VARIATION OF BOAR TAINT COMPOUNDS IN BACKFAT FROM DIVERGENT GENETIC LINES

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Background.

Androstenone (5α-androst-16-en-3-one) and skatole (3-methyl-indole) are the compounds responsible of an objectionable odour well known as boar taint, which appears specially during cooking pigmeat from entire males. Androstenone biosynthesis is produced by the Leydig cells in the testicles and skatole and indole are produced in the colon by microorganisms which degrade tryptophan. These compounds are accumulated in the fatty tissue. Entire males are more efficient in lean growth rate and carcasses are leaner compared to gilts and castrated. Also, animal welfare is not compromise avoiding castration. Many efforts have been done to clarify the factors affecting androstenone and skatole in backfat entire males. Between and within breed variation have been demonstrated as well as evidences of major genes controlling the variation of these compounds.

Objective.

This study examine the variation of androstenone, skatole and indole in pig genetic lines with different selection strategies.

Methods.

A total of 50 boar backfat samples, from five genetic lines with different selection objectives, were analysed to determine androstenone, skatole and indole. All the animals were reared in the same farm and using the same diet. Backfat and muscle depth were measured using FOM automatic grading probe and used to estimate carcass lean content. The proportion of ham and loin was obtained by commercial cutting. Correlation coefficients were calculated overall as well as adjusted within genetic line.

For androstenone determinations 2µg of 5α-androstan-3-one and 2µg 5α-androstan-3α-ol were added to 1g of backfat as internal standards according to *Rius et al. (1999)*. Extraction was achieved with 50ml of dichloromethane and 5 ml were taken and evaporated to dryness. The residue was dissolved in 2 ml of methanol to remove the fat by precipitation at room temperature. A clean-up step was performed over a solid-phase extraction column (octadecyl) prewashed with 10 ml of methanol. The methanolic extract was applied at the top of the column and washed with 2ml of methanol. The methanolic fractions were collected and evaporated to dryness. The residue was dissolved in 20 µl iso-octane and injected into an CG-MS system. The separation of compounds were performed in a column HP-5MS (30m, 250X0,25µm) and the programme temperature used was 70ºC-10ºC/min-190ºC-5ºC/min-270ºC(5min). The temperatures of the injector and detector were set at 270ºC y 280ºC, respectively. The detection were performed in a SIM mode and the selected ions were m/z 274, 272, 258, 257, 243, 241 and 202.

For skatole and indole determinations 0,5 µg of 7-ethylindole were added to 1g of sample and dissolved in 10 ml of hexane-2:propanol (92:8) during 30 minutes according to *Garcia-Regueiro and Rius (1998)*. The extract was filtered (0,45 µm, PTFE) and injected into an HPLC system. An Hypersil aminopropylsilica column (APS2 5µm, 250x4.6mm) was used to achieve the separation of compounds and hexane-2:propanol (92:8) at 1,5 ml/min as a mobile phase. The detection was carried out by fluorescence detection (λemission 280nm/ λexcitation 360 nm).

Results and discussion.

There were significant differences in live weight, carcass weight, and carcass traits among the genetic lines analysed (Table 1). Also the genetic line influenced significatively the concentrations of androstenone, skatole and indole in backfat. The E and C pigs had the highest concentration of androstenone and E pigs had the highest concentration of skatole and indole among the genetic lines. These results support previous founding of genetic differences in fat levels of androstenone *(Bonneau et al., 1979)* but no clear genetic differences in skatole levels have been reported. However, *Lundstrom et al. (1994)* suggested that a genetic effect can be due to a major gene, though environmental conditions can explain skatole variation more clearly. Androstenone differences among breeds are explained due to differing rates of sexual maturation. In fact, *Prunier et al. (1987)* reported higher levels of androstenone in Meishan boars at lower weights than Large White boars. The Meishan breed reaches sexual maturity much earlier than improved breeds *(Xue, 1991)*. In our case, line E was originated from the Meishan and this can explain its higher level of androstenone than lines A, B and D. Line C originated from the Duroc breed had as much androstenone as line E although their fatness was not significatively different than A, B, and D. The very high percentages of pigs with androstenone and skatole concentrations above the cut-off levels of 0.5 and 0.1 µg/g respectively found in line E indicate than the crosses of this line should be avoid for boar pigmeat production, and although non line C pigs were above the skatole cut-off the high percentage of pigs from this line above the androstenone cut-off also indicates it is not recommended for entire male pigmeat production. The correlation coefficients across and within lines indicate that boar taint compounds are highly associated (Table 2). According to the correlation coefficients between boar taint compounds and carcass traits, genetic improvement for increase leaness and valuable joints decrease boar taint incidence. However, carcass weight was negatively correlated to androstenone in contradiction to previous works (Walstra and Garssen, 1995). If line E is drop from the analysis carcass weight and androstenone concentrations are not correlated and moreover if line C is drop together with E the correlation turns positive. In fact, lines E and C had lower carcass weights and had higher levels of androstenone among the lines studied.

Conclusions

As increasing carcass leaness due to selection the concentration of the compounds responsible of boar taint decrease. These results confirms that the genetic differences in backfat boar taint compounds may be due to differing rates of sexual maturation. Therefore, the meat industry should select lean genetic lines for entire male pigmeat production in order to avoid consumers rejection.

Table 1. Means for carcass and boar taint traits, and the incidence of tainted carcasses in five genetic lines°.

	Genetic lines					
	A	B	C	D	E	sig
Live weight (kg)	102.8^{b}	104.5^{a}	96.8^{bc}	103.1^{ab}	94.5^{c}	**
Carcass weight (kg)	76.1^{bc}	78.6^{ab}	73.4^{c}	80.9^{a}	73.1^{c}	**
¾ last rib fat (mm)	13.3^{b}	13.6^{b}	16.0^{b}	12.3^{b}	24.9^{a}	***
¾ last rib loin (mm)	53.3^{b}	53.3^{b}	52.7^{b}	63.5^{a}	50.4^{b}	**
Carcass lean (%)	61.8^{a}	61.7^{a}	60.1^{a}	63.3^{a}	55.1^{b}	***
Loin weight (kg)	9.17^{a}	9.08^{a}	8.77^{a}	8.94^{a}	7.88^{b}	***
Ham weight (kg)	9.57^{b}	9.72^{b}	9.81^{b}	10.52^{a}	9.21^{b}	**
Androstenone (µg/g)	0.177^{b}	0.277^{b}	1.067^{a}	0.339^{b}	3.024^{a}	**
Skatole (µg/g)	0.031^{b}	0.038^{b}	0.029^{b}	0.057^{b}	0.219^{a}	***
Indole (µg/g)	0.014^{b}	0.017^{b}	0.018^{b}	0.022^{b}	0.267^{a}	***
Androstenone >1µg/g (%)	0	0	60	0	80	
Androstenone >0.5µg/g (%)	0	0	90	33	80	
Skatole >0.22 µg/g (%)	0	0	0	11	50	
Skatole >0.1 µg/g (%)	0	0	0	11	60	

Means with different superscripts are significantly different (* p<.05; ** p<.01; *** p<.001)

Table 2. Overall and within line correlations (x100) between log androstenone, log skatole, log indole and carcass traits°.

	Log androstenone		Log skatole		Log indole	
	Overall	Within line	Overall	Within line	Overall	Within line
Log skatole	**48**	25	-	-	-	-
Log indole	**58**	**33**	**85**	**78**	-	-
Carcass weight	**-32**	**-32**	-25	-24	-17	-15
¾ last rib fat	**50**	22	**58**	**34**	**67**	**44**
¾ last rib loin	-26	**-30**	-29	**-33**	**-31**	**-40**
Lean content	**-51**	**-33**	**-55**	**-38**	**-65**	**-49**
Loin weight	**-51**	**-38**	**-51**	**-38**	**-52**	**-36**
Ham weight	**-29**	**-37**	-27	**-33**	-28	**-41**

° Numbers in bolds are statiscally significative (p<.05).

rtinent literature.

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