Effect of an orally active progestin on follicular dynamics in cycling and anestrous postpartum beef cows

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ABSTRACT: Although treatment of cycling cows with low concentrations of melengestrol acetate (MGA) results in formation of persistent follicles, in the absence of corpora lutea, it is not known whether persistent follicles form in anestrous cows in response to a similar treatment. The objective of this experiment was to determine the effect of long-term MGA treatment (14 d) on follicular dynamics and the secretion of estradiol in anestrous postpartum beef cows. Treatment groups (replicated over 2 yr) included the following: anestrous control (AC; n = 11), anestrous MGA (AM; n = 16), and cycling MGA (CM; positive control; n = 16). Angus-crossbred cows were assigned to treatment by age, cow body condition, and days postpartum. Cows were fed carrier (AC group) or 0.5 mg MGA/animal−1·d−1 (AM and CM groups) for 14 d beginning approximately 38 d postpartum. Cows allotted to the CM group were injected with PGF2α on the first day of MGA treatment to induce luteolysis. The preceding treatment (CM) results in formation of persistent follicles and secretion of elevated concentrations of estradiol. Ovaries of each cow were examined daily by transrectal ultrasonography beginning 5 to 7 d preceding the initiation of feeding MGA or carrier and continued until ovulation or 7 d following MGA feeding. There was no difference among groups in the stage of follicular wave or diameter of the largest follicle at the start of carrier or MGA feeding. The length of the follicular wave present at the start of MGA feeding was greater (P < 0.01) for cows in the CM (14.5 d, yr 1; 18.3 d, yr 2) group compared to the AM (9.4 d, yr 1; 7.9 d, yr 2) or AC (9.7 d, yr 1; 10.7 d, yr 2) groups. Maximum follicular diameter over both years was greater (P < 0.01) for the CM (20.6 mm) group than the AM (15.1 mm) or AC (16.4 mm) groups. Circulating concentrations of estradiol were also increased (P < 0.05) in the CM group compared to the AM or AC groups. However, MGA appeared to have no effect (P > 0.05) on the number of follicles recruited, growth rate of the dominant follicle during the first 6 d of treatment, or growth rate to the maximum follicular diameter. In summary, MGA treatment did not increase the duration of the follicular wave, maximum follicular diameter, or secretion of estradiol in anestrous postpartum cows, nor did MGA affect the number of follicles recruited or growth rate of dominant follicles in cycling or anestrous animals.

Key Words: Beef Cows, Follicles, Progestogens

Introduction

Progestins have been shown to be an effective method of inducing and synchronizing ovulation in beef females (Odde, 1990), and melengestrol acetate (MGA), an orally active progestin, has been used to synchronize estrus in postpartum beef cows (Patterson et al., 1995). However, treatment of cycling heifers or cows with a progestin, after normal CL regression caused the formation of persistent follicles that were characterized by an extended dominant life span and increased estradiol production (Zimbelman and Smith, 1966; Siriois and Fortune, 1990; Fortune and Rivera, 1999). The progestin also increased LH pulse frequency, which may promote formation of persistent follicles (Savio et al., 1993b; Kojima et al., 1995). An increase in LH pulse frequency may be responsible for the increased estrogen production by persistent follicles because LH has been shown to increase androgen production in follicles (Fortune, 1986).

Although the formation of persistent follicles in cycling animals treated with low levels of progesterone/progestin, in the absence of a CL, is well known (see review by Fortune and Rivera, 1999), the effect of low levels of progestin on the follicular development of anestrous animals has not been characterized. In postpartum beef cows, follicular waves resume shortly after parturition with a rise in concentrations of follicle-stim-
ulating hormone (Schallenberger, 1985). In beef cows, the first dominant follicle appeared between 7 and 15 d after parturition (Murphy et al., 1990; Crowe et al., 1993); however, only 11% of the dominant follicles ovulated (Murphy et al., 1990). The inability of the first dominant follicle to ovulate is thought to be due to the absence of a preovulatory gonadotropin surge because a single injection of a GnRH agonist can induce ovulation (Crowe et al., 1993). The objective of this experiment was to determine the effect of MGA on follicular dynamics and estradiol production in anestrous postpartum cows.

**Materials and Methods**

**Experimental Design**

Postpartum multiparous (3 to 8 yr old) Angus-Simmental or Angus-Charolais beef cows (body condition score 5–6; 1 = emaciated and 9 = obese) were divided into three treatment groups (anestrous control [AC], anestrous MGA [AM], and cycling MGA [CM]) according to days postpartum (yr 1 AC, 35.4 ± 1.8 d; AM, 38.0 ± 1.9 d; CM, 38.7 ± 1.9 d and yr 2 AC, 38.1 ± 1.4 d; AM, 37.6 ± 1.2 d; CM, 37.2 ± 1.2 d), age, cow body condition score, and cycling status. Calves were maintained with cows at all times and allowed to suckle without restriction. Anestrous cows had serum concentrations of progesterone that were less than 1 ng/mL and no luteal tissue present as determined daily by radioimmunoassay and ultrasonography for 5 to 7 d before the initiation of treatment. Cycling cows had serum concentrations of progesterone that were greater than 1 ng/mL and luteal tissue present as determined daily by radioimmunoassay and ultrasonography for 5 to 7 d before the initiation of treatment. Cycling and anestrous cows were selected to be at a similar stage of a follicular wave at the initiation of treatment.

The experiment was conducted in two replications over 2 yr. In both years, each group consisted of 8 or 10 animals, and all animals were fed carrier (pretreatment) for 5 to 7 d before the start of MGA treatment. The purpose of the pretreatment was to determine stage of follicular wave at the start of MGA treatment. All cows were maintained as a group except during the 14 d during which MGA was fed; during that time, both MGA treatments were fed together and the anestrous control group was fed separately. Cycling cows received 25 mg of prostaglandin F₂₀ (Lutalyse; Pharmacia Animal Health, Kalamazoo, MI) i.m. on the first and second days of MGA treatment to induce luteolysis. Anestrous control animals were fed carrier (1.8 kg-animal⁻¹·d⁻¹; Cattle Charge; MFA, Columbia, MO), whereas the anestrous MGA and cycling MGA groups were fed 1.8 kg-animal⁻¹·d⁻¹ of carrier containing 0.278 mg MGA/kg (Cattle Charge Estrus Control; MFA, Columbia, MO) for 14 d. In both years, one animal from the anestrous MGA group, and one animal from the cycling MGA group ovulated during MGA treatment and were removed from the experiment. Thus, data from these two groups reflect seven animals per group in yr 1 and nine animals per group in yr 2. Animals from the anestrous control group were removed from the study if the dominant follicle from the wave present at the start of treatment ovulated. During yr 1, four animals were removed from the study, leaving four animals to be reported, and, in yr 2, three animals were removed, leaving seven in that group.

**Blood Sampling**

Daily blood samples were collected by jugular venipuncture into 10-mL Vacutainer tubes (Fisher Scientific, Pittsburgh, PA) beginning 5 to 7 d before the start of MGA treatment and continuing until 7 d following MGA treatment or ovulation. Blood was allowed to clot and was stored at 4°C for 24 h, and serum was harvested for determination of serum concentrations of progesterone and estradiol-17β by radioimmunoassay.

**Radioimmunoassays**

Serum concentrations of progesterone were used to determine cycling status before beginning the treatment. Animals with a concentration of progesterone greater than 1 ng/mL were considered to have luteal tissue, and presence of luteal tissue was confirmed by transrectal ultrasonography. After PGF₂₀α-induced luteolysis in the cycling MGA group, no animals had serum concentrations of progesterone above 1 ng/mL until after the treatment had been terminated and ovulation occurred. All anestrous animals had progesterone concentrations less than 1 ng/mL.

Concentrations of progesterone were analyzed in all daily serum samples by radioimmunoassay (Kirby et al., 1997; Diagnostic Products Corporation, Los Angeles, CA). Intra- and interassay coefficients of variation for progesterone assays were < 2.0 and < 7.0%, respectively, and assay sensitivity was 0.5 ng/mL of serum. Concentrations of estradiol-17β were analyzed in all daily serum samples by RIA (Kirby et al., 1997). Intra- and interassay coefficient of variation for estradiol-17β assays were < 7.0 and < 18.0%, respectively, and assay sensitivity was 0.5 pg/mL of serum.

**Ultrasonography**

All cows were examined daily by transrectal ultrasonography to record follicular development using an Aloka 500 ultrasound with a 7.5-MHz transrectal linear probe (Aloka, Wallingford, CT). All follicles (≥ 5 mm) were recorded. Cows were examined daily from 5 to 7 d before the start of MGA treatment until either ovulation or 7 d following the withdrawal of MGA. A dominant follicle was defined as a large follicle, with other follicles regressed or regressing. Dominance was considered to have ended when a new follicular wave emerged. Follicle size (≥ 5 mm) was determined by measuring follicular diameter at the widest point of the
follicle and at a right angle to the first measurement using the internal calipers on the Aloka 500. These two measurements were averaged to obtain follicular diameter. All follicles were classified into three size classes: class I, ≤ 6 mm; class II, 6.5 to 9 mm; and class III, ≥ 9.5 mm. Length of a follicular wave was defined as the interval from when the follicle that became dominant was first detected until a new follicular wave began. Initiation of a new follicular wave was defined as the growth of at least two follicles that attained a diameter of at least 6 mm. Ovulation was determined by the disappearance of a large dominant follicle from an ovary and the appearance of luteal tissue on the same ovary.

**Statistical Analysis**

Age of the follicular wave at the start of treatment, size of the dominant follicle at the start of treatment, maximum diameter of the dominant follicle, length of the follicular wave, follicular growth rate, number of follicles in each follicular size class, size of dominant follicle at the end of treatment, age of the follicular wave at the end of treatment, and serum concentrations of estradiol on the last day of treatment were analyzed by analysis of variance for repeated measures (SAS; proc mixed; Littell et al., 1998). The effect of treatment was tested using animal within treatment as the error term. Differences in the percentage of animals that ovulated between the two MGA treatments and 1–6 b diameter of at least 6 mm. Ovulation was determined by analysis of variance (SAS; proc mixed; Littell et al., 1998). The effect of treatment was tested using animal within treatment as the error term, and treatment × day. When the F-statistic was significant (P < 0.05), a mean separation was performed using the least significant difference test (means ± SEM; SAS Inst. Inc., Cary, NC; Snedecor and Cochran 1989). Serum concentrations of estradiol-17β and number of follicles recruited during the initiation of a follicular wave were analyzed by analysis of variance for repeated measures (SAS; proc mixed; Littell et al., 1998). The effect of treatment on serum concentrations of estradiol during the first follicular wave and the effect of treatment on serum concentrations of estradiol during the second follicular wave were analyzed by analysis of variance for repeated measures (SAS; proc mixed; Littell et al., 1998). The effect of treatment was tested using animal within treatment as the error term, and day and treatment × day were tested using the residual as the error term. Differences in the percentage of animals that ovulated between the two MGA treatments were analyzed by chi square analysis in SAS (Snedecor and Cochran, 1989).

**Results**

**Follicular Dynamics**

Because age of the follicular wave present at the start of MGA treatment differed (P < 0.01) between yr 1 and yr 2, the data were analyzed separately. Age of the follicular wave at the start of treatment with MGA or carrier was similar (P > 0.05) among treatment groups for yr 1 (CM, 3.8 ± 1.6; AM, 2.8 ± 1.0; AC, 3.0 ± 0.93 d) and 2 (CM, 7.4 ± 0.72; AM, 6.2 ± 0.57; AC, 6.1 ± 0.57 d). Similarly, there was no difference (P > 0.05) among groups in the size of the dominant follicle at the start of treatment in yr 1 (CM, 13 ± 2.3; AM, 11.4 ± 1.5; AC, 12.2 ± 1.5 mm) and yr 2 (CM, 13.6 ± 1.03; AM, 11.5 ± 1.03; AC, 11.1 ± 1.3 mm). There was a treatment × year interaction for length of the follicular wave; therefore, the data were analyzed separately for yr 1 and 2 (Figure 1). Regardless of year, length of the follicular wave present at the start of MGA was greater (P < 0.02) for cows in the CM group compared to the AM or AC group. Length of the follicular wave was similar in the AC and AM groups for yr 1, but longer (P < 0.02) in the AC group compared to the AM group in yr 2.

As a result of the experimental model used in this study, the follicular wave present at the start of treatment in the CM group was exposed to progesterone for a few days before initiation of MGA treatment (and PGF2α, injection), whereas the follicular waves at the start of treatment in the AC and AM groups were not exposed to progesterone. To adjust the data for a potential effect of progesterone on the growth of the dominant follicle in the CM group, the maximum size of the dominant follicle during the follicular wave was analyzed.

**Table 1. Effect of treatment on the growth rate of the dominant follicle**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>n</th>
<th>d 1-6</th>
<th>To maximum diameter</th>
</tr>
</thead>
<tbody>
<tr>
<td>AC</td>
<td>11</td>
<td>0.85 ± 0.18</td>
<td>1.13 ± 0.24</td>
</tr>
<tr>
<td>AM</td>
<td>16</td>
<td>0.72 ± 0.15</td>
<td>1.21 ± 0.19</td>
</tr>
<tr>
<td>CM</td>
<td>16</td>
<td>1.03 ± 0.15</td>
<td>0.88 ± 0.19</td>
</tr>
</tbody>
</table>

*aAC = anestrous control, AM = anestrous MGA, CM = cycling MGA.

bDay 1 = the first day of treatment (carrier or MGA).

cMean ± SEM. Means within a column (P > 0.05).
using dominant follicle diameter at the start of treatment as a covariate. Regardless of year, maximum size of the dominant follicle was larger ($P < 0.02$) for the cycling MGA group (20.6 ± 0.72 mm) compared to the anestrous MGA group (15.1 ± 0.71 mm) or the anestrous control group (16.4 ± 0.99 mm). However, the growth rate (millimeters per day) of the dominant follicle for the first 6 d of treatment or from the start of treatment until the maximum diameter was reached was not different ($P > 0.05$) between treatments (Table 1).

Because all anestrous cows and the majority of cycling cows (14/16) initiated a new follicular wave during MGA treatment, the effect of MGA treatment on the number of follicles recruited was analyzed. The number of follicles recruited at the initiation of the follicular wave preceding treatment was similar ($P > 0.05$) to the number of follicles recruited at the initiation of follicular waves during MGA treatment, or after MGA treatment ended (Table 2). However, on d 7 and 10 of treatment, the AC and AM groups had a greater ($P < 0.01$) number of follicles in size class II (AC, 4.9 and 4.6; AM, 3.9 and 5.0) compared to the CM group (1.5 and 0.6). The AM group also had a greater ($P < 0.01$) number of follicles in size class III (6.5–9 mm) compared to the CM group (1.5 and 0.6). However, on d 7 and 10 of treatment, the AC and AM groups had a greater ($P < 0.01$) number of follicles in size class I (AC, 4.9 and 4.6; AM, 3.9 and 5.0) compared to the CM group (1.5 and 0.6). The AM group also had a greater ($P < 0.01$) number of follicles in size class II (1.5 and 0.6; d 7 and 10, respectively), compared to the CM group (0.4 and 0.4; d 7 and 10, respectively). In addition, the AC group had a greater ($P < 0.01$) number of follicles, in size class II, compared to the CM group on day 10 (AC, 1.7; CM, 0.4; Table 3).

The number of animals that ovulated within 7 d following MGA withdrawal was greater ($P < 0.02$) in the CM group than the AM group (Table 4). However, only 56% of the CM animals ovulated within 7 d following MGA withdrawal.

Hormone Concentrations

The effects of treatment on daily serum concentrations of estradiol-17β were determined during the follicular wave present at the start of treatment. In yr 1, there was no significant difference among groups in serum concentrations of estradiol-17β for 2 d before treatment or for the first 4 d of treatment. Beginning on d 5 or 6 of treatment, serum concentrations of estradiol-17β were increased ($P < 0.02$) in the cycling MGA group compared to anestrous MGA and anestrous control groups (Figure 2A). In yr 2, there was no difference among groups in serum concentrations of estradiol-17β for the 5 d before treatment began. However, beginning on the first day of treatment, serum concentrations of estradiol-17β were increased ($P < 0.04$) in the cycling MGA group compared to the other groups (Figure 2B).

On the last day of MGA treatment, there was no difference ($P > 0.05$) between the two MGA treatments in serum concentrations of estradiol (AM, 3.6 ± 0.78 and CM, 3.2 ± 0.52 pg/mL), age of the follicular wave (AM, 5.4 ± 1.8 and CM, 4.8 ± 1.2 d), or in the size of the dominant follicle (AM, 10.4 ± 1.3 and CM, 10.5 ± 0.99 mm). However, the growth rate (millimeters per day) of the dominant follicle for the first 6 d of treatment or from the start of treatment until the maximum diameter was reached was not different ($P > 0.05$) between treatments (Table 1).

### Table 2. Effect of treatment on the number of follicles recruited during the initiation of follicular wave 1, 2, and 3

<table>
<thead>
<tr>
<th>Treatmenta</th>
<th>Follicular waveb</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1c</td>
</tr>
<tr>
<td>AC</td>
<td>7.5 ± 3.1, n = 10</td>
</tr>
<tr>
<td>AM</td>
<td>7.6 ± 2.4, n = 14</td>
</tr>
<tr>
<td>CM</td>
<td>7.0 ± 3.4, n = 15</td>
</tr>
</tbody>
</table>

a AC = anestrous control; AM = anestrous MGA; CM = cycling MGA. 
bcFollicular wave 1 was initiated prior to the beginning of treatment. 
dFollicular wave 2 was initiated during treatment. 
eFollicular wave 3 was initiated after treatment ended.

| Table 3. Effect of treatment on mean number of follicles in class I (≤ 6 mm), II (6.5–9 mm), and III (≥ 9.5 mm) by day of treatment

<table>
<thead>
<tr>
<th>Daya</th>
<th>Class I (≤ 6 mm)</th>
<th>Class II (6.5–9 mm)</th>
<th>Class II (≥ 9.5 mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>AC</td>
<td>AM</td>
<td>CM</td>
<td>AC</td>
</tr>
<tr>
<td>0</td>
<td>2.1</td>
<td>3.3</td>
<td>2.2</td>
</tr>
<tr>
<td>3</td>
<td>3.1</td>
<td>3.7</td>
<td>2.2</td>
</tr>
<tr>
<td>7</td>
<td>4.9c</td>
<td>3.9c</td>
<td>1.5d</td>
</tr>
<tr>
<td>10</td>
<td>4.6c</td>
<td>5.0c</td>
<td>0.6d</td>
</tr>
<tr>
<td>14</td>
<td>4.0c</td>
<td>3.9c</td>
<td>4.0c</td>
</tr>
</tbody>
</table>

a Day = day of treatment; d 0 = start of treatment (MGA or carrier). 
bcAC = anestrous control; AM = anestrous MGA; CM = cycling MGA. 
cdMeans within a row in each size class having different superscripts are different, $P < 0.01$. 

Table 4. Proportion of cows ovulating within 7 d following the end of MGA treatment

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>AM</td>
<td>1/7 (14%)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1/9 (11%)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>2/16 (13%)&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>CM</td>
<td>3/7 (43%)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>6/9 (67%)&lt;sup&gt;c&lt;/sup&gt;</td>
<td>9/16 (56%)&lt;sup&gt;e&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<sup>a</sup>AM = anestrous MGA, CM = cycling MGA.
<sup>b,c,d,e</sup>Means within a column having different superscripts are different (<sup>b,c</sup>P < 0.02, <sup>d,e</sup>P < 0.01).

± 1.9 mm). In addition, when animals that ovulated following MGA treatment were compared to animals that did not ovulate following treatment, there was no difference (P > 0.05) in serum concentrations of estradiol (ovulated, 3.1 ± 0.8 and not ovulated, 3.6 ± 0.5 pg/mL), age of the follicular wave (ovulated, 4.0 ± 1.8 and not ovulated, 5.7 ± 1.1 d), or in the size of the dominant follicle (ovulated, 11.4 ± 2.0 and not ovulated, 10.0 ± 1.2 mm) on the last day of MGA treatment.

**Discussion**

Persistent follicles have been defined as dominant follicles having a prolonged life span, increased maximum diameter, and increased serum concentrations of estradiol (Siriois and Fortune, 1990; Savio et al., 1993a,b). In the present study, MGA treatment resulted in the formation of a persistent follicle (increased length of the follicular wave, increased maximum diameter of the dominant follicle, and elevated serum concentrations of estradiol-17β) in cycling but not anestrous beef cows at a similar stage postpartum. Similar results have been reported in postpartum dairy cows in which treatment of cycling and anestrous Jersey cows with low concentrations of progesterone increased the proportion of persistent follicles in cycling compared to anestrous cows (Rhodes et al., 1997). The increase in maximum diameter obtained by a persistent follicle does not appear to be caused by an increase in the growth rate of the follicle but instead by an increase in the duration of growth as seen in the present study and others (Cooperative Regional Research Project, 1996).

The presence of a persistent follicle (CM group) also decreased the numbers of follicles in small- (class 1) and medium- (class 2) sized follicles on d 7 and 10 of treatment. Suppression of small- and medium-sized follicles corresponded to the time when persistent follicles appeared to form. Similar results have been reported by administering low concentrations of progesterone by an intervaginal releasing device (Savio et al., 1993b). Persistent follicles suppress an increase in FSH (Adams et al., 1992) and thereby inhibit initiation of a new follicular wave. Once persistent follicles were no longer dominant, there is a transient rise in FSH and initiation of a new follicular wave. Follicular recruitment during the initiation of a new follicular wave does not appear to be affected by MGA treatment because similar numbers of follicles were recruited among treatments and among follicular waves. This is further supported by the fact that MGA treatment has been shown to have no effect on FSH concentrations during long-term treatment (10 d; Kojima et al., 1995).

The effect of MGA on follicular dynamics and serum concentrations of estradiol-17β in cycling postpartum beef cows in the present study was similar to results from previous studies with cycling cows and heifers (see review by Fortune and Rivera, 1999). Specifically, low concentrations of progesterone, in the absence of a corpus luteum, in cycling dairy cows and heifers resulted in formation of persistent follicles (Cooperative Regional Research Project, 1996). In addition, the feeding of MGA in the absence of a corpus luteum has been shown to cause formation of persistent follicles in cycling beef cows and heifers (Guthrie et al., 1970; Anderson and Day, 1994; Yelich et al., 1997).
The physiological mechanism underlying the difference between cycling and anestrous postpartum beef cows in response to MGA treatment is not known. However, a difference in the response of the hypothalamic-pituitary axis to MGA treatment seems to be a likely mechanism. Persistent follicles are reported to form in cycling cattle in response to an increase in the pulse frequency of LH (Duffy et al., 2000). Circulating LH pulse frequency was increased in cows with persistent follicles (Savio et al., 1993a), and infusion of exogenous LH into cycling postpartum cows induced formation of persistent follicles (Duffy et al., 2000). In addition, LH pulse frequency was increased in cycling postpartum dairy cows compared to anestrous postpartum cows when treated with low concentrations of progesterone (Rhodes et al., 1997). However, in anestrous postpartum dairy cows, LH pulse frequency was increased in response to progesterone treatment compared to the control group even though there was no difference among groups in incidence of persistent follicles (Rhodes et al., 1997). Consequently, an inability of dominant follicles to respond to increased LH pulse frequency in anestrous cows cannot be ruled out. Further elucidation of the endocrine mechanisms associated with a failure of persistent follicles to develop in anestrous beef cows treated with MGA awaits characterization of LH pulse frequency during the treatment period.

The relatively low proportion of cows (56%) that ovulated in the cycling MGA group within 7 d of MGA withdrawal was not unexpected. Kojima et al. (1995) reported that the proportion of cycling cows that had initiated a preovulatory surge of LH within 169 h following the end of long-term MGA treatment (0.5 mg-animal$^{-1}$·d$^{-1}$) was 86% and 23%, respectively, for cows that did or did not have a corpus luteum present during treatment. The mechanism underlying the delay from MGA withdrawal to the preovulatory surge of LH or ovulation in animals that did not have luteal tissue during MGA treatment is not known. However, we hypothesize that the delay in the preovulatory surge of LH or ovulation is due to the preceding extended period of elevated serum concentrations of estradiol-17β by the persistent follicle. Ozturk et al. (1998) reported that ovariecetomized ewes treated with physiological or pharmacological doses of estradiol for 2 to 12 d totally blocked the subsequent estradiol-induced LH surge in 87% of ewes treated. In the present study, a new follicular wave began in almost all of the animals in the CM group before MGA withdrawal and the dominant follicle that formed subsequent to the persistent follicle might not have produced sufficient amounts of estradiol-17β to induce a preovulatory surge of LH.

Progestin-based estrus synchronization systems reportedly induce an ovulatory estrus in prepubertal heifers (Hall et al., 1997) and anestrous cows (Yavas and Walton, 2000). Although the primary objective of the present study was not to critically examine the effectiveness of MGA (14 d) treatment on induction of ovulation, the proportion of anestrous postpartum cows that ovulated within 7 d following MGA was 13% (2 of 16). When prepubertal heifers were treated with MGA (10 d), there was no difference between the control and MGA-treated groups in the number of heifers that attained puberty by d 7 after MGA treatment. However, by d 10 following MGA treatment, 50% more of the treated heifers attained puberty compared to the control animals (Imwalle et al., 1998). To our knowledge, the ability of MGA treatment to induce an ovulatory estrus in anestrous postpartum cows has not been rigorously tested.

**Implications**

Melengestrol acetate is an orally active progestin that has been used to synchronize estrus in beef heifers and postpartum beef cows. In beef herds, an effective estrous synchronization protocol needs to synchronize a fertile ovulatory estrus in both cycling and anestrous postpartum cows. In the present study, the effect of melengestrol acetate on follicular dynamics differed between cycling and anestrous postpartum beef cows. Specifically, melengestrol acetate treatment resulted in formation of persistent follicles in cycling but not anestrous postpartum cows. Some of the variation in fertility at a synchronized estrus following progestin-based protocols may be due to the presence or absence of a persistent follicle, a characteristic that may be influenced by cycling status of postpartum cows.

**Literature Cited**


