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Chapter 3

SOMATIC CELL COUNT AS THE FACTOR CONDITIONING PRODUCTIVITY OF VARIOUS BREEDS OF COWS AND TECHNOLOGICAL SUITABILITY OF MILK

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ABSTRACT

Early detection of mastitis with subclinical symptoms is possible by determining somatic cell count (SCC). SCC is the most widely accepted indicator of the mammary gland health as well as milk quality and its technological suitability. The authors' research has revealed that an increase of SCC (independently of a breed of cows) mainly causes a rise in a total crude protein content and a distinct reduction in lactose level ($P \leq 0.01$). Moreover, SCC also lengthens the time of milk enzymatic coagulation ($P \leq 0.01$) but it does not influence its thermal stability. Distinct negative relationship between casein content and SCC is confirmed by relatively high value of correlation coefficient ($r = -0.59$). In

the authors' studies the significant interactions (breed of cows x SCC) for the daily yield of cows, content of protein, casein and lactose, protein to fat ratio and rennet-induced milk coagulation time also have been stated, which indicates a differentiated response of various breeds of cows to udder inflammations. Holstein-Friesian cows are more sensitive to decline of daily yield, that is reflected in higher negative value of correlation coefficient between SCC and milk yield (-0.245). In Simmental and Jersey cows the correlations were negative as well but their values were substantially lower ($r=-0.123$ and $r=-0.148$) and statistically insignificant. With the age of cows increase in SCC was noted and in the cows of local breeds (Polish Red, Polish Black and White, Whitebacked) and Jersey that rise was much smaller in comparison to Polish Holstein-Friesian cows. Significant interaction ($P\leq 0.05$) for SCC between breed of cows and subsequent lactation was indicated. However, the significant changes in milk constituents were recorded only when the SCC exceeded 500 thous. ml^{-1} , that is in milk that does not meet the current regulatory quality standards.

Somatic cell count also affects the changes in whey protein content. Rise of SCC decreased the content of major albumins, i.e. alpha-LA and beta-LG, by small degree, and that was confirmed by very low statistically insignificant correlation coefficients ($r=-0.07$ i $r=-0.05$). Negative value of both correlations, though, indicates a direction of changes and may imply that in more advanced stages of udder diseases the decrease of milk proteins is likely to be higher. However, with rise of SCC, content of immunoreactive proteins (lactoferrin and lysozyme) as well as bovine albumin serum (BSA) significantly increased. The significant impact of SCC on content of these proteins in milk is confirmed by relatively high positive values of computed correlation coefficients (lactoferrin $r=0.65$, lysozyme $r=0.63$ and BSA $r=0.59$). In the case of BSA that correlations were clearly differentiated in particular breeds of cows, i.e. $r=0.711$ for Holstein-Friesian, $r=0.577$ for Simmental and $r=0.472$ for Jersey. Thus, it can be assumed that there is a differentiated degree of permeability of mammary gland cell membranes in cows of various breeds.

1. INTRODUCTION

Mammary gland inflammations are induced by as many as even over 140 various types of microorganisms inhabiting both animal and its environment [Malinowski and Kłossowska, 2000]. The cause of mastitis can be a variety of microorganisms such as bacteria, mycoplasmas, yeast like fungi, algae and in the rare cases viruses [Bradley, 2002; Khan and Khan, 2006]. The principal

etiological factors inducing the inflammation of mammary gland of cows are bacteria. The severity of clinical symptoms and course of mammary gland inflammation is largely dependent on the type of bacteria causing the infection. With regard to the etiological factor, a classic division of mastitis into two categories, depending on a method of infection, was established, i.e. mammary gland inflammation caused by “infectious” factors (e.g. *Staphylococcus aureus*) and “environmental” (e.g. *Escherichia coli*). Microorganisms inducing the infectious mastitis exist on the skin of udder and they are well adapted to a survival and proliferation in host organism, in particular in the mammary gland. Nevertheless, they are not adjusted to life outside the body of a host. These bacteria are primarily responsible for subclinical and chronic inflammation of the mammary gland. The “environmental” factors, i.e. bacteria living in an environment of cows (bedding, water, earth), are less virulent pathogens and generally rapidly eradicated by the immune system of a host [Blowey and Edmondson, 1995; Bradley, 2002]. However, *Staphylococcus aureus* and *Streptococcus agalactiae* as well as the environmental bacteria, mainly from the *coli* group: *Escherichia coli* (most frequently) and *Klebsiella pneumoniae* perform a primary function in the inflammation induction [Smulski et al., 2011].

According to Klastrup et al. [1987], the susceptibility to mastitis was in 25% a result of environmental factors, 20% – genetic factors and 50% – herd management.

2. SOMATIC CELL COUNT AS THE INDICATOR OF MAMMARY GLAND HEALTH STATUS

In numerous research it was shown that somatic cell count (SCC) in cow milk is a good indicator of mammary gland health status. Furthermore, occurring subclinical and clinical inflammations are associated with the significant increase in the count of somatic cells [Rainard and Riollot, 2006]. This fact was confirmed by generally high positive correlation coefficients between SCC and inflammation status ($r=0.30-0.97$) [Carlen et al., 2004; Bloemhof et al., 2009].

It should be emphasized that interbreed differences in the susceptibility of cows to mastitis exist. Rupp and Boichard [2003] reported that cows of dairy breeds originating from the eastern France (Montbéliarde and Abondance) and central Europe (Simmental and Brown Swiss) are less frequent in clinical form

of mastitis and their milk contains lower level of somatic cells compared to Holstein breed. Gołębiowski and Brzozowski [2007], who claimed that Montbéliarde breed characterizes a higher susceptibility to mastitis than Holstein, due to the lower by 23-38% somatic cell count in milk, also confirmed this fact.

The SCC of milk includes leucocytes derived from the blood (75%), i.e. neutrophils, macrophages, lymphocytes and erythrocytes, and epithelium cells of udder (25%). In milk obtained from a healthy udder leucocytes are consisted in $50\pm 10\%$ of neutrophils (polymorphonuclear granulocytes – PMNs), in $36\pm 9\%$ of lymphocytes and in $14\pm 2\%$ of macrophages. It should be noted that the percentage of particular types of cells varies depending on the udder health, age and stage of lactation. Share of leukocytes increase in response to bacterial infection, tissue injury, stress and final stadium of lactation. Leucocytes are transported into the milk gland directly from the blood as a response to the chemical substances, released by mammary glands during inflammation [Aniulis et al., 2003; Sharma et al., 2011]. The increase in somatic cell count in milk, primarily neutrophil granulocytes (neutrophils), is the first signal informing about changes in the health status of mammary gland. In uninfected quarters of mammary gland, i.e. without mastitis, SCC is lower than $150 \text{ thous. ml}^{-1}$, at simultaneously lower percentage of neutrophils (5-25% of the total SCC). It is assumed that the somatic cell count in cow milk exceeding $150 \text{ thous. ml}^{-1}$ is one of the first signals of mammary gland infection. During inflammation an elevation in milk SCC is mainly conditioned by the increase of share of the polymorphonuclear cells by 99-100% [Schukken et al., 2003; Tao and Mallard, 2007]. Scheppers et al. [1997] established the so called “physiological threshold level”, i.e. the SCC limit of $200 \text{ thous. ml}^{-1}$, to differentiate between a healthy and infected quarter. Furthermore, they stated that each doubling of SCC above $50 \text{ thous. ml}^{-1}$ results in losses in milk production. Griffin et al. [1987] established, however, the SCC lower limit of subclinical stage of mastitis at $125 \text{ thous. cells ml}^{-1}$, whereas the upper limit at $250 \text{ thous. cells ml}^{-1}$. Whereas Kherli and Shuster [1994] as a limit value of SCC $100 \text{ thous. ml}^{-1}$ and Harmon [1994] accepted only $50 \text{ thous. ml}^{-1}$.

For the dairy industry the most pernicious are subclinical (non-symptomatic) inflammations since milk obtained often becomes the commercial milk. In Europe, in accordance to the Commission Regulation (EC) No 1662/2006 of 6 November 2006 (No L230/4) modifying the Regulation (EC) No 853/2004 of the European Parliament and of the Council of 29 April 2004, milk intended for human consumption should not contain

more than 400 thous. somatic cells ml^{-1} . Furthermore, the identical limits apply in Canada and New Zealand. In the U.S. the official standard was up to 750 thous. cells ml^{-1} until 2011, however, a gradual reduction of SCC limit to the requirements of the European Union is planned, in order to allow the American producers to export dairy products to European markets [National Mastitis Council, 2011].

3. ECONOMIC EFFECTS OF UDDER INFLAMMATION

Mastitis remains the most expensive and hard to overcome by veterinary methods the disease affecting dairy cattle. According to SABRE [2006], the total losses due to mastitis in the dairy industry in the EU in 2005 amounted to EUR 1.55 billion.

Bovine mammary gland inflammations result not only in reduction of milk synthesis but also in alterations in milk constituents, leading to deterioration of its nutritive value and technological suitability. Simultaneously, they belong to the most common causes (except for infertility and lameness) of culling of cows from herd. The udder inflammations constitutes approximately 70% of all economic farm losses [Huijps et al., 2008]. Costs associated with the mammary gland inflammation differentiate depending on the etiological factor, course of clinical form, animal and milk prices, fodders, services and drugs [Halasa et al., 2007; Malinowski et al., 2011]. In various countries these costs are varied, however, each time they are a large financial burden in dairy farms (Table 1). For instance, in the United States in the case of one clinical mastitis the costs are estimated at USD 107 and in Sweden even the USD 735 [Malinowski et al., 2011]. The size of losses associated with an occurrence of mammary gland inflammations also results in a number of cows in herd and a form of mastitis. Huijps et al. [2008] reported that in smaller herds, consisting of approximately 30 dairy cows, losses were definitely lower (EUR 17 /cow per year) in relation to the herds comprising of approximately 160 heads (EUR 198 /cow per year). In the extreme cases, at the presence of inflammations in clinical form, the average losses were estimated at EUR 210, varying from EUR 164 to EUR 235 /cow/month, respectively, in the final and the initial stage of lactation. In the case of subclinical affliction in the herd of cows with productivity of 8,500 kg/cow/305 days of lactation, at SCC in bulk tank milk amounted to approximately 200 thous. ml^{-1} , losses were estimated at EUR 20 /cow per year. Chassagne et al. [2005] and Aniulis et al. [2003] stated that the most susceptible to mastitis were animals in the initial period of lactation.

Table 1. Costs bearing to treatment and prevention of mastitis according to various authors

Cost	Mastitis status	Additional information	Country	References
Treatment				
EUR 28 /cow/year		average cost	USA, Michigan	Kaneene and Hurd, 1990
EUR 31 /cow/year		average cost	USA, Ohio	Miller and Dorn, 1990
EUR 22 /cow/year		average cost	USA, California	Sischo et al., 1991
EUR 279 /cow/year		average cost in summer	England	Hillerton et al., 1992
EUR 102 /cow/year	subclinical	average cost	England	McInerney et al., 1992
EUR 20 /cow/year		average cost	Germany	Reinsch and Dempfle, 1997
EUR 287 /average case of cow/year		average cost	England	Kossabati and Essleniant, 1997
EUR 26 (19-32) /cow/year		cost of control of udder health status because of mastitis	France	Fourichon et al., 2001
EUR 65-182 /cow/year	subclinical and clinical		the Netherlands	Huijps et al., 2008
EUR 117 /cow/lactation			Spain	Pérez-Cabal et al., 2008
EUR 71 /cow/year			USA, New York	Bar et al., 2008
EUR 179 /cow		cost of complete treatment	USA, New York	Bar et al., 2008
EUR 78 (17-198) /cow/year		in dependence on herd size, assessed by farmers	the Netherlands	Huijps et al., 2008
EUR 210 (164-235) /cow/month	clinical	cost in () respectively in the final and the initial stage of lactation	the Netherlands	Huijps et al., 2008

Cost	Mastitis status	Additional information	Country	References
EUR 275 /cow/year	Clinical		Sweden	Nielsen, 2009
EUR 60 /cow/year	subclinical		Sweden	Nielsen, 2009
USD 224-275 /cow	clinical	average total cost of treatment	the Netherlands and Canada	Steeneveld et al., 2011
USD 107 (161.8-344.2)	clinical		USA	Malinowski et al., 2011
USD 735	clinical	cost of treatment of one case	Sweden	Malinowski et al., 2011
EGP* 15.80±5.25 – 1734±281 /cow/lactation		in dependence on SCC, respectively: ≤50 – >2000 thous. cells ml ⁻¹	Egypt Friesian	El-Awady and Oudah, 2011
Prevention				
EUR 3.56 (0-22) /cow/year			USA, Michigan	Kaneene and Hurd, 1990
EUR 4-12 /cow/year			USA, Ohio	Miller and Dorn, 1990
EUR 4 /cow/year			USA, California	Sischo et al., 1991
EUR 3 /cow/year			Germany	Reinsch and Dempfle, 1997
EUR 50 /cow/year			Sweden	Nielsen, 2009

*EGP – Egyptian pounds.

According to the French data [Chassagne et al., 2005], it results that in the first 30 days after calving, even in a well-managed herd, the percentage of those animals amounts to approximately 30%. This is due to an attenuation of immune defense reaction of cow in a perinatal period and more frequent incidence of mammary gland inflammation associated with that. According to the data provided by SABRE [2006], in the European Union 30% of cows remains infected by mastitis in 2005. Furthermore, the analyses conducted in the same period in Poland proved that the percentage of animals with symptoms of mastitis is at a similar level but clinical forms of the disease are recognized in 2-5% of cows [Głowacki, 2006]. The research conducted in Lithuania by Aniulis et al. [2003] demonstrated that about 42-47% of cows in

that country had symptoms of subclinical mastitis. It should be emphasized that subclinical states are more serious problem in dairy herds. Averagely approximately 40% of cows are constantly affected by changes specific to subclinical mammary gland inflammations [Malinowski and Kłossowska, 2000]. The changes are usually long-term and often unrecognized by farmer and ipso facto undiagnosed, especially in herds without SCC monitoring. According to Östensson et al. [2012], the degree of spreading of subclinical form of mastitis is very high, both on the level of quarter (63.2%) and cow (88.6%). However, these results are much higher than those obtained by other authors [Nam et al., 2010; Petrovski et al., 2011], i.e. 35% and 55%, respectively.

4. EFFECT OF SCC ON PRODUCTIVITY OF COWS AND BASIC COMPOSITION OF MILK

Susceptibility or resistance to mastitis of dairy cows is genetically conditioned. The genetic antagonism between milk yield and udder health status has been proved, resting on a deterioration of health status of mammary gland as a result of selection for increase in milk yield [Rupp and Boichard, 2003].

The disease is therefore a problem in high productive herds, in which an intensive husbandry conditions are used. However, direct selection for mastitis resistance is not widely applied because of its low heritability. The results of numerous research (Table 2 and 3) indicate that the values of heritability for somatic cell count (SCC) or natural logarithm of SCC (SCS – somatic cell score) are not exceeded 0.20. However, the results obtained by Dube et al. [2008] point to lower value of heritability in primiparous Jersey cows ($h^2=0.07$) and slightly higher at multiparous cows ($h^2=0.11$) (Table 3). The heritability estimates for udder inflammations are even lower than for SCC (Table 3). Low values of heritability for susceptibility of cows to mastitis do not mean, however, the absence of genetic variation. Nevertheless, the environmental factors strongly influence on phenotypic effect.

Inflammation primarily results in a reduction in milk production. Seegers et al. [2003] reported that the total loss in milk production arising from clinical mastitis was 375 kg, i.e. approximately 5% of lactation yield. However, production losses were highly variable and mainly eventuate from a lactation period in which cows suffering from mastitis.

Table 2. Heritability estimates (h^2) and genetic correlations (r) among lactational somatic cell score (SCS) [Dube et al., 2008]

	Heritability estimates			Genetic correlations		
	SCS ₁	SCS ₂	SCS ₃	SCS ₁	SCS ₂	SCS ₃
SCS ₁	0.07±0.01				0.82±0.09	0.85±0.05
SCS ₂		0.11±0.01				0.96±0.06
SCS ₃			0.11±0.02			

SCS₁ – SCS for first lactation; SCS₂ – SCS for second lactation; SCS₃ – SCS for third lactation.

Table 3. Heritability estimates (h^2) for somatic cell count and mastitis

h^2	Parameter	Additional information	References
0.16	SCC	Holstein	Welper and Freeman, 1992
0.18	SCC	Danish Holstein	Lund et al., 1994
0.11±0.04	SCC		Mrode and Swanson, 1996
0.10-0.14	SCC		Mrode et al., 1998
0.136±0.028 0.096±0.016	SCC	during the first lactation for all lactations	Sender, 2001
0.06±0.02	SCS	Finnish Ayrshire	Ikonen et al., 2004
0.11-0.22	SCS		Ptak et al., 2007
0.07 0.11	SCC	in primiparous cows in multiparous cows Jersey	Dube et al., 2008
0.14-0.15	SCC	Danish Holstein, Danish Red and Danish Jersey	Norberg et al., 2009
0.07-0.14	SCC		Ptak et al., 2009
0.05 (0.01- 0.11)	SCS	Burlina	Penasa et al., 2010
0.15 0.20	SCS	lactations 1 and 2 lactation 3	National Research Institute of Animal Production, Poland, 2010
0.09-0.18 0.11-0.14	SCC	lactations 1-3 daily average for lactations 1-3 Holstein-Friesian	Rzewuska et al., 2011
0.025	mastitis		Lund et al., 1994
ca. 0.04	mastitis		Mrode and Swanson, 1996

Table 3. (Continued)

h^2	Parameter	Additional information	References
0.02-0.04	clinical mastitis		Rupp and Boichard, 1999
0.01-0.17	mastitis		Pösö and Mäntysaari, 1996; Rupp and Boichard, 1999; Carlen et al., 2004; Bloemhof et al., 2009

SCC – somatic cell count; SCS – somatic cell score (natural logarithm of SCC).

Table 4. Genetic (r_g) and phenotypic (r_p) correlation coefficients between milk yield and SCC

Correlation coefficient (r)	Type of correlation	Additional information	References
-0.16 (for SCS) -0.02 (for SCS)	genetic phenotypic	primiparous cows Holstein	Welper and Freeman, 1992
0.14±0.04 (for SCS)	genetic		Mrode and Swanson, 1996
-0.07 (for SCS) -0.13 (for SCS)	genetic phenotypic	test-day milk yield Finnish Ayrshire	Ikonen et al., 2004
-0.19 (for SCC)	beside genetic (phenotypic)		Sawa et al., 2007
0.12 (-0.48-0.91) (for SCS)	genetic	test-day milk yield Burlina	Penasa et al., 2010
-0.55 (for SCC) -0.47 (for SCC)	genetic phenotypic	lactational milk yield Friesian	El-Awady and Oudah, 2011
-0.157 (for SCC, see Table 5)		total for Polish Holstein-Friesian, Simmental and Jersey	Litwińczuk et al., 2011
-0.22 – -0.16 (for SCC)			Jakiel et al., 2011

SCC – somatic cell count; SCS – somatic cell score (natural logarithm of SCC).

Thus, Hagnestam et al. [2007] noticed that the reduction in milk production in the period of 305-day lactation ranged from 0 to 902 kg, depending on the stage of lactation and subsequent lactation, in which the

Swedish Holstein and Swedish Red cows become ill. Hortet et al. [1999] analyzed 32,148 control milkings from 4,968 cows of French Holstein breed, in the milk samples in which SCC was lower than 600 thous. cells ml^{-1} . Value of 50 thous. cells ml^{-1} was accepted as a reference level of somatic cell. The authors found that daily milk yield of primiparous cows decreased by 0.30 kg at the SCC raise up to 100 thous. cells ml^{-1} , by 0.61 kg at SCC to 200 thous. cells ml^{-1} and by 1.09 kg at SCC to 600 thous. cells ml^{-1} . Numerous studies (Table 4) show that an increase in the somatic cell count affects the losses of daily milk production to a lesser extent (r_p from -0.02 to -0.19). Nevertheless, in a case of lactational milk production, however, higher negative correlation ($r=-0.55$) exists.

The study of Litwińczuk et al. [2011] indicate on sundry sensitivity of cow breeds to an increase of SCC in milk, that is confirmed by differences in correlation coefficients obtained between the SCC and daily yield. Out of three breeds, i.e. Polish Holstein-Friesian, Simmental and Jersey, Polish Holstein-Friesian cows were distinguished by the highest sensitivity to udder infections ($r=-0.245$ at $P\leq 0.01$). At the other two cow breeds (characterized by lower productivity), i.e. Simmental and Jersey, the values of r were substantially lower ($r=-0.123$ and $r=-0.148$, respectively).

Increase in the somatic cell count in milk also affects the changes in the basic composition. The most sensitive milk component to mammary gland inflammation is lactose. It is responsible, along with some elements (primarily Na and Cl), for regulating the osmotic pressure in udder, which must be in balance with osmotic pressure of blood [Bleck et al., 2009; Litwińczuk, 2012]. Rise in SCC effects on decline in lactose content, as the result of synthetic activity of mammary gland reduced [Ikonen et al., 2004; Bleck et al., 2009]. This leads to flow of higher quantity of Cl^- and Na^+ from blood to milk and K^+ into the blood to compensate for the osmotic pressure in mammary gland [Bansal et al., 2005; Litwińczuk, 2012]. Forsbäck et al. [2009] reported that at the SCC level ≤ 100 thous. ml^{-1} (healthy udder) lactose content was 4.66 g 100 ml^{-1} and at the $\text{SCC} > 200$ thous. ml^{-1} (threshold physiological limit) – 4.51 g 100 ml^{-1} . The research of Barłowska et al. [2009], carried out on milk obtained from four breeds of cows (Black-White and Red-White variety of Polish Holstein-Friesian, Simmental and Whiteback), proves that a significant decrease in lactose content also followed by the cellular element level ≥ 200 thous. ml^{-1} . Nevertheless, the highest decline in this component percentage (by 0.24%) was recorded at the level of $\text{SCC} \geq 1$ million ml^{-1} ($P\leq 0.01$), while in the Black-White variety of Polish breed Holstein-Friesian cows it was the lowest (by 0.13%) and the highest in Whiteback (0.35%). The similar tendencies were

noticed by Król et al. [2010] and Litwińczuk et al. [2011] in milk of cows of the greatest importance in global milk production (Holstein-Friesian, Simmental and Jersey). Ogola et al. [2007] demonstrated also the reduction in lactose content from 48.8 g l⁻¹ at the SCC level <250 thous. ml⁻¹ to 43.8 g l⁻¹ at SCC>750 thous. ml⁻¹. Relatively high negative correlation coefficients between somatic cell count in milk and lactose concentration (from r=-0.38 to r=-0.59) determined by a number of authors are a confirmation of this relationship [Zumbo et al., 2004; Bansal et al., 2005; Sawa et al., 2007; Bleck et al., 2009; Pazzola et al., 2012]. Juozaitiene et al. [2004] indicated that a correlation coefficient between SCC in milk and lactose concentration was relatively high and oscillated between -0.38 in lactation I and -0.44 in lactation III (P≤0.01), while this dependence for protein or fat content was very low and found within -0.1 – -0.04 range. Lindmark-Månsson et al. [2000] reported (as a few), though, a totally different relationships between log₁₀SCC and lactose percentage (0.073) and higher for fat, and protein level, respectively: r=0.277 and r=0.374 (in milk of Swedish Holstein cows).

With the increase in somatic cell count in milk protein percentage is generally unchanged or could slightly rise. However, significant changes occur in particular fractions. Accordingly to Lindmark-Månsson et al. [2006], concentration of total protein usually remains unchanged at SCC to 1 million ml⁻¹. Whereas, the results of Litwińczuk et al. [2011], obtained on the basis of research performed on milk of cows of four breeds, indicates that content of this component decreased to SCC level in the range of 401-500 thous. ml⁻¹, and then slightly increased (at SCC: 501-1,000 thous. ml⁻¹). El-Awady and Oudah [2011], analyzing monthly and lactational losses in protein production in the milk of Friesian cows, stated that they successively followed and occurred at SCC level >100 thous. ml⁻¹. At SCC>2 million ml⁻¹ the losses amounted to 2.12 kg monthly and 21.0 kg during lactation. The results of other authors indicate that there is no clear correlation between somatic cell count and protein content [Ikonen et al., 2004; Ogola et al., 2007; Barłowska et al., 2009; Forsbäck et al., 2009]. Generally low correlation coefficients obtained are (as already was mentioned) the confirmation of absence of an unequivocal relationship between SCC and protein content.

There is also no unequivocal opinion among researchers in the case of the relationship between somatic cell count and fat content in milk. Król et al. [2010] pointed at a downward tendency of this component with an increase in SCC, but the differences were generally statistically insignificant in almost all breeds. However, Barłowska et al. [2009] indicated a reverse tendency. In the opinion of Ogola et al. [2007] and Forsbäck et al. [2009], changes in protein

and fat content in milk at elevated somatic cell count aroused from an increased risk of proteolysis and lipolysis. El-Awady and Oudah [2011], analyzing monthly and lactational wastage in fat production in the milk of Friesian cows, stated that the losses occurred at SCC level >100 thous. ml^{-1} . At $\text{SCC} > 2$ million ml^{-1} the losses amounted to 3.01 kg monthly and 27.9 kg during lactation. According to sundry authors [Lindmark-Månsson et al., 2000; Ikonen et al., 2004; Zumbo et al., 2004; Sawa et al., 2007; Pazzola et al., 2010], the correlation coefficients between SCC and fat content in milk are generally low and oscillated from negative ($r=-0.05$) to positive (in a range of $r=0.07$ to $r=0.37$). Whereas in the research of Sender et al. [2001], the genetic correlations between somatic cell count and yield of fat and protein were positive and higher in the first lactation ($r=0.39$) than those estimated for all lactations ($r=0.16$).

Heuer et al. [1999] and Windig et al. [2005] postulated that a higher risk of mastitis incidence appeared with an elevation in fat to protein ratio in milk. According to Heuer et al. [1999], the acute inflammatory response is more likely to occur when 1.5 value of this ratio has been exceeded.

5. SCC AND MILK WHEY PROTEINS

Somatic cell count also affects the changes in whey protein content. They represent 20-25% of milk proteins, including approximately 75% of albumin, i.e. α -lactalbumin (α -LA), β -lactoglobulin (β -LG) and bovine serum albumin (BSA). The remainder constitutes immunoglobulins, protezo-peptones, glycomacropetides, lactoferrin, growth factors, hormones and various enzymes – including lysozyme. These compounds are globular proteins with a three-dimensional structure. It should be noted that the aforementioned proteins, except BSA and immunoglobulins deriving from the blood, are synthesized in mammary gland of cow [Farrell et al., 2004; Chatterton et al., 2006; Michaelidou and Steijns, 2006; El-Loly and Farrag, 2007; Smithers, 2008].

Whey proteins demonstrate multidirectional pro-health effects on the human body. They affect, among others, the digestive, immune, cardiovascular and nervous systems and play a significant role in reducing a risk of occurrence of various social diseases [Pan et al., 2006; Liu et al., 2007; Król et al., 2008; Smithers, 2008].

The dominant whey protein presenting in the milk of cows and other ruminants is β -lactoglobulin. It plays an important antioxidant role in milk. It

also exhibits anticancerogenic, antiviral and antibacterial properties. With the immunoglobulins G β -Lg is probably involved in forming the passive immunity [Sutton and Alston-Mills, 2006; Hernández-Ledesma et al., 2008]. α -lactalbumin actively contributes in a control of lactation and milk secretion, forming together with galactosyltransferase (GT) an indispensable component of lactose synthetase. It also shows anticancerogenic, antibacterial (against Gram-positive) and antiviral activity. Furthermore, α -La functions as an immunological factor, inter alia, modulating (increasing) a neonatal immunity [Séverin and Wenshui, 2005; Zimecki and Artym, 2005; Chatterton et al., 2006; Riley et al., 2008; Kanwar et al., 2009]. Thus, bovine serum albumin in milk is physically and immunologically identical to that of blood serum albumin, as it is not synthesized in the mammary secretory cells but derives from the so-called nonspecific “leakage” from the blood to milk. According to Litwińczuk et al. [2011], BSA concentration in milk is an indicator of permeability of the blood-milk barrier in the mammary gland. Mammary gland inflammations are a major factor increasing the permeability of cell membranes of the udder. Furthermore, lactoferrin is also involved in a natural defense of body against bacterial infections. By binding and sequestering of iron, lactoferrin exhibits antibacterial properties against Gram-positive and Gram-negative bacteria, non-capsular and capsular viruses as well as various types of fungi and parasites. It functions both bacteriostatic and bactericidal against *Staphylococcus aureus*, *Escherichia coli*, *Pseudomonas aeruginosa* and *Clebsiella pneumonia*. An advantage of lactoferrin in a fight against bacterial infections is possibility of increasing a bacteria sensitivity to certain antibiotics (vancomycin, penicillin) and lowering their effective doses. The combination of penicillin with lactoferrin doubles the inhibitory activity of antibiotic against *Staphylococcus aureus* [Diarra et al., 2002]. Lactoferrin also determines the innate immune.

The increase in concentration of this protein (cow milk contains an average of 20-200 mg dm⁻³) is not only due to the secretion of colostrum and development of the mammary glands but also it testifies of occurring infections, inflammations or injuries. Lactoferrin is a component of the secondary granules of neutrophils, where in the case of an injury, infection or inflammation is released into the blood [Baker and Barker, 2005; Artym, 2010; Garcia-Mantoya, 2011]. Large variability of single-nucleotide polymorphism – SNPs (more than 140) in the lactoferrin gene indicates that there is a high probability of existing of a mastitis-resistance marker or eventually also a marker of milk yield in this gene [Wojdak-Maksymiec et al., 2006; Żukiewicz et al., 2012]. Furthermore, this protein determines the

maturation of immune cells. Besides lactoferrin, lysozyme also occurs to be one of the most crucial component of the non-specific humoral immune response, i.e. as a bactericidal component of various glandular secretions, including mammary gland. Concentration of the protein varies according to an udder health, i.e. is much higher in cow colostrum and mastitis milk than in normal milk, what can be used in the mastitis diagnosis. Synergistic effect of the enzyme with immunoglobulins and lactoferrin against *Escherichia coli* and *Micrococcus luteus* was also noticed [Séverin and Wenshui, 2005; Moatsou, 2010]. Immunoglobulins are very important group of proteins exhibiting antimicrobial activity. These compounds are globulins of high molecular weight and occur in plasma and body fluids. Three major classes of immunoglobulins are distinguished, i.e. IgG, IgM and IgA, depending on physical-chemical structure and biological activity. IgG dominate in milk of ruminants (approximately 80%), while IgA in other mammalian milk, including human (approximately 90%). They determine the specific humoral immunity of the body [El-Loly and Farrag, 2007]. During the process of antigens binding as well as phagocytosis or complement activation these proteins are involved in the destruction of pathogenic microorganisms, i.e. *Escherichia coli*, *Candida albicans*, *Clostridium difficile*, *Shigella flexneri*, *Streptococcus mutans* and *Helicobacter pylori*. Furthermore, immunoglobulins block the action of toxins and viruses [Gapper et al., 2007].

The study of Litwińczuk et al. [2011], carried out on milk of cows of four breeds, i.e. Black-White and Red-White variety of Polish Holstein-Friesian, Simmental and Jersey, proved an effect of SCC on changes in whey protein content (Table 6). It was found that the rise of somatic cell count decreased the content of major albumins, i.e. α -LA and β -LG, to a small extent and that was confirmed by very low, statistically insignificant correlation coefficient obtained ($r=-0.07$ and $r=-0.05$, respectively). Negative value of the correlations indicates, however, the direction of changes and may suggest that in more advanced disease states a decline in these protein concentrations in milk can be higher. Wickström et al. [2009], found a significant decrease in α -LA concentration with an increase in somatic cell count in bulk tank milk obtained in Sweden.

The authors also observed a reduction in β -LG concentration, along with the decline in α -LA content. Similar tendencies were obtained in the study of Bleck et al. [2009], including two herds maintained in U.S., i.e. the University Illinois herd and the University Minnesota herd, wherein for the first herd achieved a negative correlation between logSCC and α -LA ($r=-0.059$) and for the second herd – positive ($r=0.177$).

Table 5. Correlation coefficients (r) between SCC and milk yield as well chosen milk components [Litwińczuk et al., 2011; Król et al., 2012]

Breed	Milk yield	Casein	BSA	Lactoferrin	Lysozyme	IgG
Polish Holstein-Friesian	-0.245**	-0.573***	0.711***	0.687***	0.694***	-
Simmental	-0.123	-0.691***	0.577***	0.710***	0.603***	-
Jersey	-0.148	-0.723***	0.472***	0.540***	0.693***	-
Total	-0.157	-0.591***	0.591***	0.652***	0.632***	0.790

** – significant at P<0.01; *** – significant at P<0.001.

Table 6. Comparison of values (%) of normal milk with that of mastitis milk having high somatic cell count [Jones and Bailey, 2009; Litwińczuk et al., 2011]

	Jones and Bailey, 2009		Litwińczuk et al., 2011		Direction of changes
	Normal milk	Milk with high SCC	Normal milk	Milk with high SCC	
Fat	3.5	3.2	-	-	↓
Lactose	4.9	4.4	-	-	↓
Total protein	3.61	3.56	3.75-4.08	3.56-4.06	↓
Casein	2.80	2.30	2.81-3.14	2.44-2.82	↓
Whey proteins	0.8	1.3	-	-	↑
Serum albumin	0.02	0.07	0.04-0.05	0.06-0.08	↑
Lactoferrin	0.020	0.100	0.007-0.009	0.010-0.012	↑
Immunoglobulins	0.1	0.6	-	-	↑
α-LA	-	-	0.100-0.120	0.092-0.098	↓
β-LG	-	-	0.31-0.38	0.27-0.33	↓

Content of immunoactive proteins (lactoferrin and lysozyme) and bovine albumin serum increases, however, with a rise of somatic cell count. In the research of Litwińczuk et al. [2011], milk with the highest SCC (501-1,000 thous. ml⁻¹) included averagely more lactoferrin (by 34.8%), lysozyme (by 60.2%) and BSA (by 64.7%), in comparison to milk with SCC below 100 thous. ml⁻¹. The authors confirmed a substantial effect of SCC on immunoactive proteins and bovine albumin serum content by relatively high

positive values of the correlation coefficients calculated. For lactoferrin content r was amounted to 0.65, for lysozyme – $r=0.63$ and BSA – $r=0.59$. In the case of bovine albumin serum, these correlations were clearly differentiated at certain breeds of cows (Table 5). The highest relationship, i.e. at the level of $r=0.711$, was obtained for Holstein-Friesian cows, while a noticeably lower dependence was found for Simmental ($r=0.577$) and Jersey cows ($r=0.472$). Higher sensitivity of Holstein-Friesian cows to udder infections was manifested by significantly greater drops in their daily yield of milk. Therefore, it can be assumed that in Simmental and Jersey cows the SCC growth is not followed by such intensive permeation of bovine serum albumin from blood to milk, as it was observed in Holstein-Friesian cows. This would indicate a higher resistance of Simmental and Jersey cows to mammary gland infections. Furthermore, Urech et al. [1999], in the study of quarter milk, noticed similar tendencies when 100 thous. cells ml^{-1} were recognized as the threshold limit of somatic cell count. Quarter milk, obtained from clinically healthy mammary glands, contained an average of 84 thous. somatic cells ml^{-1} , whereas the milk from infected glands included 293 thous. cells ml^{-1} . The authors showed the significant rise in the content of lactoferrin (by 0.45%), BSA (by 0.10%) and immunoglobulins (by 0.39%) in milk from affected udder. Hamann [2002] defined “the gold standard” for SCC at the level of 100 thous. ml^{-1} . Higher somatic cell count testifies, in his opinion, to a dysfunctional milk secretion, what leads to reduction in daily milk production, changes in milk chemical composition and deterioration of technological properties. However, similar tendencies and statements are also included in the papers of other authors [Piccinini et al., 2006; Berlung et al., 2007; Barłowska et al., 2009; Forsbäck et al., 2009]. Lindmark-Månsson et al. [2000 and 2006] observed that a somatic cell count of over 5 thous. ml^{-1} increases lactoferrin content in milk, and a close relationship between this component and udder health status has been confirmed by very high correlation coefficients between milk lactoferrin concentration and somatic cell count ($r=0.962$ and $r=0.918$) obtained in two independent studies. Furthermore, Nudda et al. [2001], in the research carried out on the milk obtained from Sarda sheep, also showed the significant differences in whey protein content between SCC groups, and for lactoferrin, BSA and IgG the significant positive correlations with somatic cell count were noticed, amounting respectively: $r=0.39$, $r=0.31$ and $r=0.35$. Higher correlation coefficient between SCC and BSA ($r=0.53$) was stated in the research of Poutrel et al. [1983], conducted on milk of Holstein and Friesian-Holstein breed. Król et al. [2012] received, however, high correlation coefficient between SCC and IgG ($r=0.790$). A significant increase in

lactoferrin, albumin and IgG content with deterioration of mammary gland health status was also confirmed in the research of other authors [Leitner et al., 2004; Piccinini et al., 2006; Liu et al., 2007].

6. SCC AND TECHNOLOGICAL PARAMETERS OF MILK

Raw material intended for processing should be characterized by the appropriate technological indicators, predisposing it to produce a dairy product. It should be noticed that basic chemical composition, acidity, thermal stability of milk and rennet clotting time are the most major indicators.

In the production of many dairy products, content of non-fat dry matter, especially total protein, including casein, is considered to be of great importance. Casein fraction determines the clotting time, curd firmness and cheese yield from a milk volume specified. κ -casein is the sole casein fraction sensitive to an addition of rennet (chymosin enzyme). However, it has no sensitivity to calcium presence. κ -casein stabilizes other casein fraction toward calcium, forming with them the micelles. It is precipitated at pH value 4.6 at 20°C. κ -casein specific feature is the ability to coagulate, both enzymatic and acid, which is used in the production of rennet and curd cheese. Raw material with high protein content is also needed in the production of fermented beverages as it is responsible for binding an appropriate amount of water in product. As a consequence, the product is stable and resistant to syneresis. The whey protein with an importance in milk processing is β -lactoglobulin. During the thermal treatment it is denatured, causing an exposure of -SH groups. -SH groups bind the metal ions (especially copper and iron) and as a result they inhibit the oxidation of milk fat in dairy products. The negative effect of β -lactoglobulin denaturation is a binding of the molecules denatured with κ -casein. Nevertheless, the binding hinders the process of enzymatic coagulation. In the cheese production also a great importance has protein to fat ratio. Furthermore, lactose presence in milk enables an acidification of milk, owing to the fact that as a sugar lactose is a food for lactic acid bacteria [Barłowska, 2007].

The preparation of stable dairy products such as UHT milk and cream, sterilized drinking milk, unsweetened condensed milk and fermented beverages requires a high temperature or a little lower but with a longer time of duration. With regard to this, the raw material which is capable of physical endurance of heat treatment used is needed, and thus of a high thermal stability. This term is generally understood as the ability to retain the colloidal

properties of milk, in particular proteins, during high temperature operation. In fact, low stability may cause sedimentation of proteins denatured in the final product and occasionally even its gelification during the storage [Singh and Creamer, 1992; Kruk, 2001; Faka et al., 2009]. Thermal stability of milk is not constant and depends directly on its chemical composition and physical characteristics. Milk as a raw material meets the quality criteria in this regard if it withstands the time of heat treatment in temperature of $140^{\circ}\text{C} \geq 8$ min [Kruk, 2001; Litwińczuk, 2012]. One of the direct factors that determine thermal stability of milk is its acidity. The research of Barłowska et al. [2010] indicates a significant negative correlation between active acidity (pH values) and thermal stability of milk ($r=-0.30$). Thermal stability of milk increases in the pH value range from 6.4 to 6.7 and at pH value above 6.7 it begins to reduce significantly, reaching a minimum at pH value 6.9. The parameter value begins to rise afresh at $\text{pH}>6.9$ [Singh and Fox, 1986, 1987; Rattay and Jelena, 1996]. Singh and Fox [1985a, b, 1986, 1987] explained the reduction in thermal stability of milk at pH value proximate to 6.9 as the result of dissociation of κ -casein complex with whey proteins. This contributes to higher sensitivity of micelles to high temperature and calcium ions operation [Singh, 1995]. van Boekel et al. [1989a, b] stated that high thermal stability of casein micelles at $\text{pH}<6.7$ (where whey proteins deposit and bind) was caused by binding of calcium ions in the initial phase of the process, which reduced the concentration of Ca^{2+} in milk. Furthermore, thermal stability of milk is determined by the content of whey proteins and calcium ions. In milk with normal concentration of whey proteins a protective activity of these proteins in relation to casein and vice versa, i.e. casein with regard to whey proteins, is observed.

On the one hand, whey proteins denatured undergo a microfloculation on the casein micelle surface (in the order: serum albumin, β -lactoglobulin and α -lactalbumin, according to the heat resistance), which prevents their stronger aggregation and loss out of solution. On the other hand, they block calcium access to the micelles (by interacting with casein) and as a result milk obtains better stability. The micelle size also greatly influences on milk heat stability. Small micelles contain relatively more κ -casein which stabilizes the remaining casein fractions to calcium ions. Therefore they are more resistant to high temperature (140°C) [O'Connell and Fox, 2000]. Barłowska et al. [2010] reported significant positive correlation between milk thermal stability and rennet clotting time ($r=0.32$).

A crucial indicator determining the suitability of milk for rennet cheese production is rennet clotting time. Defective milk, i.e. obtained from cows

with udder inflammation, may clot after 20 minutes or does not coagulate at all. The rate of clot formation and degree of firmness are largely dependent on the casein content in milk [Ikonen et al., 2004], proportion of calcium to nitrate compounds (0.2 – for slowly coagulating milk and 0.23 – for normal and quickly coagulating milk) and casein micelle size [Litwińczuk, 2012]. It is considered that rennet clotting time of milk is dependent on protein content, in that casein primarily. Nevertheless, research results are quite divergent with regard to these relationships. Analyzing milk of Finnish Ayrshire cows, Ikonen et al. [2004] stated the relationship between coagulation time (or curd firming time) and content of protein and casein. Similar analyses, which were performed by Oloffs et al. [1992] on milk of Friesian cows and Ikonen et al. [1999] on milk of Finnish cows, showed that shorter coagulation time was associated with lower protein content. However, Oloffs et al. [1992], evaluating milk of Angler cows, did not show any dependences. Results of curd firmness assessment obtained by the various authors were also divergent. In Oloffs et al. [1992], high values for curd firmness correlated with high protein and casein content, whereas Ikonen et al. [1999, 2004] indicated a reverse tendency, i.e. high values for curd firmness correlated with low protein and casein content. Ability of milk to enzymatic coagulation depends greatly on the size of casein micelles. Large micelles contain relatively less κ -casein, which functions as a stabilizer for micelle particles, and thereupon milk coagulates quickly [Ziajka, 2008]. Content of calcium in milk is considered of great importance to enzymatic coagulation processes [Nájera et al., 2003; Barłowska et al., 2010]. Milk coagulation time significantly affects the firmness of curd formed. Ikonen et al. [2004] confirmed this strong relationship by very high correlation coefficients ($r_g=-0.97$ and $r_p=-0.92$).

Breed of cows is crucial factor determining milk suitability to cheese production [Okigbo et al., 1985a, b; Malossini et al., 1996; Tyrisevä et al., 2004]. Research of numerous authors indicate that cows of local breeds produce milk which faster coagulates and curd obtained is more firm, in comparison with highly productive breeds [Chiofalo et al., 2000; Barłowska and Litwińczuk, 2006; De Marchi et al., 2007; Litwińczuk et al., 2012]. Furthermore, in the latter a large proportion of noncoagulating milk samples is stated. Ikonen et al. [2004] found that in 4,700 milk samples collected from Finnish Ayrshire cows approximately 13% of them (618 samples) had not coagulated, i.e. they had not aggregated and formed any curd. Kübarsepp et al. [2003] stated 8% of noncoagulating milk samples in dairy cattle breeds raised in Estonia (Estonian Holstein, Estonian Red and Estonian native breeds) and

slightly more (11%) in Red and White Holsteins. Ikonen [2000], however, did not noticed such problem in milk of Holstein-Friesian and Finnish cows.

Casein, as an essential milk component deciding about its suitability for processing, particularly for cheese production, usually declines in inflammations with accordance to mastitis status. This is due to the fact that protein fraction is synthesized whole in the mammary gland. Mammary gland infection results in the changes in milk component secretion, inter alia, the noncasein fraction (NCN fraction) is found to be elevated, while the casein content is decreased. It occurs partly due to increased proteolysis leading to a reduction in the casein to total protein ratio in infected quarters. And this may be linked to increased endogenous proteolysis which eventuates from the elevation of plasmin or other proteases derived from somatic cells.

The result of increased permeability of mammary epithelium is the influx of blood proteins (immunoglobulins, especially IgG, and bovine serum albumin) into milk, which results in an elevated NCN content [Ogola et al., 2007]. Forsbäck et al. [2009], analyzing milk of Swedish Red Breed and Swedish Holstein, confirmed the fact of reduction in casein content in milk with somatic cell count rise, i.e. from 2.61% at $SCC < 100$ thous. ml^{-1} to 2.56% at $SCC > 300$ thous. ml^{-1} ($P \leq 0.05$). In the research of Litwińczuk et al. [2011], carried out on milk of cows of four breeds, i.e. Black-White and Red-White variety of Polish Holstein-Friesian, Simmental and Jersey, a downward tendency for casein content was also showed. Its level declined in milk with SCC rise, regardless of the breed of cows and production season. A distinct negative relationship between milk casein and SCC was confirmed by a relatively high value of correlation coefficient ($r = -0.591$ at $P < 0.001$). Nevertheless, it should be emphasized that the values of this coefficient were various for particular breeds (Polish Holstein-Friesian – $r = -0.573$, Simmental – $r = -0.691$ and Jersey – $r = -0.723$), which indicates the sundry sensitivity of these cow breeds to udder inflammations.

As already mentioned, the milk acidity has significant effect on its resistance during heat treatment. Normal (fresh) milk, derived from cows with healthy udder, should have pH range from 6.5 to 6.8. Such pH guarantees the colloidal stability of milk [Litwińczuk, 2012]. Many authors [Ikonen et al., 2004; Bansal et al., 2005; Ogola et al., 2007] indicated the rise in values of active acidity in milk taken from infected quarters. Higher levels of citrate and bicarbonate found during udder inflammation may be responsible for elevated pH levels. Ogola et al. [2007] showed the increase in pH value to 6.81 in milk with elevated somatic cells count ($SCC > 750$ thous. ml^{-1}), while in milk contained less than 250 thous. ml^{-1} this value was equal to 6.63. However, the

differences were statistically insignificant. Results obtained by Ikonen et al. [2004] confirmed these relationships by the positive correlation coefficients between SCC and pH values ($r_g=0.16$ and $r_p=0.26$).

Udder inflammations also cause the changes in proportions of milk proteins and mineral components, and consequently the reduction of milk colloidal stability. In mastitis milk content of whey proteins increases, and as the result a protective effect of casein in relation to them becomes insufficient. This causes an imbalance between κ -casein and β -lactoglobulin, for which the optimum molar ratio should be equal to 1 in normal milk [Żbikowska and Szerszunowicz, 2002]. Furthermore, an increase in Ca and P content in milk also lowers the thermal stability.

The balance between calcium and magnesium ions as well as phosphates and citrates is greatly important for this parameter of milk technological suitability [Ziajka, 2008].

Increase of SCC in milk causes an elongation of rennet clotting time or its absence. Longer coagulation time of milk results in a decrease of curd firmness, impairment of syneresis processes, raised cheese moisture and reduced cheese yield efficiency (high losses of casein which passes to whey). This is mainly due to the changes in content of milk components (the changes in distribution of proteins, including casein, decline in lactose content, changes in concentration of minerals, mainly calcium reduction).

The fact of clotting time lengthening due to milk SCC increase confirms the results of many authors [Nudda et al., 2001; Ikonen et al., 2004; Zumbo et al., 2004; Barłowska et al., 2009]. Ikonen et al. [1999, 2004] indicated strong genetic correlations between milk coagulating properties (MCP), i.e. rennet clotting time (RCT), curd firming time (k_{20}) and curd firmness (a_{30}), and SCS estimated for cows of Finnish Ayrshire breed. They suggested that selection for low SCC could improve MCP and reduce the occurrence of noncoagulating milk samples. On the basis of the research of Barłowska et al. [2009], conducted on cow milk of four breeds (Black-White and Red-White variety of Polish Holstein-Friesian, Simmental and Whiteback), it should be also emphasized that cows of Simmental and native Polish Whiteback breed produced milk of noticeably better suitability to cheese production. It was proved by evidently shorter milk enzymatic coagulation time (moment of the first flakes of casein loss) for Simmental and Whiteback breed, which amounted from 4:32 to 5:41 min at $SCC < 400$ thous. ml^{-1} , in comparison with milk of Holstein-Friesian cows (from 7:05 to 8:32 min) – Table 7.

They also showed that milk with somatic cell count above 1,000 thous. ml⁻¹ had longer coagulation time by approximately 20-30% (depending on breed), with regard to the milk samples at SCC from 201 to 400 thous. ml⁻¹. Ikonen et al. [2004] confirmed these strong relationships between SCC and milk technological parameters to cheese production by genetic correlation coefficients obtained, i.e. between SCC and RCT – $r_g=0.29$ as well as SCC and a_{30} – $r_g=-0.45$. With regard to milk of Sarda sheep breed the similar relationships between SCC and coagulation properties of milk were also noted [Nudda et al., 2001]. Rennet clotting time significantly ($P<0.01$) lengthen from 16.05 min at $SCC<300$ thous. ml⁻¹ to 23.64 min at $SCC>2,000$ thous. ml⁻¹. Furthermore, correlation coefficients obtained between SCC and RCT ($r=0.43$), SCC and k_{20} ($r=0.41$) as well as SCC and a_{30} ($r=-0.43$) were statistically significant ($P<0.01$).

As mentioned above, a right behavior of milk during various technological processes is determined by the content and proportions of individual minerals. Udder inflammations cause a decline in Ca, P and K concentration but level of Cl and Na significantly increases [Bruckmaier et al., 2004; Ogola et al., 2007; Litwińczuk, 2012]. Ogola et al. [2007] analyzed effect of SCC on Ca, K, Na and Cl content in 396 samples of quarter milk taken from cows of Holstein-Friesian and Zebu crossbreed. They showed that Na content was 46.5 mg 100 g⁻¹, K – 146.3 mg 100 g⁻¹, Ca – 119.5 mg 100 g⁻¹ and Cl – 100.5 mg 100 g⁻¹ at $SCC<250$ thous. ml⁻¹. Rise in SCC over 750 thous. ml⁻¹ elevated Na concentration to 78.5 mg 100 g⁻¹ (by over 68%) and Cl to 183.5 mg 100 g⁻¹ (by over 72%), however, reduced K content to 108.9 mg 100 g⁻¹ (by over 34%) and Ca to 97.8 mg 100 g⁻¹ (by over 22%). Rise in chlorine concentration in mastitis milk follows due to decline in lactose content which is an essential component conditioning milk fermentation and acidification, and as a result also the syneresis processes in cheese grains. This affects an increase in cheese moisture and decrease in whey yield. A major mineral component of milk, which appropriate concentration determines the rennet curd formation, is Ca. It shields negative functional groups on the micelle surface and plays a crucial role in formation of intermicelle calcium bridges [Ziajka, 2008]. Therefore, the decrease in calcium content in mastitis milk is one the factors lengthening its coagulation time or even contributing to the lack of coagulation. As most milk Ca is associated with casein micelles, Ogola et al. [2007] reported that reduced casein content could explain the lowered calcium levels in infected quarters. Increasing of cellular component content in milk negatively affects not only the raw material but also its products. During the ripening process of cheese made from milk with high SCC undesired proteolytic processes occur, which leads to the reduction in quality of cheeses or disqualification [Marino et al., 2005; Wickström et al., 2009].

Table 7. Productivity, basic chemical composition and technological suitability of milk obtained from different cow breeds, with regard to somatic cell count [Barłowska et al., 2009]

Breed	SCC group (thous. ml ⁻¹)	Specification							
		Daily milk yield.(kg)	Dry matter (%)	Protein (%)	Fat (%)	Protein/Fat	Lactose (%)	Clotting time (min)	Thermal stability (min)
Black-White variety of Polish Holstein-Frisian	I	26.1 ^b	13.24 ^a	3.40 ^a	4.27 ^a	0.81	4.91 ^c	7:05 ^A	4:40
	II	26.7 ^b	13.17 ^a	3.42 ^a	4.26 ^a	0.82	4.84 ^b	7:52 ^A	4:20
	III	23.8 ^a	13.62 ^b	3.66 ^b	4.46 ^b	0.84	4.84 ^a	7:46 ^A	4:51
	IV	23.7 ^a	13.38 ^{ab}	3.59 ^b	4.36 ^{ab}	0.84	4.78 ^a	9:46 ^B	4:25
Red-White variety of Polish Holstein-Frisian	I	20.6 ^c	13.30 ^b	3.34	4.40 ^{ab}	0.77	4.91 ^c	7:09 ^a	4:32
	II	20.5 ^{bc}	12.96 ^a	3.26	4.23 ^a	0.79	4.82 ^b	8:32 ^{ab}	4:53
	III	18.1 ^{ab}	13.19 ^{ab}	3.33	4.49 ^b	0.76	4.73 ^b	8:37 ^b	4:48
	IV	16.5 ^a	13.08 ^{ab}	3.40	4.43 ^{ab}	0.78	4.60 ^a	11:02 ^c	4:45
Simmental	I	19.5 ^b	13.32	3.50	4.29	0.83	4.89 ^c	5:41 ^a	3:57
	II	17.6 ^a	13.16	3.53	4.24	0.85	4.74 ^b	5:16 ^a	3:50
	III	18.0 ^{ab}	13.19	3.50	4.38	0.82	4.66 ^a	5:48 ^a	3:54
	IV	16.5 ^a	13.29	3.56	4.45	0.82	4.63 ^a	7:40 ^b	3:42
Whiteback	I	12.7 ^b	13.15	3.28 ^a	4.36	0.77	4.85 ^C	4:58 ^{ab}	4:46
	II	9.3 ^a	13.12	3.51 ^b	4.25	0.83	4.71 ^B	4:32 ^a	3:56
	III	10.5 ^a	13.08	3.54 ^b	4.24	0.85	4.65 ^B	4:56 ^{ab}	4:04
	IV	11.2 ^{ab}	13.02	3.54 ^b	4.33	0.83	4.50 ^A	6:12 ^b	4:25
Average	I	21.7 ^b	13.27 ^b	3.42 ^A	4.30 ^{ab}	0.81 ^a	4.90 ^D	6:24 ^A	4:24
	II	20.5 ^b	13.13 ^a	3.44 ^{AB}	4.25 ^a	0.83 ^b	4.78 ^C	6:40 ^{AB}	4:13
	III	18.9 ^a	13.31 ^b	3.51 ^B	4.41 ^c	0.81 ^{ab}	4.74 ^B	7:02 ^{BC}	4:16
	IV	18.6 ^a	13.22 ^{ab}	3.52 ^B	4.40 ^{bc}	0.82 ^{ab}	4.66 ^A	9:20 ^C	4:32

I – to 200 thous. ml⁻¹, II – 201-400 thous. ml⁻¹, III – 401-1000 thous. ml⁻¹, IV – above 1000 thous. ml⁻¹;

A, B, C, a, b, c – differences between SCC within a breed; a, b, c – differences significant at P≤0.05; A,B, C – differences significant at P≤0.01.

CONCLUSION

Udder inflammation remains the most frequent and expensive disease affecting dairy cattle. For the dairy industry the most pernicious are subclinical (non-symptomatic) inflammations, which are very often undiagnosed. As a result, milk with lowered quality becomes a commercial milk. SCC in cow milk is a good indicator of the mammary gland health status, that is why clinical and subclinical inflammations occurred are associated with a significant increase in their number in milk. Inflammations primarily affect a reduction in the milk production. On the basis of the fact mentioned above, it could be concluded that highly productive cow breeds are more susceptible to inflammation, which in turn results in higher losses in milk production in such herds. Decreased synthetic activity of infected mammary gland are also revealed in the changes in individual milk component content and in the ratios between them. As a consequence, milk nutritional value and suitability for processing is reduced.

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