Genetic Evaluation of Somatic Cell Counts of Holstein Cattle in Zimbabwe

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ABSTRACT : The objectives of the study were to examine non-genetic factors that influence somatic cell counts in dairy cattle and to estimate the genetic parameters of somatic cell counts. A total of 34, 097-test day somatic cell count records were obtained from the Zimbabwe Dairy Services Association (ZDSA). The data were from 5, 615 Holstein daughters of 390 sires and 2, 541 dams tested between May 1994 and December 1998. First lactation cows contributed 22, 147 records to the data set, while 11, 950 records were from second and later parity cows. The model for analysis included fixed effects of month of calving, year of calving, stage of lactation, calving interval and test date. Milk yield and age on test day were fitted in the model as covariates. The additive genetic effects pertaining to cows, sires and dams and the residual error were the random effects. The Average Information Restricted Maximum Likelihood algorithm was used for analysis. The heritability of somatic cell scores was low at 0.027 ± 0.013 for parity one cows and 0.087 ± 0.031 for parity two and above. Repeatability estimates were 0.22 ± 0.01 and 0.30 ± 0.01 for the two lactation groups, respectively. Genetic and phenotypic correlations between the somatic cell scores and test day milk production were small and negative. It seems that there is no genetic link between somatic cell counts and milk yield in Holstein cattle in Zimbabwe. The results also seem to indicate that somatic cell count is a trait that is mainly governed by environmental factors. (*Asian-Aus. J. Anim. Sci. 2000. Vol. 13, No. 10 : 1347-1352*)

Key Words : Individual Somatic Cell Counts, Genetic Parameters, Holstein Cattle, Zimbabwe

INTRODUCTION

The somatic cells in milk are leukocytes, including lymphocytes, macrophages, eosinophils and polymophonuclear neutrophils, and some epithelial cells from the udder. These cells are primarily concerned with defense of the mammary gland from infection. The numbers of somatic cells in milk is, therefore, a reliable indicator of the health status of the udder, especially of subclinical mastitis.

Mastitis is the most important infectious disease of dairy cattle that affects both the quality and quantity of milk (Giraudo et al., 1997). It is the most costly disease affecting dairy cows (Sischo, 1997). The losses as a result of mastitis are due to decreased milk yield, changed milk composition, discarded milk, drug costs and veterinary costs, among others. Control of mastitis is achieved mainly through high quality management practices, which are usually costly. There is a recent school of thought that mastitis can be reduced by indirect selection for low somatic cell counts (Schutz et al., 1990). This is because somatic cell counts in milk are highly positively correlated with mastitis. The possibility of reducing somatic cell counts through selection has not been explored in developing countries including Zimbabwe.

The objectives of this study were to determine the non-genetic factors that influence the levels of somatic cell counts and to estimate the variance components of individual somatic cell counts for additive genetic, permanent environment and residual effects.

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Received December 20, 1999; Accepted April 11, 2000

MATERIALS AND METHODS

Data

The data consisted of 34, 097-test day records of Holstein cows. The records were obtained by the Zimbabwe Dairy Services Association (ZDSA) milk records from 78 herds tested between March 1994 and May 1998. The following conditions were set for records to be included in the analysis:

1. The age at test day was restricted to be between 16 and 124 months,
2. The first test had to be between day 5 and day 45 of lactation, and the interval between two consecutive tests was kept between 28 and 50 days,
3. All sires had at least 5 daughters and all cows had at least 4 test day measurements.

After editing, two data sets were constructed separating the primiparous cows from their multiparous counterparts. The data was separated by parity in accordance with Zimbabwe's genetic evaluation procedures and according to other studies in literature (Khan et al., 1997). The number of days in milk was grouped by 5-day intervals making a total of 62 stage of lactation classes. The multiparous data set had calving intervals divided into 7 classes as follows: 240 -300, 301-390, 391-570, 571-750, 751-840, 841-1,020 and 1,021-1,170 days.

Statistical analysis

The following model was used for preliminary analysis within the parity groups.

$$Y_{ijklmnop} = \mu + Mc_j + YR_k + TD_l + DIM_m + H_n + CI_o +$$

$$b_1(AGE)+b_2(AGE)^2+b_1MTD+COWni+\varepsilon_{ijklmnop}$$

where:

$Y_{ijklmnop}$ = $\log_{10}$ somatic cell scores.

μ = test day mean level of performance.

Mc_j = fixed effect of month of calving with j = 1,2.....12.

YR_k = fixed effect of year of calving with k = 1,2,3 and 4.

TD_l = fixed effect of test date with l = 1,2.....802.

DIM_m = fixed effect of stage of lactation with m = 1 March 1994.....5 May 1998.

H_n = fixed effect of herd with n = 1,2,.....78.

CI_O = fixed effect of calving interval class with o = 1,2.....7.

$(AGE)_{ijklmnop}$ = age of cows on test day as a co-variate with b_1 and b_2 being the linear and quadratic regression co- efficients, respectively.

$(MTD)_{ijklmno}$ = milk yield on test day as a covariate with b_{ith} as the regression coefficient.

$COW_{(n)i}$ = random cow effect nested within herd n, with cow distributed as $N(0,\sigma^2_c)$.

$\varepsilon_{ijklmnop}$ = random residual error with distributed as $N(0,\sigma^2_e)$.

The objective of this model was to quantify the sources of variation in somatic cell counts. Since, there were repeated records per individual cow varying from 5 to 12 records per lactation, cow was included in the model as a random factor. In this preliminary analysis the model did not account for the relationships among animals. Equations pertaining to herd and random cow effects were absorbed during the analysis. In order to achieve a normal distribution and homogeneity of variance, somatic cell count data was transformed into logarithmic scale (log 10). The Henderson Method (III) in SAS (1996) was used for analysis.

Animal model

For estimation of genetic parameters a single trait animal model containing fixed effects identified in the above model was used. With this model, the (co)variance matrix for permanent environmental effects was proportional to an identity matrix and uncorrelated with other random effects. Maximum likelihood solutions for both random and fixed effects and genetic parameters were estimated using the Average Information Restricted Maximum Likelihood (AIREML) algorithm of Gilmour (1995). The following model represents the individual animal model in matrix notation.

$$Y = Xb + Z_1a + Z_2p + e$$

Where: Y is a vector of observations (log transformed somatic cell scores) of an individual cow; b is a vector of fixed effects of herd, month of calving, year of calving, stage of lactation, calving interval, test date, test day milk yield as a covariate and age on test as a covariate; a is a vector of random animal additive genetic effects, pertaining to 5, 615 cows, 390 sires and 2, 541 dams; p is a vector of permanent environmental effects; Z_1 and Z_2 are known incidence matrices linking elements of a and p to Y, respectively and e is a vector of random residuals. The expectations and (co)variances were:

$$E\begin{bmatrix} Y \\ a \\ p \\ e \end{bmatrix} = \begin{bmatrix} Xb \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

and

$$V\begin{bmatrix} a \\ b \\ e \end{bmatrix} = \begin{bmatrix} AG_0 & 0 & 0 \\ 0 & IPE_0 & 0 \\ 0 & 0 & IR_0 \end{bmatrix}$$

Where G_0, PE_0 and R_0 denote matrices for additive genetic, permanent environment and residual effects. A is the additive numerator relationship matrix and I is the identity matrix. The underlying assumptions were normality and independence of random errors as well as homogeneity of variance.

RESULTS AND DISCUSSION

Effect of month of calving

For parity one cows, individual somatic cell counts were highest for cows that calved in June and January. However, for multiparous cows, the trend was different. Somatic cell counts increased from January to reach a peak in December (figure 1). As reported by Kennedy et al. (1982), changes in somatic cell counts are season dependent for mature cows.

Although the trends for the two parity groups were not similar, it is clear that somatic cell counts were lower for first parity cows than for their multiparous counterparts. This can be ascribed to the fact that successive lactation and milking increase the incidence of mammary infection and the permanent damage to the mammary gland due to resolved infections (Dahoo et al., 1982).

Effect of year of calving

Figure 2 shows a dramatic reduction in somatic cell counts of Holstein cattle in Zimbabwe from 1996 to 1997. This could be due to improved management in 1997. Mangwiro (1998) who also observed a dramatic increase in milk yield over the same years made the same conclusion. This seems to support the

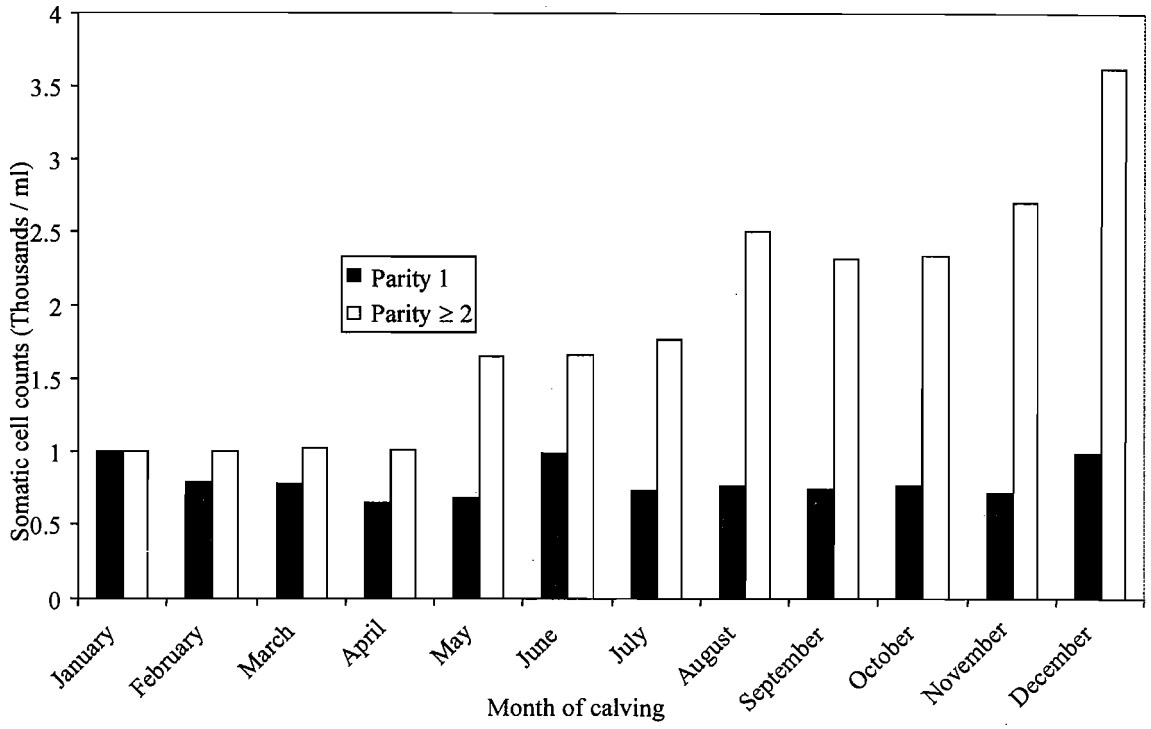

Figure 1. Effect of month of calving on somatic cell counts

fact that when incidences of sub-clinical mastitis are low, the somatic cell counts are also low, and milk production is elevated.

Effect of stage of lactation

Maximum likelihood solutions for days in milk are shown in figure 3. The curve resembles an inverted milk yield lactation curve as reported by Mangwiro (1998). These results indicate that somatic cell counts were high during early lactation or shortly after calving, decreased to a minimum by day ninety of lactation. The reverse is true for milk yield (Mangwiro, 1998). These observations seem to suggest a negative relationship between milk yield and somatic cell counts. The results are in agreement with previous work by other researchers (Schutz et al., 1993). Dahoo and Meek (1982) also reported that somatic cell counts were elevated immediately after calving, regardless of whether the cow was infected or not. High somatic cell counts shortly after calving can be caused by excessive shedding of epithelial cells as the mammary gland resumes or commences functioning (Kennedy et al., 1982). Oliver and Sordillo (1988) noted that the periparturient period was associated with rapid differentiation of the secretory parenchyma, intense mammary growth, copious synthesis and secretion, and marked accumulation of colostrum and milk. These processes could be the cause of this excessive shedding. A similar shedding occurs again at the end of lactation when the cow dries off. On the other hand, high somatic cell counts during early lactation indicate that dairy cows during this period are susceptible to mastitis. Oliver and Sordillo (1988) have reported that environmental pathogens, particularly coliform bacteria may also be involved in the elevation of somatic cell counts during early lactation.

Effect of calving interval

Figure 4 shows that somatic cell count was lowest for cows with short calving intervals. The optimum calving interval was 390 days. Cows with calving intervals of 660 days had the highest somatic cell

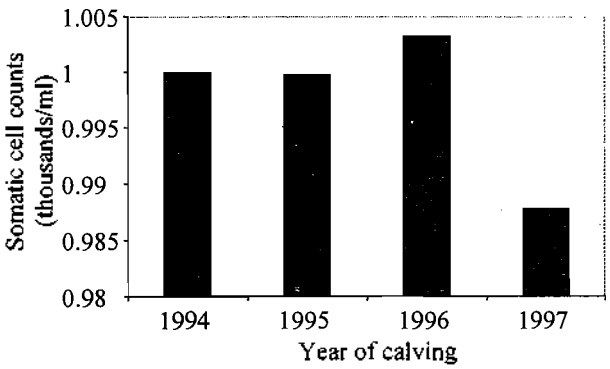

Figure 2. Effect of year of calving on individual somatic cell counts

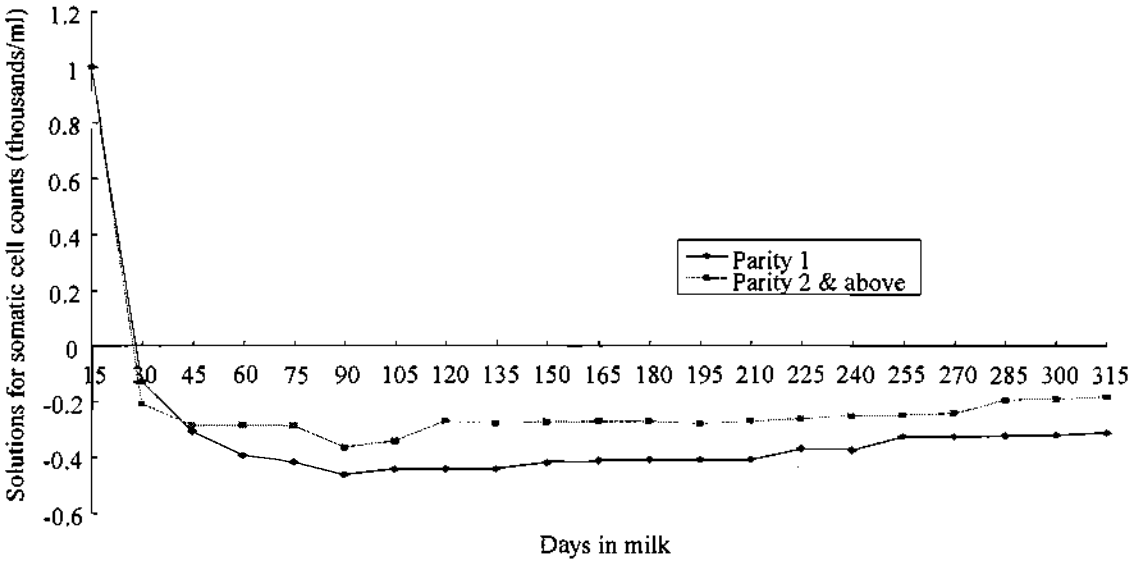

Figure 3. Effect of stage of lactation on individual somatic cell counts

numbers. The longer calving interval was associated with cows of higher milk yields in a previous study by Mangwiro (1998). Therefore, when milk yield is high, somatic cell counts are low. This is because both milk yield and somatic cell counts are negatively correlated.

Covariates

The age on the day of testing both as a linear or quadratic term significantly affected somatic cell counts ($p<0.001$). Kennedy et al. (1982) found a linear increase in somatic cell counts with advancing age of cows. The influence of age increased in parity 2 and above. This increase in somatic cell counts as the cow gets older was expected. Harmon and Reneau (1993) noted that somatic cell counts generally increased with advancing age. This may be attributable to an increased cellular response of older cows to infection (Dahoo et al., 1982). As the lactation number increases the number of chronically infected quarters may also increase. There will also be more extensive chronic tissue damage through long standing infections. In addition, the cellular response to infection may be greater in quarters that have been previously infected.

Genetic parameters

Heritability indicates the relative contribution of additive genetic effects and environment to phenotypic differences among animals. Table 1 shows the heritabilities, repeatabilities and the variance components for the somatic cell counts of Holstein cattle in Zimbabwe. The heritability estimates of 0.027 and 0.087 for somatic cell counts of primiparous and multiparous cows, respectively, were low. Studies elsewhere have reported low estimates of heritability of somatic cell counts. Schutz et al. (1993) reported estimates of heritability for somatic cell counts ranging from 0.05 to 0.27. Boettcher et al. (1992) reported estimates as low as 0.08 to 0.16 for primiparous Holstein cattle in five regions in the United States of America (USA). The same researchers reported 0.10 as the overall national estimate for the USA. Duda (1992) reported low heritability estimates for somatic cell count of 0.06, 0.07 and 0.09 for first parity, second parity and third parity animals respectively. Banos and Shook (1990) found heritability estimates for somatic cell count, which averaged 0.12. More recently, Reents et al. (1995) gave estimates for somatic cell scores of 0.09 for primiparous cows, 0.09 for cows in parity 2 and 0.11 for cows in parity 3. A low heritability estimate shows a lesser genetic

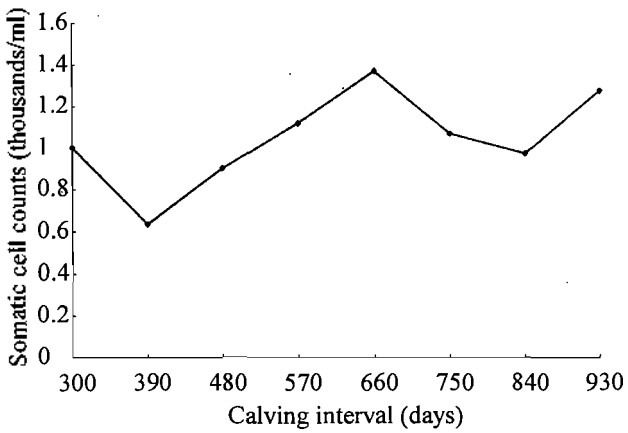

Figure 4. Effect of calving interval on individual somatic cell counts

Table 1. Residual (σ^2_r), permanent environmental (σ^2_{pe}), additive genetic (σ_a^2) and phenotypic (σ^2_p) variance components, heritabilities (h^2), repeatabilities (r) and their respective standard errors (se) for log somatic cell counts (LSCC)

Trait	Parity	σ^2_r	σ^2_{pe}	σ^2_a	σ^2_p	h^2	se	r	se
LSCC	1	0.617	0.152	0.0216	0.889	0.027	0.013	0.22	0.014
LSCC	>=2	0.647	0.199	0.0803	0.962	0.087	0.031	0.30	0.030

influence and a greater importance of the environment, including management, as a cause of variability among somatic cell counts. Low heritability results in slow genetic progress implying that selection for low somatic cell counts in Zimbabwe will not substantially reduce somatic cell counts or mastitis. Proper milking practices and improved management remain the most effective ways to reduce somatic cell counts for Zimbabwean Holstein cows. In this regard, milkers have a vital role to play in the milking parlor. It is, therefore, equally vital that milkers are properly trained and motivated to carry out their work conscientiously.

Genetic correlation

Genetic and phenotypic correlations between test day milk production and somatic cell counts are given in table 2. Low negative genetic and phenotypic relationships were present between milk yield and individual somatic cell counts. The genetic and phenotypic correlation coefficients were -0.069 and -0.107 for first parity and -0.020 and -0.201 for multiparous cows. These results are in agreement with the work of Schutz et al. (1990) who obtained a genetic correlation coefficient of -0.13 between the two traits. Banos and Shook (1990) reported a genetic correlation of -0.15 between the two traits. The results from the current study imply that there is no genetic antagonism between milk yield produced on test day and individual somatic cell counts. Thus, genetically high milk yielding cows do not necessarily have a tendency towards a higher somatic cell count. The low negative genetic trend between milk yield and somatic cell counts should also be expected since the heretability for somatic cell count is very low.

IMPLICATIONS

In this study heritability estimates were low for somatic cell counts. Estimates of genetic and phenotypic correlations between somatic cell scores and test day milk yield were low and negative. Since the heritability of somatic cells is low, it can be concluded that it is not easy to achieve genetic gain for somatic cell scores in the short term. In order to reduce somatic cell counts in Zimbabwean Holstein cattle, there is a need to direct most efforts to proper management practices.

Table 2. Genetic (r_g) and phenotypic (r_p) correlations between test day milk yield and individual somatic cell counts

Traits	Parity	r_g	r_p
Test day milk & LSCC	1	-0.069	-0.107
Test day milk & LSCC	=>2	-0.020	-0.201

ACKNOWLEDGEMENTS

The authors would like to thank the Zimbabwe Dairy Services Association for providing the records for the study. Financial assistance from the Belgian Agency for Development Co-operation is sincerely appreciated.

REFERENCES

Banos, G. and G. E. Shook. 1990. Genotype by environment interaction and genetic correlations among parities for somatic cell count and milk yield. J. Dairy Sci. 73:2563-2573.

Boettcher, P. J., L. B. Hansen, P. M. Van Raden and C. A. Ernst. 1992. Genetic evaluation of Holstein bulls for somatic cell counts and milk yield. J. Dairy Sci. 75:1127-1137.

Dahoo, I. R and A. H. Meek. 1982. Somatic cell counts in bovine milk. Can. Vet. J. 23:119-127.

Duda, J. 1992. Improvement of somatic cell counts by selection. Anim. Breed Abstr. 61(2):90.

Gilmour, A. R. 1995. Average Information Restricted Maximum Likelihood Manual. Polycopy.

Giraudo, J. A., A. Calzolari, H. Rampone, A. Rampone, A. Giraudo, C. Boghi, A. Larriestra and R. Nagel. 1997. Field trials of a vaccine against bovine mastitis. 1. Evaluation in heifers. J. Dairy Sci. 80:845-853.

Harmon. R. J. and J. K. Reneau. 1993. Factors affecting somatic cell counts in milk. Proceedings of the National Mastitis Council Annual Meeting. p. 48.

Khan, M. S., G. E. Shook, A. A. Asghar, M. A. Chaudhary and R. E. Mcdowell. 1997. Genetic parameters of milk yield and adjustment for age at calving in Nili Ravi buffaloes. Asian-Aus. J. Anim. Sci. 10:505-509.

Kennedy, B. W., M. S. Sethar, J. E. Moxley and B. R. Downey. 1982. Heritability of somatic cell count and its relationship with milk yield and composition in Holsteins. J. Dairy Sci. 65:843-847.

Mangwiro, F. K. 1998. Utilization of test day milk yield and somatic cell counts for genetic evaluation of dairy cattle. MSc. thesis. University of Zimbabwe.

Oliver, S. P. and L. M. Sordillo. 1988. Udder health in the peripaturient period. J. Dairy Sci. 71:2584-2606.

Reents, R., L. R. Schaeffer and J. C. M. Dekkers. 1995. Genetic evaluation for somatic cell score with a test day model for multiple lactations. J. Dairy Sci. 78:2847-2857.

SAS. 1996. Users Guide, SAS Institute Inc., Cary, North Carolina, USA.

Schutz, M. M., L. B. Hansen, G. R. Steuernagel, J. K. Reneau and A. L. Kuck. 1990. Variation of milk, protein, fat, and somatic cell counts for dairy cattle. J. Dairy Sci. 73:484-493.

Schutz, M. M., P. M. Van Raden and G. R. Wiggans. 1993. Genetic variation in lactation means of somatic cell scores for six breeds of dairy cows. J. Dairy Sci. 77:284-293.

Sischo, W. M. 1997. Use of physiologic variables to predict milk yield after clinical mastitis in dairy cattle. J. Am. Vet. Med. Assoc. 211:470-475.