulatory estrus" and has been described in beef heifer (Swanson et al., 1972; Nelson et al., 1985; Rutter and Randel, 1986) and could reflect growth of large dominant follicles (Pebbles et al., 1991; Roche and Boland, 1991; Evans et al., 1993; Hooper et al., 1993) a primary source of estradiol in prepubertal animals (Tortonese et al., 1990). Occurrence of the nonovulatory estrus ranges from 13 to 63% (Nelson et al., 1985; Rutter and Randel, 1986). Transient increases in circulating progesterone concentrations are consistently found during the weeks prior to pubertal estrus (Gonzalez-Padilla et al., 1975; Berardinelli et al., 1979) and is ovarian in nature (Berardinelli et al., 1979). More heifers that ovulated at their first estrus had increased progesterone concentrations before the first estrus and the progesterone concentration was greater than in heifers that had a nonovulatory first estrus (Rutter and Randel, 1986). Therefore, observation of estrus should not be used as the sole criterion for puberty. Short estrous cycles, 8 to 11 d in length, are frequently observed after the first estrus in heifers (Swanson et al., 1972; Nelson et al., 1985; Rutter and Randel, 1986). Utilizing ultrasonography, short estrous cycles are a result of a short-lived corpora lutea preceded by ovulation and not luteinized follicles (Fajersson and Edquist, 1993). Knowledge of these prepubertal or pubertal events could prove to be important since fertility of the pubertal estrus is decreased compared to the third estrus in heifers (Byerley et al., 1987) which could have a major effect on pregnancy rates for short breeding seasons.

Factors Affecting Age at Puberty

Age/Weight

Age at puberty is a term that is used quiet loosely in terms of its relationship with puberty. Age is a continuous time variable that cannot be modified by human intervention. Therefore, the importance of age on initiation of puberty is difficult to determine because it cannot be experimentally altered. Weight, however, is a variable that can be altered experimentally and is a function of birth weight, nutrient intake, and age. The effect of weight on initiation of puberty has been extensively studied. There is a negative relationship between age and weight at puberty with faster growing heifers being younger but heavier at puberty (Short and Bellows, 1971; Arjie and Wiltbank, 1974; Grass et al., 1982; Ferrell, 1982; Greer et al., 1983).

Onset of puberty after weaning may be limited by age in heavyweight heifers and by weight in lighterweight contemporaries (Mosely et al., 1982). Puberty is a physiological event that can be altered by nutrition and environment to occur at either younger or older ages. Short et al. (1994) concluded that age and weight at puberty are primarily a function of genetics and nutrition. The difficulty is determining experimentally which variable, age or weight, has more influence on initiation of puberty.

Genetic

Genetics have a major influence on age and weight at puberty (Short et al., 1994). The importance of genetics may be masked by increased nutrient intake, which does not allow heifers to express their genetic potential for younger ages at puberty. In general, faster gaining breed groups of large mature size (e.g, Charolais, Chianina) are older and weigh more at puberty than slower-gaining breed groups of smaller mature size (e.g.,

Hereford, Angus; Martin et al., 1992). Furthermore, breeds that have been historically selected for milk production (e.g., Holstein) reach puberty at younger ages than breeds that have not been selected for milk production (Laster et al., 1976, 1979; Gregory et al., 1979; Ferrell, 1982; Grass et al., 1982; Martin et al., 1992). However, the positive relationship that exists between mature size and puberty at older ages can be offset by selection for milk production within breed groups that also exhibit large mature size (e.g, Simmental, Holstein, Brown Swiss, and Gelbvieh) compared with those of similar mature size with no selection for milk production (e.g, Charolais and Chianina; Martin et al., 1992). Brahman and Brahman-based cattle consistently reach puberty at an older age than British breeds (Gregory et al., 1979; Stewart et al., 1980). Individual sire differences also effect age at puberty suggesting that physiological maturity is influenced by genetic variation within a breed (Laster et al., 1976, 1979)

Heterosis is defined as the difference between the mean for the reciprocal F1 crosses (e.g., Angus sire x Hereford dam and Hereford sire x Angus dam) and the mean for the parental pure breeds (e.g., Hereford and Angus) contributing to the cross. In general, crossbred cattle reach puberty at a younger age than straightbreds used in the cross. Hereford x Angus calves reached puberty 20 d earlier than straightbreds with no effect on weight at puberty (Laster et al., 1979). In a four breed diallel cross design consisting of Red Poll, Brown Swiss, Hereford, and Angus breeds, crossbred calves were 9.4 d younger at puberty than contemporary straightbreds (Laster et al., 1979). In a study involving a diallel matings of Angus, Brahman, Hereford, Holstein, and Jersey cattle and their 10 reciprocals, crossbred heifers raised on pasture were 15 d younger at puberty than straightbreds, whereas pen fed crossbreeds were 11 d older at puberty than contemporary straightbreds (Stewart et al., 1980). The authors suggested that the heterotic effects were decreased by increased nutrition of pen fed heifers.

Dam breed can also effect age at puberty. Hereford sired calves out of Angus dams are younger at puberty than their reciprocal cross contemporaries (Laster et al.,

1976, 1979; Gregory et al., 1978a, 1979). Gregory et al., (1979) indicated that the decreased age at puberty in calves from Angus dams was due to breed maternal and breed transmitted effects.

Age at puberty heretability estimates average around 50% (Brinks, 1994), similar to heritablity estimates for scrotal circumference (Martin et al., 1992). Scrotal circumference is negatively correlated (genetic correlation -.71) with age at puberty of a sire's female offspring in Angus, Red Angus, and Hereford cattle (Brinks et al., 1978) while Lunstra et al. (1982) reported a correlation of -.98 among breed means for Gelbvieh, Brown Swiss, Red Poll, Angus, Simmental, Hereford, Charolais, and Limousin. Therefore, selecting for early and large scrotal circumference, should result in daughters that reach puberty earlier. Smith et al. (1989) reported a regression coefficient of age at puberty on sire scrotal circumference of -.80 d/cm. Utilizing sires of high and low scrotal circumference EPD's but with similar growth and maternal EPD's, resulted in females sired by high scrotal circumference EPD bulls that were 62 d younger at puberty compared with low EPD sires (Hough et al., 1991). These data should be viewed cautiously due to small experimental numbers.

The reproductive tract score was developed by researchers at Colorado State University (Andersen et al., 1991) as a method to evaluate and estimate puberal status by rectal palpation of uterine horns and ovaries. Heifers are assigned a score of 1 through 5. A heifer with a score of 1 has a small immature tract and ovaries with no palpable structures and is definitely not cycling , whereas a heifer with a score of 3 has a larger uterus with good tone and palpable follicles on the ovary. A heifer with a score of 5 has a palpable corpus luteum and is cycling. Heritablity estimates for RTS are small (.28) probably due in part to the distribution of RTS when the measurement is about 1 mo prior to the breeding season, and the precision of the measure. However, RTS is a predictor of reproductive performance of heifers in relation to pregnancy rates to synchronized breeding programs and breeding season pregnancy rates. Heifers with more mature

reproductive tracts had greater pregnancy rates and became pregnant earlier in the breeding season (Andersen et al., 1991).

The most important genetical approach to producing reproductively efficient females is sire selection (Brinks, 1994). Replacement heifer sires should excel in maternal and reproductive traits including optimum cow size, milk production, and large scrotal circumference. Combining sire selection with RTS to measure puberal status and to predict pregnancy rates are low cost selection methods that can be utilized in an attempt to improve reproductive efficiency. Using selection to improve reproduction efficiency should result in a permanent improvement in reproductive efficiency (Martin et al., 1992).

Preweaning Growth

Environment, genetics, growth promotants, or creep feeding can influence age at puberty. Heifers with greater preweaning growth were younger and heavier at puberty than their lighter contemporaries (Arije and Wiltbank, 1971; Varner et al., 1977: Laster et al., 1979). Increased or decreased gains, could be due to positive or negative environment effects (Gregory et al., 1979) or due to breed maternal and breed transmitted effects (Laster et al., 1979).

Creep feeding of calves increases preweaning gains. In general, free choice feeding of grain based creep feeds increases weaning weight but alters postweaning gains of heifer calves. Feeding excessive energy preweaning has a detrimental effect on subsequent producing ability of the calf as measured by number of calves produced, progeny with lighter weaning weights, and longevity (Mangus and Brinks, 1971; Kress and Burfening, 1972; Martin et al., 1981). The excess condition of the suckling calves influences subsequent development of important maternal traits (Arnett et al., 1971; Holloway and Totusek, 1973) which are due to increased fat deposition in the udder that reduces subsequent milk production (Gardner et al., 1977). However, increased

conditioning of heifers can be overcome by either limit feeding of creep or feeding high protein creep feeds (Lusby, 1989). Limit feeding of cottonseed meal for 133 d in spring born calves increased gain by 14 kg over controls and increased gain was attributed to increased forage intake of creep fed calves (Lusby, 1986). Similar results have been reported by others (Wyatt et al., 1986; Kuhl et al., 1987).

Growth Promotants

Growth promoting implants are used during the pre- and post-weaning growth phases to increase gain and in some instances pelvic area. However, their use in replacement heifers is questioned because of possible detrimental effects on reproduction. Implants of steroidal and non-steroidal compounds are available . The major steroidal implants are a combination of estradiol benzoate plus either progesterone or testosterone propionate. Non-steroidal implants mimic the action of steroids and contain either the estrogenic compound zeranol or the androgenic compound trenbolone acetate. The only implant currently approved for use in suckling replacement heifers contains estradiol benzoate plus progesterone and is sold under the trade name is Synovex-C (Syntex Agribusiness, Inc., Des Moines, IA).

Most implant programs are instituted under the premise that growth stimulants will increase growth rate of muscle and skeletal structure via increased feed efficiency while decreasing the cost of developing replacement heifers. Few studies have examined implant effect on age at puberty and the mode of action.

The effect of one implant given during the first 3 mo after birth is inconclusive. No difference in age at puberty was reported for 3 mo old heifers given one 19 mg estradiol implant (Moran et al., 1990) while two 19 mg estradiol implants given at 3 mo of age increased age at puberty by 29 d over non implanted heifers (Moran et al., 1990). A non significant increase in age at puberty of 20 d was observed in heifers receiving one

zeranol implant (Deutscher et al., 1986). Pubertal age was unchanged in 2 mo old heifers receiving one estradiol benzoate plus progesterone implant (Hancock et al., 1994) while pubertal age increased in 3 mo old heifers receiving one implant (Rusk et al., 1992). No difference was observed in percent cycling at start of breeding season, conception rate to artificial insemination (AI), or breeding season pregnancy rate for the previous two studies. Breeding season pregnancy rates were similar in heifers receiving one zeranol implant at 42 d of age compared with non-implanted controls (Muncy et al., 1979). Irrespective of implant type, a greater number of non-ovulatory estrous periods were observed for implanted heifers compared with non-implanted controls (Deutscher et al., 1986; Moran et al., 1990; Hancock et al., 1994).

Multiple implant schemes between birth and start of the breeding season are common. Heifers implanted with zeranol at approximately 42 d of age and three times at 100 d intervals had significantly reduced breeding season pregnancy rates compared with nonimplanted controls (Muncy et al., 1979). In a comprehensive study conducted by Deutscher et al. (1986), heifers received a single zeranol implant at 1, or 6, or 9 mo of age and all possible combinations of 2 and 3 implants. All implanted heifers were older at puberty compared with non-implanted but only the 6 mo implant group was significantly older. In contrast, Staigmiller et al. (1983) reported no difference in age at puberty in heifers implanted with zeranol at 8 and 11 mo of age (Staigmiller et al., 1983). In both studies, the percent puberal at the start of the breeding season and conception rates were similar to non-implanted heifers. Heifers implanted with estradiol benzoate plus progesterone at 2 and 6 mo of age had similar age at puberty, and percent puberal at the start of the breeding season (Hancock et al., 1994). In contrast, Rusk et al.(1990) found a significant decrease in both percent puberal at the start of the breeding season and breeding season pregnancy rate of heifers implanted at 3 and 6 mo with estradiol benzoate plus progesterone compared to non-implanted heifers. Moran et al. (1990) implanted approximately 84 d old heifers (start of treatment) on d 1, 84, 168, and 252 of experiment

with either trenbolone acetate (TBA), zeranol, or TBA + 38 mg estradiol and reported increased age at puberty for all treatments compared to nonimplanted controls. As with single implants, an increased number of non-ovulatory estrous periods were observed in implanted heifers compared with nonimplanted controls (Deutscher et al., 1986; Moran et al., 1990: Hancock et al., 1994).

Staigmiller et al. (1983), Deutscher et al. (1986), and Cohen et al. (1987) reported that increased nutrient intake fed to achieve ADG of greater than .50 kg/d during the postweaning phase tended to decrease the negative effects of zeranol on age at puberty and pregnancy rate in yearling heifers. However, when an additional implant was given at birth, increased nutrition did not decrease the negative effects on pregnancy rates (Cohen et al., 1987). Heifers in the Hancock et al. (1994) study were gaining greater than .50 kg/d while heifers in the Rusk et al. (1992) study were gaining well under the .50 kg/d. This may have attributed to the decrease in percent heifers cycling at the start of the breeding season in the Rusk et al. (1992) study while decreased pregnancy rates were probably a result of decreased fertility at the pubertal and second estrus (Byerley et al., 1987). Based on an extensive review of growth promotants in replacement heifers, Hargrove (1994) recommended that if replacement heifers can be identified before 2 mo of age they should not be implanted but if replacement heifers can not be identified until weaning, implant heifers at approximately 2 mo of age but not less than 1 mo of age and if a second implant is used heifers should be gaining at approximately .5 kg/day or greater.

Postweaning Gain

Undernutrition delays reproductive maturation in female rats (Bronson, 1986), sheep (Fitzgerald et al., 1982; Foster and Olster, 1985), and beef cattle (Wiltbank et al., 1966, 1969; Arjie and Wiltbank, 1971; Short and Bellows, 1971; Day et al., 1986). However, this response can be reversed by increased nutrient intake. Nutrient intake is

negatively associated with age at puberty in dairy (Sorenson et al., 1959; Menge et al., 1960) and beef heifers (Wiltbank et al., 1966, 1969; Arjie and Wiltbank, 1971: Short and Bellows, 1971; Ferrell, 1982; Greer et al., 1983;) and positively associated with weight at puberty (Wiltbank et al., 1966; Arjie and Wiltbank, 1971: Short and Bellows, 1971; Ferrell, 1982; Grass et al., 1982; Greer et al., 1983). As postweaning nutrient intake is increased resulting in greater ADG, a respective decrease in age at puberty is observed (Short and Bellows, 1971; Arjie and Wiltbank, 1974; Grass et al., 1982; Ferrell, 1982; Greer et al., 1983). However, the influence of increased ADG on age at puberty can depend on age and/or weight of heifers at weaning (Mosely et al., 1982). It has been suggested that puberty occurs at a genetically predetermined weight and height among individual animals (Lamond, 1970; Taylor and Fitzhugh, 1971). This theory was indirectly tested by Clanton et al. (1983) in Angus x Hereford heifers. Weaned calves were fed to attain a target weight at puberty at either a continuous rate of .45 kg/d, or no gain for the first half of treatment and then fed to gain .91 kg/d for the second half, or the reciprocal of the last treatment. There was no difference in age at puberty and very little difference in weight at puberty. Rates of gain never exceeded 1.1 kg/d in this study.

Optimum growth rates necessary for attainment of puberty are dependent on weight and frame size (Fox et al., 1988). However, because differences in age at puberty exist between breeds (Laster et al., 1976, 1979; Gregory et al., 1979; Ferrell, 1982; Grass et al., 1982; Martin et al., 1992), weight and frame score measurements necessary for attainment of puberty are probably going to be different between breeds.

Several studies have addressed sorting heifers based on weaning weight (Varner et al., 1977; Wiltbank et al., 1985). Heifers considered above (heavy) and below (light) the average weaning weight of contemporaries were randomly allotted to groups of light, heavy, and a combination of light/heavy heifers and fed to a single target weight (Varner et al., 1977) or to a light and heavy target weight (Wiltbank et al., 1985). Varner et al. (1977) found an advantage to sorting heifers whereas Wiltbank et al. (1985) found no

advantage irrespective of target weight. The validity of the conclusions by Varner et al. (1977) are questionable since experimental numbers were very few. Sorting of heifers may be beneficial only if lightweight heifers are given preferential treatment in the form of increased nutrients. Mosely et al. (1982) found that onset of puberty at weaning maybe limited by age in heavyweight weight heifers and by weight in lighter weight contemporaries.

Ionophores (e.g., monensin) are feed additives that increase feed efficiency and alter the propionate:acetate ratio in the rumen. Propionate is an important precursor for glucose in gluconeogenesis (Trenkle, 1981) and has a positive influence on LH secretion (discussed earlier in this review). Age at puberty was hastened in heifers fed monensin (Mosely et al., 1977, 1982; McCartor et al., 1979; Purvis et al., 1993). Puberty was hastened by feeding heifers monensin that had above average weaning weights In contrast, age at puberty was not decreased when monensin was added to the diet of heifers with below average weaning weights (Mosely et al., 1982).

In several mammalian species pheromones, play a role in regulating puberty. Presence of the male hastens the onset of puberty in mice (Vandenbergh, 1969), rats (Vandenbergh, 1976), pigs (Brooks and Cole, 1970), and cows (Izard and Vandenbergh, 1982). The presence of rams induces ovulation in anestrous ewes (Schinckel, 1954; Radford and Watson, 1957). The ability of bull exposure to hasten the onset of puberty in beef heifers is inconclusive. Short (Berardinelli et al., 1979; MacMillan et al., 1979) and long term bull exposure (Roberson et al., 1987) did not influence age at puberty while, longterm bull exposure decreased age at puberty (Roberson, et al., 1991). It has been theorized that enhanced response to bull exposure was dependent on heifer body weight (Izard and Vandenbergh, 1982) and plane of nutrition (Roberson et al., 1991). How social cues enhance reproductive function is unknown although the hypothalamic-pituitary axis has been implicated. Increased circulating LH concentrations have been found in

female mice (Bronson and Desjardins, 1974) and sheep (Martin et al., 1980) exposed to males.

Environmental Effects On Puberty

Season/Photoperiod

The effect of season on reproduction has been well documented in sheep (Karsch et al., 1984; Foster et al., 1985) and extensively studied in the *Bos indicus* female (Plasse et al., 1968; Neuendorff et al., 1984; Randel, 1984; Stahringer et al., 1990) although results are less convincing compared with sheep. Effect of season on reproduction in the *Bos taurus* female remains inconclusive.

Spring born heifers reached puberty at a younger age than fall born heifers (Hawk et al., 1954; Menge et al., 1960; Roy et al., 1980; Grass et al., 1982), while others showed the opposite (Schillo et al., 1982a) or no effect (Greer et el., 1983) of season of birth on age at puberty. Heifers born later in the spring were younger (Arjie and Wiltbank. 1971; Swiestra et al., 1977; Hansen et al., 1983; King et al., 1983) and lighter (Arjie and Wiltbank. 1971; Swiestra et al., 1977) at puberty than contemporaries born earlier. The reason for this phenomenon is unclear, and a photoperiod effect has been suggested (Hansen et al., 1983). All these studies were conducted at geographical locations where climatic stress is prevalent during spring calving. Therefore, climatic stress may have a negative impact on early born calves which is manifested in delayed age at puberty. Season of birth can interact with nutrition to alter attainment of puberty. Heifers on high nutrient intake (ADG=.86 kg/d) and born in the spring reached puberty earlier than winter-born heifers on the same diet, while the opposite occurred in heifers on low nutrient intake (ADG=.50 kg/d; Grass et al., 1982). Limited animal numbers were used in this study so it precludes one from making generalizations about seasonal effects on puberty. A subsequent study was conducted to separate seasonal effects during the first 6

mo of life from the second 6 mo of life, utilizing environmental chambers (Schillo et al., 1983). Fall and spring born heifers were exposed to periods characteristic of spring and summer or fall and winter. Heifers exposed to spring and summer conditions, regardless of season of birth, were younger at puberty than those exposed to fall and winter conditions. The authors concluded that season can influence the onset of puberty by events that occur earlier and later during sexual development. This theory is supported by Tortonese and Inskeep (1992) who treated late winter born heifers with melatonin for 5 wk early in their first summer and hastened onset of puberty. They theorized that treatment with melatonin mimicked short days and enhanced the facilitative effects of increased day length during the subsequent spring.

Photoperiod is an important environmental cue that modulates reproductive activity in sheep (Karsch et al., 1984). The pineal gland is capable of transducing changes in photoperiod into a hormonal signal, melatonin, that codes for daylength. Melatonin is secreted in response to darkness and allows animals to discern seasonal differences due to daylength. Consequently, the prepubertal female lamb uses the long days of spring and summer to time the onset of puberty in the short days of fall. It is speculated that this sequence of melatonin secretion provides the signal for a decrease of the estradiol negative feedback on gonadotropin secretion resulting in increased activity of the GnRH pulse generator and attainment of puberty.

Supplemental lighting of spring born heifers during the subsequent winter hastened the onset of puberty (Peters et al., 1978; Hansen et al., 1983; Petitclerc et al., 1983). Whether decreased age at puberty was due to increased growth during supplemental lighting (Peters et al., 1978, 1980; Petitclerc et al., 1983) or strictly a photoperiod effect (Hansen et al., 1983) remains inconclusive. It has been proposed that exposure to short days during early stages of development in the heifer may enhance the stimulatory effects of a subsequent long-day photoperiod on the onset of puberty (Tortonese and Inskeep,

1992). This would be in contrast to the sequence of events that occur in the prepuberal lamb (Foster et al., 1984).

Body Composition Effects on Puberty

Body Fat

The theory that a "critical amount of body fat" is required for attainment of puberty was originally proposed in the rat (Kennedy and Mitra, 1969). This theory was later applied to young girls (Frisch and Revelle, 1970) in which body fat was more constant than weight at menarche in normally growing girls. The correlation between menarche and body weight was improved if body composition was considered in relation to body weight (Frisch and McArthur, 1974) and a minimum amount of body fat was necessary for ovulation and menstrual cycles in the human (Frisch and McArthur, 1974). Furthermore, menstrual abnormalities are common in athletes (Frisch et al., 1981, 1993; Sanborn et al., 1987) and ballet dancers (Frisch et al., 1980) and are usually associated with decreased weight and fat reserves. Rats fed either a high-fat or low-fat diet until first estrus had similar body composition at estrus, although the high-fat rats showed estrus earlier and at a lighter body weight than low-fat rats (Frisch et al., 1977). They concluded that a metabolic signal, related to fat stores, was the signal for puberty in the rat.

The validity of the critical body fat theory has been questioned in many species as body fat is not constant at puberty in nutritionally manipulated rats (Glass, et al., 1979; Bronson, 1987), mice (Hansen et al., 1983), and cattle (Grass et al., 1982; Brooks et al., 1985). Studies in humans also question this theory since reduced body fat is not consistently associated with amenorrhea in runners (Sanborn et al., 1987) and oarswomen (Frisch et al., 1993). Body energy reserves are important in resumption of estrous cycles in humans (Sanborn et al., 1987; Frisch et al., 1993) and rodents (Schneider and Wade,

1989) and after parturition in beef cattle (Dunn and Kaltenbach, 1980; Richards et al., 1986; Selk et al., 1988). The relationship between body fat and the occurrence of menstrual cycles in humans and estrous cycles in many animal species does not always mean cause and effect. Most methods used to estimate body fatness in humans are indirect (e.g., body water measurements, underwater weighing, skinfold measurements) and fail to consider regional fat deposition. Magnetic resonance imaging of overall and regional body fat in well trained female athletes indicated that body weight and body mass index are misleading indicators of body composition (Frisch et al., 1993). The ratio of subcutaneous fat to internal fat was 80:20% among athletes and nonathletes, even though athletes had significantly less total fat. Furthermore, athletes with menstrual disorders had significantly decreased subcutaneous fat, internal fat, and fat at regional sites (e.g., aortic arch, upper thigh, psoas muscle insertion, umbilicus, midiliac crest, femoral heads) compared with non-athletic controls. A subgroup of ovulatory athletes had similar internal fat at selected anatomical sites compared with non-athletic controls, whereas subcutaneous fat was decreased at these sites compared to controls. It has been theorized that body fat distribution rather than body fat mass is related to puberal development in humans (de Ridder et al., 1990).

Evidence to support the regional fat theory may exist in the dairy (e.g., Jersey, Brown Swiss; high milk production) or maternal beef cattle (e.g., Angus, Gelbvieh; moderate milk production). Dairy breeds deposit less subcutaneous fat and more internal fat than do beef breeds (Callow et al., 1961; Charles and Johnson, 1976; Kempster, 1978; Fortin et al., 1981). Dairy breeds have historically been selected for milk production and reach puberty at younger ages than those not selected for milk production (Gregory et ., 1978). Furthermore, carcasses of steers sired by high milk production breeds have greater marbling scores than steers sired by low milk production breeds (Gregory et al., 1978b; Koch et al., 1982). Data from the first three cycles of the Germ Plasm Breeding Program at the U.S. Meat Animal Research Center indicated that breed group means for steers out

of Angus dams had greater marbling scores (Koch et al., 1982) and younger ages at puberty (Gregory et al., 1982) compared with breed group means for steers out of Hereford dams. Consequently, dairy and maternal beef breed heifers may attain puberty at younger ages because they deposit internal body fat more rapidly than beef breeds with less milk production ability. This may allow dairy and maternal beef bred heifers to attain the proper physiological state necessary for puberty at a younger age. MacNeil et al., (1984) determined the genetic correlations between reproductive and maternal traits of females with growth and carcass traits of their steer paternal half-sibs (MacNeil et al., 1984). Breeds used in the analysis included straight bred Hereford and Angus cows mated to either Hereford, Angus, Jersey, South Devon, Limousin, Charolais or Simmental sires. They concluded that selection for increased carcass weight and retail product of steers should result in heifers that are older and heavier at puberty, and females from sires selected for reduced fat trim of steer progeny would be older and heavier at puberty. Therefore, the genetic antagonisms between growth rate and reproductive efficiency stress the importance of balancing growth traits and maternal traits in a selection program.

Fat does not appear to play a direct role in attainment of puberty, but the amount of energy stored in adipose tissue is an important component of total body energy balance (Bronson and Manning, 1991). This theory is supported by work primarily in rodents (Morin, 1986; Schnider and Wade, 1989 and sheep (Hileman et al., 1991; Bucholtz et al., 1992) indicating that ovulation is dependent on availability of oxidizable metabolic fuels - glucose and fatty acids. It has also been theorized that adipose can influence reproductive function through conversion, metabolism, and storage of estrogen (Frisch, 1985). Furthermore, Frisch (1985) speculates that sex hormone-binding globulin can regulate the availability of estrogen to the brain and other target tissues because obese women have decreased ability to bind sex hormone-binding globulin resulting in increased free serum estrogen. Currently, no known hormone, metabolite, or neuroendocrine pathway exist that connects adipose tissue directly with the hypothalamus to influence GnRH secretion.

Due to the complexity of the control of gonadotropins, several factors are probably involved in the regulation of secretion of GnRH.

Summary and Conclusions

Age at puberty is a very complex physiological process that is not determined by weight but by physiological conditions that result in a given weight (Greer et al., 1993). Probably the primary limiting factor influencing onset of puberty is the pulsatile secretion of LH (Kinder et al., 1987). The pituitary (Schams et al., 1981) as well as the ovaries (Casida et al., 1943; Seidel et al., 1971) are functional prior to puberty, but lack of a consistent endogenous release of GnRH from the hypothalamus (Foster et al., 1989: McShane and Keisler, 1991) may be the most limiting component in onset of puberty. Therefore, factors that influence the GnRH pulse generator including interactions between hormones and metabolites need further investigation. The principle factor controlling hormones and metabolites is nutrition.

The net influence of nutrition on reproductive function is through enhanced LH secretion (Day et al., 1984). The influence of nutrition on LH secretion may be either direct and/or indirect and is influenced by metabolic fuel availability (Schneider and Wade, 1989; Hilemann et al., 1991; Bucholtz et al., 1992), excitatory amino acids (Bucholtz et al., 1988; Hall et al., 1990), NPY (McShane et al., 1992) and possibly factors yet to be discovered. The mechanism by which all these factors influence the GnRH pulse generator or possibly the anterior pituitary have yet to be completely elucidated.

Nutrition also contributes significantly to modulation of GH and IGF-I secretion. Growth hormone and IGF-I are important in somatic and bone growth (Tanner et al., 1976; Aynsley-Green et al., 1976; Behringer et al., 1990) with increased evidence supporting the role IGF-I in reproductive function (Echternkamp et al., 1990; Lucy et al., 1992a; Spicer et al., 1993). Nutrient intake enhances IGF-I, therefore, IGF-I may be one

of the important signals by which nutrition communicates and quite possibly enhances cellular activity important in growth and reproduction.

Increased nutrient intake can hasten the onset of puberty resulting in heavier heifers at puberty (Wiltbank et al., 1966; Short and Bellow, 1971: Ferrell, 1982). In turn, gain required to attain puberty can be influenced by breed type (Martin et al., 1992), milk production ability of the breed (Laster et al., 1976, 1979; Gregory et al., 1979; Ferrell, 1982), weight and frame size at weaning (Fox et al., 1988), and possibly mature size (Taylor and Fitzhugh, 1971). Therefore it becomes imperative that heifer biological type be identified so that adequate prepuberal weight gains are made to ensure onset of puberty before the start of the breeding season. Onset of puberty after weaning is probably limited by age in heavyweight heifers and weight in lightweight heifers (Mosely et al., 1982).

The effect of body composition on age at puberty has received very little attention in beef cattle. Body energy reserves are important for normal cycles in humans (Sanborn et al., 1987; Frisch et al., 1993) and rodents (Schneider and Wade, 1989) and for resumption of cycles after parturition in beef cattle (Dunn and Kaltenbach, 1980; Richards et al., 1986; Selk et al., 1988). Body fat is not constant at puberty in nutritionally manipulated rats (Glass, et al., 1979; Bronson, 1987), mice (Hansen et al., 1983), and cattle (Grass et al., 1982; Brooks et al., 1985). Whether body fat influences onset of puberty compared with resumption of estrous cycles is inconclusive. Body composition of beef cattle at puberty has not been directly measured but only estimated. Partitioning of body fat in cattle at puberty has not been studied. Furthermore, the importance of bone and lean growth during the prepuberal period has received little attention. Due to the genetic antagonism that exist between growth rate and reproduction (MacNeil et al., 1984), body composition of animals may have more influence on the onset of puberty than has been determined.

Therefore, the objectives of our experiments were to evaluate the effect of growth rate on percentage body fat, partitioning of body fat, age at puberty, and endocrine function in beef heifers.

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Chapter III

Running Head: HORMONES AND PUBERTY IN BEEF HEIFERS

Effect of Growth Rate on Growth Hormone, Insulin-Like Growth Factor I, Insulin, and Metabolites Prior to Puberty in Beef Heifers[1,2]

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ABSTRACT: The effect of growth rate on age at puberty and concentrations of growth hormone (GH), IGF-I, insulin, glucose and nonesterified fatty acids (NEFA) in plasma prior to puberty was evaluated in 38 Angus x Hereford heifers. At 8 mo of age, heifers

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were allotted by BW and age to three nutritional treatments in each of two years: full fed (n = 13; FF) to gain 1.36 kg/d; limit fed (n = 12; LF) to gain .68 kg/d; maintenance-full fed (n = 13; MFF) to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d. Progesterone, GH, IGF-I, insulin, glucose and NEFA were quantified in weekly plasma samples. Heifers were slaughtered within 10 d after the onset of puberty (based on plasma progesterone) to determine percentage of separable body fat. During year 1, LF heifers (431 d) were older ($P<.05$) than MFF heifers (371 d) at puberty, but age of FF heifers (389 d) was not different from LF and MFF heifers. In year 2, FF heifers (351 d) were younger ($P<.05$) than LF and MFF heifers (398, 434 d; respectively). Heifers on the FF diets were heavier ($P<.05$) at puberty than the LF and MFF heifers (350; 305; 310; kg, respectively). At puberty, FF heifers had greater ($P<.01$) BCS (6.4) and percentage of total separable body fat (29.3%) than the LF (5.6; 19.5%) and MFF (5.4; 18.7%) heifers. During the first 16 wk of treatment FF heifers had decreased ($P<.001$) GH and NEFA concentrations compared with LF and MFF heifers. Increased nutrient intake was associated with increased IGF-I, insulin, and glucose concentrations during the first 16 wk of treatment. Insulin, glucose, and IGF-I were positively ($P<.001$) correlated with BCS while GH and NEFA were negatively correlated ($P<.001$) with BCS during the first 16 wk of treatment. In conclusion, diet significantly altered GH, IGF-I, insulin, and NEFA concentrations in the prepuberal period. Percentage of separable body fat was not constant at puberty and decreased percentage of separable body fat was associated with increased GH and decreased insulin, glucose, and IGF-I concentrations. Body fat per se, is not the sole regulator of puberty but is probably involved in the interaction of several hormones and metabolites with no one factor being the sole regulator.

Key Words: Body Fat, Growth Hormone, Heifers, IGF-I, Nutrition, Puberty

Introduction

The amount of body fat is associated with reproductive function in beef cows (Dzuik and Bellows, 1983; Selk et al., 1988; Randel, 1990). A critical amount of body fat may be necessary for puberty to occur in humans (Frisch et al., 1973; Frisch and McArthur, 1974) and rodents (Kennedy, 1969; Frisch et al., 1975, 1977). Conversely, other studies in rodents (Glass et al., 1979; Bronson, 1987) and beef heifers (Brooks et al., 1985) indicate that puberty may not occur at a constant percentage of body fat.

Concentrations of IGF-I in serum increased prior to puberty in beef heifers (Jones et al., 1991) and decreased IGF-I concentrations were positively associated with delayed puberty in heifers fed low quality hay compared with heifers fed hay plus supplement (Granger et al., 1989). Prepuberal heifers immunized against growth hormone-releasing factor (GRF) had reduced growth hormone and IGF-I concentrations, deposited more body fat, but reached puberty at older ages than nonimmunized heifers that had normal growth hormone concentrations (Simpson et al., 1991). Heifers immunized against GRF also had decreased feed intakes compared with nonimmunized controls; however, it was not determined if a high energy diet fed to GRF immunized heifers would increase IGF-I secretion irrespective of growth hormone concentrations.

Puberty in heifers is associated with decreased negative feedback of estradiol on LH secretion (Kinder et al., 1987). Schillo (1992) suggests that body fat alone does not link nutritional status to LH release, but that metabolic changes resulting from parallel changes in nutrition and body fatness might regulate LH release. Therefore, metabolic changes prior to puberty may be involved, along with decreased estradiol negative feedback, in enhancing LH secretion and subsequent ovulation.

The objectives of this experiment were to characterize the concentrations of hormones (IGF-I, Insulin, growth hormone - GH) and metabolites (glucose, nonesterified

fatty acids - NEFA) in plasma of beef heifers fed to attain different percentages of body fat at puberty.

Materials and Methods

Thirty-eight spring born Angus x Hereford heifers were allotted by BW and age into groups and within a group they were randomly assigned to three treatments in each of two years (yr 1, n=18; yr 2, n=20). Heifers were fed diets (Table 1) to achieve the following growth rates: 1) full fed (diet A) to gain 1.36 kg/d (n = 13; FF); 2) limit fed (diet A) to gain .68 kg/d (n = 12; LF); 3) maintenance fed (diet B) to gain .23 kg/d for 16 wk, then full fed (diet A) to gain 1.36 kg/d (n = 13; MFF). Nonshrunk BW (once per week), shrunk BW (every 4 wk after 16 h withdrawal of feed and water) and body condition score (every 2 wk; BCS: 1 = emaciated; 9 = obese; Wagner et al., 1988) were recorded for all animals. Weekly blood samples (20 mL) were collected via tail venepuncture at 0800 before feeding heifers and oxalic acid (2.5 mg) was added to each blood tube to prevent clotting. Samples were placed on ice and centrifuged (3,000 x g for 20 min) within 4 h. Plasma was decanted and stored at -20°C for subsequent hormone and metabolite analyses. Heifers were slaughtered within 10 d after the onset of puberty to determine percentage of separable body fat (total separable fat/hot carcass wt +KPH + udder + omental fat; see chapter 4 of thesis). This study was approved by the Committee for Animal Studies at Oklahoma State University.

Weekly progesterone concentrations in plasma were quantified by RIA (Bishop and Wettemann, 1993) to determine onset of puberty. Progesterone concentrations greater than 1 ng/mL for two consecutive weeks were the criterion for the onset of puberty. Date of the first of the consecutive samples with progesterone greater than 1 ng/mL was considered age at puberty. Pubertal weight is expressed as a shrunk weight and was determined by multiplying the ADG calculated from the last shrunk weight before

puberty, by the days from the last shrunk weight until puberty, and adding the estimated weight gain to the last shrunk weight before puberty.

Concentrations of glucose, NEFA, insulin, GH and IGF-I were determined in all weekly plasma samples prior to puberty. Glucose was determined by an enzymatic colorimetric procedure (Sigma, No. 510, Sigma Chemical Co., St. Louis, MO). Intra- and interassay CV (n = 12 assays) were 4.0 and 5.1%, respectively. Concentrations of NEFA were determined by an enzymatic colorimetric procedure (Wako-NEFA C, Wako Chemicals Inc., Dallas, TX) with modification as described by McCutcheon and Bauman (1986). Intra- and interassay CV (n = 21 assays) were 4.3 and 13.6%, respectively. Insulin in plasma was quantified by RIA as described by Selk (1986). Intra- and interassay CV (n = 5 assays) were 16.2 and 25.0%, respectively. Growth hormone was quantified by RIA (see Appendix). Intra- and interassay CV (n = 4 assays) were 7.9 and 7.9%, respectively. Concentrations of IGF-I were quantified by RIA (Echternkamp et al., 1990) after an acid ethanol extraction. Intra- and interassay CV (n=8 assays) were 9.7 and 16.9, respectively.

Analysis of variance were used to analyze age, BW, BCS, and percentage of separable body fat at puberty. Analyses of variance of hormones, metabolites, and BCS responses were split-plot designs (Gill and Hafs, 1971). Main effects tested were year, block within year, treatment and treatment x year. Blocks were utilized to assign samples for lab analysis to remove assay variation. Each treatment was represented in a block and samples were randomly assayed within the block. Heifer within treatment x year was the error term for the main plot. Week, week x year , treatment x year and treatment x year x week were the subplot. When treatment x year was significant, years were analyzed separately. If a significant treatment x week or treatment x year x week existed, response curves for hormone and metabolite concentrations were characterized by time trends that were analyzed via regression (SAS, 1988). If the two and three way interaction were not significant, analyses of variance was used. Test of homogeneity of regression were

utilized to determine differences among time trends for hormone and metabolite responses for FF, LF and MFF treatments (Snedecor and Cochran, 1968). During the first 16 wk of treatment, orthogonal contrast between FF and LF vs MFF and FF vs LF were evaluated. During the 10 wk period prior to puberty, orthogonal contrast between FF and MFF vs LF and FF vs MFF were evaluated. Partial correlation coefficients were determined with the Proc Corr procedure of SAS (1988).

Results

Age, BW and percentage of separable body fat at puberty (Table 2) were influenced by rate of gain. There was a treatment x year effect on age at puberty so means are reported by year. In year 1, MFF heifers were younger (371 d; $P<.05$) at puberty compared to LF heifers (431 d) but MFF heifers were similar in age to the FF heifers (389 d). In year 2, the MFF (434 d) and LF (398 d) heifers were older ($P<.05$) at puberty than FF (351 d) heifers. BW at puberty for the FF heifers (350 kg) was greater ($P<.05$) compared to both the LF (305 kg) and MFF (310 kg) heifers. The FF heifers also had greater ($P<.05$) BCS (6.4) and percentage of separable body fat (29.3%) compared to the the LF and MFF heifers (5.6, 5.4; 19.5, 18.7%; respectively).

There was a treatment x year effect on BCS during treatment so means are reported by year and there was a treatment effect ($P<.05$) for both years. During the first 16 wk of treatment, BCS of heifers were best described by a second order polynomial regression equation for year 1 (Figure 1a) and a linear regression equation during year 2 (Figure 1b). Time trends were similar ($P>.10$) for all preplanned orthogonal contrast.

There was a year effect on concentrations of GH during treatment. During the first 16 wk of treatment, concentrations of GH in plasma of heifers during year 1 (Figure 2a) and year 2 (Figure 2b) were best described by a second-order polynomial regression equation. Concentrations of GH were different ($P<.05$) for FF and LF heifers compared

with MFF heifers for both years and FF heifers had reduced (P<.05) GH concentrations compared with LF heifers for both years. Concentrations of IGF-I in plasma were influenced by year. In year 1 (Figure 3a), concentrations of IGF-I were best described by a third-order polynomial regression equation, but were not influenced by treatment. IGF-I concentrations for year 2 were best described by a second order polynomial regression equation (Figure 3b). During year 2, analysis of time trends indicated that FF and LF heifers had greater (P<.01) IGF-I concentrations than MFF heifers, and concentrations of IGF-I were greater (P<.01) than concentrations in LF heifers.

There was a year effect for concentrations of insulin during treatment. During year 1 (Figure 4a), concentrations of insulin in plasma were best described by a linear regression equation and concentrations were not influenced by treatment. A second-order polynomial regression equation best described concentration of insulin during year 2 (Figure 4b) and there was a treatment effect (P<.09). Preplanned othogonal contrast resulted in concentrations of insulin that were similar (P>.10) for all comparisons.

There was a treatment x year effect for concentrations of glucose in plasma during treatment. Concentrations of glucose for year 1 were fit by a second-order polynomial regression equation and concentrations (Figure 5a) were not influenced by treatment. For year 2, a third-order polynomial regression equation best described glucose concentrations (Figure 5b). FF and LF heifers had greater (P<.01) glucose concentrations than MFF heifers and FF heifers had greater (P<.05) concentrations of glucose than LF heifers.

There was a treatment x year effect for concentrations of NEFA in plasma during treatment. Concentrations of NEFA were influenced (P<.001) by treatment for both years and were best described by a linear regression equation for year 1 (Figure 6a) and year 2 (Figure 6b). During both years, FF and LF heifers had reduced (P<.001) concentrations of NEFA compared to MFF heifers, and FF heifers had reduced (P<.001) concentrations of NEFA compared with LF heifers.

Partial correlation coefficients adjusted for week between BCS and hormone and metabolite concentrations (Table 3) were signifacnt for the first 16 wk of treatment. Body condition score was positively correlated with insulin, IGF-I, and glucose concentrations but negatively correlated with GH and NEFA concentrations.

There was a treatment x year effect on concentrations of GH in plasma during the 10 wk prior to puberty. A second-order polynomial regression equation best described the concentrations of GH for the 10 wk prior to puberty in year 1 (Figure 7b). The FF and MFF heifers had reduced ($P<.05$) GH concentrations compared with LF heifers. Concentrations of GH for year 2 (Figure 7b) were best fit by a linear regression equation and were not influenced by treatment. There was a treatment x year effect on IGF-I concentrations during the 10 wk prior to puberty. Concentrations of IGF-I for the 10 wk prior to puberty for year 1 and year 2 were best described by a third-order polynomial regression equation (Figures 8a and 8b; respectively). Analysis of time trends indicated that concentrations of IGF-I were different ($P<.01$) for the FF and MFF heifers compared to the LF heifers for both years. The FF and MFF heifers also had different concentrations of IGF-I during the 10 wk period preceding puberty; FF heifers had greater ($P<.05$) concentrations of IGF-I than MFF heifers during both years.

Concentrations of insulin during the 10 wk prior to puberty were similar for both years and were best described by a third-order polynomial regression equation (Figure 9). The concentrations of insulin for the FF and MFF heifers were different ($P<.05$) compared with the LF heifers. The MFF heifers had reduced ($P<.01$) insulin concentrations compared with the FF heifers. Concentrations of glucose in plasma during the 10 wk prior to puberty were not influenced by wk but there were year and treatment effects. During year 1, treatment did not influence concentrations of glucose in plasma during the 10 wk prior to puberty (Table 4). However, during year 2, FF heifers had greater ($P<.05$) glucose concentrations (92.7 mg/dL) than both the LF (83.1mg/dL) and MFF (81.6 mg/dL) heifers for the 10 wk period prior to puberty (Table 4).

Concentrations of NEFA in plasma during the 10 wk of treatment prior to puberty were not influenced by week or year. Nutritional treatment influenced NEFA concentration for the 10 wk prior to puberty (Table 4). During both years, NEFA concentrations were less for FF heifers (365.9 mEq/mL; P<.01) compared with to NEFA concentrations for the MFF heifers (542.7 mEq/mL). Concentrations of NEFA were also less (P<.09) in LF heifers (485.0 mEq/mL) compared with the MFF heifers (542.7 mEq/mL)

During the 10 wk prior to puberty, treatment had a significant effect on BCS. The BCS of the FF heifers was greater (P<.01) than the BCS of the LF and MFF heifers (Table 5). Only at 1 and 3 wk prior to puberty was the BCS of the MFF heifers different (P<.01) than the BCS of the LF and FF heifers.

Discussion

The greater nutrient intake and increased gain of heifers on the FF treatment resulted in heifers that were heavier at puberty, and younger during year 2 compared with the LF and MFF heifers. This observation agrees with reports in dairy (Sorenson et al., 1959; Menge et al., 1960) and beef heifers (Wiltbank et al., 1966; Arije and Wiltbank, 1971; Hall et al., 1993) that enhanced nutrition can hasten the onset of puberty while undernutrition can delay puberty (Wiltbank et al., 1966; Arjie and Wiltbank, 1971; Day et al., 1986). During year 1, MFF heifers were of similar age at puberty compared to FF heifers. At the initiation of treatment, MFF heifers were given ad libitum access to the low energy diet, expecting to achieve gains of about .23 kg/d. The heifers gained greater than their calculated gain, and at week 4, MFF heifers were switched to a once a day limit feeding program to achieve maintenance gains. The increased gain during the first 16 wk of treatment is reflected in the similar BCS of the MFF and LF heifers during year 1 while the MFF heifers had decreased BCS compared with LF heifers during year 2. The

increased gain during the first 4 wk of treatment may have been enough to decrease the age at puberty in the MFF heifers during year 1.

The increased BCS and percentage of separable body fat for the FF heifers confirms earlier observations that nutrition can alter body composition at puberty in beef heifers (Short and Bellows, 1971; Brooks et al., 1985; Hooper et al., 1993). To be discussed later in this thesis, increased BW of the FF heifers was a result of increased fat deposition as the amount of lean and bone were similar among treatments at puberty. Furthermore, BCS is significantly correlated (r=.75; chapter IV) with percentage of separable body fat, therefore BCS can be utilized as a predictor of total fat in the animal (Wagner et al., 1988). The FF heifers had increased fat deposition during the first 16 wk of treatment compared to both LF and MFF heifers during both years. Body fat may have a role in regulating the onset of puberty; however, age may modulate the effect. If heifers gained rapidly commencing at 8 mo of age, they may not have been old enough to start estrous cycles even though they were fat enough. Although MFF heifers gained at the same rate as FF after 16 wk of minimal gain (.23 kg/d), the MFF heifers had similar BCS and percentage of separable body fat at puberty as LF heifers. These results and others in beef heifers (Brooks et al., 1985) do not support the proposed theory in rats (Kennedy, 1969) and humans (Frisch et al., 1973; Frisch and McArthur, 1974) that a critical amount of body fat is necessary for initiation of puberty.

Concentrations of GH may have acted to mediate the decrease in percentage of separable body fat of the LF and MFF heifers. GH concentrations in plasma were less in FF heifers than in LF and MFF heifers. Concentrations of GH were negatively correlated with BCS during the first 16 wk of treatment (r = -.19). Concentrations of GH are inversely proportional to nutritient intake in ewes (Foster et al., 1989; Kile et al., 1991), steers (Hayden et al., 1993) and heifers (Sejrsen et al., 1983; Villa-Godoy et al., 1990). Increased GH concentrations in heifers immunized against growth hormone releasing factor(GRF) (Simpson et al., 1991) or administered exogenous recombinant DNA-derived

somatotrophin (STH) (McShane et al., 1989) resulted in heifers with decreased backfat thickness. GRF immunized heifers (Simpson et al., 1991) had decreased ADG and delayed puberty while exogenous STH treatment (McShane et al., 1989) caused increased ADG but did not affect the onset of puberty.

The changes in body fatness in the present study could result from a combination of increased lipolysis (Vernon, 1982; Eisemann et al., 1986) and decreased lipogenesis (Vernon, 1982; Peters, 1986) due to decreased substrate availability. Compared with FF heifers, LF and MFF heifers had increased NEFA concentrations throughout the treatment period. Body condition score of the MFF heifers remained low and unchanged during year 2 and was associated with the greatest NEFA concentrations of any of the treatments. Concentrations of NEFA were negatively correlated ($r = -.42$) with BCS during the first 16 k of treatment. Increased concentrations of NEFA are indicative of a negative energy balance (Brier et al., 1986; Peters, 1986; Richards et al., 1989a) and fatty acid release from the adipocytes (Bines and Hart, 1982). Decreased reproductive activity of dairy cows in negative energy was associated with increased NEFA concentrations (Canfield and Butler, 1991). When nutritional anestrus was induced in cows, concentrations of NEFA in plasma increased (Richards et al., 1989a) and LH pulse frequency was decreased (Richards et al., 1989b). Nutritional anestrus was also associated with a decrease in LH pulse frequency in heifers (Imakawa et al., 1986). However, short term infusion of free fatty acids in ovariectomized, positive energy balance ewes suppressed pulsatile GH release but did not alter pulsatile LH secretion (Estienne et al.,1989, 1990).

Concentrations of IGF-I in plasma were associated with increased nutrient intake and gain during the first 16 wk of treatment for year 2 but not for year 1. The similar IGF-I concentrations in all heifers during year 1 may be attributed to the inability to achieve appropriate ADG during the first 4 wk of treatment in the MFF heifers. Concentrations of IGF-I increased while GH concentrations decreased for the FF heifers during the first 16 wk of treatment. Whereas, concentrations of IGF-I decreased and GH

increased in MFF heifers compared with FF heifers. The positive relationship that exists between GH and IGF-I is uncoupled in feed restricted ruminants (Elsassar et al., 1989: Granger et al., 1989; Armstrong et al., 1993). Concentrations of IGF-I tended to increase 4 to 8 wk prior to puberty for heifers on all treatments and then decreased slightly during the 4 wk prior to puberty. Jones et al. (1991) reported increased IGF-I concentrations prior to puberty while Granger et al. (1989) indicated that decreased IGF-I concentrations were positively associated with delayed puberty in heifers fed low quality hay compared to heifers fed hay plus supplement. Circulating concentrations of IGF-I increase prior to puberty in female rodents (Handelsman et al., 1987), primates (Copeland et al., 1982) and humans (Hiney et al., 1991).

The locations where IGF-I may mediate cellular activities to influnce the onset of puberty are enumerable. Ovarian follicular fluid and serum concentrations of IGF-I are positively correlated ($r = .69$; Echternkamp et al., 1990) and IGF-I may have direct local effects on granulosa cell production of estradiol (Spicer et al., 1993) to help stimulate follicular growth and eventual ovulation. In the present study, heifers fed to gain at differing rates to attain puberty at different percentages of separable body fat all had similar bone and lean mass at puberty which will be dicussed in a subsequent chapter of this thesis. Postnatal bone growth is dependent on IGF-I (Ellis et al., 1981; Schoenle et al., 1982) and skeletal muscle growth in rats appeared to be mediated by local production of IGF-I within the muscle (Bates et al., 1993) even during nutritional restriction when GH must prioritize nutrient partitioning and increase its anabolic drive toward skeletal muscle production. Therefore, it is possible that muscle and bone must develop to a certain physiological state before nutrients are partitioned to initiate events necessary for sexual maturity.

Concentrations of insulin in plasma were related to feed intake, similar to other studies involving nutritional manipulation in ruminants (Bassett et al., 1971; Richards et al., 1989a). The FF heifers had greater insulin concentrations than LF and MFF heifers

during treatment. Concentrations of insulin were positively correlated with BCS (r = .42) during the first 16 wk of treatment and are probably responsible for the greater BCS of the FF heifers (Trenkle and Topel, 1978). Concentrations of glucose in plasma were not influenced by treatment in year 1. During year 2, insulin concentrations of FF heifers increased and concentrations of glucose in plasma were also greater than LF and MFF heifers. This suggest a state of insulin resistance and alteration in the glucoregulatory effects of insulin. Infusion of insulin in heifers (Harrison and Randel, 1986) and postpartum cows (Garmendia, 1986) did not influence LH secretion and changes in serum LH concentrations have not been observed in hypoglycemic cows (Rutter and Manns, 1987, 1988). In nutritional anestrus cows (Richards et al.,1989a,b), suggest that reduced nutrient intake reduces glucose in plasma, and the lack of available substrate will not permit adequate secretion of LH to stimulate ovarian function. Schneider and Wade (1989) blocked glycolysis with 2-deoxy-D-glucose (2DG) and had no effect on estrous activity in hamsters, but when fatty acid oxidation was blocked with methyl palmoxirate (MP) concurrent with 2DG administration immediate cessation of estrous cycles occurred. Treatment of ovariectomized lambs concurrently with 2DG and MP completely blocked pulsatile release of LH (Hileman et al., 1991). Castrated , growth retarded lambs also had decreased circulating LH concentrations when 2DG and MP were given together (Bucholtz et al., 1992). These findings indicate that availabilty of metabolic fuels can influence hypothalamic-pituitary-axis function but the exact mechanism of their role in regulating GnRH release require further investigation.

Daily BW gains of heifers commencing at 8 mo of age altered the age, percentage of separable body fat and, concentrations of GH, IGF-I, insulin, NEFA, and glucose at puberty. Increased nutrient intake resulted in decreased GH and NEFA concentrations during the first 16 wk of treatment and GH concentrations during the 10 wk prior to puberty. Increased nutrient intake also resulted in greater IGF-I, insulin, and glucose concentrations during the first 16 wk of treatment which was associated with early

attainment of puberty. Therefore, a proper hormone milieu which can be modulated by nutrient intake must be available for the onset of puberty. Increased nutrient intake decreased age at puberty but resulted in heifers with increased BW and percentage of separable body fat. The increase in body fat was associated with decreased GH and increased insulin concentrations which allowed for increased lipogenesis. Body fat per se, is not the sole regulator of puberty but is probably involved in the interaction of several hormones and metabolites with no one factor being the sole regulator. Age of heifers appears to have a permissive role in the regulation of puberty. Increased nutrient intake early in the treatment period resulted in heifers that were heavy enough but were not old enough for attainment of puberty.

Implications

Feeding programs that enhance adequate growth and development of replacement heifers could greatly increase the biological and economic efficiency of beef production. Alternate growing programs will allow producers to develop replacement heifers of different biological types (maturities) so that they will produce a calf at 24 mo of age and have a productive lifetime. Alterations of nutrient intake of heifers during growth will allow researchers to develop experimental models to investigate physiological factors that regulate the onset of puberty.

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Appendix

Serum or plasma growth hormone (GH) was measured by a heterologous radioimmunoassay using monkey anti-bovine GH serum (AFP-55), bovine GH (NIH-GH-B17) for standards and bovine GH (AFP-7698B) to label with ^{125}I. The second antibody against monkey (rhesus) gamma globulin (Sigma Chemical, St. Louis, MO) was produced in sheep.

Radioiodinated bovine GH was prepared by adding 14 μg of chloramine T in 8 μL sodium phospate buffer (PB, .05 *M*, pH 7.5) to a reaction vial containg 5 μg bovine GH in 20 μL .01 *M* NaH_2OH (pH 10.0), 25 μL buffer (.5 *M* sodium phosphate pH 7.5) and 1.0 mCi ^{125}I in 10 μL of H_2O. The reaction was stopped after 40 sec by adding 31.25 μg of sodium metabisulfite in 25 μL PB. Three hundred microliters of transfer solution (1 % potassium iodide, 16 % sucrose in distilled water) was added to the reaction vial, mixed and the contents were transferred to a Bio-Gel P-60 (Bio-Rad Laboratories, Richmond, CA) column (1 cm X 10 cm) for separation of ^{125}I-GH from free ^{125}I. Prior to transfer, the column had been eluted with 2 mL 2% bovine serum albumin (BSA) in .01 *M* phosphate-buffered saline (PBS; pH 7.0). After transfer of iodinated GH to the column, the column was eluted with PB and 30, 1 mL fractions were collected in tubes containing 1 mL 1% BSA in PBS. Iodinated GH eluted in the first peak (fractions 11 to 16) while free ^{125}I eluted in the later fractions.

Antiserum was diluted 1:200,000 in PBS plus .05 *M* ethylene diamine tetraacetic acid disodium salt (EDTA; pH 7.0) with 1:400 normal monkey serum (Antibodies Incorporated, Davis CA). Diluted antiserum (200 μL) was added to 12 x 75 mm glass tubes containing comtaining either standards in 500 μL PBS plus 1% BSA or unknown (100 μL of serum or plasma, plus 400 μL PBS plus 1% BSA). Tubes were incubated for 24 h at 4° C, then 100 μL of ^{125}I-GH (20,000 cpm in 1% BSA in PBS) was added and tubes were incubated for 24 h at 4° C. Then 200 μL sheep anti monkey gamma globulin

(1:20 dilution in PBS plus EDTA) was added and the tubes were incubated for 72 h at 4° C. Prior to centrifugation (2,500 x g for 30 min) 1 mL PBS (pH 7.0) was added to each tube. After centrifugation, the supernatant was aspirated from the tubes and ^{125}I was quantified in the tubes with a gamma counter.

When 1000 ng of FSH (NIH-FSH-B1), LH (NIH-LH-4) or prolactin (NIH-P-B4) were quantified in the assay, each had < 2ng of GH and 1000 ng thyroid-stimulating hormone (NIH-TSH-B7) was equivalent to 2.5 ng GH. Displacement of ^{125}I-GH by increasing volumes (25, 50, 100, 200, 250 µl) of serum or plasma was parallel to the GH standard (NIH-GH-B17) curve. When 5.0 ng of GH were added to 1 mL of serum or plasma, 95% was recovered. Intra- and interassay CV were 4.5 and 13.9% for serum and 6.6 and 12.1% for plasma, respectively.

Table 1. Composition of diets

Item	Diet A	Diet B
Ingredients, DM%		
Rolled corn	73.00	-
Alfalfa hay	-	70.77
Alfalfa pellets	5.00	-
Cottonseed hulls	5.50	25.42
Soybean meal (44.0% CP)	11.64	-
Dical phosphate	-	.50
Limestone	1.53	-
Vitamin A, 30,000 IU/gm	.02	.02
Molasses	2.97	2.98
Salt	.30	.30
Calculated values		
NEm, Mcal/kg	2.04	1.23
NEg, Mcal/kg	1.31	.61
CP,%	13.7	14.6

Table 2. Effect of rate of gain on BW, age, body condition score (BCS)[a] and percentage of separable body fat (PFAT)[b] at puberty in beef heifers (least squares means)

	Treatment[c]		
Measure	FF	LF	MFF
Heifer, no	13	12	13
Yr 1	7	5	6
Yr 2	6	7	7
BW, kg	350 ± 12.7[e]	305 ± 13.1[f]	310 ±12.7[f]
Age[d], d			
Yr 1	389 ± 16.2[ef]	431 ± 19.2[e]	371 ± 17.6[f]
Yr 2	351 ± 16.7[e]	398 ± 15.5[f]	434 ± 15.5[f]
BCS	6.4 ± .2[e]	5.6 ± .2[f]	5.4 ± .2[f]
PFAT, kg	29.3 ± 1.3[e]	19.5 ± 1.4[f]	18.7 ± 1.3[f]

[a]BCS: 1 = emaciated, 9 = obese.
[b]PFAT = (total separable fat/hot carcass wt + udder + omental fat).
[c]Treatments = FF (fed to gain 1.36 kg/d), LF (fed to gain .68 kg/d), MFF (fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d).
[d]Treatment x year interaction (P<.006).
[e,f]Means within a row lacking a common superscript differ (P<.05).

Table 3. Partial correlation coefficients[a] between body condition score (BCS)[b] and insulin, IGF-I, growth hormone (GH), nonesterified fatty acids (NEFA), and glucose for the first 16 wk of treatment

	Insulin	IGF-I	GH	NEFA	Glucose
BCS	.33[c]	.42[c]	-.19[d]	-.41[c]	.39[c]

[a] Adjusted for week.
[b] BCS: 1 = emaciated, 9 = obese.
[c] P<.0001
[d] P<.002

Table 4. Least squares means (±SE) for nonesterified fatty acids (NEFA; mEq/mL) and glucose (mg/dL) in plasma 10 wk prior to puberty in heifers fed to gain at different rates

	Treatment		
	FF	LF	MFF
NEFA	365.9 ± 48.8^{ac}	485.0 ± 50.1^{abd}	542.7 ± 49.3^{b}
Glucose			
Year 1	83.3 ± 1.8	79.3 ± 2.2	82.5 ± 1.9
Year 2	92.7 ± 2.6^{a}	83.1 ± 2.4^{b}	81.6 ± 2.5^{b}

[a,b] Means within a row lacking a commom superscript differ (P<.01).
[c,d] Means within a row lacking a commom superscript differ (P<.09).

Table 5. Least squares means (MSE = .1) for body condition score[a] for the 9 wk prior to puberty in heifers fed to gain at different rates

	Treatment		
Wk	FF	LF	MFF
1	6.3[b]	5.7[c]	5.5[d]
3	6.1[b]	5.7[c]	5.3[d]
5	5.9[b]	5.4[c]	5.2[c]
7	5.7[b]	5.2[c]	5.0[c]
9	5.6[b]	5.2[c]	5.0[c]

[a]Body Condition score: 1 = emaciated, 9 = obese.
[b,c,d]Means within a row lacking a commom superscript differ (P<.01).

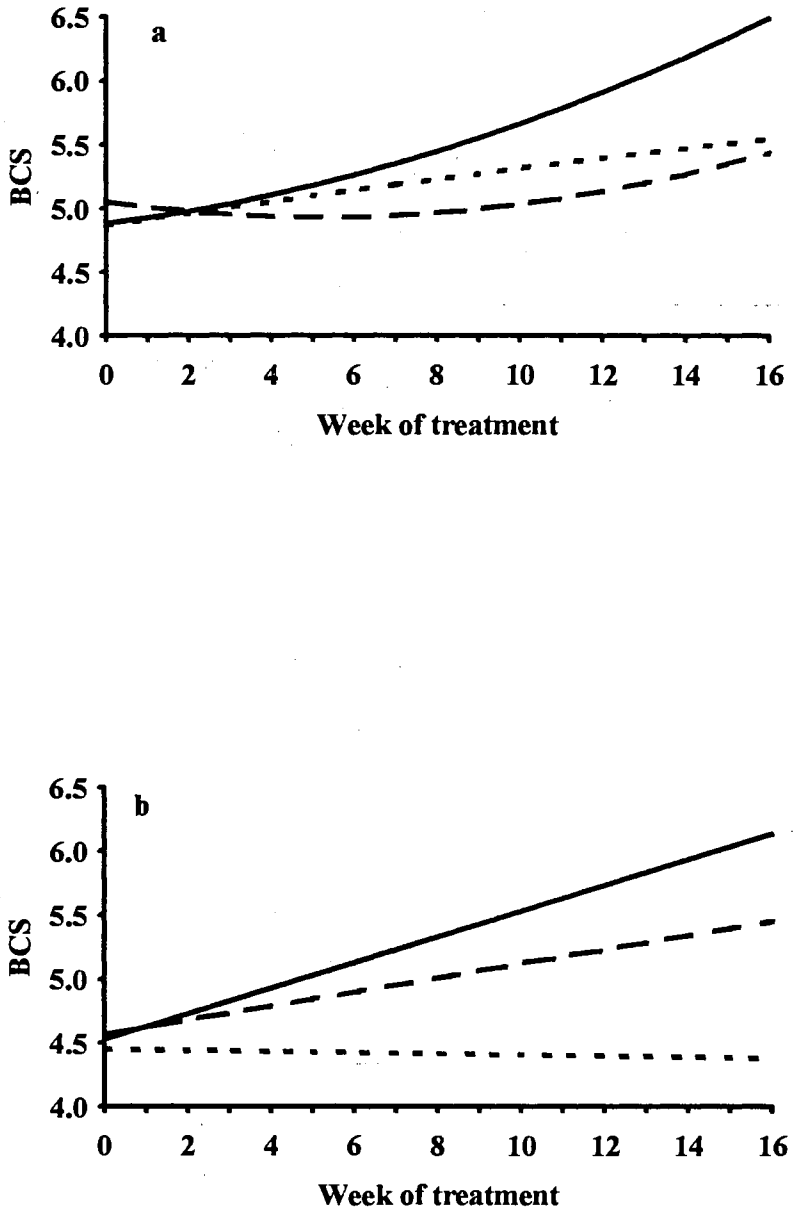

Figure 1. Least squares regressions for body condition score (BCS) for the first 16 wk of treatment for year 1 (a, MSE = .076) and 2 (b, MSE = .069) for FF(———), LF (— —) and MFF (- - - - - -) treatments.

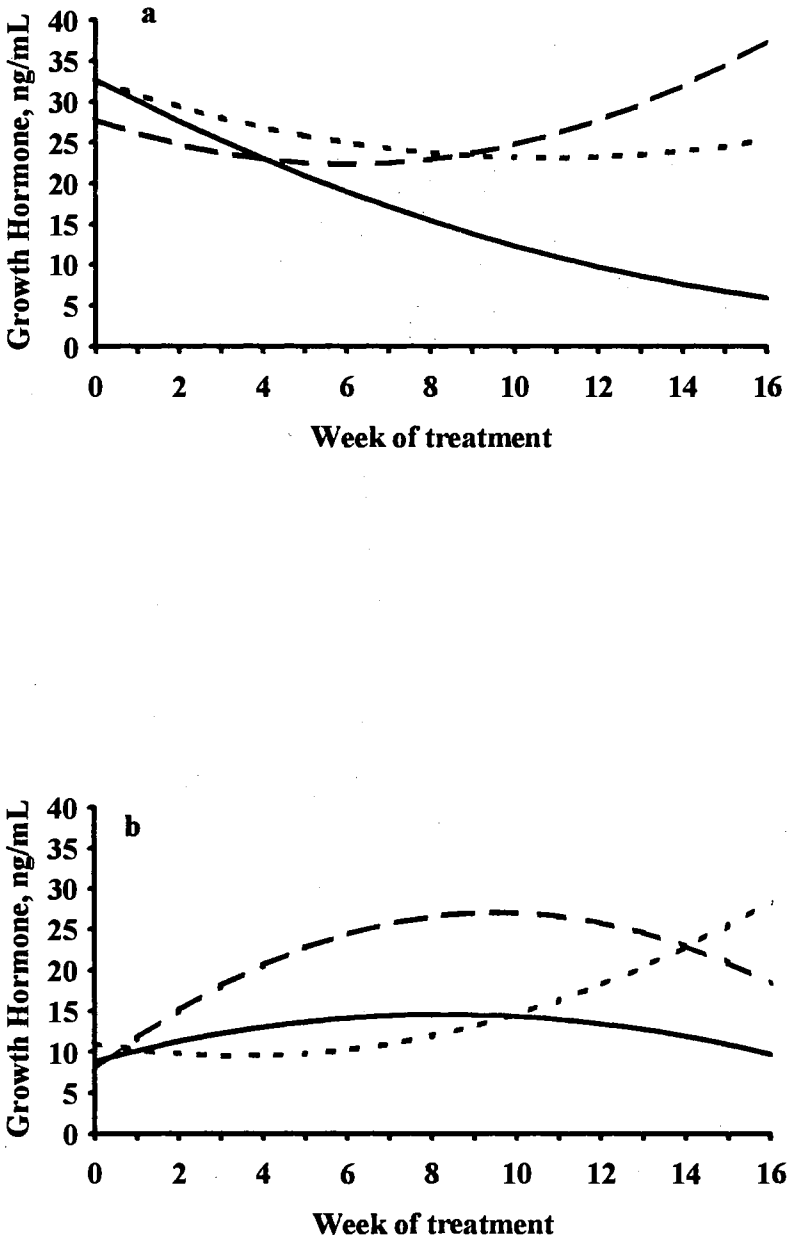

Figure 2. Least squares regressions for concentrations of growth hormone in plasma samples for the first 16 wk of treatment for year 1 (a, MSE = 264) and 2 (b, MSE = 361) for FF(———), LF (— —) and MFF (- - - - - -) treatments.

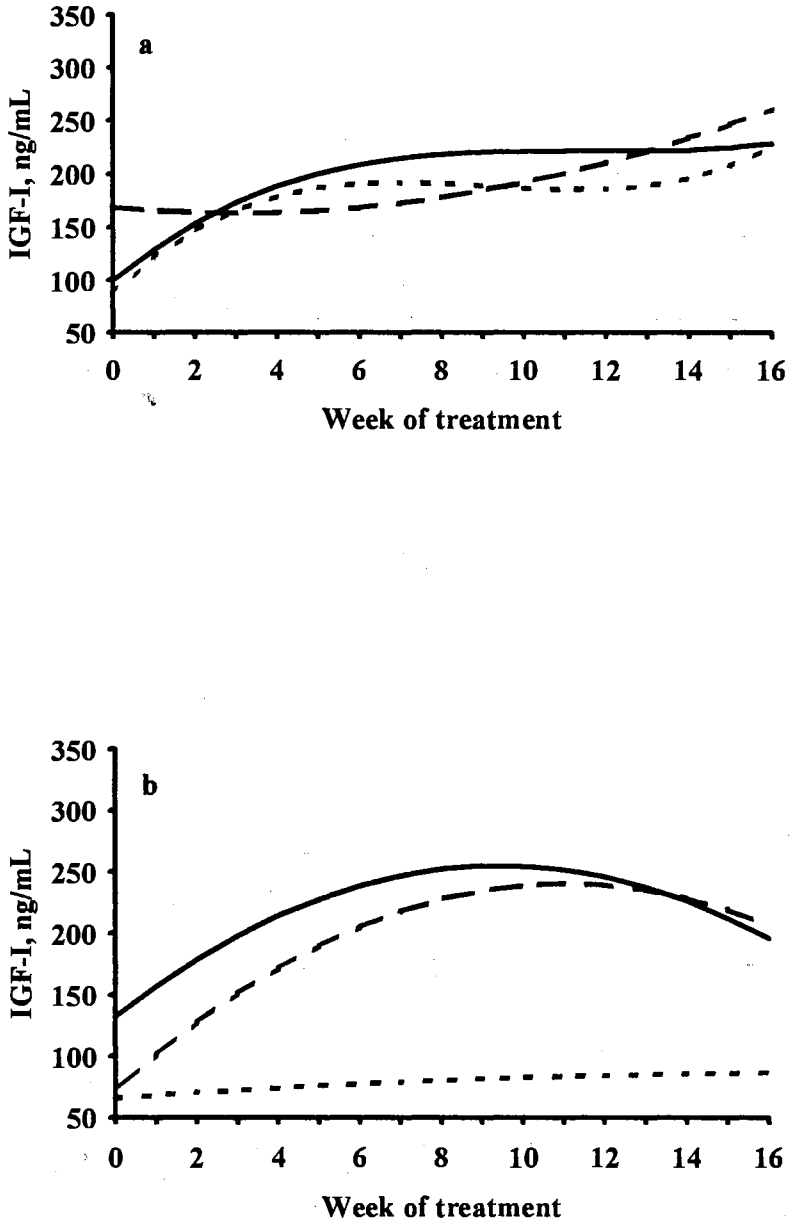

Figure 3. Least squares regressions for concentrations of IGF-I in plasma samples for the first 16 wk of treatment for year 1 (a, MSE = 3,613) and 2 (b, MSE = 1,970) for FF(———), LF (— —) and MFF (- - - - - -) treatments.

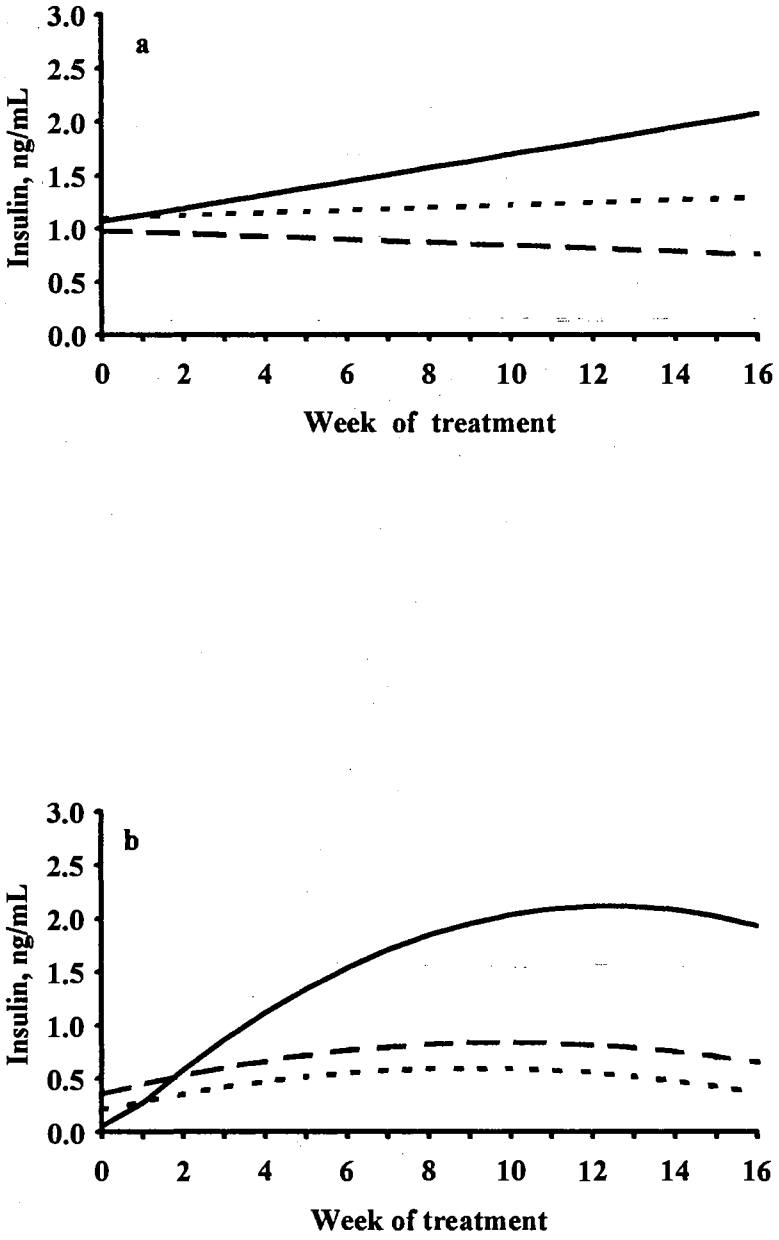

Figure 4. Least squares regressions for concentrations of insulin in plasma samples for the first 16 wk of treatment for year 1 (a, MSE = 1.1) and 2 (b, MSE = .7) for FF(———), LF (— —) and MFF (- - - - - -) treatments.

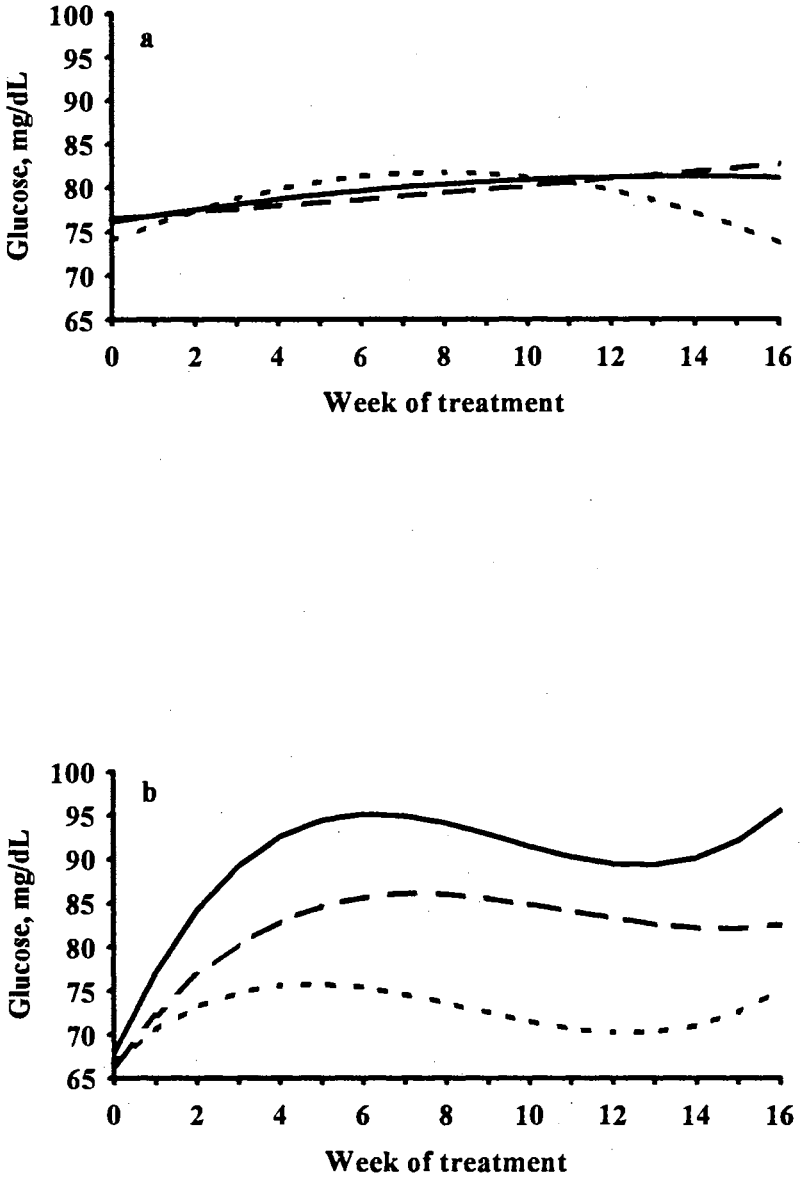

Figure 5. Least squares regressions for concentrations of glucose in plasma samples for the first 16 wk of treatment for year 1 (a, MSE = 42.5) and 2 (b, MSE = 43.3) for FF(———), LF (— —) and MFF (- - - - - -) treatments.

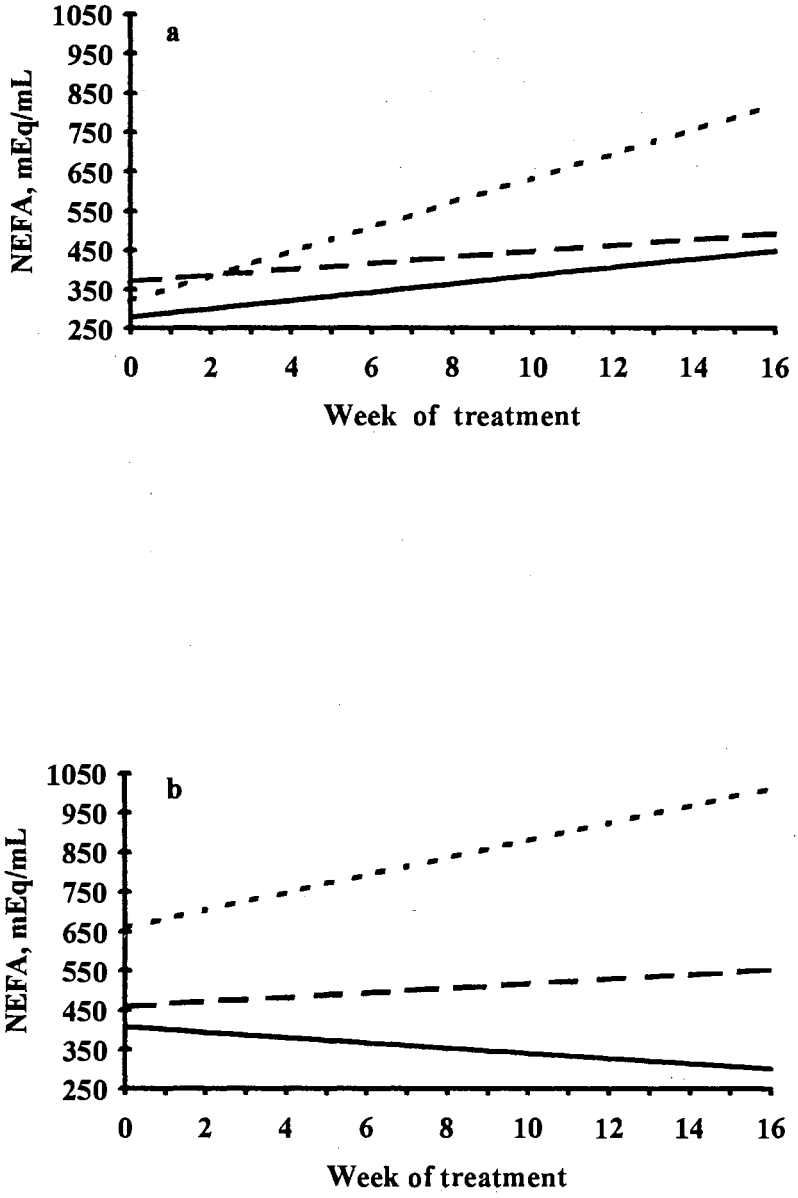

Figure 6. Least squares regressions for concentrations of NEFA in plasma samples for the first 16 wk of treatment for year 1 (a, MSE = 21,652) and 2 (b, MSE = 44,165) for FF(———), LF (— —) and MFF (- - - - - -) treatments.

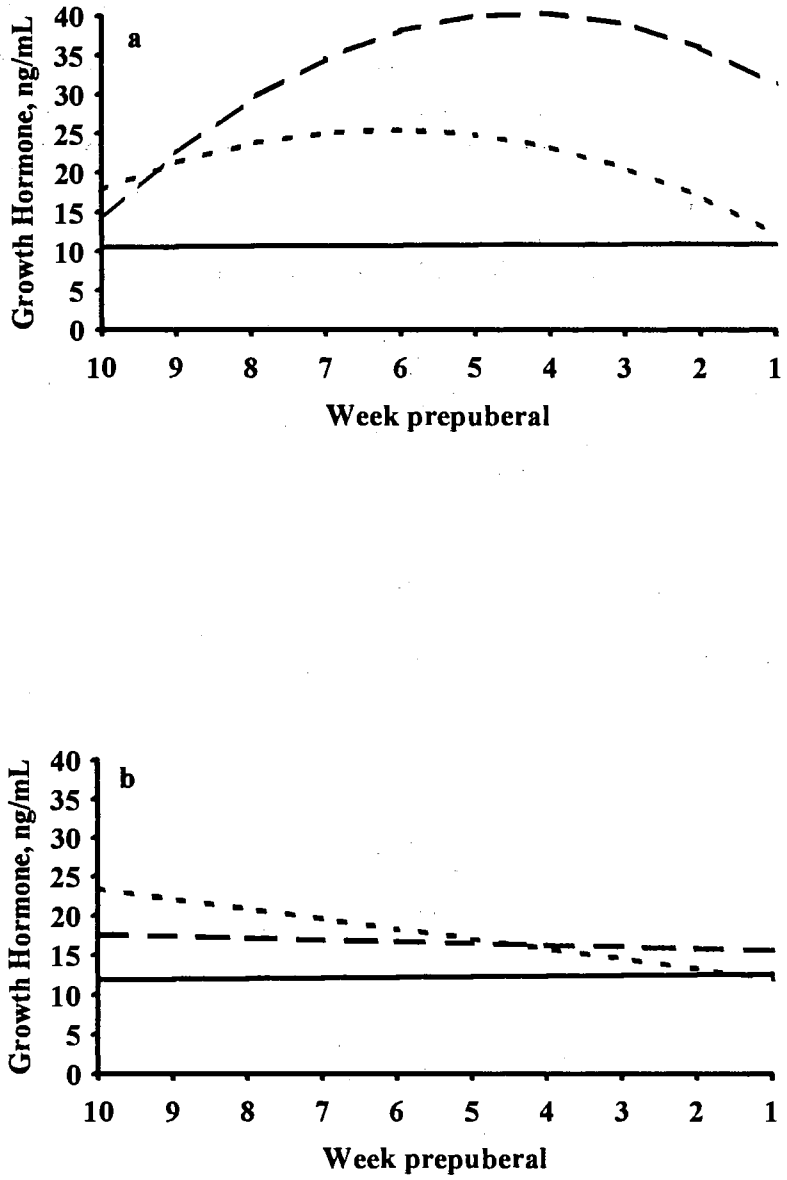

Figure 7. Least squares regressions for concentrations of growth hormone in plasma samples for the 10 wk period prior to puberty for year 1 (a, MSE =156.7) and 2 (b, MSE = 140.5) for FF(———), LF (— —) and MFF (- - - - - -) treatments.

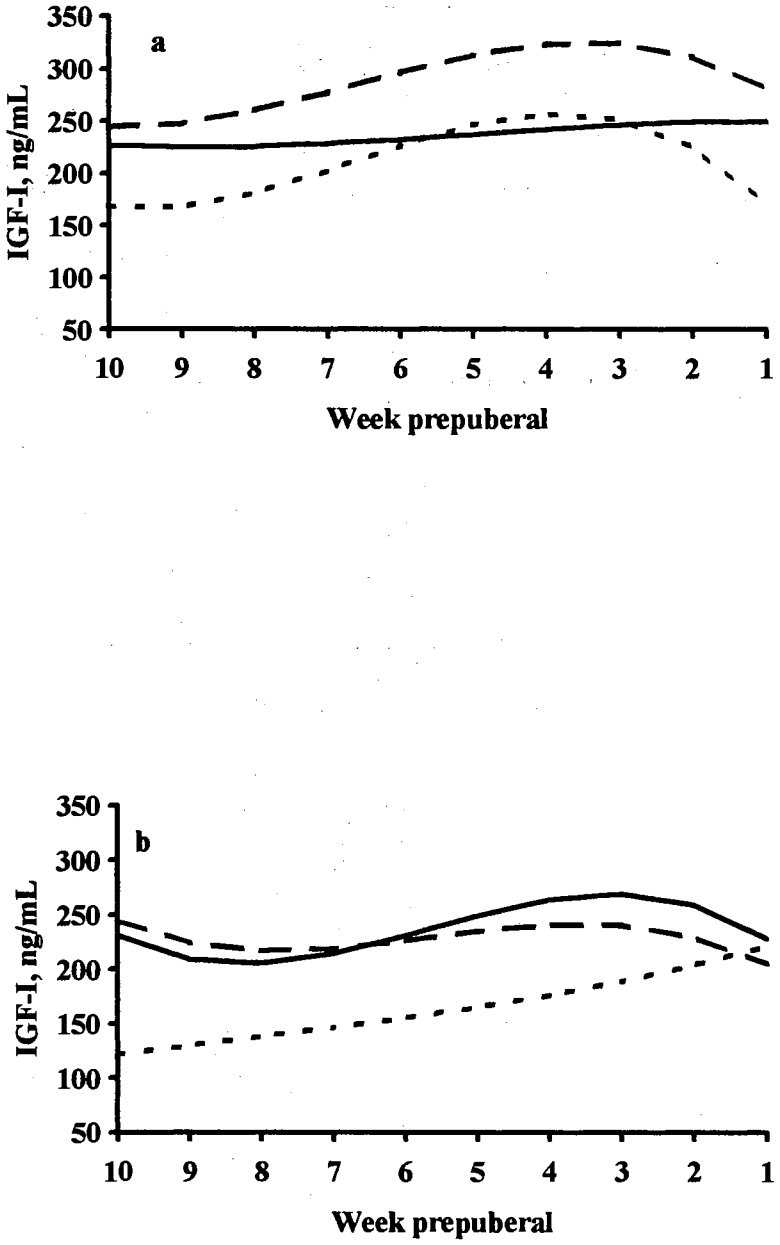

Figure 8. Least squares regressions for concentrations of IGF-I in plasma samples for the 10 wk period prior to puberty for year 1 (a, MSE = 4,584) and 2 (b, MSE = 3,471) for FF(———), LF (— —) and MFF (- - - - - -) treatments.

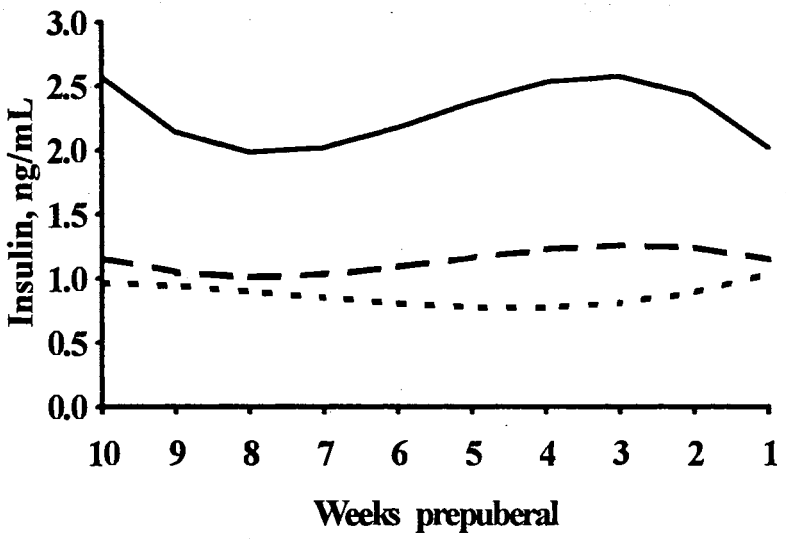

Figure 9. Least squares regressions for concentrations of insulin in plasma samples for the 10 wk period prior to puberty for both years (MSE = 1.1) for FF(———), LF (— —) and MFF (- - - - - -) treatments.

Chapter IV

Running Head: CARCASS COMPOSITION AND PUBERTY IN BEEF HEIFERS

Effect of Growth Rate on Carcass Composition and Lipid Partitioning at Puberty in Beef Heifers[1,2]

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ABSTRACT: The effect of growth rate of heifers on carcass composition, lipid partitioning, age, and BW at puberty was evaluated in 38 Angus x Hereford heifers. At 8 mo of age, heifers were allotted by BW and age to three nutritional treatments with a replication in each of two years: full fed (n = 13; FF) to gain 1.36 kg/d; limit fed (n = 12; LF) to gain .68 kg/d; maintenance-full fed (n = 13; MFF) to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d. Heifers were slaughtered within 10 d after the onset of puberty (based on plasma progesterone). At slaughter, kidney-pelvic-heart (KPH) and UDDER were separated from the carcass and fat surrounding viscera was physically separated and

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[3]To whom reprint request should be sent.

designated as omental-mesenteric fat (OM). After 48 h at 4°C, one random side of the carcass was physically dissected into subcutaneous fat (SC), intermuscular fat (SEAM), soft tissue (SFT - inseparable lean and fat), LEAN, and BONE to determine carcass composition. During year 1, LF heifers (431 d) were older (P<.05) than MFF heifers (371 d) at puberty, but age of FF heifers (389 d) was not different from LF and MFF heifers. In year 2, FF heifers (351 d) were younger (P<.05) than LF and MFF heifers (398, 434 d; respectively). Year did not influence any other traits at puberty. Heifers on the FF diets were heavier (P<.05) at puberty than the LF and MFF heifers (350, 305, 310, kg, respectively) and FF heifers had greater (P<.01) BCS (6.4) and percentage of total separable carcass + OM (29.3%) than the LF (5.6; 19.5%) and MFF (5.4; 18.7%) heifers. Carcass weights were greater (P<.001) for FF compared with LF and MFF heifers due to increased fat as lean and bone weights were not affected by treatment. FF heifers had greater (P<.05) extracted lipid in OM, SC, KPH, SEAM and LEAN compared with LF and MFF heifers which were not different from each other. Treatment affected lipid partitioning as a percentage of carcass + OM lipid for OM, KPH and LEAN depots. Total lipid as a percentage of carcass + OM weight was greater (P<.05) for FF heifers compared with LF and MFF heifers, while total moisture and protein as a percentage of carcass tissue + OM weight were less (P<.05) in FF heifers compared with LF and MFF heifers. In conclusion, diet significantly altered age, BW, carcass composition, and lipid partitioning at puberty in beef heifers. Percentage carcass + OM fat is not the sole regulator of puberty in the beef heifer.

Key Words: Body Fat, Carcass Composition, Heifers, Lipid, Nutrition, Puberty

Introduction

Beef production systems have become more intensive over the last few decades with more producers breeding replacement heifers as yearlings to calve at two years of age

(Short et al., 1994). Consequently, heifers must reach puberty at 12 to 14 mo of age and preferably before the start of the breeding season to avoid decreased fertility associated with the pubertal estrus (Byerly et al., 1987). Therefore, feeding programs that enhance adequate growth and development of replacement heifers could greatly increase the percentage of heifers with normal estrous cycles at the start of the breeding season.

Reproductive performance of beef cows varies with body energy reserves (Dunn and Kaltenbach, 1980; Richards et al., 1986; Selk et al., 1988) and body condition score can be utilized to estimate body energy reserves (Wagner et al., 1988). A critical amount of body fat may be required for attainment of puberty in rodents (Kennedy, 1969; Frisch et al. 1975, 1977) and humans (Frisch et al., 1973;Frisch and McArthur, 1974) although other studies in rodents (Glass et al., 1979; Bronson, 1987) and beef heifers (Brooks et al., 1985; Hooper et al., 1993) indicate that puberty may not occur at a constant percentage of body fat. Body fat distribution rather than body fat mass may be related to puberty in humans (de Ridder et al., 1990), however, it has not been determined if fat distribution amoung carcass and visceral tissues is related to puberty in beef cattle. Rate of gain can influence carcass composition of cattle. Steers fed to achieve greater daily gains had increased fat in their carcasses (Waldman et al., 1971). Alterations in growth rate by feed intake may influence carcass composition which, in turn, could affect age and/or BW at puberty. The objectives of this experiment were to evaluate the effect of growth rate of heifers on carcass composition, lipid partitioning, age, and weight at puberty.

Materials and Methods

Angus x Hereford heifers were allotted by BW and within a group they were randomly assigned to three treatments with a replication in each of two years (yr 1, n=18; yr 2, n=20). Heifers were fed diets (Table 1) to achieve the following growth rates: 1) full fed (diet A) to gain 1.36 kg/d (n = 13; FF); 2) limit fed (diet A) to gain .68 kg/d (n =

12; LF); 3) maintenance fed (diet B) to gain .23 kg/d for 16 wk, then full fed (diet A) to gain 1.36 kg/d (n = 13; MFF). Blood samples (20 mL) were collected weekly via tail venepuncture and oxalic acid (2.5 mg) was added to each blood tube to prevent clotting. Samples were placed on ice and centrifuged within 4 h. Plasma was decanted and stored at -20°C for subsequent progesterone analysis (Bishop and Wettemann, 1993). Nonshrunk BW (once per wk), shrunk BW (every 4 wk after 16 h withdrawal of feed and water) and body condition score (every 2 wk; BCS: 1 = emaciated; 9 = obese; Wagner et al., 1988) were recorded for all animals. Progesterone concentrations greater than 1 ng/ml for two consecutive weeks was the criterion for the onset of puberty. Date of the first of the consecutive samples with progesterone greater than 1 ng/ml was considered the day of puberty.

Heifers were slaughtered at the Oklahoma State University Meat Laboratory within 10 d after the second progesterone greater than 1 ng/ml. After evisceration, omental-mesenteric fat (OM) was physically separated from the gastrointestinal tract, weighed and stored at -20°C. Kidney-pelvic-heart fat (KPH) and the udder (UDDER) were physically separated from the carcass, weighed and stored at -20°C. Hot carcass weight was then taken. Approximately 24 h postmortem, marbling score, lean and skeletal maturities, longissimus area and fat thickness were recorded for USDA yield and quality grades (USDA, 1987). After a 48 to 72 h chill at 4°C, a random side of each carcass was selected and physically separated into subcutaneous fat (SC), intermuscular fat (SEAM), lean (LEAN), soft tissue (SFT; inseparable fat and lean), and bone (BONE). Each depot including OM and KPH but excluding bone was individually weighed, thoroughly mixed, ground through a coarse (1.25 cm) plate, remixed and reground through the coarse plate for three 50 g subsamples, obtained at random and composited. All samples were stored at - 20°C for subsequent chemical analyses. Chemical analyses was not performed on bone. Samples were pulverized in liquid nitrogen and duplicate samples were analyzed for crude protein by the Kjedahl procedure and percent protein was calculated as Kjeldahl

nitrogen x 6.25 (AOAC, 1984). Crude fat was determined by extracting with petroleum ether for 8 h (AOAC, 1984). Moisture was determined by weight loss after drying at 100°C for 24 h, and mineral content by ashing at 600°C for 8 h (AOAC, 1984).

Age at puberty was the age at the date of the first of two consecutive progesterone samples > than 1 ng/ml. Pubertal weight is expressed as a shrunk weight and was determined by multiplying the ADG during the last shrunk weight period closest to puberty, by the additional days from that shrunk weight until puberty, and adding the weight gained to the last shrunk weight. Carcass composition was calculated using depot weights adjusted for moisture loss that occurred between slaughter and physical dissection. (see Appendix Table 2). The amounts of lipid, moisture, protein and ash for each respective depot were determined by using depot weights, adjusted for moisture loss, and values from proximate analysis (see Appendix Table 3). Separable fat and lipid for respective depots were expressed as a percentage of separable fat and lipid in the carcass + OM; they were also expressed as a percentage of carcass + OM weight (see Appendix Table 2). Carcass + OM weight was defined as hot carcass weight plus OM, KPH, and UDDER. This allowed for an examination of partitioning of separable fat or lipid in each depot in relation to both fatness and carcass + OM weight. Analyses of variance were conducted using the Proc GLM procedure of SAS (1988). Treatment and year were the independent sources of variation in the model and if the treatment x year interaction was significant data were analyzed by year. This study was approved by the Committee for Animal Studies at Oklahoma State University.

Results

Age , BW and BCS at puberty (Table 2) were influenced by rate of gain. BW and BCS at puberty for the FF heifers (350 kg; 6.4) were greater ($P<.05$) compared with both the LF (305 kg; 5.6) and MFF (310 kg; 5.4) heifers. Treatment x year was significant for

age at puberty so means are reported by year. In year 1, MFF heifers were younger (371 d; P<.05) at puberty compared with LF heifers (431 d) but MFF heifers were similar in age to the FF heifers (389 d). In year 2, the MFF (434 d) and LF (398 d) heifers were older (P<.05) at puberty than FF (351 d) heifers.

Carcass measurements at puberty were affected by nutritional treatment (Table 3). The FF heifers had greater (P<.001) carcass weight, percentage KPH, and fat thickness than both the LF and MFF heifers. LF and MFF heifers had similar carcass weight, percentage KPH, and fat thickness. Maturity score, marbling score, longissimus area, and yield grade were not affected by treatment. Lean and bone weights were not influenced by treatment (Table 4) but separable carcass fat and separable carcass + OM fat (TFAT; carcass fat and OM, Table 5) were influenced by treatment. There was a treatment x year affect on carcass fat and TFAT. The interaction was due to a difference in magnitude, not the order of magnitude of the treatment effects each year. During both years 1 and 2, FF heifers had greater (P<.05) carcass fat and TFAT than LF and MFF heifers. FF and MFF heifers had similar carcass fat and TFAT. Each depot of TFAT was altered by nutritional treatment (Table 5). FF heifers had greater (P<.001) amounts of fat in OM, KPH, SFT and UDDER depots compared with the LF and MFF heifers while the LF and MFF heifers had similar OM, KPH, SFT and UDDER. There was a tendency for treatment x year effects for OM (P<.09), KPH (P<.10), and SC (P<.07) that were associated with differences in the magnitude of the responses. There was a treatment x year effect on SEAM (Table 5). During year 1, FF heifers had greater (P<.05) SEAM than both LF and MFF heifers and SEAM was similar for LF and MFF heifers. During year 2, FF heifers had greater (P<.05) SEAM compared with LF heifers but not MFF heifers,and LF and MFF heifers had similar amounts SEAM. The simple correlation coefficients were high (P<.0001) between BCS and OM (.76), KPH (.74), UDDER (.72), SQ (.76), SEAM (.75), and BFAT (.77).

The proportion of separable carcass + OM fat expressed as a percentage of carcass + OM weight was affected by treatment (Table 6). The percentage separable carcass + OM fat was greater (P<.0001) for the FF (29.3%) compared with LF (19.5%) and MFF (18.7%) heifers which were similar to each other. When separable fat pools were expressed as a percentage of separable carcass + OM fat (Table 6), there was no treatment effect on the percentages of SC and UDDER, however there was a treatment effect on KPH. FF and MFF heifers had a lesser (P<.05) proportions of separable carcass + OM fat as KPH compared with LF heifers. There was a treatment x year effect on the percentage OM (P<.05) and SEAM (P<.04). During year 1, treatment did not significantly effect partitioning of SEAM and OM. During year 2, FF heifers had a greater percentage (P<.05) of fat as OM than LF heifers, but were not different than MFF heifers, while FF and MFF heifers had similar OM fat partitioning. During year 2, FF heifers had a decreased (P<.05) percentage of fat as SEAM compared with both LF and MFF heifers. The LF and MFF heifers had similar percentages of SEAM. Simple correlation coefficient between BCS and percentage separable carcass + OM fat was .75 (P<.001).

Treatment influenced the proportion of separable carcass + OM fat depots expressed as percentages of carcass + OM weight (Table 7). The FF heifers had a greater percentage of OM, KPH, SC, and UDDER fat compared with the LF and MFF heifers, which were similar to each other. There was a treatment x year effect on PSEAM. During year 1, FF heifers had a greater (P<.05) proportion of fat as SEAM compared with LF and MFF heifers. LF and MFF heifers were not different from each other. During year 2, FF heifers had a greater (P<.05) proportion of fat as SEAM compared with LF but were not different from MFF heifers. LF and MFF heifers had similar PSEAM.

Nutritional treatment influenced both carcass + OM lipid (TLIPID) and weight of lipid in a depot (Table 8). There was a treatment x year effect on TLIPID, total SEAM lipid (SEAML), total OM lipid (OML), total SC lipid (SCL), and total intramuscular lipid (LEANL). During both years, the FF heifers had greater (P<.05) TLIPID, SEAML, and

SCL compared with LF and MFF heifers while LFF and MFF heifers had similar TLIPID, SEAML and SCL. During year 1, FF heifers had greater (P<.05) OML than both LF and MFF heifers, while LF had greater (P<.05) OML than MFF heifers. During year 2, the FF heifers had greater (P<.05) OML compared with LF and MFF heifers, which had similar OML. The FF heifers had greater LEANL than both LF and MFF heifers during year 1. There was no treatment effect on LEANL during year 2. There was a treatment effect on body KPH lipid (KPHL). The FF heifers had greater KPHL (P<.05) compared with LF and MFF heifers.

Lipid pools expressed as a percentage of carcass + OM lipid were also influenced by treatment (Table 9). The FF heifers had a greater (P<.001) percentage of OML, KPHL, and LEANL compared with LF and MFF, which were similar to each other. There was a treatment x year effect on the proprtion of SEAML. During year 1, the FF heifers had a greater (P<.05) percentage of total lipid as SEAML than LF and MFF heifers, which were similar to each other. Treatment had no effect on SEAML as a percentage of carcass + OM lipid during year 2. The partitioning of SCL was not influenced by treatment.

Lipid pools, as a percentage of carcass + OM weight, were influenced by treatment (Table 10; mammary lipid has not been analyzed and is not included in carcass + OM weight). The FF heifers had a greater percentage of (P<.01) OML, KPHL, and SCL of carcass + OM weight than both LF and MFF, which were similar to each other. There was a tendency toward a treatment x year effect on SEAML (P<.09) and LEANL (P<.07). During both years, FF heifers had greater (P<.05) SEAML as a percentage of carcass + OM weight compared with LF and MFF heifers which were not different from each other. During year 1, the FF heifers had greater (P<.05) LEANL as a percentage of carcass + OM weight compared with LF and MFF heifers which were not different from each other, while there were no treatment differences during year 2.

Chemical composition based on a carcass tissue + OM weight basis was influenced by nutritional treatment (Table 11). The FF heifers had a greater (P<.0001) percentage carcass tissue + OM (PLIPID; 34.0%) but a decreased (P<.0001) percentage carcass tissue + OM moisture (PMST; 50.5%) compared with both LF (23.6, 58.8%; respectively) and MFF (22.1, 59.8%; respectively) heifers. LF and MFF heifers had similar PLIPID and PMST. There was no treatment effect on the percentage carcass tissue + OM ASH (PASH) or fat free lean weight (FFL). The percentage of FFL (PFFL) was decreased in the FF heifers (51.4%) compared with the LF (59.0%) and MFF (60.2%) heifers, which were similar to each other. The simple correlation coefficient between PLIPID and BCS was .77 (P<.001).

Discussion

The increased rate of gain due to increased nutrient intake resulted in FF heifers that were younger, heavier, and had greater BCS at puberty compared with LF and MFF heifers. These results confirm previous observations that increased rates of gain results in younger ages but heavier weights at puberty (Arije and Wiltbank, 1971; Short & Bellows, 1971; Ferrell, 1982) while undernutrition can delay puberty (Wiltbank et al., 1966, 1969; Arije and Wiltbank, 1971; Day et al., 1986). The treatment x year effect for age at puberty suggests how vulnerable the prepubertal animal is to nutritional manipulation. During year 1, MFF heifers were of similar age at puberty compared to FF heifers. At the initiation of treatment, MFF heifers were fed the low energy diet ad lib, expecting to achieve maintenance gains. The heifers gained above their calculated gain, and at week 4, MFF heifers were switched to a once a day limit feeding program to achieve gains of .23 kg/d. The increased gain during the first 4 wk of treatment may have been sufficient to decrease the age at puberty in the MFF heifers during year 1.

The increased BCS, carcass weight, fat thickness, and percentage of separable carcass + OM fat for the FF heifers confirms earlier observations that nutrition may alter body composition at puberty in beef heifers (Short and Bellows, 1971; Brooks et al., 1985; Hooper et al., 1993). Increased BCS, BW and carcass weight of the FF heifers was a result of increased fat deposition as absolute amounts of lean and bone were similar among heifers on the three treatments at puberty. Although the correlation between BCS and percentage carcass + OM lipid was less (r=.77) than that reported by Wagner et al. (1988) in cows (r=.91), BCS can be used to estimate total carcass fat in heifers. Wagner et al. (1988) determined total body lipid whereas the present study determined carcass tissue + OM lipid, also differences in BCS distribution between the two studies, 2 to 8 for Wagner et al. (1988) compared with 4 to 7 in the present study, are probably the primary reasons for correlation coefficient differences. Absolute chemical lipid can also be used as an indicator of body energy reserves. FF heifers had increased proportions of chemical lipid as a percentage of carcass + OM weight compared with LF and MFF heifers. This is in agreement with studies with steers (Williams et al., 1983; Keane et el., 1991) and heifers (Hooper et al., 1993) fed diets to achieve differing rates of gain.

Therefore, FF heifers did not initiate estrous cylces when they attained a similar carcass + OM fat or body energy reserve percentage as the LF and MFF heifers had at puberty, possibly because the FF heifers were too young. These results and others in beef heifers (Brooks et al., 1985) do not support the proposed theory in rats (Kennedy, 1969) and humans (Frisch et al., 1973; Frisch and McArthur, 1974) that a critical amount of body fat is necessary for initiation of puberty. Recent evidence in humans, (de Ridder et al., 1990) suggest that body fat distribution rather than body fat mass may be related to puberty. Body fat distribution may have a role in regulating the onset of puberty, however, age may modulate that effect. Decreased body fat of the LF and MFF compared to FF heifers is probably due to treatments effects on growth hormone secretion. The role

of growth hormone secretion in relation to body composition will be discussed in the next chapter of this thesis. Results of the present study indicate that BCS can be effectively used to estimate the amont of fat in different carcass depots of cattle of similar breed composition because of the high high correlation ($r > .72$) between BCS and separable fat depots.

The role of lean and bone in initiation of puberty has received little attention. In the present study, heifers attained puberty at different percentages of separable carcass + OM fat but bone and lean mass were similar for heifers on the three treatments. This suggest that the onset of puberty may be more closely associated with lean and bone mass of the animal than fat. In heifers fed to gain .5 kg/d or 1.0 kg/d, Hooper et al. (1993) found that treatment did not effect separable lean weight at puberty. Lean weight in that study was estimated from prediction equations (Hankins and Howe, 1946) developed for 9-10-11 rib sections. Accuracy of this method is questioned as there is considerable room for error in dissection techniques not accounted for in the equation (Berg & Butterfield, 1976). Postnatal bone growth is dependent on IGF-I (Ellis et al., 1981; Schoenle et al., 1982), and skeletal muscle growth in rats appeared to be mediated by local production of IGF-I within the muscle (Bates et al., 1993) even during nutritional restriction when GH must prioritize nutrient partitioning and increase its anabolic drive toward skeletal muscle production. Studies with rodents (Handelsman et al., 1987), primates (Copeland et al., 1982), humans (Hiney et al., 1991) and beef heifers (Granger et al., 1989; Jones et al., 1991) indicated that IGF-I increased prior to puberty. Therefore, it is possible that muscle and bone must develop to a certain physiological state before nutrients are partitioned to initiate events necessary for sexual maturity. The role of IGF-I secretion in relation to body composition were discussed in the previous chapter of this thesis.

The increased gain of the FF heifers resulted in heavier udder weights compared to udders of the LF and MFF heifers. Much of this increase could be attributed to increases

in fat deposition in the udder of the FF heifers. Studies with dairy heifers (Gardner et al., 1977: Sejrsen, 1978) suggest that high levels of concentrate feeding can reduce subsequent milk production by increased deposition of udder fat and this would probably apply to FF heifers in the present study. Feed intake after puberty of dairy heifers did not influence milk production (Lacasse et al., 1993). In a study conducted by Marston (1993), a group of heifers were fed to gain in a similar fashion as MFF heifers in the present study and his results suggest short term feeding of a high concentrate diet prior to breeding did not affect subsequent milk producing ability.

Carcass composition comparisons between the present study with others is difficult because of different slaughter endpoints. Most carcass composition studies use an age, weight, or time on feed constant as a slaughter endpoint. Puberty was the endpoint in the present study; consequently, carcass data is confounded with both age and weight. Therefore, this must be taken into consideration when making comparisons between the present study and those in the literature. Puberty is a dynamic process controlled by several factors. Using puberty as an endpoint allows a better understanding of the physiological important events that preceed puberty which could be masked by feeding animals to a constant age or weight basis, and then trying to interpolate data as to its relation to puberty.

Absolute fat or lipid partitioning among depots varies within a heifer and within each treatment. When numerically ranked, SEAM is the largest depot followed by OM, SC, and KPH irrespective of treatment. These results agree with Wright & Russel (1984) in mature non-lactating cows with a BCS of 5 to 6. When carcass fat, minus OM, is taken into consideration, fat partitioning agrees with steers (Dolezal et al., 1993) where SEAM > SC > KPH. Dairy breeds deposit less SC and more internal fat than do beef breeds (Callow, 1961; Charles & Johnson, 1976; Kempster, 1978; Fortin et al., 1981).

Increased rate of gain resulted in increased fat deposition in all depots of the FF heifers compared with LF and MFF heifers. This was true for absolute fat amounts, separable fat as a percentage of carcass + OM weight, absolute lipid, and lipid as a percentage of carcass + OM weight. Therefore, increased nutrient intake results in a greater amount of fat deposited in the body. This in agreement with studies with steers (Waldman et al., 1971; Murray et al., 1974; Kempster et al., 1976) and non-lactating cows (Keane et al., 1991). Growth rate has little effect on the development of any particular depot as a proportion of total fat (Murray et al., 1974; Berg and Butterfield, 1976; Kempster et al., 1976). In the present study, rate of gain altered partitioning of separable fat and lipid. The FF and MFF heifers had decreased percentages of separable and lipid KPH compared with LF heifers. Although chemical composition of each respective pool is not reported in this paper, percentage KPH lipid was greater while percent moisture was significantly less for the FF and LF heifers compared to the MFF heifers. Hence, an increased amount of lipid was deposited in KPH depots of the FF and LF heifers compared with MFF heifers. FF heifers also had increased lipid in OM compared to LF and MFF heifers. The same rational used for describing KPH lipid partitioning can be made for the FF heifers in relation to percent OM lipid. Perinephric fat had the greatest lipid content of fat depots in steer carcasses although a high versus low energy diet effect was not observed (Keane et al., 1991) in that study. The present results also support the view that increased energy intake is associated with increased lipid and decreased moisture and protein concentrations in soft tissue (Fortin et al., 1980; Brooks et al., 1985; Nour and Thonney, 1987; Keane et al., 1993).

The importance of body fat in maintenance of estrous cycles in rodents (Morin, 1986), humans (Bronson and Manning, 1991) and beef cattle (Imakawa et al., 1986; Richards et al., 1989) as well as its importance in postpartum reproduction in beef cattle (Dunn and Kaltenbach et al., 1980; Richards et al., 1986; Selk et al., 1986) are well

documented. However, evidence against the critical body fat (Kennedy, 1969; Frisch and McArthur, 1974) hypothesis for the onset of puberty is accumulating for several species including rodents (Hansen et al, 1983; Bronson, 1987), monkeys (Schwartz et al., 1988), and cattle (Brooks et al., 1985). There is no overwhleming evidence to suggest that there is a metabolic/endocrine signal that links fat with the GnRH pulse generator to initiate gonadotropin secretion and onset of puberty. The current "whole-body energy balance " hypothesis, suggest that availability of total body energy, of which energy stored in adipose tissue is an important component of, modulates the activity of the GnRH pulse generator in regulating ovulation in the prepubertal and adult females (Bronson and Manning, 1991). Total energy available, rather than specific substrates, metabolites, and/or hormones, may mediate the effects of nutrition on the GnRH pulse generator (Schillo et al., 1992). This theory is supported by research linking the importance of metabolic glucose and fatty acids to estrous cycle termination in rodents and sheep. An acute fast, blocked estrous cylces immediately in lean hamsters but not until several estrous cycles in obese hamsters (Schneider and Wade, 1989). Administering drugs to block glycolysis and fatty acid oxidation blocked estrous cycles in ad lib fed hamsters (Schneider and Wade, 1989) and altered LH secretion in sheep (Hileman et al., 1991; Bucholtz et al., 1992). Experiments in sheep (Crump et al., 1983) and cattle (Rutter and Manns, 1987, 1988) to modify LH secretion by altering glucose metabolism have been inconclusive. However, fermentation products in ruminants like the VFA, propionate, can alter glucose availability (Trenkle, 1981) and enhance LH release (Randel and Rhodes, 1980; Rutter et al., 1983) and decrease age at puberty in beef heifers (Mosely et al., 1977, 1982; McCartor et al., 1979).

The role of body fat in attainment of puberty in the female must be elucidated. An animal which could serve as an experimental model and has indirectly been selected for decreased age at puberty is the dairy heifer. Dairy breeds, historically selected for milk production, reach puberty at significantly younger ages than those not selected for milk

production (Gregory et al., 1978). Furthermore, muscle to bone ratios, an indicator of muscle mass, are greater for beef breeds than for dairy breeds (Cole et al., 1964; Berg, 1968; Broadbent et al., 1976). Therefore, in light of results of the present study where muscle:bone ratios were not affected by treatment, it seems logical to assume that dairy heifers may attain puberty earlier than beef heifers because they have less muscle mass to physiologically develop and can deposit more internal fat at a quicker rate which allows them to reach the proper physiological state to attain puberty at younger ages. This suggest that percentage body fat mass may not be the indicator of puberty but body fat distribution in association with lean and bone mass may function as a signal in the attainment of puberty.

Differing rates of gain, commencing at 8 mo of age, altered age, BW, carcass composition and lipid partitioning at puberty in beef heifers. Increased nutrient intake resulted in greater separable carcass + OM fat and a greater percentage carcass + OM lipid at puberty. Bone and lean mass were not influenced by treatment. Therfore, our study does not support the proposed theory in rats and humans that females reach puberty at a critical body composition. Our results do not preclude the possible importance of a critical body composition of heifers if age is not limiting, since heifers on reduced diets (LF and MFF) attained puberty at similar body weights and composition. Hence, body fat mass per se , is not the sole regulator of puberty, a minimal age appears to have a permissive effect for the initiation of estrous cycles.

Implications

Feeding programs that enhance adequate growth and development of replacement heifers could greatly increase the number of heifers reaching puberty at a young age. Alternate growing programs will also allow producers to develop replacement heifers of

different biological types (maturities) so that they will produce a calf at 24 mo of age and have a productive life. Alterations in nutrient intake of heifers during growth will allow researchers to develop experimental models to investigate physiological factors that regulate the onset of puberty.

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Table 1. Composition of diets

Item	Diet A	Diet B
Ingredients, DM%		
Rolled corn	73.00	-
Alfalfa hay	-	70.77
Alfalfa pellets	5.00	-
Cottonseed hulls	5.50	25.42
Soybean meal (44.0% CP)	11.64	-
Dical phosphate	-	.50
Limestone	1.53	-
Vitamin A, 30,000 IU/gm	.02	.02
Molasses	2.97	2.98
Salt	.30	.30
Calculated values		
NEm, Mcal/kg	2.04	1.23
NEg, Mcal/kg	1.31	.61
CP,%	13.7	14.6

Table 2. Effect of rate of gain on age, BW, and body condition score (BCS)[a] at puberty in beef heifers (least squares means)

	Treatment[b]				P - Value		
Measure	FF	LF	MFF	MSE	TRT	YR	TRT x YR
No. of heifers	13	12	13	-	-	-	-
Yr 1	7	5	6				
Yr 2	6	7	7				
Initial BW, kg	207	208	201	471	.69	.009	.73
Yr 1	218	220	207				
Yr 2	195	196	195				
Initial age, d	263	269	262	255	.51	.26	.80
Yr 1	265	274	263				
Yr 2	261	263	260				
Puberty BW, kg	350[c]	305[d]	310[d]	2089	.03	.27	.23
Yr 1	375	313	300				
Yr 2	323	305	310				
Puberty age, d	370	415	403	-	.03	.85	.006
Yr 1	389[c,d]	431[c]	371[d]	1849			
Yr 2	351[c]	398[d]	434[d]	1686			
BCS at puberty	6.4[c]	5.6[d]	5.4[d]	.28	.001	.05	.35
Yr 1	6.7	5.7	5.5				
Yr 2	6.1	5.6	5.3				

[a]BCS: 1 = emaciated, 9 = obese.
[b]Treatments = FF (full fed to gain 1.36 kg/d), LF (limit fed to gain .68 kg/d), MFF (full fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d).
[c,d]Means within a row lacking a common superscript differ (P<.05).

Table 3. Effect of rate of gain on carcass measurements at puberty in beef heifers (least squares means)

	Treatment[a]				P - Value		
Measure	FF	LF	MFF	MSE	TRT	YR	TRTxYR
Carcass wt[b], kg	221[e]	188[f]	178[f]	761	.001	.04	.11
Yr 1	243	195	178				
Yr 2	199	181	178				
KPH, %	3.8[e]	2.8[f]	2.2[f]	.68	.0001	.62	.42
Yr 1	4.2	2.8	2.1				
Yr 2	3.5	2.8	2.3				
Overall maturity[c]	117	136	131	557	.13	.18	.31
Yr 1	106	139	123				
Yr 2	128	132	138				
Marbling score[d]	334	281	281	10059	.24	.83	.77
Yr 1	349	257	290				
Yr 2	320	283	271				
Longissimus area, cm^2	61.9	60.6	56.8	28.58	.66	.83	.89
Yr 1	61.3	60.6	58.7				
Yr 2	62.6	60.0	54.8				
Fat thickness, mm	8.9[e]	5.5[f]	5.4[f]	.10	.01	.80	.53
Yr 1	9.9	5.5	4.9				
Yr 2	8.1	5.5	5.8				
Yield grade	2.8	2.5	2.5	.33	.58	.70	.97
Yr 1	2.7	2.5	2.4				
Yr 2	2.8	2.6	2.5				

[a]Treatment = FF (full fed to gain 1.36 kg/d), LF (limit fed to gain .68 kg/d), MFF (full fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d).
[b]Carcass wt = hot carcass wt + KPH + UDDER.
[c]Maturity: A = 100 to 199.
[d]Marbling score: slight = 300 to 399; traces= 200 to 299.
[e,f]Means within a row lacking a common superscript differ (P<.05).

Table 4. Effect of rate of gain on physically separable lean, bone, fat and soft tissue[a] carcass depots at puberty in beef heifers (least squares means)

	Treatment[b]				P - Value		
Measure	FF	LF	MFF	MSE	TRT	YR	TRT x YR
Lean, kg	102.0	100.0	96.5	283	.73	.29	.47
Yr 1	103.4	95.9	90.3				
Yr 2	100.1	104.6	102.7				
Bone, kg	33.0	33.0	30.5	12	.11	.83	.91
Yr 1	33.5	32.9	30.4				
Yr 2	32.6	33.1	30.4				
Carcass fat[c], kg	54.3	31.0	28.4	-	.0001	.26	.04
Yr 1	63.6[d]	32.1[e]	25.0[e]	153			
Yr 2	45.0[d]	30.0[e]	32.0[e]	154			
Soft tissue, kg	31.8[d]	23.8[e]	23.3[e]	36	.001	.001	.71
Yr 1	42.9	33.8	32.6				
Yr 2	20.6	13.9	14.0				

[a]Soft tissue = inseparable fat and lean.
[b]Treatment = FF (full fed to gain 1.36 kg/d), LF (limit fed to gain .68 kg/d), MFF (full fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d).
[c]Carcass fat = KPH + UDDER + SEAM + SC .
[d,e]Means within a row lacking a common superscript differ (P<.05).

Table 5. Effect of rate of gain on total physically separable body fat (BFAT)[a], omental-mesenteric fat (OM), kidney-pelvic-heart fat (KPH), subcutaneous fat (SC), intermuscular fat (SEAM), and UDDER at puberty in beef heifers (least squares means)

	Treatment[b]				P - Value		
Measure	FF	LF	MFF	MSE	TRT	YR	TRT x YR
TFAT, kg	70.0	39.0	36.4	-	.0001	.24	.05
Yr 1	81.3[c]	41.2[d]	31.8[d]	220.5			
Yr 2	58.6[c]	36.8[d]	40.9[d]	252.9			
OM, kg	15.8[c]	8.0[d]	7.9[d]	-	.0001	.24	.09
Yr 1	17.7	9.0	6.9	8.3			
Yr 2	13.8	7.0	9.0	13.6			
KPH, kg	8.6[c]	5.4[d]	4.1[d]	-	.0001	.17	.10
Yr 1	10.1	5.6	3.8	4.4			
Yr 2	7.1	5.2	4.3	4.4			
SC, kg	16.2[c]	8.9[d]	8.4[d]	-	.0001	.08	.07
Yr 1	19.6	10.2	7.6	21.6			
Yr 2	12.8	7.6	9.1	15.8			
SEAM, kg	25.5	14.5	13.8	-	.0001	.61	.02
Yr 1	29.6[c]	14.1[d]	11.5[d]	28.5			
Yr 2	21.4[c]	14.9[d]	16.0[c,d]	34.4			
UDDER, kg	3.9[c]	2.2[d]	2.2[d]	.8	.0001	.59	.22
Yr 1	4.3	2.3	2.0				
Yr 2	3.5	2.1	2.5				

[a]TFAT = OM + KPH + SC + SEAM + UDDER.
[b]Treatment = FF (full fed to gain 1.36 kg/d), LF (limit fed to gain .68 kg/d), MFF (full fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d).
[c,d]Means within a row lacking a common superscript differ (P<.05).

Table 6. Effect of rate of gain on percentages of separable carcass + OM fat (PFAT)[a] and the proportion of separable fat in omental-mesenteric (OM), kidney-pelvic-heart (KPH), intermuscular (SEAM) , subcutaneous (SC), and UDDER depots as a percentage of separable carcass + OM fat at puberty in beef heifers(least squares means)

	Treatment[b]				P - Value		
Measure	FF	LF	MFF	MSE	TRT	YR	TRT x YR
PFAT, %	29.3[c]	19.5[d]	18.7[d]	23.4	.0001	.93	.22
Yr 1	31.0	19.6	17.1				
Yr 2	27.6	19.4	20.4				
OM,%	22.8	21.2	21.1	-	.25	.47	.05
Yr 1	21.9	23.1	21.1	9.6			
Yr 2	23.6[c]	19.3[d]	21.2[c]	5.9			
KPH, %	12.2[c]	13.8[d]	11.3[c]	3.5	.008	.63	.67
Yr 1	12.4	13.6	11.7				
Yr 2	11.8	14.1	10.9				
SEAM, %	36.4	37.3	38.8	-	.11	.001	.04
Yr 1	36.3	34.2	37.1	9.8			
Yr 2	36.5[c]	40.3[d]	40.4[d]	7.2			
SC, %	22.9	21.9	22.6	8.1	.68	.01	.89
Yr 1	23.9	23.4	23.8				
Yr 2	21.9	20.4	21.3				
UDDER, %	5.7	5.7	6.1	.78	.43	.51	.37
Yr 1	5.4	5.7	6.3				
Yr 2	6.1	5.8	6.0				

[a]PFAT = (separable body fat/carcass weight + KPH + UDDER +OM).
[b]Treatments = FF (full fed to gain 1.36 kg/d), LF (limit fed to gain .68 kg/d), MFF (full fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d).
[c,d]Means within a row lacking a common superscript differ (P<.05).

Table 7. Effect of rate of gain on the proportion of separable fat in omental-mesenteric (OM), kidney-pelvic-heart (KPH), intermuscular (SEAM) , subcutaneous (SC), and UDDER depots as a percentage of carcass + OM weight[a] at puberty in beef heifers(least squares means)

	Treatment[b]				P - Value		
Measure	FF	LF	MFF	MSE	TRT	YR	TRT x YR
POM,%	6.6[c]	4.0[d]	4.0[d]	1.2	.0001	.90	.26
Yr 1	6.7	4.4	3.7				
Yr 2	6.5	3.7	4.4				
PKPH, %	3.6[c]	2.7[d]	2.1[d]	.6	.001	.63	.45
Yr 1	3.9	2.7	2.1				
Yr 2	3.3	2.7	2.1				
PSEAM, %	10.7	7.3	7.1	-	.0001	.26	.05
Yr 1	11.3[c]	6.7[d]	6.2[d]	2.2			
Yr 2	10.1[c]	7.8[d]	8.1[c,d]	3.1			
PSC, %	6.7[c]	4.4[d]	4.3[d]	2.3	.0002	.23	.31
Yr 1	7.5	4.8	4.1				
Yr 2	6.1	4.0	4.5				
PUDDER, %	1.7[c]	1.1[d]	1.1[d]	.11	.0003	.61	.82
Yr 1	1.7	1.1	1.1				
Yr 2	1.7	1.1	1.2				

[a] Carcass + OM weight = carcass weight + KPH + UDDER +OM.
[b]Treatments = FF (full fed to gain 1.36 kg/d), LF (limit fed to gain .68 kg/d), MFF (full fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d).
[c,d]Means within a row lacking a common superscript differ (P<.05).

Table 8. Effect of rate of gain on total carcass + OM lipid (TLIPID)[a] and omental-mesenteric lipid (OML), kidney-pelvic-and heart lipid (KPHL), subcutaneous lipid (SCL), intermuscular lipid (SEAML), and intramuscular lipid (LEANL)[b] at puberty in beef heifers (least squares means)

	Treatment[c]				P - Value		
Measure	FF	LF	MFF	MSE	TRT	YR	TRT x YR
TLIPID, kg	68.5	39.1	35.4	-	.0001	.13	.03
Yr 1	81.3[d]	43.1[e]	31.0[e]	292			
Yr 2	55.7[d]	35.4[e]	39.8[e]	242			
OML, kg	13.6	6.6	6.3	-	.0001	.32	.07
Yr 1	15.4[d]	7.4[e]	5.3[f]	7.7			
Yr 2	11.8[d]	5.8[e]	7.4[e]	11.0			
KPHL, kg	7.6[d]	4.7[e]	3.4[e]	3.9	.0001	.25	.12
Yr 1	8.9	4.8	3.1				
Yr 2	6.3	4.6	3.6				
SCL, kg	13.1	7.0	6.3	-	.0002	.05	.08
Yr 1	16.1[d]	8.4[e]	5.8[e]	20.1			
Yr 2	10.0[d]	5.7[e]	6.8[e]	11.7			
SEAML, kg	19.7	10.6	9.9	-	.0001	.31	.02
Yr 1	23.5[d]	11.1[e]	8.2[e]	26.2			
Yr 2	16.0[d]	10.2[e]	11.6[e]	22.3			
LEANL, kg	14.5	10.2	9.5	-	.001	.06	.03
Yr 1	17.4[d]	11.4[e]	8.6[e]	13.6			
Yr 2	11.6	9.1	10.4	10.5			

[a]TLIPID = OML + KPHL + SCL + SEAML + LEANL.
[b]LEANL = total lean lipid + total soft tissue lipid
[c]Treatment = FF (full fed to gain 1.36 kg/d), LF (limit fed to gain .68 kg/d), MFF (full fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d).
[d,e,f]Means within a row lacking a common superscript differ (P<.05).

Table 9. Effect of rate of gain on lipid partitioning in omental-mesenteric (OML), kidney-pelvic-heart (KPHL), intermuscular (SEAML), subcutaneous (SCL), and intramuscular (LEANL)[a] depots as a percentage of carcass + OM lipid (TLIPID)[b] at puberty in beef heifers (least squares means)

	Treatment[c]				P - Value		
Measure	FF	LF	MFF	MSE	TRT	YR	TRT x YR
OML, %	20.2[d]	17.3[e]	17.0[e]	6.7	.006	.48	.19
Yr 1	19.1	18.0	16.4				
Yr 2	21.3	16.5	17.5				
KPHL, %	11.1[d]	12.1[e]	9.5[d]	3.8	.009	.57	.45
Yr 1	11.0	11.3	9.8				
Yr 2	11.2	12.8	9.2				
SCL, %	18.9	17.0	16.8	9.2	.21	.02	.86
Yr 1	19.6	18.1	18.3				
Yr 2	17.9	15.9	15.3				
SEAML, %	28.7	27.1	27.8	-	.13	.003	.07
Yr 1	28.7[d]	25.5[e]	26.5[e]	3.2			
Yr 2	28.6	28.7	29.1	3.7			
LEANL, %	21.2[d]	26.5[e]	28.9[e]	23.3	.001	.70	.97
Yr 1	21.5	26.9	28.9				
Yr 2	20.9	26.0	28.8				

[a]LEANL = total lean lipid + total SFT lipid.
[b]TLIPID = OML + KPHL + SCL + SEAML + LEANL.
[c]Treatment = FF (full fed to gain 1.36 kg/d), LF (limit fed to gain .68 kg/d), MFF (full fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d).
[d,e]Means within a row lacking a common superscript differ (P<.05).

Table 10. Effect of rate of gain on lipid partitioning in omental-mesenteric (POML), kidney-pelvic-heart (PKPHL), intermuscular (PSEAML), subcutaneous (PSCL), and intramuscular (PLEANL)[a] depots as a percentage of carcass + OM weight[b] at puberty in beef heifers (least squares means)

	Treatment[c]				P - Value		
Measure	FF	LF	MFF	MSE	TRT	YR	TRT x YR
POML, %	5.8[d]	3.3[e]	3.2[e]	1.1	.0001	.95	.25
Yr 1	6.0	3.6	2.8				
Yr 2	5.6	3.1	3.6				
PKPHL, %	3.2[d]	2.4[e]	1.8[f]	.55	.0001	.78	.50
Yr 1	3.5	2.3	1.7				
Yr 2	3.0	2.4	1.9				
PSCL, %	5.5[d]	3.5[e]	3.2[e]	2.1	.0004	.13	.34
Yr 1	6.2	3.9	3.1				
Yr 2	4.8	3.0	3.3				
PSEAML, %	8.3	5.4	5.2	-	.0001	.94	.09
Yr 1	9.1[d]	5.3[e]	4.4[e]	2.6			
Yr 2	7.6[d]	5.4[e]	5.9[e]	2.5			
LEANL, %	6.1	5.2	5.5	-	.01	.33	.07
Yr 1	6.7[d]	5.5[e]	4.7[e]	1.0			
Yr 2	5.5	4.8	5.4	1.2			

[a]LEANL = total lean lipid + total SFT lipid.
[b]Carcass + OM weight = carcass weight + KPH + OM.
[c]Treatment = FF (full fed to gain 1.36 kg/d), LF (limit fed to gain .68 kg/d), MFF (full fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d).
[d,e,f]Means within a row lacking a common superscript differ (P<.05).

Table 11. Effect of rate of gain on the proportion of lipid (PLIPID), protein (PPROT), moisture (PMST), ash (PASH), fat free lean (FFL)[a] and percent fat free lean (PFFL)[b] as a percentage of carcass tissue + OM weight [c] at puberty in beef heifers (least squares means)

	Treatment[d]				P - Value		
Measure	FF	LF	MFF	MSE	TRT	YR	TRT x YR
PLIPID, %	34.0[f]	23.6[g]	22.1[g]	32.9	.0001	.67	.15
Yr 1	36.1	24.6	20.0				
Yr 2	31.4	22.9	24.0				
PPROT, %	14.5[f]	16.8[g]	17.2[g]	1.8	.0001	.44	.18
Yr 1	13.8	16.7	17.5				
Yr 2	1.2	16.8	16.9				
PMST, %	50.5[f]	58.8[g]	59.8[g]	20.3	.0001	.88	.13
Yr 1	49.0	58.1	61.7				
Yr 2	52.4	59.2	58.1				
PCASH, %	1.00	.85	.90	.4	.63	.26	.48
Yr 1	1.08	.55	.77				
Yr 2	.99	1.06	1.01				
FFL, kg	119	114	110	220	.33	.01	.53
Yr 1	129	118	114				
Yr 2	109	109	106				
PFFL, %	51.4[f]	59.0[g]	60.2[g]	14.7	.0001	.42	.10
Yr 1	50.4	59.3	62.4				
Yr 2	52.4	58.8	57.9				

[a]FFL = (carcass lean + carcass soft tissue) - (total lean lipid + total soft tissue lipid).
[b]PFFL = FFL/LEAN + SFT + SC + SEAM + KPH + OM.
[c]Carcass tissue + OM = (carcass weight + KPH + OM) - BONE.
[d]Treatment = FF (full fed to gain 1.36 kg/d), LF (limit fed to gain .68 kg/d), MFF (full fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d).
[f,g]Means within a row lacking a common superscript differ (P<.05).

Chapter V

Running Head: HORMONES AND PUBERTY IN BEEF HEIFERS

Changes in Luteinizing Hormone, Growth Hormone, Insulin-Like Growth Factor -I, Insulin and Metabolites Prior to Puberty in Heifers Fed to Gain at Two Rates [1,2]

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ABSTRACT: Fall born Angus x Hereford heifers were allotted to treatments to achieve the following growth rates: 1) full fed (n=10; FF) to gain 1.36 kg/d, and 2) maintenance-full fed (n=9; MFF) to gain .23 kg/d for 16 wk, then fed to gain 1.36kg/d. Growth hormone (GH), IGF-I, insulin, glucose, non-esterified fatty acids (NEFA), and progesterone were quantified in twice weekly blood samples until onset of puberty (based

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on plasma progesterone). Body weight, hip height, and pelvic area were recorded every 28 d. Frequent blood samples (n = 8 heifers/treatment) were collected every 14 d commencing on d 29 of treatment until onset of puberty to evaluate secretion of LH and GH. FF heifers were younger (368 d; P<.001), had less hip height (115 cm; P<.05) and smaller pelvic area (140 cm^2; P<.10) but had similar BW (320 kg) at puberty compared with MFF heifers (460 d; 119 cm;155 cm^2; 347 kg). FF heifers had greater (P<.05) concentrations of LH, IGF-I, and insulin in serum and glucose and plasma in plasma in twice weekly blood samples during the first 84 d of treatment than MFF heifers, while MFF heifers had greater (P<.05) serum GH and plasma NEFA concentrations compared with FF heifers. On day 68 of treatment, FF heifers had a greater (P<.05) LH pulse frequency than MFF heifers while pulse amplitude and mean LH were similar for heifers on both treatments. MFF heifers had greater mean serum GH concentrations (28.9ng/mL) and greater (P<.10) pulse amplitude (61.5 ng/mL) compared with FF heifers (16.9; 33.5 ng/mL; respectively), but pulse frequency was not influenced by treatment on d 68. Treatment did not influence secretion of LH and GH 24 d prior to puberty. Mean serum GH (12.9ng/mL) and GH pulse amplitude (40.7 mg/mL) in all heifers were greater (P<.05) 2 to 9 d than 16 to 23 d before puberty (10.4, 20.0 ng/ml, respectively). Nutrient restriction decreased LH pulse frequency and delayed puberty, and GH secretion increased just prior to puberty in heifers.

Key words: Growth Hormone, Heifer, IGF-I, LH, Nutrition, Puberty

Introduction

The major reason heifers do not become pregnant during the breeding season is due to failure of heifers to reach puberty (Bellows et al., 1979). Replacement heifers that conceive early in their first breeding season continue to calve early during the subsequent calving seasons and wean heavier calves throughout their lifetimes (Lesmeister et al., 1987). Fertility at the pubertal estrus is decreased compared to the third estrus in heifers

(Byerley et al., 1987). Therefore, it is imperative that replacement heifers reach puberty prior to the start of the breeding season to ensure optimum reproductive performance at breeding.

Increased rates of gain in beef heifers results in younger ages but heavier weights at puberty (Short & Bellows, 1971; See Chapter 4) while undernutrition can delay puberty (Arije and Wiltbank, 1971; Day et al., 1986; Kurz et al., 1990). A critical amount of body fat does not appear to be the only factor necessary for initiation of puberty in beef heifers (Brooks et al., 1985; See Chapter 4). Puberty appears to be associated with an age dependent decrease in estradiol negative feedback on LH secretion resulting in increased LH pulse frequency (Day et al ., 1984; Kinder et al., 1987). Dietary energy restriction results in decreased LH pulse frequency and delays the onset of puberty in beef heifers (Day et al., 1986; Kurz et al., 1990) and increased energy intake results in an increase in LH pulse frequency within 14 d (Kurz et al., 1990). The role of growth hormone (GH) in initiation of puberty is not clearly understood while IGF-I has been implicated (Granger et al., 1989; Jones et al., 1991; Simpson et al., 1991) as an important mediator for the initiation of puberty.

Increased nutrient intake during the prepubertal period could increase LH pulse frequency and initiate puberty (Kurz et al.,1990). Alterations in nutritional programs could allow producers to minimize feed cost without sacrificing reproductive efficiency. The objectives of this study were to determine the effect of rate of gain on secretion of LH and GH and to characterize IGF-I and metabolites during the prepubertal period.

Materials and Methods

Twenty fall born Angus x Hereford heifers were allotted by BW and age (273 d) into groups and within a group they were randomly assigned to two treatments. Heifers were fed diets (Table 1) to achieve the following growth rates: 1) full fed (diet A) to gain

1.36 kg/d (n = 10; FF); 2) maintenance fed (diet B) to gain .23 kg/d for 16 wk, then full fed (diet A) to gain 1.36 kg/d (n = 10; MFF). Within treatments, heifers were allotted by similar BW and fed in groups of two commencing on July 18. Nonshrunk BW was obtained once per wk. Shrunk BW, hip height, and pelvic area were determined every 4 wk after 16 h withdrawal of feed and water, and body condition scores (BCS: 1 = emaciated; 9 = odese; Wagner et al., 1988) were recorded for all animals every 2 wk. Twice weekly (Tuesday and Thursday) plasma and serum samples (20 mL) were collected via jugular venepuncture at approximately 1530 h. Plasma samples were collected in tubes containing EDTA (.10 mL of a 15% solution), placed on ice and centrifuged (3,000 x g for 20 min) within 1 h and plasma was decanted. Serum samples were allowed to clot (24 h at 4°C), centrifuged (3,000 x g for 20 min), and serum was decanted. Serum and plasma were stored at -20°C for subsequent hormone and metabolite analysis.

Twice weekly progesterone concentrations in plasma were quantified by RIA (Bishop and Wettemann, 1993) to determine onset of puberty. Progesterone concentrations greater than 1 ng/mL for four consecutive samples was the criterion for the onset of puberty. Date of the first of the four consecutive samples with progesterone greater than 1 ng/mL was considered age at puberty. Pubertal BW was expressed as a shrunk wt and was determined by multiplying the estimated ADG between shrunk weights, by the days from weighing until puberty, and adding the weight gained to the last shrunk weight before puberty. Hip height and pelvic area at puberty were determined in a similar fashion.

Starting on day 29 of treatment and approximately every 14 d until puberty, serum (15 mL) was collected every 10 min for 6 h from eight heifers per treatment to assess LH and GH secretion. A polyvinyl cannula was inserted into a jugular vein of each heifer the day prior to sampling and heifers were confined in stalls overnight. Heifer receiving the FF diet had access to feed and water overnight while heifers receiving the LF diet had access only to water. On the morning of sampling , LF and FF heifers were allowed

access to feed and water for 1 h, then feed was removed. Three weeks prior to the first intensive sampling, heifers were placed in stalls a minimum of three times weekly to acclimate heifers and to minimize handling stress.

Concentrations of glucose and NEFA in plasma and insulin, LH, GH, and IGF-I in serum were determined in all twice weekly samples collected prior to puberty. LH and GH in serum were determined in intensive samples taken on day 68 of treatment and retrospectively for the two sampling periods (wk 1 = 2 to 9 d and wk 3 = 16 to 23 d) prior to the onset of puberty. Glucose was determined by an enzymatic colorimetric procedure (Sigma, No. 510, Sigma Chemical Co., St. Louis, MO). Intra- and interassay CV (n = 10 assays) were 4.5 and 6.7%, respectively. Concentrations of NEFA were determined by an enzymatic colorimetric procedure (Wako-NEFA C, Wako Chemicals Inc., Dallas, TX) with modification as described McCutcheon and Bauman (1986). Intra- and interassay CV (n = 11 assays) were 8.7 and 12.7%, respectively. Insulin in plasma was quantified by RIA as described by Selk (1986) and intra- and interassay CV (n = 4 assays) were 16.0 and 14.1%, respectively. Growth hormone was quantified by RIA (see chapter III) and intra- and interassay CV (n = 10 assays) were 4.8 and 16.4%, respectively. Concentrations of LH were quantified by RIA (Bishop et al., 1993) and intra- and interassay CV (n = 4 assays) were 7.6 and 28.0%, respectively. Concentrations of IGF-I were quantified by RIA (Echternkamp et al., 1990) after an acid ethanol extraction. Intra- and interassay CV (n=5 assays) were 9.5 and 16.6%, respectively.

An LH pulse in frequent samples was defined as a concentrations of LH that was greater than one standard deviation above the mean for a heifer on a day and was followed by at least two samples of lesser concentration. Amplitude of a LH pulse was the difference between the greatest value during a pulse and the nadir within 30 min prior to the pulse. A GH pulse was defined as a concentration of GH that was greater than one-half a standard deviation above the mean for a heifer on a day and was followed by at least

two values of lesser concentration. Amplitude of a GH pulse was the difference between the greatest concentration during a pulse and the nadir within 40 min prior to the pulse.

Analyses of variance were used to determine treatment effects on age, BW, BCS, hip height,and pelvic area at puberty, and LH and GH pulse frequency, amplitude, and mean concentrations on day 68 of treatment. One MFF heifer did not attain puberty by 280 d of treatment and was removed from all analyses. Analyses of variance of twice weekly hormone and metabolite samples and the LH and GH pulse frequency, amplitude, and mean concentrations at the two periods prior to puberty were analyzed as a split plot analyses of variance (Gill and Hafs, 1971). Treatment was the main plot and heifer within treatment was the error term for the main plot. Day was the subplot. If a significant treatment x day interaction existed, response curves for hormone and metabolite concentrations were characterized by time trends that were analyzed via regression (SAS, 1988). Test of homogeneity of regression were utilized to determine differences among time trends for hormone and metabolite responses for FF and MFF treatments (Snedecor and Cochran, 1968).

Results

Age, BW, BCS, hip height, and pelvic area were similar for FF and MFF heifers pretreatment (Table 2). The FF heifers were younger (368 d; $P<.001$; Table 2)than the MFF heifers (460 d) at puberty. Treatment did not influence BW at puberty (321 and 347 kgfr FF and MFF, respectively). Although, the FF heifers tended to have greater BCS (5.8) at puberty than the MFF heifers (5.4) the difference was not significant ($P.>18$). Hip height ($P<.05$) and pelvic area were greater ($P<.10$) for the MFF heifers (119.5 cm; 155.7 cm^2; respectively) compared with the FF heifers (115.6 cm; 140.4 cm^2; respectively) at puberty.

There was much variation in the age at puberty for the MFF heifers. Puberty occured within 48 d after increased nutrient intake (d 160 of treatment) for more than half (n=5; Figure 1) of the MFF heifers (MFF1), while the remainder of the heifers attained puberty between 169 and 280 d of treatment (n=4;MFF2). The MFF2 heifers tended to weigh less (P<.10) and had less (P<.02) BCS at initiation of treatment and on day 112 of treatment (day nutrient intake was increased) compared with MFF1 heifers (data not presented). MFF1 heifers that attained puberty early had similar BW, hip height and pelvic area as FF heifers, but were older (P<.05) and had less BCS (P<.09) than FF heifers at puberty. The MFF2 heifers had greater (P<.05) BW, hip height, pelvic area, and age at puberty but similar BCS as FF and early MFF1 heifers.

Because 70% of the FF heifers had attained puberty by d 112 of treatment, twice weekly blood samples were analyzed during the first 84 d of treatment and concentrations of LH, IGF-I, and GH in serum presented in figure 2. There was a treatment x day effect on serum LH concentration during the first 84 d of treatment and concentrations were best described by a third order polynomial equation . The FF heifers had greater (P<.01) serum LH concentration than the MFF heifers. Average LH concentrations in FF heifers were reduced when some heifers had initiated estrous cycles. There was a treatment x day effect on serum IGF-I concentrations and concentrations were best described by a second order polynomial equation. The FF heifers had greater (P<.001) serum IGF-I concentration than the MFF heifers. There was also a treatment x day effect on serum GH concentrations for the first 84 d of treatment and concentrations were best described by a second order polynomial equation. The MFF heifers had greater (P<.001) serum GH concentrations than the heifers.

Concentrations of glucose and NEFA in plasma and insulin in serum for the first 84 d of treatment are in figure 3. There was a treatment x day effect for glucose concentrations and data were best described by a second order polynomial equation. The FF heifers tended (P<.15) to have greater plasma glucose concentrations than the MFF

heifers. There was a treatment x day effect for NEFA concentrations and data were best described by a quadratic equation. The MFF heifers had greater (P<.001) concentrations of NEFA than the FF heifers. There was also a treatment x day effect on serum insulin concentrations and concentrations were best described by a linear equation during the first 84 days of treatment. The FF heifers had greater (P<.001) insulin concentrations than the MFF heifers.

Eight heifers on each treatment were frequently bled on day 68 to evaluate secretion of LH and GH . FF heifers had more (P<.03) LH pulses (2.3 pulses/6 h) than MFF (1.2 pulses/6 h) heifers (Table 3). However, pulse amplitude and mean concentrations of LH were not different for FF and MFF heifers. Serum GH concentrations for the MFF (28.9 ng/mL) heifers were greater than for FF (16.9 ng/mL) heifers on d 68 of treatment but the number of GH pulses was not influenced by treatment. Amplitude of the GH pulses tended (P<.10) to greater for the MFF (61.5 ng/mL) compared with the FF (33.5 ng/mL) heifers.

Treatment did not effect serum concentrations of LH, GH, and insulin, and plasma NEFA concentrations in twice weekly samples during the 10 wk preceding puberty (Table 4). There was a treatment x day (P<.10) effect on concentrations of glucose during the 10 wk before puberty and the response was best described by a linear equation (Figure 4). Glucose concentrations for MFF heifers increased linearly prior to puberty while glucose concentrations for FF heifers did not change. There was a treatment (P<.05) and day (P<.001) effect on serum IGF-I concentrations during the 10 wk prior to puberty and the response was best described by third order polynomial equation (Figure 4). Concentartions of IGF-I were greater and remained constant during the 10 wk before puberty in FF heifers, and IGF-I concentrations were less in MFF heifers at 10 wk before puberty and increased to concentrations similar to those in FF heifers at puberty.

LH and GH secretion were evaluated at one (wk 1) and three (wk 3) prior to puberty (Figure 5, 6). Concentration, number of pulses, and pulse amplitude of LH were

not influenced by treatment or period prior to puberty (Figure 5). Concentration, number of pulses, and pulse amplitude of GH were not influenced by treatment but were influenced by days prior to puberty (Figure 6). Concentrations of GH were greater ($P<.06$) during 1 (12.9 ng/mL) compared with 3 wk (10.4 ng/mL) prior to puberty. Pulse amplitude was greater ($P<.001$) during 1 (40.7 ng/mL) compared with 3 wk (20.0 ng/mL) prior to puberty, while pulse frequencywas not influenced by the interval before puberty.

Discussion

Increased nutrient intake of heifers on the FF treatment resulted in a younger age but a similar BW heifers at puberty compared with MFF heifers. A similar treatment effect on age at puberty was reported in chapter III and IV of this thesis. Body condition score of FF heifers tended ($P<.18$) to be greater than the BCS of MFF heifers. In the previous experiment, we found that BW was heavier and BCS was greater for FF heifers compared with MFF heifers at puberty, and increased BW and BCS were due to increased fat deposition in FF heifers. Increased nutrient intake is negatively correlated with age at puberty in dairy (Sorenson et al., 1959: Menge et al., 1960) and beef heifers (Wiltbank et al., 1966, 1969; Aije and Wiltbank, 1971: Short and Bellows, 1971; Greer et al., 1983;) and positively related to weight at puberty (Wiltbank et al., 1966; Aije and Wiltbank, 1971: Short and Bellows, 1971). In the present experiment, age but not weight at puberty was altered in heifers on the diet which caused greater daily gains. This was probably related to increased variation in age and BW at puberty of MFF heifers. Some of the MFF heifers (n=5) reached puberty soon after increased nutrient intake on d 136 of treatment, and had similar BW, pelvic area, hip height, and less BCS, but were older at puberty than FF heifers. Conversely, the remaining MFF heifers reached puberty later after increased nutrient intake (on an average of d 237 of treatment) and had significantly greater BW, pelvic area, hip height and age at puberty than the FF and MFF early heifers. Heifers with

larger pelvic widths reached puberty at younger ages. Effect of rate of gain on skeletal structure and its role in prepuberal develoment is inconclusive. Decreased rate of gain resulted in older heifers with increased pelvic areas (Short and Bellows, 1971; Varner et al., 1977) and hip heights (Grass et al., 1982) at puberty. Increased rate of gain also increased pelvic area (Wiltbank et al., 1985) and decreased age at puberty without effecting hip height at puberty (Ferrell 1982; Nelson et al., 1982). Joubert (1954) and Short and Bellows (1971) concluded that increased nutrient intake has a greater effect on soft tissue development than on skeletal growth. Results from chapter 3 indicated that bone weights were similar at puberty for both FF and MFF heifers.

Although the sample size was small in the present study, increased age and weight variations at puberty in the MFF heifers could be due to several factors: 1) pretreatment BW, 2) genetic variation within the sample, 3) season of birth, 4) photoperiod, or 5) a combination of these factors . Sources of genetic variation for age and weight at puberty are quite large for straight bred (Cundiff et al., 1986) and crossbred (Laster et al ., 1976) catttle. Photoperiod (Peters et al., 1978; Hansen et al., 1983) and season (Schillo et al., 1983) may alter age at puberty. However, the role of photoperiod and season on attainment of puberty is inconclusive.

During the first 84 d of treatment, concentrations of LH in serum were determined by twice weekly sampling. The FF heifers had greater LH concentrations compared with MFF heifers. Pulsatile secretion of LH is necessary to initiate ovarian cycles in monkeys (Knobil, 1981) and heifers (Day et al., 1984; Kinder et al., 1987). Less nutrient intake of MFF heifers on d 68 of treatment resulted in decreased pulsatile secretion of LH; however, pulse amplitude and LH concentrations were not influenced by diet. The short sampling period (6 h) utilized and/or the large variation in pulse amplitude may have influenced our ability to assess treatment effects on pulse amplitude and concentrations of LH. A decrease in pulse frequency, amplitude and mean concentrations of LH due to

decreased nutrition resulted in delayed puberty in ewes (Foster et al., 1989) and beef heifers (Day et al., 1986; Kurz et al., 1990).

Concentrations of LH in serum of FF heifers increased soon after the initiation of treatment. Diet of FF heifers was primarily grain based. Grain diets cause increased production of the VFA, propionate, and provide more precursors for gluconeogenesis (Trenkle, 1981). Diets that increase the propionate:acetate ratio decrease age at puberty in beef heifers (Mosely et al., 1977; McCartor et al., 1979; Mosely et al., 1982). Abomasal infusion of propionate resulted in an increased release of LH from the pituitary after administration of LHRH (Rutter et al., 1983). However, the experiment could not distinguish whether it was a direct propionate effect or a general energy effect. Therefore, increased propionate may be involved in enhancing LH secretion in the FF heifers resulting in earlier puberty while undernutirtion resulted in decreased LH pulse frequency and delayed puberty of MFF heifers.

There was a negative relationship between IGF-I and GH secretion during the first 84 days of treatment. Serum IGF-I concentrations were positively associated with increased nutrient intake. IGF-I concentrations in the FF heifers increased with day of treatment, while IGF-I concentrations decreased with time during nutritional restriction of MFF heifers. Nutritional restriction decreases IGF-I concentrations in ram lambs (Clarke et al., 1993) steers (Brier et al., 1986; McKinnon et al., 1993) cows (Richards et al., 1991; Nugent et al., 1993) and heifers (Houseknecht et al., 1988:Granger et al., 1989; Armstrong et al., 1993). In contrast , decreased nutrient intake resulted in greater GH concentration in MFF heifers compared with FF heifers. Growth hormone decreased after the treatment was initiated in FF heifers and then gradually increased during the period when FF heifers began to attain puberty. Decreased nutrition increases GH concentrations in rams (Clarke et al., 1993) steers (Brier et al., 1986; Mosely et al., 1988; McKinnon et al., 1993) and heifers (Houseknecht et al., 1988:Granger et al., 1989; Simpson et al., 1991; Armstrong et al., 1993). The positive relationship that usually exist between GH

and IGF-I appears to be uncoupled when nutrient intake is restricted (Elsassar et al., 1989: Granger et al., 1989; Armstrong et al., 1993). Uncoupling of GH and IGF-I appears to occur only when nutrition is limiting or in excess. IGF-I and GH concentrations are positively associated in animals on moderate nutrient intake (Granger et al., 1989; Experiment I, Chapter IV). When BW is increasing rapidly (soft tissue growth - lean and fat), GH concentrations are decreased and IGF-I concentrations are increased while the converse is true during periods of nutrient restriction when tissue mobilization is occurring to supply nutrients for body maintenance. Growth hormone treatment of hypophysectomized rats decreased body fat and increased protein:fat in animals on restricted nutrient intake (Bates et al.,1993). In ram lambs sired by either lean or fat genotype rams, increased GH concentrations resulted in decreased carcass fat and IGF-I had a positive relationship with fat deposition within and between genotypes (Suttie at al., 1993). In experiment 1 (chapter III) we observed that increased GH concentrations during nutrient restriction of MFF heifers resulted in decreased carcass fat at puberty.

Nutrient intake did not influence the frequency of GH pulses on d 68 of treatment, but heifers on the maintenance diet had a greater amplitude of pulses which probably caused the increased serum GH concentrations. This agrees with reports in sheep (Thomas at al., 1991; Clarke et al., 1993), steers (Brier et al., 1986), and heifers (Houseknecht et al., 1988) while others have attributed the differences in GH concentrations induced by dietary restrictions to only differences in pulse frequency (Villa-Godoy et al., 1990; Armstrong et al., 1993). The number of pulses identified is related to the method used to define pulses (Vizcarra et al., 1993).

When nutrients are restricted, GH acts to repartition nutrients away from fat deposition and into protein and skeletal development, and nutrients are not as available to stimulate gonadotropin secretion for normal estrous cycles. When diets contain adequate energy, the lipolytic effects of GH to provide substrate are no longer necessary. As skeletal and muscle requirements are met, excess energy can be partitioned to events

necessary for reproduction, and, finally, fat deposition. IGF-I may be a more active regulator of metabolic processes during increased nutrient intake than during restricted nutrient intake. Whether enhanced reproductive function is the result of increased IGF-I concentration, or the increased energy necessary to stimulate endocrine function, is open to much debate.

During the first 84 d of treatment, concentrations of insulin in serum and glucose and NEFA in plasma in twice weekly blood samples are indicative of nutrient intake. Concentrations of insulin and glucose were positively associated with feed intake. Similar to other results for ruminants (Bassett et al., 1971; Peters, 1986; Richards et al., 1989a) the FF heifer had greater insulin concentrations due to increased nutrient intake compared with MFF heifers. Ruminants fed greater amounts of grain have significantly greater concentrations of plasma insulin (Trenkle, 1981). Increased nutrient intake of FF heifers tended to increase plasma glucose compared with MFF heifers. The influence of insulin and glucose on LH secretion remains unclear. Infusion of insulin in heifers (Harrison and Randel, 1986) and postpartum cows (Garmendia, 1986) did not influence secretion of LH and changes in serum LH concentrations have not been observed in hypoglycemic cows (Rutter and Manns, 1987, 1988). Decreased nutrient intake reduces plasma glucose in nutritional anestrus cows (Richards et al.,1989b), and the lack of available substrate will not permit adequate secretion of LH to stimulate ovarian function. However, this may only occur with severe nutrient restriction.

In contrast to insulin concentrations, mean plasma NEFA concentrations were negatively associated with feed intake during the first 84 d of treatment. Greater nutrient intake of the FF heifers resulted in decreased NEFA concentrations compared with MFF heifers. Increased plasma concentrations of NEFA are indicative of negative energy balance (Brier et al., 1986; Eisemann et al., 1986; Peters, 1986; Richards et al., 1989a) and fatty acid release from the adipocyte (Bines and Hart, 1982). Hence, increased plasma NEFA concentrations in MFF heifers were probably a direct result of increased serum GH

concentrations and the effect on increased lipolysis and altered lipogenesis (DiMarco et al., 1981; Eisemann et al., 1986; Peters, 1986). Decreased reproductive activity of dairy cows in negative energy balance was associated with increased NEFA concentrations (Canfield and Butler, 1991). When nutritional anestrus was induced in cows, concentrations of NEFA in plasma increased (Richards et al., 1989b) and LH pulse frequency was decreased (Richards et al., 1989a). Nutritional anestrus was also associated with a decrease in LH pulse frequency in heifers (Imakawa et al., 1986). Short term infusion of free fatty acids in ovariectomized ewes in a positive energy balance, suppressed pulsatile GH release but did not alter pulsatile LH secretion (Estienne et al.,1989, 1990). Whether NEFA's have an inhibitory effect on GnRH secretion has yet to be determined.

Mobilizaion of fatty acids provides energy for many cellular processes. Growth hormone may act as a homeorhetic regulator in animals on maintenance diets to support lean tissue accretion (Eisemann et al., 1986; Peters, 1986) by altering metabolism of other body tissues. The role of GH as a mediator in other physiological actions such as reproduction are less clear. However, when Schneider and Wade (1989) blocked glycolysis in hamsters, estrous activity was unaltered, but when glycolysis and fatty acid oxidation were blocked there was an immediate cessation of estrous cycles. Pharmacological blocking of glycolysis and fatty acid oxidation resulted in cessation of pulsatile LH release in lambs (Hilemann et al., 1991) and decreased circulating LH concentrations in growth retarded lambs (Bucholtz et al., 1992). Modified glucose metabolism altered LH release in hamsters (Morin, 1986), sheep (Crump et al., 1982), and cattle (Rutter and Manns, 1987, 1988).

During the 10 wk prior to puberty, treatment did not influence concentrations of LH, GH, and insulin in serum, and concentrations of NEFA in plasma. Concentrations of glucose in plasma of FF heifers did not change during the 10 wk before puberty, while concentrations of glucose in MFF heifers increased linearly prior to puberty.

Concentrations IGF-I in serum of FF heifers during the 10 wk prior to puberty were greater than those for MFF heifers. As MFF heifers approached the time of puberty, IGF-I concentrations increased to concentrations similar to those of FF heifers. All MFF heifers were on increased nutrition for at least 4 wk before the onset of puberty, therefore part of this increase could be attributed to nutrient intake (Houseknecht et al., 1988:Granger et al., 1989; Armstrong et al., 1993). Jones et al. (1991) found increased IGF-I concentrations prior to puberty and Granger et al. (1989) indicated that decreased IGF-I concentrations were positively associated with delayed puberty in heifers fed low quality hay compared to heifers fed hay plus supplement. In postpartum cows (Rutter et al., 1989; Nugent et al., 1993) IGF-I concentrations were positively correlated with reproductive endocrine function prior to ovulation. Houseknecht et al. (1988) concluded that dietary energy had a greater influence than energy source on IGF-I concentrations. Concentrations of IGF-I increase prior to puberty in female rodents (Handelsman et al., 1987), primates (Copeland et al., 1982) and humans (Hiney et al., 1991). Ovarian follicular fluid and serum concentrations of IGF-I are positively correlated (Echternkamp et al., 1990; Lucy et al., 1992) and IGF-I may have direct local effects on granulosa cell production of estradiol (Spicer et al., 1993), to stimulate follicular growth and ovulation. Most studies in ruminants indicate a positive association between concentrations of IGF-I and nutrient intake.

Pulsatile secretion of LH is the best predictor of age at puberty (Kinder et al., 1987) and an increase in LH pulse frequency occurs 50 d prior to puberty in beef heifers. When LH secretion was examined retrospectively from puberty in the present study, there was no treatment or week effect on serum concentration of LH, pulse amplitude or pulse frequency at 1 and 3 wk prior to puberty during the fall and winter months. Season may influence LH secretion in cows (Day et al., 1986b; Stumpf et al., 1988). However, it is difficult to determine if increased nutrient intake prior to puberty masked any season effects.

Treatment had no influence on concentrations of GH in serum when determined retrospecively from puberty but there was a significant day effect. Concentrations of GH and pulse amplitude were significanlty greater at 1 wk prior to puberty compared with 3 wk prior to puberty. Pulse frequency was not influenced by treatment (P>.14) at 1 and 3 wk prior to puberty. Secretion of GH has not been characterized in heifers prior to puberty and a possible role of GH as a mediator of puberty has not been adequately evaluated. Studies attempting to link GH with attainmment of puberty in other species have been inconclusive. Bucholtz et al. (1990) infused prepuberal lambs with pituitary derived bovine GH and concluded that neuroendocrine sexual maturity was not compromized compared to untreated lambs. Immunizing male rats (Arsenijevic et al., 1989) and heifers (Simpson et al.,1991) against growth hormone-releasing factor delayed onset of puberty and IGF-I production was decreased in immunized animals. Growth hormone treatment stimulates follicular growth in pigs (Spicer et al., 1990), heifers (Gong et al., 1993), and cows (De La Sota et al., 1991) and growth hormone-releasing factor increased the size of large follicles (Spicer & Enright, 1991) in heifers. Growth hormone also influences bovine granulosa cell funciton in vitro (Langhout et al., 1991) and granulosa cell function in rats and pigs (Advis et al., 1981; Jia et al., 1986). Growth hormone, in coordination with IGF-I, may indirectly stimulate follicular growth and steriodogenesis during the prepuberal period.

In summary, decreased nutrient intake of prepuberal heifers resulted in decreased concentrations of insulin, glucose, LH and IGF-I in blood and increased GH concentrations compared with heifers on ad libitum intake with rapid weight gains. Increased nutrient intake and rapid gain of FF heifers decreased age at puberty compared with heifers on restricted intake. Decreased LH pulse frequency was associated with delayed puberty in restricted heifers heifers. Secretion of LH during the 3 wk prior to puberty was not altered by age at puberty. Growth hormone secretion prior to puberty

was not influenced by nutritional treatments but mean serum GH concentrations and pulse amplitude were increased just prior to puberty.

Implications

Numerous factors influence age at puberty in heifers. Faster growing heifers reach puberty at younger ages compared with slower growing heifers. Increased nutrient intake, after an extended period of restricted nutrient intake, hastened the onset of puberty. Feeding programs, like the one used in this study, can be designed to reduce feed cost in replacement heifer development; however, additional research to determine proper duration and level of nutrient intake, both during the restricted and increased nutrient intake period, without compromising reproductive efficiency must be conducted. As a result, feeding programs could be developed for cattle of different maturities (biological types) and provide producers increased management flexibility from both a biological and economic standpoint in develoing replacement heifers. Nutritional manipulation will also allow the researcher to better understand the physiological factors that affect age at puberty.

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Table 1. Composition of diets

Item	Diet A	Diet B
Ingredients, DM%		
Rolled corn	73.00	-
Alfalfa hay	-	70.77
Alfalfa pellets	5.00	-
Cottonseed hulls	5.50	25.42
Soybean meal (44.0% CP)	11.64	-
Dical phosphate	-	.50
Limestone	1.53	-
Vitamin A, 30,000 IU/gm	.02	.02
Molasses	2.97	2.98
Salt	.30	.30
Calculated values		
NEm, Mcal/kg	2.04	1.23
NEg, Mcal/kg	1.31	.61
CP,%	13.7	14.6

Table 2. Effect of rate of gain on age, BW, body condition score (BCS)[a] , hip height and pelvic area at puberty in beef heifers (least squares means; ±SE)

	Treatment[b]		
Measure	FF	MFF	P-value
No. of heifers	10	9	
Pretreatment			
Age, days	272.0 ± 2.2	273.6 ± 2.3	-
BW, kg	202.5 ± 8.3	201.5 ± 8.8	-
BCS	4.5 ± .1	4.5 ± .1	-
Hip height, cm	106.7 ± 1.1	109.5 ± 1.2	-
Pelvic area, cm^2	112.4 ± 4.6	113.2 ± 4.8	-
Pubertal			
Age, d	368.5 ± 16.0	460.3 ± 16.9	.001
BW, kg	320.7 ± 16.9	347.0 ± .29	.29
BCS	5.8 ± .2	5.4 ± .2	.18
Hip height, cm	115.6 ± 1.3	119.5 ± 1.4	.05
Pelvic area, cm2	140.4 ± 6.1	155.7 ± 6.4	.10

[a]BCS: 1 = emaciated, 9 = obese.
[b]Treatments = FF (full fed to gain 1.36 kg/d), MFF (full fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d).

Table 3. Least squares means (±SE) for pulse frequency, pulse amplitude, and mean concentrations of LH and growth hormone (GH) on day 68 of treatment

Measure	Treatment[a] FF	Treatment[a] MFF	P-value
Pulses/6 h			
LH	2.3 ± .3	1.2 ± .4	.03
GH	3.8 ± .5	3.5 ± .5	.67
Pulse amplitude, ng/mL[b]			
LH	3.5 ± 1.3	5.1 ± 1.5	.45
GH	33.5 ± 10.9	61.5 ± 10.9	.10
Mean, ng/mL			
LH	3.6 ± .1	3.4 ± .1	.65
GH	16.9 ± 2.4	28.9 ± 2.4	.01

[a]Treatments = FF (full fed to gain 1.36 kg/d), MFF (full fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d).
[b]Difference between peak and previous nadir.

Table 4. Least squares means (±SE) for concentrations of growth hormone (GH), LH, and insulin in serum, and nonesterified fatty acids (NEFA) in plasma during the 10 wk prior to puberty in heifers fed to gain at different rates

Measure	Treatment[a]						P-value
	FF			MFF			
GH, ng/mL	16.9	±	2.5	14.5	±	2.4	.73
LH, ng/mL	5.9	±	.4	5.6	±	.4	.57
Insulin, ng/mL	2.3	±	.3	2.1	±	.3	.68
NEFA, mEq/mL	180.4	±	18.1	198.2	±	17.9	.31

[a]Treatments = FF (full fed to gain 1.36 kg/d), MFF (full fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d).

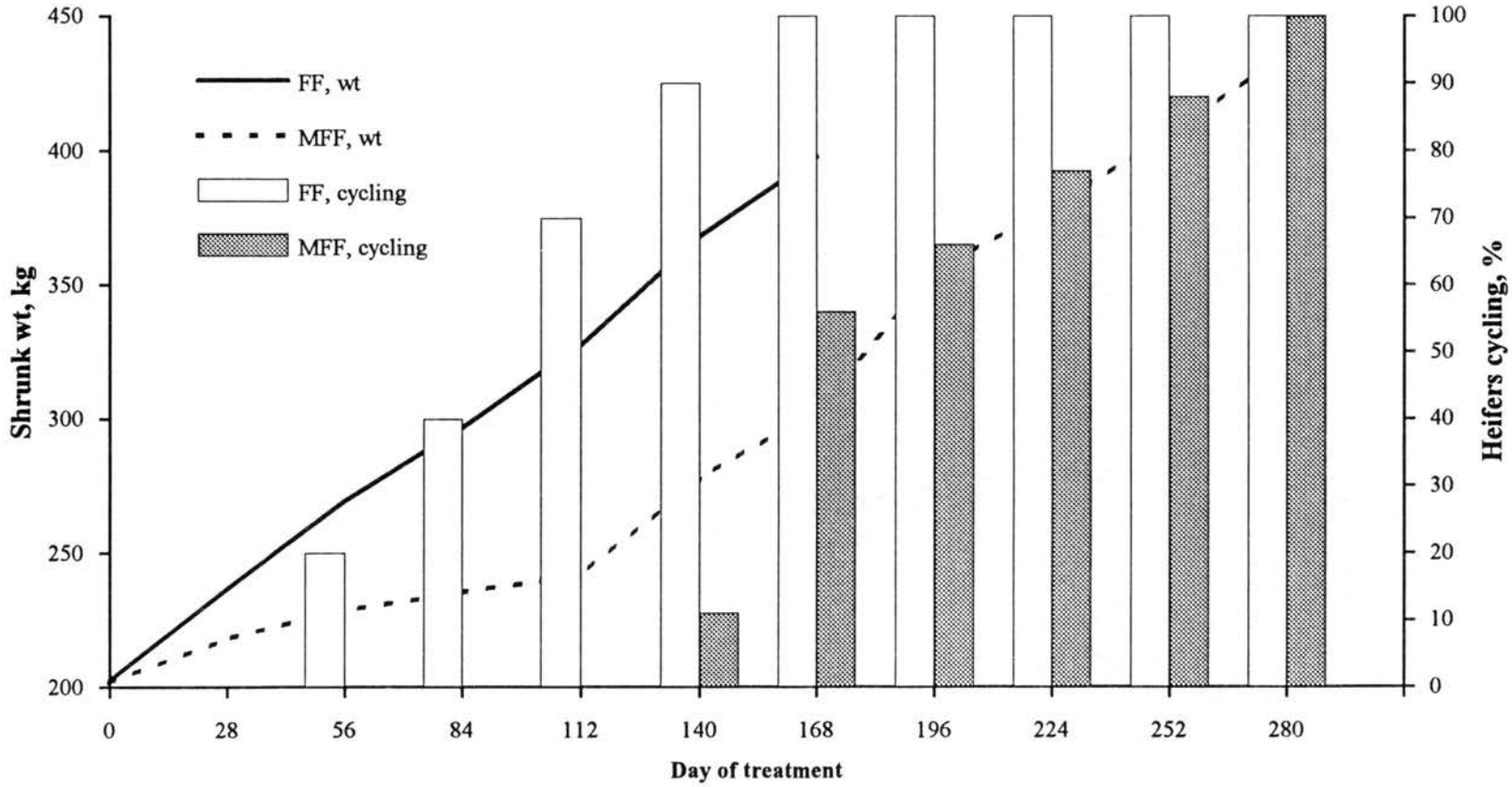

Figure 1 Shrunk BW(kg) and percentage of FF (full fed to gain 1.36 kg/d) and MFF (full fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d) heifers cycling during treatment.

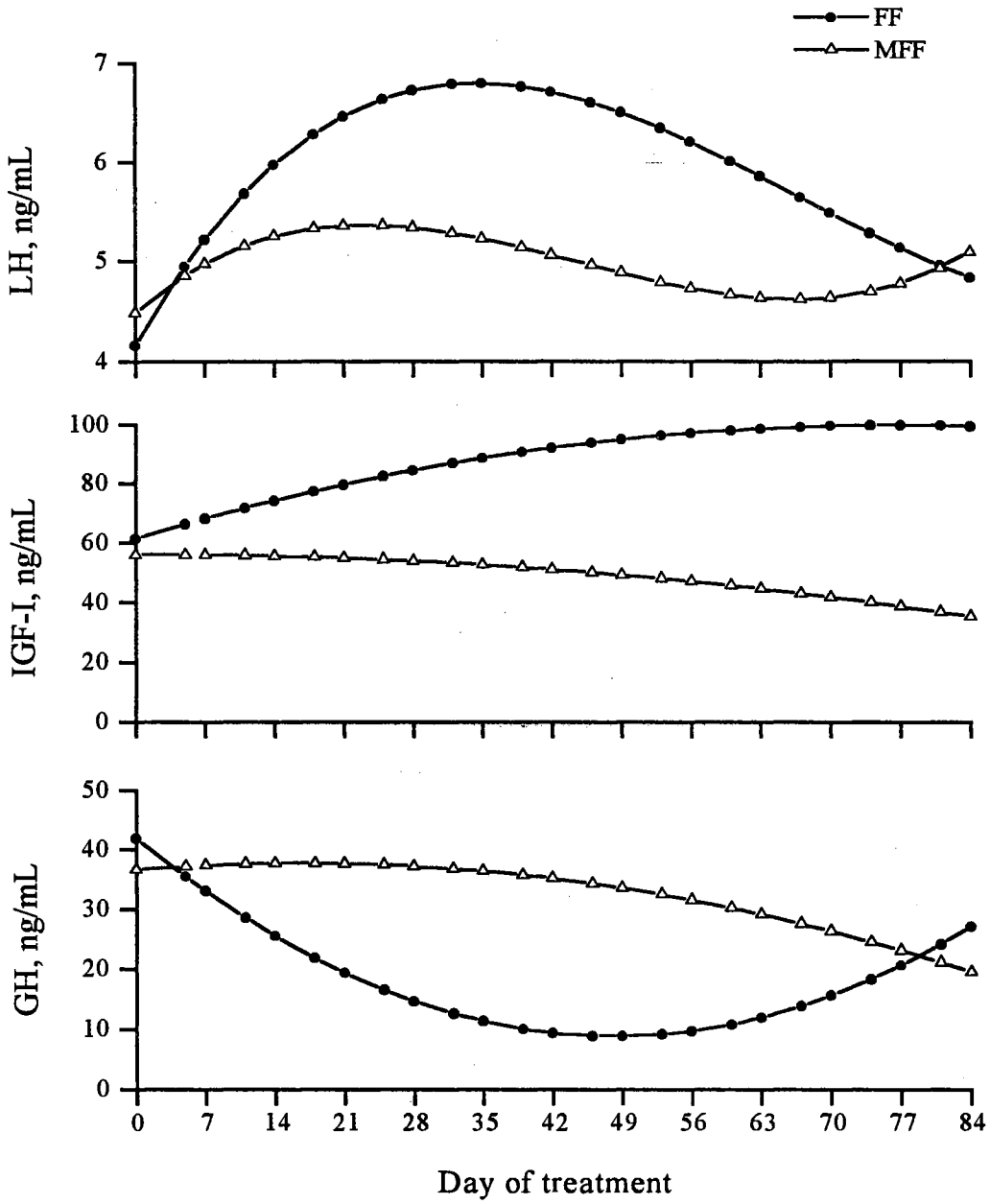

Figure 2. Least squares regressions for concentrations of LH, IGF-I, and growth hormone (GH) in serum of FF (full fed to gain 1.36 kg/d) and MFF (full fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d) heifers during the first 84 d of treatment.

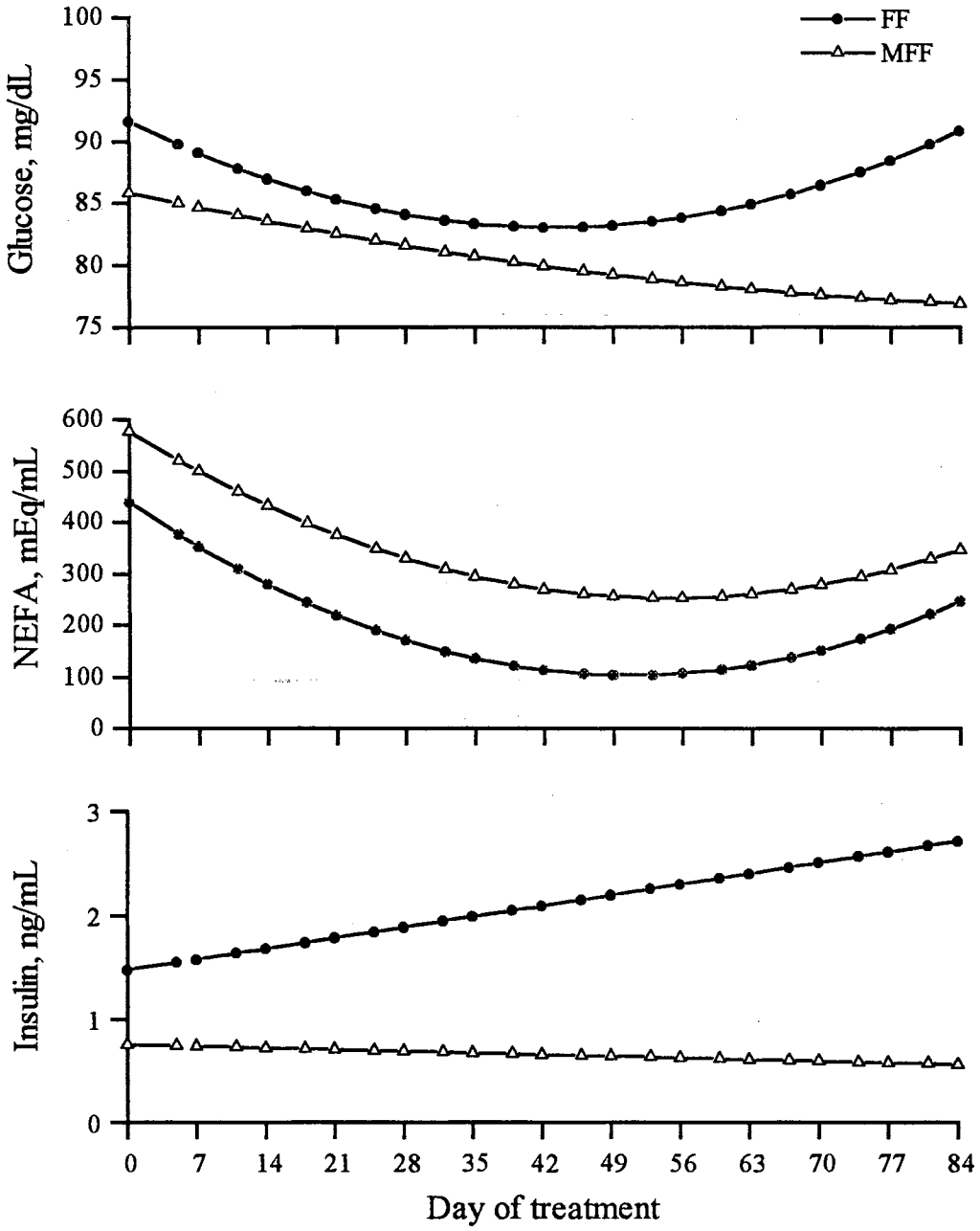

Figure 3. Least squares regressions for concentrations of glucose and nonesterified fatty acids (NEFA) in plasma, and insulin in serum of FF (full fed to gain 1.36 kg/d) and MFF (full fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d) heifers during the first 84 d of treatment.

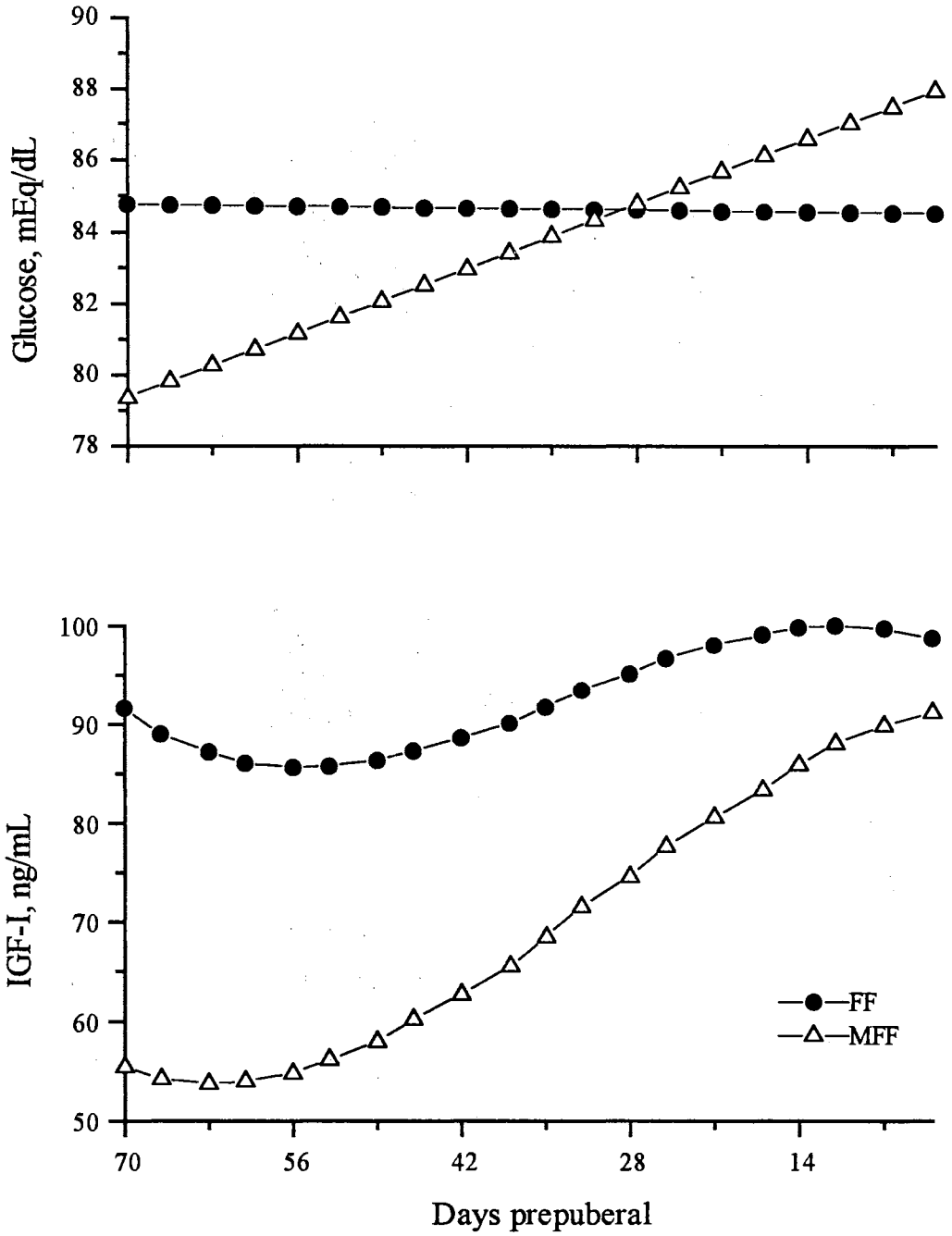

Figure 4. Least squares regressions for concentrations of glucose in plasma and IGF-I in serum of FF (full fed to gain 1.36 kg/d) and MFF (full fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d) heifers during the 70 d prior to puberty.

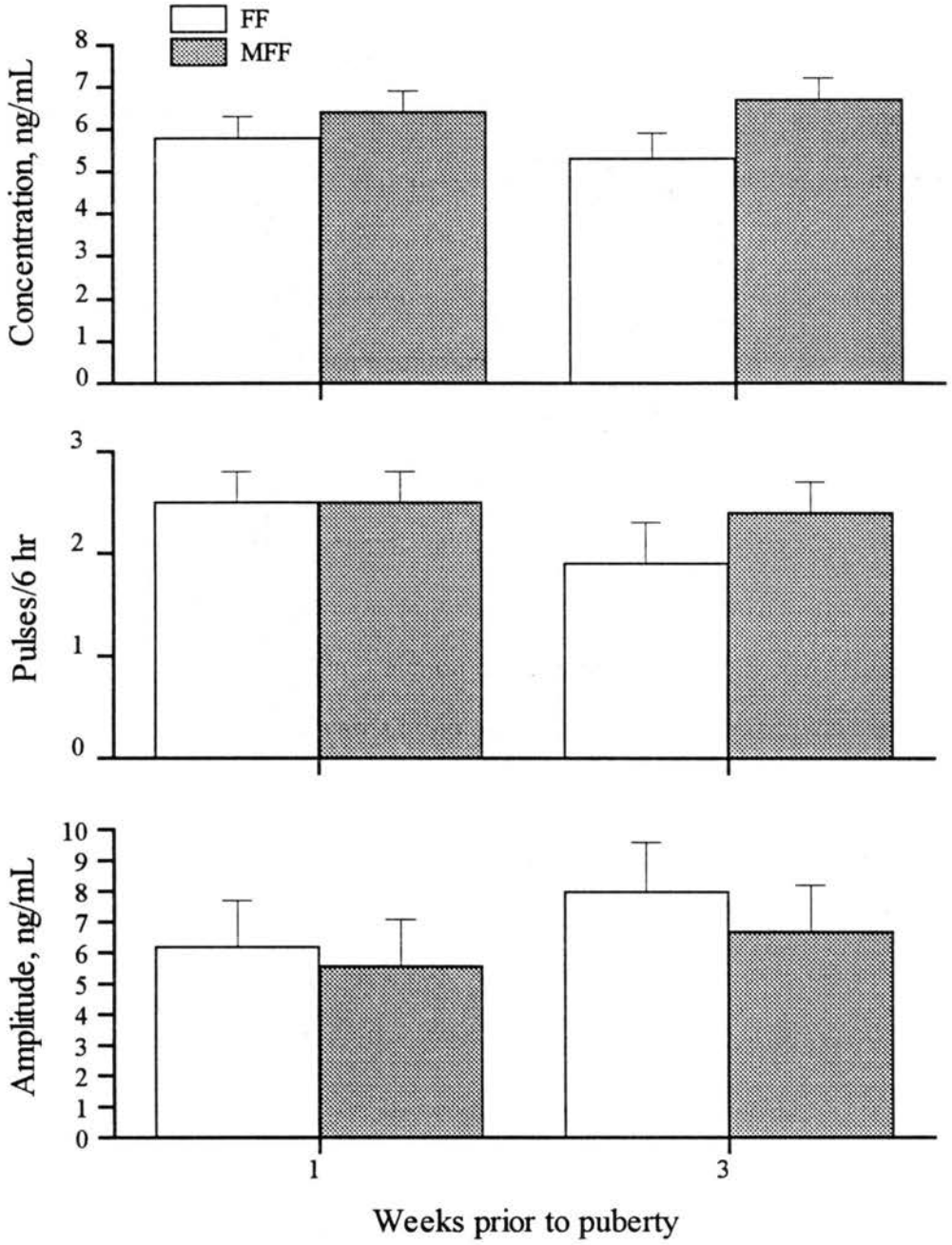

Figure 5. Concentration, pulse frequency, and pulse amplitude of LH of FF (full fed to gain 1.36 kg/d) and MFF (full fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d) heifers at 1 and 3 wk prior to puberty.

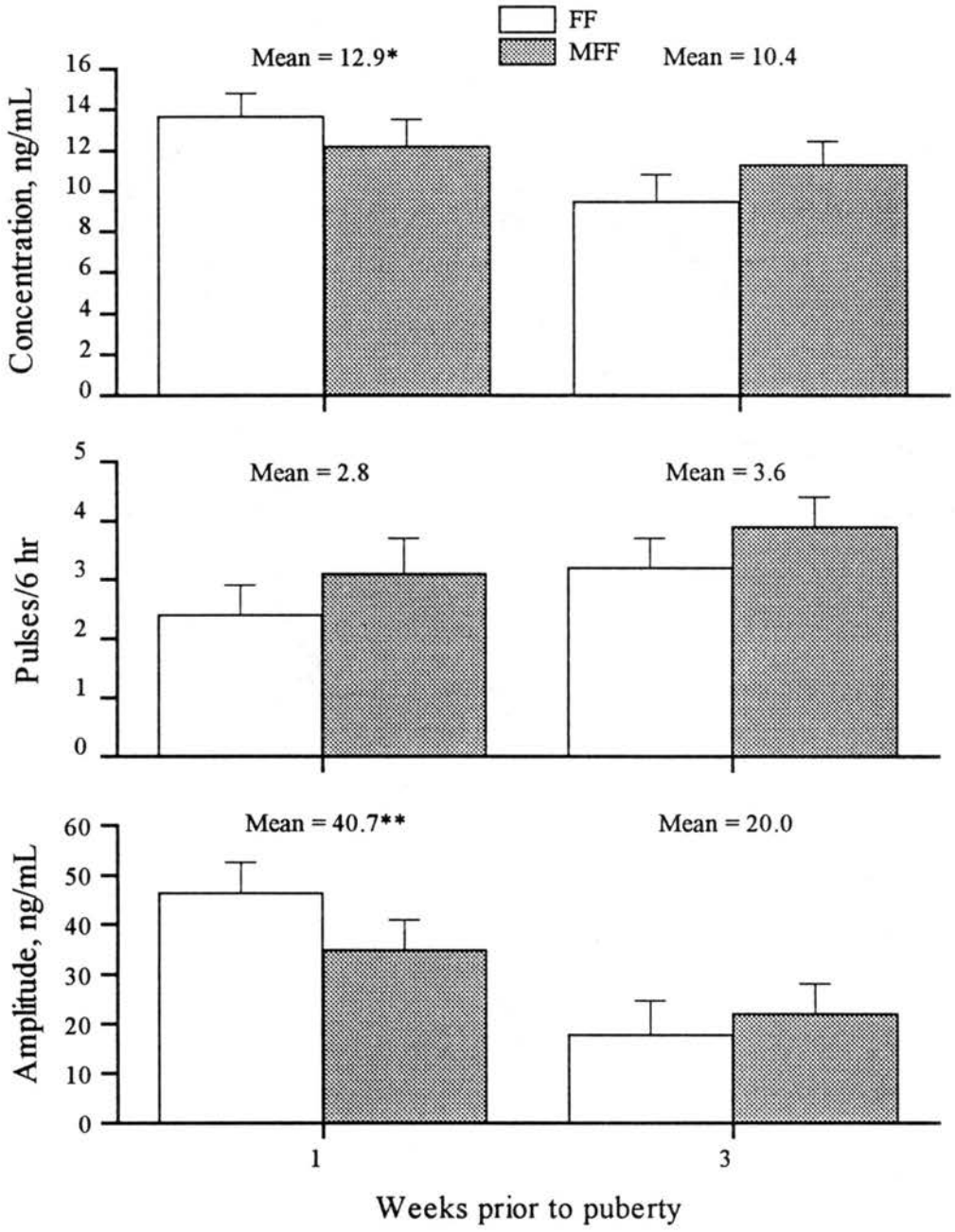

Figure 6. Concentration, pulse frequency, and pulse amplitude of growth hormone (GH) of FF (full fed to gain 1.36 kg/d) and MFF (full fed to gain .23 kg/d for 16 wk, then fed to gain 1.36 kg/d) heifers at 1 and 3 wk prior to puberty (* P<.06; **P<.005).

Chapter VI

Summary and Conclusions

The majority of replacement heifers are bred to calve at two years of age. Consequently, heifers must attain puberty at approximately 12 to 14 mo of age and before the start of the breeding season. Calving heifers at two years of age increases lifetime productivity compared with calving at three years of age, as long as heifers are not over conditioned which results in decreased milk production due to increased fat deposition in the mammary glands. Age at puberty is affected primarily by nutrition and genotype of the animal. Feeding strategies that could increase the percentage of heifers cycling at the beginning of the breeding season could help to improve profitability of the cow-calf operation.

Two experiments were conducted to examine the effects of rate of gain on percentage body fat, age at puberty, and endocrine function in crossbred beef heifers. In the first study, heifers were fed to gain .68 kg/d (moderate), 1.36 kg/d (full), or .23 kg/d for 16 wk then fed to gain 1.36 kg/d (low-high) until attainment of puberty. Weekly blood samples were taken to determine luteal activity as an indication of pubertal status. The influence of treatment on concentrations of growth hormone (GH), Insulin-like growth factor-I (IGF-I), insulin, nonesterified fatty acids (NEFA) and glucose were determined. Heifers were slaughtered within 10 d after puberty to determine carcass composition and percentage body fat.

Increased rate of gain resulted in younger but heavier heifers at puberty. The moderate and low-high rates of gain resulted in heifers of similar body weight and age at

puberty. Heavier weights at puberty for increased nutrient intake heifers was due to increased fat deposition as lean and bone mass were similar for heifers on all treatments. Heifers on increased rates of gain had increased fat on both an absolute and as a percentage of carcass + OM weight. Heifers on increased nutrition had greater concentrations of IGF-I, insulin, and glucose and decreased concentrations of GH and NEFA compared with heifers on reduced nutrient diets. After nutrient intake was increased in low gain heifers, hormone and metabolite concentrations at puberty were similar to those of the heifers that had greater nutrient intake.

Puberty did not occur at a constant percentage body fat which does not support the "critical body fat theory" proposed for mice and humans. However, it does not totally eliminate body fat as a regulator in the initiation of puberty. Body fat reserves are a critical energy source for the attainment of estrous cycles in rodents, humans, and beef cattle. Whether body fat serves a similar function in the prepubertal beef heifer remains to be answered. Prepubertal animals are physiologically different from the mature cycling animals in that they are still rapidly growing in skeletal and muscle mass while concurrently trying to attain enough weight and age to reach reproductive maturity. The present study indicated a strong association of onset of puberty with bone and muscle mass, reflected by similar bone and muscle mass for heifers on all treatments at puberty . Dairy heifers reach puberty at younger ages than beef heifers. Furthermore, dairy cattle deposit increased internal fat compared with beef cattle. Beef cattle selected for muscle attain puberty at older ages and heavier weights compared with beef cattle selected for increased milk production. When selecting for increased milk production in beef cattle, we may be selecting for decreased muscle to bone ratios, and increased internal fat deposition resulting in younger ages at puberty. Beef cattle selected strictly for muscling may reach puberty at older ages because they have increased muscle to bone ratios and decreased internal fat. Whether one of these factors is more critical in attainment of puberty than the other has yet to be determined.

Undernutrition can delay the onset of puberty while increased nutrition can hasten the process. The effects of nutrition are mediated through modification of the hormonal and metabolite milieu. Insulin, glucose, and IGF-I probably all play important roles in stimulating the hypothalamic-pituitary-ovarian axis. Whether GH has a direct role on attainment of puberty is unclear. GH may mediate its effects indirectly through IGF-I, or it may modulate of fat, bone and/or muscle growth. Insulin and glucose probably influence the central nervous system to stimulate a more synchronous and frequent firing of the GnRH neurons which stimulates LH synthesis and release from the pituitary. Pulsatile LH release appears to be the major determinant of onset of puberty. However, that does not preclude that insulin and glucose may have direct effects on the ovary. Insulin may exert part of its effect through IGF-I, which in itself probably has a more direct effect on the ovarian function through its mitogenic activity on granulosa cells and its potentiating effects on gonadotropins by augmenting steroid production. Insulin, glucose, GH, and IGF-I, whether through direct or indirect action, enhance LH release in some manner. These hormones and metabolites probably facilitate LH release as the negative feedback of estradiol on LH secretion diminishes with age.

A second study was conducted to determine the effect of rate of gain on the secretory patterns of LH and GH during the prepubertal period. Heifers were fed to gain 1.36 kg/d, or .23 kg/d for 16 wk then fed to gain 1.36 kg/d until attainment of puberty. Starting on day 29 of treatment, blood serum was obtained every 10 min for 6 h every 14 d until attainment of puberty. Twice weekly samples were also taken to quantify progesterone to assess luteal activity. Concentrations of GH, IGF-I, insulin, glucose and NEFA were also quantified in twice weekly samples. As observed in experiment 1, increased nutrient intake resulted in younger heifers at puberty. However, in contrast to experiment 1, body weights were similar for the two treatment groups. Delayed puberty was probably a result of decreased LH pulse frequency in nutrient restricted heifers. Undernutrition significantly increased GH and NEFA concentrations in plasma. Upon

increased nutrient intake of heifers on reduced nutrient intake, about half of the heifers attained puberty within several weeks, while the remainder of the heifers reached puberty over the next several months. The reason for this response is difficult to determine, but is probably due to genotypic differences in the heifers' ability to reach puberty. Heifers that reached puberty at an older age were similar in weight than fast gain heifers that reached puberty earlier but weighed less than their contemporaries that reached puberty later.

The secretory patterns of LH and GH were not influenced by treatment in the 3 wk prior to puberty. The secretory patterns of LH were similar at 1 wk and 3 wk prior to onset of puberty. This would suggest that when heifers are on similar planes of nutrition, but different ages, the events directly preceding puberty are similar and not influenced by heifer age. The amplitudes of GH pulses were greater 1 wk prior to puberty compared with 3 wk, although pulse frequency was unaffected. The importance of this finding is unclear. Increased GH amplitude may be important in the final follicular growth and development prior to ovulation. Increased pulse amplitude of GH occurs in late prepuberal humans and its release is augmented by increased gonadal steroids. Humans are also insulin resistant during the prepubertal period and it is speculated that increased GH concentrations are responsible for this resistance. Whether a similar situation occurs in beef cattle remains to be determined.

The initiation of puberty in the bovine is a very complex physiological process with no single "magic factor" that signals when puberty is to occur. Nutrition, genotype, and age all have major roles in when puberty occurs. Age and weight at puberty can be modified either positively and/or negatively by nutrient intake and genotype of the heifer. Hence, a producer must develop a selection program that results in production of a genotype that complements the management system, available feed resources, and environment in which that animal is reared and utilized.

Additional research must be conducted to evaluate the role of lean and bone mass in attainment of puberty. A comparison of carcass composition and endocrine events

necessary for the initiation of puberty in dairy breeds with breeds selected for muscle growth may be a good place to begin. Furthermore, follicular growth in the prepuberal heifer must be characterized and factors (e.g., nutrition, hormones, metabolites) that influence follicular growth and development must be investigated. Evaluation of alternative feeding programs, similar to the low to high program described in this thesis, needs to be conducted to determine the time and duration of increased nutrient intake necessary to enhance the onset of the pubertal estrous cycle.

APPENDIXES

Table 1. Equations used to adjust physically separated carcass components for moisture loss in the cooler with the assumption that moisture loss is the same for all pools.

1) Cut weight (CWT) of side after physical disection into respective pools.

CWT = LEAN + SFT + SEAM + SC + BONE

SFT - soft tissue, inseparable lean and fat
SEAM - intermuscular fat
SC - subcutaneous fat

2) Calculate percent shrink (PS) from hot carcass side weight (HCSW) to CWT.

PS = 100 - ((CWT/HCSW)*100)

3) Calculate the amount of moisture in each pools' cut weight (PMCW**x**; **x** = L, SFT, SEAM, SC) using proximate analysis value for moisture for each respective pool.

PMCW**x** = (pool CWT**x** * percent moisture in pool **x**)/100

4) Calculate the total carcass moisture side weight (CMSW).

CMSW = PMCWL + PMCWSM + PMCWSC + PMCWSFT

5) Calculate the percentage pool moisture (PPM**x**) as a percentage of CMSW.

PPM**x** = (PMCW**x** / CMSW) * 100

6) Calculate the total moisture pool weight (TMPW**x**) as a proportion of HCSW

TMPW**x** = ((PPM**x***(HCSW*PS))/100

7) Calculate the total carcass weight moisture (TCWM), the sum of TMPW**x** for all pools. Should be equal to HCSW-CWT.

TCWM = TMPWL + TMPSM + TMPSC +TMP SFT

8) Calculate adjusted pool weight (APW**x**) to account for moisture lost for each pool.

APW**x** = pool CWT**x** + TMPW**x**

9) Calculate adjusted carcass weight (ACW) with pool moisture lost added back.

ACW = APWL + APWSC + APWSM + APWSFT

10) Calculate a final adjusted pool weight (FAPW**x**) as a percent of HCW

Table 1. (cont)

FAPW**x** = (APW**x**/ACW) * HCW

11) Calculate final adjusted HCW (FAHCW), check against actual HCW

FAHCW = FAPWL + FAPWSC + FAPWSM +FAPWSFT + BONE
KPH + UDDER

KPH - kidney, pelvic, heart fat physically separated from carcass
UDDER - physically separated udder

x - indicates that calculation is made for all pools LEAN, SC, SEAM, SFT.

Table 2. Equations used to calculate carcass composition.

1. Adjusted hot body wt (ADJHBW) - accounting for shrink.

ADJHBW = ADJLEAN + ADJSFT + ADJSC + ADJSEAM + BONE+OM + KPH + UDDER

All variables are adjusted for moisture loss as calculated in appendix Table 1.

ADJLEAN - lean
ADJSFT - soft tissue = inseparable fat and lean
ADJSC - subcutaneous fat
ADJSEAM - intermuscular fat

These variables were not adjusted for moisture loss

OM - omental mesenteric fat dissected from viscera
KPH - kidney, pelvic, heart dissected from carcass
UDDER - udder dissected from carcass

2. Total separable body fat (SEPFAT).

SEPFAT = OM + KPH + ADJSC + ADJSEAM + UDDER

3. Percent separable pool fat (PSEPFATx), pool fat wt as a percentage of SEPFAT.

PSEPFATx = (x/SEPFAT) (*x = values used to derive ADJHBW)*

4. Percent separable body fat (PSEPFAT), SEPFAT as a percentage of ADJHBW.

PSEPFAT = (SEPFAT/ADJHBW)

5. Adjusted hot carcass wt (ADJHCW), adjusted for shrink with out UDDER, KPH and OM.

ADJHCW = ADJLEAN + ADJSFT + ADJSC + ADJSEAM + BONE

6. Percent x hot carcass wt (PxHCW), pool wt as a percentage of ADJHCW.

PxHCWT = (x/ADJHCW) (x is calculated for ADJLEAN, ADJSFT, ADJSC, ADJSEAM)

7. Amount, on a wt basis, that carcass pool x (Cx) is of ADJHCW.

Cx = (Px * ADJHCW) (x calculated for ADJLEAN, ADJSFT, ADJSC, ADJSEAM, BONE)

Table 2. (cont.)

Steps 8 through 12 are used to calculate the amount of chemical lipid, protein, moisture and ash in each respective pool and for percentage of lipid, protein, moisture and ash that comprise empty body wt (See number 8). Lipid will be used as an example in calculations. Protein, moisture and ash can be inserted in for lipid and each respective value derived.

8. BASEWT was used as empty body weight in calculations.

   BASEWT = ADJLEAN + ADJSFT + ADJSC + ADJSEAM + BONE+OM + KPH

9. Amount of body lipid (BLIPID) in each pool **x**, **x**.is calculated for OM, KPH. SC, SEAM, SFT, LEAN

   BLIPID**x** = (C**x** * percent lipid in **x** from proximate analysis)/100

10. Total body lipid (TBLIPID) on a wt basis.

    TBLIPID = OML + KPHL+ SCL + SEAML + LEAN-LIPID + SFTL

11. Percent carcass lipid (PBLIPID), BLIPID as a percentrage of BASEWT.

    PBLIPID = (BLIPID/BASEWT)*100

12. Percent pool lipid (PPLIPID), lipid in each pool as a percentage of BLIPID (**x** is calculated for OML, KPHL, SEAML, SCL, TLL (LEAN-LIPID + SFTL).

    PPLIPID**x** = (**x**/BLIPID)

13. Amount of lean in the carcass devoid of fat - Fat Free LEAN (FFL)..
    FFL = (CLEANWT + CSFT) - (TLL)

14. Percent FFL (PFFL), lean free of fat as a percent of BASEWT
    PFFL = FFL/BASEWT

VITA 2

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Candidate for the Degree of

Doctor of Philosophy

Thesis: EFFECT OF GROWTH RATE ON CARCASS COMPOSITION, HORMONES, AND METABOLITES AT PUBERTY IN BEEF HEIFERS

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