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Phenotypic and genetic analysis of udder health using SCC in Valle del Belice dairy sheep

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INTRODUCTION

Intramammary infections (IMI) are a complex of inflammatory diseases which are defined as an inflammation of the mammary gland resulting from the introduction and multiplication of pathogenic micro-organisms. Mastitis is one of the most frequent IMI affecting small dairy ruminants. IMI are mainly of economical, hygienic (consumption of dairy products) and legal importance in Europe (EU directives 46/92 and 71/94 defining the bacteriological quality of milk). Although management is the most effective way to prevent IMI, selection for IMI resistance is an alternative to be considered, at least to prevent any detrimental effect of milk yield on udder health. Direct selection against clinical mastitis is difficult because clinical mastitis is not widely recorded; on the contrary, somatic cell count (SCC) is promoted as selection criterion for mastitis resistance. However, the relationship between SCC and mastitis is far from clear. Some authors (Coffey et al., 1986; Kehrli and Shuster, 1994) were concerned by the recommendation of continuously decreasing SCC by selection and argued that this trend could impair the cow’s capacity for leukocyte recruitment and therefore its ability to respond to IMI. Animals with very low SCC would be more susceptible to mastitis. A punctual approach with a single threshold is a simple methodology which proposes the punctual or instantaneous discrimination between ‘healthy’ and ‘infected’ udders (Bergonier et al., 2003). In ewes, with the fluoro-optoelectronic method, single thresholds were proposed surprisingly ranging from 200,000 to 1.5x10⁶ cells/ml (Ftenakis 1996; Bergonier and Berthelot, 2003). The objective of this study was the phenotypic and genetic analysis of IMI estimates from SCC with a single trait threshold model.

MATERIAL AND METHODS

The dataset contained 2,475 first-lactation Valle del Belice ewes from 14 flocks recorded from 1998 to 2003. In total 116 sires with at least four daughters with a record were included in the pedigree file. All first-lactation test-day records were required to have SCC information. The average number of SCC test-day records per ewe was 7.62. For the threshold model, the cut-off value was fixed at...
750,000 cells/ml (Di Marco et al., 1997). Mastitis or IMI events were declared with a binary trait \( L \) set to one, when the SCC was higher than the cut-off value in one test-day record within lactation, while if SCC within lactation was still lower than the cut-off value the ewes were considered as 'doubtful or healthy' and the binary trait \( L \) was set to zero. Analyses of the risk factor for the binary IMI trait were based on logistic regression model using the SAS LOGISTIC procedure (SAS®, 2000). The overall significance of main effects in the model was assessed by a Wald chi-square test. This statistic takes the form of a squared ratio of an estimate to its standard error and asymptotically follows an approximate chi-square distribution with one degree of freedom. The odds ratio (OR) and OR 95% confidence interval were computed according to Hosmer and Lemeshow (1989). The OR measures how much more (or less) likely the outcome is among observations with a given level of a risk factor, compared with those with a reference level of the risk factor. A single trait threshold sire model was applied to the data using the ASReml package (Gilmour et al., 2002). The underlying liability of IMI in first-lactation was modeled as: 

\[
L_{\text{imm}} = \mu + \beta_1 \times (\text{season}) + \beta_2 \times \text{year} + \beta_3 \times \text{season} \times \text{year} + \epsilon \text{imm} ,
\]

where \( L \) is the IMI event with mean \( \mu \) for ewe \( m \), daughter of sire \( l \) (\( l = 1, ..., 116 \)) with random effect \( s_l \) and lambing \( i \) in herd \( h_i \), at season \( j \) (\( j = 1, ..., 3 \)) and year \( k \) (\( k = 1998, ..., 2003 \)). The binary trait is not normally distributed, so it is necessary to perform analyses that account for this distribution. Binary analyses were carried out, fitting a Generalized Linear Model, assuming a binomial distribution and using a logit function:

\[
\text{logit} \left( \pi(x) \right) = \beta_0 + \sum_{i=1}^{n} \beta_i x_i = X\beta \quad \text{where} \quad \logit \left( \pi(x) \right) = \ln \left( \frac{\pi}{1 - \pi} \right)
\]

and where \( \pi \) is the expected probability. The heritability estimated from normal analyses, treating the IMI trait as a continuous variable, was converted to the logit scale of liability with a simple relationship (Dempster and Lerner, 1950): 

\[
t_c = t_0 \left( \frac{1 - \pi}{\pi} \right) .
\]

\( t_0 \) is the heritability on the (0,1) scale and \( t_c \) is the heritability on the continuous scale (Falconer, 1989).

RESULTS AND CONCLUSIONS – The overall Wald chi-square test of the model showed a value of 336.9 with \( P<0.0001 \). The results of the logistic regression analysis of effects, investigating risk factors for “infected” and “doubtful or healthy” udder are presented in Table 1. All the effects in the model were significant. The herd effect was the major risk factor of IMI. The risk of culling for IMI increased for late season of lambing. In the Dec-Mar and Apr-May seasons of lambing, the IMI incidence was respectively 1.55 and 1.76 times that of the Jun-Nov season of lambing.

Table 1. Risk factors for the IMI trait in first lactation, expressed as odds ratio (OR) with 95% confidence interval (CI) relative to ewes lambing in 2003 and in season 3.

| Risk factor | Levels | \( P^{[1]} \) | \( OR^{[2]} \) | 95% CI |
|-------------|--------|-------------|--------|--------|
| Herd        |        | <0.001      |        |        |
| Year        |        | 0.0021      |        |        |
|             | 1998   | 0.90        | 0.48-1.67 |
|             | 1999   | 0.55*       | 0.34-0.90 |
|             | 2000   | 0.48*       | 0.30-0.77 |
|             | 2001   | 0.54*       | 0.34-0.87 |
|             | 2002   | 0.80        | 0.49-1.32 |
|             | 2003   | 1           | -       |
| Season      |        | 0.0005      |        |        |
|             | Dec-Mar| 1.55*       | 1.23-1.96 |
|             | Apr-May| 1.76*       | 1.17-2.63 |
|             | Jun-Nov| 1           | -       |

\[\text{[1]}\ P = \text{Global significance of variable (Wald statistics).} \]

\[\text{[2]}\ \text{OR significantly different from 1.0 (}P<0.05\text{) are identified by an asterisk.} \]
The effect of year of lambing showed lower risk for the IMI trait than what has been reported by Barillet et al. (2001). The OR of the years 1999 to 2001 indicate significantly lower risk than 2003, whereas Dec-May had higher risk as Jun-Nov. The genetic variance was equal to 0.095 and heritability for IMI was 0.16±0.072. This value was higher than those reported by Chang et al. (2004) in Norwegian cattle ranged from 0.03 to 0.11 and Heringstad et al. (2003) from 0.06 to 0.07. The heritability calculated from logit analyses was very close to the expected value of 0.14 calculated with Dempster and Lerner’s formula (1950). The results of this paper must be interpreted with caution. Only with breeding using information on clinical mastitis, one is selecting for the resultant of biological processes that improve mastitis resistance. With SCC, the situation is different: a high value is indicative of a diseased udder while a low value is not necessarily an indicator of a healthy udder. This is because a steady reduction of SCC by breeding may impair the innate immune system. Before relying too much on SCC in breeding programmes, a thorough examination of the linearity of the relationship between IMI and low levels of SCC should be carried out.

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