Scope of the thesis

Introduction

This thesis describes research on the essential-fatty acid supply of weanling piglets. Prior to outlining the scope of the thesis, the polyunsaturated fatty acids and their functions are briefly summarised.

Nomenclature of polyunsaturated fatty acids

Table 1 gives the names and shorthand notations of selected polyunsaturated fatty acids.

Table 1. Names, shorthand notation and abbreviations of selected polyunsaturated fatty acids

Fatty acid	Shorthand notation	Abbreviation
Linoleic acid	C18:2 n-6	LA
α-Linolenic acid	C18:3 n-3	ALA
γ-Linolenic acid	C18:3 n-6	GLA
Dihomo- γ-linolenic acid	C20:3 n-6	DGLA
Arachidonic acid	C20:4 n-6	AA
Eicosapentaenoic acid	C20:5 n-3	EPA
Docosahexaenoic acid	C22:6 n-3	DHA

Polyunsaturated fatty acids (PUFAs) are classified by the length of the carbon chain, the number of double bounds and the location of the first double bound. Holman (1964) introduced the 'omega' (ω or n) nomenclature for the identification of PUFAs. The omega number refers to the position of the first double bound as counted from the methyl end of the carbon chain. For example, linoleic acid, in short hand notation C18:2 n-6, has 18 carbons and 2 double bounds with the first double bound at the sixth carbon atom from the methyl end of the chain. All metabolic conversions in vertebrates, i.e. desaturation and elongation, occur beyond the ninth carbon atom from the methyl end of the chain. Therefore, the omega nomenclature classifies PUFAs into two families with fixed structure at the methyl end of the molecule. Vertebrates lack the enzymes to introduce double bounds within the first 9 carbons from the methyl end, and thus require dietary sources of the essential fatty acids linoleic acid (LA, C18:2 n-6) and α-linolenic acid (ALA, C18:3 n-3) which are considered the parent compounds of the n-6 and n-3 families of PUFAs, respectively. The parent fatty acids can be elongated and desaturated to other PUFAs, like arachidonic acid (AA, C20:4 n-6) or

eicosapentaenoic acid (EPA, C20:5 n-3) (Fig. 1). However, the two parent fatty acids and their metabolites compete with each other for the desaturase and elongase enzymes, which generally have more affinity for n-3 than for n-6 PUFAs.

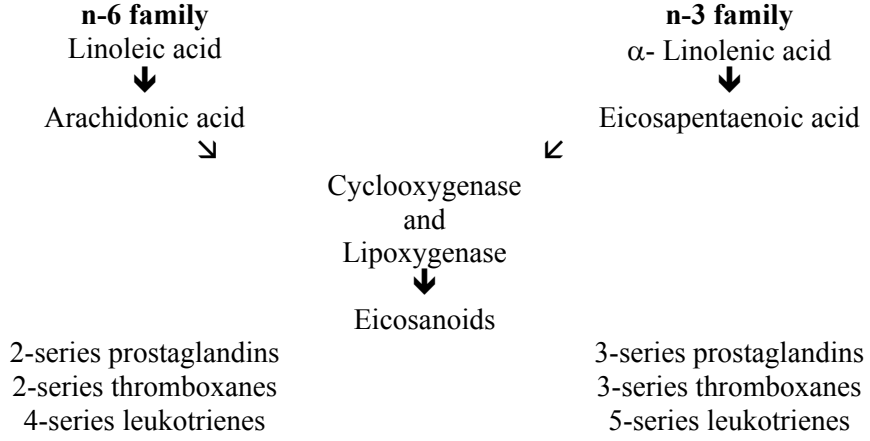

Fig 1. Simplified scheme of fatty acid metabolism and the production of eicosanoids (prostaglandins, thromboxanes and leukotrienes).

Functions of polyunsaturated fatty acids

Essential fatty acids have two main functions. First, PUFAs, especially AA, are structural components of cellular membranes. In retina and brain, EPA and docosahexaenoic acid (DHA, C22:6 n-3) are essential structural components. These PUFAs render to the cell membranes their fluid nature. Without the availability of PUFAs, membranes will incorporate more saturated fatty acids, resulting in less fluid and instable membranes. As a result, the tissue permeability increases and leads to nutrient and water loss, change of receptor function, enzyme activity and altered cytokine production (Wan et al., 1988). Secondly, PUFAs play an important role in the immune response. The fatty acids with 20 carbons, AA and EPA are precursors for the eicosanoids, i.e. the prostaglandins, thromboxanes and leukotrienes (Fig. 1). Eicosanoids affect processes such as immunity, platelet aggregation and vasoconstriction. In general, the eicosanoids derived from the n-6 PUFAs have effects opposite to those derived from the n-3 PUFAs. Due to the competition between n-3 and n-6 PUFAs for the desaturases and elongages, the net effects of the eicosanoids depend on the amounts and on the ratio of n-3 and n-6 PUFAs present in the diet.

Scope of the thesis

Weanling piglets are prone to the development of the so-called post-weaning syndrome which is associated with atrophy of the villi, inflammation of the gut (Cera et al., 1988; Hall and Byrne, 1989; Hampson, 1986; Kenworthy, 1976; Nabuurs, 1991) and depressed performance (Jahn and Uecker, 1987; Svedsen et al., 1974; Svensmark et al., 1989). 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 with n-3 PUFAs reduced the degree of necrosis (Akisu et al., 1998). Mucosal damage in food-sensitive enteropathy in mice was prevented by supplementation of the diet with n-3 PUFAs (Ohtsuka et al., 1997).

The general scope of the research in the present thesis was to investigate the influence of n-3 and n-6 PUFAs on performance and health of weanling piglets. It was anticipated that the information thus obtained would provide clues as to the ideal fatty acid composition of the diet for weanling piglets. The specific objectives were as follows:

1. To gain information as to the fatty acid supply and status of piglets from birth to two weeks after weaning.
2. To investigate the effect of supplemental n-3 PUFAs and the n-3:n-6 ratio on small intestinal morphology and growth performance.
3. To study whether the dietary fatty acid composition influences the response to a challenge with *Escherichia coli*.
4. To describe the determinants of essential-fatty acid status of piglets at weaning.
5. To put into perspective the effects of dietary fatty acid composition and the intake of dietary dry matter.

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