Genetics of a range of health traits in dairy and beef cattle

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Summary

The objective of the present study was to estimate genetic parameters for a range of different health traits in dairy and beef cattle; the validation of the resulting genetic evaluations was also undertaken. The traits focused on related to viral diseases (bovine herpesvirus-1), bacterial diseases (tuberculosis), parasitic diseases (liver-fluke, neospora, ostertagia) and production diseases (lameness, mobility score and hoof health). Data used were obtained from nationally collected data by dairy and beef producers as well as abattoirs together with a research study which extensively phenotyped up to 11,000 dairy cows on 68 herds. Heritability estimates of the traits varied from 0.01 to 0.31; considerable exploitable genetic variation existed for all traits – the genetic standard deviation of the binary traits varied from 0.04 to 0.12 units.

Keywords: health, disease, respiratory, bacterial, viral, production, parasiteauthor, guide

Introduction

With the exception of a few populations (e.g., Scandanavia), animal health is poorly represented in many dairy cow breeding objectives and moreso in beef breeding objectives. This is unsustainable. A range of different diseases exist, each likely to have different prevalence but also in their cost of recording; for example, some health traits are already routinely recorded either because testing or treatment is being undertaken either as part of day-to-day herd management or as part of national monitoring schemes (e.g., tuberculosis and liver fluke). The objective of the present study was to estimate genetic parameters for a range of health traits in cattle; validation of genetic evaluations for tuberculosis and liver fluke are presented.

Material and methods

Data pertaining to bovine herpesvirus-1 (BoHV-1; the causative agent of Infectious Bovine Rhinotracheitis) were derived from two Irish research studies between the years 2010 and 2015, inclusive. In total, 19,353 BoHV-1 antibody test results were available on 16,242 female dairy cattle from 81 dairy herds. To maximise the likelihood of equal lifetime exposure to BoHV-1, only animals born in the herd that they were blood sampled in were retained. Exposure to BoHV-1 was defined in each herd-management group (i.e., nulliparous females vs. multiparous females) separately; an animal that was both born and tested for BoHV-1 antibodies in the same year as an animal that returned a positive BoHV-1 test result was deemed exposed. Only the most recent test result from animals deemed exposed to BoHV-1 were considered; 7,501 animals from 58 herds remained.

All Irish cattle are subjected to compulsory screening for *Mycobacterium bovis* at least once annually. Test results from animals that yielded a positive antibody response to the single intradermal comparative tuberculin test as well as confirmed cases of *M.bovis* infection, based on post-mortem examination, were available for the years 2000 to 2017, inclusive. Periods of infection were defined within herd-management groups (i.e., cows vs. heifers vs. steers vs. steers) as *M.bovis* screening periods that contained ≥2 positive test-results. Only herd-management groups comprising of at least five animals and a positive result were retained. Following extraction of a random sample of data, 65,475 skin test records and 65,869 abbatoir records remained.

Irish cattle are inspected by veterinarians for the presence of liver damage caused by *F. hepatica* at abbatoirs nationally. Cattle with a *F. hepatica* are recorded either to have "live *F. hepatica* present in the liver" or a "damaged liver caused by *F. hepatica*"; otherwise cattle were recorded as "negative for *F. hepatica*-damaged liver". Between the years of 2012 and 2016, inclusive, records from 1,042,929 dairy and beef cattle slaughtered in 7 abattoirs were available. In addition, blood samples from 10,879 cows collected in Autumn 2015 from 68 dairy herds, tested for the presence of an antibody response to *F. hepatica*, *O. ostertagi* and *N. caininum* were also available. Animals were deemed exposed to *F. hepatica*-damaged liver if they were herd-mates of an animal recorded with a *F. hepatica*-damaged liver; 19,542 dairy cows and 68,048 young animals (i.e., males and females < 1096 days of age that were not a registered sire or had no recorded calving event) remained. Cows were deemed positive and negative for *F. hepatica* and *N. caninum*. Cows in herds with >5 cows positive for *F. hepatica* and a within-herd prevalence of ≥5% positive for *F. hepatica* were deemed exposed to *F. hepatica*; cows in herds with a within-herd cow prevalence of >1% positive for *N. caninum* were deemed exposed. All cows were regarded as exposed to *O. ostertagi* since Irish herds graze for the majority of the year; 6,892, 5,289 and 9,260 cows remained with an antibody response to *F. hepatica*, *N. caninum* and *O. ostertagi*, respectively.

National records of binary-score lameness (n = 378,524 records) recorded by dairy producers on a per lactation basis were available for the years 2012 to 2016, inclusive. In addition, hoof health scores (scale of 0 to 3) recorded at one herd-visit by profession hoof-trimmers, as well as repeated measures of both mobility scores (i.e., scored from 0 (perfect gait) to 3 (severely impaired mobility) and body condition score (BCS; scale 1 to 5 with 0.25 unit increments) were collected during the 2015 calendar year from 11,282 cows in 68 Irish dairy herds; all cows in the herd on the day of visit were scored. For the hoof health traits, both the presence and the intensity of observed traits (i.e., scored from 0 (not affected) to 3 (severely affected)) were recorded for both back feet of all cows; the hoof health traits observed were overgrown sole (OG), white line disease (WL), and sole hemorrhage (SH). The average hoof score per cow was used as the phenotype.

Analysis

Variance components were estimated for each trait separately using univariate animal linear mixed models; covariances were estimated using bivariate models. All models included the relevant contemporary group as well as heterosis and recombination loss coefficients. When the data originated from cows, a fixed effect of parity and, where relevant, stage of lactation was also included; if the animal had not calved then the age of the animal as well as its gender were included as fixed effects. When repeated records existed (e.g., lameness, MS, BCS, *M.bovis*), a permanent environmental effect was included as a random effect. Individual animal estimated breeding values (EBV) for *M.bovis* and *F. hepatica* were calculated using the MiX99 software suite (MiX99 Development Team, 2015). For the genetic evaluation of

M.bovis, the pedigree of all animals in 1,194 herds was included in the pedigree file while the phenotypes of these were not included for the generation of EBVs. For the genetic evaluation of *F. hepatica*, the pedigree of all animals in the abattoir dataset (i.e. cows and young animals) was included in the pedigree file; however, no phenotypes of the cows were included to generate their EBVs thus avoiding any environmental covariance. Logistic regression was used to quantify the accuracy of predicting *F. hepatica*-damaged liver in cows based on their EBV.

Results and discussion

Viral diseases

The heritability for antibody response to BoHV-1 (as a binary trait) for vaccinated and non-vaccinated animals were 0.13 (SE=0.036) and 0.12 (SE=0.051), respectively. The genetic standard deviation for vaccinated and non-vaccinated animals were 0.12 units and 0.10 units, respectively. Where vaccinated and non-vaccinated BoHV-1 test results were considered as the same trait, the heritability estimate for antibody response to BoHV-1 was 0.12 (SE=0.028); the corresponding additive genetic standard deviation was 0.11 units. BoHV-1 genetic correlations with milk production traits were all close to zero (Table 1). Although, not different from zero, genetic corrlations between BoHV-1 and fertility traits had an unfavourable association, with the exception of calving to first service interval (Table 2). To our knowledge, the present study is the first to report variance components for antibody response to BoHV-1. Nevertheless, variance components are similar to those estimated for clinical signs of respiratory disease in beef (Snowder et al., 2006; Schneider et al., 2010) and dairy (Heringstad et al., 2008) cattle.

Bacterial diseases

The prevalence of *M. bovis* positive skin test results and confirmed lesions were 9% and 11%, respectively; the corresponding heritability estimates were 0.08 (SE=0.009) and 0.05 (SE=0.007) for the skin tests and lesions, repectively. The additive genetic standard deviation were similar (i.e., range 0.07 to 0.08) to that for BoHV-1.

Considering the EBVs from animals that did not have phenotypic data available for the generation of EBVs, the animals that were deemed of very high risk of being diagnosed positive for *M.bovis* were 1.45 times (95% CI: 1.32 to 1.59; $P < 0.001$) times more likely to be diagnosed positive for *M.bovis* than their contemporaries that were deemed of very low risk of being diagnosed positive for *M.bovis.* There was a 1.87 percentage unit difference in prevalence between both extremes.

Parasites

The prevalence of *F. hepatica*-damaged liver was 46% and 20% for cows and young animals, repectively. For *F. hepatica*-damaged liver in cows, the heritability estimate was 0.02 (SE=0.008) and additive genetic standard deviation was 0.05 units. Similarly for *F. hepatica*-damaged liver in young animals, the heritability was 0.01 (SE=0.005) and the additive genetic standard deviation was 0.036 units. The coefficients of genetic variation for immune reponse to parasites was similar to milk and fertility traits, ranging from 0.04 (*O. ostertagi*) to 0.20 (*F. hepatica*). The heritability estimates for the continuous trait of antibody response to

F. hepatica, *N. caninum* and *O. ostertagi* were 0.13 (SE=0.026), 0.09 (SE=0.030) and 0.07 (SE=0.018), respectively. Antibody response to *F. hepatica* had a positive genetic correlation with antibody response to *O. ostertagi* (0.91;SE=0.103) and had a negative correlation with antibody response to *N. caninum* (-0.29; SE=0.175). The genetic correlation between antibody response to *O. ostertagi* and antibody response to *N. caninum* was -0.67 (SE=0.160). The genetic correlations between the parasite phenotypes and milk production traits were all close to zero (Table 1). Antibody response to *F. hepatica* and *O. ostertagi* were favourably genetically correlated with fertility traits (Table 2). However, antibody response to *N. caninum* and *F. hepatica*-damaged liver were unfavourably genetically correlated with fertility traits (Table 2). Therefore, current breeding programs are likely having a benefical impact on *F. hepatica*-damaged liver in cattle.

Using only phenotypic data from young animals, cows with an EBV for *F. hepatica* in the top (i.e., worse) 10% were 1.28 (95% CI: 1.05 to 1.36; $P < 0.01$) times more likely to have *F. hepatica*-damaged livers compared to their contemporaries in the bottom 10%. Averaged across all contemporaries groups, there was a 6 percentage unit difference in prevalence between these extremes. Introduction of *F. hepatica*-damaged liver in cattle breeding programs would therefore complement current control practices (i.e., anthelmintic treatment).

Production (lameness)

Heritability, genetic standard deviation and the repeatability for lameness, MS and BCS and the different hoof health traits are in Table 3. Genetic correlations among the hoof health traits were all positive ranging from 0.18 (SE = 0.131; WL with SH) to 0.45 (SE = 0.128; OG with WL). Genetic correlations between the hoof health traits and BCS were weak (-0.19 to 0.24). Nevertheless, genetic correlations between the hoof health traits and MS were all positive and moderate to strong (0.58 to 0.64). In addition, cows genetically predisposed to lameness were genetically more likely to be affected by OG, WL and SH (correlations ranged from 0.27 to 0.41) as well as to have severely impaired mobility (0.64; SE = 0.140). Estimated variance components for the hoof health traits are higher than other studies (Van der Waaij et al., 2005; Chapinal et al., 2013) which may be for many reasons. The present study examined each rear-hoof of all milking cows in a herd as opposed to only cows that required hoof attention. In addition, herds that participated in the research study were selected based on their history of accurate and timely data recording on an abundance of traits.

Conclusions

Considerable genetic variability was shown to exist for a whole range of traits; moreover, the heritability of some of the health traits was not very low suggesting that not necessarily large quantities of data are required to achieve a high accuracy of selection. The validation of these genetic evaluations is useful in disproving the acquisations of some commentators that breeding for improved animal health is not a good use of resources. On the contrary in fact, breeding programs could be a useful complementary strategy to reducing or eradication diseases.

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Table 1. Genetic correlations (standard errors in parentheses) between health phenotypes and milk production traits.

Trait	Antibody response				
	BoHV-1	*F. hepatica*	*O. ostertagi*	*N. caninum*	*F. hepatica*-damaged liver
Milk yield	0.06	-0.07	-0.04	-0.01	0.10
	(0.073)	(0.060)	(0.062)	(0.052)	(0.159)
Fat yield	0.04	-0.12	-0.01	0.04	0.00
	(0.074)	(0.061)	(0.064)	(0.084)	(0.163)
Protein yield	0.04	-0.14	-0.05	0.02	0.05
	(0.077)	(0.063)	(0.065)	(0.086)	(0.175)
Fat percentage	-0.01	-0.09	0.00	0.02	-0.06
	(0.056)	(0.045)	(0.047)	(0.062)	(0.138)
Protein precentage	0.01	-0.09	-0.04	0.02	-0.08
	(0.056)	(0.046)	(0.047)	(0.062)	(0.128)
Fat-to-protein ratio	-0.01	-0.04	0.04	0.01	-0.07
	(0.064)	(0.051)	(0.052)	(0.069)	(0.143)
SCS	-0.13	-0.05	0.07	0.13	0.32
	(0.099)	(0.082)	(0.084)	(0.110)	(0.195)

Table 2. Genetic correlations (standard errors in parentheses) between health phenotypes and fertility traits.

	Antibody response				
	BoHV-1	*F. hepatica*	*O. ostertagi*	*N. caninum*	*F. hepatica*-damaged liver
Age of first calving	0.05 (0.193)	0.08 (0.374)	0.09 (0.359)	-0.48 (0.337)	-0.18 (0.375)
Calving interval	0.26 (0.182)	0.09 (0.174)	-0.11 (0.171)	0.43 (0.220)	0.48 (0.217)
Calving to first service interval	-0.29 (0.134)	-0.46 (0.138)	-0.31 (0.144)	0.56 (0.175)	0.36 (0.245)
Number of services	0.22 (0.147)	0.19 (0.145)	0.01 (0.150)	0.37 (0.192)	0.21 (0.250)
Survival	-0.059 (0.155)	0.36 (0.180)	-0.01 (0.184)	0.24 (0.223)	-0.41 (0.228)
Submission rate in 24 d	-0.06 (0.142)	0.13 (0.135)	0.15 (0.134)	-0.13 (0.172)	-0.30 (0.260)
Calving rate in 42 d	-0.24 (0.169)	-0.25 (0.200)	-0.06 (0.208)	0.18 (0.273)	0.00 (0.000)
Pregnancy rate in 42 d	-0.10 (0.160)	0.15 (0.169)	0.10 (0.168)	-0.73 (0.182)	-0.41 (0.248)
Pregnancy rate to first service	-0.20 (0.168)	0.01 (0.167)	0.05 (0.165)	-0.43 (0.208)	-0.30 (0.266)

Table 3. Mean value or incidence as well as genetic standard deviation (SDg), heritability (h^2; standard error in parenthesis) and repeatability (t; standard error in parenthesis) for a selection of the health traits

Trait	Mean/incidence	SDg	h^2	t
Lameness	0.11	0.05	0.03 (0.005)	0.12 (0.008)
Mobility score	0.43		0.07 (0.014)	0.17 (0.011)
Body condition score	2.915		0.31 (0.023)	0.50 (0.009)
Overgrown	0.71	0.3	0.16 (0.027)	
While line	0.75	0.38	0.20 (0.030)	
Sole hemorrhage	0.79	0.43	0.27 (0.032)	