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Boron Supplementation Prevents Osteochondrosis in Growing Swine

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The effect of adding 25 ppm boron as experimental supplement (XPI-296) plus 0 or 250 ppm ascorbic acid to a commercial swine diet containing 10 ppm boron and 18% crude protein was evaluated in rapidly growing, heavily muscled pigs with history of lameness due to osteochondrosis (OCD). Fifty-six pigs (35 Durocs, 21 Yorkshires) with mean BW 42 lb were allocated to the three available pens in a modern deep-straw-bedded curtain-sided growfinish house. One pen of 19 pigs was fed commercial diet plus 25 ppm boron, another group of 19 pigs were fed that diet plus 25 ppm boron and 250 ppm ascorbic acid. Eighteen pigs received only commercial diet. Pigs were weighed individually every 3 wk and observed for signs of lameness. Pigs were scored on leg soundness from 1 to 5 on the following scale: 1) sound, no defects. 2) minor structural and mobility issues, but animal suitable for breeding purposes 3) unsound, not suitable for retention as breeding animal but could be sold to slaughter. 4) severely unsound, animal likely to be rejected as unsuitable for slaughter 5) crippled, in need of euthanasia for humane reasons. Scores 1 and 2 were interpreted as “sound”, scores 3 to 5 as “lame”. Evaluations of lameness were verified by an experienced treatment-blind evaluator. At the end of the 3-mo trial, pigs were euthanized and joints evaluated for visible lesions of OCD.

Among the pigs receiving supplemental boron, 1 of 38 was lame at end of trial as compared with 11 of 18 of the unsupplemented pigs (OR for lameness without supplemental boron = 58.1, $p < 0.001$). Growth rate did not differ across treatments. Two severely lame pigs from the no supplemental boron control group were removed from the trial. Among Durocs, 8 of 11 (73%) of the no-boron control pigs were persistently lame compared with 1 of 23 (4%) in the boron-treated groups ($p = .001$). For Yorkshires, lameness rate was 3 of 7 (43%) among the no-boron pigs. 0 of 14 (0 %) in

boron-treated groups ($p = 0.008$). Lameness rate did not differ ($p = 0.15$) between breeds, but Yorkshires were more likely to have a normal soundness score of 1 ($p = 0.003$). No significant difference in any variable was found between pigs that received boron plus ascorbic acid and those receiving only boron. Hock and elbow OCD scores (1 = normal to 4 = severe) were lower in boron-supplemented pigs ($p < 0.01$).

These results give evidence confirming the hypothesis that supplemental boron at 25 ppm can reduce the prevalence of lameness due to osteochondrosis in growing swine. The level of boron (25 ppm added plus 10 ppm background) fed here might be similar to what a foraging pig in the wild would consume.

Utility of boron for plants was demonstrated in 1910, and boron has been considered essential for plants since the 1920s. A major function of boron in plants is structural stabilization of the cell wall by cross-linkage of hydroxyl bearing moieties. Boron deficiency in plants is characterized by pathologic changes in the cell wall at the growing points. Boron-deficient plant tissues are brittle or fragile. Plants grown on adequate boron levels are flexible or resilient. Animals do not possess cell walls, but some tissues such as cartilage and neural tissue have abundant hydroxyl-rich extracellular matrix. The physis pathology in OCD parallels boron-deficiency effects in the cambium of plants.

From these results, it may be concluded that osteochondrosis in swine apparently is a boron-responsive disease. But boron is not yet FDA-approved in any form for use in animal feeds. Osteochondrosis may be one manifestation of pandemic boron deficiency in swine fed low-boron-grain-based diets without boron supplementation. Further research should determine dose and other biologic effects.