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Effects of dietary arginine supplementation during gestation and lactation on the performance of lactating primiparous sows and nursing piglets[1]

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ABSTRACT: A 2 × 2 factorial arrangement of treatments in a randomized block design was utilized to determine the effects of dietary Arg supplementation during gestation and lactation on the lactation performance of 38 first-parity sows. At 30 d of gestation, pregnant gilts were allotted based on BW to 1 of 2 diets supplemented with 1% L-Arg-HCl or 1.7% L-Ala (isonitrogenous control). After farrowing, sows were further allotted based on BW within previous gestation treatment groups to 1 of 2 lactation diets supplemented with 1% L-Arg-HCl or 1.7% L-Ala (isonitrogenous control). All gestation diets contained 3.1 Mcal/kg and 12.2% CP and were fed 2 kg/d in two equal-sized meals, whereas all lactation diets contained 3.2 Mcal/kg and 18.6% CP and were fed ad libitum. Litter size was standardized to 10 piglets by cross-fostering within 24 h post-farrowing. On a weekly basis, BW and backfat (**BF**) thickness of sows, as well as piglet BW were measured, and blood and milk samples were obtained from sows. Number of days from wean to estrus and ADFI were also recorded. There were no differences in BW, BF thickness, ADFI, or days until return-to-estrus among treatment groups. There was no effect of gestation diet or gestation × lactation diet interaction on any parameter measured. On d 7 of lactation, plasma concentrations of Arg and insulin in sows, as well as concentrations of most AA in milk, were greater ($P < 0.05$) in response to Arg supplementation during lactation compared to the control. Weight gain of piglets from sows fed the Arg-supplemented diet during lactation was greater between d 0 and 7 ($P < 0.01$) and between d 0 and 21 ($P < 0.05$) of lactation, compared to piglets from sows fed the control diet. Collectively, results from this study indicate the potential beneficial effects of dietary Arg supplementation in improving the lactation performance of first-parity sows.

Key words: L-Arg, lactation performance, litter weight gain, sows

INTRODUCTION

Young animals have a high requirement for Arg (Southern and Baker, 1983; Fickler et al., 1994) due to its utilization by multiple metabolic pathways (Wu and Morris, 1998). However, Arg intake from sow's milk is low relative to the need for protein deposition in piglets (Davis et al., 1994; Wu and Knabe, 1994). Estimates based on the supply of Arg from sow's milk and Arg requirement of piglets revealed that sow's milk provides less than 40% of the daily requirement in 7-d-old suckling pigs (Wu et al., 2004). Both metabolic and growth data indicate that an Arg deficiency is a major factor limiting maximal weight gain of milk-fed piglets (Kim and Wu, 2004; Wu et al., 2004; Frank et al., 2007).

Increasing Arg intake from milk by suckling piglets could be an effective means to enhance their growth. In addition to the feed intake of sows and suckling intensity of piglets, milk production is also influenced by the angiogenesis of mammary tissue and blood flow to mammary glands, which enhance nutrient delivery to the mammary gland for milk synthesis (Trottier et al., 1997). Mammary blood flow and angiogenesis are regulated by Arg-derived nitric oxide (Meininger and Wu, 2002; Lacasse and Prosser, 2003). Furthermore, milk production is highly correlated with mammary gland growth (Ceriani, 1974; Kim et al., 2000) and Arg is required for optimal mammary gland growth (Pau and Milner, 1982). At a high dosage, Arg stimulates the secretion of prolactin and growth hormone that are necessary for mammary development (Knopf et al., 1968; Davis, 1972). We hypothesized that supplementing Arg to diets for first-parity sows during gestation and lactation may stimulate the weight gain of sow-reared piglets possibly by increasing nutrient utilization, and therefore increasing milk production and altering nutrient composition in milk.

MATERIALS AND METHODS

Animals, Experimental Diets, and Design

A 2 × 2 factorial study was conducted to determine the effects of L-Arg supplementation in gestation in combination with lactation diets on lactation performance of 38 first-parity sows (Camborough 22, Pig Improvement Co., Franklyn, KY). The animal care and use protocol was approved by Animal Care and Use Committee of Texas Tech University.

At d 30 of gestation, pregnant gilts with average BW of 166.3 ± 1.8 kg and backfat (**BF**) thickness of 13.3 ± 0.2 mm were housed in individual gestation crates (2.1 × 0.6 m) and gilts with similar BW were paired and then randomly allotted to 2 dietary treatments which consisted of corn-soybean meal based diets supplemented with either 1% L-Arg-HCl or 1.7% L-Ala (isonitrogenous control; Table 1). At 110 d of gestation, pregnant gilts were transferred to individual farrowing crates (1.5 × 2.2 m). Within 24 h post-farrowing, sows in each treatment group were assigned randomly to corn-soybean meal based lactation diets supplemented with either 1% L-Arg-HCl or 1.7% L-Ala (isonitrogenous control; Table 1). Litter size was standardized to 10 piglets depending on their availability by cross-fostering within 24 h post-farrowing. The number of piglets per sow ranged from 9 to 13 piglets but was equalized within weight groups (blocks). Alanine was chosen for isonitrogenous control because Ala is not toxic and is not a substrate for Arg synthesis, but is extensively catabolized by pigs (Kim and Wu, 2004; Kohli et al., 2004). Furthermore, previous studies have shown no differences in the reproductive performance between first-parity sows provided either conventional diets or diets supplemented with 1% Ala (Ji, 2004; Mateo et al., 2007). The supplemental level of 1% L-Arg-HCl was chosen because it was shown in our previous study to increase plasma concentration of

87 Arg in pregnant pigs by 65% at 2 h after feeding, indicating that providing Arg through dietary
88 supplementation can successfully be delivered to the body for further metabolism (Wu et al.,
89 2007).

90 Gestation diets contained 3.1 Mcal/kg and 12.2% CP and lactation diets contained 3.2
91 Mcal of ME/kg and 18.7% CP. These diets were designed to meet or exceed the nutrient
92 requirements for both gestating and lactating sows set forth by the NRC (1998). Gestation diets
93 (2 kg/d) were fed twice daily at 0700 and 1800 h between d 30 of gestation and farrowing.
94 Lactation diets were provided to sows ad libitum throughout the lactation period. Water was
95 available ad libitum during both gestation and lactation periods. Farrowing room temperature
96 was maintained at 25ºC with supplemental heat for piglets provided by heat lamps. During the
97 entire 21-d lactation period, feed disappearance of sows was recorded and piglets had no access
98 to creep feed. Body weight of sows was obtained within 24 h post-farrowing and on d 7, 14, and
99 21 of lactation. Backfat thickness of sows was measured by ultrasound (Keiki LS-1000, Tokimec
100 Inc., Tokyo, Japan) at the P2 position (left side of the 10^{th} rib and 6 cm away from the spine)
101 during each weighing period. Piglets were weighed post-farrowing and at d 7, 14, and 21 of the
102 lactation period. Mature milk samples were collected at 1000, 2 h after feeding by manual
103 extraction after thorough cleaning of the udder with water and intramuscular injection of
104 oxytocin (20 IU; Phoenix Pharmaceutical, Inc. St. Joseph, MO). Milk samples were collected
105 from all functional teats of all sows about 30 min after piglets were separated from the dam on d
106 7 and 21 of lactation.

107 Blood samples were collected from sows at 1000, 2 h after feeding via jugular
108 venepuncture using heparinized tubes (Becton-Dickinson Vacutainer Systems, Rutherford, NJ)

on d 7 and 21 of lactation. Blood samples were centrifuged at 2,000 × *g* for 15 min. Plasma was separated using transfer pipettes into 1.5 mL microcentrifuge tubes (National Scientific, San Rafael, CA) and stored at -20ºC until further AA, insulin, and urea analyses. All litters were weaned and sows returned to individual gestation crates at d 21 of lactation. The days until return-to-estrus were also recorded.

Chemical Analyses

Plasma samples (1 mL) were deproteinized with an equal volume of 1.5 *M* $HClO_4$ and neutralized with 0.5 mL of 2 *M* K_2CO_3. The extracts were analyzed for urea concentrations using a colorimetric method that involved a reaction with phenol and hypochlorite (Wu and Knabe, 1994). For analysis of all milk AA, except for Trp, 0.2 mL of whole milk was hydrolyzed in 6 mL of 6 *N* HCl at 110°C for 24 h under N_2 (Wu and Knabe, 1994). For analysis of Trp, milk samples (0.2 mL) were hydrolyzed in 6 mL of 4.2 *M* NaOH plus 0.1 mL of thiodiglycol (25% aqueous solution, an antioxidant) as described by Wu et al., 1999. Amino acids in plasma and milk hydrosylates were analyzed by HPLC methods involving precolumn derivatization with *o*-phthaldialdehyde (Wu et al., 1997). Amino acid standards and other chemicals were obtained from Sigma Chemical Company (St. Louis, MO). An enzyme immunoassay was utilized for the quantification of plasma insulin concentrations according to the manufacturer's instruction (Porcine insulin ELISA kit, Mercodia Inc., Winston Salem, NC).

Statistical Analysis

Data were analyzed using the MIXED procedures (SAS Inst., Inc., Cary, NC) for a factorial arrangement following a randomized complete block design. Sow was considered as experimental unit. Separation of means was done using the PDIFF option of SAS. Probability

values less than 0.05 were considered statistically significant and between 0.05 and 0.07 as trends.

RESULTS

Piglet Performance

Both litter size after cross-fostering and at d 7, 14, and 21 did not differ among treatment groups. Litter sizes at d 0, 7, 14, and 21 of lactation were 10.9 ± 0.20, 10.6 ± 0.20, 10.3 ± 0.19 (pooled means ± SEM), respectively. Both gestation and lactation diets did not affect BW, BF thickness, ADFI, or days until return-to-estrus. Main effects of gestation and gestation × lactation interactions were not significant for all piglet performance data during lactation. Piglet BW at the initiation of cross-fostering did not differ among treatment groups. However, BW of piglets from sows fed the Arg-supplemented diets during lactation were greater ($P < 0.05$) at d 7 (2.62 ± 0.11 vs. 2.44 ± 0.11 kg), 14 (4.18 ± 0.20 vs. 3.86 ± 0.21 kg), and 21 (5.76 ± 0.22 vs. 5.36 ± 0.23 kg) of lactation, compared to piglets from control-fed sows. Weight gains of piglets from sows fed the Arg-supplemented diet during lactation were greater between d 0 and 7 (1.26 ± 0.09 vs. 1.00 ± 0.09 kg; $P < 0.01$) and between d 0 and 21 (4.34 ± 0.21 vs. 3.92 ± 0.22 kg; $P < 0.05$), compared to piglets from the control-fed sows. However, there was no difference in piglet weight gain during either d 7 to 14 or d 14 to 21 of lactation.

Plasma Urea Concentrations in Sows

No significant gestation × lactation interaction effect on plasma urea concentrations in sows was noted among the different treatment groups on d 7 or 21 of lactation (Table 3). There was a trend ($P = 0.071$) for sows fed the Arg-supplemented diet during gestation to have decreased plasma concentrations of urea (4.6 ± 0.07 vs. 4.8 ± 0.06 mmol/L) at d 7 of lactation,

153 compared to sows fed the control diets. In addition, sows fed Arg-supplemented diets during the
154 lactation period had decreased plasma concentrations of urea (4.5 ± 0.08 vs. 4.8 ± 0.07 mmol/L;
155 $P < 0.05$) at 7 d of lactation compared to sows fed the control diet.

156 ***Plasma AA Concentrations in Sows***

157 Plasma concentrations of AA in first-parity sows at d 7 of lactation are summarized in
158 Table 4. No significant gestation effect among treatment groups were observed for all AA
159 measured. Except for Met, no gestation × lactation diet interaction effect was noted for all other
160 AA. Arginine supplementation to sows during the lactation period resulted in greater ($P < 0.01$)
161 plasma concentrations of Pro, Gly, Arg, and ornithine, compared to the control sows. However,
162 plasma concentrations of Ser, Gln, His, citrulline, and Ala were decreased for sows fed Arg-
163 supplemented diets during lactation, when compared to sows fed isonitrogenous control diets. A
164 gestation × lactation diet interaction effect was noted for Met at 7 d of lactation ($P < 0.05$). There
165 were no differences in plasma concentrations of other AA between control and Arg-
166 supplemented sows. Similar results were obtained for plasma concentrations of AA in control
167 and Arg-supplemented sows at d 21 of lactation (data not shown).

168 ***Concentrations of Total AA in Milk***

169 ***Day 7 of Lactation.*** Concentrations of total AA (both protein-bound and free) in the milk
170 of first parity sows at 7 d of lactation are summarized in Table 5. No main effect of gestation or
171 gestation × lactation interaction effects were noted for all AA measured. There was a trend ($P <$
172 0.09) for concentrations of Ala, Val, Ile, Pro, Cys, and Trp in milk to be greater for sows fed
173 Arg-supplemented diets, compared to the control-fed sows. However, concentrations of Glu, Ser,
174 Gly, Thr, Tyr, and Phe in milk were greater ($P < 0.05$) for sows fed the Arg-supplemented diets

in comparison with the control-fed sows. There were no differences in concentrations of other AA in milk between control and Arg-supplemented sows. Total AA content in milk was greater ($P < 0.05$) for sows fed the Arg-supplemented diets compared to the control-fed sows (Table 5).

Day 21 of Lactation. Concentrations of total AA in the sow's milk at 21 d of lactation are summarized in Table 6. No main effects of gestation or gestation × lactation interactions were noted for all AA measured. There was a trend ($P < 0.10$) for concentrations of Ser, Thr, Tyr, Met, Phe, Leu, and Pro in milk to be greater for sows fed Arg-supplemented diets, compared to the control-fed group. Concentrations of both Asp and Gly in milk were greater ($P < 0.05$) for sows fed the Arg-supplemented diets compared to the control-fed sows. There were no differences in concentrations of other AA or total AA in milk between control and Arg-supplemented sows.

Plasma Insulin Concentrations in Sows

No significant gestation or gestation × lactation interaction effect on maternal plasma insulin concentrations were noted among the different treatment groups at d 7 or 21 d of lactation (Table 7). However, plasma insulin concentration was greater ($P < 0.05$) at both d 7 and d 21 of gestation in sows fed the Arg-supplemented diets, compared to sows fed the control diets (Table 7).

DISCUSSION

Results from the present study demonstrate that supplementing Arg to the diet for sows during the entire lactation period increased concentrations of total AA in milk and improved piglet growth performance. These findings provide a new strategy for the nutritional management of sow-reared neonates.

196 Litter weight gain is known to be correlated with milk production or nutrient
197 concentrations in milk (Noblet and Etienne, 1987; King et al., 1993). Increased piglet or litter
198 weight gain in Arg-supplemented sows may be indicative of increased milk production or
199 increased nutrient concentrations in milk. Results of the present study indicate that voluntary
200 feed intake and body weight changes of sows were not affected by dietary Arg supplementation
201 (Table 2), suggesting that increased concentrations of total AA in milk were not due to
202 alterations in dietary protein intake or whole-body protein mobilization. On the basis of reduced
203 levels of urea in plasma, Arg supplementation appears to enhance the efficiency of utilization of
204 dietary protein utilization for milk protein synthesis. By increasing the synthesis of nitric oxide
205 (a major vasodilator) in endothelial cells of blood vessels (Moncada et al., 1989; Wu and
206 Meininger, 2000), dietary Arg supplementation can enhance blood flow and nutrient supply to
207 the mammary gland for milk protein, resulting in improved weight gain of suckling piglets. The
208 increased concentrations of total AA in milk are associated with the increased weight gain of
209 piglets during the first week of lactation, which affected the overall improvement of piglet
210 growth performance during the entire lactation period.
211 On average, piglets from Arg-supplemented sows gained 20 g more BW per d, or 420 g
212 more during the 21-d lactation period, compared to the piglet from sows in control groups (Table
213 2). Considering that body composition in neonatal pigs is about 25% DM and 12.5% protein
214 (McPherson et al., 2004), 420 g weight gain is translated into 50 g protein gain in 3 wk for a
215 piglet. As shown in Table 5, the increase in concentrations of total AA in the milk of Arg-
216 supplemented sows is about 3.4 g/L. Considering that a piglet with 200 g daily weight gain
217 obtains 0.78 L milk per d (Wu et al. 2004), the arginine treatment would provide 2.65 g of

218 additional protein to the piglet per d, or 56 g during the 21-d lactation period. Because the
219 digestibility of milk protein is high (95 to 100%) in neonatal pigs (Lin et al., 2006), the intake of
220 an additional 56 g protein from milk is sufficient to support the gain of an additional 50 g protein
221 in each piglet during a 21-d lactation period.

222 Furthermore, mammary blood flow and substrate concentrations in blood are major
223 factors that determine substrate availability for milk synthesis (Davis and Collier, 1985) and
224 therefore nutrient delivery to the neonate. Arginine is the physiological precursor for the
225 synthesis of nitric oxide (NO), the endothelium derived relaxing factor (Wu and Meininger,
226 2000) and a key angiogenic factor (Meininger and Wu, 2002). Increasing NO availability has
227 been reported to rapidly increase mammary blood flow in ruminants (Lacasse et al., 1996;
228 Lacasse and Prosser, 2003). Interestingly, a short-term increase in NO provision within several h
229 may not lead to increased milk production (Prosser et al., 1996; Lacasse and Prosser, 2003),
230 possibly due to a lack of increase in the number of secreting cells and the synthesis of proteins,
231 fat and lactose.

232 As noted above, rapidly-growing piglets have a high requirement for Arg. However,
233 previous studies have clearly demonstrated that limited Arg availability from both sow's milk
234 (Wu and Knabe, 1994; Wu et al., 2004) and limited capability endogenous Arg synthesis (Wu
235 and Knabe, 1995; Flynn and Wu, 1996) are major obstacles in realizing the maximum growth
236 potential of sow-reared piglets (Kim and Wu, 2004; Wu et al., 2004). The marked decrease in the
237 availability of Arg coincides with the period when sub-maximal growth in piglets occurs (Boyd
238 et al., 1995; Flynn et al., 2000; Kim and Wu, 2004). In support of this view, Kim and Wu (2004)

239 demonstrated that dietary Arg supplementation dose-dependently enhanced the growth
240 performance of artificially reared piglets.

241 It has also been reported that Arg uptake by the mammary gland is much greater than
242 milk Arg output (Trottier et al., 1997) which reflects the high capacity of the porcine mammary
243 gland to catabolize Arg (O'Quinn et al., 2002). Thus, Arg supplementation did not result in a
244 substantially greater Arg concentration in sow's milk. However, an increase in the volume of
245 milk consumed by piglets (Kirchgessner et al., 1991) would translate into an increase in the
246 provision of Arg and other nutrients to the neonates for supporting their growth. This was
247 clearly observed for suckling piglets on d 0 to d 7 (Table 2). However, there was lack of a
248 significant increase in piglet weight gain during wk 2 and 3 in response to Arg supplementation
249 (Table 2). The underlying reasons are not known at present, but may be related to unaltered
250 concentrations of total AA in milk after the first wk of lactation (Table 6).

251 As expected, Ala-supplemented sows had greater concentrations of Ala in plasma,
252 compared with Arg-supplemented sows. However, an interesting observation from the present
253 study is that dietary Arg supplementation to lactating sows decreased plasma concentrations of
254 Ser, Glu, His, and Thr at d 7 of lactation. It is possible that there is an increase in the utilization
255 of these AA by the mammary gland for the synthesis of proteins, peptides, and other milk
256 components. In support of this suggestion, we showed that concentrations of total AA (primarily
257 protein) in milk increased at d 7 of lactation in Arg-supplemented sows. We surmise that Arg
258 supplementation to gestating sows may have stimulated mammary growth (including vascular
259 growth), thereby promoting blood flow and AA uptake by the mammary gland to increase milk
260 protein synthesis during the lactation period. This suggestion is consistent with the finding that

261 the majority of growth in these tissues occurs during the later parts of gestation (Kim et al.,
262 1999). Tucker et al. (1966) and Kim et al. (2000) showed that total DNA content is an indicator
263 of the number of mammary cells and is highly correlated with litter weight gain in pigs and
264 rodents. Measuring DNA content in mammary tissue would provide a useful indicator in both
265 control and Arg-supplemented sows; however, mammary DNA was not measured in this study.
266 The size of suckling piglets is positively correlated with the mass of mammary gland
267 suckled (Nielsen and Sorensen, 1998; Kim et al., 2000). Consistent with this observation, we
268 observed that piglets suckling from Arg-supplemented sows were heavier throughout lactation
269 with an increase in weight gain. Furthermore, the secretagogue effects of Arg on anabolic
270 hormones, such as insulin (Floyd et al., 1966; Kim and Wu, 2004; Laspiur et al., 2006), may also
271 play a role in the increased uptake of AA by the mammary gland (Laarveld et al., 1981).
272 Previous reports from studies with other species have shown that the mammary gland becomes
273 highly sensitive to insulin during lactation (Burnol et al., 1990). Thus, an increase in
274 concentrations of plasma insulin and its sensitivity in Arg-supplemented lactating sows may
275 stimulate the utilization of AA by the mammary gland to produce proteins. In dairy cows
276 subjected to an insulin clamp, there was an increase in both mammary blood flow and the
277 efficiency of extraction of blood AA by the mammary gland (Mackle et al., 2000). The increase
278 in insulin secretion during lactation may also explain, in part, the decreased plasma
279 concentrations of several AA measured (Fukagawa et al., 1986). These results suggest that
280 supplementing Arg to the diets for lactating sows may increase the uptake of substrates (e.g.,
281 AA) by the porcine mammary gland for milk protein synthesis. Although this effect was more

apparent during the initial period of lactation, the increased piglet performance during the first week of life translated to the overall improvement in piglet performance.

Urea is the major end product of AA oxidation in mammals (Meijer et al., 1990). Previous studies suggest that plasma urea concentrations in lactating sows may be an indicator of efficiency of whole-body nitrogen utilization (Coma et al., 1995). There were no differences in feed intake among all groups of lactating sows (Table 2). Thus, a reduction in plasma urea levels in Arg-supplemented sows may reflect an increase in the use of dietary AA for tissue or milk protein synthesis, as previously reported for Arg-supplemented gestating gilts (Mateo et al., 2007), lactating sows (Laspiur and Trottier, 2001), and neonatal pigs (Kim and Wu, 2004). Interestingly, no differences in plasma urea concentration among sows were observed at d 21 of lactation which agrees to the observation that piglet weight gain in wk 2 and 3 was not affected by Arg supplementation to the sow's diet (Table 2).

In summary, supplementing dietary Arg to lactating sows enhanced the growth performance of suckling piglets. The increased litter weight gain was associated with increased concentrations of total AA in milk at d 7 of lactation. We propose that the Arg treatment may increase mammary blood flow and extraction of AA during lactation. However, further studies are necessary to test this new hypothesis.

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Table 1. Composition of gestation and lactation diets, as-fed basis

	Experimental diets[1], %			
	Gestation		Lactation	
Ingredient	Control	Arg	Control	Arg
Corn grain	71.20	71.20	57.50	57.50
Soybean meal, 44% CP	10.50	10.50	27.00	27.00
Alfalfa meal, 17% CP	5.00	5.00	-	-
Molasses cane	4.30	5.00	2.95	3.65
Potassium chloride	0.75	0.75	0.10	0.10
Salt	0.35	0.35	0.35	0.35
Vitamin-mineral premix[2]	3.00	3.00	3.00	3.00
Vegetable oil	0.50	0.50	3.00	3.00
Dicalcium phosphate	2.20	2.20	2.50	2.50
Limestone	0.50	0.50	0.70	0.70
L-Arg-HCl	-	1.00	-	1.00
L-Ala	1.70	-	1.70	-
Chemical composition				
DM, %	89.3		90.0	
ME, Mcal/kg	3.1		3.3	
CP, %	12.2		18.7	
Lys, %	0.56		0.96	
Met+Cys, %	0.44		0.59	

Try, %	0.13	0.21
Thr, %	0.45	0.67
Ca, %	0.94	1.04
Available P, %	0.47	0.54
Total P, %	0.69	0.79

[1] Gestation diets were provided at 2 kg/d in 2 separate meals (0700 and 1800 h); lactation diets were provided ad libitum from farrowing to 21 d of lactation. Control diets were made isonitrogenous with the addition of L-Ala at 1.7% at the expense of molasses cane; eArg diets with added L-Arg HCl at 1% at the expense of molasses cane. (Supplemental AA were obtained from Ajinomoto Co., Inc. Tokyo, Japan). Analyzed CP (as-fed basis) content of diets were as follows: 12.5% for the arginine-supplemented gestation diet; 12.4% for the control gestation diet; 18.5% for the arginine-supplemented lactation diet; and 18.9% for the control lactation diet.

[2] The vitamin premix provided the following per kilogram of complete diet: 46.7 mg of Mn as manganous oxide; 75 mg of Fe as iron sulfate; 103.8 mg of Zn as zinc oxide; 9.5 mg of Cu as copper sulfate; 0.72 mg of I as ethylenediamine dihydroiodide; 0.23 mg of Se as sodium selenite; 7,556 IU of vitamin A as vitamin A acetate; 825 IU of vitamin D_3; 61.9 IU of vitamin E; 4.4 IU of vitamin K as menadione sodium bisulfate; 54.9 μg of vitamin B_{12}; 13.7 mg of riboflavin; 43.9 mg of D-pantothenic acid as calcium panthonate; 54.9 mg of niacin; and 1,650 mg of choline as choline chloride.

Table 2. Lactation performance of first parity sows fed diets supplemented with or without 1% L-Arg-HCl

	Treatment[1]							
Gestation diet	Control		Arg		SEM	*P*-value		
Lactation diet	Control	Arg	Control	Arg		Gestation	Lactation	G × L[2]
No. of observations	8	9	10	11				
Piglet BW, kg								
0	1.43	1.43	1.45	1.42	0.025	0.895	0.749	0.753
7	2.42	2.62	2.48	2.80	0.049	0.279	0.011	0.678
14	3.81	4.14	3.91	4.22	0.069	0.521	0.023	0.955
21	5.26	5.66	5.46	5.86	0.089	0.244	0.024	0.987
Piglet weight gain, g/d								
0 to 7 d	140.0	170.0	147.1	191.4	0.046	0.234	0.004	0.576
7 to 14 d	202.9	220.0	202.9	208.6	0.036	0.588	0.290	0.589
14 to 21 d	205.7	217.1	221.4	234.3	0.050	0.264	0.409	0.963
Overall	182.9	202.4	191.0	211.4	0.093	0.326	0.024	0.955

Sow								
BW, kg								
After farrowing	180.4	178.6	177.7	181.1	1.931	0.971	0.845	0.523
7 d	177.4	175.0	174.0	176.2	1.750	0.767	0.985	0.533
14 d	174.7	171.7	174.1	173.9	1.661	0.820	0.640	0.688
21 d	168.5	167.9	164.5	168.7	1.886	0.684	0.654	0.547
BW loss, kg	12.1	11.0	13.3	12.6	2.064	0.748	0.835	0.957
Backfat, mm								
After farrowing	15.4	15.3	15.5	15.3	0.186	0.934	0.732	0.813
7 d	13.1	13.1	13.1	13.1	0.209	0.953	0.987	0.996
14 d	11.5	11.1	11.7	11.6	0.219	0.429	0.621	0.722
21 d	10.9	10.2	10.8	10.5	0.227	0.793	0.342	0.675
Backfat loss, mm	4.5	5.1	4.7	4.7	0.201	0.826	0.448	0.487
ADFI, kg	6.1	6.0	5.9	6.0	0.114	0.906	0.997	0.715
Return to estrus, d	4.9	4.9	4.8	4.9	0.093	0.889	0.756	0.809

[1] Gestation diets were fed at 2 kg/d in 2 separate meals (0700 and 1800); Lactation diets were fed ad libitum up to weaning at 21d. The Arg diets were supplemented with 1% L-Arg-HCl, control diets were supplemented with 1.7% L-Ala. The number of piglets per sow ranged from 9 to 13 piglets but was equalized within weight groups (blocks).

[2] G × L = gestation × lactation interaction effect.

Table 3. Plasma urea concentrations in first parity sows fed diets supplemented with or without 1% L-Arg-HCl

	Treatment[1]							
Gestation diet	Control		Arg		SEM	*P*-value		
Lactation diet	Control	Arg	Control	Arg		Gestation	Lactation	G × L[2]
No. of observations	8	9	10	11				
Lactation, d	mmol/L							
7	4.9	4.6	4.7	4.5	0.051	0.071	0.024	0.671
21	4.6	4.5	4.7	4.6	0.056	0.351	0.218	0.434

[1] Gestation diets were fed at 2 kg/d in 2 equal-size meals (0700 and 1800 h); Lactation diets were fed ad libitum up to weaning at 21d. The Arg diets were supplemented with 1% L-Arg-HCl, control diets were supplemented with 1.7% L-Ala.

[2] G × L = gestation × lactation interaction effect.

Table 4. Plasma AA concentrations in first parity sows fed diets supplemented with or without 1% L-Arg-HCl

	Treatment[1]							
Gestation diet	Control		Arg		SEM	*P*-value		
Lactation diet	Control	Arg	Control	Arg		Gestation	Lactation	G × L[2]
No. of observations	8	9	10	11				
AA	µmol/L							
Ala	882	451	778	504	61.84	0.802	0.002	0.442
Asn	109	80	77	88	7.92	0.468	0.555	0.233
Asp	31	25	26	30	1.96	0.880	0.872	0.197
Arg	178	325	140	361	27.68	0.981	< 0.001	0.321
B-Ala	45	44	49	49	1.45	0.162	0.942	0.940
Cit	67	52	69	56	3.27	0.607	0.032	0.878
Cys	298	301	304	310	4.53	0.478	0.625	0.886
Glu	244	165	177	128	24.09	0.301	0.204	0.757
Gln	623	466	554	451	22.39	0.231	0.001	0.432

Gly	595	1356	1106	1489	106.14	0.517	0.002	0.235
His	122	74	110	77	5.64	0.505	< 0.001	0.264
Ile	110	108	104	108	4.64	0.750	0.937	0.807
Leu	168	162	151	153	6.82	0.486	0.756	0.928
Lys	143	118	93	133	12.31	0.488	0.757	0.215
Met	37x	28y	28y	29y	1.33	0.100	0.113	0.022
Orn	93	159	75	137	11.61	0.302	0.003	0.910
Phe	77	73	68	74	2.92	0.534	0.834	0.481
Pro	308	527	303	549	27.60	0.585	< 0.001	0.391
Ser	163	125	149	127	5.31	0.453	0.002	0.285
Tau	39	28	35	32	2.08	0.999	0.085	0.331
Thr	135	104	123	121	5.15	0.802	0.093	0.147
Try	54	50	48	47	3.17	0.550	0.737	0.786
Tyr	110	112	101	113	4.98	0.734	0.545	0.626
Val	195	183	174	191	11.75	0.798	0.900	0.577

[1] Gestation diets were fed at 2 kg/d in 2 equal-size meals (0700 and 1800 h); Lactation diets were fed ad libitum up to weaning at 21d. The Arg diets were supplemented with 1% L-Arg-HCl, control diets were supplemented with 1.7% L-Ala. Blood samples were obtained 2 h after feeding in the morning.

[2] G × L = gestation × lactation interaction effect.

Table 5. Concentrations of total AA, on d 7 of lactation, in milk of first parity lactating sows fed diets supplemented with or without 1% L-Arg-HCl

	Treatment[1]							
Gestation diet	Control		Arg		SEM	*P*-value		
Lactation diet	Control	Arg	Control	Arg		Gestation	Lactation	G × L[2]
No. of observations	8	9	10	11				
AA	mmol/L							
Ala	23.10	25.30	24.10	26.20	0.550	0.398	0.056	0.989
Asp	40.00	42.80	41.30	44.40	0.848	0.433	0.103	0.947
Arg	8.40	9.20	8.70	9.40	0.203	0.569	0.110	0.944
Cys	6.20	6.60	6.40	6.80	0.114	0.349	0.065	0.948
Glu	65.40	69.20	66.60	71.80	1.010	0.359	0.034	0.714
Gly	15.30	16.80	15.50	17.80	0.399	0.489	0.018	0.651
His	6.20	6.60	6.40	6.80	0.129	0.391	0.112	0.833
Ile	18.20	19.90	18.80	20.60	0.453	0.494	0.060	0.976

Leu	35.50	38.20	35.90	38.70	0.847	0.831	0.114	0.982
Lys	29.40	31.60	30.50	32.60	0.656	0.431	0.117	0.946
Met	7.10	7.60	7.20	7.80	0.157	0.552	0.100	0.764
Phe	13.10	14.40	13.30	15.30	0.312	0.376	0.007	0.532
Pro	51.00	54.10	52.30	55.60	0.901	0.423	0.081	0.952
Ser	23.60	26.10	25.10	27.60	0.604	0.198	0.039	0.982
Thr	20.10	22.00	20.60	23.30	0.471	0.335	0.014	0.672
Try	3.40	3.60	3.40	3.70	0.070	0.585	0.056	0.520
Tyr	11.10	12.80	11.60	13.60	0.332	0.338	0.006	0.838
Val	22.60	24.20	23.20	25.50	0.489	0.357	0.052	0.720
TP[3], g/L	39.97	43.09	41.08	44.75	0.759	0.371	0.030	0.856

[1] Gestation diets were fed at 2 kg/d in 2 equal-size meals (0700 and 1800 h); Lactation diets were fed ad libitum up to weaning at 21d. The Arg diets were supplemented with 1% L-Arg-HCl, control diets were supplemented with 1.7% L-Ala.

[2] G × L = gestation × lactation interaction effect.

[3] Total protein.

Table 6. Concentrations of total AA, on d 21 of lactation, in milk of first parity lactating sows fed diets supplemented with or without 1% L-Arg-HCl

	Treatment[1]							
Gestation diet	Control		Arg		SEM	*P*-value		
Lactation diet	Control	Arg	Control	Arg		Gestation	Lactation	G × L[2]
No. of observations	8	9	10	11				
AA	mmol/L							
Ala	20.7	22.00	20.60	23.10	0.560	0.679	0.102	0.598
Asp	38.90	41.10	37.90	43.60	1.000	0.695	0.049	0.372
Arg	8.40	8.70	8.30	9.00	0.180	0.811	0.185	0.637
Cys	6.00	6.30	6.00	6.50	0.130	0.747	0.186	0.753
Glu	64.40	67.10	63.60	70.04	1.590	0.704	0.148	0.531
Gly	14.20	15.30	14.20	16.20	0.360	0.572	0.039	0.569
His	5.90	6.00	6.00	6.50	0.140	0.284	0.215	0.418
Ile	16.80	17.90	16.70	19.00	0.500	0.615	0.110	0.576

Leu	33.70	35.90	33.90	38.30	0.990	0.495	0.097	0.568
Lys	27.30	28.90	27.50	30.60	0.810	0.560	0.143	0.617
Met	6.90	7.30	6.90	7.50	0.130	0.602	0.069	0.672
Phe	11.80	12.40	11.14	13.20	0.340	0.745	0.075	0.349
Pro	49.10	52.40	49.30	50.43	1.070	0.624	0.052	0.685
Ser	22.20	23.50	21.90	25.00	0.550	0.562	0.057	0.429
Thr	18.4	19.50	18.20	20.60	0.470	0.630	0.077	0.511
Try	3.30	3.40	3.20	3.50	0.070	0.786	0.150	0.641
Tyr	10.30	10.90	10.10	11.80	0.290	0.541	0.069	0.407
Val	20.80	21.70	20.50	23.30	0.590	0.550	0.101	0.439
TP[3], g/L	37.93	39.15	38.56	42.29	0.882	0.290	0.166	0.482

[1] Gestation diets were fed at 2 kg/d in 2 equal-size meals (0700 and 1800 h); Lactation diets were fed ad libitum up to weaning at 21d. The Arg diets were supplemented with 1% L-Arg-HCl, control diets were supplemented with 1.7% L-Ala.

[2] G × L = gestation × lactation interaction effect.

[3] Total protein.

Table 7. Plasma insulin concentrations in first parity sows fed diets supplemented with or without 1% L-Arg HCl

	Treatment[1]							
Gestation diet	Control		Arg		SEM	*P*-value		
Lactation diet	Control	Arg	Control	Arg		Gestation	Lactation	G × L[2]
No. of observations	8	9	10	11				
Lactation, d	μg/L							
7	2.19	3.25	2.25	3.00	0.14	0.735	0.002	0.565
21	3.20	3.73	3.29	3.86	0.10	0.621	0.010	0.974

[1] Gestation diets were fed at 2 kg/d in two equal-size meals (0700 and 1800 h); Lactation diets were fed ad libitum up to weaning at 21 d. The Arg diets were supplemented with 1% L-Arg-HCl, and control diets were supplemented with 1.7% L-Ala.

[2] G × L = gestation × lactation interaction effects.