

Social Behaviour In Pigs

**S.P. Turner, *R. Roehle and *A.B. Lawrence*

Introduction

The expression of social behavioural traits is harmful when they challenge animal welfare or profitability. In pigs, these traits include intra-specific aggression, oral manipulation of pen mates (tail biting, ear biting and belly nosing) and savaging of piglets by their dams (here regarded as a social trait, although partly a neophobic response; Baxter 2008). These traits occur with high frequency (eg around 10% of grower-stage pigs receive more than 50 skin lesions from aggression within 24h of regrouping (Turner et al. 2009), between 14 and 20% of outdoor reared pigs show evidence of tail biting lesions at slaughter (Walker and Bilkei 2006) and between 6 and 15% of primiparous sows savage their piglets (Knap and Merks 1987; van der Steen et al. 1988; Chen et al. 2008)). Such behaviours are not observed in the wild, although aggression occurs in certain contexts, and their expression highlights a mismatch between the animal and its environment. These traits have complex causation, development and expression. Furthermore, the outcomes are usually most apparent in the recipient, rather than the actor. Manipulating the expression of these traits is technically challenging but has the potential to lead to many correlated benefits.

The role of breeding

Major changes in husbandry approaches are capable of minimising the expression of harmful social behaviours but are prohibitively expensive. Harmful behavioural traits are therefore tackled by low-cost, low-benefit approaches (eg provision of chains to minimise tail biting, use of scent masking sprays to reduce fighting) and otherwise tolerated as an insurmountable outcome of modern production methods. Faced with management change being highly constrained, a logical, but to some unpalatable alternative is to selectively breed genotypes which thrive in current and future production systems without the expression of harmful social behaviours. Breeding has the advantage of delivering cumulative and permanent benefits at relatively little cost to individual producers. Arguments have also been made that past selection for economic traits may have led to correlated increases in expression of harmful social behaviours in pigs (e.g. tail-biting, Breuer et al. 2005) and it may be appropriate to reverse this trend through the use of broader breeding goals. In this paper we explore the progress made in estimating the genetic contribution to harmful social behavioural traits in pigs and the probable consequences of selection on these for other traits of economic or welfare significance. We also consider the barriers that have prevented uptake of selection to date and how these may be overcome.

Genetic contribution to social behavioural traits in pigs

Aggression at regrouping. Pigs fight when mixed into new social groups. This compromises the rate and efficiency of weight gain, meat eating quality, carcass gradings

and maternal ability (Rundgren and Löfquist 1989; Tan et al. 1991; Løvendahl et al. 2005; D'Eath et al. 2010a). It also heightens the risk of infection, reduces immunocompetence and increases the risk of disease spread (Muirhead 1983; Morrow-Tesch et al. 1994). Aggressive behaviour can be classified into reciprocal fighting, and the delivery or receipt of non-reciprocated bullying. In line with aggressive traits in other species (e.g. male rodents $h^2 = 0.22$ to 0.34 , Miczek et al. 2001; aggressive anti-social behavior in humans $h^2 = 0.46$, Eley et al. 2003), these traits in pigs have moderate heritabilities ($h^2=0.17-0.43$), although that of the receipt of bullying is low ($h^2=0.04-0.08$, Løvendahl et al. 2005; Turner et al. 2009). Observations of aggressive behaviour are time consuming and Turner et al. (2009) have estimated the genetic correlations between aggressive behaviour and the number of resulting skin lesions as an indicator of involvement in aggressive behaviour. These correlations suggest that a genetic merit index could use lesions to the front as one trait and to the middle and rear as a second trait to select against pigs which fight and bully others. Counting the number of lesions takes around 30-60 seconds per pig. Turner et al. (2006) found no significant genetic correlation between the number of lesions and either growth rate over a 3 month period or backfat depth in grower stage pigs. The relationship to reproductive traits is currently being assessed, but data from Løvendahl et al. (2005) suggest that selection to reduce fighting would not compromise maternal care of the piglets. However, McLean et al. (1998) showed a phenotypic relationship between low aggressiveness in pregnant sows and a subsequently elevated risk of savaging the piglets after birth.

Selection to reduce lesions at mixing is expected to also reduce their number under more stable social conditions after the dominance hierarchy is believed to have been established (r_g between lesions at mixing and 3 weeks post-mixing = $0.28-0.50$, Turner et al. 2009). Behaviourally unaggressive pigs do not appear to be less active than more aggressive animals, but they respond to handling differently requiring more encouragement to enter a weigh crate but struggling less once inside (D'Eath et al. 2009). Other evidence at the phenotypic level suggests that less aggressive pigs (fewer lesions) are more fearful of human contact (Brown et al. 2009). Handling ease might therefore be affected by selection to reduce lesions. Using lesions as an easily measured indicator trait, aggression can therefore be reduced at mixing with anticipated longer-term benefits. This is not expected to result in antagonistic genetic relationships with key existing traits in an index.

Quantitative selection could be augmented by knowledge of polymorphisms affecting aggression. SNPs in genes encoding components of the hypothalamic-pituitary-adrenal stress axis (*vasopressin receptor 1 β* and *glucocorticoid receptor*) affect lesions and stress reactivity (plasma cortisol concentration; Murani et al. 2009). These findings are in line with those of Wersinger et al. (2007) where vasopressin receptor 1 β knock-out mice showed reductions in intra-specific aggression. Such results provide the basis for future studies directed at identifying the causal functional DNA sequence variations that could allow the development of markers for use in breeding.

Oral manipulation of pen mates. Tail biting is perhaps the most problematic of the non-aggressive oral behaviours directed at pen mates. It is triggered by a number of environmental and nutritional causes (reviewed by Schroder-Petersen and Simonsen 2001 and Moinard et al. 2003) and is phenotypically associated with depressed growth rate in both

actor and receiver and an increased risk of carcass condemnation (Beattie et al. 2005; Walker and Bilkei 2006). It can, additionally, contribute to clinical infection and is a cause of mortality (Muirhead 1983; Hsu et al. 1985). Although only a small percentage of pigs bite the tails of others (2.8-3.5%, Breuer et al. 2005), the percentage of animals showing lesions from the receipt of tail-biting can be substantially higher, even outdoors (14-20%, Walker and Bilkei 2006). Tail biting commonly occurs alongside other forms of harmful oral manipulation, such as ear and vulva biting and belly nosing (Rizvi et al. 1998; Hunter et al. 1999; Beattie et al. 2005) which can also occur at high levels (Rizvi et al. 1998). Although vulva biting originates as an aggressive act (van Putten and van de Burgwal 1990), it may develop into a vice that has parallels with other oral behaviours. Breed effects have been reported for tail biting and ear chewing (Breuer et al. 2003) and both breed and individual sire effects have been found for belly nosing (Breuer et al. 2003; Bench and Gonyou 2007). Breuer et al. (2005) estimated a heritability of 0.05 ± 0.02 for tail biting behaviour as a binary trait in Landrace pigs with an equivalent heritability of 0.27 as a continuously distributed trait. The heritability was not different from zero in Large White pigs. Low but significant unfavourable genetic correlations between tail biting and lean tissue growth rate and back fat thickness were apparent in the Landrace population, suggesting that selection on tail biting may be justified on the basis of slowing or reversing the effects of selection on economic traits. Similarly, Torrey et al. (2001) found evidence of increased belly nosing in a line of pigs selected for loin eye muscle area.

Maternal aggression towards piglets. Between 6 and 15% of primiparous sows show deliberate maternal aggression resulting in severe piglet injury or death (savaging; Knap and Merks 1987; van der Steen et al. 1988; Chen et al. 2008), although Edwards (2002) reports much lower levels. Sedation offers the only preventative approach, but the onset of savaging may simply be delayed (Chen et al. 2008).

The heritability of savaging ranged from 0.4-0.9 in three purebred lines based on paternal half-sib and daughter-dam analyses in a study by Knap and Merks (1987). Values of between 0.12 and 0.25 using a paternal half sib analysis reported by van der Steen et al. (1988) confirm that savaging should respond to selection. More recently, four QTL associated with savaging have been identified (Quilter et al. 2007) and evidence of differential gene expression at these loci has been reported (Quilter et al. 2008). A finding by Baxter et al. (2008) that selection of a high survival line resulted in reduced crushing of piglets indoors but heightened savaging suggests that these behaviours may be genetically associated and that selection to reduce savaging may increase crushing in some environments. Selection against savaging offers the potential of significant welfare and economic gains, but the implications for other components of maternal behaviour must be more fully understood.

Consequences of selection for underlying motivations

It seems technically possible to reduce the expression of harmful social behaviour in pigs through selection. This ought to benefit the recipients of such behaviour but the impacts on the emotional experiences of the actor are more difficult to determine. Sandøe et al. (1999) and D'Eath et al. (2010b) have discussed how selection may prevent the expression of harmful behaviours without suppressing the motivation to perform these behaviours and

therefore may not improve the mental suffering of the actors. As one example, Turner et al. (2010) have proposed that reduced aggression may be achieved through a greater tolerance of unfamiliar individuals or conversely a greater fear of involvement in aggression (eg through heightened pain perception) or the perception of greater stress from previous defeat. Effort must be directed at understanding in more detail the phenotypes that would result from selection, the cognitive processes that underlie those phenotypes and by inference, the emotional experiences of the animals. Assuming that selection would lead to animals that were less stressed by their environment, this may still be achieved at the expense of compromising the animal's integrity, making it in some way less complete than it was previously, which some regard as unethical (D'Eath et al. 2010b). Conversely, it may be regarded as a moral imperative to utilise selection where this can eliminate the need for routine and painful interventions such as tail docking. Reconciling these views is probably necessary on a case by case basis making reference to the presence of the behaviour in the behavioural repertoire of wild pigs, an understanding of the cognitive basis of post-selection behavioural expression and the likely welfare improvements to both actor and receiver.

Practical barriers to implementation and their resolution

Costs of phenotyping. Phenotyping complex traits for quantitative selection is a substantial but poorly quantified cost that discourages selection against harmful traits. The development and validation of easily measured indicator traits is crucial to minimising this cost. Further constraints are the need to standardise the environment to allow all animals equal opportunity to show their phenotype and the possibility that changes in a trait may be highly context or age specific, limiting the benefits of selection. The expectation that genetic markers can be used to test for favourable alleles with major effects is probably optimistic for such traits. Combining the minor contributions of multiple loci into a genomic breeding value offers one route for selection on traits that are costly to measure, although periodic phenotyping is still required and the costs saved by routine phenotyping must be offset against the costs of genotyping, which at present remain substantial. Using a 60,000 base pair SNP chip, we are currently assessing the feasibility of developing genomic breeding values for aggressiveness in a small population of pigs.

Estimation of benefits. Critical to implementation is the full quantification of the costs and benefits of selection, including the interactions with traits in existing indexes. Harmful traits have both an economic and non-economic value in which the latter reflects the societal good resulting from their improvement. Kanis et al. (2005) have described methods to quantify the non-economic benefits in monetary terms which is necessary to place these traits in an index alongside economic traits. Currently even the more readily quantifiable economic values of these traits have not been estimated. Genetically correlated improvements in disease resistance, infection risk, lameness, longevity, reproductive performance and wastage from carcass trim and condemnation may be realisable through selection. Taking a wider perspective, a starting point to quantify the benefits of selection, which at this stage would necessarily involve multiple assumptions until data are available, would be to estimate benefit functions based on inputs (costs of implementing intervention strategies) and outputs (for welfare and profitability) to identify impacts of alternative strategies to minimise harmful behaviours (Stott and Gunn 2008). Benefit functions provide a well defined way of

comparing different collections of benefits and could allow comparison of the benefits of selection versus other approaches to reduce harmful behaviours such as providing greater opportunity to escape from harmful behaviours. Optimum management and selection scenarios could also be modelled by linear programming methodology to identify maximum welfare advantages achievable by different options based on imposed constraints such as a pre-determined maximum implementation cost (Pla et al. 2009).

Selection on associative effects

For animals living in groups, an individual's phenotype has a heritable impact on the phenotype of others (Wolf et al. 1998; Agrawal et al. 2001). Bijma et al. (2007a) has shown that incorporation of these associative effects into selection indexes for economic traits ought to considerably increase the heritable variation open to selection. Additionally, in competitive environments where direct selection for performance traits will favour competitive over less competitive individuals, ignoring associative effects risks resulting in a response to selection opposite to that intended (Muir and Schinckel 2002; Chen et al. 2007). As social behaviour is the major route through which associate effects are likely to operate, they will be important for any trait affected by social interactions or competition for limited resources (Ellen et al. 2007). Beneficial changes in social behaviour may therefore result as a by-product of selection on associative effects to improve economic traits without the need for behavioural phenotyping, estimation of benefits or inclusion of new traits in an index.

Based on these arguments Muir (2005) and Bijma et al. (2007b) have proposed that associative effects be incorporated into selection indexes for economic traits whenever social interactions between individuals may be important. In pigs, estimates of the heritability of associative effects on growth rate appear to be small (up to 0.03; Arango et al. 2005; Chen et al. 2007, 2009). However, the variance of associative effects can be large (Bergsma et al. 2008) and where this is the case it may be worthwhile incorporating them in genetic analyses (Chen et al. 2009).

It has been suggested that selection on associative effects may benefit animal welfare (Muir, 2005; Bijma et al. 2007b). Rodenburg et al. (2010) has described how selection for low mortality reduced cannibalism in laying hens and resulted in a reduction in behavioural indicators of stress and corticosterone in a range of behavioural tests. However, the behavioural mechanisms through which an individual has a beneficial associative effect on other group members is largely unknown in pigs. These may be the net result of several phenotypic traits expressed within a single individual or by different individuals (Bijma et al. 2007b) and include low aggressiveness, the absence of oral manipulation of pen mates, reduced activity, reduced sexual mounting or altered feeding behaviour. D'Eath et al. (2010b) have highlighted the risks of selecting for traits with potentially wide ranging effects. To fully understand the long-term behavioural and welfare consequences of selection on associative effects, it is necessary to understand the behavioural phenotypes that would be favoured by the use of such methodology. As a first step towards developing this understanding, the genetic associations between pig aggressiveness and associative effects on growth rate of group members were studied during a 3 week period following regrouping (Canario et al. 2009). Correlations between breeding values indicated that a beneficial

associative effect on growth occurred through involvement in decisive fights and the accumulation of lesions to the front of the body immediately following mixing and the subsequent receipt of aggression and reduced activity 3 weeks post-mixing. This was interpreted as evidence of beneficial associative effects encouraging the more rapid establishment of dominance relationships. Additional behavioural traits may also have been important but were not measured. These results are in line with those described by Rodenburg et al. (2010) who found an increased number of lesions on the front of pigs estimated to have a beneficial associative breeding value.

It is probable that the behavioural traits conferring a beneficial associative effect will vary between environments and potentially over time as selection alters the balance of behavioural strategies played by others in the population. Whilst selection on associative effects may offer a mechanism to reduce the expression of some harmful behavioural traits as a welcome by-product of improving existing economic traits, it requires an initial and thereafter periodic assessment of the behavioural phenotypes generated in order to understand the consequences of selection for welfare.

Conclusions

Harmful social behaviours in pigs occur at a high frequency and have impacts on welfare and profitability. These traits have a low to moderate heritability and selection against their expression could be justified in light of the costs of alternative strategies of improving management approaches. The response to selection is likely to be slow, except for post-mixing aggression. There appear to be three areas of activity needed to facilitate a well informed decision on the merits of selection. Firstly, there is a need to develop and validate easily measured indicator traits for some behaviours. Secondly, the consequences of selection on these traits for other traits of welfare and economic significance needs to be addressed, as is on-going in the case of aggression. This step must also allow inferences to be made about how selection against behavioural expression may affect the emotional experiences of the actor based on detailed knowledge about the causal mechanism resulting in the reduced expression of the behaviour. Thirdly, for all of the traits described, there is a clear lack of quantitative data demonstrating the economic and non-economic value of their improvement. Currently, the costs of implementing selection are estimable, but the benefits of doing so are intangible. Until this is resolved, direct selection using quantitative methodology, marker or genome wide selection is unlikely to be implemented. Selection on associative effects offers real potential to by-pass these challenges, but introduces challenges of its own to understand its behavioural mechanisms of action. The behaviours likely to be affected by this approach, their sensitivity to physical and social environmental conditions and their dynamics over time require detailed study. This process is now beginning. Whichever selection approach may be adopted in the future, selection must not be used to justify further changes in management that challenge the adaptability of livestock.

References

- Agrawal, A.F., Brodie III, E.D. and Wade, M.J. (2001). *Am. Naturalist*, 158:308-323.
- Arango, J., Misztal, I., Tsuruta, S. *et al.* (2005). *J. Anim. Sci.*, 83:1241-1246.

- Baxter, E.M. (2008). PhD thesis, University of Newcastle.
- Baxter, E., Jarvis, S., Sherwood, L. *et al.* (2008). In *Proc Brit. Soc. Anim. Sci.*, page 11.
- Beattie, V.E., Breuer, K., O'Connell, N.E. *et al.* (2005). *Anim. Sci.*, 80:307-312.
- Bench, C.J. and Gonyou, H.W. (2007). *Appl. Anim. Behav. Sci.*, 105:26-41.
- Bergsma, R., Kanis, E., Knol, E.K. *et al.* (2008). *Genetics*, 178:1559-1570.
- Bijma, P., Muir, W.M. and Van Arendonk, J.A.M. (2007a). *Genetics*, 175:277-288.
- Bijma, P., Muir, W.M., Ellen, E.D. *et al.* (2007b). *Genetics*, 175:289-299.
- Breuer, K., Sutcliffe, M.E.M., Mercer, J.T. *et al.* (2003). *Appl. Anim. Behav. Sci.*, 84:59-74.
- Breuer, K., Sutcliffe, M.E.M., Mercer, J.T. *et al.* (2005). *Livest. Prod. Sci.*, 93:87-94.
- Brown, J.A., Dewey, C., Delange, C.F.M. (2009). *Appl. Anim. Behav. Sci.*, 118:28-35.
- Canario, L., Bergsma, R., D'Eath, R.B. *et al.* (2009). In *Proc UFAW International Symposium*, Bristol, page 31.
- Chen, C.Y., Johnson, R.K., Newman, S. *et al.* (2007). *Genet. Molecular Res.*, 6:594-606.
- Chen, C., Gilbert, C., Yang, G. *et al.* (2008). *Appl. Anim. Behav. Sci.*, 109:238-248.
- Chen, C.Y., Johnson, R.K., Newman, S. *et al.* (2009). *J. Anim. Sci.*, 87:844-849.
- D'Eath, R.B., Turner, S.P., Kurt, E. *et al.* (2010a). *Animal*, 4:604-616.
- D'Eath, R.B., Conington, J., Lawrence, A.B. *et al.* (2010b). *Anim. Welfare*, in press.
- D'Eath, R.B., Roehe, R., Turner, S.P. *et al.* (2009). *Animal*, 3:1544-1554.
- Edwards, S.A. (2002). *Livest. Prod. Sci.*, 78:3-12.
- Eley, T.C., Lichtenstein, P. and Moffitt, T.E. (2003). *Develop. Psychopathol.*, 15:383-402.
- Ellen, E.D., Muir, W.M., Teuscher, F. *et al.* (2007). *Genetics*, 176:489-499.
- Hsu, F.S., Chung, W.B., Hu, D.K. *et al.* (1985). *J. Chinese Soc. Vet. Med.*, 11:93-102.
- Hunter, E.J., Jones, T.A., Guise, H.J. *et al.* (1999). *Pig J.*, 43:18-32.
- Kanis, E., De Greef, K.H., Hiemstra, A. *et al.* (2005). *J. Anim. Sci.*, 83:948-957.
- Knap, P.W. and Merks, J.W.M. (1987). *Livest. Prod. Sci.*, 17:161-167.
- Løvendahl, P., Damgaard, L.H., Nielsen, B.L. *et al.* (2005). *Livest. Prod. Sci.*, 93:73-85.
- McLean, K.A., Lawrence, A.B., Petherick, J.C. *et al.* (1998). *Anim. Repro. Sci.*, 50:95-109.
- Miczek, K.A., Maxson, S.C., Fish, E.W. *et al.* (2001). *Behav. Brain Res.*, 125:167-181.
- Moinard, C., Mendl, M., Nicol, C.J. *et al.* (2003). *Appl. Anim. Behav. Sci.*, 81:333-355.

- Morrow-Tesch, J.L., McGlone, J.J. and Salak-Johnson, J.L. (1994). *J. Anim. Sci.*, 72:2599-2609.
- Muir, W.M. (2005). *Genetics*, 170:1247-1259.
- Muir, W.M. and Schinckel, A. 2002. In *Proc 7th WCGALP*, volume 27, communication 14-07.
- Muirhead, M.R. (1883). *Vet. Rec.*, 113:587-593.
- Murani, E., D'Eath, R.B., Turner, S.P. *et al.* 2009. In *Proc. European Assoc. Anim. Prod.*, Barcelona, page 282.
- Pla, L.M., Faulin, J. and Rodriguez, S.V. (2009). *J. Operational Res. Soc.*, 60:619-625.
- van Putten, G. and van de Burgwal, J.A. (1990). *Appl. Anim. Behav. Sci.*, 26:181-186.
- Quilter, C.R., Blott, S.C., Wilson, A.E. *et al.* (2007). *Am. J. Medical Genetics, Part B-Neuropsychiatric Genetics*, 144B:862-868.
- Quilter, C.R., Gilbert, C.L., Oliver, G.L. *et al.* (2008). *Am. J. Medical Genetics, Part B-Neuropsychiatric Genetics*, 147B:1126-1137.
- Rizvi, S., Nicol, C.J. and Green, L.E. (1998). *Vet. Rec.*, 143:654-658.
- Rodenburg, T.B., Bijma, P., Ellen, E.D. *et al.* (2010). *Anim. Welfare*, in press.
- Rundgren, M. and Löfquist, I. (1989). *Anim. Prod.*, 49:311-315.
- Sandøe, P., Nielsen, B.L., Christensen, L.G. *et al.* (1999). *Anim. Welf.*, 8:313-328.
- Schroder-Petersen, D.L. and Simonsen, H.B. (2001). *Vet. J.*, 162:196-210.
- van der Steen, H.A.M., Schaeffer, L.R., de Jong, H. *et al.* (1988). *J. Anim. Sci.*, 66:271-279.
- Stevens, L., Goodnight, C.J. and Kalisz, S. (1995). *Am. Naturalist*, 145:513-526.
- Stott, A.W. and Gunn, G.J. (2008). *Prevent. Vet. Med.*, 84:179-193.
- Tan, S.S.L., Shackleton, D.M. and Beames, R.M. (1991). *Anim. Prod.*, 52:201-206.
- Torrey, S., Pajor, E., Weaver, S. *et al.* (2001). *J. Anim. Sci.*, 79 (suppl. 1):14-15.
- Turner, S.P., White, I.M.S., Brotherstone, S. *et al.* (2006). *Animal Science*, 82:615-620.
- Turner, S.P., Roehe, R., D'Eath, R.B. *et al.* (2009). *J. Anim. Sci.*, 87:3076-3082.
- Turner, S.P., D'Eath, R.B., Roehe, R. *et al.* (2010). *Animal Welfare*, in press.
- Walker, P.K. and Bilkei, G. (2006). *Vet. J.*, 171:367-369.
- Wersinger, S.R., Caldwell, H.K., Christiansen, M. *et al.* (2007). *Genes Brain Behav.*, 6:653-660.
- Wolf, J.B., Brodie III, E.D., Cheverud, J.M. *et al.* (1998). *TREE*, 13:64-69.