Maternal low-dose porcine somatotropin treatment in late gestation increases progeny weight at birth and weaning in sows, but not in gilts


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INTRODUCTION

Birth weight positively predicts piglet milk intake, growth and survival, feed efficiency, and carcass and meat quality (Hemsworth et al., 1976; Campbell and Dunkin, 1982; Dwyer et al., 1993; Rehfeldt and Kuhn, 2006). Daily pST injections from d 25 to 50 or d 30 to 43 of pregnancy increase fetal growth in gilts (first pregnancy) or mature sows, even at low doses of 2 mg/d (Sterle et al., 1995; Gatford et al., 2000, 2009). Continuing pST treatment to d 100 of pregnancy also increases progeny birth weight (Gatford et al., 2004, 2010). Shorter periods of maternal pST treatment in late pregnancy might also increase birth weight. Increased maternal pST from d 80 to term in gilts and d 80 to 94 in sows, increased birth weight but increased stillbirth rates of gilts (Rehfeldt et al., 1993; Trujillo-Ortega et al., 2006). Increased maternal pST from d 90 or 100 to term also improved progeny postnatal growth and viability markers (Kveragas et al., 1986; Etienne et al., 1992; Farmer et al., 1992). Electroporating the semiten-
dinosus muscle of gilts with a GHRH-expressing plasmid at d 85 of pregnancy, to increase endogenous pST production, increased piglet birth and weaning weights and growth rates from birth to 100 kg, with similar effects in subsequent parities (Khan et al., 2003; Person et al., 2008). Large-dose maternal pST treatment (6 to 10 mg/d) in very late pregnancy caused sow deaths, but 2 mg/d ending at d 100 did not (Kveragas et al., 1986; Cromwell et al., 1992; Gatford et al., 2010). Together, these studies suggest that maternal pST treatment in late gestation can increase piglet birth and weaning weights and postnatal performance, but large doses and continuation of treatment to term may increase stillbirths or maternal deaths. We therefore investigated whether daily maternal injection with lower doses of pST (~13 to 14 μg kg⁻¹ d⁻¹) from d 75 to 100 of pregnancy increases birth and weaning weight of progeny in sows and gilts under commercial conditions.

MATERIALS AND METHODS

The study was designed in accordance with the Australian Code of Practice for the Care and Use of Animals for Scientific Purposes (National Health and Medical Research Council of Australia, 2004) and approved by the University of Adelaide and the Rivalea Australia (formerly QAF Meat Industries) Animal Ethics Committees. The studies were conducted at Rivalea Australia, Corowa, New South Wales.

Animals

Large White × Landrace (PrimeGro Genetics) parity 0 sows (gilts) were mated at 32 wk of age (average 221 ± 1.2 d), and mature Large White × Landrace (PrimeGro Genetics) sows (parities 2 or 3 at mating) were mated at the first postweaning estrus. Gilts were group housed before mating, commenced boar exposure at 29 wk of age, and were then bred on estrus occurring between 30 and 35 wk of age. All gilts were fed a gestating sow diet (13.5 MJ of DE/kg, 16.0% CP, 0.80% total lysine) ad libitum before mating. Gilts and sows bred over a 12-wk period were subsequently allocated to treatment, with approximately equal numbers of sows and gilts allocated to each treatment in each replicate week of the study (2 × 2 factorial design, n = 69 to 74 dams per treatment and parity in total, 284 dams allocated to entire study). All matings occurred by AI using 3 × 10⁹ sperm cells/mL. Mixed boar semen was used from a commercial Large White × Landrace cross, supplied by PrimeGro Genetics, Rivalea, Australia. Gilts were served in an AM/PM/AM mating regimen. Weaned sows were mated in an AM/AM regimen. Gilts and sows were housed in groups of 75 (2.7 m² per sow) in ecoshelters (Croft Polythene Greenhouses, Yarra Glen, Australia) within 6 d of mating until entry to the farrowing house at approximately d 109 of pregnancy.

Nutrition and Treatments

Gilts and sows were housed together and fed 2.7 kg·d⁻¹ of a dry sow diet (13.5 MJ of DE/kg, 16.0% total protein, 0.80% total lysine), once daily in the morning during group housing and until entry to the farrowing shed. After confirmation of pregnancy, gilts and sows were ranked by BW within parity for each replicate week of the study and allocated alternately to each treatment group. Control gilts and sows were not injected to enable comparison with performance under current commercial management. Gilts and sows allocated to the pST-injection groups were injected intramuscularly daily with 1 mL of sterile water containing 2.5 or 4.0 mg of pST, respectively, from d 75 of pregnancy until d 100 (referred to as treatment pregnancy). Dams were housed in mixed-treatment and mixed-parity groups, and treatment group was identified using colored ear tags. Each animal was tagged after the daily injection using spray paint with alternating sites and colors. This pST dose increased fetal growth and progeny birth weight in gilts and sows when continued from d 25 to 100 of pregnancy (Gatford et al., 2010). In the present study, this treatment delivered a dose of 12.9 ± 0.1 μg of pST·kg⁻¹·d⁻¹ in gilts and 14.2 ± 0.1 μg of pST·kg⁻¹·d⁻¹ in sows at the start of treatment at d 75 of pregnancy. Dams entered the farrowing house at d 110.4 ± 0.1 of pregnancy and were fed 3 kg·d⁻¹ of a lactation diet (14.9 MJ of DE/kg, 19.2% total protein, 0.89% total lysine) until farrowing, and were then fed 3 times per day to appetite with this lactation diet until weaning. Gilts and sows farrowed naturally at term (no induction was performed). Within 24 h of farrowing, a minimal cross-fostering approach was used to equalize litter size to 11 piglets in gilt litters and 12 piglets in sow litters where possible. Fostering was performed across all treatment groups to have sufficient numbers of piglets available to equalize litter sizes. Dams were weaned in the fourth week of lactation on a set day each week at 26.9 ± 0.1 d after birth. Dam removals and reasons for removal were recorded throughout the treatment pregnancy and subsequent lactation. Farrowing rates of all dams were recorded for the subsequent mating, and litter sizes were recorded for all dams that became pregnant and farrowed from the first mating after the treatment pregnancy and lactation.

Maternal and Progeny Measures

Pregnant dams were weighed at d 70 of pregnancy, on transfer to the farrowing shed at d 104 to 111 of pregnancy, and at weaning. Backfat depth was measured on the same days by ultrasound at the P2 site (65 mm from
the midline over the final rib, using linear array 3.5-mHz live ultrasound; Noveko, Montreal, Canada). A subset (~60% of dams to minimize disturbance of dams on day of birth) was weighed on the day of farrowing for each replicate week of the study. Numbers of live piglets, still births, and mummified piglets were recorded within 24 h of birth. Total litter weight (live born) and number were recorded on the day of delivery, after fostering, at d 14 of lactation, and at weaning. Preweaning piglet losses, reasons for removal, and dates at removal were recorded throughout lactation.

**Statistical Analyses**

The effects of maternal parity group (gilts vs. sows), treatment, and their interaction on maternal and litter outcomes were analyzed using a 2-way ANOVA model. Replicate week was also included as a factor in full factorial statistical models in analyses of maternal weight, P2 backfat depth, and lactation feed intake, measures of which varied between replicate week. Analyses of litter average birth weight at birth included litter size as a covariate. Outcomes for individual progeny were analyzed using a repeated-measures mixed model, including maternal parity group and treatment (between factors), progeny sex (within factor), and interactions between these factors, including total litter size at birth as a covariate (for birth weight only), and treating litters as repeated measures on the dam. Only piglets suckled by their birth mothers were included in repeated measures analysis of weaning weight and growth rate. Chi-squared analysis was used to separately test overall effects of maternal parity group and treatment, and of treatment within each parity group, on dam removals. All tests were carried out using PASW Statistics 17.0 (SPSS Inc., Chicago, IL), and data are presented as mean ± SEM unless otherwise indicated.

**RESULTS**

**Maternal BW and Backfat Depth**

Maternal BW (Figure 1) did not differ between control and pST-treated dams before treatment ($P > 0.4$), at farrowing house entry ($P = 0.17$), or immediately after farrowing ($P > 0.7$) and was greater in sows than gilts throughout the study ($P > 0.001$ at each time). Maternal BW was not affected by replicate week before treatment start at d 71 ($P = 0.2$), nor after farrowing or weaning (each $P > 0.7$), whereas BW at farrowing house entry did differ between replicate weeks ($P = 0.002$, data not shown). At weaning, pST-treated dams tended to be heavier than control dams ($P = 0.087$; control: 240 ± 2 kg; pST-treated: 245 ± 2 kg). During late gestation (d 71 to farrowing house entry, encompassing treatment period), maternal BW gain was greater in pST-treated than in control dams ($P > 0.001$; control: 24.6 ± 0.8 kg; pST-treated: 29.6 ± 0.8 kg) and was greater in gilts than in sows ($P > 0.001$; gilts: 30.5 ± 0.9 kg; sows: 23.8 ± 0.9 kg), whereas treatment effects did not differ between parity groups ($P > 0.5$; data not shown). Body weight change between farrowing house entry and weaning was not affected by maternal treatment ($P > 0.5$) and BW loss was greater in sows than gilts ($P = 0.001$, data not shown). Lactation BW change (day after farrowing to day of weaning) was similarly unaffected by maternal treatment ($P > 0.7$; data not shown) and BW loss was greater in sows than gilts ($P = 0.040$; gilts: −9.0 ± 2.5 kg; sows: −16.2 ± 2.5 kg).

Maternal P2 backfat depth (Figure 1) was less in pST-treated than control dams on entry to the farrowing house in late pregnancy ($P = 0.003$), but did not differ between treatment groups before treatment or at weaning ($P > 0.4$ for each), and was greater in sows than in gilts throughout the study ($P < 0.001$ at each time). Maternal P2 backfat depth varied between replicate weeks at d 71 of pregan-
cy ($P = 0.010$), and at entry to the farrowing house and after weaning (each $P < 0.001$, data not shown). During late gestation (d 71 to farrowing house entry, encompassing treatment period), P2 backfat loss was greater in pST-treated dams than control dams ($P < 0.001$; control: $-0.10 \pm 0.30$ mm; pST-treated: $-1.76 \pm 0.30$ mm) and greater in sows than gilts ($P = 0.001$, data not shown), whereas effects of pST treatment were similar between parity groups ($P > 0.3$). Between farrowing house entry and weaning, P2 backfat depth change (overall mean: $-1.88 \pm 0.29$ mm) was similar between treatment groups ($P > 0.2$).

### Maternal Lactation Feed Intake

Daily lactation feed intake did not differ between control and pST-treated dams ($P > 0.7$; Figure 2) and increased more with advancing lactation in sows than in gilts ($P = 0.011$, linear contrast), such that daily feed intake was greater in sows than in gilts from the second week of lactation (wk 1: $P > 0.1$; wk 2: $P = 0.015$; wk 3: $P = 0.002$; wk 4: $P = 0.001$; Figure 2).

### Maternal Removals During Pregnancy and Lactation

Between d 75 of pregnancy to scheduled farrowing house entry, similar proportions of dams were removed from each treatment group ($P = 0.594$; control: 5 of 139, 3.6%; pST-treated: 5 of 146, 3.4%). Of the 10 dams removed after treatment commenced, 2 were not pregnant, 3 aborted between d 75 and 100 of gestation, 3 died suddenly, and 2 were culled for soundness. Similarly, dam removals during lactation were unaffected by maternal treatment ($P = 0.506$; control: 21 of 139, 15.1%; pST-treated: 23 of 146, 15.8%). Of the 44 sows culled at or after farrowing, 4 were culled for lameness (1 control, 3 pST-treated), 2 died suddenly (2 pST-treated), 22 were culled for lactation failure (8 control, 14 pST-treated, $\chi^2$ treatment effect = 0.225), 7 were culled for farrowing failure (7 control, 0 pST-treated, $\chi^2$ treatment effect = 0.006, this includes not pregnant, still born litters, and maternal complications during or resulting from farrowing), and 9 were culled as the result of loss of litters because of disease (5 control, 4 pST-treated).

### Litter Outcomes for Treatment Pregnancy

Gestation length did not differ between treatments ($P > 0.8$) and tended to be shorter in sows than in gilts (sows: $116.4 \pm 0.1$ d; gilts: $116.7 \pm 0.1$ d; $P = 0.092$). Maternal pST treatment did not alter the numbers of total or live-born piglets ($P = 0.541$ and $P = 0.231$, respectively; Table 1). Effects of maternal pST treatment on total and live-born litter sizes were also not significant in gilts ($P > 0.8$ and $P > 0.9$ respectively) or in sows ($P > 0.5$ and $P = 0.128$ respectively) when analyzed separately. Maternal pST treatment tended to affect numbers of stillborn piglets in a parity group-dependent manner (treatment × parity group interaction, $P = 0.082$; Table 1). In gilts, maternal pST treatment did not affect the number of stillborn piglets ($P > 0.8$; Table 1), but in sows, maternal pST treatment increased the number of stillborn piglets by 0.4 piglets per litter ($P = 0.034$; Table 1). Average piglet birth weights (Table 1) were increased by pST treatment in sows ($+96$ g, $P = 0.034$), but not in gilts ($P > 0.5$) and were greater in progeny of sows than in progeny of gilts ($+157$ g, $P < 0.001$), with similar results when birth weight was corrected for total litter size at birth ($P < 0.001$, Table 1). Sows tended to suckle more piglets than gilts after fostering ($P = 0.064$; gilts: $8.1 \pm 0.2$; sows: $9.5 \pm 0.2$) and remained so at weaning ($P < 0.001$; gilts: $7.7 \pm 0.2$; sows: $9.1 \pm 0.2$). Litter size from fostering to weaning was greater in sows than in gilts ($P < 0.001$), and this parity group difference in litter size thus became very significant at d 14 of lactation ($P < 0.001$; gilts: $8.1 \pm 0.2$; sows: $9.5 \pm 0.2$) and remained so at weaning ($P < 0.001$; gilts: $7.7 \pm 0.2$; sows: $9.1 \pm 0.2$). Litter size from fostering to weaning was not affected by maternal treatment during the preceding pregnancy ($P > 0.2$ at each age, data not shown).

Despite the larger litters being suckled by sows, average, as well as total, litter weight was greater in sow litters than in gilt litters at each age ($P > 0.001$ for each; Figure 3). Effects of treatment on litter weight were not significant overall ($P > 0.19$ for each) and tended to vary with parity group postfostering ($P = 0.072$) and at wean-
ing ($P = 0.082$). We therefore analyzed effects of maternal pST treatment on litter weight separately within each parity group. In gilts, maternal pST treatment did not affect litter average progeny weight (Figure 3) in their litter postfostering ($P > 0.7$), at d 14 of lactation ($P > 0.4$), or at weaning ($P > 0.5$). In sows, litter average progeny weight (Figure 3) was greater in litters of pST-treated mothers than in control mothers postfostering (+93 g, $P = 0.025$) and at weaning (+430 g, $P = 0.038$), but was not different from progeny of control sows at d 14 of lactation ($P > 0.4$).

**Individual Piglet Responses**

Consistent with analysis of litter average weights, effects of maternal treatment on individual piglet birth weights differed between parity groups (Table 2). In gilt progeny, individual birth weights were negatively related to total litter size ($P > 0.8$) and was greater in pST-treated sows than in control sows ($P = 0.034$). The average litter birth weight did not differ between treatments in gilts ($P > 0.5$) and was greater in pST-treated sows than in control sows ($P = 0.038$). The average litter birth weight, corrected for total litter size, did not differ between treatments in gilts ($P > 0.5$) and was greater in pST-treated sows than in control sows ($P = 0.039$). Corrected to an average total litter size of 12.29 piglets overall and within each parity group (gilts: 11.65 piglets born, sows: 12.88 piglets born) for analyses of the treatment × parity group interaction.

![Figure 3](image)

**Figure 3.** Effects of maternal porcine ST (pST) treatment in late pregnancy (d 75 to 100) and parity group on litter average piglet weights during lactation. Data are means and SEM of average data for each litter at each postnatal age, shown separately for gilts (plain bars) and sows (cross-hatched bars) in control (white bars) and pST-treated groups (shaded bars). Litter average piglet BW include all piglets suckled by a dam (i.e., piglets suckled by the birth dam as well as piglets fostered onto the dam). Significance values for effects of parity group and maternal pST treatment in sows only are shown for each postnatal age.
suckled by sows (gilt progeny: $167 \pm 8 \text{ g} \cdot \text{d}^{-1}$, sow progeny $205 \pm 7 \text{ g} \cdot \text{d}^{-1}$; $P > 0.001$). Overall, effects of maternal pST treatment on piglet ADG varied with piglet sex ($P = 0.040$) but not with parity group ($P > 0.9$). Maternal pST treatment did not affect ADG in male piglets ($P > 0.6$) but tended to increase ADG in female piglets ($+20 \text{ g} \cdot \text{d}^{-1}$, $P = 0.082$).

**Subsequent Reproductive Performance of Dams**

Weaning-remating interval did not differ between treatment groups ($P > 0.4$) and tended to be longer in those that were gilts during the treatment pregnancy than those that were treated as sows (gilts: $74.4 \pm 0.68 \text{ d}$, sows: $5.64 \pm 0.68 \text{ d}$; $P = 0.064$). Farrowing rates were similar in sows that had been controls or that were pST-treated in the previous (treatment) pregnancy (74 and 78%, respectively, $P = 0.293$) and were similar between parity groups (73 and 79%, respectively; $P = 0.159$). Maternal treatment and parity group did not affect numbers of live-born ($11.68 \pm 0.23$), stillborn ($0.73 \pm 0.08$), or mummified ($0.23 \pm 0.04$) piglets in the posttreatment litter (each $P > 0.2$).

**DISCUSSION**

Daily maternal injection with pST for 25 d in late gestation at $\pm 13$ to $14 \mu$g of pST-$\text{kg}^{-1} \text{d}^{-1}$ increased birth weight of progeny, but only in sows, with no increase seen in the birth weight of progeny from gilts. Even in sows, late-gestation pST only increased average birth weight of progeny by $96 \text{ g}$, a smaller increase than the $180 \text{ g}$ gain in average progeny birth weight seen in sows treated with pST from d 25 to 100 of pregnancy under similar commercial conditions (Gatford et al., 2010). In contrast to this smaller gain in birth weight, late-gestation pST treatment gave greater gains in average progeny weaning weight (sow progeny, $+430 \text{ g}$) than shown previously using sustained pST (sow progeny, $+251 \text{ g}$), although once again this was only in sows, whereas sustained pST also increased the weaning weight of progeny of gilts ($+340 \text{ g}$) in our previous study (Gatford et al., 2010, 2010). The effects of pST itself may actually be slightly greater than apparent in these studies, given that comparisons are with untreated control pigs, and any stress due to repeated maternal injections would probably decrease fetal weight.

Differential growth responses in sows and gilts suggest different capacities to increase fetal nutrient supply in response to late gestation pST. In the present study, sows and gilts were group housed and fed at the same rates during pregnancy, which because of their greater body size would be expected to be more restrictive in sows. Indeed, P2 backfat decreased during mid

**Table 2. Effects of maternal late pregnancy porcine ST (pST) treatment, parity group, and piglet sex on individual piglet outcomes**

<table>
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<tr>
<th>Outcome</th>
<th>Sows</th>
<th>Pigs</th>
<th>Treatment</th>
<th>Parity</th>
<th>Piglet sex</th>
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<td>422</td>
<td>420</td>
<td>392</td>
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<td>Birth weight, kg</td>
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<td>6.63 ± 0.19</td>
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<td>Weaning weight, kg</td>
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<td>18.8 ± 2.1</td>
<td>18.7 ± 2.1</td>
<td>20.1 ± 2.1</td>
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<tr>
<td>ADG, weaning, g/d</td>
<td>16.1 ± 2.1</td>
<td>15.9 ± 2.1</td>
<td>18.8 ± 2.1</td>
<td>18.7 ± 2.1</td>
<td>20.1 ± 2.1</td>
</tr>
</tbody>
</table>

**Footnote:**
1. This row indicates the number of piglets within each group included in analyses of the T × P.
2. Piglet birth weight did not differ between treatments in gilts ($P > 0.5$) and was greater in pST-treated sows than in control sows ($P = 0.029$).
3. Piglet weaning weight did not differ between treatments in gilts ($P > 0.4$) and was greater in pST-treated sows than in control sows ($P = 0.015$).
4. Piglet ADG from birth to weaning did not differ between treatments in male piglets ($P > 0.6$) and tended to be greater in female progeny from pST-treated sows than in those from control dams ($P = 0.029$).
5. Piglet ADG from birth to weaning did not differ between treatments in male piglets ($P > 0.6$) and tended to be greater in female progeny from pST-treated sows than in those from control dams ($P = 0.029$).
to late pregnancy in sows, suggesting that this feeding regimen might be limiting for sows. Greater birth weight increases in progeny of sows than in gilts in these studies might therefore reflect a greater capacity to mobilize maternal reserves and increase maternal plasma nutrient concentrations, possibly in part because of decreased rates of lean tissue deposition in the dam, or because of a greater capacity to increase nutrient transfer across the placenta in response to maternal pST treatment in sows than in gilts. In our previous studies of pST treatment in early-mid gestation, maternal plasma IGF-I concentrations increased similarly and maternal and fetal plasma urea concentrations decreased similarly in sows and gilts (Gatford et al., 2009). Maternal plasma insulin and maternal and fetal plasma glucose were not altered by pST treatment during early-mid gestation in either parity (Gatford et al., 2009). Dams were not blood sampled in the present study, although effects of pST treatment on BW and P2 backfat depth, which are crude measures of maternal nutrient availability, as often occurs in commercial management systems.

Our results do confirm that maternal pST treatment can increase fetal growth even under conditions of limited maternal nutrient availability, as often occurs in commercial management systems.

Similar to birth weight responses to pST, late-gestation maternal pST increased weaning weight in progeny of sows, but not in progeny of gilts, in the present study. The increase in average weaning weight in progeny of sows treated with pST in late gestation (+430 g) was greater than that shown previously (+251 g) after sustained maternal pST treatment from d 25 to 100 of gestation (Gatford et al., 2010). This is somewhat surprising given the smaller increase in birth weight after late-gestation pST treatment (+96 g) than that shown after sustained maternal pST treatment (+180 g) from d 25 to 100 of pregnancy (Gatford et al., 2010). These suggest that the growth of the heavier piglets from sows after longer pST treatment is constrained by milk production capacity of sows during lactation. Maternal feed intake during lactation was not altered by pST treatment from d 75 to 100 in the present study or from d 25 to 100 in our previous study (Gatford et al., 2010). Decreased maternal reserves at farrowing after the longer-term pST treatment also does not appear to explain the inability to maintain piglet BW differences, because the decrease in P2 backfat depth was similar after pST treatment from d 75 to 100 or d 25 to 100 (both ~2 mm less than controls at ~d 110 of pregnancy), and maternal BW at farrowing was increased by long-term pST treatment but not by late gestation pST treatment (Gatford et al., 2010). Analysis of data for individual piglets in the present study also showed that although birth weight responses to late-gestation pST were greatest in male piglets, effects on weaning weight were similar between sexes and growth rates during lactation tended to be increased by pST in female but not male piglets, suggesting a greater capacity for improved preweanling growth rates in female piglets.

Late-gestation pST treatment did not affect gilt or sow removal rates during or after treatment in the present study. In contrast, sustained pST treatment from d 25 to 100 of pregnancy increased removal of sows after weaning, due to foot and leg problems or poor condition, although removal rates of gilts were not affected by sustained pST treatment (Gatford et al., 2010). Weaning-remating interval, conception rates, and litter size in the subsequent pregnancy were not altered by late pregnancy pST treatment (d 75 to 100) in our current study or by sustained pST treatment (d 25 to 100) in our previous study (Gatford et al., 2010), in gilts or sows. Improved sow retention, as well as greater increases in progeny weaning weight, suggest that restricting pST treatment to late gestation may be more economically beneficial than sustained pST treatment from early to late pregnancy in mature sows, although effects on progeny performance after weaning remain to be evaluated.

Together with the results of previous studies, our results suggest that maternal pST treatment restricted to late gestation does not increase progeny birth or weaning
weights in gilts unless high doses are used, which have adverse effects on stillbirth rates, at least when continued to term (Trujillo-Ortega et al., 2006). Therefore, approaches to increase endogenous pST throughout most of gestation will probably be needed to increase birth weight and postnatal growth of gilts. In contrast, this shorter period of maternal pST treatment increases progeny birth weight in sows, albeit to a lesser extent than when maternal pST treatment is continued from early to late pregnancy. Late-gestation pST treatment increased weaning weight of sow progeny to a greater extent than when maternal pST treatment is continued from early to late pregnancy. Late-gestation pST treatment increased weaning weight of sow progeny to a greater extent than seen after sustained maternal pST treatment through most of gestation, without the adverse effects on sow retention seen after sustained pST treatment. Whether increasing maternal pST concentrations in late gestation only is the best strategy for sow management in commercial production systems will depend on postweaning performance responses of progeny.

LITERATURE CITED


