

Effects of supplementing organic microminerals and methionine with or without limiting growth during the rearing phase of replacement gilts on lameness, growth, and body composition¹

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ABSTRACT: Previous research suggested that lameness in growing pigs could be reduced using feeding strategies, such as limiting growth rate and supplementing trace minerals (TM) and (or) methionine (Met). The present study evaluates effects of 1) TM and Met and 2) limiting total lysine (Lys) during the rearing phase (90 d) of gilts (as a means to limit growth rate) on lameness, performance, and sow claw health and productivity (to first parity). Gilts ($n = 240$; 58.0 ± 11.1 kg body weight [BW]) were blocked, distributed into pens of 10 gilts, and pens were allocated to a 2×2 factorial arrangement. Factors were: 1) control or TM plus Met, which provided additional 10, 20, and 50 mg/kg of chelated copper, manganese, and zinc, respectively (0.1%, Aplomotec Plus, Tecnología & Vitaminas, S.L.; Alforja, Spain), and a 1.01 Met:Lys ratio and 2) standard Lys was formulated to meet growth requirements or low Lys to 19% below growth requirements. Feeding was provided through two phases, first between 119 and 163 d of age (phase I) and the second between 163 and 209 d of age (phase II). Diets had 2.43 and 2.31 Mcal net energy/kg for phases I and II, respectively,

and were offered ad libitum. Low Lys did not affect feed intake but rather reduced average daily gain (ADG) by 6.35% and the final BW by 3.80% compared with standard Lys ($P < 0.001$). Low Lys reduced ADG ($P < 0.001$) and gain:feed ($P = 0.012$) during phase I but not during phase II. Lameness prevalence was 7.92% during rearing and increased with time ($P < 0.001$). Final BW (151 kg) and ADG (989 g) were similar ($P > 0.05$) whether gilts displayed lameness or not. Lameness was low in severity and not affected by dietary factors. However, TM- plus Met-fed gilts were 19.2 kg heavier ($P = 0.016$) than were control at lameness detection. On the sow farm, there was no evidence for differences in lameness or claw lesions among previous dietary treatments. In conclusion, lameness prevalence during the rearing phase was similar, independent of TM plus Met supplement, low Lys, or the interaction. Insufficient reduction of ADG and low severity in lameness may have limited the potential of dietary treatments. Moreover, a greater deficiency of Lys would be needed to achieve the degree of growth reduction previously reported to lessen lameness through feed restriction.

Key words: growth, lameness, lysine, minerals, pigs

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INTRODUCTION

Lameness is a persistent problem and a welfare concern in the swine industry (Kilbride et al., 2009; Pluym et al., 2013) as a primary reason for gilt failure (Engblom et al., 2008; Jensen et al., 2010). Disorders such as osteochondrosis (OC, a defective endochondral ossification) and claw lesions are important causes of lameness (Yazdi et al., 2000; de Koning et al., 2015). Lameness includes a high cost per lame sow, estimated at up to \$180 in the United States (Deen et al., 2008) and between \$330 and \$380 in Finland (Niemi et al., 2017).

In a previous study, lameness was reduced when supplementing organic trace minerals (TM) copper (Cu), manganese (Mn), and zinc (Zn) in combination or not with high methionine (Met) such as a 1.02 Met:lysine (Lys) ratio (Fabà et al., 2018). In a subsample study, TM also increased tibia breakage force, bone density, and metacarpal weight and ash content (Fabà et al., 2019). Similarly, Frantz et al. (2008) reported effects of Cu–Mn and combined Met and threonine to reduce OC scores. TM, acknowledged as being necessary in the keratinization and horn production process (van Riet et al., 2013), improve claw health (Varagka et al., 2016). Nonetheless, extended research supports that other factors such as genotype, feed composition, average daily gain (ADG), housing conditions, and mechanical stress interact and influence OC, claw health, and therefore lameness (Nakano and Aherne 1988; Heinonen et al., 2013; Pluym et al., 2013; de Koning et al., 2014a, 2014b; Quinn et al., 2015; Le et al., 2016).

Fast growth rate has become controversial for increasing OC and lameness in pigs (Busch and Wachmann 2011; Quinn et al., 2015; de Koning et al., 2014b) or not (Ytrehus et al., 2004a, 2004b, 2007; Tóth et al., 2016). Therefore, intentionally limiting ADG may have a positive effect on lameness. Previous studies mainly used feed restriction (Quinn et al., 2015; de Koning et al., 2014b); however, growth rate could be modulated by altering amino acid density (Rozeboom and Johnston, 2007; Díaz et al., 2017), which would be a more practical approach.

The present objective is to evaluate replacement gilt diets on lameness, body composition, performance, and carryover effects on lameness and

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claws of gilts entering into production, using two different strategies in a factorial approach: 1) supplementing organic TM and Met and 2) reducing ADG by lowering dietary Lys by 19%.

MATERIAL AND METHODS

Animal Care and Use

The animals used were produced and housed in commercial swine facilities in northern Spain (Lleida). A rearing facility provided gilts to two sow sites with similar performance and management conditions. The Ethical Committee on Animal Experimentation (CEEAH) at the Universitat Autònoma de Barcelona reviewed and approved the procedures and protocols for this experiment according to the guidelines of the European Union (Directive 2010/63/EU).

Experimental Design, Housing, and Dietary Treatments

Maternal-line gilts ($n = 240$; DanAvl Dania Hybrid line, Landrace \times Yorkshire; DanBred International; Sant Cugat del Vallés, Spain) acquired from a breeding production company were used in a 90-d experiment. Gilts of 15 to 17 wk of age and 58.0 ± 11.1 kg of body weight (BW) were individually identified with ear tags, weighed, and blocked into pens of 10. Pens ($0.90 \text{ m}^2/\text{gilt}$; 60% slatted and 40% solid floor) were randomly allocated to one of four dietary treatments in a 2×2 factorial arrangement, the main factors being: 1) TM and Met levels and 2) Lys concentration. The TM and Met levels were control as 10, 40, and 110 mg/kg Cu, Mn, and Zn, respectively, and of inorganic sources and 0.31 to 0.39 Met:Lys ratio or TM plus Met, which provided the diet with an additional 10, 20, and 50 mg/kg of chelated Cu, Mn, and Zn, respectively (0.1%, Aplomotec Plus, Tecnología & Vitaminas, S.L.; Alforja, Spain), and a 1.01 Met:Lys ratio. Lys levels were standard Lys, which provided 165 g crude protein (CP)/kg with 10.0 g Lys/kg between 119 and 163 d of age (phase I) and 140 g CP/kg with 8.0 g Lys/kg between 163 and 209 d of age (phase II) or low Lys with 155 g CP/kg CP with 8.1 g Lys/kg in phase I and 140 g CP/kg with 6.5 g Lys/kg in phase II. A total of six pen replicates per treatment were

Table 1. Composition of the experimental diets (phases I and II) offered to growing gilts (as-fed basis), %

Ingredient, kg	Phase I (119–163 d of age)				Phase II (164–209 d)			
	Control		TM plus Met		Control		TM plus Met	
	Standard	Low Lys	Standard	Low Lys	Standard	Low Lys	Standard	Low Lys
Corn	20.4	23.8	19.8	22.6	16.5	16.6	15.0	15.6
Wheat	25.0	25.0	25.0	25.0	25.0	25.0	25.0	25.0
Barley	25.0	24.8	24.8	25.0	25.0	25.0	25.0	25.0
Wheat middlings	–	–	–	–	6.00	6.00	5.95	6.00
Soybean meal	15.6	13.8	15.8	13.6	7.00	7.55	7.05	7.70
Sunflower meal	6.00	5.65	6.00	6.00	7.00	7.00	7.00	7.00
Bakery byproduct	–	–	–	–	9.15	8.90	10.0	9.25
Fat	2.90	2.45	2.85	2.50	0.30	0.30	0.30	0.30
Calcium carbonate	1.08	1.10	1.06	1.03	1.60	1.60	1.54	1.55
Dicalcium phosphate	1.35	1.38	1.35	1.38	1.01	0.98	1.00	0.99
Salt	0.40	0.40	0.40	0.40	0.40	0.40	0.40	0.40
Met	0.13	–	0.84	0.63	–	–	0.63	0.45
hydroxy analogue								
L-Lys HCl	0.50	0.23	0.50	0.24	0.48	0.18	0.48	0.17
L-threonine	0.09	–	0.09	–	0.06	–	0.06	–
L-tryptophan	0.03	–	0.03	–	–	–	–	–
Premix ¹	0.40	0.40	0.40	0.40	0.40	0.40	0.40	0.40
Premix TM ²	–	–	0.10	0.10	–	–	0.10	0.10
Calculated composition, %								
Net Energy, kcal kg ⁻¹	2,425	2,425	2,425	2,425	2,310	2,310	2,310	2,310
Crude protein	16.5	15.5	16.5	15.5	14.0	14.0	14.0	14.0
Total Lys	1.00	0.81	1.00	0.81	0.80	0.65	0.80	0.65
Total Met	0.39	0.28	1.02	0.82	0.25	0.25	0.80	0.64
Met:Lys	0.39	0.35	1.02	1.01	0.31	0.38	1.01	0.98
SID ³ Lys	0.90	0.72	0.90	0.72	0.70	0.56	0.70	0.56
SID Met	0.36	0.24	0.99	0.79	0.22	0.23	0.77	0.61
SID Met + cysteine	0.65	0.52	1.28	1.07	0.49	0.49	1.03	0.88
SID tryptophan	0.21	0.17	0.21	0.17	0.15	0.15	0.15	0.15
SID threonine	0.58	0.48	0.58	0.47	0.46	0.41	0.46	0.41
Calcium, %	0.90	0.91	0.91	0.90	1.02	1.01	1.01	1.02
Phosphorus, %	0.59	0.59	0.59	0.59	0.57	0.57	0.57	0.57

¹Vitamin–minerals premix provided per kg of feed: vitamin B2, 3.5 mg; vitamin B12, 0.035 mg; nicotinamide, 20 mg; folic acid, 1.25 mg; vitamin D3, 2,000 IU; vitamin A, 10,000 IU; vitamin E, 30 mg; vitamin K3, 1 mg; vitamin B1, 1 mg; vitamin B6, 2.4 mg; D-calcium pantothenate, 14 mg; biotin, 0.125 mg; choline chloride, 400 mg; Fe (from FeSO₄·H₂O), 120 mg; I (from Ca(IO₃)₂), 0.5 mg; Cu (from CuSO₄·5H₂O), 10 mg; Mn (from MnO₂), 40 mg; Zn (from ZnO₂), 110 mg; Se (from Na₂SeO₃), 0.4 mg; phytase EC 3.1.3.26, 1,500 FTU; and butylhydroxytoluene, 25 mg.

²Vitamin–minerals premix provided per kg of feed: 10, 20, and 50 mg/kg of chelated copper, manganese, and zinc, respectively (Aplomotec Plus, Tecnología & Vitaminas, S.L., Alforja, Spain).

³Standard ileal digestible; calculated using SID coefficients for the various ingredients obtained from the NRC (2012).

used. Feed was provided ad libitum and in pelleted form using a single-space dry feeder in each pen. Also, gilts had free access to fresh water provided through drinking nipples. The entire rearing was divided into two feed phases of 44 and 46 d, with diets containing 2,425 and 2,310 kcal net energy (NE)/kg, respectively (Table 1). For each experimental period, feeds were formulated to meet or exceed nutrient requirements (FEDNA, 2013) and according to the genetics recommendations (Tybirk, 2015), except for Lys.

In the overall rearing phase, six gilts died and four had nonviable leg conformation. Therefore, the remaining 230 gilts across dietary treatments and

blocks were equally divided into two similar destination farms. Details of the farms' management and characteristics were previously described by Fabà et al. (2018). Briefly, the sow farms had a similar inventory (1700–1800 sows) and were in the same region (20 km) and integrative company and fed by the same feed mill. Equivalent feed and routines were provided. Gilts were penned (5–8 gilts/pen) for 30 d in a quarantine-adaptation barn (4 × 3 m; 40% solid floor and 60% concrete slatted floor). Then, gilts were placed in crates for a minimum of 2 wk before service after at least the second detected heat. After confirming gestation (28 d), gilts and sows were moved to group housing pens of 70 females (1.5

m²/female; 30% solid floor and 70% concrete slatted floor) with an electronic feeding system (EFS). The concrete slatted floor areas had 80-mm wide beams and a 20-mm opening between beams.

The first diet in the sow farm was the phase II control (standard Lys), which was provided for 30 d to all gilts and offered *ad libitum*. Subsequently, gestation feed formulated to 2,200 kcal NE/kg, and 5.7 g/kg standardized ileal digestible (SID) Lys was provided. From service to 28 d of gestation, feed was individually adjusted according to the body composition score (2.2 to 3.2 kg/d). After confirmed gestation, the feeding level was 2.2 kg/d for gilts and a range between 2.3 and 2.5 kg/d in multiparous sows depending on the body composition score. In the lactation room, sows received 2 kg/d of lactation feed (2,340 kcal NE/kg and 9.3 g/kg SID Lys) until farrowing day, and from the day after onward, feed was progressively increased to *ad libitum*.

Performance and Body Composition Measurements

Measurements of BW, backfat (BF), and loin depth were individually collected every 3 wk (days 119, 141, 164, 184, and 209 of age). The BF and loin depth were always collected by the same trained person at point P2 (6 cm from the midline and the last rib level) using an ultrasound scanner (AV-3000V Digital Handheld Electronic B Ultrasound Scanner, AMBISEA Technology Corp., Ltd; Hong Kong, China). Feed intake was measured by pen on a weekly basis. The ADG, average daily feed intake (ADFI), and gain:feed ratio (G:F) were calculated by pen for each feeding phase and overall. Similarly, SID Lys intake per kg BW gain was also calculated by phases using formulated feed composition.

On the sow farm, BW was measured at first service and BF was collected at service, before farrowing, and at weaning. Productive data were also recorded for the first parity. The reproductive performance included the number of total born, born alive, stillborn, and weaned piglets. The breeding performances recorded were returning to service and weaning-to-estrus interval. Also, removal reasons and the date were recorded. Removals classified as reproductive disorder included not displaying a first estrus, abortion, or consecutive repeating estrus.

Lameness and Gait Score

During rearing, lameness and gait score were evaluated by studying walking pattern on a hard, solid floor for a distance of 6 m at an average age of 119, 141, 164, 184, and 209 d by the same

observer. The gait scoring was a subjective methodology adapted from Welfare Quality (2009) and Mustonen et al. (2011) as: not lame (0); moderate difficulties with gilts having reluctance to move and reduced walking speed or some difficulties with exercise including slightly shortened stride to obvious limp (1), and, severe difficulties with gilts being minimum weight bearing to incapable of standing (2). The evaluation was performed allowing the gilt to advance and freely explore a familiar (two trainings) corridor that was well illuminated (daylight plus led lamps) without obstacles ahead. Also, gilts were classified as lame (obvious limping) or not lame (no limping), and within the lame category whether or not they were severe (barely bearing weight on the affected limb) for binomial analysis.

On the sow farm, gait score and lameness were evaluated by the same observer at three different times: 1) before first service; 2) at day 109 of gestation, and 3) at weaning. Lameness evaluation was performed during a 2-min observation by walking (6–10 m) on a solid floor corridor (moving females to the insemination line at first service and at weaning) or a 2-min observation inside the group-housing pens in gestation.

Claw Lesions

In the farrowing room, females were evaluated for claw lesions following the protocol previously reported by Fabà et al. (2018). Briefly, the lesions evaluated were: toe dewclaws; heel sole overgrowth and cracks; white line; hoof wall cracks; long hoofs; and asymmetry between digit claws. The score grades were: 0 = none, 1 = mild moderate (without affecting the integrity of the claw), and 2 = severe (affecting the integrity of the claw, including gait difficulties or indicating clinical signs of pain). The assessment included all claws and both outer and inner digits. Overall, only few ($n = 5$) lesions were classified as severe, and final analysis was for complete prevalence independent of severity. The toe dewclaw, white line, and hoof wall crack are lesions associated with horn properties and influenced after supplementing organic TM (Varagka et al., 2016). Those lesions were also grouped as hoof lesions, including all females that had at least one of the three lesions.

Statistical Analysis

The statistical procedures were performed using SAS v9.4 (SAS Inst. Inc.; Cary, NC). The experimental unit was the gilt for lameness, claw lesions, and number of piglets, whereas the pen (10 gilts)

was the unit for BW, BF, loin depth, ADG, ADFI, Lys intake per kg of BW gain, and G:F ratio. The different variables were evaluated for normality and homoscedasticity by using the Shapiro–Wilk and Levene's test and examining the normal plot (PROC UNIVARIATE). For all parametric analysis, the Tukey–Kramer adjustment was used to determine significant ($P < 0.05$) and marginal ($P < 0.10$) differences among dietary treatments.

The BW, loin depth, BF, and lameness occurrence over the experimental time were analyzed using repeated-measures models, including dietary treatment and time (days of experimental study) as fixed effects and block as a random effect using mixed-model methods (PROC GLIMMIX). Additionally, ADG was analyzed using repeated-measures with lameness and time as fixed effects and block and pen as the random effects. In this case, it was analyzed separately for the dietary supplements strategy and the limited Lys strategy due to the low incidence of lameness (PROC GLIMMIX). Furthermore, phase and overall ADG, ADFI, Lys intake per BW gain, and G:F were analyzed using the general linear model (PROC GLM).

Sow farm measurements of BW, BF, and sow productivity were analyzed using the general linear model (PROC GLM). Claw lesions, grouped hoof lesions, and lameness occurrence at different times were analyzed with a Binomial model (PROC GENMOD). Fisher's Exact test was used for testing removal reasons (PROC FREQ). The main fixed effects included dietary treatment and sow farm. Reproductive performances of females classified as lame (eventually displayed lameness) or not lame (never displayed lameness) on the sow farm were analyzed using a non-parametric Binomial model (PROC GENMOD).

RESULTS

Limiting dietary Lys reduced growth; however, neither such reduction, nor the dietary supplementation of TM and Met or the combination of the slower growth and dietary supplements were capable of reducing lameness prevalence compared with the control, although age and BW at lameness detection were higher on gilts supplemented with TM and Met than on the control gilts. Results are presented first for performance and lameness during rearing and second for the measurements in the sow farm.

Rearing

Overall performance showed no interactions between limited Lys and dietary supplements.

Feeding low Lys did not affect ADFI but rather reduced overall ADG ($P < 0.001$) by 6.35% and final BW ($P < 0.001$) by 3.80%, compared with standard Lys (Table 2). According to the different feed phases, gilts fed low Lys had a lowered ADG ($P < 0.001$) and G:F ratio ($P = 0.012$) between 119 and 163 d but not from 163 to 209 d. There was no evidence of main effects due to TM plus Met supplements on gilt ADG, ADFI, or G:F. Throughout the growing phase, gilts fed low Lys had a lower BW than with standard Lys ($P < 0.05$). However, an interaction at day 184 showed that gilts fed TM plus Met and low Lys were 2.8 kg heavier (SE = 1.796; $P = 0.034$) than were control gilts fed low Lys, not different than control gilts fed the standard Lys but lighter ($P = 0.034$) than gilts fed TM plus Met and standard Lys.

The BF was not different amongst dietary treatments until the end of rearing. Then, an interaction occurred where gilts fed control and low Lys had reduced ($P = 0.049$) BF as compared with the gilts fed TM plus Met with low Lys and the control with standard Lys, with TM plus Met with standard Lys being intermediate. Interactions for loin depth were observed with some inconsistency but seemed to align with those of BW at 184 d. Gilts fed low Lys without supplements (control) had lower loin depth than with standard Lys ($P < 0.02$), whilst low Lys supplemented was intermediate at 163 and 184 d of age (Table 2). Differently, at the end of rearing (day 209 of age), gilts fed TM plus Met with low Lys had a lower loin depth than with standard Lys in control, with others being not different. More clearly, at 163 and 184 d of age, gilts fed low Lys diets had a reduced loin depth when compared with gilts fed standard Lys ($P = 0.05$).

Results from the lameness assessments are presented in Table 3. During the rearing period, lameness prevalence was 7.92% and appeared in a CI between 119.6 and 139.5 kg of BW. The proportion of lameness at a given moment increased with time and BW ($P < 0.001$). The final BW and overall ADG were not different ($P > 0.05$) when comparing gilts that eventually undergo lameness (988 g and 150.1 kg, respectively) and those that never had signs of lameness (990 g and 151.2 kg, respectively).

Lameness prevalence was not different among dietary treatments. However, lame gilts fed the TM plus Met diets were 19.2 kg heavier (SE = 5.04, $P = 0.016$) and 17.9 d older (SE = 5.17, $P = 0.034$) at lameness detection when compared with control-fed lame gilts. Figure 1 presents ADG by phases and for gilts fed TM plus Met that were classified as lame (a gilt that eventually undergoes lameness) or

Table 2. Effects of organic TM plus Met and lowering Lys dietary treatments provided (90 d) to rearing gilts on performance and body composition

	Control ¹		TM plus Met ¹		SEM	P-value		
	Standard Lys ²	Low Lys ²	Standard Lys ²	Low Lys ²		Min Met	Lys	MinMet × Lys
Body weight, kg								
119 d	58.3	58.1	58.2	57.8	1.796	0.813	0.889	0.940
141 d	87.2 ^x	84.1 ^y	87.2 ^x	85.2 ^{xy}		0.685	0.047	0.091
163 d	112.1	106.2	112.5	108.0		0.543	<0.001	0.491
184 d	132.7 ^{ab}	126.1 ^c	134.5 ^a	129.9 ^b		0.030	<0.001	0.034
209 d	153.9	146.9	154.3	149.6		0.189	<0.001	0.141
Backfat depth, mm								
119 d	7.19	7.33	7.04	7.36	0.353	0.805	0.352	0.695
141 d	9.04	9.32	9.04	9.58		0.640	0.145	0.173
163 d	11.2	10.8	11.1	11.5		0.429	0.953	0.348
184 d	12.9	12.7	13.0	13.3		0.270	0.945	0.407
209 d	15.0 ^a	14.2 ^b	14.3 ^{ab}	14.9 ^a		0.998	0.603	0.049
Loin depth, mm								
119 d	30.1	30.4	29.4	29.9	0.742	0.605	0.628	0.242
141 d	39.9	39.5	39.9	39.6		0.452	0.271	0.729
163 d	49.5 ^a	48.9 ^b	50.2 ^{ab}	49.2 ^{ab}		0.939	0.036	0.019
184 d	62.8 ^a	59.7 ^b	64.1 ^a	61.1 ^{ab}		0.074	<0.001	<0.01
209 d	67.7 ^a	66.3 ^{ab}	66.4 ^{ab}	65.3 ^b		0.129	0.106	0.030
Average daily gain, g								
119–163 d	1,220	1,090	1,225	1,133	17.5	0.172	<0.001	0.275
163–209 d	907	875	908	900	41.5	0.751	0.635	0.781
Overall	1,018	944	1,028	972	23.4	0.439	0.011	0.705
Average daily feed intake, g								
119–163 d	3,055	2,953	3,040	3,001	51.2	0.761	0.183	0.554
163–209 d	3,403	3,263	3,393	3,245	103	0.892	0.179	0.968
Overall	3,238	3,204	3,215	3,220	98.8	0.887	0.971	0.885
Gain:feed ratio								
119–163 d	0.40	0.37	0.40	0.38	0.010	0.609	0.012	0.807
163–209 d	0.27	0.27	0.27	0.28	0.011	0.834	0.727	0.727
Overall	0.32	0.30	0.32	0.30	0.013	0.173	0.905	0.999

¹Control, basal diet and TM plus Met (MinMet), with additional 10, 20, and 50 mg/kg of chelated copper, manganese, and zinc, respectively (0.1%, Aplomotec Plus, Tecnología & Vitaminas, S.L., Alforja, Spain), and increased Met:Lys ratio as 1.01.

²Standard Lys provided 165 crude protein (CP)/kg with 10.0 g Lys/kg between 119 and 163 d of age and 140 g CP/kg with 8.0 g Lys/kg between 164 to 209 d of age; or low Lys, set first to 155 g CP/kg with 8.1 g Lys/kg and second phase to 140 g CP/kg with 6.5 g Lys/kg.

^{ab}Values within a row with different superscripts differ significantly at $P < 0.05$.

^{xy}Values within a row with different superscripts trend at $P < 0.10$.

never lame (a gilt that never had signs of lameness). Between 163 and 184 d of age, ADG increased ($P < 0.01$) for a mineral-plus-Met strategy (lame or never lame) as compared with a control strategy of never-lame gilts, while control-lame gilts were intermediate. Late in growth (184 to 209 d of age), lame gilts had reduced ($P = 0.015$) ADG within the control fed gilts, but gilts from TM plus Met were intermediate for both lame and never-lame gilts.

The same assessment of ADG and lameness, but for a Lys dietary strategy, is presented in Figure 2. Between 113 and 141 d of age, lame gilts fed the low Lys and never-lame gilts fed standard Lys had greater ADG than never-lame females fed low Lys ($P = 0.001$). From 141 to 163 d of age, low Lys

lowered ($P < 0.001$) ADG as compared with standard Lys for never-lame gilts; however, lame gilts were intermediate for both standard and low Lys. Finishing the rearing phase (184 to 209 d of age), lame gilts fed standard Lys tended to have reduced ($P = 0.097$) ADG compared with never-lame gilts on both standard Lys and low Lys diets; nevertheless, lame gilts on low Lys were intermediate.

Sow Farm

Lameness on the sow farm increased to 17.4% at first service, then decreased to 11.3% at the end of gestation, increasing to 15.2% at weaning. There was no evidence of differences for lameness

Table 3. Effects of TM plus Met and lowering Lys dietary treatments provided (90 d) to rearing gilts (from 58.0 ± 11.1 kg to 151 ± 14.1 kg of BW) on lameness during rearing and sow productive phases

	Control ¹		TM plus Met ¹		SEM	P-value		
	Standard Lys ²	Low Lys ²	Standard Lys ²	Low Lys ²		Min Met	Lys	MinMet × Lys
Gilts, <i>n</i>	59	58	56	57				
BW at lameness, kg	120.2	119.8	134.2	144.4	7.75	0.016	0.515	0.456
Age at lameness, d	173.8	175.8	185.3	198.2	6.92	0.034	0.346	0.462
Lameness, %								
During rearing ³	6.67	8.33	10.0	6.67	19.530	0.775	0.834	0.456
Early gestation ⁴	15.5	22.6	14.1	17.6	14.98	0.587	0.309	0.746
Late gestation ⁵	15.6	12.1	7.10	10.6	17.76	0.246	0.859	0.421
Lactation ⁶	17.1	19.1	8.88	15.8	16.04	0.211	0.304	0.503
Overall	20.5	32.8	19.6	24.0	13.49	0.447	0.532	0.625
Gait score, 0–2								
During rearing	0.15	0.13	0.15	0.15	0.052	0.854	0.789	0.152
Early gestation	0.22	0.29	0.19	0.18	0.124	0.272	0.763	0.486
Late gestation	0.19	0.17	0.09	0.12	0.057	0.140	0.742	0.587
Lactation	0.24	0.26	0.11	0.21	0.096	0.108	0.222	0.344
Overall	0.19	0.23	0.14	0.15	0.046	0.284	0.681	0.871
Severity, %								
During rearing	–	–	–	–	–	–	–	–
Early gestation	39.1	31.2	21.5	7.76	16.40	0.185	0.439	0.792
Late gestation	24.4	54.3	25.0	16.7	21.26	0.364	0.956	0.630
Lactation	47.0	67.7	42.9	32.8	22.90	0.410	0.607	0.793
Overall	66.7	53.8	46.1	38.8	17.01	0.360	0.716	0.534
All severe cases, %	13.7	17.3	8.88	10.6	21.80	0.726	0.449	0.449

¹Control, basal diet and TM plus Met (MinMet), with additional 10, 20, and 50 mg/kg of chelated cooper, manganese, and zinc, respectively (0.1%; Aplomotec Plus, Tecnología & Vitaminas, S.L., Alforja, Spain) and increased Met:Lys ratio as 1.01.

²Standard Lys provided 165 CP/kg with 10.0 g Lys/kg between 119 and 163 d of age and 140 g CP/kg with 8.0 g Lys/kg between 164 to 209 d of age; or low Lys, set first to 155 g CP/kg with 8.1 g Lys/kg and second phase to 140 g CP/kg with 6.5 g Lys/kg.

³During the rearing phase, lameness was evaluated for 60 gilts per dietary treatment; for other measurements, it is indicated in the table.

⁴Evaluated at service and at 28 d of gestation.

⁵Evaluated at 109 d of gestation.

⁶Evaluated at weaning.

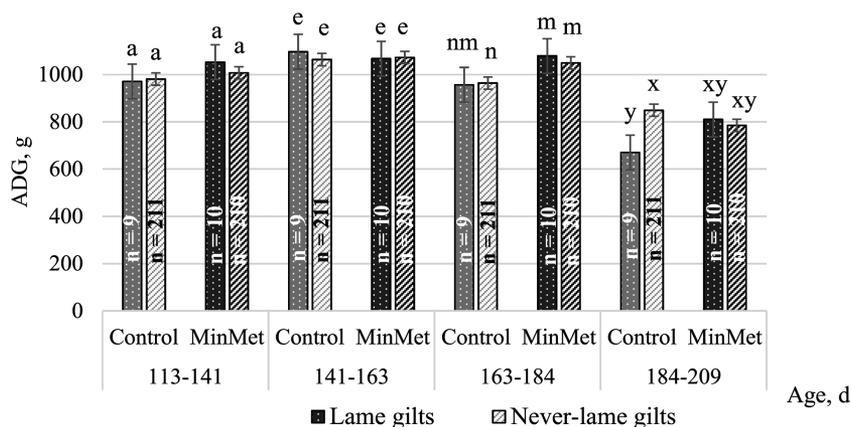


Figure 1. ADG according to different age phases for control (gray) or dietary supplemented¹ (black) gilts, comparing between females that never showed lameness (*n* = 230) and gilts that eventually became lame (*n* = 19) during rearing. ¹Control, basal diet and TM plus Met (MinMet), with additional 10, 20, and 50 mg/kg of chelated cooper, manganese, and zinc, respectively (0.1%, Aplomotec Plus, Tecnología & Vitaminas, S.L., Alforja, Spain), and increased Met:Lys ratio as 1.01. ^{a,b}Different superscripts differ significantly at *P* < 0.05 within 119 and 141 d ADG. ^{c,d}Different superscripts differ significantly at *P* < 0.05 within 141 and 163 d ADG. ^{m,n}Different superscripts differ significantly at *P* < 0.05 within 163 and 184 d ADG. ^{x,y}Different superscripts differ significantly at *P* < 0.05 within 184 and 209 d ADG.

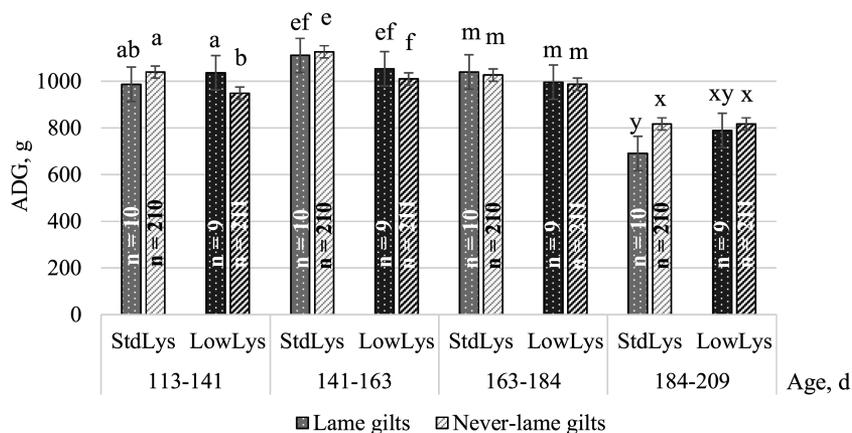


Figure 2. ADG according to the different age phases for gilts fed standard (gray) or low Lys¹ (black), comparing between females that never showed lameness ($n = 230$) and gilts that eventually became lame ($n = 19$) during rearing. ¹Standard (Std) Lys provided 165 CP/kg with 10.0 g Lys/kg between 119 and 163 d of age and 140 g CP/kg with 8.0 g Lys/kg between 164 and 209 d of age; or low Lys, set first to 155 g CP/kg with 8.1 g Lys/kg and second phase to 140 g CP/kg with 6.5 g Lys/kg. ^{ab}Different superscripts differ significantly at $P < 0.05$ within 119 and 141 d ADG. ^{ef}Different superscripts differ significantly at $P < 0.05$ within 141 and 163 d ADG. ^{mm}Different superscripts differ significantly at $P < 0.05$ within 163 and 184 d ADG. ^{xy}Different superscripts differ marginally at $P < 0.10$ within 184 and 209 d ADG.

Table 4. Effects of organic TM plus Met and lowering Lys dietary treatments provided (90 d) to rearing gilts (from 58.0 ± 11.1 kg to 151 ± 14.1 kg of BW) on first parity performance

	Control ¹		TM plus Met ¹		SEM	P-value		
	Standard Lys ²	Low Lys ²	Standard Lys ²	Low Lys ²		MinMet	Lys	MinMet × Lys
Gilts, n	59	58	56	57				
Service								
BW, kg	181.3	180.3	184.4	179.1	2.66	0.721	0.228	0.430
BF, mm	15.6	15.8	15.9	15.0	0.47	0.700	0.537	0.188
Farrowing								
BF, mm	14.4	13.8	14.4	14.9	0.46	0.206	0.936	0.195
Rate, %	91.4	82.6	87.8	89.5	4.39	0.373	0.669	0.212
Total born, n	17.3	17.4	17.4	16.4	0.59	0.403	0.351	0.332
Liveborn, n	15.6 ^{xy}	16.3 [*]	16.2 [*]	15.2 ^y	0.58	0.627	0.740	0.091
Stillborn, %	9.3	7.03	5.18	7.68	1.62	0.264	0.933	0.125
Weaning								
BF, mm	12.5	11.7	12.1	12.8	0.42	0.370	0.984	0.091
Weaned, n	14.2	14.4	13.3	14.0	0.55	0.208	0.371	0.672
Days to estrus	6.60	5.92	6.84	8.75	1.54	0.660	0.294	0.401

^{xy}Values within a row with different superscripts trend at $P < 0.10$.

¹Control, basal diet and TM plus Met (MinMet), with additional 10, 20, and 50 mg/kg of chelated cooper, manganese, and zinc, respectively (0.1%, Aplomotec Plus, Tecnologia & Vitaminas, S.L., Alforja, Spain) and increased Met:Lys ratio as 1.01.

²Standard Lys provided 165 CP/kg with 10.0 g Lys/kg between 119 and 163 d of age and 140 g CP/kg with 8.0 g Lys/kg between 164 and 209 d of age; or low Lys, set first to 155 g CP/kg with 8.1 g Lys/kg and second phase to 140 g CP/kg with 6.5 g Lys/kg.

prevalence and severity across dietary treatments, productive phases, or overall (Table 3).

The results from first parity performance are presented in Table 4 and show no major carry-over effects from dietary treatments provided during growth. Still, gilts fed TM plus Met with low Lys tended to be 16 d younger (SE = 3.63; $P = 0.009$) at first service than those fed TM plus Met with standard Lys. Additionally, the number of liveborn piglets tended to show the same interaction with gilts fed TM plus Met, with low Lys

sows having a lower number of liveborn piglets ($P = 0.091$) than did control with low Lys and mineral plus Met with standard Lys. A 17.5% lameness rate at early gestation tended to increase the odds (1.12; CI = 1.251, 1.116; $P = 0.060$) for reduced, total-born piglets (16.5 vs. 17.7) and increased the odds (1.11; CI = 1.074, 1.00; $P = 0.044$) for reduced piglets born alive (14.8 vs. 16.0). Furthermore, an 11.4% lameness rate from late gestation and 15.3% at weaning weakly increased the odds (1.04; CI = 1.008, 1.077;

$P = 0.015$; and 1.03 ; $CI = 1.001, 1.067$; $P = 0.040$, respectively) for greater weaning-to-estrus interval days than nonlame females (13.1 vs. 6.17 and 11.1 vs. 6.21, respectively).

The percentage of removals and removal reasons during first parity are presented in Table 5. Thirty-one out of 230 females (13.5%) did not reach a second gestation. Of these, 68.1% were sudden deaths, whereas 19.4% were removed due to reproductive problems, 6.9% for lameness, and 5.6% for other reasons. Over the productive phases, 16.7% of those were in gestation, 59.7% during lactation (all sudden deaths), and 23.6% at weaning. Culling and mortality showed no evidence for differences across dietary treatments. However, removals due to reproductive reasons tended to be less frequent amongst gilts fed with a low Lys strategy (6.25%) than control Lys (33.3%; $P = 0.083$). Additionally, an interaction occurred with gilts fed TM plus Met and low Lys, tending ($P < 0.10$) to have the highest incidence of sudden-death removals and total removals in lactation.

Claw Health

Incidence of claw lesions among dietary treatments was evaluated for 87.8% of the females, and results are presented in Table 6. Of all sows evaluated, 52.4% had at least one claw lesion, whereas 24.8% of them had two lesions or more. Heel lesions were the most common claw lesions (27.0%, both overgrowth and heel cracks). This was followed by lesions of asymmetry (16.1%), hoof wall cracks (10.9%), white line (7.83%), broken or missing dewclaws (4.78%), and long hoofs (3.49%). The grouped hoof lesions (wall cracks, white line,

and dewclaw) had a 23.3% prevalence. When compared across dietary treatments, no differences were observed for any type of lesion.

DISCUSSION

In agreement with our expectations, the diets with lower Lys reduced final BW (3.8%), but to a lesser extent than that expected (10%). Conversely, neither this nor the supplement of TM plus Met or both strategies together affected lameness prevalence. The only effect aligned with our hypothesis might be a delay in lameness occurrence for the gilts supplemented with TM plus Met.

The lack of a clear ADG effect on lameness was, to some extent, not surprising since a positive relationship between ADG and lameness was not detected in our previous study (Fabà et al., 2018) and because the relationship between ADG and OC is somewhat inconsistent (Orth et al., 1999; Ytrehus et al., 2007; Olstad et al., 2015). Nevertheless, young females (113 to 141 d of age) that eventually became lame had greater ADG than never-lame gilts under low Lys (Figure 2). This may suggest an initial greater potential for growth associated with lameness, although caution must be taken due to the low number of observations. Some research supports restrictive growth via dietary strategies (i.e., low energy, low Lys, and/or feed restriction) with a positive effect on reducing OC (Reiland, 1975; Goedegebuure et al., 1980; Koning et al., 2014b) and lameness (Quinn et al., 2015), whereas other studies do not support such effects (Grøndalen, 1974; Nakano et al., 1984; Woodard et al., 1987; Carlson et al., 1988). Busch and Wachmann (2011)

Table 5. Effects of organic TM plus Met lowering Lys dietary treatments provided (90 d) to rearing gilts (from 58.0 ± 11.1 kg to 151 kg ± 14.1 kg of body weight) on removal reasons during the first parity

	Control ¹		TM plus Met ¹		SEM	P-value		
	Standard Lys ²	Low Lys ²	Standard Lys ²	Low Lys ²		MinMet	Lys	MinMet \times Lys
Gilts culled, <i>n</i>	6	9	9	7				
Removals, %	10.2	15.5	16.1	12.3	4.59	0.847	0.999	0.758
Sudden death, %	50.0	77.8	44.4	100	18.60	0.999	0.736	0.069
Reproductive, %	33.3	11.1	33.3	0.0	10.60	0.685	0.083	0.365
Lameness, %	16.7	11.1	0.0	0.0	10.62	0.226	0.990	0.554
Others, %	0.0	22.2	22.2	0.0	9.90	0.484	0.226	0.232
Phases								
Gestation, %	33.3	11.1	22.2	0.0	15.73	0.654	0.172	0.488
Lactation, %	50.0	44.4	44.4	100	19.26	0.285	0.285	0.074
Postweaning, %	16.7	44.4	33.3	0.0	17.60	0.433	0.999	0.219

¹Control, basal diet and TM plus Met (MinMet), with additional 10, 20, and 50 mg/kg of chelated copper, manganese, and zinc, respectively (0.1%, Aplomotec Plus, Tecnologia & Vitaminas, S.L., Alforja, Spain), and increased Met:Lys ratio as 1.01.

²Standard Lys provided 165 CP/kg with 10.0 g Lys/kg between 119 and 163 d of age and 140 g CP/kg with 8.0 g Lys/kg between 164 and 209 d of age; or low Lys, set first to 155 g CP/kg with 8.1 g Lys/kg and second phase to 140 g CP/kg with 6.5 g Lys/kg.

associated fast ADG with an increasing risk of OC, whilst others did not (Ytrehus et al., 2004a, 2004b; Tóth et al., 2016), suggesting that other factors (i.e., genetics, age, or even location) are likely more important. For example, van Grevenhof et al. (2012) observed an inconsistent association between ADG and OC amongst joints. In their study, heavier gilts had more lesions in the elbow, but lighter females had more in femoropatellar joints.

Inherited traits associate OC with fast growth (Grøndalen and Vangen, 1974; Jørgensen and Andersen, 2000; Kadarmideen et al., 2004; Aasmundstad et al., 2013), however, if the association between fast growth with OC is related to joint overload or simply a genetic correlation for the pathology is less clear. Indeed, it was earlier noted that physiological biomechanical forces would be sufficient to affect the OC outcome (Ytrehus et al., 2007; Etterlin et al., 2014; Olstad et al., 2015); meanwhile, the initial vascular disruption cause of OC still needs to be elucidated (Olstad et al., 2015). A great degree of reduction may be required to lessen OC via weight load reduction, and, perhaps, the present lowering was insufficient as low Lys only reduced final BW by 3.8%. Quinn et al. (2015), via feed restriction, found that reducing final BW by 7.30% was associated with less lameness and OC. de Koning et al. (2014b) found that gilts fed ad libitum between 4 and 26 wk of age (final BW 132 kg) had higher odds of OC than did those fed ad libitum from 4 to 10 wk and then restricted from 10 to 26 wk (reduced 16% BW). These authors also reported that initially restricting (4–10 wk) followed by ad libitum feeding (10–26 wk; reduced 3.0% BW) increased odds of OC when compared with initially feeding ad libitum (4–10 wk) followed by restriction (10–26 wk; reduced 16% BW) or restricted throughout (reduced 19% BW). Assuming that slower ADG can minimize lameness, the ADG reduction in the present study is notable less than that in the previous studies where benefits were reported and may be insufficient.

In agreement with previous works, lowered Lys (Cia et al., 1998; Díaz et al., 2017) or Lys and CP (Li et al., 2018) in the diet reduced growth and loin area and (or) depth. Nonetheless, these authors reported greater BW reductions (7% to 12%) than that herein (3.80%). Indeed, the target was to reduce final BW by nearly 10%, which according to the above-mentioned literature is that reported with the potential to reduce lameness and lameness risk factors (OC). In a similar study, although focused on puberty, Díaz et al. (2017) used differing total Lys concentrations (low, medium, or high) of 0.81%, 0.90%, and 0.99%, respectively, from 100

to 142 d of age and of 0.65%, 0.70%, and 0.76%, respectively, from 143 to 212 d of age. Hence, our treatments provided similar Lys to their low Lys and high Lys levels (Table 1). However, present diets (with wheat, barley, corn, soybean meal, sunflower meal, and wheat byproducts) contained 2.3–2.4 Mcal/kg NE and were lower than the 2.5–2.9 Mcal/kg NE in high Lys diets (corn-soybean based) of Díaz et al. (2017). Higher Lys:energy ratio herein might result in a relatively high feed and Lys intake than the previous study because a lower energy density will increase feed intake (NRC, 2012; Li and Patience, 2017). As calculated, gilts in standard Lys consumed 25 g SID Lys/kg gain whereas low Lys consumed 19 g SID Lys/kg gain up to 163 d of age. According to Shelton et al. (2011), this would be near or slightly below sufficiency and might have been insufficient. This is especially noted as a lack of Lys deficiency, and hence, lack of ADG reduction between 163 and 209 d of age for the low Lys treatment. This indicates that present genetics is not limited with 21 g Lys daily per kg of gain between 163 and 209 d of age but slightly limited with 19 g Lys daily intake per kg of gain between 119 and 163 d. Altogether, high intake even with low Lys density in the diet, could explain the moderate but not severe reduction of BW and the lack of ADG differences in the second feed phase. A greater degree of limiting dietary Lys or ratio to energy would be needed to properly test the hypothesis herein.

Interactions for BW at day 184 and loin depth at days 163 and 184 occurred, as values were greater for gilts fed control Lys and TM plus Met than for gilts fed control and low Lys. The importance of such interactions is minor since differences were small. The reason behind the interactions is unknown. High dietary Met could support catabolism for some amino acids if control with low Lys was deficient, but other deficiencies are not expected with the present diets. Otherwise, supportive properties to the immune system attributed to Met, or an antioxidant effect, may have enhanced gilt growth under Lys deficiency (Brosnan et al., 2006; Li et al., 2007). In fact, despite extensive research, the impacts of additives and environmental conditions on amino acid requirements remain poorly defined in practical diets (NRC, 2012).

Providing extra TM plus Met did not reduce lameness prevalence though its occurrence seemed to be delayed. Furthermore, lame gilts in control diets tended to have lower ADG than never-lame gilts by the end of rearing, whereas lame gilts fed TM plus Met were intermediate, suggesting little or null intervention. These results contrast with

our previous study where identical supplementation of TM and Met, in combination or separately, reduced lameness. Likewise, in the subsample study, TM alone enhanced bone strength and density and, combined with Met, improved the OC lesions score (Fabà et al., 2019).

Over the years, bone and joint development were studied to enhance bone health and reduce lameness; however, controversy is commonly reported among intervening factors. Deficiencies in TM such as Zn, Cu, and Mn (Ott and Asquith, 1995; Shaw et al., 2006; Veum et al., 2009) and amino acid Met (Huang et al., 2014), used herein, would negatively influence bone development. Conversely, the use of TM above requirements is noted as noneffective (Orth, 1999; Creech et al., 2004; Gowanlock et al., 2013; Tóth et al., 2016) or with limited benefits on bone metabolism (Liu et al., 2016) and bones and joint lesions (Frantz et al., 2008; Quinn et al., 2015; Fabà et al., 2019). Similarly, benefits have been documented with Met (Frantz et al., 2008; Ouattara et al., 2016). The present results and published data maintain controversy and do not support a clear benefit from TM supplement on growing pigs' lameness. Using similar TM and levels to this study (all above NRC, 2012), Tóth et al. (2016) evaluated inorganic sources with additional organic ones (150 + 50 g/kg Zn, 50 + 20 mg/kg Cu, and 16.5 + 10 mg/kg Mn). Results showed no improvements in OC either early (12 wk) or later (24 wk) in the pigs' life. Further, only few severe cases of OC (3 out of 200) were detected at 24 wk of age. To discuss such results and the difference between our present and former study, lameness severity may be key. In fact, whereas in the previous study the control gilts had a lameness prevalence of 14.8%, in the present, lameness prevalence in control gilts was 6.67%.

Lameness during the rearing period (7.92%) was similar to that in our previous study (7.75%; Fabà et al., 2018), although, herein it was not associated with performance loss. Lamé gilts in our previous study had reduced ADG (90 g) and final BW (7.0 kg) compared with never-lame gilts. Such effects on performance would indicate high severity (Weary et al., 2009; Munsterhjelm et al., 2015), while absence of performance differences between lame and never-lame gilts in the present study suggests low severity. These differences were unexpected because facilities, genetics, and the observer were the same as in our previous study. A different breeder multiplier may partly explain differences because OC is inheritable, and more concentrated within populations (Aasmundstad et al., 2013; Le et al., 2016). Although it cannot

be confirmed, a lower severity of lameness in the present study may explain the lack of more consistent effects.

When entering into the sow herd, lameness prevalence doubled in our studies. This highlights the high susceptibility of young females (Pluym et al., 2013). Lameness is important and was associated with lowered prolificacy and an increased weaning-to-estrus interval. It could be that the increased stress-under-lameness condition in early gestation impairs reproductive function. Similarly, Anil et al. (2009) reported reduced liveborn piglets for lame sows. Furthermore, lameness may affect sow mobility in lactation (Anil et al., 2009; Pluym et al., 2013) and reduce feed intake, which would compromise the state of reserves at weaning and the interval to estrus. Still, this cannot be confirmed from the present data. Entering into the sow farm was a challenge associated to an increased lameness similarly for all dietary treatments. Therefore, our previous (Fabà et al., 2018) and present data suggest that dietary supplements do not act as a carryover measure to reduce lameness and claw problems. Different facilities, wider slat void, floor type, social conflicts from management (i.e., movements, animal flow, density, remixing), or training for the electronic feeding system were all new factors also known to increase lameness and claw lesions (Pluym et al., 2011; Li and Gonyou, 2013; Olsson et al., 2016). Still, if lameness prevalence were to be reduced through TM during rearing, this would enhance latter productivity (Fabà et al., 2018). Data do not negate that TM supplements in sows may reduce lameness via improving claw horn properties as previously reported (Varagka et al., 2016); probably, a continued supplement may provide a better approach. Otherwise, the change may be too severe a challenge to find a response. These data indicate that lameness should be first addressed through management and environment, especially for young sows.

In conclusion, during the rearing phase and under the present conditions, the organic TM (Cu, Mn, and Zn) and Met supplements, or the standard and low Lys, or combined treatments did not affect prevalence of lameness. The absence of performance loss in lame gilts and low gait scores suggests low lameness severity during rearing, which may have limited dietary treatment potential. Nonetheless, sow herd lameness was associated with increased odds for reduced liveborn piglets and an increased weaning-to-estrus interval. The dietary treatments during rearing did not improve sow lameness and claw health at first parity but prevalence increased

dramatically, likely due to management and environmental factors. The present low Lys reduced growth to a lesser extent as compared with previous studies that documented reduced OC and lameness via feed restriction. A greater deficiency of Lys than that herein would be needed to further test the potential of the present hypothesis.

LITERATURE CITED

- Aasmundstad, T., J. Kongsro, M. Wetten, N. I. Dolvik, and O. Vangen. 2013. Osteochondrosis in pigs diagnosed with computed tomography: heritabilities and genetic correlations to weight gain in specific age intervals. *Animal* 7:1576–1582. doi:10.1017/S1751731113001158
- Anil, S. S., L. Anil, and J. Deen. 2009. Effect of lameness on sow longevity. *J. Am. Vet. Med. Assoc.* 235:734–738. doi:10.2460/javma.235.6.734
- Brosnan, J. T., and M. E. Brosnan. 2006. The sulfur-containing amino acids: an overview. *J. Nutr.* 136(6 Suppl):1636S–1640S. doi:10.1093/jn/136.6.1636S
- Busch, M. E., and H. Wachmann. 2011. Osteochondrosis of the elbow joint in finishing pigs from three herds: associations among different types of joint changes and between osteochondrosis and growth rate. *Vet. J.* 188:197–203. doi:10.1016/j.tvjl.2010.03.021
- Carlson, C. S., H. D. Hilley, D. J. Meuten, J. M. Hagan, and R. L. Moser. 1988. Effect of reduced growth rate on the prevalence and severity of osteochondrosis in gilts. *Am. J. Vet. Res.* 49:396–402.
- Celi, P., A. J. Cowieson, F. Fru-Nji, R. E. Steinert, A. M. Klünter, and V. Verlhac. 2017. Gastrointestinal functionality in animal nutrition and health: new opportunities for sustainable animal production. *Anim. Feed Sci. Technol.* 234:88–100. doi:10.1016/j.anifeedsci.2017.09.012
- Cia, M. C., S. A. Edwards, V. L. Glasgow, M. Shanks, and H. Fraser. 1998. Modification of body composition by altering the dietary lysine to energy ratio during rearing and the effect on reproductive performance of gilts. *Anim. Sci.* 66:457–463.
- Creech, B. L., J. W. Spears, W. L. Flowers, G. M. Hill, K. E. Lloyd, T. A. Armstrong, and T. E. Engle. 2004. Effect of dietary trace mineral concentration and source (inorganic vs. Chelated) on performance, mineral status, and fecal mineral excretion in pigs from weaning through finishing. *J. Anim. Sci.* 82:2140–2147. doi:10.2527/2004.8272140x
- Deen, J., S. S. Anil, L. Anil, and S. Baidoo. 2008. Lameness overview and awareness: implications for welfare, housing, performance and economics. *Asten/Sterksel (The Netherlands): FeetFirst™ Symp. Sow Lameness.*
- Díaz, J. A. C., J. L. Vallet, R. D. Boyd, C. A. Lents, T. J. Prince, A. E. DeDecker, C. E. Phillips, G. Foxcroft, and K. J. Stalder. 2017. Effect of feeding three lysine to energy diets on growth, body composition and age at puberty in replacement gilts. *Anim. Reprod. Sci.* 184:1–10. doi:10.1016/j.anireprosci.2017.06.007.
- Engblom, L., L. Eliasson-Selling, N. Lundeheim, K. Belák, K. Andersson, and A. M. Dalin. 2008. Post mortem findings in sows and gilts euthanised or found dead in a large Swedish herd. *Acta Vet. Scand.* 50:25. doi:10.1186/1751-0147-50-25
- Etterlin, P. E., B. Ytrehus, N. Lundeheim, E. Heldmer, J. Österberg, and S. Ekman. 2014. Effects of free-range and confined housing on joint health in a herd of fattening pigs. *BMC Vet. Res.* 10:208. doi:10.1186/s12917-014-0208-5
- Ewers, B. J., D. Dvoracek-Driksna, M. W. Orth, and R. C. Haut. 2001. The extent of matrix damage and chondrocyte death in mechanically traumatized articular cartilage explants depends on rate of loading. *J. Orthop. Res.* 19:779–784. doi:10.1016/S0736-0266(01)00006-7
- Fabà, L., J. Gasa, M. D. Tokach, E. Varella, and D. Solà-Oriol. 2018. Effects of supplementing organic microminerals and methionine during the rearing phase of replacement gilts on lameness, growth, and body composition. *J. Anim. Sci.* 96:3274–3287. doi:10.1093/jas/sky195
- Fabà, L., J. Gasa, M. D. Tokach, M. Font-i-Furnols, E. Vilarrasa, and D. Solà-Oriol. 2019. Effects of additional organic microminerals and methionine on carcass composition, gait score, bone characteristics, and osteochondrosis in replacement gilts of different growth rate. *Anim. Feed Sci. Technol.* (in press).
- FEDNA. 2013. *Necesidades nutricionales para Ganado porcino.* 2th ed. Madrid (Spain): Fundación Española para el Desarrollo de la Nutrición Animal.
- Frantz, N. Z., G. A. Andrews, M. D. Tokach, J. L. Nelssen, R. D. Goodband, J. M. Derouchey, and S. S. Dritz. 2008. Effect of dietary nutrients on osteochondrosis lesions and cartilage properties in pigs. *Am. J. Vet. Res.* 69:617–624. doi:10.2460/ajvr.69.5.617
- Goedegebuure, S. A., H. J. Häni, P. C. van der Valk, and P. G. van der Wal. 1980. Osteochondrosis in six breeds of slaughter pigs. *Vet. Q.* 2:28–41. doi:10.1080/01652176.1980.9693755
- van Grevenhof, E. M., H. C. M. Heuven, P. R. van Weeren, and P. Bijma. 2012. The relationship between growth and osteochondrosis in specific joints in pigs. *Livest. Sci.* 143(1):85–90. doi:10.1016/j.livsci.2011.09.002
- Grøndalen, T. 1974. Leg weakness in pigs. I. Incidence and relationship to skeletal lesions, feed level, protein and mineral supply, exercise and exterior conformation. *Acta Vet Scand.* 15:555–73.
- Grøndalen, T., and O. Vangen 1974. Osteochondrosis and arthrosis in pigs. V. A comparison of the incidence in three different lines of the Norwegian Landrace breed. *Acta Vet Scand.* 15:61–79.
- Gowanlock, D. W., D. C. Mahan, J. S. Jolliff, S. J. Moeller, and G. M. Hill. 2013. Evaluating the NRC levels of Cu, Fe, Mn, and Zn using organic and inorganic mineral sources for grower-finisher swine. *J. Anim. Sci.* 91:5680–5686. doi:10.2527/jas2013-6608
- Heinonen, M., O. Peltoniemi, and A. Valros. 2013. Impact of lameness and claw lesions in cows on health and production. *Livest. Sci.* 156:64–70. doi:10.1016/j.livsci.2013.06.012
- Huang, T. H., J. L. Lewis, H. S. Lin, L. T. Kuo, S. W. Mao, Y. S. Tai, M. S. Chang, G. P. Ables, C. E. Perrone, and R. S. Yang. 2014. A methionine-restricted diet and endurance exercise decrease bone mass and extrinsic strength but increase intrinsic strength in growing male rats. *J. Nutr.* 144:621–630. doi:10.3945/jn.113.187922
- Jensen, T. B., M. K. Bonde, A. G. Kongsted, N. Toft, and J. T. Sørensen. 2010. The interrelationships between clinical signs and their effect on involuntary culling among pregnant sows in group-housing systems. *Animal* 4:1922–1928. doi:10.1017/S1751731110001102
- Jørgensen, B., and S. Andersen. 2000. Genetic parameters for osteochondrosis in Danish Landrace and Yorkshire boars

- and correlations with leg weakness and production traits. *Anim. Sci.* 71:427–434. doi:10.1017/S1357729800055442
- Kadarmideen, H. N., D. Schwörer, H. Ilahi, M. Malek, and A. Hofer. 2004. Genetics of osteochondral disease and its relationship with meat quality and quantity, growth, and feed conversion traits in pigs. *J. Anim. Sci.* 82:3118–3127. doi:10.2527/2004.82113118x
- KilBride, A. L., C. E. Gillman, and L. E. Green. 2009. A cross sectional study of the prevalence, risk factors and population attributable fractions for limb and body lesions in lactating sows on commercial farms in England. *BMC Vet. Res.* 5:30. doi:10.1186/1746-6148-5-30
- de Koning, D. B., E. M. van Grevenhof, B. F. Laurensen, P. R. van Weeren, W. Hazeleger, and B. Kemp. 2014a. The influence of floor type before and after 10 weeks of age on osteochondrosis in growing gilts. *J. Anim. Sci.* 92:3338–3347. doi:10.2527/jas.2014-7902
- de Koning, D. B., E. M. van Grevenhof, B. F. Laurensen, P. R. van Weeren, W. Hazeleger, and B. Kemp. 2014b. The influence of dietary restriction before and after 10 weeks of age on osteochondrosis in growing gilts. *J. Anim. Sci.* 91:5167–5176. doi:10.2527/jas.2013-6591
- de Koning, D. B., E. M. van Grevenhof, B. F. Laurensen, W. Hazeleger, and B. Kemp. 2015. Associations of conformation and locomotive characteristics in growing gilts with osteochondrosis at slaughter. *J. Anim. Sci.* 93:93–106. doi:10.2527/jas.2014-8366
- Le, T. H., P. Madsen, N. Lundeheim, K. Nilsson, and E. Norberg. 2016. Genetic association between leg conformation in young pigs and sow longevity. *J. Anim. Breed. Genet.* 133:283–290. doi:10.1111/jbg.12193
- Li, Y. Z., and H. W. Gonyou. 2013. Comparison of management options for sows kept in pens with electronic feeding stations. *Can. J. Anim. Sci.* 93:445–452. doi:10.4141/cjas2013-044
- Li, Y. H., F. N. Li, Y. H. Duan, Q. P. Guo, C. Y. Wen, W. L. Wang, X. G. Huang, Y. L. Yin. 2018. Low-protein diet improves meat quality of growing and finishing pigs through changing lipid metabolism, fiber characteristics, and free amino acid profile of the muscle. *J. Anim. Sci.* 8:3221–3232. doi:10.1093/jas/sky116
- Li, Q., and J. F. Patience. 2017. Factors involved in the regulation of feed and energy intake of pigs. *Anim. Feed Sci. Technol.* 233:22–33. doi:10.1016/j.anifeeds.2016.01.001
- Li, P., Y. L. Yin, D. Li, S. W. Kim, and G. Wu. 2007. Amino acids and immune function. *Br. J. Nutr.* 98:237–252. doi:10.1017/S000711450769936X
- Liu, B., P. Xiong, N. Chen, J. He, G. Lin, Y. Xue, W. Li, and D. Yu. 2016. Effects of replacing of inorganic trace minerals by organically bound trace minerals on growth performance, tissue mineral status, and fecal mineral excretion in commercial grower-finisher pigs. *Biol. Trace Elem. Res.* 173:316–324. doi:10.1007/s12011-016-0658-7
- Munsterhjelm, C., M. Heinonen, and A. Valros. 2015. Effects of clinical lameness and tail biting lesions on voluntary feed intake in growing pigs. *Livest. Sci.* 181:210–219. doi:10.1016/j.livsci.2015.09.003
- Mustonen, K., E. Ala-Kurikka, T. Orro, O. Peltoniemi, M. Raekallio, O. Vainio, and M. Heinonen. 2011. Oral ketoprofen is effective in the treatment of non-infectious lameness in sows. *Vet. J.* 190:55–59. doi:10.1016/j.tvjl.2010.09.017
- Nakano, T., and F. X. Aherne. 1988. Involvement of trauma in the pathogenesis of osteochondritis dissecans in swine. *Can. J. Vet. Res.* 52:154–155.
- Nakano T., F. X. Aherne, J. J. Brennan, and J. R. Thompson. 1984. Effect of growth rate on the incidence of osteochondrosis in growing swine. *Can. J. Anim. Sci.* 64:139–146. doi:10.4141/cjas84-017
- Niemi, J. K., P. Bergman, S. Ovaska, M. -L. Sevón-Aimonen, and M. Heinonen. 2017. Modeling the costs of postpartum dysgalactia syndrome and locomotory disorders on sow productivity and replacement. *Front. Vet. Sci.* 4:1–12. doi:10.3389/fvets.2017.00181.
- NRC. 2012. Nutrient requirements of swine. 11th rev. ed. Washington (DC): National Academies of Press.
- Olsson, A. C., J. Svendsen, J. Botermans, and C. Bergsten. 2016. An experimental model for studying claw lesions in growing female pigs. *Livest. Sci.* 184:58–63. doi:10.1016/j.livsci.2015.12.005
- Olstad, K., S. Ekman, and C. S. Carlson. 2015. An update on the pathogenesis of osteochondrosis. *Vet Pathol* 52(5):785–802. doi:10.1177/0300985815588778
- Orth, M. W. 1999. The regulation of growth plate cartilage turnover. *J. Anim. Sci.* 77(Suppl 2):183–189. doi:10.2527/1999.77suppl_2183x
- Ott, E. A., and R. L. Asquith. 1995. Trace mineral supplementation of yearling horses. *J. Anim. Sci.* 73:466–471. doi:10.1016/S0737-0806(06)81884-8
- Ouattara, A., D. Cooke, R. Gopalakrishnan, T. H. Huang, and G. P. Ables. 2016. Methionine restriction alters bone morphology and affects osteoblast differentiation. *Bone Rep.* 5:33–42. doi:10.1016/j.bonr.2016.02.002
- Pluym, L. M., S. Hoorebeke, A. Lopez, and R. Jeroen. 2011. Prevalence and risk factors of lameness and claw lesions in two types of group housing for pregnant sows. *Vet. Med.* 56:101–109. doi:10.17221/3159-VETMED.
- Pluym, L. M., A. Nuffel, S. Weyenberg, and D. Maes. 2013. Prevalence of lameness and claw lesions during different stages in the reproductive cycle of sows and the impact on reproduction results. *Animal.* 7:1174–1181. doi:10.1017/S1751731113000232
- Quinn, A. J., L. E. Green, P. G. Lawlor, and L. A. Boyle. 2015. The effect of feeding a diet formulated for developing gilts between 70kg and ~140kg on lameness indicators and carcass traits. *Livest. Sci.* 174:87–95. doi:10.1016/j.livsci.2014.12.016
- Reiland, S. 1975. Osteochondrosis in the pig [PhD thesis]. Stockholm (Sweden): The Royal Veterinary College.
- van Riet, M. M. J., S. Millet, M. Aluwé, and G. P. J. Janssens. 2013. Impact of nutrition on lameness and claw health in sows. *Livest. Sci.* 156:24–35. doi:10.1016/j.livsci.2013.06.005
- Shaw, D. T., D. W. Rozeboom, G. M. Hill, M. W. Orth, D. S. Rosenstein, and J. E. Link. 2006. Impact of supplement withdrawal and wheat middling inclusion on bone metabolism, bone strength, and the incidence of bone fractures occurring at slaughter in pigs. *J. Anim. Sci.* 84:1138–1146. doi:10.2527/2006.8451138x
- Shelton, N. W., M. D. Tokach, S. S. Dritz, R. D. Goodband, J. L. Nelssen, and J. M. DeRouchey. 2011. Effects of increasing dietary standardized ileal digestible lysine for gilts grown in a commercial finishing environment. *J. Anim. Sci.* 89:3587–3595. doi:10.2527/jas.2010-3030
- Tybirik, P. E. R. 2015. Nutrient recommendations for pigs in Denmark. 17th ed. Copenhagen (DK): SEGES-VSP Danis Pig Res. Cent..
- Tóth, F., J. L. Torrison, L. Harper, D. Bussieres, M. E. Wilson, T. D. Crenshaw, and C. S. Carlson. 2016.

- Osteochondrosis prevalence and severity at 12 and 24 weeks of age in commercial pigs with and without organic-complexed trace mineral supplementation. *J. Anim. Sci.* 94:3817–3825. doi:10.2527/jas.2015-9950
- Varagka, N., M. Lisgara, V. Skampardonis, V. Psychas, and L. Leontides. 2016. Partial substitution, with their chelated complexes, of the inorganic zinc, copper and manganese in sow diets reduced the laminitic lesions in the claws and improved the morphometric characteristics of the hoof horn of sows from three greek herds. *Porcine Health Manag.* 2:26. doi:10.1186/s40813-016-0040-3
- Veum, T. L., D. R. Ledoux, M. C. Shannon, and V. Raboy. 2009. Effect of graded levels of iron, zinc, and copper supplementation in diets with low-phytate or normal barley on growth performance, bone characteristics, hematocrit volume, and zinc and copper balance of young swine. *J. Anim. Sci.* 87:2625–2634. doi:10.2527/jas.2008-1604
- Weary, D. M., J. M. Huzzey, and M. A. von Keyserlingk. 2009. Board-invited review: using behavior to predict and identify ill health in animals. *J. Anim. Sci.* 87:770–777. doi:10.2527/jas.2008-1297
- Welfare Quality. 2009. Welfare quality assessment protocol for pigs (sows and piglets, growing and finishing pigs). Lelystad (The Netherlands): Welfare Quality Consortium.
- Woodard, J. C., H. N. Becker, and P. W. Poulos, Jr. 1987. Effect of diet on longitudinal bone growth and osteochondrosis in swine. *Vet. Pathol.* 24:109–117. doi:10.1177/030098588702400202
- Yazdi, M. H., N. Lundeheim, L. Rydhmer, E. Ringmar-Cederberg, and K. Johansson. 2000. Survival of Swedish Landrace and Yorkshire sows in relation to osteochondrosis: a genetic study. *Anim. Sci.* 71:1–9. doi:10.1017/S1357729800054849
- Ytrehus, B., C. S. Carlson, and S. Ekman. 2007. Etiology and pathogenesis of osteochondrosis. *Vet. Pathol.* 44:429–448. doi:10.1354/vp.44-4-429
- Ytrehus, B., C. S. Carlson, N. Lundeheim, L. Mathisen, F. P. Reinholt, J. Teige, and S. Ekman. 2004a. Vascularisation and osteochondrosis of the epiphyseal growth cartilage of the distal femur in pigs—development with age, growth rate, weight and joint shape. *Bone.* 34:454–465. doi:10.1016/j.bone.2003.07.011.
- Ytrehus, B., E. Grindflek, J. Teige, E. Stubsjøen, T. Grøndalen, C. S. Carlson, and S. Ekman. 2004b. The effect of parentage on the prevalence, severity and location of lesions of osteochondrosis in swine. *J. Vet. Med. A. Physiol. Pathol. Clin. Med.* 51:188–195. doi:10.1111/j.1439-0442.2004.00621.x