

## FACTORS AFFECTING SOMATIC CELL COUNTS (SCC) IN BRAZILIAN DAIRY COWS

Guilherme N. Souza<sup>(1)</sup>, José R. F. Brito<sup>(1)</sup>, Maria A. V. P. Brito<sup>(1)</sup>, Élvio C. Moreira<sup>(2)</sup>,  
Marcos V. G. B. da Silva<sup>(1)</sup>.

<sup>1</sup>National Center for Research on Dairy Cattle, Milk Quality Laboratory, Juiz de Fora, MG, Brazil. 36.038-330.

<sup>2</sup>Federal University of Minas Gerais, Department of Preventive Veterinary Medicine, Belo Horizonte, MG, Brazil. E-mail: [gnsouza@cnpqgl.embrapa.br](mailto:gnsouza@cnpqgl.embrapa.br)

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### Introduction

Somatic cell count (SCC) of individual cows has been used as a useful tool to monitor subclinical mastitis in dairy herds. Several factors, including age of the cow, parity order, lactation period, month and season of the year may influence SCC (Schepers et al., 1997; Leavens et al., 1997). However, the presence of pathogens in the udder is generally considered the main factor responsible for the increase of somatic cells in milk (Harmon, 1994; Haas et al., 2002).

The occurrence of mastitis in a herd seems to be strongly related to the herd management. Up to 90% of the cases may be controlled by improving management (National Mastitis Council, 1996). In spite of this, a greater variation has been reported among individual animals, even under the same management conditions (Kennedy et al., 1982; Vecht et al., 1989; Schepers et al., 1997). Scarce information is available on factors that may influence SCC in dairy cows submitted to a tropical environment, where the cows are mainly kept on pasture, as compared to the currently available data for cows maintained on temperate conditions. The aim of this study was to identify the main factors influencing SCC in dairy cows under Brazilian conditions.

### Material and methods

The work was conducted in 24 dairy herds located in the Minas Gerais and Rio de Janeiro States (Southeast Region). A total of 2,657 cows were examined at least once during a 13-month period (June, 2002 – July, 2003). Milk samples (n=3,987) were examined for SCC and mastitis pathogens identification, using standard techniques (Harmon et al. 1990; Brito et al., 1999). A questionnaire was applied at each farm visit to obtain information on the animal, such as age, parity order, and lactation stage.

Statistical analysis was conducted using the SAS/STAT ® (1990). SCC variation was evaluated by the generalized linear models (GLM) (Dohoo et al. 2003). SCC values were transformed to linear score (LSSCC) according to Philpot and Nickerson (1991). Lactation period was coded at 19-days intervals, and parity order was coded as 1, 2, 3 and 4 (for more than 3 parities), according to Schepers et al. (1997). The results of three GLM were evaluated. A more complete model included the pathogens involved in intramammary (IMI) infection, as follows:  $Y_{ijklmno} = \mu + REB_i + ANI_j (H_i) + ANO_k + EST_l + OPA_m + PEL_n + STAPHA_o + STRAG_p + STREP_q + STACN_r + DIPT_s + e_{ijklmno}$ , where: *S. aureus*=STAPHA, *S. agalactiae*=STRAG, non-*agalactiae* streptococci=STREP, coagulase-negative staphylococci=STACN and *Corynebacterium* spp.=DIPT; and 1=absence and 2=presence of each pathogen. Descriptive statistics (geometric and arithmetic means, standard deviation and median) were used to evaluate the cows SCC, in relation to mastitis pathogens, and the T test to independent samples was applied to evaluate SCC according to parity order and IMI status.

## Results

The results of the bacteriological cultures were as follows: negative (n=1,139, 30%); single-pathogen isolation (2,614; 70%), mixed culture (404; 15%) and contaminated samples (238; 6%). *Corynebacterium* spp. was the most frequent agent (826; 32%), followed by *S. aureus* (790; 30%), *S. agalactiae* (551; 21%), coagulase-negative staphylococci (466; 18%) and non-*agalactiae* streptococci (351; 13%). SCC data are presented in Table 1.

All three GLM were statistically significant (p<0.01) and explained 80.5% to 81.7% of SCC variation. The main sources of SCC variation were the effect of animals nested in the herds, followed by the herd effect.

Table 1. SCC data according to the presence/absence of IMI and type of pathogen.

Source of variation	Category	N	SCC (x 1,000/ml)			
			Arithmetic mean	Standard deviation	Geometric mean	Median
Pathogen isolation	No	1,137	264	611	22	24
	Yes	2,612	779	1,070	228	342
Pathogen	STAPHA	790	966	1,072	371	509
	STRAG	551	1,520	1,559	662	923
	STREP	351	894	922	449	641
	STACN	466	422	633	125	205
	DIPT	826	410	561	94	166

STAPHA: *S. aureus*; STRAG: *S. agalactiae*; STREP: non-*agalactiae* streptococci; STACN: coagulase-negative staphylococci; DIPT: *Corynebacterium* spp.

Animals nested in the herds, herds, parity order, year season, IMI infection and *S. agalactiae* and non-*agalactiae* streptococci were the sources of variation (p<0.05), as

identified by GLM. Average linear scores of SCC according to the parity order and IMI infection are presented in Table 2.

Table 2. Average LSSCC according to the parity order and IMI presence.

Parity order	Presence of IMI			
	No		Yes	
	n	Mean	n	Mean
1	265	1.45 <sup>aA</sup>	401	3.83 <sup>aB</sup>
2	184	2.17 <sup>bA</sup>	438	4.41 <sup>bB</sup>
> = 3	309	2.78 <sup>cA</sup>	764	4.81 <sup>cB</sup>
Total	1,137	2.22 <sup>A</sup>	2,612	4.53 <sup>B</sup>

<sup>a,b,c</sup> Values within the same column with different superscripts differ ( $p < 0.05$ ). <sup>A,B</sup> Values within the same row with different superscripts differ ( $p < 0.05$ ).

## Discussion

Our findings and those of Brito et al. (1999) showed a great variation on SCC according to the pathogen causing the IMI.

The influence of parity order, year season, animal nested in the herd, herd, IMI and *S. agalactiae* and non-*agalactiae* streptococci infection on the SCC values has also been reported by others (Harmon 1994; Schepers et al., 1997; Leavens et al., 1997). However, the lactation period did not affect SCC variation, which differs from these authors. SCC variation among animals with the same pathogen was probably due to individual characteristics such as age, parity order and lactation period as have been reported previously (Schepers et al., 1997; Leavens et al., 1997).

Of all pathogens, *S. agalactiae* and non-*agalactiae* streptococci were the main responsible for increasing SCC. *S. aureus* did not seem to influence the SCC variation by the GLM, but the descriptive statistics showed that this pathogen was associated with high SCC. Coagulase-negative staphylococci and *Corynebacterium* spp. were responsible for a discrete increase of SCC as compared to culture-negative animals. These data are comparable to those reported elsewhere (Wilson et al., 1997; Haas et al., 2002).

The LSSCC increased in parallel with the advancement of parity order, irrespective of presence or absence of IMI. However, animals with IMI presented higher values of LSSCC in relation to their counterparts without IMI of the same parity order.

The present results point out those individual animal characteristics had a more significant effect on the SCC variation than the aspects related do the herds, as has been reported before (Kennedy et al., 1982; Vecht et al., 1989 and Schepers et al., 1997).

## Conclusion

IMI was the main factor responsible for SCC increase in dairy cows, kept under tropical conditions. Among the mastitis pathogens, *S. agalactiae* was responsible for the higher SSC values. Other factors that influenced SCC increase were the same as reported for cows kept under less warm conditions. These data suggest that programs of mastitis control could be easily adapted from temperate climate to tropical conditions.

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## References

1. Brito, M. A. V. P.; Brito, J. R. F.; Ribeiro, M. T.; Veiga, V. M. O. Padrão de infecção intramamária em rebanhos leiteiros: exame de todos os quartos mamários das vacas em lactação. *Arq. Bras. Méd. Vet. Zootec.*, v. 51, n. 2, p. 129-135, 1999.
2. Dohoo, I.; Martin, W.; Stryhn, H. *Veterinary epidemiologic research*. Charlottetown: AVC Inc. , 2003. 706 p.
3. Haas, Y.; Barkema, H. W.; Veerkamp, R. F. The effect of pathogen-specific clinical mastitis on the lactation curve for somatic cell count. *Journal Dairy Science*, v. 85, n. 5, p. 1314-1323, 2002.
4. Harmon, R. J. Physiology of mastitis and factors affecting somatic cell counts. *Journal Dairy Science*, v. 77, n. 7, p. 2103-2113, 1994.
5. Harmon, R. J.; Eberhart, R. J.; Jasper, D. E. et al. *Microbiological procedures for diagnosis of bovine udder infection*. Arlington: National Mastitis Council, 1990. 34 p.
6. Kennedy, B. W.; Sethar, M. S.; Tong, A. K.; W.; Moxley, J. E. Environmental factors influencing test-day somatic cell counts in Holstein. *Journal Dairy Science*, v. 65, n.2, p. 275-280, 1982.
7. Leavens, H.; Deluyker, H.; Schukken, Y. H.; Meulemeester, L.; Vandermeersch, R.; Muëlenaere, E.; Kruif, A. Influence of parity and stage of lactation on the somatic cell count in bacteriologically negative dairy cows. *Journal Dairy Science*, v. 80, n. 12, p. 3219-3226, 1997.
8. NATIONAL MASTITIS COUNCIL. *Current concepts of bovine mastitis*. Madison: National Mastitis Council, 1996. 64 p.
9. Philpot, W. N.; Nickerson, S. C. *Mastitis: counter attack. A strategy to combat mastitis*. Naperville: Babson Bros., 1991. 150p.
10. SAS Institute (Cary, NC). *SAS/STAT user's guide, version 6*. Cary: SAS, 1990.
11. Schepers, A. J.; Lam, T. J. G. M.; Schukken, Y. H. et al., Estimation of variance components for somatic cell counts to determine thresholds for uninfected quarters. *Journal of Dairy Science*, v.80, n.8, p.1833-1840, 1997.
12. Vecht, U.; Wisselink, H. J.; Defize, P. R. Dutch national mastitis survey. The effect of herd and animal factors on somatic cell count. *Netherland Milk Dairy Journal*, v. 43, n.4, p. 425-435, 1989.