Effect of timing of feeding a high-concentrate diet on growth and attainment of puberty in early-weaned heifers


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ABSTRACT: Precocious puberty (<300 d of age) can be successfully induced in a majority of heifers with early weaning and continuous feeding of a high-concentrate diet. The objective of this experiment was to determine the relative effects of timing of feeding a high-concentrate diet on age at puberty in early-weaned heifers. Sixty crossbred Angus and Simmental heifer calves were weaned at 112 ± 2 d of age and 155 ± 3 kg of BW and were fed a receiving diet for 2 wk. Heifers were blocked by age and BW, and assigned randomly to receive a high-concentrate (60% corn; H) or control (30% corn; C) diet during phase 1 (mean age 126 to 196 d) and H or C during phase 2 (mean age 196 to 402 d), resulting in 4 treatments (HH, n = 15; HC, n = 15; CH, n = 15; and CC, n = 15). Blood samples were collected weekly beginning at a mean age of 175 d and assayed for progesterone concentration to determine age at puberty. After 56 d on the experimental diets, BW of heifers fed the H diet during phase 1 were greater (P < 0.05) than those of heifers fed the C diet (mean age of 182 d; treatment × mean age, P < 0.01). After 70 d on the new diets (mean age of 266 d), heifers fed the H diet during phase 2 reached heavier BW (P < 0.05) than heifers fed the C diet, when compared within phase 1 diet groups (HH > HC; CH > CC). Body weights in HC and CH treatments differed from a mean age of 169 through 238 d, after which BW did not differ between these treatments. The ADG over the entire experimental period was greatest for the HH treatment (1.2 ± 0.04 kg/d; P < 0.05), followed by the HC and CH treatments (1.0 ± 0.03 and 1.0 ± 0.02 kg/d, respectively), which were not different, and the CC treatment gained the least (0.7 ± 0.04 kg/d; P < 0.05). Precocious puberty occurred in 67, 47, 47, and 20% of heifers in the HH, HC, CH, and CC treatments, respectively (HH > CC; P < 0.05). Mean age at puberty for the HH and HC treatments (271 ± 17 and 283 ± 17 d of age, respectively) was earlier (P < 0.05) than for the CC treatment (331 ± 11 d of age). Age at puberty in the CH treatment (304 ± 13 d of age) was intermediate to and not different from the other treatments. Heifers fed the H diet during phase 1 attained puberty earlier (P < 0.05) than heifers fed the C diet during phase 1. In conclusion, increasing dietary energy intake in early-weaned heifers, through feeding a high-concentrate diet from 126 to 196 d of age, decreased age at puberty regardless of the diet fed after 196 d of age.

Key words: early weaning, heifer, phase feeding, puberty

INTRODUCTION

To achieve optimal lifetime productivity, heifers should conceive early in their initial breeding season (Lesmeister et al., 1973). Heifers that have experienced multiple estrous cycles prior to onset of the breeding season have a greater probability for early conception in the initial breeding season (Byerly et al., 1987; Bagley, 1993).

Traditionally, beef heifers are weaned around 7 mo of age, and the impact of postweaning growth and nutritional status on age at puberty in heifers has been well documented (Clanton et al., 1983; Lynch et al., 1997; Grings et al., 1999). Less attention has been focused specifically on the impact of nutritional status before 7 mo of age on puberty. However, heifer calves that achieve greater weaning BW, increased growth rates from birth to weaning, or both have been reported to attain puberty at earlier ages (Wiltbank et al., 1966; Arije and Wiltbank, 1971; Buskirk et al., 1995).

We have also induced precocious puberty in 85 to 100% of heifers through early weaning and continuous...
feeding of a high-concentrate (60% corn) diet (Gasser et al., 2006a,b,c). It is unknown whether continuous feeding of the high-concentrate diet through puberty is necessary to sustain the physiological changes that lead to precocious puberty or if a similar response can be achieved with a temporary feeding of the high-concentrate diet.

We hypothesized that feeding a high-concentrate diet from immediately after early weaning (3 to 4 mo of age) through approximately 6 mo of age would effectively induce precocious puberty in heifers, similar to that achieved when heifers are weaned early and continuously fed a high-concentrate diet (Gasser et al., 2006a,b,c). We further postulated that in animals receiving the high-concentrate diet at this early age, puberty would be attained at a younger age than in heifers continuously fed a control diet or in heifers receiving the high-concentrate diet only after 6 mo of age.

**MATERIALS AND METHODS**

**Animals and Treatments**

All animals were handled in accordance with procedures approved by The Ohio State University Agricultural Animal Care and Use Committee. Sixty crossbred Angus and Simmental heifers were weaned at 112 ± 2 d of age and 155 ± 3 kg of BW and were fed a receiving diet (Table 1) for 2 wk after weaning. After the receiving period, heifers were stratified by age and BW and randomly assigned to receive a high-concentrate diet (H; n = 30) or a control diet (C; n = 30) during the first 70 d of the experiment (phase 1; mean age 126 to 196 d). Half of the heifers receiving the H and C diets were preassigned to remain on their respective diets for phase 2 (mean age 196 to 402 d) or to be fed the other diet during phase 2. The resulting treatments are hereafter designated as HH (H diet during phase 1 and phase 2), HC, CH, and CC, with 15 heifers in each treatment. The transition between diets in the HC and CH groups was accomplished over a 10-d period. The

### Table 1. Ingredients and chemical composition of experimental diets fed to heifers

<table>
<thead>
<tr>
<th>Item</th>
<th>Diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ingredient, % as-fed</td>
<td></td>
</tr>
<tr>
<td>Whole shelled corn</td>
<td>Receiving 60 High-concentrate 30 Control</td>
</tr>
<tr>
<td>13% CP alfalfa pellets</td>
<td>15 10 30</td>
</tr>
<tr>
<td>Pelleted soybean hulls</td>
<td>15 10 30</td>
</tr>
<tr>
<td>Supplement1</td>
<td>20 20 10</td>
</tr>
<tr>
<td>Chemical composition</td>
<td></td>
</tr>
<tr>
<td>CP, %</td>
<td>17.2 14.1 14.1</td>
</tr>
<tr>
<td>NE\text{\text{m}}, Mcal/kg</td>
<td>1.93 2.02 1.70</td>
</tr>
<tr>
<td>NE\text{\text{g}}, Mcal/kg</td>
<td>1.29 1.37 1.09</td>
</tr>
</tbody>
</table>

1Supplement contained ground corn, soybean meal, urea (0.4% of the diet), vitamins, minerals, Rumensin (0.015% of the diet), and fat (1% of the diet).

Blood samples were collected weekly beginning at a mean age of 175 d via jugular venipuncture, centrifuged at 2,785 × g for 20 min immediately after collection, and the plasma was harvested and frozen at −20°C until it was analyzed for progesterone concentration. Age at puberty was defined as 7 d before the date of collection of the first blood sample that contained >2 ng/mL or 7 d before the collection date of the first of 2 consecutive blood samples with >1 ng/mL of plasma progesterone. To ensure the continuation of estrous cycles, blood sampling was discontinued in individual heifers after the attainment of puberty confirmed by observation of at least 1 subsequent estrous cycle of normal duration, based on progesterone concentration. Heifers that reached puberty before 300 d of age were considered to have experienced precocious puberty (Gasser et al., 2006a,b,c).

**Progesterone Quantification**

Concentrations of progesterone were determined using a commercially available RIA kit (Coat-a-Count, Diagnostic Products Corporation, Los Angeles, CA) as described previously for our laboratory (Burke et al., 2003). Interassay CV were 8.7, 7.5, and 9.2% for standard concentrations of 1.6, 2.7, and 11.7 ng/mL, respectively. Average intraassay CV was 3.2%, and the assay sensitivity was 0.1 ng/mL.

**Statistical Analyses**

Effects of treatment, mean day of age, and the interaction of treatment and mean day of age on BW were analyzed by ANOVA using the MIXED procedure (SAS Inst. Inc., Cary, NC), with repeated measures analysis appropriate for a split-plot design. The repeated measures model was

\[ Y_{ijk} = \mu + T_i + h_{ji} + D_k + (TD)_{ik} + e_{ijk}, \]

where \( Y_{ijk} \) = observation of the \( j \)th heifer in the \( i \)th treatment on the \( k \)th day, \( \mu \) = overall mean, \( T_i \) = \( i \)th
treatment, $h_{ji} = \text{random effect of the } j\text{th heifer within the } i\text{th treatment} (h_{ji} \sim N(0, \sigma^2_{hi}))$, $D_k = k\text{th day}$, $(TD)_{ijk} = \text{treatment } \times \text{day interaction term}$, and $e_{ijk} = \text{random residual effect} (e_{ijk} \sim N(0, \Sigma))$, where $\Sigma$ is the variance-covariance structure of the residual errors with a first-order autoregressive structure for repeated measurements within heifers.

The effect of treatment on ADG, age at puberty, and BW at puberty was analyzed by ANOVA appropriate for a completely random design using the MIXED procedure of SAS. In addition, we analyzed the effect of diet during each of the 2 phases of the experiment on ADG, age at puberty, and BW at puberty for subsequent analyses. The effect of treatment on the percentage of heifers experiencing precocious puberty was tested using $\chi^2$ analysis.

RESULTS

Heifers that received the H diet in phase 1 (HH and HC treatments) had greater BW ($P < 0.05$) than heifers fed the C diet during this phase (CC and CH treatments) after 56 d on experimental diets (mean age of 182 d; treatment $\times$ mean age, $P < 0.01$; Figure 1). Heifers fed the H diet during phase 2 reached greater BW ($P < 0.05$) than heifers fed the C diet during this phase, when compared within their respective phase 1 diet groups (HH > HC; CH > CC), after 70 d on new diets (mean age of 266 d). Body weight of heifers in the HC and CH treatments differed from mean age of 169 to 238 d ($P < 0.05$) but were similar among these treatments thereafter.

During phase 1 of the experiment, ADG was greater ($P < 0.05$) in HH and HC treatments ($1.0 \pm 0.03 \text{ kg/d}$) than the CH and CC treatments ($0.6 \pm 0.03 \text{ kg/d}$; Figure 2). During phase 2, ADG for HH and CH treatments ($1.4 \pm 0.05$ and $1.3 \pm 0.04 \text{ kg/d}$, respectively) did not differ from each other, but both were greater ($P < 0.05$) than the HC treatment ($1.0 \pm 0.03 \text{ kg/d}$). In the CC treatment, ADG was less ($P < 0.05$) than in all other treatments ($0.8 \pm 0.03 \text{ kg/d}$) during phase 2. In addition, phase 2 ADG was influenced by phase 1 diet ($P < 0.05$), with heifers that were fed H during phase 1 gaining more BW during phase 2 compared with heifers that were fed C during phase 1. The ADG over the entire experimental period was greatest for heifers in the HH treatment ($1.2 \pm 0.04 \text{ kg/d}$; $P < 0.05$), followed by HC and CH treatments ($1.0 \pm 0.03$ and $1.0 \pm 0.02 \text{ kg/d}$, respectively), which were not different, and heifers in the CC treatment gained the least ($0.7 \pm 0.04 \text{ kg/d}$; $P < 0.05$).

Precocious puberty occurred in 67, 47, 47, and 20% of heifers in the HH, HC, CH, and CC treatments, respectively, which was greater ($P < 0.05$) in the HH than the CC treatment. The cumulative proportion of pubertal heifers throughout the experimental period is shown in Figure 3. The experiment was terminated when heifers reached a mean age of 402 d, at which time 1 heifer in the HH treatment and 1 in the CH treatment had not reached puberty. For these 2 heifers, age and BW at termination of the experiment were used as their age and BW at puberty for subsequent analyses.

Average age at puberty for heifers in the HH and HC treatments ($271 \pm 17$ and $283 \pm 17 \text{ d of age}$, respectively) was earlier ($P < 0.05$) than for those in the CC treatment ($331 \pm 11 \text{ d of age}$; Figure 4). Age at puberty in the CH treatment ($304 \pm 13 \text{ d of age}$) was intermediate to and not different than the other treatments. Additionally, age at puberty was influenced by phase 1 diet ($P < 0.05$),

Figure 1. Body weight (kg) of heifers that were weaned at 112 ± 2 d of age and fed a high-concentrate diet continuously (60% corn; HH), the high-concentrate diet from a mean age of 126 to 196 d and then transitioned to a control diet (30% corn; HC), the control diet transitioned to the high-concentrate diet (CH), or the control diet continuously (CC). The arrow denotes the time at which the diet transition occurred in HC and CH treatments.

Figure 2. The ADG (kg/d) of heifers that were weaned at 112 ± 2 d of age and fed a high-concentrate diet continuously (60% corn; HH), the high-concentrate diet from a mean age of 126 to 196 d and then transitioned to a control diet (30% corn; HC), the control diet transitioned to the high-concentrate diet (CH), or the control diet continuously (CC). Within variable, treatment means with different superscript letters differ ($P < 0.05$).
Timing of diet feeding and puberty in heifers

The present experiment provides evidence that in early-weaned heifers, the type of diet consumed during phase 1 was a more important determinant of age at puberty than the type of diet consumed during phase 2. For a majority of previous reports regarding the influence of nutrition on age at puberty, phase 1 in the present experiment would correspond approximately with the preweaning period, whereas phase 2 approximates the postweaning period. The importance of nutritional plane during the period preceding 7 mo of age demonstrated in the current study is not unprecedented. Wiltbank et al. (1966) reported that preweaning ADG had more consistent influence on age at puberty than postweaning ADG in heifers that were weaned around 200 d of age. Others (Arije and Wiltbank, 1971; Buskirk et al., 1995) have also demonstrated that increased preweaning growth rates, larger weaning BW, or both were associated with earlier onset of puberty in heifers. Apparently, weaning before the traditional 7 mo of age is not essential for the effects of increased early growth rate to induce earlier puberty. In contrast, in a similar experiment in which Holstein heifers beginning at 143 d of age were fed at 2 nutritional planes for an initial phase of 100 d and a final phase ending at the second postpubertal estrus, it was concluded that the final phase diet had a more dramatic influence on age at puberty than the diet consumed during the initial phase (Dufour, 1975). However, the fast-growing group of heifers in that experiment exhibited ADG similar to heifers fed the control diet in the present experiment, limiting comparison with the present experiment.

Phase 1 of the current study corresponds with a period during which dynamic developmental changes in gonadotropin secretion and follicular growth are occurring in heifers (reviewed by Day and Anderson, 1998). We have previously demonstrated that, compared with heifers fed a similar control diet, feeding a similar high-concentrate diet during this period results in greater secretion of LH, increased ovarian follicular development, and hastened reduction of estradiol negative feedback on secretion of LH (Gasser et al., 2006a,b,c). Other types of treatment during this early age in heifers have also been successful at stimulating growth and hastening onset of puberty. Madgwick et al. (2005) treated heifer calves with exogenous GnRH from 4 to 8 wk of age and observed subsequent increases in BW gain, mean LH concentrations, and LH pulse frequency that occurred at similar ages to the changes in BW observed in heifers from the present experiment. The GnRH treatment also reduced age at puberty. Heifers that were administered ivermectin from birth through puberty grew faster and reached puberty earlier than nontreated heifers (Mejia et al., 1999; Lacau-Mengido et al., 2000). Lacau-Mengido et al. (2000) also detected increased concentrations of LH and IGF-I in the ivermectin-treated heifers. The mechanisms by which environmental manipulation during the period...
Several researchers have suggested that there is considerable latitude in the rate and timing of growth in replacement heifers, as long as they reach a target BW before first breeding (Clanton et al., 1983; Lynch et al., 1997; Freetly et al., 2001). However, most of these experiments have focused on nutritional management of heifers from weaning (typically around 200 d of age) to breeding. Results from the present experiment and from Wiltbank et al. (1966) would agree that the timing of growth after approximately 200 d of age can be varied to some extent without a detrimental influence on pubertal development. However, this latitude in later growth may be facilitated by the influence of growth prior to that age period, which was shown to have a greater influence on the timing of attainment of puberty in heifers in the present experiment and that of Wiltbank et al. (1966). From these results it appears the same latitude for timing of growth may not exist during this earlier age period.

The incidence of precocious puberty in the present experiment was not as great as had been previously observed using a similar model (Gasser et al., 2006a,b,c). This may have been influenced to some extent by the fact that heifers in the present experiment were weaned at a slightly greater age. Later weaning could have reduced the potential window of time available during the first several months of age to promote advancement of puberty. Although there was a lesser incidence of precocious puberty in this experiment, those heifers that attained precocious puberty did so at lighter BW, as has also been observed in the previous experiments.

In conclusion, attainment of puberty in early-weaned heifers can be hastened through feeding a high-concentrate diet from approximately 4 to 6.5 mo of age in a manner similar to that of heifers that are continuously fed the high-concentrate diet after early weaning. Nutritional status during this early sexual maturation period is an important determinant of age at puberty in heifers. The nutritional status of heifers during this time may determine the impact of feeding programs initiated later in life (i.e., postweaning) for replacement heifer development.

**LITERATURE CITED**


