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Impacts of Nutrition and Environmental Stressors on Lipid Metabolism

Heather M. White, Brian T. Richert and Mickey A. Latour

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1. Introduction

Mediation of nutrition and environmental stressors through hormonal and physiological responses alters growth performance and lipid metabolism in nonruminants, resulting in substantial impacts on carcass lipid quality. Understanding and managing the factors that control carcass fat quality is a challenge for the swine industry yet provides opportunities to improve final carcass quality and profitability of pork production. Three major contributors to lipid quality in swine are regulation of *de novo* lipogenesis, dietary lipid composition, and environmental stressors. This chapter will evaluate these contributors and their effects on lipid deposition and quality, as well as nutritional and managerial interventions.

2. De novo lipogenesis

In general, the fatty acid profiles of swine carcass lipids are reflective of dietary fatty acid composition and *de novo* lipogenesis. The level of unsaturation in dietary fat sources is mimicked in the carcass fatty acid profile, altering the lipid firmness by increasing the degree of unsaturation. Stress has also been shown to impact growth performance, and can have an impact on the swine industry both by altering growth performance and carcass lipid firmness. Fatty acids synthesized *de novo* are products of pathways tightly regulated by rate-limiting enzymes. Nutritional and hormonal regulators of the enzymes which regulate these pathways can alter rates in lipid synthesis, oxidation, and desaturation.

The first step in *de novo* lipogenesis is the generation of the main fatty acid subunit, malonyl-CoA. The production of malonyl-CoA from acetyl-CoA is catalyzed by acetyl-CoA carboxylase (ACC; EC 6.4.1.2) [1]. Acetyl-CoA is a single polypeptide chain which contains a biotin carboxyl carrier protein, biotin carboxylase, and carboxyl transferase domains [2, 3]. Acetyl-CoA is present as ACC α (~265 kDa) in liver and adipose tissue and catalyzes fatty

acid synthesis [2, 3, 4]. In liver, heart, and muscle tissues ACC β (~280 kDa) controls fatty acid oxidation [2, 3, 4]. The ACC reaction is a two-step reaction in which the biotin molecule, covalently attached by holo-carboxylase synthetase to the ϵ -amino group of a lysine residue, acts as the carboxyl carrier [5]. The first step results in the formation of carboxy-biotinyl-ACC at the biotin carboxylase active site and is ATP-dependent. During the second step, the carboxyl group is transferred from biotin to acetyl-CoA forming the malonyl-CoA product [5].

The fatty acid synthase (FAS; EC 2.3.1.85) pathway is responsible for *de novo* lipogenesis which stores excess energy as fatty acids in liver and adipose tissue [1,6]. This pathway occurs within the cytosol and is a sequence of seven steps which are NADPH-dependent and utilize one acetyl-CoA and seven malonyl-CoAs as the base molecules to produce palmitate [1]. The NADPH required for each reaction is derived from activity of malic enzyme and the pentose phosphate shunt [7]. Though palmitate is the main product, stearic, myristic and shorter fatty acids may also be produced [7]. Fatty acids produced from *de novo* lipogenesis are primarily saturated or monounsaturated and may be used in phospholipid and triacylglycerol synthesis [7].

Fatty acid synthase is a multifunctional enzyme composed of two identical monomers, each ~270 kDa [3]. Each monomer contains six functional domains which are β -ketoacyl synthase (KS), acetyl/malonyl transacylase (AT/MT), β -hydroxyacyl dehydratase (DH), enoyl reductase (ER), β -ketoacyl reductase (KR), acyl carrier protein (ACP) and thioesterase (TE) in order from the N-terminus [3]. The condensation of seven C₂ moieties to the acetyl unit involves specific functions of the monomer components [3]. The reaction begins when the two substrates, acetyl-CoA and malonyl-CoA, are transferred to the KS and ACP, respectively, which is catalyzed by acetyl and malonyl transacylases. The condensation of these two substrates is catalyzed by KS and thus acetoacetyl-ACP is formed and CO₂ is released. Acetoacetyl is reduced to a β -hydroxyacyl chain by KR and the product is then dehydrated and reduced a second time by DH and ER, respectively. The resulting product is a four-carbon fatty acid which is attached to ACP and transferred, by KS, from the ACP to a Cys-SH group on the KS. The ACP is then free to accept another malonyl unit. The addition of two carbon units from malonyl-CoA to the growing acyl chain leads to the synthesis of palmitate which is released after being hydrolyzed by TE [3].

2.1. Regulation of *de novo* lipogenesis

Regulation of ACC and FAS are important as they are the rate limiting steps of lipogenesis. Transcriptional regulation of ACC- α and - β is controlled by three promoters, PI, II, and III [5]. These promoters respond to glucose, insulin, thyroid hormone, catabolic hormones, and leptin [5]. Additional regulation occurs by sterol-regulatory-element-binding protein 1c (SREBP1c) and peroxisome-proliferator-activated receptors (PPAR) [5]. Fasting inhibits ACC expression though re-feeding returns expression to normal levels. Insulin exposure activates ACC, while catecholamines or glucagon exposure will inhibit ACC [5]. Activation and inhibition by insulin, catecholamines and glucagon, respectively, occur within minutes of exposure [5].

Acetyl-CoA carboxylase is also allosterically regulated, resulting in active and inactive protein conformations [1,5]. Phosphorylation of four or more serine residues on ACC results in inactivation [5, 8]. Phosphorylation of ACC α is by AMP-activated protein kinase (AMPK) while phosphorylation of ACC β is by protein kinase A (PKA) [5, 8]. In liver and heart cells, insulin activates ACC α by dephosphorylating the AMPK site although this mechanism has not been observed in fat or liver cells [5].

Transcriptional regulation is the primary means of controlling FAS [1, 7]. The FAS promoter has been studied in the rat, human, and chicken and the sequence is highly conserved among species [7]. The 5' flanking region of the promoter is 2.1 kb long and has transcription factor binding sites which determine tissue specificity of expression [7]. Promoter activity and FAS expression have been shown to increase in transgenic mice when high carbohydrate diets are fed, after fasting, and with increased insulin and glucocorticoid levels. Dietary polyunsaturated fatty acids (PUFA) decrease hepatic and adipose FAS mRNA levels and is a part of the mechanism of dietary fats to reduce *de novo* fatty acid synthesis [7]. Another mode of FAS regulation is stability of FAS mRNA [7]. In diabetic rats, thyroid hormone regulates FAS mRNA stability and in fetal rat lung, glucocorticoids stabilize FAS mRNA [7].

2.2. Desaturation of fatty acids

Stearoyl-CoA desaturase (SCD; EC 1.14.19.1), also known as Δ^9 desaturase, is an endoplasmic reticulum associated enzyme that catalyzes the conversion of saturated fatty acids to monounsaturated fatty acids (MUFA) [9, 10]. Palmitoyl-CoA and stearoyl-CoA are the primary substrates of the desaturation reaction and are converted to palmitoleoyl-CoA and oleoyl-CoA, respectively [10, 11]. In liver, SCD is also required for synthesis of cholesteryl esters [9]. There are four isomers of SCD: SCD-1, found in adipose and liver tissue; SCD-2 and -3, found in the brain and harderian gland; and SCD-4, found in the heart [9]. The action of SCD to add a double bond to the Δ^9 position of a saturated fatty acid starts the desaturation process. More double bonds can then be added by the elongation pathways discussed below [11]. Desaturation of 12 to 19 carbon fatty acyl-CoAs catalyzed by SCD-1, -2, -3, and -4 results in the addition of a *cis*-double bond between carbons nine and 10 and this reaction requires NADH, oxygen, NADH-cytochrome *b5* reductase and cytochrome *b5* [9].

Control of SCD-1, -2, -3, and -4 is mainly by transcriptional regulation [9]. Dietary omega-3 and -6 PUFAs, thyroid hormone, glucagon, thiazolidinediones, and leptin suppress SCD-1 expression, while cholesterol, vitamin A, PPAR α , SREBP-1c, and high carbohydrate feeding induce expression [9, 12]. Increased SCD-1 activity thus increases the conversion of saturated fatty acids to unsaturated fatty acids and changes the ratio of carcass fatty acids.

2.3. β -oxidation

β -oxidation is the catabolic process, occurring primarily in the mitochondria of the cell, that breaks down fatty acids into acyl-CoA molecules. These two carbon molecules can then enter the tricarboxylic acid cycle for energy production. β -oxidation involves three key

components: activation of fatty acids in the cytosol of the cell, transport of activated fatty acids into the cell mitochondria, and oxidation.

The mechanism of the carnitine pathway is an ordered reaction where the binding of acyl-CoA begins the transport action [13]. Long chain fatty acids are converted to acyl-CoAs by acyl-CoA synthetase [14]. Acyl-CoAs are converted to acyl-carnitine molecules and transferred across the outer mitochondrial membrane by carnitine palmitoyltransferase-I (CPT; EC 2.3.1.21) [14]. Carnitine palmitoyltransferase-II is located on the inner mitochondrial membrane and liberates the carnitine from the acylcarnitine after transfer across the inner mitochondrial membrane [15]. After liberation, the acyl-CoA units are available for β -oxidation within the mitochondrial matrix [14]. Because CPT-II is not regulated [1] it is not pertinent to this discussion.

Carnitine palmitoyltransferase-I is located on the outer mitochondrial membrane and limits the rate of fatty acid oxidation by controlling the transportation of fatty acyl-CoA to the mitochondrial matrix where β -oxidation occurs [13, 1]. Two transmembrane domains anchor CPT-I to the outer mitochondrial membrane [13]. There are three isoforms of CPT-I [1]. In liver, kidney, lung, and heart tissue, CPT-Ia is present; CPT-Ib is present in skeletal muscle, heart, and adipose tissue; and CPT-Ic is brain tissue specific [1].

Regulation of β -oxidation occurs during the initial transport step. The main route of CPT-I regulation is by malonyl-CoA, the first product of lipogenesis, which inhibits CPT-I and aids to prevent simultaneous oxidation and synthesis [1, 16]. Regulation of CPT-I allows β -oxidation to be regulated by controlling the availability of acyl-CoA in the mitochondrial matrix [1, 14]. Though the sensitivity of the CPT-Ia and CPT-Ib to malonyl-CoA are different, they both contain binding sites on the same side of the membrane as the active site [13]. The N-terminus of the enzyme, which is not required for catalytic activity, controls the response to malonyl-CoA [13]. The kinetics of inhibition by malonyl-CoA are responsive to temperature, pH, and lipids [13, 14].

Fasting and glucagon increases CPT-I gene expression while hypothyroidism decreases expression by regulating the transcription level [13]. The insulin growth factor I receptor also controls CPT-I expression by mediating the inhibitory effects of insulin [13, 14]. Expression of CPT-I is also transcriptionally upregulated by PPAR α [13]. Long chain fatty acids increase CPT-Ia mRNA expression in liver tissue by both increasing transcription levels as well as improving CPT-I mRNA stability [14].

2.4. Regulation of lipid metabolism by transcription factors

Sterol regulatory element binding proteins (SREBP) are helix loop helix proteins that are within the leucine zipper family of transcription factors [9]. The SREBPs are present as two isoforms, SREBP-1 (a and c subforms) and SREBP-2 [9]. While SREBP-2 is primarily involved in activation of cholesterol synthesis and metabolism, SREBP-1c is involved solely in regulation of fatty acid synthesis and SREBP-1a is capable of inducing both synthesis of cholesterol and fatty acids [9]. In the liver, SREBP-1c increases expression of SCD, ACC, FAS and acetyl CoA synthase [9].

Long-chain fatty acids are oxidized in the peroxisome by catalase, producing acetyl-CoA and hydrogen peroxide [15]. The catalase enzyme is induced by high-fat diets and proliferation of the peroxisomes is controlled by the peroxisome proliferator activated receptor (PPAR), which is part of the nuclear receptor family [9, 15]. The PPAR α form is involved in regulation of β -oxidation and lipolysis in hepatocytes while PPAR γ is involved in regulation of fatty acid synthesis in adipocytes [17, 18, 19]. The PPAR binding site contains both a hydrophobic ligand-binding pocket and a DNA-binding domain [9]. Stimulation of fatty acid oxidation by PPAR α is by induction of CPT-I. Peroxisome proliferators also stimulate SCD-1 transcription levels [9].

Intracellular fatty acids contribute to the overall regulation of synthesis and oxidative pathways. Fatty acids enter cells through diffusion or transporters, specifically fatty acid transport protein (FATP) or fatty acid transporter CD36 (FAT). Fatty acyl CoA synthetases or FATP then convert fatty acids into fatty acyl CoA (FACoA). Fatty acid binding proteins (FABP) then bind to and transport FACoA into intracellular compartments where they influence transcription through regulation of PPAR α , γ and SREBP-1 [17, 20]. Intracellular PUFA inhibit SREBP-1 by downregulating enzymes involved in fatty acid synthesis [17, 18]. Intracellular PUFA activate PPAR to upregulate the transcription of the corresponding enzymes [17].

3. Dietary lipid composition

Dietary triacylglycerol composition plays a major role in determining adipose tissue composition. Monogastric animals incorporate dietary fatty acids directly into tissue lipid deposits [21, 22] and, therefore, to manipulate carcass lipid quality, it is important to understand the interactions of dietary lipids with carcass lipid. Carcass fatty acid profiles closely mimic dietary fatty acid profile [21, 23], and therefore, potential exists to modify carcass lipid properties (i.e., firmness, fatty acid profile, etc.) by altering dietary lipid composition.

One of the strongest determinants of carcass fat quality in pigs is the level and composition of lipids in the diet [24]. Because the utilization efficiency of dietary fat is 90% in pigs fed above maintenance [24] and the transfer coefficient of dietary fat to carcass lipid is as high as 31-40% [25] the carcass lipid composition is a reflection of dietary fat. The impact of dietary lipids on carcass lipid may differ depending on the timing of feeding relative to growth and finishing, levels included in the diet, and interactions with other stressors.

3.1. Dietary fat

Dietary triacylglycerols alter carcass lipid composition at the level of the fatty acid profile [21]. Saturated fatty acids lack double bonds and have melting temperatures above 40°C. Mono-, di- and poly-unsaturated fatty acids have one, two, or many double bonds, respectively and as the level of unsaturation increases, the melting point decreases [21]. The ratio of saturated to unsaturated fatty acids is a way of describing the relative saturation of a fatty acid profile [21]. Iodine value, a measure of double bonds in a lipid, is a method used to composite characteristics of lipids in regard to fluidity [21, 26]. Saturated to unsaturated ratios and iodine values can be utilized to describe the composition of lipids in both feedstuffs, total rations, and animal tissue.

Fat is commonly added in swine diets from 0.5% up to 7% of the ration and increases growth rate, reduces feed intake, and improves feed efficiency [21]. Because of the previously mentioned utilization efficiency and transfer coefficients, the level of saturation and iodine value of the feed lipid source will be strongly reflected in the carcass fatty acid profile and therefore, sources of dietary fats play a critical role in final carcass lipid quality. Vegetable oils are typically high in linoleic acid, have an unsaturated to saturated fatty acid ratio of 12:1 [22] and an iodine value greater than 100 [21]. Diets high in these unsaturated vegetable oils will result in oily, soft carcass fat [21]. Conversely, tallow, which is high in palmitate and stearate, has a saturated to unsaturated fatty acid ratio of 1:1 [22], an iodine value between 40 and 45 [21] and will result in firmer carcass fat when fed in the diet. Greater saturated:unsaturated fatty acid ratio in fat contained in pig carcasses results in fewer difficulties during processing [27] due to increased firmness at typical processing temperatures (2 to 4°C). Due to differences in calculation of these indices, some variations in fatty acid profile are captured with one ration but not the other, as seen in Figure 1. For this reason, it is best to utilize both the IV and saturated:unsaturated indices when characterizing fat quality, in order to identify all variations in fatty acid profile.

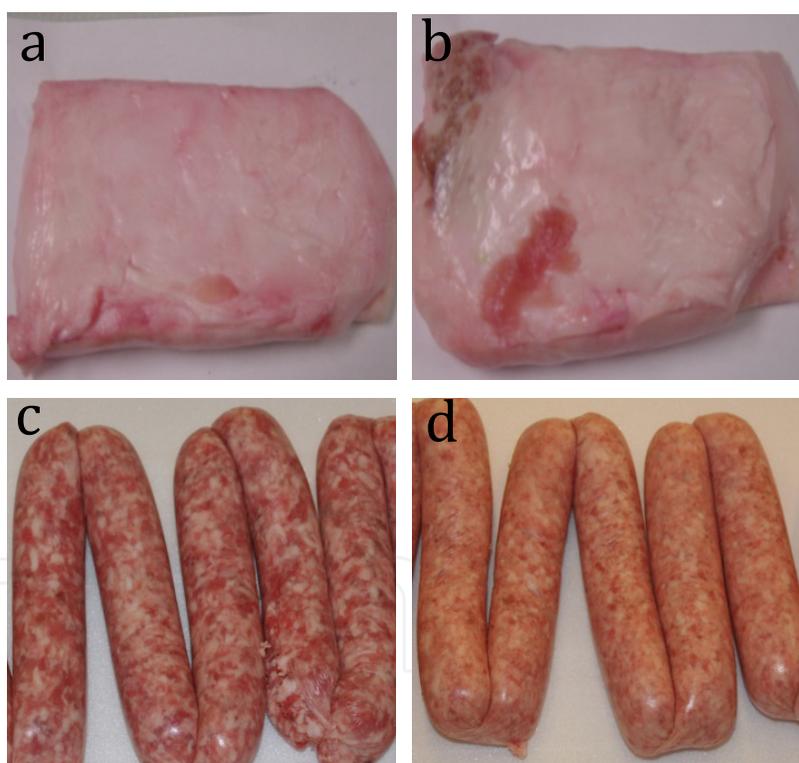


Figure 1. Differences in carcass lipid quality alter final product characteristics. Higher iodine values (IV) are associated with fat that is softer, resulting in increased difficulty slicing and processing. Panel *a* is backfat with an IV of 69 which represents fat that is firm and maintains shape and structure, while panel *b* is backfat with an IV of 79 which represents fat that will lack the firmness required for processing. Saturated to unsaturated fatty acid ratios are also used to characterize fatty acid profiles. While sausages (bottom panels) made from different animals have the same IV (59), the differences in saturated:unsaturated fatty acids results in a higher quality, firmer product in panel *c* (0.62) that has less smearing compared with panel *d* (0.59).

3.2. Dried Distillers Grains with Solubles

Dried distillers grains with solubles (DDGS) is the by-product of yeast fermentation of grains such as corn for ethanol production [28]. During fermentation, corn starch is converted into alcohol and the remaining grain components, protein, fat, fiber, minerals, and vitamins are concentrated in the fermentative co-product approximately 3-times that of corn [28]. The nutritional value of corn DDGS is variable and a function of DDGS processing [28, 29, 30].

There are two processes by which ethanol can be extracted from corn, wet milling and dry grinding. Dry grinding is more commonly used and accounts for 70% of ethanol production processes [31]. Dry grinding yields the maximum ethanol from corn while wet milling yields other products including corn oil and corn gluten meal [31, 32]. The dry grind process begins by grinding the corn and mixing it with water (Figure 2). The resulting mash is then heated with enzymes to convert the starches to sugars which can be fermented by yeast. The product contains particulates and solubles which are distilled and dehydrated, producing ethanol and wet distiller's grains. The distiller's grains are then dried in order to increase shelf life [31, 32].

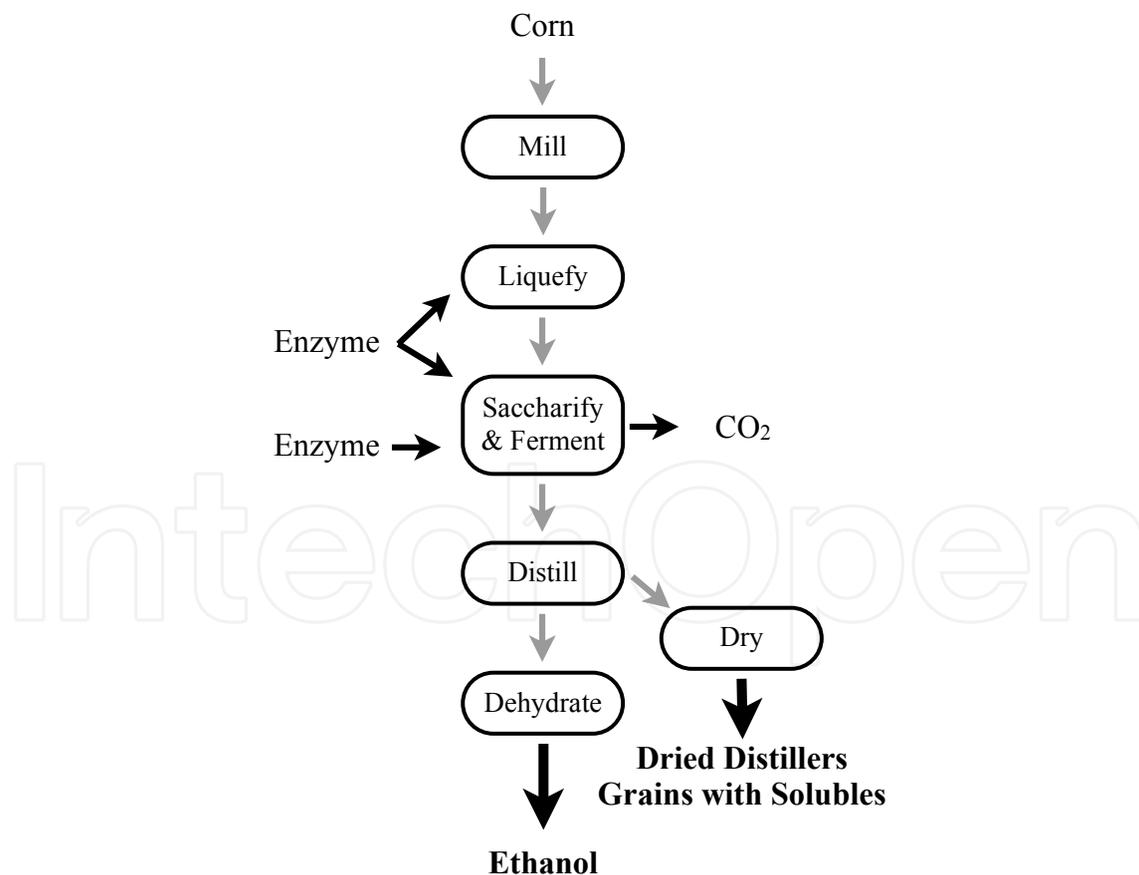


Figure 2. Dry grind processing of corn to produce ethanol. Progression of processing steps are shown in ovals and gray arrows, with inputs and outputs indicated by black arrows. The major byproduct of ethanol production is dried distillers grains with solubles.

The nutritional value of DDGS for pigs is influenced by the processing procedure and production plant equipment and techniques [33, 34]. The nutrient profile of DDGS remains highly variable even within the same production site [30, 35]. The NRC published content for DDGS is 93% dry matter, 2.82 Mcal/kg metabolizable energy, 27.7% crude protein, 8.4% crude fat and 34.6% neutral detergent fiber [36], however there is significant plant to plant variation, as noted above.

Two limiting factors for including DDGS in swine diets are the high level of unsaturation in the dietary fatty acid profile and the high fiber content [28, 31]. As discussed above, the composition of these fat sources is important when considering the carcass fat firmness [21]. Dietary fiber has also shown beneficial effects in swine diets including reduction of gastric ulceration and restriction of pathogenic bacteria in the intestinal tract; however, when fiber content of the diet exceeds 7%, growth is inhibited [37]. The high level of fat and fiber in DDGS have been shown to result in both decreased feed intake and increased unsaturated content of adipose tissue. In a trial utilizing 0, 10, 20, and 30% DDGS in grow-finish diets, pigs fed 20 or 30% DDGS had decreased growth performance and increased IV when compared to control fed pigs [38]. Incorporation of 0, 20, or 40% DDGS in diets during the final 30 days of the finishing phase resulted in reduced percent lean in bacon and decreased carcass firmness (based on IV and saturation); however, no effect on growth performance was observed [39].

The future direction of DDGS as a feed ingredient will likely be defined by the final use in global energy needs and not how it might be valued as a feed ingredient; that is, DDGS still contains a considerable amount of oil, a highly valued potential energy source. Today, DDGS is well suited for non-ruminants in terms of energy and protein content, price, and availability; however, the high linoleic acid content known to alter fat quality must be considered when determining dietary inclusions. As refiners investigate new approaches to removing the oil and protein, which may be of more value extracted, the future product could resemble a more fiber-like product, which would have wide range implications on non-ruminant animals and likely reduce its future use in swine diets.

3.3. Omega-3 and -6 fatty acids

The levels of omega-3 and omega-6 fatty acids in the human diet are important for optimal health. Animals, including humans, lack the enzymes required to add double bonds between the methyl group and ninth carbon and therefore cannot synthesize omega-3 and -6 fatty acids, making these fatty acids essential in the diet [22]. Fatty acids in the omega-6 family, linoleic (LA; 18:2n-6) and arachidonic (AA; 20:4n-6), and those in the omega-3 family, α -linolenic (ALA; 18:3n-3) and subsequently eicosapentaenoic acid (EPA; 20:5n-3) and docosahexaenoic acid (DHA; 22:6n-3), must be supplied in animal diets [16, 21, 22]. The synthesis pathways of omega-3 and -6 fatty acids and the parallel omega-9 pathways are shown in Figure 3. The omega-3 and -6 pathways compete for the Δ^5 and Δ^6 desaturases though both enzymes preferentially catalyze the reactions of the omega-3 pathway [40, 41].

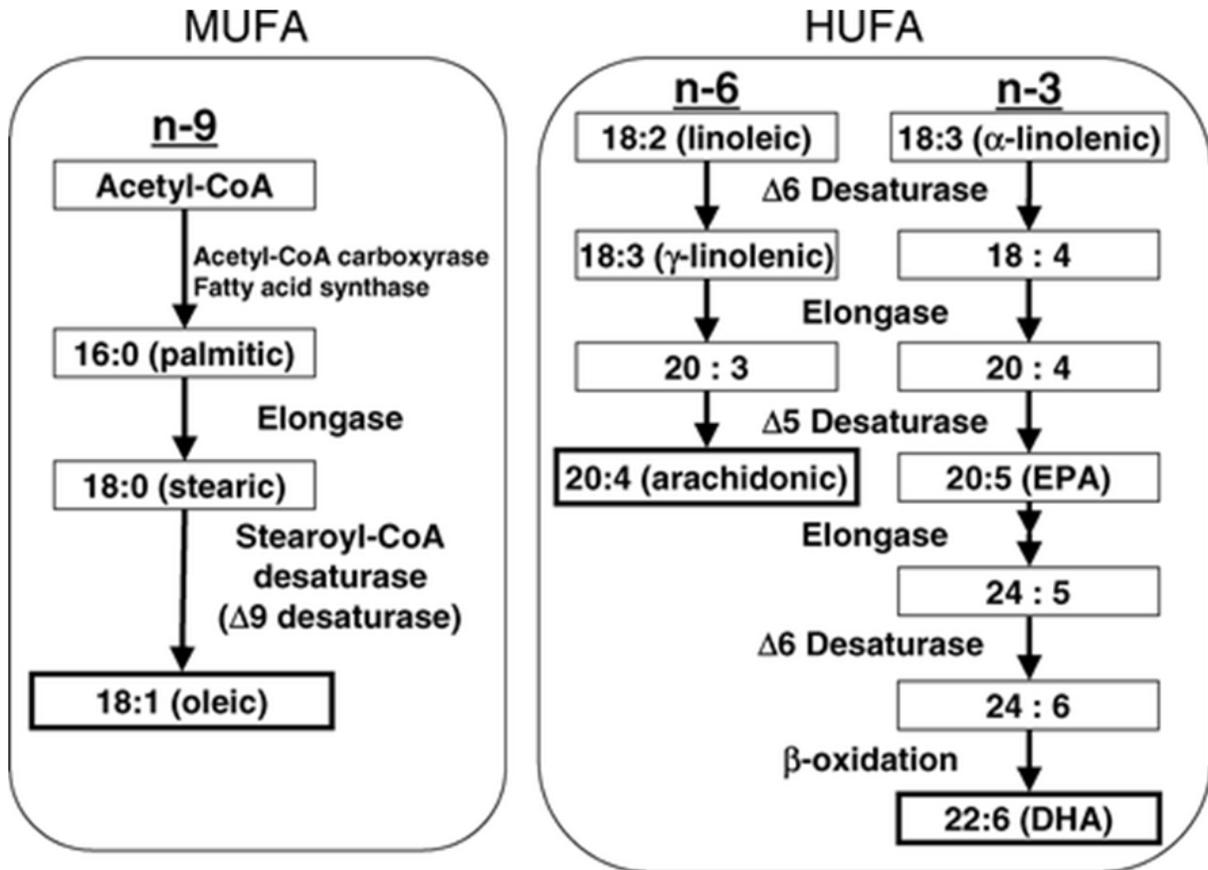


Figure 3. Synthesis pathways for omega-3, -6, and -9 fatty acids in mammals. MUFA, monounsaturated fatty acid; HUFA, highly unsaturated fatty acid; EPA, eicosapentaenoic acid; DHA, docosahexaenoic acid [9].

The ideal ratio of omega-6 to omega-3 fatty acids in human diets is between 4 to 6:1, although the average American diet is between 10 to 30:1 [42]. The change in this ratio is due to the increase in omega-6 intake relative to the level of omega-3 fatty acids [41]. The need to increase dietary intake of omega-3 fatty acids, specifically EPA and DHA, has increased demand for products with a ratio of omega-6 to -3 fatty acids more closely related to American Heart Association (AHA) recommendations.

The many health benefits of omega-3 fatty acids, such as lowering serum cholesterol and triacylglycerol concentrations, reduce platelet aggregation, reduction of blood pressure, and decreasing very-low-density and low-density lipoproteins, make dietary inclusion important [40]. The overall anti-inflammatory effects of omega-3 fatty acids have shown beneficial effects for arthritis and joint health in rats and humans [40]. Though it has not been directly studied in swine, omega-3 fatty acids could decrease the prevalence of lameness in sows if they result in the same joint and anti-inflammatory benefits noted in humans and rats. In Canada, 8-11% of sows culled were due to lameness [43] and in the United States lameness accounts for about 10% of culled sows during parity zero, one, and two or more, respectively [44]. Lameness results in the removal of sows at a younger age than other culling reasons, thus decreasing breeding herd productivity [44].

Omega-6 fatty acids are the precursors of eicosanoids which include prostaglandins, thromboxanes and leukotrienes. These metabolites of n-6 fatty acids exhibit inflammatory effects [45]. Omega-3 fatty acids inhibit eicosanoid synthesis by decreasing the available arachidonic acid available for eicosanoid production [18, 45]. In addition to decreasing eicosanoid production, omega-3 fatty acids also decrease other inflammatory cytokines such as interleukin-1 and -6, and tumour necrosis factor [18, 45].

3.4. Conjugated linoleic acid

Conjugated linoleic acids (CLA) are a group of polyunsaturated fatty acids that are positional and geometric isomers of linoleic acid (C18:2). Because CLA and its precursor, *trans* vaccenic acid, are naturally produced during bacterial fermentation in the rumen of ruminant animals, the main sources of CLA in human nutrition are ruminant milk and meats [46, 47]. The main isomers of CLA are *cis*-9, *trans*-11(c9t11) and *trans*-10, *cis*-12 (t10c12; Figure 4). Though the main isomer produced by ruminants is c9t11, commercially available products commonly contain equal proportions of c9t11 and t10c12 [46, 47]. Research in rodents, pigs, and humans has been conducted on the effects of CLA and has shown beneficial effects of CLA against obesity, cancer, atherosclerosis, and diabetes, some of which are isomer specific [46, 47, 48].

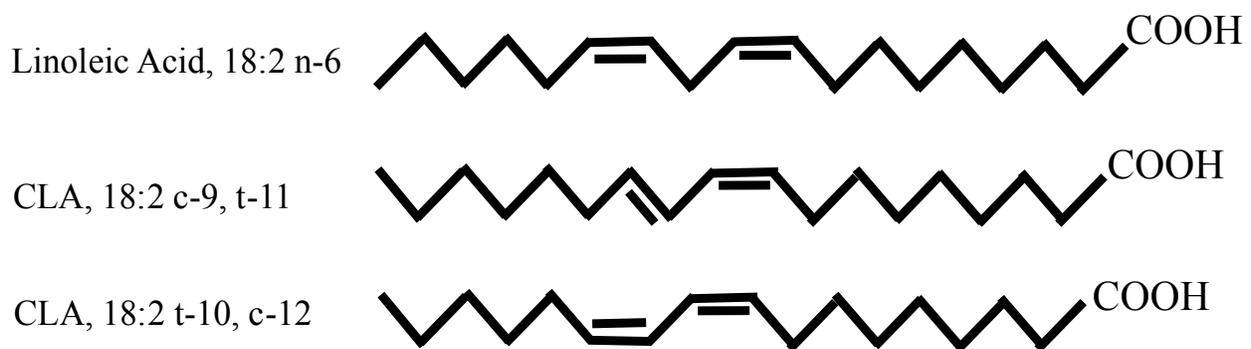


Figure 4. Structure of linoleic acid compared with *cis*-9, *trans*-11 and *trans*-10, *cis*-12 conjugated linoleic acid (CLA).

Many studies have shown CLA mixtures are able to reduce adipose tissue depots in rodents, pigs, and humans and that this effect is specific to the t10c12 isomer or a mixture containing greater than 50% t10c12 [46, 48]. Postweanling mice fed 1% CLA for 28-30 d had a 50% reduction in total adipose tissue compared to control mice [49]. In pigs, CLA inclusion in feed has resulted in decreased backfat thickness at finishing [50, 51]. Overweight or obese humans supplemented with CLA for 12 weeks also demonstrated reduced body fat mass, although their body mass index remained unchanged [52].

Another noted effect of CLA is the inhibition of cancer, specifically, mammary, prostate, skin, colon, and stomach cancers [48]. The anti-carcinogenic effects of CLA have been mainly attributed to the c9t11 isomer [46]. In studies of mammary and prostate cancer cell lines, feeding 1% CLA significantly reduced growth of the cancerous cells. Other studies of the same cell lines have not demonstrated these effects of CLA [48].

Atherosclerotic plaque formation is reduced by CLA [48]. Inclusion of 0.5 g/day in hypercholesterolemic diets fed to rabbits for 12 weeks resulted in significantly reduced serum triacylglycerols, low density lipoprotein (LDL) cholesterol levels and atherosclerotic plaque formation in the aorta [53]. The reduction of plaque deposits by CLA was proposed to be due to changes in LDL oxidative susceptibility [48].

Effects of CLA on the onset of diabetes and insulin resistance are inconsistent. Rats fed CLA have shown significantly reduced fasting glucose, insulinemia, triglyceridemia, free fatty acids, and leptinemia [48]. Butter enriched with c9t11 CLA failed to reduce glucose tolerance, lower adipose tissue or enhance glucose uptake leading to the conclusion that perhaps it is the t10c12 isomer which is responsible for the antidiabetogenic responses [48]. Insulin tolerance testing on CLA-fed mice showed marked insulin resistance without changes to blood glucose concentrations after oral glucose tolerance testing [54]. Other studies have examined the reduction of plasma leptin by CLA and the concomitant changes in blood glucose level due to regulation by leptin [46]. Feeding male mice high-fat diets with 1% CLA has resulted in reduced plasma leptin levels in one study [55] while resulting in no change in plasma leptin or glucose levels in another [56].

3.4.1. Feeding CLA to pigs

The effects of feeding CLA to pigs have been evaluated in regard to fat quality [57]. Gilts fed 1% CLA for seven weeks had firmer bellies, higher levels of saturated fatty acids, lower levels of unsaturated fatty acids and decreased IV when compared to controls [58]. Barrows fed CLA had improved feed efficiency, decreased backfat, and improved loin marbling and firmness when CLA was included at 0.75% of grow-finish diets [51]. When CLA was fed to genetically lean gilts for eight weeks, an increase in average daily gain and gain:feed was observed [59]. The same study also noted an increase in saturated fatty acids, decrease in unsaturated fatty acids, and an increased level of saturation of the belly tissue [59]. Several studies have shown that CLA feeding increases fatty acid saturation, and firmness in back fat and belly fat [60, 61, 62]. Additionally, use of CLA when feeding by-products may alleviate some or all of the negative impact on carcass quality. When feeding 0, 20, or 40% DDGS during the final 30 days of the finishing period, the addition of 0.6% CLA minimized the negative impact of 20% DDGS inclusion on carcass lipid quality but was unable to overcome the negative effects of feeding 40% DDGS [63].

3.4.2. Mechanism of CLA to alter lipid metabolism

Dietary CLA in several species alters the activity of SCD-1, FAS, and ACC in adipose and liver. Conjugated linoleic acids decrease mRNA for FAS and ACC to significantly inhibit the capacity for *de novo* lipogenesis [47, 60]. In barrows and gilts fed 0.25 or 0.5% CLA for the finishing diet from 97 to 172 kg, ACC activity was significantly reduced compared to control pigs [64]. Alleviation of negative impacts of nutritional stress of lipid quality, such as during DDGS feeding, is likely through altered lipid metabolism as adipose mRNA expression of ACC was decreased with CLA supplementation with all inclusion levels of DDGS [39].

Reductions in SCD-1 expression were observed with CLA feeding in both mouse liver and cultured preadipocytes [48]. Previous studies indicate that CLA tends to decrease both SCD-1 [65] and decreases the Δ^9 desaturase index in pigs [65, 66]. Decreasing SCD-1 mRNA expression, and thereby decreasing the amount of saturated fatty acids being converted to unsaturated fatty acids, may be responsible for the increased levels of saturated fatty acids observed after feeding CLA [65, 66].

The c10-t12 isomer of CLA decreases the expression of PPAR γ in adipose tissue and increases the expression of PPAR α in liver tissue [67, 68, 69]. By acting as a PPAR γ modulator, CLA is able to prevent lipid accumulation as shown in cultured adipocytes [70]. Conjugated linoleic acid also acts as a PPAR α activator and induces accumulation of PPAR-responsive mRNAs in hepatic cells [67] serving to upregulate PPAR-responsive pathways.

4. Environmental stressors

Environmental stressors on pigs can impact lipid metabolism and overall carcass quality. Impacts of environmental stressors, including thermal stress and housing density, are through both direct effects of decreased growth efficiency and indirect effects of altered regulation of *de novo* lipogenesis. Managerial and nutritional strategies during critical growth periods may alleviate the impact of these environmental stressors. Additionally, the regulation of *de novo* lipogenesis is influenced by the health status of the animal. Insults to health through disease or constant stress decrease feed intake and reduce *de novo* lipid synthesis. This decrease in *de novo* synthesis shifts the ratios of fatty acids in the adipose tissue to more unsaturated FA, further reducing lipid quality.

4.1. Spatial allocation, growth, and carcass composition

Decreasing space allocation reduces growth performance and the minimal spatial requirements for grow-finish pigs have been examined [36, 71]. Housing densities between 0.76 and 0.93 m²/pig have been reported as the threshold for grow-finish swine, below which ADG and ADFI are reduced [71].

Stress from spatial allocation is not a simple reflection of floor space, it is also reflective of pen dimensions, size, location of feeders and waters, and size of the pigs. One allometric calculation for spatial allocation is $f = k \times BW^{.667}$ (f = floor allowance, m²; k = coefficient of housing area; BW = body weight, kg) which accounts for the relationship between body weight and surface area [71]. In a study using this approach, housing densities of 0.578, 0.761 and 0.942 m²/pig corresponding to housing area coefficients of 0.030, 0.039 and 0.048, respectively; resulted in decreased ADG and ADFI in pigs housed at floor area allowances with coefficients between 0.030 and 0.039 [71]. These results were within in the range of other reported housing threshold values [71].

4.2. Heat stress, pork quality and animal growth

The thermal neutral zone of a mammal is the range of ambient temperatures within which the animal can control its core body temperature without elevating its metabolic rate [72].

Within the thermoneutral zone of mammals, core body temperature is maintained without expending additional energy to warm or cool basal body temperature [72, 73]. If the environmental conditions are below this zone, additional energy of metabolism is devoted to generating heat to maintain the desired core temperature [72]. Conversely, at temperatures above this zone, the animal must dissipate energy to maintain core body temperature through additional heat loss mechanisms such as evaporative heat loss, convection, and conduction [72]. When environmental temperature rises above the point where heat production and heat loss are balanced, the animal is in a state of heat stress [74]. In swine, evaporative heat loss is limited due to their inability to sweat; therefore, heat loss is primarily by respiration, evaporation, and exposure to cool air and wet surfaces for convection and conduction, respectively [72, 73, 75]. As an adaptive mechanism to further cool the body and maintain a homeothermic temperature, the animal decreases feed intake in order to decrease the thermal effect of feeding [74].

The optimum temperature for a finishing pig between 54.5 and 118.2 kg of body weight is 18.3°C, with a desirable temperature range between 10°C to 23.9°C [76]. The heat stress index (HSI; Figure 5), published by Iowa State University [77], is a practical guideline outlining temperature and humidity ranges for growing pigs. The HSI classifies environmental temperature and humidity conditions into three zones: alert, danger, and emergency. Within the alert range, producers are advised to monitor animal behavior, increase ventilation, and ensure that water is readily available. The danger range requires additional cooling by spraying or misting with water and increasing air flow. Under emergency conditions, producers are advised to avoid transporting animals, withdraw feed during the hottest part of the day, and reduce light levels. For example, when relative humidity is between 45 and 60%, 25.6°C is the alert threshold, 27.2°C is the danger threshold and 30°C is the emergency threshold.

For grow-finish swine, housing temperatures above 23.9°C decrease voluntary feed intake and growth rate compared to optimum housing temperatures [76, 78]. Voluntary decreases in feed intake decrease metabolic heat production to help maintain homeothermy [79]. Pigs challenged with heat-stress will have decreased feed consumption and average daily gain; however, feed efficiency is maintained when compared to control animals [80].

Nienaber et al. [81] noted that elevated temperature decreased daily feed consumption in both cattle and swine, through decreases in meal size and frequency. Feed intake was reduced by 55 g per degree of temperature increase above 22°C [82]. A similar decrease in feed intake observed by Collin et al. [75] was coupled to reductions in thermic effect of feeding and heat production.

Humidity is also influential in the animal's ability to dissipate heat by evaporative heat loss [74]. In a study comparing 50, 65, and 80% humidity levels, respiration rate and rectal temperatures were increased at lower temperatures and 80% humidity compared to when humidity was 50 and 65% [74]. Increasing environmental humidity decreases the efficiency of evaporative cooling, resulting in symptoms of heat-stress occurring at lower temperatures.

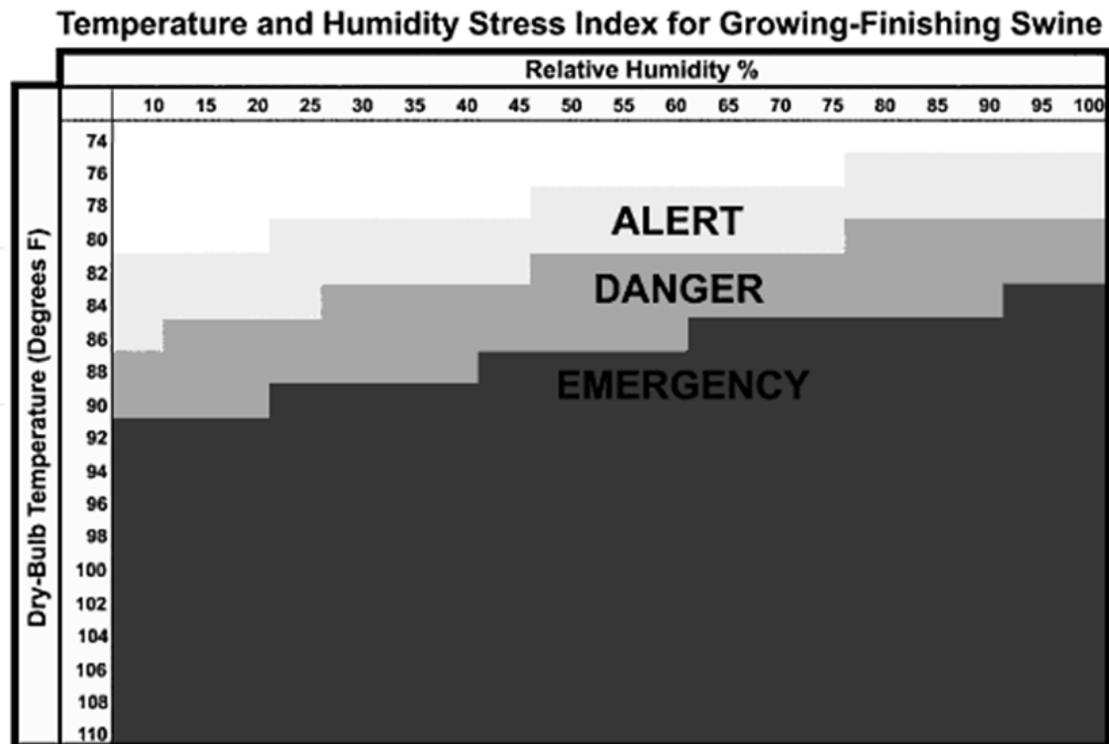


Figure 5. Heat Stress Index for Swine published by Iowa State University [77].

Physiological response to stressors, such as heat, results in the activation of the stress-activated sympathetic nervous system and the release of catecholamines and glucocorticoids [83]. When animals are exposed to a stressor, the hypothalamus releases corticotrophin-releasing hormone which stimulates adrenocorticotropin hormone (ACTH) from the pituitary gland [84]. The release of ACTH stimulates the adrenal cortex to release cortisol [84]. Cortisol regulates growth, immunity, and intermediary metabolism including gluconeogenesis, glycogen synthesis, and lipogenesis [7, 85]. The regulation of these processes by stress-activated hormones is one source of altered metabolism during periods of stress that may contribute to changes in feed intake, weight gain, and carcass lipid quality.

Decreases in acetyl-CoA-carboxylase and stearoyl-CoA-desaturase in adipose and liver tissues have been noted in heat-stressed pigs [79]. Kouba et al. [79] noted a decrease in acetyl-CoA-carboxylase activity in heat-stressed pigs and a decrease in *de novo* fatty acid synthesis. Acetyl-CoA-carboxylase and stearoyl-CoA-desaturase catalyze the first step of the synthesis of fatty acids and the synthesis of monounsaturated fatty acids from saturated fatty acids, respectively, and therefore would be key points of potential change in lipid metabolism.

Kouba et al. [86] noted 20-35 kg pigs maintained at 31°C compared to 20°C had slightly thicker backfat with a greater lipid content and an increase in lipoprotein lipase expression in backfat and an increase in VLDL-lipid concentration in heat-stressed pigs. The increase in fat thickness of heat-stressed pigs was thus attributed to increases in lipid metabolism in the liver, and in adipose tissue, noted through increased VLDL production and LPL activity, respectively. Increased lipid circulation between liver and adipose tissue is also part of the adaptation of pigs to high environmental temperatures [86].

Interactions between environmental stressors can amplify or alleviate the impact of an individual stressor. Pigs challenged with increased temperature and decreased spatial allocation demonstrated that both temperature and spatial allocation affected growth performance and carcass quality [63]. Temperature stress decreased ADG, ADFI, and G:F ratios. Pigs housed at minimum required spatial allocation of 0.66 m²/pig [36] and high environmental temperatures (32.2°C) had a 50% reduction in ADFI and an 85% reduction in ADG when compared with pigs housed in their thermal neutral zone; when pigs were housed at increased spatial allocation (0.93 m²/pig) and a temperature above 23.9°C, there was a 29% reduction of ADFI and a 36% reduction in ADG. Additionally, the level of saturation in adipose tissue was decreased in heat stressed pigs; however, increasing the spatial allocation in the 32.2°C environment ameliorated these effects and increased the fatty acid saturation to match the 23.9°C-housed pigs. The effects of spatial allocation on carcass quality demonstrate that challenging pigs with elevated temperature and reduced spatial allocation decreased feed intake, as demonstrated in the literature [87], and also decreased carcass lipid firmness. These relationships demonstrate that almost 50% of the negative growth performance effects of temperature can be ameliorated by a 28% increase in spatial allocation. In addition, an increase in housing allocation during heat stress may ameliorate the negative effects of temperature on belly weight, carcass quality, and growth performance.

5. Carcass quality

5.1. Bacon quality

The belly is the most expensive cut of the carcass, thus, the quality of bacon produced from the belly is linked to overall carcass value. Bacon is scored according to lean content and slice thickness to identify premium quality slices [88]. Premium slices have greater than 50% lean content and are wider than 1.9 cm at all points [88]. Accordingly, bacon slices are graded as either number one slices, number two slices, or as ends and pieces [88]. Pork bellies that are classified below standard based on these characteristics represent a decrease in carcass value.

The swine industry has shifted to genetically lean lines with decreased backfat and thus, bellies have become thinner, leaner, and softer [89, 90]. Thinner bellies are generally softer, produce fewer grade one slices, and present more problems with processing and storage [89, 90]. Providing saturated fat in the diet of pigs increases belly thickness and improves belly firmness [90]. Likewise, feeding CLA improves belly firmness in finishing pigs [59, 90].

5.2. Carcass lipid quality

Many processors utilize IV as numerical evaluation of carcass fat quality and thus have target IV values. An IV greater than 65, for some processors may be unacceptably high [58], while an IV greater than 75 may be the threshold for other processors. Increased IV [29] and decreased saturated to unsaturated fatty acid ratios [21] indicate decreases in carcass quality due to decreased fat firmness. High levels of unsaturated fatty acids result in rapid oxidation which decreases shelf life [91]. Furthermore, high levels of unsaturated fatty acids in the diets also produce bacon which is smeary, separates and causes processing difficulties

[88]. As discussed above, dietary fatty acid composition contributes to the carcass fatty acid composition; therefore, feeding more saturated dietary lipid sources will result in firmer carcass lipids with decreased IV [21].

5.3. Shelf-life of meat products

Shelf-life is defined as the period of time between packaging of a product and its end use when product properties remain acceptable to the consumer [92]. Shelf-life properties may include appearance, texture, flavor, color, and nutritive value [93]. One of the major factors affecting the shelf-life of meat products is rancidity or lipid oxidation, which occurs when fatty acids react to oxygen sources in the environment [94]. Oxidation produces low molecular weight aldehydes, acids, and ketones that cause the meat to exhibit distinct odors and flavors, typically unacceptable to consumers [94]. The level of unsaturation greatly affects the susceptibility of fat to oxidation with high degrees of unsaturation resulting in rapid oxidation and subsequently decreased shelf-life [95].

6. Summary

The fatty acid profiles of swine carcass lipids are reflective of dietary fatty acid composition and *de novo* lipogenesis [21]. The level of unsaturation in dietary fat sources is mimicked in the carcass fatty acid profile, altering the lipid firmness by increasing the degree of unsaturation [21, 22]. Feed alternatives such as DDGS, which are high in PUFA, decrease carcass lipid firmness and bacon lean when fed to grow-finish pigs [38]. Conversely, feeding CLA positively impacts growth performance and carcass fat quality [57, 58]. Stress has also been shown to impact growth performance, and low spatial allocation and heat stress have an impact on the swine industry both by altering growth performance and carcass lipid firmness [36, 76]. Fatty acids synthesized *de novo* are products of pathways tightly regulated by rate-limiting enzymes. Nutritional and hormonal regulators of the enzymes, which regulate these pathways, can alter rates in lipid synthesis, oxidation, and desaturation [5, 7, 13].

Environmental and nutritional stressors on pigs can impact lipid metabolism and carcass quality and thus alter final product quality and profitability. While the interactions of these stressors can additively worsen the impact on growth or lipid quality, understanding these interactions can also be used as a basis for managerial or nutritional interventions to alleviate the negative impact of unavoidable stressors.

Author details

Heather M. White

Department of Animal Science, University of Connecticut, USA

Brian T. Richert

Department of Animal Science, Purdue University, Brian, USA

Mickey A. Latour

Department of Animal Science, Southern Illinois University, Carbondale, USA

7. References

- [1] Ronnett, G. V., A. M. Kleman, E. K. Kim, L. E. Landree, and Y. Tu. 2006. Fatty acid metabolism, the central nervous system, and feeding. *Obesity*. 14Suppl:201-207.
- [2] Munday, M. R. 2002. Regulation of mammalian acetyl-CoA carboxylase. *Biochem Soc Trans*. 30:1059-1064.
- [3] Chirala, S. S., and S. J. Wakil. 2004. Structure and function of animal fatty acid synthase. *Lipids*. 39(11):1045-1053.
- [4] Tong, L. 2005. Acetyl-coenzyme A carboxylase: crucial metabolic enzyme and attractive target for drug discovery. *Cell Mol Life Sci*. 62:1784-1803.
- [5] Brownsey, R. W., A. N. Boone, J. E. Kulpa, and W. M. Lee. 2006. Regulation of acetyl-CoA carboxylase. *Biochem Soc Trans*. 34:223-227.
- [6] Ronnett, G. V., A. K. Kim, L. E. Landree, and Y. Tu. 2005. Fatty acid metabolism as a target for obesity treatment. *Physiol Behav*. 85:25-35.
- [7] Semenkovich, C. F. 1997. Regulation of fatty acid synthase (FAS). *Prog Lipid Res*. 36(1):43-53.
- [8] Boone, A. N., A. Chan, J. E. Kulpa, and R. W. Brownsey. 2000. Bimodal activation of acetyl-CoA carboxylase by glutamate. *J Biol Chem*. 275(15):10819-10825.
- [9] Nakamura, M. T., and T. Y. Nara. 2004. Structure, function and dietary regulation of $\Delta 6$, $\Delta 5$, and $\Delta 9$ desaturases. *Annu Rev Nutr*. 24:345-376.
- [10] Dobrzyn, A., and J. M. Ntambi. 2005. The role of stearoyl-CoA desaturase in the control of metabolism *Prostaglandins Leukot Essent Fatty Acids*. 73(1):35-41.
- [11] Mayes, P. A., and K. M. Botham. 2003a. Metabolism of unsaturated fatty acids & Eicosanoids. Pg. 190 in Harper's Illustrated Biochemistry. R.K. Murray, D.K. Granner, P.A. Mayes and V.W. Rodwell, ed. McGraw-Hill, New York.
- [12] Ntambi, J. M., M. Miyazaki, and A. Dobrzyn. 2004. Regulation of stearoyl-CoA desaturase expression. *Lipids*. 39(11):1061-1065.
- [13] Ramsay, R. R., R. D. Gandour, and F. R. van der Leij. 2001. Molecular enzymology of carnitine transfer and transport. *Biochim Biophys Acta*. 1546:21-43.
- [14] Louet, J. F., C. L. May, J. P. Pegorier, J. F. Decaux, and J. Girard. 2001. Regulation of liver carnitine palmitoyltransferase I gene expression by hormones and fatty acids. *Biochem Soc Trans*. 29:310-316.
- [15] Mayes, P. A., and K. M. Botham. 2003b. Oxidation of fatty acids: ketogenesis. Pg. 180 in Harper's Illustrated Biochemistry. R.K. Murray, D.K. Granner, P.A. Mayes and V.W. Rodwell, ed. McGraw-Hill, New York.
- [16] Drackley, J. K. 2000. Lipid Metabolism. Page 97 in Farm Animal Metabolism and Nutrition. J.P.F. D'Mello, ed. CABI Publishing, Oxon, UK.
- [17] Jump, D. B., and S. D. Clarke. 1999. Regulation of gene expression by dietary fat. *Annu Rev Nutr*. 19:63-90.
- [18] Jump, D. B. 2002. The biochemistry of n-3 polyunsaturated fatty acids. *J Biol Chem*. 277(11):8755-8758.
- [19] Sampath, H., and J. M. Ntambi. 2005. Polyunsaturated fatty acid regulation of genes of lipid metabolism. *Ann Rev Nutr*. 25:317-340.

- [20] Jump, D. B., D. Botolin, Y. Wang, J. Xu, B. Christian, and O. Demeure. 2005. Fatty acid regulation of hepatic gene transcription. *J Nutr.* 135:2503-2506.
- [21] Azain, M. J. 2001. Fat in swine nutrition. Pg 95 in *Swine Nutrition 2nd Ed.* A.J. Lewis and L.L. Southern, ed. CRC Press, Boca Raton.
- [22] Wiseman, J. 2006. Value of fats and oils in pig diets. Page 368 in *Whittemore's Science and Practice of Pig Production.* 3rd ed. I. Kyriazakis and C.T. Whittemore, ed. Blackwell Publishing, Oxford, UK.
- [23] Allen, C. E., D. C. Beitz, B. A. Cramer, and R. G. Kauggman. 1976. Biology of fat in meat animals. Research Division, College of Agricultural and Life Sciences, University of Wisconsin-Madison.
- [24] Freeman, C. P. 1983. Fat supplementation in animal production -- monogastric animals. *Proc Nutr Soc.* 42:351-359.
- [25] Kloareg, M., J. Noblet, and J. van Milgen. 2007. Deposition of dietary fatty acids, *de novo* synthesis and anatomical partitioning of fatty acids in finishing pigs. *Brit J Nutr.* 97:35-44.
- [26] Madsen, A., K. Jakoben, and H. P. Mortensen. 1992. Influence of dietary fat on carcass fat quality in pigs. A review. *Acta Agric Scand, Sect. A., Animal Sci.* 42:220-225.
- [27] Teye, G. A., J. D. Wood, F. M. Whittington, A. Stewart, and P. R. Sheard. 2006. Influence of dietary oils and protein level on pork quality. 2. Effects on properties of fat and processing characteristics of bacon and frankfurter-style sausages. *Meat Science.* 73: 166-177.
- [28] Newland, H. W., and D. C. Mahan. 1990. Distillers By-Products. Page 161 in *Nontraditional Feed Sources for Use in Swine Production.* P.A. Thacker and R.N. Kirkwood, ed. Butterworths, Stoneham, MA.
- [29] Cromwell, G. L., K. L. Herkelman, and T. S. Stahly. 1993. Physical, chemical, and nutritional characteristics of distillers dried grains with solubles from chicks and pigs. *J Anim Sci.* 71:679-686.
- [30] Pahm, A.A., C. Pedersen, D. Hoehler, and H.H. Stein. 2008. Factors affecting the variability in ileal amino acid digestibility in corn distillers dried grains with solubles fed to growing pigs. *J. Anim. Sci.* 86:2180-2189.
- [31] Rausch, K. D., and R. L. Belyea. 2006. The future of coproducts from corn processing. *Appl Biochem Biotechnol.* 128:47-86.
- [32] Bothast, R. J., and M. A. Schlicher. 2005. Biotechnological processes for conversion of corn into ethanol. *App Microbiol Biotechnol.* 67:19-25.
- [33] Spiehs, M. J., M. H. Whitney, and G. C. Shurson. 2002. Nutrient database for distiller's dried grains with solubles produced from new ethanol plants in Minnesota and South Dakota. *J Anim Sci.* 80:2639-2645.
- [34] Belyea, R. L., K. D. Rausch, and M. E. Tumbleson. 2004. Composition of corn and distillers dried grains with solubles from dry grind ethanol processing. *Bioresour Technol.* 94:293-298.
- [35] Shurson, G., M. Spiehs, and M. Whitney. 2004. The use of maize distiller's dried grains with solubles in pig diets. *Pig news and information.* 25(2):75N-83N.

- [36] NRC. 1998. Nutrient Requirements of Swine. 10th rev. ed. Natl. Acad. Press, Washington, DC.
- [37] Varel, V. H. and J. T. Yen. 1997. Microbial perspective on fiber utilization by swine. *J Anim Sci.* 75:2715-2722.
- [38] Whitney, M. H., G. C. Shurson, L. J. Johnston, D. M. Wulf, and B. C. Shanks. 2006. Growth performance and carcass characteristics of grower-finisher pigs fed high-quality corn distillers dried grain with solubles originating from a modern Midwestern ethanol plant. *J Anim Sci.* 84:3356-3363.
- [39] White, H. M., B. T. Richert, J. S. Radcliffe, A. P. Schinckel, J. R. Burgess, S. L. Koser, S. S. Donkin, and M. A. Latour. 2009. Feeding CLA partially recovers carcass quality in pigs fed dried distillers grains with solubles. *J. Anim. Sci.* 87:157-66.
- [40] Simopoulos, A. P. 1991. Omega-3 fatty acids in health and disease and in growth and development. *Am J Clin Nutr.* 54:438-463.
- [41] Kris-Etherton, P. M., W. S. Harris, and L. J. Appel. 2002. Fish consumption, fish oil, omega-3 fatty acids, and cardiovascular disease. *Circulation.* 106:2747-2757.
- [42] Leskanich, C. O., K. R. Matthews, C. C. Warkup, R. C. Noble, and M. Hazzledine. 1997. The effect of dietary oil containing (n-3) fatty acids on the fatty acid, physicochemical, and organoleptic characteristics of pig meat and fat. *J Anim Sci.* 75(3):673-683.
- [43] Dewey, C. E., R. M. Friendship, and M. R. Wilson. 1992. Lameness in breeding age swine – a case study. *Can Vet J.* 33:747-748.
- [44] Anil, S. S., L. Anil, and J. Deen. 2005. Elavulation of patterns of removal and associations among culling because of lameness and sow productivity traits in swine breeding herds. *JAVMA.* 226(6):956-961.
- [45] Calder, P. C. 2006. Polyunsaturated fatty acids and inflammation. *Prostaglandins Leukot Essent Fatty Acids.* 75:197-202.
- [46] Wang, Y. W. and P. J. H. Jones. 2004. Conjugated linoleic acid and obesity control: efficacy and mechanisms. *Int J Obes.* 28:941-955.
- [47] House, R. L., J. P. Cassady, E. J. Eisen, M. K. McIntosh, and J. Odle. 2005. Conjugated linoleic acid evokes de-lipidation through the regulation of genes controlling lipid metabolism in adipose and liver tissue. *Obes Rev.* 6:247-258.
- [48] Belury, M. A. 2002. Dietary Conjugated Linoleic Acid in Health: Physiological Effects and Mechanisms of Action. *Annu Rev Nutr.* 22:505-531.
- [49] Park Y., K. J. Albright, J. M. Storkson, W. Liu, M.E. Cook, and M. W. Pariza. 2001. Changes in body composition in mice during feeding and withdrawl of conjugated linoleic acid. *Lipids.* 34:243-248.
- [50] Tischendorf, F., F. Schone, U. Kirchheim, and G. Jahreis. 2002. Influence of a conjugated linoleic acid mixture on growth, organ weights, carcass traits and meat quality in growing pigs. *J Anim Physiol Anim Nutr (Berl).* 86:117-128.
- [51] Wiegand, B. R., F. C. Parrish, Jr., J. E. Swan, S. T. Larsen, and T. J. Baas. 2001. Conjugated linoleic acid improves feed efficiency, decreases subcutaneous fat, and improves certain aspects of meat quality in Stress-Genotype pigs. *J Anim Sci.* 79:2187-2195.

- [52] Blankson, H., J. A. Stakkestad, H. Fagertun, E. Thom, J. Wadstein, and O. Gudmundsen. 2000. Conjugated linoleic acid reduces body fat mass in overweight and obese humans. *J Nutr.* 130:2943-2948.
- [53] Lee, K. N., D. Kritchevsky and M. W. Pariza. 1994. Conjugated linoleic acid and atherosclerosis in rabbits. *Atherosclerosis.* 108:19-25.
- [54] Tsuboyama-Kasaoka, N., M. Takahashi, K. Tanemura, H. J. Kim, T. Tange, H. Okuyama, M. Kasai, S. Ikemoto, and O. Ezaki. 2000. Conjugated linoleic acid supplementation reduces adipose tissue by apoptosis and evelops lipodystrophy in mice. *Diabetes.* 49:1534-1542.
- [55] DeLany, J. P., F. Blohm, A. A. Truett, J. A. Scimeca, and D. B. West. 1999. Conjugated linoleic acid rapidly reduces body fat content in mice without affecting energy intake. *Am J Physiol.* 276:R1172-R1179.
- [56] West, D. B., F. Y. Blohm, A. A. Truett, and J. P. DeLany. 2000. Conjugated linoleic acid persistently increases total energy expenditure in AKR/J mice without increasing uncoupling protein gene expression. *J Nutr.* 130:2471-2477.
- [57] Cox, A. D. 2005. Added dietary fat effects on market pigs and sows. M.S. Thesis, Purdue University, West Lafayette.
- [58] Eggert, J. M., M. A. Belury, A. Kempa-Steczko, S. E. Mills, and A. P. Schinckel. 2001. Effects of conjugated linoleic acid on the belly firmness and fatty acid composition of genetically lean pigs. *J Anim Sci.* 79:2866-2872.
- [59] Weber, T. E., B. T. Richert, M. A. Belury, Y. Gu, K. Enright, and A. P. Schinckel. 2006. Evaluation of the effects of dietary fat, conjugated linoleic acid, and ractopamine on growth performance, pork quality, and fatty acid profiles in genetically lean gilts. *J Anim Sci.* 84:720-732.
- [60] Ostrowska, E., M. Muralitharan, R. F. Cross, D. E. Bauman, and F. R. Dunshea. 1999. Dietary conjugated linoleic acids increase lean tissue and decrease fat deposition in growing pigs. *J Nutr.* 129:2037-2042.
- [61] Aalhus, J. L., and M. E. R. Dugan. 2001. Improving meat quality through nutrition. *Advances in Pork Production.* 12:145.
- [62] Dugan, M. E. R., J. L. Aalhus, and B. Uttaro. 2004. Nutritional manipulation of pork quality: current opportunities. *Advances in Pork Production.* 15:237.
- [63] White, H. M., J. R. Burgess, A. P. Schinckel, S. S. Donkin, and M. A. Latour. 2008. Effects of temperature stress on growth performance and bacon quality in grow-finish pigs housed at two densities. *J. Anim. Sci.* 86:1789-98.
- [64] Corino, C., S. Magni, G. Pastorelli, R. Rossi, and J. Mourot. 2003. Effect of conjugated linoleic acid on meat quality, lipid metabolism, and sensory characteristics of dry-cured hams from heavy pigs. *J Anim Sci.* 81:2219-2229.
- [65] Smith, S. B., T. S. Hively, G. M. Cortese, J. J. Han, K. Y. Chung, P. Castenada, C. D. Gilbert, V. L. Adams, and H. J. Mersmann. 2002. Conjugated linoleic acid depresses the delta9 desaturase index and stearyl coenzyme A desaturase enzyme activity in porcine subcutaneous adipose tissue. *J Anim Sci.* 80:2110-2115.
- [66] Demaree, S. R., C. D. Gilbert, H. J. Mersmann, and S. B. Smith. 2002. Conjugated Linoleic Acid Differentially Modifies Fatty Acid Composition in Subcellular Fractions of

- Muscle and Adipose Tissue but Not Adiposity of Postweanling Pigs. *J Nutr.* 132:3272-3279.
- [67] Moya-Camarena, S. Y., J. P. Vanden Heuvel, S. G. Blanchard, L. A. Leesnitzer, and M. A. Belury. 1999. Conjugated linoleic acid is a potent naturally occurring ligand and activator of PPAR α . *J Lipid Res.* 40:1426-1433.
- [68] Evans, M. E., J. M. Brown, and M. K. McIntosh. 2002. Isomer-specific effects of conjugated linoleic acid (CLA) on adiposity and lipid metabolism. *J Nutr Biochem.* 13:508-516.
- [69] Kang, K., W. Liu, K. J. Albright, Y. Park, and M. W. Pariza. 2003. *trans*-10, *cis*-12 CLA inhibits differentiation of 3T3-L1 adipocytes and decreases PPAR γ expression. *Biochem Biophys Res Commun.* 303:795-799.
- [70] Granlund, L., L. K. Juvet, J. I. Pedersen, and H. I. Nebb. 2003. *Trans*10, *cis*12-conjugated linoleic acid prevents triacylglycerol accumulation in adipocytes by acting as a PPAR γ modulator. *J Lipid Res.* 44:1441-1452.
- [71] Gonyou, H. W. and W. R. Stricklin. 1998. Effects of floor area allowance and group size on the productivity of growing/finishing pigs. *J Anim Sci.* 76:1326-1330.
- [72] Mount, L. E. 1976. Heat loss in relation to plane of nutrition and thermal environment. *Proc Nutr Soc.* 35:81-86.
- [73] Noblet, J., J. L. Dividich, and J. Van Milgen. 2001. Thermal environment and swine nutrition. Page 519 in Swine Nutrition 2nd Ed. A.J. Lewis and L.L. Southern, ed. CRC Press, Boca Raton.
- [74] Huynh, T. T. T., A. J. A. Aarnink, M. W. A. Verstegen, W. J. J. Gerrits, M. J. W. Heetkamp, B. Kemp, and T. T. Canh. 2005. Effects of increasing temperatures on physiological changes in pigs at different relative humidities. *J Anim Sci.* 83:1385-1396.
- [75] Collin, A., J. van Milgen, S. Dubois, and J. Noblet. 2001. Effect of high temperature on feeding behavior and heat production in group-housed young pigs. *Br J Nutr.* 86:63-70.
- [76] Myer, R., and R. Bucklin. 2001. Influence of hot-humid environment on growth performance and reproduction of swine. University of Florida, IFAS Extension. Available: <http://edis.ifas.ufl.edu/AN107>. Accessed May 30, 2007.
- [77] Iowa State University. 2002. Heat stress index chart for swine producers. Available: <http://www.thepigsite.com/articles/5/housing-and-environment/669/heat-stress-index-chart-for-swine-producers>. Accessed May 30, 2007.
- [78] Verstegen, M. W. A., W. H. Close, I. B. Start, and L. E. Mount. 1973. The effects of environmental temperature and plane of nutrition on heat loss, energy retention and deposition of protein and fat in groups of growing pigs. *Br J Nutr.* 30:21-35.
- [79] Kouba, M., D. Hermier, and J. Le Dividich. 1999. Influence of a high ambient temperature on stearyl-CoA desaturase activity in the growing pig. *Comp Biochem Physiol.* 124B:7-13.
- [80] Lopez, J., G. W. Jesse, B. A. Becker, and M. R. Ellersieck. 1991. Effects of temperature on the performance of finishing swine: I. Effects of a hot, diurnal temperature on average daily gain, feed intake, and feed efficiency. *J Anim Sci.* 69:1843-1849.
- [81] Nienaber, J. A., G. L. Hahn, and R. A. Eigenberg. 1999. Quantifying livestock responses for heat-stress management: a review. *Int J Biometeorol.* 42:183-188.

- [82] Le Bellego, L., J. van Milgen, and J. Noblet. 2002. Effect of high temperature and low-protein diets on the performance of growing-finishing pigs. *J Anim Sci.* 80:691-701.86.
- Kouba, M., D. Hermier, and J. Le Dividich. 2001. Influence of a high ambient temperature on lipid metabolism in the growing pig. *J Anim Sci.* 79:81-87.
- [83] Breinekova, K., M. Svoboda, M. Smutna, L. Vorlova. 2006. Markers of acute stress in pigs. *Physiological Research Pre-Press Article.*
- [84] Becker, B. A., J. A. Nienaber, R. K. Christenson, R. C. Manak, J. A. DeShazer, and G. L. Hahn. 1985. Peripheral concentrations of cortisol as an indicator of stress in the pig. *Am J Vet Res.* 46(5):1034-1038.
- [85] Chrousos, G. P. 2007. Adrenocorticosteroids & Adrenocortical Antagonists. Ch 39 in *Basic & Clinical Pharmacology*, 10th Ed. B.G. Katzung, ed. McGraw-Hill, New York, NY.
- [86] Missing Reference
- [87] Kerr, C. A., L. R. Giles, M. R. Jones, and A. Reverter. 2005. Effects of grouping unfamiliar cohorts, high ambient temperature and stocking density on live performance of growing pigs. *J Anim Sci.* 83:908-915.
- [88] Person, R. C., D. R. McKenna, D. B. Griffin, F. K. McKeith, J. A. Scanga, K. E. Belk, G. C. Smith, and J. W. Savell. 2005. Benchmarking value in the pork supply chain: Processing characteristics and consumer evaluations of pork bellies of different thicknesses when manufactured into bacon. *Meat Science.* 70:121-131.
- [89] Morgan, J. B., G. C. Smith, J. Cannon, F. McKeith, and J. Heavner. 1994. Pork distribution channel audit report. In: *Pork Chain Quality Audit-Progress Report*. D. Meeker and S. Sonka, ed. NCCP, Des Moines, IA.
- [90] Gatlin, L. A., M. T. See, J. A. Hansen, D. Sutton, and J. Odle. 2002. The effects of dietary fat sources, levels, and feeding intervals on pork fatty acid composition. *J Anim Sci.* 80:1606-1615.
- [91] Wood, J. D., R. I. Richardson, G. R. Nute, A. V. Fisher, M. M. Campo, E. Kasapidou, P. R. Sheard, and M. Enser. 2003. Effects of fatty acids on meat quality: a review. *Meat Science.* 66:21-32.
- [92] Delmore, R. J. (2009). Beef Shelf-life. Cattlemen's Beef Board and National Cattlemen's Beef Association.
- [93] Singh, R. K., & Singh, N. (2005). Quality of packaged foods. In J. H. Han (Ed.), *Innovations in Food Packaging*, (pp. 22-24). Amsterdam: Elsevier Academic Press.
- [94] Gerrard, D. E. and A. L. Grant. 2003. *Principles of Animal Growth and Development* (1st ed.). Dubuque, IA: Kendall/Hunt Publishing Co.
- [95] Wood, J. D., R. I. Richardson, G. R. Nute, A. V. Fisher, M. M. Campo, E. Kasapidou, P. R. Sheard, and M. Enser. 2004. Effects of fatty acids on meat quality: a review. *Meat Science*, 66:21-32.