ABSTRACT: We compared the effects of exogenous insulin and porcine ST (pST) on follicular development after weaning. Crossbred primiparous sows received saline (1.5 mL i.m.; n = 9), insulin (.4 IU/kg BW s.c.; Eli Lilly Lente Iletin II; n = 10), or pST (40 μg/kg BW i.m.; n = 10) from d 1 to 5 after weaning (d 0). Ovaries were collected, the diameter of each follicle ≥ 2 mm was measured, and fluid from the 20 largest follicles was assessed for IGF-I, IGFBP, estradiol, progesterone, and testosterone. The total number (27.7, 25.3, and 29.1 for saline, insulin, and pST, respectively; SEM = 3.2) and average diameter (4.7, 5.2, and 5.5 mm for saline, insulin, and pST treatments, respectively; SEM = .3 mm) of ovarian follicles were not affected by insulin or pST treatment. The pST and insulin increased follicular fluid estradiol and testosterone in medium and large follicles compared to fluid from saline-treated sows, but the increase was greater for insulin than for pST treatment (treatment × size interaction, \( P < .01 \)). Similarly, progesterone concentrations in follicular fluid were higher in medium and large follicles after insulin treatment, and pST treatment induced higher progesterone concentrations in small follicles and increasingly lower concentrations of progesterone in medium and large follicles (treatment × size interaction, \( P < .0007 \)) compared to saline treatment. Follicular fluid IGF-I was greater (treatment × health interaction, \( P < .0001 \)) in atretic and nonatretic follicles from pST-treated sows than in those from insulin- and saline-treated sows. Follicular fluid IGFBP-2 (tendency, \( P < .07 \)) and IGFBP, possibly representing IGFBP-5 (30 kDa) and IGFBP-4 (22 kDa), were higher in atretic follicles than in nonatretic follicles (\( P < .05 \)), whereas IGFBP-3 was not influenced by health status. The 30- and 22-kDa IGFBP were also influenced by treatment, increasing due to pST compared with saline or insulin treatments (\( P < .008 \)). Follicular fluid IGFBP-2 and IGFBP-3 were not influenced by treatment. In conclusion, pST and insulin positively influenced follicular steroidogenesis and possibly follicular development, although through different mechanisms.

Key Words: Sows, Insulin, Estradiol, Somatotropin, Insulin-Like Growth Factor, Follicles

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Introduction

Metabolic hormones, such as insulin and porcine somatotropin (pST), have been established as modulators of reproductive function. Insulin administered during preovulatory follicle development increased ovulation rate (Cox et al., 1987b) and decreased follicular atresia (Matamoros et al., 1990) in cyclic gilts and increased farrowing rate or litter size (Ramirez et al., 1997) in sows. In contrast, reports on the influence of in vivo administration of pST on reproduction in swine have been somewhat contradictory. Administration of pST to gilts decreased the number of gilts with a second estrus but increased ovulation rate in gilts exhibiting a second estrus (Kirkwood et al., 1988) or increased the number of medium-sized follicles (Echternkamp et al., 1994). In mixed-parity sows, pST given from 2 d before weaning until breeding had no effect on serum insulin concen-

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Weaning, and the intrafollicular IGF-I system in primiparous sows after insulin and pST on follicular development and the present study was to compare the actions of IGF-I and an increased number of medium-sized follicles (Echternkamp et al., 1994). The objective of the present study was to compare the actions of insulin and pST on follicular development and the intrafollicular IGF-I system in primiparous sows after weaning.

Materials and Methods

Animals

Thirty-two crossbred gilts (1/2 Landrace, 1/4 or more Yorkshire, 1/4 or less Duroc or Hampshire) were mated on the second postpubertal estrus and were maintained on a 14% protein, corn, and soybean meal diet (NRC, 1988) during pregnancy (2.27 kg/d), lactation (ad libitum), and after weaning (2.73 kg/d). Average feed intake (based on feed offered) during lactation was 5.8 ± 1 kg/d, and sows nursed 8.8 ± .3 pigs an average of 21.3 ± .3 d with no differences among treatments. Ten sows received .4 IU insulin/kg of BW s.c. (Eli Lilly Lente Iletin II); 11 sows received 40 μg pST/kg of BW i.m. (Monsanto, St. Louis, MO), and 11 sows received 1.5 mL saline s.c. for 5 d. This time period was chosen due to the rapid growth of potentially preovulatory follicles occurring then (Cox and Britt, 1982; Dyck, 1983) and the interval to estrus (6.9 ± .56) in our previous experiments (unpublished data). Doses of insulin and pST were chosen based on previous data from our laboratory (insulin) or data reported in studies from other laboratories (pST; Kirkwood et al., 1993). Treatments were given at the time of feeding and began the day after weaning (weaning = d 0). One pST-treated sow and one saline-treated sow ovulated before slaughter, and one saline-treated sow did not consume the allotted amount of feed during the treatment period, so data from these sows were excluded from all analyses.

Sampling

Sows were slaughtered by electrical stunning followed by exsanguination as approved by the Missippi State University Institutional Animal Care and Use Committee. Ovaries were removed approximately 4 h after the last injection. Blood samples were collected at slaughter for determination of glucose, LH, IGF-I, IGFBP, and ST concentrations. Blood was centrifuged for 20 min 4 h after sampling to collect serum. Serum samples were sent to USDA-ARS, Athens, GA, and assayed for LH with a RIA as previously described by Kesner et al. (1987) for detection of a possible LH surge that might confound the estradiol results. None of the animals sampled had LH concentrations indicative of the onset of a preovulatory surge. This information, combined with observation of estrual behavior and serum estradiol concentrations, implied that follicles were preovulatory.

Ovaries were aseptically collected and placed in sterile saline on ice for transport. Sampling was accomplished within 1 h after collection. The surface diameter of each follicle 2 mm or greater from each ovary was recorded without dissection. Fluid from the 20 largest follicles 2 mm or greater in diameter was aspirated using a 1-mL sterile syringe, centrifuged at 10 × g for 5 min, and transferred to preweighed vials for volume determination. The 20 largest follicles were considered to contain preovulatory population, but it is recognized that not all of the follicles would have ovulated.

Radioimmunoassay

Follicular fluid and serum estradiol concentrations were measured with RIA procedures previously validated in our laboratory (Cox et al., 1987a,b). Follicular fluid was diluted 1:500 in assay buffer. The sensitivity of the assay for follicular fluid was 1 pg/mL and the intra- and interassay CV were 8.74 and 10.55%, respectively. For serum estradiol, the intra- and interassay CV were 4.37 and 7.96%, respectively. Serum IGF-I was analyzed in one determination of a possible LH surge that might confound the estradiol results. None of the animals sampled had LH concentrations indicative of the onset of a preovulatory surge. This information, combined with observation of estrual behavior and serum estradiol concentrations, implied that follicles were preovulatory.

Concentrations of LH, IGF-I, IGFBP, and ST concentrations. Blood was centrifuged for 20 min 4 h after sampling to collect serum. Serum samples were sent to USDA-ARS, Athens, GA, and assayed for LH with a RIA as previously described by Kesner et al. (1987) for detection of a possible LH surge that might confound the estradiol results. None of the animals sampled had LH concentrations indicative of the onset of a preovulatory surge. This information, combined with observation of estrual behavior and serum estradiol concentrations, implied that follicles were preovulatory.

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standards were incubated with 125I-labeled hormone at 4°C for 18 to 20 h. The sensitivity of the progesterone assay was .075 ng/mL and the intra- and interassay coefficients of variation were 5.15 and 15.57%, respectively. The sensitivity of the testosterone assay was .025 ng/mL and the intra- and interassay CV were 10.47 and 14.03%, respectively.

**Results**

**Follicle Number and Size**

The total number (27.7, 25.3, and 29.1 for saline, insulin, and pST, respectively; SEM = 3.2) and average diameter of all ovarian follicles ≥ 2 mm were not affected by insulin or pST treatment. The average diameters of all follicles measured were 4.7, 5.2, and 5.5 mm (SEM = .3 mm) for saline, insulin, and pST treatments, respectively (P > .10). The total number of follicles and average diameter for each size class (small, medium, or large) were also unaffected by treatment (data not shown).

The number of follicles meeting the sampling criteria (up to 20 follicles that were ≥ 2 mm in diameter per sow) averaged 19.1 ± .3 and was not influenced by treatment. The diameter of the sampled follicles also was not influenced by treatment and averaged 5.4, 6.0, and 6.2 mm for saline, insulin, and pST treatments, respectively (SEM = .3 mm). The numbers of follicles comprising each of the three size categories were also not affected by treatment (data not shown). The percentages of follicles classified as atretic based on estradiol:testosterone ratio were not influenced by treatment and averaged 33.3%. The mean diameters of atretic follicles were lower (P < .0001) than those of nonatretic follicles but were not influenced by treatment (5.2 mm for atretic compared to 6.1 mm for nonatretic; SEM = .1 mm).

**Growth Hormone and Glucose**

Serum growth hormone was similar for saline- and insulin-treated sows but was increased in pST-treated sows (P < .0001) with mean concentrations of 2.12, 2.21, and 39.89 ng/mL for saline, insulin, and pST treatments, respectively (SEM = 2.27).

As expected, glucose concentrations were decreased by insulin (62.7 mg/dL) and increased by pST (113.8 mg/dL) compared to saline treatment (82.9 mg/dL; P < .005; SEM = 4.3).

**Steroid Hormones**

Follicular fluid estradiol concentration was influenced by the interactions of treatment × size class (P < .01) and treatment × health status (P < .03). Insulin and pST treatments increased (P < .03) estradiol concentrations in follicular fluid from medium and large follicles compared to saline treatment, but the increase was greater for insulin than for pST (P < .0001; Figure 1). Insulin treatment increased estradiol in atretic follicles compared to the other treatments, which had similar concentrations (P < .0001; Figure 2), and insulin and pST treatment increased estradiol in nonatretic follicles, although the increase was higher for insulin treatment (P < .0001; Figure 2). Serum estradiol was not influenced by treatment and averaged 5.3, 13.3, and 9.8 pg/mL for
Figure 1. Follicular fluid estradiol (upper panel), testosterone (middle panel), and progesterone (lower panel) concentrations in primiparous sows for saline, insulin, and porcine somatotropin (pST) treatment were influenced by a treatment × size class interaction \((P < .01)\). Size classes were small (2 to 3 mm), medium (4 to 6 mm) and large (7 to 10 mm) based on diameter. The SEM are in rectangles. Bars with different capital letters differ among size class in the same treatment, and bars with different lowercase letters differ among treatment in the same size class \((P < .05)\).

There was a treatment × size class interaction for testosterone \((P < .0001)\) such that insulin treatment resulted in lower testosterone in small follicles \((P < .002)\), and the insulin and pST treatments resulted in higher testosterone in medium and large follicles compared to saline, with a greater increase \((P < .03)\) for insulin than for pST treatment (Figure 1). Follicular fluid testosterone was also influenced by a treatment × health status interaction \((P < .02)\), with insulin treatment causing increased \((P < .004)\) testosterone in atretic compared to nonatretic follicles, and saline and pST treatments had similar levels of testosterone in atretic compared to nonatretic follicles. The atretic and nonatretic follicles had greater testosterone concentrations in insulin treatment compared to the other treatments \((P < .004)\), and pST was associated with increased testosterone in nonatretic follicles only \((P < .0001; \text{Figure 2})\).

Insulin and saline treatments resulted in similar patterns of follicular fluid progesterone concentrations in relation to follicle size class, although insulin
Table 1. Influence of follicle size on ratios of follicular fluid estradiol:testosterone and estradiol:progesterone in postlactational primiparous sows

<table>
<thead>
<tr>
<th>Ratio</th>
<th>Small</th>
<th>Medium</th>
<th>Large</th>
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</thead>
<tbody>
<tr>
<td>Estradiol:testosterone</td>
<td>.10</td>
<td>.18</td>
<td>.20</td>
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<tr>
<td>SEM</td>
<td>.08</td>
<td>.07</td>
<td>.09</td>
</tr>
<tr>
<td>Estradiol:progesterone</td>
<td>.12</td>
<td>.10</td>
<td>.10</td>
</tr>
<tr>
<td>SEM</td>
<td>.05</td>
<td>.04</td>
<td>.07</td>
</tr>
</tbody>
</table>

*Based on diameter: small = 2 to 3 mm, medium = 4 to 6 mm, large = 7 to 10 mm.

**Ratios different (P < .002).**

produced greater (P < .001) progesterone concentrations in medium and large follicles than saline treatment. In contrast, progesterone decreased as size class increased after pST, and there were greater (P < .02) progesterone concentrations in small and medium follicles than for saline treatment (treatment × size class interaction, P < .0007; Figure 1). Serum progesterone concentrations were not influenced by treatment and averaged .15 ± .03 ng/mL. Progesterone and testosterone in follicular fluid were positively correlated (r = .36; P < .0001).

Ratios of estradiol to testosterone and estradiol to progesterone were analyzed to estimate follicle health. As expected, both ratios increased with size class, with small follicles having the lowest estradiol:testosterone or estradiol:progesterone ratios and large follicles exhibiting the highest ratios (Table 1). Treatment did not influence either estradiol:testosterone ratios at 1.64 ± .61, 1.61 ± .67, and 1.74 ± .86 or estradiol:progesterone ratios at .40 ± .27, .57 ± .29, and .53 ± .38 for saline, insulin, and pST treatments, respectively.

**Insulin-Like Growth Factor I and Insulin-Like Growth Factor Binding Proteins**

Follicular fluid IGF-I concentrations were greater (treatment × status interaction, P < .0001) in atretic and nonatretic follicles from pST-treated sows compared to follicles from saline- and insulin-treated sows (Figure 2). In addition, IGF-I in pST-treated animals was greater (P < .0001) in nonatretic than in atretic follicles, and saline and insulin treatments produced similar IGF-I concentrations regardless of health status (Figure 2). Follicular fluid IGF-I content was also influenced by a treatment × size class interaction (P < .003). Follicular fluid IGF-I content increased with pST treatment in all size classes compared to the other treatments (P < .006; data not shown). Serum IGF-I concentrations were greater (P < .0005) for pST (572 ± 46.6 ng/mL) than for saline (298 ± 49.1 ng/mL) or insulin (286 ± 46.6 ng/mL) treatments. Follicular fluid IGF-I was weakly, positively correlated with the estradiol:progesterone (r = .13) and estradiol:testosterone (r = .20) ratios as well as the 30-kDa IGFBP (r = .13; P < .003).

Follicular fluid IGFBP-2 (tendency, P < .07) and IGFBP at 30 and 22 kDa (P < .05) were higher in atretic follicles than in nonatretic follicles, but IGFBP-3 was not influenced by health status (Figure 3). The 30- and 22-kDa IGFBP were also influenced by treatment, increasing due to pST treatment when compared to saline or insulin treatments (P < .008), but follicular fluid IGFBP-2 and IGFBP-3 were not influenced by treatment (Figure 3). None of the
binding proteins were influenced by size class (Figure 3). Serum IGFBP-3 tended to be increased (P < .10) by pST, but the lower molecular weight IGFBP (IGFBP-2, 30-kDa IGFBP, and 22-kDa IGFBP) in serum were not influenced by treatment (data not shown). The IGFBP-2 was negatively correlated with follicle diameter (r = −.13; P < .002) but was positively correlated (P < .0001) with IGFBP-3 (r = .24), the 30-kDa IGFBP (r = .46), and the 22-kDa IGFBP (r = .50). In addition, IGFBP-3 was positively correlated with diameter (r = .17; P < .0001).

Discussion

In the present study, administration of insulin and pST increased overall follicular fluid steroid production, with insulin generally more potent than pST. Several studies support an increase in estradiol due to insulin or pST treatment. Heifers treated with bovine somatotropin had higher serum estradiol concentrations than control heifers (Gong et al., 1991), and bovine granulosa cells exhibited increased estradiol production in response to insulin or bST plus insulin in vitro (Spicer and Stewart, 1996). In contrast, porcine granulosa cells in vitro had lowered or unchanged production of estradiol in response to insulin or pST treatments (Xu et al., 1995), and 4-mm porcine follicles exposed to insulin and follicles with no insulin treatment had similar concentrations of estradiol (Purvis et al., 1997). Insulin injections also failed to influence follicular estradiol production in crossbred, mixed-parity ewes (Kirkwood et al., 1991). These contrasting results may be due simply to differences between in vitro and in vivo situations as well as species or parity differences. However, a previous study from our laboratory also using primiparous sows indicated that insulin treatment dramatically decreased estradiol production in large follicles and did not affect estradiol in small- and medium-sized follicles (Whitley et al., 1997). The previous study used sows lactating for 30 d instead of 21 d and fed .45 kg less feed per day during the postweaning treatment period. Therefore, it is possible that metabolic balance before or during insulin treatment affects the potential of insulin to influence steroidogenesis. In support of this theory, insulin-infused ewes fed a low-quality straw diet had ovulation rates similar to those of control ewes but a slower rate of preovulatory FSH decline following luteolysis, possibly indicating delayed follicular growth and estradiol production (Downing and Scaramuzzi, 1997). The concept of metabolic balance or status altering effects of treatment with metabolic modifiers could be extended to pST treatment. For example, in vivo administration of pST decreased follicular fluid estradiol in lean prepubertal gilts but increased estradiol production in follicles of obese and geneti-

The increase in testosterone production due to insulin and pST treatment followed the pattern of estradiol production, as expected (Grant et al., 1989). Administration of testosterone from d 13 to estrus increased ovulation rate and embryo survival in gilts (Cárdenas and Pope, 1997), perhaps through increasing follicular fluid estradiol and(or) improving some unknown aspect of oocyte quality. If this is the case, production of androgen precursors such as testosterone would be a limiting factor controlling ovulation rate or embryo survival. Therefore, the increased follicular fluid testosterone and estradiol due to the insulin and pST treatments in the present study may indicate a potential for more ovulations and(or) improved embryo survival.

Many in vitro studies reported increased progesterone production in response to insulin. For example, Kirkwood et al. (1992) and Xu et al. (1995) reported increased progesterone production by porcine granulosa cells in response to 100 ng/mL of insulin, and Spicer et al. (1993) reported increased progesterone production by bovine granulosa cells. Insulin also increased progesterone secretion from whole porcine follicles (Purvis et al., 1997). Similarly, insulin administration increased progesterone production from medium and large follicles in the present study. However, pST treatment produced higher concentrations of progesterone in small and medium follicles but the concentration decreased in large follicles. Similarly, pST-treated prepubertal gilts, which have mostly small and some medium-sized follicles, had follicular fluid progesterone concentrations that tended to increase with dose and with daily administration compared to daily administration on alternate weeks (Bryan et al., 1992). Echternkamp et al. (1994) also found that pST administration to gilts over a period of 6 wk resulted in increased follicular fluid progesterone in small and medium follicles. In contrast, Spicer et al. (1992) found follicular fluid progesterone unaffected by pST treatment in gilts. In the present study, alterations in steroid hormone production in follicles of different size classes due to treatment indicates that steroidogenesis may be differentially regulated by insulin and pST. However, overall follicle health did not seem to be altered because ratios of estradiol:testosterone and estradiol:progesterone were not influenced by treatment.

The induction of steroidogenesis by pST could be attributed to the increase in follicular fluid IGF-I production. In vitro, IGF-I stimulates estradiol
and (or) progesterone production in granulosa cells from a variety of animals, including rats (Davoren et al., 1986), humans (Erickson et al., 1990), cattle (Spicer et al., 1993), and pigs (Yoshimura et al., 1994; Xu et al., 1995). The increased production of steroids by IGF-I could in turn contribute to follicle or ovum health and result in greater embryo survival. For example, in vitro exposure to IGF-I increased porcine granulosa cell progesterone production and percentage of total oocyte cleavage and improved embryo development beyond the 8-cell stage (Xia et al., 1994). However, in the present study, insulin also increased steroid production but without increasing follicular fluid IGF-I concentrations. Although the idea of insulin working through the IGF-I system is not supported by the present study, the possibility of increased IGF-I cross-reacting with the insulin receptor to induce similar but less potent responses has not been ruled out. However, the differential response of steroid hormones to insulin and pST in regard to follicle size class (with estradiol increasing over size class for both treatments but testosterone decreasing over size class with pST treatment) supports the idea of different primary modes of action for insulin and pST, not a common pathway working through the IGF-I system.

It is widely accepted that ovarian follicular fluid IGFBP-2 levels are associated with atresia. Negative relationships between IGFBP-2 and follicular growth have been demonstrated in postlactational sows (Howard and Ford, 1992) and cyclic gilts (Zhou et al., 1996). Using a more direct method of assessing atresia, IGFBP-2 was positively correlated with percentage of apoptotic granulosa cells in swine (Guthrie et al., 1995). In addition to IGFBP-2, IGFBP-4, and IGFBP-5 decrease during follicular growth (Mondshein et al., 1991; Howard and Ford, 1992; Monger et al., 1993), and IGFBP-2 and IGFBP-4 increase during follicular atresia (Grimes et al., 1994; Guthrie et al., 1995). Therefore, the increase in IGFBP-2 and the 22-kDa IGFBP corresponding with IGFBP-4 in atretic follicles and the negative correlation of IGFBP-2 with follicle diameter found in the present study are consistent with previous reports in swine, sheep, and cattle. The observed correlation of IGFBP-3 with diameter and the similar level of this binding protein in atretic and nonatretic follicles is also in agreement with previous studies (Howard and Ford, 1992; Grimes et al., 1994; Guthrie et al., 1995).

Although pST treatment increased presumed IGFBP-4 and IGFBP-5 in the present study, it did not affect IGFBP-2 (a more common marker of atresia) or number of atretic follicles and actually seemed to stimulate follicle growth, as evidenced by increased steroidogenic capacity of follicles in all size classes with a greater increase in medium and large follicles. Therefore, the vast increase in IGF-I concentration may have overwhelmed the capacity of the binding proteins and allowed follicular growth to continue unchecked. Although long-term (6 wk) pST treatment increased follicular fluid and serum IGFBP-3, decreased serum IGFBP-2, and increased follicular fluid IGFBP-2 in gilts (Echternkamp et al., 1994), our short-term administration of pST to sows did not influence IGFBP-2 or follicular fluid IGFBP-3 but tended to increase IGFBP-3 in serum, perhaps indicating that a longer treatment period is required to substantially alter IGFBP.

In summary, insulin and pST administration seemed to influence follicular growth in a positive manner, as evidenced by increased steroid production. However, the primary mechanism of action of the two metabolic hormones seems to be somewhat different and may target follicles at different stages of development. The increased IGF-I due to pST treatment may have induced the smaller follicles to become steroidogenic, but insulin did not increase IGF-I and did not influence steroid production in small follicles, pointing to a different mechanism of action. In addition, increased steroid production, as seen in medium and large follicles, may indicate superior maturity and health that could in turn lead to more ovulations or healthier oocytes.

Implications

The present results indicate that application of metabolic modifiers after weaning can alter preovulatory follicle hormone production. Administration of insulin and porcine somatotropin positively influenced follicular development, which might be indicative of increased follicle health. A healthy follicle may then relate to more healthy viable ova available to develop into more pigs born. Therefore, although not directly measured in the present study, metabolic hormones may have the potential to increase fertility in primiparous sows.

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