Genotype-by-environment interactions for health traits across productivity level or health status of herds in Normande

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Abstract

Genotype-by-environment (GxE) interactions for somatic cell score (SCS) and clinical mastitis (CM) were investigated in first lactation Normande cows. Milk and SCS herd-year effects estimated in the French national evaluation were used as environment descriptors of productivity level and health environment of the herds, respectively. Variance and covariance components were estimated using bivariate and reaction norm models. For SCS, heritability estimates ranged from 0.17 to 0.23 according to productivity level. Genetic correlations higher than 0.9 were estimated between groups, showing no GxE interaction for this trait with both models. For CM, the estimates of heritability ranged from to 0.01 to 0.07. Genetic correlations between and 0.3 and 0.99 were estimated across productivity levels and health environments, indicating GxE interactions for this trait.

Introduction

Genotype-by-environment (GxE) interactions reflect the ability of genotypes to respond differently to a change in the environment. These interactions are currently ignored in the national genetic evaluation in dairy cattle in France. But the question arises as to whether these interactions should be taken into account due to the diversity of environments and herd management practices.

In a previous study, Huquet et al. (2012) investigated GxE interactions for production traits in French dairy cattle. No re-ranking was observed but they found a significant scaling effect for milk yield, with a higher genetic variance in the herds with more intensive management practices.

The aim of this paper was to investigate the magnitude of GxE interactions on SCS and CM in Normande cattle breed. Very diverse definitions of the herd environment have been used in the literature. In this study, herd environment description was based on herd-year effects estimated in the French genetic evaluations of milk production and SCS. The former is assumed to be a descriptor of the productivity level of a given herd, while the latter describes its sanitary status, regardless to its genetic level or the age of the animals.

Material & Methods

Description of the dataset. Data were obtained from the French national database.

First parity phenotypes of Normande cows recorded between 2015 and 2020 were included in the analyses. Phenotypes were lactation somatic cell scores (SCS) and clinical mastitis events (CM). Lactation SCS was computed as a weighted average of the test-day SCS ($SCS=\log_2(\text{Somatic Cell Count}/100{,}000)+3$)) adjusted for days in milk. CM phenotype was a

binary variable set to 1 when at least one clinical mastitis event was recorded during the first 150 days of lactation, and 0 otherwise.
Milk and SCS herd-year effects, reflecting productivity level and sanitary status of the herds, respectively, were used as descriptors of the environment. These estimates were obtained as a by-product of the French national genetic evaluation of milk yield and SCS performed in October 2020.

Data analysis. Only cows having completed their entire lactation within the same herd have been included in the analyses to ensure the phenotype was expressed in the right environment. Herds with less than 15 cows, therefore with limited accuracy of herd-year effects, were discarded.
The environment description was based on the deciles of the herd-year effect distribution. Two methods were investigated to estimate variance and covariance components for SCS and CM. First, a bivariate analysis was performed, in which the herds with extreme levels of productivity or sanitary status were selected. Then, performances were analysed with a reaction norm model, using the herd-year effects to describe the environment. SCS performances were analysed in relation to the productivity level only. For CM, two separate analyses were performed for the productivity level and the sanitary status, respectively.
For the bivariate analysis, herds randomly selected in the deciles 2-3 and deciles 7-8 defined two groups with contrasted situations. Bivariate models included the fixed effects of herd-year, age at first calving, month of calving, the breeding value as a random effect, and the residual. Pedigree information was traced back on three generations and included 100,111 and 112,810 individuals for SCS and CM, respectively.
For reaction norm models, herds were randomly selected in each decile. The average herd-year effects for each decile were used as regression variable. Reaction norm models included Legendre polynomials of order 2 for both traits:

$$\mathbf{y} = \mathbf{X\beta} + \sum_{r=0}^{2} \boldsymbol{Z}_r(\boldsymbol{t})\boldsymbol{g}_{ir} + \mathbf{e} \quad (1)$$

The pedigree file included 85,541 animals for the SCS analysis and 75,681 for the CM analyses.
The analyses were performed using Wombat software (Meyer, 2007).

Results

With bivariate models, heritabilities of 0.17 and 0.20 (±0.02) were estimated for SCS in low and high levels of productivity groups respectively (Table 1). No GxE interaction was found for SCS, with a genetic correlation of 0.99 (±0.02) between the two groups.
For CM, heritabilities between 0.01 (±0.006) and 0.03 (±0.008) were estimated (Table 1). Genetic correlations between low and high productivity level groups as well as between good and poor health environment groups were 0.7 (±0.2) indicating the existence of GxE interactions.
With reaction norm models, heritabilities of SCS along the milk herd-year effects ranged from 0.18 to 0.23 (Table 2). Genetic correlations between SCS at different milk herd-year effects were all higher than 0.9 (s.e. ranging from 0.003 between close groups to 0.52 between the most distant groups), indicating no GxE interaction for this trait. Heritabilities of CM along milk herd-year effects were low, ranging from 0.02 to 0.06 (Table 2). Similar values were obtained for the heritabilities of CM along SCS herd-year effects (0.01 to 0.07).
Genetic correlations between CM at different milk herd-year effects ranged from 0.44 between groups with the most distant herd-year effects to 0.99 between groups with the

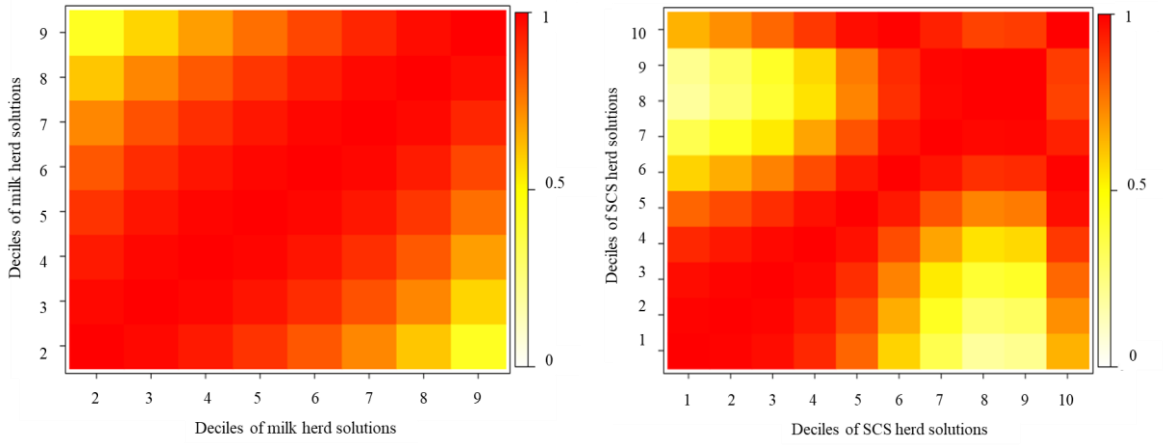
closest herd-year effects (Figure 1.a). The same trend could be observed for genetic correlations between CM along the SCS herd-year effects with values between 0.3 and 0.99 (Figure 1.b). However, the sampling errors were quite high and the estimates were not significantly different from 0 when using SCS herd-year effects as environment descriptor.

Table 1. Heritability and genetic correlation estimates for SCS and CM in herds with low and high productivity level and in herds with good and poor health environments with bivariate models.

Trait	Herd-year effects	Group	h^2	r_g
SCS	Milk	Low	0.17 ± 0.02	0.99 ± 0.02
		High	0.20 ± 0.02	
CM	Milk	Low	0.014 ± 0.006	0.70 ± 0.20
		High	0.025 ± 0.008	
	SCS	Good	0.020 ± 0.007	0.70 ± 0.20
		Poor	0.025 ± 0.007	

Table 2. Ranges of heritability estimates for SCS and CM according to productivity level and health environment with reaction norm model.

Trait	Herd-year effects	h^2
SCS	Milk	0.18 - 0.23
CM	Milk	0.02 - 0.06
	SCS	0.01 - 0.07



(a) (b)

Figure 1. Heatmaps of the genetic correlations between clinical mastitis at different milk herd-year effects (a) and SCS herd-year effects (b).

Discussion

Our estimates for SCS and CM heritabilities were consistent with the literature (Boichard and Rupp, 1999; Druet et al., 2005; Govignon-Gion et al, 2016).

Regarding GxE interactions, the analyses with bivariate and reaction norm models gave similar results. Both models supported the existence of GxE interactions for CM, but not for SCS. Our results are in agreement with previous studies reporting genetic correlations between 0.8 and 1 for SCS at the lactation level across environments (Carlén et al., 2005).

However, studies on test-day level are more informative and tend to show the existence of GxE for SCS in early lactation related to a higher incidence of mastitis in early stages of lactation (Calus et al., 2006). Similarly to Carlén et al. (2005), our bivariate analysis suggested the existence of GxE interactions for mastitis in different SCS environments.
The use of milk and SCS herd-year effects is an original approach to describe the environment in GxE interactions studies. They represent good estimates of the overall productivity level and health status of the herds and they are easily accessible since they are computed as part of the national genetic evaluations. The use of SCS herd-year effects was particularly interesting since few indicators on the health status of herds are available, these data being difficult to collect routinely. Proxies could be used as shown by Calus et al. (2006) who combined bulk milk SCC as an indicator of hygienic conditions to days in milk in their GxE analyses; and Carlén et al. (2005) who used herd-year average of somatic cell count and mastitis. Our results indicate that SCS herd-year effects are another useful proxy for health environment.
Different breeding strategies could be considered to take into account GxE interactions in the breeding programs. A guideline was provided by Mulder et al. (2006) who showed that it was optimal to have environment-specific breeding programs if the genetic correlation between environments was lower than 0.61.

Conclusion

This study showed that milk and SCS herd-year effects are useful proxies for the productivity level and health environment of the herds. We report the existence of GxE interaction for clinical mastitis in first lactation Normande cows. Taking these interactions into account in breeding programs would allow to adapt breeding strategies to various environments.

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