30

MICRONUTRIENTS AND REPRODUCTION

B. HARDY and D. FRAPE,
Dalgety Spillers Ltd., Bristol, UK

All micronutrients are required for reproduction in a general sense for maintenance, cell enlargement and multiplication and for various secretions including milk, but there is little published evidence on the interactions between micronutrients and control of the endocrine system. An interaction between diet and reproduction is likely to occur where the supply of nutrients is only marginally adequate and variable. Evidence of marginality in traditional diets has been found for several nutrients in recent years, largely as a result of changes in two other environmental factors: (1) the confinement of sows and intensification of pig production and (2) developments in crop husbandry and production.

It is very difficult to establish the micronutrient requirements of the breeding pig due to the complex nature of pregnancy, the ability of the animal to mobilize stored nutrients at different stages of the reproductive cycle and the interactions that occur between the micronutrients. Many estimates of their requirement are based on the minimum level required to prevent deficiency symptoms and therefore bear no relation to the amount

Table 30.1 MICRONUTRIENT REQUIREMENTS OF BREEDING PIGS (DRY MATTER BASIS)

	Current UK estimate	*NRC (1979)*	
	All breeding pigs	*Bred gilts and sows Young and adult boars*	*Lactating gilts and sows*
Fat-soluble vitamins			
Retinol (mg/kg) or	0.7	1.3	0.7
β-Carotene (mg/kg)	8.4	17.8	8.9
Cholecalciferol (mg/kg) or Ergocalciferol	-	5.5	5.5
α-Tocopherol (mg/kg)	10.2	11.1	11.1
Water-soluble vitamins			
Riboflavin (mg/kg)	3.0	3.3	3.3
Pantothenic acid (mg/kg)	10.0	13.3	13.3
Cyanocobalamin (μg/kg)	15.0	16.7	16.7
Pyridoxine (mg/kg)	1.5	1.1	1.1
Choline (mg/kg)	1000–1900	1400	1400
Minerals and trace elements			
Calcium (%)	0.90	0.83	0.83
Phosphorus (%)	0.70	0.67	0.55
Manganese (mg/kg)	10	11	11
Iodine (mg/kg)	0.50	0.15	0.15

of the micronutrient required for maximization of reproductive performance. The best current UK estimates of micronutrient requirements for breeding pigs have been compared with those given by the NRC (1979) in *Table 30.1*. There are insufficient data on which to base an estimate of the requirement for those vitamins, minerals and trace elements not shown in *Table 30.1*.

There is a marked difference in the total quantity of a micronutrient supplied to the animal in its diet and the 'requirement' as currently known. The relationship between the requirement, the supply from natural sources and that normally added to the diet by means of a vitamin–mineral supplement is shown in *Table 30.2*. In most cases the average vitamin supplementation rate more than covers the level of minimum requirement and the total cost of this is currently £1.15/tonne of finished feed. The addition of minerals and trace elements to the diet will add approximately 85p/tonne of finished feed. The total micronutrient supplementation represents 1–1.5% of the total cost of the feed.

Fat-soluble vitamins

RETINOL

Where sows are kept on pasture the likelihood of reproductive problems occurring as a result of deficiencies in any of the fat-soluble vitamins is very slight. The vitamin A requirements are provided as the precursor β-carotene which has a potency in the pig of approximately 9% that of retinol. A deficiency of retinol leads not only to a decline in general health but more specifically to a decrease in ovarian size and to testicular atrophy. These symptoms are associated with a lowering of the concentration of retinol in the colostrum and milk and a diminution of the hepatic reserves in the neonate. Ocular lesions and other abnormalities are also apparent in the offspring. Where the diet contains between 7 and 10 mg β-carotene/kg dietary dry matter, normality is achieved. Spring and summer pasture would normally provide adequate amounts, including sufficient hepatic reserves to sustain the sow during the winter period. Cereal-based diets given to confined sows should contain 700 μg preformed retinol/kg dietary dry matter (14 μg/kg liveweight daily).

Although the needs of gestation and lactation are likely to differ it is not possible to consider the states of gestation and lactation independently because hepatic reserves in the dam provide a source of retinol for the milk. The amount of retinol transferred from the mother to the foetus is closely related to the amount available during pregnancy (Hjarde *et al.*, 1961) both from liver reserves and from feed. Nevertheless it has been calculated (Frape *et al.*, 1969) that total placental transfer is only 1–2% of the amount given during gestation. Large doses of retinol given during the later stages of pregnancy (Thomas, Loosli and Willman, 1947; Whiting, Loosli and Willman, 1949) have increased the concentration in the colostrum and in the livers of the offspring but the amount of retinol ingested by the piglet in the colostrum under normal conditions greatly exceeds that found in the livers at birth and exceeds the concentration found in the milk (Braude *et al.*, 1947; Hjarde *et al.*, 1961).

Table 30.2 COMPARISON OF MINIMUM REQUIREMENT, AMOUNT NATURALLY PRESENT AND AVERAGE SUPPLEMENTATION RATES AND THE AVERAGE COST OF SUPPLEMENTS OF SOW DIETS IN THE UK

	Minimum requirement (mg/kg DM)	*Naturally present in diet* (mg/kg DM)	*Average supplementation rate* (mg/kg DM)	*Average cost of supplementation* (p/tonne feed)	*Total dietary vitamin content/minimum requirement*
Vitamin A as retinol	0.7	Nil	4:7	21.6	6.7
Vitamin D_3 as cholecalciferol	(0.003)	Nil	0.057	2.0	19.2
DL-α tocopheryl acetate	10.2	8	13.2	12.6	2.1
Menaphthone salts	(0.2)	1	4.6	2.4	28.0
Riboflavin	3.0	1.1	5.7	9.0	2.3
Pantothenic acid	10.0	6	13.8	7.2	2.0
Pyridoxine	1.5	4	2.6	4.0	4.4
Cyanocobalamin	0.015	Nil	0.019	2.8	1.3
Choline	1000–1900	1000	121	12.7	0.6–1.1
Thiamin	(1.5)	4	1.1	1.5	3.4
Available Nicotinic acid	(14.0)	2	17.2	3.0	1.4
Biotin	(0.15)	0.1	0.10	27.0	1.4
Folic acid	(0.4)	0.4	0.25	1.2	1.6
Ascorbic acid	Nil	Nil	7.5	7.5	

Figures in parentheses are based on limited data and cannot be considered as an estimate of requirement.

Little objective evidence is available on a positive effect of retinol on the reproductive processes of pigs under practical conditions. A direct effect on conception or implantation may exist (Sevkovic *et al.*, 1973; Saryceva, 1968). As a result, some interest has been shown in the parenteral use of retinol in large doses given by injection at the time of mating. Evidence from Eastern Europe (Gondos *et al.*, 1970, Jugina, 1966) suggests increases in the number and weight of foetuses in gilts at the 60th day of pregnancy and an increase in litter size at birth as a result of such injections. The injection of 30 mg retinol (Sevkovic *et al.*, 1969) before mating has been shown to increase litter size and weight at birth. Even better results have been obtained with the injection of between 75 and 300 mg before mating, at mid-pregnancy and at the end of pregnancy. Similar injections (Jancic *et al.*, 1970; Stumpf, 1968; Petrenko and Zirnov, 1968) have been claimed to reduce the numbers stillborn and to lower neonatal mortality. However, responses of this nature have not been produced in western countries under practical conditions.

VITAMIN D_3

Two forms of vitamin D are of importance, vitamin D_2 (ergocalciferol) and vitamin D_3 (cholecalciferol). There is some evidence that pigs do not absorb ergocalciferol from feedstuffs and therefore cholecalciferol is the main precursor of vitamin D. In the absence of any meaningful data relating to the vitamin D requirement of sows, no further information is given.

α-TOCOPHEROL

The concentration of tocopherols per unit dry matter in fresh herbage is between five and ten times as great as that in some cereals or their products. Tocopherols are labile and the preservation of cereals by ensilage has been demonstrated to cause almost the complete loss of vitamin E activity. Symptoms of a deficiency of the vitamin have, therefore, been described amongst sows receiving ensiled cereals. It is uncertain whether the requirements/unit of bodyweight for reproduction are higher or lower than those for growth. Several reports suggest that the requirement of young pigs is greater than that of their dams. Sows maintained on a diet deficient in vitamin E and selenium for five reproductive cycles (Glienke and Ewan, 1974) produced piglets which died between 30 and 48 days of age in the fifth parity. The symptoms in the young include muscular dystrophy (Adamstone, Krider and James, 1949; Aydin, Pond and Kirtland, 1973) not generally seen in their dams, low piglet survival and depressed growth rate. Considerable reserves of α-tocopherol normally occur in the sow which meet the demands of the neonatal pig for several weeks by transference across the placenta and by secretion in the colostrum. Thus, sows maintained on a diet deficient in vitamin E and selenium have been shown to produce normal piglets during the first reproductive cycle of the deficiency and symptoms occurred only after five such cycles (Glienke and Ewan, 1974). The requirement for milk secretion could be considered to be greater than that for foetal growth as the milk provides an important source for the offspring (Cline, Mahan and Moxon 1974), although a deficient gestation diet has been shown to depress milk yield (Nielsen *et al.*, 1973). This effect could be ascribed to the vitamin E potency of the diet, although it could be ascribed more directly to the oxidized herring oil also present in the sow's diet. In the absence of the herring oil (Nielsen, 1971) additional α-tocopherol had no effect on litter size at birth or at three weeks, suggesting that the vitamin E potency of the basal diet may have been higher than in the more recent study.

The tissue aberrations appear to have a more damaging effect on the neonate than on the sow. They include abnormalities of cardiac and striated muscle, blood and the liver (Eggert *et al.*, 1957; Michel, 1968; Reid, 1968; Reid *et al.*, 1968; Money, 1970; Piper *et al.*, 1975). The effects of deficiency in these animals are exacerbated by some forms of injectable complexed iron used as a prevention of anaemia in piglets. The consequences are severe bruising, a rise in serum aspartate-amino-transferase and increased mortality as ferric iron imposes a severe strain on the redox system of the pig (Tollerz and Lannek, 1964; Pedersen, 1966; Tollerz, 1973; Miller *et al.*, 1973). Protection from iron intoxication has been achieved by the intramuscular injection, not only of α-tocopherol, but also of several synthetic antioxidants. A number of such synthetic compounds, including methylene blue, butylated hydroxyanisole (BHA), butylated hydroxytoluene (BHT) and ethoxyquin, apparently possess some biological activity comparable with that of the tocopherols, substituting for them in some functions provided they penetrate to and are retained by the tissue. These functions include suppression of iron intoxication/bruising (Tollerz, 1973), prevention of steatitis, nutritional muscular dystrophy and liver necrosis (Michel, 1968) and stimulation of erythropoiesis.

Selenium and vitamin E

It is appropriate to consider the role of selenium in vitamin E metabolism at this stage as the symptoms of vitamin E deficiency in the field can frequently be overcome by the dietary provision, or the injection, of selenium as sodium selenite. In areas where hepatic necrosis and mulberry heart disease have been endemic, probably associated with soils deficient in selenium, sows treated by intramuscular injection have a lowered incidence of stillbirths, neonatal mortality and hepatic necrosis (Van Vleet, Meyer and Olander, 1973). Similar responses in deficient sows have been obtained by providing diets containing 0.13 mg selenium/kg (Mahan *et al.*, 1974). The concentration required in the diet depends to some extent upon the source of the selenium as some natural sources have a greater availability than sodium selenite (Ku *et al.*, 1972). Supplementation of the sow results in the production of milk with a higher selenium content (Mahan, Moxon and Hubbard, 1975; 1977), but the growing pig is unlikely to obtain sufficient selenium from this source to meet its needs (Mahan *et al.*, 1974; Mahan, Moxon and Hubbard, 1975; 1977). A risk attached to the injection of selenium compounds results from the narrow limits of dose within which a response can be obtained, but which avoids toxicity. Experimental evidence has demonstrated that 1.5 mg selenium/kg liveweight daily is apparently not toxic (Van Vleet, Meyer and Olander, 1974) but similar evidence (Diehl, Mahan and Moxon, 1975) has suggested that an injection at the rate of 1.65 mg/kg liveweight is toxic. A toxic level in the diet of sows lies between 5 and 10 mg/kg diet. Storage of selenium occurs in the blood and other tissues and gilts reared for breeding on a selenium-deficient diet either die before breeding or suffer reproductive failure. An adequate level in the diet from a mixture of natural and synthetic sources appears to be in the region of 0.15 mg/kg for breeding pigs.

The results of numerous experiments conducted with pigs deficient of both α-tocopherol and selenium suggest that the one nutrient can substitute for the other. This outcome undoubtedly results from a sparing effect rather than a substitution as more recent experiments entailing extended depletion periods have indicated that both are required. Such experiments (Ewan *et al.*, 1969; Wastell *et al.*, 1972) have demonstrated the requirements for both nutrients and have shown that the requirements of the young pig appear to exceed those of their dams. A major function of α-tocopherol is suggested as being a protector of the essential fatty acids of cell membranes, whereas selenium acts in glutathione peroxidase and functions to mop up toxic tissue peroxides, production of which the vitamin has failed to inhibit.

Essential fatty acids

No information of note is available on the needs of the breeding pig for essential fatty acids (EFA). In view of the role of EFAs in prostaglandin synthesis, and the evidence in breeding rats, it is speculated that intensively managed breeding boars and sows have a particularly important requirement for EFA. The germ of cereal grains is a source of oil which is a rich source of linoleic acid (C18:2ω6)—a precursor of arachidonic acid (C20:4ω6) and an essential component of cellular lipids. Relatively few unsaturated fatty acids are precursors of this C20 acid and these must be provided by the diet. Certain prostaglandins, for which a role has been demonstrated in reproduction are synthesized from arachidonic acid. Whereas the oil from cereal grains is a rich source of the ω6 acid-linoleic, that oil is a relatively poor source of the ω3 fatty acid α-linolenic (C18:3) and timnodonic acid (C20:5). These unsaturated fatty acids are precursors of another group of prostaglandins and are found abundantly in herbage lipids and fish oils. It remains to be seen whether pasture species can confer any breeding advantage in pigs through this medium.

Water-soluble vitamins

A dietary requirement in breeding stock for many of the water-soluble vitamins has not been demonstrated. This situation may arise as a consequence of the relatively voluminous hind gut of the mature pig in which a vast population of microorganisms synthesize a number of the vitamins of this group. The absorption from the intestines of some members of the B vitamin group synthesized by the gut microflora has been demonstrated in several mammalian species. The extent, however, to which this acts as a biologically important source for direct absorption remains to be determined, although undoubtedly a portion of this source is made available to the pig through the mechanism of coprophagy. Several experiments in pigs have demonstrated a better reproductive performance in sows having access to their faeces, but whether this results from an enhanced supply of B vitamins and vitamin K, or from other nutrients, or indeed from a general reduction in environmental stress, has not been determined.

RIBOFLAVIN

An indirect effect on breeding performance of sows might be anticipated from the known enhanced antibody response in the young pig to injected antigens when deficient diets are supplemented with either pantothenic acid, pyridoxine or riboflavin (Harmon *et al.*, 1963). Whether the requirements for normality of this function differ from those for optimum growth is, however, not known. Fresh pasture herbage is a relatively rich source of riboflavin, but supplementation of cereal-based diets is generally required. During absorption riboflavin is phosphorylated in the wall of the small intestine and in its phosphorylated form acts as a coenzyme in many reactions. In a deficient state sows lose their appetite and the sows either do not conceive or a high neonatal mortality occurs amongst piglets. The dietary level found adequate for growing pigs (Miller and Ellis, 1951) is inadequate for satisfactory reproduction and lactation in the sow. Breeding sows appear to require a minimum of 3.0 mg riboflavin/kg dietary dry matter (Miller and Ellis 1951; Pochernnyaeva, 1976).

NICOTINIC ACID AND PANTOTHENIC ACID

Nicotinic acid is present in cereals in a bound form which is completely, or largely, unavailable to the pig. Diets based upon maize which are low in tryptophan have been demonstrated repeatedly to cause nicotinic acid deficiency in growing pigs. On the other hand, the only experiment in which it was attempted to produce a deficiency in sows (Ensminger, Colby and Cunha, 1951) failed to produce definite symptoms, despite an absence of gut synthesis. Symptoms of pantothenic acid deficiency have, on the other hand, been produced in gilts (Ensminger, Colby and Cunha, 1951). Diets containing up to only 7.8 mg/kg dietary dry matter (Teague, Palmer and Grifo, 1970; Teague, Grifo and Palmer, 1971) caused a marginally reduced reproductive performance and symptoms in the progeny of second generation gilts. As a part of the coenzyme A molecule the vitamin has a key role in many metabolic reactions, not confined to any particular reproductive function. Supplementation of breeding diets is recommended and some evidence suggests that the breeding performance of boars, particularly under intensive conditions, can be affected by an inadequacy in natural diets causing abnormalities in gait.

PYRIDOXINE AND CYANOCOBALAMIN

Experiments on the response of gilts and sows to pyridoxine have been both small in scale and short in duration. The supplementation of diets containing 1.0–2.3 mg pyridoxine/kg dietary dry matter was associated with a non-significant increase in the number of piglets weaned per litter (Ritchie *et al.*, 1960; Wöhlbier and Siegel, 1967). Supplementation gave an increase in the pyridoxine content of the sow's milk.

Pyridoxine is widely distributed in feed ingredients and is synthesized by gut bacteria. Errors may, however, be made in estimating the potency of diets as the standard test organism *S. uvarum* underestimates the potency of pyridoxal and pyridoxamine for the pig. Pyridoxine functions biologically in the phosphorylated form as a coenzyme in transaminase and decarboxylase activities. Therefore it functions in the synthesis of porphyrin rings in haemoglobin formation and in lymphocyte production and hence in antibody production. More specifically for the breeding pig its role in the synthesis of endocrine secretions awaits investigation. Studies with rats have demonstrated such an association with the thyroid gland, adrenal glands and the testes. (The intensification of sow production with their maintenance in stalls lends itself to such investigation.)

Cyanocobalamin is also synthesized by gut bacteria and therefore sows maintained in deep litter yards may be less susceptible to deficiency than those housed in stalls. The vitamin is, however, stored in the blood and liver so that symptoms of deficiency only develop gradually. Conversely supplementation of the diet of sows is of more benefit than the supplementation of gilt diets (Frederick and Brisson, 1961) and successive litters produced by sows given a diet low in the vitamin are progressively weaker (Kralovanszky, Eöri and Kallai, 1954; Teague and Grifo, 1966). Cyanocobalamin is involved in the transfer of the so-called C1 fragment in association with folic acid. The two vitamins tend to partially spare the requirement for each other and the need for them is reduced if the diet contains a good supply of compounds acting as a source of C1 groups such as choline, betaine and methionine. An increasing scarcity of animal proteins may imply that supplementation of breeding pig diets is increasingly based on vegetable products, less reliance being placed on animal proteins, and therefore supplementation with cyanocobalamin is becoming more necessary.

Cyanocobalamin in milk is strongly bound to minor whey proteins which are present in excess in sow's milk (Gregory and Holdsworth, 1955) and it has been postulated that this increases the amount of the vitamin available to the piglet directly, but also indirectly, by preventing its uptake by intestinal microorganisms (Ford, 1974). The much higher binding capacity of sow's milk, compared with cow's milk, may have implications from the point of view of the relative requirements for the vitamin of piglets reared on sow's milk and those reared on diets based on cow's milk. The general conclusion concerning the requirements for the vitamin in sows is that they should be provided with 15 μg/kg dietary dry matter, but in the light of evidence with older pigs (Frederick, 1962; Teague and Grifo, 1966) the needs may be increased considerably for older sows.

BIOTIN

Biotin is again synthesized by gut bacteria, but there appears to be a definite need for the vitamin in the diet of sows. Much of this is undoubtedly met by natural feed sources, but the relative availability to the pig of different sources is unknown. The availability of microbiologically assessed biotin to the chick is low for barley, wheat, milo and some protein feeds of animal origin but is high for maize and oilseed meals. It is widely involved metabolically, including its involvement in the synthesis of fatty acids found in healthy skin. There is considerable storage in the liver and probably similar arguments apply to it as apply to cyanocobalamin and the age of the sow. Supplementation either by injection (Cunha, Adams and Richardson, 1968) or in the feed of sows (Brooks, Smith and Irwin, 1977) receiving a basal diet containing just over 100 μg biotin/kg according to microbiological assay, reduced the incidence of foot lesions and dry or eczematous skin. Sows given supplements (Brooks, Smith and Irwin, 1977) produced more piglets and were remated significantly sooner after weaning than were control sows.

FOLIC ACID

Folic acid is another vitamin synthesized by gut bacteria and although normally no dietary requirement occurs for the growing pig, there may be a need in sows under some conditions, particularly where vitamin B_{12} intake is limited. Folate in sow's milk is strongly bound to minor whey proteins and it has been postulated that this increases the amount of the vitamin available to piglets (Ford and Scott, 1975). The authors suggested that in view of the low concentration of folate in sow's milk and the rapid postnatal increase in liver folate, folate-synthesizing bacteria in the intestine may make a significant contribution to the folic acid nutrition of the piglet. The possibility is suggested that in acting to sequester the milk folate the unsaturated binder protein in milk may reduce the growth of folate-dependent bacteria in the gut, and hence encourage the growth of bacteria that contribute to the folate needs of the piglet.

CHOLINE

The requirement for dietary choline depends upon the quantitative presence of other methyl donors and the dietary content of vitamin B_{12} (Dyer and Krider, 1950). Earlier evidence has suggested that choline deficiency in gilts is manifested by a splay-leg condition in their neonatal progeny but this claim could not be clearly substantiated by more recent work. Experiments covering six parities (Kornegay and Meacham, 1973) have shown that sows receiving a maize–soyabean meal diet containing 150 g crude protein/kg produced fewer piglets in the fifth and sixth litters when no supplementary choline was given. An experiment in which similar diets were involved, but conducted at nine centres, showed that the addition of 770 mg choline/kg diet increased the total number of live pigs born per litter and the number of pigs alive at two weeks post-partum. A further experiment (Stockland and Blaylock, 1974) in which maize–soya diets were supplemented with 410 mg and 820 mg choline/kg showed that conception rate, farrowing rate and total and live piglets farrowed were increased by the choline addition.

The evidence in total suggests that sow diets containing 150 g of crude protein, including 2 g methionine, also require the diet to contain between 1400 mg and 1900 mg choline/kg dry matter.

Trace minerals

IRON

A number of attempts has been made to increase the haemoglobin level in the blood and the hepatic iron reserves of piglets at birth by administering various iron salts in chelated and free forms to the sow. Evidence on the success of these measures is conflicting, although iron from more complex molecules may be transferred across the placenta (Agapitova, 1970; Brady *et al.*, 1975). Higher than normal concentrations of copper in pregnancy diets have also been shown to increase the amount of iron in piglets at birth (Hemingway, Brown and Luscombe, 1974). The piglet at birth is relatively well endowed with iron (blood haemoglobin is approximately 80 g/l) and anaemia arises principally as a consequence of rapid growth rate on a diet of mother's milk deficient in iron. Regardless of the scale of iron storage at birth, it is probable that pigs denied access to iron other than that supplied by sow's milk will develop anaemia within a few weeks when blood haemoglobin may fall to 40 g/l. Various sources of iron given to the lactating sow may bring about an increase in the iron content of the milk but owing to the low concentration this can have only a small effect on the piglet. If the piglet has access to iron provided by the sow's feed and faeces, then performance may be improved even if the piglet is denied iron from other sources. It is very unlikely that an iron deficiency anaemia would develop in breeding sows or boars given conventional diets as the iron content of most natural ingredients would normally meet their needs. If the diet is, on the other hand, imbalanced in respect of some other minerals, viz. calcium, copper, manganese or phosphorus, then interference with iron metabolism may occur in the sow. For example, feeds containing growth promotion levels of copper could induce hypochromic microcytic anaemia through impairment of iron absorption (Gipp *et al.*, 1974).

ZINC

Zinc deficiency problems in pigs have arisen largely as a consequence of interactions with other mineral elements. Evidence in the growing pig has indicated that where copper is used as a growth promoter this has led to the development of parakeratosis in pigs fed vegetable protein-based diets unless the zinc supplementation has been adequate. Increased dietary concentrations of calcium and phytic acid also increase the requirement for zinc in that phytic acid adversely affects the zinc availability and a rise in the dietary content of calcium increases urinary excretion of zinc (Beardsley and Forbes, 1957). Whether high levels of dietary calcium also influence absorption from the intestines is not clear. Dry diets are more likely to produce parakeratosis than the same diets fed wet (Lewis, Grummer and Hoekstra, 1957) possibly due to the hydrolysis of phytates in diets which are soaked (Frape, Wayman and Tuck, 1979). Furthermore, iron may act as an antagonist to zinc in relieving parakeratosis (Hoefer *et al.*, 1960) and high levels of dietary zinc may depress the utilization of dietary iron (Cox and Hale, 1962). No evidence of these deficiencies and interactions has been demonstrated in sows in practice.

Observations made in a range of species other than the pig have shown that spermatogenesis and the development of the primary and secondary sex organs in the male and all phases of the reproductive process in the female from oestrus to parturition and lactation can be adversely affected by zinc deficiency. The impaired development of the secondary sex glands may be subsidiary to the inanition of zinc deficiency which could result in a reduced gonadotrophin output and consequential fall in androgen production. Testicular atrophy and failure of spermatogenesis, on the other hand, is due directly to lack of zinc. In the female congenital abnormalities and failure to suckle at birth have been observed, together with an impairment of lactation. Zinc levels in the milk are reduced by a deficiency of this element so that the offspring may suffer further. Zinc-deficient females deliver their offspring with extreme difficulty and suffer excessive bleeding.

Limited evidence in sows shows that suboptimal dietary levels of zinc reduce the size of litters and the zinc content of some of the tissues in the young. No abnormalities in foetal development or in maternal behaviour have been shown (Hoekstra *et al.*, 1967). Skeletal abnormalities have been demonstrated in foetuses of several species resulting from both zinc and manganese deficiency and a reduction in the size and strength of the femur in zinc-deficient baby pigs has been reported by Miller *et al.* (1968); but the effect appeared to be largely a consequence of reduced food intake. However, in another study with weanling pigs, the reduced skeletal growth associated with zinc deficiency was most apparent in the low activity of the epiphyseal growth plate and at other points of osteoblastic prominence (Norrdin *et al.*, 1973). Zinc is involved primarily in nucleic acid and protein metabolism and hence in the fundamental processes of cell replication. The utilization of amino acids in the synthesis of protein is impaired in zinc deficiency and the general consensus is that growing boars and gilts have a higher requirement for zinc than do castrates, probably resulting from their higher protein demands. Two papers, however, suggest that zinc requirements of breeding sows do not exceed 40 mg/kg diet containing 1.4% calcium (Pond and Jones, 1964; Hennig, 1965). One of these experiments entailed the use of fishmeal as a source of protein and as other work has shown that the availability of zinc in soyabean meal is less than that in milk

(Smith, Plumlee and Beeson, 1962), it is generally considered that the diet of breeding pigs should contain 50 mg zinc/kg, particularly where it is based on vegetable proteins. This would, in addition, allow for some body reserves in the offspring (Palludan and Wegger, 1972; Hedges, Kornegay and Thomas, 1976). The recommendation assumes normal levels of other mineral elements for breeding stock. Zinc supplementation of a maize–soyabean meal diet containing 30–34 mg Zn/kg and 1.6% calcium significantly increased the number of live pigs per litter without affecting the birth or weaning weights (Hoekstra *et al.*, 1967).

MANGANESE

The requirement of pigs for satisfactory reproduction may be higher than that required for body growth. In fact satisfactory growth in young pigs has been reported with diets supplying only 1 mg/kg (Johnson, 1943; Plumlee *et al.*, 1956). This level incurs a marked tissue manganese depletion and when such diets are fed throughout gestation and lactation, skeletal abnormalities and impaired reproduction become apparent. Other studies have shown that diets containing less than 3 mg/kg manganese cause a depression in reproductive performance (Johnson 1943) but concentrations of 6 or 12 mg/kg appear to be adequate (Johnson, 1943; Grummer *et al.*, 1950). The precise mode of action of manganese in preventing reproductive defects in both males and females has not yet been established. It has been suggested (Doisy, 1974) that lack of manganese inhibits the synthesis of cholesterol, which in turn limits the synthesis of sex hormones. In laboratory species a deficiency of manganese is associated with defective ovulation, testicular degeneration and infant mortality.

COPPER

Very little work has been conducted on the copper requirements of the breeding pig. One report (Carpenter, 1946/47) showed that by increasing the copper content of the diet from 7 to 35 mg/kg an increase in litter size of two to three pigs occurred. In the female rat and guinea pig a copper deficiency results in reproductive failure due to foetal death and resorption (Dutt and Mills, 1960; Hall and Howell, 1969; Howell and Hall, 1969; 1970). The oestrous cycle remains unaffected and conception appears to be uninhibited. Normal foetal development in copper-deficient rats was shown to cease on the 13th day of pregnancy (Howell and Hall, 1969) and necrosis of the placenta became apparent on the 15th day.

IODINE

Little work has been conducted on the iodine requirements of breeding pigs. Growing pigs fed maize–soyabean meal diets have a requirement of iodine in the region of 0.1 mg/kg (Sihombing, Cromwell and Hays, 1974) and the results of other experiments suggest that the requirement does not exceed 0.14 mg/kg. Iodine requirements are influenced by the presence of goitrogens and certain other elements in the diet. Commonly found goitrogens are the thiocyanates. Some elements including rubidium and arsenic can interfere with iodine uptake and induce goitre.

Changes in thyroxine secretion are reflected in changes in the adrenal cortex and a relationship between the thyroid and the gonads is apparent in all male and female mammals. Reproductive failure is often the outstanding manifestation of iodine deficiency with the birth of weak, dead or hairless young. Foetal development may be arrested at any stage leading to early death and resorption, abortion and stillbirth. The live birth of weak young frequently associated with prolonged gestation often occurs together with retention of foetal membranes at parturition. The occurrence of hairless piglets has been prevented by adding potassium iodide, equivalent to 0.17 mg iodine/kg diet given to pregnant sows (Hart and Steenbock, 1918) and intrauterine death has been reduced by feeding sows 0.77 mg iodine/100 kg liveweight daily from three months before service (Varganov, 1965). Supplements are frequently given to breeding females by including iodized salt which provides 0.35 mg iodine/kg diet (Andrews *et al.*, 1948). A roughly similar quantity of 0.4 mg in diets containing 12% of extracted rapeseed meal was sufficient to prevent intrauterine and neonatal deaths in piglets if fed to females throughout pregnancy (Devilat and Skoknic, 1971). Suckling piglets should receive sufficient quantities from their dams as it has been demonstrated (Iwarsson, Bengtsson and Ekman, 1973) that between 20% and 45% of the iodine intake of sows is secreted in the milk.

No scientific evidence has been produced in breeding pigs to establish their dietary requirement for vitamins D_3, K, thiamine and ascorbic acid. There appears not to be a need for the supplementation of practical diets with any of them, although the claims that navel bleeding is caused by a dietary deficiency of vitamin K and ascorbic acid may require further investigation.

Conclusion

The amount of evidence on which to base requirement figures for micronutrients for breeding pigs is sparse. The high cost of purified or semi-purified diets for sows, plus the difficulty in measuring the potency and controlling the potency of micronutrients in natural diets, has been restrictive to experimentation. Developments in instrumentation and assay techniques, coupled with the individual housing and management of sows, should lend themselves to easier research in the future.

More information is required on the total micronutrient content and availability values in raw materials and on the effect of processing techniques used in feed manufacture on micronutrient stability. This, linked to better understanding of interactions between macro and micronutrients, should enable the breeding pig's requirement, once known, to be satisfied within a given daily feed allowance, thereby ensuring the maximization of economic production of weaned pigs.

References

ADAMSTONE, F.B., KRIDER, J.L. and JAMES, M.F. (1949). Response of swine to vitamin E deficient rations. *Ann. N.Y. Acad. Sci.* **52**, 260

AGAPITOVA, G.N. (1970). Iron glycerophosphate in rations for pregnant sows. *Sb. nauch. Rab. vses. nauchno-issled. Inst. Zhivotn.* **20**, 55–57 (*Nutr. Abstr. Rev.* **42**, 769)

ANDREWS, F.N., SHREWSBURY, C.L., HARPER, C., VESTAL, C.M. and DOYLE L.P. (1948). Iodine deficiency in newborn sheep and swine. *J. Anim. Sci.* **7**, 298–310

AYDIN, A., POND, W.G. and KIRTLAND, D. (1973). Dietary PUFA – Vitamin E: Dam–progeny effects in pigs. *J. Anim. Sci.* **37**, 274 (Abstract)

BEARDSLEY, D.W. and FORBES, R.M. (1957). Growth and chemical studies on zinc deficiency in the baby pig. *J. Anim. Sci.* **16**, 1038 (Abstract)

BRADY, P.S., KU, P.K., GREEN, F.F., ULLREY, D.E. and MILLER, E.R. (1975). Evaluation of an amino acid–iron chelate hematinic. *J. Anim. Sci.* **41**, 308 (Abstract)

BRAUDE, R., COATES, M.E., HENRY, K.M., KON, S.K., ROWLAND, S.J., THOMPSON, S.Y. and WALKER, D.M. (1947). A study of the composition of sow's milk. *Br. J. Nutr.* **1**, 64–77

BROOKS, P.H. SMITH, D.A. and IRWIN, V.C.R. (1977). Biotin-supplementation of diets; the incidence of foot lesions and the reproductive performance of sows. *Vet. Rec.* **101**, 46–50

CARPENTER, L.E. (1946/47). *Rep. Hormel Inst. Univ. Minn.* p.19

CLINE, J.H., MAHAN, D.C. and MOXON, A.L. (1974). Progeny effects of supplemental vitamin E in sow diets. *J. Anim. Sci.* **39**, 974 (Abstract)

COX, D.H. and HALE, O.M. (1962). Liver iron depletion without copper loss in swine fed excess zinc. *J. Nutr.* **77**, 225–228

CUNHA, T.J., ADAMS, C.R. and RICHARDSON, C.E. (1968). Observations on biotin needs of the pig. *Feedstuffs, Minneap.* **40**, 22

DEVILAT, J. and SKOKNIC, A. (1971). Feeding high levels of rapeseed meal to pregnant gilts. *Can. J. Anim. Sci.* **51**, 715–719

DIEHL, J.S., MAHAN, D.C. and MOXON, A.L. (1975). Effects of single intramuscular injections of selenium at various levels to young swine. *J. Anim. Sci.* **40**, 844–850

DOISY, E.A. JR. (1974). Effects of deficiency in manganese upon plasma levels and clotting protein and cholesterol in man. In *Trace Element Metabolism in Animals Vol. 2*, (W.G. Hoekstra *et al.*, Eds.), pp. 664–667. Baltimore, Univ. Park Press

DUTT, B. and MILLS, C.F. (1960). Reproductive failure in rats due to copper deficiency. *J. comp. Path. Ther.* **70**, 120–125

DYER, I.A. and KRIDER, J.L. (1950). Choline versus betaine and expeller versus solvent soyabean meal for weanling pigs. *J. Anim. Sci.* **9**, 176–179

EGGERT, R.G., PATTERSON, E., AKERS, W.T. and STOKSTAD, E.L.R. (1957). The role of vitamin E and selenium in the nutrition of the pig. *J. Anim. Sci.* **16**, 1037 (Abstract)

ENSMINGER, M.E., COLBY, R.W. and CUNHA T.J. (1951). Effect of certain B-complex vitamins on gestation and lactation in swine. *Stn Circ. For. Facts agric. Exp. Stn Wash. St. Coll.*, No. 134

EWAN, R.C., WASTELL, M.E., BICKNEL, E.J. and SPEER, V.C. (1969). Performance and deficiency symptoms of young pigs fed diets low in vitamin E and selenium. *J. Anim. Sci.* **29**, 912–915

FORD, J.E. (1974). Some observations on the possible nutritional significance of vitamin B_{12} and folate-binding proteins in milk. *Br. J. Nutr.* **31**, 243–257

FORD, J.E. and SCOTT, K.J. (1975). Uptake of vitamin B_{12} in the piglet. *Rep. natn. Inst. Res. Dairy.* 1973–74, 75

FRAPE, D.L., WAYMAN, B.J. and TUCK, M.G. (1979). The utilisation of phosphorus and nitrogen in wheat offal by growing pigs. *J. agric. Sci., Camb.* **93**, 133–146

FRAPE, D.L., WOLF, K.L., WILKINSON, J. and CHUBB, L.G. (1969). Liver weight and its N and vitamin A contents in piglets from sows fed two levels of protein and food. *J. agric. Sci., Camb.* **73**, 33–40

FREDERICK, G.L. (1962). Practical and physiological studies of the relationship between vitamin B_{12} and reproduction in swine. In *Vitamin B_{12} and Intrinsic Factor: 2. Europaisches Symposium*, (H.C. Heinrich, Ed.), p. 580. Stuttgart, Ferdinand Enke Verlag

FREDERICK, G.L. and BRISSON, G.J. (1961). Some observations on the relationship between vitamin B_{12} and reproduction in swine. *Can. J. Anim. Sci.* **41**, 212–219

GIPP, W.F., POND, W.G., KALLFELZ, F.A., TASKERN, J.B., VAN CAMPEN, D.R. KROOK, L. and VISEK, W.J. (1974). Effect of dietary copper, iron and ascorbic acid levels on hematology, blood and tissue copper, iron and zinc concentrations and ^{64}Cu and ^{59}Fe metabolism in young pigs. *J. Nutr.* **104**, 532–541

GLIENKE, L.R. and EWAN, R.C. (1974). Selenium in the nutrition of the young pig. *J. Anim. Sci.* **39**, 975 (Abstract)

GONDOS, M., PALAMARU, E., HARSIAN, A., HARSIAN, E., NICHITIN, A., MAXIM, V. and NICOLOF, E. (1970). Optimum amounts of vitamin A for pigs. *Lucr. stiint. Inst. Cerc. zooteh.* **27**, 551–564

GREGORY, M.E. and HOLDSWORTH, E.S. (1955). The occurrence of a cyanocobalamin-binding protein in milk and the isolation of a cyanocobalamin–protein complex from sow's milk. *Biochem. J.* **59**, 329–334

GRUMMER, R.H., BENTLEY, O.G., PHILLIPS, P.H. and BOHSTEDT, G. (1950). The role of manganese in growth, reproduction and lactation of swine. *J. Anim. Sci.* **9**, 170–175

HALL, G.A. and HOWELL, J. McC. (1969). The effect of copper deficiency on reproduction in the female rat. *Br. J. Nutr.* **23**, 41–45

HARMON, B.G., MILLER, E.R., HOEFER, J.A., ULREY, D.E. and LUECKE, R.W. (1963). Relationship of specific nutrient deficiencies to antibody production in swine. II. Pantothenic acid, pyridoxine or riboflavin. *J. Nutr.* **79**, 269–275

HART, E.B. and STEENBOCK, H. (1918). Thyroid hyperplasia and the relation of iodine to the hairless pig malady. *J. biol. Chem.* **33**, 313–323

HEDGES, J.D., KORNEGAY, E.T. and THOMAS, H.R. (1976). Comparison of dietary zinc levels for reproducing sows and the effect of dietary zinc and calcium on the subsequent performance of their progeny. *J. Anim. Sci.* **43**, 453–463

HEMINGWAY, R.G., BROWN, N.A. and LUSCOMBE, J. (1974). The effect of iron and copper supplementation of the diet of sows during pregnancy and lactation on the iron and copper status of their piglets. In *Trace Element Metabolism in Animals Vol. 2*, (W.G. Hoekstra *et al.*, Eds.), pp. 601–604. Baltimore, Univ. Park Press

HENNIG, A. (1965). Effect of supplementing mother's ration with Ca and Zn on composition of the piglet and of sow's milk. *Arch. Tierernähr.* **15**, 331–383

HJARDE, W., NEIMANN-SORENSEN, A., PALLUDAN, B. and HAVSKOV SORENSEN, P. (1961). Investigations concerning vitamin A requirement utilisation and deficiency symptoms in pigs. *Acta Agric. scand.* **11**, 13–53

HOEFER, J.A., MILLER, E.R., ULLREY, D.E, RITCHIE, H.D. and LUECKE, R.W. (1960). Interrelationships between calcium, zinc, iron and copper in swine feeding. *J. Anim. Sci.* **19**, 249–259

HOEKSTRA, W.G., FALTIN, E.C., LIN, C.W., ROBERTS, H.F. and GRUMMER, R.H. (1967). Zinc deficiency in reproducing gilts fed a diet high in calcium and its effect on tissue zinc and blood serum alkaline phosphatase. *J. Anim. Sci.* **26**, 1348–1357

HOWELL, J.McC. and HALL, G.A. (1969). Histological observations on foetal resorption in copper deficient rats. *Br. J. Nutr.* **23**, 47–50

HOWELL, J.McC. and HALL, G.A. (1970). Infertility associated with experimental copper deficiency in sheep, guinea-pigs and rats. In *Trace Element Metabolism in Animals Vol. 1*, (C.F. Mills, Ed.), pp. 106–109. Edinburgh, Livingstone

IWARSSON, K. BENGTSSON, G. and EKMAN, L. (1973). Iodine content in colostrum and milk of cows and sows. *Acta vet. scand.* **14**, 254–262

JANCIC, S., PESUT, M., CRNOJEVIC, Z., CRNOJEVIC, T., and COSIC, H. (1970). Effect of large doses of vitamin A and lucerne carotene on reproduction in sows and vitamin A in blood of the sows and blood and liver of their piglets. *Symp. Pig Prod. Anim. Nutr., Zagreb,* pp. 72–89

JOHNSON, S.R. (1943). Studies with swine on rations extremely low in manganese. *J. Anim. Sci.* **2**, 14–22

JUGINA, A.D. (1966). Effect of a mixture of vitamins on the fertility of gilts. *Svinovodstvo* **20**, 30–32

KORNEGAY, E.T. and MEACHAM, T.N. (1973). Evaluation of supplemental choline for reproducing sows housed in total confinement on concrete or in dirt lots. *J. Anim. Sci.* **37**, 506–509

KRALOVANSZKY, U.P., EORI, E. and KALLAI, L. (1954). The influence of vitamin B_{12} on the reproduction of Mangalica sows. *Allattenyesztes* **3**, 331

KU, P.K., MILLER, E.R., WAHLSTROM, R.C. GROCE, A.W. and ULLREY, D.E. (1972). Supplementation of naturally high selenium diets. *J. Anim. Sci.* **35**, 218 (Abstract No. 207)

LEWIS, P.K. Jr., GRUMMER, R.H. and HOEKSTRA, W.G. (1957). The effect of method of feeding upon the susceptibility of the pig for parakeratosis. *J. Anim. Sci.* **16**, 927–936

MAHAN, D.C., MOXON, A.L. and HUBBARD, M.D. (1975). Efficiency of supplemental Se on sow and progeny tissue Se. *J. Anim. Sci.* **41**, 319 (Abstract No. 297)

MAHAN, D.C., MOXON, A.L. and HUBBARD, M.D. (1977). Efficacy of inorganic selenium supplementation to sow diets on resulting carry-over to their progeny. *J. Anim. Sci.* **45**, 738–746

MAHAN, D.C., PENHALE, L.H., CLINE, J.H., MOXON, A.L., FETTER, A.W. and YARRINGTON, J.T. (1974). Efficacy of supplemental selenium in reproductive diets on sow and progeny performance. *J. Anim. Sci.* **39**, 536–543

MICHEL, R.L. (1968). The role of vitamin E, selenium and methionine in dietary liver necrosis and nutritional muscular dystrophy in the pig. *Diss. Abstr.* **28**, 3764B

MILLER, C.O. and ELLIS, N.R. (1951). The riboflavin requirements of growing swine. *J. Anim. Sci.* **10**, 807–812

MILLER, E.R., LUECKE, R.W., ULLREY, D.E., BALTZER, B.V., BRADLEY, B.L. and HOEFER, J.A. (1968). Biochemical, skeletal and allometric changes due to zinc deficiency in baby pigs. *J. Nutr.* **95**, 278–286

MILLER, E.R., HITCHCOCK, J.P., KUAN, K.K., KU, P.K., ULLREY, D.E. and KEAHEY, K.K. (1973). Iron tolerance and E–Se status of young swine. *J. Anim. Sci.* **37**, 287 (Abstract)

MONEY, D.F.L. (1970). Vitamin E and selenium deficiencies and their possible aetiological role in the sudden death in infants syndrome. *N.Z. med. J.* **70**, 32

NATIONAL RESEARCH COUNCIL (1979). *Nutrient Requirements of Domestic Animals, No. 2 Nutrient Requirements of Swine,* 8th Revised Edition. Washington, National Research Council

NIELSEN, H.E. (1971). The influence of nutrition on the reproduction performance of boars and sows. *Svind Symp. Zagreb* **1**,

NIELSEN, H.E., HØJGAARD-OLSEN, N.J., HJARDE, W. and LEERBECK, E. (1973). Vitamin E content in colostrum and sow's milk and sow milk yield at two levels of dietary fat. *Acta Agric. scand., Suppl.* **19**, 35

NORRDIN, R.W., KROOK, L., POND, W.G. and WALKER, E.F. (1973). Experimental zinc deficiency in weanling pigs on high and low calcium diets. *Cornell Vet.* **63**, 264–290

PALLUDAN, B. and WEGGER, I. (1972). Zinc metabolism in pigs III. Placental transfer of zinc in normal and zinc deficient gilts and its influence on fetal development. *Arsberetn. Inst. Sterilitetsforsk.* p.27

PEDERSEN, J.G.A. (1966). Om nedsat tolerance for jern hos grise. *Nord. VetMed.* **18**, 1–18

PETRENKO, G.G. and ZIRNOV, I.I. (1968). Injection of vitamin A for pregnant sows. *Svinovodstvo* **22**, 36 (*Nutr. Abstr. Rev.* **39**, 3985)

PIPER, R.C., FROSETH, J.A., McDOWELL, L.R., KROENING, G.H. and DYER, I.A. (1975). Selenium–Vitamin E deficiency in swine fed peas (*Pisum sativum*). *Am. J. vet. Res.* **36**, 273–281

PLUMLEE, M.P., THRASHER, D.M., BEESON, W.M. ANDREWS, F.N. and PARKER, H.E. (1953). The effect of manganese deficiency on the growth and development of swine. *J. Anim. Sci.* **14**, 996 (Abstr.)

PLUMLEE, M.P., THRASHER, D.M., BEESON, W.M., ANDREWS, F.N. and PARKER, H.E. (1956). The effect of a manganese deficiency upon the growth development and reproduction in swine. *J. Anim. Sci.* **15**, 352–367

POCHERNNYAEVA, G. (1976). Riboflavin in the diet and reproductive capacity of replacement gilts. *Svinovodstvo* **4**, 42–43. (*Nutr. Abstr. Rev.* **47**, 5542)

POND, W.G. and JONES, J.R. (1964). Effect of level of zinc in high calcium diets on pigs from weaning through reproductive cycle and on subsequent growth of their offspring. *J. Anim. Sci.* **23**, 1057–1060

REID, I.M. (1968). Chemical and histologic pathology of protein deficiency alone and complicated by vitamin E deficiency in the young pig. *Diss. Abstr.* **28**, 3962B

REID, I.M., BARNES, R.H., POND, W.G. and KROOK, L. (1968). Methionine-responsive liver damage in young pigs fed a diet low in protein and vitamin E. *J. Nutr.* **95**, 499–508

RITCHIE, H.D., MILLER, E.R., ULLREY, D.E., HOEFER, J.A. and LUECKE, R.W. (1960). Supplementation of the swine gestation diet with pyridoxine. *J. Nutr.* **70**, 491–496

SARYCEVA, M.M. (1968). Effect of vitamin A on reproduction in sows. *Svinovodstvo* **22**, 39 (*Nutr. Abstr. Rev.* **38**, 8313)

SEVKOVIC, N., PUJIN, D., TESARZ, I. and VUKOVIC, S. (1969). Effect of different amounts of vitamin A on reproduction in sows. *Vet. Glasn.* **23**, 21–27 (*Nutr. Abstr. Rev.* **39**, 5962)

SEVKOVIC, N., RAJIC, I., VUJOSEVIC, J. and PETRIC, M. (1973). The effect of vitamin A on embryonic mortality in gilts. *Acta vet. Beogr.* **23**, 197–202 (*Nutr. Abstr. Rev.* **44**, 6875)

SIHOMBING, D.T.H., CROMWELL, G.L. and HAYS, V.W. (1974). Effect of protein source, goitrogens and iodine level on performance and thyroid status of pigs. *J. Anim. Sci.* **39**, 1106–1112

SMITH, W.H., PLUMLEE, M.P. and BEESON, W.M. (1962). Effect of source of protein on zinc requirement of the growing pig. *J. Anim. Sci.* **21**, 399–405

STOCKLAND, W.L. and BLAYLOCK, L.G. (1974). Choline requirement of pregnant sows and gilts under restricted feeding conditions. *J. Anim. Sci.* **39**, 1113–1116

STUMPF, J. (1968). Effect of vitamin A on production of piglets. *Veterinaria, Spofa* **10**, 127–133 (*Nutr. Abstr. Rev.* **39**, 3984)

TEAGUE, H.S. and GRIFO, A.P., Jr. (1966). Vitamin B_{12} supplementation of sow rations. *J. Anim. Sci.* **25**, 895 (Abstract)

TEAGUE, H.S., GRIFO, A.P., Jr. and PALMER, W.M. (1971). Pantothenic acid deficiency in the sow. *J. Anim. Sci.* **33**, 239 (Abstract)

TEAGUE, H.S., PALMER, W.M. and GRIFO, A.P., Jr. (1970). Pantothenic acid deficiency in the reproducing sow. *Ohio agric. Res. Dev. Center Anim. Sci.* Mimeo No. 200, pp. 1–5

THOMAS, J.W., LOOSLI, J.K. and WILLMAN, J.P. (1947). Placental and mammary transfer of vitamin A in swine and goats as affected by the prepartum diet. *J. Anim. Sci.* **6**, 141–145

TOLLERZ, G. (1973). Vitamin, selenium (and some related compounds) and iron-tolerance in piglets. *Acta Agric. scand., Suppl.* **19**, 184

TOLLERZ, G. and LANNEK, N. (1964). Protection against iron toxicity in vitamin E deficient piglets and mice by vitamin E and synthetic antioxidants. *Nature, Lond.* **201**, 846–847

VAN VLEET, J.F., MEYER, K.B. and OLANDER, H.J. (1973). Control of selenium–vitamin E deficiency in growing swine by parenteral administration of selenium–vitamin E preparations to baby pigs or to pregnant sows and their baby pigs. *J. Am. vet. med Ass.* **163**, 452–456

VAN VLEET, J.F., MEYER, K.B. AND OLANDER, H.J. (1974). Acute selenium toxicosis induced in baby pigs by parenteral administration of selenium–vitamin E preparations. *J. Am. vet. med. Ass.* **165**, 543–547

VARGANOV, A.I. (1965). Fertility of pigs in conditions of I deficiency. *Svinovodstvo* 4, 31–32 (*Nutr. Abstr. Rev.* **35**, 7109)

WASTELL, M.E., EWAN, R.C., VORHIES, M.W. and SPEER, V.C. (1972). Vitamin E and selenium for growing and finishing pigs. *J. Anim. Sci.* **34**, 969–973

WHITING, F., LOOSLI, J.K. and WILLMAN, J.P. (1949). The influence of tocopherols upon the mammary and placental transfer of vitamin A in the sheep, goat and pig. *J. Anim. Sci.* **8**, 35–40

WÖHLBIER, W. and SIEGEL, A. (1967). Vitamin B_6 supply of sucking pigs. *Arch. Tierernähr.* **17**, 257–262