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NUTRITIONAL MANIPULATION OF MEAT QUALITY

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Abstract

Pork quality is still evolving as an area of intense interest as consumer demand for tender, juicy and tasty meat strongly influences the focus of the industry towards the quality of its products. The metabolism of glycogen is highlighted as a key determinant of pork quality through its influence on the rate and extent of pH decline post-slaughter. There is considerable genetic and nutritional scope for increasing muscle glycogen concentration pre-slaughter so as to prevent dark, firm, dry (DFD) pork but the resulting associated risk of increased pale, soft, exudative (PSE) pork would suggest that increasing muscle glycogen is not beneficial. However dietary manipulation to reduce the rate of glycolysis in muscle pre- and post-slaughter is of special interest since it will be associated with a reduced incidence of PSE pork. The role of dietary tryptophan and its effects on brain serotonin levels is discussed as a way of 'calming' pigs pre-slaughter. An additional approach is to utilize dietary magnesium as a means of reducing the secretion of catecholamines and so reduce the rate of post-slaughter glycolysis and PSE pork. The regulation of fat texture at both the subcutaneous and intramuscular sites is then discussed. The modern pig tends to show low rates of lipogenesis *de novo* and so relatively small changes in the ratio of saturated/unsaturated fatty acids in the diet can influence fat texture with decreases in the ratio being associated with softer fat. While the consumer associates more unsaturated fat with the perception of improved health, unsaturated fat has a softer texture and it is less desirable for the meat processing sector. This dichotomy needs to be addressed. Finally the role of vitamin E in promoting meat colour and reducing the incidence of PSE is discussed. Vitamin E supplementation is associated with improved meat colour during storage and reduced lipid oxidation, however the incidence of PSE is not reduced.

Introduction

Meat quality is a complex term involving many attributes that affect the technological and sensory quality of pork. This review focuses on the potential for nutritional modification of meat quality. The regulation of glycogen metabolism and the potential for dietary control is first discussed. It is followed by considering the dietary factors which can modify fat texture and quality.

Glycogen and pH

Regulation of glycogen metabolism

The rate and extent of post-slaughter change in the pH of pork is considered the single most important cause of variation in pork quality (Bendall and Swatland, 1988). The rate of pH and temperature decline in meat post slaughter influences protein denaturation, and when the rate of muscle pH decline is rapid while the carcass temperature is still high, then pale, soft, exudative (PSE) pork eventuates. When the decline in pH is not sufficient the meat becomes dark, firm and dry (DFD), and when the decline is too extensive the meat becomes pale and loses water resulting in lower cooking yield ('acid' pork; Sellier and Monin, 1994). The post-slaughter change in pH is largely based on the degradation of glycogen to lactic acid by glycogenolysis and glycolysis.

Glycogen represents the store of body carbohydrate with the quantitatively most important reserves found in the liver and skeletal muscle. The role of hepatic glycogen is primarily for the maintenance of blood glucose while the glycogen in skeletal muscle represents an energy reserve that can be rapidly mobilised. Typically glycogen in muscle

is mobilised during exercise, especially when the exercise calls upon a significant level of anaerobic metabolism.

Glycogen is a large (MW=10^7) branched polymer of glucose with each glycogen molecule associated with a protein primer and the enzymes of glycogen metabolism. The protein primer, glycogenin is required to form the template for initial synthesis of glycogen. The physiological role of glycogenin in the regulation of glycogen concentrations is poorly understood. However there is potential for regulation of glycogen concentrations at this step since it is the number of glycogenin primer molecules that will determine the number of glycogen granules (Alonso *et al.*, 1995). The maximum size of each granule is thought to be limited due to inhibition of glycogen synthase as the glycogen molecule increases in size.

The relative balance between glycogen biosynthesis and breakdown is controlled by regulation of glycogen phosphorylase and glycogen synthase. Anaerobic breakdown of glycogen is designed to allow for a very rapid acceleration (<5 secs to attain Vmax) and high final activity (high Vmax) (Sahlin, 1986). In contrast, synthesis is a more chronic process lasting hours to days.

Glycogen synthesis in skeletal muscle typically requires blood glucose as the substrate and is classically thought to be regulated at two steps; firstly, by the entry of glucose into the cell, which is regulated by the transport protein GLUT4, and secondly by the activity of glycogen synthase (Sugden and Holness, 1997). There is evidence for regulation at both levels with glucose availability and insulin concentration in the blood being key positive influences. Hormones such as the catecholamines strongly inhibit glycogen synthesis as a result of 3',5'-cyclic adenosine monophosphate (cAMP) induced phosphorylation of glycogen synthase. In addition, there is a more novel pathway for glycogen resynthesis in muscle which has been shown to exist in the type IIb muscle fibres of the rat after high intensity (sprint) exercise. During the resting phase glycogen resynthesis occurs within the muscle fibre via a reversal of glycolysis which can become thermodynamically favourable as a result of very high lactic acid accumulation (Palmer and Fournier, 1997).

Regulation of glycogenolysis is the most relevant metabolic pathway affecting pork quality, since this determines the rate and extent of pH decline. Glycogenolysis involves activation of glycogen phosphorylase which breaks down glycogen to release glucose-1-P for entry into glycolysis. The regulation of this step in skeletal muscle is by three primary mechanisms (Murray *et al.*, 1996). The classical pathway for activation of glycogen phosphorylase is by the cAMP dependant cascade resulting in phosphorylation of the enzyme. Catecholamine hormones and/or neurotransmitters are thought to be the primary agents initiating this process, and accordingly physical activity and/or stress is sufficient to elevate catecholamines concentrations which initiate glycogenolysis (Sahlin, 1986). This could lead to glycogen depletion pre-slaughter and associated DFD meat. Alternatively, acute stress before or at stunning may result in an accelerated muscle pH decline while the carcass temperature is still high, resulting in PSE pork. The mechanisms for this are not entirely clear but may be related to changes in calcium metabolism. Calcium is a potent activator of muscle contraction and glycogenolysis and the resultant effect is for the rate of muscle glycolysis and pH decline to increase. Stunning procedures such as CO_2 are associated with lower rates of PSE (Barton Gade, 1997) and this is likely related to reduced catecholamine and calcium release at and after stunning.

Muscle fibre type

Skeletal muscle is not a uniform tissue but instead consists of several different fibre types. Type I fibres are slow contracting, while type II are fast contracting. The type II fibres are split further into type IIa and type IIb. Type IIa fibres are fast contracting but also have good aerobic activity and give muscle a red colour (along with type I fibres). The metabolism of glycogen is different in the various fibres - type I fibres have low levels of glycogen. Type IIa fibres have high levels of glycogen, a high rate of glycogen resynthesis and loss of glycogen is least affected by acute stress. Type IIb fibres have lower glycogen levels, slower rates of glycogen synthesis and are the most susceptible to acute stress-induced glycogen depletion (Monin, 1981; Holness *et al.*, 1997). The metabolic differences between muscle fibres can be largely explained by the different

enzyme complement of each fibre type (Table 3). The very high activity of glycogen phosphorylase in combination with low activities of glycogen synthase and hexokinase mean that type IIb muscle fibres rapidly deplete and slowly replete glycogen levels.

Table 3. Enzyme activities for carbohydrate metabolism in rat skeletal muscle (Adapted from Saltin and Gollnick (1983).

	Enzyme activity for each fibre type (μmol of glucose converted/min/g of muscle)		
	Type I	Type IIa	Type IIb
Enzyme			
Glycogen phosphorylase	14	115	171
Glycogen synthase	6	10	5
Hexokinase	2	2	0.8

The skeletal muscle of the pig generally has much higher levels of type IIb fibres than the 'red meat' animals such as sheep and cattle. For example the *m. longissimus dorsi* of ruminants has a ratio of type I:IIa:IIb muscle fibres of 50:40:10 (Suzuki, 1971; Aalhus and Price, 1991) while in the pig the ratio is 8:8:84 (Karlsson *et al.*, 1994). Theoretically this would make the pig susceptible to (i) loss of glycogen from muscle pre-slaughter (i.e., DFD) and (ii) to an accelerated rate of glycogenolysis post-slaughter (i.e., PSE). In practice the incidence of DFD in pigs is not different to those rates reported in ruminants (Barton Gade, 1997; Fabiansson *et al.*, 1989). This may be attributed to the pig having a reduced sensitivity to catecholamines when compared to the ruminant (Pethick and Dunshea, 1996). In addition the decline in muscle glycogen during fasting is slower in type II fibres compared to type 1 (Fernandez *et al.*, 1995).

Fasting and sugar feeding

Fasting of pigs for up to 72 h pre-slaughter, in the absence of other stressors, has minimal effect on muscle glycogen content, certainly insufficient to cause DFD meat (Fernandez and Tornberg, 1991). It is well-known that the stressful events that occur between farm and slaughter, including loading and unloading, trucking, unfamiliar environments, mixing and fighting in lairage can induce muscle glycogen depletion and cause the occurrence of DFD pork (Fernandez and Tornberg, 1991). Activation of the sympathetic nervous system is enhanced in rats undergoing food deprivation (Weick *et al.*, 1983) and this may also occur for pigs although no evidence has been produced to date.

A long waiting time at the abattoir is known to increase the occurrence of DFD pork (Fernandez and Tornberg, 1991), thus sugar feeding to prevent the depletion of muscle glycogen has been investigated. Diet composition does not generally affect the muscle glycogen content of pigs if conventional energy sources are used. Sugar feeding in the last few days prior to slaughter can increase muscle glycogen content (Sayre *et al.*, 1963). Compared to controls, pigs provided with glucose in the water overnight during lairage have higher muscle glycogen concentrations. This has the effect of reducing the ultimate pH (pHu) of the meat from the high levels seen in DFD pork (pHu > 6.0) to the pHu of normal pork (5.5-5.9), and thus prevents the occurrence of DFD pork (Fernandez *et al.*, 1979; Gallwey and Tarrant, 1979; Gardner and Cooper, 1979). It is not clear from these studies whether muscle glycogen depletion was prevented or if repletion was enhanced. The mechanisms need to be understood so that sugar feeding can be used optimally under the range of situations which occur in commercial practice, and to determine if the sugar feeding should occur on the farm or at the abattoir. The advantage of sugar feeding is that it increases muscle glycogen content but by decreasing the propensity for the occurrence of DFD, the propensity for PSE may be increased. The occurrence of DFD pork has negative welfare implications as well as problems with reduced shelf life and potential problems with food safety. Thus DFD is as undesirable as PSE and both need to be prevented. The tendency towards producing PSE with sugar feeding would particularly be the case for stress susceptible animals or abattoir systems where pre-

slaughter stress is high. Also, it is clear that the occurrence of PSE pork results in an economic loss to the pig industry as it is unacceptable to the consumer due to its poor appearance and unacceptable palatability. However, the palatability of DFD is highly acceptable to the consumer as the meat is very tender and juicy due to its high water-holding capacity (Warner, 1994), and it is preferred for sausage manufacture. Thus a decrease in the occurrence of DFD at the expense of a possible increase in the occurrence of PSE pork is probably undesirable.

Exercise

Physical training is known to increase glycogen concentrations of skeletal muscle of the pig (Essen-Gustavsson *et al.*, 1988). The mechanism is poorly understood but in part relates to changes in fibre type. The pig is usually housed intensively and so this mode of control is probably of little practical importance.

Genetic influences

The discovery of the RN gene points to some level of genetic control of glycogen concentrations in skeletal muscle . This gene is associated with an 80% increase in glycogen level of muscle and a lower ultimate pH (pHu) of meat. The rate of muscle pH decline in pigs carrying the RN gene is similar to genetically normal animals (Monin and Sellier, 1985; Hermesch, 1997) suggesting that PSE pork is not associated with elevated glycogen in muscle at slaughter. The high drip loss and poor cooking yield in pigs carrying the RN gene is thought to be directly related to the lower than normal pHu (Lundström *et al.*, 1996). It is thought that the elevated glycogen levels drives the pHu lower than normal. However the relationship between the pHu of meat and glycogen levels of the muscle in RN gene carriers is not strong (Lundström *et al.*, 1996) suggesting that factors in addition to glycogen levels are involved.

The report of positive effects of the RN gene on taste and aroma intensity of cooked pork is of interest and may be related to the low pHu (Lundström *et al.*, 1996). However it is possible that flavour- and aroma-facilitating compounds arise from the interactions between protein and the 'extra' carbohydrate during cooking (i.e., the Maillard reaction; Farmer, 1992). On this basis, elevated residual glycogen levels in meat may be associated with improved flavours upon cooking.

Given the current level of understanding producers need to make a decision on managing glycogen levels. Options include procedures for manipulation of glycogen levels or utilising nutritional tools for slowing glycogen breakdown. The most appropriate 'post farm' gate procedures will depend upon the financial incentives offered by processors.

Dietary regulation of glycogen breakdown

Tryptophan

The main hormonal responses to sudden stress are the release of neurotransmitters in the brain which results in stimulation of the nervous system, and the release of stress hormones into the blood stream which results in stimulation of muscle metabolism. The relationship between stress and neurotransmitters in the brain may indicate ways of reducing the response of pigs to stress and thus reduce glycogen breakdown pre- and post-slaughter. Dietary tryptophan has been used to alleviate 'hysteria' in laying hens (Laycock and Ball, unpublished), reduce the stress response of horses to transport, and reduce pain sensitivity in mice and rats. Ball (1988) reported a relationship between brain serotonin and stress susceptibility in market pigs and subsequently showed that adding 5g of tryptophan/kg to the diet of finisher pigs for 5 d pre-slaughter resulted in increased concentrations of serotonin in the hypothalamus and a reduction in the incidence and severity of PSE (Ball *et al.*, 1988). Warner *et al.* (1990) fed 5g of tryptophan/kg of feed to finisher pigs for 5 d pre-slaughter and monitored their behaviour in pens on the farm and at the abattoir. There was no difference in behaviour on the farm between treated and untreated groups of pigs. When the pigs were exposed

to the unfamiliar environment of the abattoir and mixed in lairage pens (within treatments), the tryptophan-treated pigs were markedly less aggressive (see Figure 3) and exhibited less mounting activity in the lairage pens than the control pigs. The tryptophan-treated carcasses also had less blemishes but there was no difference in the meat quality.

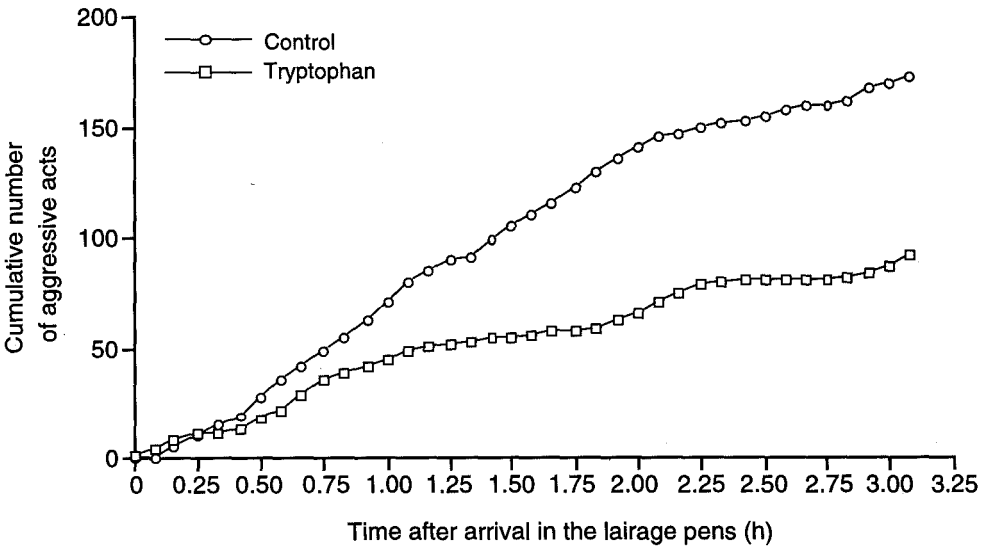

Figure 3. *Cumulative number of aggressive acts of pigs in separate lairage pens for the control and tryptophan-treated pigs during the 3 h in lairage subsequent to arrival at the abattoir.*

Magnesium Compounds

Niemack *et al.* (1979) and Kietzman and Jablonski (1985) have shown that dietary magnesium supplementation is effective in reducing the effect of stress in pigs by reducing plasma cortisol, noradrenaline, adrenaline and dopamine concentrations. This has led to the suggestion that magnesium supplementation may be a viable option for reducing glycolysis pre- and post-slaughter and therefore improving meat quality (Kuhn *et al.*, 1981) and reducing the incidence of PSE (Otten *et al.*, 1992; Schaefer *et al.*, 1993). Magnesium is an essential cofactor for numerous metabolic and enzymatic pathways (Stryer, 1988). Magnesium directly depresses skeletal muscle activity by antagonising calcium at the site of the voltage gated channels in the pre-synaptic terminal which prevents migration and exocytosis of vesicles containing the neurotransmitters to the surface of the pre-synaptic junction. Thus magnesium causes a reduction in the secretion of acetylcholine by motor-nerve impulses, which in turn reduces neuromuscular stimulation (Hubbard, 1973; Hagiwara *et al.*, 1974). Magnesium may be similarly involved in reducing the release of catecholamines (noradrenaline and adrenaline) from both nerve terminals and the adrenal glands (Classen *et al.*, 1983; Herman and Brown, 1983; Kuhn *et al.*, 1981; Kietzmann and Jablonski, 1985).

Otten *et al.* (1992) have reported that long-term (from grower to slaughter weights) dietary magnesium fumarate supplementation (10 and 20 g magnesium fumarate/kg diet; 30-100 kg live weight) in pigs resulted in higher initial muscle pH and conductivity values and less pale meat compared to pigs which were fed a standard finisher diet. Schaefer *et al.* (1993) also reported that meat from pigs fed short-term dietary supplementation of magnesium aspartate (40 g magnesium aspartate-HCl/pig per d for 5 d) displayed reduced muscle temperatures at 45 min post-slaughter and a reduced % drip loss. Dietary supplementation of pigs with magnesium aspartate at 40 g/pig per d for 5 d increased plasma magnesium concentrations by 6%, reduced plasma noradrenaline concentrations and resulted in lower % drip loss and less pale meat compared to pigs fed

the control diet (D'Souza *et al.*, 1997; see Table 4). While negative handling significantly increased the % drip loss (2.4 %) in pigs from the control diet, there was no difference in % drip loss in pigs fed the magnesium aspartate supplemented diet irrespective of the handling treatment. The results presented in Table 4 demonstrate that the use dietary magnesium aspartate supplementation in pigs can be used to reduce the effects of pre-slaughter 'stress' in pigs and hence improve meat quality and reduce the incidence of PSE meat.

Table 4. The effect of dietary magnesium aspartate (Mg Asp) supplementation and pre-slaughter handling (minimum or negative) on plasma noradrenaline and adrenaline concentrations, muscle glycogen and lactic acid concentrations in the *Longissimus thoracis* at slaughter, and muscle pH decline and meat quality indicators in the *Longissimus thoracis* at 24 h post-slaughter.

Diet (D)	Control		Mg Asp			P - values		
Handling (H)	Minimum	Negative	Minimum	Negative	sed	D	H	DxH
Noradrenaline[1] (nmol/ml)	1.79	1.24	0.89	1.05	0.380	0.048	0.470	0.194
Adrenaline[1] (nmol/ml)	0.40	0.43	0.32	0.33	0.085	0.150	0.729	0.945
Glycogen[1] (mg/g)	8.4	6.9	9.6	9.4	0.818	0.003	0.136	0.292
Lactic acid[1] (mg/g)	3.8	4.2	3.2	3.5	0.420	0.036	0.229	0.671
pH at 40 min post-slaughter	6.60	6.59	6.79	6.69	0.074	0.007	0.285	0.431
Ultimate pH[2]	5.48	5.51	5.61	5.57	0.045	0.004	0.864	0.224
% Drip Loss[2]	4.0	6.4	3.5	3.5	0.824	0.006	0.047	0.047
Lightness-L*[2]	48.7	49.1	45.2	47.4	1.109	0.002	0.115	0.247
% PSE[2,3]	8	33	0	0	-	0.050	0.280	0.093

[1]Measured at slaughter. [2]Measured at 24 h post-slaughter. [3]Exact contingency table test used.

Recently reported compounds

The addition of high levels of L-carnitine (Vitamin Bt, up to 300mg/kg) has been reported to improve the in vitro digestibility of muscle (Bonomi, 1995; as cited by Mordenti and Marchetti, 1996) and reduce the paleness of pork (Sardi *et al.*, 1996; as cited by Mordenti and Marchetti, 1996). The addition of up to 150 mg/kg of niacin (Vitamin PP) in the diet for 7 d pre-slaughter may increase muscle glycogen content although the effects on meat quality would need to be investigated in more detail (Piva *et al.*, 1995; as cited by Mordenti and Marchetti, 1996).

Acid and alkaline salts administered in the drinking water for 4 d pre-slaughter have been found to influence pork quality; oral loading with 8g/l of ammonium chloride detrimentally affected pork quality whereas 12.6g/l of sodium bicarbonate tended to improve pork quality (Boles *et al.*, 1993; 1994).

Dietary and other regulation of fat quality

Dietary fat and fat texture

Genetic selection and nutritional manipulation over the last 15 years have focused on reducing the amount of subcutaneous fat with a corresponding increase in feed conversion efficiency by the animal. However, anecdotal and scientific reports suggest

that these putative improvements have been to the detriment of eating or processing quality (Wood, 1993). Many of the flavour components (both positive and negative) are found in fat and the reductions in fat content of the pig can lead to alterations in eating quality. Although from a productive efficiency point of view it is desirable to reduce excessive deposition of fat subcutaneously it may be advantageous to ensure that intramuscular fat is not reduced to levels that compromise meat quality. Coupled to this is the desire by consumers and health authorities to reduce the consumption of saturated fatty acids while increasing the consumption of some of the n-3-polyunsaturated fatty acids. Thus, many in the butchering and processing sector would prefer fat rich in saturated fatty acids whereas the marketing sector and the health professionals would prefer unsaturated fatty acids.

From a processors perspective poor fat quality manifests itself as soft fat and lean/fat separation. With the trend towards producing leaner carcasses through either genetic selection, use of intact males or dietary and hormonal manipulation there has been an increase in the incidence of soft fat. In a review of the genetic effects on fat quality in the growing pig, Metz (1985) concluded that the prevalence of soft fat in lean pigs was due to decreased *de novo* lipogenesis and increased reliance on dietary fatty acids rather than to any genetic predisposition towards preferentially depositing unsaturated fatty acids. Subcutaneous adipose tissue triglyceride fatty acids are derived from either *de novo* lipogenesis or are incorporated directly from dietary fatty acids. The principle fatty acids produced *de novo* in pigs fed a fat free diet are the saturated fatty acids, palmitic acid and stearic acid and the mono-unsaturated fatty acid, oleic acid (Leat *et al.*, 1964; Metz and Decker, 1981). However, inclusion of vegetable oils in the diet dramatically increases the unsaturated fatty acid content of adipose tissue triglyceride (Leat *et al.*, 1964; Metz and Decker, 1981; Marchello *et al.*, 1983; St John *et al.*, 1987; Leskanich *et al.*, 1997). Fat firmness is largely governed by the degree of saturation of the triglyceride fatty acids with soft fat being associated with unsaturated fatty acids. Therefore, there is potential for dietary fatty acids to influence carcass fat quality. For example, in pigs fed an isocaloric diet but differing widely in source and type of fat, there were high correlations between individual fatty acids in the diet and in carcass fat (Hertzman *et al.*, 1988) with diets high in unsaturated fatty acids resulting in softer fat. However, carcass fat can be influenced by even moderate changes in dietary fat. Leskanich *et al.* (1997) recently conducted an experiment with a relatively lean genotype where changing the added dietary fat from 3% tallow: soybean oil (4:1) with 3% canola: fish oil (2:1) resulted in softer subcutaneous fat, particularly over the shoulder.

In many parts of the world high dietary copper levels are used to promote growth but high dietary copper has also been shown to increase the ratio of oleic to stearic acid in the backfat of pigs through stimulation of desaturase activity (Moore *et al.*, 1969). However, the effects upon backfat softness are more profound than can be attributed to gross fatty acid composition alone and other factors need to be considered. For example, Moore *et al.* (1969) selected some back fat samples with similar fatty acid composition but widely different melting points. After chemical randomisation of the position of the fatty acids on the triglyceride molecule *in vitro*, the melting points of the backfat were more uniform. Although individual fatty acids are preferentially incorporated into specific positions in the triglyceride molecule, considerable scope does exist for positional isomers (Brockerhoffe *et al.*, 1966). The synthesis of positional isomers in turn may be related to whether the fatty acid is synthesised *de novo* or of dietary source. Therefore, in addition to fatty acid composition, position of the fatty acids within the triglyceride molecule can be an important determinant of fat quality. Leskanich *et al.* (1997) cites work from his PhD dissertation where inclusion of dietary polyunsaturated fatty acids actually increased backfat firmness in the presence of high levels of linoleic acid, presumably through specific changes in the distribution of triglyceride molecular species (Leskanich, 1995).

Biotin is an important vitamin involved in the formation of malonyl CoA, a rate limiting step in *de novo* fatty acid synthesis and chain elongation of linoleic and linolenic acids. Therefore, lipid obtained from the subcutaneous adipose tissue of biotin deficient pigs has low levels of the major saturated fatty acids synthesised *de novo* (palmitic and stearic acids) while linoleic acid accumulates (Glattli *et al.*, 1973). Increasing the level of

biotin reduces the ratio of unsaturated to saturated fatty acids and so can have marked effects upon fat quality.

While there is little evidence of genetic control over the deposition of polyunsaturated fatty acids in subcutaneous fat (Metz, 1985), there are quite marked genetic influences on the amount and type of fat deposited intramuscularly (Hermesch, 1997). There is also potential to alter the composition of intramuscular fat by dietary means although not to the same extent as subcutaneous fat (Marchello *et al.*, 1983). For example, inclusion of sunflower oil in the diet increased the linoleic acid and decreased the oleic acid content of intramuscular fat of pigs (Marchello *et al.*, 1983). Leskanich *et al.* (1997) were able to use dietary manipulation with canola and fish meal to increase the levels of desirable unsaturated fatty acids in intramuscular fat. However, changes in the fatty acid profile were not at the expense of saturated fatty acids nor was total intramuscular fat altered. Rather, monounsaturated fatty acids were decreased. Many fish meals are also rich in oils containing n-3 fatty acids and fishmeal is often used as a protein supplement in pig diets. A problem with this approach is that meat can become tainted with a "fishy flavour" if fishmeal is included at too high a level or for extended periods of time. However, recent research has suggested that feeding 20% Porcmega fishmeal for 6 to 10 weeks followed by a one week withdrawal can increase intramuscular n-3 fatty acids without compromising handling and processing quality (Howe *et al.*, 1996).

A potential novel method of altering fat deposition for the future may be through the dietary inclusion of conjugated linoleic acid (CLA). Conjugated linoleic acid has one double bond in the *cis* and the other in the *trans* configuration with no methylene interruption probably giving it a shape more like oleic acid. The fatty acid is found in appreciable levels in dairy products and has been shown to increase feed efficiency in rats, mice and chickens (Chin *et al.*, 1994), and to decrease carcass fat content in mice (Albright *et al.*, 1996). It is possible that this fatty acid can be used to manipulate the amount and type of fat deposited in pigs. It is worth noting that CLA itself is a potent antioxidant and anticarcinogen. The former characteristic may ensure some protection against oxidative rancidity while the health and marketing benefits of any anticarcinogen effects are obvious.

Vitamin E

The beneficial effects of dietary supplementation with Vitamin E on aspects of meat quality in all major meat-producing species has been reported by various investigators (see Mitsumoto *et al.*, 1993 for review). The improvement in lipid and colour stability during retail display of beef from cattle supplemented with Vitamin E (Mitsumoto *et al.*, 1991) and in the colour and lipid stability of pork from pigs supplemented with Vitamin E (Monahan *et al.*, 1992) can be attributed to the antioxidant effect preventing both lipid and myoglobin oxidation. Dietary vitamin E has been shown to reduce drip loss of thawed pork (Asghar *et al.*, 1991; Monahan *et al.*, 1994) and fresh beef muscle (Mitsumoto *et al.*, 1995). It has been suggested that the basic mechanism reducing drip loss in muscles from carcasses of vitamin E-fed pigs is alterations in muscle cell membrane permeability to calcium. This is postulated to result in the reduction in rate of muscle glycolysis at slaughter. Rapid rates of glycolysis post-slaughter result in the PSE defect, particularly in pigs di-mutant for the *ryr1* gene (previously called the 'halothane' gene) (Duthie *et al.*, 1989, 1992; Cheah and Cheah, 1985). Pigs which are di-mutant for the *ryr*1 gene have increased requirement for anti-oxidant defence mechanisms in the cells which results in sustained oxidative stress. This oxidative stress that *ryr1* pigs undergo is reported to be alleviated by supplementation of the diet with Vitamin E (Duthie *et al.*, 1989). However, Asghar *et al.* (1991) and Monahan *et al.* (1994) attribute the reduced drip loss in the meat of Vitamin E-fed pigs to a reduction in lipid oxidation in cell membranes. Conceivably, this could reduce the movement of water across the muscle cell membrane post-slaughter. Warner *et al.* (1995) found that feeding pigs di-mutant for the *ryr*1 gene 200 IU of Vitamin E/kg of feed for 40 d pre-slaughter did not prevent the PSE condition and had no major effects on water-holding capacity. However dietary supplementation with vitamin E prevented lipid oxidation until 9 d post-slaughter whereas lipid oxidation in control

samples increased to unacceptably high levels after 2 d post-slaughter and continued to increase (Figure 4).

Figure 4. *Changes in lipid oxidation (TBARS value, mg malonaldehyde/kg of meat) for each treatment with days of suspension post slaughter. Each point is a least squares mean of 3 (control; SE ± 0.093) or 5 (vitamin E; SE ± 0.072) pigs.*

Conclusion

Given the relative intensive nature of modern pig production and the increasing use of specific diets, there is considerable potential to improve meat quality through nutritional manipulation. The key role of glycogen and its metabolites in determining final meat quality suggest that manipulation of muscle glycogen depletion and repletion is an area to target. There is a risk that increasing muscle glycogen may result in an increase in PSE pork. On the other hand, reducing the rate of glycolysis in muscle pre- and post-slaughter through inclusion of dietary magnesium may be useful. The relatively low rate of *de novo* lipogenesis in the modern pig means that deposited fat reflects dietary fat and so can be easily manipulated. The difficulty here is balancing the requirements of the processor with that of the consumer. Another means of manipulating meat quality is through the inclusion of Vitamin E which has been shown to improve meat colour and reduce lipid oxidation during storage.

Symposium continued on next page