Prediction of reduction in aggressive behaviour of growing pigs using skin lesion traits as selection criteria

S. Desire[1], S.P. Turner[1], R.B. D'Eath[1], A.B. Doeschl-Wilson[2], C.R.G. Lewis[3], and R. Roehe[1]

[1] Animal and Veterinary Sciences Group, Scotland's Rural College, West Mains Road, Edinburgh, Scotland, EH9 3JG.

[2] The Roslin Institute, University of Edinburgh, Division of Genetics and Genomics, Easter Bush, Midlothian, Scotland, EH25 9RG.

[3] Genus, PIC, Avda. Ragull, 80, 08173, Sant Cugat del Valles (Barcelona), Spain

Corresponding author: Suzanne Desire

Email: suzanne.desire@sruc.ac.uk

Abstract

Aggression at regrouping is a common issue in pig farming. Skin lesions are genetically and phenotypically correlated with aggression and have been shown to have a significant heritable component. This study predicts the magnitude of reduction in complex aggressive behavioural traits when using lesion numbers on different body regions at two different time points as selection criteria, to identify the optimum skin lesion trait for selection purposes. 1,146 pigs were mixed into new social groups, and skin lesions were counted 24 hours (SL24h) and 3 weeks (SL3wk) post-mixing, on the anterior, centre and posterior regions of the body. An animal model was used to estimate genetic parameters for skin lesion traits and 14 aggressive behavioural traits. Estimated breeding values (EBVs) and phenotypic values were scaled and standardized to allow direct comparison across multiple traits. For each body region, individuals with SL24h and SL3wk EBVs in the least aggressive 10% of the population and compared to the population mean to predict the expected genetic and phenotypic response in aggressive behaviour to selection. At mixing, selection for low anterior lesions was predicted to affect substantially more behavioural traits of aggressiveness than lesions obtained on other body parts, with EBVs between -0.21 and -1.17 SD below the population mean. Individuals with low central SL24h EBVs also had low EBVs for aggressive traits (-0.33 to -0.55). Three weeks later, individuals with high SL3wk EBVs had low EBVs for aggression at mixing (between -0.24 and -0.53 SD below the population mean), although this was predicted to affect fewer traits than selection against SL24h. These results suggest that selection against anterior SL24h would result in the greatest genetic and phenotypic reduction in aggressive behaviour recorded at mixing. Selection for increased SL3wk was predicted to reduce aggression at mixing; however current

51 understanding about aggressive behaviour under stable social conditions is
52 insufficient to recommend using this trait for selection purposes.
53

Keywords: pigs, aggression, skin lesions, selection, genetics

Implications

Pigs fight to establish dominance when mixed into new social groups. This aggressive behaviour affects growth and is a welfare concern. We estimated the effect of selective breeding on aggressive behaviour, using the number of skin lesions (scratches received) on different regions of the body to identify the least aggressive individuals to breed from. The results suggest that selection for reduced skin lesions at the front of the body at 24hrs post-mixing would result in the greatest reduction in aggressive behaviour and is therefore the best selection criterion of all analysed lesion traits to reduce aggressiveness at mixing.

Introduction

In indoor commercial farming systems pigs are housed in inflexible group sizes under space-limited conditions. In order to create groups of a predetermined and uniform size, growing pigs are often mixed with unfamiliar individuals throughout the production cycle. Physical aggression usually occurs at mixing, which serves to establish dominance relationships (Meese and Ewbank, 1973). Mixing induced aggression has been associated with stress and injury (Mendl *et al*., 1992), suppressed immune responses (de Groot *et al*., 2001), and reduced growth (Stookey and Gonyou, 1994), carcass (Faucitano, 2001) and meat quality (D'Eath *et al*., 2010). Although a number of practical interventions continue to be explored, an under-explored solution to reduce aggression is via genetic selection. This study aimed to examine the best trait to select upon in order to reduce aggressive behaviour.

Phenotyping aggressive behaviour by direct observation or from video is highly labour intensive; therefore skin lesions - which occur as a result of physical aggression – may constitute valuable proxies for aggressive behaviour (Turner *et al*., 2006; Guy *et al*., 2009). Skin lesions are genetically and phenotypically correlated with aggression and have been shown to have a significant heritable component (h^2: 0.08 to 0.43); therefore it is expected that selection against the quantity and body location of skin lesions will result in a corresponding reduction in aggression (Turner *et al*., 2009; Desire *et al*., 2015a).

Deciding how skin lesions should be used to select against aggression requires understanding of the complex relationship between skin lesions and aggressive

behaviour. Skin lesions on one animal can be an indication of the behaviour of other members of the social group (via an unreciprocated attack), as well as the individual in question (via willing involvement in aggression). Failing to distinguish between the underlying causes of lesions may lead to biased estimates of individual aggression. On a phenotypic level, Turner *et al*. (2006) showed that skin lesions to the anterior region of the body are strongly correlated with the proportion of time involved in reciprocal aggression, whereas lesions to the rear of the body indicated a larger proportion of time spent receiving non-reciprocal aggression.

Previous work suggests this relationship is partly under genetic regulation (Turner *et al*., 2009). In addition to measuring aggression performed immediately post-mixing, skin lesions have been explored as a method of phenotyping aggression experienced under socially stable conditions (several weeks post-mixing). Studies on both on a phenotypic (Desire *et al*., 2015a) and genetic (Turner *et al*., 2009) level have found that individuals involved in much aggression at mixing tend to have fewer lesions several weeks later. These results suggest that avoidance of aggression upon first mixing may be detrimental to the individual's long term welfare. Finally, genetic variation and heritabilities of skin lesion traits differ between different body regions and time points. In light of the above, it is important to carefully assess the potential impact of selection for reduced aggression via skin lesion traits. Although genetic correlations between skin lesion traits and some aggressive behavioural traits have been previously published (Turner *et al*., 2009) these correlations do not give an indication of the magnitude of the expected response to selection. In addition, the estimated genetic correlations among skin lesion and behaviour traits are complex, often in conflict with one another, and associated with high errors of

116 estimation. This means that predicting the selection response based on genetic
117 correlations can be difficult. Due to time constraints, only one skin lesion trait is likely
118 to be recorded under practical conditions, therefore it is necessary to identify the
119 single best skin lesion trait for selection. The objective of this study was to identify
120 the optimum skin lesion trait for selection purposes, by determining the magnitude of
121 the reduction in aggressive behavioural traits at mixing, when using lesion numbers
122 recorded on different body regions at mixing and in the stable group as selection
123 criteria.

Materials and methods

Animals and housing

Data were collected from a commercial herd. Three individuals (average age 71 days, SD 4.5) taken from each of five litters were mixed to form a new social group of 15 same sex, same breed individuals. Animals with all skin lesion and behavioural phenotypes were included in the analyses, and the final dataset contained 1,146 individuals (698 purebred Yorkshire and 448 Yorkshire x Landrace) from 77 social groups. Experimental animals were the progeny of 82 sires and 217 dams, and a 2-generation pedigree was used (total 1,862 animals). Groups mixed on the same day were classed as the same batch. At time of mixing, animals were approximately matched for body weight in order to minimise variation between pen mates. Pigs were weighed 24 hours following mixing. Animals were housed indoors in partially slatted pens (30% slats, 70% solid flooring with light straw bedding) and were provided with pelleted feed and water ad libitum.

Skin lesion traits

Fresh skin lesions were counted separately on the anterior (head, neck, forelegs and shoulders), centre (flanks and back), and posterior (hind legs and rump) regions of the body 24 hours post-mixing (SL24h). In order to ensure injuries inflicted prior to mixing were not included in the analyses, lesions were also counted immediately prior to mixing, and pre-mix lesion counts deducted from the post-mixing count. Lesions were deemed to be fresh if they were a vivid red colour, bleeding or recently scabbed. Lesions were counted in the same manner three weeks post-mixing (SL3wk) as a measure of aggression under stable social conditions.

149

150 *Behavioural traits*

151 Animals were video recorded for 24 hours following mixing. The behavioural traits
152 used in this study were based on data recorded during these behavioural
153 observations. Each interaction was classed as either reciprocal aggression (RA) or
154 non-reciprocal aggression (NRA) as defined by Turner *et al*., (2006). Behavioural
155 traits used in the analyses are defined in Table 1.

156

157 *Characteristics of the data*

158 Only aggressive behavioural traits that had been previously shown to be predictive of
159 skin lesion traits on a phenotypic level in the same population were chosen for
160 analysis (Desire *et al*., 2015a). Skin lesion and aggressive behavioural traits showed
161 considerably skewed distributions (Supplementary Table S1), therefore a log
162 transformation ($y = \log_e+1$) was used to approach the normal distribution.

163

164 *Statistical Analyses*

165 Univariate analyses were used to estimate genetic components and estimated
166 breeding values (EBVs) of all log transformed skin lesion and behavioural traits using
167 the following animal model:

168 $$\mathbf{y = Xb + Za + Wc + e}$$
169

170 where **y** is the vector of records for skin lesions (SL24h and SL3wk) and aggressive
171 behaviour, and **X**, **Z** and **W** are the incidence matrices of fixed effects, genetic
172 effects, and environmental (pen) effects, respectively. Vectors **b**, **a**, **c** and **e**
173 represent fixed effects, additive direct genetic effects, common environmental effects
174 (shared by all pigs in a pen), and residual error, respectively. Genetic line, sex, and

batch were included in all models as fixed categorical effects, while bodyweight at time of mixing was fitted as a covariate. Age at time of mixing was included for SL24h and aggressive behavioural traits. Bivariate analyses were used to estimate genetic and group level correlations between skin lesion traits and aggressive behavioural traits using the same fixed and random effects described for the univariate analyses. Genetic analyses were performed using ASReml (Gilmour *et al*., 2009).

As skin lesion and behavioural traits are measured on different scales, it is impractical to directly compare genetic and phenotypic values across multiple traits. Breeding values and untransformed phenotypic values were therefore scaled and standardised, and expressed in terms of standard deviations from a population mean of zero. Individuals were chosen for inclusion in each subsequent analysis based on either SL24h EBVs in the lowest 10% of the population, or SL3wk EBVs in the highest 10% of the population. This methodology was chosen as it allows the selection response to be predicted based on the given data, rather than complex genetic correlations that have a high level of estimation error, which might affect the accuracy of predicted response using population genetics theory.

Results

Heritabilities and common environmental effects

Heritabilities estimated for skin lesion traits ranged from 0.11 to 0.43 (Table 2). A substantially higher heritability was estimated for anterior SL3wk than anterior SL24h. Heritabilities for behavioural traits ranged from 0.09 to 0.44 (Table 2). The proportion of variance attributed to common environmental effects was generally lower than estimated heritabilities for skin lesion and behavioural traits (c^2 = 0.06 to 0.15), except for posterior SL24h, average fight duration, and duration of time spent receiving NRA.

Genetic and pen level correlations between skin lesion and behaviour traits

Genetic correlations. Where significant, genetic correlations between anterior SL24h and aggressive behavioural traits were positive (Table 3). The proportion of fights won was negatively correlated with central and posterior SL24h, while the duration of NRA received, and the number of pen mates that NRA was received from were positively correlated with these traits (Table 3). Genetic correlations between anterior SL3wk and aggressive behavioural traits were generally negative (Table 4), except duration of NRA received and number of pen mates that NRA was received from. Positive correlations were found between central SL3wk and duration of NRA received, and number of pen mates that NRA was received from (Table 4). No significant genetic correlations were found between posterior SL3wk and aggressive behavioural traits (Table 4).

Pen level correlations. Most pen level correlations between skin lesions and aggressive traits did not significantly differ from zero. Those that did were mainly positive for SL24h (Table 3). Statistically significant negative pen level correlations were found between posterior SL24h and the number of reciprocal fights involved with, and duration of NRA received (-0.08, SE 0.03; Table 3). Negative pen level correlations were found between all SL3wk traits and the number of pigs attacked by (RA), and between anterior or central SL3wk and the duration of NRA received (Table 4).

Low EBVs for SL24h

Associations with SL24h. Individuals with low EBVs for anterior, central or posterior SL24h had low EBVs (-0.69 SD and -1.89 SD; (Figure 1 [a, c, e]) and phenotypic values (-0.38 SD to -0.94 SD; Figure 2 [a, c, e]) for all skin lesion traits at mixing

Associations with SL3wk. Individuals with low EBVs for SL24h had low EBVs for SL3wk (-0.15 SD to -0.41 SD (Figure 1 [a, c, e]). Phenotypically, individuals with low EBVs for SL24h did not differ significantly in the number of SL3wk in comparison to the population as a whole (-0.05 SD to 0.07 SD; Figure 2 [a, c, e]).

Associations with aggressive behaviour. Individuals with low EBVs for anterior SL24h also had low EBVs for all aggressive behavioural traits (-0.21 to -1.17 SD; Figure 3 [a]). Other than proportion of fights won, duration of NRA initiated, and number of pen mates attacked (NRA), individuals with low EBVs for central SL24h had significantly lower EBVs for aggressive behavioural traits (-0.28 to -0.51 SD), compared to the population average. Individuals with low EBVs for posterior SL24h

had EBVs that were significantly lower than the population mean for duration of NRA received (-0.74 SD); duration of RA received (-0.41 SD); number of pigs attacked by (RA) (-0.27 SD); total number of RA received (-0.27 SD), and higher than the population average for proportion of fights won (0.45 SD) and duration of RA initiated (0.19 SD; Figure 3 [e]).

Phenotypic values for individuals with low EBVs for anterior, central and posterior SL24h largely mirrored those observed on the genetic level (Figure 4 [a, c, e]). Individuals with low EBVs for anterior SL24h received non-reciprocal attacks for 11.55 seconds less than the population mean (Supplementary Table S2).

High EBVs for SL3wk

Associations with SL24h. Individuals with high EBVs for SL3wk did not differ significantly from the population mean for anterior SL24h EBVs but had higher than average EBVs for central and posterior SL24h (0.19 to 0.42 SD; Figure 1 [b, d, f]). On a phenotypic level, only central SL24h significantly differed from the population mean in individuals with low EBVs for central SL3wk (0.25 SD ; Figure 2 [d]). No other significant associations were found between SL24h and SL3wk at the phenotypic level(Figure 2 [b, d, f]).

Associations with SL3wk. Individuals with high SL3wk EBVs had high EBVs for all other skin lesion traits at this time point (0.91 to 1.69 SD; Figure 1 [b, d, f]). These individuals also had high skin lesion numbers on a phenotypic level compared to the population mean (0.52 to 1.45 SD; Figure 2 [b, d, f]).

267 *Response on aggressive behaviour.* Except for the duration of NRA received, high
268 EBVs for anterior SL3wk corresponded with low EBVs for all aggressive behavioural
269 traits (-0.37 to -0.54 SD; Figure 3 [b]). The same trends were also observed for high
270 central SL3wk EBVs (-0.30 to -0.53 SD; Figure 3[d]). Individuals with high EBVs for
271 posterior SL3wk had low mean EBVs for all behavioural traits (-0.28 to -0.46 SD),
272 except for proportion of fights won and duration of NRA received, which did not
273 significantly differ from zero (Figure 3 [f]).
274
275 Phenotypic values for individuals with low EBVs for anterior, central and posterior
276 SL24h largely mirrored those observed on the genetic level (Figure 4 [b, d, f]).
277 Individuals with low EBVs for anterior SL3wk were involved in 2.39 fewer reciprocal
278 interactions than the population mean (Supplementary Table S3).
279

Discussion

Heritabilities

Heritabilities for skin lesion traits were of a low to moderate magnitude. These estimates differed from those reported by Turner *et al.* (2009) for the same population, as only those animals with behavioural data available were used in this analysis. Heritabilities for SL3wk were higher than SL24h, which is likely to be due to lower environmental variance 3 weeks post-mixing compared to 24 hours post-mixing. The lowest heritability estimated for behavioural traits was for receipt of NRA. Receipt of NRA results from the behaviour of other individuals in a pen, and not the individual itself, which may explain why direct genetic effects account for so little of the genetic variation in this trait. The highest heritabilities were estimated for traits related to RA. During engagement in RA, the individual animal is actively involved in the event, choosing to either attack or respond to an attack, which may explain why these traits showed the highest heritabilities.

Social genetic effects describe genetic variation due to interactions between pen-mates (Bijma and Wade, 2008). It is likely that social genetic effects contribute significantly to mixing-related aggression in pigs. Ideally, both direct and social genetic effects would be considered when assessing the genetic basis of aggression in pigs, however these effects are difficult to estimate, optimally requiring several hundred groups composed of few families (Bijma, 2010). It was therefore not possible to include social effects in this study, however common environmental effects were included in the genetic model to approximate social effects. Common environmental effects had a low influence on the number of skin lesions and

involvement in aggression. As expected, traits that related to behaviour of other group members, for example the receipt of NRA, tended to have higher common environmental effects.

Expected response following selection for reduced SL24h

Consistent with the strength and direction of genetic correlations published previously (Turner *et al*., 2009), individuals with low SL24h EBVs had significantly lower genetic and phenotypic values for SL24h to all body regions compared to the population as a whole. The results suggest that selection for anterior SL24h would have the lowest effect on posterior SL24h and vice versa. This is likely to be because lesions to these body regions reflect involvement in opposing behaviours. On a genetic level, there was generally a positive association between SL24h and SL3wk, in that individuals with low SL24h EBVs had slightly reduced EBVs for SL3wk compared to the whole population, and vice versa. However, this relationship was not universally observed on a phenotypic level.

If the aim of using skin lesions for selection purposes were to simply reduce lesion numbers, central or anterior SL24h should be used. However, the main goal of any breeding program incorporating skin lesions would be to reduce aggression, preferably on both a short and long term basis; therefore the results suggest that selection against anterior SL24h would have the greatest effect on aggressive behaviour. Associations between EBVs in the bottom or top 10% of skin lesion traits with aggressive traits were generally in accordance with genetic correlations between the same traits. The results suggest that selecting individuals based on low

anterior SL24h would result in the greatest reduction in mean EBVs for behavioural traits relating to RA. Reciprocal contests make up the majority of time spent engaged in physical aggression and carry the biggest risk of injury, therefore reducing this behaviour is highly desirable. A slightly greater reduction in receipt of RA was predicted, in comparison to initiation of RA, suggesting that the recipient of an attack may be more likely to become injured than the initiator. This may be because the initiator is more likely to win a contest, inflicting more damage in the process (Stukenborg *et al*., 2011)*.*

The possible role of social genetic effects on social aggression was mentioned above. Where there is a negative correlation between direct and social genetic effects, selection based on direct breeding values alone can result in an undesirable result (for example, selecting for reduced SL24h could theoretically result in increased aggression; Ellen *et al*., 2014). Previous studies suggest a positive correlation between direct and social effects for aggressive behaviour, meaning that animals with a low genetic propensity to become involved in aggression also have a low chance of being attacked (Wilson *et al*., 2011; Alemu *et al*., 2014). Negative correlations between social and direct effects have been found for dominance traits (Wilson *et al*., 2009; Sartori and Mantovani, 2012) however social effects accounted for little of the variation in these studies. If a positive correlation exists between social and direct effects for aggressive traits, combined selection for social and direct EBVs for SL24h may reduce aggressive behaviour to a greater degree than our results suggest.

Individuals with low EBVs for anterior SL24h had EBVs that were close to the population mean for the proportion of fights won. This suggests that selection for low anterior SL24h would not result in a strong selection for individuals that win a high proportion of fights. It is likely that the low genetic correlation between anterior SL24h and proportion of fights won is due to the fact that proportion of fights won is independent from the duration of time spent engaged in aggression. For example, an individual with very high fight success may have spent little time engaged in aggression (receiving few lesions in the process), or much time engaged in aggression (receiving many lesions). Likewise, the same can be true for animals with a low fight success rate. Because of this relationship, individuals with low anterior SL24h EBVs were involved in low levels of aggression, but contained individuals with both high and low EBVs for proportion of fights won. These results may address the criticism that selection for low lesions may simply result in selection for meek animals, as it would seem that some dominant individuals are able to convey social rank with very little aggression, possibly via behavioural cues, or short, decisive fights.

Genetic correlations indicate that high fight success and low receipt of non-reciprocal attacks are associated with few lesions to the central and posterior regions of the body. These correlations alone would suggest that selection against either of these traits could result in selection for highly dominant individuals. The results of the analysis performed in this study suggest that selection for low central SL24h EBVs would result in a reduction of several other behaviours, including initiation of reciprocal fighting. This suggests that individuals with low EBVs for central SL24h have a low propensity to be involved in both reciprocal and NRA. In contrast to

379 genetic correlations, traits likely to be related to dominance were not predicted to be
380 affected by selection for low central SL24h. This conflict seems to suggest that
381 central lesions are an ambiguous proxy measure of aggression, as they appear to
382 capture both aggressive and unaggressive individuals. In contrast, individuals with
383 low posterior SL24h had high values for proportion of fights won and duration of RA
384 initiated, and a decrease in all traits relating to the amount of aggression received.
385 This suggests that selecting against posterior SL24h would result in selection for
386 dominance related behaviours. Correlations between central or posterior SL24h and
387 aggressive traits presented in this study sometimes conflicted with those previously
388 calculated by Turner *et al.* (2008) using similar phenotyping methods. However, the
389 strength and direction of genetic correlations between anterior SL24h and behaviour
390 traits were similar between the two populations, providing further evidence that
391 anterior SL24h is the best trait overall for reducing aggression at mixing.
392
393 *Expected response following selection for increased SL3wk*
394 Due to lower and opposing genetic correlations between aggressive traits at mixing
395 and SL3wk, selection for increased SL3wk was predicted to reduce mean levels of
396 aggressive behaviour to a lesser extent than selection for low anterior SL24h.
397 Despite this, aggressive EBVs and phenotypes were still significantly lower than the
398 population mean in individuals with high EBVs for SL3wk. Selecting for increased
399 anterior or central SL3wk is expected to change mean EBVs for all aggressive traits
400 to a similar degree. Most behavioural EBVs were lower than the population as a
401 whole in this cohort; suggesting selection for increased lesions under stable social
402 conditions would result in a reduction in aggressive behaviour at mixing.
403

In accordance with genetic correlations, individuals with high EBVs for central SL3wk had significantly higher EBVs for the duration of NRA received, which conflicts with the aim of reducing aggression via selection. However, selection for increased central SL24h is predicted to result in a higher proportion of unaggressive animals in subsequent generations, and a lower proportion of aggressive animals. It would therefore also be expected that duration of NRA received would actually decrease in subsequent populations, despite positive genetic correlations between skin lesions and this behavioural trait. Similarly, it is expected that the number of skin lesions would also reduce under stable conditions, despite selecting for increased lesions at this time, as this would ultimately reduce the amount of aggression experienced by subordinate animals as hypothesised above. From a behavioural perspective, the results suggest there would be little difference between using anterior or central SL3wk for selection purposes.

Aggression is most intense upon first mixing and it is behaviour at this time point that has been the focus of most research. It is worth considering the implications of aggression under stable social conditions as, once mixed, animals are often housed for several weeks or months within these groups. Practically, counting skin lesions on larger, older animals in a socially stable environment is less time consuming than counting lesions on younger animals, as there are fewer lesions, the animals are more settled and tend to show less avoidance of an observer present in the pen. Furthermore, heritability estimates of skin lesion numbers under stable social conditions have been found to be of a higher magnitude to those inflicted under newly mixed conditions, possibly due to less environmental noise, (Turner *et al*.,

2009; Desire *et al*., 2015b), potentially increasing the response to selection for these traits.

At present, it is still not well understood how lesions three weeks post-mixing are related to longer-term aggressive behaviour. No study has yet looked at long-term aggressive behaviour in sufficient detail to allow for thorough investigation into the genetic and phenotypic relationships between skin lesion traits and aggression under stable and unstable social conditions. Lower correlations between behaviour at mixing and SL3wk suggests that individuals with the most lesions at three weeks may not always be the least aggressive individuals at this time point. Without behavioural information it is unknown what factors contribute to aggression under stable social conditions, and under what circumstances individuals engage in aggression. For example, lesions received under stable social conditions may be the result of attacks by dominant individuals or reciprocal fighting between subordinate individuals, perhaps partly due to unstable or ambiguous dominance hierarchies. Genetic correlations between SL24h, SL3wk and aggressive behaviour at mixing provide a conflicting narrative. Positive genetic correlations between SL24h and SL3wk (Turner *et al*., 2009; Desire *et al.* 2015b) suggest individuals that receive many lesions at mixing go on to receive many lesions under stable social conditions, whereas negative correlations between most behavioural traits at mixing and SL3wk suggest that on the whole, animals that are aggressive at mixing go on to have fewer lesions 3 weeks later. Direct behavioural observations on animals under stable social conditions are required to explore this further. Until long-term aggressive behaviour is better understood, skin lesions recorded under stable social conditions only provide information on the aggression performed by a group as a whole and not the

453 individual in question. In contrast, the relationship between skin lesions at mixing and
454 aggressive behaviour is well established. In particular, anterior SL24h are highly
455 correlated with RA, meaning that skin lesions on this body region result from the
456 actions of the individual in question. When anterior SL24h are used as a phenotype,
457 it is a good proxy measure of an individual's behaviour, rather than other animals in
458 the social group. Moreover, although mixing aggression has been studied for several
459 decades, the damaging effects of long-term aggression have not been quantified. It
460 should be noted that in this study skin lesion numbers recorded immediately prior to
461 mixing were subtracted from those counted 24 hours later, to ensure only those
462 lesions resulting from mixing aggression were included in the analysis. This
463 methodology effectively doubles the amount of labour required to record SL24h,
464 however correlations between raw anterior lesion numbers recorded 24 hours post-
465 mixing and lesion numbers adjusted for pre-mix counts were very high (0.95; $P < 0.001$) suggesting that recording skin lesions prior to mixing is not necessary.
466
467
468 This study provides evidence that significant reductions in social aggression could be
469 achieved via selection for skin lesions. Much of the variation in skin lesion numbers
470 is attributed to environmental factors, and previous research has demonstrated that
471 variation in management systems can affect the phenotypic expression of
472 aggression (Arey and Edwards, 1998), however information regarding how
473 environmental factors affect the genetic expression of these traits is limited. Although
474 previous studies have found phenotypic correlations between skin lesions and
475 aggression (Stukenborg *et al*., 2011; Tönepöhl *et al*., 2013; Turner *et al*., 2006) few
476 studies have estimated genetic correlations across traits. Results from populations
477 housed under different management systems suggest anterior SL24h is a reliable

measure of social aggression in growing pigs (Turner *et al*., 2008; Turner *et al*., 2009). In practice, selection for skin lesions would be incorporated into a selection index tailored to a wider breeding goal. Therefore, further research is required to estimate the genetic correlation with other traits in the breeding goal and derive the marginal economic and non-economic value of skin lesions to allow these traits to be weighted within a multi-trait commercial index.

Conclusion

Results suggest that selection against anterior SL24h would have the greatest effect on behaviour at mixing, both on a genetic and phenotypic level. The results also suggest that anterior SL24h are a more accurate representation of the behaviour of separate individuals, as opposed to other skin lesion traits which may be more representative of the behaviour of others in the pen. There is also evidence that selection for increased SL3wk would have the favourable effect of reducing aggressive behaviour at mixing, although to a lesser degree than selection against anterior SL24h. Although there are several advantages to using skin lesions recorded under stable social conditions to phenotype individuals for selection purposes, more research into the relationship between aggressive behaviour at mixing and aggression under stable social conditions is needed. In conclusion, with the evidence currently available, anterior SL24h would be the preferable trait for genetic selection, as it has the potential to significantly reduce levels of aggression observed in the first 24 hours post-mixing, and also reduce the genetic trend in longer-term aggression (three weeks post-mixing).

Acknowledgements

503 The authors gratefully acknowledge BBSRC, KTN, and Genus PLC for PhD
504 studentship funding, as well as the Scottish Government. The data used are from a
505 previous project funded by the European Union Sixth Framework Programme and
506 Scottish Government. The assistance of data recording of Quality Genetics, S. Ison,
507 M. Farish, M. Jack, B. Olsson, U. Schmidt, J. Vallgårda, E. Merin, and Vallrums
508 Lantbruk (Sweden) staff is gratefully acknowledged.

509

510

511 **References**

512 Alemu SW, Bijma P Møller, SH Janss, L and Berg, P 2014. Indirect genetic effects
513 contribute substantially to heritable variation in aggression-related traits in
514 group-housed mink (Neovison vison). Genetics Selection Evolution 46:30.

515 Arey D and Edwards S 1998. Factors influencing aggression between sows after
516 mixing and the consequences for welfare and production. Livestock Production
517 Science 56, 61–70.

518 Bijma P. 2010. Estimating indirect genetic effects: precision of estimates and
519 optimum designs. Genetics 186, 1013–1028.

520 Bijma P and Wade, M. J 2008. The joint effects of kin, multilevel selection and
521 indirect genetic effects on response to genetic selection. Journal of Evolutionary
522 Biology 21, 1175–1188.

523 D'Eath RB, Turner SP, Kurt E, Evans G Thölking L, Looft H, Wimmers K, Murani E,
524 Klont R, Foury A, Ison SH, Mormède P 2010. Pigs' aggressive temperament
525 affects pre-slaughter mixing aggression, stress and meat quality. Animal 4, 604–
526 616.

527 De Groot J, Ruis MA, Scholten JW, Koolhaas JM and Boersma WJ 2001. Long-term
528 effects of social stress on antiviral immunity in pigs. Physiology and Behavior
529 73, 145–158.

530 Ellen ED, Rodenburg TB, Albers GAA, Bolhuis JE, Camerlink I, Duijvesteijn N, Knol
531 EF, Peeters K, Reimert I, Sell-Kubiak E, Van Arendonk JAM, Visscher J, Bijma,

532 P 2014. The prospects of selection for social genetic effects to improve welfare
533 and productivity in livestock. Frontiers in Genetics 5, 1–14.

534 Faucitano L 2001. Causes of skin damage to pig carcasses. Canadian Journal of
535 Animal Science 3, 39-45

536 Gilmour AR, Gogel BJ, Cullis BR and Thompson R 2009. ASReml User Guide
537 release 3.0. VSN International Ltd, Hemel Hempstead, UK. www.vsni.co.uk

538 Guy JH, Burns SE, Barker JM and Edwards SA 2009. Reducing post-mixing
539 aggression and skin lesions in weaned pigs by application of a synthetic
540 maternal pheromone. Animal Welfare 18, 249–255.

541 Meese GB and Ewbank R 1973. The establishment and nature of the dominance
542 hierarchy in the domesticated pig. Animal Behaviour 21, 326-334.

543 Mendl M, Zanella AJ and Broom DM 1992. Physiological and reproductive correlates
544 of behavioural strategies in female domestic pigs. Animal Behaviour 44, 1107-
545 1121.

546 Sartori C and Mantovani R 2012. Indirect genetic effects and the genetic bases of
547 social dominance: evidence from cattle. Heredity 110, 3–9.

548 Stookey JM and Gonyou HW 1994. The effects of regrouping on behavioral and
549 production parameters in finishing swine. Journal of Animal Science 72, 2804–
550 2811.

551 Stukenborg A, Traulsen I, Puppe B, Presuhn U, and Krieter J 2011. Agonistic
552 behaviour after mixing in pigs under commercial farm conditions. Applied Animal
553 Behaviour Science 129, 28–35.

554 Tönepöhl B, Appel AK, Voß B, König von Borstel U and Gauly M 2013. Interaction
555 between sows' aggressiveness post-mixing and skin lesions recorded several
556 weeks later. Applied Animal Behaviour Science 44, 108-115.

557 Turner SP, D'Eath RB, Roehe R and Lawrence AB 2010. Selection against
558 aggressiveness in pigs at re-grouping: practical application and implications for
559 long-term behavioural patterns. Animal Welfare 19, 123-132

560 Turner SP, Farnworth MJ, White IM, Brotherstone S, Mendl M, Knap P, Penny P,
561 Lawrence AB 2006. The accumulation of skin lesions and their use as a
562 predictor of individual aggressiveness in pigs. Applied Animal Behaviour
563 Science 96, 245–259.

564 Turner SP, Roehe R, D'Eath RB, Ison SH, Farish M, Jack MC, Lundeheim N,
565 Rydhmer L, Lawrence AB 2009. Genetic validation of postmixing skin injuries in
566 pigs as an indicator of aggressiveness and the relationship with injuries under
567 more stable social conditions. Journal of Animal Science 87, 3076–3082.

568 Turner SP, Roehe R, Mekkawy W, Farnworth MJ, Knap PW and Lawrence AB 2008.
569 Bayesian analysis of genetic associations of skin lesions and behavioural traits
570 to identify genetic components of individual aggressiveness in pigs. Behavior
571 Genetics 38, 57-75.

572 Wilson AJ, Morrissey MB, Adams MJ, Walling CA, Guinness FE, Pemberton JM,
573 Clutton-Brock TH, Kruuk LEB 2011. Indirect genetics effects and evolutionary
574 constraint: An analysis of social dominance in red deer, *Cervus elaphus*. Journal
575 of Evolutionary Biology 24, 772–783.

576 Wilson AJ, Gelin U, Perron MC and Réale D 2009. Indirect genetic effects and the
577 evolution of aggression in a vertebrate system. Proceedings. Biological
578 Sciences / The Royal Society, 276(October 2008), 533–541.

579

580 **Tables**

581 Table 1 Definitions of skin lesion traits and behavioural traits used in the analyses

Trait	Description
Reciprocal aggression (RA)	A fight lasting >1s in which the recipient of the attack retaliated
Non-reciprocal aggression (NRA)	An attack in which the recipient did not retaliate
Number of RA involved with	Total number of reciprocal fights the focal pig was involved with, regardless of which pig initiated the attack
Proportion of fights won	Proportion of all reciprocal fights which the focal pig won
Average duration RA & NRA involved (s)	Average duration (in seconds) of all aggressive encounters in which the focal pig was involved
Duration of RA/NRA initiated (s)	Duration of time (in seconds) spent in aggression in which the focal pig was the initiator
Duration of RA/NRA received (s)	Duration of time (in seconds) spent in aggression in which the focal pig was the recipient of the attack
Number of pen mates attacked (RA/NRA)	The number of pen mates the focal pig attacked
Number of pen mates attacked by (RA/NRA)	The number of pen mates the focal pig was attacked by
Pen mates interacted with	Total number of pen mates with which the focal pig had any aggressive interactions

582
583

584

585

586 **Table 2** Heritabilities (h^2), phenotypic proportions of pen variances (c^2), phenotypic (σ^2p), and genetic (σ^2a) variances for skin lesion traits
587 recorded on pigs 24 hours post-mixing (SL24h) and 3 weeks post-mixing (SL3wk) and aggressive behavioural traits

588

	Trait	h^2	c^2	σ^2p	σ^2a
SL24h	Anterior	0.13 (0.05)	0.06 (0.02)	1.14	0.15
	Central	0.21 (0.06)	0.10 (0.03)	1.22	0.25
	Posterior	0.12 (0.05)	0.14 (0.03)	0.87	0.11
SL3wk	Anterior	0.43 (0.08)	0.03 (0.02)	0.31	0.13
	Central	0.39 (0.08)	0.06 (0.02)	0.35	0.13
	Posterior	0.11 (0.05)	0.07 (0.02)	0.44	0.05
Behaviour	Number of RA involved with	0.44 (0.08)	0.07 (0.02)	0.82	0.36
	Proportion of fights won	0.34 (0.08)	0.01 (0.02)	0.04	0.01
	Average duration of NA and NRA involved (s)	0.14 (0.05)	0.15 (0.03)	0.38	0.05
	Duration NRA initiated (s)	0.33 (0.07)	0.03 (0.02)	3.42	1.12
	Duration NRA received (s)	0.09 (0.04)	0.13 (0.03)	1.82	0.16
	Duration of RA initiated (s)	0.35 (0.08)	0.01 (0.02)	5.44	1.92
	Duration RA received (s)	0.28 (0.07)	0.06 (0.02)	2.08	0.58
	Number of pen mates attacked (RA)	0.40 (0.08)	0.04 (0.02)	0.48	0.19
	Number of pen mates attacked by (RA)	0.33 (0.07)	0.07 (0.02)	0.40	0.13
	Number of pen mates attacked (NRA)	0.31 (0.07)	0.02 (0.02)	0.57	0.18
	Number of pen mates attacked by (NRA)	0.11 (0.05)	0.19 (0.04)	0.43	0.05
	Number of attacks initiated (RA)	0.42 (0.08)	0.03 (0.02)	0.72	0.30
	Number of attacked received (RA)	0.32 (0.07)	0.07 (0.02)	0.61	0.20
	Number of pen mates interacted with	0.37 (0.08)	0.09 (0.03)	0.24	0.09

589

590 RA = reciprocal aggression; NRA = non-reciprocal aggression

591 **Table 3** Genetic (r_G) and pen level (r_C) correlations[1] between anterior, central and posterior skin lesion traits recorded on pigs 24 hours post-
592 mixing (SL24h), with aggressive behavioural traits recorded 24 hours following mixing (standard errors presented in parentheses)

	Anterior SL24h		Central SL24h		Posterior SL24h	
	r_G	r_C	r_G	r_C	r_G	r_C
Number of RA involved with	**0.78 (0.10)**	0.42 (0.21)	-0.02 (0.19)	-0.41 (0.23)	-0.11 (0.22)	**-0.47 (0.21)**
Proportion of fights won	0.24 (0.22)	0.01 (0.56)	**-0.49 (0.18)**	-0.46 (0.56)	**-0.52 (0.21)**	-0.48 (0.48)
Average duration of NA and NRA involved (s)	**0.66 (0.18)**	0.14 (0.22)	-0.17 (0.26)	-0.11 (0.20)	-0.36 (0.27)	-0.15 (0.19)
Duration of NRA initiated (s)	**0.56 (0.17)**	0.10 (0.36)	-0.21 (0.22)	0.01 (0.32)	-0.19 (0.23)	-0.02 (0.30)
Duration of NRA received (s)	0.34 (0.29)	**0.17 (0.03)**	**0.54 (0.24)**	-0.07 (0.04)	**0.67 (0.28)**	**-0.08 (0.03)**
Duration of RA initiated (s)	**0.72 (0.12)**	0.38 (0.39)	-0.10 (0.20)	-0.34 (0.43)	-0.32 (0.22)	-0.47 (0.40)
Duration of RA received (s)	**0.77 (0.12)**	**0.51 (0.20)**	0.10 (0.21)	-0.12 (0.25)	0.06 (0.24)	-0.02 (0.23)
Number of pen mates focal pig attacked (RA)	**0.75 (0.12)**	**0.21 (0.03)**	-0.07 (0.20)	**0.23 (0.03)**	-0.30 (0.22)	**0.22 (0.03)**
Number of pigs attacked by (RA)	**0.84 (0.10)**	**0.50 (0.02)**	0.08 (0.20)	**0.19 (0.03)**	0.16 (0.22)	**0.11 (0.03)**
Number of pen mates attacked (NRA)	**0.59 (0.17)**	0.17 (0.38)	-0.15 (0.22)	0.00 (0.35)	-0.07 (0.23)	0.03 (0.33)
Number of pen mates attacked by (NRA)	0.21 (0.29)	0.18 (0.21)	**0.54 (0.22)**	0.28 (0.18)	**0.67 (0.26)**	0.30 (0.16)
Number of attacks initiated (RA)	**0.73 (0.12)**	0.51 (0.27)	-0.05 (0.20)	-0.35 (0.31)	-0.28 (0.22)	-0.53 (0.28)
Number of attacked received (RA)	**0.85 (0.10)**	**0.58 (0.18)**	0.09 (0.20)	-0.39 (0.23)	0.12 (0.23)	-0.40 (0.21)
Number of pen mates interacted with	**0.69 (0.13)**	0.30 (0.22)	-0.01 (0.20)	-0.16 (0.22)	0.00 (0.22)	-0.05 (0.21)

593 [1] Bold font signifies correlation significantly different from 0

594 RA = reciprocal aggression; NRA = non-reciprocal aggression

595

596

597

598 Table 4: Genetic (r_G) and pen level (r_C) correlations[1] between anterior, central and posterior skin lesion traits recorded on pigs 3 weeks post-

599 mixing (SL3wk), with aggressive behavioural traits recorded 24 hours following mixing (standard errors presented in parentheses)

	Anterior SL3wk		Central SL3wk		Posterior SL3wk	
	r_G	r_C	r_G	r_C	r_G	r_C
Number of RA involved with	**-0.34 (0.14)**	-0.13 (0.31)	-0.30 (0.15)	-0.39 (0.23)	-0.31 (0.22)	-0.04 (0.26)
Proportion of fights won	**-0.49 (0.14)**	-0.35 (0.80)	**-0.45 (0.15)**	-0.79 (0.63)	-0.30 (0.23)	-0.29 (0.67)
Average duration of NA and NRA involved (s)	-0.19 (0.20)	-0.17 (0.27)	-0.30 (0.20)	-0.13 (0.22)	-0.30 (0.28)	-0.14 (0.22)
Duration of NRA initiated (s)	**-0.38 (0.15)**	-0.22 (0.41)	**-0.33 (0.16)**	-0.34 (0.33)	-0.28 (0.24)	-0.45 (0.34)
Duration of NRA received (s)	**0.51 (0.22)**	**-0.16 (0.04)**	**0.66 (0.19)**	**-0.18 (0.04)**	0.14 (0.34)	-0.06 (0.03)
Duration of RA initiated (s)	**-0.36 (0.15)**	0.05 (0.54)	-0.21 (0.16)	-0.44 (0.43)	-0.35 (0.23)	0.11 (0.45)
Duration of RA received (s)	-0.20 (0.17)	-0.31 (0.31)	-0.25 (0.17)	-0.29 (0.25)	-0.15 (0.25)	-0.17 (0.26)
Number of pen mates focal pig attacked (RA)	**-0.33 (0.15)**	0.05 (0.03)	-0.21 (0.16)	0.08 (0.04)	-0.33 (0.23)	-0.02 (0.03)
Number of pigs attacked by (RA)	**-0.33 (0.15)**	**-0.15 (0.04)**	**-0.39 (0.15)**	**-0.18 (0.04)**	-0.26 (0.24)	**-0.08 (0.03)**
Number of pen mates attacked (NRA)	-0.31 (0.16)	-0.18 (0.45)	-0.30 (0.16)	-0.32 (0.36)	-0.21 (0.25)	-0.41 (0.38)
Number of pen mates attacked by (NRA)	**0.47 (0.20)**	0.14 (0.25)	**0.58 (0.18)**	-0.12 (0.21)	0.22 (0.32)	-0.09 (0.21)
Number of attacks initiated (RA)	**-0.33 (0.15)**	-0.23 (0.39)	-0.20 (0.16)	-0.52 (0.30)	-0.25 (0.23)	-0.05 (0.34)
Number of attacked received (RA)	-0.32 (0.16)	-0.22 (0.31)	**-0.42 (0.15)**	-0.40 (0.23)	-0.32 (0.24)	-0.05 (0.26)
Number of pen mates interacted with	**-0.37 (0.15)**	-0.11 (0.30)	-0.20 (0.16)	-0.32 (0.23)	-0.28 (0.23)	-0.27 (0.24)

600 [1] Bold font signifies correlation significantly different from 0

601 RA = reciprocal aggression; NRA = non-reciprocal aggression

602 **Figure captions**

603 **Figure 1** Mean estimated breeding values (EBVs) for skin lesion traits of pigs with EBVs in
604 the lowest 10% for either anterior (a), central (c) or posterior (e) skin lesions recorded 24
605 hours post-mixing (SL24h), or highest 10% EBVs for anterior (b), central (d) or posterior (f)
606 skin lesions recorded 3 weeks post-mixing (SL3wk). Skin lesion trait that selection was
607 based on is indicated above each panel and shaded black.

608 **Figure 2** Mean phenotypic values for skin lesion traits of pigs with EBVs in the lowest 10%
609 for either anterior (a), central (c) or posterior (e) skin lesions recorded 24 hours post-mixing
610 (SL24h), or highest 10% EBVs for anterior (b), central (d) or posterior (f) skin lesions
611 recorded 3 weeks post-mixing (SL3wk). Skin lesion trait that selection was based on is
612 indicated above each panel and shaded black.

613 **Figure 3** Mean estimated breeding values (EBVs) for aggressive behavioural traits of pigs
614 with EBVs in the lowest 10% for either anterior (a), central (c) or posterior (e) skin lesions
615 recorded 24 hours post-mixing (SL24h), or highest 10% EBVs for anterior (b), central (d) or
616 posterior (f) skin lesions recorded 3 weeks post-mixing (SL3wk). Skin lesion trait that
617 selection is based on is indicated beneath each panel. Numbers on horizontal axes
618 correspond with the following behavioural traits: 1 - number of RA involved with; 2 -
619 proportion of fights won; 3 - average duration of RA and NRA involved (s); 4 - duration of
620 NRA initiated (s); 5 - duration of NRA received (s); 6 - duration of RA initiated (s); 7 -
621 duration of RA received (s); 8 - number of pen mates focal pig attacked (RA); 9 - number of
622 pigs attacked by (RA); 10 - number of pen mates attacked (NRA); 11 - number of pen mates
623 attacked by (NRA); 12 - total RA initiated; 13 - total RA received; 14 - number of pen mates
624 interacted with. RA = reciprocal aggression; NRA = non-reciprocal aggression.

625 **Figure 4** Mean phenotypic values for aggressive behavioural traits of pigs with EBVs in the
626 lowest 10% for either anterior (a), central (c) or posterior (e) skin lesions recorded 24 hours

post-mixing (SL24h), or highest 10% EBVs for anterior (b), central (d) or posterior (f) skin lesions recorded 3 weeks post-mixing (SL3wk). Skin lesion trait that selection is based on is indicated beneath each panel. Numbers on horizontal axes correspond with the following behavioural traits: 1 - number of RA involved with; 2 - proportion of fights won; 3 - average duration of RA and NRA involved (s); 4 - duration of NRA initiated (s); 5 - duration of NRA received (s); 6 - duration of RA initiated (s); 7 - duration of RA received (s); 8 - number of pen mates focal pig attacked (RA); 9 - number of pigs attacked by (RA); 10 - number of pen mates attacked (NRA); 11 - number of pen mates attacked by (NRA); 12 - total RA initiated; 13 - total RA received; 14 - number of pen mates interacted with. RA = reciprocal aggression; NRA = non-reciprocal aggression.

Supplementary Table S1 Characteristics of skin lesion traits recorded on pigs 24h post-mixing (SL24h) and 3 weeks post-mixing (SL3wk) and behavioural data for all animals included in the statistical analysis

		Original scale				Transformed scale		
Trait	***N***	***Min-Max***	***Mean (SD)***	***SK***	***K***	***Mean (SD)***	***SK***	***K***
Anterior SL24h	1146	0 - 99	19.07 (17.35)	1.37	2.27	2.58 (1.07)	-0.90	0.43
Central SL24h	1146	0 - 100	10.82 (12.03)	1.43	6.03	2.06 (1.1)	-0.64	-0.52
Posterior SL24h	1146	0 - 41	3.69 (8.30)	-0.73	4.08	1.36 (1.02)	-0.12	-1.30
Anterior SL3wk	1146	0 - 63	10.40 (5.62)	1.59	8.89	2.3 (0.56)	-1.15	2.66
Central SL3wk	1146	0 - 40	10.36 (5.93)	1.03	1.90	2.28 (0.6)	-0.92	1.54
Posterior SL3wk	1146	0 - 30	4.53 (3.49)	1.16	2.92	1.49 (0.71)	-0.52	-0.34
Number of RA involved with	1146	0 - 56	8.43 (7.16)	1.37	3.04	1.91 (0.9)	-0.60	-0.35
Proportion of fights won	1047	0 - 1	0.30 (0.25)	0.54	-0.27	0.25 (0.19)	0.20	-0.85
Average duration NA and NRA involved (s)	1138	1 - 250	42.70 (27.97)	2.03	8.24	3.59 (0.64)	-0.39	0.46
Duration NRA initiated (s)	1146	0 - 996	41.71 (68.81)	4.64	40.72	2.53 (1.82)	-0.19	-1.27
Duration NRA received (s)	1146	0 - 444	41.47 (46.53)	2.88	13.9	3.12 (1.34)	-0.88	0.36
Duration of RA initiated (s)	1146	0 - 2394	289.8 (366.2)	2.07	5.30	4.3 (2.34)	-0.88	-0.53
Duration RA received (s)	1146	0 - 2997	329.6 (353)	2.08	6.62	5.1 (1.45)	-1.08	1.45
Number of pen mates attacked (RA)	1146	0 - 11	2.86 (2.32)	0.65	-0.20	1.14 (0.69)	-0.40	-0.94
Number of pen mates attacked by (RA)	1146	0 - 9	2.86 (2.06)	0.40	-0.52	1.18 (0.63)	-0.59	-0.66
Number of pen mates attacked (NRA)	1146	0 - 14	2.57 (2.69)	1.32	1.54	1.00 (0.75)	0.06	-1.10
Number of pen mates attacked by (NRA)	1146	0 - 9	2.57 (1.67)	0.61	0.1	1.26 (0.45)	-1.64	2.39
Number of attacks initiated (RA)	1146	0 - 36	4.23 (4.30)	1.75	4.97	1.32 (0.85)	-0.14	-0.89
Number of attacked received (RA)	1146	0 - 25	4.20 (3.78)	1.44	3.04	1.37 (0.79)	-0.32	-0.72
Number of pen mates interacted with	1146	0 - 14	6.69 (3.06)	0.02	-0.66	1.94 (0.49)	-1.13	1.48

RA = reciprocal aggression; NRA = non-reciprocal aggression; SK = skewness; K = kurtosis

Supplementary Table S2 Comparison of lesion traits and aggressive behavioural traits for all pigs and pigs in the lowest 10th percentile of EBVs for anterior skin lesions recorded 24 hours post mixing (SL24h). Raw mean values for all pigs, mean phenotypic differences (in SD) between all pigs and selected pigs, and expected mean change (in trait units) after selection for SL24h.

	Trait	Raw mean of all pigs	Mean difference between all and selected pigs in SD (SE)[1]	Expected change after selection[2]
SL24h	Anterior (selection trait)	19.07	**-0.91 (0.03)**	-15.85
	Central	10.82	**-0.61 (0.05)**	-7.35
	Posterior	3.69	**-0.43 (0.07)**	-3.60
SL3wk	Anterior	10.40	-0.06 (0.08)	-0.34
	Central	10.36	0.07 (0.10)	0.43
	Posterior	4.53	0.00 (0.07)	0.01
Behaviour	Number of RA involved with	8.43	**-0.63 (0.06)**	-4.51
	Proportion of fights won	0.30	-0.06 (0.14)	-0.01
	Average duration of NA and NRA involved (s)	42.70	**-0.53 (0.06)**	-14.76
	Duration of NRA initiated (s)	41.71	**-0.24 (0.06)**	-16.29
	Duration of NRA received (s)	41.47	**-0.25 (0.07)**	-11.55
	Duration of RA initiated (s)	289.80	**-0.44 (0.06)**	-160.42
	Duration of RA received (s)	329.60	**-0.62 (0.05)**	-217.42
	Number of pen mates attacked (RA)	2.86	**-0.57 (0.08)**	-1.32
	Number of pigs attacked by (RA)	2.86	**-0.74 (0.07)**	-1.52
	Number of pen mates attacked (NRA)	2.57	**-0.28 (0.09)**	-0.76
	Number of pen mates attacked by (NRA)	2.57	**-0.44 (0.11)**	-0.73
	Total RA initiated	4.23	**-0.47 (0.07)**	-2.03
	Total RA received	4.20	**-0.66 (0.06)**	-2.48
	Number of pen mates interacted with	6.69	**-0.60 (0.09)**	-1.83

SL24h = skin lesions recorded 24 hours post-mixing; SL3wk = skin lesions recorded 3 weeks post-mixing ; RA = reciprocal aggression; NRA = non-reciprocal aggression

[1] Bold font signifies change significantly different from 0

[2] Untransformed phenotypes were scaled and standardised (see Table S1) and the corresponding change in SD after selection based on breeding values was used to calculate the expected change in aggressive behaviour.

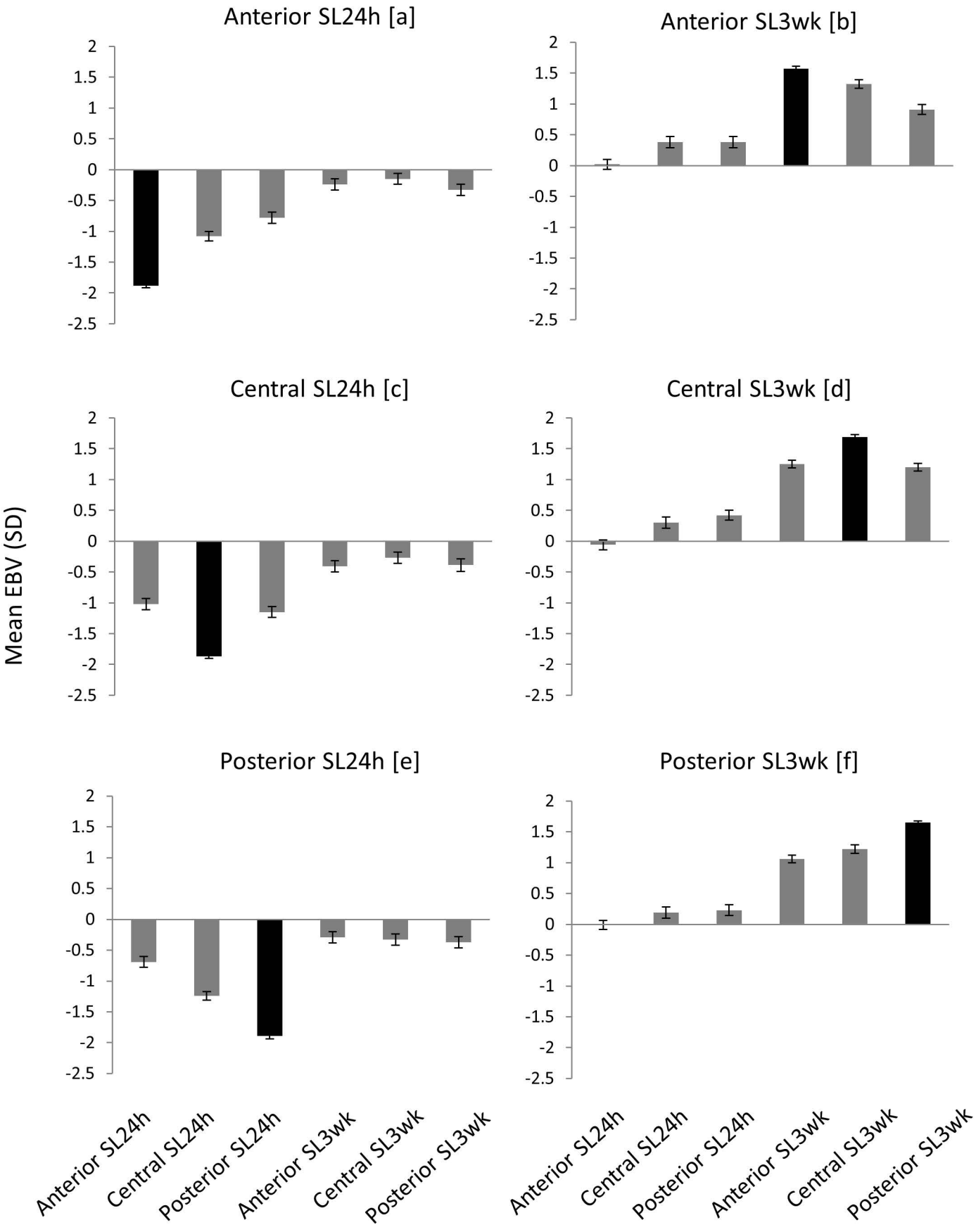
Supplementary Table S3 Comparison of lesion traits and aggressive behavioural traits for all pigs and pigs in the highest 10th percentile of EBVs for anterior skin lesions recorded 3 weeks post mixing (SL3wk). Raw mean values for all pigs, mean phenotypic differences (in SD) between all pigs and selected pigs, and expected mean change (in trait units) after selection for SL3wk

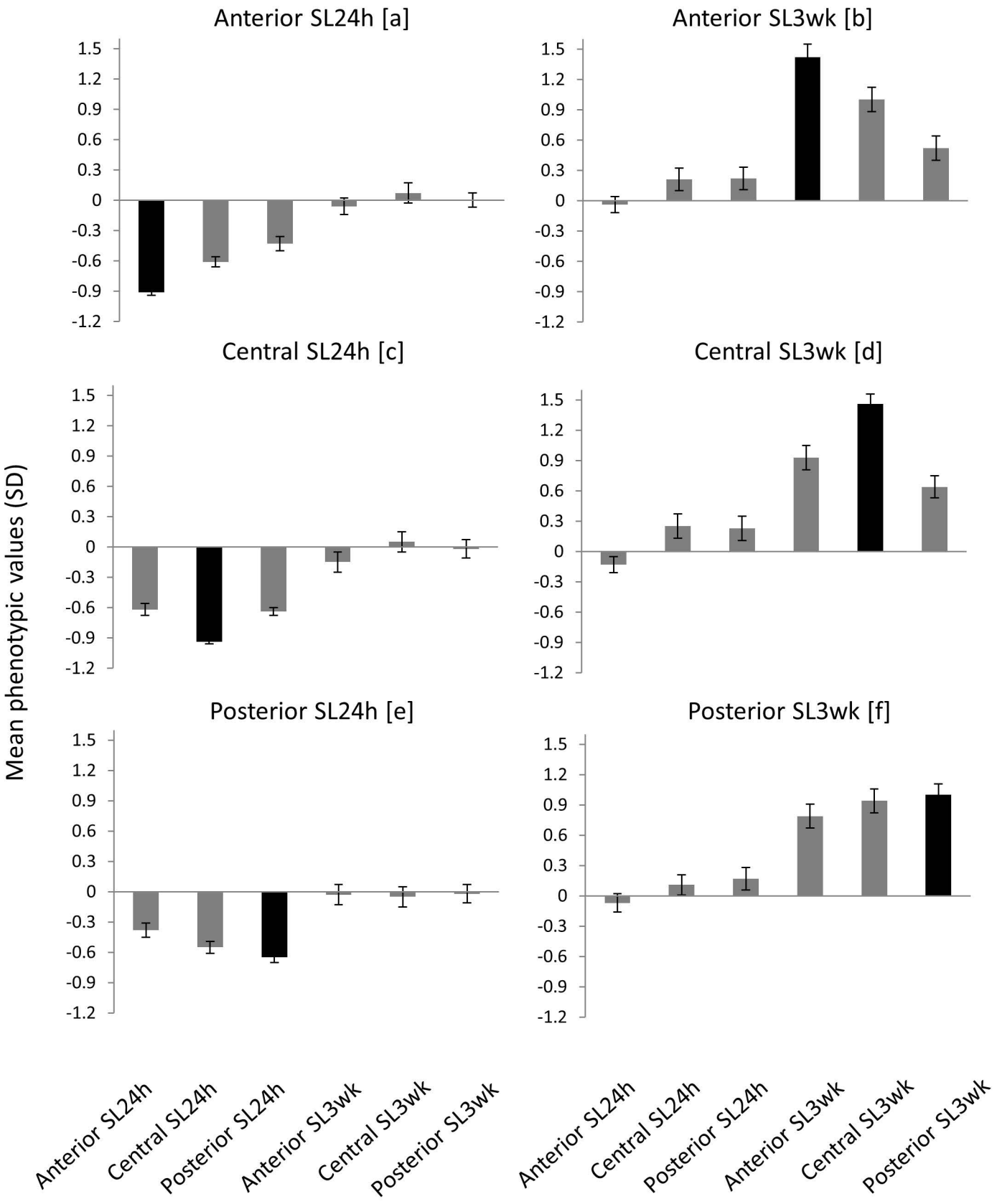
	Trait	Raw mean of all pigs	Mean difference between all and selected pigs in SD (SE)[1]	Expected change after selection[2]
SL24h	Anterior	19.07	-0.04 (0.08)	-0.76
	Central	10.82	0.21 (0.11)	2.58
	Posterior	3.69	0.22 (0.11)	1.79
SL3wk	Anterior (selection trait)	10.40	**1.42 (0.13)**	7.95
	Central	10.36	**1.00 (0.12)**	5.91
	Posterior	4.53	**0.52 (0.12)**	1.82
Behaviour	Number of RA involved with	8.43	**-0.33 (0.08)**	-2.39
	Proportion of fights won	0.30	**-0.31 (0.09)**	-0.08
	Average duration of NA and NRA involved (s)	42.70	**-0.19 (0.07)**	-5.30
	Duration of NRA initiated (s)	41.71	-0.13 (0.08)	-8.72
	Duration of NRA received (s)	41.47	-0.10 (0.07)	-4.50
	Duration of RA initiated (s)	289.80	**-0.30 (0.07)**	-108.12
	Duration of RA received (s)	329.60	**-0.28 (0.07)**	-98.06
	Number of pen mates focal pig attacked (RA)	2.86	**-0.35 (0.09)**	-0.81
	Number of pigs attacked by (RA)	2.86	**-0.35 (0.08)**	-0.71
	Number of pen mates attacked (NRA)	2.57	**-0.28 (0.08)**	-0.75
	Number of pen mates attacked by (NRA)	2.57	0.1 (0.09)	0.17
	Total RA initiated	4.23	**-0.29 (0.08)**	-1.25
	Total RA received	4.20	**-0.3 (0.07)**	-1.14
	Number of pen mates interacted with	6.69	**-0.39 (0.09)**	-1.19

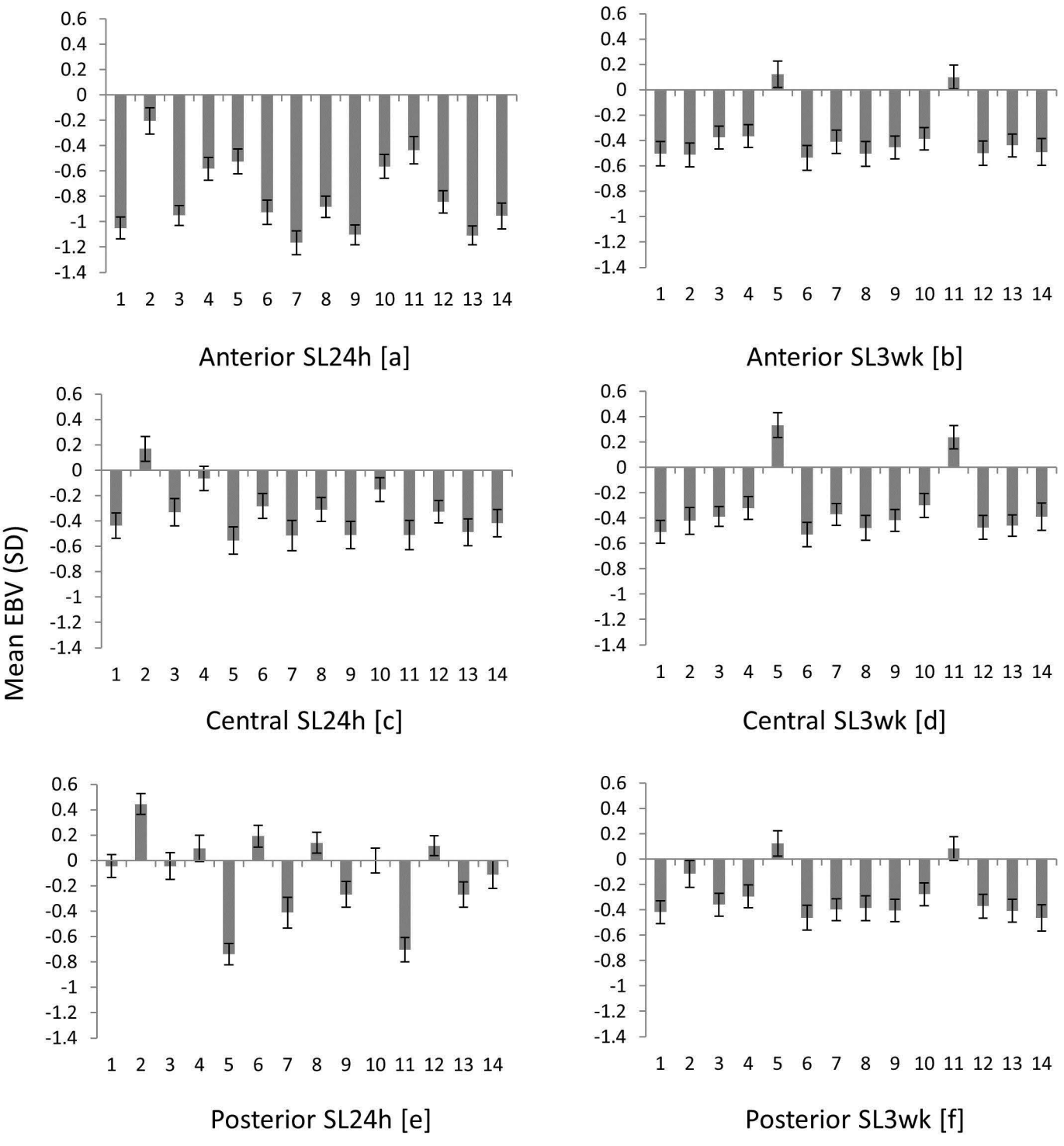
SL24h = skin lesions recorded 24 hours post-mixing; SL3wk = skin lesions recorded 3 weeks post-mixing; RA = reciprocal aggression; NRA = non-reciprocal aggression

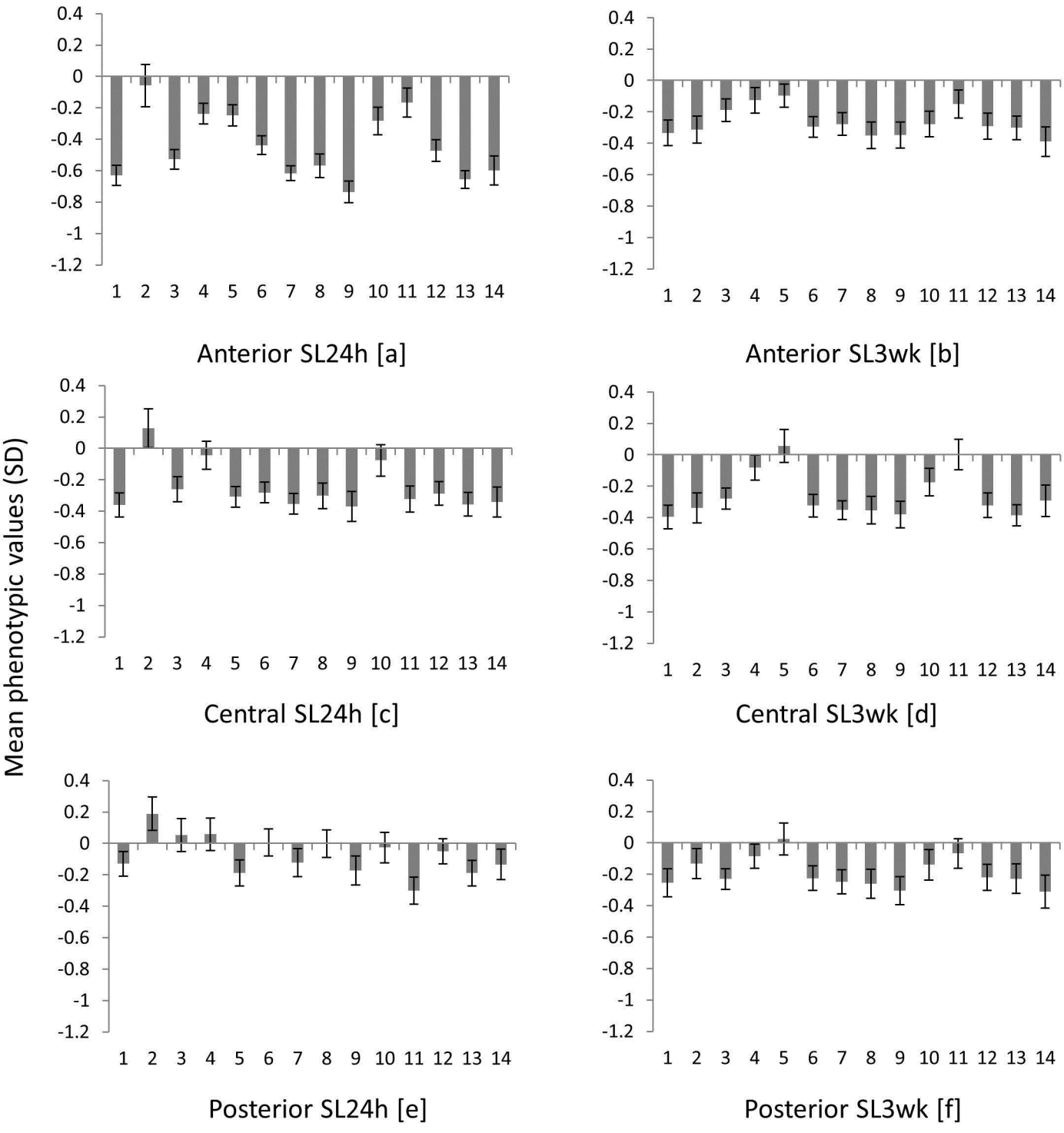
[1] Bold font signifies change significantly different from 0

[2] Untransformed phenotypes were scaled and standardised (see Table S1) and the corresponding change in SD after selection based on breeding values was used to calculate the expected change in aggressive behaviour.


Anterior SL24h [a]
Anterior SL3wk [b]
Central SL24h [c]
Central SL3wk [d]
Posterior SL24h [e]
Posterior SL3wk [f]
Mean EBV (SD)
2
1.5
1
0.5
0
-0.5
-1
-1.5
-2
-2.5
Anterior SL24h
Central SL24h
Posterior SL24h
Anterior SL3wk
Central SL3wk
Posterior SL3wk


Anterior SL24h [a]
Anterior SL3wk [b]
Central SL24h [c]
Central SL3wk [d]
Posterior SL24h [e]
Posterior SL3wk [f]
Mean phenotypic values (SD)
1.5
1.2
0.9
0.6
0.3
0
-0.3
-0.6
-0.9
-1.2
Anterior SL24h
Central SL24h
Posterior SL24h
Anterior SL3wk
Central SL3wk
Posterior SL3wk


Mean EBV (SD)
0.6
0.4
0.2
0
-0.2
-0.4
-0.6
-0.8
-1
-1.2
-1.4
1 2 3 4 5 6 7 8 9 10 11 12 13 14
Anterior SL24h [a]
Anterior SL3wk [b]
Central SL24h [c]
Central SL3wk [d]
Posterior SL24h [e]
Posterior SL3wk [f]


Mean phenotypic values (SD)
0.4
0.2
0
-0.2
-0.4
-0.6
-0.8
-1
-1.2
1 2 3 4 5 6 7 8 9 10 11 12 13 14
Anterior SL24h [a]
Anterior SL3wk [b]
Central SL24h [c]
Central SL3wk [d]
Posterior SL24h [e]
Posterior SL3wk [f]