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ORIGINAL ARTICLE

High heritability for *Ascaris* and *Trichuris* infection levels in pigs

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Aggregated distributions of macroparasites within their host populations are characteristic of most natural and experimental infections. We designed this study to measure the amount of variation that is attributable to host genetic factors in a pig–helminth system. In total, 195 piglets were produced after artificial insemination of 19 sows (Danish Landrace–Yorkshire crossbreds) with semen selected from 13 individual Duroc boars (1 or 2 sows per boar; mean litter size: 10.3; 5–14 piglets per litter). Starting at 10 weeks of age, piglets were repeatedly infected with the gastrointestinal helminths *Trichuris suis* and *Ascaris suum* by administering eggs in the feed for 14 weeks until necropsy. Faecal egg counts (FECs) were estimated regularly and *A. suum* worm burden was obtained at necropsy. Heritability calculations for log (FEC + 1) at weeks 7–10 post-infection (p.i.) showed that 0.32–0.73 of the phenotypic variation for *T. suis* could be attributed to genetic factors. For *A. suum*, heritabilities of 0.29–0.31 were estimated for log (FEC + 1) at weeks 7–14 p.i., whereas the heritability of log worm counts was 0.45. Strong positive genetic correlations (0.75–0.89) between *T. suis* and *A. suum* FECs suggest that resistance to both infections involves regulation by overlapping genes. Our data demonstrate that there is a strong genetic component in resistance to *A. suum* and *T. suis* infections in pigs. Identification of responsible genes would enhance our understanding of the host immune response to these common nematodes and for the closely related species (*T. trichiura* and *A. lumbricoides*) in man infecting more than a billion people.
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Introduction

Helminth infections are typically overdispersed (aggregated) within the host population, with a minority of the population harbouring the majority of the worm load (for example, Anderson and May, 1985). For example, in both natural and experimental (single and trickle) *Ascaris suum* infections of pigs, a typical aggregated distribution of parasites within the host population has been shown (reviewed by Roepstorff, 2003), with around 20% of the host population harbouring 80% of the parasites. Furthermore, 'wormy' pigs seem to be predisposed to high infection levels, as they become reinfected to a higher extent than their less wormy penmates after an anthelmintic treatment (Boes *et al*., 1998b). In single infections, the aggregated distribution is the result of expulsion of high numbers of larvae from the small intestine during the early phase of infection where after only few pigs are worm positive (Roepstorff *et al*., 1997). Results from a low number of trickle-infected pigs indicate the same (Eriksen *et al*., 1992). In these experimental infections, a number of potential 'aggregation factors' such as different exposure/behaviour, uneven level of acquired resistance prior to infection, sex, age, breed and infection doses were controlled, which suggest a regulating role of genetic factors on worm load. These factors may very likely be associated with host genetics. In humans, Williams-Blangero *et al*. (1999) found strong support for genetic factors accounting for 30–50% of the variation in *A. lumbricoides* infection levels in a Nepalese population. Three significant and three suggestive quantitative trait loci influencing susceptibility to *A. lumbricoides* infection have now been identified (Williams-Blangero *et al*., 2008a).

For *Trichuris suis*, we are not aware of any published studies focusing on the effect of host genetics on infection levels, but several studies have shown familial or household effects on *T. trichiura* infections in human populations (Forrester *et al*., 1990; Anderson *et al*., 1993; Chan *et al*., 1994a, b; Ellis *et al*., 2007). Recently, Williams-Blangero *et al*. (2002) have been able to disentangle the genetic effects on *T. trichiura* infection levels (as measured by faecal egg counts (FECs)) in two Asian populations. Approximately 28% of the variation within these two populations could be attributed to genetic factors, whereas household effects only accounted for 4%. This finding strongly suggests that susceptibility to

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T. trichiura has a host genetic component, which recently has been supported by localization of two significant quantitative trait loci in one of these two populations (Williams-Blangero *et al*., 2008b).

Genetic markers with influence on helminth resistance in ruminants have been identified (for example, Sayers *et al*., 2005) as have markers for *Escherichia coli* resistance in pigs (Meijerink *et al*., 1997; Jørgensen *et al*., 2004). In recent years, marker-assisted selection has received increased attention as this approach allows the selection of animals without producing the phenotype (Dekkers, 2004). In human populations, identification of the genetic factors underlying a specific phenotype such as resistance to pathogens is challenging because populations are heterogeneous in age, exposure to infection, nutritional and immune status and many other factors. In contrast, domestic animals, such as pigs constitute a more experimentally tractable resource for understanding the genetic basis of phenotypic variation (for example, Milan *et al*. 2000; Van Laere *et al*., 2003). In animal studies, it is possible to establish 'ideal' pedigrees (resource families) in which specific traits can be predicted to segregate and family sizes are typically higher than in humans, which improve the power to detect heritable traits. Most importantly, controlled infections of pigs can be conducted and environmental variation can be minimized—as such we are able to examine infection dynamics at defined intervals following infection.

The present study aims at assessing the contribution of host genetic factors in determining infection outcomes in pigs exposed to infection with *A. suum* and *T. suis.* To do this, we used controlled trickle infections, which mimic natural transmission, in a resource population with known pedigree.

Materials and methods

Inoculation material

A. suum eggs (DCEP batch 03–02) were isolated as described by Oksanen *et al*. (1990) from *A. suum* females collected at a local abattoir and stored in 0.05 M H_2SO_4 with less than 20 eggs μl^{-1}. The infectivity of the batch was tested by inoculating each of two pigs (10 weeks of age) by stomach tube with 1000 embryonated eggs. The pigs were killed at day 12 post-infection (p.i.) and *A. suum* larvae were isolated from the small intestine and enumerated. Recovery rates were 38 and 62%. The *T. suis* egg batch, originally isolated from an organic farm (Roepstorff and Murrell, 1997), had an infectivity rate of approximately 100%, as described by Kringel and Roepstorff (2006).

The resource population

From two commercial specific pathogen-free farms, 111 and 84 weaned piglets were obtained from 10 and 9 sows, respectively (Table 1), with an overall mean litter size at 10.3 (minimum–maximum: 5–14). The piglets were the outcome of artificial insemination of 19 Danish Landrace–Yorkshire crossbred sows with individual semen from 13 Duroc boars. The pigs were produced in two rounds 2 weeks apart (8 and 11 litters, respectively). All pigs were ear-tagged and males were castrated.

Table 1 Numbers of piglets produced on two farms by inseminating sows with semen from specific boars

Sow	*Boar*	*Farm*	*Delivery*	*No. of pigs*
1	1	1	2	8
2	1	2	2	10
3	2	1	1	13
4	2	2	2	8
5	3	1	1	13
6	3	2	2	11
7	4	1	1	13
8	4	2	2	10
9	5	1	2	11
10	5	2	2	8
11	6	1	1	8
12	6	2	2	5
13	7	1	1	14
14	8	1	1	9
15	9	1	1	12
16	10	1	2	10
17	11	2	2	10
18	12	2	2	9
19	13	2	2	13

Experimental design and laboratory analysis

The infection study was performed under outdoor conditions at the research farm of Copenhagen University during wintertime to avoid autoinfection (Larsen and Roepstorff, 1999). At 8 weeks of age, the pigs were randomly allocated, after stratification according to farm, sex and litter, to six helminth-free paddocks of equal size. The pigs from the two deliveries (2 weeks in between) were allocated into two and four paddocks, respectively. The pigs were fed a standard diet consisting of ground barley with protein/mineral supplement and had free access to water. After 2 weeks of acclimatization, the pigs received *A. suum* and *T. suis* trickle infection (25 and 5 eggs kg^{-1} day^{-1}, respectively) in the feed twice weekly until slaughter at week 14 post start of infection (p.i.). The dose was adjusted weekly on a pen level according to mean weight of the pigs. Faecal samples were taken from each pig at weeks 0, 6, 7, 8, 9, 10, 12 and 14 p.i. and FECs were determined by a modified McMaster technique with saturated NaCl with 500 g glucose per litre (specific gravity 1.27 g ml^{-1}), with a lower detection limit of 20 eggs per gram of faeces (Roepstorff and Murrell, 1998). False-positive egg counts due to coprophagia have been shown for *A. suum* and *T. suis* (Boes *et al*., 1997, 1998a) and an arbitrary cutoffs at FEC <41 and <21 for *A. suum* and *T. suis*, respectively, were applied to define non-infected pigs for the prevalences and frequency distributions of worm load, whereas heritability calculations were based on raw data. Pig body weights were obtained at weeks 0, 7 and 14 p.i. At slaughter, the pigs were exviscerated and the small intestine was cut open. The contents were sieved and *A. suum* visible by the naked eye were recovered and stored in 70% ethanol. All worms ($n=4758$) were subsequently sexed, length measured, weighed and enumerated. Actual worm counts for *T. suis* were not determined as very few pigs were coprologically positive at slaughter (week 14 p.i.).

Calculation of the phenotypic traits

All FECs for *A. suum* and *T. suis* for each week were used as phenotypic traits in the heritability calculations. For

FECs of *T. suis* at weeks 6, 12 and 14 p.i., we were not able to keep the residual kurtosis within normal range despite transformation, and these results were omitted. A number of other ways of describing the phenotypes were included: *T. suis* FEC weeks 7 + 8, *T. suis* FEC weeks 8 + 9 and *T. suis* FEC weeks 7–9 as the sum of the FECs for these given weeks; *T. suis* and *A. suum* total FECs (the areas under the curves) as the sums of the FECs from week 6 to 14 p.i., where FECs weeks 11 and 13 p.i. were calculated as the average of the two flanking weeks; *A. suum* and *T. suis* FEC peaks were defined as the maximum FECs observed for a given pig. In addition, as phenotypes for *A. suum* we also used worm count, *A. suum* biomass (total weight of the worms obtained from each pig), mean worm length and mean worm weight, being aware that storing in 70% ethanol may have affected weight and length. Lastly, mean FEC per female ($n = 129$) was calculated as FEC week 14 p.i. divided by the number of females obtained at slaughter.

Statistical analysis

We performed heritability analysis using a variance components approach and software package SOLAR (Almasy and Blangero, 1998) where all parameter estimates are maximum likelihood estimates, and all hypothesis testing is conducted using likelihood ratio testing. Variance components-based heritability analysis is based on decomposition of the phenotypic variance into the effects of genetic factors, shared environmental factors (for example, using a 'household' model), and individual-specific factors due to the unique environmental exposures and measurement error. The effects of measured covariates (for example, sex) can be included in the analysis as predictors of the phenotypic mean (for example, using a linear regression model). We first estimated the (narrow sense) heritability (h^2), which is the proportion of the total phenotypic variance that can be attributed to the additive effects of genetic factors. We also estimated to which degree two phenotypic traits are under the regulation of overlapping genetic factors, by performing bivariate heritability analysis. The additive genetic correlation (ρ_G) and the random environmental correlation (ρ_E) were estimated using SOLAR, and the phenotypic correlation (ρ_P) was subsequently calculated by:

$$\rho_P = \sqrt{h_1^2}\cdot\sqrt{h_2^2}\cdot\rho_G + \sqrt{(1-h_1^2)}\cdot\sqrt{(1-h_2^2)}\cdot\rho_E$$

where h_1^2 is the heritability of trait 1 and h_2^2 is the heritability of trait 2 (Czerwinski *et al*., 1999). The entire pedigree relationships were used in the analyses. It was assumed that the founding sows and boars were unrelated.

All significant covariates from the univariate analysis were included in the bivariate analysis. Owing to the aggregated distribution of *A. suum* and *T. suis* within the host population, FECs and worm counts were all transformed with ln(trait + 1) to correct for skewness. FEC per female worm was inverse normal transformed, whereas pig body weight week 0 p.i. and pig weight gain remained untransformed.

For each trait, the heritability was calculated with paddock (1–6) included as shared environmental effect ('household') and body weight at week 0 p.i., sex, farm (1 and 2) and delivery (first or second) as covariates. A permissive threshold was used including covariates in the final model with $P < 0.1$. Significant covariates were included in the bivariate analysis.

The parameter k of the negative binomial distribution was calculated for all *A. suum* and *T. suis* FEC for each week 7–14 p.i. and *A. suum* worm load at necropsy to determine how levels of aggregation change over time. Decreasing value of k indicates increase in aggregation. k was computed as follows: $k = \text{mean}^2/(\text{variance} - \text{mean})$.

Results

Descriptive information

All faecal samples week 0 p. i. were found to be negative and are therefore omitted from the following figures and tables. Approximately two-third of the pigs became positive for *A. suum* and continued to excrete eggs (Figure 1). A peak in prevalence around week 8 p.i. was observed for *T. suis* after which expulsion took place leaving 5% FEC-positive pigs by week 14 p.i.

Figure 2 shows the aggregation of *A. suum* within the host population with 27 and 22% of the pigs harbouring 80% of the total worm population as measured by worm count at necropsy and FEC by week 8 p.i., respectively ($k = 0.25$ and 0.32). The geometric mean for worm counts was 8 (all pigs). In infected pigs, the mean intensity of *A. suum* was 29 (s.d. = 46; minimum–maximum: 1–422). For *A. suum* FEC weeks 7–14 p.i, the minimum–maximum estimate for k was 0.18–0.4 as depicted in Figure 1. As for *A. suum*, a skewed distribution was also observed for *T. suis* (Figure 2), with 22% of the pig population carrying 80% of the total infection ($k = 0.33$), as measured by FECs at week 8 p.i. The minimum–maximum of k for *T. suis* FEC weeks 7–14 p.i. was 0.03–0.33 (Figure 1).

Heritability estimates

The heritabilities for *A. suum* FECs were consistently around 0.3 (all h^2, $P < 0.002$) from week 7 p.i. and onwards (Figure 3). In contrast, the heritability estimates for *T. suis* FECs (all h^2, $P < 0.001$) peaked by week 8 p.i. with 73% of the phenotypic variation explained by genetic components, where after it declined following the same pattern as the prevalence. *T. suis* FEC weeks 7 and 8 p.i., *T. suis* FEC weeks 7–9 p.i., total FEC and peak of egg excretion had heritabilities around 0.62, whereas for *T. suis* FEC weeks 8 and 9 p.i. 72% of the variation could be explained by genetic components (Table 2).

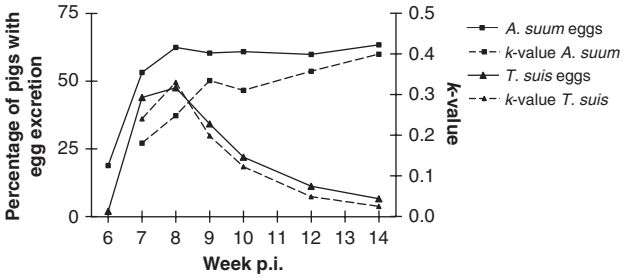

Figure 1 Percentage of *A. suum* and *T. suis* trickle-infected pigs ($n = 195$) with egg excretion (>40 and >20 faecal egg count (FEC) used as cutoffs, respectively) and the respective k-values as a function of week p.i.

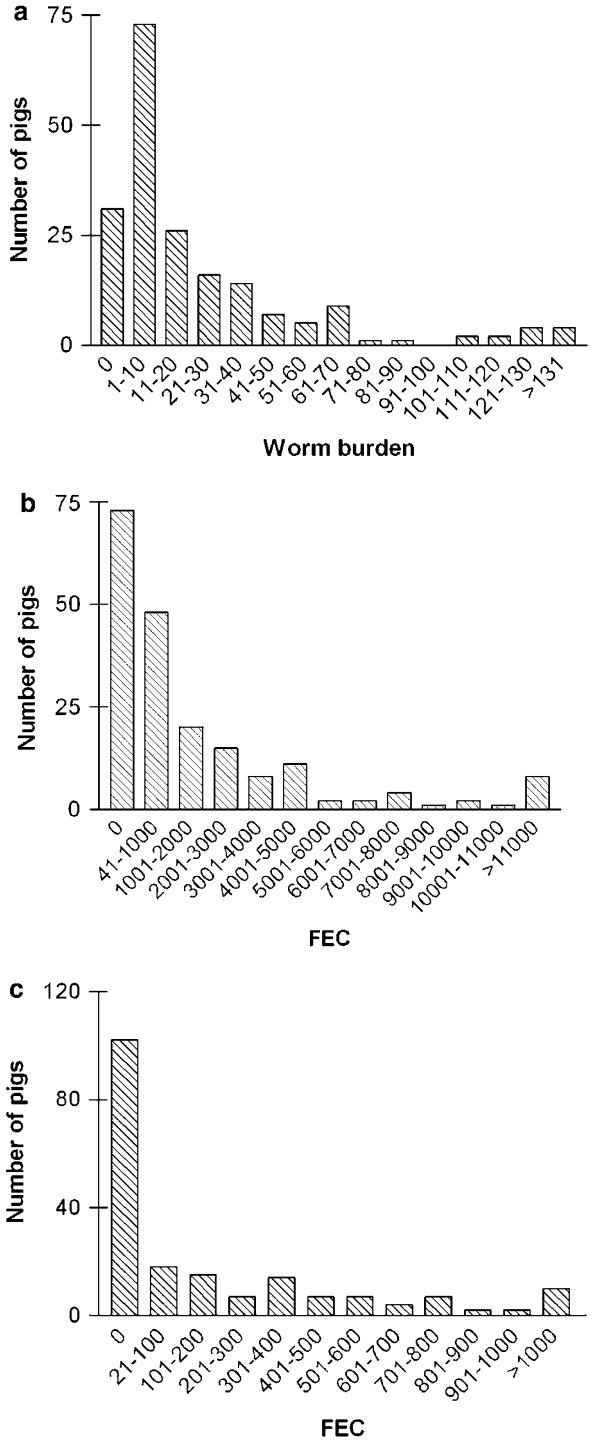

Figure 2 Frequency distribution of *A. suum* and *T. suis* in trickle-infected pigs ($n = 195$). (**a**) Worm counts for *A. suum* at week 14 p.i. (**b**) Faecal egg count (FEC) for *A. suum* week 8 p.i. (FEC < 41 was considered 'false positive'). (**c**) FEC for *T. suis* at week 8 p.i. (FEC = 20 was considered 'false positive').

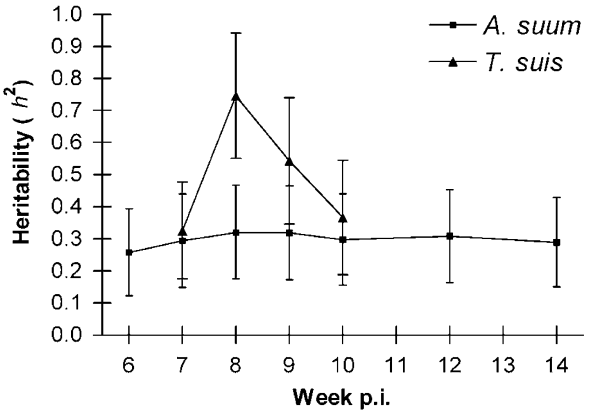

Figure 3 Heritability estimates by week p.i. for *A. suum* and *T. suis* faecal egg counts (FECs) in pigs during trickle infections (for all h^2, $P < 0.002$). Heritability for *Trichuris* at weeks 6, 12 and 14 p.i. had very skew distribution due to few infected pigs and were omitted. Bars: s.e.

For *A. suum*, there was no significant effect of paddock (that is, common effect, c^2) or any of the covariates, accounting for less than 2% of the variation. Paddock and sex were also found to be nonsignificant for *T. suis*, whereas delivery and farm had significant ($P < 0.05$) effects on the variance of FECs, with highest figures from pigs originating from delivery 2 and farm 1, respectively. The significant covariates accounted for 18–26% of the total variation.

The worm count gave the highest heritability estimate (0.44) obtained for *A. suum*, whereas total FEC, peak of egg excretion and *A. suum* worm biomass had heritabilities around 0.35 (Table 3). The heritability of mean FEC per female worm was found to be low and nonsignificant. All covariates and the effect of paddock were nonsignificant. Heritabilities of mean length and mean weight of worms, both in total and by sex of worm, were found to be low and nonsignificant (data not shown).

The heritabilities for weight were 0.26 (0.13), 0.22 (0.12) and 0.25 (0.12) at week 0, 7 and 14 p.i., respectively (standard error in brackets) and significant at $P < 0.01$. Delivery and sex had significant ($P < 0.05$) effects on weight at week 7 and 14 p.i., respectively.

Bivariate analyses

High genetic (1.00) and phenotypic correlations (0.81–0.99) were found between *T. suis* traits, and similar results were obtained for *A. suum* with genetic correlations of 0.97–1.00 and phenotypic correlations of 0.73–1.00 (Table 4). Genetic correlations between *A. suum* and *T. suis* traits were all found to be high (0.75–0.89) and significant, with the highest value obtained for *A. suum* FEC and *T. suis* FEC week 8 p.i. ($P = 0.007$). In contrast, the phenotypic correlations between *A. suum* and *T. suis* traits were relatively low with estimates from 0.15 to 0.32. The genetic correlations between the parasite-related traits and weight gain of the pigs were all negative (−0.01 to −0.38) but not significantly different from zero.

Bivariate analysis of worm counts and other traits for *A. suum* week 14 p.i. showed high genetic and phenotypic correlations (Table 5).

Discussion

In this experimental infection study, we have found a classical aggregated distribution of *A. suum* ($k = 0.18$–0.4) with approximately 20–30% of the host population

Table 2 Heritability estimates for *T. suis*

Trait	h^2	*s.e*	P-*value*	P-*value for delivery*[a]	P-*value for farm*[b]	*Percentage of variance due to all final covariates*
FEC weeks 7 and 8 p.i.	0.61	0.19	<0.0001	0.02	0.001	21.2
FEC weeks 8 and 9 p.i.	0.76	0.20	<0.0001	0.018	0.003	22.5
FEC weeks 7–9 p.i.	0.64	0.19	<0.0001	0.013	0.0008	24.3
Total FEC output	0.64	0.19	<0.0001	0.007	0.0006	25.8
Peak value of FEC	0.64	0.19	<0.0001	0.009	0.0008	24.3

Abbreviations: FEC, faecal egg count; s.e., standard error.
[a]Deliveries 1 and 2.
[b]Farms 1 and 2.

Table 3 Heritability for *A. suum*

Trait	h^2	*s.e.*	P-*value*
Total FEC output	0.36	0.15	<0.0001
Peak value of FEC	0.37	0.16	<0.0001
Worm count	0.45	0.17	<0.0001
Biomass of worms	0.35	0.15	<0.0001
Mean FEC per female ($n = 129$)	0.06	0.12	0.21

Abbreviations: FEC, faecal egg count; s.e., standard error.

carrying 80% of the worm population as seen in other studies (for example, Boes *et al*., 1998b). For *T. suis* FECs in trickle-infected pigs, we have for the first time shown a similar skewed distribution of the infection ($k = 0.03$–0.33). Eggs can be detected in faeces from around week 6 p.i. for both parasites. After week 6 p.i., the prevalences for both parasites increase. Despite this initial similarity, the population dynamics of the two parasites differ dramatically (Figure 1). The majority of *A. suum* larvae get expelled from the small intestine during the early phase of infection (~week 3 p.i.) where after the small remaining *A. suum* population seems unaffected by host immunity and these worms grow and give rise to the stable *A. suum* FEC around week 8 p.i. and onwards when they are fully mature (Roepstorff *et al*., 1997). For *T. suis*, the prevalence drops dramatically at week 8 p.i. (Figure 1), indicating population dynamics similar to single infections with very few FEC positive hosts at week 11 p.i. (Kringel and Roepstorff, 2006). The observed difference in population dynamics between *A. suum* and *T. suis* based on FEC is therefore related to the time point for the onset of main immune response and the nature of the response mounted by the host.

The heritability estimates for *A. suum* FECs week 7 p.i. onwards were around 0.30, which means that genetic factors account for 30% of the variation in FECs. A heritability of 0.35 was obtained when using the phenotype 'total *A. suum* eggs excretion' (area under the curve), which one may argue is a better measure of the phenotype as it covers the whole infection period. However, the highest heritability was obtained using the actual worm counts (0.44). Our heritability estimates are of similar magnitude as the ones estimated for *A. lumbricoides* in a human population in Nepal, where Williams-Blangero *et al*. (1999) found that genetics accounted for 30–50% of the variation in FEC, worm count and worm biomass (estimated after treatment). These findings are mutually encouraging, due to the close genetic relationship between *A. suum* and *A. lumbricoides* (Anderson, 2001) and the physiological similarities of the two hosts. In contrast, Ellis *et al*. (2007) did not find a genetic component to *A. lumbricoides* infections in China but only a household effect accounting for 32% of the variation. But as mentioned by these authors, this may be due to their use of binary phenotypes (infected versus non-infected) and associated lack of quantitative power, which are important when studying helminth infections (Quinnell, 2003).

Heritability estimates obtained for fecundity measures (for example, number of eggs in uterus) and length of worms have shown to be higher than for actual worm counts in lambs infected with *Teladorsagia circumcincta* (Stear *et al*., 1997) and appear to be associated with IgA levels (Stear *et al*., 1999). In contrast, for *A. suum*, we have found low and nonsignificant heritabilities both for FEC per female and worm size. This indicates that *A. suum* fecundity may be under the regulation of mechanisms other than worm counts, for example, responding in a different way to host immunity.

In human populations, heritability for *T. trichiura* infection has been found to be approximately 0.30 (Williams-Blangero *et al*., 2002; Ellis *et al*., 2007), with no or low effect of household (4%). In contrast, we have found heritability of 0.6–0.7 for *T. suis* FECs. This difference could reflect the degree of controlled variables in the two studies but could also be due to differences in host reaction. The prevalence for *T. trichiura* infection as measured by egg excretion is generally peaking around the second decade of life for humans and is chronic by nature (Stephenson *et al*., 2000), whereas the peak is reached early in life for pigs (Roepstorff *et al*., 1992) or early after exposure followed by expulsion (Figure 1).

In contrast to *A. suum* where there were no significant effects observed for any of the included covariates, pigs from farm 1 and delivery 2 had significant higher *T. suis* FEC. We do not have any plausible explanation for these discrepancies between farms and deliveries except perhaps that effect of farm must be due to different underlying genetics of the sows used at the two farms.

A caveat in our heritability estimates is, however, that the genetic effect obtained here is confounded with any litter environment effect as the piglets were kept together with the sow for the first 5 weeks of life. A potential litter effect could be due to different levels of nutrition received by piglets in different litters, or more specifically related to transfer of maternal immunity by colostrum or an infection during the suckling period. We consider litter effects due to maternal immunity to be unlikely because all sows were tested negative for *T. suis* and *A. suum* except one for *A. suum* (FEC, 140). In

Table 4 Genetic (r_g, above diagonal) and phenotypic correlations (r_p, below diagonal) for some of the phenotypic traits

	T. suis *FEC week 8 p.i*	T. suis *peak value of FEC*	T. suis *total FEC output*	A. suum *FEC week 8 p.i*	A. suum *peak value of FEC*	A. suum *total FEC output*	A. suum *worm count*	*Weight gain*
T. suis FEC week 8 p.i	—	1.00**	1.00**	0.89** (0.21)	0.83* (0.21)	0.83* (0.22)	0.79* (0.22)	−0.01 (0.43)
T. suis peak value of FEC	0.81	—	1.00	0.87* (0.27)	0.79* (0.26)	0.79* (0.26)	0.76* (0.27)	−0.10 (0.45)
T. suis total FEC output	0.82	0.99	—	0.85* (0.28)	0.78* (0.26)	0.78* (0.27)	0.75* (0.27)	−0.08 (0.08)
A. suum FEC week 8 p.i	0.25	0.15	0.15	—	0.99* (0.05)	0.99* (0.04)	0.97* (0.07)	−0.19 (0.50)
A. suum peak value of FEC	0.23	0.16	0.16	0.89	—	1.00	0.99* (0.06)	−0.38 (0.45)
A. suum total FEC output	0.24	0.15	0.15	0.89	1.00	—	0.98* (0.06)	−0.38 (0.46)
A. suum worm count	0.32	0.22	0.22	0.75	0.81	0.81	—	−0.02 (0.47)
Weight gain	−0.19	−0.10	−0.11	−0.03	−0.07	−0.08	−0.07	—

Abbreviation: FEC, faecal egg count.
Total egg output, area under the curve.
Standard error in brackets.
*$P<0.05$, **$P<0.01$.

Table 5 Genetic (r_g) and phenotypic (r_p) correlations for *A. suum* worms at slaughter

	r_g	*s.e.*	P_g	r_p
Worm burden, *A. suum* FEC week 14 p.i.	1	—	0.07	0.86
Worm burden, *A. suum* biomass	1	—	0.03	0.96
A. suum FEC week 14 p.i., *A. suum* biomass	1	—	0.19	0.90

Abbreviations: FEC, faecal egg count; s.e., standard error.

addition, all pigs were initially (week 0 p.i.) FEC negative.

Genetic correlation close to one was found between *Haemonchus contortus* and *Trichostrongylus colubriformis* infection levels in sheep (Gruner *et al*., 2004). We have also found evidence for genetic correlation between *A. suum* and *T. suis* infections (0.75–0.89), suggesting that some of the same genes may play a role in the regulation of the worm loads. This observation is interesting when considering the different population dynamics of the two parasites (Figure 1). The majority of the pigs are able to completely expel all of the *T. suis* (Kringel and Roepstorff, 2006) and hinder the establishment of new incoming worms (unpublished data), whereas a stable parasite population situation seems to be established for *A. suum*. Correlation between *A. lumbricoides* and *T. trichiura* in the human population is normally explained by similar transmission routes and egg biology, but may also be due to the regulation of some of the same genes as our finding in the pig suggest. However, Williams-Blangero *et al*. (2008a, b) did not identify overlapping quantitative trait loci for *A. lumbricoides* and *T. trichiura* infections in humans, suggesting that different genes are involved here, even though the population dynamics and prevalences of the two parasites show similar patterns in the human population (for example, Anderson *et al*., 1993).

As the high heritability for *T. suis* is linked to the short period where the prevalence is peaking (week 8 p.i.), this makes the identification of high- and low-responder pigs more problematic than for *A. suum* (for example, if high responder pigs should be identified for breeding). For *T. suis*, exposure period needs to be known to predict when FEC is peaking. It would therefore be beneficial, if a more 'stable' phenotype (for example, an ELISA) could be used to identify high- and low-responder *T. suis* pigs. For example, Davies *et al*. (2005) found high genetic correlations between a range of indicator traits and nematode infections in lambs, and this could aid in the selection for increased resistance to gastrointestinal nematodes. Douch *et al*. (1995) have shown that in sheep infected with *T. colubriformis*, selection based on serum antibodies would result in 51–67% of the genetic gain in FEC compared with that obtained by FEC selection directly. However, as mentioned by these authors, FEC in itself is an indirect measure of worm number and antibody titre may be as good or a better measure of a host's ability to resist parasite infection.

In naturally infected sheep, a negative genetic correlation has been described between FEC and weight gain, suggesting that resistant animals have a higher growth rate (Bishop *et al*., 1996; Bouix *et al*., 1998; Gauly and Erhardt, 2001). We have also found a negative correlation between *A. suum* and *T. suis* FECs and weight gain. However, this correlation was not different from zero, suggesting that selection for more *A. suum*- and *T. suis*-resistant pig will not influence the growth performance in this breed of pigs.

Although aggregation and predisposition of *A. suum* within a pig population previously have been shown in single and trickle infections (Roepstorff *et al*., 1997; Boes *et al*., 1998b), the present study for the first time demonstrates the role of host genetic. Our findings suggest that future whole genome scan would be useful to identify regions and specific genes, which are involved in the regulation of both *A. suum* and *T. suis* infection levels in pigs. Genetic markers may be useful in breeding programs (for example, marker-assisted selection) and thereby production of more parasite-resistant pigs or in choosing 'the right animal for the right purpose'. In addition, if specific genes can be identified these would undoubtedly enhance our understanding of the host immunological response to infection with these parasites in the pig and with related species in humans.

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