Genotype by environment interaction for udder health traits in Swedish Holstein cattle

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Abstract

Genotype by environment interaction for somatic cell count and mastitis in the first lactation of the Swedish Holstein breed was studied, using a reaction norm model and multiple trait analysis. Data from the Swedish milk-recording scheme containing more than 200 000 cows having their first calving from 1995-01-01 and onwards was used. Somatic cell count was defined as the average value of the monthly milk sampling results until 150 days after calving expressed in 10 000 cells per millilitre and transformed to a logarithmic scale to the base 10 (LSCC). Mastitis was defined as an all-or-none trait observed from 10 days before calving to 150 days after calving. Environments were defined as: herd-year averages of 305-day protein yield, somatic cell count, and mastitis, all measured in first lactation; and herd size, expressed as the average of 1995-2000 herd-year sizes. The multiple trait analysis was done using the highest and lowest quartiles of the environments herd-year protein yield, herd-year somatic cell count, herd-year mastitis, and herd size. The genetic correlation for LSCC and mastitis between low and high environments indicated GxE for LSCC in somatic cell count environment. Variances of the slope and the level of the reaction norm were analyzed by regressing phenotypic values of somatic cell count and mastitis on herd-year environments of protein yield, somatic cell count, mastitis and on the environment herd size. Significant genetic variation in slope was also detected for LSCC in somatic cell count environments and the correlation between predicted offspring performance in low and high somatic cell count environments showed GxE and indicated re-ranking of sires. The heritability of somatic cell count and mastitis estimated as functions of the environments tended to be lowest in average environments and increased with the distance from the average. Neither the multiple trait analysis nor the reaction norm model provided us with complete results when using the environmental scale mastitis. This is probably due to that the mastitis frequency is close to zero in low mastitis environments, resulting in a lack of phenotypic variation.

Introduction

Living organisms respond to changes in their environment, and the ability to alter the phenotype in response to changes in the environment is called phenotypic plasticity or environmental sensitivity. Differences in environmental sensitivity between individuals result in genotype by environment interaction (GxE), *i.e.* the difference between the phenotypic values of two genotypes is not the same in two environments. If the difference changes sign between environments, the effect of GxE is re-ranking of individuals. If the difference changes in magnitude, but not in sign, there is a scaling effect (Falconer & Mackay, 1996; Kolmodin, 2003).

There are basically three different methods to describe the extent of GxE. For all methods, observations on the same or related individuals in two or more different environments are needed to study GxE (de Jong, 1995). The common use of artificial insemination in dairy cat-

tle makes it possible to compare the performance of daughters of the same sires in different environments.

Interaction term model. In the first method the phenotypic value of an individual is simply described as the sum of the genotypic value, the environmental value and the residual, and when an interaction between genotype and environment exists an interaction component, GxE, is added: P=G + E + GxE + e.

Multiple trait model. In the second method the phenotypic expressions in the two environments are seen as two separate traits and r_g can be studied to see whether re-ranking GxE exists. The difference in genetic between two environments is a measure of whether scaling GxE exists. When r_g between the phenotypic values of the same genotype expressed in different environments is high, the phenotypic expression is considered as the same trait in the different environments and there is no (re-ranking) GxE (Robertson, 1959; Falconer & Mackay, 1996). When r_g is low, the phenotypic expressions in the different environments are not the same trait and this is an indication of GxE. The genetic correlation (r_g) can be estimated using a multiple trait analysis based on grouping herds with similar production environments to clusters and treating the observation from the different clusters as separate traits. GxE is indicated by low r_g between clusters (Falconer & Mackay, 1996).

Reaction norm model. When the production environment can be described as a continuous variable, a third method, called the reaction norm model, is possible to use (de Jong, 1995). The phenotypic expression of a genotype as a function of the environment is described by the reaction norm (Kolmodin, 2003). A given difference of an environment can have a greater effect on one genotype than on another (Falconer & Mackay, 1996).

The increasing co-operation and exchange of semen between countries has raised the question of GxE in dairy cattle breeding evaluation. If GxE is large, this would mean that the same bulls should not be selected in all countries. In the international sire evaluation for bulls, performed by the International Bull Evaluation Service (INTERBULL), the evaluation is based on a multiple trait across-country evaluation (MACE) and a sire model. The current estimates, among the member countries, for SCC and mastitis are in the range from 0.85 to 0.96 and 0.73 to 0.86, respectively (Interbull, 2004). Even within countries, there could be GxE, e.g., within large countries covering several different climates and production systems. If so, this would indicate the need for selection of different bulls for different environments (but not necessarily within country) (Kolmodin *et al*., 2002).

Previous studies have examined the existence of GxE for production and fertility traits (Kolmodin, 2003) and longevity (Petersson *et al.*, 2005) in dairy cattle for Swedish and Nordic countries. To make the picture more complete, knowledge about GxE for mastitis and somatic cell count (SCC) is desirable, especially because udder health is a trait of major importance in dairy cattle and few studies have been performed within this area (Weigel *et al.,* 2001). No or small evidence for the existence of GxE for SCC have been reported (Castillo-Juarez *et al*., 2000; Boettcher *et al*., 2003; Kearney *et al*., 2004; Mulder *et al.*, 2004). The objective of this study was to quantify GxE for both mastitis and SCC between different environments in the first lactation of Swedish Holstein cows. The methods used were multiple trait analysis and analysis with a reaction norm model.

Materials and methods

Data editing

The data set was collected in a previous study (Carlén *et al.,* 2004) and contained more than 200 000 cows of the Swedish Holstein breed. The data, originally received from the Swedish milk-recording scheme, contained information about identification number and year of birth of the cows and their sires, as well as the cow's proportion North American Holstein and proportion heterosis. There were also records of herd, year, month and age of first calving and information about protein production, SCC and clinical mastitis from first lactation. Mastitis was defined as an all-or-none trait observed from 10 days before calving to 150 days after calving. Since mastitis is a binary trait the record of a cow can be either 1 (the cow has at least one treatment of mastitis) or 0 (when the cow has not been treated for mastitis). The somatic cell count was defined as the average value of the monthly milk sampling results until 150 days after calving expressed in 10,000 cells per ml and transformed to a logarithmic scale to the base 10 (LSCC). The production of protein was defined as kg of the completed 305-days first lactation. Average LSCC in the data set was 0.787 (SD 0.426), which corresponds to about 60 000 cells per ml, and average mastitis was 0.099 (SD 0.299).

Herd-years were excluded if there were less than two cows with observations on either LSCC, protein production or mastitis in that herd-year. After editing, the dataset contained 221 104 cows belonging to 27 410 herd-year classes.

The phenotypic values of LSCC, protein production and mastitis were pre-adjusted before estimating herd-year mean values for the random regression model. The following fixed model using the GLM procedure in the SAS package (SAS Institute Inc., 2000) was used for the pre-adjustment:

$$y_{ijk} = \mu + a_i + m_j + am_{ij} + e_{ijk} \qquad [1]$$

where,

y_{ijk} = the value of LSCC, protein production or mastitis in first lactation of cow k
μ = overall mean,
a_i = fixed effect of ith age at first calving (in months),
m_j = fixed effect of jth month at first calving,
am_{ij} = interaction effect between age and month at first calving,
e_{ijk} = random residual effect.

The residuals were used to calculate mean values of LSCC, protein production and mastitis for each herd-year class. The mean value for overall herd size corresponded to a mean from the 1995-2000 herd-year sizes, number of cows with first calvings in a herd per year. The average was taken to get an estimate of herd size less influenced by fluctuating numbers of first-calvers, especially in small herds.

Multiple trait analysis

For the multiple trait analysis the observations were divided into low and high herd-year clusters with regard to LSCC, protein production, and clinical mastitis and in herd clusters with regard to overall herd size, using the UNIVARIATE procedure in the SAS package (SAS Institute INC., 2000). Observations in low and high quartile of each environmental variable were chosen for analysis and are illustrated in Table 1.

Table 1. Number of cows in low and high quartiles of each production environment and the number of bulls with daughters in each quartile.

	Low quartile		High quartile	
Environment	**Cows**	**Bulls**	**Cows**	**Bulls**
Protein yield	52 513	836	52 512	835
Somatic cell count	52 514	835	52 504	838
Mastitis	52 503	836	52 499	834
Overall herd size	54 183	834	54 669	837

The sires born before 1991 could not be considered as young test bulls because they had reached too high an age and only the selected bulls had daughters in the material. In an attempt to get more unbiased estimates of the variances, the older bulls (n=311 with 66 399 daughters) were considered as fixed and the young bulls (n=527 with 154 705 daughters) were considered as random. The following bivariate multiple trait model was used for LSCC and clinical mastitis:

$$y_{ijkmn} = \mu + ym_i + age_j + hy_k + sire_n + sire_o + b_1Het_m + b_2AmH_m + e_{ijkmno} \quad [2]$$

where:

μ = overall mean
ym_i = fixed effect of i^{th} year by month of calving
age_j = fixed effect of j^{th} age in months at calving
hy_k = fixed effect of k^{th} herd by year of calving
$sire_n$ = random effect of sire *n* in unselected batches
$sire_o$ = fixed effect of sire *o* in selected batches
b_1Het_m = fixed regression of coefficient of the proportion heterosis of animal *m*
b_2AmH_m = fixed regression of coefficient of the proportion American Holstein of animal *m*
$e_{ijklmn(o)}$ = random residual effect

Variance and covariance components were estimated with the DMU package (version 6, release 4) developed by Madsen & Jensen (2000). Both convergence criteria were set to 10^{-6} and in order to reach the convergence criteria faster the values from the previous run multiple trait analyses were used as starting values in subsequent analyses. The random effects were assumed to have zero means and the covariance structure was:

$$V\begin{bmatrix}\mathbf{s}_1\\ \mathbf{s}_2\\ \mathbf{e}_1\\ \mathbf{e}_2\end{bmatrix} = \begin{bmatrix}\mathbf{A}\sigma^2_{s_1} & \mathbf{A}\sigma_{s_{1,2}} & 0 & 0\\ & \mathbf{A}\sigma^2_{s_2} & 0 & 0\\ & \textit{symm.} & \mathbf{I}\sigma^2_{e_1} & \mathbf{I}\sigma_{e_{1,2}}\\ & & & \mathbf{I}\sigma^2_{e_2}\end{bmatrix}$$

where **A** is the additive relationship matrix and **I** is the identity matrix, and the indexes represent the two traits in the bivariate analysis.

Reaction norm model

In order to avoid dependence between the dependent and independent variables, new herd-year mean values for LSCC (hylscc), protein production (hyprot) and clinical mastitis (hymast) were calculated for the reaction norm model. The value of the environmental scale for a particular individual was corrected for its own observation, to avoid including a cow´s obser-

vation both in the dependent and independent variable. The following linear random regression sire model was used to study the data:

$$y_{ijklm} = \mu + ym_i + age_j + hy_k + b_1Het_m + b_2AmH_m + s_{a_l} + s_{b_l}X_{ml} + e_{ijklm} \quad [3]$$

where:

$y_{ij(k)lmn,}$ μ, $ym_{i,}$, age_j, $hy_{k,}$, b_1Het_m and b_2AmH_m are as before and

s_{a_l} = random intercept or level of the random regression for sire l

s_{b_l} = random linear coefficient or slope of the random regression of y on $X_{ml,}$ for sire l

X_{ml} = the environment daughter m of sire l produced in

e_{ijklm} = random residual

The random effects were assumed to have zero means and the covariance structure was:

$$V\begin{bmatrix}\mathbf{s_a}\\ \mathbf{s_b}\end{bmatrix} = \begin{bmatrix}\sigma^2_{s_a} & \sigma_{s_{a,b}}\\ \sigma_{s_{a,b}} & \sigma^2_{s_b}\end{bmatrix} \otimes \mathbf{A} = \mathbf{S} \otimes \mathbf{A} \qquad V[\mathbf{e}] = \mathbf{I}\sigma^2_e \quad [4]$$

where **A** is the additive relationship matrix and **I** is the identity matrix, and the indexes represent the random intercept (level) and the random linear coefficient (slope). The sire effects and the residual were assumed to be uncorrelated. The matrix **S** is equal to one quarter of the genetic variance matrix for the level and slope. As for the multiple trait analysis, the DMU package was used to estimate variance and covariance components (Jensen and Madsen, 2000).

The model [3] has the random regression and the environmental variable, $X_{ml,}$ added. For each sire the level and the slope of a linear reaction norm were estimated for the environments based on herd-year averages for protein, LSCC and mastitis and for herd size. Predicted offspring performance (POP), depends on the environment the offspring will produce in. The formula used to calculate the POP for sire l in environment X is:

$$POP_{l|X} = \mu + s_{a_l} + s_{b_l}X \quad [5]$$

The POPs were calculated from the herd year means of protein yield, LSCC, mastitis and herd size. The predicted offspring performance was calculated in the range of ±3 standard deviations from the mean. The correlation between POP in the average environment and POP in deviating environment was calculated to illustrate the potential re-ranking of sires between environments. To have more accurate correlation curves these were based on only the sires with daughters in the data set. The range of environments in these curves contained 95 % of the observations.

The sire variance was calculated as the variance of [5] since POP, and also the heritability, varies with the environment. Also the range of environments in the heritability curves contained 95 % of the observations.

$$\sigma^2_{s|X} = \sigma^2_{s_a} + X_i^2\sigma^2_{s_b} + 2X_i\sigma_{a,b} \quad [6]$$

$$h^2|X = \frac{4\sigma^2_{s|X}}{4\sigma^2_{s|X} + \sigma^2_E} \quad [7]$$

where,

$$\sigma^2_E = \sigma^2_e - 3\sigma^2_{s|X=0} \quad [8]$$

Results

Multiple trait analysis

Average LSCC and mastitis in low and high quartiles of the herd-year environments protein yield, LSCC, mastitis and herd size from the multi-trait analysis are shown in Table 2. Neither LSCC nor mastitis differed between low and high herd sizes and there was little difference between low and high production herds. As expected, there was a large difference in LSCC and mastitis, respectively, between low and high LSCC and mastitis herds, respectively. In fact, the mastitis mean and standard deviation are near zero in the low quartile of mastitis environment. When the environment variable was LSCC or mastitis, respectively, and the trait studied was mastitis or LSCC, respectively, the difference was less pronounced.

Table 2. Average and standard deviation (SD) of LSCC and mastitis in low and high quartiles of different environments.

	LSCC[1] mean ± SD		**Mastitis mean ± SD**	
Environment	**Low quartile**	**High quartile**	**Low quartile**	**High quartile**
Protein yield	0.824 ± 0.425	0.752 ± 0.421	0.098 ± 0.297	0.107 ± 0.309
LSCC	0.547 ± 0.316	1.039 ± 0.452	0.089 ± 0.285	0.123 ± 0.328
Mastitis	0.760 ± 0.413	0.814 ± 0.443	0.000 ± 0.000	0.285 ± 0.451
Overall herd size	0.788 ± 0.430	0.791 ± 0.421	0.103 ± 0.304	0.101 ± 0.301

[1]LSCC = the average value of the monthly milk sampling results until 150 days after calving expressed in 10,000 cells per ml and transformed to a logarithmic scale to the base 10. For instance, the value 0.824 transforms into 66 681 cells/ml.

Genetic correlations for LSCC and mastitis in low and high herd-year protein yield, LSCC, mastitis and herd size environment from the multiple trait analysis are presented in Table 3. The analysis to estimate the genetic correlation between mastitis in low and high mastitis environments did not converge owing to the low variation in herds with low mastitis frequency (Table 2). The correlation for both LSCC and mastitis was high and not different from unity in the environments protein yield and herd-year size. Genetic correlation for LSCC in low and high LSCC environments was significantly lower than unity. The genetic correlation for LSCC in low and high mastitis environment was also low but with too large a standard error to make it different from unity. The same is the case for the correlation for mastitis in low and high environments of LSCC and herd size.

Table 3. Genetic correlations and standard errors from the multiple trait analysis for LSCC and mastitis in low and high quartiles of different environments.

	Environment			
Model and trait	**Protein**	**LSCC**	**Mastitis**	**Overall herd size**
LSCC	0.980 ± 0.0627	**0.803** ± 0.0829	0.887 ± 0.0689	1.00 ± 0.0588
Mastitis	0.950 ± 0.2535	0.891 ± 0.2265		0.857 ± 0.1869

The heritability estimates of LSCC and mastitis in various environments are shown in Table 4. For LSCC in the environments LSCC and herd size the heritability tended to be higher in the high quartiles of the environment.

Table 4. Heritability of LSCC and mastitis in low and high environment quartiles from the multiple trait analysis.[1]

	Environment							
	Protein		**LSCC**		**Mastitis**		**Herd size**	
Model and trait	**Low**	**High**	**Low**	**High**	**Low**	**High**	**Low**	**High**
LSCC	0.135	0.137	0.109	0.170	0.124	0.137	0.135	0.158
Mastitis	0.027	0.024	0.038	0.034			0.054	0.042

[1] The standard errors of the heritability estimates of LSCC ranged from 0.11 to 0.17, and the standard errors of the mastitis heritability ranged from 0.001 to 0.002.

Reaction norm model

Variance components for the level and the slope, and the genetic correlation between the level and the slope of the reaction norm for various environments are shown in Table 5. The heritability of LSCC and mastitis in the average environment for each environment variable are presented in Table 6. The heritability of LSCC is highest in the average herd-year protein yield environments and lowest in average herd-year LSCC environments. For mastitis, on the other hand, the heritability is highest in average herd-year mastitis environment and lowest in average herd-year LSCC environment.

Table 5. Genetic variances and standard errors, correlations and standard errors for effects of level (a) and slope (b) of the reaction norm and residual variances and standard errors.

Trait and model	**Environment**	$\sigma^2_{s_a}$	$\sigma^2_{s_b}$	$r_{g_{a,b}}$	σ^2_e
LSCC					
	hyprot	4.884E-03 ± 3.604E-04	1.310 ± 0.141	0.00 ± 0.300	0.148 ± 5.236E-04
	hylscc	1.723E-03 ± 1.454E-04	0.761 ± 0.040	0.15 ± 0.240	0.085 ± 2.894E-04
	hymast	4.750E-03 ± 3.450E-04	72 323 ± 7 866	0.03 ± 0.290	0.158 ± 5.389E-04
	hsize	4.742E-03 ± 4.137E-04	0.062 ± 0.167	0.44 ± 3.240	0.160 ± 5.440E-04
Mastitis					
	hyprot	4.450E-04 ± 6.678E-05	2.897E-07± 4.585E-08	0.14 ± 0.494	0.082 ± 2.871E-04
	hylscc	4.226E-04 ± 6.304E-05	3.763E-08 ± 4.023E-09	-0.04± 0.390	0.083 ± 2.826E-04
	hymast	3.628E-04 ± 4.426E-05	1.000 ± 5.166E-02	0.78 ± 0.154	0.046 ± 1.520E-04
	hsize	6.404E-04 ± 1.040E-04	3.667E-08 ± 8.961E-08	-0.16± 0.533	0.088 ± 2.929E-04

Table 6. Heritability of LSCC and mastitis in the average environment for each environment variable.

Model	**Environment**	**LSCC**	**Mastitis**
	hyprot	0.130	0.024
	hylscc	0.083	0.020
	hymast	0.117	0.054
	hsize	0.120	0.028

The heritability of LSCC and mastitis as a function of the environmental scales protein yield, LSCC and mastitis in model C is shown in Figure 1 and Figure 2, respectively. The range of environments shown contains 95 % of the observations. The range in SD units around average for prothy, proth, lscchy, lscch, masthy and masth were -2 to +2, –2.2 to +1.9, –1.9 to +2.2, -1.9 to +2.1, -1 to +2.8 and –1.4 to +2.4, respectively. For LSCC in hylscc and for mastitis in hymast environments the heritability was high (near 1) in most deviating environments.

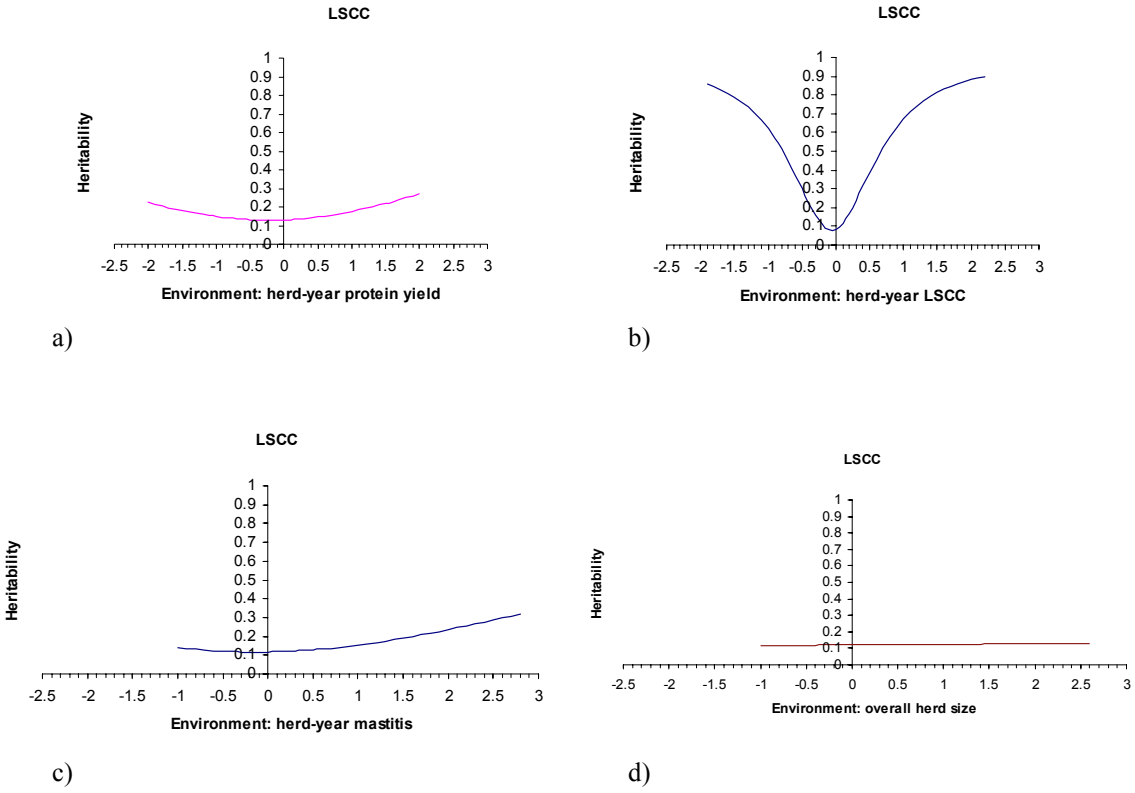

Figure 2. Heritability of LSCC as a function of a) herd-year protein yield, b) LSCC, c) mastitis and d) herd size.

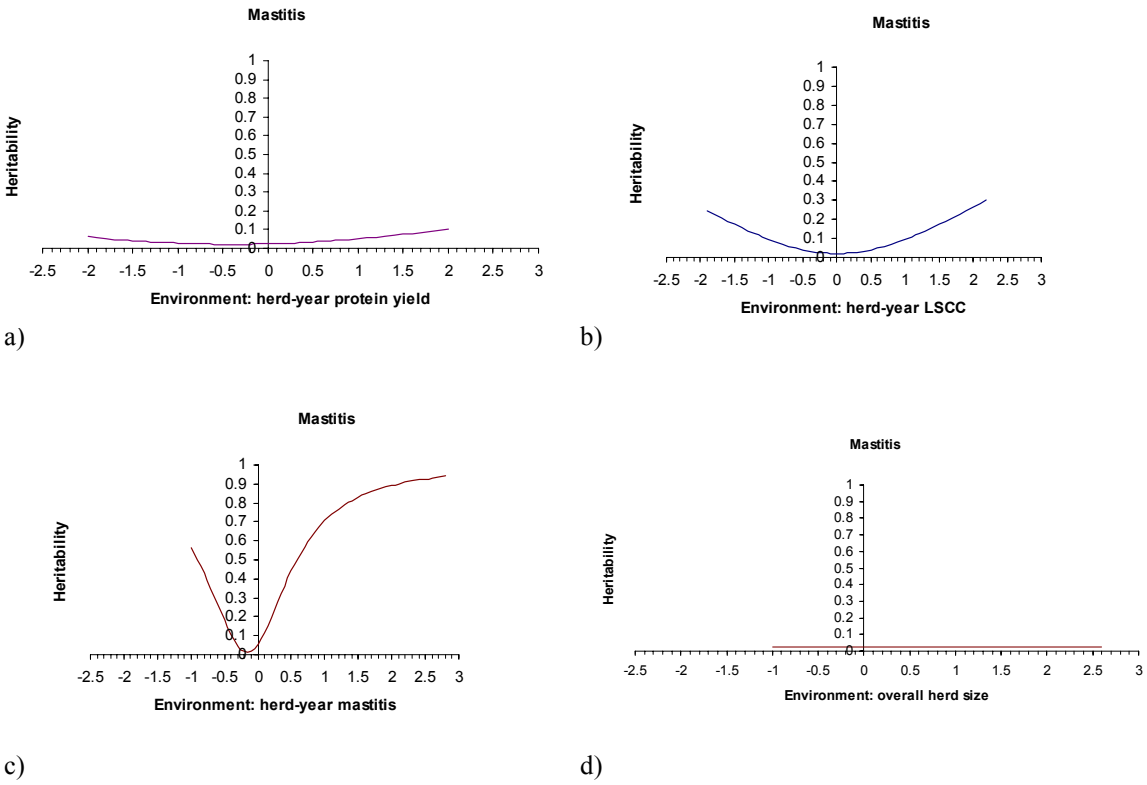

Figure 3. Heritability of mastitis as a function of herd-year a) protein yield, b) LSCC, c) mastitis and d) herd size

For LSCC and mastitis, correlations between POP in average and deviating environment of protein yield, LSCC and mastitis are shown in Figures 3 and 4, respectively. For these environments there is a tendency of re-ranking of sires in regard both to LSCC and mastitis. In the environment hylscc there is most evidence of GxE for the two traits. The correlation in environment overall herd size showed no GxE for neither of the two traits and is therefore not shown.

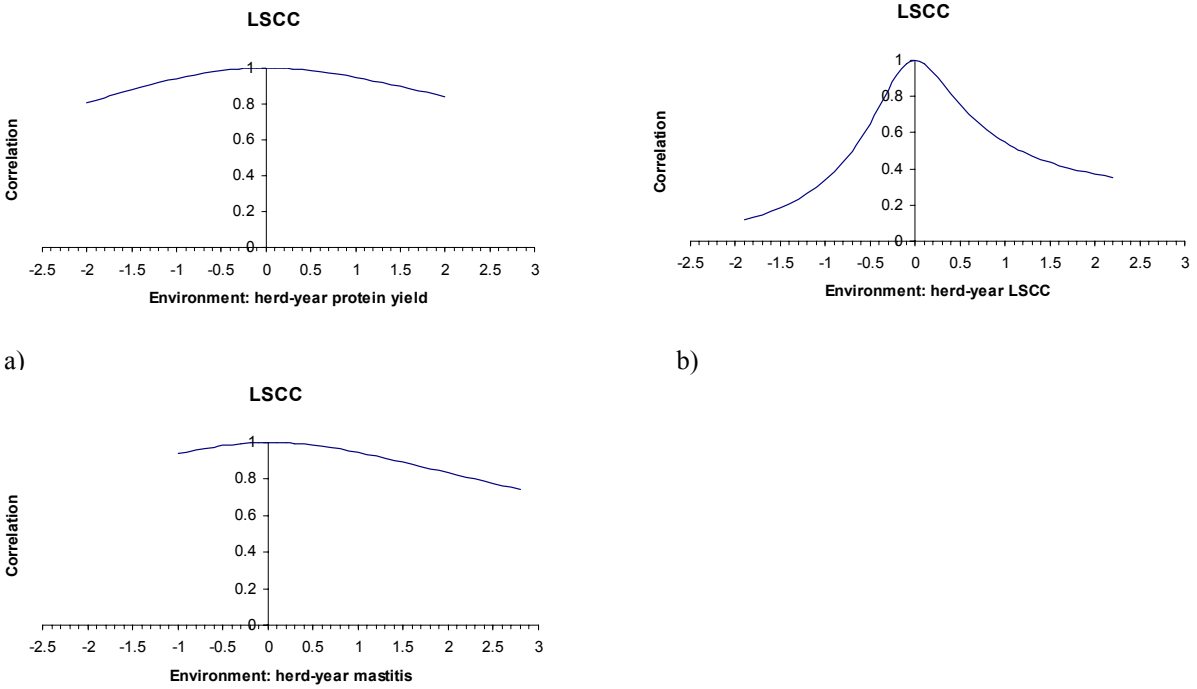

Figure 4. Correlations between POP in the average environment (0) and deviating environments for the environmental scales herd-year a) protein yield, b) LSCC and c) mastitis for LSCC.

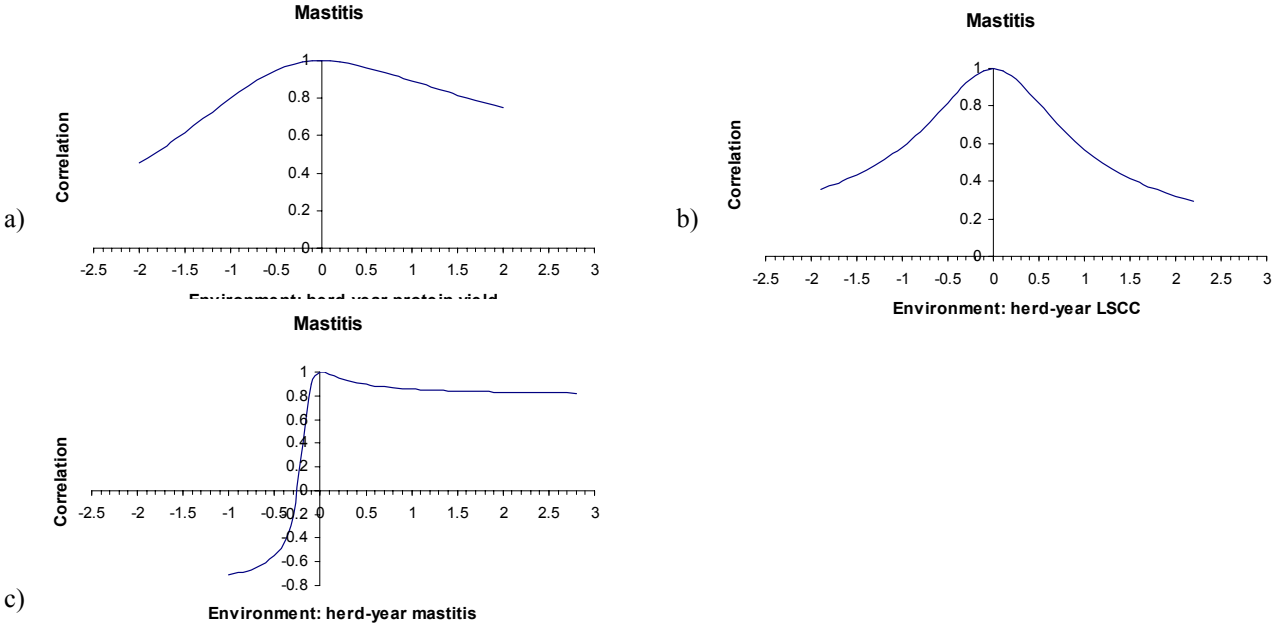

Figure 5. Correlations between POP in the average environment (0) and deviating environments for the environmental scales herd-year a) protein yield , b) LSCC and c) mastitis for mastitis.

Discussion

The genetic correlations between LSCC and mastitis in low and high quartile herd-year environments estimated in the multiple trait analysis indicated some re-ranking in a few environments. This was the case for LSCC in the environment LSCC (0.80). For LSCC in mastitis environment (0.89) and for mastitis in LSCC environment (0.89) and herd size environments (0.86), correlations were also below 1, however, not significantly so. To set these correlations in perspective, they can be compared with the correlations used by Interbull for production traits between countries in the Northern hemisphere (e.g. USA and Europe) on one hand and New Zealand/Australia on the other (around 0.75-0.85) (Interbull, 2004).

The fact that this analysis did not provide us with the genetic correlation between mastitis in low and high mastitis environment, is not surprising considering the all-or–none character of mastitis. This means that in a low mastitis environment the mastitis frequency is zero and consequently there is a lack of phenotypic variation. Even if this becomes very obvious for the mastitis environment the same problem does occur also for other traits as was pointed out by Kolmodin et al. (2002) for the trait days open. This phenomenon is most problematic when the environmental scale is based on the same trait as the dependent variable. In the current study we avoided the obvious dependency by excluding the individual's own observation from the herd-year average, however, the does not solve the problem of low variation in herds with low mastitis treatment incidence. This phenomenon highlights a crucial point in the study of GxE, the definition of the environmental scale. This applies to all methods of analyzing GxE. There is a need for a better and hopefully generally accepted approach for defining the environment.

An alternative model was tested in the multiple trait analysis, that did not account for the fact that only some sires of a batch had daughters in the data (so-called selected sires), but the results from this analysis are not presented. The model presented, which accounted for the fact that some sires were selected provided a few more genetic correlations below unity and also tended to give higher heritability of LSCC and mastitis in most environments. This latter result may be due to that the sire variance was estimated only from complete bull batches of young bulls. However, the standard errors of both heritabilities and genetic correlations were higher, probably owing to less information used (fewer sires and fewer records of cows).

There was a tendency to higher heritability of LSCC and mastitis in high protein yield. Previous studies (Castillo-Juarez *et al,* 2000, 2002) have reported the opposite relationship between the heritability of LSCC and yield environment.

For the reaction norm model there were some problems updating the parameter vector using the AI-algorithm when running mastitis in the environment hylscc, and the EM-algorithm was used instead.

The genetic variance components of level of reaction norms were always significantly different from zero for LSCC and mastitis, regardless of the environmental scale. Genetic variance in slope, indicating GxE, was detected for LSCC for all environments except herd size. For mastitis, the sire variance for slope was significantly different from zero for both mastitis and LSCC environments, and also for the environment hyprot.

When studying the correlation curves between POP in average and deviating environments, it becomes clear that the largest re-ranking would be expected for LSCC between average and

low and high LSCC environments (Figure 4). These results were in agreement with the multiple trait analysis (Table 3) where the genetic correlations between low and high quartiles were 0.80-0.84. For mastitis there were also low correlations between POP in average and low or high LSCC environments, indicating potential re-ranking (Figure 5). The genetic correlation for mastitis between low and high LSCC quartile herds in the multiple trait analysis also indicated re-ranking (Table 3), however, the correlation (0.89) was not significantly different from unity. Previous performed studies have shown evidence of GxE due to scaling in dairy cattle but little evidence of GxE due to re-ranking (Cromie *et al.*, 1998).

For both LSCC and mastitis, there was some indication of re-ranking also when the environment was herd-year average protein yield, and for LSCC also when the environmental scale was mastitis average. The latter indication if GxE was found also in the multiple trait analysis, however again, the correlation (0.89) was nor significantly different from unity. For mastitis, the correlation dropped sharply when the environment changed from average to low herd-year average mastitis and went to negative values (Figure 5). This was not corroborated from the multiple trait analysis, in fact, that analysis did not converge owing to too low variation in the trait mastitis in low mastitis herds. It is likely that the same phenomenon has influenced the estimates of the reaction norms. If there is no or little (genetic) variation at a certain point on the environmental scale, all reaction norms would tend to cross at that point. This would automatically lead to a change of sign of the correlation of POP across this point. This negative correlation should probably be interpreted with caution.

The genetic correlation between the reaction norm level and slope for mastitis against mastitis environments and against hprot environment was high. This means that animals with high breeding values for level of mastitis are sensitive to changes in the herd production environment as well as in the mastitis frequency environment. This is an example of the scaling effect of GxE (Kolmodin *et al,* 2002).

The genetic correlation for LSCC between the reaction norm level and slope against herd-year protein yield and mastitis environments are low. The low genetic correlation between level and slope means that the animals can be sensitive to environmental changes regardless of their breeding value for the level (Kolmodin *et al,* 2002).

The heritability estimates in average environment of LSCC and mastitis vary from 0.083 to 0.130 and from 0.020 to 0.083, respectively (Table 7). When estimating the heritability as a function of the environment the lowest heritability of LSCC is found in average environment. With increased distance from the average the heritability also increased, especially for LSCC in LSCC environment. The heritability of mastitis as a function of the environment was projected in the same way except in mastitis environments where the lowest heritability was found below the average environment. In environments over the average environment the heritability increased more then in low environment.

The results from the multiple trait analysis are not easily comparable to the results from the reaction norm model since the models used in the analyses differ and the multiple trait analysis uses only half of the herds.

Conclusions

GxE was detected with both the multiple trait analysis, as a low genetic correlation between the trait in low and high environment, and the reaction norm model, as a significant variation in the slopes of the reaction norms. The two analyses used to detect GxE for LSCC and mastitis were possible to use, even if there were some problems with the trait mastitis in mastitis environments.

The genetic correlation estimated in the multiple trait analysis indicated some re-ranking for LSCC between low and high LSCC environments. There was also indication of re-ranking for LSCC in mastitis environment and for mastitis in LSCC and herd size environments, but they were not significant. The results from the multiple trait analysis corroborates the results from the reaction norm model as the genetic variance in slope indicated re-ranking for LSCC in LSCC environments. The correlation between POP in average and deviating environments also show that the largest re-ranking would be expected for LSCC between low and high LSCC environments.

In practice, the detected GxE for LSCC in LSCC environment could affect the choice of bulls when selecting for udder health for the next generation of dairy cows within a dairy herd.

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