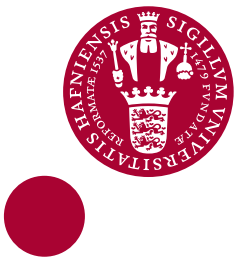

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Running title

Heritability for *Ascaris* and *Trichuris* infections

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Abstract

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. 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 gastro-intestinal helminths *Trichuris suis* and *Ascaris suum* by administering eggs in the feed for 14 weeks until necropsy. Faecal egg counts (FEC) were estimated regularly and *A. suum* worm burden obtained at necropsy. Heritability calculations for log (FEC+1) in 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) by 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.

Key words: *Ascaris suum*, *Trichuris suis*, heritability, host genotypes, resistance, breeding

Introduction

Helminth infections are typically overdispersed (aggregated) within the host population with a minority of the population harbouring the majority of the worm load (e.g. 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' like 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 (QTL) influencing susceptibility to *A. lumbricoides* infection have now been identified (Williams-Blangero et al., 2008a).

For *T. 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;

Chan et al., 1994b; 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) 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 *T. trichiura* has a host genetic component which recently has been supported by localisation of two significant QTLs in one of these two populations (Williams-Blangero et al., 2008b).

Genetic markers with influence on helminth resistance in ruminants have been identified (e.g. Sayers et al., 2005) as have markers for *E. coli* resistance in pigs (Meijerink et al., 1997; Jørgensen et al., 2004). In recent years marker assisted selection has received increased attention since 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, like pigs constitute a more experimentally tractable resource for understanding the genetic basis of phenotypic variation (e.g 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 power to detect heritable traits. Most importantly, controlled infections of pigs can be conducted and environmental variation can be minimised – as such we are able to examine infection dynamics at defined intervals following infection.

91 The present study aims at assessing the contribution of host genetic factors in determining infection
92 outcomes in pigs exposed to infection with *A. suum* and *T. suis*. To do this we used controlled
93 trickle infections, which mimic natural transmission, in a resource population with known pedigree.
94

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 0.05M H_2SO_4 with less than 20 eggs/µl. 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 approx. 100%, as described by Kringel and Roepstorff (2006).

The resource population

From two commercial specific pathogen-free (SPF) 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 (min-max: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.

Experimental design and laboratory analysis

The infection study was performed under outdoor conditions at the research farm of Copenhagen University during winter time in order 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 6 helminth free paddocks of equal size. The pigs from the two deliveries (two weeks in between)

119 were allocated into 2 and 4 paddocks, respectively. The pigs were fed a standard diet consisting of
120 ground barley with protein/mineral supplement and had free access to water. After 2 weeks of
121 acclimatization, the pigs received *A. suum* and *T. suis* trickle infection (25 and 5 eggs/kg/day,
122 respectively) in the feed twice weekly until slaughter at week 14 post start of infection (p.i.). The
123 dose was adjusted weekly on a pen level according to mean weight of the pigs. Faecal samples were
124 taken from each pig at weeks 0, 6, 7, 8, 9, 10, 12, and 14 p.i. and faecal egg counts (FEC) were
125 determined by a modified McMaster technique with saturated NaCl with 500 g glucose per litre
126 (specific gravity 1.27 g/ml) with a lower detection limit of 20 eggs per gram of faeces (epg)
127 (Roepstorff and Murrell, 1998). False-positive egg counts due to coprophagia have been shown for
128 *A. suum* and *T. suis* (Boes et al., 1997; Boes et al., 1998a) and an arbitrary cut-offs at FEC <41 and
129 <21 for *A. suum* and *T. suis*, respectively, was applied to define non-infected pigs for the
130 prevalences and frequency distributions of worm load whereas heritability calculations were based
131 on raw data. Pig body weights were obtained at weeks 0, 7, and 14 p.i. At slaughter, the pigs were
132 exviscerated and the small intestine cut open. The contents were sieved and *A. suum* visible by the
133 naked eye were recovered and stored in 70% ethanol. All worms (n=4758) were subsequently
134 sexed, length measured, weighed and enumerated. Actual worm counts for *T. suis* were not
135 determined since very few pigs were coprologically positive at slaughter (week 14 p.i.).
136
137 Calculation of the phenotypic traits
138 All FECs for *A. suum* and *T. suis* for each week were used as phenotypic traits in the heritability
139 calculations. For FECs of *T. suis* at weeks 6, 12, and 14 p.i. we were not able to keep the residual
140 kurtosis within normal range despite transformation, and these results were omitted. A number of
141 other ways of describing the phenotypes were included: *T. suis* FEC weeks 7+8, *T. suis* FEC weeks
142 8+9 and *T. suis* FEC weeks 7-9 as the sum of the FECs for these given weeks; *T. suis* and *A. suum*

143 total FECs (the areas under the curves) as the sums of the FECs from week 6 to week 14 p.i. where
144 FECs weeks 11 and 13 p.i. were calculated as the average of the two flanking weeks; *A. suum* and
145 *T. suis* FEC peaks were defined as the maximum FECs observed for a given pig. In addition, as
146 phenotypes for *A. suum* we also used worm count, *A. suum* biomass (total weight of the worms
147 obtained from each pig), mean worm length and mean worm weight, being aware that storing in
148 70% ethanol may have affected weight and length. Lastly, mean FEC per female (n=129) was
149 calculated as FEC week 14 p.i. divided with number of females obtained at slaughter.
150
151 Statistical analysis
152 We performed heritability analysis using a variance components approach and software package
153 SOLAR (Almasy and Blangero, 1998) where all parameter estimates are maximum likelihood
154 estimates, and all hypothesis testing is conducted using likelihood ratio testing. Variance
155 components-based heritability analysis is based on decomposition of the phenotypic variance into
156 the effects of genetic factors, shared environmental factors (e.g., using a "household" model), and
157 individual-specific factors due to the unique environmental exposures and measurement error. The
158 effects of measured covariates (e.g., sex) can be included in the analysis as predictors of the
159 phenotypic mean (e.g. using a linear regression model). We first estimated the (narrow sense)
160 heritability (h^2), which is the proportion of the total phenotypic variance that can be attributed to the
161 additive effects of genetic factors. We also estimated to which degree two phenotypic traits are
162 under regulation of overlapping genetic factors, by performing bivariate heritability analysis. The
163 additive genetic correlation (ρ_G) and the random environmental correlation (ρ_E) were estimated
164 using SOLAR and the phenotypic correlation (ρ_P) was subsequently calculated by:
165

166 $$\rho_P = \sqrt{h_1^2} \cdot \sqrt{h_2^2} \cdot \rho_G + \sqrt{\left(1 - h_1^2\right)} \cdot \sqrt{\left(1 - h_2^2\right)} \cdot \rho_E$$

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. Due 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* indicate increase in aggregation. *k* was computed as follows: $k = \text{mean}^2/(\text{variance-mean})$.

Results

Descriptive information

191 All faecal samples week 0 p.i. were found to be negative and are therefore omitted from the
192 following figures and tables. Approximately two third of the pigs became positive for *A. suum* and
193 continued to excrete eggs (Figure 1). A peak in prevalence around week 8 p.i. was observed for *T.*
194 *suis* after which expulsion took place leaving 5% FEC positive pigs by week 14 p.i.
195
196 Figure 2 shows the aggregation of *A. suum* within the host population with 27% and 22% of the
197 pigs harbouring 80% of the total worm population as measured by worm count at necropsy and
198 FEC by week 8 p.i., respectively (k = 0.25 and 0.32). The geometric mean for worm counts was 8
199 (all pigs). In infected pigs, the mean intensity of *A. suum* was 29 (SD = 46; min-max: 1-422). For *A.*
200 *suum* FEC weeks 7-14 p.i the min-max estimate for k were 0.18-0.4 as depicted in Figure 1. As for
201 *A. suum,* a skewed distribution was also observed for *T. suis* (Figure 2) with 22% of the pig
202 population carrying 80% of the total infection (k = 0.33), as measured by FECs at week 8 p.i. The
203 min-max of k for *T. suis* FEC weeks 7-14 p.i. were 0.03-0.33 (Figure 1).
204
205 Heritability estimates
206 The heritabilities for *A. suum* FECs were consistently around 0.3 (all h^2, $P < 0.002$) from week 7
207 p.i. and onwards (Figure 3). In contrast, the heritability estimates for *T. suis* FECs (all h^2, $P < 0.001$)
208 peaked by week 8 p.i. with 73% of the phenotypic variation explained by genetic components,
209 where after it declined following the same pattern as the prevalence. *T. suis* FEC weeks 7 and 8 p.i.,
210 *T. suis* FEC weeks 7-9 p.i., total FEC, and peak of egg excretion had heritabilities around 0.62,
211 whereas for *T. suis* FEC weeks 8 and 9 p.i. 72% of the variation could be explained by genetic
212 components (Table 3).
213

For *A. suum,* there was no significant effect of paddock (i.e. 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 non-significant 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 2). The heritability of mean FEC per female worm was found to be low and non-significant. All covariates and the effect of paddock were non-significant. Heritabilities of mean length and mean weight of worms, both in total and by sex of worm, were found to be low and non-significant (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

238 the parasite-related traits and weight gain of the pigs were all negative (-0.01 to -0.38) but not
239 significantly different from zero.
240
241 Bivariate analysis of worm counts and other traits for *A. suum* week 14 p.i. showed high genetic and
242 phenotypic correlations (Table 5).
243
244 **Discussion**
245 In this experimental infection study, we have found a classical aggregated distribution of *A. suum* (*k*
246 = 0.18-0.4) with approximately 20-30% of the host population carrying 80% of the worm
247 population as seen in other studies (e.g. Boes et al., 1998b). For *T. suis* faecal egg counts in trickle
248 infected pigs, we have for the first time shown a similar skewed distribution of the infection (*k* =
249 0.03-0.33). Eggs can be detected in faeces from around week 6 p.i. for both parasites. After week 6
250 p.i. the prevalences for both parasites increase. Despite this initial similarity the population
251 dynamics of the two parasites differ dramatically (Figure 1). The majority of *A. suum* larvae get
252 expelled from the small intestine during the early phase of infection (~ week 3 p.i.) where after the
253 small remaining *A. suum* population seems unaffected by host immunity and these worms grow and
254 gives rise to the stable *A. suum* FEC around week 8 p.i. and onwards when they are fully mature
255 (Roepstorff et al., 1997). For *T. suis* the prevalence drops dramatically at week 8 p.i. (Figure 1)
256 indicating population dynamics similar to single infections with very few FEC positive hosts at
257 week 11 p.i. (Kringel and Roepstorff, 2006). The observed difference in population dynamic
258 between *A. suum* and *T. suis* based on FEC is therefore related to the time point for the onset of
259 main immune response and the nature of the response mounted by the host.
260

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 since 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 (e.g. 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 non-significant heritabilities both for FEC per female and worm size. This indicates that *A. suum* fecundity may be under regulation of other mechanisms than worm counts, e.g. 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

285 contrast, we have found heritability of 0.6-0.7 for *T. suis* FECs. This difference could reflect the
286 degree of controlled variables in the two studies but could also be due to differences in host
287 reaction. The prevalence for *T. trichiura* infection as measured by egg excretion is generally
288 peaking around the second decade of life for humans and is chronic by nature (Stephenson et al.,
289 2000), whereas the peak is reached early in life for pigs (Roepstorff et al., 1992) or early after
290 exposure followed by expulsion (Figure 1).
291
292 In contrast to *A. suum* where there were no significant effects observed for any of the included
293 covariates, pigs from farm 1 and delivery 2 had significant higher *T. suis* FEC. We do not have any
294 plausible explanation for these discrepancies between farms and deliveries except perhaps that
295 effect of farm must be due to different underlying genetics of the sows used at the two farms.
296
297 A caveat in our heritability estimates is however that the genetic effect obtained here is confounded
298 with any litter environment effect since the piglets were kept together with the sow for the first five
299 weeks of life. A potential litter effect could be due to different levels of nutrition received by piglets
300 in different litters, or more specifically related to transfer of maternal immunity by colostrum or an
301 infection during the suckling period. We consider litter effects due to maternal immunity to be
302 unlikely because all sows were tested negative for *T. suis* and *A. suum* except one for *A. suum* (FEC,
303 140). In addition, all pigs were initially (week 0 p.i.) FEC negative.
304
305 Genetic correlation close to one was found between *Haemonchus contortus* and *Trichostrongylus*
306 *colubriformis* infection levels in sheep (Gruner et al., 2004). We have also found evidence for
307 genetic correlation between *A. suum* and *T. suis* infections (0.75-0.89), suggesting that some of the
308 same genes may play a role in the regulation of the worm loads. This observation is interesting

309 when considering the different population dynamics of the two parasites (Figure 1). The majority of
310 the pigs are able to completely expel all of the *T. suis* (Kringel and Roepstorff, 2006) and hinder the
311 establishment of new incoming worms (unpublished data) whereas a stable parasite population
312 situation seems to be established for *A. suum*. Correlation between *A. lumbricoides* and *T. trichiura*
313 in the human population are normally explained by similar transmissions routes and egg biology but
314 may also be due to regulation of some of the same genes as our finding in the pig suggest. However,
315 Williams-Blangero (2008a; 2008b) did not identify overlapping QTLs for *A. lumbricoides* and *T.*
316 *trichiura* infections in humans, suggesting that different genes are involved here, even though the
317 population dynamics and prevalences of the two parasites show similar patterns in the human
318 population (e.g. Anderson et al., 1993).
319
320 Since the high heritability for *T. suis* is linked to the short period where the prevalence is peaking
321 (week 8 p.i.) this makes the identification of high and low responder pigs more problematic than for
322 *A. suum* (e.g. if high responder pigs should be identified for breeding). For *T. suis* exposure period
323 needs to be known in order to predict when FEC is peaking. It would therefore be beneficial, if a
324 more 'stable' phenotype (e.g. an ELISA) could be used to identify high and low responder *T. suis*
325 pigs. For example, Davis et al. (2005) found high genetic correlations between a range of indicator
326 traits and nematode infections in lambs, and this could aid in the selection for increased resistance
327 to gastrointestinal nematodes. Douch et al. (1995) have shown that in sheep infected with
328 *Trichostrongylus colubriformis*, selection based on serum antibodies would result in 51-67% of the
329 genetic gain in FEC compared to that obtained by FEC selection directly. However, as mentioned
330 by these authors, FEC in itself is an indirect measure of worm number and antibody titre may be as
331 good or a better measure of a host ability to resist parasite infection.
332

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.

While 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 demonstrate the role of host genetic. Our findings suggest that future whole genome scan would be useful in order 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 (e.g. 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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358

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472

473 **Titles and legends to figures**

474

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

477

478

479 Figure 2. Frequency distribution of *A. suum* and *T. suis* in trickle infected pigs (n=195). (A) Worm
480 counts for *A. suum* at week 14 p.i. (B) FEC for *A. suum* week 8 p.i. (FEC < 41 was considered 'false
481 positive') (C) FEC for *T. suis* at week 8 p.i. (FEC = 20 was considered 'false positive')

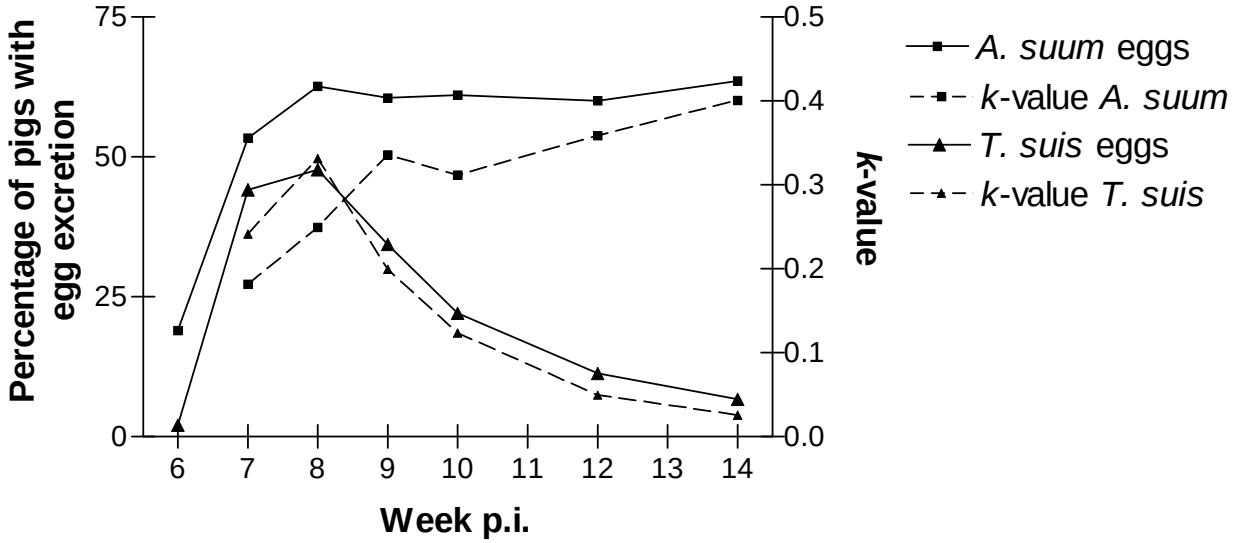
482

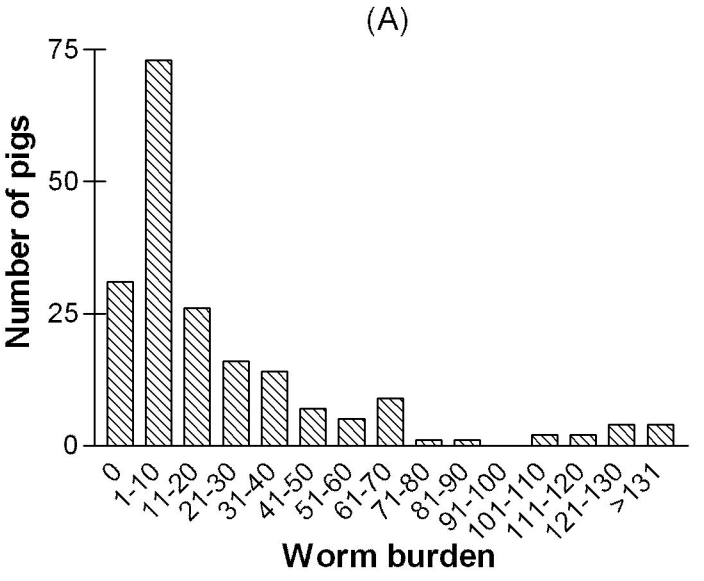
483

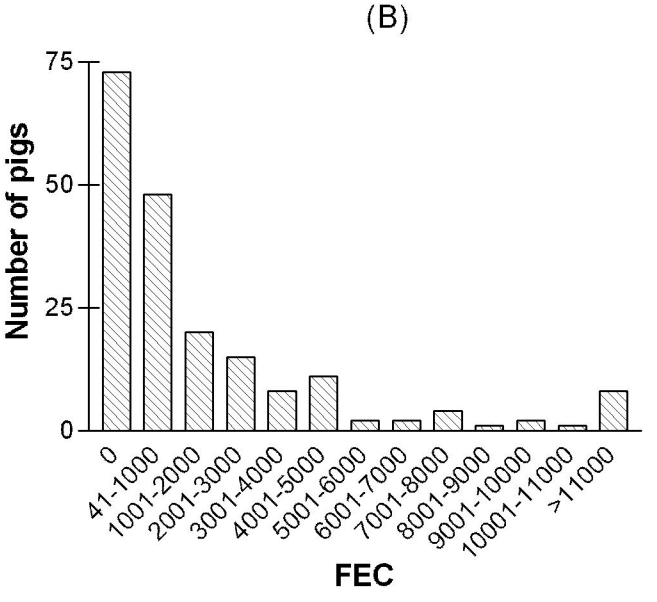
484 Figure 3. Heritability estimates by week p.i. for *A. suum* and *T. suis* FECs in pigs during trickle
485 infections (for all h^2, $P < 0.002$). Heritability for *Trichuris* at weeks 6, 12, and 14 p.i. had very skew
486 distribution due to few infected pigs and were omitted. Bars: s.e.

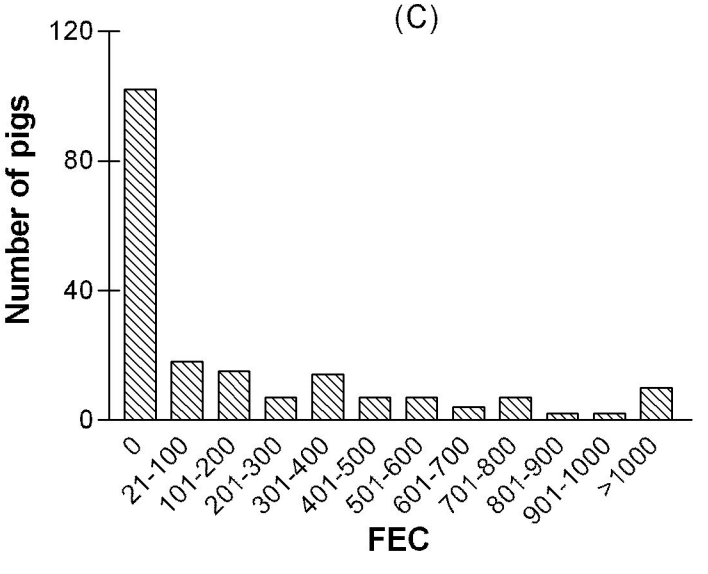
487

488


Percentage of pigs with egg excretion
75
50
25
0
0.5
0.4
0.3
0.2
0.1
0.0
k-value
6
7
8
9
10
11
12
13
14
Week p.i.
A. suum eggs
k-value A. suum
T. suis eggs
k-value T. suis


(A)
75
50
25
0
Number of pigs
0
1-10
11-20
21-30
31-40
41-50
51-60
61-70
71-80
81-90
91-100
101-110
111-120
121-130
>131
Worm burden


(B)
75
50
25
0
Number of pigs
0
41-1000
1001-2000
2001-3000
3001-4000
4001-5000
5001-6000
6001-7000
7001-8000
8001-9000
9001-10000
10001-11000
>11000
FEC


(C)
120
80
40
0
Number of pigs
0
21-100
101-200
201-300
301-400
401-500
501-600
601-700
701-800
801-900
901-1000
>1000
FEC

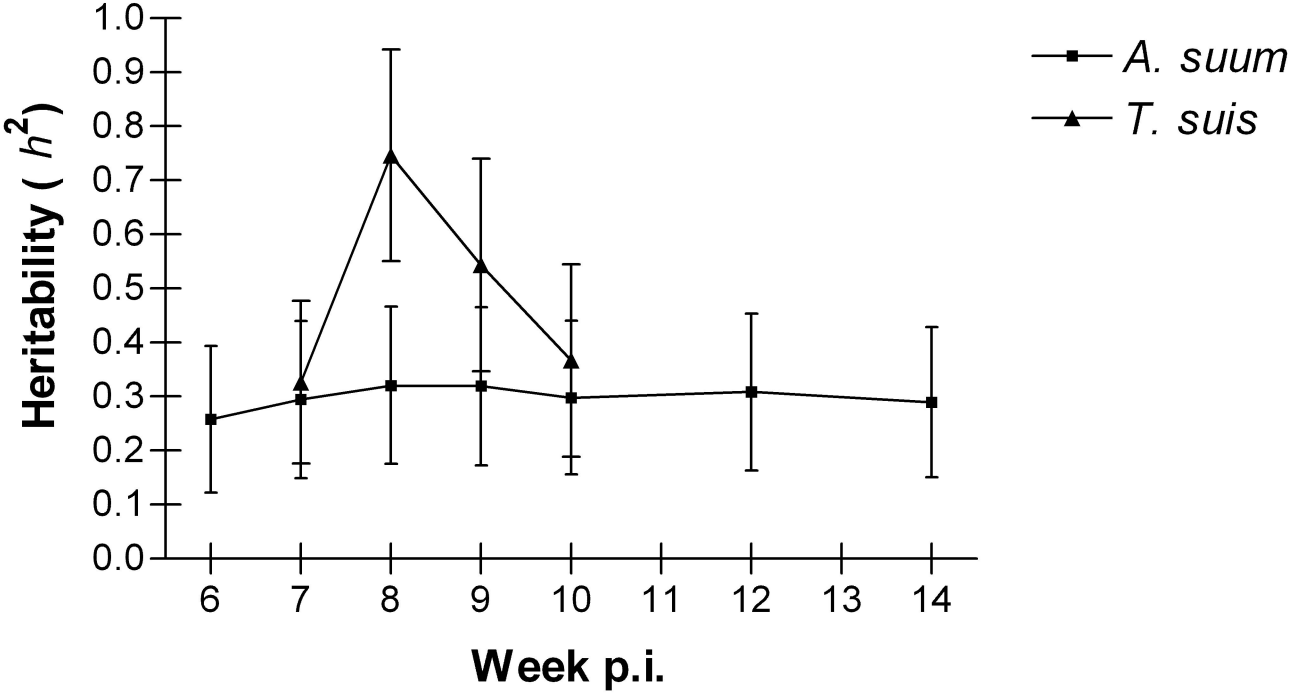

1.0
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
Heritability (h^2)
6
7
8
9
10
11
12
13
14
Week p.i.
A. suum
T. suis

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

Table 2
Heritability for *A. suum*

Trait	h^2	s.e	*P*
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

FEC, faecal egg count
s.e., standard error

Table 3
Heritability estimates for *T. suis*

Trait	h^2	s.e	*P*	*P* for delivery[a]	*P* 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

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

Tabel 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	-

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

FEC, faecal egg count

s.e., standard error