

Nutritional Regulation of Precocious Puberty in Beef Heifers

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Introduction

Age at puberty has an important impact on the productive, reproductive and economic efficiency of female cattle. The occurrence of this physiological event implies an adequate growth rate and development of the animal that supports the endocrinological mechanisms that lead to sexual maturity. Age at puberty and first conception will influence lifetime productivity of cattle, reflected in the number of calves (Bagley, 1993). In spite of the importance of this physiological endpoint, the metabolic signals that activate the endocrine mechanisms that trigger puberty are still unknown. Puberty has been defined as the manifestation of reproductive competence, with the ability to ovulate an oocyte accompanied by the presentation of estrous behavior and the development and maintenance of a functional corpus luteum (Kinder et al, 1987). In cattle as well as other species, maturation of the reproductive axis occurs in a gradual fashion and is associated with changes in body composition and in the somatotrophic axis that regulates metabolic status of the animal. While changes in the reproductive mechanisms that lead to puberty have been well established, metabolic signals that modulate the processes of sexual maturation are not well known.

Post-weaning growth and age at puberty

The age at which cattle reach puberty can be associated with genetic and nutritional factors. Weight gain after weaning is a major variable that influences age and weight at puberty (for review, see Patterson et al., 1992). The influence of different diets during the post-weaning period on age of puberty has been extensively investigated. Growth rate between traditional weaning (6 to 8 months of age) and puberty was negatively associated with age at puberty (Smith et al. 1976). Target weights at breeding have been developed to reflect these relationships and suggest that heifers should achieve a specific weight by the beginning of the breeding season in order to achieve high pregnancy rates (Lamond, 1970, Wiltbank, 1969). Early reports established that heifers should be fed to achieve 60 to 65% of their projected mature weight before their first breeding season to ensure high pregnancy rates (Lamond 1970; Taylor and Fitzhugh, 1971). Ferrell (1982) evaluated the effect of feeding to achieve different average daily gain (ADG; low 0.4 kg/d, medium 0.6 kg/d and high 0.8 kg/d) in different beef cattle breeds. It was demonstrated that both breed and post-weaning ADG were significant sources of variation for both age and weight at puberty. Across breeds, heifers that were fed to achieve the lowest ADG tended to be older and lighter

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at puberty. More recently, Thallman et al, (1999) investigated the effect of breed on weight at puberty in different breeds in the USA. Across breeds, age and weight at puberty were approximately 357 d of age and 320 kg body weight (BW) although differences between breeds in both variables were noted. Postweaning nutrition plays a critical role in determining age at puberty in heifers.

Influence of preweaning growth rate on age at puberty

While less emphasis has been placed on the influence of preweaning growth rate on puberty, it has been demonstrated that this is also a significant source of variation. Wiltbank et al. (1966) demonstrated that preweaning BW gain influenced age at puberty in heifers more consistently than postweaning BW gain, with increased preweaning BW gain resulting in earlier onset of puberty. Others have also observed a reduction in age at puberty with increased weaning weight (Arije and Wiltbank, 1971; Greer et al, 1983). Recently, Roberts et al (2007) found that age at puberty is affected more by growth rate pre-weaning and the growth rate at early phases after weaning (~8 months of age) than growth in the period just before breeding.

Altered preweaning nutrition and precocious puberty in heifers

The influence of preweaning growth rate on age at puberty in heifers managed in a typical manner (i.e. weaned at approximately 7 months of age) has been well established. However, several years ago we observed that a significant proportion of heifers had attained puberty by 7 to 8 months of age when they were weaned early and fed a high concentrate diet beginning at 3 to 4 months of age (Day and Anderson, 1998). This casual observation led to the hypothesis that the reproductive axis could be precociously activated by feeding of a high concentrate diet beginning at approximately 3 months of age in beef heifers. We initiated a series of studies designed to assess the impact of diet, beginning after weaning at 3 to 4 months of age, on induction of precocious puberty (before 300 d of age), and the alterations in the reproductive axis that led to this premature event.

The primary objective of the first study (Gasser et al, 2006a; EXPT 1) was to test the hypothesis stated in the previous paragraph and to verify previous observations of precocious puberty. To accomplish this, eighteen heifers were weaned at 73 ± 3 d of age (early weaned; EW) and 115 ± 3 kg of BW. After a 20 d receiving period, heifers were assigned to be fed one of two diets. The control (C) diet (Table 1) consisted predominantly of roughage, and was formulated to achieve BW gain (0.75 kg/d) similar to those that would have been achieved by heifer calves had they remained with their dams on pasture. This diet was formulated to be fed at approximately 2.5% of BW. The high concentrate (H) diet (Table 1) was similar to that fed previously when induction of precocious puberty was observed (Day and Anderson, 1998) and was formulated to support target BW gain of 1.5 kg/d when fed at the rate of 2.5% of BW. Heifers were individually fed, and intakes were adjusted to attempt to achieve the target ADG. Heifers in the EWH treatment were fed the H diet throughout the experiment whereas those in the EWC treatment received the C diet from 99 to 286 d of age, and the H diet

thereafter (Figure 1). Realized ADG during the experimental phase of the study (99 to 286 d of age) was 1.27 ± 0.05 kg/d for the EWH and 0.85 ± 0.05 kg/d for the EWC treatment. Eight of nine heifers in the EWH treatment experienced precocious puberty whereas none of the heifers in the EWC treatment reached puberty before 300 d of age (Figure 2, Table 2). Heifers in the EWH treatment reached puberty at a lesser BW than in the EWC treatment (Figure 3).

In the subsequent 3 experiments (Gasser et al., 2006b, EXPT 2; 2006c, EXPT 3; 2006d, EXPT 4) similar differences in age at puberty (Table 2) were observed between heifers that received the EWC and EWH treatments described for EXPT 1, although the proportion of heifers that experienced precocious puberty varied somewhat between experiments. An unexpected observation was that in 56% of heifers in the EWC treatment in EXPT 2 experienced precocious puberty. Pasture feed resources preceding the time of early weaning were excellent during the year in which this experiment was conducted and the heifers used in the experiment were approximately 35 kg heavier at approximately 100 d of age than heifers used in the other 3 studies. We speculate that the nutritional plane provided to these heifer calves and their dams, during their first months of life and before early weaning was a potential source of stimulation for the occurrence of precocious puberty in some heifers. Indeed, spontaneous precocious puberty has been verified in a significant proportion of heifers weaned at 6 to 8 months of age (Wehrman et al., 1996). As expected, ADG was greater in the EWH than EWC treatment in all experiments (Figure 4). Although BW at puberty was less in the EWH than EWC treatment in EXPT 1, this relationship was not detected in subsequent studies (Figure 4).

In EXPT 2 (Gasser et al., 2006b) a treatment that consisted of heifers that remained with their dams until weaning at 208 ± 3 d of age and 276 ± 7 kg BW (normal weaning; NW) and were then fed the C diet postweaning (NWC treatment) was included to determine if age at weaning was a significant source of variation. In this study, ADG of heifers in EWC and NWC treatments was similar (1.03 ± 0.05 kg/d and 1.07 ± 0.03 kg/d respectively) and age at puberty and incidence of precocious puberty did not differ between the EWC (308 ± 26 d of age; 56%) and the NWC (330 ± 25 d of age; 50%) treatments. Thus, in heifers growing at a typical and similar rate, large differences in timing of weaning and compositional differences in the diet (EWC – control diet, NWC – milk and pasture followed by the C diet) did not appear to influence the rate of sexual maturation.

In EXPT 4 (Gasser et al., 2006d) two treatments, in addition to the EWC and EWH were included to test the influence of the timing of feeding of the H diet on occurrence of precocious puberty. Diets were initiated at 126 d of age and the experiment was divided into two feeding phases. Phase 1 was from 126 to 196 d of age and Phase 2 was from 196 d of age until the end of the experiment. Heifers in the EWC and EWH treatments received their indicated diets throughout both phases. Other heifers received the H diet for Phase 1 and the C diet for Phase 2 (EWH-C) while heifers in the fourth treatment received the C diet for Phase 1 and the H diet for Phase 2 (EWC-H). Body weight changes over the experiment are illustrated in Figure 5.

Incidence of precocious puberty ranged from 67% in the EWH to 20% in the EWC treatment (Table 3). Age at puberty for the EWH and EWH-C treatments did not differ (Table 3) and was earlier than for the EWC treatment. Age at puberty in the EWC-H treatment was intermediate to and not different from the other treatments. Across treatments, heifers fed the H diet during Phase 1 attained puberty earlier than heifers fed the C diet during Phase 1. It was concluded that early weaning and increased dietary energy intake through feeding the H diet from approximately 4 to 7 months of age in heifers advanced age at puberty; regardless of the diet fed after 7 months of age.

Based upon the findings of these 4 experiments, we accept our hypothesis that feeding a high energy, concentrate-based diet to heifers that are weaned at 3 to 4 months of age will induce precocious puberty in a majority of heifers. Precocious puberty occurred sporadically in heifers on the control diet across experiments and the incidence of this spontaneous precocious puberty appeared to be related to the nutritional status of the heifer calves up to the time of early weaning. Consistent with this speculation, feeding of the high energy diet from early weaning to approximately 200 d of age had a similar impact on age at puberty to feeding this diet through 11 to 12 months of age. Hence, the signal that induces precocious activation of the reproductive axis may well occur early in life of heifers and in some cases, before the age at which early weaning was performed in the experiments described above. This series of experiments suggest the existence of a critical time in which nutritional management can induce the activation of the reproductive axis and consequently the initiation of the reproductive cycles. The specific dietary or metabolic signals that promote this response have not yet been identified and this question is the focus of an ongoing experiment.

In an ongoing experiment (Maquivar et al., unpublished), the objective was to test qualitative influences of feeding a high energy diet to early weaned heifers on age and BW at puberty and to assess the impact of these diets on metabolic and reproductive variables. Thirty six animals were weaned at an average of 80 d of age and 120 kg BW. After a 21 d receiving period, animals were transitioned onto one of three experimental diets (Table 4). Experimental diets were either a High starch diet (HIGH-S) that was similar to the H diet fed in previous experiments, a low starch diet (LOW-S) in which a large proportion diet was composed of feeds that are high in energy and low in starch (DDGS and soy hulls) and a roughage-based control diet (CONT). Target ADG for the HIGH-S and LOW-S diets was 1.50 kg/d, and was 0.75 kg/d for the CONT diet. Diets were designed to be fed at approximately 2.5% of BW. Amount of intake was adjusted to achieve the desired ADG within treatment. No heifers in the CONT treatment exhibited precocious puberty, whereas a similar proportion of heifers in the LOW-S (42%) and HIGH-S (60%) treatments had precocious puberty (Figure 6). Age at puberty was greater in the CONT (377 ± 26) than in the LOW-S (269 ± 69 d) and HIGH-S (262 ± 56 d) treatments. Average daily gain and BW throughout the experiment did not differ between the LOW-S and HIGH-S treatment, and both were greater than ADG and BW in the CONT treatment (Figure 7). These results suggest that the quantitative effect of the diet (amount of energy provided to heifers) appears to be more important than the qualitative aspects of the two high energy diets used in this experiment.

Endocrinology of precocious puberty

From an endocrine standpoint, the occurrence of puberty is the result of a decrease in estradiol negative feedback on secretion of luteinizing hormone (LH), which leads to an increase in the secretion of LH in response to an increment of gonadotropin-releasing hormone (GnRH) release from the hypothalamus (Rodriguez and Wise, 1989), and final growth and maturation of ovarian follicles leading to a the first ovulation (Figure 8; Day et al., 1984; Day et al., 1986, Kinder et al., 1987). The 40 to 60 d period before first ovulation when a majority of these dynamic changes occur is referred to as the peripubertal period. The timing of the peripubertal period in heifers varies with age at puberty, but appears to be initiated between 10 to 12 months of age in most well managed *Bos taurus* heifers. We have proposed that the period from birth to puberty in heifers can be divided into 4 periods, beginning with an infantile period (birth to 2 months of age), developmental period (2 to 6 months of age) a static phase (6 to 10 months of age) and the peripubertal period (Day and Anderson, 1998; Figure 9). As described earlier, feeding a high energy diet during the developmental phase hastens puberty. Dynamic changes occur in the reproductive axis during the developmental phase (for review, see Day and Anderson, 1998). Increased GnRH secretion from the hypothalamus leads to increased LH secretion. Rising gonadotropin secretion stimulates follicular growth and results in increased peripheral concentrations of estradiol. The presence of estradiol negative feedback on LH secretion is verified by the decline in LH secretion at 3 to 6 mo of age in response to increasing production of estradiol by ovarian follicles. Following this period of development, secretion of LH stabilizes at a relatively low level by 6 months of age, signifying the initiation of the static phase in heifers. During the static phase in heifers, the hypothalamo-pituitary axis is fully competent, but is unable to generate follicular phase levels of LH secretion due to restraint by estradiol negative feedback. The peripubertal decline in estradiol negative feedback leads to increased LH secretion, hence estradiol secretion, and the initial LH surge and ovulation.

Clearly, when heifers are weaned early and fed a high energy diet, the timing of the events that culminate in puberty is altered. We hypothesized that the key changes that occur between 10 and 12 months of age (peripubertal period) in traditionally managed heifers were instead occurring between 6 and 8 months in heifers that were weaned early and fed a high energy diet. We investigated changes in various components of the reproductive axis in three of the previously described experiments. In the first experiment (Gasser et al., 2006a, EXPT 1) we determined that secretion of LH was greater in the EWH treatment, beginning at 190 d of age up to the time of puberty. In the second experiment (Gasser et al., 2006b, EXPT 2) follicular growth was enhanced in the EWH treatment by 196 d of age, as evidenced by a greater diameter of the dominant ovarian follicle. In the same study, it was demonstrated that animals which experience precocious puberty had a larger dominant ovarian follicle, a longer, follicular wave, and greater estradiol concentrations than heifers that reached puberty after 300 d of age. These differences were evident as early as 120 d of age between heifers that experienced precocious puberty and those that did not. Finally, in EXPT 3 (Gasser et al., 2006c) we demonstrated that timing of the peripubertal decline in

estradiol negative feedback on LH secretion was hastened in the EWH treatment. We have concluded that when heifers are weaned early and fed a high energy diet, changes in LH secretion, follicular development and estradiol negative feedback are similar to those that occur in traditionally managed heifers, but are initiated around 6 months of age. It appears that with this dietary manipulation, heifers forego the static phase described in Figure 9, moving directly from the developmental phase to the peripubertal period.

The somatotropic axis and precocious activation of the reproductive axis

We know that feeding of a high energy diet precociously activates the reproductive axis. It appears that this nutritional stimulus is most profound between 3 and 6 months of age and is not dependent upon a high content of starch in the diet. Radcliff et al, (1997; 2004) evaluated the effect of two different diets from 120 d of age until puberty in Holstein heifers. The diets fed (High diet containing 75% grain and 25% haylage and Control diet with 10% grain and 90% haylage) were similar to the H and C diets we have used in most of our experiments. Heifers fed the High diet showed puberty at 266 ± 9 d of age while puberty occurred at 313 ± 10 d of age when fed the Control diet (Radcliff et al., 1997). Similar to our findings in beef heifers (Gasser et al., 2006a; 2006b, 2006c, and 2006d) feeding a high concentrate, high energy diet early in life induced precocious puberty in dairy heifers. In the latter report (Radcliff et al., 2004), the authors presented the response of the somatotropic axis of these heifers to these diets. Heifers fed the High diet had greater peripheral insulin like growth factor-1 (IGF-1) concentrations, and lesser IGF binding protein-2 (IGFBP-2) concentrations than those fed the Control diet. Concentrations of growth hormone (GH) were unaffected by diet. However, the liver concentrations of growth hormone receptor (GHR) 1A and IGF-1 mRNA were increased, and concentration of IGFBP-2 mRNA was decreased in heifers fed the High diet; explaining the effect of diet on peripheral concentrations of IGF-1 and IGFBP-2. Since IGFBP-2 has been suggested to inhibit IGF-1 activity, the relative influence of the High vs. Control diet on IGF-1 may be even greater than that reflected by concentrations of IGF-1 in circulation. Similar responses in IGF-1 were noted in the previously described experiment (Maquivar et al., unpublished) in which heifers were fed a control diet (CONT), or one of two high energy diets that had a high (HIGH-S) or low (LOW-S) starch content (Table 4). Concentrations of IGF-1 in the heifers fed the HIGH-S and LOW-S diets were greater than in heifers fed the CONT diet. Between the HIGH-S and LOW-S, IGF-1 concentrations did not differ between treatments at a majority of sampling periods. In a couple of instances, IGF-1 was greater in the HIGH-S than LOW-S treatment. Of particular relevance to the putative role of IGF-1 to induction of precocious puberty, concentrations of IGF-1 were greater in those heifers that experienced precocious puberty than in those that did not; regardless of the formulation of the high energy diet (Figure 10). These findings support the possibility that IGF-1 may trigger the cascade of events that lead to precocious puberty.

Others have investigated the influence of IGF-1 on puberty in heifers. Yelich et al (1996) working with beef heifers from nine months of age until the onset of puberty, fed heifers either a high gain diet, or a low gain diet for 16 weeks followed by the high gain

diet). Heifers that received the high gain diet throughout reached puberty at younger ages than with the low to high gain diet (369 ± 16 vs. 460 ± 17 d of age, respectively). Simpson et al, (1991) evaluated the effect of serial active immunization against growth hormone releasing factor (GRF) in 6 month-old beef heifers. As anticipated, concentrations of GH were decreased by immunization. Accordingly, immunized heifers had much lower concentrations of IGF-1 than non-immunized heifers at approximately 8 months of age (41 ± 5 and 112 ± 4 ng/ml, respectively). At 12.9 months of age 81% of the non-immunized heifers reached puberty, whereas only the 40% of immunized heifers were pubertal by this age. The impact of GRF immunization beginning at 104 d of age on a variety of reproductive characteristics was investigated by Schoppee et al, (1996). Decreased IGF-1 concentrations were detected by 169 d of age in the immunized heifers. At 6 months of age, serum estradiol concentrations and intrafollicular concentrations of IGF-I were lower in immunized than non-immunized heifers. By 393 d of age, 32% of immunized heifers had reached puberty whereas 71% of non-immunized heifers were pubertal by this time. These authors suggested that the deficiency in IGF-1 induced by immunization against GRF impairs the ability of ovarian follicles to synthesize estradiol and thereby, delays puberty.

Across reports in which prepubertal concentrations of IGF-1 have been evaluated, age at puberty has been demonstrated to be negatively associated with concentrations of IGF-1. Schoppee et al., 1996 reported that when lower concentrations of IGF-1 were experimentally induced beginning at 3 months of age, follicular development at 6 months of age was impaired, and puberty was delayed. Conversely, we (Gasser et al., 2006b) have demonstrated that feeding of a high energy diet that would increase IGF-1 resulted in enhanced follicular development and precocious puberty. Radcliff et al. (1997; 2004) also demonstrated that precocious puberty was associated with increased concentrations of IGF-1 and decreased concentrations of IGFBP-2. Finally, we have recently demonstrated in heifers fed two high energy diets that differed markedly in composition, those that experienced precocious puberty had greater concentrations of IGF-1; regardless of diet (Figure 10).

Ovarian follicles are a likely target for IGF-1 in prepubertal heifers as both IGF-1 and IGFBP are involved in the regulation of follicular growth and development (Webb et al, 1999; Renaville et al, 2002). In the ovary, IGF-1 increases proliferation and differentiation of granulosa cells and promotes the synthesis of steroid hormones through stimulating the aromatase enzymatic system (for review, see Zulu et al, 2002).

Our working hypothesis regarding the mechanism that trigger precocious puberty when heifers are fed a high energy diet beginning at 3 months of age is that IGF-1 acts at the level of ovarian follicles to induce increased growth and estradiol production by follicles which initiates a cascade of endocrine events that leads to precocious puberty. In the first experiment to test this hypothesis, the impact of feeding a high energy diet to early weaned heifers for approximately 50 d on follicular growth, estradiol production and other aspects of follicular function will be evaluated.

Conclusions

Nutritive signals responsible for precocious activation of the reproductive axis are most important between 3 to 6 months of age. Recent data regarding qualitative and quantitative aspects of diets fed beginning at 3 months of age indicate that diets that differ widely in composition can induce precocious puberty. The initial response to feeding of a high energy diet at this age appears to occur in ovarian follicles. It is proposed that the increase in IGF-1 and/or the decrease in IGFBP-2 that results from feeding a high energy diet to 3 month old heifers stimulate granulosa cells of ovarian follicles to secrete more estradiol. Estradiol in turn, will act at the hypothalamic level to accelerate the decline in estradiol negative feedback, resulting in an increase in GnRH release that will be reflected in an increase in LH secretion. The increase in secretion of LH will further stimulate follicular maturation and progress to the point that sufficient concentrations of estradiol are present to cause the estradiol-induced LH surge and precocious puberty.

Table 1. Experimental diets fed to heifers^a

Item	Diet ^b	
	H	C
Ingredient, % as-fed		
Whole shelled corn	60	30
13% CP alfalfa pellets	10	30
Pelleted soybean hulls	10	30
Supplement	20	10
Chemical analyses		
CP, %	14.1	14.1
NEm, Mcal/kg	2.02	1.70
NEg, Mcal/kg	1.37	1.09

^aDiets used in Gasser et al., 2006a, 2006b, 2006c and 2006d.

^bH = high-concentrate diet; C = control diet.

Table 2. The percentage of heifers that experienced precocious puberty and age at puberty^a

Experiment	n	Early weaning, high concentrate diet (EWH)		Early weaning, control diet (EWC)	
		% Precocious puberty	Age at Puberty (d)	% Precocious puberty	Age at Puberty (d)
EXPT 1	18	89 (8/9)	262 ± 10	0 (0/9)	368 ± 10
EXPT 2	18	100 (9/9)	252 ± 9	56 (5/9)	308 ± 26
EXPT 3	10	80 (4/5)	275 ± 30	0 (0/5)	385 ± 14
EXPT 4	30	67 (10/15)	271 ± 17	20 (3/15)	331 ± 11

^aData from Gasser et al, 2006a, EXPT 1; 2006b, EXPT 2; 2006c, EXPT 3; and 2006d, EXPT 4.

Table 3. The percentage of heifers that experienced precocious puberty and age at puberty^a

Treatment	n	Reproductive Variable	
		% Precocious puberty	Age at Puberty (d)
EWH	15	67 (10/15)	271 ± 17
EWH-C	15	47 (7/15)	283 ± 17
EWC-H	15	47 (7/15)	304 ± 13
EWC	15	20 (3/15)	331 ± 11

^aHeifers were weaned early and fed a high concentrate diet (H) throughout the experiment (EWH), the H diet during Phase 1 and the control diet (C) during Phase 2 (EWH-C), the C diet during Phase 1 and the H diet during Phase 2 (EWC-H) or the C diet throughout the experiment (EWC). Phase 1 was from 126 to 196 d of age and Phase 2 was from 196 d of age to the end of the experiment (Gasser et al., 2006d).

Table 4. Composition of experimental diets fed to heifers (% as fed).

Item	Diets ^a		
	HIGH-S	LOW-S	CONT
Whole shelled corn	50.00	-	-
Orchard grass hay	30.00	23.00	70.00
DDGS		37.00	-
Soybean hulls	-	20.00	-
Supplement	20.00	20.00	30.00
Chemical analysis			
CP, %	15.99	16.14	12.85
Starch,%	46.40	14.48	19.03
Fat,%	4.03	6.43	3.73
NEm, Mcal/kg	1.83	1.84	1.40
NEg, Mcal/kg	1.20	1.20	0.81

^aHIGH-S=High starch diet, LOW-S=Low starch diet, CONT=Control diet.

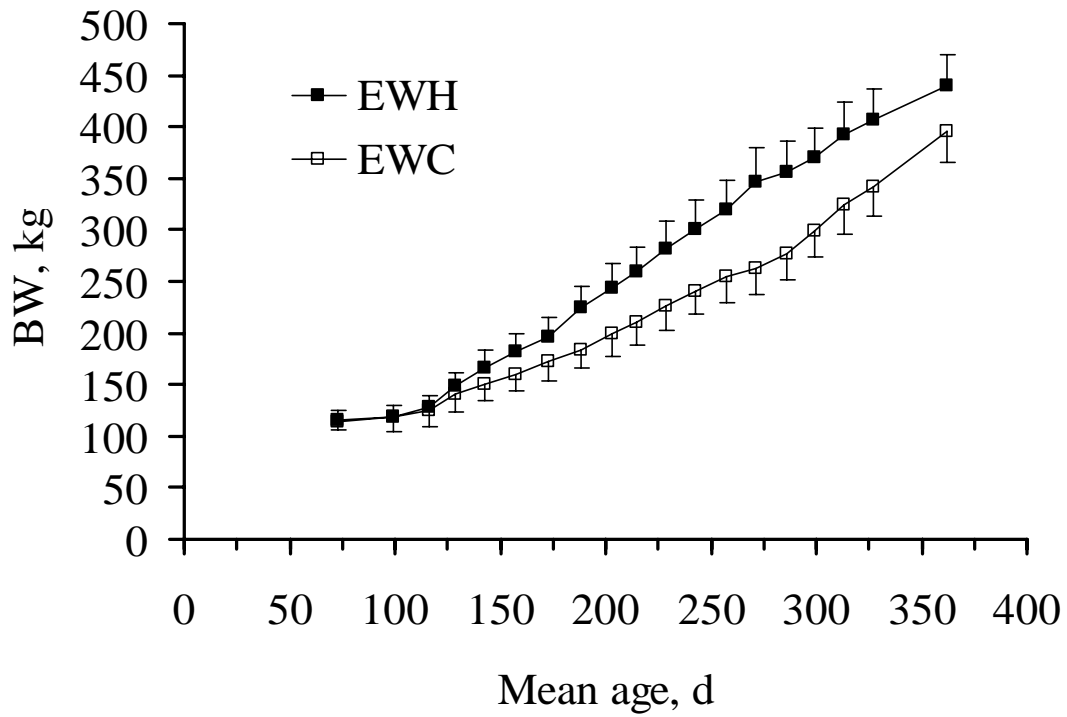


Figure 1. Body weight gain in heifers that were weaned early and fed a high concentrate diet (EWH) or control diet (EWC). Adapted from Gasser et al.,2006a, *Journal of Animal Science*. 84:2035- 41.

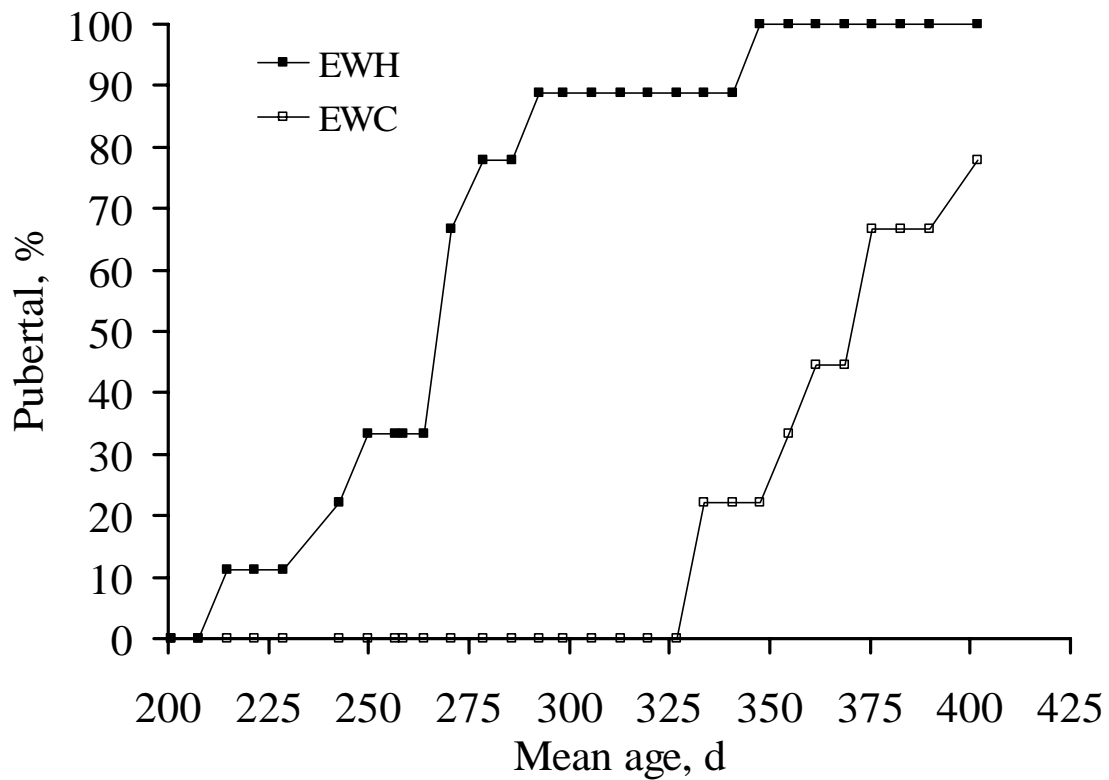


Figure 2. Cumulative percentage of heifers that attained puberty when weaned early and fed a high concentrate diet (EWH) or control diet (EWC). Adapted from Gasser et al., 2006a, *Journal of Animal Science*. 84:2035- 41.

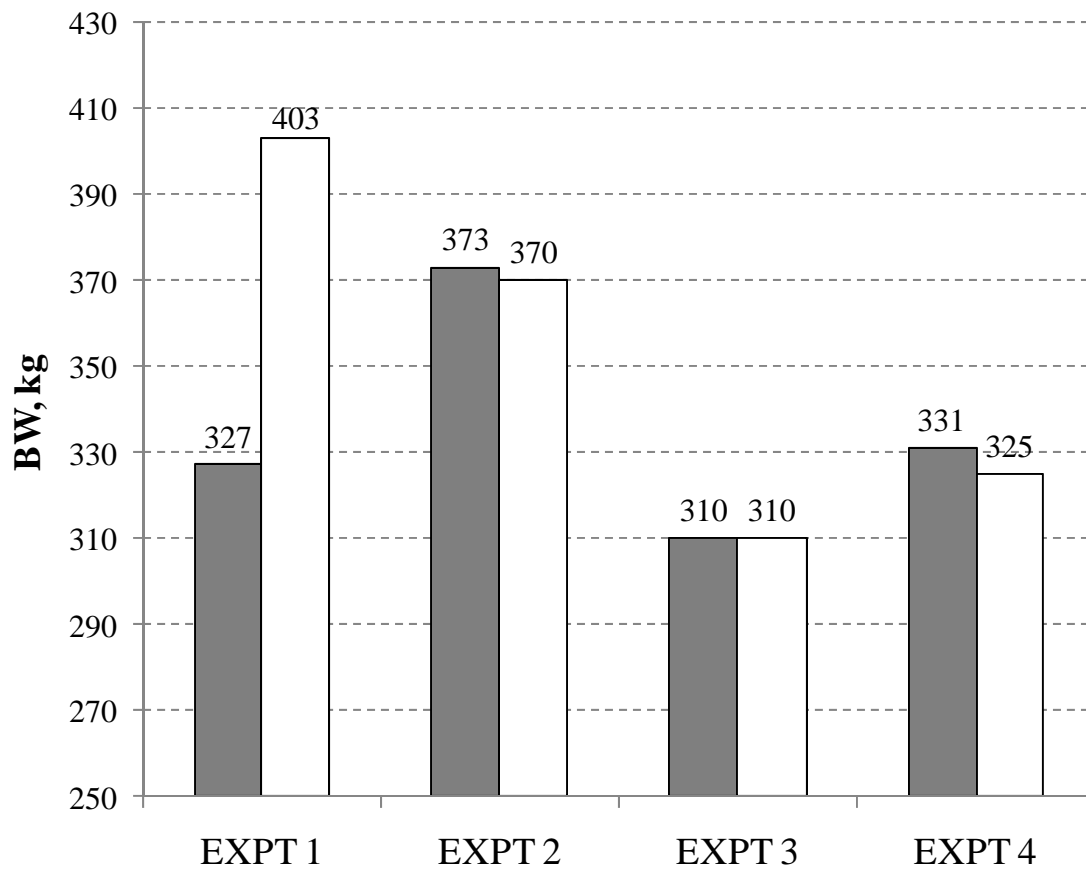


Figure 3. Body weight at puberty in heifers that were weaned early and fed a high concentrate diet (EWH; dark bars) or control diet (EWC; white bars) in Gasser et al., 2006a (EXPT 1), 2006b (EXPT 2), 2006c (EXPT 3) and 2006d (EXPT 4).

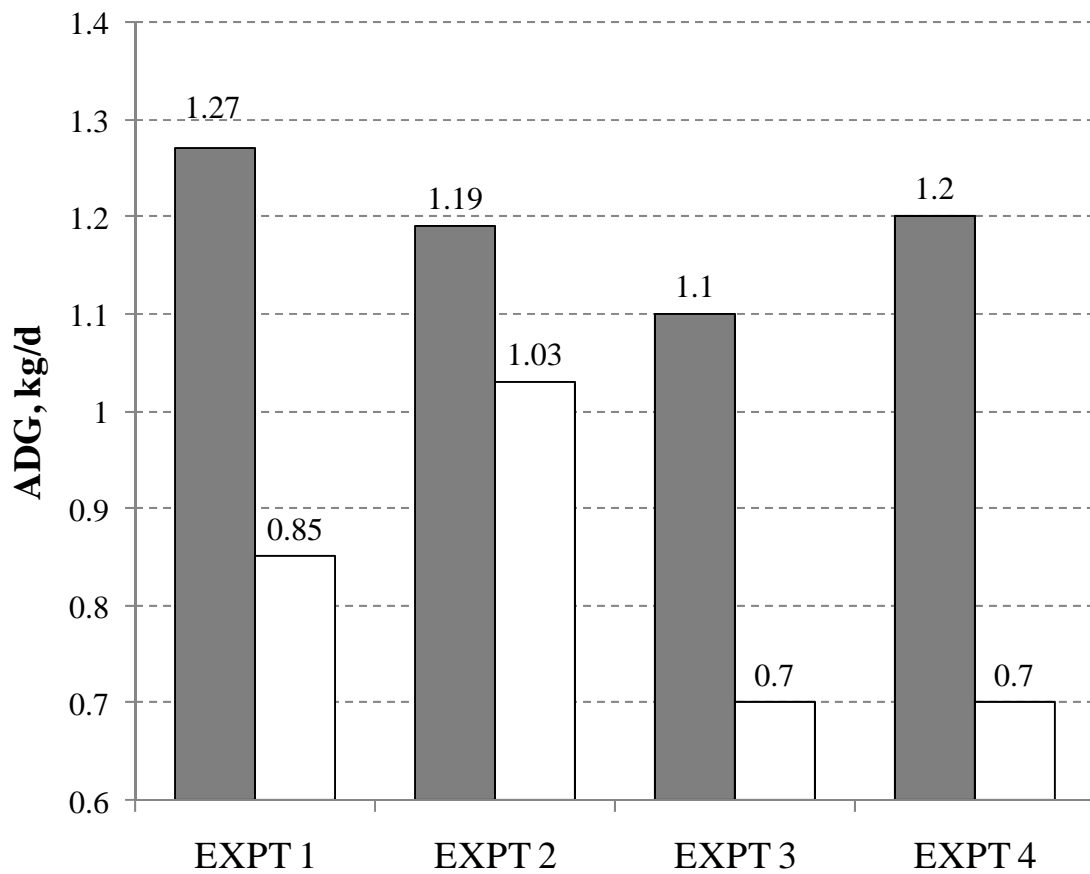


Figure 4. Average daily gain during the experiment in heifers that were weaned early and fed a high concentrate diet (EWH; dark bars) or control diet (EWC; white bars) in Gasser et al., 2006a (EXPT 1), 2006b (EXPT 2), 2006c (EXPT 3) and 2006d (EXPT 4).

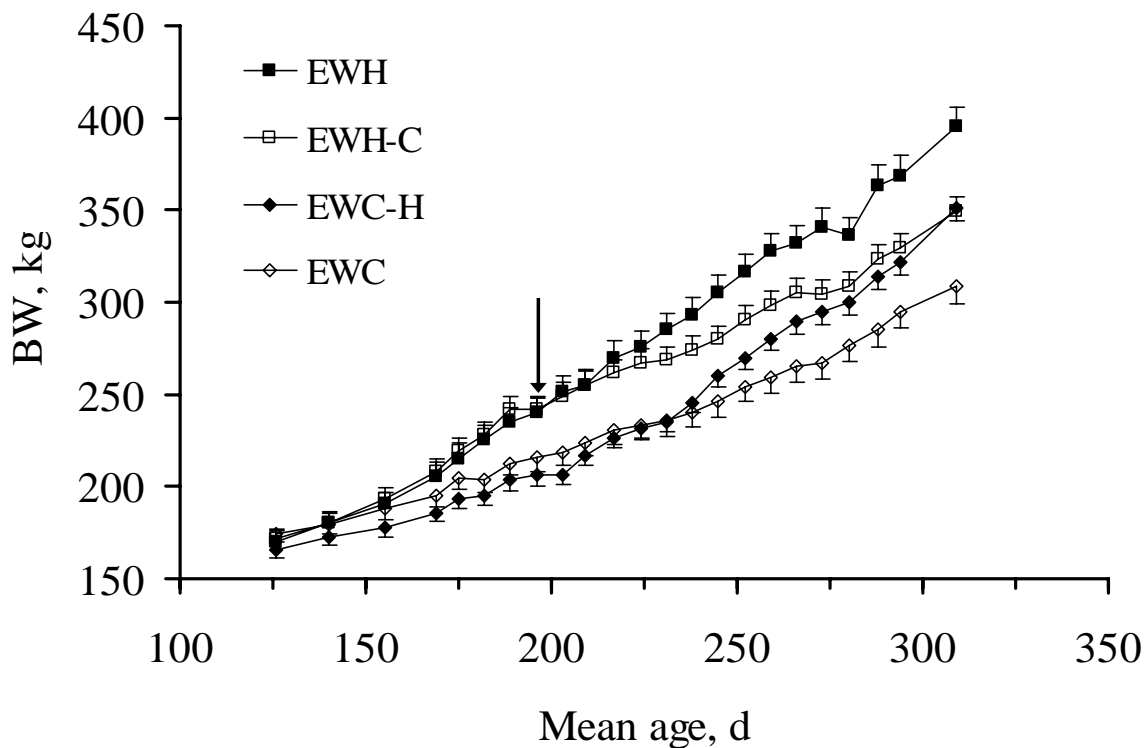


Figure 5. Body weight gain in heifers that were weaned early and fed a high concentrate diet (H) throughout the experiment (EWH), the H diet during Phase 1 and the control diet (C) during Phase 2 (EWH-C), the C diet during Phase 1 and the H diet during Phase 2 (EWC-H) or the C diet throughout the experiment (EWC). Phases are delineated by the arrow (Phase 1, 126 to 196 d of age; Phase 2, 196 d of age to the end of the experiment) Adapted from Gasser et al., 2006d, *Journal of Animal Science*. 84:3118-3122.

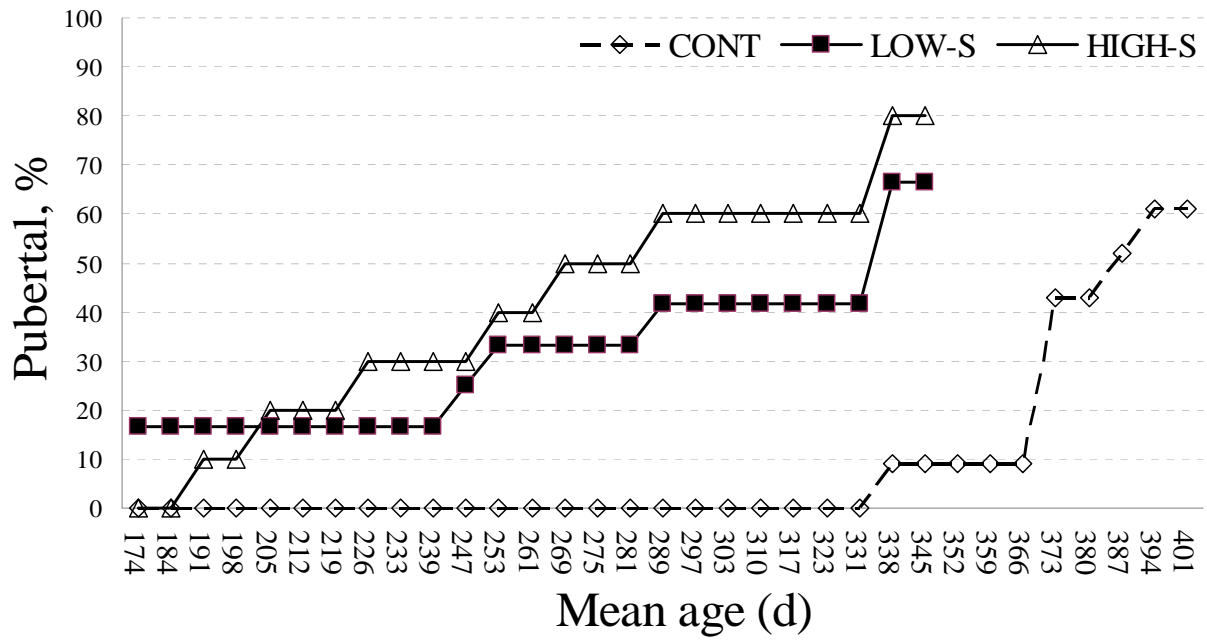


Figure 6. Cumulative percentage of pubertal heifers fed a control diet (CONT), high energy, low starch diet (LOW-S) or high energy, high starch diet (HIGH-S).

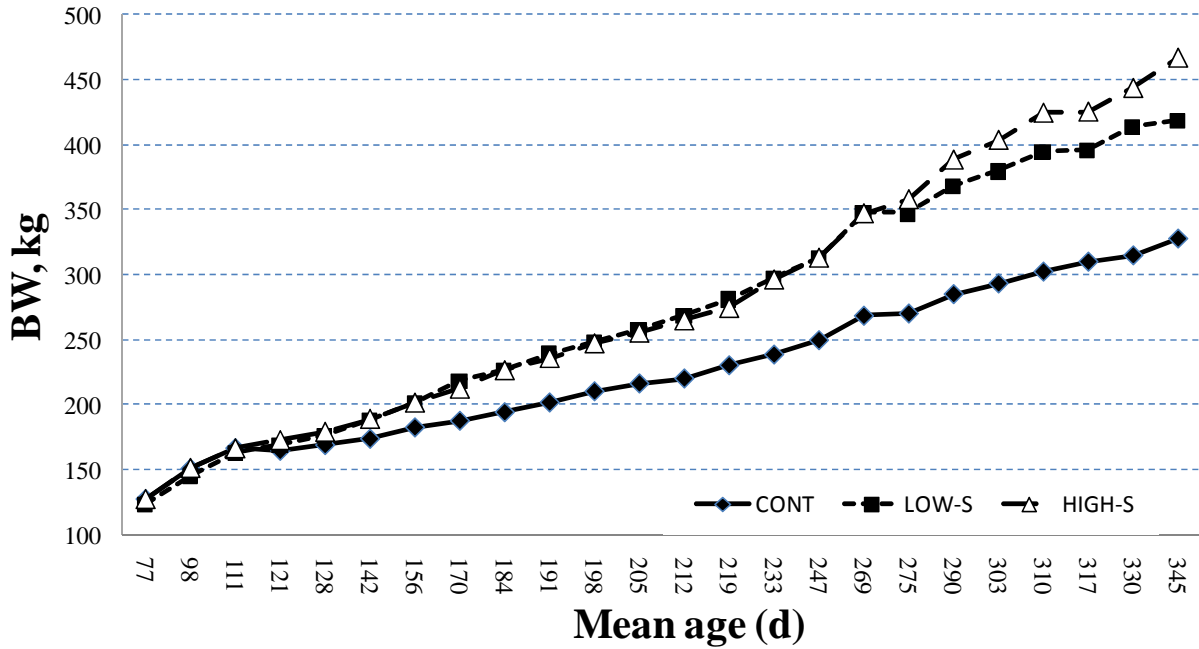


Figure 7. Growth rate fed of pre-pubertal heifers fed a control diet (CONT), high energy, low starch diet (LOW-S) or high energy, high starch diet (HIGH-S).

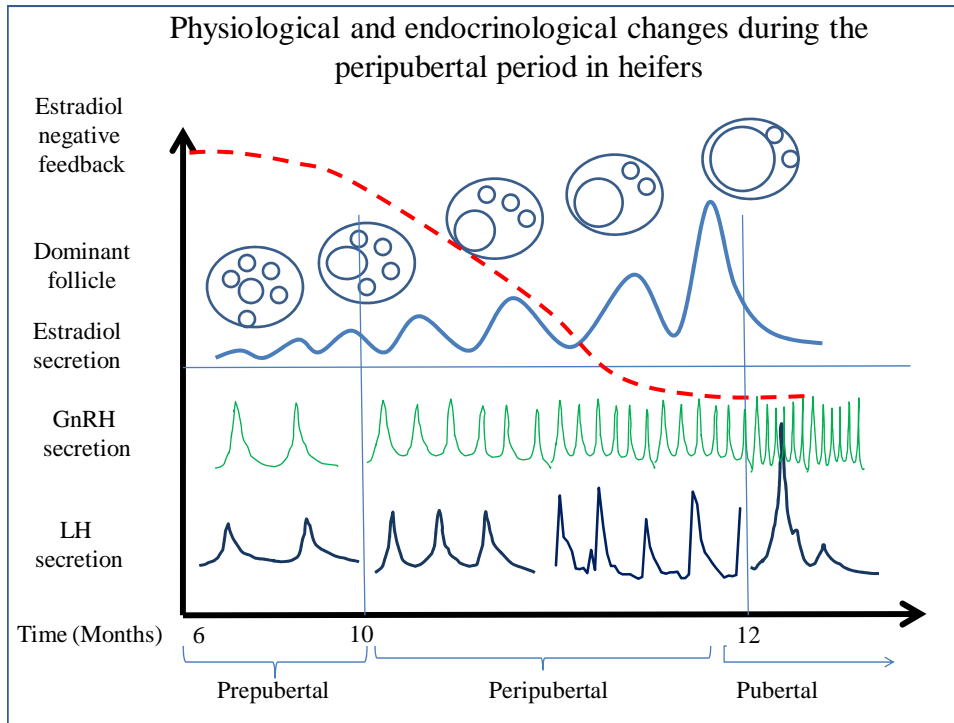


Figure 8. Endocrine changes during the peripubertal period that result in puberty in heifers. The peripubertal period includes the 50 to 60 d preceding puberty in heifers. The dashed line represents estradiol negative feedback on secretion of LH. The secretion of GnRH is highly sensitive to estradiol negative feedback during the prepubertal period. As the peripubertal period and the associated decline in estradiol negative feedback begins, secretion of GnRH, and hence LH, increases, resulting in increased growth and estradiol secretion by dominant ovarian follicles. As a result of the progressive decline in estradiol negative feedback and increase in LH secretion during the peripubertal period, estradiol concentrations eventually attain levels sufficient to induce the pubertal surge of LH.

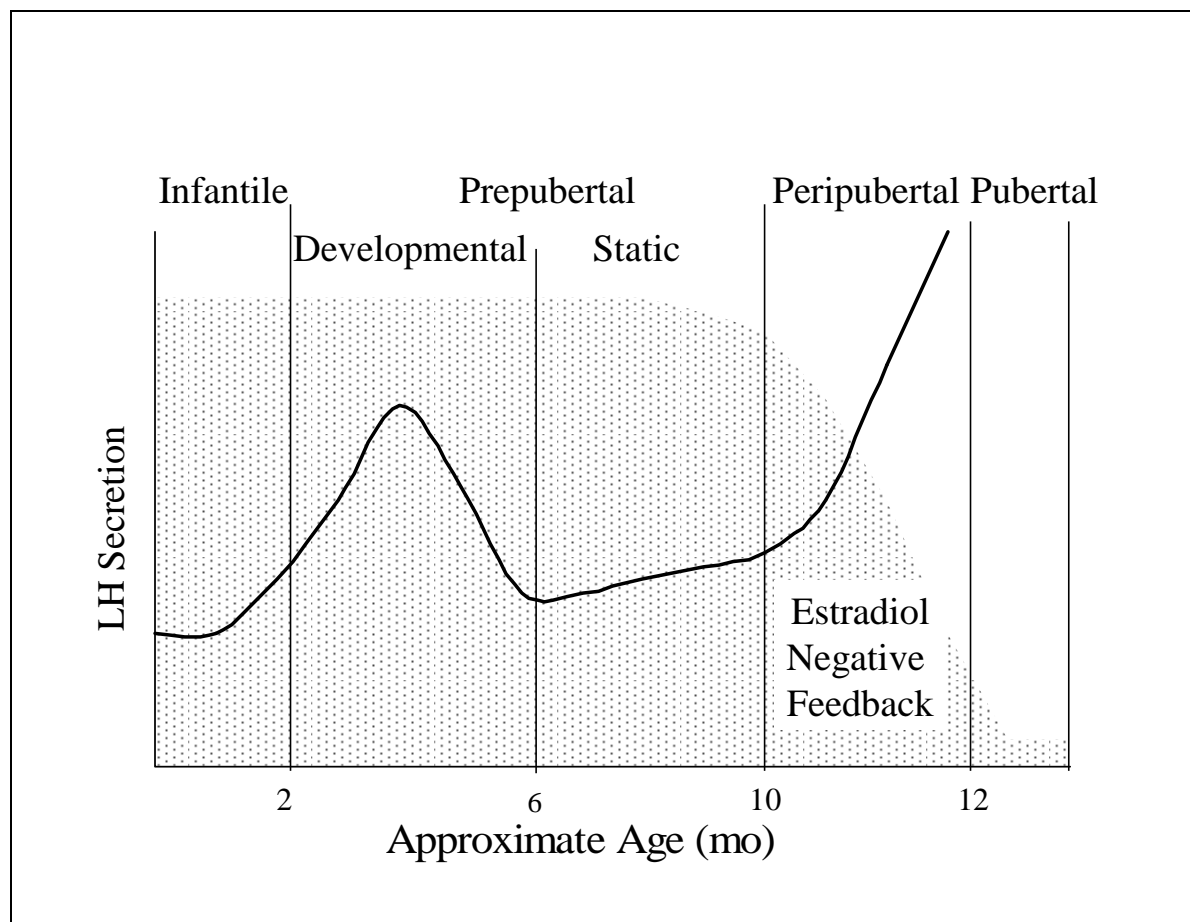


Figure 9. Model for stages of sexual maturation in heifers. The shaded area denotes the sensitivity of GnRH secretion to estradiol negative feedback. Changing patterns of LH secretion during the prepubertal period are the result of increased GnRH secretion followed by increased estradiol production by ovarian follicles. The peripubertal increase in LH is the result of a decline in estradiol negative feedback on LH. (Adapted from Day and Anderson, (1998) *Journal of Animal Science*. 76 (Suppl. 3): 1 – 15.

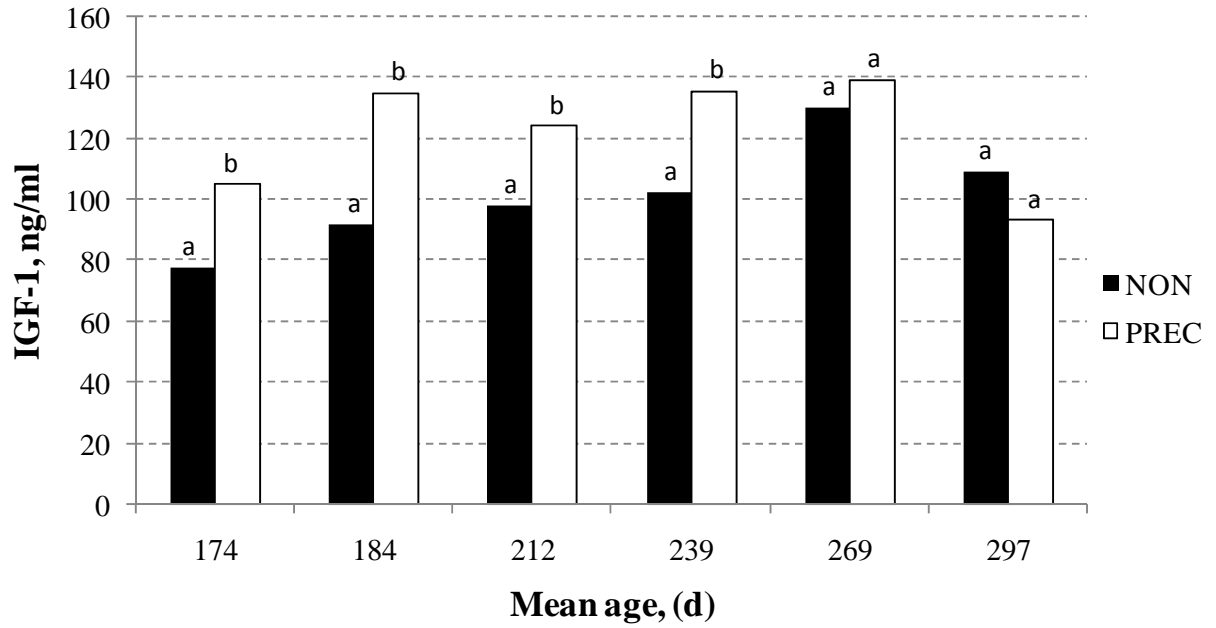


Figure 10. Serum concentration of IGF-1 in heifers that had precocious puberty (PREC) and those that did not (NON) in the HIGH-S and LOW-S treatments. Different superscript within age differ ($P < 0.05$)

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SESSION NOTES