Genetic variation in serological response to *Mycobacterium avium* subspecies *paratuberculosis* and its association with performance in Irish Holstein-Friesian cows

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Introduction Paratuberculosis, also referred to as Johne's disease, is a contagious and chronic disease in ruminants caused by *Mycobacterium avium* subspecies *paratuberculosis* (MAP). Few estimates of the genetic variation in measures of susceptibility to MAP are available and even less have attempted to elucidate the genetic associations between measures of susceptibility to MAP and performance in dairy cattle. The objective was to quantify the genetic variation in susceptibility to MAP, as measured by serology, and to determine the genetic association between MAP serological response and performance in Irish Holstein-Friesian dairy cows.

Material and methods Data on animal serological response to MAP originated from two sources. In 2004 and 2005 blood samples were collected from all lactating and non lactating animals >12 months in 34 herds with suspected MAP infection. The second data base, originating from a paratuberculosis prevalence survey in 2007, was conducted on a random sample of Irish cattle herds. Serological response to MAP was measured using a MAP ELISA (Institut Pourquier, France). The sample to positive (S/P) ratio was used for interpreting test results. A positive animal had an S/P ratio of ≥ 70. The last paratuberculosis test observation in time per animal was retained and animals with no known sire or maternal grandsire were discarded. Only herds with at least two ELISA positive animals (see definition below), of which at least one was home bred were retained. Three herds with less than 10 animals remaining following all previous edits were discarded. The final dataset consisted of 4,789 cows from 44 herds. (Co)variance components were estimated in ASREML (Gilmour *et al*., 2008). Heritability estimates for serological response to MAP were estimated using both a univariate animal linear mixed model and a univariate animal threshold model. In the animal linear mixed model, the dependent variable was the normalised S/P ratio as well as the S/P ratio dichotomised into infected (S/P ratio ≥70) or not-infected (S/P ratio <70). For the threshold model analysis, a generalized linear mixed model with a logit link function was used. Fixed effects included in both models were herd-year of test, month of test, parity of animal (1, 2, 3, 4, ≥5), stage of lactation (6 stages), heterosis, recombination loss, and Holstein breed proportion. Genetic correlations between serological response to MAP and performance were estimated using a series of bivariate sire mixed linear models. Fixed effects included in the models for the performance traits included contemporary group, Holstein breed proportion, heterosis, and recombination loss.

Results Within the edited dataset, 211 (4.4%) of the 4,789 animals were MAP-positive (i.e., S/P ratio ≥70). Note that these estimates of herd-prevalence should not be extrapolated to the general Irish cattle population. The heritability for serological response to MAP measured on a continuous scale was 0.04 (SE=0.02) when estimated using an animal linear mixed model. Heritability of serological response to MAP when analysed as a binary trait was 0.07 (SE=0.028) and 0.14 (SE=0.069) when estimated using a linear and threshold animal model, respectively. Table 1 summarises the genetic correlations between serological response to MAP and performance. The genetic correlations with milk, fat and protein yield were negative or close to zero with the strength of the negative genetic correlations being greater for yields in parity 2 and 3 animals. Serological response to MAP was positively correlated with milk protein percent and negatively correlated with calving interval.

Table 1 Genetic correlations between MAP (treated as a dichotomous variable) and milk production, fertility and survival.

Trait	Parity 1	Parity 2	Parity 3	Range of standard errors
Milk yield	-0.07	-0.15	-0.08	0.086 to 0.094
Fat yield	-0.04	-0.41	-0.41	0.080 to 0.095
Protein yield	0.05	-0.18	-0.12	0.090 to 0.105
Fat %	0.07	-0.18	0.03	0.082 to 0.097
Protein %	0.16	-0.03	0.06	0.082 to 0.091
Somatic cell score	0.12	0.15	0.05	0.115 to 0.134
Calving interval	-0.59	-0.34	-0.13	0.132 to 0.179
Survival	0.06	0.24	0.16	0.239 to 0.248

Conclusions These results clearly indicate significant genetic variation in serological response to MAP exist and are consistent with previous international estimates. Although the response variable used in the present study only measures the immune response and is not a measure of the clinical symptoms, it may, however, be an approximation of the ability to control the infection. No strong genetic correlations with performance were evident with the possible exception of calving interval.

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References

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