The Causal Relationship between Specific Pathogens and Milk Somatic Cells in Valdostana Cattle Breed

A.B. Samoré[*], S.I. Roman-Ponce[*†], M. Vevey[¥], C. Maltecca[‡], A. Bagnato[*]

Introduction

Mastitis is a costly health problem in dairy cattle (Hilleron and Berry, 2005) generally caused by bacteria infection of the udder that results in an increase of somatic cell count (SCC) in milk. In dairy cattle therefore SCC has been used as an indirect trait for genetic improvement of mastitis resistance for the large genetic correlation between SCC and direct mastitis and for the possibility of data collection of SCC during routine milk recording (Shook and Schutz, 1994). Even if high SCC in milk is indicative of both clinical and subclinical mastitis, the increase and the pattern of SCC in milk during a mastitis episode depends on several environmental factors (Mrode and Swanson, 1996) and on the pathogens causing the infection (De Haas et al., 2002). Objective of this study is the estimation of recursive effects associated to the presence of specific pathogens in milk to the SCC levels in the dual purposes Valdostana cattle breed.

Material and methods

Data. Data were provided from the Italian National Association of Breeders of Valdostana dairy cattle and included bacteriological information collected on a herd basis from 2001 to 2008. All cows in herds with almost a case of clinical mastitis were tested for bacteria presence. Specific pathogens information were collected for *Staphylococcus aureus* (STAUR), and *Streptococcus agalactiae* (STREA). Samples with a detected pathogen not pertaining to the other two microorganisms were tagged as ENV. Each pathogen infection status is coded as 1 or 0 according to its presence or absence, respectively. Information on SCC was included in the analysis extracting the value of the routine milk recording test nearest to the bacteriological analysis date. SCC were transformed to SCS according to Wiggans and Shook (1987).

Data were edited to ensure a minimum class size of 5 daughters per sire and 5 cows per herd and resulted in 20,868 records of daughters of 858 sires producing in 848 herds. Three generations of ancestors of sires were used resulting in 4,773 animals.

Recursive model. Data were analyzed using the SirBayes package, (Wu et al., 2007). A sire multiple trait model was fitted including the random effect of sire and fixed effects of herd, year-month of test (58 levels), month of calving (12 levels), days in milk (10 classes of 30 days), and parity number (3 levels: 1,2, and 3+). A total of 150K iterations were performed

[*] Università degli Studi di Milano. VSA Department, via Celoria 10, 20133 Milano, Italy
[†] INIFAP. C.E. Valles Centrales - CIRPAS. Oaxaca, Oax, México. 68200
[¥] ANA.Bo.Ra.Va. Gressan, Italia. 11020.
[‡] Department of Animal Science NCSU. 27695 Raleigh NC, USA

with a burn-in of 30K. Posterior samples were saved every10 iterations after burn-in. Convergence was assessed by visual plot of the posterior distributions. Traits included in the analysis were SCS, STAUR, STREA, and ENV.

Results and discussion

Descriptive data. A total of 16.95% of data reported the presence of STREA, 22.89% of STAUR and 50.75% of ENV pathogens. ENV group was large because it included all samples with a detected pathogen of any type. SCS was in average 2.88±2.10 with clear differences in levels between infected and non-infected samples for STREA (4.25 vs 2.60) and STAUR (3.48 vs 2.70). In contrast, the difference in SCS levels was almost reverse in ENV group (2.71 vs 3.04).

Recursive effects. The posterior distribution of the recursive effect on SCS for each pathogen class is presented in Figure 1. The recursive effects from the categorical trait (the pathogen) to the continuous trait of SCS were inferred on the underlying scale, the liability to the presence of the specific pathogen (Wu et al., 2008). The recursive effects (figure 1) of STAUR and STREA on SCS had positive posterior means (0.128±0.015 and 0.200±0.015 increase of SCS for 1 unit increase of liability) while the effect of ENV on SCS was almost null (0.024±0.013). These results suggested that an increase incidence of (or liability to) the presence of one of the two major pathogens increased SCS in milk at the nearest test day.

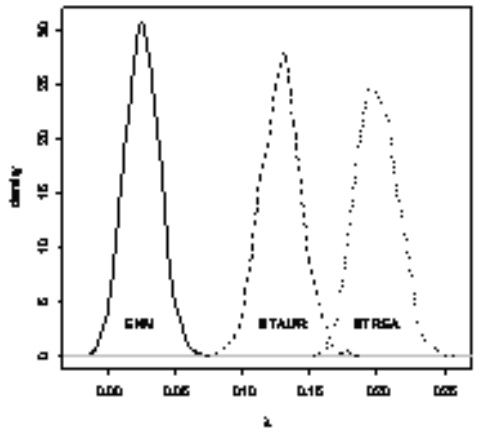

Figure 1: Posterior distributions of direct effect from the specific pathogen (STAUR=*Staphylococcus aureus*, STREA= *Streptococcus agalactiae* and ENV=other pathogens in milk) to SCS.

The effect from ENV group of pathogens to SCS was weak reflecting actually the non-evident difference in SCS phenotypic averages between infected and non-infected samples. The increase in SCS associated to udder inflammations due to infections was clearly evident in the case of most important bacteria involved in mastitis cases. In contrast the concentration in SCS did not seem to change with the presence of other pathogens. Indeed

the group of ENV pathogens included several different microorganisms and possibly for this reason the whole group of pathogens, not identified as STAUR and STREA, was not always causing a significant increase in SCS.

Genetic parameters. The estimated heritabilities (Table 1) for liability of STREA and STAUR had values of 0.07 indicating the presence of a similar genetic predisposition of contracting infection from STAUR and STREA. In contrast the heritability for liability to ENV was small (0.035) suggesting a limited genetic variation related to the predisposition to develop infection from environmental and others pathogens. Nevertheless it should be noticed that the group of ENV pathogens included all samples with an identified bacteria not pertaining to the two major pathogens STAUR and STREA. The low heritability value for ENV could therefore be the result of a complex host-pathogens (many different microorganisms) interaction having different effect on the SCS increase. Considering single microorganism, as in this study for STREA and STAUR, might therefore lead to different results. Finally the posterior mean of SCS heritability was 0.07 similarly to previous estimates in the same breed (Roman-Ponce et al., 2010).
The genetic correlation between STAUR and STREA was moderately high (0.52) while correlations of these pathogens with ENV were negative in both cases (-0.28 and -0.32 respectively for STAUR and STREA). These values suggested that the tendency to develop mastitis because of one of the two major pathogens partially relied on common genetic variation but it was not exactly the same (genetic correlation of 0.52 far from 1). Also considering the limits in ENV group definitions, the presence of STAUR and STREA resulted in this study in an antagonistic relationship with the joint infection due to ENV pathogens. The genetic correlations of STAUR and STREA with SCS were comparable (0.51 and 0.76 respectively), indicating that the presence of these two bacteria is generally genetically associated with an increase in SCS. Indirect genetic selection for mastitis resistance through SCS in milk could therefore result in a reduction of STREA and STAUR infections with a stronger effect on STREA than STAUR. In contrast, the genetic correlation between SCS and ENV was close to zero (-0.140 ±0.179) indicating that indirect selection for mastitis resistance using SCS would poorly affect ENV infections.

Table 1: Posterior mean (standard deviation) of heritabilities and of genetic correlations for SCS and specific pathogens (STAUR= *Staphylococcus aureus*, STREA= *Streptococcus agalactiae* and ENV=other pathogens in milk).[α]

Traits	SCS	ENV	STAUR	STREA
SCS	0.071(0.013)			
ENV	-0.140(0.179)	0.035(0.010)		
STAUR	0.513(0.150)	-0.283(0.194)	0.071(0.021)	
STREA	0.764(0.096)	-0.321(0.186)	0.520(0.189)	0.070(0.021)

[α]Heritabilities (standard deviation) on the diagonal and genetic correlations (standard deviation) below.

Conclusion

The pattern of genetic correlations between SCS and specific pathogens and the evidence of recursive effects from STAUR and STREA to SCS suggested that the worldwide common

genetic selection for mastitis resistance in dairy cattle based on the indirect trait SCS is promising in reducing the predisposition to udder infections by the major pathogens of STAUR and STREA. Nevertheless the same selection scheme, does not appear equally efficient in the reduction of ENV infection for the small recursive effect from ENV to SCS and for the genetic correlation, close to zero, between these two traits. A more detail analysis of this group of pathogens would help in design a clear pattern of association between SCS and pathogens.

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