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In this issue:

Reducing Vitamin D, Calcium & Phosphorus

As a result of a feeding error at the University of Wisconsin Swine Research and Teaching Center in 2008, a spontaneous outbreak and subsidence of kyphosis occurred in a four month period. The error was an accidental omission of vitamin D_3 in one of two premixes used in sow diets during an experiment which involved feeding diets with marginal calcium and phosphorus concentrations. The deficient premix was fed to gestating sows that delivered the pigs affected with kyphosis. Because of this mistake, this reported experiment looked to duplicate the omission of the supplemental vitamin D_3 in one of the two vitamin premixes used in sow diets and then assess the incidence of kyphosis and skeletal integrity of the offspring.

The experiment:

The experimental design factors included gestation, lactation, nursery and grower phases. The University used crossbred (Landrace x Large White), multiparous sows who were bred via AI with semen from Line 19 (PIC Inc., Hendersonville, TN) boars. The sows were randomly assigned in groups of four, and were fed one of two dietary treatments that varied in vitamin D_3 supplementation at breeding.

At the research and training center, routine sow diets are supplemented with vitamin D_3 (325 IU/kg), using a combination of two custom-mixed vitamin-trace mineral mixes. One premix was formulated for growth with 280 IU/kg of vitamin D_3. The second custom-mixed vitamin-trace mineral premix was used in sow diets to supply additional vitamins and trace minerals required by gestating and lactating sows beyond the amounts needed in diets for grow/finish pigs. This mix contained an additional 45 IU vitamin D_3/kg. In this experiment, two treatment diets were formulated. The control diet for sows contained the 325 IU vitamin D_3/kg and was made up of the routine premixes (**+D**). The second diet was formulated to be deficient of supplemental vitamin D_3 (**-D**). The -D experimental diet was calculated to supply 45 IU vitamin D_3/kg. Sows were fed either the +D or the -D diets from breeding (0d) until parturition. At parturition, the sows continued in their treatment groups, but the diets were switched to –D or +D lactation diets.

Pigs were weaned at 26 ± 1 d of the lactation period (at week 4) and assigned to one of the two nursery treatment groups. Again, in the attempt to duplicate the earlier omission, the growth vitamin-trace mineral mix did not include supplemental vitamin D_3. The nursery

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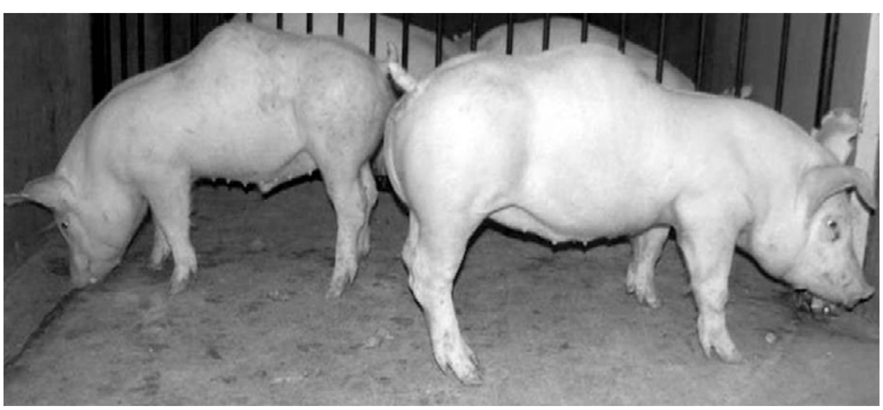
diets were also formulated to either supply 120% of the calcium and phosphorus requirements or 80% of the requirements. In the first phase after weaning, the treatment diets were formulated for 5- to 10-kg pigs, and the phase 2 diets were for 10- to 20-kg pigs. Phase 1 treatment diets were fed for 12 d following the assigned phase 2 diets until the end of the trial. At 40 d, three pigs were selected and subjected to dual energy X-ray absorptiometry scans and then euthanized and bone samples were collected. The scans were used to determine bone mineral content and bone mineral density. At week 9 poor mobility, mild tetany and ataxia in pigs on the reduced (80%) calcium and phosphorus diets were observed. Because of these observations, all pigs were then put on the higher calcium and phosphorus (120%) diets for the remainder of the experiment (wk 9-13). This allowed for observations on the incidence of kyphosis in the remaining pigs.

Researchers recorded gross, visual symptoms of kyphosis in pigs at wk 4, 9, and 13. A score was assigned to the pigs based on the visual evidence of spinal curvature on a scale of one to three. A score of one indicated no evidence of curvature and score of three indicated clear evidence of abnormal spinal column curvature. The picture below shows the incidence of kyphosis from the 2008 outbreak at the University of Wisconsin Swine Research and Teaching Center that this current experiment was attempting to duplicate.

What the research found:

This study primarily attempted to determine the effect of supplemental vitamin D deletion in maternal diets on the incidence of kyphosis in their offspring. At weaning, there was no visual evidence of kyphosis. Pigs produced by -D sows on the low calcium-phosphorus diets exhibited a 17% incidence of kyphosis at week 9. By week 13, the incidence of kyphosis increased to 32%. This is because two additional pigs were displaying symptoms.



Picture 1. Example of kyphosis symptoms observed in the 2008 outbreak at the University of Wisconsin Swine Research and Treatment Center. Kyphosis was observed in both gilts and barrows. Symptoms were not clearly evident until around 5-8 weeks of age.

At week 9, pigs from –D sows with high calcium and phosphorus concentration nursery diets and those on the +D diets regardless of calcium and phosphorus levels in the nursery diets did not show symptoms of kyphosis. However, at week 13, the pigs on +D diets and low calcium and phosphorus levels showed a 26% incidence of kyphosis. Since it took until week 13 for pigs on diets with higher levels of calcium and phosphorus to show symptoms, it appears that the amount of maternal vitamin-D supplementation alters the amount of time required to display kyphosis in pigs from maternal diets devoid of vitamin-D supplementation and nursery diets of marginal calcium and phosphorus concentrations. This study may be the first documentation reporting diet-induced kyphosis.

In addition to the incidence of kyphosis, the researchers also looked at other swine traits to characterize responses to dietary items. These traits included pig body weight, growth, serum calcium and phosphorus concentrations and measurements of femur mineral content and mechanical properties.

Average pig body weight and growth of pigs from sows fed vitamin-D deficient diets were reduced by more than 10% compared to pigs from sows on the routine vitamin-D3 supplemented diets. The growth responses were expected and are most likely attributed to the reduced dietary phosphorus concentrations. Across the dietary treatments, pigs fed diets of lower calcium and phosphorus concentrations experienced more than a 20% reduction in growth at weeks 9 and 13.

The interaction between the maternal and nursery diets was expected because of the interrelationships between vitamin-D, calcium and phosphorus. In this study, there were no

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detected differences in growth because of an interaction between the maternal and nursery diets at week 9 or 13. Pigs fed diets with higher concentrations of calcium and phosphorus had approximately 0.10 kg/day reduced growth rates if produced by sows on the -D diets compared to those from sows on the +D diets. The growth rates for pigs fed the lower concentrations of calcium and phosphorus nursery diet were almost identical regardless of the maternal diets. Again, it is important to remember that all pigs were fed the same diet of higher calcium and phosphorus concentrations during the week 9-13 period. There was not conclusive evidence that pigs from maternal diets of routine vitamin-D supplementation differed in their ability to recover from suppressed growth when compared to pigs from vitamin-D deficient sows.

The whole body bone mineral content responses were similar to growth responses across the dietary treatment groups. During the experiment, pig bone mineral content increased approximately 1.5- to 3.5-fold between weeks 4 and 9. Further growth increases were 2.5- to 3-fold between weeks 9 and 13. No significant differences were in the bone mineral content in pigs from the different maternal diets (-D or +D). There were significant differences in the bone mineral content between the pigs' nursery diet treatments (varying levels of calcium and phosphorus). Those differences were dependent on the maternal diet.

At week 13, the pattern was reversed. Pigs from -D sows accumulated approximately 45% less bone mineral content when fed the lower calcium and phosphorus concentration nursery diet compared to the pigs fed the nursery diet with higher concentrations of these nutrients. Pigs from +D sows and who were fed lower nutrient (Ca & P) concentrations only accumulated approximately 25% less bone mineral content compared to maternal +D diets and higher nutrient concentrations nursery diet treatment. It is unclear if the bone mineral content between weeks 9 and 13 is a carryover from the maternal diets or an age-related maturity of skeletal tissue mineralization that is outside of this experiment. Further, bone mineral content differences were confounded by the differences in body weight.

The authors looked at bone mass density to correct for the differences in skeletal size. Bone mineral density is calculated by dividing bone mineral content by the skeletal tissue area. Using this bone mineral density correction for difference in pig size did not nullify the observed responses to maternal and nursery treatments. The extent of the bone mineral density differences between treatment groups was less than the differences in the bone mineral content, but differences were observed. At week 9, pigs from vitamin-D deficient sows had approximately 5% less bone mineral density than those from the maternal diets with routine vitamin-D supplementation. By week 13, the differences due to maternal diets were approximately 2% and not significantly different. When comparing nursery diet treatments, the pigs fed lower nutrient concentrations had a 10-20% bone mineral density reduction compared to pigs fed the higher nutrient concentrations regardless of maternal diets. This trend for the greater difference due to nursery diets in pigs from vitamin-D deficient diets compared to pigs from sows fed routine vitamin-D supplementation is consistent with the bone mineral content observations. This finding means the responses on the nursery diets were not just a reflection of differences in pig body weight.

Femur samples were collected to compare dietary treatments in this experiment. The femur bone mineral content and bone mineral density values were similar to the whole body bone mineral values in response to maternal and nursery diets. Femurs from pigs on the nursery diet of lower nutrient concentrations were approximately 5 and 12% shorter than the femurs of pigs on the higher calcium and phosphorus concentrations, at weeks 9 and 13 respectively. These results of shorter femur lengths were consistent with the smaller body weight of pigs on lower nutrient concentration nursery diet. There was a difference in the midshaft diameter of the femurs. At week 9, the femur diameters of the pigs on the nursery diet of lower calcium and phosphorus concentrations tended to be larger than those fed the higher nutrient concentration nursery diet. The trend was more evident in pigs on the lower nutrient concentrations nursery diet produced by sows on the +D maternal diets.

In addition, the experiment evaluated the bending moment of the femur samples. This is a measure of the force corrected for the span over which the load was applied. The femurs from pigs fed the lower nutrient concentration nursery diet endured

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significantly greater strain during testing. This means that those bones bent more during the testing at week 13.

The calcium and phosphorus serum concentrations were collected during the experiment at weeks 4, 9 and 13. Serum calcium concentrations were significantly greater at week 4 in pigs from the +D maternal diets than -D sows. While the finding was statistically significant, the magnitude of the difference was small. The difference between the serum phosphorus concentrations was greater both statistically and in magnitude for the same maternal diet treatments (-D vs. +D) at week 4. Serum calcium concentrations reached the upper physiological range (14 mg/dL) at weeks 9 and 13. In the pigs from sows fed -D maternal diets, serum calcium concentrations were significantly greater than for those fed the +D maternal diets at week 9. Serum calcium concentrations were greater in pigs fed the nursery diet of lower nutrient concentration versus the higher nutrient concentrations at week 9. By week 13, there were no differences observed across treatment groups in serum calcium concentrations. These results led the researchers to imply that serum calcium does not follow the growth and bone mineralizing responses to diets.

Growth and bone trait responses to dietary inputs were mirrored by serum phosphorus concentrations. Differences due to maternal diets were observed at week 9. Serum phosphorus concentrations in pigs from the sows fed -D maternal diet were reduced by approximately 10% compared to the pigs from sows fed the +D maternal diet. Pigs fed the lower nutrient concentration nursery diet had serum phosphorus concentrations of approximately half that of pigs fed the higher nutrient concentration nursery diet. The serum phosphorus concentrations of pigs from -D sows were reduced 20% if the pigs were fed the lower nutrient concentration nursery diet.

What this means:

This lengthy report confirms that a deletion of vitamin-D from the vitamin premix induced kyphosis in young pigs. Concentrations of vitamin-D in maternal diets affects the period of time for visible symptoms of kyphosis to be displayed.

Another factor in inducing skeletal defects is the reduction of dietary calcium and phosphorus concentrations. While the marginal deficiencies of the experiment were not dramatic deficiencies, they may have been exacerbated by the limited supplemental vitamin D. The researchers suggest that the marginal deficiencies of all three nutrients (vitamin -D, calcium and phosphorus) exacerbated response symptoms or the life-cycle phases when the deficiencies were imposed were critical. To know more definitively, the role of maternal vitamin-D status in fetal and neonatal skeletal tissue development needs to be clarified.

There is evidence that vitamin $D_{3,}$ and hydroxylated metabolites of vitamin D readily cross the placenta. As a result of this transfer, maternal vitamin D status may potentially influence the reservoir of vitamin-D in the fetus, and thus influence neonatal development of the pig. In addition, research has shown the presence of 1, 25-dihydroxyvitamin D_3 receptors before ossification (bone formation) and in patterns consistent with skeletal tissue differentiation (Johnson et al. 1996). This indicates a functional role for vitamin D as stem cells differentiate into cartilage and bone during neonatal development.

More research is necessary to determine if a vitamin-D deficiency during fetal development will directly affect vertebrae in the deformed regions of the spinal column in pigs displaying kyphosis. This research will help understand the maternal-fetal vitamin-D relationship. As this research expands, it may even be possible to gain a better understanding of this maternal-fetal nutrient relationship in human gestation and fetal development as it pertains to understanding and preventing the development of Scheuermann's Juvenille Kyphosis in humans.

Information for this article taken from:

Rortvedt, L. A and T.D. Crenshaw. 2012. Expression of kyphosis in young pigs is induced by a reduction of supplemental vitamin D in maternal diets and vitamin D_3, Ca and P concentrations in nursery diets. Journal of Animal Science. 90:4905-4915.

Johnson, J.A., J.P. Grande, P.C. Roche, and R. Kumar. 1996. Ontogeny of the 1,25-dihydroxyvitamin D_3 receptor in fetal rat bone. Journal of Bone and Mineral Research. 11:56-61.