

Chapter 6

Essential-fatty acid status of piglets at weaning in relation to post-weaning health: a brief review

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Abstract

From the outset, it is assumed that a sufficient status of polyunsaturated fatty acids (PUFAs) in combination with a high ratio of n-3:n-6 PUFAs will be beneficial for piglets so as to coping with the multiple stressors at weaning. The fatty acid status at weaning is essentially determined by the fatty acid composition of the sow's milk which is in turn determined by the fatty acid composition of fat mobilized by the sow and that of the lactation diet.

Introduction

In current swine production, piglets are weaned at the age of 21-28 days. The weaning transition is associated with abrupt changes in social, physical, microbial and chemical environment, including the diet. For a period of a few days after weaning, feed intake is below maintenance requirement (Pluske et al., 1996) which causes atrophy of small intestinal villi and oedema disease (Nabuurs, 1998). The low feed intake after weaning causes a low intake of polyunsaturated fatty acids (PUFAs), including the essential fatty acids, linoleic acid (LA, C18:2 n-6) and α -linolenic acid (ALA, C18:3 n-3). LA and ALA are the parent compounds of the so-called n-6 and n-3 families of PUFAs, respectively. The two essential fatty acids and their metabolic products of desaturation and elongation are important structural components of membranes, influencing membrane fluidity, receptor function and enzyme activity (Burns et al., 1979). In addition, the metabolites arachidonic acid (AA, C20:4 n-6) and eicosapentaenoic acid (EPA, C20:5 n-3) are the precursors of eicosanoids affecting a variety of biological functions, including immunity (Wu and Meydani, 1998; Fritsche et al., 1993a). There is evidence that dietary n-3 PUFAs may antagonize atrophy of villi and have anti-inflammatory activity. In growing chicks, the intake of extra n-3 PUFAs has been shown to improve performance and decrease the inflammatory response to LPS from *S. typhimurium* and *S. aureus* (Korver and Klasing, 1997). In young mice with hypoxia-induced bowel necrosis, supplementation of the diet with n-3 PUFAs reduced the degree of necrosis (Akisu et al., 1998). Mucosal damage in food-sensitive enteropathy in mice was prevented by fortification of the diet with n-3 PUFAs (Ohtsuka et al., 1997).

We have addressed the question whether the fatty acid composition of the diet of weanling piglets affects their villus:crypt ratio of small intestinal mucosa and growth performance. Weanling piglets were given access to diets containing different amounts of LA and ALA, in the form of corn oil and linseed oil, so that the n-3:n-6 ratio of dietary PUFAs ranged between 0.03 and 0.32. Corn oil contains about 55% LA whereas linseed oil contains 55% ALA. The different n-3:n-6 ratios did not affect growth performance (Chapter 2). Fish oil may contain 20% EPA plus docosahexaenoic acid (DHA, C22:6 n-3), and it was hypothesized (Chapter 3) that fish-oil feeding would be more effective than linseed-oil feeding because weanling piglets might have low capacity to convert ALA into EPA (Bee, 2000). However, there was no consistent effect of fish oil in the diet on either the villus:crypt ratio of small intestinal mucosa or growth performance (Chapters 3 and 4).

Just before weaning at the age of about 21 days, piglets consume about 1 kg of milk/day (Taugbol et al., 1993). We have calculated that an intake of 300 g weaner diet/day provides a similar amount of n-3 and n-6 PUFAs as does an intake of 1 kg of sow milk (Chapter 1). Clearly, the outcome of our calculations is

dependent on the fatty acid composition and fat content of both the weaner diet and sow milk. It takes about 12 days after weaning before piglets reach an intake of 300 g of dry feed (Bruininx et al., 2001). The period of low PUFA intake may not affect fatty acid status of the piglets. Indeed, we have shown that fatty acid status of weanling piglets, as represented by the fatty acid composition of erythrocytes, subcutaneous adipose tissue and lymph nodular fat, does not alter within 7 days (Chapter 1). Thus, it is likely that the observed (Chapters 3 and 4) lack of effect of fatty acid composition of the weaner diet on villus:crypt ratio of small intestinal mucosa and growth performance of weanling piglets is explained by sufficient body stores of n-6 and n-3 PUFAs at the time of weaning. If PUFAs indeed influence gut integrity, then the status of essential fatty acids of piglets at the time of weaning may determine their susceptibility to post-weaning disorders.

The fatty acid status of suckling piglets is determined by the fatty acid content of sow milk. In the case that the piglets have access to creep feed, the intake of creep feed will be too low (Bruininx et al., 2002) to influence fatty acid status. New-born piglets weighing 1.5 kg have a body-fat content of only 1%, whereas weanling piglets weighing about 8 kg have a fat content of 5% (Murry et al., 1999). It follows that the fatty acid status of new-born piglets, which is related to the sow diet fed during gestation (Rooke et al., 1999; Rooke et al., 1998; Farnworth and Kramer, 1989; Arbuckle and Innis, 1993), has no impact on the fatty acid composition of the piglets' carcass at weaning. This paper summarizes the literature data on the influence of the fatty acid composition of the lactation diet on the fatty acid status of piglets at weaning. It is assumed that a sufficient status of PUFAs in combination with a high n-3:n-6 ratio in erythrocyte membrane PUFAs will be beneficial so as to cope with the multiple stressors at weaning. Prior to describing the relation between the fatty acid composition of the lactation diet and fatty acid status of the weanling piglet, the conditions of deficient and optimum fatty acid status are discussed.

Essential-fatty acid deficiency

In order to evaluate the fatty acid status of weanling piglets, as mirrored by the fatty acid composition of selected blood components or tissues, it is helpful to know when the condition of deficiency occurs. Unfortunately, there is limited information on essential-fatty acid deficiency in suckling and weanling piglets. To study the effects of deficiency, suckling piglets have to be fed on artificial diets, because, obviously, sow milk is not deficient in essential fatty acids. Overt deficiency is associated with signs such as impaired growth. Daveloose et al. (1993) fed 10-day old piglets semipurified diets containing 7% fat either high or low in LA and ALA. Table 1 shows the fatty acid compositions of the two diets. The diets were fed for 6 weeks. Weight gain of piglets fed the high-PUFA diet was

Table 1. Fatty acid composition of the diet and that of plasma phospholipids in piglets fed either sufficient or deficient in LA

Fatty acids	Diet	
	Sufficient	Deficient
	% of total fatty acids	
LA	50.9	4.5
ALA	0.6	0.0
	Plasma phospholipids	
	Sufficient	Deficient
LA	16.0	6.9
ALA	0.2	0.1
AA	11.0	5.4
EPA	1.6	1.0
DHA	4.1	0.8
EA	0.3	4.3

Based on Daveloose et al. (1993)

236 ± 15 g/day (mean ± SEM, n=6) and for those fed the low-PUFA diet it was 137 ± 15 g/day. Thus, the low-PUFA diet can indeed be considered deficient. Table 1 documents the fatty acid composition of the PUFA-deficient piglets. It is clear that PUFA deficiency is associated with various changes in the fatty acid profile of plasma phospholipids. PUFA deficiency elicited an increase in the percentages of oleic acid (OA, C18:1 n-9) and eicosatrienoic acid (EA, C20:3 n-9). As would be expected, the percentages of LA, AA, ALA, EPA and DHA were lowered after feeding the PUFA-deficient diet (Table 1). The study of Daveloose et al. (1993) illustrates that PUFA deficiency impairs growth of piglets and alters their fatty acid status. It should be stressed that the feeding trial started when the piglets were aged 10 days and ended when they were older than 7 weeks. In the context of this paper the main interest is in new-born piglets fed variable amounts of PUFAs until the age of about 3 weeks.

LA and ALA levels in tissues vary with LA and ALA intakes and therefore the levels by themselves do not readily indicate the status of deficiency. In other words, the tissue levels can be considered deficient only when they cause metabolic aberrations. It is generally accepted that PUFA deficiency in various animal species causes an increase in the levels of EA. The study described above showed a similar effect (Table 1). The increase in n-9 fatty acids with a

simultaneous decrease in n-6 and n-3 PUFAs may maintain membrane fluidity and thereby membrane-dependent functions (Beynen et al., 1984). Thus, it is reasonable to use the tissue level of OA and EA acid as an indicator of essential-fatty acid deficiency, which may even point at a deficient intake earlier than does impaired growth. The implication would be that with varying intakes of PUFA, essential-fatty acid deficiency is about to develop when OA and EA begin to increase. The condition of deficiency may then be defined as the tissue level of LA and/or ALA at which OA and EA is about to increase. In three studies (Goustard-Langelier et al. 1999; Huang and Craig-Schmidt 1996; Alessandri et al. 1996) new-born piglets have been fed diets with different fatty acid compositions. However, the results of these studies do not provide information as to conditions that elicit essential-fatty acid deficiency.

Optimum n-3:n-6 ratio in tissues

Clearly, a condition of essential-fatty acid deficiency in weaning piglets does not readily occur under practical conditions. An important issue is to define the fatty acid status that provides optimum disease resistance. Fritsche (1993 a) fed sows a diet containing either 7% menhaden fish oil or lard from day 107 of gestation until farrowing and studied the effect on immune cell fatty acid composition and eicosanoid production in the nursed piglets. The fatty acid profile of total diets and sow milk were not given. Fatty acid compositions of serum, liver, thymus, splenocytes and alveolar macrophages in weaning piglets were affected by the sows diet. The sow's diet influenced prostaglandin production by alveolar macrophages isolated from weaning piglets, but it cannot be concluded which effect should be considered advantageous.

Fatty acid status of piglets at weaning

Rooke (1998) fed sows diets with either soyabean oil or tuna oil (30 g/kg diet) for the last three weeks of gestation. Shortly after birth piglets from tuna-oil-fed sows had a higher amount of n-3 polyunsaturated fatty acids in their tissues compared to the soyabean-oil fed other group. Feeding sows with different oil supplements for the last three weeks of pregnancy did not cause differences in litter size or piglet weight. However, the piglets from the tuna-oil-fed sows had a lower viability score, being based on heart rate, onset of breathing and first attempt to stand. This was probably due to the induced farrowing on day 113-114, because intake of n-3 fatty acids may prolong gestation time, so the sows fed soyabean oil were closer to their natural farrowing time. The sows diet influenced plasma fatty acids and the fatty acid composition of the milk (Table 2).

Fritsche (1993 b) fed sows from day 107 of gestation experimental diets in which fish oil was substituted for lard at 0, 3.5 or 7% of the diet. The fatty acid

Table 2. Fatty acid composition of the diet and that of milk fat and of plasma of piglets

Fatty acid (% FAME ¹)	Diet	
	Soyabean oil	Tuna oil
LA	51.1	2.2
ALA	7.2	3.0
EPA	0.2	3.9
DHA	0.4	17.6
	Plasma	
	Soyabean oil	Tuna oil
LA	12.9	10.0
ALA	ND ²	ND
EPA	1.3	5.2
DHA	3.6	7.7
	Milk fat	
	Soyabean oil	Tuna oil
LA	25.9	14.4
ALA	2.9	1.6
EPA	0.6	1.7
DHA	2.1	10.1

Based on Rooke et al. (1998)

¹ FAME = Fatty Acid MethylEsters

² ND = not detectable

composition of milk fat was influenced by the diet. The fatty acid composition of the experimental diets was not given, but the fatty acid composition of milk fat and that of total serum of piglets differed significantly. The suckling piglets had an increase in serum n-3 levels within 24 hours after birth when the mothers were fed 3.5 or 7% fish oil. This increase lasted throughout the lactation period (Table 3). The increasing amount of fish oil at the expense of lard from day 107 from gestation did not result in differences among the number of live piglets born per litter, their birth and weaning weights. In conclusion, manipulation of fatty acid supply in the diet of the sow from day 107 of gestation up to weaning can modify the fatty acid composition of the milk fat and the piglets fatty acid composition of plasma or serum.

Table 3. Fatty acid composition of milk fat and fat of serum of piglets (mean value over total lactation period)

Fatty acid (% FAME) ¹	Milk fat		
	7% Lard	3.5% Lard 3.5% Fish oil	7% Fish oil
LA	13.1	12.6	11.3
ALA	0.6	0.8	0.9
EPA	0.5	2.3	3.3
DHA	0.6	2.4	3.5
	Serum		
	7% Lard	3.5% Lard 3.5% Fish oil	7% Fish oil
LA	23.0	19.1	17.1
ALA	0.5	0.5	0.5
EPA	0.3	7.6	12.4
DHA	1.2	4.6	5.6

Based on Fritsche et al (1993b)

¹ FAME = Fatty Acid MethylEsters

Conclusion

The major determinant of the fatty acid status of the weanling piglet is the fatty acid composition of the pregnancy and lactation diet fed to the sow. There is insufficient data to suggest the optimum composition of these diets.

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