Breeding Pigs For Resistance To Disease Is Difficult Even With Genomic Selection

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Introduction
Most attempts to breed pigs for resistance to clinical and sub-clinical disease have failed. The reason is that it is difficult to identify individuals that are resistant to disease. Resistance is a lowly-heritable trait and indicator traits, traits that are genetically correlated with resistance and act as additional selection criteria, have yet to be discovered. The advent of genomic selection provides a new approach to breed for resistance. Selection candidates are genotyped for dense markers and these markers are used to predict genomic breeding values. The potential benefit of genomic selection is that pigs can be differentiated for their ability to resist disease, opening the way for genetic gains. This led us to believe that including genomic selection for resistance in pig breeding schemes will generate genetic gains in resistance. Using stochastic simulation, we estimated the amount of gain that could be generated by including genomic selection for resistance to respiratory disease in a pig breeding scheme. We generated phenotypic marker information for genotyped animals and assumed that the marker information could predict true breeding values for resistance with pre-defined levels of accuracy.

Material and methods

Procedure. We simulated four variants of the current breeding scheme for Duroc pigs in Denmark, albeit on a smaller scale. Seven traits were included in the breeding objective: growth rate from birth to 30 kg, growth rate from 30 to 100 kg, feed-conversion ratio, meat yield, leg strength, slaughter loss, and resistance to respiratory disease. The variants were:

- Control. Current breeding scheme with no economic value for resistance, no pigs observed for resistance, and no pigs genotyped.
- Semi-control. Current breeding scheme with an economic value for resistance, performance-tested boars observed for resistance, but no pigs genotyped.

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Each variant was simulated assuming (i) no correlations between resistance and the other traits in the breeding objective, and (ii) unfavourable genetic and favourable residual correlations between resistance and growth rate from birth to 30 kg \((r_g = -0.20, r_e = 0.10)\), growth rate from 30 to 100 kg \((-0.20, 0.10)\), and feed-conversion ratio \((0.20, -0.20)\). These correlations were derived from other livestock species (Rauw et al. (1998)). The \textit{sire genotyping} and \textit{sire-dam genotyping} variants were simulated assuming that phenotypic marker information generated for the genotyped boars and sows could predict true breeding values for resistance with accuracies of 0.45, 0.63, and 0.77. This corresponds to 20, 40, and 60% of the genetic variation for resistance being explained by the marker information.

**Breeding scheme.** The breeding scheme consisted of 400 breeding sows in four herds (100 sows/ herd) and 10 breeding boars at a test station. Each sow produced 10 piglets, resulting in 4000 piglets (1000 piglets/ herd). Two-hundred of the male piglets were performance-tested at the test station. The remaining males and all female piglets were tested at the herds. Following testing, the 300 best performance-tested boars and the 250 best gilts in each herd (1000 sows in total) were selected. The 10 new breeding boars at the test station were selected from the 300 best performance-tested boars and the 10 breeding boars currently at the test station. The 100 new breeding sows in each herd were selected from the 250 best performance-tested gilts and the 100 breeding sows currently at the herds. All selection decisions were based on BLUP breeding values.

**Traits.** Resistance to respiratory disease was observed for boars performance tested at the test station in the \textit{semi-control}, \textit{sire-genotyping}, and \textit{sire-dam genotyping} variants. We assumed that resistance could be observed on an underlying liability scale; the underlying liability was normally distributed \(N(0,1)\) with heritability 0.05 and economic value €0.95/s.d. liability. The economic value was derived from a commercial cost of respiratory disease of €2.64/pig. Twenty percent of pigs in the base population were resistant; genetic gains in resistance were transferred from the underlying to the observed percentage scale.

Phenotypic marker-information was observed for genotyped boars and sows. The 300 best performance-tested boars were genotyped in the \textit{sire-genotyping} and \textit{sire-dam genotyping} variants; the 100 best performance-tested gilts in each herd were also genotyped in the \textit{sire-dam genotyping} variant. The marker information had heritability 0.99 and genetic correlations to the true breeding values for resistance of 0.45, 0.63, and 0.77; equivalent to the assumed accuracies of 0.45, 0.63, and 0.77. Including marker information as an additional trait in a multivariate-BLUP model resulted in genomically-enhanced breeding values for resistance (after Dekkers (2007)). Marker-information had no economic value.

Growth rate from 30 to 100 kg, meat yield, and leg strength were observed for all performance-tested pigs; growth rate from birth to 30 kg was observed for pigs performance tested at the herds; feed-conversion ratio for boars performance tested at the test station, and slaughter loss for boars performance tested at the test station but not selected. Genetic variances, heritabilities, economic values, and genetic and residual correlations for these traits are presented in Table 1.
Table 1: Additive genetic variances ($V_a$), heritabilities ($h^2$), economic values (EV), and genetic and residual correlations $\alpha$ for growth rate from birth to 30 kg (GR$_{30}$), growth rate from 30 to 100 kg (GR$_{100}$), feed-conversion ratio (FCR), meat yield (MY), leg strength (LS), and slaughter loss (SL)

<table>
<thead>
<tr>
<th>Traits</th>
<th>$V_a$</th>
<th>$h^2$</th>
<th>EV (€/pig)</th>
<th>GR$_{30}$</th>
<th>GR$_{100}$</th>
<th>FCR</th>
<th>MY</th>
<th>LS</th>
<th>SL</th>
</tr>
</thead>
<tbody>
<tr>
<td>GR$_{30}$</td>
<td>185</td>
<td>0.29</td>
<td>0.016/g/day</td>
<td>0.46</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>GR$_{100}$</td>
<td>1536</td>
<td>0.33</td>
<td>0.016/g/day</td>
<td>0.06</td>
<td>-0.30</td>
<td>-0.20</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>FCR</td>
<td>0.006</td>
<td>0.31</td>
<td>-17.6/ratio unit</td>
<td>0</td>
<td>-0.52</td>
<td>-0.34</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>MY</td>
<td>0.275</td>
<td>0.44</td>
<td>1.15/%-point</td>
<td>0</td>
<td>0.04</td>
<td>-0.30</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>LS</td>
<td>0.10</td>
<td>0.19</td>
<td>1.67/point</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>SL</td>
<td>0.89</td>
<td>0.42</td>
<td>-0.67/kg</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
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</tr>
</tbody>
</table>

$\alpha$ Genetic correlations above and residual correlations below the diagonal

**Simulations.** Simulations were run for the equivalent of 15 years. Genetic gains for resistance are presented as means of 50 replicates on the observed scale. The simulations were carried out in ADAM (Pedersen et al. (2007)). The BLUP breeding values were predicted using DMU6 (Madsen et al. (2006)).

**Results**

Genomic selection generated only small genetic gains in resistance when there were no correlations between resistance and the other traits in the breeding objective; it generated no gains when there were unfavourable genetic and favourable residual correlations with resistance (Figure 1). When there were no correlations, genomic selection in the sire genotyping and sire-dam genotyping variants increased resistance from 20% to approximately 24, 27, and 30% at year 15 with accuracies 0.45, 0.63, and 0.77. Genotyping sows in the sire-dam genotyping variant did not generate more genetic gain than the sire genotyping variant. The semi-control variant, where resistance was observed on the underlying scale without using genomic selection, did not increase resistance by any more than 1%. There was no change in resistance in the control variant.

With unfavourable genetic and favourable residual correlations, genomic selection did not generate any genetic gain. Resistance fell from 20% to approximately 9% at year 15 in the sire genotyping, sire-dam genotyping, semi-control, and control variants.

**Discussion**

Our findings partly supported our premise that including genomic selection for resistance in pig breeding schemes will generate genetic gains in resistance. The fact that we generated gains when there were no correlations with resistance suggests that it is possible to increase resistance. However, the small magnitude of the gains demonstrates that breeding pigs for resistance is difficult even with genomic selection where breeding values are predicted accurately. The level of the difficulty was merely amplified when there were unfavourable genetic and favourable residual correlations with resistance. There was presumably so much selection emphasis on the other traits in the breeding objective that including selection for resistance, a lowly-heritable trait with moderate economic value, produces little selection
differential. If pig breeders are to increase resistance, they will have no option but to re-evaluate its economic value and accept that gains for the other traits will probably be reduced. Re-evaluating the economic value could involve desired-gains selection indices or allocating a monetary value to non-commercial, marketing, and animal welfare concerns. So, genomic selection still remains a potential approach to breed for resistance, but in order to generate gains, pig breeders will need to re-evaluate its economic value and accept reduced gains for other traits.

![Figure 1: Proportion (%) of resistant pigs in four variants of a pig breeding scheme when there were (a) no correlations between resistance and other traits in the breeding objective and (b) unfavourable genetic and favourable residual correlations to resistance. The variants are control (—), semi-control (---), sire-genotyping where true breeding values for resistance are predicted by genomic selection with accuracies 0.45 (--), 0.63 (--), and 0.77 (--), and sire-dam genotyping where the true breeding values are predicted with accuracies 0.45 (--), 0.63 (--), and 0.77 (--).](image)

The lack of additional gains from genotyping the sows was surprising given that it increased the accuracy of the sow’s breeding values. The lack of gains was most likely because sows were selected with lower intensity than the boars and because the number of performance-tested gilts that were genotyped did not exceed the number of breeding sows. The selection differential applied to the sows for resistance was simply too low. Therefore, it is likely that larger numbers of sows will need to be genotyped before any additional gains are generated.

References


