

Estimation of (co)variance components of nematode parasites resistance and somatic cell count in dairy sheep

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ABSTRACT - Nematode parasites and mastitis are the major animal health constraints in sheep. The aim of this study was estimating the genetic (co)variances of nematode parasites resistance and somatic cell count in dairy sheep. From 2000 to 2008, Somatic Cell Score (SCS) and Faecal Egg Count (FEC) records were available on an experimental population consisting of 949 backcross ewes and 806 their daughters. Data were processed independently for each subpopulation in order to adjust for specific environmental effects and to obtain lactation records for both traits to be used in the genetic analysis. **Variance components** estimation was performed by using the REML method applied to a bi-trait repeatability animal model. Heritabilities of lactation SCS (LSCS) and FEC were 0.19 and 0.16. Genetic correlation was 0.21, whereas phenotypic correlation was 0.01. The estimated heritabilities confirm that both traits could be selected by the classical quantitative approach. **The genetic correlation estimate between LSCS and FEC suggests** that selection for one of the two traits would not have any detrimental effect on the other one.

Key words: Genetic parameters, SCS, Nematode parasite resistance.

Introduction – Nematode parasites and mastitis are the major animal health constraints in sheep breeding also with a great impact upon productivity. In Italy, the prevalence of gastro-intestinal (GI) parasites, primarily affecting growing lambs and lactating ewes, ranges from 32% to 94% (Garippa *et al.*, 2008). The incidence of clinical mastitis in small ruminants is generally lower than 5%, whereas the prevalence of subclinical mastitis ranges from 10 to 50% (Bergonier *et al.*, 2003). Evidence for genetic variation amongst sheep in their resistance to GI nematode parasites is well documented in many breeds (Bishop and Morris, 2007). Somatic Cell Count (SCC) has been used as an indicator to enable selection for increased resistance to subclinical mastitis (Barillet, 2007). Breeding programs selecting commercial animals for enhanced resistance against nematode parasites are used for meat and wool breeds (Nieuwhof and Evans, 2003). In the French Lacaune breeding scheme, SCC is used as selection criterion for resistance to mastitis (Rupp *et al.*, 2002). Implementation of nematode resistance and SCC in breeding programs requires the knowledge of the relationships between these major health traits. The aim of this study was estimating the genetic (co)variances of nematode parasites resistance and SCC in dairy sheep.

Material and methods – From 2000 to 2008, measurements were recorded on an experimental population consisting of 949 Sardinian x Lacaune backcross (BC) ewes and 806 their daughters procreated by mating the BC ewes with Sardinian rams. SCC (cells/ml) were measured by a Fossomatic cell counter from milk samples bimonthly collected at a.m. and p.m. milking. Daily SCC were computed as the arithmetic mean of evening and morning values. Somatic Cell Score (SCS) was obtained with a log-

transformation of test-day SCC. Lactation SCS (LSCS) was computed as the arithmetic mean of test-day SCS per lactation. Nematode resistance was measured by Faecal Egg Count (FEC) under natural conditions of infection. Periodically, a sample of around 50 animals were monitored in order to evaluate the percentage of infected animals and to decide whether or not to sample the whole flock. After that, FEC were measured on the whole flock 1 to 3 times per year, more frequently in September and July. Faeces were processed by floatation in saturated salt solution in a McMaster slide and the eggs counted (Raynaud, 1970). **FEC measurements were log-transformed prior to further analysis. Individual lnFEC were considered pertaining to a given parity when they were realized by the month of September following the dry-off (July).** Descriptive statistics of analysed phenotypes are reported in Table 1.

Table 1. Means and standard deviations per parity of the two traits for the backcross (BC) and the progeny populations.

Parity	N	Lactation Somatic Cell Score $\text{Log}_2(\text{SCC}[\text{cells/ml}]/100)+3$			N	Faecal Egg Count $\text{Ln}(\text{FEC}[\text{eggs number}]+14)$		
		BC	N	Progeny		BC	N	Progeny
1 st	880	3.73±0.98	712	3.60±1.05	949	4.93±1.26	806	5.13±1.26
2 nd	845	4.33±1.15	726	4.00±1.23	910	4.87±1.15	714	4.70±1.26
3 rd	782	4.98±1.28	654	4.46±1.32	874	4.27±1.20	239	4.61±1.36
4 th	674	5.68±1.41	607	4.84±1.35	734	3.61±1.03	390	4.81±1.40

All ewes were bred according to a particular management depending on the specific experimental needs. The main difference was that BC ewes were contemporary, whereas the progeny's flock was constituted by three age classes. Thus, data were first processed independently. To produce LSCS and lactation lnFEC for the genetic analyses, data were analysed using a repeated measures model including fixed effects specific of each population and the random individual effect. The fixed effects used for LSCS were year-group of management for BC and year-parity-age at lambing for the progeny, whereas for lnFEC were the sampling date and parity for both populations. Lactation phenotypes for the genetic analysis were calculated summing the individual solutions to the corresponding residuals. When more than one sampling date occurred during the lactation, residuals were averaged. Finally, 5,880 LSCS records and 5,616 lactation lnFEC records, corresponding to 1,673 animals for LSCS and 1,768 animals for lnFEC, were retained. Variance components estimation was performed by using the REML method applied to a bi-trait repeatability animal model. The pedigree file including 4,838 individuals, born between 1967 and 2004, was set up using all available relationships between animals.

Results and conclusions – In each population, an increase of LSCS according to parity was observed (Table 1) in agreement with Gonzalo *et al.* (1994). Concerning lnFEC, first lactation ewes showed higher values than following lactations (Table 1). The mechanism of acquired immunity against nematodes underlined by Stear *et al.* (1999) could explain this trend, already reported also for lactating ewes (Bishop and Stear, 2001). Heritability and repeatability estimates for each trait are given in Table 2. Heritability of LSCS was 0.19, slightly higher than estimates found in literature (El-Saied *et al.*, 1999; Rupp *et al.*, 2002; Serrano *et al.*, 2003).

Heritability of lnFEC was 0.16 that is in the range of values found in literature for meat and wool breeds (Bishop and Stear, 2001). In our study variance values could be inflated by other sources of covariation between daughters of the same sire. In fact, the BC population was created by mating each sire with ewes homogeneous for age and flock of origin and each family was managed in a homogeneous way. However, this fact is likely to affect mainly production traits than health traits. Repeatability of LSCS was in the range of values reported by Serrano *et al.* (2003). Repeatability of lnFEC was slightly higher than

Table 2. Estimates of heritability and, in bracket, repeatability (on the diagonal), genetic (above the diagonal) and phenotypic (below the diagonal) correlations between LSCS and lnFEC and relative standard errors.

	LSCS	Ln(FEC+14)
LSCS	0.19±0.04 (0.48±0.01)	0.21±0.16
Ln(FEC+14)	-0.01±0.02	0.16±0.03 (0.29±0.02)

values found by Bishop and Stear (2001) in lactating meat ewes (0.23). For LSCS, the high amount of variance due to permanent environment would be related to chronic intramammary infections. As a whole, the large difference between repeatabilities and heritabilities could indicate that part of the genetic variance has been estimated as permanent environmental variance due to the limited number of ancestors' records. **Genetic correlation between LSCS and lnFEC was moderately favourable with a large standard error.** The phenotypic correlation was close to 0 and not significant. The estimated heritabilities confirm that both traits could be selected by the classical quantitative approach. **The genetic correlation estimate between LSCS and lnFEC suggests that selection for one of the two traits would**

not have any detrimental effect on the other one. Further analyses on more structured population will be useful to better precise the genetic relationships between the two traits. Moreover, further indications will derive from several ongoing QTL detection projects on both traits (Rupp *et al.*, 2003; Moreno *et al.*, 2006).

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REFERENCES – Barillet, F., 2007. Genetic improvement for dairy production in sheep and goats. *Small Rumin. Res.* 70:60-75. **Bergonier, D.**, de Crémoux, R., Rupp, R., Lagriffoul, G., Berthelot, X., 2003. Mastitis of dairy small ruminants. *Vet. Res.* 34:689-716. **Bishop, S.C.**, Stear, M.J., 2001. Inheritance of faecal egg counts during early lactation in Scottish Blackface ewes facing mixed, natural nematode infections. *Anim. Sci.* 73:389-395. **Bishop, S.C.**, Morris, C.A., 2007. Genetics of disease resistance in sheep and goats. *Small Rumin. Res.* 70:48-59. **El-Saied, U.M.**, Carriedo, J.A., De La Fuente, L.F., 1999. **Genetic parameters of lactation cell counts and milk and protein yields in dairy ewes.** *J. Dairy Sci.* 82:639-644. **Garippa, G.**, Bufano, G., Caroli, A., Carta, A., Cringoli, G., Nardo, F.E., Filippini, G., Leori, S.G., Moniello, G., Ronchi, B., 2008. Realtà e prospettive dell'allevamento dei piccoli ruminanti in Italia. *Proc. 18th SIPAOC*, pp. 40-43. **Gonzalo, C.**, Carriedo, J.A., Baro, J.A., San Primitivo, F., 1994. **Factors influencing variation of test day milk yield, somatic cell count, fat, and protein in dairy sheep.** *J. Dairy Sci.* 77:1537-1542. **Moreno, C.R.**, Gruner, L., Scala, A., Mura, L., Schibler, L., Amigues, Y., Sechi, T., Jacquiet, P., François, D., Sechi, S., Roig, A., Casu, Sara, Barillet, F., Brunel, J.C., Bouix, J., Carta, A., Rupp, R., 2006. QTL for resistance to internal parasites in two designs based on natural and experimental condition of infection. *Proc. 8th WCGALP, Communication 15-05.* **Nieuwhof, G.J.**, Evans, J.L., 2003. Inclusions of selection for nematode resistance in British sheep reference schemes. *Options Méditerranée.* 55:47-54. **Raynaud, J.P.**, 1970. Etude de l'efficacité d'une technique de coproscopie quantitative pour le diagnostic de routine et le contrôle des infestations parasitaires des bovins, ovins, équins et porcins. *Ann. Parasitol. Hum. Comp.* 45:321-342. **Rupp, R.**, Boichard, D., Barbat, A., Astruc, J.M., Lagriffoul, G., Barillet, F., 2002. **Selection for mastitis resistance in French dairy sheep.** *Proc. 7th WCGALP, Communication 09-28.* **Rupp, R.**, Schibler, L., Cribiu, E., Amigues, Y., Boscher, M.Y., Mura, L., Sechi, T., Fraghi, A., Casu, Sara, Barillet, F., Carta, A., 2003. Evidence of chromosomal regions controlling somatic cell counts in dairy sheep from two QTL detection projects. *Proc. Int. Workshop on Major Genes and QTL in Sheep and Goats. INRA. Toulouse. France, CD-ROM n° 2-32.* **Serrano, M.**, Perez-Guzman, M.D., Montoro, V., Jurado, J.J., 2003. Genetic analysis of somatic cell count and milk traits in Manchega ewes: mean lactation and test-day approaches. *Livest. Prod. Sci.* 84:1-10. **Stear, M.J.**, Strain, S. Bishop, S.C., 1999. Mechanisms underlying resistance to nematode infection. *Int. J. Paras.* 29:51-56.