Vitamins and pig reproduction

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The premise of pig reproduction is to produce the maximum number of pigs weaned per sow per year at the lowest cost. Besides genetic improvements and modern production management techniques, vitamin nutrition adds another dimension of improving the efficiency of pig reproduction. Vitamin C improves semen quality in boars for alleviating summer infertility syndrome. Maximising bone density with adequate vitamin D nutrition in replacement gilts may offer protection against not only skeletal disorders precipitated by confinement housing in subsequent reproductive years but also early culling from the breeding herd. Riboflavin lowers the incidence of anoestrus in post pubertal gilts. Vitamin E improves litter size, whereas biotin and folic acid improves the survival of embryos and foetuses during pregnancy. Feeding extra vitamin E to lactating sows results in a carry-over of vitamin E for the piglets up to three weeks after weaning, with the positive implication for livability and immunity. Biotin reduces the interval from weaning to oestrus. In addition, biotin improves the integrity of the claw and, therefore, lowers the incidence of foot lesions.

Swine reproduction presents a lot of challenges to be overcome by genetic improvements, modern management techniques and advanced nutritional technologies. In understanding swine reproduction, one must have a good understanding of the chronological sequence of reproductive events in sows. In addition, knowledge must also be extended to the management of breeding boars, gilts and sows and the vitality of piglets.

Traditionally, producers focused breeding on sows relative to fat thickness, maximum growth and large litter sizes.

The focus now is extended to piglet vitality (number of pigs born alive and number of weaned piglets) and to good mothering characteristics.

From a nutritional standpoint, vitamins play an important role in swine reproduction in that they allow breeding swine to improve reproductive efficiency, longevity and piglet vitality.

The vitamin supplementation guidelines for swine are shown in Table 1.

Boars and semen quality

The use of boars in sow herds is decreasing as a result of the increased use of artificial insemination. Besides semen production, boars are able to provide tactile, olfactory, auditory and visual cues that can be very functional to optimise reproductive output.

The presence of boars in sow herds can prevent prolonged weaning to oestrus intervals and help predict when ovulation takes place.

Semen quality gradually deteriorates when boars age as well as when they are heat stressed. Supplementing boar diets with selenium and vitamin E improves semen quality in boars. Feeding supplemental vitamin C to boars during heat stress helps to improve the quality and quantity of sperm produced by boars during the summer infertility period.

Selection of gilts

Selection of gilts and first breeding them at the right age and body weight are prerequisites to get large litter size in gilt litters and through their reproductive lifetime.

Replacement gilts must also be ensured that they have built up maximum bone reserve to enjoy longevity. Skeletal soundness is essential for the longevity of sows in the breeding herd. Physical activity is required for maintaining bone density.

Confinement of sows in gestation and lactation crates coupled with a lack of physical activity may aggravate bone loss, resulting in the possibility of culling of sows earlier. 25-hydroxy-vitamin D_3 has been shown to effectively improve porcine bone density and bone strength.

Prebreeding/breeding

● Ovulation/fertilisation.

The number of ova released varies between 15-25, depending on the age of the sow. Young sows have lower ovulation numbers than older sows. Feeding extra dietary riboflavin to postpubertal gilts helps to lower the incidence of anoestrus gilts. Fertilisation of the ova is generally 90-100% and thus has fairly minor influence on litter size.

Acute or prolonged stress may precipitate summer infertility syndrome which may be lowered by dietary supplementation of vitamin C which is in high demand for the synthesis of glucocorticoids during stress.

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Table 1. DSM vitamin supplementation guidelines for domestic animals 2004 – Optimum Vitamin Nutrition for Swine.

Swine[1] OVN	A	D_3	E[2]	K_3 (Menadione)	B_1	B_2	B_6	B_{12}	Niacin	D-Pantothenic acid	Folic acid	Biotin	C[7]	Choline	Beta-Carotene
	IU	IU	mg	mg	mg	mg	mg	mg	mg	mg	mg	mg	mg	mg	mg
Prestarter, up to 10 kg	10000–20000	1800–2000	60–100[3]	2.0–4.0	3.0–5.0	6–10	4.0–6.0	0.040–0.070	40–60	20–40	1.5–2.5	0.15–0.40	100–200[6]	500–800	
Starter, 10-20 kg	10000–15000	1800–2000	60–100	2.0–4.0	2.0–4.0	6–10	4.0–6.0	0.030–0.050	30–50	20–40	1.0–2.0	0.15–0.40	100–200[6]	200–400	
Growing, 20-50 kg	7000–10000	1500–2000	40–60	1.5–3.0	1.0–2.0	5–10	2.0–4.0	0.020–0.040	20–30	20–40	0.6–1.0	0.15–0.25		150–300	
Finisher, 50 kg - market	5000–8000	1000–1500	30–50[4]	1.0–1.5	0.5–1.5	3–8	1.5–3.0	0.015–0.030	20–30	20–40	0.5–1.0	0.10–0.20		100–200	
Sows	10000–15000	1500–2000	60–80[5]	1.0–2.0	1.0–2.0	5–9	3.0–5.0	0.020–0.040	25–45	18–25	3.0–5.0	0.30–0.50	200–300	500–800	300[8]
Boars	10000–15000	1500–2000	60–80	1.0–2.0	1.0–2.0	5–9	3.0–5.0	0.020–0.040	25–45	18–25	3.0–5.0	0.30–0.50	200–500	500–800	

[1] Added per kg air-dry feed. [2] Dietary fat higher than 3%; additional 5mg/kg feed for each 1% dietary fat
[3] For optimum piglet health: additional 150mg/kg feed. [4] For optimum meat quality: additional 150mg/kg feed
[5] For optimum piglet health during late pregnancy and lactation; total 250mg/kg feed (maternal transfer through colostrum/milk). [6] Recommended in stress conditions. [7] Vitamin C activity in phosphorylated form.
[8] For improved sow fertility; to be fed per animal per day immediately after weaning until confirmed conception

Treatment	No. of boars	Total no. of collections	Sperm density per ejaculate (billion/mL)	Total sperm ejaculate (billion)	Doses/ ejaculate	Assessment score
Control	6	43	0.267	58.89	15.28	1.49
Vitamin C	8	62	0.288	66.99	21.55	1.81

Table 2. Effects of dietary addition of vitamin C (200mg/kg diet) on sperm quality of young boars (Henman et al., 2001).

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Fortifying sow diets with extra vitamin E increases litter size. The likely mechanism of vitamin E on fertility may be due to its role in modulating the metabolism of arachidonic acid, which is the precursor of prostaglandins.

● **Pregnancy/foetal growth.**
80-90% of the losses between ovulation and birth occurs during the first 25 days of gestation (embryonic phase), with the remaining 10-20% occurring at the end of gestation (foetal phase).

High embryonic losses may be caused by the critically high growth rate during embryonic development. The foetal losses are largely the result of restriction of the available space per piglet in the uterus which is more likely to be a limiting factor in young sows.

The main vitamins affecting survival of the embryo or foetus include biotin and folic acid. Embryonic cells with a high degree of proliferation require extra folic acid for the synthesis of methyl groups and of purines and pyrimidines which are indispensable components of RNA and DNA.

The positive influence of folic aid on litter size is due to reduction of embryonic death rather than to any increase in the number of released ova. How biotin positively influences litter size is largely unknown. Basically, litter size is a function of ovulation and embryo survival. Scherf and Scott (1989) suggested the possibility for biotin to increase uterine space and placental development during mid pregnancy.

Simmins (1985) reported a non-significant increase of 20% in the length of the uterine horns by supplementing biotin in gilt diets. It is known that horn length is a significant factor in determining the final volume of the uterus. Scherf and Scott (1989) speculated that it is conceivable that relaxing prostaglandins are involved in the stretching phase of uterine enlargement.

Prostaglandins are metabolic products of polyunsaturated fatty acid metabolism from which biotin plays a key metabolic role in carboxylation reactions. Consider the muscle tone needed by the uterus in order to stretch and accommodate the developing litter in late pregnancy.

The foetus undergoes rapid development during pregnancy, resulting in greater demands of vitamins to fuel metabolic processes. Limitations in the maternal supply for vitamins may lead to foetal malformation or death. A deficiency of vitamin A may result in the growth of foetal lung and liver being disproportionate to the rest of the body.

Farrowing/weaning

● **Lactation/maternal nutrient transfer.**
Advances in genetics have resulted in a gradual increase in litter sizes and in the growth rate of piglets. An increase in litter size must also come with a concomitant increase in milk production and improved mothering characteristics to ensure that the piglets are strong enough to survive.

Vitamin E (mg/kg diet)	22	44	66
No. of litters	134	126	120
Total no. piglets born	11.85	11.97	12.27
No. of stillborn piglets	0.40	0.31	0.60
No. of piglets born alive	11.45	11.66	11.60
Litter weight (kg)	16.90	17.90	17.90
Piglet birth weight (kg)	1.46	1.63	1.51

Table 3. Effects of dietary vitamin E on sow reproductive performance over a five parity period (Mahan, 1994).

Both colostrum and milk contain high levels of calcium. It is, therefore, important that dietary levels of calcium and vitamin D_3 are adequate to support optimum milk production and piglet growth. It Is known that fat-soluble vitamins do not cross the placenta. In addition, all piglets are born deficient in both fat-soluble vitamins and energy.

Therefore, it is of paramount importance for piglets to obtain these nutrients during the suckling period initially through colostrums and subsequently through milk.

About 96% of fat soluble nutrients can be digested during the suckling period. Colostrum and milk are enriched in vitamin E that is readily transferred to the piglet. During lactation, the piglet relies on the sow's milk to receive its vitamin nutrition.

Enriching the milk by feeding extra vitamins in the sow diet is able to improve the vitamin status of the piglet. Raising the vitamin status of the pre-weaned piglet can be very helpful to overcome their stresses during the weaning process.

Similarly, supplementing extra dietary vitamins in the postweaned diet is also beneficial to the weaned piglet. Lauridsen and Jensen (2005) reported that α-tocopherol concentrations in piglet tissues were increased when extra vitamin E was added to the lactating sow diet (Table 4).

● **Rebreeding (weaning to oestrus interval).**
Biotin is able to reduce the interval from weaning to oestrus in sows through its involvement in energy metabolism. In addition, biotin stimulates oestrogen production and, therefore, reduces the incidence of weak and silent heats.

Sow longevity

Sow longevity introduces a continued economic and welfare concern for the pig industry. Culling younger sows at high levels result in decreased lifetime sow productivity. Producers should assess the reasons why sows are culled from the breeding herd. Reproductive failure, old age and skeletal disorders and foot lesions are the three predominant reasons.

Serenius and Stadler (2004) concluded that leg conformation is genetically correlated with the length of sow reproductive life. Skeletal disorders are often observed in sows with poor body frame conditions.

Adequate vitamin D_3 and calcium nutrition is, therefore, of prime importance in maximising bone mineral density in replacement gilts before incorporating them in the breeding herd. Foot lesion associated lameness is precipitated by biotin deficiency.

Biotin, involved in keratinisation processes, improves the integrity of the claw and therefore lowers the incidence of foot lesions.

In conclusion, supplementing vitamins at adequate or optimum levels in breeding swine diets is undisputed. Vitamins are proven to provide multiple benefits to swine reproduction for reproductive efficiency, longevity and piglet vitality ■

Table 4. Effects of supplemental dietary dl-α-tocopheryl acetate to sow feed (from one week before farrowing until weaning) on the concentration of α-tocopherol in plasma and tissues of piglets at weaning at 28 days of age (Lauridsen and Jensen, 2005).

dl-α-tocopheryl acetate (mg/kg diet)	70	150	250
Plasma α-tocopherol (mcg/mL)	3.75	5.18	3.48
Tissue α-tocopherol (mg/kg of wet tissue)			
Liver	5.42	9.15	12.24
Heart	3.05	6.59	9.31
Adipose tissue	7.48	10.29	13.38
Muscle	4.26	6.24	6.49