

## Genetic parameters for components of boar taint and their relation with carcass quality and female fertility

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#### Abstract

Of an anticipated 2000 entire males we analysed the first 693 animals for incidence of androstenone (AND) ( $1.7 \pm 1.5 \mu\text{g/g}$ ), skatole (SK) ( $0.082 \pm 0.09 \mu\text{g/g}$ ) and indole (IND) ( $0.065 \pm 0.07 \mu\text{g/g}$ ). Boars were culled from a synthetic sire line, slaughtered at  $124 \pm 12$  kg. Traits were log transformed and analysed using ASReml, with pen, age at start test, days tested and days from end test till slaughter as fixed effects, and slaughterday and animal effect as random. Resulting heritability estimates ( $h^2 \pm \text{SE}$ ): AND ( $0.75 \pm 0.14$ ), SK ( $0.53 \pm 0.13$ ) and IND ( $0.29 \pm 0.10$ ), in line with literature. Genetic correlation between AND, SK and IND was high ( $0.75 \pm 0.10$ ), for AND and SK, resp. IND  $0.22 \pm 0.19$  and  $0.30 \pm 0.20$ . Parameters for female fertility traits were estimated using 5320 litters of 1800 sows. Litter size ( $0.15 \pm 0.03$ ) and age until first insemination ( $0.20 \pm 0.06$ ) had low genetic correlations ( $-0.15 < r_g < 0.05$ ) with the three components of boar taint. Genetic correlation between interval weaning to 2nd insemination and AND was negative ( $-0.58 \pm 0.34$ ), but positive with SK and IND ( $0.62 \pm 0.38$  and  $0.62 \pm 0.37$ ).

Next step is the estimation of genetic correlations with finishing traits. Tissue of animals and their dams was stored for QTL studies. Given current results a substantial change in levels of boar taint seems to be feasible. To shift the distribution towards a zero boar taint perception for *all* consumers might be a bridge too far.

Key words: boar taint, carcass quality, female fertility, heritability, genetic parameters

#### Introduction

Meat with an unpleasant smell or taste is not accepted by consumers. Boar taint can occur in pork from entire male pigs, resulting in meat with an unfavourable odour and flavour. To reduce the risk of boar taint, in most of Europe, male pigs are being castrated shortly after birth. However, castration of boars has negative effects on feed efficiency and lean yield of the carcass. Furthermore, castration of piglets is done without anaesthesia and this causes a growing welfare concern in society. In the near future, European regulation might prohibit castration without sedation, and alternatives for castration have to be identified.

The main compounds related to the occurrence of boar taint are androstenone, skatole and indole in fat tissue (Vold, 1970; Walstra and Maarse, 1970; Bonneau 1982, Moss et al., 1988). Androstenone is a male sex pheromone which promotes sexual behaviour in female pigs (reviewed by Zamaratskaia, 2004), and is associated with a urine and perspiration odour. Skatole is produced in the lower gut by microbial degradation of the amino acid tryptophan, and is associated with naphthalene and faecal odour (Vold, 1970;

Walstra and Maarse, 1970). Indole is also synthesized in the large intestine from the same precursor tryptophan and is therefore considered to be related to skatole (Jensen and Jensen, 1998). Indole has been implicated in the development of boar taint, although to a much lesser degree than skatole (Moss *et al.*, 1988). Androstenone seems to affect skatole degradation in the liver by repression of the expression of the enzyme P450IIE1 (CYP2E1) (Babol, Squires and Lundström, 1999; Doran *et al.*, 2004). A higher level of androstenone will result in less breakdown of skatole and therefore higher levels of skatole in liver and also fat tissue.

Selection against boar taint seems to be feasible because heritabilities of androstenone, skatole and indole found in literature are quite high (0.25 - 0.88) (Jonsson and Pederson, 1974; Jonsson and Andresen, 1979; Willeke *et al.*, 1987; Sellier and Bonneau, 1988; Jonsson and Joergensen, 1989; Willeke, 1993; Fouilloux *et al.*, 1997). However, consequences for female fertility and carcass quality traits are not yet well understood. Negative effects of selection against androstenone on growth performance and onset of puberty in male and female pigs were found by Sellier and Bonneau (1988) and Willeke and Pirchner (1989). The aim of this study is to find out whether selection against high levels of androstenone, skatole and indole might have a negative influence on carcass quality and female fertility traits in a commercial purebred sire line.

## Material and Methods

### *Animals and measurements during test phase*

A total of 995 entire males from a synthetic sire line were born and raised in 2006 on a nucleus sire line farm in The Netherlands. After parturition males were not castrated. During the finishing test phase animals were housed in single-sex pens. Pigs were fed ad lib a commercial feed and had free access to water. One or two days after end of test where, weight and backfat measurements were done, most of the animals were slaughtered as culls. Others were slaughtered up to two months later as a result of negative AI ability scores. Backfat was recorded as the average value of four ultrasonic backfat measurements taken on the back of the pig. First two measurement points were located on the line between shoulder and last rib, third measurement point at the last rib and fourth measurement point behind the last rib. All measurements were taken on the left side of the spine, 5 cm from the mid-line, and distance between all points was equal. Growth during live was calculated as:  $\text{live growth} = \text{weight at end test} / \text{age in days}$ .

### *Measurements at slaughter and fertility traits*

Boars were slaughtered at an average live weight of  $124 \pm 12$  kg. Fat samples from the boars were taken at slaughter from the neck region and were analysed for the level of androstenone, skatole and indole. A Hennessy Grading Probe was used to record backfat and loin depth. Subsequently, meat percentage was calculated as:  $\text{meat percentage} = 60.9 - (0.755 * \text{backfat}) + (0.137 * \text{loin depth})$ .

Female fertility traits were recorded from 1800 sows (female sibs and parents of boars tested) with 5320 litters (total number born, litter mortality, age first insemination and interval weaning-second insemination).

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### *Analysis of androstenone, skatole and indole*

Androstenone concentrations in fat tissue were analysed by time-resolved fluoroimmunoassay as described by Tuomola et al. (1997). Skatole and indole were extracted from the fat sample using a mixture of methanol and hexane at 40°C in an ultrasonic bath. Skatole and indole were separated by HPLC on a reversed phase column. Fluorescence was measured at 285 nm and 340 nm (CCL B.V., Veghel, The Netherlands).

### *Statistical analysis*

Heritabilities and genetic correlations were estimated with ASReml (version 1.1, Gilmour et al., 2002). Data of androstenone, skatole and indole were not normally distributed, therefore log-transformed values of these traits were used. For androstenone, skatole and indole a multivariate individual animal model was used:

$$Y_{ijk} = \mu + \text{Pen}_i + b_1 * \text{Age at penning} + b_2 * \text{Test days} + b_3 * \text{Interval test to slaughter} + \text{Slaughter day}_j + \text{Animal}_k + e_{ijk}$$

Where  $Y_{ijk}$  = trait under study; Pen = fixed effect of  $i$ th pen number; Age at penning = age at start test; Test days = length of the test in days as a covariate; Interval test to slaughter = days between end of the test and day of slaughter as a covariate; Slaughter day = random effect of  $j$ th day of slaughter; Animal = additive genetic effect of  $k$ th animal;  $e_{ijk}$  = residual effect. For fertility and carcass traits, also a multivariate individual model was used. The multivariate individual model for carcass traits and live growth:

$$Y_{ij} = \mu + \text{Pen}_i + b_1 * \text{Month} + \text{Animal}_j + e_{ij}$$

Where Pen = fixed effect of  $i$ th pen number; Month = month of slaughter as a covariate; Animal = additive genetic effect of  $j$ th animal;  $e_{ij}$  = residual effect.

Models for fertility traits were the same as used in routine selection work. Effects in the model varied depending on the analysed trait (subscripts are ignored):

$$\begin{aligned} \text{AFI} &= \mu + \text{Farm} * b_1 * \text{Year} + \text{Sec ins} + e \\ \text{TNB} &= \mu + \text{Farm} * \text{Year} + \text{Sec ins} + \text{Parity} * \text{Line} + \text{Line litter} + \text{Perm} + \text{Boar} + e \\ \text{LM} &= \mu + \text{Farm} * \text{Year} + \text{Parity} + \text{Line litter} + \text{Perm} + e \\ \text{IW12} &= \mu + \text{Farm} * \text{Year} + b_1 * \text{Lact period} * \text{Lact period} + b_2 * \text{Weaned} + e \end{aligned}$$

Where Farm = fixed effect of farm of the sow; Year = fixed effect of year of farrowing; Sec ins = fixed effect of second insemination (yes or no); Parity = fixed effect of parity number; Line = fixed effect of line of the sow; Line litter = fixed effect of line of the litter; Lact period = lactation length as a covariate; Weaned = number piglets weaned in previous litter as a covariate; Boar = random effect of influence of the service sire; Perm = random effect of permanent environment;  $e$  = residual effect.

## **Results**

In Table 1, a description of the raw data is given. Levels of androstenone and skatole found in the present study are not extremely high (highest levels of androstenone and

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skatole: 9.15 µg/g and 0.93 µg/g, respectively), and some boars showed very low levels of androstenone and skatole (<0.05 µg/g) although the average live weight at slaughter is rather high (124 kg). Nevertheless, taking into account a threshold level for androstenone of 1.0 µg/g, 59% of the boars in the present study are suspected to show boar taint. But for skatole levels, only 6% of the boars in present study had values above the threshold of 0.2 µg/g.

Table 1. Summary statistics for traits measured: abbreviations used in text, number of animals per trait (N), means, SD and minimum (min) and maximum (max) values.

Trait	Abbreviation	N	Mean	SD	Min	Max
Androstenone, µg/g	AND	901	1.59	1.38	0.02	9.15
Skatole, µg/g	SKA	985	0.08	0.08	0.01	0.93
Indole, µg/g	IND	980	0.05	0.06	0.01	0.68
Ln (androstenone)	LNA	901	0.16	0.81	-3.91	2.21
Ln (skatole)	LNS	985	-2.82	0.69	-4.83	-0.07
Ln (indole)	LNI	980	-3.16	0.60	-4.61	-0.39
Weight at end test, kg	WE	798	122.4	11.9	82.0	160.0
Ultrasonic backfat, mm	BF	798	11.0	1.7	6.3	18.3
Loin depth HGP, mm	HGPloin	995	61.4	6.8	33.2	86.0
Backfat depth HGP, mm	HGPbf	995	14.7	2.7	7.6	25.2
Meat % (calculated)	HGPlean	995	58.1	2.1	49.1	64.2
Live growth, g	LGR	777	683.7	68.8	410.0	869.6
Age at first insemination, d	AFI	1677	250.6	17.6	208.0	346.0
Total number piglets born	TNB	5313	10.6	2.7	3.0	22.0
Litter mortality, %	LM	3944	9.6	13.2	0.0	100.0
Interval weaning-2nd insemination, d	IWI2	1677	0.1	0.4	0.0	1.0

### *Genetic parameters*

Heritabilities and genetic correlations of the logarithms of androstenone (LNA), skatole (LNS) and indole (LNI) are given in Table 2. The highest heritability was found for androstenone ( $h^2 = 0.72 \pm 0.11$ ) followed by skatole ( $h^2 = 0.63 \pm 0.11$ ) and indole ( $h^2 = 0.44 \pm 0.10$ ). The genetic correlation between skatole and indole was high ( $r_g = 0.75 \pm 0.10$ ), whereas the genetic correlations between skatole and androstenone and indole and androstenone were relatively low ( $r_g = 0.22 \pm 0.19$ ;  $r_g = 0.30 \pm 0.20$ , respectively).

Table 2. Heritabilities (diagonal) and genetic correlations (above diagonal) of androstenone, skatole and indole.

	LNA	LNS	LNI
Ln (androstenone)	<b>0.72 ±0.11</b>	0.22 ±0.19	0.30 ±0.20
Ln (skatole)		<b>0.63 ±0.11</b>	0.75 ±0.10
Ln (indole)			<b>0.44 ±0.10</b>

Estimates of the genetic correlations of LNA, LNS and LNI with carcass quality traits (ultrasonic backfat depth, HGP loin depth, HGP backfat depth, HGP lean meat percentage and live growth) are presented in Table 3.

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Table 3. Genetic correlations and standard error between androstenone, skatole and indole with carcass quality traits.

	LNA	LNS	LNI
Ultrasonic backfat (BF)	0.00 ±0.16	0.07 ±0.16	0.13 ±0.18
Loin depth (HGPloin)	-0.01 ±0.24	-0.11 ±0.25	0.09 ±0.26
Backfat (HGPbf)	0.20 ±0.18	0.17 ±0.19	0.27 ±0.20
Lean meat % (HGPllean)	-0.20 ±0.18	-0.19 ±0.19	-0.22 ±0.21
Live growth (LGR)	0.34 ±0.16	0.18 ±0.19	0.35 ±0.19

Genetic correlations of LNA, LNS and LNI with BF (ultrasonic) and HGPloin were very weak (-0.11 – +0.13) whereas backfat recorded in the slaughter house (HGPbf) was moderately and positively correlated with all three boar taint compounds (LNA=0.20, LNS=0.17, and LNI=0.27). For lean meat %, similar but negative correlations were observed (LNA=-0.20, LNS=-0.19, and LNI=-0.22). Genetic correlations between boar taint components and live growth were all positive (LNA=0.34, LNS=0.18, and LNI=0.35).

Table 4. Genetic correlations and standard error between androstenone, skatole and indole with female fertility traits.

	LNA	LNS	LNI
Age first insemination (AFI)	-0.24 ±0.24	0.03 ±0.26	0.12 ±0.26
Total number piglets born (TNB)	-0.06 ±0.20	0.16 ±0.20	0.08 ±0.21
Litter mortality (LM)	-0.59 ±0.54	-0.35 ±0.49	- *
Interval weaning-2nd insemination (IWI2)	-0.44 ±0.31	0.34 ±0.36	0.29 ±0.36

\* Genetic correlation could not be estimated.

In Table 4 genetic correlations of LNA, LNS and LNI with four female fertility traits are given. Only LNA showed clear negative genetic correlations with litter mortality (-0.59) and interval weaning- 2<sup>nd</sup> insemination IWI2 (-0.44), and a somewhat lower correlation with age at first insemination (AFI) (-0.24). Litter size appeared not to be related to LNA. LNS and LNI showed very low and positive genetic correlations with AFI and TNB (0.03-0.16), and somewhat higher and positive values for IWI2 (0.34 and 0.29). However, litter mortality showed a moderate negative genetic correlation with LNS (-0.35).

## Discussion

Excessive concentrations of androstenone in pork fat may reach levels of 100 µg/g, while the concentration threshold for perception is about 0.5 µg/g with a range from 0.2 to 1.0 µg/g (Rhodes, 1971; Claus et al. 1994; Di Natale et al. 2003; Aldal et al. 2004) and a mean group threshold of 0.426 µg/g (Annor-Frempong *et al.* 1997a). The concentration of skatole in pork fat may reach 4.0 µg/g while the concentration threshold for acceptability is about 0.2 µg/g. In Norway, skatole levels ≥ 0.21 µg/g fat have been used as cut off level (Aldal *et al.* 2004). The androstenone levels found in this study (59% above 1.0 µg/g) are clearly higher than in a large international study of Walstra *et al.*, (1999) where about 30% had levels above 1.0 µg/g. To the contrary only 6% of the boars

in the present study had skatole levels above the threshold of 0.2 µg/g. This percentage is lower than the results found in the study of Walstra et al. (1999), where more than 15% of the entire males had levels above 0.2 µg/g and more than 10% had levels above 0.25 µg/g; with a large variation between countries.

Heritability of LNA was high but in line with literature. Heritabilities of LNS and LNI were somewhat higher than heritabilities found in other studies (skatole: 0.19- 0.34, Pedersen, 1998; skatole and indole: 0.55 and 0.38, respectively, Tajet et al., 2005). These heritabilities indicate the possibility for effective selection against androstenone, skatole and indole. Genetic correlations between LNA, LNS and LNI were comparable to the study of Tajet et al. (2005).

Effect of selection against androstenone, skatole or indole might affect carcass quality. However, the genetic correlations of LNA, LNS and LNI with BF (ultrasonic) and HGPloin were negligible (-0.11 – 0.13), and a low but positive genetic correlation was found between androstenone and backfat. Comparable results were found in the study of Sellier et al. (2000). Backfat recorded in the slaughter house (HGPbf) was moderately and also positively correlated with LNA, LNS and LNI, which was expected to have the same genetic correlation as ultrasonic backfat. The number of animals with HGP backfat measurements was larger than the animals with ultrasonic backfat measurements (n= 995 vs. 798), and this might explain the difference. The positive correlations of LNA, LNS and LNI with HGPbf and negative correlations with HGPlan are favourable. Selecting against androstenone, skatole or indole might result in a decrease of backfat depth and increase of lean meat percentage. Comparable relationships of androstenone with backfat and lean meat percentage were found by Willeke and Pirchner (1989). In the latter study a selection experiment was done with entire males, which were selected against androstenone. The selected line had lower androstenone levels, backfat and body weight was lower and lean meat percentage was higher. In the present study, also the genetic correlation between live growth and boar taint compounds were calculated and turned out to be positive, which is unfavourable. Because the effects on backfat, lean meat percentage and live growth are adverse, we might conclude that selection against androstenone, skatole or indole will not have large effects on finishing traits. Additionally, selection in boar lines is not expected to have large consequences for boar taint, based on the results of the present study.

Although a decrease of boar taint might be achieved quite simple and fast, consequences of selection against androstenone, skatole or indole for female fertility are not yet well determined. Selection experiments for high or low levels of androstenone have been successful but were associated with delayed puberty in litter-mate gilts (Willeke *et al.*, 1987; Sellier and Bonneau, 1988). In a French selection experiment, index selection for a reduction in androstenone content without any adverse effects on reproduction - by controlling the size of the bulbourethral glands - was only partly successful, due to deviations of the estimated genetic parameters from the expected ones and thus the use of erroneous weights in the selection index (Sellier *et al.*, 2000). The results of the present study point also towards negative effects on female fertility. Especially breeding for low levels of androstenone might result in late maturing animals. This problem will not be relevant for finishing pigs, but selection against boartaint only in sire lines might not be enough if fertility selection in dam lines will favour boartaint components. The relation between LNA and IW12 also was clearly negative, animals might be later in heat after the first litter when selecting for a low level of androstenone. However, genetic correlations

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of LNS and LNI with AFI were low and even positive with IWI2, indicating possibilities for selection against skatole and indole without having problems like late maturing animals or animals being later in heat after first litter. It is known that skatole increases too around puberty (Bonneau, 1982), and selection against high levels of skatole might delay puberty onset resulting in later maturing animals. Genetic correlations between LNA and LNI with TNB were small and therefore litter size is not expected to be affected by selection against high levels of androstenone or indole. Correlation between LNS and TNB was low; therefore a negative effect on litter size is not expected. Litter mortality might also increase when selecting against skatole because of the moderate and negative correlation of LNS with litter mortality. To conclude, selection against boar taint compounds might have negative effects on dam lines. Selection in dam lines more than selection in sire lines might be a necessity for reduction of boar taint in finishing pigs.

## Conclusions

- Heritabilities show ample opportunities for selection against androstenone, skatole and indole.
- Genetic correlations of androstenone, skatole and indole with lean meat are low, but favourable; with gain correlation was a bit negative; current selection does not seem to increase boar taint too much.
- Genetic correlations of androstenone with female fertility, however, indicate risks in terms of higher litter mortality, later sexual maturation and animals later in heat after the first litter. Current selection in dam lines might be a larger risk than in sire lines.

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