IOWA STATE UNIVERSITY
Digital Repository

Animal Science Publications | Animal Science

3-2007

Effects of breed, sex, and halothane genotype on fatty acid composition of pork longissimus muscle

S. Zhang
Iowa State University

Travis J. Knight
Iowa State University

Kenneth J. Stalder
Iowa State University, stalder@iastate.edu

Rodney N. Goodwin
Iowa State University

Steven M. Lonergan
Iowa State University, slonerga@iastate.edu

See next page for additional authors

Follow this and additional works at: http://lib.dr.iastate.edu/ans_pubs

Part of the Agriculture Commons, and the Meat Science Commons

The complete bibliographic information for this item can be found at http://lib.dr.iastate.edu/ans_pubs/13. For information on how to cite this item, please visit http://lib.dr.iastate.edu/howtocite.html.

This Article is brought to you for free and open access by the Animal Science at Iowa State University Digital Repository. It has been accepted for inclusion in Animal Science Publications by an authorized administrator of Iowa State University Digital Repository. For more information, please contact digirep@iastate.edu.

Effects of breed, sex, and halothane genotype on fatty acid composition of pork longissimus muscle

Abstract

The objective of this study was to estimate the effects of breed, sex, and halothane genotype on fatty acid composition and several fatty acid indices of lipid extracted from porcine LM. Purebred Yorkshire (n = 436), Duroc (n = 353), Hampshire (n = 218), Spotted (n = 187), Chester White (n = 173), Poland China (n = 124), Berkshire (n = 256), and Landrace (n = 187) pigs (n = 1,934; 1,128 barrows and 806 gilts) from 1991, 1992, 1994, and 2001 National Barrow Show Sire Progeny Tests were used. Pigs were classified as the HAL-1843 normal (NN) genotype (n = 1,718) or the HAL-1843 carrier (Nn) genotype (n = 216). For statistical analysis, a mixed model was used that included fixed effects of breed, sex, halothane genotype, test, slaughter date, interaction of breed × sex, and random effects of sire and dam within breed. Breed significantly affected the concentration of individual fatty acids, total lipid content, and the values of several fatty acid indices of LM. Duroc pigs had the greatest (P< 0.01) content of total SFA. Total MUFA concentration in Poland China pigs was greater ($P < 0.05$) than in all other breeds except the Spotted (P> 0.05). The concentrations of total PUFA were greater ($P < 0.01$) in Hampshire, Landrace, and Yorkshire pigs compared with those of other breeds. Significant sex differences for individual fatty acids were detected. Compared with gilts, barrows had greater ($P < 0.01$) concentrations of SFA and MUFA but lower ($P < 0.01$) total PUFA. Halothane genotype was a significant source of variation for the percentages of some fatty acids. Pigs with the carrier (Nn) genotype had lower concentrations of SFA ($P < 0.05$) and MUFA ($P < 0.01$) but a greater concentration of PUFA ($P < 0.01$) compared with NN pigs. There were significant negative correlations between total lipid content and individual PUFA and significant positive correlations between lipid concentration and most individual SFA and MUFA. In conclusion, the results suggest that breed and sex are important sources of variation for fatty acid composition of LM.

Keywords

Biochemistry Biophysics and Molecular Biology, breed, fatty acid, halothane genotype, longissimus muscle, pig, sex

Disciplines

Agriculture | Animal Sciences | Meat Science

Comments

This article is from *Journal of Animal Science* 85 (2007): 583–591, doi:10.2527/jas.2006-239. Posted with permission.

Authors

S. Zhang, Travis J. Knight, Kenneth J. Stalder, Rodney N. Goodwin, Steven M. Lonergan, and Donald C. Beitz

This article is available at Iowa State University Digital Repository: http://lib.dr.iastate.edu/ans_pubs/13

JOURNAL OF ANIMAL SCIENCE
The Premier Journal and Leading Source of New Knowledge and Perspective in Animal Science

Effects of breed, sex, and halothane genotype on fatty acid composition of pork longissimus muscle

S. Zhang, T. J. Knight, K. J. Stalder, R. N. Goodwin, S. M. Lonergan and D. C. Beitz

J ANIM SCI 2007, 85:583-591.
doi: 10.2527/jas.2006-239 originally published online October 23, 2006

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://www.journalofanimalscience.org/content/85/3/583

American Society of Animal Science

www.asas.org

Downloaded from www.journalofanimalscience.org at Serials Acquisitions Dept on February 21, 2014

Effects of breed, sex, and halothane genotype on fatty acid composition of pork longissimus muscle[1]

S. Zhang,* T. J. Knight,† K. J. Stalder,†[2] R. N. Goodwin,† S. M. Lonergan,† and D. C. Beitz†

*Department of Biochemistry, Biophysics, and Molecular Biology, and †Department of Animal Science, Iowa State University, Ames 50010

ABSTRACT: The objective of this study was to estimate the effects of breed, sex, and halothane genotype on fatty acid composition and several fatty acid indices of lipid extracted from porcine LM. Purebred Yorkshire (n = 436), Duroc (n = 353), Hampshire (n = 218), Spotted (n = 187), Chester White (n = 173), Poland China (n = 124), Berkshire (n = 256), and Landrace (n = 187) pigs (n = 1,934; 1,128 barrows and 806 gilts) from 1991, 1992, 1994, and 2001 National Barrow Show Sire Progeny Tests were used. Pigs were classified as the HAL-1843 normal (NN) genotype (n = 1,718) or the HAL-1843 carrier (Nn) genotype (n = 216). For statistical analysis, a mixed model was used that included fixed effects of breed, sex, halothane genotype, test, slaughter date, interaction of breed × sex, and random effects of sire and dam within breed. Breed significantly affected the concentration of individual fatty acids, total lipid content, and the values of several fatty acid indices of LM. Duroc pigs had the greatest ($P < 0.01$) content of total SFA. Total MUFA concentration in Poland China pigs was greater ($P < 0.05$) than in all other breeds except the Spotted ($P > 0.05$). The concentrations of total PUFA were greater ($P < 0.01$) in Hampshire, Landrace, and Yorkshire pigs compared with those of other breeds. Significant sex differences for individual fatty acids were detected. Compared with gilts, barrows had greater ($P < 0.01$) concentrations of SFA and MUFA but lower ($P < 0.01$) total PUFA. Halothane genotype was a significant source of variation for the percentages of some fatty acids. Pigs with the carrier (Nn) genotype had lower concentrations of SFA ($P < 0.05$) and MUFA ($P < 0.01$) but a greater concentration of PUFA ($P < 0.01$) compared with NN pigs. There were significant negative correlations between total lipid content and individual PUFA and significant positive correlations between lipid concentration and most individual SFA and MUFA. In conclusion, the results suggest that breed and sex are important sources of variation for fatty acid composition of LM.

Key words: breed, fatty acid, halothane genotype, longissimus muscle, pig, sex

©2007 American Society of Animal Science. All rights reserved.
J. Anim. Sci. 2007. 85:583–591
doi:10.2527/jas.2006-239

INTRODUCTION

Meat fatty acid composition is of great interest because of its implications for human health. High intake of SFA causes elevated plasma cholesterol, which contributes to cardiovascular disease (Bronte-Stewart et al., 1956). In contrast, PUFA and MUFA increase hepatic low-density lipoprotein receptor activity, thereby decreasing the circulating concentration of LDL-cholesterol (Woollett et al., 1992; Rudel et al., 1995). Nutritional concern about dietary fat, especially SFA, has stimulated the production of leaner pigs in the past 2 decades. However, low content of intramuscular fat (**IMF**) is believed to negatively impact pork flavor (DeVol et al., 1988). Manipulation of the fatty acid composition of IMF, therefore, plays an important role in producing healthier pork without compromising consumer acceptable palatability.

Fatty acid composition of IMF in pigs is affected by environmental factors, such as diet, and by genetic factors, such as breed, sex, and halothane genotype (DeSmet et al., 2004). Numerous studies have shown that fatty acid composition of pig muscle and adipose tissue can be changed through modified feeding strategies (Gatlin et al., 2002, Kouba et al., 2003; Nürnberg et al., 2005). In contrast, the effects of genetic factors, especially breed and halothane genotype, have received less attention (Piedrafita et al., 2001; Wood et al., 2004; LoFiego et al., 2005). This study is the first to investigate the effects of breed on fatty acid composition using

[1]This paper of the Iowa Agriculture and Home Economics Experiment Station, Ames, Project No. 3801, was supported by Hatch Act and State of Iowa funds.
[2]Corresponding author: stalder@iastate.edu
Received April 14, 2006.
Accepted October 17, 2006.

Downloaded from www.journalofanimalscience.org at Serials Acquisitions Dept on February 21, 2014

pigs of all of the 8 major swine breeds in the United States when fed similar diets. Determining the extent to which genetic factors affect the variation of fatty acid composition will provide breeders a tool to modify the fatty acid composition of pork and minimize the negative consequences for pork quality.

The objective of this study was to determine the influence of breed, sex, and halothane genotype on the fatty acid composition of LM lipids of purebred pigs fed the same diets.

MATERIALS AND METHODS

Animals

Animal care and use approval was not obtained for this study because the data were from an existing database. In brief, the following procedures were used to generate the data within the database.

Pigs (n = 1,934) of 8 purebred breeds from the 1991, 1992, 1994, and 2001 National Barrow Show Sire Progeny Test program (George A. Hormel and Co., Austin, MN) were used in the current study. The breeds were Yorkshire (n = 436), Duroc (n = 353), Hampshire (n = 218), Spotted (n = 187), Chester White (n = 173), Poland China (n = 124), Berkshire (n = 256), and Landrace (n = 187). Pigs were delivered to the Northwest Iowa Testing Station (New Hampton, IA) at approximately 8 wk of age and were penned by breed, sire group, and weight in a solid-concrete-floored, open-front building and given 1.4 m^2 of pen space per pig. Pigs within contemporary groups were fed the same diets. The diets of all pigs consisted of ground shelled corn, soybean meal, and supplements, which met or exceeded NRC (1988, 1998) requirements for all phases throughout these grow-finish studies. Additionally, incorporation of contemporary group within the statistical model used to evaluate these data accounts for dietary differences across contemporary groups. Halothane genotypes of the pigs were determined using the HAL-1843 test described by Fujii et al. (1991). Distribution of the breeds by year, sex, and HAL-1843 genotype is shown in Table 1. Pigs were weighed weekly and transported to George A. Hormel Company for slaughter at a BW of approximately 103 kg.

Sample Collection

A 3-rib (10th to 12th) section of the LM was excised from 1 side of each carcass, packaged individually, and stored at 4°C. Samples were transported to the Iowa State University Meat Laboratory and processed approximately 24 h later. The LM section then was trimmed of bone and external adipose tissue and cut into 3 equal-sized portions. The 10th rib section was ground in a food processor until finely homogenized and stored at −20°C for further analysis.

Fatty Acid Analysis

Total lipids were extracted from the LM samples using a chloroform and methanol (2:1, vol:vol) mixture (Folch et al., 1957). The lipids were methylated directly with acetyl chloride and methanol according to Lepage and Roy (1986). Fatty acid methyl esters (**FAME**) were quantified by a gas chromatograph (model 3400, Varian, Palo Alto, CA) equipped with a Supelco SP-2380 column (30 m long × 0.25 mm i.d. × 0.20 μm film thickness) and a flame ionization detector. The column began at a temperature of 100°C and was ramped to 170°C at a rate of 2°C/min, followed by an increase to 180°C at 0.5°C/min and to 250°C at 10°C/min. The total running time was 62 min. The temperature of the injector was programmed to increase from a beginning temperature of 68°C to a final temperature of 250°C at a rate of 250°C/min. The detector was maintained at 220°C.

Peaks of FAME were identified by comparing the retention time with the commercially available FAME standards (Nu-Chek-Prep Inc., Elysian, MN). The fatty acid compositions were calculated using the peak areas and expressed on a weight percentage basis. The activities of Δ9-desaturase and elongase were estimated by relating the percentage of product to the percentage of precursor (Pan et al., 1995; Okada et al., 2005). Specifically, the Δ9-desaturase (16) index was calculated as 100 times the ratio of the palmitoleic acid (C16:1) percentage to the sum of C16:1 and palmitic acid (C16:0). The Δ9-desaturase (18) index was calculated as 100 times the ratio of oleic acid (C18:1) to the sum of C18:1 and stearic acid (C18:0). The Δ9-desaturase (16+18) index was calculated as 100 times the ratio of the sum of C16:1 and C18:1 to the sum of C16:1, C16:0, C18:1, and C18:0. The elongase index was calculated as the ratio of C18:0 to C16:0. The thioesterase index was calculated as the ratio of C16:0 to myristic acid (C14:0). The index of atherogenicity (**IA**) was calculated according to Ulbricht and Southgate (1991).

Statistical Analysis

Least squares means (±SE) were determined using a mixed linear model (PROC MIXED, SAS Inst. Inc., Cary, NC) that included the fixed effects of breed, sex, halothane genotype, test, slaughter date (contemporary group), the interaction of breed × sex, and random effects of sire and dam within breed. Means were compared using pairwise *t*-tests (PDIFF option of SAS) and declared to be different at $P < 0.05$. Residual correlation coefficients between the traits were calculated using a fixed effects model with breed, sex, halothane genotype, test, slaughter date, and the interaction between breed × sex in a multivariate ANOVA in PROC GLM of SAS.

RESULTS AND DISCUSSION

Breed Effects

Breed was a significant source of variation for the percentage of individual fatty acids and the content of

Downloaded from www.journalofanimalscience.org at Serials Acquisitions Dept on February 21, 2014

Table 1. Distribution of pigs from the National Barrow Show Sire Progeny Test Program by breed, year, sex, and HAL 1843 genotype[1]

		Breed								
Year	Sex/Genotype	Yorkshire	Duroc	Hampshire	Spotted	Chester White	Poland China	Berkshire	Landrace	Total
1991	Barrows	123	80	74	62	27	29	35	42	472
	Gilts	81	52	51	37	36	28	31	42	358
1992	Barrows	66	58	27	31	26	12	15	17	252
	Gilts	44	30	23	25	10	8	7	9	156
1994	Barrows	49	21	23	13	11	13	12	33	175
	Gilts	33	19	20	13	4	11	7	21	128
2001	Barrows	29	49	0	5	35	11	94	6	229
	Gilts	11	44	0	1	24	12	55	17	164
Total	Barrows	267	208	124	111	99	65	156	98	1,128
	Gilts	169	145	94	76	74	59	100	89	806
1991	NN[2]	193	131	117	82	63	10	53	72	721
	Nn[3]	11	1	8	17	0	47	13	12	109
1992	NN	104	77	46	45	36	9	16	25	358
	Nn	6	11	4	11	0	11	6	1	50
1994	NN	82	38	39	20	15	9	16	53	272
	Nn	0	2	4	6	0	15	3	1	31
2001	NN	40	93	0	4	59	6	142	23	367
	Nn	0	0	0	2	0	17	7	0	26
Total	NN	419	339	202	151	173	34	227	173	1,718
	Nn	17	14	16	36	0	90	29	14	216

[1]Values in the table refer to the number of pigs.
[2]HAL-1843 normal genotype.
[3]HAL-1843 carrier genotype.

total lipids (Table 2). The concentration of each fatty acid in LM of Duroc pigs differed from that of Yorkshire and Landrace pigs ($P < 0.05$). Similarly, the percentage of each fatty acid differed between Chester White and Yorkshire pigs ($P < 0.05$). No significant fatty acid percentage differences were observed between Yorkshire and Landrace pigs. The lipid content of LM was greatest in Duroc pigs ($P < 0.01$). Berkshire pigs had the second greatest ($P < 0.05$) concentration of lipid but did not differ ($P > 0.05$) from Poland China pigs. In contrast, Yorkshire, Landrace, and Hampshire pigs had the lowest lipid content ($P < 0.01$). It has been shown that Duroc pigs have greater lipid content than Berkshire (Suzuki et al., 2003) and Landrace pigs (Lo et al., 1992). Similarly, Wood et al. (2004) reported that IMF content was relatively high and backfat lipid content was relatively low in Duroc pigs, whereas marbling and backfat lipid contents were high in Berkshire pigs. Newcom et al. (2004) reported that Duroc and Chester White pigs had the greatest IMF, whereas Landrace and Yorkshire pigs had the lowest IMF, which were not different from Hampshire and Poland China pigs.

The concentration of myristic acid (C14:0) in Duroc, Chester White, and Berkshire pigs was greater ($P < 0.01$) than in pigs from the other breeds (Table 2). Palmitic acid (C16:0) concentrations were significantly greater ($P < 0.01$) in Duroc and Berkshire pigs than in all other breeds. Furthermore, Chester White pigs had greater C16:0 content ($P < 0.01$) than did Yorkshire, Hampshire, Spotted, or Landrace pigs. The percentage of stearic acid (C18:0) was greatest in Duroc pigs ($P < 0.01$). In addition, Hampshire and Spotted pigs had lower C18:0 content ($P < 0.01$) than did pigs of all other breeds except Poland China ($P > 0.05$). Consequently, Duroc pigs had the greatest content of total SFA ($P < 0.01$), and Berkshire pigs had the second greatest concentration of total SFA ($P < 0.01$). Likewise, Cameron and Enser (1991) compared the fatty acid composition of LM of Duroc and Landrace pigs and found the SFA concentration was greater in Durocs.

Breed was a significant source of variation for both the thioesterase and elongase indices (Table 3). Landrace, Yorkshire, Hampshire, and Spotted pigs had a greater thioesterase index than Duroc and Chester White pigs ($P < 0.05$). Thioesterase in the fatty acid synthase complex is responsible for terminating the cycles of fatty acid synthesis and release of the newly synthesized fatty acid. Both C14-acyl ACP and C16-acyl ACP are substrates for thioesterase, even though C16:0 is the major product. The ratio of C16:0 to C14:0 was utilized to reflect the selective cleavage of thioesterase on C14-acyl ACP or C16-acyl ACP; the greater the thioesterase index, the less cleavage of C14-acyl ACP. The elongase index was greater ($P < 0.05$) in Landrace, Yorkshire, and Duroc than in the other breeds. The elongation of fatty acids occurs in the mitochondria and microsomal membranes, but the predominant site for elongation is the endoplasmic reticulum membrane. Generally, the mitochondrial elongation system uses fatty acyl CoA substrates in the range of C10-C14, whereas microsomal elongases act on C16 and longer fatty acids (Harwood, 1994). In the current study, the

Downloaded from www.journalofanimalscience.org at Serials Acquisitions Dept on February 21, 2014

Table 2. Effects of breeds from the National Barrow Show Sire Progeny Test Program on fatty acid composition, total lipid content, and fatty acid indices of LM[1]

Item	Breed							
	Duroc	Chester White	Berkshire	Poland China	Spotted	Yorkshire	Landrace	Hampshire
Fatty acid[2]								
14:0	10.46 ± 0.02[a]	10.39 ± 0.03[a,b]	10.38 ± 0.03[b]	10.25 ± 0.03[c]	10.21 ± 0.03[c]	10.24 ± 0.02[c]	10.20 ± 0.03[c]	10.18 ± 0.03[c]
16:0	260.01 ± 0.13[a]	250.19 ± 0.17[b]	260.01 ± 0.15[a]	240.76 ± 0.18[b,c]	240.52 ± 0.15[c,d]	240.62 ± 0.11[c]	240.53 ± 0.16[c,d]	240.27 ± 0.15[d]
16:1 n-7	40.03 ± 0.05[b]	40.08 ± 0.07[b]	40.38 ± 0.06[a]	40.11 ± 0.07[b]	40.05 ± 0.06[b]	30.76 ± 0.05[c]	30.80 ± 0.06[c]	40.14 ± 0.06[b]
18:0	130.04 ± 0.09[a]	120.14 ± 0.13[c,d]	120.29 ± 0.11[b,c,d]	110.98 ± 0.13[d,e]	110.69 ± 0.12[e]	120.47 ± 0.09[b]	120.40 ± 0.12[b,c]	110.70 ± 0.11[e]
18:1 n-9	450.86 ± 0.23[c]	460.41 ± 0.32[b,c]	440.85 ± 0.28[d]	470.48 ± 0.34[a]	460.97 ± 0.29[a,b]	440.81 ± 0.21[d]	440.91 ± 0.31[d]	440.87 ± 0.28[d]
18:2 n-6	70.85 ± 0.20[e]	80.65 ± 0.27[c,d]	80.76 ± 0.23[c,d]	80.38 ± 0.28[d,e]	90.19 ± 0.24[c]	100.30 ± 0.18[b]	100.23 ± 0.26[b]	110.07 ± 0.24[a]
20:4 n-6	10.76 ± 0.08[d]	20.13 ± 0.11[b,c]	20.34 ± 0.09[b]	20.03 ± 0.11[c,d]	20.36 ± 0.10[b]	20.80 ± 0.07[a]	20.93 ± 0.10[a]	20.77 ± 0.09[a]
Total SFA	400.50 ± 0.17[a]	380.73 ± 0.23[c,d]	390.68 ± 0.20[b]	370.99 ± 0.25[e,f]	370.42 ± 0.21[f,g]	380.33 ± 0.16[d,e]	380.13 ± 0.22[d,e]	370.16 ± 0.21[g]
Total MUFA	490.89 ± 0.25[c,d]	500.48 ± 0.34[b,c]	490.22 ± 0.29[d,e]	510.60 ± 0.36[a]	510.02 ± 0.31[a,b]	480.57 ± 0.23[e]	480.70 ± 0.33[e]	490.00 ± 0.30[e]
Total PUFA	90.61 ± 0.27[e]	100.79 ± 0.36[c,d]	110.10 ± 0.32[c,d]	100.41 ± 0.38[d,e]	110.55 ± 0.33[c]	130.10 ± 0.25[b]	130.16 ± 0.35[a,b]	130.84 ± 0.32[a]
Total lipids[3]	30.73 ± 0.09[a]	20.76 ± 00.12[c]	30.05 ± 00.10[b]	20.84 ± 00.13[b,c]	20.67 ± 00.11[c]	20.06 ± 00.08[d]	20.17 ± 00.12[d]	20.23 ± 00.11[d]
P:S[4]	0.24 ± 0.01[e]	0.28 ± 0.01[d]	0.28 ± 0.01[d]	0.28 ± 0.01[d]	0.31 ± 0.01[c]	0.35 ± 0.01[b]	0.35 ± 0.01[b]	0.37 ± 0.01[a]
IA[5]	0.54 ± 0.02[a]	0.50 ± 0.02[c]	0.52 ± 0.02[b]	0.48 ± 0.02[d]	0.47 ± 0.02[d,e]	0.48 ± 0.01[d]	0.47 ± 0.02[d]	0.46 ± 0.02[e]
Δ^9-desaturase (16) index[6]	130.42 ± 0.14[c]	130.93 ± 0.19[b]	140.43 ± 0.16[a]	140.24 ± 0.20[a,b]	140.18 ± 0.17[a,b]	130.26 ± 0.13[c]	130.36 ± 0.18[c]	140.57 ± 0.16[a]
Δ^9-desaturase (18) index[7]	770.85 ± 0.18[d]	790.24 ± 0.24[b]	780.49 ± 0.21[c]	790.84 ± 0.25[a,b]	800.05 ± 0.22[a]	780.24 ± 0.16[c,d]	780.33 ± 0.23[c,d]	790.29 ± 0.21[b]
Δ^9-desaturase (16+18) index[8]	560.08 ± 0.18[e]	570.46 ± 0.25[b,c]	560.22 ± 0.22[d,e]	580.42 ± 0.26[a]	580.45 ± 0.23[a]	560.69 ± 0.17[d]	560.83 ± 0.24[c,d]	570.61 ± 0.22[b]
Thioesterase index[9]	180.47 ± 0.35[c]	180.63 ± 0.48[c]	190.35 ± 0.41[b,c]	200.18 ± 0.50[a,b]	210.00 ± 0.43[a]	200.89 ± 0.32[a]	210.29 ± 0.46[a]	210.07 ± 0.42[a]
Elongase index[10]	0.50 ± 0.004[a]	0.48 ± 0.006[b]	0.48 ± 0.005[b]	0.49 ± 0.006[b]	0.48 ± 0.005[b]	0.51 ± 0.004[a]	0.51 ± 0.005[a]	0.49 ± 0.005[b]

[a–g]Values in the same row with different superscripts differ at $P < 0.05$.
[1]Values are expressed as least squares means ± SE.
[2]Fatty acid values are g/100 g of total lipids.
[3]Total lipids are g/100 g of muscle.
[4]The ratio of total PUFA to total SFA.
[5]Index of atherogenicity, calculated as $(4 \times 14{:}0 + 16{:}0)/(\Sigma\ \text{MUFA} + \Sigma\ \text{PUFA})$.
[6]Calculated as $100 \times [16{:}1\text{n-}9/(16{:}1\text{n-}9 + 16{:}0)]$.
[7]Calculated as $100 \times [18{:}1\text{n-}9/(18{:}1\text{n-}9 + 18{:}0)]$.
[8]Calculated as $100 \times [(16{:}1\text{n-}9 + 18{:}1\text{n-}9)/(16{:}1\text{n-}9 + 16{:}0 + 18{:}1\text{n-}9 + 18{:}0)]$.
[9]Calculated as 16:0/14:0.
[10]Calculated as 18:0/16:0.

Downloaded from www.journalofanimalscience.org at Serials Acquisitions Dept on February 21, 2014

Table 3. Effects of sex on fatty acid composition, total lipid content, and fatty acid indices of LM[1]

Item	Sex		Significance
	Barrows	Gilts	
Fatty acid[2]			
14:0	1.33 ± 0.01	1.25 ± 0.01	**
16:0	25.41 ± 0.07	24.56 ± 0.08	**
16:1 n-7	4.13 ± 0.03	3.96 ± 0.03	**
18:0	12.36 ± 0.06	12.06 ± 0.06	**
18:1 n-9	46.19 ± 0.14	45.35 ± 0.14	**
18:2 n-6	8.55 ± 0.11	10.06 ± 0.12	**
20:4 n-6	2.03 ± 0.05	2.75 ± 0.05	**
Total SFA	39.11 ± 0.10	37.87 ± 0.11	**
Total MUFA	50.31 ± 0.14	49.31 ± 0.15	**
Total PUFA	10.58 ± 0.15	12.81 ± 0.16	**
Total lipids[3]	3.03 ± 0.05	2.35 ± 0.05	**
P:S[4]	0.27 ± 0.00	0.34 ± 0.00	**
IA[5]	0.51 ± 0.01	0.48 ± 0.01	**
Δ^9-desaturase (C16) index[6]	13.96 ± 0.08	13.22 ± 0.09	NS
Δ^9-desaturase (C18) index[7]	73.86 ± 0.10	78.97 ± 0.11	NS
Δ^9-desaturase (C16+C18) index[8]	57.09 ± 0.11	57.35 ± 0.11	NS
Thioesterase index[9]	19.76 ± 0.21	20.46 ± 0.22	NS
Elongase index[10]	0.49 ± 0.002	0.49 ± 0.003	NS

[1]Values are expressed as least squares means ± SE.
[2]Fatty acid values are g/100 g of total lipids.
[3]Total lipids are g/100 g of muscle.
[4]The ratio of total PUFA to total SFA.
[5]Index of atherogenicity, calculated as (4 × 14:0 + 16:0)/(Σ MUFA +Σ PUFA).
[6]Calculated as 100 × [16:1n-9/(16:1n-9 + 16:0)].
[7]Calculated as 100 × [18:1n-9/(18:1n-9 + 18:0)].
[8]Calculated as 100 × [(16:1n-9 + 18:1n-9)/(16:1n-9 + 16:0 + 18:1n-9 + 18:0)].
[9]Calculated as 16:0/14:0.
[10]Calculated as 18:0/16:0.
NS = no significant difference ($P > 0.05$); ** $P < 0.01$.

ratio of C18:0 to C16:0 was used to represent the enzyme activity of elongase on C16:0.

Stearoyl-CoA desaturase (**SCD**), or Δ^9-desaturase, catalyzes the conversion of C16:0 and C18:0 to C16:1 and C18:1, the 2 major MUFA of pork lipids (Warnants et al., 1996). Kouba et al. (2003) reported that a decrease in total MUFA in pigs fed crushed linseed for 60 d could be attributed to decreased SCD activity. Some studies on beef cattle also suggested that the elevated concentration of C16:1 between breeds could be attributed to increased SCD activity (Sturdivant et al., 1992; Laborde et al., 2001). Therefore, 3 different ratios were used for the indices of SCD activity (Table 2). Greater index values mean greater desaturase activity. The Δ^9-desaturase (C16) index, which is an indicator of the SCD influence on the conversion of C16:0 to C16:1, was lower ($P < 0.05$) in Duroc, Yorkshire, and Landrace pigs than in all other breeds (Table 2). In addition, the Δ9-desaturase (16) index in Chester White pigs was lower ($P < 0.05$) than that in Hampshire and Berkshire pigs. Similarly, the Δ^9-desaturase (C18) index, which is an indicator of the influence of SCD on the conversion of C18:0 to C18:1, was lower in Duroc, Yorkshire, and Landrace pigs ($P < 0.01$) than in Hampshire, Spotted, Chester White, and Poland China pigs (Table 2). Furthermore, Spotted pigs had a greater Δ^9-desaturase (18) index ($P < 0.01$) than did Hampshire and Chester White pigs (Table 2). Because the concentration of C18:1 was much greater than that of C16:1, the orders of the values of the Δ^9-desaturase (18) index were similar to those of the Δ^9-desaturase (C16+C18) index, which is an indicator of the Δ^9-desaturase activity on the conversion of C16:0 and C18:0 to C16:1 and C18:1.

Berkshire pigs had the greatest percentage of C16:1, whereas Yorkshire and Landrace had the lowest concentrations of C16:1 ($P < 0.05$; Table 2). Oleic acid (C18:1) content was greater ($P < 0.01$) in Poland China, Spotted, Chester White, and Duroc pigs than that in Berkshire, Yorkshire, Landrace, and Hampshire. In addition, Poland China pigs had greater concentration of C18:1 ($P < 0.05$) than did Chester White and Duroc pigs. Because C18:1 is a major component of the total lipid, approximately 45% of the total fatty acids, the trend for differences in total MUFA content was similar to that of C18:1. The concentration of total MUFA was greater in Poland China ($P < 0.05$) than in all other breeds except Spotted. Our results were consistent with those of Mason et al. (2005), who found that Landrace pigs had lower C16:1 and C18:1 in LM than did Duroc pigs. Loins from Duroc pigs had greater content of lipid than did loins from Landrace, Berkshire, and Hampshire pigs. The increase in IMF content is in large part attributed to the increase in neutral lipid content, but not phospholipid (**PL**) content (Fernandez et al., 1999;

Downloaded from www.journalofanimalscience.org at Serials Acquisitions Dept on February 21, 2014

Wood et al., 2004). Consequently, the percentage of triacylglycerol (**TAG**) in total lipid tended to be greater with increasing IMF content. Oleic acid makes up approximately 40% of total fatty acid content of TAG, whereas it makes up only about 10% of total fatty acid content of PL (Wood et al., 2004). It is possible that the greater content of TAG in LM of Duroc pigs can be attributed, at least in part, to the greater concentration of C18:1 in total lipid of Duroc pigs compared with Landrace, Berkshire, and Hampshire pigs.

The concentration of linoleic acid (C18:2) was greatest in Hampshire pigs ($P < 0.05$; Table 2). Yorkshire and Landrace pigs had the second greatest ($P < 0.01$) content of C18:2. The content of C18:2 in Duroc pigs was lower ($P < 0.01$) than that of all other breeds except Poland China ($P > 0.05$; Table 2). Linoleic acid cannot be synthesized in vivo in pigs and, therefore, exclusively reflects dietary intake. The concentration of C18:2 was negatively correlated with the lipid content with an overall correlation of –0.68 ($P < 0.01$). This correlation occurs because pigs with less IMF content tend to have lower de novo fatty acid synthesis and, consequently, have greater concentration of dietary fatty acids incorporated into their tissues (Wood et al., 1989). In the current study, pigs from breeds with less lipid content in LM, such as Hampshire, Yorkshire, and Landrace, had greater concentration of C18:2, whereas pigs from breeds with greater lipid content in LM had lower concentration of C18:2 (Table 2). These results indicate that the differences in C18:2 content observed in the current study may be the result of differences in lipid content of LM among breeds, which might be attributed further to differences in amount of de novo fatty acid synthesis among breeds. Similarly, arachidonic acid (C20:4) and total PUFA concentrations were greater ($P < 0.01$) in Hampshire, Yorkshire, and Landrace than in the other breeds (Table 2). These results were consistent with those of Cameron and Enser (1991), which showed that Duroc pigs had greater concentration of total SFA and MUFA and lower content of PUFA in lipid of their LM than did Landrace pigs.

Of the SFA, including C14:0, C16:0, and C18:0, dietary C14:0 is considered to have the most harmful cardiovascular effect on humans, with almost 4 times the effect of C16:0 (Hegsted et al., 1965; Keys et al., 1974), whereas C18:0 is believed to be neutral in effect (Bonanome and Grundy, 1988). The ratio of total PUFA to SFA (**P:S ratio**) has been widely used as a lipid quality indicator in relation to atherogenicity. However, the IA developed by Ulbricht and Southgate (1991) is considered to be a better atherogenicity measure of dietary lipids because it includes MUFA and places more weight on C14:0 (Kinsella et al., 1990; Ulbricht and Southgate, 1991). In the current study, Hampshire pigs had the greatest and Landrace and Yorkshire had the second greatest P:S ratio (Table 2), whereas the P:S ratio for the Duroc pigs was the lowest. The IA of LM from Hampshire pigs (IA = 0.46) was lower ($P < 0.05$) than that of all other breeds except the Spotted. In contrast, Duroc pigs had the greatest IA, which was 17% greater than that of Hampshire pigs ($P < 0.05$). Chester White, Berkshire, and Poland China pigs had the same P:S ratio, but their IA differed significantly from each other (Table 2). The P:S ratio of Spotted pigs was lower than that of Yorkshire, Landrace, and Hampshire pigs, but the IA of Spotted pigs did not differ from that of these 3 breeds (Table 2). Leszczynski et al. (1992) found that the P:S ratio of lipid in LM increased 25 or 54% in the pigs fed a 10 or 20% extruded full-fat soybean (**FFS**) diet for 6 wk compared with pigs fed a corn-soybean meal diet. However, the atherogenic indices calculated from their data were 0.48, 0.46, and 0.48 for 10% FFS, 20% FFS, and control groups, respectively, which were very similar. These results suggest that the assessment of lipid quality depends on the selection of indicators.

Sex Effects

Sex was a highly significant source of variation for the percentage of each individual fatty acid (Table 3). The concentrations of C14:0, C16:0, C18:0, C16:1, and C18:1 were greater ($P < 0.01$) in barrows than in gilts. In contrast, the percentages of 18:2 and 20:4 were greater ($P < 0.01$) in gilts than in barrows. Consequently, barrows had greater ($P < 0.01$) total SFA and MUFA than gilts, whereas gilts had 21% more ($P < 0.01$) total PUFA than barrows (Table 3). The P:S ratio was 26% greater ($P < 0.01$) in gilts than in barrows. Gilts also had a smaller ($P < 0.01$) IA than barrows. The indices of Δ^9-desaturase, thioesterase, and elongase did not differ between the 2 sex groups ($P < 0.05$), indicating similar enzyme activities in barrows and gilts. The effects of sex were different in the 8 breeds, as indicated by significant breed × sex interactions for the percentages of C16:0, C16:1, C18:0, C18:2, and C20:4, total SFA, MUFA, and PUFA, IA, and P:S ratio.

The sex effect results from the current study were consistent with those of Nürnberg et al. (2005), who reported that barrows had greater SFA and lower PUFA than gilts in total lipid of LM. A previous study of the fatty acid composition of longissimus muscle showed significant effects of sex on several fatty acids (Leszczynski et al., 1992). Warnants et al. (1999) reported that barrows had greater concentration of C16:0, C18:0, and total SFA in backfat and greater content of C16:0 in the total lipid of IMF than gilts. In agreement with previous studies (Leszczynski et al., 1992; Warnants et al., 1996), barrows have greater IMF content than gilts. The PL content of LM in barrows is similar to that of gilts, and the difference in IMF content between barrows and gilts is likely attributable to the difference in the content of TAG (Warnants et al., 1996). The IMF of barrows, therefore, contains more TAG than that of gilts. Because the fatty acid composition of TAG differs greatly from that of PL, the differences in fatty acid composition of LM between gilts and barrows observed

Downloaded from www.journalofanimalscience.org at Serials Acquisitions Dept on February 21, 2014

Table 4. Effects of halothane genotype on fatty acid composition, total lipid content, and fatty acid indices of LM[1]

Trait	Genotype		Significance
	HAL-1843 normal (NN)	HAL-1843 carrier (Nn)	
Fatty acid[2]			
14:0	1.30 ± 0.02	1.27 ± 0.02	NS
16:0	25.15 ± 0.06	24.83 ± 0.11	**
16:1 *n*-7	4.07 ± 0.02	4.02 ± 0.05	NS
18:0	12.23 ± 0.04	12.20 ± 0.09	NS
18:1 *n*-9	46.19 ± 0.11	45.35 ± 0.22	**
18:2 *n*-6	8.87 ± 0.09	9.74 ± 0.18	**
20:4 *n*-6	2.19 ± 0.04	2.59 ± 0.07	**
Total SFA	38.68 ± 0.08	38.30 ± 0.16	*
Total MUFA	50.26 ± 0.11	49.37 ± 0.23	**
Total PUFA	11.06 ± 0.12	12.33 ± 0.24	**
Total lipids[3]	2.88 ± 0.04	2.50 ± 0.08	**
P:S[4]	0.29 ± 0.003	0.33 ± 0.007	**
IA[5]	0.50 ± 0.001	0.49 ± 0.003	**
Δ^9-desaturase (C16) index[6]	13.92 ± 0.06	13.92 ± 0.13	NS
Δ^9-desaturase (C18) index[7]	79.04 ± 0.08	78.79 ± 0.16	NS
Δ^9-desaturase (C16+C18) index[8]	57.33 ± 0.08	57.11 ± 0.17	NS
ΔThioesterase index[9]	19.93 ± 0.16	20.29 ± 0.33	NS
ΔElongase index[10]	0.49 ± 0.002	0.49 ± 0.004	NS

[1]Values are expressed as least squares means ± SE.
[2]Fatty acid values are g/100 g of total lipids.
[3]Total lipids are g/100 g of muscle.
[4]The ratio of total PUFA to total SFA.
[5]Index of atherogenicity, calculated as $(4 \times 14{:}0 + 16{:}0)/(\Sigma \text{ MUFA} + \Sigma \text{ PUFA})$.
[6]Calculated as 100 × [16:1n-9/(16:1n-9 + 16:0)].
[7]Calculated as 100 × [18:1n-9/(18:1n-9 + 18:0)].
[8]Calculated as 100 × [(16:1n-9 + 18:1n-9)/(16:1n-9 + 16:0 + 18:1n-9 + 18:0)].
[9]Calculated as 16:0/14:0.
[10]Calculated as 18:0/16:0.
NS = no significant difference ($P > 0.05$); ** $P < 0.01$; * $P < 0.05$.

in the current study are in part the result of differences in TAG content.

Halothane Genotype Effects

Table 4 shows the fatty acid composition of LM by halothane genotype. All 3 HAL-1843 genotypes, NN, Nn, and HAL-1843 positive (nn), were detected in the test pigs. However, there were so few nn pigs that they were dropped from the current study. Halothane genotype was a significant source of variation for the percentages of some, but not all, fatty acids. Pigs with NN genotype had more 16:0 and 18:1 and less 18:2 and 20:4 than did Nn pigs ($P < 0.01$; Table 4). Consequently, the total SFA and MUFA concentrations and IA were greater ($P < 0.05$ and $P < 0.01$, respectively) in NN pigs, whereas the total PUFA concentration and P:S ratio were less ($P < 0.01$) in Nn pigs. No significant difference in the indices of Δ^9 desaturase, thioesterase, and elongase were observed between the 2 HAL 1843 genotypes, indicating similar enzyme activities. Our results were contrary to those of Piedrafita et al. (2001), which suggested that HAL 1843 genotype had significant effects on the percentages of several fatty acids in adipose tissue of NN, Nn, and HAL 1843 positive (nn) pigs, but the mean values for NN and Nn pigs were very similar in their study. Pigs with the NN genotype have greater marbling than pigs with nn (Stalder et al., 1998; Piedrafita et al., 2001) and Nn (Hamilton et al., 2000; Maddock et al., 2002). Our data supported these results by showing that the lipid content was greater ($P < 0.01$) in NN than in Nn pigs (Table 4).

Relationship Between Lipid Content and Fatty Acid Composition

Table 5 shows the Pearson's correlation and the residual correlation coefficients between lipid content and fatty acid composition. The content of IMF was negatively correlated with C18:2 and C20:4 percentages and positively correlated with the percentages of the other fatty acids, except C18:0, based on the residual correlation coefficients ($P < 0.01$; Table 5). Similarly, Suzuki et al. (2003) reported a significant positive partial correlation between IMF content and percentages of C14:0, C16:0, C18:0, and C18:1 and a strong negative correlation between lipid content and C18:2 percentage (r = –0.87). Cameron and Enser (1991) showed that the concentrations of SFA and MUFA tended to increase, whereas the concentration of PUFA tended to decrease, with increasing IMF content. A similar tendency was found in the current study. The residual correlations

Downloaded from www.journalofanimalscience.org at Serials Acquisitions Dept on February 21, 2014

Table 5. Pearson's correlation[1] and residual correlation[2] coefficients between lipid content and fatty acid composition of LM from the National Barrow Show Sire Progeny Test Program[3]

	Lipid content	14:0	16:0	16:1 n-7	18:0	18:1 n-9	18:2 n-6	20:4 n-6	P:S[4]	IA[5]
Lipid content		0.38	0.40	0.29	0.18	0.44	–0.68	–0.63	–0.64	0.43
14:0	0.27		0.40	0.27		0.05	–0.35	–0.35	–0.35	0.63
16:0	0.37	0.43		0.19		–0.13	–0.43	–0.50	–0.51	0.93
16:1 n-7	0.21	0.35	0.28		–0.20	0.27	–0.43	–0.37	–0.38	0.18
18:0			0.12	–0.48		–0.10	–0.27	–0.18	–0.28	0.14
18:1 n-9	0.43		–0.12	0.21	–0.30		–0.72	–0.65	–0.63	–0.17
18:2 n-6	–0.60	–0.34	–0.47	–0.36	–0.07	–0.74		0.84	0.97	–0.45
20:4 n-6	–0.57	–0.32	–0.47	–0.31		–0.67	0.81		0.90	–0.48
P:S[4]	–0.58	–0.38	–0.58	–0.33	–0.17	–0.64	0.97	0.89		–0.57
IA[5]	0.38	0.69	0.92	0.25	0.27	–0.15	–0.48	–0.47	–0.59	

[1]Upper diagonal.
[2]Lower diagonal; calculated from a multivariate, fixed linear model with fixed effects of breed, sex, halothane genotype, and the interaction between breed and sex.
[3]Values different from zero ($P < 0.05$) are presented.
[4]The ratio of total PUFA to total SFA.
[5]Index of atherogenicity, calculated as $(4 \times 14{:}0 + 16{:}0)/(\Sigma\text{MUFA} + \Sigma\text{PUFA})$.

of C18:1 with C18:2 and C20:4 were highly negative (r = –0.74 and –0.67, respectively) and were almost the same as Pearson's correlations (Table 5). Malmfors et al. (1978) hypothesized that the concentration of C18:1 in subcutaneous adipose tissue of pigs decreases in response to an increase in the content of C18:2 to regulate the degree of saturation of subcutaneous lipids.

The correlation between lipid content and P:S ratio was moderate (r = –0.58), whereas the correlation between lipid content and IA was weak (r = 0.38) based on residual correlation coefficients (Table 5). The P:S ratio was highly correlated with C18:2 (r = 0.97) and C20:4 (r = 0.89; Table 5). The correlation between P:S ratio and C14:0, however, was weak (r = –0.38; Table 5). Moreover, P:S ratio had a strong negative correlation with C18:1 (r = –0.64; Table 5), which is considered to have beneficial effects on health (Rudel et al., 1995). As stated before, C18:1 had strong negative correlations with C18:2 and C20:4, whereas P:S ratio had high positive correlations with C18:2 and C20:4, which resulted in a strong negative correlation between C18:1 and P:S ratio. In contrast, IA had strong positive correlations with C16:0 and C14:0 (r = 0.92 and 0.69, respectively) and moderate negative correlations with C18:2 and C20:4 (r = –0.48 and –0.47, respectively; Table 5). Similar results were obtained using Pearson correlation coefficients. Previous research findings have shown that health conscious consumers tend to select a fat-modified rather than a low-fat diet (Thorogood et al., 1990). Questions have arisen regarding which term is the most suitable measure of the atherogenicity of a diet. Our results suggest that P:S ratio and IA provide different healthfulness assessments of dietary lipids, as indicated by their different correlations with individual fatty acids.

IMPLICATIONS

Per capita pork consumption in the United States decreased by 10% between 1960 and 2003, according to the United States Department of Agriculture, Economic Research Service. Health concerns over fat intake, especially saturated fatty acids, is one of the factors contributing to this decline. Significant breed differences in fatty acid composition of longissimus muscle were observed in the current study. These differences may be sufficient to be exploited through selection to design a more healthful pork product for nutrition-conscious consumers.

LITERATURE CITED

Bonanome, A., and S. Grundy. 1988. Effect of dietary stearic acid on plasma cholesterol and lipoprotein concentrations. N. Engl. J. Med. 318:1244–1248.

Bronte-Stewart, B., A. Antonis, L. Eales, and J. F. Brock. 1956. Effects of feeding different fats on serum-cholesterol level. Lancet 270:521–526.

Cameron, N. D., and M. B. Enser. 1991. Fatty acid composition of lipids in LM muscle of Duroc and British Landrace pigs and its relationship with eating quality. Meat Sci. 29:295–307.

DeSmet, S., K. Raes, and D. Demeyer. 2004. Meat fatty acid composition as affected by fatness and genetic factors: A review. Anim. Res. 53:81–98.

DeVol, D. L., F. K. McKeith, P. J. Bechtel, J. E. Novakofski, R. D. Shanks, and T. R. Carr. 1988. Variation in composition and palatability traits and relationships between muscle characteristics and palatability in a random sample of pork carcasses. J. Anim. Sci. 66:385–395.

Fernandez, X., G. Monin, A. Talmant, J. Mourot, and B. Lebret. 1999. Influence of intramuscular fat content on the quality of pig meat - 1. Composition of the lipid fraction and sensory characteristics of m. longissimus lumborum. Meat Sci. 53:59–65.

Folch, J. M., M. Lees, and G. H. Sloane Stanley. 1957. A simple method for the isolation and purification of total lipids from animal tissue. J. Biol. Chem. 226:497–509.

Downloaded from www.journalofanimalscience.org at Serials Acquisitions Dept on February 21, 2014

Fujii, J., K. Otzu, F. Zorzato, S. de Leon, V. K. Khanna, J. E. Weiler, P. J. O'Brien, and D. H. MacLennan. 1991. Identification of a mutation in porcine ryanodine receptor associated with malignant hyperthermia. Science 253:448–451.

Gatlin, A. L., M. T. See, J. A. Hansen, D. Sutton, and J. Odle. 2002. The effects of dietary fat sources, concentrations, and feeding intervals on pork fatty acid composition. J. Anim. Sci. 80:1606–1615.

Hamilton, D. N., M. Ellis, K. D. Miller, F. K. McKeith, and D. F. Parrett. 2000. The effect of halothane and rendement Napole gene on carcass and meat quality characteristics of pigs. J. Anim. Sci. 78:2862–2867.

Harwood, J. K. 1994. Lipid Metabolism. Pages 605–664 in The Lipid Handbook. 2nd ed. Chapman & Hall, London, UK.

Hegsted, D. M., R. B. McGandy, M. L. Myers, and F. J. Stare. 1965. Quantitative effects of dietary fat on serum cholesterol in man. Am. J. Clin. Nutr. 17:281–295.

Keys, A., F. Grande, and J. T. Anderson. 1974. Bias and misrepresentation revisited— "Perspective" in saturated fat. Am. J. Clin. Nutr. 27:188–212.

Kinsella, J. E., B. Lokesh, and R. A. Stone. 1990. Dietary n-3 polyunsaturated fatty acids and amelioration of cardiovascular disease: Possible mechanism. Am. J. Clin. Nutr. 52:1–28.

Kouba, M., M. Enser, F. M. Whittington, G. R. Nute, and J. D. Wood. 2003. Effect of a high-linolenic acid diet on lipogenic enzyme activities, fatty acid composition, and meat quality in the growing pig. J. Anim. Sci. 81:1967–1979.

Laborde, F. L., I. B. Mandell, J. J. Tosh, J. W. Wilton, and J. G. Buchanan-Smith. 2001. Breed effects on growth performance, carcass characteristics, fatty acid composition, and palatability attributes in finishing steers. J. Anim. Sci. 79:355–365.

Lepage, G., and C. C. Roy. 1986. Direct transesterification of all classes of lipids in a one-step reaction. J. Lipid Res. 27:114–120.

Leszczynski, D. E., J. Pikul, R. A. Easter, F. K. McKeith, D. G. McLaren, J. Novakofski, P. J. Bechtel, and D. E. Jewell. 1992. Characterization of lipid in loin and bacon from finishing pigs fed full-fat soybeans or tallow. J. Anim. Sci. 70:2175–2181.

Lo, L. L., D. G. McLaren, F. K. McKeith, R. L. Fernando, and J. Novakofski. 1992. Genetic analyses of growth, real-time ultrasound, carcass, and pork quality traits in Duroc and Landrace pigs: I. Breed effects. J. Anim. Sci. 70:2373–2386.

LoFiego, D. P., P. Santoro, P. Macchioni, and E. DeLeonibus. 2005. Influence of genetic type, live weight at slaughter and carcass fatness on fatty acid composition of subcutaneous adipose tissue of raw ham in the heavy pig. Meat Sci. 69:107–114.

Maddock, R. J., B. S. Bidner, S. N. Carr, F. K. McKeith, E. P. Berg, and J. W. Savell. 2002. Creatine monohydrate supplementation and the quality of fresh pork in normal and halothane carrier pigs. J. Anim. Sci. 80:997–1004.

Malmfors, B., K. Lundstrom, and I. Hanson. 1978. Fatty acid composition of porcine backfat and muscle lipids as affected by sex, weight and anatomical location. Swedish J. Agric. Res. 8:25–38.

Mason, L. M., S. A. Hogan, A. Lynch, K. O'Sullivan, P. G. Lawlor, and J. P. Kerry. 2005. Effects of restricted feeding and antioxidant supplementation on pig performance and quality characteristics of LM muscle from Landrace and Duroc pigs. Meat Sci. 70:307–317.

Newcom, D. W., K. J. Stalder, T. J. Bass, R. N. Goodwin, F. C. Parrish, and B. R. Wiegand. 2004. Breed differences and genetic parameters of myoglobin concentration in porcine longissimus muscle. J. Anim. Sci. 82:2264–2268.

NRC. 1988. Nutrient Requirements of Swine. 9th ed. Natl. Acad. Press, Washington, DC.

NRC. 1998. Nutrient Requirements of Swine 10th ed. Natl. Acad. Press, Washington, DC.

Nürnberg, K., K. Fischer, G. Nürnberg, U. Kuechenmeister, D. Klosowska, G. Eliminowska-Wenda, I. Fiedler, and K. Ender. 2005. Effects of dietary olive and linseed oil on lipid composition, meat quality, sensory characteristics and muscle structure in pigs. Meat Sci. 70:63–74.

Okada, T., N. Furuhashi, Y. Kuromori, M. Miyashita, F. Iwata, and K. Harada. 2005. Plasma palmitoleic acid content and obesity in children. Am. J. Clin. Nutr. 82:747–750.

Pan, D. A., S. Lillioja, M. R. Milner, A. D. Kriketos, L. A. Baur, C. Bogardus, and L. H. Storlien. 1995. Skeletal muscle membrane lipid composition is related to adiposity and insulin action. J. Clin. Invest. 96:2802–2808.

Piedrafita, J., L. L. Christian, and S. M. Lonergan. 2001. Fatty acid profiles in three stress genotypes of swine and relationship with performance, carcass, and meat quality traits. Meat Sci. 57:71–77.

Rudel, L. L., J. S. Park, and J. K. Sawyer. 1995. Compared with dietary monounsaturated and saturated fat, polyunsaturated fat protects African green monkeys from coronary artery atherosclerosis. Arterioscler. Thromb. Vasc. Biol. 15:2101–2110.

Stalder, K. J., J. Maya, L. L. Christian, S. J. Moeller, and K. J. Prusa. 1998. Effects of preslaughter management on the quality of carcasses from porcine stress syndrome heterozygous market hogs. J. Anim. Sci. 76:2435–2443.

Sturdivant, C. A., D. K. Lunt, G. C. Smith, and S. B. Smith. 1992. Fatty acid composition of subcutaneous and intramuscular adipose tissues and M. LM of Wagyu cattle. Meat Sci. 32:449–458.

Suzuki, K., T. Shibata, H. Kadowaki, H. Abe, and T. Toyoshima. 2003. Meat quality comparison of Berkshire, Duroc and crossbred pigs sired by Berkshire and Duroc. Meat Sci. 64:35–42.

Thorogood, M., L. Roe, K. McPherson, and J. Mann. 1990. Dietary intake and plasma lipid levels: Lessons from a study of the diet of health conscious groups. BMJ 300:1297–1301.

Ulbricht, T. L. V., and D. A. T. Southgate. 1991. Coronary heart disease: Seven dietary factors. Lancet 338:985–992.

Warnants, N., M. J. Van Oeckel, and C. V. Boucqué. 1996. Incorporation of dietary polyunsaturated fatty acids in pork fatty tissues and its implication for the quality of the end products. Meat Sci. 44:125–144.

Warnants, N., M. J. Van Oeckel, and C. V. Boucqué. 1999. Incorporation of dietary polyunsaturated fatty acids into pork fatty tissues. J. Anim. Sci. 77:2478–2490.

Wood, J. D., M. Enser, F. M. Whittington, C. B. Moncrieff, and A. J. Kempster. 1989. Backfat composition in pigs: Differences between fat thickness groups and sexes. Livest. Prod. Sci. 22:351–362.

Wood, J. D., G. R. Nute, R. I. Richardson, F. M. Whittington, O. Southwood, G. Plastow, R. Mansbridge, N. da Costa, and K. C. Chang. 2004. Effects of breed, diet and muscle on fat deposition and eating quality in pigs. Meat Sci. 67:651–667.

Woollett, L. A., D. K. Spady, and J. M. Dietschy. 1992. Saturated and unsaturated fatty acid independently regulate low density lipoprotein receptor activity and production rate. J. Lipid Res. 33:77–88.

Downloaded from www.journalofanimalscience.org at Serials Acquisitions Dept on February 21, 2014

Related Articles A related article has been published:
http://www.journalofanimalscience.org/content/animalsci/85/5/1351.full.html

Supplementary Material Supplementary material can be found at:
http://www.journalofanimalscience.org/content/suppl/2007/03/20/jas.2006-239.DC1.html

References This article cites 38 articles, 19 of which you can access for free at:
http://www.journalofanimalscience.org/content/85/3/583#BIBL

Citations This article has been cited by 7 HighWire-hosted articles:
http://www.journalofanimalscience.org/content/85/3/583#otherarticles

Errata An erratum has been published regarding this article. Please see next page or:
http://www.journalofanimalscience.orghttp://www.journalofanimalscience.org/content/85/5/1351.full.pdf

Downloaded from www.journalofanimalscience.org at Serials Acquisitions Dept on February 21, 2014

Erratum

In the published manuscript, Effects of breed, sex, and halothane genotype on fatty acid composition of pork longissimus muscle (J. Anim. Sci. 2007, 85:583–591), the numbers in the body of the table had the decimal place incorrect, and also had incorrect fatty acid designations in footnotes 6 and 8. The correct table is provided below.

Table 2. Effects of breeds from the National Barrow Show Sire Progeny Test Program on fatty acid composition, total lipid content, and fatty acid indices of LM[1]

	Breed							
Item	Duroc	Chester White	Berkshire	Poland China	Spotted	Yorkshire	Landrace	Hampshire
Fatty acid[2]								
14:0	1.46 ± 0.02a	1.39 ± 0.03[a,b]	1.38 ± 0.03[b]	1.25 ± 0.03[c]	1.21 ± 0.03[c]	1.24 ± 0.02[c]	1.20 ± 0.03[c]	1.18 ± 0.03[c]
16:0	26.01 ± 0.13[a]	25.19 ± 0.17[b]	26.01 ± 0.15[a]	24.76 ± 0.18[b,c]	24.52 ± 0.15[c,d]	24.62 ± 0.11[c]	24.53 ± 0.16[c,d]	24.27 ± 0.15[d]
16:1 *n*-7	4.03 ± 0.05[b]	4.08 ± 0.07[b]	4.38 ± 0.06[a]	4.11 ± 0.07[b]	4.05 ± 0.06[b]	3.76 ± 0.05[c]	3.80 ± 0.06[c]	4.14 ± 0.06[b]
18:0	13.04 ± 0.09[a]	12.14 ± 0.13[c,d]	12.29 ± 0.11[b,c,d]	11.98 ± 0.13[d,e]	11.69 ± 0.12[e]	12.47 ± 0.09[b]	12.40 ± 0.12[b,c]	11.70 ± 0.11[e]
18:1 *n*-9	45.86 ± 0.23[c]	46.41 ± 0.32[b,c]	44.85 ± 0.28[d]	47.48 ± 0.34[a]	46.97 ± 0.29[a,b]	44.81 ± 0.21[e]	44.91 ± 0.31[e]	44.87 ± 0.28[e]
18:2 *n*-6	7.85 ± 0.20[e]	8.65 ± 0.27[c,d]	8.76 ± 0.23[c,d]	8.38 ± 0.28[d,e]	9.19 ± 0.24[c]	10.30 ± 0.18[b]	10.23 ± 0.26[b]	11.07 ± 0.24[a]
20:4 *n*-6	1.76 ± 0.08[d]	2.13 ± 0.11[b,c]	2.34 ± 0.09[b]	2.03 ± 0.11[c,d]	2.36 ± 0.10[b]	2.80 ± 0.07[a]	2.93 ± 0.10[a]	2.77 ± 0.09[a]
Total SFA	40.50 ± 0.17[a]	38.73 ± 0.23[c,d]	39.68 ± 0.20[b]	37.99 ± 0.25[e,f]	37.42 ± 0.21[f,g]	38.33 ± 0.16[d,e]	38.13 ± 0.22[d,e]	37.16 ± 0.21[g]
Total MUFA	49.89 ± 0.25[c,d]	50.48 ± 0.34[b,c]	49.22 ± 0.29[d,e]	51.60 ± 0.36[a]	51.02 ± 0.31[a,b]	48.57 ± 0.23[e]	48.70 ± 0.33[e]	49.00 ± 0.30[e]
Total PUFA	9.61 ± 0.27[e]	10.79 ± 0.36[c,d]	11.10 ± 0.32[c,d]	10.41 ± 0.38[d,e]	11.55 ± 0.33[c]	13.10 ± 0.25[b]	13.16 ± 0.35[a]	13.84 ± 0.32[a]
Total lipids[3]	3.73 ± 0.09[a]	2.76 ± 0.12[c]	3.05 ± 0.10[b]	2.84 ± 0.13[b,c]	2.67 ± 0.11[c]	2.06 ± 0.08[d]	2.17 ± 0.12[d]	2.23 ± 0.11[d]
P:S[4]	0.24 ± 0.01[e]	0.28 ± 0.01[d]	0.28 ± 0.01[d]	0.28 ± 0.01[d]	0.31 ± 0.01[c]	0.35 ± 0.01[b]	0.35 ± 0.01[b]	0.37 ± 0.01[a]
AI[5]	0.54 ± 0.02[a]	0.50 ± 0.02[c]	0.52 ± 0.02[b]	0.48 ± 0.02[d]	0.47 ± 0.02[d,e]	0.48 ± 0.01[d]	0.47 ± 0.02[d]	0.46 ± 0.02[e]
Δ^9-desaturase (16) index[6]	13.42 ± 0.14[c]	13.93 ± 0.19[b]	14.43 ± 0.16[a]	14.24 ± 0.20[a,b]	14.18 ± 0.17[a,b]	13.26 ± 0.13[c]	13.36 ± 0.18[c]	14.57 ± 0.16[a]
Δ^9-desaturase (18) index[7]	77.85 ± 0.18[d]	79.24 ± 0.24[b]	78.49 ± 0.21[c]	79.84 ± 0.25[a,b]	80.05 ± 0.22[a]	78.24 ± 0.16[c,d]	78.33 ± 0.23[c,d]	79.29 ± 0.21[b]
Δ^9-desaturase (16+18) index[8]	56.08 ± 0.18[e]	57.46 ± 0.25[b,c]	56.22 ± 0.22[d,e]	58.42 ± 0.26[a]	58.45 ± 0.23[a]	56.69 ± 0.17[d]	56.83 ± 0.24[c,d]	57.61 ± 0.22[b]
Thioesterase index[9]	18.47 ± 0.35[c]	18.63 ± 0.48[c]	19.35 ± 0.41[b,c]	20.18 ± 0.50[a,b]	21.00 ± 0.43[a]	20.89 ± 0.32[a]	21.29 ± 0.46[a]	21.07 ± 0.42[a]
Elongase index[10]	0.50 ± 0.004[a]	0.48 ± 0.006[b]	0.48 ± 0.005[b]	0.49 ± 0.006[b]	0.48 ± 0.005[b]	0.51 ± 0.004[a]	0.51 ± 0.005[a]	0.49 ± 0.005[b]

[a–g]Values in the same row with different subscripts differ at $P < 0.05$.
[1]Values are expressed as least squares means ± SE.
[2]Fatty acid values are g/100 g of total lipids.
[3]Total lipids are g/100 g of muscle.
[4]The ratio of total PUFA to total SFA.
[5]Index of atherogenicity, calculated as $(4 \times 14{:}0 + 16{:}0)/(\Sigma\text{MUFA} + \Sigma\text{PUFA})$.
[6]Calculated as 100 × [16:1*n*-7/(16:1*n*-7 + 16:0)].
[7]Calculated as 100 × [18:1*n*-9/(18:1*n*-9 + 18:0)].
[8]Calculated as 100 × [(16:1*n*-7 + 18:1*n*-9)/(16:1*n*-7 + 16:0 + 18:1*n*-9 + 18:0)].
[9]Calculated as 16:0/14:0.
[10]Calculated as 18:0/16:0.

Additional corrections to the manuscript include the following (indicated in **bold**):

On pg. 585, the sentence should read “Breed was a significant source of variation for both the thioesterase and elongase indicies (**Table 2**).”;

On pg. 588, the sentence should read “The indicies of Δ^9-desaturase, thioesterase, and elongase did not differ between the 2 sex groups (***P* > 0.05**), indicating similar enzyme activities in barrows and gilts.”;

On pg. 589, the sentence should read “Consequently, the total SFA and MUFA concentrations and IA were greater ($P < 0.05$ and $P < 0.01$, respectively) in NN pigs, whereas the total PUFA concentration and P:S ration were **greater** ($P < 0.01$) in Nn pigs.”; and

In Table 3, footnote 6 should read “Calculated as 100 × [16:1n-**7**/(16:1n-**7** + 16:0)].” and footnote 8 should read “Calculated as 100 × [16:1n-**7** + 18:1n-9/(16:1n-**7** + 16:0 + 18:1n-9 + 18:0.”