EFFECT OF DIETARY FOLIC ACID SUPPLEMENTATION ON SOW PERFORMANCE THROUGH TWO PARITIES1,2


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ABSTRACT

One hundred fifty-three gilts were maintained in three breeding groups and fed gestation-lactation diets supplemented with either 0 (control), 1.65 or 6.62 mg of supplemental folic acid/kg of diet for two consecutive parities. Serum folate concentrations of sows were linearly (P < .05) increased by dietary additions of folic acid during both gestation and lactation, but serum glucose and urea concentrations were unaffected by treatment. Serum folate concentrations decreased from breeding to d 60 and 90 of gestation and then increased through lactation for all treatments. Number of pigs born and live pigs at birth, d 14 and d 21 were quadratically (P < .05) increased by folic acid additions. Average pig weights were similar among treatments (P > .10) on both d 0 and 14 of lactation but were less (P < .01) than the other treatment groups on d 21 for pigs from sows fed the 1.65 mg/kg treatment. Litter weights were quadratically (P < .01) increased on d 0 and d 14 by folic acid supplementation. Sow weight gain and backfat thickness loss were unaffected by treatment during gestation (P > .06); sow weight loss and backfat thickness loss increased quadratically with increasing level of folic acid during lactation (P < .06 and .05, respectively). More control sows exhibited estrus by d 7 postweaning than sows receiving folic acid supplementation in parity I (P < .05); however, no differences (P > .10) were detected among treatments by d 14, nor were any differences observed by d 7 in parity II. Conception rate was unaffected by folic acid additions. Dietary folic acid supplementation improved sow reproductive performance by increasing the number of pigs born alive.

(Key Words: Folic Acid, Sows, Gestation Period, Lactation, Litter Size.)

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Introduction

Folic acid is an essential B-complex vitamin for swine (NRC, 1988). It participates in many enzymatic reactions requiring the transfer of single carbon units, most notably those associated with amino acid and nucleotide synthesis (Herbert and Das, 1976; Pike and Brown, 1986). The pig's requirement for folic acid is met mainly through dietary sources and, to a lesser extent, by bacterial synthesis in the lower gut.

Conflicting evidence exists as to the efficacy of folic acid supplementation during gestation. Easter et al. (1983) reported that sows consuming corn-soybean meal diets did not require folic acid supplementation during gestation. In contrast, Matte et al. (1984a) reported that sow serum folate concentrations drastically decreased during gestation, suggesting a possible deficiency condition. Later work by Matte et al. (1984b) and Tremblay et al.
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(1986) supports that observation. Also, when sows were injected i.m. with folic acid during gestation, serum folate concentrations were elevated and concomitant increases in number of total and live pigs born per litter were observed (Matte et al., 1984b).

Because injections of folic acid would require additional labor and management, we studied the addition of folic acid to sow diets. Tremblay et al. (1986) has shown that folic acid supplementations of 3, 9 and 27 mg/kg of feed during gestation elevated serum folate concentrations, whereas Lindemann and Kornegay (1988) reported that 1 mg of folic acid/kg of feed during gestation and lactation increased total pigs born and pigs born alive. However, no research to date has been conducted to investigate the effects of dietary folic acid additions on both sow performance and serum folate concentrations. Therefore, the objective of this study was to determine the effects of three levels of folic acid supplementation during two consecutive gestation-lactation periods on both sow reproductive performance and serum criteria.

Experimental Procedure

Animals. A total of 153 Yorkshire × Duroc × Chester White gilts from three breeding groups was utilized for two consecutive parities. Approximately 34 d before breeding, gilts were moved to an outside lot, regrouped and exposed to a mature boar (>18 mo of age). A standard 14% CP grain sorghum-soybean meal finishing diet that met or exceeded all NRC (1979) requirements was available ad libitum during this period. On d 30 and d 19 prebreeding, gilts were bled via anterior vena cava puncture and serum was analyzed for concentration of progesterone (Davis et al., 1985) to determine pubertal status. Gilts with serum progesterone concentration exceeding 2 ng/ml in either sample was considered to have attained puberty before the start of breeding. If progesterone concentrations of both samples were <2 ng/ml, the gilt was considered to have been bred on the pubertal estrus. On d 18 prebreeding, animals were moved to individual gestation stalls in a breeding barn and allotted to one of three dietary treatments based on initial weight and ancestry. Treatments consisted of either 0 (control), 1.65 or 6.62 mg of supplemental folic acid/kg of feed were obtained by adding 7.5 and 30.0 g of a 20% folic acid mix to 907 kg of the basal gestation and lactation diets containing .05 mg of folic acid/kg of feed. Dietary folic acid levels were determined by microbiological assay and were similar to calculated concentrations.

*Diets containing 1.65 and 6.62 mg of supplemental folic acid/kg of feed were obtained by adding 7.5 and 30.0 g of a 20% folic acid mix to 907 kg of the basal gestation and lactation diets containing .05 mg of folic acid/kg of feed. Dietary folic acid levels were determined by microbiological assay and were similar to calculated concentrations.

$^b$Supplied (per kg of premix): vitamin A, 1,810,000 IU; vitamin D₃, 181,000 IU; vitamin E, 7,240 IU; menadione sodium bisulfite complex, 724 mg; riboflavin, 1.81 g; pantothenic acid, 4.5 g; niacin, 10 g; choline chloride, 181 g; vitamin B₁₂, 9 mg.

$^c$Contained: Fe, 10%; Zn, 10%; Mn, 10%; Cu, 1%; I, .3%; Co, .1%.

$^d$Selenium premix provided .1 ppm Se in diet; biotin premix provided .44 mg/day per sow during gestation and .88 mg/day per sow during lactation.

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6Merrick Foods 4-80 R, Unionville, WI.
7Technicare 210 DX, Johnson & Johnson Co., Princeton, NJ.
From breeding to d 90 of gestation, gilts were fed 1.81 kg/d of the gestation diet (Table 1). Gilts that were inseminated were tested for pregnancy 30 to 40 d postbreeding, and pregnant sows were moved to outside gestation lots. On d 90, the feeding level was increased to 2.27 kg/d until farrowing. On d 108 of gestation, gilts were moved to a farrowing house, weighed, scanned for backfat thickness and housed in individual farrowing crates.

Sows and litters were weighed 24 h postfarrowing, and litters were equalized within treatment by 48 h postfarrowing. Each sow was fed 4.55 kg/d of the lactation diets (Table 1) through the 21-d lactation period to keep nutrient intake constant across all treatments. One-half the daily allowance was offered at 0800 and the remaining half was offered at 1600. Creep feed was not provided for the piglets. On d 14 and d 21 of lactation, sows and litters were weighed, and sows were scanned for backfat thickness on d 21.

Following weaning, sows were moved to the breeding unit and fed 1.81 kg/d of the gestation diets. Estrous detection was initiated and sows were managed in a manner similar to parity I. After weaning in parity II, sows were returned to the breeding barn and checked twice daily for estrus, and days from weaning to first estrus were recorded.

Blood Sampling. Blood samples were obtained by anterior vena cava puncture 3 h postprandial on d 0 and 108 of gestation and on d 14 and 21 of lactation for determination of serum folate, urea and glucose concentrations. Sows also were bled on d 30, 60 and 90 of gestation 3 h postprandial for determination of serum folate concentration. Samples were placed on ice immediately and stored overnight at 5°C. The serum was harvested by centrifugation and stored in cryogenic tubes at –60°C for later analysis. Folate concentrations were measured in duplicate using a human radioassay adopted for porcine serum by Matte et al. (1984a). Urea concentrations were determined by a modification of the automated procedures described by Marsh et al. (1965), and glucose concentrations were measured by a modification of the automated procedures described by Gochman and Schmitz (1972).

Statistical Analysis. Data were analyzed as a randomized complete block design using the GLM procedures of SAS (1984) with the effects of parities I and II included in the model. Treatments effects were separated into linear and quadratic components (Snedecor and Cochran, 1980). Treatment effects on the percentage of sows exhibiting estrus by 7, 14, and 21 d postweaning were tested as a categorical data using the FUNCAT procedure (SAS, 1984). Differences in conception rates for parities I and II were analyzed by chi-square.

Results

Reproductive Performance. Because few treatment × parity interactions existed, data from parities I and II were pooled. The least square means for the individual parities and for the pooled data are reported. The effects of folic acid supplementation on litter criteria are presented in Table 2. Number of pigs born dead was not affected by folic acid addition (P > .10). However, total number of pigs born and number of pigs born alive were increased (quadratic effect; P > .05) by folic acid treatment. The increased litter size resulting from folic acid supplementation was maintained through d 14 and 21 of lactation (quadratic; P < .001). The majority of the response in increased litter (~1 pig/litter) at birth and at 14 and 21 d occurred with the supplemental level of 1.65 mg of folic acid/kg. No differences in average pig weight at birth on d 14 of lactation were observed (P > .10) among treatment groups. However, on d 21 of lactation, average pig weight was quadratically affected (P < .01) by folic acid supplementation; pigs from sows receiving the 1.65 mg treatment weighed the least. Total litter weights were greater at birth and or on d 14 for sows receiving diets containing 1.65 mg of folic acid/kg of feed (quadratic effect; P = .01) and were numerically greater on d 21 due to folic acid supplementation (45.6 vs 47.0 vs 45.9 kg for the 0, 1.65 and 6.62 mg folic acid treatments, respectively). Though few treatments × parity interactions existed, the magnitude of response for litter criteria was greater in parity I than in parity II; in parity II, treatment differences were never significant.

8Quantaphase Folate, Folate Radioassay, Bio-Rad Laboratories, Hercules, CA.
9Industrial method #339-01, Technicon Instruments Corp., Tarrytown, NY.
10Industrial method #SE4-0036FJ4, Technicon Instruments Corp., Tarrytown, NY.
The effects of folic acid supplementation on sow performance are shown in Table 3. Gestational weight gain was unaffected by dietary treatment, but backfat thickness loss tended to decrease linearly \((P < .07)\) during gestation with increasing level of folic acid supplementation. Sow weight loss on d 21 of lactation \((P < .06)\) and lactational backfat thickness loss \((P < .05)\) were quadratically affected by folic acid supplementation; sows...
TABLE 3. EFFECT OF FOLIC ACID ADDITIONS ON SOW CRITERIA

<table>
<thead>
<tr>
<th>Item</th>
<th>Supplemental folic acid level, mg/kg</th>
<th>0</th>
<th>1.65</th>
<th>6.62</th>
<th>SE</th>
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<td>Gestation</td>
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<td>136.58</td>
<td>136.06</td>
<td>135.75</td>
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<td>Wt at breeding, kg</td>
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<td>39.74</td>
<td>40.45</td>
<td>40.32</td>
<td>.87</td>
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<td>Wt gain, kg</td>
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<td>26.23</td>
<td>25.68</td>
<td>26.16</td>
<td>.44</td>
</tr>
<tr>
<td>Backfat thickness at breeding, mm</td>
<td></td>
<td>1.68</td>
<td>.69</td>
<td>.49</td>
<td>.39</td>
</tr>
<tr>
<td>Backfat thickness loss, mm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
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<td>Lactation</td>
<td></td>
<td>162.90</td>
<td>162.43</td>
<td>161.03</td>
<td>1.18</td>
</tr>
<tr>
<td>Wt post-farrowing, kg</td>
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<td>10.97</td>
<td>13.03</td>
<td>11.99</td>
<td>.88</td>
</tr>
<tr>
<td>Wt loss, kg</td>
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<td>24.80</td>
<td>24.66</td>
<td>25.67</td>
<td>.49</td>
</tr>
<tr>
<td>Backfat thickness at d 108 of gestation, mm</td>
<td></td>
<td>1.97</td>
<td>3.09</td>
<td>2.46</td>
<td>.39</td>
</tr>
<tr>
<td>Backfat thickness loss, mm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conception rate, %^e</td>
<td>Parity I</td>
<td>76.5</td>
<td>72.5</td>
<td>72.5</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Parity II</td>
<td>71.8</td>
<td>70.3</td>
<td>70.3</td>
<td></td>
</tr>
<tr>
<td>Returned to estrus</td>
<td>Parity I</td>
<td>91.4</td>
<td>61.3</td>
<td>74.2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Parity II</td>
<td>100.0</td>
<td>100.0</td>
<td>96.8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>By d 7, %^f</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>By d 14, %</td>
<td></td>
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</tr>
</tbody>
</table>

^aParities I and II combined.
^bLinear folic acid effect (P < .07).
^cQuadratic folic acid effect (P < .06).
^dQuadratic folic acid effect (P < .05).
^e(Number farrowed/number exposed) x 100.
^fFolic acid effect (P < .05).

receiving the intermediate level of folic acid supplementation exhibited the greatest loss of weight and backfat thickness.

Conception rates for parity I (P > .75) and parity II (P > .99) were unaffected by dietary folic acid additions. However, in parity I, there was a decrease (P < .05) in number of sows fed folic acid exhibiting estrus by d 7 postweaning. By d 14 postweaning in parity I, there were no differences in the number of sows exhibiting estrus, nor were any differences observed in parity II by d 7 postweaning. Average days from weaning to estrus for parity I were 6.20, 7.45 and 6.85 and for parity II were 4.96, 4.72 and 4.88 for sows fed diets supplemented with 0, 1.65 and 6.62 mg of folic acid/kg, respectively.

Progestosterone concentrations in serum on d 15 and d 4 prebreeding indicated that only one, four and five sows in the 0, 1.65 and 6.62 mg/kg treatments, respectively, were bred on their pubertal estrus.

Serum Criteria. Mean serum folate concentrations are presented in Figures 1 and 2 (parities I and II, respectively). Folic acid additions of 1.65 and 6.62 mg/kg of feed linearly increased (P < .01) serum folate concentrations in both gestation and lactation of parity I. In parity II, except on d 30 of gestation, serum folate concentrations were linearly increased (P < .05) by dietary folic acid supplementation. However, the increase was not of the magnitude observed in parity I.

Stage of the reproductive cycle also affected circulating concentrations of folate. In parity I, folate concentrations decreased from breeding to d 60 of gestation, and this decrease was more pronounced (27.2%) for sows receiving no supplemental folic acid than for sows receiving 1.65 or 6.62 mg of folic acid/kg of diet (11.1 and 9.2%, respectively). From d 60 of gestation through d 21 of lactation in parity I, folate concentrations increased in all treatment groups. Serum folate concentrations in parity II followed the same trends observed in parity I, with the exception that the decrease in serum folate concentration in gestation continued to d 90.

Serum urea and glucose concentrations are presented in Table 4. Serum urea concentrations did not differ (P > .10) among treatments on any day measured except for a linear
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decrease ($P < .05$) with supplemental folic acid on d 108 of gestation in parity II. However, in both parities, a sampling time effect was noted; serum urea concentration decreased during gestation and increased during lactation ($P < .01$). Serum glucose concentrations were similar ($P > .10$) for all days measured except on d 108 of gestation in parity II, when folic acid additions resulted in a quadratic decrease in serum glucose concentrations ($P < .05$). Across treatments in both parities, serum glucose concentrations remained constant during gestation, rose until d 14 of lactation, and then decreased through d 21 of lactation.

Discussion

Folic acid addition of 1.65 mg/kg diet resulted in an increase in litter size at birth and on d 14 and 21 of lactation by about one pig per litter. Matte et al. (1984a,b) observed a similar response and suggested that this increase in number of pigs born due to supplemental folic acid resulted from a decrease in embryonic mortality. Embryo loss is the major factor determining litter size and accounts for 58% of the variation in litter size (Paterson et al., 1980). Bazer and First (1983) suggested that pregnancy wastage or embryonic loss was a function of conceptus deficiencies. Research with women (Pritchard et al., 1969; Baker et al., 1981) and guinea-pigs (Habibzadeh et al., 1986) has shown that a folic acid deficiency during gestation increases embryonic death and fetal resorptions. Therefore, adequate levels of folic acid appear to be essential in maintaining embryo survival.

Folic acid is an essential cofactor in DNA, RNA and amino acid synthesis (Herbert and Das, 1976). Through its role in DNA synthesis, folic acid is thought to enhance embryonic survival (Baker et al., 1981). A lack of folic acid not only inhibits DNA synthesis (Blair and Newsome, 1985), but also it can result in defective DNA synthesis by causing the misincorporation of uracil instead of thymine into DNA (Anonymous, 1983; Li and Zhou, 1985). In a folic acid deficiency, the deoxyuridine triphosphate (dUTP):deoxycytidine triphosphate (dTTP) ratio is increased and uracil becomes incorporated in DNA. As the dUTP:dTTP ratio becomes larger, all the misincorporated uracil cannot be removed from the DNA by excision repair. The resulting defective DNA synthesis in the fetus could cause an increase in embryonic abnormality and/or death. Because dietary additions of folic acid have been shown to increase serum folate concentrations in sows in this study and in others (Tremblay et al., 1986), folic acid supplementation may have decreased embryonic mortality by increasing fetal folate con-
### TABLE 4. EFFECT OF FOLIC ACID ON SERUM UREA AND GLUCOSE CONCENTRATIONS

<table>
<thead>
<tr>
<th>Item</th>
<th>Supplemental folic acid level, mg/kg</th>
<th>0</th>
<th>1.65</th>
<th>6.62</th>
<th>SE</th>
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<tbody>
<tr>
<td><strong>Urea, mg/dl</strong></td>
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<tr>
<td>Parity I</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>d 0 of gestation</td>
<td></td>
<td>22.01</td>
<td>20.93</td>
<td>22.50</td>
<td>.75</td>
</tr>
<tr>
<td>d 108 of gestation</td>
<td></td>
<td>16.10</td>
<td>15.69</td>
<td>17.36</td>
<td>.63</td>
</tr>
<tr>
<td>d 14 of lactation</td>
<td></td>
<td>21.57</td>
<td>22.18</td>
<td>20.71</td>
<td>.83</td>
</tr>
<tr>
<td>d 21 of lactation</td>
<td></td>
<td>22.79</td>
<td>22.24</td>
<td>22.82</td>
<td>.76</td>
</tr>
<tr>
<td>Parity II</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>d 108 of gestation</td>
<td></td>
<td>19.28</td>
<td>19.09</td>
<td>18.72</td>
<td>.69</td>
</tr>
<tr>
<td>d 14 of lactation</td>
<td></td>
<td>21.98</td>
<td>23.37</td>
<td>24.44</td>
<td>.81</td>
</tr>
<tr>
<td>d 21 of lactation</td>
<td></td>
<td>23.18</td>
<td>24.64</td>
<td>25.13</td>
<td>1.12</td>
</tr>
<tr>
<td><strong>Glucose, mg/dl</strong></td>
<td></td>
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<tr>
<td>Parity I</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>d 0 of gestation</td>
<td></td>
<td>82.27</td>
<td>83.85</td>
<td>78.35</td>
<td>1.58</td>
</tr>
<tr>
<td>d 108 of gestation</td>
<td></td>
<td>83.60</td>
<td>81.30</td>
<td>83.70</td>
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<td>d 14 of lactation</td>
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<td>95.49</td>
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<td>d 21 of lactation</td>
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<td>90.51</td>
<td>94.31</td>
<td>1.78</td>
</tr>
<tr>
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<td></td>
<td></td>
</tr>
<tr>
<td>d 108 of gestation</td>
<td></td>
<td>82.93</td>
<td>76.25</td>
<td>82.49</td>
<td>2.04</td>
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<td>d 14 of lactation</td>
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<td>90.13</td>
<td>87.20</td>
<td>90.43</td>
<td>2.30</td>
</tr>
<tr>
<td>d 21 of lactation</td>
<td></td>
<td>83.92</td>
<td>82.60</td>
<td>79.70</td>
<td>2.22</td>
</tr>
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</table>

*aLinear folic acid effect (P < .05).*

*bQuadratic folic acid effect (P < .05).*

Concentrations and supporting normal embryonic DNA synthesis during gestation in our study. The increase in litter weight for sows supplemented with folic acid observed at birth and on d 14 of lactation is attributed mainly to the larger litter size. Even though the larger litter size was maintained on d 21 of lactation, the lack of a treatment response (P > .10) on d 21 is because of the decrease in average pig weight on d 21. Even though there were more pigs from sows fed the 1.65 mg folic acid diets on d 21, their average weight was less, so total litter weight was not affected. A similar negative relationship between litter size and average pig weight was observed by Cromwell et al. (1989). Average pig weight on d 21 may have been decreased due to a deficiency of nutrients between d 14 and 21 of lactation for pigs nursing sows receiving the 1.65 mg folic acid treatment. Because sow milk production peaks approximately on wk 3 of lactation (Pond and Maner, 1984) and no creep feed was offered in this study, pigs were competing for a fixed amount of nutrients. Also, maximum milk production may not have been attained in the latter stages of lactation due to the limited amount of feed fed (4.55 kg/d). Further exacerbating the situation is the increase in the daily total nutrient requirements as the pig gets older. Therefore, because available nutrients were fixed and sows receiving the intermediate folic acid treatment had more pigs on d 21, each pig presumably received a smaller daily amount of total nutrients than did pigs from sows fed diets supplemented with either 0 or 6.62 mg of folic acid/kg of feed. If the amount of daily nutrients was not meeting the pig's increased nutrient requirements on d 21, a decrease in pig weight gain, as was observed in this study, would result.

The effect of folic acid additions on litter criteria was more pronounced in parity I than in parity II. One possible explanation may be related to the stage of growth in the dam. Because the gilt is still in a growing stage during the first gestation, more nutrients are required to sustain the growth. This decreases the total amount of nutrients, including folic acid, available to the embryo. Therefore, supplemental folic acid may replace some of the folic acid used for tissue accretion. When the sow is in the second gestation, less growth occurs, making more folic acid available to the fetus, which decreases the effect of supplemental folic acid.

Davis et al. (1987) reported that gilts bred on their pubertal estrus had smaller litters than gilts bred on a later estrus. Because only 10 of the 153 gilts were bred on their pubertal estrus, pubertal estrus had little, if any, effect on litter data.
Lactational losses of backfat thickness and sow body weight can be attributed to the sow catabolizing body tissue to provide metabolites for milk synthesis (Stahly et al., 1979; Nelssen et al., 1985). Because there was a quadratic response to folic acid in the number of pigs during lactation, one would expect quadratic decreases in lactational weight and backfat thickness as the sow catabolizes more body tissue trying to meet the increased nutrient requirement due to the extra pig per litter.

Supplementation with folic acid caused a delay in return to estrus following weaning in parity I. In humans, days to return to menstruation after parturition is correlated highly \((r = -0.81)\) with lactational weight gain (Anonymous, 1982a). Delay in return to estrus has been observed in primiparous sows fed low-energy (Nelssen et al., 1985) and low-protein (Brendemuhl et al., 1987) lactation diets. Because sows fed folic acid-supplemented diets lost more weight and backfat thickness than control sows, they may have experienced lactational nutrient deficiencies that delayed their return to estrus by approximately 1 d.

In this study, serum folate concentrations were increased due to dietary folic acid additions. Other researchers have reported elevated serum folate concentrations in women (Baker et al., 1981) and sows (Tremblay et al., 1986) receiving gestation diets supplemented with folic acid. These data indicate that sows can absorb increasing amounts of dietary folic acid. Absorption of folic acid occurs primarily at the proximal portion of the small intestine (Rose et al., 1978; Coleman et al., 1979; Halsted, 1980). However, the exact method of absorption has not been ascertained. Possibilities include passive diffusion (Spencer and Bow, 1964; Bertino et al., 1975), a Na-dependent saturable carrier mechanism (Rose et al., 1978; Selhub et al., 1984) or a combination of these two (Halsted 1980; Vincent et al., 1985). However, in a comprehensive study, Schron et al. (1985) suggested that folic acid is absorbed via a carrier-mediated folate/OH-exchange mechanism.

Research in swine (Matte et al., 1984a; Tremblay et al., 1986), humans (Temperley et al., 1968; Anonymous, 1982b) and rats (Tigner and Roe, 1978) corroborate the gestational decrease in serum folate concentrations observed in this study. Rapid growth of fetal membranes and fetuses during mid-gestation (Pond and Houpt, 1978) increases the requirement for all nutrients involved in protein synthesis. Because folic acid is involved intimately in amino acid and nucleotide synthesis, the requirement for this vitamin by the dam would be expected to increase during gestation. An increase in folic acid requirements of the gravid uterus might decrease serum folate concentration during gestation as observed in this study.

After mid-gestation, serum folate concentrations increased through lactation. This increase in folate concentrations may be related to placental growth and maintenance. During gestation, the placenta grows proportionally earlier than the fetus (Baker et al., 1981). Due to this larger growth, the placenta's nutrient requirements are proportionally greater during the early stages of gestation. However, by d 70 of gestation, most of the growth of the placenta is complete (Pond and Maner, 1984). Therefore, the placenta would sequester less folate from the maternal serum for protein synthesis, resulting in an elevation of serum folate concentrations during the latter stages of gestation.

After parturition, the sow no longer has to provide for the placenta and growing fetuses. These changes result in a decrease in the amount of protein metabolism occurring and thereby decrease the need for folic acid. Also, during lactation, feeding level was increased 2.25 times over that of gestation, so daily intake of folic acid was increased by this amount. The combination of a decreased requirement and increased intake of folic acid could elevate serum folate concentrations, as observed during lactation.

Many of the criteria measured responded in a quadratic fashion. This is difficult to explain because the range of folic acid concentrations investigated is not large enough to cause toxicity in the animal. One possible explanation is that the fetus may be much more sensitive to nutrient concentrations than the postnatal animal. If true, a toxicity or an imbalance could exist, causing responses to be quadratic. However, no data are available to substantiate or refute this hypothesis.

In our study, dietary additions of folic acid increased gestational and lactational serum folate concentrations and 1.65 mg of folic acid/kg of feed improved sow performance by increasing the number of pigs per litter. Only three levels of folic acid supplementation were
investigated; therefore, further dose-titration studies are needed to determine the folic acid requirement of gestating and lactating sows. However, because most of the responses in our experiment were quadratic, the sow’s folic acid requirement presumably is less than 6.62 mg of folic acid/kg of feed.

Implications

Number of pigs born alive is one of the major factors influencing the profitability of a swine operation. By increasing number of pigs born alive and weaned, production, and possibly profitability, are increased. Folic acid addition of 1.65 mg/kg feed in gestation and lactation diets increased the number of pigs born alive by approximately one pig/litter through two parities in this study. Though more work needs to be done in determining its optimum level, folic acid supplementation increased the number of pigs born alive, and thereby enhanced swine production.

Literature Cited


Cary, NC.


