Genetic relationships between measures of sexual development, boar taint, health, and aggressiveness in pigs

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ABSTRACT: Breeding intact boars is a promising alternative to surgical castration of piglets. Genetic selection should enable farmers to solve problems due to boar taint and aggressiveness while taking into account potential consequences on other traits of interest. The aim of the study was to estimate genetic relations between sexual development, boar taint, health, and aggressiveness. About 1,600 Pietrain (purebred) or Pietrain × Large White (crossbred) boars were raised in a testing station. Blood samples were collected at about 105 kg BW for measuring sex hormones (testosterone and estradiol) and indicators of the inflammatory status (C-reactive protein [CRP], pig major acute-phase protein [pigMAP], and blood formula). Animals were slaughtered 9 d later and measured for boar taint compounds present in fat (androstenone and skatole) and skin lesions on carcass, an indicator of aggressiveness. For both genetic types, heritability was moderate for sex hormones (from 0.17 to 0.29) and skatole (0.24 for purebred and 0.37 for crossbred), and high for androstenone (0.63 and 0.70 for purebred and crossbred, respectively). Genetic correlations between sex hormones and boar taint compounds were moderate to high (from 0.31 to 0.95). Heritability was moderate for CRP (0.24 and 0.46 for purebred and crossbred, respectively) and very low for pigMAP (0.06 and 0.05 for purebred and crossbred, respectively). Numbers of leukocytes had moderate to high heritabilities according to the genetic type (from 0.21 to 0.52). Heritability of skin lesions was moderate for both genetic types (0.31). Genetic correlations were negative between sex hormones and inflammatory measures (from –0.46 to –0.05), positive between testosterone and number of lesions (0.43 and 0.53 for purebred and crossbred, respectively), and low between androstenone and lesions (–0.06 and –0.17 for purebred and crossbred, respectively). Overall, both breeds of pigs had very similar estimations of heritabilities, but estimates of genetic correlations were different for some pairs of traits. It would be possible to select boars based on their plasma concentration of sex hormones to decrease boar taint and aggressiveness without important consequences on the immune response. However, because of the strong links between boar taint and reproductive function, the possible consequences on the reproductive performance should be evaluated.

Key words: acute-phase proteins, boar taint, genetic parameters, pig, sex hormones, skin lesions

INTRODUCTION

For welfare reasons, the European pig industry is engaged in a voluntary ban of surgical castration of male piglets by 2018. This practice is mainly performed to avoid sexual odors (i.e., boar taint) in pork meat (Lundström et al., 2009). Genetic selection to decrease boar taint is possible (Windig et al., 2012; Haberland et al., 2014) but could have consequences on other characteristics of pigs.

Androstenone, one major odorous compound (Patterson, 1968), is produced increasingly by the testes in parallel to testicular steroids as puberty progresses (Zamaratskaia and Squires, 2009). Skatole, the other major odorous compound, comes from the degradation of tryptophan in the gut of pigs (Jensen et al., 1995). Production of skatole depends mainly on environmental factors but its degradation and fat storage are mainly under the control of testicular steroids (Zamaratskaia and Squires, 2009).

Sex hormones are able to modulate various components of the pig immune system (Merlot et al., 2013), and reciprocally, cytokines may influence Leydig cells activity (Diemer et al., 2003). Among the blood parameters existing to monitor the immune inflammatory response to a threat, proteins of the acute phase reaction (Heegaard et al., 1998) and blood formula (Le Floc’h et al., 2014) are commonly used. Sexual development may increase aggressiveness through testosterone (Soma et al., 2008; Prunier et al., 2013). Number of skin lesions is a good indicator of aggressiveness when measured on pigs after social mixing (Turner et al., 2006a).

There are no genetic information linking boar taint compounds, sex hormones, skin lesions, and immune parameters. Therefore, the aim of the present study was to estimate heritabilities of those parameters and genetic correlations between them in Pietrain and Large White × Pietrain pigs. This will allow predicting possible consequences of a genetic selection against androstenone, skatole, or sex hormones on boar taint and important traits for health and welfare.

MATERIAL AND METHODS

Animals and Management

A total of 749 purebred Pietrain boars and 791 crossbred Pietrain × Large White boars were raised under the same conditions from postweaning to slaughter (110 kg) in a testing station (Le Rheu, France). All animals were raised in 11 successive batches (n = 36 to 118 pigs per genetic type per batch). Purebred and crossbred pigs were offspring from the same 96 purebred Pietrain sires. Pedigree information was available for all boars up to 6 generations. Boars were housed in groups of 10 to 12, with free access to water. Animals were fed ad libitum, at an electronic single-space feeder, with pellets of standard composition (NE = 9.5 MJ/kg, total nitrogenous matter = 163 g/kg, digestible lysine content = 0.94 g/MJ NE, and digestible tryptophan content = 1.7 g/kg).

The experiment was conducted according to the French guidelines for animal care and use (http://ethique.ipbs.fr/sdv/charteexpeanimal.pdf; accessed 15 January 2011).

Traits and Measurements

A blood sample, about 8 mL, was collected in EDTA tubes by direct puncture from the external jugular vein, between 0930 and 1100 h, at 105.4 ± 6.5 kg BW and 9.1 ± 3.8 d before slaughter. Total numbers of lymphocytes and granulocytes were measured with a hematology automatic cell counter calibrated for pigs (MS-9; Melet Schloesing Laboratories, Osny, France). Thereafter, blood samples were centrifuged at 2,500 × g for 10 min at +4°C, and plasma was stored at −20°C until analysis of sex hormones (testosterone and 17β-estradiol) and acute-phase proteins (APP; C-reactive protein [CRP] and pig major acute-phase protein [pigMAP]).

A piece of backfat was sampled on the carcass in the neck region (between cervical and first dorsal ribs) and frozen at −20°C until analysis 53 ± 40 d later for boar taint compounds.

Each slaughter batch was composed, on average, of 36 boars (between 19 and 50). Animals were transferred directly from their pen to the truck without waiting in a lairage area. During transport and lairage at the slaughterhouse, purebred and crossbred boars were separated. However, in each of these 2 subgroups, boars from different pens were grouped together, which could cause aggressions between boars. The total mean duration between departure from the farm and slaughter was 173 min (between 102 and 240 min), with about 20 min of transport and 150 min of lairage at the slaughterhouse.

Skin lesions at slaughter were counted, by experienced staff, on both sides of carcasses without head and legs. All lesions above 2 cm in length were registered. When at least 3 lesions below 2 cm were grouped within 2 cm, they were scored as 1 lesion. The variable analyzed was the cumulated number of skin lesions measured on both sides.

Sex hormones were measured using RIA kits (testosterone: Immunotech, Prague, Czech Republic; estradiol: Orion Diagnostica, Espoo, Finland); CRP and pigMAP were measured using ELISA kits developed for pig APP in plasma (CRP: Genway Biotech, San...
Estimates of genetic parameters in boars

Diego, CA; pigMAP: PigCHAMP, Segovia, Spain). For testosterone, detection limit was 0.2 ng/mL, intra-assay CV was 4.1% at 0.75 ng/mL, and interassay CV was 9.4% at 0.5 ng/mL. For estradiol, detection limit was 2.5 pg/mL; intra-assay CV was 18.1 and 5.0% at 4.6 and 278 pg/mL, respectively; and interassay CV was 17.6 and 9.7% at 3.3 and 490 pg/mL, respectively. For CRP, detection limit was 6.25 ng/mL, interassay CV was 4.6 and 278 pg/mL, respectively; and interassay CV was 11.2% at 55.6 μg/mL. For pigMAP, detection limit was 2.5 pg/mL; intra-assay CV was 17.6 and 9.7% at 3.3 and 490 pg/mL, respectively.

Boar taint compounds (androstenone, skatole, and indole) were measured by HPLC in the backfat sample after heating a piece of fat of about 10 to 20 g, centrifugation at 11,200 × g for 20 min at +4°C, sampling of 2 mL of the supernatant, and extraction of the compounds with methanol (Batorek et al., 2012). Concentrations were expressed per gram of the lipid fraction from adipose tissue. The detection limits of the method were 0.24 μg/g for androstenone and 0.03 μg/g for skatole and indole (Batorek et al., 2012).

Statistical Analyses and Genetic Parameter Estimations

Sex hormones, boar taint compounds, immune blood cells, and skin lesions were normalized by logarithmic transformation; APP were normalized by square root transformation. Detection limits of the assays were assigned to pigs with levels below those limits. This procedure overestimates means of the variables.

First, analyses were conducted to test the breed effect using the PROC MIXED procedure of the software SAS/STAT 9.4 (SAS Inst. Inc., Cary, NC). For each trait, the initial model included the breed, the batch, and the date of sampling as fixed effects and the live weight at measure as a covariate. When a factor or a covariate had a nonsignificant effect (P > 0.10), it was removed from the model, except the breed effect, which was always included. The final statistical models used for all traits are summarized in Table 1.

Second, statistical analyses were performed separately for the 2 genetic types to estimate genetic parameters. The following linear model in matrix notation was used for all traits: y = XB + Za + e, in which y is a vector of observations; β, a, and e are vectors of fixed, additive genetic, and residual effects, respectively; and X and Z are known incidence matrices. The fixed effect retained for each trait was the same one as described in Table 1, without the breed effect. Assumptions for random effects were a ~ N(0, Aσ2a) and e ~ N(0, Ieσ2e), in which A is the matrix of additive relationships among animals, Ie is the identity matrix of appropriate order, and σ2a and σ2e are the additive genetic and residual variances, respectively. Heritability values and genetic correlations were estimated using the REML methodology applied to a multiple trait animal model with the VCE6 software (Neumaier and Groeneveld, 1998). Heritabilities and correlations were considered low (range from 0.0 to 0.2), moderate (range from 0.2 to 0.4), or high (range from 0.4 to 1). As analyses included 4 traits at the same time, heritabilities and genetic correlations were estimated several times.

Table 1. Statistical models used to estimate the breed effect for boar taint compounds, sex hormones, immune response traits, and aggressive behavior

<table>
<thead>
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<td>Slaughter</td>
</tr>
<tr>
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<td>×</td>
<td>×</td>
</tr>
<tr>
<td>E2</td>
<td>×</td>
<td>×</td>
<td>×</td>
</tr>
<tr>
<td>AND</td>
<td>×</td>
<td>×</td>
<td>×</td>
</tr>
<tr>
<td>SKA</td>
<td>×</td>
<td>×</td>
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</tr>
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<td>×</td>
<td>×</td>
<td>×</td>
</tr>
<tr>
<td>cCRP</td>
<td>×</td>
<td>×</td>
<td>×</td>
</tr>
<tr>
<td>PM</td>
<td>×</td>
<td>×</td>
<td>×</td>
</tr>
<tr>
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<td>×</td>
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</tr>
<tr>
<td>GRA</td>
<td>×</td>
<td>×</td>
<td>×</td>
</tr>
<tr>
<td>NSL</td>
<td>×</td>
<td>×</td>
<td>×</td>
</tr>
</tbody>
</table>

¹TES = concentration of testosterone in plasma (ng/mL); E2 = concentration of 17β-estradiol in plasma (pg/mL); AND = concentration of androstenone in fat (μg/g); SKA = concentration of skatole in fat (μg/g); IND = concentration of indole in fat (μg/g); cCRP = concentration of C-reactive protein (μg/mL) in plasma; PM = concentration of pig major acute-phase protein in plasma (μg/mL); LYM = number of lymphocytes in blood (1,000 cells/mm³); GRA = number of granulocytes in blood (1,000 cells/mm³); NSL = number of skin lesions on the carcass.

RESULTS

The number of animals, means, and SD of untransformed and transformed data regarding all traits for both breed types of boars are shown in Table 2. Purebred boars had a lower live weight at slaughter compared with crossbred boars (P < 0.0001). Moreover, age at slaughter was greater in purebred than in crossbred boars (P < 0.0001). Comparison of means between the 2 breeds for sex hormones and boar taint compounds showed significantly lower values in purebred than in crossbred pigs, except for estradiol.

Phenotypic correlations calculated between the residuals of the statistical mixed models between traits were significant for some pairs of variables (Table 3). Indeed, correlations between testosterone or estradiol on the one hand and androstenone on the other hand were moderately to highly positive in both types of pigs and were greater than correlations with skatole.
Mean values of heritabilities and genetic correlations as well as SE of genetic correlations are presented in Table 4. Testosterone in both breeds and estradiol in purebred boars had a moderate heritability. Androstenone had a high heritability and skatole had a moderate one in both breeds. Heritabilities of immune response traits were moderate or high in both breeds, except for pigMAP, which had a low heritability both for purebred and crossbred boars. Heritability of skin lesions was high in both breeds. Genetic correlations between plasma testosterone and estradiol, fat androstenone, and skatole were moderate or high in both breeds of pigs. Regarding other genetic correlations, only 3 were consistent in both breeds: a moderate negative one between plasma testosterone and number of lymphocytes, a high positive one between numbers of lymphocytes and granulocytes, and high positive one between plasma testosterone and number of carcass skin lesions.

### DISCUSSION

In the present study, we used the Pietrain either in pure or in cross-breeding with Large White sows because the Pietrain breed is the predominant terminal sire line used in France and the Large White breed is frequently used in crossbred dam lines. Purebred pigs were older and lighter at slaughter than crossbred pigs due to a slower growth rate. This result was expected. Indeed, Large White pigs grow faster than Pietrain pigs (Saintilan et al., 2013), and crossbred pigs are expected to present intermediary growth rate between parental lines.

#### Sex Hormones and Boar Taint Compounds

Meat from boar carcasses may have a very unpleasant odor of urine, feces, and perspiration when cooking (Dijksterhuis et al., 2000). This unpleasant odor in boar meat, known as “boar taint,” is mainly...
due to the accumulation of 2 smelling components: 5α-androst-16-en-3-one (androstenone) and 3-methylindole (skatole). Other substances may play a role in the overall perception of boar taint—indole, 4-phenyl-3-buten-2-one, and short-chain fatty acids—but with a minor role (Lundström et al., 2009). In the present study, fat indole was measured in addition to skatole and androstenone.

Overall, our data show very low levels of boar taint compounds. They are similar to those reported in purebred Pietrain boars (Aluwé et al., 2011) as well as in crossbred Pietrain specially selected for very low levels of androstenone and skatole (Morlein and Tholen, 2014). Very few studies exist comparing the Pietrain breed with another one. Data obtained on 48 pigs per breed slaughtered either around 90 or 110 kg

Table 3. Pearson’s coefficients of correlation on residual of transformed variables in purebred (above diagonal) and crossbred (below diagonal) boars1

<table>
<thead>
<tr>
<th>Trait</th>
<th>ITES</th>
<th>IE2</th>
<th>IAND</th>
<th>ISKA</th>
<th>IND</th>
<th>sCRP</th>
<th>sPM</th>
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1 Within the table values, in bold have a P-value < 0.05.

Table 4. Estimates of heritabilities (diagonal), genetic correlations (above diagonal), and SE estimated (below diagonal) in purebred and crossbred boars1

A. Purebred

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<td>0.42</td>
<td>0.05</td>
<td>0.44</td>
<td>0.12</td>
<td>0.22</td>
</tr>
<tr>
<td>ILYM</td>
<td>0.17</td>
<td>0.22</td>
<td>0.12</td>
<td>0.13</td>
<td>0.19</td>
<td>0.09</td>
<td>0.53</td>
<td>0.52</td>
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</tr>
<tr>
<td>IGRA</td>
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<td>0.26</td>
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<td>0.32</td>
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<td>0.30</td>
<td>0.13</td>
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<td>0.08</td>
</tr>
<tr>
<td>INS</td>
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<td>0.27</td>
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<td>0.07</td>
<td>0.38</td>
<td>0.17</td>
<td>0.18</td>
<td>0.31</td>
</tr>
</tbody>
</table>

1 Standard errors for heritabilities were between 0.05 and 0.10 both for purebred and crossbred boars.

2 ITES = log(testosterone); IE2 = log(17β-estradiol); IAND = log(androstenone); ISKA = log(skatole); IND = log(indole); sCRP = sqrt(C-reactive protein); sPM = sqrt(pig major acute-phase protein); ILYM = log(lymphocytes); IGRA = log(granulocytes); INS = log(number of skin lesions on the carcass + 1).
live weight showed that fat skatole and fat androstene none were about one-third lower in Pietrain than in Landrace and Large White boars (Aluwé et al., 2011). Using the commonly accepted upper limits of 1.7 μg/g of pure fat (equivalent to 1.0 μg/g of fat tissue) for androsteneone and 0.34 μg/g (equivalent to 0.2 μg/g of fat tissue) for skatole (Lundström et al., 2009; Mathur et al., 2012), the percentage of carcasses that would have been considered tainted was low for both purebred (3.5%) and crossbred (2.5%) pigs. Because no consensus exists for these thresholds, we have also calculated the percentage of downgraded carcasses using 3 μg/g of pure fat for androsteneone and 0.25 μg/g for skatole as recommended by Bonneau and Chevillon (2012). In this situation, only 2.1% of purebred and 2.4% of crossbred boar carcasses would have been rejected. Using slightly more severe limits (0.5 and 0.2 μg/g fat tissue, respectively, for androsteneone and skatole), Aluwé et al. (2011) observed about 10% of tainted carcasses in the Pietrain breed and more than 20% of tainted carcasses in the Large White and Landrace breeds. In commercial farms using various breeds that were not specified, it was estimated that 9 to 44% of boars were affected by boar taint depending on the thresholds chosen (Walstra et al., 1999; Mathur et al., 2012). However, comparison across studies should be taken with caution because of variations between analytical methods and between laboratories for a given analytical method (Ampuero Kragten et al., 2011).

Blood samples for hormone measurements were performed at a fixed time of day to avoid diurnal variations. They were performed, on average, 9 d before slaughter, similarly to Grindflek et al. (2011), who took blood samples up to 2 wk before slaughter. Mean values and SD for sex hormones were similar to those observed in Duroc and Landrace boars of similar age and BW by Grindflek et al. (2011), except for testosterone that was lower in our experience. To our knowledge, this is the first time that those parameters are reported in purebred and crossbred Pietrain boars.

**Heritabilities.** Androsteneone had a high heritability whereas skatole had a moderate one in agreement with previous studies (Robic et al., 2008; Grindflek et al., 2011). This agrees well with many observations showing that skatole depends more on the environment and androsteneone on the genetic type (Zamaratskaia and Squires, 2009). In our study, samples were obtained after slaughter, which is a stressful event and may be a source of variation. Indeed, estimate of skatole heritability from fat samples collected via biopsy performed in the farm without the stress arising from the prest slaughter conditions (e.g., transport, social mixing, and new environment) resulted in a greater value (Baes et al., 2013). Furthermore, it was shown that skatole in fat was greatly influenced by transport and pre-unloading duration (Wesoly et al., 2015). Therefore, skatole levels could have been influenced by nongenetic factors that could not be included in the statistical model before calculating the heritability.

Unexpectedly, heritability of indole was very different between purebred and crossbred boars. This influence of breed type may be explained by the fact that nearly 90% of the measures from Pietrain × Large White boars were under the threshold of detection. In Pietrain boars, Mathur et al. (2013) reported a much lower heritability for indole \((h^2 = 0.29)\). Estimates in other breeds showed heritabilities between 0.27 and 0.55 (Grindflek et al., 2011; Baes et al., 2013). Both testosterone and estradiol had moderate heritabilities in agreement with previous estimates in pigs (Grindflek et al., 2011).

**Correlations.** A positive phenotypic correlation between androsteneone and skatole was observed in accordance with previous studies (Babol et al., 1999; Tajet and And resen, 2006; Grindflek et al., 2011). The genetic correlation between the 2 variables was also positive, was moderate for the crossbred boars, and was high for the purebred boars. This link is probably due to an antagonist action of androsteneone on skatole degradation by the liver (Doran et al., 2002) and hence on the level of skatole available for storage in the adipose tissue. Genetic correlations between testosterone and estradiol were high, similar to results from Grindflek et al. (2011; 0.80 and 0.93 in Landrace and Duroc breeds, respectively). High genetic correlation between testosterone and estradiol was expected because of common mechanisms of control regarding testicular activity. Both hormones are already known to be good indicators of the sexual development (Prunier et al., 2013).

Greater phenotypic correlations between sex hormones and androsteneone than between sex hormones and skatole were expected. Indeed, testosterone, estradiol, and androsteneone are produced by the testes and hence depend on common mechanisms of regulation, whereas skatole derives from the degradation of tryptophan by bacteria in the large intestine (Deslandes et al., 2001) and hence its production depends on very different mechanisms of regulation. We also estimated very high genetic correlations between estradiol and androsteneone and very high or moderate genetic correlations between testosterone and androsteneone, respectively, for purebred and crossbred boars. Positive genetic correlations were also observed between sex hormones and skatole. These moderate or high correlations were expected and confirmed values previously reported between these traits in Duroc and Landrace breeds (Grindflek et al., 2011). In good agreement with the genetic correlations, these authors showed
that boar taint compounds and sex hormones are under
the control of similar genomic regions.

Positive genetic correlations between estradiol or
testosterone and boar taint compounds support the
possibility to select boars on plasma sex hormone
concentrations to reduce boar taint. Blood samples
offer the advantage of being easier to perform in liv-
ing pigs compared with biopsies of backfat, although
this latter measure was already approved (Baes et al.,
2013). In addition, estradiol and testosterone can be
measured in plasma, without any extraction procedure,
with numerous commercial kits giving a result within
a few hours, contrarily to androstenone and skatole.
Because of the strong links between boar taint and re-
productive function (Zamaratskaia and Squires, 2009),
selection should be planned differently for the sire and
dam lines. Besides delaying age at full sexual develop-
ment, decreasing testosterone by selection is expected
to have limited effect on male reproduction traits such
as sperm production and fertility traits (Walker et al.,
2004) but detrimental effects are expected on female
reproduction traits (Robison et al., 1994). Therefore,
a possible strategy to reduce boar taint compounds in
crossbred pigs would be to select against testosterone
and estradiol in sire lines. The aim of this selection
would be to delay the age at which boars reach their
full capacity to produce steroids. Consequently, age at
full steroid production would also be delayed in cross-
bred offspring and boar taint compounds would not
have time to accumulate before slaughter. To avoid a
possible decrease in the reproductive ability of mature
pigs, 2 measures of plasma sex steroids should be per-
formed, one before the usual slaughter weight, when
full sexual development is not reached (for example
around 90–100 kg live weight), and one several weeks
later when boars are fully sexually mature. Animals
should be selected for low levels of sex steroids at the
first sample but normal levels at the second one.

Immune Response Traits and Relationships
with Other Traits

Mean values for APP and immune blood cells
were in the range of expected values for growing pigs
(Segalés et al., 2004; Clapperton et al., 2009). Number
of granulocytes and plasma pigMAP concentration
were similar in both breeds whereas CRP concentra-
tion and number of lymphocytes differed significantly
between breeds. A breed effect on immune response
traits has already been observed in growing pigs
(Clapperton et al., 2005, 2007; Merlot et al., 2012). The
same authors have also shown that some inflammatory
markers were influenced by the genotype contrarily to
others. For example, the number of circulating granu-
locytes was greater whereas that of lymphocytes was
lower and plasma level of haptoglobin was similar in
Meishan compared with Large White growing pigs
(Clapperton et al., 2005). The different inflammatory
response markers are under various mechanisms of
regulation. Therefore, it is not surprising that breed
differences exist in the immune response of pigs to
the same sanitary conditions.

Heritabilities. C-reactive protein heritability was
moderate in purebred boars in accordance with previ-
ous studies (Clapperton et al., 2009; Flori et al., 2011)
and was greater in crossbred boars. Heritability for pig-
MAP was very low in both breeds. To our best knowl-
edge, no previous estimation of pigMAP heritability
has been reported in literature. This very low heritabil-
ity for pigMAP suggests a more profound influence of
nongenetic individual factors on this trait compared
with CRP. Numbers of lymphocytes and granulocytes
had moderate or high heritabilities, in agreement with
previous studies (Clapperton et al., 2008; Flori et al.,
2011). However, a null heritability was also previously
estimated for the total number of lymphocytes in pigs
8 wk old (Edfors-Lilja et al., 1994).

Correlations. Phenotypic correlations within im-
une response traits were relatively low in both ge-
etic types, sometimes negative and sometimes posi-
tive, and often inconsistent from one breed to the other,
except for lymphocyte and granulocyte numbers. The
close positive link between these 2 variables was ex-
pected because they have a common hematopoietic
origin. Inconsistent correlations between different APP
in unchallenged conventional pigs were also observed
in a previous study (Clapperton et al., 2007). Due to
very low heritabilities of pigMAP, all genetic correla-
tions with this parameter should be considered with
cautions. Genetic correlations between plasma CRP and
numbers of lymphocytes and granulocytes were close
to zero, as previously observed (Flori et al., 2011).
Both APP and leukocytes play an important role in the
immune response but they are under the control of very
different mechanisms and low genetic correlations
were expected. Numbers of lymphocytes and granulo-
cytes were highly genetically correlated, as expected.

A moderate negative genetic correlation was ob-
served between the number of lymphocytes and testos-
terone in both breeds of pigs. To our best knowledge,
the negative correlation between the number of lym-
phocytes and plasma testosterone was not previously
described in pigs and in other species. However, the
negative role of testosterone on T and B cells produc-
tion by bone marrow and thymus in sexually mature
animals is well described (Viselli et al., 1997; Hirakata
et al., 2010) and fits well with the negative correla-
tion observed in our pigs. From the present state of
knowledge, it cannot be concluded whether increasing or decreasing the number of lymphocytes by genetic selection would be positive or not. Due to that incertitude, monitoring the blood formula together with specific health parameters (e.g., lung lesions at slaughter) would be recommended in any selection against levels of plasma steroids designed to decrease boar taint.

Phenotypic correlations between immune response traits and skin lesions were relatively low in both genetic types. This is not surprising because skin lesions were observed at slaughter well after blood was sampled and hence could not be at the origin of any inflammatory reaction at the time of the blood sampling. Genetic correlations between immune response traits and number of skin lesions or androstenone or skatole were moderate or high in some instances but were never consistent in the 2 breeds. Therefore, they should be interpreted with cautious.

Skin Lesions and Relationships with Sex Hormones and Boar Taint

Skin lesion number is an indicator of individual aggressiveness during the postmixing period (Turner et al., 2006a, 2009). In entire males, mounting behavior can also be a source of lesions (Rydmer et al., 2006). Therefore, the significantly lower number of skin lesions in purebred than in crossbred boars is probably due to a lower level of aggressive interactions and/or of mounting attempts.

Heritabilities. Heritability of carcass skin lesions was moderate and similar in purebred and crossbred boars, in agreement with a previous study, in a different breed of pigs, where lesions were counted on the farm after mixing (Turner et al., 2006b). This moderate heritability also suggests the possibility to decrease aggressiveness of boars by genetic selection.

Correlations. Testosterone is known to stimulate aggressiveness (Lumia et al., 1994; Breuer et al., 2001; Soma et al., 2008). Therefore, a positive phenotypic correlation was expected between plasma testosterone and the number of skin lesions as already observed by Prunier et al. (2013). This is not the case in the present experiment. However, skin lesions were counted on the carcasses about 8 d after measuring plasma testosterone and social groups of pigs were different for the 2 measures because animals of different pens were grouped before slaughter. This may have contributed to lower the phenotypic correlation calculated on the residuals. Contrary to what was observed for the phenotypic correlation, a high positive genetic correlation between testosterone and skin lesions was observed in both breeds of pigs, as expected. This is in full agreement with the stimulatory influence of testosterone on aggressiveness.

The genetic correlation of skin lesions with estradiol was also high in purebred but not in crossbred pigs. This lack of consistency of the genetic correlation between estradiol and skin lesions is probably due to the fact that estradiol does not influence directly the social behavior. In both breeds, the genetic correlations between skin lesions and androstenone or skatol levels were low.

Conclusion

Purebred and crossbred boars had very similar estimations of heritabilities but estimates of genetic correlations were different for some pairs of trait. Considering the heritabilities and genetic correlations estimated in our 2 populations of boars with low boar taint levels, it would be possible to decrease the occurrence of boar taint and limit aggressive behaviors in boars by combining a selection for low plasma estradiol and testosterone levels at a given live weight before full sexual development and unchanged levels of sex steroids at the usual slaughter weight when boars are fully mature. A direct selection against androstenone and skatole would be efficient to decrease the occurrence of boar taint but would have no or very low incidence on aggressive behaviors. Both types of selection would have low consequences on immune response traits, even though a possible increase in the number of lymphocytes could be expected by selecting against testosterone. Therefore, one strategy to decrease boar taint and improve welfare by less aggressiveness would be to select against plasma testosterone and estradiol. However, potential consequences of such a selection on health should be evaluated by monitoring the blood formula and parameters more directly linked to the health status. In addition, the consequences on the reproductive performances should be evaluated.

LITERATURE CITED


