AN ASSESSMENT OF THE EFFECTS OF DIETARY OIL SUPPLEMENTATION ON FETAL SURVIVAL IN GILTS AT 40 DAYS OF GESTATION

by

Alberto Pérez Rigau

Thesis submitted to the Faculty of the

Virginia Polytechnic Institute and State University

in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

in

Animal Science
(Animal Nutrition)

APPROVED:

E. T. Kornegay, Co-chairman

M. D. Lindemann, Co-chairman

K. E. Webb Jr.

June, 1993

Blacksburg, Virginia

AN ASSESSMENT OF THE EFFECTS OF DIETARY OIL SUPPLEMENTATION ON FETAL SURVIVAL IN GILTS AT 40 DAYS OF GESTATION

by

Alberto Pérez Rigau

Committee Co-chairmen: E. T. Kornegay and M. D. Lindemann
Animal Science

(ABSTRACT)

Eighty-six crossbred (Duroc x Yorkshire) gilts were used in two trials (50 gilts in Trial 1 and 36 gilts in Trial 2) for an assessment of the effect of supplemental dietary fat during early gestation on fetal survival, fetal development, and fatty acid concentration in gilt plasma and fetal head and body. Three diets contained 4% (w/w) added fat either as coconut, soybean, or fish (menhaden) oils. A fourth diet was used as a control. On d 37 to 45 postbreeding, gilts were slaughtered and numerous fetal and ovarian measurements made. Two sets of four randomly selected fetuses per gilt from Trial 1 were prepared. Blood samples from each gilt were obtained on the day of slaughter for determination of the plasma fatty acid profile. Across both trials, percentage fetal survival did not differ according to treatment, but in Trial 2 fetal survival was higher ($P < .06$) for gilts fed fish oil, compared with the controls. The fatty acid profile of plasma of gilts and the conceptus tissues were similiar; both were influenced by

the fatty acid concentration of the diets. The ratio of n-3/n-6 fatty acids was higher in conceptus tissue than in maternal plasma and the ratio was higher ($P < .05$) for the fish oil diet compared with the other diets. The relatively high concentration of omega-3 fatty acids in fetal tissues supports the hypothesis that omega-3 fatty acids play a role in the development of the pig conceptus and contributes to improve fetal survival. However, the high percentage fetal survival observed in all the treatments may have masked benefits of supplemental oil.

Key Words: Fatty Acids, Pigs, Reproduction, Polyenoic Acids, Embryo Mortality.

ACKNOWLEDGEMENTS

First of all I wish to express my most sincere gratitude to Dr. E. T. Kornegay for giving me the opportunity to make this big step forward in my professional career and for many helpful suggestions, discussions, and comments that have helped to complete this thesis. I would like to extend my deepest appreciation to Dr. Merlin Lindemann who created the idea for the research and assisted in the development of this project day after day and for his encouragement, optimism and friendship. I also thank Dr. K. E. Webb, Jr. for serving on my committee.

I am also very pleased to have had the friendship and invaluable expertise of Dr. V. Ravindran. I thank him for his tireless patience and helpfulness in writing this thesis. I will miss him very much.

To my fellow graduate students and staff, especially to Lisa Flory for her patience and her sympathy when I was having a "fight" with the lab equipment. I express my appreciation to Anne Dudley for her assistance in the statistical analyses, and to Joan Sabaté and Eduardo Romano for their sensible advice on metaphysical problems and other flowers, and to the Spanish students, well?...thank you!.

A special thanks to my friend Lazar Boskov for being always there for me to help when things were not going right especially during the first year.

My deep appreciation to Dr. A. Puchal, animal nutrition professor in the Veterinary College of Barcelona for encouraging and helping me to get in this "American adventure".

Finally, I thank my parents and in laws for their love, support and their help without which my graduate studies would have never been completed. Specially, I thank my father for believing in me with this latest endeavor.

The task of working on this thesis though stimulating was made much easier by the love and support of my wife Patricia, to her I dedicate this thesis.

TABLE OF CONTENTS

Appendices

LIST OF TABLES

Table **Page**

Text Tables

Appendix Tables

LIST OF FIGURES

Chapter I

INTRODUCTION

The need for greater efficiency in swine production has led to extensive research on factors influencing litter size and prenatal mortality. Prenatal mortality is divided into embryonic mortality (before d 30) and fetal mortality (after d 30) (Lambert et al., 1991). Life-time sow performance records show that low litter size is a major source of loss to the swine industry. Litter size is determined by ovulation rate, fertilization rate and embryonic and fetal mortality. Day (1979) observed that fertilization rate approaches 100% but approximately 40% of fertilized ova and fetuses were reabsorbed or were aborted during pregnancy in gilts and in sows. Pope and First (1985) pointed out that a major portion of embryonic loss occurs during the second and the fourth week of gestation thus, embryo survival during this period is a major factor in determining litter size at farrowing. The specific causes of porcine embryo deaths are still unclear.

The maternal diet must supply the nutrients needed to meet the additional demands for uterine, placental, and fetal growth. An area of nutrition that has seen little research attention in swine, but one that may have importance, is the specific fatty acid needs of the developing embryo/fetus. Quantitatively, lipid is the most important component of mammalian brain. Brain tissue is extremely rich in polyunsaturated fatty acids (PUFA), with a great predominance of docosahexaenoic acid (Century et al., 1963;

O'Brien et al., 1964; Crawford et al., 1976). In the rat and several species more than 50% of the PUFA have been found to accumulate in the central nervous system during the perinatal period of their development, primarily in the last third of pregnancy and during the first weeks of life (Sinclair and Crawford, 1972). Studies using the developing rat suggested that maternal dietary PUFA during gestation were potential sources of PUFA in the fetal brain (Sinclair and Crawford, 1972; Sinclair, 1975; Hassam and Crawford, 1976). Furthermore, there is indirect evidence that the fertilized egg and developing embryo may have a requirement for one of the omega-3 fatty acids, such as linolenic acid (Naughton, 1981; Samulski and Walker, 1982). To meet a specific need for linolenic acid may require greater attention to dietary formulation and this need probably cannot be met by a simple grain-soybean meal diet. Some types of fish are particularly rich in omega-3 fatty acids and in the longer chain omega-3 fatty acids, eicosapentaenoic, docosahexaenoic acids (Stansby, 1990) but the use of fish oil in animal feedstuffs is limited because of its cost and the potential off-flavor problems they cause in meat. Available sources of linolenic acid in the United States are oils of vegetable origin such as soybean oil, canola oil and linseed oil.

The objectives of the present study were to determine if soybean oil and fish (menhaden) oil supplementation would enhance embryo survival in pregnant gilts and to demonstrate that the response, if observed, was related to their linolenic acid content.

Chapter II

REVIEW OF LITERATURE

Lipid Metabolism in the Pig

There are three major forms of fat found in the diet of mammals. These are: 1) glycerides, principally triglycerides (TG), the form in which fat is stored for energy, 2) phospholipids, and 3) steroids, principally cholesterol. Triglycerides account for 95 to 98% of the fats ingested in all forms of food and for a similar percentage of the fat in the animal (Mead et al.,1986). The phospholipids and cholesterol are present mainly as constituents of cell membranes and myelin sheaths of the nerves. Cholesterol is not found in foods of plant origin (Krausser and Mahan, 1979). The structure of lipids is characterized by a relative lack of oxygen. Fats consist almost exclusively of carbon and hydrogen (Krausser and Mahan, 1979), a condition that makes them hydrophobic and mostly immiscible with water, and also makes them richer in calories since they are in a more reduced form (Stryer, 1988).

During the last three decades, there has been an increased health awareness of the undesirability of excessive dietary intakes of lipids. Epidemiological evidence linking high-fat diets, particularly diets rich in saturated fat and cholesterol, with a higher incidence of cardiovascular diseases and cancer has promoted interest in studying the metabolism of fat in

mammalian animals. In general terms, the pathways of fatty acid biosynthesis are qualitatively similar in the liver and adipose tissue of monogastric, ruminant, and avian species (Scholssman and Bell, 1976). There are, however, quantitative differences between species and these have been discussed by several authors (Fiennes et al., 1973; Pearce, 1983). Swine diets, in most production systems, are generally based on feedstuffs derived from plants. These diets normally contain a small amount of fat (2 to 4%); diets that are supplemented with fat may contain 10 to 15% total fat. Higher levels of fat will significantly reduce the intake of pigs (Stahly, 1984). The present review summarizes some relevant information on the anabolic and catabolic metabolism of lipids in swine.

Biosynthesis of Fatty Acids

Numerous reviews are available regarding mammalian fatty acid biosynthesis. Anabolic activities provide fatty acids for the synthesis of TG, the major adipose form of storage, and component of lipoproteins.

Metabolic Pathways. In swine, the incorporation of glucose and other carbon sources into lipids of the adipose tissue proceeds through the typical mammalian pathways for fatty acid biosynthesis (Figure 1). There is evidence that glucose carbons flux through the pentose-phosphate pathway in swine adipose tissue. This has been reported by several researchers by measuring activities of glucose 6-phosphate and

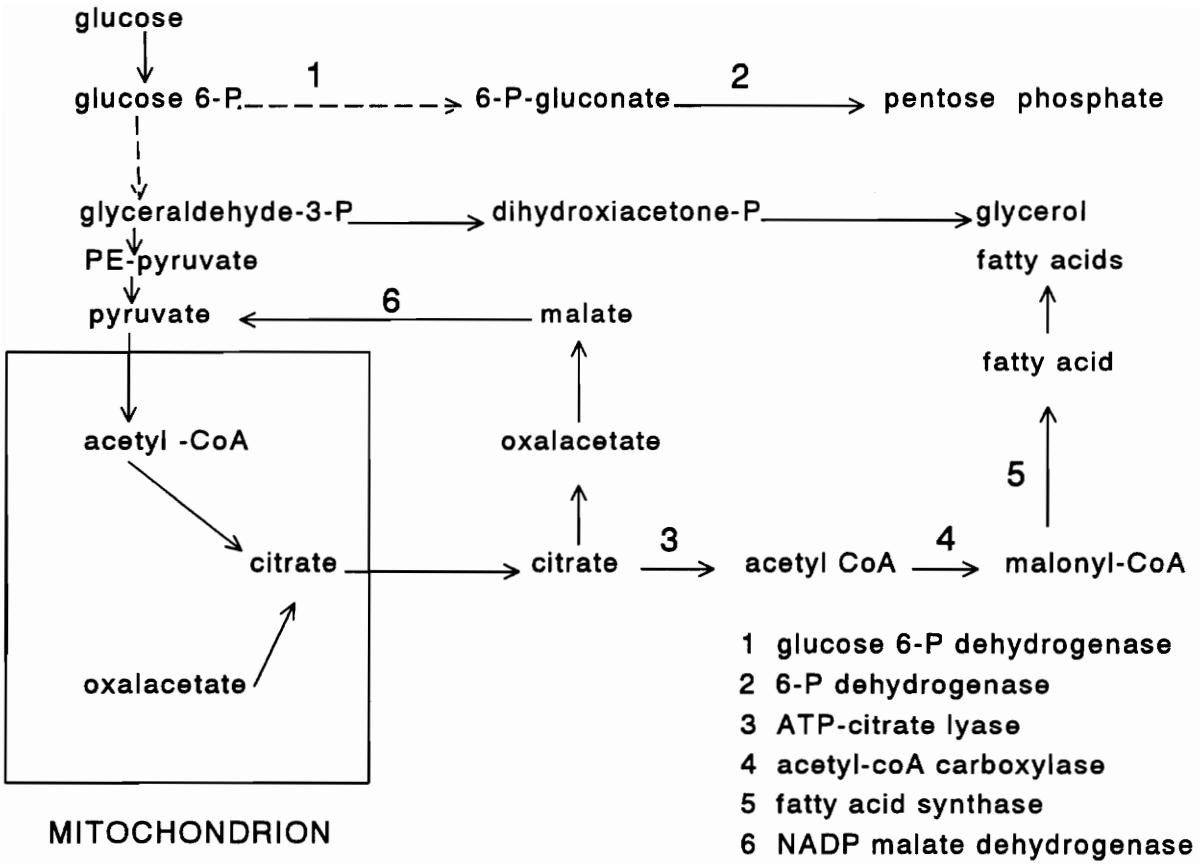


Figure 1. Synthesis of fatty acids. (Adapted from Hahn, 1972).

6-phosphogluconate dehydrogenase coupled with the incorporation of glucose-1-^{14}C into $^{14}CO_2$ (O'Hea and Leveille, 1969; Anderson et al., 1972; Anderson and Kauffman, 1973). Glucose utilization by this pathway provides reducing equivalents for fatty acid synthesis, but the extent of this contribution has not been quantified (Anderson and Kauffman, 1973; Mersmann et al., 1976). Supporting evidence that glucose also follows the glycolysis pathway was provided by studies of carbon incorporation into glyceride-glycerol and glyceride fatty acids. The key glycolytic enzyme, pyruvate kinase, has been measured in swine adipose tissue (Martin and Herbein, 1976). The entry of pyruvate into the mitochondria, the carboxylic acid cycle, and the exit of citrate from the mitochondria in swine adipose tissue have not been studied. However, it is assumed that glucose carbon proceeds via these pathways because citrate and lactate are incorporated into fatty acids, and hydroxycitrate, an inhibitor of ATP-cytrate lyase, inhibits fatty acid biosynthesis from glucose . There is evidence of the activities of ATP-cytrate lyase, acetyl-CoA carboxylase, NADP-malic deshydrogenase, NADP-isocitrate deshydrogenase, and the complex enzyme fatty acid synthase in swine adipose tissue (Mersmann et al., 1976). However, more research is needed regarding the quantification of metabolic pathways in swine adipose tissue to provide information on the movement of carbon from glucose and other substrates to fatty acids and complex lipids.

Tissue Sites. In many species, the liver is a major locus for synthesis of fatty acids. In swine, glucose, acetate, pyruvate, and lactate carbon are incorporated in vitro into liver lipids and fatty acids; however, the rates of incorporation are quite low relative to those found in adipose tissue (O'Hea and Leveille, 1969; Mersmann et al, 1973a). Martin and Herbein (1976) and Kasser et al. (1981), reported that all enzyme activities associated with fatty acid biosynthesis in adipose tissue are observed in the liver of swine. Glucose carbon is incorporated in vitro into swine hepatic fatty acids, but at low rates. Acetic acid but not pyruvate carbon is incorporated at rates greater than for glucose carbon. However, Dutler et al. (1971), proposed that the carbon incorporated from glucose into swine hepatic fatty acids represents fatty acid chain elongation activity rather than de novo synthesis.

Pearce (1983) reviewed the differences between species in regard to the relative contributions of adipose tissue and liver to de novo fatty acid biosynthesis. He reported that rats and mice synthesize fatty acids in both adipose and liver tissues at considerable rates with a tendency to shift more to the adipose tissue at older ages. In humans and chickens, fatty acid synthesis is predominantly in the liver with low activity in adipose tissue. Vernon (1980) gave evidence that ruminants have little hepatic fatty acid synthesis activity at any age. Only the birds have majority of fatty acid synthesis in the liver (Pearce,

1983). In addition to liver, several other swine tissues incorporate glucose or acetate carbon in its lipids at detectable, but low, levels. Intestinal mucosa incorporates acetate and, to a small extent, glucose into lipids (Huang and Kummerow, 1976). Acetate, and to a lesser extent, glucose are incorporated *in vitro* into swine skeletal muscle lipids (Yanovich et al., 1982).

Glucose and acetate are readily incorporated *in vitro* into fatty acids by the mammary gland in sows (Bauman et al., 1970). Swine cardiac muscle only minimally incorporates acetate into lipids *in vitro* (Huang and Kummerow, 1976). Cholesterol synthesis in swine liver and adipose tissues, but not heart and aorta tissues, are important for *in vitro* cholesterol synthesis from acetate (Huang and Kummerow, 1976). Huang and Kummerow (1976) also reported that the incorporation of glucose into cholesterol was minimal in liver but occurred in larger amounts in adipose tissue.

When animals are fed high-carbohydrate diets and/or when the ratio of insulin to glucagon in the blood is high, the biosynthesis of fatty acids is increased. Glucose is considered the major carbohydrate precursor for the *de novo* fatty acid biosynthesis in nonruminant mammals. Glucose carbon is metabolized via glycolysis to pyruvate, enters the mitochondria, traverses the initial steps of the tricarboxylic acid cycle to citrate, exits the mitochondria, is cleaved to acetyl-CoA by ATP-citrate lyase, is carboxylated to malonyl-CoA by acetyl-CoA

carboxylase, and then is polymerized to a long-chain fatty acid (Figure 1). Most of the carbon destined for fatty acid synthesis flows through the pyruvate pool. Glucose also provides most of the glyceride-glycerol for TG synthesis as a branch from the glycolytic pathway. The reducing equivalents for fatty acid biosynthesis are provided from a combination of the pentose phosphate pathway dehydrogenases, NADP-malate dehydrogenase, and cytoplasm NADP-Isocitrate dehydrogenase.

It is well established that swine adipose tissue in vitro converts glucose to fatty acids and to glyceride-glycerol moiety of complex lipids (O'Hea and Leveille, 1969; Christensen and Goel, 1972; Mersmann et al., 1973b ; Steele et al, 1974). More than 70% of the glucose carbon is in glyceride fatty acids in growing pigs, but at younger ages especially before weaning, much less glucose carbon is in glyceride fatty acids (O'Hea and Leveille, 1969; Steele et al., 1974). In vitro studies have shown that glucose is readily incorporated to adipose tissue in swine (O'Hea and Leveille, 1969) and is considered to be a major precursor for lipid synthesis in this species.

Carbon precursors, other than glucose, such as acetate, lactate or citrate are incorporated into lipids in vitro and may be important physiological substrates when swine are subjected to particular nutritional or enviromental circumstances. O'Hea and Leveille (1969) reported that acetate is readily incorporated in swine adipose tissue fatty acids suggesting that it is transported into cells and converted to acetyl-CoA. However,

Imoto and Namioka (1983) reported that the physiological significance of acetate as a lipid precursor in pigs is unclear, because in vitro studies use about 10 mM acetate whereas circulating concentrations are less than .2 mM and approach 1 mM only when pigs are fed 10% of the metabolizable energy as acetate. Other potential precursors such as leucine and glycerol (Martin and Herbein, 1976) or aspartate (Mersmann et al., 1976) are also incorporated into lipids in swine adipose tissues in vitro, although the physiological role is not known.

Control of Lipogenesis. The hormone insulin is the major anabolic hormone regulating the adipose tissue lipogenic capacity in non-ruminant mammalian species. Insulin capacity to stimulate lipogenic activity in swine adipose tissue is low in vitro (Etherton and Chung, 1981). In contrast to other species, insulin has long term regulatory effects on the lipogenic activity in swine adipose tissue. Romsos et al. (1971) produced diabetic pigs with the use of alloxan and found that they had almost totally depressed lipogenic activity that was restored by insulin injection. Furthermore, Kasser et al. (1981) found that fetuses at 112 d of gestation from pregnant diabetic sows had greater adipose tissue lipogenic rates than those from non-diabetic sows. However, in subsequent similar experiments, such effects were not so clear (Kasser et al., 1982). The effects of other hormones associated with metabolic control, such as glucagon, adrenocorticotropin, corticosteroids, growth hormone, thyroid

hormones, and catecholamines, have not been examined to any extent in swine adipose tissue. Experiments using decapitated swine fetuses have shown an increased adipose tissue lipogenic rate suggesting a role for central nervous system or pituitary factors in the control of lipogenesis (Kasser et al., 1983).

Influence of the Diet on Adipose Tissue Lipogenesis. As with other nonruminant mammalian species, diet composition influences the rates of swine adipose tissue lipogenesis (Romsos and Leveille, 1974). Compared with high-carbohydrate diets, feeding of high-fat diets produces lower in vitro fatty acid synthesis and lipogenic enzyme activities in swine adipose tissue (Allee et al., 1971a). Suppression of lipogenesis by feeding high-fat diets has been observed in pigs as young as 14 d of age (Wolfe et al., 1977) although in very young pigs, the regulation of the adipose tissue lipogenic rate appears to be compromised by other dietary components and the level of feed intake (Mersmann et al., 1973c; Mersmann et al., 1976). Allee et al. (1971a) suggested that the low lipogenic rates observed in adipose tissue before weaning probably result, at least in part, from the relatively high-fat milk diet. The same authors (Allee et al., 1972) reported later that several different fats, both saturated and unsaturated, when fed at about 10% in the diet, inhibited fatty acid synthesis in swine adipose tissue to the same extent; and that dietary medium-chain triglycerides are not as inhibitory as long-chain

triglycerides. Waterman et al. (1975) reported that at higher dietary levels, a saturated fat (tallow) inhibited fatty acid synthesis to a greater extent than an unsaturated fat (sunflower oil). The sources of protein and carbohydrate in diets may also influence the adipose tissue lipogenic capacity. Allee et al. (1971b) fed a low-fat, 12% protein diet to growing pigs (46 kg) and found that they had greater in vitro adipose tissue lipogenic rates as well as the amount of carcass fat than pigs fed a 24% protein diet. However, when pigs were about 12 kg BW, dietary protein did not influence in vitro lipogenic rates.

The Role of Lipoprotein Lipase. The lipoprotein lipase enzyme is produced by numerous tissues in the animal body (Cryer et al., 1978). Lipoprotein lipase has been purified from swine adipose tissue (Bensadoun et al., 1974). Lipoprotein and serum lipid profiles of fetal, neonatal, and adult swine have also been reported (Johansson and Karlsson, 1982). Lipoprotein lipase activity is observed histochemically in adipose tissue from swine fetuses about midway through gestation (Hausman, 1982). The physiological function of adipose tissue lipoprotein lipase in the poorly developed fat depots of the swine fetus is not clear. The enzyme activity rapidly increases after birth and levels are maintained throughout development (Hausman, 1982). Lipoprotein lipase is probably important for the supply of fatty acids to adipose tissue during the suckling period when the diet is relatively high in fat, the adipose tissue de novo fatty acid

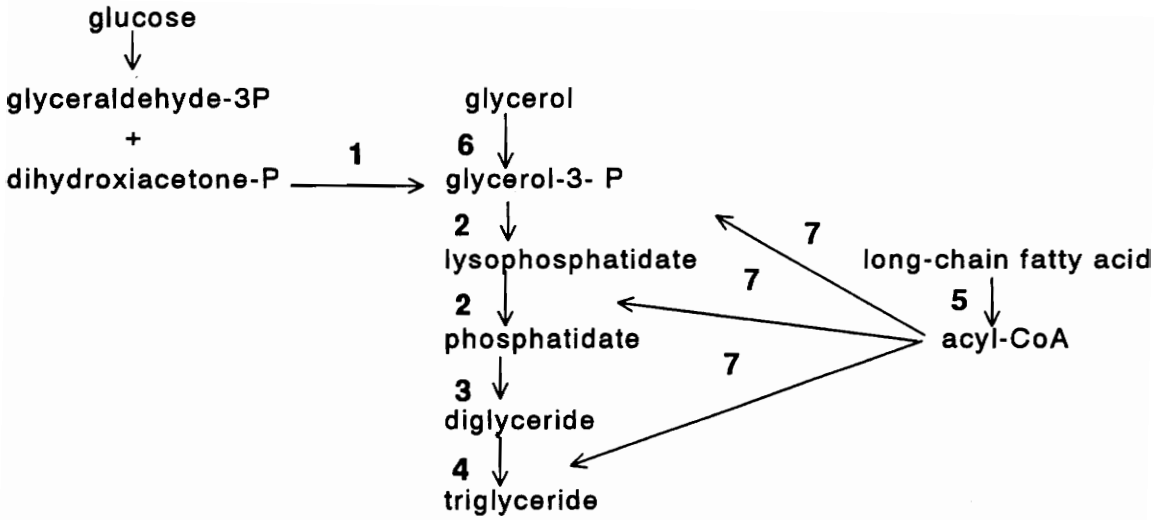
synthesis capacity is low, and the relative deposition of fat is great. Lee and Kauffman (1974) reported that lipoprotein lipase is probably important for maintenance of the dynamics of lipid metabolism to provide fatty acids to the adipose tissue esterification from lipoproteins in blood of either dietary or hepatic origin. Dietary energy intake levels control lipoprotein lipase activity; adipose tissue enzyme activity is decreased during low caloric intake, whereas, in some other tissues, the enzyme activity remains elevated, presumably to provide fatty acids for oxidation. Starvation decreases swine adipose tissue lipase activity (Steffen et al., 1981). There is limited specific information available on this aspect; however most reports indicate that lipase activity is regulated similarly to that of other mammalian species. The metabolism of very low density, low density, and high density lipoproteins have been described (Carew et al., 1976; Hannan et al., 1980; Bartner et al., 1982). Lipoprotein synthesis, degradation, and transport will not be discussed in this review.

Triacylglycerol Biosynthesis. De novo synthesis of fatty acids, followed by esterification into TG are the major metabolic functions in swine adipose tissue. At 2 wk of age, porcine adipose tissue is composed of about 50% TG; this can be increased to as much as 90% of the depot weight in older growing animals (Mersmann et al., 1973c). The biosynthesis of adipose tissue TG has received little attention in swine, but is considered more important in the control of fat deposition than the synthesis of

the precursors, fatty acids.

Metabolic Pathways. The major pathway for synthesis of TG in most mammalian tissues appears to be the glycerophosphate pathway (Figure 2), the enzymes of which are in the endoplasmic reticulum. However, Schlossman and Bell (1976) pointed out that the enzymes in other pathways may also play roles in the synthesis of TG. For example, dihydroxyacetone phosphate may be directly acylated by the same enzyme that acylates alpha-glycerolphosphate. Biosynthesis of TG, including the cellular localization of enzymatic activities, has been previously reviewed (Bell and Coleman, 1980).

The glycerol phosphate pathway in swine adipose tissue microsomes incorporates glycerolphosphate and long chain fatty acids into phospholipids, diacylglycerol, and triacylglycerol (Steffen et al., 1979). Swine adipose tissue, unlike that of many other species, has a large amount of saturated acylgroups on the second carbon of glyceride-glycerol in triacylglycerol (Raju and Six, 1975). Palmitate is readily incorporated into adipose tissue slices obtained from pigs at several ages (Etherton and Allen, 1980). Glycerol has been demonstrated to be incorporated to a lesser extent. Mersmann et al. (1973a) reported that the amount of glycerokinase activity in porcine adipose tissue is probably a limiting factor. There are few studies of nutritional influences on swine adipose tissue TG biosynthesis. In young weaning pigs, high-fat diets do not change enzyme activities relative to those observed in pigs fed low-fat diets, whereas a 72-h fast



1 alpha-glycerolphosphate deshydrogenase
2 glycerolphosphate acyltransferase
3 phosphatidate phosphohydrolase
4 diglyceride acyltransferase
5 acyl-CoA synthase
6 glycerol kinase
7 acyl transferase

Figure 2. Synthesis of triglycerides. (Adapted from Stryer, 1988).

reduces enzyme activities relative to those observed in tissue from fed pigs (Steffen et al., 1978). Endocrine influences on triacylglycerol biosynthesis have not been investigated in swine adipose tissue.

Tissue Sites. The glycerophosphate pathway has been demonstrated in swine liver in vitro where it is assumed to be utilized to assemble TG for storage and for further processing to lipoproteins for secretion. In swine, the process of biosynthesis of TG occurs in microsomes of hepatocyte (Akesson, 1969; Sundler and Akesson, 1970). Much of the fatty acid incorporated into liver lipids is in the phospholipid fraction primarily in the form of phosphatydilcholine (Huang and Kummerow, 1982). There is little information on the TG synthesis in other swine tissues.

Catabolism of Lipids

Lipolysis. The degradative or lipolytic process is highly regulated in adipose tissue and is under endocrine control. The cascade-system regulation is indicated by formation of a hormone-receptor complex at the cell surface followed by intracellular generation of cAMP, activation of protein kinase, and finally phosphorylation and consequent activation of a specific lipase that is the rate limiting factor in the degradation of triacylglycerol (Figure 3). Fatty acids released by lipolysis mostly enter the plasma and are utilized by other tissues for

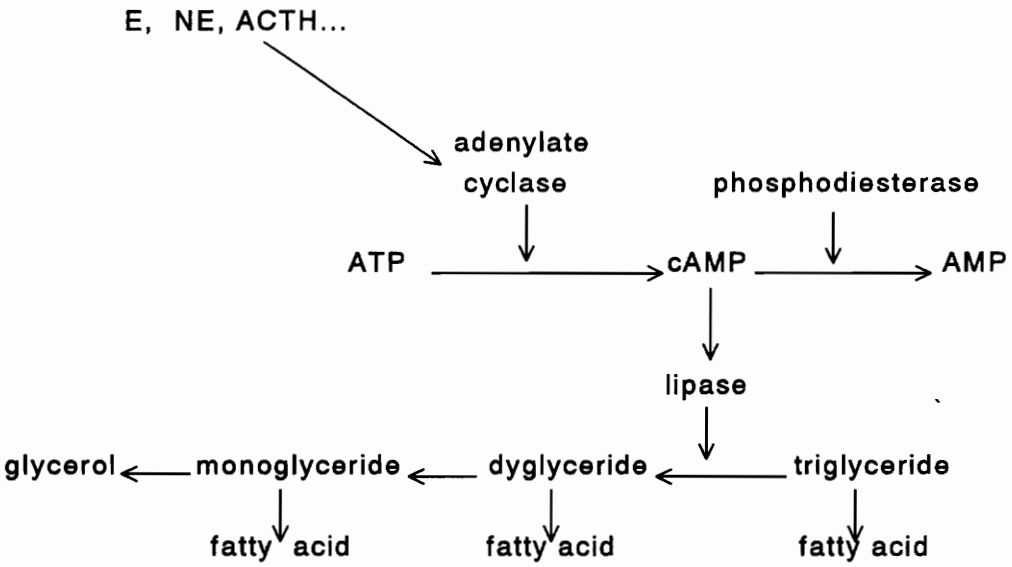


Figure 3. Mechanisms of lipolysis. (Adapted from Hahn, 1972)

oxidation or in the synthesis of various complex lipids. Some fatty acids can be reesterified into triacylglycerol within the adipose tissue cell.

Hormonal Control. Lipolysis in adipose tissue from mammals is readily regulated by beta-adrenergic agonists (Mersmann et al., 1974). The synthetic noreprinephrine analogue, isoprotenerol, acts as a beta-agonist and is very effective and potent (Fain, 1973). Likewise, adipose tissue lipolysis in swine is regulated by beta-adrenergic compounds (Mersmann, 1984). The swine adipose tissue adrenergic receptor cannot be classified into a $beta_1$- or $beta_2$-subtype because swine adipose tissue has an extreme agonist structural specificity for stimulation of lipolysis. Adipose tissue lipolysis is also stimulated by other hormones. Porcine adipose tissue lipolysis <u>in vitro</u> is marginally stimulated by adrenocorticotropin and somatotropin, but glucagon does not stimulate lipolysis at any age (Mersmann et al., 1975). The inhibition of lipolysis in adipose tissue of swine is carried out by adrenergic receptors (Fain, 1973). Inhibition of lipolysis readily occurs in the presence of adenosine, and this inhibition may operate physiologically in swine adipose tissue (Fain, 1973). Prostaglandins, particularly the E-type, inhibit lipolysis. There is evidence that insulin may act as a physiological inhibitor of lipolysis (Himms-Hagen, 1970). Research also indicates that there are a wide diversity of hormones controlling lipolysis for the different species of animals.

Ontogeny. Hahn and Novak (1975) reported that adipose tissue lipolytic activity is high in late fetal and newborn humans. In pigs, epinephrine-stimulated adipose tissue lipolytic rates are low at birth but increase dramatically by post partum d 2 (Mersmann et al., 1975). The deficit in porcine newborn lipolitic rates is attributed to a lower hormone-sensitive lipase activity and to the generally minimum development of adipose tissue in the newborn pig (Steffen et al., 1978). Newborn pigs have low concentrations of plasma free fatty acids and these are only marginally elevated by fasting (Swiatek et al., 1968). Both the plasma concentration and the capacity to mobilize fatty acids increase progressively during the first day of life. Newborn wild piglets have been shown to have more capacity to mobilize fatty acids than domesticated piglets; this was due to a greater adipose tissue hormone-sensitive lipase activity (Horn et al., 1973).

Fatty Acid Oxidation. Miller et al. (1971), in their review, concluded that most of the long chain fatty acids produced by adipose tissue lipolysis are transported via the plasma to various tissues of the animal body. Fatty acids are readily used as an oxidative energy source by many tissues of the body including skeletal muscle, liver, cardiac muscle, and adipose tissue. Mitochondrial oxidation of fatty acids yields CO_2 and ketone bodies as products. Pigs have been shown to oxidize fatty acids readily in many studies. The respiratory quotient is depressed by exposure of piglets to cold (Noblet and Le Dividich,

1981) indicating fat catabolism. Newborn piglets have very small fat stores, marginal ability to mobilize fatty acids, and little ability to oxidize fatty acids (Noblet and Le Dividich, 1981). Older pigs oxidize a variety of long-chain fatty acids, and the rates are increased by fasting (Freeman et al., 1970). Long-chain fatty acids are transferred across the mitochondrial membrane as carnitine derivatives. Newborn pigs have reduced ability to oxidize fatty acids in vitro in liver, and skeletal muscle, but apparently not in heart and kidney (Wolfe et al., 1978). The newborn pig heart utilizes fatty acids in preference to glucose as an oxidative substrate but in the fetal pig heart, fatty acid oxidation is suppressed (Werner et al., 1983).

Essential Fatty Acids in Swine

Research work between the 1930s to 1960s (Burr and Burr, 1929; Holman, 1964), led to the conclusion that the term essential fatty acids should include only those substances which were active both for growth and for maintenance of dermal integrity. At present, there is no clear agreement among scientists regarding this concept. Questions are being raised on whether a compound is essential for mammals only because they cannot synthesize it or whether all n-6 (linoleic, eicosatrienoic, gamma-linolenic, arachidonic) and n-3 (eicosapentanoic, linolenic, docosahexaenoic) acids are equally effective in meeting the definition of essentiality. However,

most authors agree that linoleic acid is the EFA by definition. Animals have the enzymatic capacity to synthesize arachidonic (20:4n-6) from linoleic acid (18:2n-6) and the intermediate fatty acids namely, gamma-linolenic acid (18:3n-6) and dihomo-gamma-linolenic acid (20:3n-6) and its derivative docosapentaenoic acid (22:5n-6). In this way, ingested linoleic acid (18:2n-6) is converted to gamma-linolenic (18:3n-6), dihomo-gamma-linolenic acid (20:3n-6), arachidonic (20:4n-6), docosatetraenoic (22:4n-6) and docosapentaenoic (22:5n-6) acids (Holman, 1978). Dietary alpha-linolenic (18:3n-3) acid is converted to docosahexaenoic acid (22:6n-3) and eicosatetraenoic (20:4n-3). However, animals do not posses enzymes capable of inserting double bonds between the 9-carbon and the terminal methyl group. Fatty acids with methylene-interrupted double bonds in this region are required in the diet and are termed essential fatty acids (Watkins, 1991). The two essential fatty acids most common in the diets are linoleic and linolenic acid. These two fatty acids and oleic acid give rise to series of PUFA designated n-9, n-6, and n-3 according to the position of the double bond nearest to the methyl group.

However, Whitehead (1984) pointed out that there are some factors which must be taken into account when judging the concept of nutritional essentiality of fatty acids. Thus, interconversion of the fatty acid provided in the diet need not be the acid ultimately responsible for alleviating the deficiency; it need only have appropriate double bond configuration to allow

synthesis of the metabolically active higher member of the series. Consequently, a fatty acid may have EFA activity without being a necessary dietary constituent.

Another reason why the role of EFA in nutrition is not fully recognized is that their metabolism is dependent on a number of dietary cofactors (Cunnane, 1984). These cofactors, which include zinc and selenium and possibly vitamins, are essential in their own way (Dib and Carreau, 1987; Watkins and Kratzer, 1987). Thus, the utilization of dietary EFA and other lipids require the presence of those cofactors to regulate functions such as desaturation, elongation, esterification, and prostaglandin synthesis. A further complication arises from the wide range of metabolic processes in which fatty acids are involved. A dietary EFA deficiency may have many manifestations, but those may respond differently to treatments with different fatty acids (Holman, 1978). Furthermore, Whitehead (1984) also reported that, when assessing responses of various parameters to different dietary fatty acids, it is sometimes difficult to differentiate between responses to a fatty acid or family of acids. In summary, there is still a need to further investigate the physiological functions of EFA. These aspects are reviewed herein.

Physiological Importance of PUFA. It is widely accepted that the n-6 and n-3 PUFA are of physiological importance. Because of their low melting points which are essential to maintain the gross fluidity of adipose tissue and the fluidity of phospholipids in membranes, they also function as precursors for

the synthesis of prostaglandins, prostacyclins, thromboxanes and leukotrienes (Needleman et al., 1979; Neuringer and Connor, 1986).

The ability of erythrocytes to resist rupture in response to haemolytic stress is an example of the membrane integrity function of PUFA. The function of PUFA as prostaglandin precursors is also important because, among other activities, these compounds affect blood preassure (Hoffmann and Forster, 1986), blood clotting (Driss et al., 1981), the immune response (Mertin 1981; Kinsella et al., 1990) and pregnancy (Pope et al., 1982; Geisert et al., 1986). Both omega-6 and omega-3 PUFA alter properties of some proteins; they inhibit the binding of steroids to certain proteins such as the estrophile alpha-fetoprotein, the sex steroids-binding protein and the corticosteroid-binding globulin (Holman, 1978). These functions are all of specific significance during fetal and postnatal life in mammals when the developmental processes concentrated in a limited time interval, occur in a critical sequence and are highly demanding in terms of lipid requirement.

Growing tissues have a high EFA requirement for the synthesis of structural lipids (Pope and Fist, 1985). The use of dietary EFA involves their conversion to the 20- and 22-carbon chain length PUFA derivatives, in order to be actively incorporated into membrane structures, especially in the central nervous system (Samulski, 1982), in which the main period of cell

division is prenatal. Both linoleic and linolenic acids and their longer chain derivatives are found in fetal and neonatal structural lipids in humans and other mammalian species (Mersmann et al., 1973a). Thus, both families of fatty acids should be provided in the diet. Furthermore, the long-chain PUFA of the n-3 series may have a specific role in the development and functioning of brain (Bourre et al., 1989) and retina (Neuringer et al., 1986), whereas, dietary alpha-linolenic acid has been shown to prevent skin lesions in monkeys, even in the presence of dietary linoleic acid (Fiennes et al., 1973). Among the long-chain polyunsaturated derivatives of dietary PUFA, only 20-carbon members are converted to active prostanoids, at least in mammals.

If the dietary supply of unsaturated fatty acids is insufficient, they can be synthesized in the animal body. Essential fatty acids, linoleic and linolenic are transformed by processes of desaturation and elongation into more unsaturated longer-chain derivatives which are needed for normal growth and maintenance.

The recommended linoleic acid content in swine diets is 1.5% of the digestible energy for the young pig and .7% of the digestible energy for the finishing pig (NRC, 1988). However it is difficult to produce overt signs of EFA deficiency in pigs and normal growth has been reported in finishing pigs fed on diets containing .1% dietary linoleic acid. The backfat of pigs usually contains approximately 10% linoleic acid, double that in humans,

and the softening effect of excess linoleic acid is likely to be more of a problem than a deficiency (Allen and Foegeding, 1981). The actual requirement of linolenic acid has not yet been specified.

Deficiency of Essential Fatty Acids in Animals

The importance of essential fatty acids (EFA) in animal nutrition and metabolism has long been known. Burr and Burr (1929) were the first to report that total deprivation of fat in the diets of weaning rats induced a deficiency which was corrected by adding certain components of fat. Production of EFA deficiency in weaning rats is usually carried out by starting them on an artificial diet either fat-free or containing only fully saturated fat. However, Holman (1978) reported that it is difficult to produce deficiency symptoms in the adult rat presumably because stored EFA satisfy the needs of the adult for protracted periods. The deficiency state was also reported to be extremely difficult to attain in swine and humans (Holman, 1978). The regular diets fed to pigs usually contain adequate amounts of EFA (Stahly, 1984); thus the EFA deficiency is far rarer than those of protein, vitamins, and minerals. Externally recognizable EFA deficiency induced by a fat-free diet requires approximately 3 mo to produce dermal lesions in rats (Holman, 1978).

The fatty acids necessary to prevent the deficiency symptoms have been proved to be n-6 linoleic acid of plant origin, and

arachidonic, formed in animals consuming linoleic acid. Eicosatrienoic and gamma-linolenic acids, two acids from the same n-6, family are also effective in preventing EFA deficiency. In the omega-3 family of fatty acids, alpha-linolenic acid, present in certain seed oils (ex: soybean oil or linseed oil) and fish oils (ex: menhaden, mackerel), seems to be effective for growth promotion but do not prevent the skin symptoms.

Linolenic acid deficiency was first described in humans by Holman et al. (1982). The deficiency was caused by long-term intake of fat-free parenteral nutrition. Total parenteral nutrition (TPN) is a medical technique utilized commonly to help patients who are unable to tolerate oral food for extended periods to survive. Total parenteral nutrition is commonly administered as the continous infusion of glucose-containing solution that results in a constant elevation of serum insulin and consequently depresses the release of fats including EFA from adipose fat stores (Jungas and Ball, 1963; Mascioli et al., 1979). Attempts to deplete the omega-3 docosahexaenoic acid (DHA) from tissue lipids by the use of fat-free diets or diets low in PUFA have been reported to be only partially effective, and have greater effects on n-6 fatty acid levels than n-3 fatty acids (Neuringer and Connor, 1986). Holman (1978) pointed out that the dietary ratio of n-6 and n-3 fatty acids appears to be more important than the absolute amount of n-3 fatty acids in determining the degree of DHA depletion. This effect has been

attributed to be caused by competitive inhibition between these two fatty acid families for the same desaturating enzyme system (Holman, 1978; Mohrhauer and Holman, 1963); high dietary levels of linoleic acid suppress the conversion of linolenic acid to DHA. Consequently, greater DHA depletion has been produced in studies which used purified linoleic acid as the only dietary fat, or used natural fats such as safflower or sunflower oil in which the n-6/n-3 ratio is very high. Increased levels of the triene 20:3n-9, which are characteristic of n-6 deficiency, do not occur in specific n-3 fatty acid deficiency but only when both n-6 and n-3 fatty acids are unavailable (Neuringer and Connor, 1986). However, results of experiments with rats by Tinoco et al., (1971, 1977) showed that deprivation of dietary n-3 fatty acids for two or three generations caused no effects on fertility, birth weight, growth, or organ weights.

Symptomatology. The effects of an EFA deficiency involve all organs. This is because all polyunsaturated fatty acids formed from dietary EFA are essential for the structural and normal functions of the cell membranes. Essential fatty acids are known to be required for the synthesis of lipids of vital membranous structures. Thus, during periods of rapid growth, such as in fetal and in early life periods, a more severe manifestation of EFA deficiency will be manifested (Guesnet et al., 1986).

Signs of EFA deficiency have been described in rats by Holman (1978). The first sign is retarded growth rate. A scaly

dermatitis may also develop. This appears first on the back of the paw. Scaliness of the skin occurs over the entire surface of the body and tail and in increasing severity, from erythema through light scaliness, flakes and concentric scales (which somewhat resemble fish scales). The scales can become 1 or 2 mm long, projecting from the trunk of the tail. In most cases, before the entire tail is covered with such scales, necrosis begins, usually at the tip of the tail.

Additional symptoms of EFA deficiency in rats have been reported by other workers (Cunnane, 1984) and include loss of muscle tone, inability to reproduce, modifications in organ weights and size, fatty liver, kidney damage, and changes in lung structure, both macroscopic and microscopic, which leads to elevated respiratory quotient and high metabolic rate. All these symptoms are prevented or reversed by feeding fatty acids of the n-6 family. Although n-3 fatty acids are also partially effective in some cases, they are not specifically necessary (Holman, 1978). Only some species of fish show specific dietary deficiency of n-3 fatty acids where similar symptoms include impaired pigmentation and growth, necrosis of the tail fin, pale and fatty liver, and stress-induced "fainting" (Nicolaides and Woodall, 1962).

An EFA deficiency has been induced in weaning pigs (Hill et al., 1961; Howard et al, 1965). The symptoms include hair loss, scaly dermatitis, skin necrosis, poor growth, inanition, and early mortality. Aortic lesions involving calcification but not

lipid deposition have also been reported. Early studies by Hill et al. (1957) reported that, unlike other species, swine do not consistently show a scaly dermatitis. However, Hill et al. (1961) explained that this differences could be due to relative humidity which is known to affect the degree of dermatitis in EFA-deficient rats. Hanson et al. (1958) found that the age of pig also influences the deficiency symptoms due to EFA. The younger pigs were more susceptible than the older pigs.

Holman et al. (1982) described a case of linolenic acid deficiency of a 6-year-old girl who sustained an accidental gunshot wound to the abdomen and was maintained on home TPN that included safflower oil emulsion rich in linoleic acid. After 5 months, she experienced episodes of numbness, paresthesia, weakness, inability to walk, leg pain, and blurred vision. Total parenteral nutrition was then changed to include soybean oil emulsion, which contains linolenic acid. All the symptoms disappeared over the next 3 mo.

Use of Soybean Oil and Menhaden Oil in the Diets of Pigs

In recent years there has been a heightened interest to decrease the consumption of dietary fats and cholesterol, and to increase the polyunsaturated to saturated fatty acids ratio in diets to reduce the risk of coronary heart disease. There has been a greater awareness in the consumption of various oils containing PUFA and no cholesterol. Many investigators have shown that, in addition to their effects on the cardiovascular system,

PUFA can affect the immune system (Marshall et al,, 1983) and platelet function (Culp et al., 1979; Culp et al., 1980) as well as contribute to the formation of the nervous system of the fetus (Lamptey and Walker, 1976; Fayard et al., 1990; Wainwright et al., 1992).

The physiological effects of vegetable oils are primarily due to their fatty acid composition, which vary depending on the specific oil. The health aspects of the soybean and fish oil has been reviewed previously (Sanders and Younger, 1986; Dyerberg, 1986; Kaizer et al., 1989). The present review evaluates the value of soybean and menhaden oils as sources of the essential fatty acids (EFA), linoleic and linolenic acids, and the suitability of inclusion of soybean and menhaden oils in the diets of pigs with respect to the fatty acid composition of these oils.

The world annual production of fats and oils has been estimated to be 75 million metric tons (Bimbo, 1990). Soybean oil and animal fats make up almost 50% of the total while marine oils, consisting of marine mammal oils, fish liver oils, and fish body oils, account for only 2% of the world supply of fats and oils. Fish body oils make up to 97% of the total marine supply (Bimbo, 1990). The U.S. production of edible fats and oils was about 7.6 million metric tons in 1988, with soybean oil accounting for about 67% of the total production of all fats and oils. The U.S. production of marine oils totalled 135,000 metric

tons in 1987 and, was composed of 134,000 metric tons of menhaden oil and 1,500 metric tons of oil from miscellaneous fish (USDC, 1988).

Animal and vegetable oils have been incorporated into animal feeds for many years. They have growth-promoting effects, and fish oils contain significant amounts of vitamins A and D (Higashi, 1961; Stansby, 1982). Fish oils are also used because of the benefits of their broad spectrum of fatty acids. Research with swine has demonstrated that carcass fat may be influenced among other factors by the dietary fatty acid profile (Dahl and Personn, 1965; Brooks, 1971). Numerous experiments have shown that changing the carcass fat composition can be accomplished more readily in the pig than in any other large farm animal (Allee et al., 1976; Enser et al., 1984; Busboom et al., 1991). Therefore, a practical way to modify quality characteristics of pork may be through the diet.

The digestible energy of a fat is influenced by the degree of saturation, length of the carbon chain, and free fatty acid contained (Wiseman, 1986). In the monogastric animal, the dietary FA are absorbed unchanged, hence changes in the type and amount of fat in the diet will be reflected in the FA composition of adipose tissue.

Soybeans (glycine hispida) have an oil content of about 20% on a dry weight basis, which makes them a relatively good oil source. Soybean oil production around the world as well as in the

United States has increased markedly from 1940 to 1980s (Meydani et al., 1991). The change in its economic position came about with improvements in industrial practices in processing. Soybean oil has currently been used in the diets of pigs as a way to increase energy density of the diets of pigs as well as reduce dustiness in the facilities (Gore et al., 1986).

The use of fats in sow diets during late gestation periods to improve piglet survival and consequent litter size has been controversial. The wide variety of experimental designs and conditions employed make it difficult to draw definitive conclusions (Seerly, 1984), but an overall summary of the results from several experiments on this issue suggests a small improvement in piglet survival. Furthermore, the type of fat (Fengler, 1990), the level of fat (Pettigrew, 1981), and the stage of pregnancy are important factors in determining the degree of improvement. However, the major factor limiting the use of supplemental fat in pig diets is economics. Available data indicates that there is an improvement in pig survival by the use of supplemental fat; thus, it is estimated that if the average number of pigs marketed per sow per year in the U. S. is about 14 pigs, increasing this number by even 1 to 2 pigs per sow per year or 1/2 to 1 pig/litter) can change the amount of costs radically (Lindemann, unpublished).

The marine fish menhaden (bug-fish, pogy) is an abundant fat and oily fish ranging along the Atlantic coast of N. America from Nova Scotia to Florida, and represented in the Gulf of

Mexico by similar forms (Wheeler, 1985). Menhaden is one of the most important commercial fish in the E. coast of the U.S., currently being used for the production of fish meal, oil, and fertilizers. Although it is edible, it is too oily to appeal to man (Vaughan et al., 1988). Pigs can utilize fish oils as a source of energy, but the amount included in their diets must be limited to maintain the quality of carcass, since, "fishy" flavors are sometimes found in pork (Garton and Duncan, 1954). Karrick (1990) recommended that fish oils could be added at a level of about .5% in the diet, and they suggested that the oil be removed from diet for a period from 2 to 4 wks prior to slaughter.

Soybean oil is pale yellow in appearance and typically has a bland flavor when it is properly processed and fresh. Crude soybean oil contain 1.5 to 2.5% non-glyceride materials, consisting mainly of phosphatides which are removed during refining (Landers and Rathmann, 1981). Refined soybean oil contain about 99% triacylglycerols (Hammond and Fehr, 1984). A typical FA distribution for soybean oil is shown in the table 8 chapter III). It can be seen that soybean oil has 59.6% PUFA, 21.4% monounsaturated fatty acids and 19.1% saturated fatty acids. In this table the fatty acid profile of fish (menhaden) and coconut oils, a saturated oil, is also shown. A major difference between these oils is the amount of PUFA. Soybean oil and fish (menhaden) are relatively rich sources of PUFA; the polyunsaturated:saturated ratio (P:S) for coconut, soybean and

fish (menhaden) oils is .02, 3.1, and 1.1 respectively. The PUFA levels of soybean oil and fish oil compare favorably with other major vegetable oils consumed in the United States (Landers and Rathmann, 1981).

The amounts of linoleic and linolenic acids are low in fish oils but the amounts of the linolenic acid family (omega-3 FA) are high. The growth-promoting activity of the omega-3 fatty acids from fish oils was demonstrated in studies by Privett et al. (1960). They fed EFA-deficient rats with supplements of C_{16}, C_{18}, C_{20}, and C_{22} fatty acid esters fractionated from tuna oil and compared the results with a supplement of ethyl linoleate. Supplements did not cure dermal symptoms, but all except the C_{16} fraction had growth-promoting activity equivalent to the ethyl linoleate. These data on omega-3 fatty acids were supported in subsequent studies by Privett et al. (1960), who showed that menhaden, herring, and tuna oils fed to EFA-deficient rats at a 10% level in the diet not only stimulated growth but also cured dermal symptoms. Metabolizable energy of fish oils is high. Renner and Hill (1958) reported that the metabolizable energy value of menhaden oil for chicks is 8.150 kcal/kg, whereas tallow had a value of 6.388 kcal/kg.

Soybean oil has been supplemented in corn-soybean meal based pig starter diets with good results. Howard et al. (1987) fed pigs at a rate up to 6% of the diet obtaining significant improvements in average daily gain, feed intake and gain per feed. Coefficients of digestibility of oils reflect their feeding

and their growth-promoting values (Artman, 1964).

Some of the nutritional problems attributed to fish oils are related to their rancidative oxidation which leads to the formation of peroxides that are toxic to animals (Kinsella, 1987). The amount of vitamin E present may influence the stability of various oils. It is known that vitamin E plays a role as an intracellular antioxidant (Privett and Cortesi, 1972). Einset et al. (1957) reported that menhaden oil contains 70 micrograms of vitamin E per gram of oil. Adequate vitamin E is critical in diets rich in PUFA. Higher dietary levels of PUFA increase the requirement of some animals for vitamin E (Malm et al., 1976). Watanabe (1982) reported that the effects of vitamin E in animals are related to the dietary form of tocopherol, as well as to the presence or absence of selenium and ascorbic acid.

The high contents of PUFA in both menhaden and soybean oils however, makes them susceptible to oxidative deterioration and reduced flavor stability. In particular, the high concentration (7%) of linolenic acid in soybean oil increases its oxidative instability (Dutton, 1981). Nonetheless, unhydrogenated soybean oil has been shown to be stable for several months of storage.

Role of Linolenic Acid on the Reproductive Performance in Mammals

Lipids are the main energy store used during the course of pregnancy, lactation, and weaning. Polyunsaturated fatty acids have been shown to play a fundamental physiological role in

animals and humans (Holman, 1978; Holman et al., 1982). A PUFA deficiency can be the direct cause of delayed development (Aaes-Jørgensen and Holmer, 1969), skin disorders (Hanson et al., 1958 ; Wiese et al., 1966), and reproductive disorders (Guesnet et al., 1986). In addition to the work of Holman et al. (1982), several studies have indicated that omega-3 fatty acids are essential and have a major role in the structure and function of cell membranes, particularly in the central nervous system (Mohrhauer and Holman, 1963; Clandinin et al., 1980).

The role of EFA in pregnancy and fetal growth in humans and animals has been examined only in recent years. The relationship between maternal nutrition during pregnancy and fetal lipids is controversial. Hansen et al. (1964) found no correlation between maternal nutrition during the third trimester and serum lipids of their infants. However, Churchill et al. (1967) hypothesized that the lower intelligence quotients observed in children born from mothers with biliary tract disease during gestation could be related to decreased availability of EFA for fetal brain development. Irreversible impairment of learning behavior also has been described in mice born from mothers kept on EFA-deficient diet during the last third of pregnancy (Lamptey and Walker, 1976).

Fats are currently used in intensive swine production systems to supplement the diets of pigs because of their high energy content (approximately 2.25 that of the carbohydrate) and their favourable cost relative to other sources of energy. Fat

supplementation of the diet of sows during late gestation has been shown to improve piglet survival rate during lactation (Pettigrew, 1981). However, little is known about the role of specific types and sources of fatty acids during pregnancy. A recent experiment by Fengler et al. (1990) in swine indicate an increase in the length of embryos from gilts supplemented with safflower oil (rich in linoleic acid) suggesting that the ingestion of safflower oil may play a role in fetal development and growth. Normal fetal development and growth require a high level of EFA for the synthesis of structural lipids. These observations, however, need to be further investigated since n-6 fatty acids may not be adequate for conditions that specifically require n-3 fatty acids which are essential during early development of the nervous system.

The central nervous system in swine and other species undergoes an accelerated growth-phase during the last stage of pregnancy and during the first month of extrauterine life and requires a high supply of EFA for structural expansion (Lamptey and Walker, 1976; Samulski and Walker, 1982). Brain and peripheral nerve tissues contain high levels of 22-carbon n-3 PUFA (Naughton, 1981) and several investigators (Mohrhauer and Holman, 1963; Galli et al., 1972; Sanders et al., 1984) have insisted on the essentiality of these n-3 PUFA during early development. The visual function of the retina in monkeys (Neuringer et al., 1984) and learning ability in rats (Lamptey and Walker, 1976; Yamamoto et al., 1987) have been shown to be

impaired by diets low in n-3 fatty acids. Furthermore, Pope and First (1985) in their review, concluded that the levels of prenatal mortality in the first 40 d of gestation accounted for the majority of the 30% prenatal deaths. In contrast mortality of fetuses that occur during the latter half of pregnancy is low (10%) when insufficient luteal development (Webel et al., 1975) or insufficient uterine space (Webel and Dziuk, 1974) occur. The specific causes of porcine embryo and early fetus death are still unclear. Bazer et al. (1969) postulated that some factors influencing uterine environment, and consequently the implantation and nutrition of the embryo, may influence the embryo survival. The influence of level of nutrition in early gestation on embryo survival in swine has been studied by several authors in the sow (Lodge et al. 1966; Hawton and Meade 1971; Dick and Strain 1983), and in the gilt (Scofield, 1972; Hartog and Kemper, 1980). The common conclusion reached was that a high level of feeding during this period is detrimental to embryonic survival. Furthermore, a reduction in conception rate was produced if sows were underfed before or during early pregnancy (Anderson, 1975; Dick and Strain, 1983). These data demonstrate that the level of nutrition is an important factor influencing the reproductive performance in swine.

Chapter III

EFFECT OF DIETARY FATTY ACID SUPPLEMENTATION ON FETAL SURVIVAL IN GILTS AT 40 DAYS OF GESTATION

Abstract

Eighty-six crossbred (Duroc x Yorkshire) gilts were used in two trials (50 gilts in Trial 1 and 36 gilts in Trial 2) for an assessment of the effect of supplemental dietary fat during early gestation on fetal survival, fetal development, and fatty acid concentration in gilt plasma and fetal head and body. Three diets contained 4% (w/w) added fat either as coconut, soybean, or fish (menhaden) oils. A fourth diet was used as a control. On d 37 to 45 postbreeding, gilts were slaughtered and numerous fetal and ovarian measurements made. Two sets of four randomly selected fetuses per gilt from Trial 1 were prepared. Blood samples from each gilt were obtained on the day of slaughter for determination of the plasma fatty acid profile. Across both trials, percentage fetal survival did not differ according to treatment, but in Trial 2 fetal survival was higher ($P < .06$) for gilts fed fish oil, compared with the controls. The fatty acid profile of plasma of gilts and the conceptus tissues were similar; both were influenced by the fatty acid concentration of the diets. The ratio of n-3/n-6 fatty acids was higher in conceptus tissue than in maternal plasma and the ratio was higher ($P < .05$) for the fish oil diet compared with the other diets. The relatively high concentration of omega-3 fatty acids in fetal tissues

supports the hypothesis that omega-3 fatty acids play a role in the development of the pig conceptus and contributes to improve fetal survival. However, the high percentage fetal survival observed in all the treatments may have masked benefits of supplemental oil.

Key Words: Fatty Acids, Pigs, Reproduction, Polyenoic Acids, Embryo Mortality.

Introduction

The need for greater efficiency in swine production has led to extensive research on factors influencing litter size and prenatal mortality. Prenatal mortality is divided into embryonic mortality (before d 30) and fetal mortality (after d 30) (Lambert et al., 1991). Day (1979) observed that fertilization rate approaches 100%, but approximately 40% of fertilized ova and fetuses were reabsorbed or were aborted during pregnancy in gilts and in sows. Pope and First (1985) pointed out that a major portion of embryonic loss occurs during the second and the fourth week of gestation. The specific causes of porcine embryo deaths are still unclear.

The maternal diet must supply the additional nutrients needed for uterine, placental, and fetal growth. An area of nutrition that has seen little research attention in swine, but which may have importance, is the specific fatty acid needs of the developing embryo/fetus. Results from studies using the developing rat suggested that maternal dietary PUFA during

gestation were potential sources of PUFA in the fetal brain (Sinclair and Crawford, 1972; Sinclair, 1975; Hassam and Crawford, 1976). Furthermore, there is indirect evidence that the fertilized egg and developing embryo may have a requirement for one of the omega-3 fatty acid such as linolenic acid (Naughton, 1981; Samulski and Walker, 1982). Some available sources of linolenic acid in the United States are oils of vegetable origin such as soybean oil, canola oil, and linseed oil. Some types of fish oils are particularly rich in omega-3 fatty acids (Stansby, 1990) and in the longer chain omega-3 fatty acids, eicosapentaenoic and docosahexaenoic acids, but their use in animal feedstuffs is limited because of their cost and the potential off-flavor problems they create in meat.

The objectives of the present study were to determine if soybean oil and fish (menhaden) oil supplementation would enhance embryo survival in pregnant gilts and to demonstrate that the response, if observed, was related to their linolenic acid content.

Experimental Procedures

A total of 86 crossbred (Duroc x Yorkshire) gilts were used in two trials (50 gilts in Trial 1 and 36 gilts in Trial 2) to study the effects of three sources of supplemental dietary fatty acids (FA) during early gestation on fetal survival, fetal development, and fatty acid concentration in sow plasma and fetal head and body. Beginning at 160 d of age, the gilts were checked daily by the use of mature boars to detect estrus. Ten to 17 d

after the first standing heat, each gilt was randomly assigned to one of four dietary treatments with the restriction that genetic background be balanced as much as possible across the dietary treatments.

Dietary treatments consisted of three diets with 4% (w/w) added oil, and a fourth diet with added corn starch[1] equal to 225% of the amount of added oil to balance the energy content with that of the oil-added diets. Composition and calculated analysis of the basal corn-soybean meal diet is shown in Table 1. The added oils were coconut oil[2], soybean oil[3], or fish oil[4]. The coconut oil was selected as a source of primarily short and medium chain FA.
The soybean oil supplied linolenic acid (18:3n-3, an omega-3 fatty acid), and menhaden oil supplied preformed long chain omega-3 FA (e.g. docosahexanoic (22:6n-3) and eicosapentaenoic (20:5n-3) FA). The basal diet contained primarily fatty acids from corn oil.

Gilts were fed 1,816 g/d of the oil-added diet or 1,906 g/d of the added corn starch diet. Daily dietary allowances were therefore, essentially isocaloric. Diets were also equal with respect to all amino acids, minerals, and vitamins. It is known that diets high in PUFA increase

---

[1]Corn starch, Cargill, Eddyville, IA.
[2]Coconut oil, Merricks, Union Center, WI 53962.
[3]Refined, bleached and deodorized soybean oil with TBHQ added as antioxidant. Louisiana Filling, Inc. Wilmington, NC 28402.
[4]Menhaden oil refined, bleached undeodorized-dietary grade, stabilized with 50 ppm ethoxyquin. Zapata Hayne Corp, Reedville, VA 22539.

Table 1. Composition and calculated analysis of the basal diet[ab]

Ingredient	g/kg
Ground corn	761.25
Soybean meal (48% CP)	197.00
Dicalcium phosphate	20.00
Ground limestone	8.00
Salt	5.00
Vitamin premix[c]	2.50
Trace mineral premix[d]	.75
Selenium premix[e]	.50
Tylan-10[f]	5.00
Calculated analysis:	
Crude protein	160.30
Lysine	8.00
Crude fat	29.17
Calcium	8.20
Phosphorus	7.00
Metabolizable energy, kcal/kg	3270

[a]The gilts were fed the basal diet at the rate of 2,724 g per d until assigned to an experimental diet. Then, either 1,743 g per d of basal diet plus 163 g of starch (6,280 kcal ME/d) or 1,743 g per d of basal diet plus 73 g of supplemental oil (6,220 to 6,290 kcal ME/d) was fed.

[b]Upon mixing the basal diet with the starch or oils, .65 grams of premix were added per kilogram of basal diet that supplied an additional 20 IU vit E, .2mg biotin and 100 mg ethoxyquin.

[c]Provided per kilogram of diet: vitamin A 6,875 IU, vitamin D_3 441 IU, vitamin E 27.5 IU, vitamin K 2.20 mg, riboflavin 4.41 mg, niacin 22.0 mg, d-pantothenic acid 22.0 mg, choline 550.7 mg, folic acid 2.2 mg, biotin .28 mg, vitamin B_{12} .02 mg.

[d]Provided per kilogram of diet: Mn 33.0 mg , Zn 99.0 mg, Fe 66.0 mg, Cu 13.2 mg, I .33 mg.

[e]Provided .3 mg of Se per kilogram of diet.

[f]Provided 110 g of tylosin per kilogram of diet.

the vitamin E requirement (Malm et al., 1976); therefore, a premix that supplied an additional 20 IU of vitamin E per kilogram of diet was added as well as biotin for fatty acid chain elongation and desaturation, and ethoxyquin as an antioxidant. The number of gilts assigned to each of the treatments in Trials 1 and 2 respectively, were as follows: 12 and 9 on the corn-starch diet, 13 and 9 on the coconut oil diet, 13 and 9 on the soybean oil diet and 12 and 9 on the menhaden oil diet . The number of gilts slaughtered on each treatment were 11 and 8 for corn-starch, 13 and 9 coconut oil, 13 and 9 for soybean oil, and 11 and 8 for menhaden oil, respectively, for trials 1 and 2.

Two to four gilts were penned as a group by diet and were fed once daily at 0600 to 0800. Diets were prepared biweekly in a vertical single screw mixer. Small amounts of oil were added during the mixing operation to assure that a homogeneous blend of ingredients was obtained. Representative samples of the different diets at the different mixings were pooled and analyzed for fatty acid profile.

At d 35 post-estrus expression, daily heat detection was reinitiated. Gilts were bred twice at first standing heat, immediately and then 8 to 10 hr later, using different boars by either natural service or by artificial insemination of freshly collected semen. This was the third estrus cycle for all gilts and removed the possible effect of variation of estrus cycle on reproductive responses. Gilts that were bred successfully were

grouped by treatment and kept in pens measuring 1.7 m x 3.0 m. Gilts were slaughtered on d 37 to 45 postbreeding. The gravid uterus was removed and separated from the mesometrium. Then, each placental unit was separated by opening the uterus at the cranial end of the right horn and progressing towards the left horn. Sex, length, weight, and position of the fetuses were recorded as well as placental-fetal units in the process of reabsorption. Number of corpora lutea were also determined. Placental weights were taken and recorded including the necrotic tips but excluding allantoic and amniotic fluids. Calculations and analyses of the conceptus were determined on both a wet and dry basis.

Conceptus samples were stored in plastic bags and frozen at -20°C for subsequent analysis. The fetal and placental samples were randomly divided into three sets. The first set was utilized for the analysis of total fat content and fatty acid profile, the second was used for dry matter and total protein determinations. The third set was retained for possible future analyses or repeats.

The gilts were weighed at breeding and on the day prior to slaughter. Blood samples were taken after a 24-h fast from each gilt on d 1 postbreeding and on the day of slaughter by venipuncture of the jugular vein. Blood samples were placed on ice and then centrifuged for 15 min at 500 x g. Plasma samples were collected into plastic tubes and frozen immediately at -20°C for later determination of fatty acid profiles in the laboratory of Dr. Bruce Watkins, Department of Food Science, Smith Hall,

Purdue University, West Lafayette Indiana 24061.

Percent fetal survival was calculated using the number of presumably live fetuses compared with the number of corpora lutea counted on both the right and left ovaries, which was assumed to be an estimate of the ovulation rate (Pope and First, 1985).

The following procedure was used for the determinations of protein and dry matter for both fetus and placenta (Harper, 1991). Tissues were thawed and weighed prior to analysis. Each fetus or placenta was placed in a plastic flask and a phosphate saline buffer (2M NaCl, .05M Na_2HPO_4, .0002M EDTA) was added to a volume of 50 or 100 mL (depending on weight of the tissue) and then homogenized using a polytron high speed homogenizer. Each homogenate was poured into a labeled centrifuge tube and stored at -80°C for subsequent analysis.

Dry matter was determined using a drying oven at 110°C for 5 d. Total protein was determined by the bicinchoninic acid assay method described by Smith et al. (1985). The commercial assay kit (BCA Protein Assay Reagent, Pierce Co., Rockford, Illinois 61105) included the standard (bovine serum albumin) and the bicinchoninic acid reagent. Three aliquots of well mixed homogenate were placed on microtiter plates. Protein was determined by measuring absorbance at 540 nm using an ELISA reader Titertek MCC/340^R (ICN Biomedicals, Huntsville, AL). The fatty acid components of the fetal tissues were determined by gas chromatographic analysis of the methylated lipid samples extracted from a total of four fetuses per diet totalling 2 and

4 g of tissue from the head and body portions, respectively.

Statistical Analysis. Nineteen dependent variables were identified. Days of gestation was used as a covariant for the following variables: weight at slaughter, number of corpora lutea, total number of fetuses, gestational weight gain, percentage fetal survival, number of fetuses alive, placental weight, crown-rump length, fetal weight, total fetal tissue protein (wet & dry basis), total placental tissue protein (wet & dry basis). Five other variables, gilt age at estrus, gilt weight at estrus, age at breeding, weight at breeding, mean estrus cycle length ((age at breeding - age at estrus)/2) were, believed not to be influenced by days of gestation and were analyzed only as an assessment of uniformity with regard to treatment allocation. Tukey's multiple comparison procedure was used to delineate differences among diets. The General Linear Model (GLM) procedure of the Statistical Analysis Systems (SAS, 1989) was used for all analysis of variation.

Results

Of the 86 gilts that were bred on their third estrus, only two gilts (one from SR diet and one from the CO diet) were not pregnant at the time of slaughter, which gave a pregnancy rate of 97.6%. The fatty acid profiles of the dietary oils and diets are shown in Tables 2 and 3. Also, the fatty acid profile of corn oil (White, 1992), which was not actually analyzed, is also included since approximately 93% of the crude fat content of the basal diet came from oil in the ground corn. Soybean oil contained the

highest levels of oleic (18:1n-9), linoleic (18:2n-6), and linolenic (18:3n-3), with the exception of corn oil for oleic and linoleic. Menhaden oil had the highest levels of palmitoleic (16:1n-7), timnodonic acid (20:5n-3), docosapentaenoic (22:5n-3), and docosahexaenoic acid (22:6n-3). The coconut oil had a much higher proportion of medium-chain fatty acids, such as lauric (12:0), myristic (14:0), and palmitic acids (16:0). The fatty acid profile of the diets generally reflected the profile of the oil that was added and the basal diet. The basal diet contained 2.91% crude fat that was made up primarily of corn oil (CN) and some soybean oil (SO). The fatty acid profile of CN was generally similar to that of SO; however, SO had a higher level of linolenic acid and CN had a higher level of linoleic and oleic acids. Thus, the fatty acid profile of the SR and SO diets were very similar, although the total amount of each of the fatty acids was greater for the SO diet. The percentage of linolenic acid (18:3n-3) in SO was about 60% of the book value (White, 1992). The total n-3 fatty acids in the FO diet (Table 3) was more than ten-fold greater than in the CO and SR diets.

The reproductive traits summarized in Table 4 were not influenced ($P > .10$) by the addition of the various types of dietary oils. Also, the various fetal and placental measurements, crown-rump length, fetal weight, and placental wet weight were not influenced ($P > .10$) by dietary treatments (Table 5). Simmilarly, different sources of supplemental oil did not affect fetal and placental tissue protein contents.

Table 2. Fatty acid profiles of oils and diets[a]

Fatty acid	Oils				Diets			
	CO	SO	FO	CN[b]	SR	CO	SO	FO
6:0	.4	—	—	—	—	.3	—	—
8:0	6.7	—	—	—	—	3.7	—	—
10:0	5.6	—	—	—	—	3.1	—	—
12:0	46.4	—	—	—	—	24.6	—	—
14:0	19.2	.3	8.6	—	.1	10.0	.2	4.4
14:1n5	—	—	.3	—	—	—	—	.2
15:0	—	—	.5	—	—	—	—	.3
15:1	—	—	.1	—	—	—	—	T[c]
16:0	9.7	14.1	16.8	12.2	12.7	11.0	13.3	14.6
16:1n7	—	.2	10.6	.1	.2	T[c]	.2	5.8
17:0	—	.1	2.1	—	.1	—	.1	1.1
17:1	—	.1	2.4	—	T[c]	—	.1	1.2
18:0	3.0	3.6	3.1	2.2	2.3	2.7	3.0	2.6
18:1n9	6.9	20.5	9.1	27.5	27.7	15.8	23.5	18.7
18:2n6	1.8	54.3	1.5	57.0	53.5	27.3	54.6	27.4
18:3n6	—	—	.3	—	—	—	—	.2
18:3n3	T[c]	4.3	1.0	.9	2.1	1.0	3.3	1.4
18:4n3	—	—	—	—	—	—	—	1.4
20:0	.1	.4	.5	.1	.5	.3	.4	.5
20:1n9	—	.3	1.0	—	.3	—	.3	.7
20:2n6	—	—	.2	—	—	—	—	.1
20:3n6	—	—	.3	—	—	—	—	.1
20:3n3	—	—	.1	—	—	—	—	—
20:4n6	—	—	.9	—	—	—	—	.4
20:4n3	—	—	1.4	—	—	—	—	.4
20:5n3	—	—	15.1	—	—	—	—	7.8
22:0	—	.3	.2	—	.3	—	.3	.2
22:1n9	—	—	.2	—	—	—	—	.1
22:4n6	—	—	.2	—	—	—	—	.1
22:5n6	—	—	.4	—	—	—	—	.2
22:5n3	—	.14	2.8	—	—	—	—	1.4
22:6n3	—	—	10.9	—	—	—	—	5.6
24:1n9	—	—	.3	—	—	—	—	.1

[a]Expressed as percent of total fatty acids. Where SR = starch; CO = coconut oil; SO = soybean oil; FO = fish oil; CN = corn oil

[b]Source: White (1992).

[c]Traces detected, values less than .05%.

Table 3. Total saturated, monounsaturated and polyunsaturated fatty acids in oils and diets[a]

	Oils[b]				Diets			
Item[c]	CO	SO	FO	CN[d]	SR	CO	SO	FO
Total SFA	91.1	18.8	31.8	14.7	16.0	55.7	17.3	23.7
Total MUFA	6.9	21.1	24.0	27.6	28.2	15.8	24.1	26.8
Total PUFA	1.8	58.7	35.1	85.4	55.6	27.3	57.9	46.5
Total n-6	1.8	58.6	3.8	57.0	53.5	27.3	54.6	28.5
Total n-3	__	4.4	31.3	.9	2.1	1.0	3.3	18.0
Ratio n-3/n-6	__	.01	8.24	.02	.04	.04	.06	.63

[a]Expressed as percent of total fatty acids.
[b]Where CO = coconut oil; SO = soybean oil; FO = fish oil; SR = starch; CN = corn oil.
[c]Where SFA = saturated fatty acids; MUFA = monounsaturated fatty acids; PUFA = polyunsaturated fatty acids.
[d]Source: White (1992).

Table 4. Effects of dietary treatments on reproductive traits of gilts fed various sources of supplemental oil[a]

Item[c]	Treatments[b]				Pooled SEM
	SR[d]	CO	SO	FO	
Age at estrus, days	178.0	177.3	177.2	179.4	2.2
Wt at estrus, kg	110.5	112.8	111.2	109.3	2.4
Age at breeding, days	218.3	216.5	216.5	221.0	2.5
Wt at breeding, kg	129.1	128.1	127.7	127.7	2.3
Days of gestation	41.9	41.9	41.9	42.0	.6
Gestational wt gain, kg[ef]	20.5	21.6	18.9	18.6	1.0
Mean estrus cycle length[g]	20.2	19.8	19.6	20.8	.6
Wt at slaughter, kg[f]	149.7	149.9	146.7	146.5	2.4
No. of corpora lutea[f]	14.3	13.8	13.6	14.0	.5
Total no. of fetuses[f]	11.6	11.8	11.2	12.2	.6
Total fetuses alive[f]	11.0	11.1	10.4	11.8	.7
Fetal survival, %[f]	77.1	80.7	76.7	84.8	4.1

[a]Number of gilts on Trials 1 and 2 combined were: Eighteen gilts on SR diet, 22 gilts on CO, 21 gilts on SO diet, 19 gilts on FO diet.

[b]Where SR = starch; CO = coconut oil; SO = soybean oil; FO = fish oil.

[c]Treatment effects were not significant ($P < .10$).

[d]Isocaloric to the oil supplemented diets.

[e]Gestational wt gain = Slaughter wt - Wt at breeding.

[f]Least square means adjusted for days of gestation using covariate analysis.

Table 5. Effects of dietary treatments on fetus measurements from gilts fed various sources of supplemental oil[ab]

Item[d]	Treatments[c]				Pooled SEM
	SR	CO	SO	FO	
Placental weight, g[e]	77.1	76.6	79.5	69.5	4.0
Crown-rump length, cm	6.1	6.0	5.9	6.0	.1
Fetal weight, g	16.9	15.7	15.2	15.3	.8
Total fetal protein, mg/g (Wet basis)	37.9	35.3	34.5	35.4	1.3
Total fetal protein, mg/g (Dry basis)	351.8	319.7	311.7	315.1	12.1
Total placenta Protein, mg/g[e] (Wet basis)	13.4	12.0	12.9	14.0	1.1
Placental Protein, mg/g[e] (Dry basis)	121.6	108.0	130.4	118.7	14.5

[a]Number of gilts on Trials 1 and 2 combined were: Eighteen gilts on SR diet, 22 gilts on CO, 21 gilts on SO diet, 19 gilts on FO diet.

[b]LS means adjusted for days of gestation using covariate analysis.

[c]Where SR = starch; CO = coconut oil; SO = soybean oil; FO = fish oil.

[d]Treatment effects of were not significant ($P < .10$).

[e]Placental measurements from Trial 1 only.

[g]Mean estrus cycle length = (Age at breeding - Age at estrus)/2.

The fatty acid profile of plasma obtained from gilts at the time of slaughter was generally positively related to the fatty acid profile of the diet fed (Table 6). Gilts fed the CO diet had the highest level of 14:0 and 16:0, whereas, gilts fed the FO diet had the highest level of 16:1n-7, 20:5n-3, and 22:6n-3.

The total amount of saturated fatty acids (SFA) in the plasma was the largest in gilts fed the CO diet compared with the other diets (Table 7). Total monounsaturated fatty acids (MUFA) levels were lower ($P < .05$) in gilts fed SO and FO diets than for SR and CO diets. Total PUFA were not significantly different. The total n-6 level was lowest and the total n-3 level of fatty acids was highest for gilts fed the FO diet; thus, the n-3/n-6 ratio was the highest for the FO diet fed gilts. The ratio of n-3/n-6 fatty acids did not differ between gilts fed SR, CO, and SO diets.

Fatty acid levels in the conceptus tended to mirror those in the plasma; but differences were observed (Tables 8, 9, 10, and 11). As with plasma, the concentration of 14:0 and 16:0 were larger for the head portion of the pig conceptus from gilts fed the CO diet compared with those from gilts fed FO diet; but the concentrations of 14:0 and 16:0 in the body portion were intermediate and generally not different. Like plasma, the concentration of 20:5n-3 and 22:5n-3 in the head and body portion of the conceptus from

Table 6. Plasma fatty acid profile of pregnant gilts fed diets with various sources of supplemental oil[ab]

Fatty acid[e]	Treatments[cd] SR	CO	SO	FO	Pooled SEM
12:0	ND[f]	6.9	ND	ND	1.13
14:0	ND[g]	13.1[i]	1.7[g]	5.9[h]	.66
16:0	120.5[gh]	141.3[h]	98.3[g]	108.4[g]	7.90
16:1n7	9.4[g]	9.0[g]	9.1[g]	23.1[h]	1.76
17:0	4.2[gh]	4.8[g]	2.6[h]	4.9[g]	.45
18:0	63.7[gh]	70.9[g]	53.3[hi]	50.1[i]	3.72
18:1n9	111.5[g]	113.9[g]	70.1[h]	60.2[h]	5.40
18:2n6	119.5[g]	158.7[h]	162.4[h]	91.7[g]	7.33
18:3n6	2.1[g]	2.5[g]	1.1[h]	.9[h]	.12
18:3n3	2.3[g]	2.5[g]	3.9[h]	2.6[g]	.19
20:2n6	2.4[g]	2.8[g]	2.4[g]	ND[h]	.24
20:3n6	2.6[jk]	3.0[j]	1.7[jk]	1.4[k]	.42
20:4n6	42.8[g]	43.1[g]	34.9[g]	13.7[h]	2.38
20:5n3	1.5[g]	1.4[g]	ND[g]	46.6[h]	1.97
22:4n6	3.9[g]	4.3[g]	2.7[h]	ND[i]	.31
22:5n3	7.3[g]	7.0[g]	5.6[g]	16.2[h]	.63
22:6n3	2.3[g]	1.9[g]	.5[g]	17.1[h]	.82

[a]Expressed as milligram of lipid per milliliter of plasma.

[b]Least square means adjusted for days of gestation using covariate analysis.

[c]Where SR = starch; CO = coconut oil; SO = soybean oil; FO = fish oil.

[d]Six gilts per treatment, only from Trial 1.

[e]Traces of: 10:0, 20:0, also determined but not presented.

[f]ND = Not detected. A value of zero was assigned for the statistical analysis.

[g,h,i]Means with different superscripts within rows differ ($P < .05$).

[j,k]Means with different superscripts within rows differ ($P < .10$).

Table 7. Total saturated, monounsaturated, and polyunsaturated fatty acids in plasma of pregnant gilts fed the experimental diets[ab]

Item[e]	Treatments[cd] SR	CO	SO	FO	Pooled SEM
Total SFA	189[f]	237[g]	156[f]	169[f]	10.48
Total MUFA	119[f]	123[f]	79[g]	83[g]	5.76
Total PUFA	187	227	216	190	11.04
Total n-6	173[f]	215[g]	205[fg]	108[h]	9.42
Total n-3	13[f]	13[f]	10[f]	83[g]	3.15
Ratio n-3/n-6	.08[f]	.06[f]	.05[f]	.77[g]	.02

[a]Expressed as milligram of lipid per milliliter of plasma.

[b]Least square means adjusted for days of gestation using covariate analysis.

[c]Where SR = starch; CO = coconut oil; SO = soybean oil; FO = fish oil.

[d]Six gilts per treatment, only from Trial 1.

[e]Where SFA = saturated fatty acids; MUFA = monounsaturated fatty acids; PUFA = polyunsaturated fatty acids.

[f,g,h]Means with different superscripts within rows differ ($P < .05$).

Table 8. Fatty acid profile of the head portion of the pig conceptus from gilts fed various sources of supplemental oil[a]

Fatty acid	Treatments[bcd]				Pooled SEM
	SR	CO	SO	FO	
14:0	237[ef]	293[e]	184[ef]	112[f]	37.8
15:0	219[ef]	286[e]	191[fg]	125[g]	22.3
16:0	3791[ef]	4641[e]	3348[ef]	2398[f]	465.7
16:1n7	704	821	545	426	97.9
17:0	217[e]	271[e]	208[ef]	144[f]	16.2
18:0	2283	2470	2113	1527	249.5
18:1n9	3159[ef]	3766[e]	2642[ef]	2289[f]	344.3
18:2n6	121	197	147	127	22.5
20:1n9	54	68	30	31	13.2
20:2n6	248	345	180	244	46.4
20:3n6	29	38	26	44	14.0
20:4n6	1528[e]	1792[e]	1521[e]	695[f]	109.8
20:5n3	60[e]	99[e]	53[e]	189[f]	19.6
22:1n9	47	23	ND[h]	ND	25.5
22:4n6	430[e]	434[e]	458[e]	70[f]	29.9
22:5n3	102[e]	100[e]	78[e]	470[f]	33.2
22:6n3	559	752	671	642	137.1

[a]Expressed as micrograms of fatty acid per gram of tissue.

[b]Where SR = starch; CO = coconut oil; SO = soybean oil; FO = fish oil.

[c]Least square means adjusted for days of gestation using covariate analysis.

[d]Nine gilts per treatment, only from Trial 1.

[e,f,g]Means with different superscripts within rows differ ($P < .05$).

[h]ND = Not detected.

Table 9. Total saturated, monounsaturated, and polyunsaturated fatty acids in the head portion of the pig conceptus from gilts fed various sources of supplemental oil[ab]

Item[d]	Treatments[c]				Pooled SEM
	SR	CO	SO	FO	
Total SFA	6747[ef]	7961[e]	6043[ef]	4305[f]	773.7
Total MUFA	3964[ef]	4678[e]	3213[ef]	2745[f]	449.7
Total PUFA	3077	3758	3133	2481	314.3
Total n-6	2356[e]	2807[e]	2333[e]	1180[f]	196.6
Total n-3	721[e]	951[ef]	801[f]	1301[f]	129.9
Ratio n3/n6	.34[e]	.33[e]	.32[e]	1.10[f]	.05

[a]Expressed as microgram of fatty acid per gram of tissue.

[b]Least square means adjusted for days of gestation using covariate analysis.

[c]Where SR = starch; CO = coconut oil; SO = soybean oil; FO = fish oil. Nine gilts per treatment, only gilts from Trial 1.

[d]Where SFA = saturated fatty acids; MUFA = monounsaturated fatty acids; PUFA = polyunsaturated fatty acids.

[e,f]Means with different superscripts within rows differ ($P < .05$).

Table 10. Fatty acid profile of the body portion of pig conceptus from gilts fed various sources of supplemental oil[a]

Fatty acid	Treatments[bcd]				Pooled SEM
	SR	CO	SO	FO	
14:0	171[e]	148[e]	75[f]	125[ef]	17.1
15:0	337[e]	293[ef]	190[f]	228[ef]	34.3
16:0	3452	3309	2257	2555	330.8
16:1n7	757	743	450	556	82.8
17:0	487	488	354	352	67.1
18:0	2710	2927	2141	2130	378.9
18:1n9	3774	3631	2619	2666	383.1
18:2n6	335	348	328	322	47.4
20:1n9	44	16	18	12	14.8
20:2n6	253	253	129	205	33.4
20:3n6	68[ef]	44[e]	76[ef]	101[ef]	12.9
20:4n6	2642	2705	2214	1178	367.6
20:5n3	72[e]	33[e]	35[e]	465[f]	28.9
22:4n6	277[e]	235[e]	270[e]	23[f]	25.6
22:5n3	111[e]	78[e]	101[e]	317[f]	27.6
22:6n3	915	1073	712	1233	179.9

[a]Expressed as micrograms per gram of tissue.
[b]Where SR = starch; CO = coconut oil; SO = soybean oil; FO = Fish oil.
[c]Least square means adjusted for days of gestation using covariate analysis.
[d]Nine gilts per treatment, only from Trial 1.
[e,f,g]Means with different superscripts within rows differ ($P < .05$).

Table 11. Total saturated, monounsaturated, and polyunsaturated fatty acids in the body portion of the pig conceptus from gilts fed various sources of supplemental oil[ab]

Item[d]	Treatments[c]				Pooled SEM
	SR	CO	SO	FO	
Total SFA	7157	7155	5016	5390	796.4
Total MUFA	4576	4391	3086	3234	458.8
Total PUFA	4637	4774	4201	3850	651.5
Total n-6	3574	3586	3017	1829	442.3
Total n-3	1092[e]	1185[ef]	921[e]	2017[f]	215.9
ratio n-3/n-6	.32[e]	.32[e]	.27[e]	1.10[f]	.04

[a]Expressed as micrograms of fatty acid per gram of tissue.

[b]Least square means adjusted for days of gestation using covariate analysis.

[c]Where STR = starch; CO = coconut oil; SBO = soybean oil; FO = fish oil. Nine gilts per treatment, only from Trial 1.

[d]Where SFA = saturated fatty acids; MUFA = monounsaturated fatty acids; PUFA = polyunsaturated fatty acids.

[e,f]Means with different superscripts within rows differ ($P < .05$).

gilts fed the FO diets were much higher than for the other treatments. However, unlike plasma, the concentration of 16:1n-7 and 22:6n-3 were not influenced by dietary oil treatments. Also, similar to plasma, the concentrations of 20:4n-6 and 22:4n-6 in the conceptus from gilts fed the FO diet was much lower than those for the other treatments.

The total SFA, MUFA, and PUFA were generally slightly larger for the body portion of the pig conceptus compared with the head portion of the pig conceptus; however, the distribution of the fatty acids within each were generally similar but some differences will be noted later. The n-3/n-6 ratio was very similar for the body and head portions and the ratio was more than three-fold larger for gilts fed the FO diet than for those fed SR, CO, and SO diets which had similar ratios.

Discussion

Several authors have reported that during late gestation of the sow very small amounts of dietary fatty acids are transferred to the fetus (Thulin et al., 1989; Ramsay et al., 1990) but in our experiment the addition of 4% fish oil containing relatively high levels of n-3 fatty acids (31.3% of total fatty acids) to the basal corn-soybean meal diet increased the concentration of n-3 fatty acids in the plasma, and in the head and body portions of the conceptus compared with the conceptus from gilts fed starch (control), coconut oil, or soybean oil. Even though soybean oil contained a low level of n-3 fatty acids

(4.4% total fatty acids), the concentration of n-3 fatty acids in the plasma and conceptus were similar to those for gilts fed corn starch and coconut oil diets.

Linolenic acid derivatives eicosapentaenoic (20:5n-3) and docosahexaenoic (22:6n-3), are known to have an important role in the development of the nervous system of mammalian animals at prenatal and early stages of gestation (Sinclair and Crawford, 1972; Lamptey and Walker, 1976; Neuringer and Connor, 1986); and consequently may be related to fetal loss. Although, in our studies, the concentrations of omega-3 fatty acids were increased for gilts fed the fish oil diet, reproductive and fetal and placental measurements were not improved compared with the SR or other treatments. Number of ovulations and fetal survival were high for all gilts and this may have been a factor masking the effect of omega-3 fatty acids. Although differences for fetal survival across treatments were not significant, embryo survival in Trial 2 was higher ($P < .05$) for gilts fed fish oil, compared with the controls, 91.2 vs 78.5%, respectively. The level of PUFA in all diets was considered high. This high level of PUFA may explain the unexpectedly high levels of linoleic and linolenic acid in tissues of gilts and fetuses and may also explain the high fetal survival rates in all treatments.

The fatty acid profile of the soybean oil and that of the soybean oil supplemented diet were similar in polyenoic acid content, and n-3/n-6 fatty acid ratio. In contrast, fish oil had a higher concentration of omega-3 fatty acids and a higher

n-3/n-6 ratio for the fish oil supplemented diet in which both concentration and ratio were lower. The polyenoic acid content in saturated oils like coconut oil were known to be minimal and the n-3/n-6 fatty acid cannot be calculated.

Although evidence on the improved embryo/fetal survival properties of certain types of dietary PUFA such as linoleic acid (18:2n-6) fed during the first weeks of gestation has been reported before (Fengler, 1990), to our knowledge this is the first study evaluating fetal survival in swine for the omega-3 fatty acid linolenic (18:3n-3). The results of our experiment suggest that the slight improvement in embryo survival can be related to fatty acids of the omega-3 family. The markedly higher concentration of docosahexaenoic acid from all maternal and fetal tissues analyzed in all the treatments provides indirect support that docosahexaenoic acid plays an important role in the developing fetus and consequently docosahexaenoic acid may contribute to improved fetal survival. There is indirect evidence that mammalian fetus have the ability to elongate linolenic (18:3n-3) fatty acid to longer chain derivatives such as eicosapentaenoic (20:5n-3), eicosahexaenoic (20:6n-3) and docosahexaenoic (22:6n-3) acids, (Purvis et al., 1983) however, the source of linolenic acid in this study, soybean oil, did not serve to provide the same results as the source of the longer chain omega-3 derivatives, fish oil. The developing fetus may not, therefore, have the ability to elongate linolenic acid (18:3n-3) and may not derive the benefit from soybean oil that it

may, from fish oil. Linolenic acid is the major fatty acid in lipids of plant origin whereas longer chain derivatives of the omega-3 family (docosahexaenoic and eicosapentaenoic acids) are rich in lipids of fish, therefore fish oil may be necessary supplying docosahexaenoic and eicosapentaenoic acids. Further research is necessary to investigate the role of docosahexaenoic acid in the development of the fetal nervous system and the possible influence of docosahexaenoic acid on fetal survival in swine.

Summary and Implications

In summary, maternal dietary fat influences the fatty acid profile of tissues of the developing fetus. The markedly higher concentration of omega-3 fatty acids in fetal tissues support the hypothesis that omega-3 fatty acids play an important role in the developing fetus. While some evidence existed of benefit due to fish oil, the high percent survival observed in all the treatments precluded from conclusions relative to the supplemental oil. The fatty acid composition of the corn-soybean diet used in the experiment may have contained sufficient amounts of essential fatty acids necessary for the normal development of the fetus.

Chapter IV

LITERATURE CITED

Aaes-Jørgensen, E. and G. Holmer. 1969. Essential fatty acid and deficient rats. I. Growth and testes development. Lipids 4:501.

Akessom, B. 1969. The acylation of diacylglycerols in pig liver. Eur. J. Biochem. 9:406.

Allee, G. L., D. Romsos., G. A. Leveille, and D. H. Baker. 1971a. Influence of age on "in vitro" lipid biosynthesis and enzymatic activity in pig adipose tissue. Proc. Soc. Exp. Biol. Med. 137:449.

Allee, G. L., E. K. O'Hea, G. A. Leveille, and D. H. Baker. 1971b. Influence of dietary protein and fat on lipogenesis and enzymatic activity in pig adipose tissue. J. Nutr. 101:869.

Allee, G. L., D. R. Romsos, G. A. Leveille, and D. H. Baker. 1972. Lipogenesis and enzymatic activity in pig adipose tissue as influences by source of dietary fat. J. Anim. Sci. 35:41.

Allee, G. L., B. A. Koch and R. H. Hines. 1976. Effect of fat level and calorie:protein ratio on performance of finishing pigs. J. Anim. Sci. 42:1349.

Allen, C. E. and E. A. Foegeding. 1981. Some lipid characteristics and interactions in aviary, aquatic and mammalian muscle foods. A review. Food Technol. 35:253.

Anderson, D. B., R. G. Kauffman, and L. L. Karstenschmidt. 1972. Lipogenic enzyme activities and cellularity of porcine adipose tissue from various anatomic locations. J. Lipid Res. 13:593.

Anderson, D. B., and R. G. Kauffman. 1973. Cellular and enzymatic changes in porcine adipose tissue during growth. J. Lipid Res. 14:160.

Anderson. L. L. 1975. Embryonic and placental development during prolonged inanition in the pig. Am. J. Physiol. 229:1687.

Artman, N. R. 1964. Interactions of fats and fatty acids as energy sources for the chick. Poultry Sci. 43:994.

Barter, P. J., G. J. Hopkins, and G. D. Calvert. 1982. Pathways for the incorporation of esterified cholesterol into very low density lipoproteins in plasma incubated "in vitro". Biochem. Biophys. Acta. 713:136.

Bauman, D., R. E. Brown, and C. L. Davis. 1970. Pathways of fatty acid synthesis and reducing equivalent generation in mammary gland of rat, sow, and cow. Arch. Biochem. Biophys. 140:237.

Bazer, F. W., O. W. Robison, A. J. Clawson, and L. C. Ulberg. 1969. Uterine capacity at two stages of gestation in gilts following embryo superinduction. J. Anim. Sci. 29:30.

Bell, R. M. and R. A. Coleman. 1980. Enzymes of glycerolipid synthesis in eukaryotes. Annu. Rev. Biochem. 49:459.

Bensadoun, A., C. Ehnholm., D. Steinberg, and W. V. Brown. 1974. Purification and characterization of lipoprotein lipase from pigs adipose tissue. J. Biol. Chem. 249:2220.

Bimbo, A. P. 1990. Production of fish oil. In: M. E. Stansby (Ed). Fish Oils in Nutrition. Van Nostrand Reinhold. New York.

Bourre, J. M., D. Georges, P. Gerard and A. Youyou. 1989. Brain cell and tissue recovery in rats made deficient in n-3 fatty acids by alteration of dietary fat. J. Nutr. 119:15.

Brooks, C. C. 1971. Fatty acid composition of pork lipids as affected by basal diet, fat source and fat level. J. Anim. Sci. 33:1224.

Burr, G. O. and M. M. Burr. 1929. A new deficiency disease produced by the rigid exclusion of fat from the diet. J. Biol. Chem. 82:345.

Busboom, J. R., D. C. Rule, D. Colin, T. Heald, and A. Mazhar. 1991. Growth, carcass characteristics and lipid composition of adipose tissue and muscle of pigs fed canola. J. Anim. Sci. 69:1101.

Carew, T. E., R. P. Saik., K. H. Johansen., C. A. Dennis, and D. Steinberg. 1976. Low density and high density lipoprotein turnover following portacaval shunt in swine. J. Lipid Res. 17:441.

Century, B., L. A. Witting, C. C. Harvey, and M. K. Horwitt. 1963. Interrelationships of dietary fatty lipids upon fatty acid composition of brain mitochondria, erythrocytes and heart tissue in chicks. Am. J. Clin. Nutr. 13:362.

Christensen, K. and V. D. Goel. 1972. The influence of various glucose and insulin concentrations on "in vitro" lipid synthesis by adipose tissue isolated from adult pigs. Int. J. Biochem. 3:591.

Churchill, J. A., M. A. Ayres, D. F. Caldwell, and A. Melvern. 1967. Intelligence of children whose mothers have biliary tract disease. J. Anim. Med. Assoc. 201:482.

Clandinin, M. T., J. E. Chappell, S. Leong, T. Haim, P. R. Swyer, and G. W. Chance. 1980. Extrauterine fatty acid accretion rates in human brain: implications for fatty acid requirements. Early Human Dev. 4:121.

Crawford, M. A., A. G. Hassam, G. Williams, and Whitehouse. 1976. Essential fatty acids and fetal brain growth. Lancet. 1:452.

Cryer, A., J. Kirtland., H. M. Jones, and M. I. Gurr. 1978. Lipoprotein lipase activity in the tissues of guinea pigs exposed to different dietary fats from conception to three months of age. J. Biochem. 170:169.

Culp, B. R., B. G. Titus, and W. E. M. Lands 1979. Inhibition of prostaglandin biosynthesis by eicosapentaenoic acid. Prostaglandins. Med. 3:269.

Culp, B. R., E. E. M. Lands., B. R. Lucchesi, B. Pitt, and J. Romson. 1980. The effect of dietary supplementation of fish oil on experimental myocardial infarction. Prostaglandins 20:1021.

Cunnane, S. C. 1984. Essential fatty-acid/mineral interaction with reference to the pig. In: J. Wiseman (Ed).Fats in Animal Nutrition. p 167. Butterworths. London. U. K.

Dahl, O., and K. Personn. 1965. Properties of animal depot fat in relation to dietary fat. J. Sci. Food Agric. 16:452.

Day, B. N. 1979. Embryo transfer in swine. Theriogenology 11:27.

Dib, A., and J. P. Carreau. 1987. Effects of gamma-linolenic acid supplementation on pregnant rats fed a zinc-deficient diet. Ann. Nutr. Metab. 31:312.

Dick, G. W. and J. N. Strain. 1983. Postmating feeding level effects on conception rate and embryonic survival in gilts. Can. J. Anim. Sci. 63:579.

Driss, F. E., M. Vericel, M. Lagarde, S. S. Dechavanne, and P. Darcet. 1981. Inhibition of plateletaggregation and thromboxane synthesis after intake of small amount of eicosapentaenoic acid. Thromb. Res. 36:389.

Dutler, H., A. Kull, and R. Mislin 1971. Fatty acid synthetase from pig liver. II. Characterization of the enzyme complex with oxidoreductase activity for alicyclic ketones as a fatty acid synthetase. Eur. J. Biochem. 22:213.

Dutton, H. J. 1981. History of the development of soy oil for edible uses. J. Am. Oil Chem. Soc. 58:234.

Dyerberg, J. 1986. Linolenate-derived polyunsaturated fatty acids and prevention of atherosclerosis. Nutr. Rev. 44:125.

Einset, E., H. S. Olcott, and M. E. Stansby. 1957. Oxidative deterioration in fish and fishery products. IV. Progress in Studies concerning oxidation of extracted oils. Com. Fish Rev. 19:35.

Enser, M., E. Dransfield, P. D. Jolley, R. C. D. Jones, and M. Leedmann. 1984. The composition and consistency of pig back fat as it affects the quality of vacuum-packed rindless bacon rashes. J. Sci. Food Agric. 34:1230.

Etherton, T. D. and C. E. Allen. 1980. Effects of age and adipocyte size on glucose and palmitate metabolism and oxidation in pigs. J. Anim. Sci. 50:1073.

Etherton, T. D. and C. S. Chung. 1981. Preparation, characterization, and insulin sensitivity of isolated swine adipocytes: comparison with adipose tissue slices. J. Lipid Res. 22:1053.

Fain, J. N. 1973. Biochemical aspects of drug and hormone action on adipose tissue. Pharmacol. Rev. 25:67.

Fayard, J. M., L. Timonyasse, P. Guesnet, G. Durand, G. Pascal, and C. Laugier. 1990. Dietary alpha-linolenic acid deficiency and early uterine development in female rats. J. Nutr. 122:1529.

Fengler, A. I., S. K. Baidoo, and F. X. Aherne. 1990. Improving embryo survival by dietary supplementation of oils in early gestation. 69th Annual U. of Alberta Feeders Day Report, p 7.

Fiennes, R. N. T. W., A. I. Sinclair, and M. A. Crawford. 1973. Essential fatty acid studies in primates. Linolenic acid requirements in capuchins. J. Med. Prim. 2:155.

Freeman, C. P., D. E. Noakes, and E. F. Annison. 1970. The metabolism of glucose, acetate, palmitate, stearate and oleate in pigs. Br. J. Nutr. 24:705.

Galli, C., H. T. Trzeciack and R. Paoletti. 1972. Effects of essential fatty acid deficiency on myelin and various subcellular structures in rat brain. J. Neurochem. 19:1863.

Garton, G. A, and W. R. H. Duncan. 1954. Dietary fat and body fat: The composition of the back fats of pigs fed on a diet rich in cod-liver oil and lard. J. Biochem. 57:120.

Geisert, R. D., R. J. Rasby, J. E. Minton, and R. P. Wettemann. 1986. Role of prostaglandins in development of porcine blastocysts. Prostaglandins 31:191.

Gore, A., E. T. Kornegay, and H. P. Veit. 1986. The effects of soyabean oil on nursery air quality and performance of weaning pigs. J. Anim. Sci. 63:1.

Guesnet P., G. Pascal, and G. Durand. 1986. Effects of alpha-linolenic acid deficiency on reproduction and growth in rats. Prog. Lipid Res. 25:391.

Hahn, P. 1972. Lipid metabolism and nutrition in the prenatal and postnatal period. In: A. Winck (Ed). Nutrition and Develpoment. P 99. Wiley and Son, New York.

Hahn, P, and M. Novak. 1975. Development of brown and white adipose tissue. J. Lipid Res. 16:79.

Hammond, E. G. and W. R. Fehr. 1984. Improving the fatty acid composition of soybean oil. J. Am. Oil Chem. Soc. 61:1713.

Hannan, S. F., N. Baker., H. Rostami., K. Lewis, and P. J. Scott. 1980. Very low density lipoprotein metabolism in domestic pigs. Atherosclerosis 37:55.

Hansen, A. E., M. F. Wiese, D. J. D. Adam, A. N. Boelsche, M. E. Haggard, H. Davis, W. T. Newsom, and Le Pegut 1964. Influence of diet on blood serum lipids in pregnant women and newborn infants. Am. J. Clin. Nutr. 15:11.

Hanson, L. J., D. K. Sorensen, and H. C. H. Kernkamp. 1958. Essential fatty acid deficiency: Its role in parakeratosis. Am. J. of Vet. Res. 30:921.

Hartog, L. A. and G. J. M. Kemper 1980. Relationship between nutrition and fertility in pigs. Neth. J. Agric. Sci. 28:211.

Harper, A. F. 1992. An Assessment of the Effects of Dietary Folic Acid Supplementation on Serum Folates Status, Conceptus Development and Reproductive Performance in Gilts and Sows. Ph.D. Dissertation. Virginia Polytechnic Institute and State University.

Hassam, A. G. and M. A. Crawford. 1976. The differential incorporation of labelled linoleic, gamma linolenic, dihomo-gamma-linolenic and arachidonic acids into the developing rat brain. J. Neurochem. 27:967.

Hausman, G. J. 1982. Histochemically detectable lipoprotein lipase activity in adipose tissue of pigs and normal and decapitated pig fetuses. Acta Anat. 114:281.

Hawton, J. D. and R. J. Meade. 1971. Influence of quantity and quality of protein fed the gravid female on reproductive performance and development of offspring in swine. J. Anim. Sci. 32:88.

Higashi, H. 1961. Vitamins in fish, with special reference to edible parts. In: G. Borgstrom (Ed). Fish as Food. Vol 1. p 411. Academic Press, New York.

Hill, E. G., E. L. Warmanen, H. Hayes, and R. T. Holman. 1957. Effects of essential fatty acid deficiency in young swine. Proc. Soc. Exp. Biol. Med. 95:274.

Hill, E. G., E. L. Warmanen, C. L. Silbernick, and R. T. Holman. 1961. Essential fatty acid nutrition in swine. I. Linoleate requirement estimated from triene:tetraene ratio of tissue lipids. J. Nutr. 74:335.

Himms-Hagen, J. 1970. Adrenergic receptors for metabolic responses in adipose tissue. Fed. Proc. Fed. Am. Soc. Exp. Biol. 29:1388.

Hoffmann, P. and W. Forster. 1986. Antihypertensive effect of dietary sunflowerseed oil and linseed oil in spontaneously hypertensive rats during a multigeneration feeding study. Prost. Leukotr. Med. 25:65.

Holman, R. T. and H. Mohrhauer. 1963. A hypothesis involving competitive inhibitions in the metabolism of polyunsaturated fatty acids. Acta. Chem. Scand. 17:S84.

Holman, R. T. 1964. Nutritional and metabolic interrelationships between fatty acids. Fed. Proc. Fed. Am. Soc. Exp. Biol. 23:1062.

Holman, R. T. 1978. Essential fatty acid deficiency in animals. In: CRC handbook series nutrition and food, Vol 2, Section E, CRC Press. West Palm Beach. Fla.

Holman, R. T., S. B. Johnson, and T. F. Hatch. 1982. A case of human linolenic acid deficiency involving neurological abnormalities. Am. J. Clin. Nutr. 35:617.

Horn, G. W., C. Foley, R. W. Seerley, and J. F. Munnell. 1973. Role of adipose tissue lipolysis in postnatal amelioration of thermogenesis in domestic and wild piglets. J. Anim. Sci. 37:1356.

Howard, A. N., W. M. Leat, G. A. Gresham, W. D. Bowyer, and E. R. Dalton. 1965. Studies on pigs reared on semisynthetic diets containing no fat, beef tallow or maize oil: Husbandry and serum biochemistry. Br. J. Nutr. 19:383.

Howard, K. A., D. M. Forsyth and T. R. Cline. 1987. Effects of soybean oil in swine starter diets. J. Anim. Sci. 65 (suppl.1): 294 (Abstr).

Huang, W. Y. and F. A. Kummerow. 1976. Cholesterol and fatty acid synthesis in swine. Lipids 11:34.

Huang, W. Y. and F. A. Kummerow. 1982. Incorporation of fatty acids into swine liver microsomes. Biochem. Med. 28:70.

Imoto, S. and S. Namioka. 1983. Acetate-glucose relationship in growing pigs. J. Anim. Sci. 56:867.

Johansson, M. B. N. and B. W. Karlsson. 1982. Lipoprotein and lipid profiles in the blood serum of the fetal, neonatal and adult pig. Biol. Neonate. 42:127.

Jungas, R. L. and E. G. Ball. 1963. Studies on the metabolism of adipose tissue. XII. The effects of insulin and epinephrine on free fatty and glycerol production in the presence and absence of glucose. Biochem. 2:383.

Kaizer, L., N. F. Boyd, V. Kriukor, and D. Tritchter. 1989. Fish consumption and breast cancer risk: an ecological study. Nutr. Cancer 12:61.

Karrick, N. L. 1990. Nutritional value of fish oils as animal fed. In: M. E. Stansby (Ed). Fish Oils in Nutrition. AVI book publishing. Van Nostrand Reinhold. New York.

Kasser, T. R., J. H. Gahagan, and R. J. Martin. 1982. Fetal hormones and neonatal survival in response to altered maternal serum glucose and free fatty acid concentrations in pigs. J. Anim. Sci. 55:1351.

Kasser, T. R., R. J. Martin, and C. E. Allen. 1981. Effect of gestational alloxan diabetes and fasting on fetal lipogenesis and lipid deposition in pigs. Biol. Neonate 40:105.

Kasser, T. R., G. J. Hausman, D. R. Campion, and R. J. Martin. 1983. Lipogenesis and pancreatic insulin release in fetal pigs. J. Anim. Sci. 56:579.

Kinsella, J. E. 1987. Components affecting the safety of fish oils. In: J. E. Kinsella (Ed). Seafoods and Fish Oils in Human Health and Disease. Marcel Dekker, Inc. New York.

Kinsella, J. E., B. Lokesh, S. Broughton, and J. Whelan. 1990. Dietary polyunsaturated fatty acids and eicosanoids: potential effects on the modulation of inflammatory and immune cells: an overview. Nutrition 6:24.

Krausser, M. V., L. K. Mahan. 1979. In: W. B. Saunders (Ed). Nutrition and diet therapy (6^{th} ed). Philadelphia.

Lambert, E., D. H. Williams, P. B. Lynch, T. J. Hanrahan, T. A. McGready, F. H. Austin, M. P. Boland, and J. H. Roche. 1991. The extent and timing of prenatal loss in gilts. Theriogenology 36:655.

Lamptey, M. S. and B. L. Walker. 1976. A possible essential role for dietary linolenic acid in the development of the young rat. J. Nutr. 106:86.

Landers, R. E., and D. M. Rathman. 1981. Vegetable oils: effects of procesing, storage and use on nutritional value. J. Am. Oil Chem. Soc. 58:255.

Lee, Y. B. and R. G. Kauffman. 1974. Cellular and enzymatic changes with animal growth in porcine intramuscular adipose tissue. J. Anim. Sci. 38:532.

Lodge, G. A., F. W. H. Elsley, and R. M. MacPherson. 1966. The effects of level of feeding of sows during pregnancy: I. Reproductive performance. Anim. Prod. 8:29.

Malm, A., W. G. Pond, E. J. Walker Jr, A. Aydin, D. Kirtland. 1976. Effect of polyunsaturated fatty acids and vitamin E level of the sow gestation diet on reproductive performance and on level of alpha-tocopherol in colostrum, milk, and dam and progeny blood serum. J. Anim. Sci. 42:393.

Marshall, L. A., A. Szczesniewski, and P. V. Johnston. 1983. Dietary alpha-linolenic acid and prostaglandin synthesis: a time course study. Am. J. Clin. Nutr. 38:895.

Martin, R. J. and J. H. Herbein. 1976. A comparison of the enzyme levels and the "in vitro" utilization of various substrates for lipogenesis in pair-fed lean and obese pigs. Proc. Soc. Exp. Biol. Med. 151:231.

Mascioli, E. A., M. F. Smith, M. S. Trerice, H. C. Meng, and G. L. Blackburn. 1979. Effect of total parenteral nutrition with cycling on essential fatty acid deficiency. J. Parent. Ent. Nutr. 3:171.

Mead, J. F., R. B. Alfin-Slater, D. R. Norton, and George Popjau. 1986. Nutritional value of lipids. In: J. F. Med (Ed). Lipids, Chemistry, Biochemistry, and Nutrition. Plenum Press. New York.

Mersmann, H. J., J. M. Houk, G. Phinney, M. C. Underwood, and L. J. Brown. 1973a. Lipogenesis by "in vitro" liver and adipose tissue preparations from neonatal swine. Am. J. Physiol. 224:1123.

Mersmann, H. J., M. C. Underwood, L. J. Brown, and J. M. Houk. 1973b. Adipose tissue composition and lipogenic capacity in developing swine. Am. J. Physiol. 224:1130.

Mersmann, H. J., J. M. Houk, G. Phinney, and M. C. Underwood. 1973c. Effect of diet and weaning age on "in vitro" lipogenesis in young swine. J. Nutr. 103:821.

Mersmann, H. J., L. J. Brown, M. C. Underwood, and H. C. Stanton. 1974. Catecholamine-induced lipolysis in swine. Comp. Biochem. Physiol. 47B:263.

Mersmann, H. J., G. Phinney, and L. J. Brown. 1975. Ontogeny of epinephrine-induced lypolisisin adipose tissue from swine *(sus domesticus)*. Gen. Pharmacol. 6:187.

Mersmann, H. J., C. D. Allen, D. G. Steffen, L. G. Brown, and D. M. Danielson. 1976. Effect of age, weaning and diet on swine adipose tissue and liver lipogenesis. J. Anim. Sci. 43:140.

Mersmann, H. J. 1984. Adrenergic control of lipolysis in swine adipose tissue. Comp. Biochem. Physiol. 77C:43.

Mertin, J. 1981. Essential fatty acids and cell-modulated immunity. Prog. Lipid Res. 20:851.

Meydani, S. N. , A. H. Lichtenstein, P. J. White, S. H. Goodnight, C. E. Elson, M. Woods, S. L. Gorbarch, and E. J. Schaefer. 1991. Food use and health effects of soyabean and sunflower oils. J. Am. Coll. Nutr. 10:406.

Miller, G. M., J. H. Conrad, T. W. Keenan, and W. R. Featherston. 1971. Fatty acid oxidation in young pigs. J. Nutr. 101:1343.

Mohrhauer, H. and R. Holman. 1963. Alteration of the fatty acid composition of brain lipids by varying levels of dietary essential fatty acids. J. Neurochem. 10:523.

Naughton, J. M. 1981. Supply of polyenoic fatty acids to the mammalian brain: the ease of conversion of the short-chain essential fatty acids to their longer chain polyunsaturated metabolities in liver, brain, placenta, and blood. Int. J. Biochem. 13:21.

Needleman, P., A. Raz, M. S. Minkes, J. A. Ferrendeli, and H. Sprecher. 1979. Triene prostaglandins: prostacyclin and thromboxane biosynthesis and unique biological properties. Proc. Natl. Acad. Sci. U.S.A. 76:944.

Neuringer, M., W. E. Connor, C. V. Petten, and L. Barstad. 1984. Dietary omega-3 fatty acid deficiency and visual loss in infant rhesus monkeys. J. Clin. Invest. 73:272.

Neuringer, M, and W. E. Connor. 1986. n-3 fatty acids in the brain and retina: evidence for their essentiality. Nutr. Res. 44:285.

Nicolaides, N. and A, N. Woodall. 1962. Impaired pigmentation in chinook salmon fed diets deficient in essential fatty acids. J. Nutr. 78:431.

Noblet, J. and J. Le Dividich. 1981. Energy metabolism of the newborn pig during the first 24 hr of life. Biol. Neonate 40:175.

NRC. 1988. Nutrient Requirements of Swine (9th). National Academic Press, Washington, DC.

O'Brien, J. S., D. L. Fillerup, and J. F. Mead. 1964. Quantification and fatty acid aldehyde composition of ethanolamine choline and serine glycerophosphatides in human cerebral grey and white matter. J. Lipid Res. 5:329.

O'Hea, E. K. and Leveille, G. A. 1969. Significance of adipose tissue and liver as sites of fatty acid synthesis in the pig and the efficiency of utilization of various substrates for lipogenesis. J. Nutr. 99:338.

Pearce, J. 1983. Fatty acid synthesis in liver and adipose tissue. Proc. Nutr. Soc. 42:263.

Pettigrew, J. E. Jr. 1981. Supplemental dietary fat for peripartal sows: A review. J. Anim. Sci. 53:107.

Pope, W. F., R. R. Maurer, and F. Stormshak. 1982. Intrauterine migration of the porcine embryo- interaction of embryo, uterine flushings and indomethacin on myometrial function "in vitro". J. Anim. Sci. 55:1169.

Pope, W. F. and N. L. First. 1985. Factors affecting the survival of pigs embryos. Theriogenology 23:91.

Privett, O. S., F. J. Push, R. T. Holman, and N. O. Lundberg. 1960. Essential fatty acids properties of tuna, herring, and menhaden oils. J. Nutr. 71:66.

Privett, O. S., and R. Cortesi. 1972. Observations on the role of vitamin E in the toxicity of oxidized fats. Lipids 7:780.

Purvis, J. M., M. T. Clandinin, and R. R. Hacker. 1983. Chain elongation-desaturation of linoleic acid during the development of the pig. Implications for the supply of polyenoic fatty acids to the developing brain. Comp. Biochem. Physiol. 75:199.

Raju, P. K. and D. M. Six. 1975. Glyceride biosynthesis in swine adipose tissue microsomes. Comp. Biochem. Physiol. 51B:171.

Ramsay, T. G., J. Karousis, M. E. White and C. K. Wolverton. 1991. Fatty acid metabolism by the porcine placenta. J. Anim. Sci. 69:3645.

Renner, R., and F. W. Hill. 1958. Metabolizable energy values of fats and fatty acids to chickens. Proc. Cornel Nutrition Conf. for Feed Manuf. p 95.

Romsos, D. R., G. A. Leveille, and G. L. Alle. 1971. "in vitro" lipogenesis in adipose tissue from alloxan-diabetic pigs (Sus domesticus). Comp. Biochem. Physiol. 40A:569.

Romsos, D. R. and G. A. Leveille. 1974. Effect of diet on activity of enzymes involved in fatty acid and cholesterol synthesis. Adv. Lipid Res. 12:97.

Samulski, M. A. and B. L. Walker. 1982. Maternal dietary fat and polyunsaturated fatty acids in the developing foetal rat brain. J. Neurochem. 39:1163.

Sanders, T. A. B., M. Mistry, and D. J. Naismith. 1984. The influence of a maternal diet rich in linoleic acid on brain and retinal docohexaenoic acid in the rat. Br. J. Nutr. 51:57.

Sanders, T. A. B., and K. M. Younger. 1986. The effect of dietary supplements of omega-3 fatty acids on the fatty acid composition of platelets and plasma choline phosphoglycerides. Br. J. Nutr. 45:613.

SAS. 1989. SAS/STAT User's Guide, Version 6, Fourth Edition. Volume 2. SAS Institute Inc. Cary, NC 27513.

Schlossman, D. M. and R. M. Bell. 1976. Triacylglycerol synthesis in isolated fat cells. J. Biol. Chem. 251:5738.

Scofield, A. 1972. Embryonic mortality. In pig Production. D. J. A. Cole (Ed). p 367. Butterworths. London. U.K.

Seerly, R. W. 1984. The use of fat in sow diets. In: J. Wiseman (Ed). Fats in Animal Nutrition. p 333. Butterworths. London. U.K.

Sinclair, A. J., and M. A. Crawford. 1972. The accumulation of arachidonate and docohexanoate in the developing rat brain. J. Neurochem. 19:1753.

Sinclair, A. J. 1975. The incorporation of radioactive polyunsaturated fatty acids into the liver and brain of the developing rat. Lipids 10:175.

Stahly, T. S. 1984. Use of fats in diets for growing pigs. In: J. Wiseman (Ed). Fats in Animal Nutrition. p 313. Butterworths. London. U.K.

Stansby, M. E. 1982. Properties of fish oils and their application to handling of fish and to nutritional and industrial use. In: R. E. Martin., G. J. Flick., C. E. Hebard, and D. R. Ward (Ed). Chemistry and Biochemistry of Marine Food products. p 75. AVI Publishing Co; West Port, CT.

Stansby, M. E.. 1990. Nutritional properties of fish oil for human consumption. In: M. E. Stansby (Ed). Fish Oils in Nutrition. p 268. Van Nostrand Reinhold, New York.

Steele, N. C., L. T. Frobish, and M. Keeney. 1974. Lipogenesis and cellularity of adipose tissue from genetically lean and obese swine. J. Anim. Sci. 39:712.

Steffen, D. G., E. Y. Chai, L. J. Brown, and H. J. Mersmann. 1978. Effects of diet on swine glyceride lipid metabolism. J. Nutr. 108:911.

Steffen, D. G., L. J. Brown, and H. J. Mersmann. 1978. Ontogenic development of swine (Sus domesticus) adipose tissue lipases. Comp. Biochem. Physiol. 59B:195.

Steffen, D. G., G. Phinney, L. J. Brown and H. J. Mersmann. 1979. Ontogeny of glycerolipid biosynthetic enzymes in swine liver and adipose tissue. J. Lipid Res. 20:246.

Steffen, D. G., M. C. Arakelian, G. Phinney, L. J. Brown, and H. J. Mersmann. 1981. Effect of nutritional status on swine adipose tissue lipolytic activities. J. Anim. Sci. 52:1306.

Stryer, L. 1988. Fatty acid metabolism. In: L. Stryer (Ed). Biochemistry (3th ed). W. H. Freeman and Company. New York.

Sundler, R. and B. Akesson. 1970. The acylation of monoacylglycerol isomers by pig liver microsomes. Biochem. Biophys. Acta. 218:89.

Swiatek, K. R., D. M. Kipnis, G. Mason, K. L. Chao, and M. Cornblath. 1968. Starvation hypoglycemia in newborn pigs. Am. J. Physiol. 214:400.

Thulin, A. J., G. L. Allee, D. L. Harmon and D. L. Davis. 1989. Utero-placental transfer of octanoic, palmitic and linoleic acids during late gestation in gilts. J. Anim. Sci. 67:738.

Tinoco, J., M. A. Williams, I. Hincenbergs, and R. L. Lyman. 1971. Evidence for non-essentiality of linolenic acid in the diet of the rat. J. Nutr. 101:937.

Tinoco, J., R. Babcock, I. Hincenbergs, B. Medwadowski, and P. Miljanich. 1977. Linolenic acid deficiency: changes in fatty acid patterns in female and male rats raised on a linolenic acid deficient diet for two generations. Lipids. 13:6.

USDC 1988. Fisheries of the United States, 1987. Current Fishery Statistics No. 8700.

Vaughan, V. S., J. V. Merriner, and J. W. Smith. 1988. The U.S. menhaden fishery: Current status and utilization. In: N. Davis (Ed). Fatty Fish Utilization: Upgrading from Feed to Food. p 15. Raleigh: University of North Carolina Sea Grant.

Vernon, R. G. 1980. Lipid metabolism in the adipose tissue of ruminant mammals. Prog. Lipid Res. 19:23.

Wainwright, P. E., Y. S. Huang, B. Bulman-Fleming, D. Dalby, D. E. Mills, P. Redden, and D. McCutcheon. 1992. The effects of dietary n-3/n-6 ratio on brain development in the mouse; a close response study with long chain n-3 fatty acids. Lipids 27:98.

Watanabe, T. 1982. Lipid Nutrition. Fish Comp. Biochem. Physiol. 73B:3.

Waterman, R. A., D. R. Romsos, A. C. Tsai, E. R. Miller, and G. A. Leveille. 1975. Influnce of dietary safflower oil and tallow on growth, plasma lipids and lipogenesis in rats, pigs and chicks. Proc. Soc. Exp. Biol. Med. 150:347.

Watkins, B. A., and F. H. Kratzer. 1987. Dietary biotin effects on polyunsaturated fatty acids in chick tissue lipids and prostaglandin E_2 levels in freeze-clamped hearts. Poult. Sci. 66:1818.

Watkins, B. A. 1991. Importance of essential fatty acids and their derivatives in poultry. J. Nutr. 121:1474.

Webel, S. K. and P. J. Dziuk. 1974. Effect of stage of gestation and uterine space on prenatal survival in the pig. J. Anim. Sci. 38:960

Webel, S. K., T. J. Reimers and P. J. Dziuk. 1975. The lack of relationship between plasma progesterone levels and number of embryos and their survival in the pig. Biol. Reprod. 13:177.

Werner, J. C., V. Whitman, R. R. Fripp, H. G. Schuler, J. Musselman, and R. L. Sham. 1983. Fatty acid and glucose utilization in isolated, working fetal pig hearts. Am. J. Physiol. 245:E19.

Wheeler, A. 1985. In: Fishes. A. Wheeler (Ed). The world Encyclopedia of Fishes. p 139. McDonald Book. London. U.K.

White, P. J. 1992. Fatty acids in Oilseeds (vegetable oils). In: C. K. Chow (Ed). Fatty Acids in Foods and Their Health Implications. Marcel Dekker, Inc. New York.

Whitehead, C. C. 1984. Essential fatty acids in poultry nutrition. In: J. Wiseman (Ed). Fats in Animal Nutrition. p 153 Butterworths. London

Wiese, H. F., W. Yamanaka, E. Coon, and S. Barber. 1966. Skin lipids of puppies as affected by kind and amount of dietary fat. J. Nutr. 89:113.

Wiseman, J. 1986. Antinutritional factors associated with dietary fats and oils. In: W. Haresing and D. J. A. Cole (Eds). Recent Advances in Animal Nutrition . p 47. Butterworths, London. U.K.

Wolfe, R. G., C. V. Maxwell, E. C. Nelson, and R. R. Johnson. 1977. Effect of dietary fat level on growth and lipogenesis in the colostrum deprived neonatal pig. J. Nutr. 107:2100.

Wolfe, R. G., C. V. Maxwell, and E. C. Nelson. 1978. Effect of age and dietary fat level on fatty acid oxidation in the neonatal pig. J. Nutr. 108:1621.

Yamamoto, N. Saitoh, A. Moriuchi, M. Nomira, and H. Okuyama. 1987. Effect of dietary alpha-linolenate/linoleate balance on brain lipid composition and learning ability of rats. J. Lipid Res. 28:144.

Yanovich, V. G., S. I. Vovk, and A. I. Shibitsyi. 1982. Ontogenetic characteristics of the synthesis of lipids in pig liver and skeletal muscles in vitro from [1-^{14}C]glucose, [1-^{14}C]acetate, and [1-^{14}C]palmitate. Appl. Biochem. Microbiol. (USSR). 18:219.

APPENDICES

Appendix A

PROCEDURES FOR HOMOGENIZATION OF FETAL AND PLACENTAL TISSUES

Phosphate Saline Buffer:

The same concentrated phosphate saline buffer solution was utilized for the homogenization of both fetal and placental tissues following the procedures described by Harper (1992). The buffer solution was prepared by combining 14.2 g Na_2HPO_4, 234 g NaCl and 1.488 g EDTA in 2 L batches of distilled water. After the complete disolution of the solutes the pH was adjusted the to 7.4 by means of 1N NaOH. The bottles were labeled and stored at room temperature.

Two further dilutions (1% and 10%) of this concentrated buffer were used in the procedures for fetal pig and placental total protein assays.

Tissue Homogenization Procedure:

1) Whole litters of fetal pigs and corresponding placentas were thawed on the day of homogenization and kept cold on ice throughout the process.

2) The thawed weight of each fetal pig and corresponding placenta were determined and recorded prior to homogenization. Placental fluids were washed out by means of a colonder in order to get a more accurate weight of the tissues.

3) The fetus or placenta was weighed and then placed in a plastic

graduated cylinder. The contents of the cilinder was brought to a volume of 70-110 ml (depending on the weight) with phosphate saline buffer and the total contents was homogenized using polytron high speed homogenizer for 30 seconds. The homogenate liquid was then poured into a labeled 50 ml plastic centrifuge tube, capped and kept on ice. The polytron probe and graduated cylinder were thoroughly rinsed between each use.

4) Once all pigs within a littermate pair replicate were homogenized (all within one day), homogenates were storaged at -80°C until assayed for protein content.

Appendix B

DETERMINATION OF TOTAL PROTEIN OF FETAL AND PLACENTAL TISSUES

The following modified procedure of the bicinchoninic acid (BCA) was described by Smith et al. (1985) was used for the determination of total protein content of fetal and placental tissues. It is based on the principle that protein will react with Cu^{++} in an alkaline medium to produce Cu^{+} which in turn will form a stable complex with BCA. It is this Cu^{+}-BCA complex that forms a purple color product which allows for spectrophotometric quantification of protein content. The reagents (BCA detection reagent and 4% copper sulfate) and bovine serum albumin (BSA) standard were obtained commercially as the (Pierce BCA Protein Assay Reagent Kit, Pierce Company, Rockford, IL 61105).

Preparation of Working Reagent:

Fifty parts of the Pierce BCA detection reagent were combined with 1 part of the 4% copper sulfate solution in a clean beaker and mixed by gentle stirring. This working reagent was prepared fresh prior to each assay.

Preparation of Working Standard:

The Pierce BSA standard (2 mg/ml) was diluted 5-fold by combining 750 μl of a 10% solution of phosphate saline buffer (described in Appendix A). This solution was mixed by thorough vortexing and 900 μl aliquots were placed in polypropylene vials,

capped and stored under refrigeration for up to 2 weeks. Each aliquot provided enough standard solution for one standard curve in triplicate per assay.

Sample Preparation and Dilution:

The 50 ml centrifuge tubes of homogenate were allowed to thaw under refrigeration overnight prior to the assay. Thawed homogenates were vortexed then slow centrifuged at 300 X g for 15 minutes to settle particulate matter. Each homogenazte was then further diluted 1:50 by pipetting 100 µl of liquid homogenate into 16 X 150 mm borosilicate glass tubes containing 4900 µl of a 1% solution of phosphate saline buffer. Each tube of diluted homogenate was then capped and vortexed.

Conducting the Assay:

1) Using a 96 well microtiter plate, the working standard was pipetted in triplicate in volumes of 0, 10, 20, 30, 40 and 50 µl for six points of the standard curve. The total volume of each working standard well was brought to 50 µl with a 1% solution of phosphate saline buffer.
2) Fifty µl of each diluted unknown sample was then pipetted in triplicate to wells on the same plate. One or two additional plates were used as needed.
3) Two hundred µl of the working reagent was then added to all wells using an 8-tip multichannel pipette. Twenty ml/whole plate.

4) The plate wells were covered with laboratory film and a plastic microtiter plate cover and allowed to incubate at room temperature for 2 hours.
5) Absorbance at 540 nm was determined and recorded electronically using a Titertek^{R} ELISA reader.
6) A standard curve was prepared by plotting the net absorbance at 540 nm versus protein concentration. The protein concentration of each unknown was then calculated from the regression equation determined for the standard curve.
7) All final protein concentration values were expressed as milligram of protein per gram of wet tissue.

Appendix C

DETERMINATION OF DRY MATTER OF FETAL AND PLACENTAL TISSUES

Procedure:

1) Selected fetal pigs were placed in numbered aluminum drying pans that had been previously oven dried and weighed.
2) The sample pans were placed in a forced air drying oven at a temperature of 110°C.
3) After a minimum 7-d drying period, samples were removed from the oven and placed in a desiccator and allowed to cool to room temperature.
4) Working from the desiccator, each sample was weighed and the results recorded. The samples were then placed back in the oven for a minimum drying period of 6 h.
5) Step 4 was repeated until no further weight loss in the fetal pigs was observed. This final weight less the drying pan weight was recorded as the fetal dry weight.

Appendix D

APPENDIX TABLES

Appendix Table 12. Effects of dietary fatty acids on reproductive traits of gilts fed various sources of supplemental oil (Trial 1)[a]

Item	Treatments[b]				Pooled SEM
	SR[c]	CO	SO	FO	
Age at estrus, days	173.3	171.0	173.2	172.5	2.18
Wt at estrus, kg	119.3	118.7	117.5	113.8	2.58
Age at breeding, days	212.5	212.3	212.6	213.7	2.40
Wt. at Breeding, kg	135.2	135.7	135.0	132.3	2.69
Days of gestation	42.5	40.6	40.8	42.0	.88
Gestational wt gain, kg[d]	21.3	21.2	17.2	21.4	1.24
Wt at slaughter, kg[d]	157.5	156.2	151.6	154.2	2.91
No. of corpora Lutea[d]	14.4	14.9	13.7	14.6	.64
Total no. of fetus[d]	12.3	13.0	10.6	11.8	.82
Total fetus alive[d]	11.5	11.6	10.1	11.6	.94
Fetal survival (%)[d]	80.2	78.0	74.0	80.0	5.94

[a]Eleven gilts on SR diet, 13 gilts on CO diet, 13 gilts on SO diet, 11 gilts on FO diet.

[b]Where SR = Starch; CO = Coconut oil; SO = Soybean oil; FO = Fish oil.

[c]Gestation gain = Slaughter wt- wt at breeding.

[d]Least square means adjusted for days of gestation using covariate analysis.

Appendix Table 13. Effects of dietary fatty acids on fetal measurements from gilts fed various sources of supplemental oil (Trial 1)[a]

Item[c]	Treatments[b]				Pooled SEM
	SR	CO	SO	FO	
Fetal weight, g	16.1	15.5	15.5	14.3	.78
Crown-rump length, cm	5.9	5.8	5.9	5.8	.10
Total Fetal Protein, mg/g (Wet Basis)	38.6	36.2	35.2	35.3	1.63
Total Fetal Protein, mg/g (Dry Basis)	351.9	326.7	317.7	317.8	15.14
Placental weight, g	77.1	76.6	79.5	69.5	4.01
Total Placenta Protein, mg/g (Wet basis)	13.4	12.0	12.9	13.9	1.05
Total Placenta Protein, mg/g (Dry basis)	121.6	108.0	130.4	118.7	14.49

[a]Eleven gilts on SR diet, 13 gilts on CO diet, 13 gilts on SO diet, 11 gilts on FO diet.

[b]Where SR = Starch; CO = Coconut oil; SO = Soybean oil; FO = Fish oil.

[c]Least square means adjusted for days of gestation using covariate analysis.

Appendix Table 14. Effects of dietary fatty acids on reproductive traits of gilts fed various sources of supplemental oil (TRIAL 2)[a]

Item	Treatments[b]				Pooled SEM
	SR	CO	SO	FO	
Age at estrus, days	183.6	185.8	181.3	186.3	4.28
Wt at estrus, kg	103.2	109.6	105.0	104.8	4.52
Age at breeding, days	225.3	223.5	220.4	228.3	4.95
Wt at breeding, kg	124.5	123.0	120.5	123.0	3.90
Days of gestation	41.3	43.2	43.0	42.0	.62
Gestational wt gain, kg[cd]	21.2	21.2	19.9	16.5	1.58
Wt at slaughter, kg[d]	141.8	143.4	141.6	138.6	4.33
No. of corpora Lutea[d]	14.2	12.6	13.5	13.5	.61
Total no. of fetus[d]	11.7	10.1	11.2	12.8	.72
Total fetus alive[d]	11.1	10.1	10.3	12.3	.70
Embryo survival (%)[d]	78.5	79.9	76.8	91.2	4.58

[a]Seven gilts on SR diet, 9 gilts on CO diet, 8 gilts on SO diet, 8 gilts on FO diet.

[b]Where SR = Starch; CO = Coconut oil; SO = Soybean oil; FO = Fish oil.

[c]Gestation gain = Slaughter wt - wt at breeding.

[d]Least square means adjusted for days of gestation using covariate analysis.

Appendix Table 15. Effects of dietary fatty acids on fetal measurements from gilts fed various sources of supplemental oil (Trial 2)[a].

Item[c]	Treatments[b]				Pooled SEM
	SR	CO	SO	FO	
Fetal weight, g	16.9	17.2	16.5	16.2	1.35
Crown-rump length, cm	6.1	6.4	6.1	6.2	.19
Total Fetal Protein, mg/g (Wet Basis)	37.4	34.6	33.9	35.5	1.99
Total Fetal Protein, mg/g (Dry Basis)	352.3	317.0	309.4	309.6	17.5

[a]Seven gilts on SR diet, 9 gilts on CO diet, 8 gilts on SO diet, 8 gilts on FO diet.

[b]Where SR = Starch; CO = Coconut oil; SO = Soybean oil; FO = Fish oil.

[c]Least square means adjusted for days of gestation using covariate analysis.

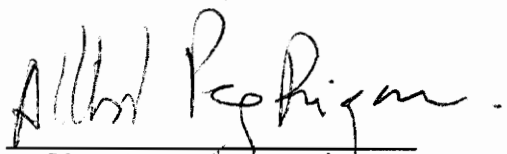
Appendix table 16. Range of fatty acid content in large batches of commercial menhaden oil taken annually from 1977 through 1988[a]

Fatty acids	Range % of total	Ratio[b]
C14: 0	7.2-12.1	1.7
C15: 0	0.4-2.3	5.8
C16: 0	15.3-25.6	1.7
C16: 1	9.3-15.8	1.7
C16: 2	0.3-2.8	9.4
C16: 3	0.9-3.5	3.9
C16: 4	0.5-2.8	5.6
C17: 0	0.2-3.0	15.0
C18: 0	2.5-4.1	1.6
C18: 1	8.3-13.8	1.7
C18: 2	0.7-2.8	4.0
C18: 3	0.8-2.3	2.9
C18: 4	1.7-4.0	2.4
C20: 0	0.1-0.6	6.0
C20: 4	1.5-2.7	1.8
C20: 5	11.1-16.3	1.5
C22: 1	0.1-1.4	10.0
C22: 5	1.3-3.8	2.9
C22: 6	4.6-13.8	3.0

Source: Zapata-Haynie Co., Anthony Bimbo, Laboratory Director.
[a]Reproduced from M. E. Stansby (1990) Fish Oils in Nutrition.
[b]Highest to Lowest

VITA

Alberto Pérez Rigau was born in Barcelona, Spain on October 19, 1961, to Juan Pérez Pérez and Francisca Rigau Nolla. He obtained his Bachelor in Veterinary Science in the Facultat de Veterinaria de Barcelona, Bellaterra (Spain) in June 1990. In August 1991 he entered graduate school at Virginia Tech to pursue the Master of Science degree in Animal Nutrition in the department of Animal Science under the direction of Dr. E. T. Kornegay.



Alberto Pérez Rigau