T309 Effect of vitamin E supplementation on its hepatic concentration in broiler chicken. M. A. Pompeu*[1], N. C. Baião[1], L. J. C. Lara[1], V. M. Barbosa[3], J. S. R. Rocha[1], P. C. Cardeal[1], R. C. Andrade[1], C. E. Cunha[1], C. W. R. Gondim[1], and L. F. L. Cavalcanti[1,2], *[1]Universidade Federal de Minas Gerais, Belo Horizonte, Minas Gerais, Brazil, [2]Texas A&M, College Station, [3]Universidade Federal da Bahia, Salvador, Bahia, Brazil.*

The main goal of vitamin E (VE) supplementation on poultry diets is to ensure good performance and welfare, rather than to attend the nutritional requirements. The liver is the main regulator of the body's VE levels and it appears to be the major site of VE metabolism and excretion, making it a good indicator of VE's body status. This study aimed to evaluate the VE liver concentration (VELC, mg/kg) in broilers with 21 or 39 d of age, fed with increasing levels of VE supplementation. The initial phase (IP) comprehended the birth to 21 d of age in which 15 animals were supplemented with 5 levels (3 animals/level) of VE, to achieve diets concentrations of 10, 30, 50, 75 or 100 mg/kg of DM. Additionally, another 15 animals (3 animals/level) were fed with the same supplementation levels from 21 to 39 d of age during the growth phase (GP). At the end of each phase, the animals were slaughtered and their livers were collected. The VELC was assessed by HPLC. A linear cubic regression was fitted to the IP ($VELC = a+b \times D+c \times D^2+d \times D^3$), whereas nonlinear logistic model was fitted to the GP phase [$VELC = A \times (1-B \times e^{-k \times D})^{-1}$]; where, A = asymptote (mg/kg), B = constant related to the intercept (mg/kg), k = growth rate (1/mg/kg), D = VE concentration in the diet (mg/kg). For both phases, was observed an increase on VELC when VE levels were increased from 10 to 30 mg/kg. However, for IP it readily tended to a plateau (61.99 ± 13.3 mg/kg) until the highest VE level, when it abruptly reached 135.3 ± 28.6 mg/kg, justifying the fitted cubic model ($a = -20.2 \pm 18.1$, $b = 4.3 \pm 1.5$, $c = -0.08 \pm 0.03$, $d = 0.0005 \pm 0.0001$, $r^2 = 0.89$). For the GP this plateau was only achieved when the diet VE levels reached 50 mg/kg, with a VELC close to the estimated A parameter of the logistic model ($A = 112.6 \pm 9.8$, $B = -151.53 \pm 429.4$, $k = 0.15 \pm 0.1$, Residual SE = 23.73). For the IP, the VE supplementation above 75mg/kg seems to overload the liver metabolism capacity, resulting in VE accumulation, what did not happen in the GP. The observed plateaus may be related to a balance between liver requirement and utilization, and the decision of the exact VE diet levels below those plateaus should be made based on additional performance results.

Key Words: diet, poultry, tocopherol

T313 Effects of a dietary antioxidant blend and vitamin E on growth performance and meat quality in broilers fed a high oxidants diet. T. Lu*[1], R. A. Dalloul[1], J. Zhao[2], and A. F. Harper[1], *[1]Virginia Tech, Blacksburg, [2]Novus International Inc., St. Charles, MO.*

The aim of this study was to determine the effects of a dietary antioxidant (AOX) (Agrado Plus, Novus Inc.) and vitamin E (VE) on performance and meat quality in broilers fed a diet high in oxidants. Cobb 500 male broilers (n = 1200, d 0) were randomly distributed into 60 floor pens across 6 treatments with 10 replicate pens of 20 chicks each. Treatments included (1) NC [negative control, VE at 10 IU/kg, 3% oxidized oil, 3% high PUFA source], (2) VE (NC + VE at 200 IU/kg), (3) AOX (NC + AOX at 135 mg/kg), (4) VE+AOX (NC + VE at 200 IU/kg + AOX at 135 mg/kg), (5) SC (standard control, VE at 10 IU/kg, 3% fresh oil, no high PUFA), and (6) PC (positive control, SC + AOX at 135 mg/kg). Performance parameters were measured on d 10, 21 and 42 and carcass quality assessed on d 42 by measuring drip loss, pH, and lactate levels. Data were analyzed using Glimmix of SAS with Tukey's multiple comparison. Compared with the SC birds, the NC, VE, AOX, VE+AOX groups had larger BW (855.4, 859.4, 901.3 and 889.5 vs. 785.2 g), ADG (38.6, 38.8, 40.8 and 40.2 vs. 35.3 g) and ADFI (58.2, 58.6, 60.9 and 60.1 vs. 53.1 g) from d 0 through d 21 ($P < 0.05$). However, the growth of birds fed the VE treatment fell behind that of other treatments (NC, AOX, AOX + VE, SC and PC, $P < 0.05$) during the 42 d trial. The AOX fed birds (AOX and VE+AOX) had heavier BW and ADG from d 10 forward, except that the SC treatment had the highest ADG from d 22–42 ($P < 0.05$). The AOX birds had the best G:F on d 10 and d 42, or throughout the experiment ($P < 0.05$). The high oxidants diet induced a higher drip loss (2.63 vs. 1.33, 1.24, 2.56, 1.46 and 2.08%) in NC fed birds, which was associated with a lower ultimate pH (5.91 vs. 5.95, 5.96, 6.06, 5.98 and 5.88) and the highest lactate concentration (76.48 vs. 68.48, 64.69, 58.44, 60.73 and 74.00 mmol/g) compared with VE, AOX, AOX + VE, SC and PC ($P < 0.05$). In conclusion, dietary addition of AOX or AOX plus VE was effective in improving growth. The addition of AOX alone and high VE reduced drip loss, but VE may exert a pro-oxidant property in the finisher phase as measured by growth.

Key Words: broiler, antioxidant, PUFA

T314 Pig bone trait responses to maternal vitamin D intake depend on nursery diet vitamin D and P concentrations. L. A. Rortvedt-Amundson* and T. D. Crenshaw, *University of Wisconsin-Madison, Madison.*

In earlier experiments, kyphosis was induced and bone mineral density (BMD) was reduced in pigs produced by sows fed no supplemental vitamin D_3 or minimum levels (325 IU/kg). This experiment was designed to evaluate carryover effects of maternal vitamin D_3 (D) intake on pig bone traits as implied by earlier results. In 2 trials, gilts were fed 1 of 3 diets (n = 6, 8, or 9 gilts/treatment, respectively) with 0, 325, or 1750 IU D/kg from breeding through lactation. Using a nested design, pigs within a litter at weaning (23 ± 2 d) were assigned to pens and fed an adjustment diet with no supplemental D for 1 wk. Then for 4 wk, pigs were fed 1 of 4 nursery diets (arranged as a 2 × 2 factorial) with 0 (-D) or 280 (+D) IU D/kg, each with 95% (95P) or 120% (120P) of the P requirement. Pigs were killed before colostrum consumption at birth (n = 23), weaning (n = 22), and a subsample at the end of the nursery (n = 185) for DXA (GE Lunar Prodigy) scans to determine whole body bone mineral content (BMC, g/pig) and BMD (g/cm^2). Individual femurs were scanned. Femur BMC and BMD responses were similar to pig DXA responses. Live births per litter (n = 14.3, 14.4, 14.1) were not different among maternal treatments. At birth and weaning, no differences due to maternal diet were detected in pig BMC and BMD. Pig BMC and BMD responses to nursery diets were dependent upon maternal diets (maternal × nursery diet interaction, $P < 0.05$). The interaction confirmed a carryover effect of maternal diets. BMD was reduced in pigs fed –D120P but increased in pigs fed +D120P nursery diets if produced by gilts fed 0 or 325 IU D/kg. However, BMD increased in pigs fed –D120P nursery diets if produced by gilts fed 1750 IU D/kg. Thus, maternal dietary D intake affects neonatal bone traits at 8 wk of age even though maternal dietary responses were not evident in pigs at birth and weaning.

Table 1.

Item	Maternal diet	Nursery diets				SEM[1]
		−D95P	−D120P	+D95P	+D120P	
BMD, g/cm^2	0	0.342	0.323	0.386	0.430	
	325	0.459	0.386	0.462	0.558	
	1750	0.473	0.580	0.515	0.514	0.028
BMC, g/pig	0	213	179	237	278	
	325	259	232	246	328	
	1750	276	310	270	312	12

[1]SEM pooled across 12 dietary treatments.

Key Words: phosphorus, maternal carryover, DXA

T315 True total-tract digestibility of P in monocalcium phosphate for 15- and 25-kg pigs. H. Zhai* and O. Adeola, *Purdue University, West Lafayette, IN.*

Two experiments were conducted to determine the true total-tract digestibility (TTTD) of P in monocalcium phosphate using the regression method. Forty-eight barrows (initial BW 15.7 ± 1.53 kg) in Exp. 1 and 24 barrows (initial BW 25.2 ± 1.04 kg) in Exp. 2 were used in a randomized complete block design with 6 replicate pigs per dietary treatment. Eight dietary treatments were established in Exp. 1 by incremental addition of 3.5 g/kg of monocalcium phosphate to a 3.3 g P/kg corn-soybean meal-based diet. In Exp. 2, 4 dietary treatments were constituted by incremental addition of 2.4 g/kg of monocalcium phosphate to a 2.96 g P/kg corn-soybean meal-based diet. In both experiments, limestone was added accordingly to maintain a constant Ca:P ratio of 1.25:1 across all diets. A 5-d adjustment period preceded a 5-d total collection of feces, and ferric oxide was used as a marker to time the initiation and termination of fecal collection. The results of Exp.1 showed that dietary P intake, fecal P output, and digested P increased linearly ($P < 0.001$) with the increasing supplementation of monocalcium phosphate. The regression of daily digested P against daily P intake gave a TTTD of 67.5% for P in monocalcium phosphate. The results of Exp. 2 showed that dietary P intake, fecal P output, and digested P increased linearly ($P < 0.05$) with the increasing supplementation of monocalcium phosphate. Regressing daily digested P against daily P intake gave the TTTD of 84.3% for P in monocalcium phosphate. In conclusion, the TTTD of P in monocalcium phosphate was determined to be 67.5 and 84.3% for, pigs with BW of 15 and 25 kg, respectively.

Key Words: monocalcium phosphate, pig, true total-tract digestibility