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Genetic correlation of average somatic cell score at different stages of lactation with milk yield and composition in Holstein cows

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Summary

Genetic and non-genetic correlations of the average somatic cell score (SCS) at different stages of lactation (100, 150, 200, 305 and 365 days in milk) with lactation performance were estimated based on the first lactation records of Iranian Holstein calving during 2000 to 2009. (Co) variance components were estimated using derivative-free REML based on multiple traits animal models. The estimated genetic correlations between average of first 100 days in milk SCS (SCS_{100}) and $milk_{100}$, fat_{100}, $milk_{305}$, fat_{305} and $protein_{305}$ were 0.14, 0.19, 0.11, 0.15 and 0.17, respectively. The corresponding environmental correlations were -0.13, -0.03, -0.18, -0.10 and -0.13, respectively. The estimated genetic correlations of the average SCS during the first 150 days in milk (SCS_{150}) with $milk_{100}$, fat_{100}, $milk_{305}$, fat_{305} and $protein_{305}$ were 0.14, 0.19, 0.11, 0.15 and 0.17, respectively. The corresponding environmental correlations were -0.13, -0.03, -0.18, -0.10 and -0.13, respectively. The estimated genetic correlations of the average SCS during 305 days in milk with milk, fat and protein yield, and fat and protein percentages were 0.12, 0.11, 0.18, -0.05 and -0.08, respectively. The estimated genetic correlation between SCS_{100} or SCS_{150} with average SCS over longer stages of lactations (SCS_{305} and SCS_{365}) was 0.99 ± 0.01. These results indicate that recording SCS data over shorter period of lactation is an alternative approach to reduce the costs of SCS data collection and therefore to expand the SCS recording in the country by increasing both the number of herds and cows involved.

Key words: Somatic cell score, Milk composition, Genetic correlation, Holstein cow

Introduction

Somatic cell score (SCS) is an important trait that considerably affects the profitability of dairy industry. High SCS is not only associated with udder health and milk loss, but also affects the casein content and processing ability of milk, and shelf-life of the pasteurized milk. SCS also negatively affects the fertility (Buch and Norberg, 2008; Rekik *et al*., 2008) and longevity (Sewalem *et al*., 2006) of dairy cows. There is a strong genetic relationship between SCS and all types of mastitis. Genetic correlation between SCS and mastitis is reported to be in the range of 0.53 to 0.91 (Ødegard *et al*., 2004; De Haas *et al*., 2008; Vallimont *et al*., 2009).

Increased resistance to mastitis has been widely studied based on indirect selection on SCS in many countries (Windig *et al*., 2010; Interbull, 2011). Although in some cases the heritability estimates for SCS were low (Mostert *et al*., 2004; Ptak *et al*., 2009), in most populations the estimates were in the range of 0.15 to 0.25 for different definition of SCS (Muir *et al*., 2007; Samoré *et al*., 2008; Bohmanova *et al*., 2009; Miglior *et al*., 2009). The heritability of SCS was significantly greater than the heritability estimates of 0.02 to 0.05 reported for mastitis (Rupp and Boichard, 1999; Carlen *et al*., 2004; Ødegard *et al*., 2004; Bloemhof *et al*., 2009), implying that SCS could be an indirect selection criteria for improvement of genetic resistance of cows to mastitis.

Extreme emphasis on selection for increasing yield exerted a negative effect on

udder health. Several studies reported moderate but unfavorable genetic correlation between milk yield and SCS (Carlen *et al*., 2004; Koivula *et al*., 2005; Samoré *et al*., 2008, 2010). On the other hand, increased level of SCS has a negative effect on subsequent milk production. Direct losses in daily milk yield due to elevated SCS are different depending on population, breed, stage of lactation, and level of production, but reported to be in the range of 0.55 to 2.45 kg per one unit increase in SCS (Dürr *et al*., 2008; Rekik *et al*., 2008). A negative phenotypic correlation of -0.14 between SCS and milk yield was reported in Finnish Ayrshire, while the genetic correlation between clinical mastitis and test day milk yield was 0.38 to 0.58 (Koivula *et al*., 2005).

The objective of the present study was to determine the genetic and non-genetic relationships among SCS averaged over different stages of lactation, milk yield and composition in Holstein cows of Iran. The possibility of recording SCS data only on short lactation period, i.e. in the first 150 DIM, was investigated in order to eventually reduce the costs of SCS data collection and therefore to expand the SCS recording of Holstein cows of Iran.

Materials and Methods

First lactation records of Iranian Holstein dairy cows, in 305 herds calved between 2000 and 2009 were obtained from the Animal Breeding Center. All herds were under official pedigree and performance recording. Initial dataset consisted of 643335 test day records of SCS on 91116 cows. The traits considered were the average SCS during 100, 150, 200, 305 and 365 days of lactation, milk yield, fat yield and percentage, protein yield and percentage, adjusted for 305 DIM as well as milk and fat yield of the first 100 and 200 days in milk. Data of cows from herds with at least 20 observations for each trait were used. Records associated with incorrect calving date and age at first calving outside 18-40 months were excluded from the analysis. Only sires with at least 10 daughters in 5 different herds were considered. The pedigree file consisted of the animal in data file plus 3 ancestor generations. Total number of animals included in the pedigree was 132656 to 160036 in different sets of analyses. As the distribution of SCC is highly skewed, in most cases, log-transformed is used for the analyses (Ali and Shook, 1980). Somatic cell count data were transformed to SCS based on the following formula:

$$SCS = \ln(SCC/100000) + 3$$

where,

ln: The natural logarithm of each test day record

Because of the dilution effect of milk yield on the somatic cell count (Green *et al*., 2006), the adjusted mean of SCS was calculated (Jones *et al*., 1984), taking into account the corresponding milk yield of each test day:

$$Adj\_SCS_i = \frac{\sum_{i=1}^{n} (m_i SCS_i)}{\sum_{i=1}^{n} m_i}$$

where,

Adj-SCS_i: The weighted average of SCS during different stages of lactation

SCS_i: Monthly SCS

m_i: The corresponding test day milk production of 1st month of recording

Observations between days 5 to 365 of lactation were used. A minimum of 2, 3, 3, 4 and 4 test day records were required to calculate SCS_{100}, SCS_{150}, SCS_{200}, SCS_{305} and SCS_{365}, respectively.

Variance components were estimated by restricted maximum likelihood (REML) based on 4-traits animal models, using VCE6 software (Kovac *et al*., 2008). The following model was used for the analyses of yield traits:

$$y_{ijk} = \mu + HYS_i + \alpha(age - \overline{age}) + \beta(age - \overline{age})^2 + a_j + e_{ijk}$$

where,

y_{ijk}: The phenotypic observations for SCS and production traits

μ: Overall mean

HYS_i: The effect of herd-year-season of calving

a_j: Random additive effect of animals

α and β: The linear and quadratic regression coefficients of the age at calving

e_{ijk}: Random residual

The model for the analysis of SCS was the same as yield traits but only consisted of the linear covariate of the age at calving.

Results

Characteristics of the data for studied traits are shown in Table 1. Tables 2 and 3 show the estimated heritabilities, genetic and non-genetic correlations of SCS with lactation performance. The protein content had stronger genetic correlations with SCS but the protein percentage showed low genetic correlations with different SCS traits. The environmental correlation between protein yield and all SCS traits were negative. On the other hand, positive but similar in magnitude, environmental correlations were observed between the protein percentage and SCSs. Negative but low genetic correlations were estimated between fat percentage and all SCS traits. On the other hand, the phenotypic and environmental correlations between the fat percentage and SCS at different stages of lactation were positive but still relatively weak.

The estimated heritability, and

Table 1: Summary statistics of SCS, milk yield and composition

Trait	No. of records	Mean	SD	Min	Max
SCS_{365}	63318	2.704	0.963	0.318	6.804
SCS_{305}	60633	2.678	0.968	0.318	6.804
SCS_{200}	59121	2.626	1.008	0.318	6.912
SCS_{150}	49269	2.597	1.020	0.258	6.985
SCS_{100}	48499	2.598	1.084	0.098	7.072
$Milk_{305}$ (kg)	59711	7654.0	1372.8	1836.0	14180.0
Fat_{305} (kg)	54205	252.6	50.55	60.57	503.93
$Prot_{305}$ (kg)	53707	232.9	39.94	61.78	423.1
$Fatper_{305}$ (%)	54201	3.33	0.45	1.40	6.40
$Protper_{305}$ (%)	53707	3.06	0.22	1.70	5.26
$Milk_{200}$ (kg)	51508	5220.5	886.5	1279.3	8717.9
Fat_{200} (kg)	47029	170.7	35.1	39.0	369.0
$Milk_{100}$ (kg)	47731	2560.4	462.7	442.0	4976.8
Fat_{100} (kg)	44527	84.9	20.2	16.4	241.4

Table 2: Heritability (h^2), genetic (r_G), environmental (r_E) and phenotypic (r_P) correlations between yield traits and average SCS of 100 and 150 DIM

Traits	h^2	SCS_{100}			SCS_{150}		
		r_G	r_E	r_P	r_G	r_E	r_P
$Milk_{305}$	0.29 ± 0.01	0.10 ± 0.06	-0.16 ± 0.01	-0.12	0.11 ± 0.06	-0.18 ± 0.01	-0.13
Fat_{305}	0.17 ± 0.01	0.14 ± 0.07	-0.09 ±0.01	-0.07	0.15 ± 0.07	-0.10 ± 0.01	-0.08
$Prot_{305}$	0.24 ± 0.01	0.17 ± 0.07	-0.13 ± 0.01	0.09	0.17 ± 0.06	-0.13 ± 0.01	-0.10
$Fatp_{305}$	0.27 ± 0.01	-0.05 ± 0.06	0.11± 0.01	0.08	-0.04 ± 0.06	0.10 ± 0.01	0.08
$Prop_{305}$	0.34 ± 0.01	0.09 ± 0.06	0.15 ± 0.10	0.18	0.08 ± 0.06	0.17 ± 0.10	0.15
$Milk_{100}$	0.19 ± 0.01	0.14 ± 0.07	-0.13 ± 0.01	-0.13	0.14 ± 0.07	-0.13 ± 0.01	-0.13
Fat_{100}	0.11 ± 0.01	0.17 ± 0.07	-0.02 ± 0.01	-0.01	0.19 ± 0.07	-0.03 ± 0.01	-0.02
$Milk_{200}$	0.27 ± 0.01	0.13 ± 0.07	-0.15 ± 0.01	-0.11	0.13 ± 0.06	-0.17 ± 0.01	-0.13

Table 3: Genetic (r_G), environmental (r_E) and phenotypic (r_P) correlations between yield traits and average SCS of 200, 305 and 365 days in milk

Traits	SCS_{200}			SCS_{305}			SCS_{365}		
	r_G	r_E	r_P	r_G	r_E	r_P	r_G	r_E	r_P
$Milk_{305}$	0.13 ± 0.06	-0.19 ± 0.01	-0.14	0.12 ± 0.05	-0.19 ± 0.01	-0.13	0.13 ± 0.05	-0.09 ± 0.01	-0.13
Fat_{305}	0.13 ± 0.06	-0.11 ± 0.01	-0.08	0.11 ± 0.05	-0.11 ± 0.01	-0.08	0.11 ± 0.05	-0.11± 0.01	-0.08
$Prot_{305}$	0.18 ± 0.06	-0.14 ± 0.01	-0.10	0.18 ± 0.05	-0.15 ± 0.01	-0.10	0.19 ± 0.05	-0.15 ± 0.01	-0.09
$Fatp_{305}$	-0.04 ± 0.06	0.10 ± 0.01	0.08	-0.05 ± 0.01	0.10 ± 0.01	0.07	-0.07± 0.06	0.10 ± 0.01	0.07
$Prop_{305}$	0.08 ± 0.06	0.17 ± 0.01	0.15	0.08 ± 0.05	0.16 ± 0.01	0.14	0.07 ± 0.06	0.16 ± 0.01	0.14
$Milk_{100}$	0.16 ± 0.06	-0.13 ± 0.01	-0.10	0.18 ± 0.06	0.11 ± 0.01	-0.08	NE	NE	NE
Fat_{100}	0.15 ± 0.06	0.15 ± 0.01	-0.13	0.16 ± 0.06	-0.04 ± 0.01	-0.02	NE	NE	NE
$Milk_{200}$	0.18 ± 0.06	0.18 ± 0.01	-0.05	NE	NE	NE	NE	NE	NE

NE: Not estimated

Table 4: Heritability (diagonal), genetic (above diagonal) and environmental (below diagonal) correlations among average SCS at different stages of lactation

	SCS_{100}	SCS_{150}	SCS_{200}	SCS_{305}	SCS_{365}
SCS_{100}	**0.04**	0.99	0.99	0.99	0.99
SCS_{150}	0.90	**0.05**	0.99	0.99	0.99
SCS_{200}	0.85	0.95	**0.06**	0.99	0.99
SCS_{305}	0.77	0.88	0.94	**0.07**	0.99
SCS_{365}	0.76	0.79	0.92	0.99	**0.08**

SE of estimates were in the range of 0.01 to 0.06

Table 5: Heritability (diagonal), genetic (above diagonal) and environmental (below diagonal) correlations among milk yield and composition

	$Milk_{305}$	Fat_{305}	$Prot_{305}$	$Fatper_{305}$	$Prtotper_{305}$
$Milk_{305}$	**0.28**	0.75	0.93	-0.60	-0.58
Fat_{305}	0.75	**0.18**	0.81	0.14	-0.06
$Prot_{305}$	0.94	0.76	**0.24**	-0.36	-0.23
$Fatper_{305}$	-0.27	0.41	-0.17	**0.27**	NE
$Prtotper_{305}$	0.37	-0.12	-0.05	NE	**0.32**

The SEs of environmental correlation in most cases was close to 0.01 and for heritabilities and genetic correlations in the range of 0.01 to 0.04. NE: Not estimated

phenotypic and genetic correlations of SCS at different stages of lactation using multivariate animal model are shown in Table 4. There were strong genetic and phenotypic correlations among SCS at different stages of lactation, implying that average SCS in different stages of lactation could be genetically regarded as the same trait. However, the phenotypic and environmental correlations were slightly smaller than respective genetic correlations. As expected, the environmental and phenotypic correlations between the adjacent stages were higher than the stages further apart, which is likely due to similarity of environmental factors among adjacent stages of lactation.

The estimated heritabilities for production traits as well as the genetic and phenotypic correlations among production traits are shown in Table 5.

Discussion

The estimates of heritability for all yield traits were within the range of values reported in the literature but slightly lower than the recent estimates obtained using random regression models (Miglior *et al*., 2009). The estimated genetic correlations between SCS and yield traits indicated that selection for higher yield tends to increase the SCS level of milk to some extent, which finally increases the susceptibility of cows to clinical mastitis. The environmental and phenotypic correlations between SCS and yield traits were negative but similar to the genetic correlations in magnitude. The environmental and phenotypic correlations were somewhat similar in sign and magnitude. Negative environmental correlation between SCS and milk was also reported in other populations (Haile-Mariam *et al*., 2001). The negative environmental correlation between yield and SCS indicates that good management and hygienic practices would be helpful in maintaining a satisfactory level of milk production as well as controlling the SCS of milk. Koivula *et al*. (2005) considered SCS before and after clinical mastitis as being two different traits and based on this definition, the genetic correlation of milk yield with SCS was reported to be 0.17 and 0.25, respectively. The existence of a positive genetic correlation between milk yield and SCS was also observed in Finish Ayrshire (Poso and Mantysaari, 1996), France Holstein (Rupp and Boichard, 1999) and Swedish Holstein cows (Carlen *et al*., 2004).

Samoré *et al*. (2008) reported genetic correlation of close to zero (0.02 to 0.08) between SCS and protein yield of Italian Holstein cows. Similar values (0.07) were

estimated for Italian Brown Swiss (Samoré *et al*., 2010). Genetic correlations of 0.22, 0.17 and 0.23 were reported between SCS and milk, fat and protein yield of Swedish Holstein, while the corresponding environmental correlations were -0.22, -0.17 and -0.19, respectively (Carlen *et al*., 2004). Positive genetic correlation between protein content and SCS were also reported in other populations (Miglior *et al*., 2007; Muir *et al*., 2007). Negative environmental correlations of -0.11 to -0.14 between SCS and protein yield were also reported in other studies (Samoré *et al*., 2008). It is clear that the interacting effects of various infections on SCS may change the sign or intensity of the correlation coefficients between milk protein content and percentage with SCS. After incidence of clinical or subclinical mastitis and subsequent elevation of the SCS, total protein content of milk may increase or remain constant. After infection of the udders with pathogens, Gamma immunoglobulin or Albumin are increased, where α, β and κ-caseins or other milk proteins are degraded. Therefore, the protein content may either increase or remain unchanged, but with a significant alteration in composition (Urech *et al*., 1999; Batavani *et al*., 2007). In Italian Holstein, the genetic correlation of protein yield with SCS was large and positive at the beginning of the first stages of first lactation but approached to zero or negative as lactation continued (Samoré *et al*., 2008).

The genetic correlations of milk yield and composition with average SCS were consistent across different stages of lactation, indicating that collection of somatic cell data over a shorter period of lactation, i.e. during the first half of lactation, in a larger number of herds could effectively be used in the breeding programs. Currently, somatic cell data are recorded only in several large herds, due to limited facilities or other infrastructures needed. It seems that data of first half stage of lactation could be informative enough to be implemented in the breeding program. Some studies showed that SCS during the first half of lactation has a stronger genetic correlation with clinical mastitis (Bloemhof *et al*., 2009).

The low heritability of SCS was in agreement with the recent estimate of 0.07 in Iranian (Safdari Shahroudi *et al*., 2010) and Polish Holstein cows (Ptak *et al*., 2009). Mostert *et al*. (2004) also reported very low heritability (0.04 to 0.07) for SCS in lactations 1 to 3 of South African Holstein and Jersey cows. However, in a more recent study, the estimated heritability of SCS in South African Holstein cows was between 0.17 and 0.19 (Dube *et al*., 2008). De Haas *et al*. (2008) reported estimates of 0.08 to 0.13 for the log-transformed average of SCC up to 100-335 days in milk. Literature values of heritability for SCS often ranged between 0.15 to 0.25 depending on data, model, population and definition of SCS (Muir *et al*., 2007; Samoré *et al*., 2008; Bohmanova *et al*., 2009; Miglior *et al*., 2009).

The results of the present study showed that cows with a high genetic potential for milk, fat or protein yield tended to have high SCS. The negative environmental correlation between SCS and yield traits implies that the environmental factors which increase milk production also are helpful in reducing the SCS. The genetic parameters estimated here could be useful in constructing the selection index for the genetic improvement of Iranian Holstein cattle. Also, the estimated genetic correlation between SCS_{100} or SCS_{150} with average SCS over longer stages of lactations (SCS_{305} and SCS_{365}) was 0.99 ± 0.01, implying that recording SCS over shorter period of lactation (first 100 or 150 DIM), but in a larger number of cows and herds would be informative enough to be applied in breeding programs, providing a cost effective alternative to collecting SCS in whole lactation.

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