# Genetic Parameters for Specific Pathogens Presence and Somatic Cell Scores in Valdostana Cattle Milk

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## Introduction

The bovine mastitis is an inflammation of the mammary gland that can be associated to infectious or non infectious etiology. The pathogens causing mastitis in dairy cattle can be classified in contagious or environmental (Blowey and Edmondson, 1995). *Staphylococcus aureus, Streptococcus dysgalactie* and *Streptococcus agalactie* are the contagious pathogens causing the largest proportion of mastitis cases. *Enterobacteriacae* as *Escherichia coli* and *Streptococcus uberi* represent the most common environmental pathogens causing udder infections (Bradley 2002; Pyörälä 2002; Zhao and Lacasse 2008). Mastitis with non-infectious etiology could be caused by mechanical or thermal traumas or chemical insults (Zhao and Lacasse 2008). About 20-35% of mastitis cases have an unknown etiology (Wellenberg *et al*., 2002).

Genetic selection is a long term strategy to control mastitis incidence. It results in a permanent change in the genetic resistance of the dairy herd (Shook, 1989), spread over all individuals of the population and cumulative over generations. Heritability estimated for clinical mastitis is generally low with values below 0.05 (Emanuelson *et al*., 1988; Weller *et al*., 1992; Mrode and Swanson, 1996). Selection to reduce the susceptibility to mastitis in dairy cows is often performed through indirect traits, normally with somatic cell count after the logarithmic transformation into somatic cell scores (**SCS**) (Wiggans and Shook, 1987). SCS have two main advantages: i) data collection is easy because it is integrated into the routine milk recording schemes; ii) the heritability for SCS is larger than for the direct measure of clinical mastitis (**CM**) with a genetic correlation between CM and SCS in average of 0.70 (for a review: Mrode and Swanson, 1996).

The incidence of specific pathogen in milk represents a further source of information that can be considered for genetic selection and therefore the estimation of genetic parameters is needed for breeding values predictions. Reported posterior means of heritabilities for pathogen specific incidence in mastitis, estimated using Gibbs Sampling procedures, are from 0.035 to 0.076 with high estimates for unspecific mastitis (0.109). Genetic correlations

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among pathogens specific varied between 0.45 and 0.77, suggesting that the presence of each specific pathogens in infected milk should be considered as a different trait (Sorensen *et al*., 2009).

The aim of this study is to estimate genetic parameters for the presence of specific pathogens in milk and for the level of SCS in milk of Valdostana cattle, an Italian dual purpose breed. Estimates obtained with linear (**LM**) and threshold (**ThrM**) models will be compared.

## Material and methods

All data were provided by the Valdostana Breeder Association and were collected on a herd basis from 2001 to 2008. All cows in herds with at least case of clinical mastitis were tested for bacteria presence. The data set included 28,063 records of 25,065 cows daughters of 2,607 sires and 18,267 dams. SCC was transformed into SCS (Wiggans and Shook, 1987). SCS averaged 2.65±2.09 with a skewness of 0.649 and a kurtosis of -0.038. Bacteriological analyses recorded the presence in milk (1) of *Staphylococcus aureus* (**STAUR**) in 22.51%, *Streptococcus agalactie* (**STREA**) in 16.10% and other pathogens in 50.12% (**MASENV**). The absence of the specific pathogen was recorded with 0. Pedigree of 5 generations of ancestors was extracted from the Italian Herd Book for a total of 54,650 animals.

Fixed effects were tested with the procedure GLM of SAS (V9.1.3, 2009) after the editing. The effects included were: combination of herd-year (5483 levels), days in milk (12 classes of 30 days), month of calving (12 levels), order of parity (5 levels: from 1 to 5 and more), and breed type (2 levels). Random effects considered in the models were additive genetic and permanent environmental effects. The same data were analyzed with two different models: LM and ThrM. In both cases the program THRGIBBS1F90 (Tsuruta, and Misztal, 2006) with Gibbs sampling for threshold-linear mixed models was used. A total of 100K iterations with a burn-in of 10K were performed. SCS were considered as continuous trait while STAUR, STREA and MASENV were continuous in the first model analyzed and threshold traits in ThrM.

## Results and discussion

Estimates of heritabilities, genetic and residual genetic correlations resulting from linear and threshold models are in Tables 1 and 2, respectively. SCS heritabilities (0.080 for LM and 0.083 for ThrM) and repeatabilities (0.147 for LM and 0.124 for ThrM) are consistent to the parameters reported by previous literature estimates (Shutz, et al., 1990; Mrode and Swanson, 1996; and Emanuelson, 1997).

Little genetic variation for pathogen specific incidence resulted with LM (heritabilities of 0.01 for STAUR and STREA and near zero for MASENV) reflecting the binary nature of pathogens traits. LM repeatabilities for pathogen incidence for STAUR (0.057), STREA (0.199) and MASENV (0.053) were lower than with ThrM (0.141, 0.252 and 0.140, respectively) suggesting a greater sub-estimation of genetic vs the phenotypic variances in binary traits. Genetic correlations obtained from LM were negative among specific

pathogens presences indicating that the presence of one microorganism would probably inhibit the presence of other pathogens in the same moment in infected milk. In contrast the positive genetic correlation between SCS and pathogens represents the genetic predisposition for an increase in SCS as the normal immune-defense of the udder against pathogen invasion. Residual correlations were smaller that genetics ones in all cases.

Genetic correlation estimated with the ThrM between STAUR and STREA was positive while the other genetic correlations among pathogens were negative. All genetic correlations among pathogens and SCS were positive confirming a common udder reaction to the two major pathogens considered but also the different effect in the SCS increase with any of the specific pathogen. The value of genetic correlation between SCS and MASENV estimated with ThrM was lower than with LM. This latter correlation seems to suggest that it could exists interaction between microorganism presences, as symbiosis or antagonism, affecting by consequence the increase in SCS associated, that it is not always so evident, with this large group of pathogens. Residual correlations were similar with LM models.

All heritabilities and genetic correlations were comparable with literature estimates. Although LM values here estimated were smaller than previous reported results ThrM heritabilities were higher and genetic correlations lower (De Haas *et al* 2002; Sorensen *et al*., 2009).

**Table 1: Posterior mean of heritabilities, genetic and residual correlation values for SCS and pathogen incidence (STAUR= *Staphylococcus aureus*, STREA= *Streptococcus agalactie* and MASENV=other pathogens in milk) resulting from the linear model.[α]**

| **LM** | SCS | STAUR | STREA | MASENV |
|---|---|---|---|---|
| SCS | **0.080** | 0.4273 | 0.5841 | 0.1868 |
| STAUR | 0.1016 | **0.008** | -0.2451 | -0.0811 |
| STREA | 0.1981 | 0.0552 | **0.010** | -0.2554 |
| MASENV | -0.0095 | -0.1214 | -0.2093 | **0.013** |

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

**Table 2: Posterior mean of heritabilities, genetic and residual correlations values for SCS and pathogens incidence (STAUR= *Staphylococcus aureus*, STREA= *Streptococcus agalactie* and MASENV=other pathogens in milk) resulting from the Threshold model.[α]**

| **ThrM** | SCS | STAUR | STREA | MASENV |
|---|---|---|---|---|
| SCS | **0.0828** | 0.4380 | 0.5730 | 0.0194 |
| STAUR | 0.1380 | **0.0398** | 0.4004 | -0.4492 |
| STREA | 0.2668 | 0.0805 | **0.0688** | -0.7078 |
| MASENV | -0.0189 | -0.0993 | -0.1311 | **0.0610** |

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

## Conclusion

Both models, linear and threshold were estimated genetic parameters for SCS and pathogen traits considered in this study consistent to values reported in literature. Nevertheless the binary natures of some traits, as the specific pathogen presence or absence in milk, require the use of ThrM. This is evident here in the smaller heritability values estimated with LM, for the difficulty of identifying variances for binary traits, when compared to ThrM estimates. Genetic correlation among SCS and both STAUR and STREA suggest that a genetic selection for mastitis resistance through the indirect trait of SCS would genetically reduce udder infection due to these microorganisms with a stronger effect on STREA than on STAUR. Genetic correlation between SCS and MASENV were not clear with values close to zero probably due to the large spectrum of microorganisms considered in the same group. A more detailed classification of this pathogen group is needed for better dissect trait variation in future analyses. Finally the joint use of bacteriological information from specific pathogens in milk and SCS would be of great value in genetic selection for mastitis resistance of Valdostana cattle breed, and in dairy cattle in general.

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