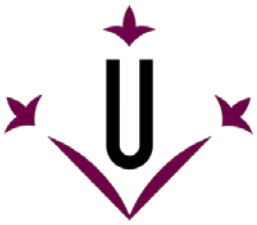

The effect of *SCD* and *LEPR* genetic polymorphisms on fat content and composition is maintained throughout fattening in Duroc pigs

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ABSTRACT

The effects of the stearoyl-CoA desaturase (*SCD; AY487830:g.2228T>C*) and leptin receptor (*LEPR; NM_001024587:g.1987C>T*) polymorphisms on fat content and fatty acid (FA) composition were investigated throughout fattening. Samples of *Longissimus thoracis* (LT) and subcutaneous fat (SF) from 214 Duroc barrows were collected from 160 days to slaughter age (220 days) using a longitudinal design. Results indicated that the positive effect of the T allele at the *SCD* gene on monounsaturated FA and of the T allele at the *LEPR* gene on saturated FA are maintained throughout the growing-finishing period, both in LT and SF. In *LEPR*, however, compositional changes, particularly in SF, are a result of increased fatness. There is very limited evidence of genotype by age interaction, and thus it is concluded that the combined selection for the *SCD* T and *LEPR* C alleles is a good strategy to increase the MUFA/SFA ratio regardless of the age at slaughter.

Key words: age; fatty acids; genetic marker; intramuscular fat; meat quality; pork.

Highlights:

- The effect of age on two SNPs affecting fat composition in Duroc pigs is examined.
- The SNP at the *SCD* gene increased monounsaturated fatty acid content.
- The SNP at the *LEPR* gene increased fatness and saturated fatty acid content.
- The effects of both SNPs are maintained throughout fattening.

1. Introduction

The pig industry mostly relates carcass quality to lean content and conformation. However, there is a constant increase of consumers who attach greater importance to pork quality. Meat quality is not straightforward to define (Wood et al., 2004) and depends on a number of meat attributes. Among them, intramuscular fat (IMF) content has a beneficial impact on tenderness, texture, taste and flavour intensity of pork, particularly for premium fresh pork niches and dry-cured products (Fernandez, Monin, Talmant, Mourot, & Lebret, 1999; Fortin, Robertson, & Tong, 2005; Jeleníková, Pipek, & Miyahara, 2008). Recently, mainly due to health promotion policies, the fatty acid (FA) composition has also entered as a new feature for pork quality. A dietary substitution of saturated fatty acids (SFA) for monounsaturated fatty acids (MUFA) may not only be beneficial against dyslipidemias (Gillingham, Harris-Janz, & Jones, 2011; Roche, 2001) but may also improve organoleptic properties and overall acceptability of pork (Cameron et al., 2000; Cameron & Enser, 1991; Tikk et al., 2007).

Due to the importance of fat content and composition for the meat industry, genes involved in lipid metabolism have been an important target of research in animal breeding. The leptin receptor (*LEPR*) and the stearoyl-CoA desaturase (*SCD*) are two of these genes. *LEPR*, as a mediator of the satiety effect of the leptin hormone, influences overall fatness (Houseknecht, Baile, Matteri, & Spurlock, 1998), while *SCD*, the rate-limiting enzyme required for the biosynthesis of MUFA from SFA, affects fatty acid composition (Ntambi & Miyazaki, 2004). In pigs, a non-synonymous exonic polymorphism in the *LEPR* gene has been reported to be strongly associated with fatness in an Iberian x Landrace (Óvilo et al., 2005) and in Duroc × Landrace/Large White (Galve et al., 2012) crossbreds. Similarly, a polymorphism has been reported in

the promoter region of the *SCD* gene affecting MUFA content in both IMF and subcutaneous fat (SF) of purebred and crossbred Duroc animals (Estany, Ros-Freixedes, Tor, & Pena, 2014; Henriquez-Rodriguez, Tor, Pena, & Estany, 2015). A recent genome-wide association study confirmed *SCD* and *LEPR* as the two main loci influencing IMF and FA composition in Duroc (Ros-Freixedes et al., 2016).

In a previous work, Bosch, Tor, Reixach, & Estany (2012) estimated the evolution of fat content and composition in both IMF and SF throughout the growing–finishing period in pigs from a Duroc line used for high-quality production. These authors showed that the age-related increase of IMF and SF is associated to modifications in the fatty acid profile, with major changes occurring in MUFA and PUFA. Therefore, the objective of this paper was to examine whether the effects of the *SCD* and *LEPR* polymorphisms on fat content and composition affect each other and/or change with age.

2. Material and methods

2.1. Animals and experimental procedures

All experimental procedures were approved by the Ethics Committee for Animal Experimentation of the University of Lleida (Agreement 2/01, March 2001) and all animal procedures and care performed in accordance with authorization AE2374 issued by the Catalan Ministry of Agriculture, Livestock, and Fisheries, Spain.

A total of 214 purebred barrows from a Duroc line (Selección Batallé, Riudarenes, Girona, Spain) were used for this research (Bosch et al. 2012). The line was closed in 1991 and since then it has been selected for an index including body weight, backfat thickness and intramuscular fat content with the primarily objective of

87 producing premium pork and high quality dry-cured hams (Solanes et al., 2009). Pigs
88 were produced by 102 sows and 36 boars and raised up to slaughter in three separate
89 batches in a commercial farm. They were allocated in pens of 12 individuals and were
90 given *ad libitum* access to feed. A pelleted growing and finishing diet were given from
91 110 to 160 days and from 160 to 220 days, respectively (**Table 1**). Pigs used in the
92 experiment were subjected to repeated sampling for muscle and subcutaneous fat (SF)
93 specimens throughout the finishing period. A biopsy of m. *Longissimus thoracis* (LT)
94 and of SF was taken in 191 pigs at around 185 days (183, SD 4.3). Additionally,
95 samples of both tissues were also taken at 160 days (158.0, SD 6.9; n=81) and at 210
96 days (207.9, SD 3.0; n=60). Before taking biopsies, the live body weight was measured
97 and backfat thickness (BT) and loin-muscle thickness at 5 cm of the midline between
98 the third and fourth last ribs ultrasonically recorded using the portable equipment Piglog
99 105 (SFK-Technology, Herlev, Denmark). Biopsies were taken 5 cm deep at the same
100 location where BT was measured and were extracted using 8-mm cannula inserted into
101 spring-loaded biopsy device (PPB-U Biotech, Nitra, Slovakia) as described in Oksbjerg,
102 Henckel, Andersen, Pedersen, & Nielsen (2004). All the necessary measures were taken
103 to prevent animal discomfort during and after the process (Bosch, Tor, Villalba,
104 Puigvert, & Estany, 2003). Muscle and fat samples were trimmed from skin and
105 separately frozen in liquid nitrogen until analysis 1 to 5 months later. Pigs were
106 slaughtered at 220 days (222, SD 3.8) in a commercial slaughterhouse equipped with a
107 carbon dioxide stunning system (Butina ApS, Holbaek, Denmark), where BT and loin-
108 muscle thickness at 6 cm off the midline between the third and fourth last ribs were
109 measured using the Autofom automatic carcass grading (SFK-Technology, Herlev,
110 Denmark). After slaughter, the carcass weight and the carcass length were measured.
111 The carcass length was measured from the anterior edge of the symphysis pubic to the

recess of the first rib. The carcass lean percentage was estimated on the basis of 35 measurements of AutoFOM points by using the official approved equation (decision 2001/775/CE, 2001) and the lean weight from carcass weight and lean percentage. After chilling for about 24 h at 2°C, each carcass was divided into primal cuts and the left side ham was weighed. Each ham was trimmed according to customary procedure used for manufacturing traditional dry-cured Spanish ham. Immediately after quartering, a sample of m. *Gluteus medius* from the left side ham was taken. In around 30 pigs per batch a sample of LT and SF at the level of the third and fourth ribs was also collected. These samples were immediately vacuum packaged and stored at –20°C until required for IMF and FA determinations.

2.2. Determination of IMF content and fatty acid composition

Frozen samples were removed from the nitrogen tank or the freezer 12 h prior to laboratory analyses. Biopsy specimens were directly freeze-dried and thereafter thoroughly homogenized by mixing with sand using a glass stirring rod. Due to their small size, dry matter in these samples was calculated as the weight difference before and after freeze-drying, and then the whole sample used for subsequent analyses. Post-mortem samples of LT and m. *Gluteus medius* were completely defrosted, vacuum drip losses were eliminated and muscle and subcutaneous fat were dissected out separately. Once minced, a small quantity of each was used to determine dry matter by drying 24 h at 100 to 102 °C in air oven whereas the rest of the sample was freeze-dried and pulverized using an electric grinder. A representative aliquot from the pulverized freeze-dried specimens was used for chemical analyses.

IMF content was estimated by quantitative determination of the fatty acids by gas chromatography following the methodology described in Bosch, Tor, Reixach, &

137 Estany (2009). Fatty acid methyl esters of both IMF and SF were directly obtained by
138 transesterification using a solution of boron trifluoride 20% in methanol (Rule, 1997).
139 Analysis of fatty acid methyl esters were performed by gas chromatography with a
140 capillary column SP2330 (Supelco, Tres Cantos, Madrid) and a flame ionization
141 detector with helium as the carrier gas at 1 mL/min. The oven temperature program
142 increased from 150 to 225 °C at 7 °C per min, and the injector and detector
143 temperatures were both 250 °C (Tor, Estany, Francesch, & Cubiló, 2005). The
144 quantification was carried out through area normalization by adding into each sample 1,
145 2, 3-Tripentadecanoylglycerol as internal standard before transesterification. IMF was
146 calculated as the sum of each individual fatty acid expressed as triglyceride equivalents
147 (AOAC, 2000) on a dry tissue basis. IMF and SF fatty acid composition was calculated
148 as the percentage of each individual fatty acid relative to total fatty acids, and expressed
149 as mg/g fatty acid. The proportion of SFA (C14:0; C16:0; C18:0 and C20:0), MUFA
150 (C16:1n−9; C18:1 and C20:1n−9) and PUFA (C18:2n−6; C18:3n−3; C20:2n−6 and
151 C20:4n−6) fatty acid contents were calculated.
152
153 *2.3. Isolation of genomic DNA and genotyping*
154 The isolation of genomic DNA was carried out from muscle samples stored at -
155 80ºC. Samples were lysed in the presence of proteinase K and DNA was purified
156 through extraction with phenol: chloroform, followed by ethanol precipitation. Finally,
157 DNA was re-suspended and stored in TE buffer. The quantification and estimation of
158 the quality and purity of genomic DNA was performed using a Nanodrop N-1000
159 spectrophotometer; DNA integrity was tested through electrophoresis in a 1% agarose
160 gel.

All pigs were genotyped for the *LEPR* NM_001024587:g.1987C>T and the *SCD AY487830:g.2228T>C* single nucleotide polymorphisms (SNP), which serve as tag SNPs for capturing the variance associated to *LEPR* and *SCD* genes, respectively. The *LEPR* NM_001024587:g.1987C>T SNP at exon 14 (Óvilo et al., 2005) was genotyped by High Resolution Melt analysis (Luminaris Color HRM Master Mix, Thermo Scientific) in a real time thermocycler (CFX-100, Bio-Rad) using 10 ng of genomic DNA and 0.4 µM of each of the following primers: LEPR-F, 5´-CAGAGGACCTGAATTTTGGAG-3´; LEPR-R, 5´-CATAAAAATCAGAAATACCTTCCAG-3´. The *SCD* AY487830:g.2228T>C SNP was genotyped using an allelic discrimination assay with the primers and probes indicated in Estany et al. (2014). The reaction mix contained 1x Universal TaqMan master mix (LifeTechnologies, Grand Island, NY), 0.2 µM Primer mix, 0.8 µM Probe mix and 10 ng of DNA in a final volume of 5 µl.

2.4. Statistical analyses

The effect of the *SCD* and *LEPR* genotypes by age on body weight, BT, loin-muscle thickness, IMF and FA of LT and SF were estimated on data from biopsies taken at 160, 185, and 210 days of age using a linear mixed model which included the batch (3 levels), the age at measurement (160, 185, and 210 days), the *SCD* genotype (TT, CT and CC), the *LEPR* genotype (CC, CT and TT) and the interaction of genotype by age at measurement as fixed effects and the pig and the residual as random effects. Moreover, data from either biopsies or carcass, were also analyzed independently at each age using a fixed model with the effects of the batch, the *SCD* and *LEPR* genotypes and age, this latter considered here as a deviation from the target age in each time-point (160, 185, 210, and at slaughter at 220 days). As in Bosch et al. (2009), in

both approaches the potential bias due to the biopsy size on IMF and FA composition was corrected including in the model for these traits a quadratic polynomial on sample weight. The interaction between genotypes was tested including in the model the corresponding term. The effect of the genotypes was tested following an F-test and multiple pairwise comparisons were done using the Tukey test. All the analyses were performed using the statistical package JMP 8 (SAS Institute Inc., Cary, NC).

3. Results

The average effects of the *SCD* and *LEPR* genotypes on body weight, BT, loin-muscle thickness, IMF and FA composition in both muscle and subcutaneous fat during the finishing period are given in **Tables 2 and 3,** respectively. The effect of both genotypes on LT and SF was consistent across tissues and throughout the finishing period. Thus, pigs carrying the T allele at *SCD* increased MUFA content (452.0 mg /g FA, for TT, and 428.9 mg/g FA, for CC, $P<0.05$, in LT; and 414.3 mg/g FA, for TT, and 400.7 mg/g FA for CC, $P<0.05$, in SF) while the T allele at *LEPR* increased SFA (423.9 mg/g FA, for TT, and 409.8 mg/g FA, for CC, $P<0.05$, in LT; and 420.6 mg/g FA, for TT, and 410.6 mg/g FA, in SF). In general, the effect of the *SCD* genotypes on the FA profile was greater than for *LEPR* genotypes and in LT than in SF. The T allele at *LEPR* also increased BT (20.9 mm, for TT, and 19.4, for CC, $P<0.05$). The *SCD* genotype did not affect neither BT nor IMF. A significant interaction of *SCD* with age was observed for BT and PUFA, both in LT and SF, and of *LEPR* with age for SFA in LT ($P<0.05$).

In order to dissect out these interactions, the data were independently analyzed at each age of measurement. The effects of the *SCD* and *LEPR* genotypes on SFA, MUFA

and PUFA in LT by age are depicted in **Figures 1 and 2**, respectively. As expected from previous works (Bosch et al., 2012), MUFA increased during the finishing period while PUFA decreased. Similar results were obtained for BT and for FA composition in SF and therefore they are not shown. It can be seen from these figures that the interaction between genotype and age was minor and limited to small changes in magnitude for SFA in *LEPR*. On the whole, the effect of the genotypes on SFA, MUFA, and PUFA showed the same pattern throughout the finishing period, with the T allele at *SCD* increasing MUFA and the T allele at *LEPR* increasing SFA. The joint effect of both genes is accounted for in **Figure 3** using the MUFA/SFA ratio as a target trait. Both in LT and in SF, the proportion of MUFA with respect to SFA was around 15% higher in the TTC- (TT, for *SCD*, and CC or CT, for *LEPR*) as compared to the CCTT (CC, for *SCD*, and TT, for *LEPR*) pigs (1.16 and 1.07, for TTC-, and 1.01 and 0.94, for CCTT, in LT and SF, respectively; $P<0.05$). The difference between this two extreme genotypes for BT, IMF and body weight was not significant ($P>0.05$; data not shown).

The effect of the *SCD* and *LEPR* genotypes on carcass traits, as well as on IMF content and FA composition of the *Gluteus medius* muscle, are presented in **Table 4**. In agreement with results obtained with live measurements, the most striking effects were on FA composition. Thus, pigs carrying the T allele at the *SCD* gene had higher MUFA (471.7 mg/g FA, for TT, and 456.2 mg/g FA, for CC, $P<0.05$) and pigs with the T allele at the *LEPR* gene higher SFA content (417.5 mg/g FA, for TT, and 402.4 mg/g FA, for CC, $P<0.05$). In contrast to the SCD-T allele, the *LEPR*-T led to higher levels of IMF (22.5% DM, for TT, and 19.5% DM, for CC, $P<0.05$). Neither of the two genotypes affected BT, loin-muscle thickness, and lean content. Evidence of synergic effects between both genes was limited, with BT and C20:2n-6 being the only traits for which the interaction between *SCD* and *LEPR* was significant ($P<0.05$).

4. Discussion

In this study we investigated the effects of two tag polymorphisms, one at the promoter of the *SCD* gene (AY487830:g.2228T>C) and another at exon 14 of the *LEPR* gene (NM_001024587:g.1987C>T), on fat content and composition during the growing-finishing period. In line with earlier research in Duroc pigs (Estany et al., 2014; Henriquez-Rodriguez et al., 2015), the results obtained confirmed the beneficial effect of the T allele at *SCD* gene on MUFA content and provided new evidence that the T allele at *LEPR*, which is segregating in Duroc, is positively associated with fatness and SFA content, both in muscle and SF. This is in agreement with previous findings with the *LEPR* gene in both Iberian (Muñoz et al., 2009; Óvilo et al., 2010) and Duroc-sired crossbreds (Galve et al., 2012; Muñoz et al., 2011). Also in line with previous reports (Gol et al., 2015), the allelic frequency of the T allele in this study was 0.41, for *SCD*, and 0.48, for *LEPR*, suggesting that both polymorphisms are present at intermediate frequencies in purebred Duroc. Such segregation pattern gives enough scope for using both SNPs to reduce the heterogeneity of Duroc-sired pig products.

The polymorphism at the *SCD* gene did not show relevant undesirable effects, particularly on carcass traits and composition. Contrarily, the *LEPR* polymorphism, although had a positive impact on IMF, it also affected overall fatness. It has been suggested that the effects of *LEPR* can be an indirect consequence of increased feed intake (Óvilo et al., 2005), since the leptin receptor mediates the satiety effect of leptin (Barb, Hausman, & Houseknecht, 2001; Houseknecht et al., 1998). This hypothesis was corroborated by the results reported by Rodríguez et al. (2010), who found a positive effect of the T allele on body weight and voluntary feed intake. In the present study we

found an effect of *LEPR* on BT and IMF, but not on body weight. However, dealing with a larger dataset on production and carcass traits from the same line used here, Gol et al. (2015) were able to detect that *LEPR* not only affect BT and IMF but also body and carcass weight. These results would confirm that, although subjected to variations due to sampling location, muscle or equipment of measurement, the T allele at *LEPR*, likely through increased feed intake, results in heavier and fatter pigs. Although BT is easy to modify with conventional breeding, it is always interesting to have available for use in genetic evaluations a genetic marker explaining a significant percentage of the genetic variation of IMF content and composition (Ros-Freixedes et al., 2016) and of the unfavorable correlation of BT with these traits (Ros-Freixedes et al, 2014).

Several authors have shown that the fatty acid profile of muscle and SF changes during fattening. Bosch et al. (2012), using the same Duroc as here, reported increased SFA and MUFA content while decreased PUFA content from 5.5 to 7.5 months of age. The same trend was observed in commercial crossbreds by Lo Fiego, Macchioni, Minelli, & Santoro (2010), from 6 to 9.5 months, and by Virgili et al. (2003), from 8 to 10 months. The results obtained here reflect the same evolution as in these experiments regardless of the markers. Interestingly, however, the effect of the markers may offset the effect of age in terms of fatty acid composition. Thus, for example, the CC pigs at the *SCD* gene had more SFA in LT at 160 days (415.9±4.1 mg/g FA) than the TT at 220 days (405.8±4.2 mg/g FA), or similarly, the TT pigs at the *LEPR* gene had more SFA at 160 days (424.4 ±4.8 mg/g FA) than the other two genotypes at 220 days (CC: 404.8±3.4 mg/g FA; CT: 411.0±2.3 mg/g FA). The combined effect of the *SCD* and *LEPR* markers was analyzed for the MUFA/SFA ratio, a trait commonly used to assess the impact of dietary fat on health (Pacheco et al., 2006; Voisin et al., 2015). Both in muscle and SF, the MUFA/SFA ratio was on average 15% higher in pigs jointly

displaying the beneficial *SCD* TT and *LEPR* C- genotypes as compared to pigs with the CCTT genotype. This result shows that the combined use of both markers could be useful to produce healthier meat. However, the use of the *LEPR* C- genotype, which is associated to lower IMF and higher PUFA, may affect negatively the technological and sensory attributes of dry-cured hams production (Ruiz-Carrascal, Ventanas, Cava, Andrés, & García, 2000; Gandemer,2009).

The effects of the *SCD* and *LEPR* SNPs have proved to be consistent throughout the whole finishing period and in both LT and SF. Rodríguez et al. (2010) observed that the magnitude of the effect of *LEPR* on feed intake and average daily gain increased with age. In this study, however, we did not observe an interaction pattern between genotype and age. It should be noted, however, that we have only investigated the age interval covering the late fattening period, from 95 to 130 kg, where the effect of *LEPR* genotypes on body composition are already manifested. In fact, Rodríguez et al. (2010) did not find any effect of *LEPR* on body weight and feed intake until 65 kg. The effect of both polymorphisms on fat composition was in general more relevant in muscle than in SF, which is in accordance with the fact that the composition of neutral lipids in IMF is more aligned to endogenous fatty acid synthesis and remodeling rather than to dietary fat (Wood et al., 2008). Not only age but fat content determine fatty composition. For SFA in particular, Bosch et al. (2012) showed that fat content is what most influences SFA. To test whether the effect of *LEPR* was mainly a matter of scale, the difference between genotypes for SFA was adjusted for IMF (in *Gluteus medius* and LT) and BT (in SF). The effect of *LEPR* on SFA at constant fat content was lower, still significant ($P<0.05$) in *Gluteus medius* and LT but not in SF (410.5±3.4 mg/g FA, for CC; 408.1±2.3 mg/g FA, for CT; and 417.5±3.4 mg/g FA, for TT). This suggests that with

regards to *LEPR*, compositional changes, particularly in SF, are due to overall increased fatness.

In a previous research we showed that the T allele at *SCD* behaved additively (Estany et al., 2014), but results are less clear and more controversial for *LEPR*, in part because only some experiments included the three genotypes. Thus, while *LEPR* effects were found to be mainly additive (Rodríguez et al., 2010; Galve et al., 2012), complete dominance is not discarded (Pérez-Montarelo et al., 2012; Uemoto et al., 2012). Even though we have not tested specifically for dominance, the results obtained (see, for instance, **Figure 1**) would support the existence of a dominant effect with allele T acting as recessive, in line with other results in purebred Duroc (Uemoto et al., 2012; Gol et al., 2015). The statistical gene-gene interactions can lead to changes in magnitude or direction of the effects observed phenotypically (Mackay, 2014). Evidence of epistatic interaction between *LEPR* and *SCD* are minor and constrained to small-magnitude changes in BT, in line with the interaction of *LEPR* with other genes related to fat metabolism, such as the leptin (Perez-Montarelo et al., 2012) or the *MC4R* (Galve et al., 2012) genes. However, there are recent reports providing clues for possible dominant by additive interactions of *LEPR* with the *SCD* (Gol et al., 2015) and *PRKAG3* (López-Buesa, Burgos, Galve, & Varona, 2013) genes. More powerful designs are needed to detect and confirm these potential dominant and epistatic effects.

5. Conclusions

The present research confirms the positive effect of the T allele at the *SCD* gene (*AY487830:g.2228T>C*) on MUFA and provides new evidence on the positive effect of the T allele at the *LEPR* gene (*NM_001024587:g.1987C>T*) on SFA, both in LT and SF

in Duroc pigs. However, contrarily to SCD, our findings show that the effect of *LEPR*, particularly in SF, is due to increased overall fatness. There is limited evidence of synergic effects between *SCD* and *LEPR* genes and of the interaction between them and age. Accordingly, their join effects are mostly additive and remain stable throughout all the finishing period. It is concluded that the combined selection for the *SCD* T and the *LEPR* C alleles is a good strategy to increase the MUFA/SFA ratio regardless of the age at slaughter.

Acknowledgments

This research was funded by the Spanish Ministry of Science and Innovation (MICINN, grant AGL2009-09779) and the Ministry of Economy and Competitiveness (MINECO, grant AGL2012-33529). EH-R is a recipient of a PhD scholarship from the University of Lleida.

References

AOAC (2000). Official method 960.39 fat (crude) or ether extract in meat. *Official methods of analysis* (pp. 2). (17th ed.). Arlington, VA: Association of Analytical Chemists.

Barb, C. ., Hausman, G. ., & Houseknecht, K. . (2001). Biology of leptin in the pig. *Domestic Animal Endocrinology*, *21*(4), 297–317. doi:10.1016/S0739-7240(01)00123-0

Bosch, L., Tor, M., Reixach, J., & Estany, J. (2009). Estimating intramuscular fat content and fatty acid composition in live and post-mortem samples in pigs. *Meat*

Science, *82*(4), 432–7. doi:10.1016/j.meatsci.2009.02.013

Bosch, L., Tor, M., Reixach, J., & Estany, J. (2012). Age-related changes in intramuscular and subcutaneous fat content and fatty acid composition in growing pigs using longitudinal data. *Meat Science*, *91*(3), 358–63. doi:10.1016/j.meatsci.2012.02.019

Bosch, L., Tor, M., Villalba, D., Puigvert, X., & Estany, J. (2003). Determinación del contenido y composición de la grasa intramuscular a partir de una biopsia en porcino. *ITEA, Volumen Extra*, *24*, 193–195.

Cameron, N.D., Enser, M., Nute, G.R., Whittington, F.M., Penman, J.C., Fisken, A.C., Perry, A.M., & Wood, J.D. (2000). Genotype with nutrition interaction on fatty acid composition of intramuscular fat and the relationship with flavour of pig meat. *Meat Science*, *55*(2), 187–195. doi:10.1016/S0309-1740(99)00142-4

Cameron, N. D., & Enser, M. B. (1991). Fatty acid composition of lipid in Longissimus dorsi muscle of Duroc and British Landrace pigs and its relationship with eating quality. *Meat Science*, *29*(4), 295–307. doi:10.1016/0309-1740(91)90009-F

Estany, J., Ros-Freixedes, R., Tor, M., & Pena, R. N. (2014). A functional variant in the stearoyl-CoA desaturase gene promoter enhances fatty acid desaturation in pork. *PloS One*, *9*(1), e86177. doi:10.1371/journal.pone.0086177

Fernandez, X., Monin, G., Talmant, A., Mourot, J., & Lebret, B. (1999). Influence of intramuscular fat content on the quality of pig meat — 2. Consumer acceptability of m. longissimus lumborum. *Meat Science*, *53*(1), 67–72. doi:10.1016/S0309-1740(99)00038-8

Fortin, A., Robertson, W. M., & Tong, A. K. W. (2005). The eating quality of Canadian pork and its relationship with intramuscular fat. *Meat Science*, *69*(2), 297–305. doi:10.1016/j.meatsci.2004.07.011

385 Galve, A., Burgos, C., Silió, L., Varona, L., Rodríguez, C., Ovilo, C., & López-Buesa,
386 P. (2012). The effects of leptin receptor (LEPR) and melanocortin-4 receptor
387 (MC4R) polymorphisms on fat content, fat distribution and fat composition in a
388 Duroc×Landrace/Large White cross. *Livestock Science*, *145*(1-3), 145–152.
389 doi:10.1016/j.livsci.2012.01.010
390 Gandemer, G, (2009). Dry cured ham quality as related to lipid quality of raw material
391 and lipid changes during processing: a review. *Grasas y Aceites*, 60(3), 297–307.
392 Gillingham, L. G., Harris-Janz, S., & Jones, P. J. H. (2011). Dietary monounsaturated
393 fatty acids are protective against metabolic syndrome and cardiovascular disease
394 risk factors. *Lipids*, *46*(3), 209–28. doi:10.1007/s11745-010-3524-y
395 Gol, S., Ros-Freixedes, R., Tor, M., Pena, R. N., Rothschild , M. F. y Estany , J. (2015).
396 Additive, dominant and epistatic effects of SCD and LEPR genes on fat content
397 and fatty acid composition in pigs. *AIDA. XVI Jornadas sobre Producción Animal*,
398 *Tomo II*, 483-485.
399 Henriquez-Rodriguez, E., Tor, M., Pena, R. N., & Estany, J. (2015). A polymorphism in
400 the stearoyl-CoA desaturase gene promoter increases monounsaturated fatty acid
401 content in dry-cured ham. *Meat Science*, *106*, 38–43.
402 doi:10.1016/j.meatsci.2015.03.019
403 Houseknecht, K. L., Baile, C. A., Matteri, R. L., & Spurlock, M. E. (1998). The
404 biology of leptin: a review. *Journal of Animal Science*, *76*(5), 1405–1420.
405 doi:/1998.7651405x
406 Jeleníková, J., Pipek, P., & Miyahara, M. (2008). The effects of breed, sex,
407 intramuscular fat and ultimate pH on pork tenderness. *European Food Research*
408 *and Technology*, *227*(4), 989–994. doi:10.1007/s00217-007-0810-x
409 Lo Fiego, D. Pietro, Macchioni, P., Minelli, G., & Santoro, P. (2010). Lipid

410 composition of covering and intramuscular fat in pigs at different slaughter age.
411 *Italian Journal of Animal Science, 9*(2), 39. doi:10.4081/ijas.2010.e39.
412 López-Buesa, P., Burgos, C., Galve, A., & Varona, L. (2013). Joint analysis of additive,
413 dominant and first-order epistatic effects of four genes (IGF2, MC4R, PRKAG3
414 and LEPR) with known effects on fat content and fat distribution in pigs. *Animal*
415 *Genetics* 45, 133–137. doi: 10.1111/age.12091
416 Mackay, T. F. C. (2014). Epistasis and quantitative traits: using model organisms to
417 study gene-gene interactions. *Nature Reviews. Genetics*, *15*(1), 22–33.
418 doi:10.1038/nrg3627
419 Muñoz, G., Alcázar, E., Fernández A., Barragán, C., Carrasco, A., de Pedro, E., Silió.,
420 L, Sánchez, J.L., & Rodríguez, M.C. (2011). Effects of porcine MC4R and LEPR
421 polymorphisms, gender and Duroc sire line on economic traits in Duroc×Iberian
422 crossbred pigs. *Meat Science*, 88, 169–173. doi:10.1016/j.meatsci.2010.12.018
423 Muñoz, G., Ovilo, C., Silió, L., Tomás,A., Noguera, J.L., & Rodríguez, M.C. (2009)
424 Single- and joint-population analyses of two experimental pig crosses to confirm
425 quantitative trait loci on Sus scrofa chromosome 6 and leptin receptor effects on
426 fatness and growth traits. *Journal of Animal Science*, *87*(2), 459–468.
427 doi:10.2527/jas.2008-1127
428 Ntambi, J., & Miyazaki, M. (2004). Regulation of stearoyl-CoA desaturases and role in
429 metabolism. *Progress in Lipid Research*, *43*(2), 91–104. doi:10.1016/S0163-
430 7827(03)00039-0
431 Oksbjerg, N., Henckel, P., Andersen, S., Pedersen, B., & Nielsen, B. (2004). Genetic
432 variation of in vivo muscle glycerol, glycogen, and pigment in Danish purebred
433 pigs. *Acta Agriculturae Scandinavica, Section A - Animal Science*, *54*(4), 187–192.
434 doi:10.1080/09064700510009225

435 Óvilo, C., Fernández, A., Fernández A.I., Folch, J.M., Varona, L., Benítez, R., Núñez,
436 Y., Rodríguez, M.C, & Silio, L. (2010). Hypothalamic expression of porcine leptin
437 receptor (LEPR), neuropeptide Y (NPY), and cocaine- and amphetamine-regulated
438 transcript (CART) genes is influenced by LEPR genotype. *Mammalian Genome*,
439 (21), 583–591. doi: 10.1007/s00335-010-9307-1
440 Óvilo, C., Fernández, A., Noguera, J. L., Barragán, C., Letón, R., Rodríguez, C.,
441 Mercadé, A., Alves, E., Folch, J. M., Varona, L., Toro, M. (2005). Fine mapping
442 of porcine chromosome 6 QTL and LEPR effects on body composition in multiple
443 generations of an Iberian by Landrace intercross. *Genetical Research*, *85*(1), 57–
444 67. doi:10.1017/S0016672305007330.
445 Pacheco, Y. M., Bermudez, B., Lopez, S., Abia, R., Villar, J., & Muriana, F. J. (2006).
446 Ratio of oleic to palmitic acid is a dietary determinant of thrombogenic and
447 fibrinolytic factors during the postprandial state in men. *Am J Clin Nutr*, *84*(2),
448 342–349. Retrieved from http://ajcn.nutrition.org/content/84/2/342.long
449 Pérez-Montarelo, D., Fernández, A., Folch, J.M., Pena, R.N, Óvilo, C, Rodríguez, M.C.,
450 Silió, L., & Fernández, A.I. (2012). Joint effects of porcine leptin and leptin
451 receptor polymorphisms on productivity and quality traits. *Animal genetics*, 43,
452 805–809. doi: 10.1111/j.1365-2052.2012.02338.x
453 Roche, H. M. (2001). Olive oil, high-oleic acid sunflower oil and CHD. *British Journal*
454 *of Nutrition*, *85*(01), 3. doi:10.1079/BJN2000242
455 Rodríguez, M.C., Fernández, A., Carrasco, C., García, A., Gómez, E., de Mercado, E.,
456 López, M.A., Ovilo, C., & Silió, L. (2010). Effect of LEPR c.2002C>T
457 polymorphism on growth and feed intake in heavy Duroc x Iberian pigs. In
458 *Proceedings of the 9th World Congress on Genetics Applied to Livestock*
459 *Production*: *1–6*, August 2010; Leipzig. Retrieved

460 from http://www.kongressband.de/wcgalp2010/assets/pdf/0479.pdf
461 Ros-Freixedes, R., Gol, S., Pena, R.N., Tor, M., Ibáñez-Escriche, N., Dekkers, J.C.M, &
462 Estany, J. (2016). Genome-wide association study singles out SCD and LEPR as
463 the two main loci influencing intramuscular fat content and fatty acid composition
464 in Duroc pigs. *PLoS ONE* 11(3): e0152496.
465 Ros-Freixedes, R., Reixach, J., Bosch, L., Tor, M. & Estany, J (2014). Genetic
466 correlations of intramuscular fat content and fatty acid composition among muscles
467 and with subcutaneous fat in Duroc pigs. *Journal of Animal Science*, 92(12):5417-
468 5425. doi:10.2527/jas2014-8202
469 Ruiz-Carrascal, J., Ventanas, J., Cava, R., Andrés, A. I., & García, C. (2000). Texture
470 and appearance of dry cured ham as affected by fat content and fatty acid
471 composition. *Food Research International*, 33, 91–95
472 Rule, D. C. (1997). Direct transesterification of total fatty acids of adipose tissue, and of
473 freeze-dried muscle and liver with boron-trifluoride in methanol. *Meat Science*,
474 *46*(1), 23–32. doi:10.1016/S0309-1740(97)00008-9
475 Solanes, F. X., Reixach, J., Tor, M., Tibau,J., & Estany J. (2009). Genetic correlations
476 and expected response for intramuscular fat content in a Duroc pig line. *Livestock.*
477 *Science*, 123:63–69.
478 Tikk, K., Tikk, M., Aaslyng, M. D., Karlsson, A. H., Lindahl, G., & Andersen, H. J.
479 (2007). Significance of fat supplemented diets on pork quality - Connections
480 between specific fatty acids and sensory attributes of pork. *Meat Science*, *77*(2),
481 275–86. doi:10.1016/j.meatsci.2007.03.019
482 Tor, M., Estany, J., Francesch, A., & Cubiló, M. D. (2005). Comparison of fatty acid
483 profiles of edible meat, adipose tissues and muscles between cocks and capons.
484 *Animal Research*, *54*(5), 413–424. doi:10.1051/animres:2005033

485 Uemoto, Y., Kikuchi, T., Nakano, H., Sato, S., Shibata, T., Kadowaki, H., Katoh, K.,
486 Kobayashi, E., & Suzuki, K. (2012). Effects of porcine leptin receptor gene
487 polymorphisms on backfat thickness, fat area ratios by image analysis, and serum
488 leptin concentrations in a Duroc purebred population. *Animal Science Journal =*
489 *Nihon Chikusan Gakkaihō*, *83*(5), 375–85. doi:10.1111/j.1740-0929.2011.00963.x
490 Virgili, R., Degni, M., Schivazappa, C., Faeti, V., Poletti, E., Marchetto, G., Pacchioli,
491 M. T., & Mordenti, A. (2003). Effect of age at slaughter on carcass traits and meat
492 quality of Italian heavy pigs. *Journal of Animal Science*, 81(10), 2448–2456.
493 doi:/2003.81102448x
494 Voisin, S., Almén, M. S., Moschonis, G., Chrousos, G. P., Manios, Y., & Schiöth, H. B.
495 (2015). Dietary fat quality impacts genome-wide DNA methylation patterns in a
496 cross-sectional study of Greek preadolescents. *European Journal of Human*
497 *Genetics : EJHG*, *23*(5), 654–62. doi:10.1038/ejhg.2014.139
498 Wood, J. D., Nute, G. R., Richardson, R. I., Whittington, F. M., Southwood, O.,
499 Plastow, G., Mansbridge, R., da Costa, N., & Chang, K. C. (2004). Effects of
500 breed, diet and muscle on fat deposition and eating quality in pigs. *Meat Science*,
501 *67*(4), 651–67. doi:10.1016/j.meatsci.2004.01.007
502 Wood, J. D., Enser, M., Fisher, A. V., Nute, G. R., Sheard, P. R., Richardson, R. I.,
503 Hughes S.I., & Whittington F.M. (2008). Fat deposition, fatty acid composition
504 and meat quality: A review. *Meat Science*, 78, 343–358. doi:
505 10.1016/j.meatsci.2007.07.019
506

Table 1. Composition of the diets (g/kg)

Item	Growing	Finishing
Dry matter	893.2	886.1
Crude lipid	56.3	61
Crude protein	193.6	181.2
Ash	60.4	69.8
Crude fiber	57.1	61.7
Nitrogen free extract	525.8	512.4
ME, MJ/kg	13.4	12.7
Fatty acids, mg/g fatty acid [A]		
C12:0, lauric	4.8	3.2
C14:0, myristic	18.3	16.2
C16:0, palmitic	220.8	229.8
C18:0, stearic	77.4	81.7
SFA	321.3	330.9
C16:1n−9, palmitoleic	22.6	23.6
C18:1n−9, oleic	301.1	294.7
C20:1n−9, eicosenoic	3.7	6.8
MUFA	327.4	325.1
C18:2n−6, linoleic	327.7	311.7
C18:3n−3, linolenic	15.5	19.7
C20:2n−6, eicosadienoic	2.8	3.1
C20:4n−6, arachidonic	1.1	1.3
PUFA	347.1	353.8

[A] SFA, saturated fatty acids (C12:0+C14:0+C16:0+C18:0; MUFA, monounsaturated fatty acids (C16:1n-9+C18:1n-9+C20:1n-9); PUFA, polyunsaturated fatty acids (C18:2n-6+C18:3n-3+C20:2n-6+C20:4n-6).

Table 2. Least square means (±SE) for production traits, intramuscular fat content (IMF) and fatty acid composition in m. *Longissimus thoracis* and subcutaneous fat by *SCD* genotype during the finishing period (from 160 to 210 days of age) and interaction of the *SCD* genotype with age [A]

	m. *Longissimus thoracis*				Subcutaneous fat			
	TT	CT	CC	SCD*age	TT	CT	CC	SCD*age
No of data	51	164	99		51	159	98	
Body weight, kg	111.2±1.7	113.0±0.9	115.3±1.2					
Backfat thickness, mm	19.2±0.6	20.3±0.3	19.8±0.4	*				
Loin thickness, mm	43.2±0.6	44.1±0.3	44.8±0.4					
IMF, % DM	16.8±0.9	17.4±0.5	16.8±0.7					
C14:0	14.0±0.8	13.8±0.5	13.1±0.6		17.6±0.4	16.6±0.2	17.0±0.3	
C16:0	255.5±2.2	258.1±1.2	257.8±1.6		246.9±2.4^{ab}	246.0±1.3^{b}	251.8±1.8^{a}	
C18:0	133.8±1.8^{c}	141.7±1.0^{b}	148.8±1.3^{a}		139.5±2.4^{b}	143.9±1.3^{b}	153.8±1.7^{a}	
C20:0	1.7±0.1	1.76±0.1	1.79±0.1		1.7±0.2	1.8±0.1	1.8±0.1	
SFA, mg/g FA	404.6±3.7^{b}	415.2±2.0^{a}	421.3±2.7^{a}		406.0±4.2^{b}	408.2±2.3^{b}	424.3±3.2^{a}	
C16:1n-9	34.0±1.1^{a}	32.1±0.6^{a}	28.8±0.8^{b}		22.4±1.1^{a}	20.8±0.6^{ab}	18.4±0.8^{b}	
C18:1	408.7±3.2^{a}	402.5±1.7^{a}	391.6±2.3^{b}		380.3±3.0^{ab}	378.7±1.6^{a}	372.0±2.2^{b}	
C20:1n-9	9.2±0.2^{a}	8.59±0.1^{b}	8.42±0.2^{b}		11.3±0.3	10.8±0.2	10.5±0.2	*
MUFA, mg/g FA	452.0±3.5^{a}	443.2±1.9^{a}	428.9±2.6^{b}		414.3±3.1^{a}	410.4±1.7^{a}	400.7±2.3^{b}	
C18:2n-6	123.2±3.6	122.3±2.0	129.9±2.7	*	158.2±3.0	157.5±1.7	153.7±2.2	*
C18:3n-3	7.7±0.2	7.46±0.1	7.76±0.2		11.9±0.5^{ab}	12.5±0.3^{a}	11.2±0.4^{b}	
C20:2n-6	5.8±0.2	5.66±0.1	5.80±0.1		8.5±0.4	8.5±0.2	7.9±0.3	
C20:4n-6	6.1±0.4	5.9±0.2	6.34±0.3		2.4±0.1	2.5±0.1	2.3±0.1	
PUFA, mg/g FA	142.9±4.1	141.5±2.3	149.9±3.0	*	181.5±3.5	181,1±1.9	175.1±2.6	*
C18:1/C18:0	3.18±0.05^{a}	2.86±0.03b	2.65±0.04^{c}		2.74±0.05^{a}	2.66±0.03^{a}	2.45±0.04^{b}	
C16:1n-9/C16:0	0.13±0.00^{a}	0.12±0.00^{a}	0.11±0.00^{b}		0.09±0.00^{a}	0.09±0.00^{a}	0.07±0.00^{b}	
MUFA/SFA	1.12±0.01^{a}	1.07±0.01^{b}	1.02±0.01^{c}		1.02±0.02^{a}	1.01±0.01^{a}	0.95±0.01^{b}	
MUFA/PUFA	3.29±0.10^{ab}	3.24±0.05^{a}	3.04±0.07^{b}		2.33±0.05	2.31±0.03	2.34±0.04	*
SFA/PUFA	2.95±0.10	3.04±0.05	2.97±0.07		2.28±0.07^{b}	2.29±0.04^{b}	2.48±0.05^{a}	

[A] SFA: C14:0+C16:0+C18:0+C20:0; MUFA: C16:1n-9+C18:1+C20:1n-9; PUFA: C18:2n-6+C18:3n-3+C20:2n-6+C20:4n-6; C18:1:C18:1n-9+C18:1n-7;
* Interaction between *SCD* genotype and age significant at p<0.05; * [a.b.c] Within row and factor, means with different superscripts differ significantly (P<0.05).

Table 3. Least square means (±SE) for production traits, intramuscular fat content (IMF) and fatty acid composition in m. *Longissimus thoracis* and subcutaneous fat by *LEPR* genotype during the finishing period (from 160 to 210 days of age) and interaction of the *LEPR* genotype with age [A]

	m. *Longissimus thoracis*				Subcutaneous fat			
	CC	CT	TT	LEPR*age	CC	CT	TT	LEPR*age
No of data	82	156	76		80	153	75	
Body weight, kg	110.8±1.4	113.5±1.0	115.1±1.4					
Backfat thickness, mm	19.4±0.5^{b}	19.0±0.3^{b}	20.9±0.5^{a}					
Loin thickness, mm	43.7±0.5	44.8±0.3	43.6±0.5					
IMF, % DM	16.2±0.7	16.5±0.5	18.3±0.7					
C14:0	13.6±0.7^{ab}	12.6±0.5^{b}	14.8±0.7^{a}		17.2±0.4	16.9±0.2	17.0±0,4	
C16:0	256.5±1.8^{ab}	253.0±1.3^{b}	261.8±1.8^{a}	*	246.7±2.0^{ab}	245.4±1.4^{b}	252.6±2.0^{a}	
C18:0	138.32±1.52^{b}	140.1±1.0^{b}	145.9±1.5^{a}		144.4±2.0	143.8±1.3	149.0±2.0	
C20:0	1.6±0.1	1.8±0.1	1.8±0.1		1.9±0.1	1.7±0.1	1.7±0.1	
SFA, mg/g FA	409.8±3.0^{b}	407.4±2.1^{b}	423.9±3.0^{a}	*	410.6±3.5^{ab}	407.4±2.4^{b}	420.6±3.5^{a}	
C16:1n-9	32.2±0.9	31.8±0.6	30.8±0.9		21.0±0.9	21.0±0.6	19.5±0.9	
C18:1	404.4±2.6	402.2±1.8	396.2±2.6		378.5±2.5	379.3±1.7	373.2±2.4	
C20:1n-9	8.6±0.2	8.7±0.1	8.9±0.2		10.7±0.3	10.7±0.2	11.1±0.3	
MUFA, mg/g FA	445.4±2.9^{a}	442.8±2.0^{ab}	435.9±2.9^{b}		410.5±2.6	410.9±1.8	403.9±2.6	
C18:2n-6	124.9±3.0	129.5±2.1	121.1±3.0		156.3±2.5	159.0±1.7	154.2±2.5	
C18:3n-3	7.6±0.2	7.8±0.1	7.4±0.2		11.9±0.4	11.9±0.3	11.8±0.4	
C20:2n-6	5.8±0.1	5.9±0.1	5.6±0.1		8.5±0.4	8.1±0.2	8.3±0.3	
C20:4n-6	6.4±0.4	6.4±0.2	5.5±0.4		2.5±0.1^{ab}	2.6±0.1^{a}	2.2±0.1^{b}	
PUFA, mg/g FA	144.9±3.4^{ab}	149.8±2.3^{a}	139.6±3.4^{b}		179.7±2.9	181.6±2.0	176.3±2.9	
C18:1/C18:0	2.96±0.04^{a}	2.90±0.03^{a}	2.74±0.04^{b}		2.65±0.04^{ab}	2.78±0.03^{a}	2.52±0.04^{b}	
C16:1n-9/C16:0	0.13±0.00	0.13±0.00	0.12±0.00		0.09±0.00	0.09±0.00	0.08±0.00	
MUFA/SFA	1.09±0.01^{a}	1.09±0.01^{a}	1.03±0.01^{b}		1.01±0.01^{ab}	1.02±0.01^{a}	0.96±0.01^{b}	
MUFA/PUFA	3.21±0.08	3.10±0.05	3.27±0.08		2.33±0.04	2.31±0.03	2.34±0.04	
SFA/PUFA	2.94±0.08^{ab}	2.84±0.06^{b}	3.17±0.08^{a}		2.33±0.06	2.29±0.04	2.44±0.06	

[A] See fatty acid abbreviations in Table 2; * Interaction between *LEPR* genotype and age significant at $p<0.05$; * [a.b.c] Within row and factor, means with different superscripts differ significantly ($P<0.05$).

Table 4. Least square means (±SE) for carcass traits, intramuscular fat content (IMF) and fatty acid composition in m. *Gluteus medius* at slaughter (220 days of age) by *SCD* and *LEPR* genotypes [A]

	SCD genotype			*LEPR* genotype			*SCD*LEPR*
	TT	CT	CC	CC	CT	TT	
No of pigs	33	110	71	58	104	50	
Carcass weight, kg	102.2±2.6	104.18±1.1	104.33±1.5	101.1±2.3	105.5±1.2	104.1±1.9	
Carcass backfat thickness, mm	24.2±0.9	24.5±0.4	23.2±0.5	24.0±0.8	23.7±0.4	24.1±0.6	*
Carcass loin thickness, mm	42.9±2.3	43.1±1.0	44.6±1.3	43.8±2.0	44.2±1.0	42.7±1.6	
Carcass length, cm	86.9±0.9	87.2±0.4	87.4±0.7	86.9±0.9	87.6±0.4	87.0±0.8	
Lean, %	42.0±1.2	41.7±0.5	43.0±0.7	42.0±1.1	42.8±0.5	42.0±0.8	
Lean weight, kg	42.8±1.4	43.3±0.6	44.5±0.8	42.3±1.2	44.9±0.6	43.4±1.0	
Ham weight, kg	12.6±0.3	13.0±0.1	13.3±0.2	12.9±0.3	13.3±0.2	12.7±0.3	
IMF, % DM	20.3±1.3	20.8±0.6	20.5±0.7	19.5±1.2^{ab}	19.6±0.6^{b}	22.5±0.9^{a}	
C14:0	16.3±1.0	15.4±0.4	14.6±0.6	15.7±0.9^{ab}	14.1±0.4^{b}	16.6±0.7^{a}	
C16:0	256.7±2.3	253.6±1.0	254.3±1.3	253.3±2.1^{b}	251.2±1.1^{b}	260.0±1.6^{a}	
C18:0	127.0±2.3^{c}	135.0±1.0^{b}	142.2±1.3^{a}	131.6±2.1^{b}	133.4±1.0^{b}	139.2±1.6^{a}	
C20:0	1.54±0.2	1.52±0.1	1.71±0.1	1.6±0.1	1.5±0.1	1.6±0.1	
SFA, mg/g FA	401.7±4.4^{ab}	405.5±1.9^{b}	412.8±2.5^{a}	402.2±4.0^{b}	400.3±2.0^{b}	417.5±3.0^{a}	
C16:1n-9	42.2±1.4^{a}	40.0±0.6^{a}	37.7±0.8^{b}	40.1±1.2	40.4±0.6	39.4±0.9	
C18:1	420.6±4.4^{ab}	419.9±1.9^{a}	410.2±2.5^{b}	418.2±4.0	417.8±2.0	414.6±3.1	
C20:1n-9	8.9±0.3	8.6±0.1	8.4±0.1	8.4±0.2^{ab}	8.4±0.1^{b}	9.0±0.2^{a}	
MUFA, mg/g FA	471.7±4.4^{a}	468.5±1.9^{a}	456.2±2.5^{b}	466.7±4.0	466.7±2.0	463.1±3.0	
C18:2n-6	108.1±3.8	107.0±1.7	111.4±2.2	111.5±3.5^{ab}	113.1±1.7^{a}	101.8±2.7^{b}	
C18:3n-3	6.2±0.3	5.9±0.1	6.1±0.1	6.3±0.2	6.1±0.1	5.8±0.2	
C20:2n-6	5.0±0.2	5.1±0.1	5.2±0.1	5.0±0.2	5.3±0.1	5.0±0.1	*
C20:4n-6	7.3±0.7	8.0±0.3	8.3±0.4	8.2±0.6^{ab}	8.5±0.3^{a}	6.8±0.5^{b}	
PUFA, mg/g FA	126.6±4.6	126.0±2.0	131.0±2.6	131.1±4.2^{ab}	133.1±2.1^{a}	119.5±3.2^{b}	
C18:1/C18:0	3.34±0.08^{a}	3.14±0.04^{b}	2.90±0.05^{c}	3.22±0.07^{a}	3.26±0.04^{a}	3.00±0.06^{b}	
C16:1n-9/C16:0	0.16±0.01^{a}	0.16±0.00^{a}	0.15±0.00^{b}	0.16±0.00	0.16±0.00	0.15±0.00	
MUFA/SFA	1.18±0.02^{a}	1.16±0.01^{a}	1.11±0.01^{b}	1.16±0.02^{a}	1.17±0.01^{a}	1.11±0.01^{b}	
MUFA/PUFA	3.81±0.13	3.8±0.06	3.61±0.08	3.65±0.12^{ab}	3.61±0.06^{b}	3.95±0.09^{a}	
SFA/PUFA	3.26±0.12	3.3±0.05	3.26±0.07	3.15±0.11^{b}	3.11±0.06^{b}	3.56±0.09^{a}	

[A] See fatty acid abbreviations in Table 2; * Interaction between *SCD* ad *LEPR* genotypes significant at p<0.05; [a.b.c] Within row and factor, means with different superscripts differ significantly (P<0.05)

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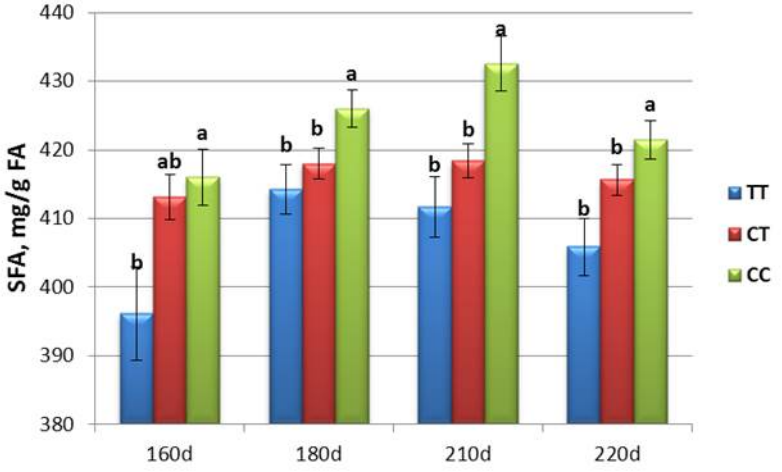
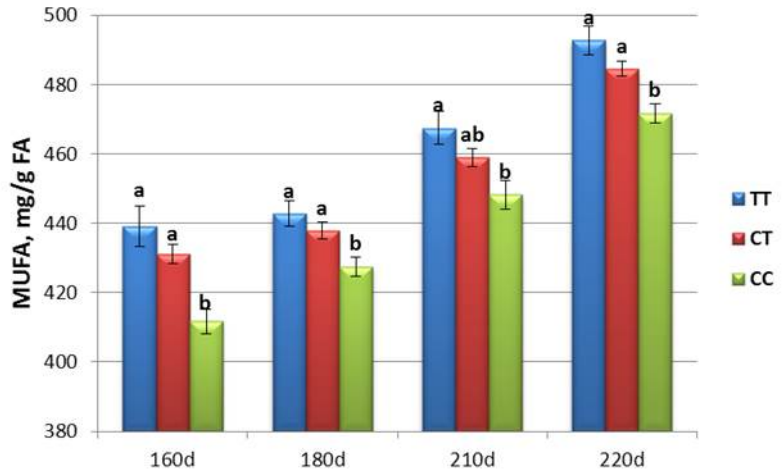
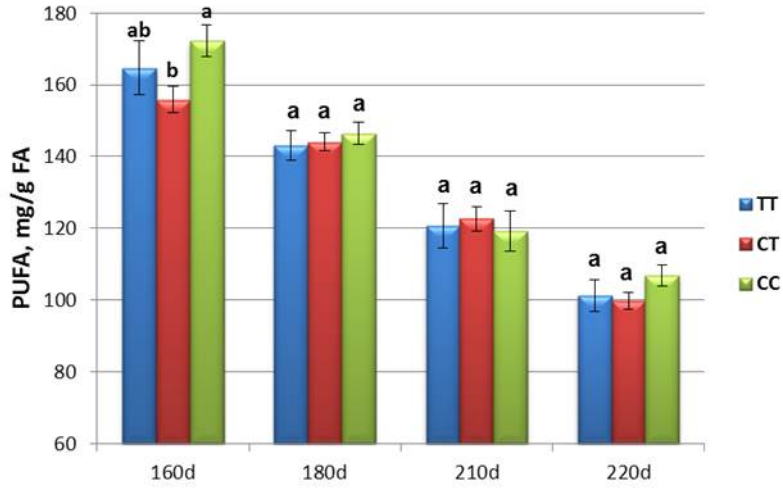
Figure 1. Effect of the *SCD* genotype on SFA, MUFA and PUFA in m. *Longissimus thoracis* by age. Means with different letters within age differ significantly (P<0.05).

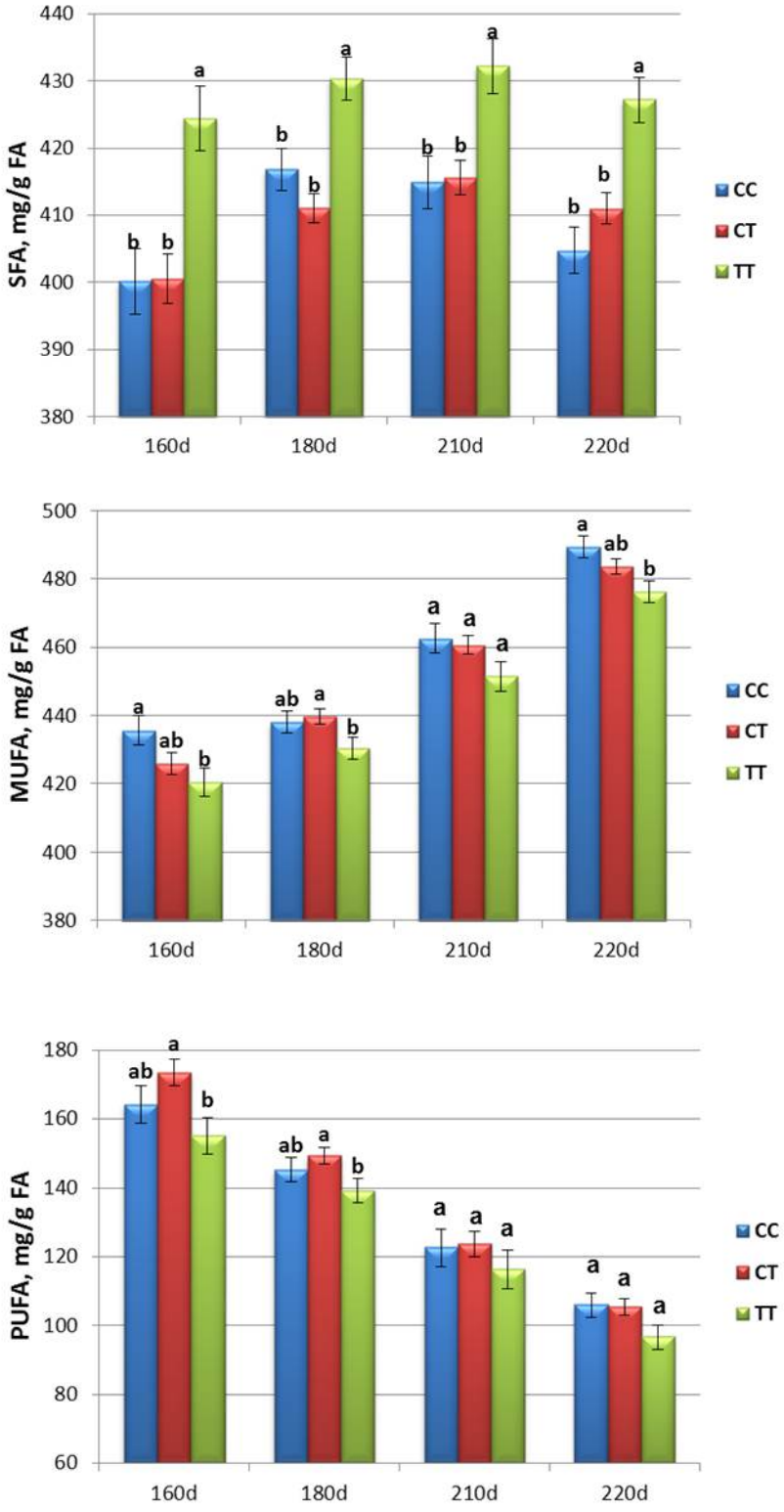
Figure 2. Effect of the *LEPR* genotype on SFA, MUFA and PUFA in m. *Longissimus thoracis* by age. Means with different letters within age differ significantly (P<0.05).

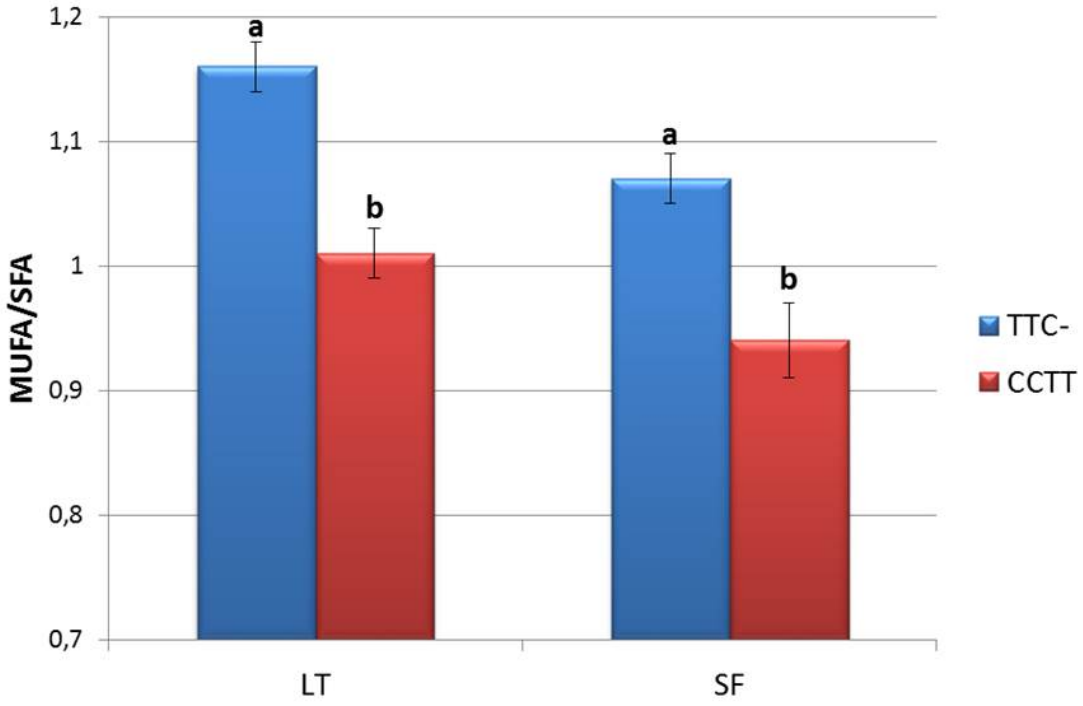
Figure 3. Least square means for the monounsaturated to saturated fatty acid ratio (MUFA/SFA) in m. *Longissimus thoracis* (LT) and subcutaneous fat (SF) the during finishing period (from 160 to 210 days of age) in the two extreme genotypes at *SCD* (first) and *LEPR* (second) genes. Means with different letters within tissues differ significantly (P<0.05).


SFA, mg/g FA
440
430
420
410
400
390
380
160d
180d
210d
220d
TT
CT
CC


MUFA, mg/g FA
500
480
460
440
420
400
380
160d
180d
210d
220d
TT
CT
CC


PUFA, mg/g FA
180
160
140
120
100
80
60
160d
180d
210d
220d
TT
CT
CC


SFA, mg/g FA
440
430
420
410
400
390
380
160d
180d
210d
220d
CC
CT
TT
MUFA, mg/g FA
500
480
460
440
420
400
380
PUFA, mg/g FA
180
160
140
120
100
80
60


1,2
1,1
1
0,9
0,8
0,7
MUFA/SFA
a
b
a
b
LT
SF
TTC-
CCTT