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Running head: Genetic Parameters for Boar Taint

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Abstract. Boar taint is an offensive odor, which affects the smell and taste of cooked pork, resulting mainly from the accumulation of skatole and androstenone in the back fat of intact males. The aim of the study was to estimate genetic parameters for skatole and androstenone and their genetic relationship to production and litter size traits. Concentrations of skatole and androstenone in the back fat were available for approximately 6,000 and 1,000 Landrace boars, respectively. The concentrations were log-transformed to align phenotypic measures to a normal distribution. Heritability estimates for Log(skatole) and Log(androstenone) were 0.33 and 0.59, respectively. The genetic correlation between the two measures of boar taint was 0.37, suggesting that genetic selection against boar taint based on only one of the chemical compounds could be insufficient. The boar taint compounds had low and mostly favorable genetic correlations with the production traits. Most noticeable, a favorable genetic correlation of −0.20 between meat percentage and Log(skatole) was estimated and hence continued selection for lean pigs can also slowly reduce the level of boar taint if the desired carcass weight is kept constant. The relationship between litter size traits (measured on sows related to boars) and boar taint compounds was low and not significantly different from zero. In conclusion, skatole and androstenone can be reduced through selection without affecting important economical production and litter size traits. Thus, animal breeding offers an effective and sustainable solution to surgical castration of male piglets.

Key words: Boar taint, breeding, animal welfare, pigs

INTRODUCTION

Boar taint is characterized by an offensive taste or odor of the meat that emits during cooking, which makes it unpleasant for consumers. It is caused primarily by the compounds androstenone and skatole, which accumulate mainly in the back fat of intact males (Zamaratskaia and Squires, 2008). A significant proportion of all carcasses from intact males are classified as tainted pork unless surgical or immunological castration of male piglets at 2-4 days of age is carried out. Castration reduces the concentrations of skatole and androstenone in fat under the threshold levels starting mostly at 0.20–0.25 ug/g for skatole and 0.5–1 ug/g for androstenone. Public pressure to abandon castration has led stakeholders (i.e., producers, meat industry, retailers, scientists, veterinarians and animal welfare NGOs) within the European Union to sign a voluntary declaration to end castration practices by January 1, 2018 (EU, 2012). It is well established that a fraction of the Danish and other pig populations carry genes that enable entire male pigs to develop boar taint (e.g. Pedersen, 1998). Reduction of boar taint through genetic selection is promising, because concentrations of boar taint compounds are moderately to highly heritable (e.g. Grindflek et al., 2011; Robic et al., 2008).

The profitability of intensive swine production systems depends mainly on the number of healthy and vital piglets born, their growth rate from birth to slaughter and their efficiency of utilization of resources, e.g. feed. Several pig breeding companies or organizations in the European Union, including Denmark, are currently exploring possibilities to include boar taint in the industry breeding goal. Hence, estimation of genetic correlations of boar taint to economically important traits such as production and litter size traits is critical because these will be used to construct a balanced selection index that places simultaneous selection emphasis on these traits and boar taint compounds.

The objective of the study was to estimate genetic parameters for boar taint compounds and their relationship to key production and litter size traits included in the breeding goal for Danish Landrace.

MATERIALS AND METHODS

Boar taint traits. The concentration of the boar taint compounds, androstenone and skatole-equivalents, were measured in carcass fat samples. The fat samples were taken post slaughter from the neck area of the carcass and were stored at -20°C. A skatole-equivalent represented a combined measurement of skatole and indole and it was measured by a calorimetric method (Mortensen and Sørensen, 1984). In addition, androstenone was measured by the Norwegian School of Veterinary Sciences (**NVH**) on a subset of the boars. Levels of androstenone were analyzed by modified time-resolved fluoroimmunoassay (Tuomola et al., 1997), using an antibody (Andresen, 1974) produced at NVH. The original dataset contained skatole-equivalent concentrations on 6,166 intact males and androstenone concentrations on 1,002 intact males. Androstenone, which is more expensive to measure, was selectively phenotyped on pairs of full sibs. More specifically, 501 pigs with very high skatole (>0.3 μg/g) at slaughter were identified and matched with a low skatole litter mate. Boar taint records that could not be matched with observations on age at slaughter and carcass weight were discarded. Furthermore, each skatole-equivalent observation had to belong to a contemporary group containing at least five records to be included in the analyses. After these data edits, the final dataset consisted of 5,936 and 920 records of skatole-equivalent and androstenone, respectively. Finally, the natural logarithm was applied to transform the boar taint traits to normality. In the remainder of the article, the transformed boar taint traits will be termed Log(skatole) and Log(androstenone).

Production traits. The production traits were average daily gain (**ADG**) from birth to 30 kg (**ADG30**) and from 30 to 100 kg BW (**ADG100**) [g/d], meat percentage (**MP**) and feed conversion ratio (**FCR**) [feed units/kg BW gain], which are traits considered in the Danish routine genetic evaluations. The meat percentage is predicted based on ultrasound recordings of back fat thickness and live weight at the time of scan, using a prediction equation that is regularly updated from data on total dissected meat. Records on ADG and MP were considered from pigs which had phenotypic records on Log(skatole), their male or female full and half sibs and all contemporary group members of these pigs. These data consisted of records on both intact males and females. Data were edited such that the contemporary group size consisted of at least five records, having 18,966 records for consideration in the analyses. Feed intake records were available from the national test station, yielding 1,102 records on FCR. Descriptive statistics for the production traits included in the dataset are presented in Table 1.

Production traits - pedigree. A pedigree for animals that had records on either boar taint or production traits was constructed. The pedigree was traced back 5 generations and it contained 25,110 animals including 18,966 pigs with phenotypic records. These descended from 241 sires and 2,829 dams. The size of the base population was 417 animals.

Litter size traits. Recordings on total number of piglets born (**TNB**) piglets and live piglets at day five (**LP5**) were considered from sows which had male full and half sibs with phenotypic records on Log(skatole) and all contemporary group members of these sows. We considered only data from the first parity, pure breed pigs, and contempory group sizes of at least five records. The survival rate until day five (**SV5**) was expressed as LP5/TNB. This yielded 35,715; 34,991

and 34,991 records for TNB, LP5 and SV5, respectively. It must be mentioned that litter size traits are recorded more frequently than production traits, because litter size traits are recorded in both nucleus and multiplier herds in Denmark, explaining the larger data size despite the fact that the traits are only measured on sows. Descriptive statistics for the litter size traits included in the analyses are presented in Table 1.

Litter size traits - pedigree. A pedigree for animals that had records on either boar taint or litter size traits was constructed. The pedigree was traced back 5 generations and it contained 55,047 animals including 43,070 animals with phenotypic records. These descended from 1,325 sires and 12,588 dams. The size of the base population was 563 animals.

Bivariate linear mixed models. All genetic parameters were estimated using animal models where informative fixed effects were derived from multiple regression analyses prior to fitting the multi-trait animal models. First, a 2-trait linear mixed model was specified and fitted to the boar taint data, establishing relationships between boar taint compounds. Second, a series of 2-trait models were specified for boar taint and production traits which were also used to generate starting values for the variance components for the final 6-trait model.

A bivariate model to estimate correlations between Log(skatole) and Log(androstenone) was as follows:

$$\begin{bmatrix}\mathbf{y}_1\\ \mathbf{y}_2\end{bmatrix}=\begin{bmatrix}\mathbf{X}_1 & \mathbf{0}\\ \mathbf{0} & \mathbf{X}_2\end{bmatrix}\begin{bmatrix}\mathbf{b}_1\\ \mathbf{b}_2\end{bmatrix}+\begin{bmatrix}\mathbf{Z}_{a_1} & \mathbf{0}\\ \mathbf{0} & \mathbf{Z}_{a_2}\end{bmatrix}\begin{bmatrix}\mathbf{a}_1\\ \mathbf{a}_2\end{bmatrix}+\begin{bmatrix}\mathbf{Z}_{c_1} & \mathbf{0}\\ \mathbf{0} & \mathbf{Z}_{c_2}\end{bmatrix}\begin{bmatrix}\mathbf{c}_1\\ \mathbf{c}_2\end{bmatrix}+\begin{bmatrix}\mathbf{e}_1\\ \mathbf{e}_2\end{bmatrix}$$

Where $\mathbf{y}_1$ and $\mathbf{y}_2$ were vectors of observations on Log(skatole) and Log(androstenone), which were denoted by indices 1 and 2, respectively; $\mathbf{b}_i$, $\mathbf{a}_i$, $\mathbf{c}_i$, and $\mathbf{e}_i$ were vectors of solutions for the two boar taint traits, which were fixed, random additive genetic, litter and residual effects,

respectively. The vectors of fixed effects for $\mathbf{y}_1$ and $\mathbf{y}_2$ were: $\mathbf{b}_1 = [\mu, HYS, ASL, WSL]'$ and $\mathbf{b}_2 = [\mu, HERD, ASL, WSL]'$, where μ = the overall mean; HYS (Herd-Year-Season) and Herd were categorical variables; *ASL* and *WSL* denoted the continuous regression variables; age (in days) and carcass weight (in kg) at slaughter. These were confounded partially, as the Spearman rank correlation was estimated to 0.40. We chose to use both variables to adjust for systematic differences in sexual maturation between boars. This approach assumed that sexual maturity is related to both weight and age. Higher order terms for *ASL* and *WSL* were not significant (P > 0.10) in preliminary multiple regressions analyses and therefore not considered further. Design matrices $\mathbf{X}_1$ and $\mathbf{X}_2$ related fixed effects to $\mathbf{y}_1$ and $\mathbf{y}_2$. Index matrices $\mathbf{Z}_a$ and $\mathbf{Z}_c$ allocated records to random effects were known indices matrices allocating records to random effects. The random effects were assumed to be independent and normally distributed with mean zero. The (co)variance structure among random effects was defined as

$$\text{var}\begin{bmatrix}\mathbf{a}\\ \mathbf{c}\\ \mathbf{e}\end{bmatrix} = \begin{bmatrix}\mathbf{A}\otimes\mathbf{G} & \mathbf{0} & \mathbf{0}\\ \mathbf{0} & \mathbf{I}\otimes\mathbf{C} & \mathbf{0}\\ \mathbf{0} & \mathbf{0} & \mathbf{I}\otimes\mathbf{R}\end{bmatrix}$$

where **A** is the matrix of additive genetic relationships among animals in the pedigree (Quaas, 1976) and **I** is an identity matrix. Here **G**, **C** and **R** were full unstructured (co)variance matrices.

Multi-trait model to estimate parameters for production and boar taint traits. A 6-trait model was specified on the basis of the bivariate mixed model analyses, i.e.

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z}_a\mathbf{a} + \mathbf{Z}_c\mathbf{c} + \mathbf{Z}_p\mathbf{p} + \mathbf{e}$$

where **y** was the vector of observations [ADG30, ADG100, FCR, MP, Log(skatole), Log(androstenone)]; **b** was the vector of fixed effects which were different for the different traits.

All equations for production traits included HYS effects and equations for traits ADG30, ADG100 and FCR included a BW continuous regression variable, which was adjusted to 30 kg BW, accounting for systematic differences in the planned BW recordings at 30 kg. In addition, fixed effects were defined above for the boar taint compounds, being continuous regression variables *ASL* and *WSL*. The random effects were: **a**, the vector of additive genetic effects; **c**, the vector of litter of birth effects; **p**, the vector of pen effects (included only for traits ADG100 and MP); and **e** was the vector of random residuals. Design matrices **X**, $\mathbf{Z}_a$, $\mathbf{Z}_c$ and $\mathbf{Z}_p$ were incidence matrices associating **b**, **a**, **c** and **p** with **y**. The random effects **a**, **c**, **p** and **e** were assumed to be mutually independent and normally distributed:

$$\mathbf{a} \sim \mathrm{N}(0, \mathbf{A} \otimes \mathbf{G}),\ \mathbf{c} \sim \mathrm{N}(0, \mathbf{I} \otimes \mathbf{C}),\ \mathbf{p} \sim \mathrm{N}(0, \mathbf{I} \otimes \mathbf{P}),\ \mathbf{e} \sim \mathrm{N}(0, \mathbf{I} \otimes \mathbf{R})$$

where **G**, **C**, **P**, and **R** were (co)variance matrices for additive genetic effects, litter of birth effects, pen effects, and residuals, respectively, **A** was the matrix of additive genetic relationships among animals in the pedigree and **I** was the identity matrix. Here **G** and **R** were full unstructured (co)variance matrices, while all covariance parameters between traits in **C** and **P** were set to zero. This was possible, because the estimated correlations between traits in these random effects were not significantly different from zero based on the bivariate animal model analyses. These variance restrictions ensured proper convergence of the AI-REML routine.

Multi-trait models to estimate parameters for litter size and boar taint traits. Estimation of genetic and environmental parameters for litter size and boar taint traits were performed in 3-[SV5, Log(skatole), Log(androstenone)] or 4-trait [TNB, LP5, Log(skatole), Log(androstenone)] linear mixed model analyses. Model descriptions were limited to the four trait model, because

the two models were similar (i.e. same explanatory effects). The 4-trait model to describe the observations for the *i*'th trait $i = \{1,...,4\}$ was

$$\begin{bmatrix}\mathbf{y}_1\\ \mathbf{y}_2\\ \mathbf{y}_3\\ \mathbf{y}_4\end{bmatrix} = \begin{bmatrix}\mathbf{X}_1 & \mathbf{0} & \mathbf{0} & \mathbf{0}\\ \mathbf{0} & \mathbf{X}_2 & \mathbf{0} & \mathbf{0}\\ \mathbf{0} & \mathbf{0} & \mathbf{X}_3 & \mathbf{0}\\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{X}_4\end{bmatrix}\begin{bmatrix}\mathbf{b}_1\\ \mathbf{b}_2\\ \mathbf{b}_3\\ \mathbf{b}_4\end{bmatrix} + \begin{bmatrix}\mathbf{Z}_{ps_1} & \mathbf{0} & \mathbf{0} & \mathbf{0}\\ \mathbf{0} & \mathbf{Z}_{ps_2} & \mathbf{0} & \mathbf{0}\\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0}\\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0}\end{bmatrix}\begin{bmatrix}\mathbf{p}_{s_1}\\ \mathbf{p}_{s_2}\\ \mathbf{0}\\ \mathbf{0}\end{bmatrix} + \begin{bmatrix}\mathbf{Z}_{d_1} & \mathbf{0} & \mathbf{0} & \mathbf{0}\\ \mathbf{0} & \mathbf{Z}_{d_2} & \mathbf{0} & \mathbf{0}\\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0}\\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0}\end{bmatrix}\begin{bmatrix}\mathbf{d}_1\\ \mathbf{d}_2\\ \mathbf{0}\\ \mathbf{0}\end{bmatrix}$$
$$+ \begin{bmatrix}\mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0}\\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0}\\ \mathbf{0} & \mathbf{0} & \mathbf{Z}_{a_3} & \mathbf{0}\\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{Z}_{a_4}\end{bmatrix}\begin{bmatrix}\mathbf{0}\\ \mathbf{0}\\ \mathbf{a}_3\\ \mathbf{a}_4\end{bmatrix} + \begin{bmatrix}\mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0}\\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0}\\ \mathbf{0} & \mathbf{0} & \mathbf{Z}_{c_3} & \mathbf{0}\\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{Z}_{c_4}\end{bmatrix}\begin{bmatrix}\mathbf{0}\\ \mathbf{0}\\ \mathbf{c}_3\\ \mathbf{c}_4\end{bmatrix} + \begin{bmatrix}\mathbf{e}_1\\ \mathbf{e}_2\\ \mathbf{e}_3\\ \mathbf{e}_4\end{bmatrix}$$

where $\mathbf{y}_1$, $\mathbf{y}_2$, $\mathbf{y}_3$ and $\mathbf{y}_4$ were a vector of records for TNB, LP5, Log(skatole) and Log(androstenone), respectively; $\mathbf{X}_1$, $\mathbf{X}_2$, $\mathbf{X}_3$ and $\mathbf{X}_4$ were design matrices relating fixed effects in $\mathbf{b}_1$, $\mathbf{b}_2$, $\mathbf{b}_3$ and $\mathbf{b}_4$ to $\mathbf{y}_1$, $\mathbf{y}_2$, $\mathbf{y}_3$ and $\mathbf{y}_4$, respectively. The fixed effects in the model involving TNB ($\mathbf{b}_1$), LP5 ($\mathbf{b}_2$), Log(skatole) ($\mathbf{b}_3$) and Log(androstenone) ($\mathbf{b}_4$) were:

$$\mathbf{b}_1 = \begin{bmatrix}\mu\\ HYQ\\ AGE\\ AGE^2\end{bmatrix},\ \mathbf{b}_2 = \begin{bmatrix}\mu\\ HYQ\\ AGE\\ AGE^2\end{bmatrix},\ \mathbf{b}_3 = \begin{bmatrix}\mu\\ HYS\\ ASL\\ WSL\end{bmatrix},\ \mathbf{b}_4 = \begin{bmatrix}\mu\\ Herd\\ ASL\\ WSL\end{bmatrix}$$

where μ = the overall mean; HYQ (Herd-Year-Quarter) effects, Herd effects and HYS were categorical variables; *AGE* and AGE^2 were linear and quadratic effects of the continuous regression variable, age at first mating (days); *ASL* and *WSL* denoted the continuous regression variables age (days) and carcass weight at slaughter (kg).

The vector $\mathbf{p}_s$ contained the permanent service sire effects; **d** was the vector of genetic effects of the sow; **a** was the vector of direct animal genetic effects; **c** was the vector of litter effects; **e** was the vector of random residuals; and $\mathbf{Z}_{ps}$, $\mathbf{Z}_d$, $\mathbf{Z}_a$ were incidence matrices associating $\mathbf{p}_s$, **d**, and **a** with **y**. The random effects were assumed to be independent of each other, except for

d and **a**, which were assumed to be correlated via the additive genetic relationship matrix **A**, describing the relationships among animals in the pedigree. All random effects were assumed to be normally distributed. Thus,

$$\mathbf{p_s} \sim N(\mathbf{0}, \mathbf{I} \otimes \mathbf{S_0}),\ \mathbf{c} \sim N(\mathbf{0}, \mathbf{I} \otimes \mathbf{C_0}),$$
$$\begin{bmatrix} \mathbf{d} \\ \mathbf{a} \end{bmatrix} \sim N(\mathbf{0}, \mathbf{A} \otimes \mathbf{G_0}),\ \mathbf{e} \sim N(\mathbf{0}, \mathbf{I} \otimes \mathbf{R_0})$$

where $\mathbf{S_0}$, $\mathbf{C_0}$, $\mathbf{G_0}$, and $\mathbf{R_0}$ represented covariance matrices for permanent effects service sire, litter effects, genetic effects of sow, and direct animal genetic effects, and residuals, respectively, **I** was the identity matrix of appropriate size. The residual covariances between litter size traits and boar taint compounds were assumed to be zero, because litter size traits were collected on sows and boar taint compounds were collected on slaughter boars. Based on the covariance structures defined above, the phenotypic variances were defined as $\sigma_p^2 = \sigma_{p_s}^2 + \sigma_d^2 + \sigma_e^2$ for litter size traits and $\sigma_p^2 = \sigma_c^2 + \sigma_a^2 + \sigma_e^2$ for boar taint compounds.

An additional analysis was conducted for the SV5 trait on the arc-sine scale, $SV5_t = \arcsin(\sqrt{SV5})$, because it is often used for binomial proportions. In the transformation, a zero survival rate was counted as 1/4n and a 100% survival rate as (n-1/4)/n, where n was litter size at birth. The inferences based on the transformed scale were almost identical to those based on the original scale and thus, we chose to present results on the original scale.

All parameters were estimated using the average information residual maximum likelihood algorithm (Jensen et al., 1997) as implemented in the DMU software (Madsen and Jensen, 2008). Standard errors of heritabilities and genetic and phenotypic correlations were calculated from the average information matrix at convergence by means of the delta method

e.g., Dodenhoff et al. (1998). The genetic parameter estimates were considered significantly different from zero when the estimate deviated by more than 1.96 x SE from zero.

RESULTS

Boar taint compounds. The raw averages of skatole-equivalent and androstenone concentrations are 0.197 and 1.080 ug/g back fat (Table 1). Variation is also substantial, as the coefficient of variation is close to 100% for both traits. The concentrations of the two boar taint compounds are plotted against each other in Figure 1 with suggested sensory thresholds added (Mortensen et al., 1986). Within the sensory thresholds (or "safe box"), a carcass can be declared free of boar taint (Mortensen et al., 1986). This means that the concentrations of skatole and androstenone in carcass back fat are below 0.25 and 1.0 ug/g back fat, respectively, for both compounds (Mortensen et al., 1986). Based on this classification, only 35% of all carcasses will be declared free of boat taint. The boar taint classification practice in Denmark is currently based on the skatole-equivalent only, ignoring the androstenone component, and hence 46% of all carcasses will be declared free of boat taint.

A significant proportion of the variation in boar taint is of genetic origin, which is confirmed by the moderate to high heritabilities for both compounds (Table 2). These amount to 0.33 (SE = 0.05) and 0.59 (SE = 0.14) for Log(skatole) and Log(androstenone), respectively. The litter variance account for a smaller ($\leq$ 5%), but significant proportion of the phenotypic variance in Log(skatole). The genetic correlation (based on the bivariate animal model) between Log(skatole) and Log(androstenone) is 0.37 (SE = 0.15), positive and significantly different from zero (Table 2).

Production traits and boar taint compounds. Heritabilities of ADG30, ADG100, MP and FCR and genetic correlations to boar taint compounds, which are estimated in either bivariate or multivariate animal model analyses, are presented in Tables 2 and 3. In general, the multi-trait estimates presented in Table 3 yield minor differences to those estimates present in Table 2. The heritabilities of ADG30 and ADG100 are moderate (i.e. 0.22 and 0.30) while MP is a highly heritable trait where the heritability is estimated to 0.48. The contribution of the litter variance to the phenotypic variance is small for most traits, but significant, while its contribution to the ADG30 phenotypic variance is substantial (i.e., 0.17). The heritability of FCR is estimated to 0.17 and 0.16 in the bivariate and multivariate analyses, respectively.

The genetic correlations between production traits and boar taint compounds are generally small, tend to be favorable, but in most cases not significantly different from zero. The exception is the ADG30 trait where low unfavorable correlations were estimated ($0.17 < r_g < 0.28$). Meat percentage and Log(skatole) is correlated genetically ($-0.22 < r_g < -0.20$), pointing in a favorable direction. Hence, selection for increasing MP will result in decreasing skatole levels. Consequently, moderate selection emphasis on boar taint in Danish Landrace is expected to have limited effects on the production traits and vice versa.

Litter size traits and boar taint compounds. The genetic parameters for litter size traits and boar taint compounds are presented in Table 4. The heritability based on the sow component is 0.09 (SE = 0.01) and 0.06 (SE = 0.01) of TNB and LP5, respectively, and the heritability of SV5 was 0.09 (SE = 0.01) and at the same level as TNB. The genetic correlation between boar taint and litter size (based on the sow component) are low ($-0.18 < r_g < 0.06$) and not significantly

different from zero. Genetic correlations between boar taint and piglet survival are low ($0.01 < r_g < 0.03$).

DISCUSSION

Boar taint compounds. The average skatole-equivalent level was substantially higher in the current data compared to the previous investigations reported by Pedersen (1998). The data collection scheme for the current study differed from the scheme described by Pedersen (1998), because slaughter boars from all Landrace nucleus herds were sampled and not only those that were reared and performance tested at the National test station. Different management procedures in nucleus herds may have affected the phenotypic skatole levels. In addition, the carcass weight was higher in the current study compared to the study of Pedersen (1998), but comparable to the current Danish industry basis of 70 to 90 kg carcass weight. For several carcasses both of the boar taint compounds were more than twice as high than the respective "safe" thresholds (Figure 1), indicating that selection against boar taint will probably be desirable in this breed, although it only contributes with ¼ of the additive genetic merit of Danish production pigs. In an earlier investigation in Danish Landrace, the heritability of skatole in back fat was reported as 0.27 (Pedersen, 1998), which was close to the current estimate. As reviewed by Robic et al. (2008), Log(skatole) show medium to high heritability values ranging from 0.19 to 0.54 and hence the current estimate was in middle of the reported range. The estimate in the present study was somewhat lower than reported by Tajet et al. (2006) who estimated a heritability of 0.55, while Grindflek et al. (2011) reported 0.41 for skatole in the same population of Norwegian Landrace. Heritability of skatole has been reported on several occasions in Dutch

populations (e.g. Engelsma et al., 2007) and Windig et al. (2012) recently reported a heritability of 0.41, which was slightly higher than our estimate.

The heritability of Log(androstenone) has been previously estimated in Danish Landrace. Jonsson and Andresen (1979), computed the heritability of Log(androstenone) to 0.54, but the estimate had limited precision, i.e. the SE was 0.32. The point estimate agreed very well with the estimate derived herein. The authors also developed a boar taint index, which was a linear combination of Log(androstenone) and an olfactory measurement, showing a heritability of 0.46. The literature survey of Robic et al. (2008) reported heritabilities ranging from 0.25 to 0.88 for Log(androstenone) and the current estimate was close to the average reported estimate in the literature. Tajet et al. (2006) and Grindflek et al. (2011) reported heritabilities of 0.54 and 0.49, respectively, for Norwegian Landrace. The average androstenone concentration in back fat in the Danish Landrace was comparable to the phenotypic level observed in Norwegian Landrace, i.e. 1.14 versus 1.08 ug/g, suggesting that the two breeds are quite comparable in the androstenone trait. Based on the current investigation and previously published work in Denmark and other European countries, it can be stated that Log(skatole) and Log(androstenone) were moderately to highly heritable, which was expected.

The genetic correlation between the two boar taint compounds in Danish Landrace has not previously been estimated. Estimates for the genetic correlation between Log(skatole) and Log(androstenone) were reported to be 0.32 – 0.36 in Norwegian Landrace (Grindflek et al., 2011; Tajet et al., 2006), which corresponds very well with the current estimates of 0.35 – 0.43 based on the different models that were fitted (Tables 2, 3 and 4). Windig et al. (2012) reported a genetic correlation of 0.37 between the two traits in the Dutch populations, a blend of different breeds and lines. We chose to use the carcass weight as a fixed effect even though the variation

in carcass is in part genetic. We explored the consequence of this approach by treating carcass weight as a third trait and obtained a heritability of 0.15 (SE = 0.04), confirming our assumption. However, it did not change the estimate of the genetic correlation between boar taint compounds significantly as the estimate was 0.42 (SE = 0.14). In addition, other recent genetic analyses of boar taint traits in other populations have included age at slaughter and carcass weight as fixed effects (Windig et al. 2012; Baes et al. 2012).

These results suggest that selection emphasis should probably be placed on both compounds if the selection is to be based directly on the chemical components. This would also result in a significant reduction in indole, because the trait (skatole-equivalent) was a mixture of skatole and indole. Moreover, there has been a report of a high genetic correlation between the pure substances indole and skatole in back fat of boars (Grindflek et al., 2011; Windig et al., 2012) and hence collecting indole as a separate trait is not necessary in a future breeding scheme. Finally, it should be noted that in the literature the boar taint effect of indole is considered secondary to that of skatole and androstenone (Zamaratskaia and Squires, 2008).

Genetic correlations among production traits and boar taint compounds. The heritabilities for ADG30, ADG100 and MP were close to those used in routine genetic evaluations, while the heritability of FCR was significantly lower than the estimate for the entire Landrace population. Two earlier studies conducted in Danish Landrace have estimated the genetic correlation between ADG100 and boar taint. Both studies found genetic correlations to be low and not significantly different from zero (Jonsson and Andresen, 1979; Pedersen, 1998). Thus, the current estimates were in agreement with these findings, which are also supported by Windig et al. (2012).

The genetic correlation between skatole and MP has been previously reported by Pedersen (1998), resulting in an estimate of -0.21, which was close to our estimates (Tables 2 and 3). Meat percentage is approximately the inverse of back fat thickness. Windig et al. (2012) reported low positive genetic correlations between carcass fat depth and boar taint compounds, supporting the results obtained in this study. Selection to increase lean meat content in pigs may slowly decrease the concentration of skatole-equivalents and androstenone and hence prevalence of boar tainted carcasses. In Denmark, the average carcass weight has increased from 77 to 82 kg during the last 10 years, and thus the correlated response to selection for lean meat content may have had a very limited effect on boar taint prevalence due to the increased carcass weight. The genetic relationship between boar taint and FCR has not been previously reported in the literature. The genetic correlation points in a favorable direction although the estimates were estimated with low precision due to the small number of records for the trait. Hence, these estimates should be treated with some caution and more data is needed to improve the accuracy of these estimates. Nonetheless, these results suggest that feed efficient animals would tend to be low in skatole. Hence, continued selection for feed efficiency would slowly reduce the concentration of skatole in backfat. The biological mechanism behind this finding cannot be deduced from the current study, but possible links between rates of nutrient metabolism in the liver and its skatole clearance capacity may exist. Finally, selection against skatole would have positive effects in both males and females because it is associated with environmental conditions and bacterial digestion of tryptophan in the gut of the pig.

Litter size traits and boar taint compounds. The heritability of TNB was higher than that of LP5, which was in agreement with Su et al. (2007). The heritability of SV5 was the same as that

of TNB, but lower than previously reported by Su et al. (2007). The genetic correlation between boar taint compounds and dam genetic effects on litter size and survival rate was low and not significantly different from zero. The literature estimates of these correlations are scarce, but Engelsma et al. (2007) reported similar estimates for TNB as presented here. Litter mortality and Log(androstenone) have been reported to be unfavorable genetically correlated (r_g = -0.59) by Engelsma et al. (2007), which are contrary to our results, being an estimate of 0.03 between Log(androstenone) and SV5. It must be noted that the results of their analysis was based on a substantially smaller dataset (5320 records) compared to the present investigation (35715 records).

The precision of the genetic correlations could be improved if the service sire or AI-boar had phenotypic records for boar taint. Thus, obtaining boar taint information on AI-boars seems to be a necessary step towards fully estimating the association between boar taint and both female and male fertility. Recently, Baes et al. (2012) presented a quantitative performance test for use in live male breeding candidates, which showed that the estimated heritabilities on the basis of data from small tissue samples obtained by biopsy were comparable to our estimates. The literature is scarce in terms of estimates of genetic correlations between economically important traits such as litter size and boar taint compounds. Further research is needed to quantify the genetic relationships between boar taint compounds and litter size traits in other breeds and especially dam lines.

In conclusion, skatole and androstenone can be reduced through selection without negatively affecting important economical production traits. The boar's fertility may be of secondary importance in a general breeding scheme, because in practice, female fertility is the

major trait of direct economic importance to pig producers. However, deterioration of male fertility should be avoided and genetic correlations between boar taint compounds and male fertility traits such as semen quality and libido may not be favorable. Hence future research should focus on estimating these correlations. Nonetheless, animal breeding seems to offer a sustainable solution to avoid surgical castration of male piglets. For an efficient eradication of boar taint, direct selection is necessary. This leads to an enhancement of sustainable pork production from both an animal welfare/ethics as well as an environmental perspective, because intact males are 10 – 15% more feed efficient than their castrated counterparts.

LITERATURE CITED

Andresen, Ø. 1974. Development of a radioimmunoassay for 5α-androst-16-en-3-one in pig peripheral plasma. Acta endocrinologica 76: 377-387.

Baes, C., S. Mattei, H. Luther, S. Ampuero, X. Sidler, G. Bee, P. Spring, and A. Hofer. 2012. A performance test for boar taint compounds in live boars. Animal 1:1-7.

Dodenhoff, J., L. D. Van Vleck, S. D. Kachman, and R. Koch. 1998. Parameter estimates for direct, maternal, and grandmaternal genetic effects for birth weight and weaning weight in Hereford cattle. J. Anim. Sci. 76: 2521-2527.

Engelsma, K., R. Bergsma, B. Harlizius, D. Ducro-Steverink, and E. Knol. 2007. Genetic parameters for components of boar taint and their relation with carcass quality and female fertility. EAAP 2007, Dublin, Ireland, August 26-29.

EU 2012. European Declaration on alternatives to surgical castration of pigs. http://ec.europa.eu/food/animal/welfare/farm/docs/castration_pigs_declaration_en.pdf. (Accessed, September 2012).

Grindflek, E., T. Aasmundstad, H. Hamland, M. Hansen, T. Nome, M. Kent, P. Torjesen, and S. Lien. 2011. Revealing genetic relationships between compounds affecting boar taint and reproduction in pigs. J. Anim. Sci. 89:680-692.

Jensen, J., E. Mäntysaari, P. Madsen, and R. Thompson. 1997. Residual maximum likelihood estimation of (co) variance components in multivariate mixed linear models using average information. Jour. Ind. Soc. Ag. Stat. 49: 215-236.

Jonsson, P., and Ø. Andresen. 1979. Experience during two generations of within lines performance testing, using 5a-androst-16-ene-3-one (5a-androstenone) and an olfactory judgement of boar taint. Ann. Genet. Sél. Anim. 11: 241-250.

Madsen, P., and J. Jensen. 2008. An User's Guide to DMU: A Package for Analysing Multivariate Mixed Models. Version 6, release 5.0. Aarhus University, Foulum, Denmark.

Mortensen, A., C. Bejerholm, and J. K. Pedersen. 1986. Consumer test of meat from entire males, in relation to skatole in backfat. Pages 23–26 in Proc. 32th Eur. Mtg. of Meat Res. Workers, Gent, Belgium.

Mortensen, A., and S. Sørensen. 1984. Relationship between boar taint and skatole determined with a new analysis method. 30th Eur. Mtg. of Meat Res. Workers, Bristol, U.K.

Pedersen, B. 1998. Heritability of skatole in back fat. In: W. K. Jensen, editor, Skatole and boar taint. Danish Meat Research Institute, Roskilde, Denmark, p. 129-136.

Quaas, R. 1976. Computing the diagonal elements and inverse of a large numerator relationship matrix. Biometrics: 949-953.

Robic, A., C. Larzul, and M. Bonneau. 2008. Genetic and metabolic aspects of androstenone and skatole deposition in pig adipose tissue: a review. Genet. Selec. Evol. 40: 581.

Su, G., M. S. Lund, and D. Sorensen. 2007. Selection for litter size at day five to improve litter size at weaning and piglet survival rate. J. Anim. Sci. 85: 1385-1392.

Tajet, H., Ø. Andresen, and T. Meuwissen. 2006. Estimation of genetic parameters of boar taint; skatole and androstenone and their correlations with sexual maturation. Acta Vet. Scand. 48:1-4.

Tuomola, M., R. Harpio, P. Knuuttila, H. Mikola, and T. Lövgren. 1997. Time-resolved fluoroimmunoassay for the measurement of androstenone in porcine serum and fat samples. J. Agric. Food Chem. 45: 3529-3534.

Windig, J., H. Mulder, J. Ten Napel, E. Knol, P. Mathur, and R. Crump. 2012. Genetic parameters for androstenone, skatole, indole and human nose scores as measures of boar taint and their relationship with finishing traits. J. Anim. Sci. 90:2120-2129

Zamaratskaia, G., and E. Squires. 2008. Biochemical, nutritional and genetic effects on boar taint in entire male pigs. Animal 3:1508-1521.

Figure 1. Androstenone concentration in back fat plotted against the corresponding skatole-equivalent concentration. Suggested sensory thresholds (0.25 ug/g skatole-equivalent and 1.0 ug/g androstenone (Mortensen et al., 1986)) have been added to the plot, indicating boar-tainted carcasses

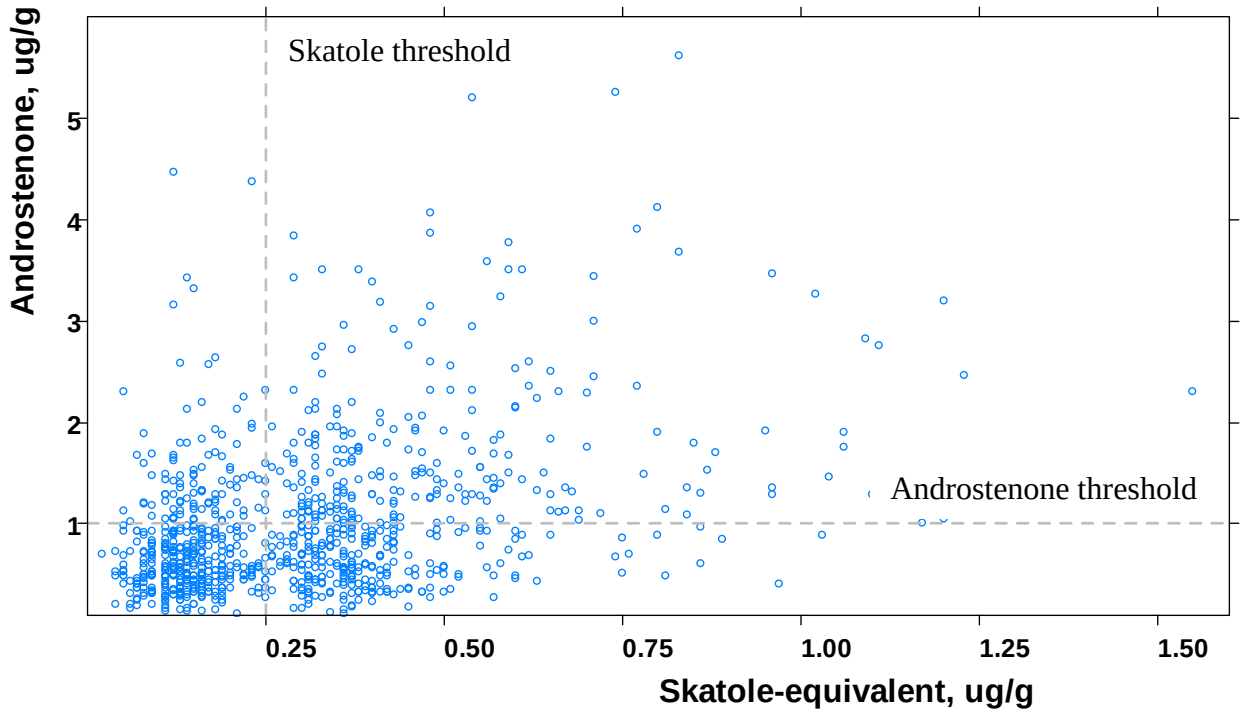

Table 1. Summary statistics for the traits[1] included in the dataset

Trait	No. records	Mean	Standard dev.	Minimum	Maximum
ADG30	17817	377.9	39.02	294.7	586.2
ADG100	18966	946.0	110.0	571.1	1307
FCR	1102	2.390	0.207	1.580	3.310
MP	18966	61.90	1.060	56.20	65.70
TBN	35715	13.56	3.832	1.000	28.00
LP5	34991	10.47	3.453	0.000	22.00
SV5	34991	0.780	0.188	0.000	1.000
Skatole	5937	0.197	0.145	0.020	2.860
Log(skatole)	5937	-1.786	0.545	-3.912	1.051
Androstenone	920	1.080	0.985	0.010	10.60
Log(androstenone)	920	-0.191	0.712	-2.207	2.363

[1]Average daily gain from birth to 30 kg BW (ADG30), average daily gain from 30 to 100 kg BW (ADG100), feed conversion ratio (FCR), meat percentage (MP), total number born (TBN), live piglets at day 5 (LP5) and survival rate until day 5 (SV5).

Table 2. Heritability of Log(skatole) and Log(Androstenone), growth traits (ADG30 and ADG100), meat percentage (MP) and feed conversion ratio (FCR) and additive genetic correlations with boar taint compounds estimated from pairwise bivariate animal model analyses.

Trait	h^2	l^2	Genetic correlations	
			Log(skatole)	Log(androstenone)
Log(skatole)	0.33 ± 0.05	0.05 ± 0.02	1.00	0.37 ± 0.15
Log(Androstenone)	0.59 ± 0.14	0.03 ± 0.06	0.37 ± 0.15	1.00
ADG30	0.22 ± 0.03	0.17 ± 0.01	0.26 ± 0.11	0.28 ± 0.15
ADG100	0.30 ± 0.03	0.04 ± 0.01	-0.06 ± 0.09	0.03 ± 0.12
MP	0.48 ± 0.03	0.03 ± 0.01	-0.22 ± 0.08	-0.13 ± 0.11
FCR	0.17 ± 0.08	0.02 ± 0.05	0.24 ± 0.19	0.06 ± 0.27

Table 3. Heritabilities of traits[1], genetic correlations (above diagonal) and phenotypic correlations (below diagonal) (±SE) among growth traits, feed conversion ratio, meat percentage and boar taint compounds

	ADG30	ADG100	FCR	MP	Log(skatole)	Log(androstenone)
Heritability	0.22 ± 0.03	0.30 ± 0.03	0.16 ± 0.07	0.48 ± 0.03	0.33 ± 0.04	0.59 ± 0.14
Trait						
ADG30		0.25 ± 0.07	-0.19 ± 0.21	-0.13 ± 0.07	0.17 ± 0.09	0.28 ± 0.13
ADG100	0.09 ± 0.01		-0.04 ± 0.18	-0.42 ± 0.05	-0.04 ± 0.08	0.10 ± 0.11
FCR	-0.04 ± 0.04	-0.32 ± 0.03		-0.24 ± 0.16	0.18 ± 0.19	-0.04 ± 0.26
MP	-0.01 ± 0.01	-0.27 ± 0.01	-0.10 ± 0.03		-0.20 ± 0.07	-0.18 ± 0.10
Log(skatole)	0.02 ± 0.02	-0.01 ± 0.02	0.04 ± 0.04	-0.12 ± 0.02		0.35 ± 0.14
Log(androstenone)	0.08 ± 0.04	0.05 ± 0.04	0.14 ± 0.10	-0.15 ± 0.03	0.25 ± 0.03	

[1]Average daily gain from birth to 30 kg BW (ADG30), average daily gain from 30 to 100 kg BW (ADG100), feed conversion ratio (FCR) and meat percentage (MP)

Table 4. The proportion of phenotypic variance that is due to sow and genetic effects on litter size traits[1] and direct animal genetic effects on boar taint compounds are presented in bold face on the diagonal. Genetic correlations between litter size traits and boar taint compounds are presented below the diagonal.

4-trait model[2]	TNB	LP5	Log(skatole)	Log(Androstenone)
TNB	**0.09 ± 0.01**			
LP5	0.57 ± 0.06	**0.06 ± 0.01**		
Log(skatole)	0.06 ± 0.11	0.05 ± 0.13	**0.33 ± 0.05**	
Log(Androstenone)	-0.12 ± 0.15	-0.18 ± 0.18	0.41 ± 0.14	**0.59 ± 0.14**

3-trait model[2]	SV5	Log(skatole)	Log(Androstenone)
SV5	**0.09 ± 0.01**		
Log(skatole)	0.01 ± 0.12	**0.33 ± 0.05**	
Log(Androstenone)	0.03 ± 0.17	0.42 ± 0.14	**0.59 ± 0.14**

[1]Total number born (TBN), live piglets at day 5 (LP5) and survival rate until day 5 = LP5 / TNB

[2]Estimation of genetic and environmental parameters for litter size and boar taint traits were performed in 4-trait [TNB, LP5, Log(skatole), Log(androstenone)] or 3-trait [SV5, Log(skatole), Log(androstenone)] linear mixed model analyses.