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J ANIM SCI 2013, 91:4043-4050.
doi: 10.2527/jas.2013-6255 originally published online August 13, 2013

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Genome-wide association of meat quality traits and tenderness in swine[1,2]

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ABSTRACT: Pork quality has a large impact on consumer preference and perception of eating quality. A genome-wide association was performed for pork quality traits [intramuscular fat (IMF)], slice shear force (SSF), color attributes, purge, cooking loss, and pH] from 531 to 1,237 records on barrows and gilts of a Landrace-Duroc-Yorkshire population using the Illumina PorcineSNP60 BeadChip. Associations were detected using MTDFREML for all traits. Intramuscular fat had the greatest number of SNP associations, followed by pH, purge, cooking loss, shear force, and color. Two regions contained associations for multiple traits; one on SSC1 at 255 Mb near calcineurin subunit B (*PPP3R2*) was associated with SSF, moisture loss, and pH, and one on SSC6 from 28 to 29.5 Mb for purge and IMF containing the candidate genes glucose-6-phosphate isomerase (*GPI*) and *KCTD15*. Some of the other compelling candidate genes in regions associated with meat quality include *CEBPA*, *SNAI1*, and *FAM132A* for IMF, *CAPN1* for SSF, *GLUL* for pH, and *PRKAG3* and *ITGB1* with cooking loss.

Key words: association, pork quality, single nucleotide polymorphism, swine, tenderness

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doi:10.2527/jas2013-6255

INTRODUCTION

Considerable variation exists in meat quality traits of pork (Moeller et al., 2010). Consumer perception of pork eating quality is largely driven by tenderness and sensory juiciness scores, which are related to shear force, cooking loss, and ultimate pH (Huff-Lonergan et al., 2002; Moeller et al., 2010). Water loss from meat during postmortem storage reduces profitability and consumer appeal. Other measures of pork quality, such as color and intramuscular fat (**IMF**), also affect consumer satisfaction ratings to a lesser extent but may influence initial purchase decisions. Factors like genetics, cooking temperature, and packing plant stunning and chilling procedures also affect meat quality and tenderness (Crawford et al., 2010; Shackelford et al., 2012). Although several candidate genes have been proposed for pork tenderness through QTL studies, only a few causative genes or quantitative trait nucleotide (**QTN**) have been identified. The most characterized gene affecting pork tenderness is calpastatin (Ciobanu et al., 2004; Meyers and Beever, 2008; Nonneman et al., 2011) and markers in calpastatin have been validated in commercial populations (Nonneman et al., 2011; Rohrer et al., 2012). Mutations affecting glycogen content in *PRKAG3* (Milan et al., 2000) affect pH, cooking loss, and color (Ciobanu et al., 2001; Choi et al., 2012; Rohrer et al., 2012), and the *RYR1* R615C mutation has large effects on drip loss (Otto et al., 2007), shear force (Rohrer et al., 2012), pH (Cherel et al., 2012), and color (Leach et al., 1996).

Numerous QTL studies have been reported for pork quality using divergent founding breeds and F2

[1]Mention of trade names or commercial products in this publication is solely for the purpose of providing specific information and does not imply recommendation or endorsement by USDA.

[2]The authors thank Patty Beska, Kathy Mihm, Pat Tammen, Dave Kohmetscher, Kenny Ostdiek, Ron Harris, Kris Simmerman, and Sue Hauver of the U.S. Meat Animal Research Center for technical assistance, and Linda Parnell for manuscript preparation.

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Received January 9, 2013.
Accepted May 16, 2013.

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or backcross populations, but limited studies have been done using high density SNP arrays in commercial-type pedigreed animals. This study was done to identify additional genes affecting economically important meat quality traits.

MATERIALS AND METHODS

All animal procedures were reviewed and approved by the U.S. Meat Animal Research Center Animal Care and Use Committee, and procedures for handling pigs complied with those specified in the Guide for the Care and Use of Agricultural Animals in Agricultural Research and Teaching (FASS, 2010).

The animals were a multigenerational Landrace-Duroc-Yorkshire composite population developed at the U.S. Meat Animal Research Center (**USMARC**), as described before (Lindholm-Perry et al., 2009). The phenotyped animals included 2005-born gilts (n = 532) and 2008- and 2009-born barrows (n = 346) and gilts (n = 364) from the fourth, seventh, and eighth filial generations, respectively. All of the 2005-born animals were harvested at the USMARC abattoir and one-half of the 2008- and 2009-born animals were harvested at a commercial processing plant, and phenotypes were collected as described before (Shackelford et al., 2004; Lindholm-Perry et al., 2009). Using conventional processing schedules for the plants, carcasses were cut at ~20 to 22 h postmortem and loins were boned shortly thereafter. The boneless loin was obtained from the left side of each carcass. Boneless loins were vacuum packaged, boxed, and transported at –2.8°C to USMARC (if harvested at a commercial abattoir). At 14 d postmortem, two 2.54-cm-thick chops were obtained from the 11th rib region of each loin and cooked (71°C) with a belt grill, and LM slice shear force (**SSF**) was measured on each of the 2 chops (Shackelford et al., 2004). The duplicate SSF values were averaged and that value was used for all analyses. A 2.54-cm-thick chop was obtained from the 14th rib region and within 1 min of cutting, color was assessed objectively on that chop, using a colorimeter (Minolta ColorTec PCM; color-tec.com, Clinton, NJ) and then ultimate pH was measured. That chop was vacuum packaged, frozen, and stored for assessment of IMF content. Subsequently, IMF chops were thawed and LM was trimmed free of surface fat and epimysium, and pulverized with a food processor. The pulverized IMF sample was divided approximately in half and duplicate random samples (~70 g each) were taken, wrapped in cheesecloth, and frozen at –30°C. Moisture content was determined after samples were thawed and subjected to oven drying at 100°C for 24 h and total lipids were obtained on dried samples by diethyl ether extraction (AOAC, 1985).

Table 1. Summary statistics of phenotypic traits measured on porcine LM and number of animals genotyped with the Illumina PorcineSNP60 BeadChip

Trait[1]	No. records	Mean	SD
SSF, kg	1,237	13.79	3.43
IMF, %	1,237	2.28	1.05
Cooking loss, %	1,237	21.18	3.06
Purge, %	675	2.95	1.26
pH	531	5.81	0.17
Color L*	706	56.14	3.75
Color a*	706	6.75	1.43
Color b*	706	12.92	1.68

[1]SSF = slice shear force; IMF = intramuscular fat; pH = ultimate pH at 24 h.

DNA Isolation, SNP Array Genotyping, and Quality Control

Genomic DNA was extracted from the frozen tail of each pig using the Wizard SV Genomic DNA Purification kit (Promega, Madison, WI). The DNA samples of 200 to 300 ng at a concentration of 75ng/µL were genotyped using the Illumina Porcine SNP60 BeadChip containing 64,232 SNP (Illumina, San Diego, CA; Ramos et al., 2009). The number of phenotyped animals that were genotyped for each trait is shown in Table 1. Genotyping was performed at the USDA-ARS Bovine Functional Genomics Laboratory (Beltsville, MD) and scan results interpreted at USMARC, using the GenCall Data Analysis software of Illumina. Genotypes were called for 59,895 SNP spanning the entire porcine genome.

The SNP with call rates ≤80% or minor allele frequencies ≤0.05 were excluded from the data set. After using these quality control measures, 52,336 SNP out of 64,232 SNP qualified for association analyses.

Marker positions were determined by alignments of marker sequences to Sscrofa builds 9.2 and 10.2 to maximize the number of markers mapped. Candidate genes were identified from build 9.2 using the University of California Santa Cruz (**UCSC**) Genome Browser (http://genome.ucsc.edu) and from build 10.2 using Map Viewer (www.ncbi.nlm.nih.gov/projects/mapview).

Statistical Analysis

Initial analyses on phenotypic data were conducted using WOMBAT (Meyer, 2007) to develop statistical models and estimate variance components of random effects for marker association analyses. WOMBAT was selected for variance component estimation because it implements average information, as well as other REML algorithms, in addition to the derivative free simplex algorithm. For genome-wide association analyses, an animal model was applied with fixed effects of gender, age at slaughter, slaughter group, and abattoir. Pedigree information for all phenotyped animals was traced back to

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Table 2. Marker associations with slice shear force of porcine LM

SNP_ID	SSC 9.2[1]	Position 9.2	SSC 10.2[1]	Position 10.2	ss#[2]	rs#[2]	Freq-B[3]	Effect[4]	*P*-value	Candidate gene	Nearest gene
MARC0060815	1	255,063,294	1	272,001,095	107840831	80859304	0.4217	1.2504	2.19E-05	*PPP3R2*	in *GRIN3A*
H3GA0005672	2	5,049,456	2	5,903,796	131185965	81357491	0.8079	-0.9191	3.64E-05	*CAPN1*	in *RELA*
MARC0055360	2	74,021,690			107832822	81245547	0.8981	1.1893	3.17E-05	*IQGAP2*	in *SV2C*
ALGA0111274	9	1,865,461	9	2,957,048	131108082	81339684	0.6705	1.0019	4.84E-06	*ZNF215/OVCH2*	in *SYT9*

[1]SSC 9.2 and SSC 10.2 refer to chromosome of the respective Sus scrofa genome build.

[2]Submitted SNP ID (ss#) and reference SNP ID (rs#) accession numbers in GenBank dbSNP.

[3]Frequency of the B allele as defined by Illumina (San Diego, CA).

[4]Estimated phenotypic effect (kg) of 1 copy of the B allele as defined by Illumina (San Diego, CA).

founders of the population. Linear marker effects, based on number of copies of the B allele, were estimated for each marker in separate analyses. Resulting marker effects and standard error estimates were used to compute t-statistics and nominal and Bonferroni-corrected *P*-values. A test between Multi Trait Derivative Free REML (**MTDFREML**; Boldman et al., 1995) and WOMBAT programs indicated that MTDFREML required less time to conduct the genome-wide association study (**GWAS**) analyses. The MTDFREML (Boldman et al., 1995) analyses used the variance components estimated for additive genetic and residual effects from initial analyses. Additive genetic and residual variance components were held constant when estimating individual marker effects. Re-estimating variance components for every marker would needlessly add time and noise to the analyses if the variance explained by individual markers is too small for detection of differences in additive genetic variance. A Bonferroni correction factor for multiple testing requires a nominal *P*-value of 1.0×10^{-6} to reach genome-wide significance of $P < 0.05$. An approximation of chromosome-wise significance reported, or suggestive associations, is 5.0×10^{-5}.

The population studied does not possess the deleterious 1843T allele for ryanodine receptor 1 (**RYR1**; Fujii et al., 1991), nor does it have the Rendement Napole (**RN**) mutant allele discovered by Milan et al. (2000) in 5'-AMP-activated protein kinase subunit gamma-3 (**PRKAG3**; 200Q allele). As we have previously reported an effect of calpastatin (**CAST**) markers on tenderness in this population (Nonneman et al., 2011), CAST genotypes were fit as a fixed effect for SSF.

A principal component analysis of the genotypic data was conducted to determine if population stratification existed in the data set. However, there was no evidence of population stratification among animals included in this analysis.

RESULTS AND DISCUSSION

Summary statistics of phenotypic traits measured are shown in Table 1. Several regions were identified for associations with meat quality traits. Intramuscular fat had the greatest number of associations, followed by pH, purge, cooking loss, shear force, and color.

Associations with Slice Shear Force

Four regions were significantly associated with SSF (Table 2). On SCC1 at 255 Mb an SNP in *GRIN3A* was associated with SSF (the estimated effect was 1.25 kg per copy of the B allele). The single exon for calcineurin subunit B (*PPP3R2*) is located within the *GRIN3A* gene. Calcineurin has been shown to be involved in skeletal muscle differentiation, regeneration, and muscle fiber conversion (Mallinson et al., 2009). One SNP, H3GA0005672, is located in transcription factor p65 (*RELA)* at 5 MB on SSC2, ~200 kb upstream from μ-calpain (*CAPN1)*. Markers in the proteolytic enzyme μ-calpain have been known to be predictive of tenderness in beef breeds (Page et al., 2002; McClure et al., 2012). μ-Calpain activity is also associated with tenderness in pork (Melody et al., 2004). Another SNP, MARC0055360, on SSC2 at 74 Mb is located near *SV2C* adjacent to *IQGAP2*, which interacts at the cytoskeleton with calmodulin for cellular signaling (Atcheson et al., 2011). Although SNP in calpastatin at 92 Mb on SSC2 have previously been shown to be associated with SSF (Nonneman et al., 2011), this region was not detected in this study. Five markers on the Illumina PorcineSNP60 BeadChip are located in calpastatin; however, 1 marker was uninformative in this population and the other 4 were not in linkage disequilibrium with published markers associated with tenderness. The most significant association was found on SSC9 at 1.8 Mb in synaptotagmin-9 (***SYT9***), near *ZNF215*, an imprinted gene which is associated with carcass weight in cattle (Magee et al., 2010).

Associations with Intramuscular Fat

Thirty-four SNP in 15 regions were associated with IMF (Table 3). Candidate genes were identified based on their involvement in promotion or regulation of adipogenesis or lipid synthesis. Several candidate genes reside on chromosome 3 from 102 to 106 Mb. Lysocardiolipin acyl transferase (*LCLAT1*) on SSC3 at 101.9 Mb is a microsomal enzyme involved in phospholipid remodeling

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Table 3. Marker associations with intramuscular fat content (IMF) of porcine LM

SNP_ID	SSC 9.2[1]	Position 9.2	SSC 10.2[1]	Position 10.2	ss#[2]	rs#[2]	Freq-B[3]	Effect[4]	*P*-value	Candidate gene	Nearest gene
ASGA0011102	2	94,873,644	2	109,699,598	131196703	81362116	0.2884	–0.3948	3.38E-05	*LOC100133050*	near *CHD1*
ASGA0015930	3	102,284,233	3	115,785,486	131227204	81375549	0.6390	0.2571	5.82E-06	*LCLAT1*	*LCLAT1*
MARC0006904	3	102,943,060	3	116,501,233	107828571	81242774	0.9628	–0.4718	4.83E-05	*LCLAT1*	*ALK*
ASGA0016042	3	~103,000,000	3	118,779,014	131228033	81375924	0.8013	–0.2485	1.46E-05	*PPP1CB/FOSL2*	*ALK*
ALGA0021085	3	105,591,226	3	119,979,845	131229325	81376507	0.7636	–0.2319	3.71E-05	*ADCY3*	*ASXL2*
INRA0014565	4	68,083,732	4	71,524,893			0.8041	–0.2347	3.3E-05	*NCOA2*	*SULF1*
MARC0030811	4	68,200,145			107800789	80850929	0.8044	–0.23386	3.47E-05	*NCOA2*	*C8orf34*
MARC0000845	4	82,163,706	4	86,747,423	107896525	80903322	0.7767	0.237532	1.65E-05	*SNAI2*	*SNAI2*
ASGA0020519	4	87,256,381	4	91,648,161	131271402		0.6253	0.235847	3.54E-05		*GPA33*
DIAS0003235	4	100,662,825	4	105,800,661			0.7269	–0.25084	8.23E-06		*LCE1E*
ASGA0021274	4	101,189,230	4	106,399,494	131276796		0.6918	–0.24114	4.24E-05	*RORC*	*RORC*
ASGA0021291	4	101,386,442	4	106,628,995	131276886		0.6723	–0.24677	2.86E-05		*SNX27*
H3GA0013675	4	101,451,421	4	106,788,826	131276903	80905326	0.6512	–0.27628	4.14E-06		*TNRC4*
ALGA0027174	4	101,663,974	4	106,858,461	131276960	80950440	0.6923	–0.2408	4.51E-05		in *CGN*
H3GA0013787	4	104,986,280			131277947	80997112	0.5857	–0.25403	3.01E-05		*PDE4DIP*
ASGA0021414	4	105,026,563	4	110,832,479	131277965		0.5783	–0.25096	3.43E-05		*SEC22B*
MARC0006175	6	29,549,244			107830472	81244026	0.8262	0.257989	2.32E-05	*KCTD15*	*UBA2*
MARC0072609	6	29,898,357	6	39,960,475	107853780	81258771	0.7982	0.254881	1.43E-05	*FFAR3*	*ZNF792*
M1GA0026195	6	42,855,840	6	58,080,576	131042301	81310348	0.6635	0.221719	4.89E-05	*FAM132A*	*TTLL10*
INRA0023564	7	11,302,669	7	11,485,232			0.4111	0.332002	3.63E-06		*GMPR*
DRGA0007577	7	52,235,348	7	51,608,179			0.5709	–0.24597	3.76E-05	*TFAP2B*	*TFAP2B*
DRGA0007578	7	52,256,047	7	51,628,878			0.5679	–0.24368	4.5E-05	*TFAP2B*	*TFAP2B*
M1GA0025365			8	76,669,178	131076611	81326148	0.3537	0.355766	2.58E-05		*SHROOM3*
ASGA0095609			13	15,785,723	131052517	81315056	0.7614	0.254795	3.69E-05		*SLC4A7*
ASGA0079000	18	13,261,264	18	15,942,579	131559489	81471911	0.1311	1.087998	1.52E-05	*SLC35B4*	*SLC35B4*
ALGA0097534	18	22,662,098	18	26,106,000	131554971	81467895	0.8788	0.30069	6.87E-06		in *CADPS2*
ASGA0079288	18	22,683,172	18	26,127,074	131554975	81467899	0.8788	0.30069	6.87E-06		in *CADPS2*
MARC0054855	18	22,750,576	18	26,194,478	107832209	81245135	0.7739	0.25873	4.77E-06		in *CADPS2*
ALGA0097562	18	23,040,335	18	26,484,237	131555057	81467972	0.7917	0.23867	4.6E-05		*CADPS2*
ALGA0097568	18	23,180,952	18	26,627,380	131555076	81467987	0.9304	0.40121	1.43E-06	*AASS*	in *AASS*
ASGA0079341	18	23,507,864	18	26,954,292	131555137	81468038	0.9304	0.403939	1.21E-06		*PTPRZ1*
ALGA0097602	18	23,628,773	18	27,075,206	131555154	81468053	0.8100	0.280188	2.48E-06		*FAM3C*
ALGA0097617	18	23,905,840	18	27,352,273	131555192	81468086	0.7716	0.242451	1.64E-05		in *CPED1*
ASGA0079377	18	24,070,999	18	27,517,432	131555217	81468108	0.9286	0.356461	1.09E-05		in *CPED1*

[1]SSC 9.2 and SSC 10.2 refer to chromosome of the respective Sus scrofa genome build.

[2]Submitted SNP ID (ss#) and reference SNP ID (rs#) accession numbers in GenBank dbSNP.

[3]Frequency of the B allele as defined by Illumina (San Diego, CA).

[4]Estimated phenotypic effect (%) of 1 copy of the B allele as defined by Illumina.

(Zhao et al., 2009). The transcription factor *FOSL2* at 103 Mb regulates leptin expression in adipocytes (Wrann et al., 2012), additional sex combs like 2 (*ASXL2*) at 105.5 Mb, which enhances adipogenesis by increasing expression of adipogenic genes through modulation of *PPARG* (Park et al., 2011), *RAB10* at 105.4 Mb which participates in *GLUT4* translocation in fat and muscle cells (Sano et al., 2008) and adenylate cyclase 3 (*ADCY3*) at 106 Mb which is associated with body mass index in humans (Wen et al., 2012). Mice deficient in *ADCY3* are obese due to adipocyte hypertrophy, hyperplasia and reduced activity (Wang et al., 2009). Eleven markers in 5 regions on SSC4 were associated with IMF. Two markers located near *NCOA2* at 68 Mb and a marker near snail homolog 2 (*SNAI2*) at 82 Mb were associated with IMF. *NCOA2* expression is correlated with IMF in porcine *longissimus* muscle (Wang et al., 2008) and mice deficient in *SNAI2* have reduced adipose tissue mass (Pérez-Mancera et al., 2007). Five markers on SSC4 from 100.6 to 101.6 Mb in high linkage disequilibrium (LD; r^2 range 0.67- 0.99) with each other were associated with IMF and the most likely candidate in this region is retinoic acid receptor (**RAR**) -related orphan receptor C (*RORC*) which has been associated with IMF in cattle (Barendse et al., 2010). Two markers at 105 Mb in vesicle-trafficking protein *SEC22B*, which is a component of the very low-density lipoprotein (**VLDL**) transport vesicle (Rahim et al., 2012), were associated with IMF. Two regions on SSC6 were identified near *UBA2* at 29.5 Mb and *ZNF792* at 29.9 Mb and another 35 kb away

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Table 4. Marker associations with objective Minolta color scores of porcine LM

Trait	SNP_ID	SSC 9.2[1]	Position 9.2	SSC 10.2[1]	Position 10.2	ss#[2]	rs#[2]	Freq-B[3]	Effect[4]	*P*-value	Candidate Gene	Nearest Gene
L*	ALGA0000698	1	8,936,120	1	9,651,655	131174661	80985205	0.7373	1.2023	1.078E-05	*SOD2*	*SOD2*
L*	MARC0099191	2	13,947,868	2	17,186,566			0.2271	–2.3124	4.581E-05	*F2*	*F2*
b*	ALGA0041291	7	51,133,715	7	50,327,355	131351290	80790299	0.4845	–0.4435	2.458E-05	*RHAG*	in *CRISP1*
L*	H3GA0024941	8	59,113,450	8	73,841,435	131370826	81401075	0.9383	2.0840	1.297E-05	*AFM*	*AFM*
b*	H3GA0024941	8	59,113,450	8	73,841,435	131370826	81401075	0.9383	0.6396	6.126E-05	*AFM*	*AFM*

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[2]Submitted SNP ID (ss#) and reference SNP ID (rs#) accession numbers in GenBank dbSNP.

[3]Frequency of the B allele as defined by Illumina (San Diego, CA).

[4]Estimated phenotypic effect of 1 copy of the B allele as defined by Illumina (San Diego, CA).

from *FAM132A* at 42.8 Mb, an insulin-sensitizing adipokine (Wei et al., 2012), that were associated with IMF. Chromosome 18 had 9 markers in high linkage disequilibrium within 1.4 Mb (22.6–24) that were associated with IMF surrounding the mitochondrial aminoadipate-semialdehyde synthase gene (*AASS*) that catalyzes lysine degradation. Nutritionally limiting lysine in swine diets can increase IMF accumulation in porcine muscle (Gondret and Lebret, 2002; Katsumata, 2011).

Associations with Color Attributes

Three associations for L* color and 1 association for b* color were found; no significant associations were detected for a* color (Table 4). The most significant association for L* was found on SSC1 at 8.9 Mb, 100 kb distal to the *SOD2* gene. *SOD2* is a mitochondrial, manganese binding superoxide dismutase converting superoxide anion to oxygen and hydrogen peroxide and inhibits oxidative inactivation of nitric oxide, thereby regulating mitochondrial oxygen consumption (Fukai and Ushio-Fukai, 2011), which can affect meat color (Mancini and Hunt, 2005). A significant association for L* was found on SSC2 at 13.9 Mb in the coagulation factor II (thrombin; *F2*) gene. Apoptosis of muscle postmortem would result in an inversion of membrane phospholipids, activation of thrombin and a change in membrane conductance (Ouali et al., 2006). A significant association for both b* and L* was found on SSC8 at 59 Mb about 10 kb distal to afamin (*AFM*). Afamin is a member of the albumin gene family localized in tandem with albumin (*ALB*) and α fetoprotein (*AFP*) and is a specific binding and transport protein for vitamin E (Voegele et al., 2002). Vitamin E reduces lipid peroxidation and numerous studies have shown that feeding exogenous vitamin E results in accumulation of α-tocopherol in muscle and improved meat color and increased color stability of beef, pork and lamb (Dikeman, 2007). The only association for b* was on SSC7 at 51.1 Mb between *CRISP1* and *PGK2*. The nearest candidate for affecting color is *RHAG* about 200 kb upstream, a red cell antigen and ammonium and carbon dioxide transporter.

Associations with Cooking Loss, pH, and Purge

Twelve markers in 9 regions were associated with cooking loss (Table 5). Two of these (ASGA0006484 and DRGA0005843) had allele frequencies of 6 to 10% and large estimated effects. Marker ASGA0006484 lies near the calcineurin B subunit (*PPP3R2*) gene on SSC1 at 255 Mb and was also associated with pH. An adjacent SNP in this same region was also associated with SSF (above). Marker DRGA0005843 is on SSC5 between ethanolamine kinase 1 and *SOX5* at 48.8 Mb. Two SNP associated with cooking loss on SSC6 at 57.8 Mb are both located in the tumor suppressor *ARID1A* gene near *PIGV*, a glycosylphosphatidylinositol mannosyltransferase of glycosylphosphatidylinositol that anchors proteins to the cell membrane. A marker 50 kb from integrin β-1 (*ITGB1*) on SSC10 at 53 Mb was associated with cooking loss. Integrin β-1 is involved in the adhesion of the cell membrane and cytoskeleton, and degradation of integrin β-1 is correlated with the formation of drip channels in pork (Lawson, 2004). Also associated with cooking loss were 2 SNP located 80 and 42 kb on either side of *PRKAG3* at 113.8 Mb on SSC15. The effects of *PRKAG3* variation on glycogen content, drip loss, cooking loss, pH, and pork quality have been well documented (Milan et al., 2000; Ciobanu et al., 2001; Otto et al., 2007; Rohrer et al., 2012).

Twelve markers were associated with pH (Table 5). Two markers on SSC2 at 46 Mb (MARC0029487 and MARC0039652) were in complete LD with each other and are near 2 candidate genes for pH: *MED16*, a coactivator of heat shock-specific transcription, and *ATP5D*, a mitochondrial ATP synthase D subunit. Further down SSC2, spanning from about 69 to 70.5 Mb, associations were found with 2 markers in strong LD, MARC0080822 and MARC0071942, near *COL23A1* at 69 Mb, a transmembrane collagen ligand for integrin β-1 found to be in a selection signature in beef cattle (Qanbari et al., 2011), and *HIGD2A* at 70.5 Mb, a hypoxia-induced mitochondrial protein involved in assembly of the respiratory complex. Two SNP on SSC9 at 116 and 116.4 Mb surrounding glutamate-ammonia ligase (***GLUL***) were associated with pH. The *GLUL*, also called glutamine synthase, plays an

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Table 5. Marker associations with measures of porcine LM pH, purge, and cooking loss (cookloss)

Trait	SNP_ID	SSC 9.2[1]	Position 9.2	SSC 10.2[1]	Position 10.2	ss#[2]	rs#[2]	Freq-B[3]	Effect[4]	*P*-value	Candidate Gene	Nearest Gene
Cookloss	ASGA0006484	1	255,150,921	1	272,280,326	131147796	80846194	0.0973	–3.3735	4.0527E-05	*PPP3R2*	in *LPPR1*
Cookloss	DRGA0005843	5	48,834,098	5	54,277,077			0.1070	–3.3631	1.4168E-05	*KCNJ8*	*ETNK1*
Cookloss	MARC0003453	6	57,758,165	6	77,637,493	107902356	81287462	0.7802	0.4972	3.0197E-05	*RPS6KA1*	in *ARID1A*
Cookloss	ALGA0105658	6	57,767,345	6	77,646,673	131092587	81333008	0.8412	0.5571	2.4187E-05	*RPS6KA1*	in *ARID1A*
Cookloss	ALGA0111085	6	59,909,655	6	81,180,148	131107592	81339481	0.4898	0.6804	2.6287E-06	*NKAIN1*	in *SDC3*
Cookloss	H3GA0053380			6	86,558,700	131113033	81341820	0.5330	–0.5652	4.6029E-05	*INPP5B*	*MTF1*
Cookloss	MARC0111479	8	2,195,668	8	2,615,211	107873907	81270877	0.3901	–0.7332	2.0274E-05		*JAKMIP1*
Cookloss	H3GA0053600	10	53,283,265	10	61,561,773	131119358	81344582	0.3615	0.7182	3.6157E-05	*ITGB1*	*ITGB1*
Cookloss	MARC0063636	14	81,808,031	14	84,774,622	107843548	80851246	0.1843	–1.3169	4.5741E-05	*KCNMA1*	in *C10orf11*
Cookloss	H3GA0041097	14	82,085,486	14	85,028,953	131518836	80962229	0.3031	–0.9246	1.1630E-05	*KCNMA1*	in *C10orf11*
Cookloss	ASGA0070623	15	113,767,195	15	133,493,709	131529089	80936921	0.8560	0.6023	2.5930E-06	*PRKAG3*	*PRKAG3*
Cookloss	DBUN0002708	15	113,892,876	15	133,836,471			0.6159	–0.5205	7.9212E-06	*PRKAG3*	*PRKAG3*
pH	ASGA0006484	1	255,150,921	1	272,280,326	131147796	80846194	0.0973	0.3966	5.5839E-06	*MURC*	in *LPPR1*
pH	MARC0029487	2	46,107,041	2	77,558,460	107801274	81224813	0.1644	0.3193	2.3526E-05	*MED16*	in *ARID3A*
pH	MARC0039652	2	~46,500,000	2	76,667,122	107812776	81232544	0.1644	0.3193	2.3526E-05	*ATP5D*	*ATP5D*
pH	MARC0080822	2	~69,000,000	2	81,125,411	107863592		0.2005	0.3113	3.2730E-05	*AGXT2L2*	*CLK4*
pH	MARC0071942	2	70,498,029	2	83,081,001	107852250	81257797	0.1659	0.3139	3.0758E-05		in *CLTB*
pH	ASGA0100307	3	88,814,365	3	101,510,973	131065211	81321027	0.4014	0.0933	1.8762E-05	*CAMKMT*	near *SIX2*
pH	ASGA0035707	7	108,914,547	7	106,906,152	131323201		0.6009	0.0674	3.7825E-05	*ADCK1*	in *ADCK1*
pH	MARC0085179	9	~700,000	9	1,121,393	107867928		0.8128	–0.0677	3.2209E-05	*IPO7*	in *TMEM41B*
pH	MARC0043119	9	~116,000,000	9	135,810,859	107817539	81235678	0.1181	0.4431	2.5963E-06	*GLUL*	*CACNA1E*
pH	ASGA0103785	9	116,442,036			131027773	81303620	0.4530	0.0888	3.5791E-05	*GLUL*	*RGS8*
pH	ALGA0110413	13	~3,400,000	13	4,156,953	131105637	80878114	0.9085	–0.0907	1.5271E-05	*OXNAD1*	in *TBC1D5*
pH	MARC0053561					107831194	81244501	0.1646	0.3193	2.3526E-05		
Purge	ALGA0001735	1	24,126,129	1	25,580,117	131141754	80991420	0.6852	–0.4160	4.2297E-05	*VTA1*	*VTA1*
Purge	DRGA0000284	1	25,188,087	1	26,745,940			0.6726	–0.4088	4.7804E-05	*VTA1*	*NMBR*
Purge	ALGA0104848	1	28,083,839			131090420	81332076	0.4619	–0.5338	4.1828E-05	*TNFAIP3*	*TNFAIP3*
Purge	MARC0038383	1	253,668,435	1	270,366,408	107811758	80995253	0.9289	–0.6613	2.3457E-05	*ERP44*	in *INVS*
Purge	ALGA0009136	1	263,494,521	1	281,551,428	131149226	80863627	0.4309	0.7772	1.2079E-09	*PALM2*	*PALM2*
Purge	ALGA0030881	5	15,701,269	5	17,611,490	131279562	81382780	0.3834	0.5129	3.6172E-05	in *SLC4A8*	in *SLC4A8*
Purge	ASGA0104230	6	~28,000,000			131566014	81475736	0.2637	1.0547	2.7381E-06	*PEPD*	*DPY19L3*
Purge	ASGA0028127	6	28,722,428	6	38,442,573	131319877	81395674	0.2625	0.8923	2.2404E-05		*SLC7A10*
Purge	MARC0045568	6	28,804,441			107820644	81237691	0.2609	1.0792	2.1313E-06		*CEBPA*
Purge	MARC0033885	6	28,906,397	6	38,728,802	107805474	81227680	0.2586	1.0776	2.2110E-06	*PEPD*	*CEBPG*
Purge	ASGA0103299	6	29,038,534			131566493	81476193	0.2632	1.0552	2.7164E-06	*RHPN2*	*CEBPG*
Purge	ASGA0097167	6	39,656,150	6	52,127,572	131566322	81476026	0.6156	0.4365	4.2071E-05		*ZNF615*
Purge	ASGA0089858					131037429	80998426	0.6854	–0.4213	3.5040E-05		

[1]SSC 9.2 and SSC 10.2 refer to chromosome of the respective Sus scrofa genome build.

[2]Submitted SNP ID (ss#) and reference SNP ID (rs#) accession numbers in GenBank dbSNP.

[3]Frequency of the B allele as defined by Illumina (San Diego, CA).

[4]Estimated phenotypic effect of 1 copy of the B allele as defined by Illumina. Purge and cooking loss (cookloss) are expressed as a percentage.

important role in controlling body pH and removing ammonia from circulation (King et al., 1983).

Thirteen markers in 7 genomic regions were associated with purge loss (Table 5). Of 4 regions found on SSC1, 1 SNP was in the *PALM2-AKAP2* read-through gene. *AKAP2* is involved in anchoring protein kinase A to the cytoskeleton and highly enriched in mitochondria (Wang et al., 2001). One maker located in the sodium bicarbonate cotransporter *SLC4A8* on SSC5 at 15.7 Mb was associated with purge loss. *SLC4A8* is expressed in heart, skeletal muscle, and kidney, and regulates intracellular pH (Loiselle et al., 2004). Ranging from the neutral AA transporter (*SLC7A10*) to peptidase D, 5 SNP in high linkage disequilibrium with each other on SSC6, spanning from ~28 to 29 MB, were associated with purge loss.

Results of the present association analyses identified are consistent with, and occur within, previously reported QTL regions (www.animalgenome.org/QTLdb); however, novel associations have also been identified. Associations for IMF on SSC3 (103 Mb), for SSF on SSC1 (255 Mb) and SSC2 (5 Mb), for color L* on SSC1 (9 Mb), SSC2 (13 Mb), and SSC8 (59 Mb) have not been reported in the pig QTL database. Novel associations were found for

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purge loss on SSC1, SSC5, and SSC6, as well as for pH on SSC1, SSC3, SSC7, and SSC13.

The proximity of significant SNP near known causative genes (i.e., *PRKAG3*) and compelling candidate genes would imply that the resolution for most QTL is quite good and causative variants are not a great distance away from associated SNP. One factor that likely contributes to the high mapping resolution is the depth of generations (4, 7, and 8 filial generations) from the time the population of phenotyped animals was developed. Over each generation, linkage disequilibrium that was initially created has eroded, allowing improved mapping resolution. A second factor is that we used animals from multiple generations so that the specific linkage disequilibrium of a certain set of sires did not dominate results of the present study.

Although many SNP associations identified reasonable candidate genes very close to an SNP, several regions did not. It is possible the linkage disequilibrium in particular genomic regions in this population extends some distance from the causative variation. Five SNP on SSC4 associated with IMF and 5 SNP on SSC6 associated with purge loss extended over 1 Mb and were in high LD with each other; therefore, the causative genes may be several hundred kilobases away from the most significant SNP. Candidate gene selection is difficult for meat quality traits, such as color and cooking loss, because a limited amount is known about biochemical changes that affect these traits, there is limited knowledge about function of genes in associated regions, there is currently a lack of detailed annotation, and there are discrepancies in different builds of the pig genome. Many of the factors affecting meat quality traits have not been studied in laboratory species and some mechanisms (i.e., IMF) tend to be species specific (Hocquette et al., 2010), or occur postmortem. The use of high-density genotyping platforms should provide greater resolution in QTL mapping and lead to identification of underlying genes.

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