EVALUATION OF THE RELATIONSHIP BETWEEN MILK SOMATIC CELL AND ABNORMAL UDDER CONFORMATION IN HOLSTEIN FRIESIAN COWS USING ANIMAL MODEL. Amin, A. A.¹; M. A. Mostafa² and T. Gere³

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ABSTRACT

Genetic, phenotypic correlations and heritability of milk somatic cell either count (SCC) or score (SCS) with mastitis udders (MU) and abnormal udder conformation (AUC) of 9368 Hungarian Holstein Friesian cows as daughters of 318 sirs of 28643 observation sample test–day of somatic cell count were estimated using animal model MTDFREML. Correlations of SCC and SCS with MU were higher than those with abnormal teat form. Genetic and phenotypic association of MU with SCS was higher than with SCC. The highest positive genetic relationships were presented for MU with non-cleft udder and unbalanced quarters. Suspended udder was phenotypic highly correlated with SCC and SCS. Long teat was strongly correlated with MU either genetic or phenotypic. Phenotypic correlation of unbalanced teat position with MU, SCC and SCS were higher than genetic estimates. Suspended udder was strongly genetic correlated with long udder, non-cleft udder, unbalanced quarters and unbalanced teat position. Short teat *vs.* long teat was great correlated genetically with suspended udder and unbalanced teat position. Estimates of heritability, variance and co-variances component are tabulated.

Keywords: udder, somatic cell, correlation, mastitis,

INTRODUCTION

Several reports have been made on the relationship between milk production and its constituents either as yields or percentages (Amin *et al.*, 1996; Amin *et al.*, 1997, Gaspardy *et al.*, 1995). However, there is controversy on which criteria to explain yield traits in genetic analysis. Linear type traits of body, udder conformation and milk somatic cell (Rogers and McDaniel, 1989; and Strandberg and Shook, 1989) are usually converted to a logarithmic form. Somatic cell scores have been identified as traits that are possibly useful for indirect selection to improve udder health and milk quality. The objective of the present study is to compute correlation estimates between milk somatic cell, as either count or score, with mastitis udder and abnormal udder conformation as an over view and guide line to construct a suitable selection index using udder traits for improving dairy farm profitability.

MATERIALS AND METHODS

Data used were the first four parities of 9368 Hungarian Holstein-Friesian cows as daughters of 318 sires, and arithmetic mean of 28643 observations as sample test-day observations of somatic cell count (SCC).

Total of 1013 animals of unknown sires was used all in estimating A⁻¹. All lactations commenced during the beginning of 1990 to the end of 1996. Somatic cell count measurements and mastitis cases were recorded for each sample daily Linear descriptive abnormal udder traits were considered as the following: -

Abnormal Udder Extension: AUE				
Extended Front Udder: AUE _{EFU}	ii) Emaciation Back Udder: AUEEBL			
Unbalanced udder Quarters: UUQ				
Low Rear Quarters: UUQ _{LRQ}	ii) Low Front Quarters: UUQ _{LFQ}			
Abnormal Udder Height: AUH				
Deep Udder: AUHDU	ii) Short Udder: <i>AUH_{SU}</i>			
4- Abnormal Udder Support: AUS				
Extreme Positive Cleft: AUSEPC	ii) Extreme Negative Cleft: AUS _{ENC}			
5- Abnormal Teat Placement: ATP				
Rear View: RV a) Close: ATF	b) Wide: ATP _{RV-W}			
Side View: SV a) Far Forward:	ATP _{SV-FF} b) Far Back: ATP _{SV-FB}			
6- Abnormal Teat Length: ATL				
Long Teat: ATLIT	ii) Short Teat: ATLst			

Figure 1. Shows form of the abnormal udders and teats that involved in the present study.

Mastitis test results were available with collecting monthly milk samples. Somatic cell count was based on the number of cells per milliliter of milk. Somatic cell count (SCC) had been transformed to SCS with the base 2-log scale as the following: -

SCS=log₂[(SCC/100)+3]

This formula has been accepted by the National Co-operative Dairy Herd Improvement

Statistical analysis of variance:

Animal Model of multi-trait derivative free REML procedure was used to obtain simultaneously genetic variance and covariances components, and residual variances of SCC, SCS, mastitis and abnormal udder and teat descriptive traits. A simplified version MTDFREML described by Boldman *et al.* (1997) was used for statistical analysis of variance in the present study.

The mathematical multi-trait animal model for investigated trait was

$$Y = X\beta + Zm + Wh + \varepsilon$$

Where: -

Y: is the vector of observation (Somatic cells counts or scores, mastitis, abnormal udder scores)

X*B*: represents all fixed factors (i-herd-year-season of calving, ii- age of cow at the testing time and stage of lactation as a covariant) associated with **Y**.

Zm: represents the total genetic value of all animals (Known and unknown sire. with or without records) associated with **Y**.

Wh: represents all other random factors in the mathematical model associated with Y, and

ε: is the vector of residual effects.

The vector *m* is the total additive genetic value of animals. Assume that $E(y) = X\beta$, and that:

RESULTS AND DISCUSSION

Relationship of somatic cell and mastitis with abnormal udder and teat forms:

Estimates of genetic and phenotypic correlation of somatic cell count (SCC), somatic cell score (SCS) mastitic udders (MU) and daily milk yield (DY) with abnormal udder and teat forms are presented in Table 1. All correlation estimates of MU with milk somatic cell (SCC, SCS) and daily milk yield (DY) were positive and showed similar trend with intermediate level. Genetic correlations of SCS with MU were higher than the corresponding estimates of SCC with MU. Genetic association of mastitis with SCS was relatively higher by 22.5% than with SCC. Thus, highly correlated response may be achieved in improving udder health through selection programs based on somatic cell score rather than that with somatic cell count. The values of phenotypic correlations between MU with SCC and SCS showed similar trends. The results (Table 1) showed also that all phenotypic correlations for MU with milk somatic cell were higher than the corresponding genetic values. These lead to the influences of environmental as an effective

power in the relationship among mastitis and milk somatic cell. Most of genetic and phenotypic correlation estimates between abnormal udder forms with milk somatic cell and MU were positive with high values Table (1). These results mean that, the abnormal udder forms could be used as a good indication for increasing and incidence of mastitis udder. On the same direction, the corresponding relationship of milk somatic cell with abnormal teat form were concisely positive and showed values of above the intermediate level. On the other direction the relationship between daily milk yield (DY) and each of abnormal udder and teat forms (Table 1) were mostly negative and of had intermediate values except DY with AUE_{EFU}, AUSEPC, AUSENC and ARLLT. The highest negative genetic relationships of DY with abnormal udder forms were obtained for AUH_{SU} and AUH_{DU} (-0.44 and -0.31), respectively. On the other hand, the corresponding estimates of phenotypic correlations had negative and higher than their genetic estimates (-0.51 and 0-0.37, respectively). All estimates of genetic and phenotypic correlations of DY with abnormal teat forms were negative except with ATLLT. The highest negative relationship of DY also with abnormal teat length-short teat (ATL_{ST}) was found (-0.44 and -0.49), respectively. As a matter of fact the genetic effect on reduced length of teat and on the milk secretary tissues in the same udder. In addition reduced teat length is considered unsuitable for a speedy milk letdown. Thus the long teats were more positively correlated with milk somatic cell and MU than short teats.

Regarding the teat placement (Table 1); indicated that the wide and close teat placement (rear view) had the same relationship with MU and milk somatic cell.

fig

fig

Close teat placement was relatively highly phenotypic correlated with MU than wide teat placement. That may due to transferring infections among infected quarters is easier in close teat than the wide teat placement.

Abnormal udder extension-emaciation back udder (AUE_{EBU}) had moderate negative genetic and phenotypic relationship with DY (r_g : -0.23 & r_p : -0.28, respectively). This may indicate that, abnormal back udder AUE_{EBU} is not favorable in selection programs intended to improve milk production since this may cause limitations in growing and development of the mammary gland.

The phenotypic correlations between abnormal udder height (deep udder only) with MU and milk somatic cell were higher than the corresponding genetic estimates (Table 1). This may indicate the importance of the environmental role in increasing rate of mastitis deep udder. On the other hand dairy cows with phenotypic abnormal short udder had low chance for mastitis occurrence. It also indicate that dairy cow with short udders had high genetic capability of mastitis resistance and increasing milk somatic cell.

Genetic correlation between DY and short udder was negative and not low (r_g : -0.44). Therefore, short udder, from economic point of view, is not considered as favorable udder form. Milk somatic cell was highly and negatively correlated with abnormal udder height-deep udder (AUH_{DU}). This indicate that extended udder under the hock or nearing the udder to the ground increases the possibility for infection by mastitis microorganisms and consequently increasing number of somatic cell count. The current results are agreement with that reported by Young *et al.* (1960).

Table 1, shows also that the extended front udders AUE_{EAU} were slightly correlated with mastitis incidence and intermediately correlated with milk somatic cell and DY. On the other hand, inheritance ability and environmental effects for mastitis resistance of emaciation back udder AUE_{EBU} showed negative and small values. This means that AUE_{EBU} had possibility for mastitis infection due to closeness of all udder teats together and with animal back legs.

Unbalanced udder quarters (UUQ_{LFQ} and UUQ_{LRQ}) had positive genetic and phenotypic correlations with mastitis incidence and milk somatic cell (Table 1). Inheritance abilities for mastitis infection of UUQ were relatively high under unsuitable environmental conditions. Also results of correlation estimates may reflect that the rate of mastitis infection of UUQ_{LRQ} were higher than UUQ_{LFQ} . This may be due to closeness low quarters to animal legs. The either positive or negative cleft abnormal udder supports ($AUS_{EPC} \& AUS_{ENC}$) were positively correlated with mastitis and slightly with DY. While MU was 50% highly correlated either phenotypically or genetic with AUS_{ENC} than with AUS_{EPC} . This may be due to losses of the connective tissue among udder quarters. This may ease the migration of mastitis infection among quarters. Also, the relationship between AUS and DY was positive with small correlation estimates. On the other hand, environmental influences seemed to have a great role in controlling the relationship between AUS_{EPC} with DY.

The abnormal teat forms either as teat placement or teat length (Table 1) had positive relationship with mastitis incidence and milk somatic cell. On the other hand all udders with abnormal teat form were negatively correlated

with DY except udders with long teats. Most estimates of genetic and phenotypic correlations among abnormal teat forms and both MU and SCS were approximately similar This may indicate that, early examination for milk somatic cell could be an appreciate toll for predicting and detecting mastitis case in early stages. Abnormal teat length-long teat (ATL_{LT}) had stronger genetic correlation with MU than the other abnormal teat forms. This may be due to less suitability of these teats for milking machine. Also udders with long teats were easier for mastitis infection through touching pens ground or dirty back legs of the animal these results are in agreement with those reported by Rathore (1977).

Relationship between abnormal udder and teat forms:

Estimates of genetic and phenotypic correlations between abnormal udder and teat forms are presented in Table 2. Estimates of phenotypic relationship among all abnormal udder and teat forms were lower than the corresponding genetic estimates.

Table 2: Estimates of heritability and correlations between abnormal udder and teat forms traits.

Gonotic	AUH	AUE	UUQ	AUS	ATP	ATL		
Genetic	Phenotypic							
AUH	.23 <u>+</u> .16	.22 <u>+</u> .09	.24 <u>+</u> .18	.25 <u>+</u> .10	.33 <u>+</u> .11	.30 <u>+</u> .14		
AUE	.31 <u>+</u> .13	.53 <u>+</u> .07	.22 <u>+</u> .11	.22 <u>+</u> .17	.19 <u>+</u> .11	.29 <u>+</u> .12		
UUQ	.71 <u>+</u> .29	.49 <u>+</u> .11	.29 <u>+</u> .11	.19 <u>+</u> .07	.32 <u>+</u> .14	.23 <u>+</u> .14		
AUS	.54 <u>+</u> .18	.37 <u>+</u> .11	.27 <u>+</u> .10	.52 <u>+</u> .13	.43 <u>+</u> .14	.37 <u>+</u> .07		
ATP	.76 <u>+</u> .21	.44 <u>+</u> .13	.39 <u>+</u> .17	.73 <u>+</u> .24	.33 <u>+</u> .17	.32 <u>+</u> .24		
ATL	.23 <u>+</u> .13	.29 <u>+</u> .14	.29 <u>+</u> .14	.41 <u>+</u> .18	.49 <u>+</u> .12	.51 <u>+</u> .15		

Abnormal udder height (AUH) had strong positive genetic correlated with UUQ and ATP. This result indicates that dairy cows had either deep or short udders had a genetic potential for having unbalanced udder quarters and abnormal teat placement. Also the results in Table 1 show that the previous examined three abnormal forms had great genetic and phenotypic correlation with MU. All the previous relationships do not favorable to increasing milk production and reducing amount of secreted milk somatic cell. Moderate genetic relationships were obtained for AUH with both ATL and AUE. Therefore, inclusion of the previous traits in selection programs for improvement mastitis resistance will be of very little impact. Current results of genetic and phenotypic estimates of correlations between abnormal udder height with other investigated abnormal teat forms were slightly higher than were reported by Brotherstone (1994). Also, the relationship between AUE and ATL may be controlled by either environmental factors or genetic factors due to similar obtained value (0.25). The values of estimates of phenotypic correlations among udder and teat forms, which were mostly, low and few of them were of intermediate level. Thus, the common environmental conditions seemed to have an effective role in controlling the relationship between ATP and ATL with AUS. Estimates of correlations either genetic or phenotypic of

the abnormal studied current were in agreement with results of some udder traits, which reported by Brotherstone (1994) and Short and Lawlor (1992). Estimates of heritability for all investigated abnormal udder and teat forms are in diagonal of Table 2. Results in table 2 shows that *AUE*, *AUS* and *ATL* seemed to have a great genetic ability (h^2 from 0.51 to .53) for transmission among successive generations than the other abnormal forms. These results are in agreement with Seykora and McDaniel (1986); Monardes *et al.* (1990), and Rogers, *et al.* (1991) in their studies on udder and body forms of dairy cattle. The lowest heritability estimates of abnormal udders was obtained for *AUH* ($h^2 = 0.23$). This may be due to more control of environmental factors (such as bad hand milking, bad evacuations, pulsation and equipment of milking machine) on this trait.

Estimates of heritability for all investigated abnormal udder and teat forms across the first successive parities are presented in Table 3. Heritability estimates of abnormal udder forms were lower than for abnormal teat forms in all parities except the last one. Heritability estimates of all investigated abnormal forms were increased with advancing order of lactation till the 4th parity. Results in Table 3 show that the lowest heritability of all abnormal udder forms was presented in the first parity. This may be due to genetic homogeneity of sub-set abnormal udder population in early stages of lifetime production. Heritability estimates of *AUS* were very low in the first parity. This result may indicate that genetic factors controlling *AUS* increase their magnitude of effect with advancing age of animal.

Conclusion and General Considerations:

Normally the evidence could be found in the variation between individual udders within each cow of dairy breed. This make reflection in the shape of the udder, teat as well as the position of teats.... etc. No doubt the effective relations between abnormal udder, teat forms and milk somatic cell and by the way the mastitis. Thus, the results of present study convermed these observation, using 28643 observation sample test-daily of milk somatic cell from 9368 Hungarian Holstein Friesian cows as daughters of 318 sires should the following results.

	1 <u>st</u>	2 <u>nd</u>	3 <u>rd</u>	4 th	5 <u>th</u>				
MU	.07 <u>+</u> .01	.13 <u>+</u> .05	.12 <u>+</u> .10	.16 <u>+</u> .11	.19 <u>+</u> .14				
Abnormal Udder Form									
AUH	.12 <u>+</u> .10	.19 <u>+</u> .11	.31 <u>+</u> .12	.26 <u>+</u> .12	.11 <u>+</u> .11				
AUE	.07 <u>+</u> .10	.13 <u>+</u> .07	.24 <u>+</u> .09	.28 <u>+</u> .11	.26 <u>+</u> .19				
UUQ	.13 <u>+</u> .01	.21 <u>+</u> .18	.26 <u>+</u> .11	.33 <u>+</u> .12	.27 <u>+</u> .17				
AUS	.09 <u>+</u> .01	.19 <u>+</u> .05	.29 <u>+</u> .17	.29 <u>+</u> .18	.24 <u>+</u> .13				
Abnormal Teat Form									
ATP	.22 <u>+</u> .13	.23 <u>+</u> .12	.31 <u>+</u> .20	.32 <u>+</u> .17	.12 <u>+</u> .09				
ATL	.18 <u>+</u> .11	.29 <u>+</u> .12	.35 <u>+</u> .11	.35 <u>+</u> .13	.17 <u>+</u> .08				

Table 3:	Heritability	estimates	of	mastitis	and	abnormal	udder	and	teat
	traits per p	parity.							

Early examination for milk somatic cell could be an appreciate toll for predicting and detecting mastitis case in early stages. Dairy cows had either deep or short udders had a genetic potential for heaving unbalanced udders guarters and abnormal teat placement and these traits do not favorable to increasing milk production and reducing amount of secreted milk somatic cell. Udders with long teats were easier for mastitis infection through touching pens ground or dirty back legs of the animal. Highly correlated response may be achieve in improving udder health through selection programs based on somatic cell scone rather than that with somatic cell count. Abnormal udder forms could be used as a good indicator for increasing and incidence mastitic udders. Using abnormal udder, abnormal teat length and abnormal other extension traits in selection programs for improvement of mastitis resistance will be very little impact. As a conclusion, the obtained results foucsing the light on the importance of the udder shape and teat as well as teat placement which lead to accuracy of introducing these traits in breeding programs, as well as the role of improving the environment factors surrounding the dairy cows to reduce the possibility of other infection.

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تقييم العلاقة بين الخلايا الجسمية في اللبن وأشكال الضرع غير الطبيعية في ماشية الهولشتين فريزيان باستخدام نموذج الحيوان . أشرف عبد الرحمن أمين أو محمد عبد الرحمن مصطفى وتيبور جرى " · قسم الإنتاج الحيواني _ كلية الزراعة بالإسماعيلية _ جامعة قناة السويس _ مصر · ۲ قسم إنتاج الحيوان – كلية الزراعة – جامعة المنصورة – مصر. ٢ كلية الزراعة _ جونجش _ المجر.

تم تقدير الارتباطات الوراثية والمظهرية والمكافئ الوراثي بين الخلايا الجسمية في اللبن سواء كعدد أو قيم إحصائية مع صفات التهاب الضرع وأشكال الضرع الغير طبيعية في ماشية الهولشتين فريزيان المجرية وذلك باستخدام نموذج الحيوان إحصائيا لعدد ٢٨٦٤٣ عينة إختبار الخلايا الجسمية في اللبن من ٩٣٦٨ بقرة هولشتين فريزيان، بنات ٣١٨ طلوقة.

- أظهرت الارتباطات بين صفة التهاب الضرع والخلايا الجسمية سواء كانت عدد أو قيم إحصائية أعلى منها مع أشكال الحلمة غير الطبيعية.
- كانت الارتباطات الوراثية والمظهرية بين صفة التهاب الضرع وقيم الخلايا الجسمية إحصائيا أعلى منها في العدد . كما ظهر إحصائيا أن أعلى ارتباط وراثي موجب بين صفة التهاب الضرع وكل من صفتى الضرع ضعيف الارتباط بالجسم، والأرباع غير المتزنة. - ظهر أن الارتباط المظهري بين القيم الإحصائية لصفة الخلايا الجسمية عالياً عن عددها مع صفة
- الضرع المتدلى.
- ، ليسرع حصي لوحظ قوة الارتباط المظهري والوراثي بين صفتي طول الحلمة والتهاب الضرع. كانت قيم الارتباط المظهري لصفة وضع الحلمة غير المتزنة مع صفات التهاب الضرع، الخلايا الجسمية كقيم إحصائية أعلى من الارتباط الوراثي.
- ارتبط الضرع المتدلى ارتباطا وراثياً موجباً مع كل من صفات الضرع الممتد، الضرع ضعيف الارتباط بالجسم، الأرباع غير المتزنَّة، وضع الحلمة غير المتزن. وقد ظهر أن الحلمة القصيرة أكثر أرتباطاً وراثياً مع صفة الضرع المتدلى ووضع الحلمة غير المتزن عن الحِلمة الطويلة.
- وقد قدرت الدراسة المكافئ الوراثي للصفات المدروسة التي جذبت الانتباه إلى أهمية الاعتماد على شكل الضرع وشكل الحلمات وأبعادها في التنبؤ بمدى قابلية الضرع للإصابة بمرض التهاب الضرع وإجراء الانتخاب في ماشية اللبن اعتماداً على الصفات المرفولوجية للضرع.

	Genetic correlations				Phenotypic correlations			
	Мυ	SCC	SCS	DY	Мυ	SCC	SCS	DY
MU		0.33 <u>+</u> 0.11	0.42 <u>+</u> 0.14	0.35 <u>+</u> 0.11		0.38 <u>+</u> 0.14	0.47 <u>+</u> 0.18	0.42 <u>+</u> 0.13
			Abn	ormal Udder l	Form			
AUH _{DU}	0.27 <u>+</u> 0.17	0.55 <u>+</u> 0.14	0.45 <u>+</u> 0.20	-0.31 <u>+</u> 0.11	0.44 <u>+</u> 0.18	0.65 <u>+</u> 0.10	0.63 <u>+</u> 0.17	-0.37 <u>+</u> 0.12
AUH _{SU}	-0.37 <u>+</u> 0.11	-0.24 <u>+</u> 0.13	-0.15 <u>+</u> 0.10	-0.44 <u>+</u> 0.11	-0.15 <u>+</u> 0.11	-0.18 <u>+</u> 0.12	-0.19 <u>+</u> 0.10	-0.51 <u>+</u> 0.09
AUE _{EFU}	0.22 <u>+</u> 0.10	0.34 <u>+</u> 0.17	0.44 <u>+</u> 0.19	0.41 <u>+</u> 0.15	0.19 <u>+</u> 0.01	0.45 <u>+</u> 0.19	0.44 <u>+</u> 0.09	0.48 <u>+</u> 0.13
AUE _{EBU}	-0.18 <u>+</u> 0.19	-0.22 <u>+</u> 0.12	-0.27 <u>+</u> 0.10	-0.23 <u>+</u> 0.09	-0.11 <u>+</u> 0.07	-0.13 <u>+</u> 0.11	-0.11 <u>+</u> 0.10	-0.28 <u>+</u> 0.11
UUQ_{LFQ}	0.41 <u>+</u> 0.01	0.21 <u>+</u> 0.18	0.17 <u>+</u> 0.07	-0.11 <u>+</u> 0.09	0.31 <u>+</u> 0.11	0.22 <u>+</u> 0.07	0.35 <u>+</u> 0.10	-0.11 <u>+</u> 0.05
UUQ_{LRQ}	0.53 <u>+</u> 0.12	0.23 <u>+</u> 0.10	0.32 <u>+</u> 0.11	-0.07 <u>+</u> 0.04	0.44 <u>+</u> 0.17	0.27 <u>+</u> 0.13	0.44 <u>+</u> 0.19	-0.09 <u>+</u> 0.03
AUS _{EPC}	0.20 <u>+</u> 0.00	0.11 <u>+</u> 0.00	0.18 <u>+</u> 0.07	0.23 <u>+</u> 0.09	0.22 <u>+</u> 0.08	0.15 <u>+</u> 0.07	0.11 <u>+</u> 0.09	0.33 <u>+</u> 0.12
AUSENC	0.42 <u>+</u> 0.11	0.31 <u>+</u> 0.09	0.43 <u>+</u> 0.07	0.10 <u>+</u> 0.10	0.41 <u>+</u> 0.13	0.25 <u>+</u> 0.09	0.31 <u>+</u> 0.11	0.12 <u>+</u> 0.09
Abnormal Teat Form								
ATP _{RVC}	0.39 <u>+</u> 0.00	0.29 <u>+</u> 0.07	0.39 <u>+</u> 0.00	-0.11 <u>+</u> 0.04	0.41 <u>+</u> 0.07	0.32 <u>+</u> 0.09	0.42 <u>+</u> 0.17	-0.20 <u>+</u> 0.08
ATP _{RVW}	0.41 <u>+</u> 0.11	0.32 <u>+</u> 0.09	0.38 <u>+</u> 0.08	-0.17 <u>+</u> 0.06	0.38 <u>+</u> 0.07	0.33 <u>+</u> 0.07	0.45 <u>+</u> 0.12	-0.22 <u>+</u> 0.11
ATP _{SV-FF}	0.22 <u>+</u> 0.04	0.45 <u>+</u> 0.11	0.22 <u>+</u> 0.11	-0.14 <u>+</u> 0.05	0.29 <u>+</u> 0.04	0.29 <u>+</u> 0.09	0.22 <u>+</u> 0.10	-0.18 <u>+</u> 0.03
ATP _{SV-FB}	0.18 <u>+</u> 0.03	0.48 <u>+</u> 0.11	0.43 <u>+</u> 0.19	-0.09 <u>+</u> 0.13	0.18 <u>+</u> 0.00	0.33 <u>+</u> 0.11	0.22 <u>+</u> 0.03	-0.23 <u>+</u> 0.07
ATL _{ST}	0.23 <u>+</u> 0.12	0.18 <u>+</u> 0.11	0.21 <u>+</u> 0.14	-0.44 <u>+</u> 0.11	0.24 <u>+</u> 0.14	0.22 <u>+</u> 0.18	0.23 <u>+</u> 0.19	-0.49 <u>+</u> 0.19
ATLLT	0.55 <u>+</u> 0.18	0.44 <u>+</u> 0.11	0.49 <u>+</u> 0.11	0.10 <u>+</u> 0.07	0.37 <u>+</u> 0.11	0.34 <u>+</u> 0.20	0.40 <u>+</u> 0.14	0.12 <u>+</u> 0.10

Table 1: Correlation estimates somatic cell with abnormal udder traits.

MU: Mastitis udder, *SCC:* Somatic cell count, *SCS:* Somatic cell score Other abbreviations see Figure 1.