

Swine nutrition and pork quality: A review¹

J. E. Pettigrew*² and M. A. Esnaola†

*Pettigrew Consulting International, LLC, Louisiana, MO 63353 and

†Animal Production International Consulting, Roseville, MN 55113

ABSTRACT: The rapidly expanding body of information concerning swine nutritional impacts on pork quality was reviewed. Energy is required to support muscle growth, but excess energy intake increases fatness. Energy restriction increases leanness but reduces marbling. If amino acid intake is inadequate to maximize protein accretion rate, pigs grow slowly and produce fatter carcasses but have more marbling. Supplementation of the diet with chromium increases muscling, but recent data do not support the early observations of reduced backfat thickness. Addition of conjugated linoleic acid (CLA) to the diet produces leaner carcasses. The softness of fat is related to the composition and level of dietary fat. Dietary fats containing a high level of *n*-3 fatty acids appear to increase the incidence of off-flavors in pork. Dietary CLA increases the firmness of carcass fat, but a high dietary level of copper de-

creases it. A high dietary level of vitamin E consistently improves the oxidative stability of pork. It appears that under some circumstances a preslaughter feed deprivation reduces the incidence of PSE. Specific inhibitors of key glycolytic enzymes appear to improve quality characteristics of pork muscle, including pH, water-holding capacity, and color. Addition of a high level of magnesium to the diet for a few days before slaughter markedly reduces the incidence of PSE. Supplemental dietary creatine may improve some muscle quality characteristics. Supplementing the diet with a high level of vitamin D has not been shown to increase tenderness of pork, as it has for beef. However, it may be that further research will identify a combination of dietary concentration and duration of feeding that will improve tenderness. Nutritional means to improve pork quality exist.

Key Words: Leanness, Meat Quality, Nutrition, Pigs, Pork, Reviews

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Introduction

Many factors affect pork quality, including genetics, preslaughter handling of the pig, and postslaughter handling of the carcass. An accumulating body of evidence shows that various nutritional factors affect pork quality. The purpose of this review is to assemble and interpret the rapidly accumulating data concerning the impacts of swine nutrition on pork quality.

Microbial safety and freedom from toxins and residues are important components of pork quality that are outside the scope of this review.

Nutritional Factors Associated with Mineral and Vitamin Levels in Pork

Some pork producers withdraw some or all of the supplemental minerals and vitamins from feed given to pigs during the last few weeks of growth as a cost-saving measure. Withdrawal of supplemental vitamins and trace minerals from the diet for 6 or 12 wk before slaughter reduced the levels of vitamin E and copper in muscle (Edmonds and Arentson, 2001). Zinc levels were clearly not affected, and the 21% reduction in tissue iron concentration was not statistically significant.

Nutritional Factors Associated with Lean:Fat Ratios (Carcass Leanness and Marbling)

The lean:fat ratio is important both in the overall carcass and within the muscle (marbling). Carcass fatness and marbling are not perfectly correlated, but in general nutritional interventions that alter one cause the other to move in the same direction also.

The relative amounts of protein (or lean) and fat in a pig's body determine leanness. We can increase lean-

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²Present address: Dept. of Animal Sciences, 206 Animal Sciences Laboratory, 1207 W. Gregory Dr., Urbana, Illinois 61801 (phone: (217) 244-6927; fax: (217) 333-7861; E-mail: jepettig@uiuc.edu).

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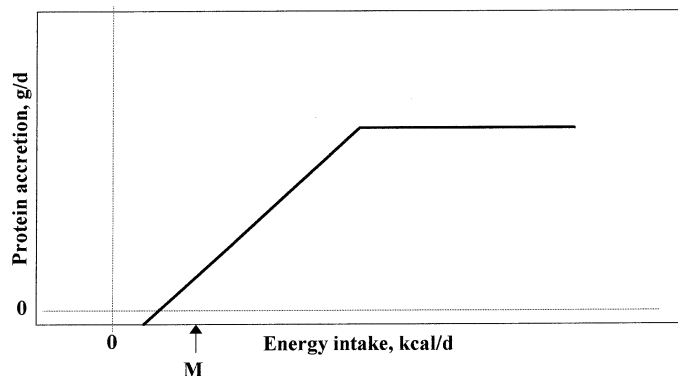


Figure 1. Conceptual relationship of protein accretion rate to energy intake. *M* is the maintenance energy requirement.

ness by increasing the amount of protein accreted (deposited), by reducing the amount of fat accreted, or both. If we can redirect the flow of nutrients that go to fat deposition and make them go to protein deposition, that will increase leanness.

The amount of protein a pig can accrete each day can be limited by either nutritional factors or nonnutritional factors. Nonnutritional factors put a ceiling on the amount of protein the pig is capable of accreting. The most important of these factors is genetics, but sex, health, and various environmental factors appear to have significant effects. The supply of nutrients also determines how much protein the pig can accrete. Therefore, protein accretion can be limited by the potential of a given pig in its environment (nonnutritional factors), by energy intake, or by amino acid intake. Protein deposition requires both energy and amino acids. If energy intake is limiting, increasing the amino acid levels of the diet does not increase protein deposition or leanness. If amino acid intake is limiting, increasing the energy intake will only make a pig fatter.

Energy

The prevailing concept of the relationship of protein accretion to energy intake is shown in Figure 1. The figure shows that as energy intake increases, protein accretion increases linearly until it reaches an upper limit. Further increases in energy intake have no effect on protein accretion (the line is flat). The upper limit can be set by the amino acid supply or by nonnutritional factors. Energy consumed in excess of that needed to reach the maximum rate of protein accretion all goes into fat accretion. Many young pigs cannot consume enough feed (energy) to reach the maximum protein accretion rate, and that situation sometimes continues until market weight. More often, pigs during at least part of the finishing period consume enough feed to pass the break-point of the line in Figure 1, and fatten rapidly. In that situation, further increases in energy intake reduce leanness dramatically.

Short of the energy intake required to reach maximum protein accretion, each increment of energy consumed is divided by the body. One part supports protein accretion, the other part supports fat accretion. The slope of the line reflects the proportion that goes to protein accretion. It is determined by the same nonnutritional factors that set a ceiling on protein accretion rate. Except in special circumstances discussed below, protein accretion is always accompanied by fat accretion.

It is important to note the lower left corner of the graph (Figure 1). It shows that at maintenance energy intake (*M*, the energy intake at which the pig neither gains nor loses body energy), the pig deposits protein. The energy to support that protein accretion comes from mobilization of body fat, so at maintenance energy intake the pig gains protein but loses fat. The importance of this occurs at higher energy intakes. Because of the starting points, each increment of energy intake increases fat accretion by a greater percentage than protein accretion. Therefore, increasing energy intake always makes pigs fatter, even if they do not reach the energy intake needed to maximize protein accretion rate. This is part of the reason that the fastest-growing pigs in a group are fatter than the others.

A reasonable target for energy intake is the amount needed to maximize protein accretion rate. That would avoid the excessive fatness that comes from higher energy intake. Pigs would be slightly leaner at lower energy intake, but they would also grow more slowly. Of course, it is not always possible to reach this target without the use of restricted feeding. The decline in popularity of restricted feeding in Europe reflects the fact that modern genotypes are much closer to this target with ad libitum consumption than were the older, fatter genotypes.

The discussion to this point has related to energy intake, not dietary energy density. Changing energy density usually changes energy intake, but the effects are often small. It is reasonable to use a high energy density during the early stages of growth and progressively lower energy density as the pig grows toward market weight. Some nutritionists use a high energy density (e.g., 3.5 Mcal ME/kg) all the way to market weight. That system increases growth rate in some situations, improves feed efficiency (reduces kilograms feed/kilogram gain) in all situations, increases dressing percentage, and makes pigs fatter. The increase in fatness is small in lean genotypes. The economics of such a system depend on nonnutritional factors, including genotype, and on the relative value of growth rate, feed efficiency, and leanness.

The empirical data generally support the concepts described above about the relationship of carcass leanness to energy intake and will not be reviewed here.

It seems the effects of energy intake on fatness extend to intramuscular fat as well as subcutaneous fat. Restricted feeding reduces the intramuscular fat concentration (Ellis et al., 1996; Blanchard et al., 1999). This

translated into reduced juiciness in two experiments (Warkup et al., 1990; Ellis et al., 1996) but not in a third (Blanchard et al., 1999).

Amino Acids

The challenge in setting dietary amino acid levels is conceptually simpler than the challenge in managing energy intake. The target is to provide just enough of each essential amino acid to maximize protein accretion. Lower levels reduce carcass leanness, but higher levels do not make pigs leaner.

However, this challenge is difficult in practice. The appropriate amino acid levels in the diet are different for different situations. Genotype is the most important factor affecting amino acid requirements, but health and environment also have impacts. Because the requirements vary from one situation to another, it is necessary to get important information about the situation at hand. That information is in the form of protein accretion rates and feed intake at each stage of growth. It must be quantitatively accurate. Gathering that information requires commitment of resources and attention.

We suggest using that information along with the mathematical model that is part of the *Nutrient Requirements of Swine*, recently published by the U.S. National Research Council (NRC, 1998). That model will take data from the farm and estimate the lysine requirement for any stage of growth. It will then use an ideal protein system to estimate the requirements for other amino acids. In addition to lysine, the amino acids threonine, methionine, cystine, and tryptophan deserve attention in diet formulation. In some situations, isoleucine or valine should be added to the list. Again, the empirical data on the response of carcass leanness to dietary amino acid levels generally support the concepts described above and will not be reviewed here.

These concepts extend to marbling, as shown in Table 1. The differences between protein-adequate and protein-deficient diets were not always statistically significant within the individual experiments, but in a total of 18 comparisons of marbling score and fat content of muscles, every one was in the direction of more marbling in pigs fed a low-protein diet. That consistency leaves no doubt about the existence of this response. On average, the deficient treatments increased marbling score by about 0.4 (on a 5-point scale) and percentage fat by about 1.4.

Unfortunately, these protein-deficient dietary treatments also impaired growth performance and increased the fatness of the carcass. These economically detrimental responses make it unlikely that the industry will choose this means of increasing marbling for general use. Perhaps low-protein diets could be used effectively in special circumstances or for specific market segments. Effects of dietary amino acid concentrations on

other aspects of pork quality are described near the end of the paper.

Chromium

Chromium is part of a compound often called the glucose tolerance factor (GTF). This GTF forms a three-way complex with insulin and the insulin receptor on the cell surface to potentiate insulin action. It seems that in some situations the amount of chromium in the body is insufficient to maximize the action of GTF, and therefore the effects of insulin. In those situations, dietary supplementation of an efficacious source of chromium improves glucose tolerance and enhances the clearance of glucose from the plasma in response to an insulin challenge, effects consistent with the accepted action of chromium. It is not clear how this physiological action of chromium is connected to the observed increases in leanness described below. Toxic effects of trivalent chromium have not been reported except at dietary levels far above the levels that cause these benefits.

Page et al. (1993) first reported that supplementation of pig diets with chromium picolinate during the finishing period increased carcass leanness. This observation was consistent with the reported use of chromium by people engaged in the sport of body building to increase muscle mass. This report stimulated a flurry of research activity to determine whether this critically important observation could be repeated.

The summary in Table 2 covers many of the results of that activity. This summary is restricted to studies of one form of chromium (chromium picolinate) at one level of dietary inclusion (200 ppb). It is restricted to data on carcass measurements at or near a conventional slaughter weight.

The first observation to be made from Table 2 is that supplemental dietary chromium picolinate appears to make pigs leaner. The unweighted averages of the data suggest that chromium reduced backfat thickness by about 1.5 mm, increased loin eye area by about 2 cm², and increased percentage lean in the carcass by about 1.5 percentage units. In each of the three measures, pigs fed chromium had more desirable mean values than did control pigs, and in about a third of the comparisons these advantages were statistically significant. In the case of loin eye area, 23 of 27 comparisons gave higher values for pigs fed chromium, three comparisons give higher values for the controls, and in one case the two treatments were equal. There were no reports of statistically significant detrimental effects of chromium.

However, Table 2 gives an overly optimistic impression of the complete body of information available. Some studies are excluded from the table because they used a different level or source of chromium, or because the studies have been reported only in abstract form without full data. We have identified 10 studies in these categories, and in eight of them dietary chromium sup-

Table 1. Effects of deficient dietary protein level on marbling

Source/description	Marbling score ^a				% Fat in muscle			
	Control	Deficient ^b	Difference	<i>P</i>	Control	Deficient ^b	Difference	<i>P</i>
Essen-Gustavsson et al., 1994								
Biceps femoris	—	—	—	—	1.3	2.0	0.70	<0.001
Longissimus	—	—	—	—	1.5	2.5	1.00	<0.01
Goerl et al., 1995								
Longissimus	—	—	—	—	3.41	9.37	5.96	<0.01
Cured ham	—	—	—	—	5.95	9.06	3.11	NS
Cisneros et al., 1996								
Longissimus, 10–11th rib	2.31	3.25	0.94	NS	3.8	5.7	1.90	<0.05
Longissimus, 3–4th lumbar vertebrae	—	—	—	—	3.0	4.8	1.80	<0.05
Semimembranosus	—	—	—	—	3.2	5.0	1.80	<0.05
Cisneros et al., 1999								
1-Wk deficiency	2.33	2.67	0.34	NS	—	—	—	—
Longissimus, 10th rib	—	—	—	—	2.93	3.22	0.29	NS
Longissimus, 3–4th lumbar vertebrae	—	—	—	—	3.37	4.12	0.75	NS
3-Wk deficiency	2.17	2.33	0.16	NS	—	—	—	—
Longissimus, 10th rib	—	—	—	—	3.05	3.08	0.03	NS
Longissimus, 3–4th lumbar vertebrae	—	—	—	—	4.69	4.73	0.04	NS
5-Wk deficiency	2.60	2.67	0.07	NS	—	—	—	—
Longissimus, 10th rib	—	—	—	—	2.82	3.34	0.52	NS
Longissimus, 3–4th lumbar vertebrae	—	—	—	—	2.83	3.46	0.63	NS
Witte et al., 2000								
Longissimus	—	—	—	—	2.93	3.48	0.55	<0.05
Mean	—	—	0.38	—	—	—	1.36	—

^aFive-point scale, where 1 is devoid of marbling and 5 is moderately abundant or greater.

^bDegree and duration of deficiency varied.

plementation apparently failed to improve leanness significantly (Evock-Clover et al., 1993; Wenk et al., 1995; Green et al., 1997; O'Quinn et al., 1998b; van de Ligt et al., 1998; O'Quinn et al., 1999a; van de Ligt et al., 1999a,b). In one additional case chromium improved carcass leanness in barrows but not in gilts (Lindemann and Purser, 1997), and in the other there appeared to be a beneficial response to chromium (Khajerern and Khajerern, 1997). It is difficult to evaluate this information realistically without full data, but it is clear that Table 2 overstates the response to supplemental dietary chromium. It is somewhat troubling that the early data showed large and consistent responses to chromium but that the more recent data tend not to show effects.

The second observation from the table and from the previous paragraph is that the response is variable. In several experiments chromium supplementation appeared to produce no effects. We have made little progress in identifying factors that make a response to chromium more or less likely, as detailed below. However, variation in response to dietary supplementation of a nutrient is not surprising. There should be a response if the basal diet is limiting in the nutrient, but no response if the basal diet is adequate. We know very little about the background chromium levels in pig feeds or ingredients or about the bioavailability of this chromium. The levels of bioavailable chromium may vary considerably, and this variation may explain much of the variation in response to added chromium.

There are no clear interactions of chromium with genotype (Page et al., 1992; Green et al., 1997; Mooney

and Cromwell, 1999). Two studies (Lindemann and Purser, 1997; Renteria and Cuaron, 1998) suggested the response was bigger in castrated males than in gilts. Evock-Clover et al. (1993) found no improvement in carcass leanness from chromium supplementation either with or without treatment of the pigs with porcine somatotropin.

One study (Boleman et al., 1995) suggested that chromium should be fed only during the finishing period (from 57 kg onward), but others (Page et al., 1993; Lindemann et al., 1995) found good responses from feeding chromium throughout the growing and finishing periods (from less than 22 kg onward). Renteria and Cuaron (1998) found that responses to chromium were larger at a heavier slaughter weight (105 vs 95 kg), when the chromium was introduced at 32 kg body weight. There are no clear interactions of supplemental chromium with dietary amino acid levels (Lindemann et al., 1995; Renteria and Cuaron, 1998; van de Ligt et al., 1999a) or dietary energy intake (van de Ligt et al., 1998; 1999b).

The data shown in Table 2 were all developed using chromium picolinate, so it is clear that this product is efficacious. It is not clear that all other chromium products will be efficacious, and we need a method for determining which forms are and which are not biologically effective. The most direct indicator of chromium efficacy is stimulation of glucose clearance from plasma during glucose tolerance tests or insulin sensitivity tests. Such effects have been shown from chromium picolinate (Amoikon et al., 1995; Matthews et al., 1997), chromium l-methionine (Kegley et al., 1999), chromium

Table 2. Effects of supplemental dietary chromium^a on carcass characteristics of pigs^b

Source/description	Backfat thickness, mm			Loin-eye area, cm ²			% Lean					
	Control	Chromium	Difference	P	Control	Chromium	Difference	P	Control	Chromium	Difference	P
Page et al., 1993												
Exp. 1	28.3	24.4	-3.9	NS	34.9	37.2	2.3	NS	52.9	54.3	1.4	NS
Exp. 2	31.5	26.3	-5.2	<0.01	34.0	39.9	5.9	<0.01	51.7	54.7	3.0	<0.01
Exp. 3	30.7	23.9	-6.8	<0.01	31.5	38.4	6.9	<0.01	52.3	55.7	3.4	<0.01
Page et al., 1992												
Genotype A	32.6	27.8	-4.8	<0.01	32.0	34.8	2.8	NS	50.4	52.6	2.2	NS
Genotype B	34.2	25.5	-8.7	<0.01	31.7	38.4	6.7	<0.01	49.6	54.1	4.5	<0.01
Lindemann et al., 1995												
Low lysine	33.6	27.8	-5.8	<0.05	29.0	33.6	4.6	<0.05	44.0	47.9	3.9	<0.05
High lysine	33.0	29.6	-3.4	<0.05	30.1	32.1	2.0	<0.05	44.7	46.8	2.1	<0.05
Mooney and Cromwell, 1995												
Cr fed 19–106 kg body weight	37.1	36.1	-1.0	NS	30.0	29.3	-0.7	NS	42.9	46.6	3.7	<0.05
Boleman et al., 1995												
Cr fed 19–106 kg body weight	30.3	32.6	2.3	NS	30.9	31.4	0.5	NS	45.4	45.2	-0.2	NS
Cr fed 57–106 kg body weight		27.6	-2.7	NS		33.2	2.3	NS		47.7	2.3	NS
Ward et al., 1995												
Control	30.0	29.0	-1.0	NS	29.6	29.9	0.3	NS	45.2	45.5	0.3	NS
Chromium	31.0	32.2	1.2	NS	29.3	31.3	2.0	<0.05	45.0	45.0	0.0	NS
Kornegay et al., 1997												
Control	34.8	34.0	-0.8	NS	28.8	30.6	1.8	NS	43.1	44.0	0.9	NS
Chromium	18.8	19.5	0.7	NS	41.5	41.9	0.4	NS	NA	NA	NA	NA
Mooney and Cromwell, 1997												
Control	28.9	29.2	0.3	NS	36.6	37.5	0.9	NS	NA	NA	NA	NA
Chromium												
Crow and Newcomb, 1997												
Control	27.9	27.1	-0.8	<0.01	41.2	41.2	0.0	NS	NA	NA	NA	NA
Chromium	31.4	29.0	-2.4	<0.01	36.0	37.5	1.5	NS	NA	NA	NA	NA
Renteria and Cuaron, 1998												
Exp. 1, females	25.8	26.3	0.5	NS	28.6	29.3	0.7	NS	NA	NA	NA	NA
Exp. 1, castrated males	26.7	29.2	2.5	NS	29.9	30.6	0.7	NS	NA	NA	NA	NA
Exp. 4, low protein, 95 kg	26.8	29.5	2.7	NS	30.0	28.9	-1.1	NS	NA	NA	NA	NA
Exp. 4, low protein + AA, 95 kg	32.2	30.9	-1.3	NS	29.0	32.9	3.9	<0.05	NA	NA	NA	NA
Exp. 4, high protein, 95 kg	32.0	29.3	-2.7	NS	26.6	29.4	2.8	<0.05	NA	NA	NA	NA
Exp. 4, low protein, 105 kg	31.9	29.0	-2.9	NS	27.5	37.1	9.6	<0.05	NA	NA	NA	NA
Exp. 4, low protein + AA, 105 kg												
Exp. 4, high protein, 105 kg												
Mooney and Cromwell, 1999												
Exp. 1, Genotype Y	31.2	31.5	0.3	NS	28.5	29.9	1.4	NS	44.8	44.9	0.1	NS
Exp. 1, Genotype Z	25.7	25.2	-0.5	NS	28.6	30.3	1.7	NS	46.8	47.5	0.7	NS
Exp. 2, no pST	27.2	29.7	2.5	NS	33.9	32.0	-1.9	NS	48.2	46.7	-1.5	NS
Exp. 2, pST	14.5	14.1	-0.4	NS	39.0	39.6	0.6	NS	55.8	55.6	-0.2	NS
Mean	29.5	28.0	-1.6	—	31.9	34.0	2.2	—	47.7	49.1	1.6	—

^a200 ppb Cr from chromium picolinate.^bMeasurements and calculations were performed differently in different experiments. The 10th-rib backfat measurement was used if available.

propionate (Matthews et al., 1997), and chromium yeast (Guan et al., 1997). Unpublished data from the University of Minnesota (Zollitsch-Stelzl et al., personal communication) found this response to a chromium nicotinate product, but in another laboratory a different chromium nicotinate product had the opposite effects (Johnston et al., 1999). With present information, it should be expected that the compounds that enhance glucose clearance will improve leanness when fed to finishing pigs. However, different forms of chromium are handled differently by the body, so it should not be assumed that all other chromium sources will be efficacious.

The efficacy of specific chromium sources can be evaluated not only by the physiological responses described above, but also by empirical measurements of leanness. Further evidence for the efficacy of chromium l-methionine is found in the data of Khajerern and Khajerern (1997), which suggest an increase in leanness in pigs fed this form of chromium. O'Quinn et al. (1999b) found no response to dietary supplementation of a low dose (50 ppb) of chromium in the nicotinate form. Other studies have shown no improvements in leanness from dietary additions of chromium as the chloride, the acetate, the oxalate, the nicotinate, or chromium yeast, but these studies also found no response to the picolinate (Ward et al., 1995; Wenk et al., 1995; O'Quinn et al., 1998b).

As noted later in this paper, chromium seems to have no other effects on pork quality.

Conjugated Linoleic Acid (CLA)

Linoleic acid is a polyunsaturated fatty acid containing 18 carbon atoms and two *cis* double bonds at carbon atoms 9 and 11. Conjugated linoleic acid is similar except that the double bonds are in unexpected places, and some of them are in the *trans* form (rather than *cis*). It is a series of positional and geometric isomers of linoleic acid, and these isomers have several physiological effects. One is that when added to the diet they make animals leaner, perhaps because they reduce the rate of lipogenesis (Heckart et al., 1999). The specific isomer composition of CLA seems to be important. There is variation in isomer composition among CLA products, but the concentration of desired isomers and the uniformity of composition may improve with advances in production technology. Most CLA products are specially produced, but modified tall oil is a by-product material that contains CLA. Studies with modified tall oil are included in the following summary.

We have not tabulated the effects of dietary CLA on carcass leanness for two reasons. First, the variation in experimental treatments, specifically dose and duration of CLA feeding, would make such a table unwieldy. Second, several of the studies summarized here have been reported only in abstracts that contain few data.

Pettigrew (1999) reviewed data from seven experiments evaluating the effect of CLA supplementation of

the diet on measures of carcass leanness, and he reported that CLA reduced backfat thickness in all seven of the experiments and increased percentage lean in the carcass in all six of the experiments that reported that value (one did not). Some of these responses were not statistically significant, and indeed some were near zero, whereas others were both large and significant. Perhaps some of the variation in response may be due to variation in isomer composition of the CLA products used in the experiments. However, the reviewed data were remarkably consistent in direction. There are now more data sets available that were not included in that summary (O'Quinn et al., 1998a; Eggert et al., 1999a,b; O'Quinn et al., 1999a,b; Woodworth et al., 1999), and they continue to show beneficial responses to dietary CLA. Among the added studies, CLA reduced backfat thickness in four of five instances and increased percentage lean in two of four. If modified tall oil is excluded from the summary, there is only one failure of CLA to move these measures in the desired direction (percentage lean in the data of Eggert et al., 1999b). We can better estimate the size of the response after more of the actual data become available in full journal articles.

In an unusual departure, CLA seems to have opposite effects on lean:fat ratios in the muscle from those in the entire carcass. Addition of CLA to the diet increased marbling scores in five experiments (Eggert et al., 1999a,b; Larsen et al., 1999; Sparks et al., 1999b; Wiegand et al., 1999), and this effect was confirmed by an increase in ether extractable fat content in loins from CLA-fed pigs (Wiegand et al., 1999). Only two other experiments (Eggert et al., 1998; Thiel-Cooper et al., 1999) failed to find this increase in marbling. Addition of CLA to the diet may be the only nutritional intervention that both reduces subcutaneous fat and increases intramuscular fat, both desired responses. Dietary supplementation with CLA also improves belly firmness and may improve pork color, as detailed later in this paper.

Betaine

Betaine (trimethyl glycine) occurs naturally in many tissues, where it functions in several metabolic processes as a methyl donor. It has been suggested that adding betaine to the diet of finishing pigs increases carcass leanness (Cadogen et al., 1993). However, reviews of the pertinent literature (Shurson, 1995; Matthews et al., 1998) show that the response is erratic and unreliable. Recent reports continue the trend; one report shows dietary betaine increases leanness (Cromwell et al., 1999), two show no effect on leanness (Matthews et al., 1998; Øverland et al., 1999), and one shows an improvement in only one measure (shoulder weight) and no response in several other measures of leanness (Kitt et al., 1999). The variation in response does not appear to be due to dietary levels of energy or amino acids (Matthews et al., 1998). It seems unlikely that

betaine alters other aspects of pork quality, as described later in this paper.

Trimethylamine Oxide

Trimethylamine oxide occurs widely in nature and shares with betaine its function as a methyl donor. Recent data suggest that its inclusion in the diet at a low level (0.2%) increases the lean content of the carcass (60.6 vs 59.2%; $P < 0.10$), with no effect at higher levels (Øverland et al., 1999). Information on the effect of trimethylamine oxide on other aspects of pork quality can be found later in this review.

Carnitine

Carnitine is a vital component of the animal's system for using fat as a fuel. If there were inadequate carnitine in the tissues, perhaps fat would be deposited and the body would switch to glucose or amino acids as fuels. An early study (Smith et al., 1994) suggested that addition of carnitine to the diet reduced backfat thickness and increased the lean content of the carcass. However, more recently addition of carnitine to the diet failed to reduce backfat thickness or increase loin-eye area (O'Quinn et al., 1999a). This observation is supported by carnitine's failure to alter nitrogen or energy balance (Frank et al., 1999). The current evidence does not show effects of dietary carnitine on other aspects of pork quality, as detailed later in this paper.

Creatine

Creatine occurs naturally in all muscle cells, where it is intimately involved in energy metabolism. An experiment was conducted to determine whether addition of creatine to the diet would alter muscle pH, and the results are described below (Berg et al., 1999). Creatine supplementation increased the chemically-determined fat content of the loin.

Nutritional Factors Associated with Fat Metabolism

In this review, fat quality relates to fatty acid composition (firmness) and oxidative state. The color (whiteness) of fat is important in some countries but will not be considered here.

Factors Affecting Leanness

The fatty acids in the pig's carcass come from two sources: some are synthesized by the pig, and others are consumed from the diet and deposited unchanged in the tissues. The fatty acids synthesized by the pig are mostly saturated and monounsaturated; those from the diet are more often polyunsaturated. Pigs that are genetically leaner usually synthesize lesser amounts of fatty acids in the tissues. Similarly, restriction of energy intake reduces the amount of energy available to the

Table 3. Effect of leanness on fat composition^a

Component	P ₂ fat thickness, mm ^b			
	8	12	16	<i>P</i>
% of Fresh weight				
Water	22.4	17.1	14.1	<0.001
Lipid	69.2	77.0	81.6	<0.001
Collagen	4.5	3.0	2.0	<0.001
% of Total fatty acids, by weight				
14:0 ^c	1.5	1.5	1.5	NS
16:0	24.5	25.4	25.9	<0.001
16:1	2.8	2.7	2.7	NS
18:0	13.1	13.8	13.9	<0.001
18:1	40.3	41.8	43.1	<0.001
18:2	14.9	12.4	10.6	<0.001
18:3	1.1	0.9	0.8	<0.001

^aWood et al., 1989.

^bBackfat thickness measured 60 mm lateral to the midline at the level of the 10th rib.

^cNumber of carbons:number of double bonds.

pig and therefore reduces the amounts of fatty acids synthesized. Either change shifts the source of deposited fats to a lower proportion from endogenous synthesis and a higher proportion from the diet. The result is that leaner pigs usually have more unsaturated (softer) fat, whether they are leaner because of genetics, sex, or energy intake. This relationship is shown in Table 3, most obviously in the case of linoleic acid (18:2). A similar relationship was shown by Scott et al. (1981).

Note also that the water content of adipose tissue is higher, and the lipid content lower, in leaner animals (Table 3). As the animal fattens, it accumulates triglycerides (fat) into existing adipocytes (fat cells). Therefore, the adipose tissue of fatter animals has a higher proportion of stored fat and a lower proportion of other cellular components, including water. The higher content of triglycerides in the adipocytes also contributes to the greater firmness of the fat.

Dietary Fat Composition and Level

As noted above, body fat comes from a combination of fats produced endogenously and those from the diet. Therefore, the body fat composition reflects the composition of the dietary fat to some extent. Fatty acids absorbed from the diet, especially polyunsaturated ones, specifically inhibit endogenous synthesis of fatty acids, inflating the effect of dietary fat composition on body fat composition. Therefore, it is possible to manipulate the composition of body fat quite dramatically by selection of dietary fats. On the other hand, this relationship between dietary and body fats may restrict the types and amounts of fats we can choose to use in the diet, in order to keep the body fat composition within acceptable limits. A voluminous literature, not reviewed in depth here, documents this relationship.

Iodine value (*IV*) is a useful measure of the unsaturation of fats, higher *IV* indicating more unsaturation. Technically it is the amount of iodine (in grams) that

can be bound by 100 grams of fat, and it works because iodine is bound only at the double bonds (unsaturation points).

Most dietary fats are more unsaturated (higher IV) than the triglycerides the pig synthesizes endogenously, so addition of fat to the diet usually makes carcass fat softer. Raising the amount of fat added to the diet increases the IV (softness) of the carcass fat, because a higher proportion of the deposited fat comes from the diet. The composition and dietary level of fat can be combined into a single expression, the iodine value product (IVP) (Madsen et al., 1992). The IVP is the IV of fat multiplied by its concentration in the diet, and the product divided by 10. Madsen et al. (1992) reported a close relationship in Danish pigs between the IV of the pig's carcass fat and the IVP of the diet, as follows: $IV(\text{carcass}) = 47.1 + 0.14 \times IVP(\text{diet})$; $R^2 = 0.86$. A maximum carcass fat IV of 70 has been proposed for Danish conditions (Barton-Gade, 1987).

This issue is of increasing importance because of the progressive leanness of pigs (less endogenous fat synthesis), and because of the trend to higher-energy (higher-fat) diets. The adoption of high-oil corn often increases both the level of fat in the diet and the IV of that fat.

There also can be concerns about specific dietary fats that can become part of pork fat. For example, a high level of fish products in the diet can cause a fishy taste in pork.

There also are detrimental effects of *n*-3 fatty acids such as linolenic acid. Some observers believe that consumption of large amounts of -3 fatty acids may protect against coronary heart disease, so there is interest in increasing the concentration of these fatty acids in pork and other foods. However, accumulation of these specific fatty acids in pork results in development of off-flavors, probably because they are unusually susceptible to oxidation. The data summarized in Table 4 show this relationship clearly and raise concern about the use of canola oil, a rich source of -3 fatty acids, in swine diets.

Conjugated Linoleic Acid (CLA)

It was noted above that CLA in the diet makes pigs leaner, and it has other effects also. It shifts the composition of body fat from unsaturated to saturated (Eggert et al., 1998; Waylan et al., 1999). One important result is a dramatic increase in the firmness of the belly, as shown by both objective (Table 5) and subjective (Eggert et al., 1999a; O'Quinn et al., 1999a; Woodworth et al., 1999) measures.

Three experiments found that dietary CLA increased firmness of the loin (Eggert et al., 1999a,b; Sparks et al., 1999b), but two others (Eggert et al., 1998; Thiel-Cooper et al., 1999) failed to find this effect. Larsen et al. (1999) found no effect of dietary CLA on firmness of the ham.

The increase in saturation of the fat is not large enough to produce consistent improvements in oxidative stability. Pork from CLA-supplemented pigs had lower thiobarbituric acid reactive substances (TBARS) values after 1 d of storage, but not later (Wiegand et al., 1999). Waylan et al. (1999) found no effect of dietary CLA on TBARS of pork. It seems that adding CLA to the diet can alleviate the problem of soft bellies, and thus make processing more efficient. Perhaps it can be used to counteract the detrimental effect on belly firmness of dietary unsaturated fats (Eggert et al., 1999b). The industry can adopt this technology easily if producers are adequately compensated for the cost of the CLA. The effects of dietary CLA on leanness and on other aspects of pork quality are detailed elsewhere in this review.

Copper

We often add a high level of copper to the pig's diet as a growth promotant. This inexpensive practice is effective in promoting growth, but it raises concerns in two areas. First, when a high level of copper is added to the diet of pigs throughout growth to market weight, it can result in a buildup of copper in the soils on which the manure is spread. Second, it makes the carcass fat softer (more unsaturated) by increasing the activities of fatty acyl desaturase enzyme systems (Ho and Elliot, 1974) and perhaps by changing the positions of specific fatty acids in the triglyceride molecules (Pethick et al., 1997).

The data on carcass fat composition are summarized in Table 6. Of 20 comparisons of the percentage of unsaturated fatty acids in backfat gleaned from seven publications, all showed that high dietary copper increased the unsaturation of backfat. Twelve of the responses were statistically significant ($P < 0.05$). The overall mean response (unweighted) was a shift of 4.9% of the fat from saturated to unsaturated fatty acids. This response is supported by measurements of the melting point of the backfat. Of 10 comparisons from five publications, all showed that high dietary copper reduced the melting point (eight of the differences were statistically significant [$P < 0.05$]). The overall mean response was a reduction in the melting point of 6.2°C. This reduction of melting point is consistent with a shift to more unsaturated fat.

The consistency of this response is impressive. The magnitude of response appears to be affected somewhat by the protein source in the diet (Elliot and Bowland, 1970). There are no big differences in response between barrows and gilts (Ho and Elliot, 1974). The response occurs soon after introduction of the high copper levels into the diet (Amer and Elliot, 1973). The response in outer subcutaneous adipose tissue is smaller than the response in perirenal adipose (Moore et al., 1969; Thompson et al., 1973).

Astrup and Matre (1987) reported that a physical measure of fat firmness appeared to be reduced by high

Table 4. Effect of *n*-3 fatty acids on acceptability of pork

Source/measurement	Dietary fat source ^a				
	Control	Animal fat	Safflower	Sunflower	Canola
Miller et al., 1990					
Longissimus fat composition ^b					
Oleic (18:1)	47.4 ^x	44.6 ^v	48.8 ^y	51.7 ^z	45.9 ^w
Linoleic (18:2)	6.7 ^w	11.5 ^z	10.4 ^y	8.4 ^x	12.3 ^z
Linolenic (18:3)	1.5 ^y	1.6 ^y	1.4 ^y	1.5 ^y	3.0 ^z
Taste panel data					
Flavor quality ^c	5.2 ^y	5.4 ^y	5.3 ^y	5.4 ^y	4.7 ^z
Overall palatability ^c	5.1 ^{yz}	5.3 ^y	5.2 ^{yz}	5.1 ^{yz}	4.6 ^z
Off-flavor percentage ^d	19.0	18.8	19.6	17.5	28.6
Shackelford et al., 1990					
Bacon fat composition ^b					
Oleic (18:1)	45.4 ^w	46 ^w	55.2 ^y	60.8 ^z	50.3 ^x
Linoleic (18:2)	8.1 ^v	14.3 ^y	12.8 ^x	9.2 ^w	16.4 ^z
Linolenic (18:3)	0.0 ^y	0.3 ^y	0.0 ^y	0.0 ^y	4.8 ^z
Taste panel data					
Flavor ^c	5.6 ^x	4.6 ^y	5.2 ^{xy}	5.1 ^{xy}	2.9 ^z
Overall palatability ^c	5.5 ^x	4.5 ^y	4.7 ^y	4.8 ^y	2.8 ^z
Off-flavor percentage ^d	5.7 ^x	23.4 ^y	11.5 ^{xy}	10.6 ^{xy}	65.1 ^z

^aIncluded at the level of 10% of the diet.

^bPercentage of total fatty acids.

^cScale from 1 to 8: 8 is extremely flavorful or palatable and 1 is extremely unflavorful or unpalatable.

^dPercentage of panelists detecting off-flavor.

^{v,w,x,y,z}Values without a common superscript differ ($P < 0.05$).

dietary copper, providing further support to the effects on unsaturation and melting point. Myer et al. (1992) failed to find an effect on firmness, but the response in unsaturation in this experiment was the smallest of the 20 comparisons.

High dietary copper may reduce the oxidative stability of carcass fat in two ways. First, the more unsaturated fat caused by copper is more susceptible to oxidation. Second, copper may be a prooxidant in tissues (Morrissey et al., 1998). Data from Amer and Elliot (1973) confirm a detrimental effect of dietary copper on oxidative stability of fat (Table 7), although Astrup and Matre (1987) were unable to demonstrate such an effect using the active oxygen method (2.7 h to rancidity for controls, 2.5 for copper-supplemented pigs). Similarly, Jensen et al. (1998) found no effect of 175 ppm supplemental dietary copper on TBARS. Feeding 175 ppm copper to pigs actually reduced the rate of iron-induced lipid oxidation in homogenates of psoas major (Laurid-

sen et al., 1999b) and liver (Lauridsen et al., 1999a). Overall, there is no clear indication that feeding a high level of copper to pigs reduces the oxidative stability of the pork.

A trained taste panel was able to distinguish between pork from pigs fed a high level of copper and pork from control pigs, but there was no clear preference for either (Ho et al., 1976). Pork from the pigs fed copper had a more pronounced flavor, perhaps because of oxidation of the fat. Overall, it is clear that the use of a high level of copper in the diet as a growth promotant makes the carcass fat softer and more unsaturated.

Vitamin E

Several tocopherols and tocotrienols have vitamin E activity, alpha tocopherol being the most active and the most commonly used as a dietary supplement (usually as alpha tocopheryl acetate). Vitamin E has a special role in the tissues as a "chain-breaking" antioxidant, an antioxidant that stops the self-perpetuating production of lipid peroxides (Benzie, 1996). It has been proposed that a high level of vitamin E in the diet might reduce the damaging oxidation of meat, and this area has been reviewed extensively (Buckley et al., 1995; Morrissey et al., 1998).

Several studies have been conducted to determine whether high dietary vitamin E actually improves the oxidative stability of pork fat, and the results are summarized in Table 8. The table shows a very consistent reduction in TBARS (fat oxidation) in pork from pigs fed a high level of vitamin E. Not all data from these papers are included in the table because of the volume

Table 5. Effects of conjugated linoleic acid (CLA) in the diet on belly firmness^a

Source	% CLA ^b					
	0	0.12	0.25	0.5	0.75	1
Thiel et al., 1998	52 ^c	55.3 ^c	56.3 ^c	67.4 ^{cd}	—	78.8 ^d
Fulghum et al., 1999	23.2 ^e	—	—	—	42.4 ^f	—

^aMeasured as belly span, distance in cm between the ends of a belly suspended over a rod, lean side up; a higher value is more desirable.

^bContains 60 to 65% actual CLA isomers.

^{c,d}Values without a common superscript differ ($P < 0.05$).

^{e,f}Values without a common superscript differ ($P < 0.0001$).

Table 6. Effects of 250 ppm dietary copper on the fatty acid composition and melting point of carcass fat

Source/description	Unsaturated fatty acids, %				Melting point, °C			
	Control	Cu	Difference	<i>P</i>	Control	Cu	Difference	<i>P</i>
Moore et al., 1969								
Exp. 1	56.3	58.9	2.6	NS	—	—	—	—
Exp. 2	54.9	58.6	3.7	NS	—	—	—	—
Exp. 3	58.6	61.0	2.4	NS	—	—	—	—
Exp. 4	52.9	54.9	2.0	NS	45	33	-12.0	NS
Exp. 5	52.2	56.1	3.9	NS	43	34	-9.0	NS
Elliot & Bowland, 1970								
Fish meal	63.7	70.9	7.2	<0.01	—	—	—	—
Meat meal	62.5	70.3	7.8	<0.01	—	—	—	—
Soybean meal	60.4	63.6	3.2	<0.05	—	—	—	—
Rapeseed meal	64.4	65.5	1.1	NS	—	—	—	—
Amer and Elliot, 1973								
Exp. 1								
Live weight at slaughter ^a								
23 kg	61.6	67.6	6.1	<0.01	26.8	23.5	-3.3	<0.01
46 kg	64.0	70.7	6.7	<0.01	25.9	20.4	-5.5	<0.01
69 kg	60.7	72.2	11.5	<0.01	28.0	19.0	-9.0	<0.01
92 kg	61.7	67.6	5.9	<0.01	27.8	24.6	-3.2	<0.01
Exp. 2 ^b	62.6	69.3	6.7	<0.05	27.9	21.1	-6.8	<0.05
Thompson et al., 1973								
Outer subcutaneous fat	60.7	62.1	1.4	NS	—	—	—	—
Perirenal fat	50.2	53.3	3.1	NS	—	—	—	—
Ho and Elliot, 1974								
Barrows	61.7	68.4	6.7	<0.01	—	—	—	—
Gilts	63.1	68.8	5.7	<0.01	—	—	—	—
Average	—	—	—	<0.01	25.1	20.5	-4.6	<0.01
Ho et al., 1976	60.8	70.2	9.4	<0.01	24.1	19.5	-4.6	<0.01
Astrup and Matre, 1987	—	—	—	—	39.2	35.4	-3.8	<0.05
Myer et al., 1992	65.5	66.1	0.6	NS	—	—	—	—
Overall mean	—	—	4.9	—	—	—	-6.2	—

^aInitial weight 13.5 kg.^b200 ppm Cu.

of data and the relative consistency of response. In addition to the data shown here, Jensen et al. (1997, 1998) showed a reduction in TBARS by inclusion of a high level of vitamin E in the diet. These data were excluded

from the table because they were expressed in different units. Corino et al. (1999) also found that high dietary vitamin E reduced TBARS, but the basal dietary level of vitamin E was too high to meet the arbitrary standard

Table 7. Effects of a high level of dietary copper on oxidative stability of fat^a

Description	Oxygen uptake, mL/g fat				TBARS ^b , mg malonaldehyde/kg fat			
	Control	Cu	Difference	<i>P</i> ^c	Control	Cu	Difference	<i>P</i> ^c
Exp. 1, 250 ppm Cu								
Live weight at slaughter ^d								
23 kg	0.061	0.108	0.047	<0.01	12.25	21.40	9.15	<0.01
46 kg	0.083	0.219	0.136	<0.01	16.60	43.95	27.35	<0.01
69 kg	0.054	0.252	0.198	<0.01	10.85	51.20	40.35	<0.01
92 kg	0.060	0.140	0.080	<0.01	12.10	25.18	13.08	<0.01
Exp. 2, 200 ppm Cu								
Added vitamin E								
0 IU/kg	0.050	0.136	0.086	<0.05	8.69	13.08	4.39	<0.05
22 IU/kg	—	0.108	—	—	—	11.56	—	—
44 IU/kg	—	0.121	—	—	—	11.21	—	—
88 IU/kg	—	0.075	—	—	—	8.76	—	—
Mean	—	—	0.109	—	—	—	18.86	—

^aAmer and Elliot, 1973.^bThiobarbituric acid reactive substances.^cSignificance of difference between control and Cu treatments.^dInitial weight 13.5 kg.

Table 8. Effects of a high level of dietary vitamin E on TBARS^a of fat

Source, description, and storage days ^c	Level of vitamin E supplementation, mg/kg diet ^b						
	Basal	100			200		
	TBARS	TBARS	Diff. ^d	P ^e	TBARS	Diff. ^d	P ^e
Asghar et al., 1991 (fed ~98 d)							
Frozen pork chops							
0 d	0.28	0.27	-0.01	NS	0.27	-0.01	NS
3 d	1.54	0.56	-0.98	<0.05	0.35	-1.19	<0.05
6 d	2.96	0.94	-2.02	<0.05	0.58	-2.38	<0.05
10 d	5.17	2.96	-2.21	NS	1.33	-3.84	<0.05
Ground pork							
1 d	1.34	0.36	-0.98	<0.05	0.22	-1.12	<0.05
4 d	3.49	1.13	-2.36	<0.05	0.41	-3.08	<0.05
8 d	5.38	3.27	-2.11	<0.05	1.59	-3.79	<0.05
Monahan et al., 1994b^f (fed from 30 to 98 kg BW)							
Refrigerated pork chops							
0 d	0.20	—	—	—	0.17	-0.03	<0.05
2 d	0.37	—	—	—	0.19	-0.18	<0.05
4 d	0.38	—	—	—	0.20	-0.18	<0.05
6 d	0.51	—	—	—	0.21	-0.30	<0.05
8 d	0.78	—	—	—	0.13	-0.65	<0.05
Monahan et al., 1994a^f (fed from 30 to 98 kg BW)							
Fresh pork chops							
Diet 3% fresh corn oil							
8 d	0.57	0.16	-0.41	<0.01	0.12	-0.45	<0.01
Diet 3% oxidized corn oil							
8 d	0.60	0.28	-0.32	<0.01	0.12	-0.48	<0.01
Diet 3% fresh corn oil							
4 mo frozen chops							
8 d	2.80	0.35	-2.45	<0.01	0.30	-2.50	<0.01
Diet 3% oxidized corn oil							
8 d	2.98	1.20	-1.78	<0.01	0.75	-2.23	<0.01
Lanari et al., 1995^f (fed 105 d)							
Longissimus lumborum							
Display in air							
0 d	0.19	—	—	—	0.07	-0.12	<0.01
2 d	0.48	—	—	—	0.11	-0.37	<0.01
4 d	0.70	—	—	—	0.11	-0.59	<0.01
6 d	0.72	—	—	—	0.18	-0.54	<0.01
8 d	0.74	—	—	—	0.21	-0.53	<0.01
10 d	0.74	—	—	—	0.42	-0.32	<0.01
Cannon et al., 1995^f (fed 84 d)							
Precooked chops							
0 d	0.42	0.31	-0.11	<0.05	—	—	—
7 d	0.63	0.54	-0.09	NS	—	—	—
14 d	0.88	0.65	-0.23	<0.05	—	—	—
28 d	0.56	0.44	-0.12	<0.05	—	—	—
56 d	0.76	0.47	-0.29	<0.05	—	—	—
Precooked roasts							
0 d	0.78	0.38	-0.40	<0.05	—	—	—
7 d	0.78	0.50	-0.28	<0.05	—	—	—
14 d	1.13	0.80	-0.33	<0.05	—	—	—
28 d	0.50	0.38	-0.12	NS	—	—	—
56 d	0.52	0.44	-0.08	NS	—	—	—

Continued

Table 8. *Continued.* Effects of a high level of dietary vitamin E on TBARS^a of fat

Source, description, and storage days ^c	Level of vitamin E supplementation, mg/kg diet ^b						
	Basal	100			200		
	TBARS	TBARS	Diff. ^d	<i>P</i> ^e	TBARS	Diff. ^d	<i>P</i> ^e
Cannon et al., 1996 (fed 84 d)							
Fresh longissimus chops							
0 + 1 d ^g	0.30	0.32	0.02	NS	—	—	—
0 + 3 d	0.51	0.30	-0.21	<0.05	—	—	—
0 + 5 d	0.74	0.41	-0.33	<0.05	—	—	—
14 + 1 d	0.50	0.50	0.00	NS	—	—	—
14 + 3 d	0.72	0.52	-0.20	<0.05	—	—	—
14 + 5 d	0.75	0.49	-0.26	<0.05	—	—	—
28 + 1 d	0.38	0.36	-0.02	NS	—	—	—
28 + 3 d	0.59	0.43	-0.16	<0.05	—	—	—
28 + 5 d	0.92	0.60	-0.32	<0.05	—	—	—
56 + 1 d	0.40	0.37	-0.03	NS	—	—	—
56 + 3 d	0.72	0.53	-0.19	<0.05	—	—	—
56 + 5 d	0.93	0.60	-0.33	<0.05	—	—	—
Houben et al., 1998^f (fed 72 d)							
Minced pork at 7°C, foil package storage							
0 d	0.02	—	—	—	0.07	0.05	NS
3 d	0.06	—	—	—	0.08	0.02	NS
9 d	0.13	—	—	—	0.05	-0.08	NS
11 d	0.24	—	—	—	0.17	-0.07	NS
Minced pork at 7°C, gas package storage							
0 d	0.05	—	—	—	0.07	0.02	NS
3 d	0.18	—	—	—	0.07	-0.11	<0.05
9 d	0.60	—	—	—	0.09	-0.51	<0.05
11 d	1.20	—	—	—	0.10	-1.10	<0.05
Hoving-Bolink et al., 1998 (fed 84 d)							
Longissimus lumborum (fresh)							
0 d	0.07	—	—	—	0.06	-0.01	NS
3 d	0.29	—	—	—	0.10	-0.19	<0.05
6 d	0.53	—	—	—	0.19	-0.34	<0.05
Longissimus lumborum (frozen)							
0 d	0.17	—	—	—	0.10	-0.07	<0.05
3 d	0.62	—	—	—	0.15	-0.47	<0.05
6 d	1.07	—	—	—	0.24	-0.83	<0.05
Zanardi et al., 1998 (6 mo)							
Fresh chops							
35 ppm dietary Cu							
0 d	0.10	0.11	0.01	—	0.12	0.02	NS
175 ppm supplemental Cu							
0 d	0.11	0.09	-0.02	—	0.11	0.00	NS
Cooked chops							
35 ppm dietary Cu							
0 d	0.06	0.06	0.00	—	0.04	-0.02	NS
175 ppm supplemental Cu							
0 d	0.06	0.06	0.00	—	0.04	-0.02	NS
Isabel et al., 1999^f (fed 42 d)							
Dry-cured Iberian hams							
3 d	0.85	—	—	—	0.70	-0.15	—
6 d	1.20	—	—	—	0.80	-0.40	—
9 d	1.65	—	—	—	1.10	-0.55	—
Overall storage days							0.015
Mean				-0.53			-0.69

^aThiobarbituric acid reactive substances, mg malonaldehyde/kg tissue.

^bApproximate amount of vitamin E added to a basal diet containing < 12 IU supplemental vitamin E; duration varied.

^cPrevious treatment and storage conditions varied.

^dDifference between supplemented and control.

^eStatistical significance (*P*-value) of difference between supplemented and control.

^fData taken from a graph.

^gDays stored under vacuum at 2°C + days in retail storage.

for incorporation into Table 8. From all of these data it is clear that a high level of vitamin E in the pig's diet improves the oxidative stability of pork.

The data in Table 8 provide little guidance regarding the appropriate duration of feeding high levels of vitamin E. All except two of the studies used apparent durations within the relatively narrow range of 72 to 105 d, and all found responses to the dietary treatments. Isabel et al. (1999) applied the dietary treatments for only 42 d and found a treatment response, whereas Zanardi et al. (1998) fed the treatments for about 6 mo and found no treatment effects.

The biggest effects of supplemental vitamin E on TBARS (oxidation) occurred when the pork had high TBARS values (when it was oxidized), whether that oxidation was caused by long storage time, storage conditions, the pigs' diet, or other factors. With relatively few exceptions, a high level of dietary vitamin E reduced the TBARS value if that value was above about 0.15 to 0.30 in the control animals but showed no effect when the control level was lower.

In summary, a high dietary level of vitamin E improves the oxidative stability of pork. The implications are that it improves the quality of pork for the consumer and that it increases the shelf life of pork. There is one significant obstacle to putting this knowledge into practice. The levels of vitamin E that produce the desired response are expensive. The investment will be made only when the investor receives an appropriate financial return for adopting the practice.

There is a further important question of whether the observed improvement in oxidative stability that results from a high dietary vitamin E level translates into improvements in other quality measures, such as water-binding capacity, color and pH. The results available to date are inconclusive, as described below.

There are two reasons why improvements in the oxidative stability of the tissue may improve the quality of pork lean. First, the loss of redness of meat during storage is caused by oxidation of the bright pinkish-red oxymyoglobin to the brown metmyoglobin. Improved oxidative stability may reduce the rate of that oxidation and preserve the red color for a longer time during storage. Note that this effect on meat color is entirely distinct from effects on the lightness of muscle related to carbohydrate metabolism and pH. Second, reducing the oxidation of membrane lipids may improve the integrity of those membranes, and thus reduce fluid leakage. That would translate into reduced drip loss (increased water-binding capacity). The following paragraphs review the empirical data to determine whether these suggested improvements in quality actually occur when a high level of vitamin E is added to the diet.

In terms of muscle color, neither lightness (L^*) nor yellowness (b^*) is influenced by high dietary levels of vitamin E (Asghar et al., 1991; Houben et al., 1998; Zanardi et al., 1998), so our summary focuses on redness (a^*). This is consistent with the logic described in the previous paragraph.

Asghar et al. (1991), Monahan et al. (1994a), and Lanari et al. (1995) found that adding high levels of vitamin E to the diet increased the intensity of redness in the pork (Table 9). Two of these papers also reported that dietary vitamin E reduced drip loss. Two reports from The Netherlands (Houben et al., 1998; Hoving-Bolink et al., 1998) show only sporadic a^* responses to dietary vitamin E level in either direction. The other three papers cited in Table 9 plus Cannon et al. (1996) show no effect of dietary vitamin E level on redness of meat. Monahan et al. (1994a) and Isabel et al. (1999) found that vitamin E in the diet slowed the loss of redness during storage, but Houben et al. (1998) found the opposite. Zanardi et al. (1998) showed slower development of the brown color of metmyoglobin in pork from pigs fed a high level of vitamin E.

Overall, these data suggest that a high dietary vitamin E level increases redness of pork in some situations but not in others. It is not obvious from Table 9 that the response in meat color to dietary vitamin E level is related to either storage time or the basal level of redness (a^*). It appears that the vitamin E level in the basal diet does not explain the variation in responses, nor does the dose or duration (data not shown) of supplemental vitamin E. We have not identified factors that determine whether dietary vitamin E level will influence meat color.

Three papers (Asghar et al., 1991; Monahan et al., 1994b; Cheah et al., 1995) reported clear reductions in drip loss when high levels of vitamin E were added to the diet (Table 10). A fourth (Cannon et al., 1996) found a nonsignificant trend toward improvement, and a fifth (Hoving-Bolink et al., 1998) reported a clear absence of response. An additional paper (Jensen et al., 1997) reported no response when the basal vitamin E level was very high.

Cheah et al. (1995) found a response in the predominantly white longissimus muscle, but not in the predominantly red masseter muscle. This difference does not explain the lack of response in the two studies mentioned above, because they used the longissimus. Dietary vitamin E improved oxidative stability in all of the experiments (except that of Cheah et al. [1995], who did not measure it), and the magnitude of that response was not clearly bigger in the experiments that showed an effect on drip loss. It appears the response does not depend on the level of vitamin E in the basal diet, because Asghar et al. (1991) and Monahan et al. (1994b) used higher basal levels of vitamin E than did the others and they found responses to further additions. There is some indication that a response is more likely if the supplemental level is greater than 100 IU/kg. There is no indication from these data that the duration of feeding a high level of vitamin E determines whether it will reduce drip loss (data not shown). We interpret these results to suggest that adding a high vitamin E level to the diet will reduce drip loss in some situations, but perhaps not in all situations. Van Laack and Spencer (1999) suggested that the fatty acid composition of the

Table 9. Effects of a high level of dietary vitamin E on pork redness (Hunter a*)

Source, description, and storage days ^b	Level of vitamin E supplementation, mg/kg diet ^a						
	Basal	100			200		
	a*	a*	Diff. ^c	P ^d	a*	Diff. ^c	P ^d
Asghar et al., 1991							
Frozen pork chops							
0 d	10.7	11.6	0.9	NS	12.6	1.9	<0.05
3 d	10.1	11.1	1.0	NS	12.4	2.3	<0.05
6 d	7.0	9.3	2.3	<0.05	10.0	3.0	<0.05
10 d	7.0	7.9	0.9	NS	8.7	1.7	NS
Monahan et al., 1994a^e							
Fresh pork chops							
Diet 3% fresh corn oil							
8 d	4.1	4.1	0.0	NS	5.9	1.8	<0.01
Diet 3% oxidized corn oil							
8 d	4.0	4.2	0.2	NS	4.5	0.5	NS
4 mo frozen chops							
Diet 3% fresh corn oil							
8 d	5.2	4.0	-1.2	NS	8.3	3.1	<0.01
Diet 3% oxidized corn oil							
8 d	4.1	6.5	2.4	<0.01	5.9	1.8	<0.01
Lanari et al., 1995^e							
Exp. 1							
Display in air							
0 d	7.0	—	—	—	8.7	1.7	—
2 d	6.0	—	—	—	7.8	1.8	—
4 d	5.0	—	—	—	7.2	2.2	—
6 d	2.9	—	—	—	6.3	3.4	—
8 d	1.9	—	—	—	5.2	3.3	—
10 d	1.3	—	—	—	4.8	3.5	—
Display in 80% O ₂ , 20% CO ₂							
0 d	7.5	—	—	—	8.3	0.8	—
2 d	6.8	—	—	—	7.5	0.7	—
4 d	5.0	—	—	—	7.5	2.5	—
6 d	3.2	—	—	—	5.7	2.5	—
8 d	1.8	—	—	—	4.1	2.3	—
10 d	1.2	—	—	—	3.2	2.0	—
Overall Exp. 1	—	—	—	—	—	—	<0.009
Exp. 2							
Illuminated display							
0 d	8.0	—	—	—	8.2	0.2	—
1 d	10.7	—	—	—	9.1	-1.6	—
2 d	7.0	—	—	—	7.2	0.2	—
4 d	6.1	—	—	—	7.0	0.9	—
6 d	4.0	—	—	—	6.3	2.3	—
8 d	3.1	—	—	—	5.4	2.3	—
10 d	2.0	—	—	—	4.3	2.3	—
Overall	—	—	—	—	—	—	<0.05
Dark display							
0 d	8.0	—	—	—	9.0	1.0	—
1 d	10.4	—	—	—	10.4	0.0	—
2 d	8.1	—	—	—	8.3	0.2	—
4 d	8.0	—	—	—	8.5	0.5	—
6 d	6.2	—	—	—	7.1	0.9	—
8 d	6.0	—	—	—	7.1	1.1	—
10 d	6.5	—	—	—	7.3	0.8	—
Overall	—	—	—	—	—	—	NS
Houben et al., 1998^f							
Minced pork at 7°C, foil package storage							
0 d	16.4	—	—	—	16.7	0.3	NS
3 d	13.5	—	—	—	11.7	-1.8	<0.05
6 d	12.6	—	—	—	10.8	-1.8	NS
9 d	12.7	—	—	—	10.9	-1.8	NS

Continued

Table 9. *Continued.* Effects of a high level of dietary vitamin E on pork redness (Hunter a*)

Source, description, and storage days ^b	Level of vitamin E supplementation, mg/kg diet ^a						
	Basal	100			200		
	a*	a*	Diff. ^c	P ^d	a*	Diff. ^c	P ^d
Minced pork at 7°C, gas package storage							
0 d	16.4	—	—	—	16.7	0.3	NS
3 d	14.8	—	—	—	14.2	-0.6	NS
Hoving-Bolink et al., 1998^e							
Longissimus lumborum (fresh)							
1 d	10.2	—	—	—	10.2	0.0	NS
2 d	10.2	—	—	—	10.4	0.2	NS
3 d	9.9	—	—	—	10.1	0.2	NS
4 d	9.3	—	—	—	9.6	0.3	NS
6 d	8.3	—	—	—	8.8	0.5	<0.05
Longissimus lumborum (frozen)							
1 d	9.2	—	—	—	9.3	0.1	NS
2 d	8.6	—	—	—	8.9	0.3	NS
3 d	8.1	—	—	—	8.4	0.3	NS
4 d	7.7	—	—	—	8.1	0.4	NS
5 d	7.4	—	—	—	8.0	0.6	NS
6 d	6.9	—	—	—	7.8	0.9	<0.05
Psoas major (fresh)							
1 d	14.1	—	—	—	13.8	-0.3	NS
2 d	14.4	—	—	—	14.3	-0.1	NS
3 d	14.0	—	—	—	14.0	0.0	NS
4 d	14.0	—	—	—	14.0	0.0	NS
6 d	10.2	—	—	—	10.4	0.2	NS
Zanardi et al., 1998							
35 ppm dietary Cu							
0 d	6.8	7.3	0.5	—	7.5	0.7	NS
175 ppm supplemental Cu							
0 d	8.2	7.1	-1.1	—	9.4	1.2	NS
Jensen et al., 1998^g							
No added Cu							
8 d	5.2	5.3	0.1	—	4.0	-1.2	NS
35 ppm supplemental Cu							
8 d	4.7	3.8	-0.9	—	4.8	0.1	NS
175 ppm supplemental Cu							
8 d	4.3	4.8	0.5	—	4.2	-0.1	NS
Isabel et al., 1999							
Cured hams (biceps femoris)							
0 d	14.9	—	—	—	14.6	-0.3	NS
1 d	13.1	—	—	—	12.9	-0.2	NS
2 d	12.1	—	—	—	12.4	0.3	NS
3 d	12.1	—	—	—	11.7	-0.4	NS
4 d	11.6	—	—	—	11.6	0.0	NS
Mean	—	—	0.4	—	—	0.8	—

^aApproximate amount of vitamin E added to a basal diet containing < 20 IU supplemental vitamin E.

^bPrevious treatment and storage conditions varied.

^cDifference between supplemented and control.

^dStatistical significance (*P*-value) of difference between supplemented and control.

^eData taken from a graph.

^fData for d 6 and 9 taken from a graph.

^gMinolta a* values.

membrane phospholipids may influence whether the oxidative state of the muscle alters water-binding capacity.

Supplemental dietary vitamin E did not affect the pH of meat measured either soon after slaughter or after storage in four studies (Cannon et al., 1996;

Houben et al., 1998; Hoving-Bolink et al., 1998; Zanardi et al., 1998); only one experiment (Lauridsen et al., 1999b) found higher pH (5.7 vs 5.5; *P* = 0.03) at 24 h after slaughter.

In summary, high dietary levels of vitamin E seem to reduce drip loss and improve color (increase redness)

Table 10. Effects of a high level of dietary vitamin E on pork drip loss^a

Source, description, and storage days ^c	Level of vitamin E supplementation, mg/kg diet ^b						
	Basal	100			200		
	Drip loss, %	Drip loss, %	Diff. ^d	<i>P</i> ^e	Drip loss, %	Diff. ^d	<i>P</i> ^e
Asghar et al., 1991							
Frozen pork chops							
3 d	19.0	16.2	-2.8	—	10.2	-8.8	<0.05
6 d	20.1	19.5	-0.6	—	12.2	-7.9	<0.05
10 d	21.3	21.2	-0.1	—	14.1	-7.2	<0.05
Monahan et al., 1994^b							
Refrigerated pork chops							
2 d	5.1	—	—	—	2.9	-2.2	<0.05
4 d	8.0	—	—	—	3.1	-4.9	<0.05
6 d	8.7	—	—	—	5.3	-3.4	<0.05
8 d	11.6	—	—	—	6.1	-5.5	<0.05
Cannon et al., 1996							
Fresh longissimus chops							
0 d	5.01	4.76	-0.25	—	—	—	—
14 d	3.81	3.30	-0.51	—	—	—	—
28 d	2.96	2.68	-0.28	—	—	—	—
56 d	2.35	2.40	0.05	—	—	—	—
Hoving-Bolink et al., 1998							
Longissimus lumborum (fresh)							
2 d	6.9	—	—	—	6.9	0.0	—
Longissimus lumborum (frozen)							
2 d	10.9	—	—	—	11.4	0.5	—
Psoas major (fresh)							
2 d	2.4	—	—	—	2.4	0.0	—
Cheah et al., 1995							
Exp. 1 ^g							
Longissimus thoracis							
Genotype NN ^h							
2 d	6.9	—	—	—	3.2	-3.7	<0.01
Genotype Nn ⁱ							
2 d	9.1	—	—	—	5.0	-4.1	<0.01
Masseter							
Genotype NN ^h							
2 d	1.1	—	—	—	1.8	0.7	—
Genotype Nn ⁱ							
2 d	1.0	—	—	—	1.0	0.0	—
Exp. 2 ^j							
2 d	8.5	—	—	—	5.0	-3.5	<0.05
Mean			-0.6			-3.3	

^aMethods for measuring drip loss varied among laboratories.

^bApproximate amount of vitamin E added to a basal diet containing < 20 IU supplemental vitamin E.

^cPrevious treatment and storage conditions varied.

^dDifference between supplemented and control.

^eStatistical significance (*P*-value) of difference between supplemented and control.

^fData taken from a graph.

^gVitamin E added to the diet at the level of 500 IU/kg.

^hRyanadine receptor genotype; halothane negative.

ⁱRyanadine receptor genotype; heterozygote.

^jVitamin E added to the diet at the level of 1,000 IU/kg.

of pork in some situations, but not in others. We need further clarification of the situations in which it provides these benefits.

Selenium

Selenium is a component of the enzyme glutathione peroxidase, an important biological antioxidant. It is

important to provide an adequate amount of selenium to support maximal antioxidant activity of glutathione peroxidase, but there is unlikely to be a benefit of providing higher levels of selenium (Benzie, 1996). In fact, caution must be exercised to protect against toxic effects, because the ratio of toxic level to requirement is quite low in the case of selenium. We did not identify any studies of the effect of dietary selenium level on

Table 11. Effect of preslaughter feed deprivation during transport and handling on pork quality^a

Measure	Time off feed, h					
	0	2	24	48	72	96
pH (ultimate)	5.52	—	5.64	5.61	—	—
L*	52.1	47.9	49.0	46.5	44.9	45.1
Water-holding capacity, %	—	58.2	60.1	62.5	63.7	66.5
Drip loss	32.0	—	20.0	—	—	—
Color (1 = pale, 5 = dark)	2.85	—	2.93	2.96	—	—

^aSchaefer et al. (1995). Summarized from three original reports.

oxidative stability of pork. See a later section for a description of the effect of dietary selenium on other aspects of pork quality.

Vitamin C

Vitamin C is a potent biological antioxidant (Benzie, 1996). Therefore, it is reasonable to postulate that addition of vitamin C to the pig's diet might improve oxidative stability of the pork. The pig synthesizes vitamin C and does not require it in the diet for normal growth, but there remains the possibility that endogenous synthesis is inadequate to maximize vitamin C's contribution to oxidative stability in some situations. Tsai et al. (1978) found no improvement in oxidative stability (TBARS, peroxide value) of pork muscle or adipose from addition of vitamin C to the diet, but high standard deviations of the measurements impaired the sensitivity of the experiment. Provision of dietary vitamin C to livestock has historically been difficult because the vitamin is very unstable in feeds. However, stabilized forms are now commercially available. Studies of the effect of these new stabilized forms on oxidative stability of pork would be useful. As noted later, vitamin C may have another role in pork quality.

Nutritional Factors Associated with Carbohydrate Metabolism and pH

Empirical observations of effects (or lack of effects) of nutritional factors on pH, color, or water-binding capacity are included in this section if they do not obviously belong elsewhere.

Preslaughter Feed Deprivation

A preslaughter feed deprivation can potentially reduce the muscle glycogen content at slaughter, and that could theoretically increase the ultimate pH. The data in Table 11 suggest this relationship occurs, and that lack of a feed deprivation can result in an increased incidence of PSE pork, whereas too long a feed deprivation can increase the incidence of the DFD condition. These data suggest that with a feed deprivation of 2 h or less, pork is light in color with a low pH and high drip loss. The longest periods of preslaughter feed deprivation

produced dark-colored pork with high water-holding capacity.

However, the response to preslaughter feed deprivation is not always so predictable. Koohmarie et al. (1991) found what appears to be the opposite response in pH decline during the first 3 h postmortem (Table 12). The issue is complicated also by the original amount of glycogen in the muscle, by preslaughter handling, and by the environmental temperature. Hot weather leads to a greater incidence of PSE, but cold weather exhausts glycogen stores in feed-deprived pigs and causes more DFD pork (Barton-Gade, 1997).

Pigs that carry the Rendement Napole gene have elevated muscle glycogen levels. An initial study (Bidner et al., 1999b) found that in those pigs a 60-h feed deprivation was not sufficient to reduce the muscle glycogen levels enough to raise the ultimate pH of longissimus muscles. In pigs without the gene, a 36-h feed deprivation was adequate to raise the ultimate pH (Table 13). The pigs in this experiment were mixed before slaughter. In a second study (Bidner et al., 1999a) in which pigs were not mixed before slaughter, a 36-h feed deprivation had no effects in either genotype. Perhaps, in the pigs without the gene, mixing before slaughter was sufficient stress to reduce muscle glycogen levels prior to slaughter enough to result in greater ultimate pH. Preslaughter feed deprivations up to 60 h did not reduce the carcass weight (F. K. McKeith, personal communication) but did reduce live weight (presumably because of reduced gut fill). Prolonged preslaughter feed deprivations have no consistent effects on tenderness,

Table 12. Effect of preslaughter feed deprivation on postmortem pH^a

Hours postmortem	Time off feed, h		
	0	48	P
0	6.40	6.49	NS ^b
3	6.44	6.06	<0.05
6	6.00	6.01	NS
9	5.78	5.84	NS
12	5.51	5.62	NS
24	5.42	5.46	NS

^aKoohmarie et al. (1991).

^bNot significant, $P > 0.05$.

Table 13. Effect of preslaughter feed deprivation on meat quality of pigs with low and high glycogen levels

Source/measurement	Glycogen levels					
	Low ^a			High ^b		
	Time off feed, h			Time off feed, h		
	12	36	60	12	36	60
Bidner et al., 1999b						
Pigs mixed						
Ultimate pH	5.45 ^c	5.59 ^d	5.65 ^d	5.36 ^c	5.34 ^c	5.36 ^c
Purge loss, %	4.10	2.46	2.37	4.48	4.66	4.05
Drip loss, %	4.17	3.11	3.50	5.49	6.22	5.25
Hunter L*	55.54 ^c	53.08 ^d	51.76 ^d	55.33 ^c	55.55 ^c	55.48 ^c
Bidner et al., 1999a						
Pigs not mixed						
Ultimate pH	5.48	5.51	—	5.46	5.42	—
Drip loss, %	7.32	6.94	—	7.31	7.96	—
Hunter L*	55.3	54.4	—	52.5	53.2	—
Minolta L*	50.2	48.9	—	46.9	48.5	—

^aPigs without the Rendement Napole gene.

^bPigs with the Rendement Napole gene.

^{c,d}Means within rows with different superscripts differ.

as detailed later. In practice an adequate preslaughter feed deprivation can often be achieved by shutting off feeders at night and shipping the pigs early the following morning. Pigs not stressed by heat or crowding eat very little at night, so they have a self-imposed feed deprivation (Hyun et al., 1997).

Sugar Feeding

If the length of the preslaughter feed deprivation is long and the pigs are cold or fighting, the glycogen levels in muscle can be reduced so far that DFD pork results. In such cases, it may be possible to restore the glycogen to desired levels by providing a sugar solution for consumption by the pigs during lairage. The evidence suggests that providing a sugar solution reduces the incidence of DFD pork. We are unlikely to adopt this practice for general use because it would increase the risk of producing PSE pork, which is a bigger problem for the industry than is DFD.

Sodium Oxalate

Sodium oxalate inhibits a key glycolytic enzyme, pyruvate kinase, so if its concentration in muscle tissue can be raised adequately that might slow postmortem glycogen metabolism and improve meat quality. Data from one experiment (Kremer et al., 1999b) suggest that adding sodium oxalate to the final meal given to pigs before slaughter resulted in higher early postmortem pH and reduced water loss from the muscle (Table 14). Ultimate pH was not affected.

Vitamin C

Vitamin C is a precursor of oxalic acid, so its supplementation in the diet may mimic the beneficial effects

of sodium oxalate described above. Data from two experiments (Kremer et al., 1999a) suggest that it does, showing improvements in pH, drip loss, and Hunter L* and a* values (Table 14). A brief discussion of the potential influence of dietary vitamin C on fat metabolism is offered earlier in this paper.

Quercetin

Quercetin, a compound found naturally in fruits and vegetables, inhibits lactate dehydrogenase, another glycolytic enzyme. When quercetin was added to the final meal given to pigs before slaughter (Kremer et al., 1999c), the early postmortem pH of muscle tended to be higher but a* values (redness) were lower (Table 14). Ultimate pH was not affected.

Magnesium

When an animal is stressed, it releases catecholamines (epinephrine and norepinephrine), and one of the effects of these hormones is stimulation of glycogenolysis. Pigs that are stressed before slaughter have a high rate of glycogenolysis, and therefore a relatively high incidence of poor-quality pork. A high level of dietary magnesium alleviates stress and reduces catecholamine release. It has been suggested that a high dietary intake of magnesium before slaughter might improve pork quality, especially in stressed pigs.

Results of three relevant studies using short-term (5 d before slaughter) dietary additions of magnesium are summarized in Table 15. In the study by Schaefer et al. (1993) with very low doses of magnesium, the only statistically significant ($P < 0.05$) effects were on redness (Minolta a*) and initial muscle temperature (data not shown). There was a trend ($P < 0.06$) to less drip loss

in one block. However, the results of two experiments by D'Souza et al. (1998, 1999) with higher doses show marked improvements in several measures of pork quality from dietary magnesium. Most strikingly, adding magnesium to the diet for 5 d before slaughter completely eliminated PSE carcasses in both experiments. The response was obtained from magnesium aspartate, magnesium sulfate, or magnesium chloride (D'Souza et al., 1999).

The important challenges remaining before magnesium supplementation can be used to improve pork quality throughout a wide section of the industry are 1) to clarify the most appropriate dose, duration, and source of magnesium and 2) to devise methods for delivering magnesium-supplemented feeds to pigs before slaughter in commercial practice.

Other Electrolytes

Preslaughter stress alters blood concentrations of certain electrolytes, changes that may contribute to detrimental effects on pork quality. An experiment by Schaefer et al. (1993) failed to show improved pork quality from potassium supplementation of the diet. Oral intake of an alkaline salt (sodium bicarbonate) before slaughter increased ($P < 0.05$) the initial muscle pH (slowed glycogen metabolism) in one experiment (Ahn et al., 1992) but had no effect in two others (Boles et al., 1993, 1994). Oral intake of an acid salt (ammonium chloride) increased ($P < 0.05$) b^* values (yellowness) in two experiments (Boles et al., 1993; 1994),

but there was a trend in the opposite direction in the work of Ahn et al. (1992). There were no effects of either salt on ultimate pH, drip loss, or L^* or a^* values in any of the three experiments. A taste panel judged loin roasts from the pigs given ammonium chloride to be significantly ($P < 0.05$) less firm, less juicy, more tender, and more mealy with less intense flavor than control pigs (Boles et al., 1993). Sodium bicarbonate had no effect on sensory qualities. These data fail to demonstrate that altering acid-base balance or increasing electrolyte intake before slaughter will improve pork quality.

Tryptophan

The amino acid tryptophan is the precursor of serotonin, a brain neurotransmitter that attenuates the animal's response to stress. In one study reviewed by Pethick et al. (1997), addition of 0.5% tryptophan to the diet (five times the requirement estimate of NRC [1998]) for 5 d before slaughter increased serotonin concentration in the hypothalamus and reduced the incidence and severity of PSE pork. However, another experiment found no effect of a similar treatment on meat quality, although the pigs receiving supplemental tryptophan were less aggressive when mixed in lairage.

Creatine

Creatine binds and holds phosphate within the muscle cell, and that phosphate can buffer the postmortem

Table 14. Effect of glycolytic inhibitors in the diet on pork quality^a

Measurement	Sodium oxalate ^b			Vitamin C			Quercetin ^d		
	0	2	10	NA	NA	NA	0	2	10
Feed allocation, g/pig ^e	545	568	557	NA	NA	NA	545	563	532
Feed intake, g/pig	342	360	100	325	371	300	342	431	377
Inhibitor intake, g/pig	0	7.0	9.7	0	0.290	0.715	0	1.08	4.67
Muscle traits postmortem ^f									
pH	5.88 ^g	5.97	5.98	6.06 ^j	6.14	6.18	5.88 ^h	5.96	6.00
Temperature °C	31.4	31.2	31.0	NA	NA	NA	31.4	31.3	31.1
During storage ⁱ									
Water loss, %	11.3 ^j	10.1	9.5	5.99 ^m	5.78	5.21	11.3	9.1	11.0 ^k
L^*	52.9	53.0	52.7	47.9 ^l	45.7	47.5	52.9	52.8	53.4
a^*	5.6	5.5	5.3	8.9 ⁿ	10.0	9.2	5.6 ^l	5.0	5.0
After storage (d 12)									
Cooking Loss, %	29.5	29.6	28.4	NA	NA	NA	29.5	30.3	30.0
Penetration resistance, kg	3.00	2.97	3.00	NA	NA	NA	3.00	2.82	2.85

^aPooled across chill rate (rapid or slow) and muscle type (longissimus and semimembranosus).

^bKremer et al. (1999b). Treatment levels (0, 2, 10) are the target tissue concentrations of the inhibitors relative to K_i .

^cKremer et al. (1999a).

^dKremer et al. (1999c). Treatment levels (0, 2, 10) are the target tissue concentrations of the inhibitors relative to K_i .

^eProvided 4 h before transport to slaughter (sodium oxalate and quercetin) or 4 h prior to stunning (vitamin C).

^fPooled across 22, 45, 90 and 180 min postmortem.

^gDifferent from higher levels, $P < 0.06$.

^hDifferent from higher levels, $P < 0.10$.

ⁱPooled across 0, 3, 6, 9, and 12 d of retail storage.

^jDifferent from higher levels, $P < 0.05$.

^kDifferent from next level, $P < 0.05$.

^lDifferent from higher levels, $P < 0.01$.

^mDifferent from higher levels, $P < 0.07$.

ⁿDifferent from higher levels, $P < 0.02$.

Table 15. Effect of dietary magnesium supplementation on pork quality

Source/measurement	Mg dose ^a	Mg source	Initial pH (40 or 45 min)			Minolta L*			Drip loss, %			PSE incidence, % ^b		
			Control	Mg	Difference	Control	Mg	Difference	Control	Mg	Difference	Control	Mg	Difference
Schaefer et al., 1993^c														
Block 1	25 mg	Aspartate	5.84	5.82	-0.02	59.4	58.7	-0.7	5.0	5.0	0.0	NA	NA	NA
Block 2	50 mg	Aspartate	5.90	5.96	0.06	55.5	54.8	-0.7	4.2	3.6 ^d	-0.6	NA	NA	NA
D'Souza et al., 1998														
Longissimus thoracis														
Minimal handling ^e	3.2 g	Aspartate	6.60	6.79	0.19	48.7	45.2	-3.5	4.0	3.5	-0.5	8	0	-8
Negative handling ^f	3.2 g	Aspartate	6.59	6.69	0.10	49.1	47.4	-1.7	6.4	3.5	-2.9	33	0	-33
Biceps femoris														
Minimal handling ^e	3.2 g	Aspartate	6.54	6.62	0.08	44.0	44.0	0.0	3.0	2.2	-0.8	NA	NA	NA
Negative handling ^f	3.2 g	Aspartate	6.42	6.49	0.07	45.7	45.3	-0.4	4.8	4.7	-0.1	NA	NA	NA
Probabilities ^g														
Diet	—	—	—	0.02	—	—	0.04	—	—	0.05	—	—	0.05	—
Diet × muscle	—	—	—	NS	—	—	0.02	—	—	NS	—	—	—	—
Diet × muscle × handling	—	—	—	NS	—	—	NS	—	—	0.03	—	—	—	—
D'Souza et al., 1999														
	3.2 g	Aspartate	6.48	6.70	0.22	48.7	47.8	-0.9	5.8	3.2	-2.6	17	0	-17
	3.2 g	Sulfate	—	6.62	0.14	—	49.1	0.4	—	2.8	-3.0	—	0	-17
	3.2 g	Chloride	—	6.52	0.04	—	48.8	0.1	—	3.1	-2.8	—	0	-17
Mean					0.10			-0.8						-1.5

^aDaily dose of supplemental magnesium during the last 5 d before slaughter.

^bDefined as L* > 50 and drip loss > 5%.

^cPooled across all three Ryanadine receptor (Halothane) genotypes; longissimus muscle.

^dDifferent from control, $P < 0.06$.

^ePigs handled gently before slaughter.

^fPigs stressed with electric prod before slaughter.

^gOnly significant ($P < 0.06$) effects involving diet are shown.

^hDifferent from magnesium-supplemented treatments, $P < 0.05$.

pH drop caused by accumulation of lactic acid. Perhaps adding creatine to the diet before slaughter could reduce the rate of pH decline and thereby improve meat quality. Results of one experiment (Berg et al., 1999) confirm that dietary supplementation with creatine for 5 or 10 d before slaughter increased both initial and ultimate pH in the semimembranosus muscle. This effect was not found in the loin. There were nonsignificant trends to reduced drip loss in both muscles and reduced L^* values in semimembranosus. The standard deviation of L^* values was reduced sharply in both muscles by the dietary creatine. These results appear promising. As noted earlier in this paper, the added creatine also appeared to increase marbling.

Chromium

In individual experiments, supplementing the diet with chromium has reduced a subjective color score (caused less intense color) or increased the Hunter a:b ratio, but these effects have not been consistent across experiments (O'Quinn et al., 1998b). It has been reported to increase (Waylan et al., 1999) or decrease (O'Quinn et al., 1998b) the saturation of the fats in pork loins but to have no effect on oxidative stability (Waylan et al., 1999). It had no effect on shear force. Overall, the evidence does not show clear effects of dietary chromium on pork quality, except for leanness.

Conjugated Linoleic Acid (CLA)

The beneficial effects of dietary CLA on carcass leanness, marbling, and belly firmness were described above. A growing body of data suggests that these benefits are derived without detrimental effects on other quality characteristics. The data are not presented in tabular form because of the wide range of experimental treatments employed, especially with regard to dose and duration of CLA feeding.

Water-holding capacity has been either improved (O'Quinn et al., 1998a) or unchanged (Eggert et al., 1998; Thiel-Cooper et al., 1999; Wiegand et al., 1999) by CLA in the diet. There seem to be no effects on tenderness (Larsen et al., 1999; Thiel-Cooper et al., 1999; Waylan et al., 1999) or ultimate pH (Eggert et al., 1998; Thiel-Cooper et al., 1999; Wiegand et al., 1999). Sensory scores are largely unaffected by dietary CLA (Larsen et al., 1999; Thiel-Cooper et al., 1999), although in one experiment a panel considered chops from CLA-fed pigs to be slightly less juicy than controls (Thiel-Cooper et al., 1999).

The effect of dietary CLA on pork color is difficult to decipher from the data now available. Most individual measures of color, whether subjective or objective, have shown no effect of CLA (Eggert et al., 1998; O'Quinn et al., 1998a; Eggert et al., 1999a). However, several experiments found an effect on some measure of color. Some of them (Eggert et al., 1999a,b; Larsen et al., 1999) found effects on subjective but not objective mea-

ures, whereas others (Thiel-Cooper et al., 1999; Waylan et al., 1999; Wiegand et al., 1999) found the opposite. Larsen et al. (1999) found a subjective increase in color uniformity. Among objective measures, there were increases in Hunter a^* values (redness; Thiel-Cooper et al., 1999), in b^* values (yellowness; Waylan et al., 1999; Wiegand et al., 1999), or both (O'Quinn et al., 1998a). There were no effects on L^* (lightness). Overall, it seems that dietary CLA probably causes a subtle increase in color intensity in pork.

Betaine

In two recent experiments, there were contrary effects of the methyl donor betaine on meat color. In one experiment (Matthews et al., 1998), adding betaine to the diet reduced a subjective color score (2.0 to 1.8; $P < 0.01$). In another experiment, betaine reduced a subjective whiteness score assigned to the longissimus muscle (Øverland et al., 1999). On a 9-point scale in which 9 is the most intense white, the control muscles scored 6.2 and muscles from the betaine treatment (1.00% of the diet) scored 5.9 ($P < 0.05$). There were no significant effects on 15 other measures of odor, flavor, color, and texture. It seems unlikely that adding betaine to the diet will alter lean quality. The potential effects of dietary betaine on leanness are discussed elsewhere.

Trimethylamine Oxide

This compound, which is a methyl donor like betaine, is found widely in biology. In one experiment (Øverland et al., 1999), adding it to the diet of finishing pigs reduced subjectively determined whiteness of the longissimus muscle from 6.2 to 5.9 on a 9-point scale ($P < 0.05$). Effects on leanness were described earlier.

Carnitine

Adding carnitine to the diet had no effect on shear force, fat saturation, or oxidative stability (Waylan et al., 1999). It had no effect on color measured subjectively or objectively except that it increased L^* (lightness) only in the presence of chromium nicotinate. The earlier discussion suggests it also has no dependable effect on leanness.

Selenium

Swine diets are routinely supplemented with sodium selenite to ensure that the pig's selenium requirement is met. Selenium-enriched yeast is an alternative selenium source for pig diets. Most of the selenium in yeast is in organic compounds such as selenomethionine and selenocystine. Mahan et al. (1999) found a tendency to increased drip loss from the loin with increasing supplementation of the diet with sodium selenite, but not with a selenium-enriched yeast. This was associated with increased ($P < 0.05$) Hunter L^* values for the loin as the dietary sodium selenite level increased, but

Table 16. Effect of a high level of vitamin D before slaughter on pork tenderness^a

Method ^b and time postmortem	Control	Vitamin D ^c	Difference	<i>P</i>
Warner-Bratzler shear, kg				
1 d	3.57	3.65	0.08	0.82
7 d	3.10	3.06	-0.04	0.88
14 d	3.01	3.06	0.05	0.83
21 d	2.92	3.04	0.12	0.51
Mean	—	—	0.05	—
Star probe				
1 d	5.73	5.92	0.19	0.58
7 d	5.26	5.65	0.39	0.54
14 d	5.71	5.46	-0.25	0.33
21 d	5.04	5.20	0.16	0.31
Mean	—	—	0.12	—

^aSparks et al. (1999a).

^bLoins were cooked at 71°C; measured at 21°C.

^c500,000 IU vitamin D₃/d for 3 d before slaughter.

not as the level of selenium yeast increased. However, Wolter et al. (1999) found no difference between supplementation with the two selenium sources on drip loss, L* values, or other measures of pork quality. Firm conclusions about the importance of dietary selenium source for pork quality must await further information. Potential effects of dietary selenium level on fat metabolism are discussed above.

Amino Acid Levels

As noted elsewhere, it has been proposed that lowering the amino acid levels of the diet can improve eating quality by increasing marbling and tenderness. That change has little other effect on pork quality. Studies have found no effects of reduced dietary amino acid concentrations on drip loss (Essén-Gustavsson et al., 1994; Goerl et al., 1995; Witte et al., 2000) or subjective firmness score (Cisneros et al., 1996; Witte et al., 2000). Goerl et al. (1995) found no effect on pH. Goerl et al. (1995) found that low-protein diets increased ($P < 0.01$) the Hunter a* and b* values, but several other studies (Essén-Gustavsson et al., 1994; Cisneros et al., 1996; Witte et al., 2000) found no effect on objective or subjective measures of color.

Vitamin D

A high dose of vitamin D before slaughter improved color and water-holding capacity of pork in one study (Enright et al., 1998), and in another (Sparks et al., 1999a) there were nonsignificant and inconsistent trends in the same direction. However, the vitamin D reduced growth rate markedly in both of these studies. A discussion of the effect of vitamin D on tenderness follows.

Nutritional Factors Associated with Calcium Metabolism

Postmortem degradation of specific structural proteins (e.g., titin and nebulin) in the Z-band of striated muscle is critical to production of tender meat. That

protein degradation appears to be catalyzed largely by calcium-activated proteases called calpains. Under physiological and postmortem conditions, the activity of these calpains seems to be sensitive to calcium concentration. Therefore, it seems reasonable that if we could increase the intracellular calcium concentration, that would increase the proteolytic activity of the calpains, and the meat would become more tender. However, the intracellular calcium concentrations are under tight homeostatic control, so it may be difficult to raise those concentrations significantly by dietary means. For convenience, observations of nutritional effects on tenderness with no clear mechanism are included in this section.

Vitamin D

Vitamin D raises tissue calcium concentrations, functioning as part of the calcium homeostatic system. Perhaps a high dietary level of vitamin D shortly before slaughter could overwhelm the homeostatic system and increase intracellular calcium levels during the critical postmortem period. In fact, such treatments have increased calcium concentrations and tenderness in beef (Swanek et al., 1999). Injection of calcium chloride into the pork loin muscle postmortem improves tenderness (McFarlane and Unruh, 1996), as it does in lamb (Koohmarie et al., 1989) and beef (Wheeler et al., 1991). This provides encouragement that the hypercalcemic effect of vitamin D can be effective in increasing tenderness of pork.

The limited data available to date in pigs do not show an effect of increasing intake of vitamin D before slaughter. Table 16 shows the data of Sparks et al. (1999a), who added 500,000 IU/d to the diet for 3 d. The treatment was successful in increasing the serum calcium concentration. Enright et al. (1998) also did not find a tenderness response to additional dietary vitamin D. Perhaps a different dosage or duration would be more effective, or perhaps there is a species difference in response. The likelihood of a reduction in growth rate, associated with reduced feed intake, may

Table 17. Effect of preslaughter feed deprivation on pork tenderness^a

Method/time postmortem	Fed	Deprived ^b	Difference	<i>P</i>
Shear force, kg ^c				
Day 1	4.99	4.79	-0.20	NS
Day 7	4.77	3.37	-1.40	<0.05
Day 14	4.23	3.59	-0.64	<0.05
Mean	—	—	-0.75	—
Myofibril fragmentation index				
Day 1	64.6	63.1	-1.5	NS
Day 3	74.7	75.2	0.5	NS
Day 7	78.1	90.7	12.6	<0.05
Day 14	85.8	90.2	4.4	NS
Mean	—	—	5.8	—

^aKoohmarie et al. (1991).

^bFasted 48 h before slaughter.

^cCooked at 70°C, refrigerated for 24 h.

affect the use of vitamin D to improve leanness, if it is eventually shown to be efficacious. See above for a discussion of potential effects of a high dietary level of vitamin D on other measures of pork quality.

Feeding Levels

Restricting the feed intake of finishing pigs results in pork that is detectably less tender (Warkup et al., 1990; Ellis et al., 1996; Blanchard et al., 1999), with a greater shear force required to penetrate it (Ellis et al., 1996). This represents an advantage for the ad libitum system most common in the United States over restricted feeding systems.

Amino Acid Levels

The beneficial effect of reduced-protein diets on marbling and the general lack of effects on other measures of pork quality have been described above. Two experiments (Essén-Gustavsson et al., 1994; Goerl et al., 1995) indicated that pork from pigs fed low-protein diets is more tender (lower shear force) than pork from control pigs, and a third (Witte et al., 1999) showed a nonsignificant trend in the same direction. There remain the problems of reduced growth performance and increased carcass fatness caused by such diets.

Preslaughter Feed Deprivation

An extended preslaughter feed deprivation increased the rate of postmortem tenderization of the pork in one study (Koohmarie et al., 1991; Table 17) but had no effect on sensory properties in two others (Bidner et al., 1999a,b). The effects of preslaughter feed deprivation on other pork quality attributes are described above.

Summary and Conclusions

We have the means to improve pork quality through nutritional changes. The pork industry has a rich sup-

ply of proven and potential nutritional means to alter the quality characteristics of pork.

We are already using some of these technologies, directed to improving carcass leanness. Carcass leanness is one of the outcomes we consider in selection of energy and amino acid densities in diets for finishing pigs. Some producers supplement finisher diets with betaine or carnitine to make carcasses leaner, although the scientific support for these additions is not strong. Chromium is available for the same purpose, but its use is limited. Conjugated linoleic acid will soon become available for use by the industry at large for improvement of carcass leanness.

The industry's aggressiveness in developing and adopting nutritional means to increase carcass leanness stands in contrast to the apparent lack of interest in methods to improve other aspects of pork quality. Of course, the reason for this apparent lack of interest is the perception that there are no financial rewards to the producer for improving pork quality. That lack of rewards is especially significant for nutritional interventions that have a direct financial cost. Perhaps we are in roughly the same stage of development in pork quality now as we were in leanness about two decades ago, before there were systematic methods for providing financial rewards to producers who produce lean pigs. If so, we can take encouragement from the widespread adoption of leanness premiums during recent years. Perhaps the entire pork chain will find ways to develop appropriate rewards for other aspects of pork quality in the near future.

There are two promising nutritional technologies for improving the quality of pork fat. The beneficial effect of high levels of dietary vitamin E on oxidative stability and shelf-life are well documented, and adoption of this technology awaits only an economic incentive. Conjugated linoleic acid provides clear benefits in belly firmness, but again there must be a method for passing this advantage to the decision-maker. The other documented nutritional effects on fat quality (leanness, dietary fat, and copper) are of use in avoiding problems

rather than in making real progress. Vitamin C deserves a closer look for possible benefits.

In terms of nutritional effects on quality of lean, perhaps the most important thing we can do now is ensure that all pigs are held off feed for an appropriate time before slaughter. Providing magnesium to pigs before slaughter improves pork quality dramatically. We can adopt this technology throughout the industry as soon as we work out some of the practical details and provide appropriate incentives. Addition of glycolytic inhibitors or creatine to the diet before slaughter deserves more research attention. We now have no nutritional technologies to improve tenderness of pork, but we should continue to investigate a possible role for vitamin D.

Implications

We have the means to improve pork quality through nutritional changes. The pork industry has a rich supply of proven and potential nutritional means to alter the quality characteristics of pork. Among the more promising are appropriate dietary levels of energy and amino acids, and conjugated linoleic acid to improve leanness; high levels of dietary vitamin E to improve oxidative stability and extend shelf-life; conjugated linoleic acid to improve fat firmness; and preslaughter feed deprivation and supplemental magnesium to improve water-binding capacity, color, and pH.

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