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Exogenous porcine somatotropin administered to neonatal pigs at high doses can alter lifetime fat but not lean tissue deposition

Frank. R. Dunshea[1,2]*, Danny Suster[1], Douglas J. Kerton[1] and Brian J. Leury[2]

[1]*Victorian Institute of Animal Science, Werribee 3030, Australia*
[2]*The University of Melbourne, Melbourne 3010, Australia*

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The growth rate of the young pig is generally much less than its potential and may be constrained by endocrine status as well as nutrient intake. The aim of the present study was to determine whether porcine (p) somatotropin (ST) treatment of the sucking pig could alter subsequent body composition. Twelve mixed-parity cross-bred sows with an average litter size of ten piglets were used to nurse pigs for the present study. On day 1 of lactation, the median two male pigs (by weight) from each litter were randomly allocated to one of two doses of pST (0 or 1 mg/kg per d) until weaning on day 21. Pigs were weaned and offered feed *ad libitum* until slaughter at 134 d of age. Body composition was measured using dual-energy X-ray absorptiometry (DXA) at 21, 49, 77, 105 and 133 d of age. There was no significant difference in growth rates between day 1 and 21 of lactation in pigs injected with either saline (9 g/l NaCl/l) or pST (258 *v.* 246 g/d for control and pST-treated pigs respectively, $P=0{\cdot}61$), and as a consequence there was no significant difference in liveweight at weaning (7·13 *v.* 6·84 kg, $P=0{\cdot}59$). However, fat mass at weaning tended to be decreased (1·18 *v.* 0·96 kg, $P=0{\cdot}064$), while the % fat in the body at weaning was significantly (16·7 *v.* 13·9 %, $P=0{\cdot}008$) decreased by exogenous pST treatment. In the immediate post-weaning period there was a reduction in lean tissue deposition (347 *v.* 300 g/d, $P=0{\cdot}021$) but no effect on fat deposition (35 *v.* 33 g/d, $P=0{\cdot}72$). Over the entire weaning-to-slaughter period, pST treatment of neonatal pigs decreased the rate of fat deposition (130 *v.* 112 g/d, $P=0{\cdot}033$), but had no effect on lean tissue deposition (550 *v.* 538 g/d, $P=0{\cdot}49$). Therefore, treatment of nursing pigs with high doses of pST for a short period before weaning may provide a means of reducing the fat content of pork and pork products.

Lactation: Somatotropin: Growth: Body composition: Neonate: Pig

The growth rate of the young pig is generally less than half its potential (Boyd *et al.* 1995) and may be constrained by endocrine status as well as nutrient intake. Although porcine (p) somatotropin (ST) increases lean tissue growth and decreases fat growth in grower and finisher pigs, the response to pST is often much less in younger pigs (Campbell *et al.* 1991). For example, young weaned pigs (about 10·0 kg, age not given) did not exhibit any growth response to pST until after at least 10 d of treatment and even then the response was inconsistent (Harrell *et al.* 1997). This was despite elevated plasma insulin-like growth factor (IGF)-I and insulin levels and reduced plasma urea as a result of only 5 d with pST treatment (Harrell *et al.* 1997). In addition, pST administration at the doses used in finisher pigs (0·06 mg/kg) failed to increase plasma IGF-I or growth in neonatal sucking pigs, although there was limited evidence of subsequent growth responses (Dunshea *et al.* 1999). In contrast, Wester *et al.* (1998) found that a relatively high dose of exogenous pST (1 mg/kg) increased plasma IGF-I and growth over the first 7 d of life in artificially reared pigs.

The ontogeny of somatotropin and its receptors in the neonate has been well studied in a variety of species, although results specific to the pig are relatively scarce. Plasma ST in the young pig is very high around parturition, declines rapidly over the first week after birth, then remains constant until the second week of life before gradually increasing again over the next 5 weeks and declining once again (Buonomo & Klindt, 1993; Matteri & Carroll, 1997). ST then gradually declines up to at least 30 weeks of age (Klindt & Stone, 1984; Owens *et al.* 1991; Harrell *et al.* 1997). These patterns of plasma growth hormone

Abbreviations: DXA, dual-energy X-ray absorptiometry; IGF, insulin-like growth factor; p, porcine; ST, somatotropin.

* **Corresponding author:** Associate Professor Frank R. Dunshea, fax +61 3 9742 0400, email Frank.Dunshea@nre.vic.gov.au

concentrations are essentially the same as *in vitro* basal and growth hormone releasing hormone-stimulated ST release from cultured pituitary cells (Matteri & Carroll, 1997). ST receptor mRNA has been found in the liver of the fetal (Duchamp *et al.* 1996) and neonatal (Brameld *et al.* 1995) pig and it increases over at least the first 20 d of life (Owens *et al.* 1990). Therefore, it appears that the ST receptor gene is being transcribed, but it is not known whether there are any functional receptors being produced. Alternatively, there may be some functional receptors that are resistant to ST but that may respond to high doses of exogenous pST.

The carry-over effects of pST treatment of the neonatal animal on growth and body composition are unclear. Evidence exists from *in vitro* studies that pST may inhibit pre-adipocyte differentiation and proliferation (Ramsey *et al.* 1987; Gerfault *et al.* 1999). If this were the case *in vivo*, neonatal pST treatment may have subsequent effects on fat deposition that would have important practical implications. Therefore, the aim of the following study was to determine whether a high dose of exogenous pST administered to neonatal pigs would alter pre-weaning and subsequent growth and body composition.

Materials and methods

All procedures involving animals were approved by the Victorian Institute of Animal Science Animal Ethics Committee.

Twelve mixed parity cross-bred sows with an average litter size of ten piglets were used to nurse pigs for the present study. All sows and litters were housed in farrowing crates in an insulated building maintained at 18–22°C. The creep area was maintained at a temperature of 30–32°C through use of infrared lamps. Sows were offered 2·0 kg lactation diet/d containing 14·3 MJ digestible energy, 160 g crude protein (N×6·25) and 9·3 g available lysine/kg prior to farrowing. After farrowing, feed allowance was increased by 0·5 kg/d until reaching *ad libitum* feed intake, for the remainder of the lactation. Piglets were denied access to both creep feed and water throughout the nursing period.

On day 1 of lactation the litters were weighed and two male pigs from each litter were selected and randomly allocated to either daily saline (9 g/l NaCl/l) or recombinant pST (1 mg/kg body weight; Alpharma Animal Health, Toorak, Australia) injection until weaning on day 21. Pigs were weighed and pST doses adjusted every 3 d. After weaning at 21 d of age pigs were placed in individual pens in the weaner shed and fed *ad libitum*. At 4 weeks post-weaning the pigs were transferred into an experimental grower shed and kept in a pen with their male littermates. For the last 4 weeks of the finishing period pigs were kept in individual pens to determine feed intake and average daily gain. Conventional wheat-based weaner (15·0 MJ digestible energy, 200 g protein and 12·0 g available lysine/kg), grower (14·0 MJ digestible energy, 185 g protein and 9·5 g available lysine/kg) and finisher diets (13·7 MJ digestible energy, 167 g protein and 7·5 g available lysine/kg) diets were fed *ad libitum* from 21 to 49, 49 to 105 and 105 to 133 d of age respectively.

Body composition of pigs was measured using dual energy X-ray absorptiometry (DXA) (Hologic QDR4500, Waltham, MA, USA). Good overviews of the DXA technology and its underlying principals can be found in Laskey & Phil (1996) and Kelly *et al.* (1998) and DXA has previously been used to measure body composition in pigs (Mitchell *et al.* 1996, 1998; Lukaski *et al.* 1999). Animals were scanned using DXA at weaning (21 d old), at transfer into the grower shed (49 d old), midway through the grower phase (77 d old), commencement of finisher phase (105 d old) and just before slaughter (133 d old). Feed was removed from pigs 16 h prior to scanning to reduce the influence of gastointestinal contents on DXA measurements. Animals were anaesthetised using an intramuscularly administered cocktail of xylazine and ketamine (0·05 and 0·10 ml/kg body weight respectively) followed by respiratory administration of isoflurane. The chemical composition of the animal as estimated from the output generated by the algorithms in the proprietary DXA software were corrected using regression equations developed in our laboratory (Suster *et al.* 2000). Calibration of DXA was performed on a weekly basis with a step phantom to ensure accurate soft tissue results, and on a daily basis with a spine phantom to minimise baseline drift. Initial body composition of the piglets was estimated from live weight and the proportions of lean (812 g/kg), fat (65 g/kg) and ash (23 g/kg) found in similar pigs from our herd (Auldist *et al.* 1997).

Pig growth and body composition data was analysed by one-way ANOVA with the main effect being dose of pST with sow as the blocking factor. All analyses were performed using GENSTAT (Payne *et al.* 1993).

Results

Growth performances of the pigs are shown in Table 1. There was no significant difference in growth rate between day 1 and 21 of growth in pigs injected with either saline (9 g/l NaCl/l) or pST (258 *v.* 246 g/d for control and pST-treated pigs respectively, P=0·61) and as a consequence there was no significant difference in live weight at weaning (7·13 *v.* 6·84 kg, P=0·59). However, pigs previously treated with pST tended to eat less (547 *v.* 469 g/d, P=0·097) and grow more slowly (426 *v.* 373 g/d, P=0·085) after weaning than their control counterparts and as a consequence tended to be lighter at the end of the weaner period (19·1 *v.* 17·0 kg, P=0·078). Despite this, there were no significant differences in growth performance or live weight over the rest of the study (Table 1).

There was no effect of pST on lean tissue deposition of sucking pigs until weaning at 21 d of age (211 *v.* 210 g/d, P=0·97; Table 2). However, lean tissue deposition over the weaner period between 21 and 49 d of age was decreased by almost 14 % by previous exogenous pST treatment (347 *v.* 300 g/d, P=0·021). Consequently, the total lean tissue mass of pigs previously treated with pST tended to be less at 49 d of age (15·5 *v.* 13·9 kg, P=0·054). Despite this, there was no significant effect of pST treatment on lean tissue deposition over any other period of growth. Over the entire birth-to-slaughter

Table 1. Effect of daily porcine (p) somatotropin (ST) injection from day 1 until 21 d of age on growth performance*
(Mean values for six pigs per treatment group)

	Saline†	pST	SED	Statistical significance of effect: *P*
Live weight (kg)				
Day 1	1·97	1·92	0·099	0·64
Day 21	7·13	6·84	0·517	0·59
Day 49	19·1	17·0	1·05	0·078
Day 77	41·4	38·2	2·15	0·17
Day 105	69·9	67·8	3·03	0·49
Day 133	101·6	99·2	3·69	0·53
Weaner performance (21–49 d)				
Daily gain (g/d)	426	373	28·7	0·097
Feed intake (g/d)	547	469	40·7	0·085
Feed conversion ratio	1·29	1·26	0·063	0·70
Finisher performance (105–133 d)				
Daily gain (g/d)	1132	1123	31·8	0·79
Feed intake (g/d)	3158	3045	125·0	0·39
Feed conversion ratio	2·79	2·72	0·076	0·33

* For details of diets and procedures, see p. 796.
† 9 g/l NaCl/l.

period there was no effect of neonatal pST on lean tissue deposition (550 *v.* 538 g/d, *P*=0·49) or final lean tissue mass (74·2 *v.* 72·5 kg, *P*=0·49). Fat mass at weaning tended to be decreased (1·18 *v.* 0·96 kg, *P*=0·064), while the % fat in the body at weaning was significantly (16·7 *v.* 13·9 %, *P*=0·008) decreased by exogenous pST treatment (Table 3 and Fig. 1). While fat deposition was numerically lower over every stage of post-weaning growth in pigs treated with pST as neonates, it did not reach significance until during the finisher phase when fat deposition was decreased by 15 % (273 *v.* 232 g/d, *P*=0·039). Over the entire weaning-to-slaughter period, neonatal pST treatment decreased the rate of fat deposition (130 *v.* 112 g/d, *P*=0·033), final fat mass (17·3 *v.* 14·9 kg, *P*=0·033) and the % fat in the body (18·5 *v.* 16·6 %, *P*=0·008). However, there was no significant effect of neonatal pST treatment on P2 backfat at slaughter (20·2 v. 18·5 mm, *P*=0·22; Fig. 1). There was no effect of neonatal pST treatment on ash deposition or whole-body ash content at any stage of growth.

Table 2. Effect of daily porcine (p) somatotropin (ST) injection from 1 until 21 d of age on subsequent rates of tissue deposition*
(Mean values for six pigs per treatment group)

	Saline†	pST	SED	Statistical significance of effect: *P*
Lean deposition (g/d)				
1–21 d	211	210	17·1	0·97
21–49 d	347	300	17·4	0·021
49–77 d	538	660	102·9	0·27
77–105 d	831	742	117·4	0·47
105–133 d	723	683	35·6	0·29
21–133 d	611	598	17·9	0·48
1–133 d	550	538	17·0	0·49
Fat deposition (g/d)				
1–21 d	52·7	41·9	5·11	0·059
21–49 d	34·9	33·4	4·09	0·72
49–77 d	98·8	89·4	9·51	0·36
77–105 d	167	146	14·1	0·20
105–133 d	273	232	16·9	0·039
21–133 d	144	125	7·8	0·045
1–133 d	130	112	6·9	0·033
Ash deposition (g/d)				
1–21 d	2·31	2·10	0·320	0·51
21–49 d	7·43	6·80	0·549	0·28
49–77 d	17·6	18·2	1·47	0·69
77–105 d	20·2	20·5	0·96	0·77
105–133 d	19·6	19·2	1·98	0·87
21–133 d	16·3	16·2	0·91	0·94
1–133 d	14·2	14·0	0·78	0·87

* For details of diets and procedures, see p. 796.
† 9 g/l NaCl/l.

Table 3. Effect of daily porcine (p) somatotropin (ST) injection from 1 until 21 d of age on subsequent body composition*
(Mean values for six pigs per treatment group)

	Saline†	pST	SED	Statistical significance of effect: *P*
Lean content (kg)				
21 d	5·82	5·77	0·388	0·90
49 d	15·5	13·9	0·74	0·054
77 d	30·7	32·6	3·15	0·56
105 d	53·9	53·4	2·09	0·80
133 d	74·2	72·5	2·29	0·49
Fat content (kg)				
21 d	1·18	0·963	0·1062	0·064
49 d	2·16	1·83	0·126	0·028
77 d	4·96	4·36	0·320	0·098
105 d	9·64	8·44	0·719	0·13
133 d	17·3	14·9	0·92	0·033
Ash content (g)				
21 d	0·092	0·086	0·0081	0·52
49 d	0·300	0·271	0·0187	0·16
77 d	0·801	0·783	0·0525	0·75
105 d	1·37	1·36	0·074	0·90
133 d	1·91	1·90	0·104	0·86

* For details of diets and procedures, see p. 796.
† 9 g/l NaCl/l.

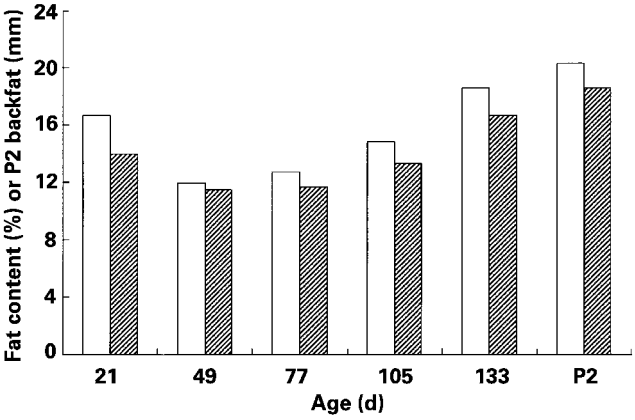

Fig. 1. Effect of neonatal porcine (p) somatotropin (ST) treatment on body fat content (%) at various ages and on P2 backfat (mm) at slaughter. □, Control; ▨, pST. For details of procedures, see p. 796. Values are means for six pigs. Mean values were significantly different from those of the control group at 21 d (P=0·001), 49 d (P=0·14), 77 d (P=0·039), 105 d (P=0·055), 133 d (P=0·008), but not in P2 (P=0·22).

Discussion

Treatment of neonatal pigs with pST caused a 20 % decrease in the rate of fat deposition, despite there being no significant effect on the rates of lean tissue, ash and whole-body accretion up to weaning. Previously, we found that a much lower dose of pST (0·06 *v.* 1·0 mg/kg) had little effect on growth of sucking piglets (Dunshea *et al.* 1999). On the other hand, Wester *et al.* (1998) administered the same dose of pST as used in the present study (1 mg/kg) to neonatal pigs that were artificially reared (from 1 d of age) and found increases in growth rate and plasma IGF-I and IGF-binding protein-3. This indicates that the somatotrophic axis was functional in the neonatal pig, but sensitivity of expressed ST receptors was specifically reduced in the neonate as compared with an older pig. Wang *et al.* (1999) obtained adipose tissue from nursing neonatal pigs (7 d old) and found that although basal and insulin-stimulated lipogenesis rates were low when compared with those observed in adipose tissue obtained from growing pigs, these rates could be further inhibited (about −78 and −43 % under basal and insulin-stimulated conditions respectively) by including pST in the medium. In another study involving pigs that were artificially reared, the antilipogenic responses to pST were evident at 25 d of age, although the response was markedly less than that normally observed in older pigs (Harrell *et al.* 1996). Similarly, nursing pigs treated with 0·3 mg pST/kg per d from 1 day of age had 12 % less backfat at the cessation of treatment at 25 d of age (Morel *et al.* 1999).

In the immediate post-weaning period there was no effect of previous pST treatment on fat deposition. However, as the animals grew older there appeared to be a progressive reduction in the rate of fat deposition, as indicated by the declining P value with advancing age. Over the finishing phase, the rate of fat deposition was 15 % lower in pigs previously treated with pST as neonates. It is possible that neonatal treatment of pigs with pST may decrease proliferation and/or differentiation of preadipocytes, thereby resulting in a reduction in total fat cells in the body. For example, Ramsay *et al.* (1987) found that the addition of pST to pre-adipocytes cultured in serum from decapitated fetuses (to remove pituitary hormones) resulted in an 80 % reduction in complete differentiation. Likewise, Gerfault *et al.* (1999) found that pST inhibited both differentiation and proliferation of porcine pre-adipocytes obtained from neonatal pigs. If pST treatment of neonatal pigs also results in a decrease in porcine pre-adipocyte proliferation and differentiation *in vivo* then there would be fewer fat cells that would fill sooner. This in turn could result in the reduction in fat deposition becoming most pronounced during the finisher phase.

An alternative explanation may be that the reduced feed intake observed in the immediate post-weaning and possibly the grower (although not measured) periods in the present study may have contributed to an inhibition of pre-adipocyte differentiation and proliferation and subsequent reduced fat deposition in the finisher period. It is not anticipated that there was any effect of pST on feed intake during the sucking period, since milk intake was likely to be already limiting growth (Boyd *et al.* 1995; Dunshea *et al.* 2002). In addition, there was no effect of pST on growth rate, which in turn is closely related to milk intake in the nursing pig (King *et al.* 1989). However, there were clearly differences in the partitioning of nutrients in the sucking pig such that less energy was available for adipose tissue development and growth. Others have attempted to manipulate subsequent growth of pigs by manipulation of nutrient intake during the neonatal period. For instance, the classical studies of McMeekan (1940*a*,*b*,*c*) suggested that the level of feed intake up to 16 weeks of age could influence subsequent growth and carcass composition. Thus, McMeekan (1940*c*) found pigs that were restricted in feed intake and were then fed *ad libitum* had fatter carcasses at slaughter at 90 kg than pigs that were fed *ad libitum* throughout. However, more targeted restrictions in energy and/or feed intake in younger pigs, such as those used in the present study, suggest that the predominant effect is to reduce body fat with more subtle effects on lean tissue content (for review, see Young & Sharma, 1973). For example, Martin *et al.* (1974) fed early weaned (3 d of age) pigs on milk replacers that were either adequate in both energy and protein or deficient in either energy or protein until 4 weeks of age, after which they were fed standard diets *ad libitum* until slaughter at 23 weeks of age. Pigs fed the low-energy diet were lighter (8·8 *v.* 5·5 kg) at 4 weeks of age than the control pigs, with this effect still evident at 23 weeks of age (87·7 *v.* 73·6 kg). There was little effect of neonatal nutrition on a representative muscle weight (*Semitendinosis*), whereas the representative fat depot (perirenal fat) was 46 % lighter in pigs fed the low-energy diet. Therefore, it is possible that the effects of neonatal pST treatment on lifetime fat deposition may be mediated indirectly through reduced feed intake during the immediate post-weaning or grower periods. Regardless of the mechanism, the practical implication is that neonatal and/or perinatal manipulation of fat metabolism can have subsequent effects on fat deposition.

It is possible that a paired-feeding regimen during the post-weaning period could have been employed to determine whether effects of pST on subsequent growth and carcass composition were direct effects or the result of decreased feed intake. In this context, Neilsen (1964) found that effects of feed restriction from weaning at 3 weeks of age until 20 kg were most pronounced in pigs that were kept on a low plane of nutrition until slaughter at 90 kg than when fed *ad libitum*. However, it was felt that since the rate of fat deposition and voluntary feed intake are so closely linked, a restricted feeding regimen may have masked any effects of neonatal pST treatment on subsequent lean and fat deposition. For example, the reduced fat deposition that occurs in finisher pigs treated with pST is due to the induction of insulin resistance and resultant reduction in feed intake (Dunshea *et al.* 1992*a*,*b*,*c*). From a practical point of view it is also unlikely that restricted feeding would be used to exploit neonatal pST treatment under commercial conditions.

Other workers have administered ST to pig and other species during the early period of growth and observed decreased backfat depths, although these effects have generally been transitory and not maintained until market weight. For example, Morel *et al.* (1999) treated neonatal pigs with 0·3 mg pST/kg per d from 1 to 24 d of age and found differences in backfat depth at the cessation of treatment at 25 but not at 70 d of age or slaughter at 95 kg. Similarly, McCutcheon *et al.* (1994) injected lambs with up to 0·3 mg bovine ST/kg per d from birth until 11 weeks of age and observed a reduction in backfat at 8 but not 13 months of age. The responses may have been more sustained in the present study because a higher dose of pST was used at a critical stage in development, which in turn could have a more profound effect upon pre-adipocyte proliferation and differentiation. While treatment of neonatal rats with bovine ST for 21 d had no effect on body fat at 21 or 60 d of age, there was a decrease in body fat at 120 d of age (Kadim *et al.* 1996). As with the present study, there was little effect of neonatal ST treatment on lean tissue mass at any age.

There was a reduction in feed intake, lean tissue deposition and growth rate during the immediate post-weaning period in the pigs that had been previously treated with pST as neonates. Very little work has been done on the effects of withdrawal of pST on subsequent growth performance of pigs, particularly in pigs so young. In one study, we found that there was very little effect of previous treatment pST of nursing pigs on growth performance in the immediate post-weaning period (Dunshea *et al.* 1999). On the other hand, Campbell *et al.* (1989*b*) reported that pigs treated with pST between 30 and 60 kg live weight had greater rates of lean and ash deposition, but unchanged fat deposition, over the growth phase between 60 and 90 kg live weight. More typically, a reduction in growth performance, often to levels below that of controls, is observed when pST treatment ceases (Bryan *et al.* 1990; Smith & Kasson, 1990; Weeden *et al.* 1993). For example, Weeden *et al.* (1993) found that while daily gain was increased (+11 %) by pST treatment of finisher gilts, growth rate during the 35 d withdrawal period was dramatically decreased (−51 %). Similarly, Sandles & Peel (1987) treated identical calves with bovine ST (0·6 mg pituitary bovine ST/kg per d) from 3·5 months old for 21 weeks and found that although there was a significant increase in live weight at the end of the treatment period, this difference had disappeared 5 weeks later. Brumby (1959) also found that the improvements in live weight of calves treated with pituitary bovine ST were lost 5 weeks after cessation of treatment. Previous pST treatment of finisher pigs causes a large (about 50 %) reduction in the amount of pST contained in the pituitary (Campbell *et al.* 1989*a*). Therefore, it may be possible that pST treatment of neonatal pigs decreases pituitary pST production and/or delays the development of somatotrophic activity in the pituitary. A decrease in endogenous pST production in the immediate post-weaning period may be the cause of the reduced lean tissue deposition in weaner pigs previously treated with pST.

Porcine somatotropin failed to stimulate lean tissue deposition in the sucking pig, most likely because sow's milk has a protein and lysine content much lower than the protein and lysine requirements of the young pig (Williams, 1976; Auldist *et al.* 1997; Dunshea *et al.* 2000). For example, the lysine content of sows' milk is about 0·7 g/MJ gross energy, whereas the requirement of the neonatal pig is about 0·95 g/MJ gross energy (Auldist *et al.* 1997). Therefore, lysine intake from sows' milk normally limits lean tissue growth of pigs. It is well established that at low levels of dietary lysine there is very little effect of pST on lean tissue deposition in grower (Campbell *et al.* 1990; Caperna *et al.* 1990; Krick *et al.* 1993) and finisher pigs (Campbell *et al.* 1991; King *et al.* 2000). Therefore, dietary lysine may have limited the lean tissue response to pST in the sucking pigs in both the present study and that of Dunshea *et al.* (1999), whereas the neonatal pigs used in the study of Wester *et al.* (1998) received a milk replacer containing adequate levels of lysine.

Conclusions

Treatment of neonatal pigs with very high doses of pST can decrease fat deposition during the neonatal period. More importantly, pST treatment of neonatal pigs can also decrease subsequent fat deposition, particularly over the finishing period. Therefore, treatment of nursing pigs with pST for a short period before weaning may provide a means of reducing the fat content of pork and pork products.

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