Estimation of Variance – Covariance Component for Somatic Cell Score in Iranian Holstein Cows

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The objective of this study was estimation of variance - covariance component for somatic cell score (LSCS, the lactation mean of the natural log of test- day somatic cell count) of Iranian Holstein across parities. Test day of somatic cell count for first, second and third parity that collected from 1993 to 2010 by Animal Breeding Center of Iran, were used. Each LSCS in the parity was considered as separate trait. Multi trait analysis was performed by ASReml software. Mean of LSCS for first, second and third parity was 7.78, 8.22 and 8.47. The heritability estimates for parities varied form 0.05 - 0.09. Genetic correlations between parities were higher than 0.90 but environmental correlations between parities were in range of 0.11 to 0.21. Genetic correlations between parity demonstrate that genes that control LSCS in parities are same and selection animal for LSCS in first parity would have affected LSCS in other parity.

Key Words: Somatic cell score, Genetic correlation, Iranian Holstein

INTRODUCTION

Profitability of a dairy herd is related to incomes and costs. Incomes included milk production and livestock sales. Factors that effect on costs of production included feed costs, fertility and health costs. Udder health effects both of incomes and cost (Ma et al., 2000). Mastitis, an inflammation of mammary gland mostly caused by microbial infection, is one of the most frequent, complex and costly diseases of dairy cattle (JiaZhong et al., 2010). This inflammation of the mammary gland, usually in response to invasive agents, can be determined by an increase in somatic cell count (SCC) in milk (Rodriguez-Zas et al., 2000). Mastitis includes clinical and subclinical infection. The economic costs of mastitis include reduced milk production, discarded milk, increased culling, increased costs of health care and labor, and reduced milk quality. In addition, mastitis contributes to consumer concerns regarding animal welfare. Strategies to control mastitis include preventive health care, hygiene, veterinary remedies, and genetic selection (Koldeweij, et al., 1999).

Direct measurement of clinical mastitis incidence is costly and difficult to implement in national recording schemes(Heringstad et al., 2000). Somatic cell count, generally log-transformed into SCS, is used for indirect selection for mastitis resistance in many formal dairy selection programs (Interbull, 2008). Heritability of SCS is greater than heritability of mastitis incidence, and genetic correlation between SCS and mastitis incidence is large (Heringstad et al., 2000). The estimates of genetic correlation between SCS and clinical mastitis were around 0.70 for all lactations (Koivula et al., 2005).

The SCC in raw milkis generally considered to be an important index for mastitis detection and udder health in dairy farming. SCC in milk from healthy udders varies between 50 and 200 thousands cells mL^{-1}, depending on the age of cows (Smith, 1996). While cows with subclinical mastitis can excrete up to a few million cells per milliliter sometimes, excretion is usually in the range from 200 to 500 thousands cells mL^{-1} (Reneau, 1986). In the past decades, the researches on variation ofSCC and relationship of SCC and milk production

traits have been paid much attention worldwide in dairy cattle.The objective of the present study was to estimate genetic parameters for SCS in across parities.

MATERIALS AND METHODS

Records of Somatic cell score that is collected by Animal Breeding Center of Iran from 1993 to 2010 were used for this study. To achieve an approximate normal distribution of test-day records, these were loge-transformed to somatic cell score and lactation mean SCS was calculated by the arithmetic mean of all observation per cows with following equation (Schukken et al. 1992; Odegard et al., 2003):

$$LSCS = \frac{1}{n}\sum_{1}^{n}\left(\log_e\left(\frac{SCC}{1000}cells/ml\right)+3\right)$$

In the equation, n is number of records for animal in parity and SCC is somatic cell score. Descriptive statistical of records after edition was shown in Table1. Genetic parameters and genetic and phenotypic Variance-covariance components for traits were estimated by multitrait animal model as following model:

y =Xb+ Zu+e

Assumptions of model were:

$E\begin{bmatrix} y_i \\ u_i \\ e_i \end{bmatrix} = \begin{bmatrix} Xb_i \\ 0 \\ 0 \end{bmatrix}$; var ($y_i$)= $ZG_{ii}Z'+R_{ii}$; cov (y_i ,y_j) =$ZG_{ij}Z'+R_{ij}$

M= $var\begin{bmatrix} u_i \\ u_j \\ e_i \\ e_j \end{bmatrix} = \begin{bmatrix} G_{ii} & G_{ij} & 0 & 0 \\ G_{ji} & G_{jj} & 0 & 0 \\ 0 & 0 & R_{ii} & R_{ij} \\ 0 & 0 & R_{ji} & R_{jj} \end{bmatrix}$; G_{ii}=A□$^2_{u_i}$; R_{ii}=I□$^2_{e_i}$;G_{ij}=G_{ji}=A□$_{u_i,u_j}$; R_{ij} = R_{ji}= I□$_{e_i,e_j}$

In the model:

Y is the column vector of the observations of traits, b is the column vector of fixed effects (Herd- year- season of calving, age at first calving, and quadratic effect of age), u is a column vector of additive genetic effects, and e is a vector of residualseffects. The matrixes X, Z arethe incidence matrix for the fixed effects and observations animals. Records of animal in different parity were supposed as separate trait. Records were analyzed with ASReml software (Gilmour et al. 2000)

RESULTS AND DISCUSSION

Table 1 shows descriptive statistical for records of LSCS in three parity for Iranian Holstein cows. The number of records in second and third parity is lower than first parity. Mean of LSCS for first, second and third parity was 7.78, 8.22 and 8.47, respectively. Mean of LSCS increased with parity. Maximum of CV for LSCS was in first parity. The increasing of mean LSCS in third parity was suggested than animal in third parity were susceptible for Mastitis disease.

Mean of LSCS in this study was higher than another studies (Ikonen et al., 2004; Wellar and Ezra, 2004; Rekik et al., 2008; Samore et al., 2008; Mostert et al., 2006; JiaZhong et al., 2010). Increase in LSCS with parity was reported for South African, Italian, Tunisian and Chinese Holsteins (Rekik et al., 2008; Samore et al., 2008; Mostert et al., 2006; JiaZhong et al., 2010).

Table 1 descriptive statistical for records

Traits	Rcord.no	Pedigree	Mean	SD**	Min	Max	CV*
LSCS1	150946	224666	7.78	1.25	3.4	14.18	16.07
LSCS2	78617	224666	8.22	1.13	3.00	13.06	13.75
LSCS3	39144	224666	8.47	1.13	3.00	12.60	13.34

** Standard Deviation
* Coefficient of Variation

Genetic variance for LSCS in third parity was higher than in first parity (Table 2). For these traits, environmental covariance between traits was higher than genetic covariance. These result showed that LSCS was affected by environmental effect and genetic is effective lowly.

The heritability estimates for LSCS1, LSCS2 and LSCS3 were 0.05, 0.09 and 0.09, respectively (Table 3). This is lower than Smore et al. (2008), Wellar and Ezra. (2004), Montaldo et al. (2010), Odegard et al. (2003) reports but close other reports (Mostert et al. (2006). Reents et al. (1995) concluded from various studies that the heritability for SCS ranges from 10-15%.

Table 2 geneticVariance (diagonal) and genetic (above the diagonal) and environmental (below) covariance

	LSCS1	LSCS2	LSCS3
LSCS1	0.050	0.060	0.063
LSCS2	0.14	0.089	0.092
LSCS3	0.11	0.19	0.095

Table 3 heritability (on diagonal), genetic (above the diagonal), and environmental (below) correlation coefficients for LSCS in different parity

	LSCS1	LSCS2	LSCS3
LSCS1	0.05	0.90	0.91
LSCS2	0.15	0.09	0.99
LSCS3	0.11	0.21	0.09

Genetic correlations between parity were in range of 0.90 to 0.99 and for environmental correlation were 0.11 to 0.21. The genetic correlationsamong parities were higher than the environmentalcorrelations. Genetic correlations between parities decreasedwith increase in the difference in parity number,but all were greater than 0.90. Genetic correlations between parity in this study were higher than reports for Israeli and Italian Holstein but the environmental correlation were low (Wellar and Ezra, 2004; Samore et al., 2002).The reason of different in result with another research could be due to of data structure, production level and model that used for analysis.

In conclusion, estimates of heritability for LSCS suggested that for improve of LSCS in Iranian Holstein must be improved environmental condition. Genetic correlation between

parity was shown that these traits were affected by common gens and selection as first parity improved LSCS in another parity.

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REFRENCES

JiaZhong, G.,L. Xiao- Lin, X. A- juin and X.Zhi. 2010. Relationship of somatic cell count with milk yield and composition in Chinese Holstein population. Agri. Sci. China. 9 (10):1492-1496.

Weller, J. I and E. Ezra.2004. Genetic analysis of the Israeli Holstein dairy cattle population for production and nonproduction traits with a multitraitanimal model. J. Dairy Sci. 87: 1519-1527.

Schukken, Y. H., K. E. Leslie, A. J. Weersink andS. W. Martin. 1992. Ontario bulk milk somatic cell count program. II. Population dynamics of bulk milk somatic cell counts. J. Dairy Sci. 75: 3359-3366.

Samoré, A. B., P. Boettcher,J.Jamrozik, A. Bagnato and A. F. Groen. 2002. Genetic parameters for production traits and somatic cell scores estimated with a multiple trait random regression model in Italian Holsteins. Proc. 7th World Congr.Genettic Applied Livestock Production. Montpellier, France. XXIX:63–66.

Samore, A. B., A. F. Groen,P. J. Boettcher, J. Jamrozik, F. Canavesi, and A. Banganato. 2008. Genetic correlation patterns between somatic cell score and protein yield in the Italian Holstein-Friesian population. J. Dairy Sci. 91: 4013-4021.

Rekik, B., N.Ajili, H. Belhani, A. Ben Gara, and H. Rouissi. 2008. Effect of somatic cell count on milk and protein yields and female fertility in Tunisian Holstein dairy cows. Livest. Sci. 11 (6): 309 – 317.

Reents, R., J.C.M.Dekkers, and L.R. Schaeffer. 1995. Genetic evaluation for somatic cell score with a testday model for multiple lactations. J. Dairy Sci. 78, 2858-2870.

Odegard, J., K. Gunnar and H.Bjőrg. 2003. Variance components and genetic trend for somatic cell count in Norwegian Cattle. Livest. Prod. Sci. 79: 135–144.

Mostert, B. E., H. E. Theron, F. H. J. Kanfer, and E. Van Marle-koster. 2006. Test-day models for South African dairy cattle for participation in international evaluations. South African J. Anim. Sci. 36(1): 58-70.

Ikonen, T., S.Morri, A.-M.Tyriseva,O. Ruottinen and M. Ojala. 2004. Genetic and phenotypic correlations between milk coagulation properties, milk production traits, somatic cell count, casein content, and pH of milk. J. Dairy Sci. 87: 458-467.

Gilmour, A.R., B.R.Cullis, S.J. Welham and R. Thompson. 2000. ASREML Reference manual. ftp.res.bbsrc.ac.uk in pub/aar.

Ma, Y., C. Ryan, D.M. Barbano, D.M. Galton, M.A. Rudan, and K.J. Boor. 2000. Effects of somatic cell count on quality and shelf-life of pasteurized fluid milk. J. Dairy Sci. 83:264-274.

Montaldo, H. H., H. Castillo-Juárez., E. G. Cienfuegos-Riva and F. J. Ruiz-López. 2010. Genetic and environmental parameters for milk production, udder health, and fertility traits in Mexican Holstein cows. J. Dairy Sci. 93:2168–2175.

Heringstad, B., G. Klemetsdal and J. Ruane. 2000. Selection for mastitis resistance in dairy cattle: A review with focus on the situation in the Nordic countries. Livest. Prod. Sci. 64:95–106.

Interbull. 2008. description of national genetic evaluation systems for dairy cattle traits as applied in different Interbull member countries. http://ww.interbull.slu.se/national_ges_info2/framesida-ges.htm.Accessed June 5, 2008.

Koivula, M., E. A. Mäntysaari, E. Negussie and T. Serenius. 2005. Genetic and phenotipic relationships among milk yield and somatic cell count before and after clinical mastitis. J. Dairy Sci. 88:827–833.

Reneau, J K. 1986. Effective use of dairy herd improvement somatic cell couns in mastitis control.J. Dairy Sci. 69: 1708-1720.

Smith, K L. 1996. Standards for somatic cells in milk: physiological and regulatory. IDF Mastitis Newsletter. 21: 7-9.

Koldeweij, E., U. Emanuelson,L.Janson. 1999. Relation of milk production loss to milk somatic cell count. Acta Vet.Scand. 40: 47-56.

Rodriguez-Zas, S. L., D. Dianola and G. E. Shook. 2000. Evaluation of models for somatic cell score lactation patterns in Holsteins. Livest. Prod. Sci. 67:19-30.