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ESTROUS SYNCHRONIZATION AND FERTILITY IN GILTS AFTER 14- OR 18-DAY FEEDING OF ALTRENOGEST BEGINNING AT ESTRUS OR DIESTRUS

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Kansas State University, Manhattan 66506

Summary
A synthetic progestogen (altrenogest) was used to synchronize estrus in 160 Yorkshire and Duroc x Yorkshire gilts (6 to 11 mo old) in four trials. Gilts were fed 15 mg altrenogest for 14 or 18 d beginning either at or near estrus (d -1, 0, +1 or +2) or at diestrus (d +3 to +21). Mean intervals to estrus after treatment did not differ between 14- and 18-d treatments (5.4 ± 0.1 vs 5.3 ± 0.1 d, respectively), but were slightly longer (P<.01) in gilts beginning treatment at or near estrus than in diestrus (5.6 ± 0.1 vs 5.2 ± 0.1, respectively). Average intervals to estrus were similar between treatment groups and among stages of the cycle even though more gilts (P<.01) beginning treatment at estrus had serum progesterone concentrations >2 ng/ml at the end of progestogen treatment. Although more 18- than 14-d treated gilts (P<.05) were in estrus on d 5 post-treatment, proportions in estrus from 3 to 10 d post-treatment were similar (> 98%). Neither stage of estrous cycle at onset of treatment nor duration of progestogen treatment affected percentages of gilts farrowing (>73%), average gestation length (116.5 d), or total (9.7) and dead (.6) pigs at birth after artificial insemination at the post-treatment estrus. We concluded that 14-d feeding of altrenogest effectively synchronized fertile estrus in gilts regardless of stage of estrous cycle at the onset of progestogen treatment. Although 18-d treatment of gilts with altrenogest improved estrous synchronization precision compared to 14-d treatment, there was no advantage of the 18-d treatment for subsequent farrowing responses.

(Key Words: Progestogen, Estrous Synchronization, Swine, Progesterone, Farrowing Responses.)

Introduction
An effective method of synchronizing estrus in gilts would reduce labor required for estrous detection, reduce the number of replacement gilts retained and facilitate using artificial insemination and group farrowing. Webel (1978) recently reviewed ovulation control in swine. Generally, the past approaches have involved the use of orally active progestogens that suppress pituitary gonadotropin secretion, follicle growth and ovulation. Many progestogens were not satisfactory because they stimulated formation of cystic follicles and decreased fertility (First et al., 1963). But a new orally active progestogen [altrenogest, allyl trenbolone or RU-2267; 17α-hydroxy-17-(2 propenyl)estra-4,9,11-trien-3-one] has effectively synchronized estrus without producing cystic follicles or decreasing fertility at the first post-treatment estrus (First et al., 1963). But a new orally active progestogen [altrenogest, allyl trenbolone or RU-2267; 17β-hydroxy-17-(2 propenyl)estra-4,9,11-tren-3-one] has effectively synchronized estrus without producing cystic follicles or decreasing fertility (Webel, 1978; Davis et al., 1979; Kraeling et al., 1981; Pursel et al., 1981). However, at doses less than 15 mg/d for 18 d, cystic follicles developed after withdrawal of altrenogest.

Webel (1978) reported that feeding the progestogen for 18 d produced more precise synchronization of estrus than did 10, 12, 14 or 16 d of treatment, but only limited numbers of animals were treated. Stage of the cycle at which treatment began did not appear to affect synchronization of estrus. But gilts in or near estrus when treatment began might be expected to vary more in response than gilts at later stages of the estrous cycle because treatment might not alter estrous cycle length. Therefore, the objective of the present study was to deter-
mine the effect of duration of altrenogest feeding (14 vs 18 d) and stage of estrous cycle at the onset of treatment (estrus vs diestrus) on synchronization of estrus and fertility in gilts.

Experimental Procedure

One hundred and sixty gilts (Yorkshire and Duroc x Yorkshire) ranging from 6 to 11 mo old were used in four trials of 40 gilts each during October, November and December, 1980 and January, 1981. Gilts were randomly assigned by breed and age to four treatments in a 2 x 2 factorial design. Duration of altrenogest feeding (14 vs 18 d; 15 mg/d) and stage of the estrous cycle (estrus vs diestrus) at treatment onset were the main effects. Stage of estrous cycle before treatment was determined by observing daily (0800 to 1000 h) for estrus (d 0) using a boar. Pretreatment estrus was detected in all but two gilts. Estrous gilts (E) were those observed on d -1, 0, +1 and +2 of the cycle and diestrous gilts (D), on d +3 to +21 of the cycle when treatment was started. Estrous detection continued after treatment began to determine if any diestrous gilts began estrus. If so, such gilts were reclassified. Twice daily detection for estrus began 3 d after the last progestogen feeding, and gilts were artificially inseminated with at least 3 x 10^9 motile sperm from at least two boars in 100 ml egg yolk extender (Dziuk, 1958) at approximately 12 and 24 h after first detected post-treatment estrus.

Blood was collected from the anterior vena cava on the first and either the last day or the day after the last progestogen feeding. Serum progesterone concentrations determined by radioimmunoassay (Stevenson et al., 1981) were used to verify cycle stage and luteolysis. Blood also was collected from 13 control gilts (no progestogen) on days 14, 15, 16 and 17 of the estrous cycle and from 15 gilts on the same cycle days during progestogen feeding to determine the effects of treatment on serum progesterone concentrations during luteolysis.

Distribution of estrus (d 3 to 10 post-treatment), average interval from last feeding of altrenogest to estrus (interval to estrus), farrow-

<table>
<thead>
<tr>
<th>TABLE 1. ESTRUS AND FERTILITY IN GILTS TREATED WITH ALTRENOGEST FOR 14 OR 18 DAYS BEGINNING AT TWO STAGES OF THE ESTROUS CYCLEa</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stage of cycle</strong></td>
</tr>
<tr>
<td>-------------------</td>
</tr>
<tr>
<td>No. gilts treated</td>
</tr>
<tr>
<td>No. gilts with P_4&gt;2 ng/mlc</td>
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<tr>
<td>Days to estrus</td>
</tr>
<tr>
<td>No. gilts bred</td>
</tr>
<tr>
<td>Age at breeding, mo</td>
</tr>
<tr>
<td>No. bred gilts farrowing</td>
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<tr>
<td>Gestation length, d</td>
</tr>
<tr>
<td>Total pigs born</td>
</tr>
<tr>
<td>Born live</td>
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<tr>
<td>Born dead</td>
</tr>
</tbody>
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^aThere were no interactions between the main effects (P>.10).
^bStage of cycle at onset of progestogen treatment (estrus = d 0). Stage of cycle was not known in two gilts.
^cBlood was collected at the end of treatment; P_4 = progesterone.
^dGreater than diestrous gilts (P<.01).
^eGreater than 18-d treatment (P<.01).
^fPercentage of gilts inseminated.
ing rate (number of bred gilts that farrowed), gestation length and number of live and dead pigs at birth were recorded. Percentage data were analyzed by chi-square (Snedecor and Cochran, 1971). Other data were analyzed by least-squares procedures where breed, age at insemination, trial, duration of treatment, stage of the estrous cycle at the onset of progestogen treatment and duration of treatment x stage of the estrous cycle at the onset of progestogen treatment interaction were incorporated.

Results

Estrus was observed in 156 gilts (97.5%) between 3 and 10 d post-treatment. Four did not return to estrus within 10 d post-treatment and two that exhibited estrus were not detected in estrus before progestogen treatment. Synchronization was precise with 133 gilts (84.1%) in estrus on d 3, 4 and 5 post-treatment.

Duration of progestogen treatment had no effect on the interval to estrus (table 1) nor on distribution of estrus after treatment (figure 1). More gilts (P<.05) were in estrus on the peak day (d 5 post-treatment) in the 18-d treatment than in the 14-d treatment group (57.1 vs 34.6%, respectively). Although the distribution of estrus was skewed slightly to the right (figure 1) and more 14- than 18-d gilts had progesterone concentrations greater than 2 ng/ml at the end of treatment (P<.01), the mean interval to estrus was not affected by duration of progestogen feeding (5.4 vs 5.3 d, respectively; table 1). After 6 d post-treatment, 89 and 96% of gilts had expressed estrus in the 14- and 18-d treatment groups, respectively.

Stage of estrous cycle at the onset of treatment had a significant effect on synchronization precision. Average interval to estrus was longer (P<.01) in E than D gilts (table 1) and the distribution of estrus was skewed slightly to the right for the E gilts (figure 2). Average interval to estrus was probably extended in some E gilts by the inherent luteal function extending beyond the treatment period, which caused more E (P<.01) than D gilts to have serum progesterone concentrations greater than 2 ng/ml at the end of treatment (table 1). Despite differences in serum progesterone concentrations at the end of treatment, 88 and 96% of the E and D gilts were in estrus by 6 d post-treatment, respectively.

Serum progesterone concentrations in control gilts and in treated gilts declined (P<.01) between d 14 and 17 of the estrous cycle (figure 3). Serum progesterone concentrations, however, tended to be higher in treated gilts on d 15 to 17 than in control gilts, indicating that either feeding progestogen during late diestrus altered or delayed luteolysis or that a metabolite of the progestogen was competing with progesterone for antiserum binding sites.

Beginning progestogen treatment of E gilts seemed to have little effect on ovulation or luteal function because 39 of 42 E gilts (92.9%) bled between 7 and 14 d after progestogen treatment started had luteal phase concentrations of serum progesterone (>10 ng/ml). Two
Figure 3. Serum progesterone concentrations in control (n = 13; no progestogen) and progestogen-treated gilts (n = 15) on d 14, 15, 16 and 17 of the cycle (d 0 = estrus). Values are mean ± SE.

E gilts bled on d 14 after the onset of treatment apparently underwent early luteolysis because progesterone levels were less than 7 ng/ml, and ovulation may have been blocked in a third E gilt. But those apparent alterations in luteal function or maintenance did not adversely affect fertility because all three E gilts returned to estrus 5 or 6 d after treatment and farrowed at least 10 pigs each.

Percentages of gilts farrowing, gestation length and number of pigs at birth, (total, live or dead) were unaffected by duration of progestogen feeding (table 1). Similarly, stage of estrous cycle at the onset of treatment had little effect on farrowing responses (table 1), except there was a tendency for fewer E (P<.15) than D gilts to farrow (73.6 vs 84.1%, respectively). There were no significant interactions between treatment duration and stage of the estrous cycle at the onset of progestogen treatment for any of the factors measured.

**Discussion**

Our data demonstrate that estrus was synchronized effectively after treatment with altrenogest, as others (Webel, 1978; Kraeling et al., 1981; Pursel et al., 1981) have reported. Duration of treatment had no effect on the interval to estrus, but the 14-d treatment tended to spread the distribution of estrus (figure 1).

Although synchronization of estrus in the 14-d treatment was less precise than the 18-d treatment, more than 98% of the gilts were in estrus in 3 to 10 d post-treatment. Webel (1978) reported similar results. An 8- to 10-d breeding period is common in most swine operations, therefore, a 14-d treatment would produce acceptable results. A reduced feeding period would also reduce the amount of drug required and possibly simplify breeding herd management. In herds that farrow weekly, a group of gilts could be fed the last treatment and a new group started on approximately the same day sows are weaned.

Our data demonstrate that feeding altrenogest for 14 d is long enough for corpora lutea to regress and for gilts to come into estrus synchronously. When treatment was begun in E gilts, average interval to estrus and the distribution of estrus did not differ from D gilts (table 1 and figure 2). Although corpora lutea did not appear to have fully regressed (based on serum progesterone concentrations) at the end of treatment in E gilts (table 1), average interval to estrus increased only .4 d. Partial or complete luteal regression may have occurred in both E and 14-d gilts despite more of these gilts having serum progesterone greater than 2 ng/ml (table 1) because progestogen-treated gilts tended to have higher serum progesterone than untreated control gilts on d 15 to 17 of the estrous cycle (figure 3). This supports a previous observation of no difference in the interval to estrus between estrous gilts and randomly cycling gilts treated for 14 d (Webel, 1978).

Farrowing rates of nearly 80% with fresh semen in this study compare favorably with previous results with natural service in both progestogen-treated and control gilts (Webel, 1978; Pursel et al., 1981). Davis et al. (1979) reported similar fertilization rates (>95%) in control and progestogen-treated gilts after first post-treatment inseminations, with ovulation rates higher and subsequent litter size often greater (Webel, 1978) in progestogen-treated gilts than in control gilts. Litter size in our study, regardless of treatment, was acceptable for gilts and indicates that acceptable farrowing responses after synchronization with altrenogest should be achieved consistently.

We concluded that 14-d treatment of gilts with altrenogest resulted in a synchronized fertile estrus, regardless of estrous-cycle stage when treatment was started. Furthermore, although 18-d treatment with altrenogest produced more precise synchronization of
estrus than the 14-d treatment, a 4 d shorter treatment regimen may reduce costs and be more simply applied under farm conditions.

**Literature Cited**


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