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# Genetic Relationships Between Somatic Cell Score and Milk Production in The First Five Lactations of Friesian Cows in Egypt

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# ABSTRACT



Records of somatic cell score (SCS) and 305 day milk yield (MY) of the Friesian cows raised at Sakha and El-Karada farms. From 2000 to 2017 were used to determine the relationships between SCS and MY between and within first five lactations. Genetic parameters were estimated by MTDFREML. Heritability estimates of SCS was ranged from 0.11 to 0.23 for five first lactations and were from 0.21 to 0.36 for MY. Heritability estimates for all lactations were 0.30 and 0.41 for SCS and MY, respectively. The genetic correlations between (SCS and MY) were moderate to high and positive (0.60 to 0.81) for first five lactations. Additionally, genetic correlations between (SCS and MY) were increased with increased lactation order, indicating a selection for MY will reduce SCS and therefore reduce the incidence of mastitis. Accuracy of (EBV's) varying from 64 to 84, 42 to 78 and 30 to 53% of sires, cows and dams, successively, indicating, the genetically improvement could be achieved with any pathway of them. Also, the trend of correlations between predicted breeding values were in the same direction with those reported for genetic correlations for the same traits. Selection of the cow on the basis of their expected breeding values should cause reduction in the SCS and therefore reduce mastitis and increase MY. The accuracy of the selection index that included SCS with MY was higher than that of other indices. It is emergent to include SCS as a criterion for selection of mastitis resistance in the breeding programme in Friesian cows.

*Keywords:* heritability, genetic and phenotypic correlation, somatic cell score, predicted breeding value, mastitis resistance and selection index.

# INTRODUCTION

For more years, milk production traits have held the leading position as influential

Breeding goals. Therefore, the research was concentrated on those factors in animal physiology and nutrition that would increase milk yield at the peak of lactation rather than on the maintenance of milk secretion from the decrease phase of lactation (Stefanon *et al.*, 2002). During the period between calving and peak yield, prevalence of most health problems, including mastitis, are high (Strapáková *et al.*, 2016).

Udder mastitis is widely realize as a single of the majority expensive health problems of dairy cattle (Hansen *et al.*,1979) and it is regard as one of the most expensive disease in dairy cattle because of its organic belongings (Heringstad *et al.*, 2000), financial losses (El-Awady and Oudah, 2011 and Sinha *et al.*, 2014) and also decrease in animal wellbeing (Kheirabadi and Razmkabir, 2016). In this respect, udder mastitis is demarcated as a mammary gland irritation that is generally due to bacterial infections and non-infections etiological causes (Santos *et al.*, 2003, Abera *et al.*, 2012) that not only affects milk yield but also has a undesirable impact on milk composition and physicchemical appearances (FAO, 2014).

Shook and Schutz (1994) recommended that SCC for individual cows be modified by base 2, commonly called linear somatic score (LCS). Ease of interpretation and comparison as well as more direct relationships to lost milk production formed the basis of this recommendation. They added that Linear scores correlate highly with lost production, infection status, and cheese processing yields. For these reasons, several dairy farm report SCC data as LCS.

Mastitis and high somatic cell score were the second leading reason for culling and accounted for about 24% of culled cows (Svensk Mjölk, 2002). Selection has classically concentrated on production traits and direct selection for resistant mastitis has been considered as inoperative because the heritability of mastitis is low and The majority of countries not recording the clinical mastitis incidences indifferent breeds (Carlén et al, 2004, Koivula et al, 2005, El-Arian and El-Awady 2008 and El-Awady 2014). Therefore, indirect measures of udder health such as SCS, have been an appealing alternative. SCS is routinely recorded in most milk recording systems, and information on SCS is easily available on a large scale. So, indirect measures for udder health trasits as SCS, have been an attractive alternative. Considered as SCS is routinely worked in recording systems of milk, and realization on SCS is readily available on widely. The efficiency selection criterion for mastitis resistance based on SCS depended on genetic correlations. The genetic correlations were moderate and positive (0.60 to 0.84) have been reported (Koivula et al, 2005; El-Awady, 2009 and Strapáková et al., 2016).

Moreover, Somatic cells exist normally in milk and consist of different many of type's cells; such as neutrophils, macrophages, lymphocytes, eosinophils, and various epithelial cells from the mammary gland. Cells in milk from a healthy udder are mainly represented by mammary gland epithelium and drain canal cells. Only 8% are leukocytes and less than 1% are macrophages (Walawski, 1999). Kehrli and Shuster (1994) reported that the concentration of somatic cells in milk is defined as somatic cell count (SCC) and it is the measure of thousands of cells per millilitre of milk. Milk obtained from a normal healthy udder usually contains less than  $10^5 x 10^3$  somatic cells per ml.

An elevated of SCC in samples of milk is a sign of udder infection. In addition, SCC are also current in milk of healthy cows and the increase in SCC is a normal cellular defense against udder infection. On the contrary, several studies shown that low lactation yield mean SCC not high the susceptibility of dairy cows to indices clinical mastitis (Rupp and Boichard, 2000, Rupp et al, 2000 and Boettcher et al, 2002 and El-Awady et al., 2014). Therefore, it is needed to understand that, while selection against dairy cows to high SCC is presumed to decrease mastitis incidence, the conundrum is whether SCC should be reduce to the lowest level (El-Awady and Oudah, 2011 and Strapáková et al., 2016). Therefore, the objective of this paper is to discuss why mastitis should be used for breeding decisions, why genetic improvement of resistance to mastitis should be based upon somatic cell scores (SCS) and investigate the relation among SCS and MY to determine whether SCS can serve as efficient indicator of mastitis infection to evaluate the efficiency of including it in a selection criterion to increase mastitis resistance in dairy cows in a hot semi-arid climate.

# MATERIALS AND METHODS

### Data and managements

Data used in this study were taken from the records of the Holstein Friesian cows in the Sakha and El-Karada farms from the Sakha milk recording unit in the International Livestock Management Training Center (ILMTC) located in the northern part of middle Delta, from 2000 to 2017. These herds belong to the Animal Production Research Institute, Ministry of Agriculture-Egypt. A total of 5696 Friesian records (2831 from Sakha and 2865 from El-Karada) of 1018 cows sired by 79 sires were used in the analysis. All sires have at least 50 daughters. Heifers were served in the first time when reached 24 months of age or 350 kg of weight. Cows were usually served two months after post parturition. The cows were kept under the same system of feeding and management.

Cows were fed on concentrate ration, through winter and spring months (December to May), and Egyptian clover (Trifolium alexandrinum), while in summer and autumn (June to November), cows were fed on concentrate, Egyptian cover hay or green sweet sorghum and rice straw Feed the cows base on live body weight, milk yield during lactations and pregnancy status. Water was available freely, Also the same thing in mineral mixture. Cows were milked twice daily by DeLaval Alpha milking machine. Milk yield was recording from day to the nearest 100 g for each Cow. Usually cows were dry off before calving date almost 60-75 days.

Milk yield (MY) and SCS consist of one to greater than or equal five lactations adjusted to a 305-day lactation record. SCS is an indicator trait for resistance to mastitis. SCC were converted to SCS using a base 2 logarithmic function: SCS = log2 (SCC/100) + 3 (Ali and Shook 1980; Shook and Schutz 1994), where somatic cells by thousand per milliliter of milk.

# Statistical analysis

Data were first analyzed by Mixed Model program of Harvey (1990) in order to determine the fixed effects. The statistical model included month (1 to 12) and year (2000 to 2017) of calving, parity (1 to  $\geq$ 5) and farm (1 = Sakha and 2 = El-Karada). All effects being significant for studied traits were included in the analytical model. Age at first calving was used -free restricted maximum as a covariate. Covariance components were estimated for traits with (REML) procedures using the MTDFREML program of Boldman *et al*, (1995). The basic multiple model was:  $Y = X\beta + Zd + Wp_e + e$ 

Y is a vector of observations,  $\beta$  = is a vector of fixed effects with incidence matrix X.

d ~ NID (0, I<sub>c</sub>  $\sigma^2_{pe}$ ) is a vector of direct additive genetic effect with incidence matrix Z, Pe ~ NID (0, A  $\sigma^2_d$ ) is a vector of random maternal permanent environmental effects with incidence matrix W, and e ~ NID (0, I<sub>n</sub>  $\sigma^2_e$ ) is a vector of random residual effects. In addition,  $\sigma^2_d$  is the direct additive genetic variance;  $\sigma^2_{pe}$  is the maternal permanent environmental variance,  $\sigma^2_e$ . is the residual variance (temporary environment), A is the additive relationship matrix, I<sub>c</sub> and I<sub>n</sub> are identity matrices of order equal to the number of maternal permanent environmental effects and the number of records, respectively.

Best linear unbiased prediction (BLUP) of estimated breeding values (EBV's) was calculated by backsolution using the MTDFREML programme for all animals in the pedigree file for multi-traits analysis. Selection index was used to compare the accuracy of selection for mastitis resistance when selection is based on SCS, MY or combination of both. Accuracy was defined as the correlation between the true breeding value using genetic and phenotypic covariances estimated in the first lactation.

# **RESULTS AND DISCSSION**

#### **Descriptive statistics:**

Means and standard deviations for MY and SCS in different lactations are presented in table 1. As expected, MY, increased until fourth lactation and so did the corresponding standard deviations. Likewise, increases in SCS with parity were observed. This is in covenant with results of Carlén *et al.*, (2004) and El-Awady and Oudah, (2011). The present milk yield increased with increase lactation order until the fourth lactation and then decreased (Table 1).

Koivula *et al.*, (2005) added that the increase of SCS or SCC in the milk leads to increase of mastitis infection, being 11.8% and 14.9% for first and second lactations. The same trend obtained by El-Arian and El-Awady (2008) with SCC of Friesian cows in Egypt, they found the frequencies of mastitis infection occurrence due to increase of SCC were 9.9%, 11.7%, 14.6% and 21.5% in

first, second, third and all lactations, respectively. All fixed effects were significantly affect (P<0.05 or P<0.01) on each of SCS and MY exclude the impact of month of calving on MY was not significant.

Table 1. Means and standard deviation for SCS and<br/>MY in the first five lactations of Holstein<br/>Friesian cows.

Trait	SCS	MY
Lactation	$\overline{X}_{\pm SD}$	$\overline{X}_{\pm SD}$
1	$6.48 \pm 0.886$	$4517 \pm 840$
2	$6.57 \pm 0.863$	$4953\pm978$
3	$6.62\pm0.981$	$5532 \pm 1015$
4	$6.63 \pm 1.016$	$5804 \pm 1127$
5	$6.60 \pm 1.015$	$5640 \pm 1082$
All	$6.62\pm0.971$	$5467 \pm 962$

Genetic parameters:

# Heritability (h<sup>2</sup>):

Estimates of heritabilities for SCS and MY are presented in Table 2 and 3. The estimates of heritability obtained for SCS ranged from 0.11 to 0.23 for first five lactations (Table 2). The present estimates were located well in the range from 0.08 to 0.23 which reported by (Carlén *et al.*, 2004, Al-Seaf *et al.*, 2007, El-Awady and Oudah, 2011 and Strapáková *et al.*, 2016). However, the higher range of  $h^2$  estimates for SCC reported by Mrode and Swanson (1996) (0.05 and 0.47) for  $1^{st}$  and  $2^{nd}$  lactations, respectively.

Table 2. Estimates of  $h^2$  (diagonal), phenotypic correlations ( $r_p$ ) (below diagonal) and genetic correlations ( $r_g$ ) (above diagonal) for SCS and MY traits in the first fifth lactations (1, 2, 3, 4 and 5) of Friesian cows.

Trait	SCS	MY
SCS		
1	$0.21 \pm 0.23$	0.60 (0.13)
2	$0.20 \pm 0.23$	0.65 (0.14)
3	$0.23 \pm 0.19$	0.73 (0.14)
4	$0.15 \pm 0.13$	0.73 (0.14)
5	$0.11 \pm 0.09$	0.81 (0.17)
MY		
1	-0.30	$0.35 \pm 0.19$
2	-0.23	$0.35 \pm 0.19$
3	-0.28	$0.30 \pm 0.13$
4	-0.25	$0.21 \pm 0.13$
5	-0.15	$0.23\pm0.17$

\* S.E for phenotypic correlations ranged from 0.07 to 0.12, 0.06 to 0.09, 0.07 to 0.10, 0.09 to 0.0.13 and 0.07 to 0.11 for first, second, third, fourth and fifth lactation, respectively.

\*\* S.E for genetic correlations are between parenthesis.

\*\*\* 1 = first lactation, 2 = second lactation, 3 = third lactation, 4 = fourth lactation and 5 = fifth lactation.

Table 3. (Co)variance components estimates for SCS and MY traits of Friesian cows in all lactations.

Componenta	Tr	aits
Components	SCS	MY
$\sigma^2_d$	0.9683	0.8993
$\sigma^2_{pe}$	1.2507	0.4640
$\sigma_e^{2^r}$	0.9970	0.7904
$\sigma_p^2$	3.2161	2.1537
$h^2_d$	0.30±0.19	0.41±0.27
c <sup>2</sup>	0.39	0.22
e <sup>2</sup>	0.31	0.37

 $\sigma_d^2$  = direct additive genetic variance,  $\sigma_{pe}^2$  = maternal permanent environmental variance,  $\sigma_e^2$  = residual,  $\sigma_p^2$  = phenotypic variance,  $h_d^2$  = direct heritability,  $c^2$  = fraction phenotypic variance due to permanent environmental effect and  $e^2$  = fraction phenotypic variance due to residual effects. The present estimate of heritability for SCS in all lactations was 0.30 as shown in Table 3. This is agreeing with Koivula *et al.*, (2005). Carlén *et al.*, (2004) reported that heritability of SCS decreased from 0.14 to 0.10 when parity increased from 1 to 3. Whereas Da *et al.*, (1992) reported an increase in heritability of SCS from 0.05 to 0.11 for the same parities.

Heritabilities of MY were moderate (0.21 to 0.35) and decrease with increasing lactation order from 1 to 4 (Table 2) and was 0.41 for all lactations as in (Table 3). The parallel trend was found by Carlén *et al.*, (2004) and Koivula *et al.*, (2005). The same trend also reported by El-Arian and El-Awady (2008). The present  $h^2$  for MY was decrease with increasing lactation number from 1 to 4 lactations (0.35 to 0.21). Additionally, El-Awady and Abu El-Naser (2017) on Friesian cows, estimated  $h^2$  for MY as 0.43.

### Genetic and phenotypic correlations:

As shown in Table 2,  $r_g$  SCS and MY were positive and increased with increasing parity. However, the magnitude of these correlations varied considerably between two traits in different parities. On the other set of data in Sakha farm, El-Arian and El-Awady (2008) reported that the SCC and mastitis had strong positive genetic correlations, ranging from 0.72 to 0.83 with highest estimate found in the third lactation. They added that, these correlations indicated that high SCS is genetically accompanied with low resistance to mastitis infection and both are one expression of the udder health and hence SCS could serve as an indirect criterion of selection improve resistance to mastitis.

The highest estimate of genetic correlation was between SCS and MY traits were in the later (fifth) lactation. However, Carlén *et al.*, (2004) reported that the highest  $r_g$  between milk and SCS was found in the 1<sup>st</sup> lactation. In Addition, Rupp *et al.*, (2011) on Saanen sheep breed, found low  $r_g$  between SCS and MY traits were generally low (0.12).

The present phenotypic correlations between SCS and milk production within parities was small and negative estimates, ranging from -0.30 to -0.15 (table 2). This implies that high SCS or SCC and mastitis incidence decrease milk production slightly. These current results were consistent with those estimated by Carlén *et al.*, (2004); Koivula *et al.*, (2005) and El-Arian and El-Awady, (2008).

However, (Banos and Shook, 1990; Pösö and Mäntysaari 1996 and El-Awady and Oudah 2011) reported that the rg between SCS or SCC and milk production, changed from positive in 1st lactation, to negative in the later lactations. Generally, the genetic relationship between occurrence of mastitis and milk traits is unfavorable and elevates with progress of the parities (Uribe et al., 1995, Nielsen et al., 1997, Heringstad et al., 1999, Rupp and Boichard, 1999 Heringstad et al., 2000, Hansen et al., 2002, Carlén et al., 2004, Koivula et al., 2005 and El-Arian and El-Awady, 2008). Two alternatives available for explaining these changes: i) Partly different genes affect SCS in first versus later lactations because different pathogens may be responsible for occurrence of mastitis (Banos and Shook, 1990). ii) Culling practices, especially during first lactation remove low-producing cows with high chance of occurrence of mastitis and high level of SCC, but Carlén *et al.*, (2004) expected no effect of culling practice on changes in genetic correlations.

The linear relationship between SCS and mastitis reported by Philipsson *et al.*, (1995), Nash *et al.*, (2000) and Koivula *et al.*, (2005), confirmed the possibility of improving resistance to mastitis by selecting for low SCS. In this respect, El-Awady and Oudah, (2011) on Frisian cattle concluded that, if the direct information on the udder health traits is not accessible, SCC measures can be included in the selection criteria to improve the revenue from dairy cattle.

On the contrary, few researches have projected a curvilinear association between the risk of the udder mastitis and SCS, which means that the risk of udder mastitis intensifications if the SCC droplets under some critical level (Kehrli and Shuster, 1994, and Peeler *et al.*, 2003).

The genetic correlations between SCS across lactations ranged from 0.79 to 0.95 and between MY, ranged from 0.80 to 0.93 as shown in Table 4. The  $r_g$  in all cases were much higher than the phenotypic correlations and those between the second and the third lactation were the higher. This suggested that susceptibility to mastitis or the SCS remain consistent at the same level across lactations. Mrode and Swanson (2003), Carlén *et al.*, (2004) and Koivula *et al.*, (2005) found similarly high positive genetic correlations across lactations for SCS.

 Table 4. Estimates of rg and rp between SCS and MY across the first fifth lactations of Friesian cows.

		Trait/c	orrelations			
Lactations	SCS	SCS		MY		
	rg	rp	$\mathbf{r}_{\mathbf{g}}$	rp		
1-2	0.91 (0.22)	0.51	0.88 (0.25)	0.63		
1-3	0.90 (0.19)	0.61	0.86 (0.21)	0.66		
1-4	0.89 (0.21)	0.59	0.83 (0.21)	0.49		
1-5	0.83 (0.20)	0.57	0.88 (0.23)	0.48		
2-3	0.95 (0.24)	0.63	0.93 (0.26)	0.59		
2-4	0.79 (0.20)	0.63	0.90(0.25)	0.55		
2-5	0.80 (0.23)	0.59	0.81 (0.23)	0.60		
3-4	0.91 (0.23)	0.51	0.88 (0.27)	0.62		
3-5	0.87 (0.19)	0.60	0.80(0.27)	0.58		
4-5	0.90 (0.24)	0.64	0.93 (0.25)	0.62		

\*S.E for genetic correlations are between parentheses.

\*\*S.E for phenotypic correlations ranged from 0.09 to 0.19.

### Predicted breeding values (EBV's):

Expected breeding values (EBV's), standard errors and accuracy for SCS and MY are in Table 5. The predicted breeding values (PBV'S) in present study for sires, varied from -0.618 to 0.509 for SCS and -453 to 323 kg for MY. While, PBV'S for cows ranged between -0.838 to 0.104 for SCS and -430 to 513 kg for MY. Similarly, PBV'S of dams ranged from -0.207 to 0.237 for SCS and -76 to 123 kg for MY.

Accuracy of PBV'S, of sires, cows and dams ranged from 64 to 84%, 42 to 78%, and from 17 to 53%, respectively. These stated the genetic improvement for MY and SCS could be attained with any both of sires, or cows or dams and shown the estimates of PBV'S could base on any of sires or cows or dams to estimated breeding values. These results agree with estimated by (Abu El-Naser 2019 and 2020) in Egyptian buffaloes.

Negative EBV's values mean that the daughters of a given sire have lower SCS than other sires and vice versed Koivula et al., (2005) indicated that the curvilinearity between mastitis and SCS is bring about by the nature of SCS as a mastitis contagion signal and seem to be a weak relation of bulls with predicted breeding values (EBV's) less of average because for the lower end of the curve, the slope appear to be much less, while of bulls for EBV's average and the relationship be sturdy. highest of However, (Boettcher et al., 2002, Carlén et al., 2004, El-Arian and El-Awady, 2008 and Strapáková et al., 2016) they noted that cows with low SCC does not occur increased of susceptibility to clinical mastitis. As well as, when has been taken the duration and severity of mastitis into account, it was cleared that daughters of sires have transmitting abilities high of SCC have sever and long constant clinical episodes (Nash et al., 2000 and Koivula et al., 2005). Furthermore, Rupp et al., (2000) and Koivula et al., (2005) suggested that cows in the first lactation have low mean of SCS. Also have in later; lactations low risk of clinical mastitis, thus, should be deign cows with low SCS from breeding goals. El-Awady et al (2016) Egyptian buffaloes, obtained the PBV's of sires, cows and dams for TMY and SCC were varied from -687 to 543 kg, -146,000 to 371,000 cell/ml, -869 and 844 kg, 21,600,195,000 cell/ml, -450 to 430 kg, and -125,000 to 407,000 cell/ml, respectively.

The present results revealed the animals of high SCS should be greater vulnerable to mastitis and culling practices that remove low producing cows with high SCS, make the remaining cows enter their second and later lactations have high milk yield vice versa for SCS. Therefore, the goal of reducing mastitis and its unfavorable genetic relationship between production and health should be taken into account. El-Arian and El-Awady, (2008) reported that the highest EBV's for SCC were associated with those highest for mastitis, whereas the EBV's for SCC was -24.6x10<sup>3</sup> cells/ml. Moreover, they added that negative EBV's values mean that the daughters of a given sire have lower SCC or mastitis than other sires.

 Table 5. Range of breeding values through Sires (SBV's), Cows (CBV's), and Dams (DBV's) and its accuracy's (%) for SCS and MY traits of Holstein Friesian cows.

	Estimate of breeding values (EBV's)					
Traits	Sires					
	Min±SE	Max±SE	Range	Accuracy%	Positive%	Negative%
SCS	-0.618±0.58	0.509±0.59	1.127	64-76	54.2	45.8
MY	-453±300	323±340	776	76-84	51.0	49.0
Cows						
SCS	-0.838±0.74	0.104±0.73	0.942	42-66	47.7	52.3
MY	-430±320	513±340	943	49-78	46.2	53.8
Dams						
SCS	-0.207±0.18	0.237±0.108	0.387	30-52	49.6	49.0
MY	-76±34	123±37	199	17-53	50.4	48.1

The correlations between breeding values of all traits (table 6). The correlations trend of predicted breeding values were in the same direction with those reported for genetic correlations for the same traits (Table 2). The correlations between the values of EBV's for SCS and MY within each path of breeding value (table 6) indicated that the trend of correlation within each path was nearly the same, since it was from 0.80 to 0.87 between them. All the correlation coefficients were positive and high significant, which revealed that the trend with each path was the same.

Table 6. Correlations between the values of breeding values of SCS and MY within each path of sires cows and dams

sires, cows and dams.		
Path	Correlations between values of EBV's SCS and MY traits	
Sire BV's	0.87**	
Cow BV's	0.80**	
Dam BV's	0.80**	

# Selection for resistance of mastitis

Table (7) show comparison of the accuracies of selection for mastitis resistance when selection is based on SCS or MY only or a combination of both SCS with MY using (Co)variances and parameters estimated from the first lactation data. The accuracy of the index number (3) which included SCS, with MY was (0.79) higher than those for other two indices and similar should be more efficient in improving mastitis resistance than direct selection on SCS or MY only. Other indices were nearly similar and close to those reported by Strandberg and Shook (1989). The results obtained by Philipsson *et al.*, (1995) and Carlén *et al.*, (2005) confirm that both SCS and MAST should be taken in the selection criterion for improve resistance to udder mastitis in dairy cattle.

Table 7. Accuracies (r<sub>IH</sub>) of selection for improve resistance to udder mastitis based on only SCS or MY and combinations of SCS with MY in the selection index of Holstein Friesian.

Index	Traits in index	r <sub>IH</sub>	
1	MY	0.49	
2	SCS	0.43	
3	SCS, MY	0.79	

El-Arian and El-Awady, (2008) found that the antagonism between udder fitness and production accentuates the need to select for strong udder mastitis resistance to prevent the increase in the frequency of occurrence mastitis as a consequence of selection for yield only. They also added that the moderate heritabilities of SCC (0.08 to 0.15) and their high positive genetic correlation with mastitis (0.72 to 0.83) make possible to use form as an indirect criterion to select against mastitis.

The present results indicate that the increased level of SCS and the high genetic correlations among SCS within lactations (0.79 to 0.95) assure the necessity of adopting selection programme seeking the improvement of mastitis resistance early in the animal life and continues in all lactations. Waiting for later lactations might create prolonged problems with respect to cow resistance to mastitis in later lactations while the latter should be improved if only the first lactation records were used.

# CONCLUSION

Producers may have unreasonable expectations from selection based on genetic evaluations for SCS. It is the responsibility of health professionals and geneticists in the dairy industry to guide producers in the optimal use of SCS assessments. The discrepancy between the healthy features of the udder and the production of milk underscores the need to select strong mastitis resistance to prevent the increase in the frequency of occurrence mastitis as a consequence of selection for yield only. The moderate heritabilities of SCS (0.11 to 0.23) and their high positive genetic correlation with MY (0.60 to 0.81) make possible to use form as an indirect criterion to select against mastitis. The increased level of SCS with parity and the high genetic correlations among inter lactations SCS (0.79 to 0.95) assured the necessity of adopting selection programme seeking the improvement of mastitis resistance early in the animal life and continues in all parities. Waiting for later lactations might create prolonged problems with respect to cow resistance to mastitis in later lactations while the latter should be improved if only the first lactation records were used.

When mastitis records are existing, direct selection can be made against mastitis and information on SCS can be used as an associated trait when estimating EBV's. The high genetic correlations between MY and SCS suggested a multitrait model with 1<sup>st</sup> and/or later lactation to select against SCS and then mastitis. In addition, if information on both traits (SCS and mastitis) are obtainable, a combined assessment will lead to more accurate prediction of BV's for resistance to mastitis.

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العلاقات الوراثية بين عدد الخلايا الجسدية وانتاج اللبن في مواسم الحليب الخمسة الأولى في أبقار الفريزيان في مصر عادل فوزي إبراهيم<sup>1</sup>، أيمن فؤاد عاشور<sup>1</sup> وحسن غازي العوضي<sup>2</sup> <sup>1</sup>معهد بحوث الإنتاج الحيواني ، وزارة الزراعة ، الدقى ، مصر. <sup>2</sup>قسم الإنتاج الحيواني - كليه الزراعة – جامعه كفر الشيخ - مصر

تم استخدام سجلات أعداد الخلايا الجسدية (SCS) و إنتاج اللبن في 305 يوم (MY) لأبقار الفريزيان الموجودة في مزرعة سخا والقرضا خلال الفترة من 2000 إلى 2017 لتقدير العلاقات بين الـ SCS و MY بين وداخل مواسم الحليب الخمسة الأولى. تم تقدير المعايير الوراثية باستخدام طريقة الاحتمالات العظمى المحددة (MTDFREML) بنموذج الحيوان. تر اوحت تقديرات معامل التوريث للـ SCS من 1.10 إلى 2.20 لمواسم الحليب الخمسة الأولى بينما كانت من 2.01 إلى 3.00 للـ MY. قدر المكافئ الوراثي للمواسم مجتمعة بـ 3.00 و 0.10 للـ SCS و الـ MY على التوالى. كانت الارتباطات الوراثية بين الـ SCS و My معتدلة إلى عالية و إيجابية (0.60 إلى 0.81) في مواسم الحليب الخمسة الأولى. وزادت الارتباطات الوراثية بين الـ ترتيب موسم الحليب، مما يشير إلى أن الانتخاب للـ MY سيقلل من الـ SCS و بالتالى يقلل من حدوث التهاب الضرع. تر اوحت دقة القيمة التربوية المتوقعة ترتيب موسم الحليب، مما يشير إلى أن الانتخاب للـ MY سيقلل من الـ SCS وبالتالى يقلل من حدوث التهاب الضرع. تر اوحت دقة القيمة التربوية المتوقعة الرتيب موسم الحليب، مما يشير إلى أن الانتخاب للـ MY سيقلل من الـ SCS وبالتالى يقلل من حدوث التهاب الضرع. تر اوحت دقة القيمة التربوية المتوقعة الرتيب موسم للحليب، مما يشير إلى أن الانتخاب للـ MY سيقلل من الـ SCS وبالتالى يقلل من حدوث التهاب الضرع. تر اوحت (SVB) من 4.64 إلى عالية و و 4.00 إلى 3.01 إلى الاماء و الأمهات على التوالى، مما يشير إلى أنه يمكن تحقيق التحسين الوراثي من خلال أى الاتيب موسم الحليب، من الـ SCS و من 30 إلى 25.3 للأباء، الأبقار و الأمهات على التوالى، مما يشير إلى أنه يمكن تحقيق التحسين الوراثي من خلال أى مسار لها. أيضا كان اتجاه الار تباط بين القيم التربوية المتوقعة في نفس الاتجاه مع تلك التي تم تقديرها في الارراثية لنفس الصفات. إن انتخاب البورة على أسلس قيم التربية المتوقعة سوف يؤدى إلى الحاف في الارحابة بالتهاب الضرع وزيادة الار تباط بين الوراثي من خلال أى على أسلس قيم التربية لمتوقعة سوف يؤدى إلى الذي توية الاتحالى يقل من الإصابة بالتهاب الصرع وزيادة الـ MY. ولن مع الربية الحليب. أوضحت النتائج أن أخذ أعداد الخلايا الجسمية وكذلك حالة الضرع الصحية في الاعتبار عذا القول العوب يقال الحلي عن الحلي عالم رع قرم المولي وزيامة التربية في أبقار الحليب. أوضحت التائج الذ