www.foodandfeed.fins.uns.ac.rs
FOOD AND FEED RESEARCH
Journal of the Institute of Food Technology – FINS
University of Novi Sad
ISSN
2217-5369
(print version ceased in 2023)
2217-5660 (online)

UDK 637.1:579.86
DOI: 10.5937/ffr0-55906

Review article

PRESENCE OF *STAPHYLOCOCCUS AUREUS* ENTEROTOXIN GENES IN MILK AND DAIRY PRODUCTS – A REVIEW

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Abstract: *Staphylococcus aureus* is a highly adaptable microorganism that is commonly found in a variety of settings, including the skin and mucous membranes of humans and animals, as well as in the air, soil, and water. It is widely known for possessing a variety of virulence factors that enable it to cause and spread diseases. The presence of the genes for TSST-1 and enterotoxin A in milk and milk products will receive the most emphasis in this review. *S. aureus* isolated from milk can carry individual or combinations of genes coding for classical enterotoxins. The expression of many virulence-related genes *S. aureus* is regulated by the *agr* (accessory gene regulator) system, a two-component signaling system that down-regulates surface proteins and up-regulates secretory proteins, including various enterotoxins such as SEB, SEC, SED, and TSST-1. Staphylococcal food poisoning (SFP) is commonly linked to the growth of staphylococci in protein-rich foods, for example, meat, milk, and dairy products. The growth of *S. aureus* and the production of enterotoxins are heavily influenced by the characteristics of the food, including salt content, pH, nutrient availability, oxygen levels, and temperature. In the dairy industry, strict control methods and ongoing monitoring are necessary to guarantee food safety. Long-term storage of milk above 8 °C greatly raises the danger of *S. aureus* growth and the possibility of enterotoxin development.

Key words: *food safety, enterotoxin A, toxic shock syndrome toxin-1, dairy*

INTRODUCTION

Staphylococcus aureus is a highly adaptable microorganism commonly found in various environments, including the human skin, mucous membranes, the air, soil, and water. It is known for possessing a wide range of virulence factors that enable it to cause disease. These factors help the bacterium invade host tissues, evade the immune system, and cause damage to the host. Due to its numerous virulence mechanisms, *S. aureus* is one of the leading bacterial pathogens responsible for many human diseases, ranging from mild skin infections to life-threatening illnesses such as pneumonia, sepsis, and toxic shock syndrome. An important characteristic of *S. aureus* is its ability to produce extracellular enzymes and heat-stable toxins,

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which are primarily responsible for food poisoning. This means that even when *S. aureus* itself is no longer present in the food, the toxin can remain, posing a risk of foodborne illness. Consequently, food contaminated with these toxins may still cause food poisoning even after the bacteria have been killed by heat. *S. aureus* frequently colonizes the papillary ducts of the mammary glands of cows, leading to subclinical mastitis, thus affecting milk quality (Fagundes et al., 2010). As one of the primary causes of mastitis in dairy cows, it poses both significant health problems for the animals and economic challenges for the dairy industry, as it can reduce milk production and quality (Oliveira, Soares e Barros, Silva and Cirqueira et al. 2011; Homsombat, Boonyayatra, Awaiwanont & Pichpol, 2021; Morar et al., 2021). Moreover, it can be present in milk and milk products that are not thermally processed, but also in thermally processed products subsequently contaminated by humans. Raw milk and milk products are considered a good substrate for the growth of *S. aureus* and enterotoxin synthesis, so they are often related to staphylococcal food poisoning (SFP).

Pathogenicity is the ability of a microorganism to cause disease. Virulence factors can be related to the structure of the bacterial cell (peptidoglycan, teichoic acid, surface proteins) and extracellular products of bacterial metabolism (toxins and enzymes). *S. aureus* has a wide range of virulence factors that can be either secreted into the surrounding environment or attached to its cell membrane (Foster, 2005; Chambers & DeLeo, 2009). Depending on their role in pathogenesis, virulence factors can be divided into those that enable adherence, and invasiveness and those that produce toxins. The main toxins of *S. aureus* that should be highlighted are hemolysins, exfoliatins, enterotoxins, toxic shock syndrome toxin 1 (TSST-1) and Panton - Valentin leukocidin (PVL) (Ahmad-Mansour et al., 2021). Virulence factors contributing to a bacterium's ability to cause disease can be encoded by genes found in various genetic elements, including the bacterial chromosome, plasmids, bacteriophages, and transposons. These genetic elements play a key role in the pathogenicity of bacteria by carrying the necessary information for producing toxins, enabling immune evasion, or facilitating bacterial adhesion to host tissues (Malachowa & DeLeo, 2010). Toxins that act as superantigens lead to polyclonal activation of a large number of lymphocytes, and this group includes enterotoxins, exfoliantin and TSST-1 produced by *S. aureus*. Toxin synthesis depends on the activity of the regulatory genes that control the expression of staphylococcal enterotoxins (SEs) and TSST-1 and in this review, the most attention will be paid to the presence of the genes for enterotoxin A (SEA) and TSST-1 in milk and milk products.

GENE EXPRESSION

S. aureus isolated from milk can carry individual or combinations of genes coding for classical enterotoxins. The expression of many virulence-related genes *S. aureus* is regulated by the *agr* (accessory gene regulator) system, a two-component signalling system that down-regulates surface proteins while up-regulating secretory proteins, including various enterotoxins like staphylococcal enterotoxin B (SEB), staphylococcal enterotoxin C (SEC), staphylococcal enterotoxin D (SED), and TSST-1. The *agr* quorum-sensing system plays a crucial role in regulating the gene expression of *S. aureus*, particularly when bacterial populations reach a critical density (Jenul & Horswill, 2019). This system regulates the expression of virulence factor genes through the production of two main RNA transcripts, RNA II and RNA III, which are controlled by the P2 and P3 operons, respecttively. The P2 operon within the *agr* locus contains four essential structural genes: *agr*B, *agr*D, *agr*C, and *agr*A. Together, these genes encode the *agr* signalling mechanism, which is primarily driven by the RNAII transcript. AgrB is a transmembrane protein responsible for the posttranslational modification of the primary *Agr*D peptide. It modifies *Agr*D into an octapeptide, which is further processed into an active cyclic form. This modified form of the peptide is known as the autoinducer peptide (AIP). *Agr*D gene encodes the precursor peptide that, after modification by *Agr*B, becomes the active AIP, which is the signalling molecule. *Agr*C is a transmembrane protein with histidine kinase activity. It binds to the AIP, and this binding activates the *Agr*C-*Agr*A two-component system. The activation of *Agr*C results in the phosphorylation of *Agr*A, a key regulator of the system. Once activated by phosphorylation, *Agr*A initiates the transcription of the P2 and P3 operons, which leads to a significant increase in the production of various virulence factors during the late logarithmic phase of bacterial

growth (Božić, 2024). The transcription of the P3 operon results in the production of RNA III, which not only promotes the synthesis of virulence factors like δ-toxin, TSST-1, and α-hemolysin but also inhibits the production of genes for cell wall-associated proteins, such as protein A. Notably, during the exponential growth phase, the synthesis of enterotoxins A and K remains unaffected by RNA III, with only partial regulation of enterotoxins B, C, and D (Božić, 2024; Huntzinger et al., 2005).

The *sea* gene is encoded by prophages, specifically carried by bacteriophages from the polymorph family, which integrate into the bacterial chromosome. Under certain stressful conditions, such as improper food storage, these prophages can reactivate, producing new bacteriophages. Recent studies have shown that transcription of the *sea* gene is linked to the life cycle of the SEA-encoding prophage, which consists of two phases (lysogenic and lytic), in contrast to other enterotoxin genes such as *seb*, *sec*, and *sed* (Schelin, Wallin-Carlquist, Thorup Cohn, Lindqvist & Barker, 2011; Grispoldi, Karama, Armani, Hadjicharalambous & Cenci-Goga, 2021). In the lysogenic phase, the bacteriophage DNA integrates into the chromosome of the host bacterium. The integrated phage DNA is called prophage. The prophage is transmitted to subsequent bacterial generations as the bacterial cell divides. In the lytic phase, under certain conditions (such as stress to the host bacterium), the prophage may be excised from the bacterial chromosome. This is triggered by events like DNA damage. New phage particles are produced, assembly occurs, and eventually, the host bacterium is lysed (broken open), releasing the newly formed phage to infect other cells (Zeaki, Budi Susilo, Pregiel, Rådström, & Schelin, 2015; Wallin-Carlquist et al., 2010). The polymorphic nature of the prophage influences the amount of Staphylococcal enterotoxin A (SEA) produced by the bacterial strain.

Research indicates that *S. aureus* isolated from milk and milk products often carries the *sea* gene, which is responsible for producing Staphylococcal Enterotoxin A (Fooladi, Tavakoli & Naderi, 2010). This enterotoxin is one of the primary contributors to foodborne illnesses associated with dairy products. Factors such as storage temperature and nutrient availability in milk can significantly influence the expression of this gene, leading to an increased risk of contamination in improperly handled products (Zhang et al., 2022). The presence of the *tst* gene, which encodes TSST-1, has been detected in certain *S. aureus* strains found in dairy products. While the primary concern with *tst* is its association with severe systemic illnesses, its expression in dairy environments poses a potential risk. Factors such as the microbial environment and processing methods can affect the levels of TSST-1 in milk products (Abril et al., 2020).

FACTORS AFFECTING *S. AUREUS* GROWTH AND ENTEROTOXIN SYNTHESIS

Staphylococcal food poisoning (SFP) is commonly linked to the growth of staphylococci in protein-rich foods like meat, milk, and dairy products. The growth of *S. aureus* and the production of enterotoxins are heavily influenced by the food's complex composition, including factors such as salt content, pH, nutrient availability, oxygen levels, and temperature. While bacterial growth is necessary for enterotoxin production, toxin synthesis can occur independently, even in non-replicating cultures, as has been observed in certain ham products (Wallin-Carlquist et al., 2010; Schelin et al., 2011).

S. aureus shows remarkable adaptability to different environmental conditions, especially concerning water activity (aw). It can grow at a minimum aw of 0.83–0.86, provided that all other parameters are optimal, but it is ideal water activity for growth is above 0.99 (Medved'ova, Valík and Studeničová, 2009). However, the synthesis of staphylococcal enterotoxins varies depending on the aw level. Enterotoxins A (SEA) and D (SED) can be produced at almost all aw values that favour the growth of *S. aureus*, while the synthesis of enterotoxin B (SEB) is more sensitive to the reduction of aw. At an aw value of 0.93, the synthesis of this enterotoxin is low, despite the intensive growth of the SEB-positive *S. aureus* strain (Qi & Miller, 2000). Also, water activity has the same effect on SEC synthesis.

S. aureus is a mesophilic microorganism with an optimal growth temperature of 37 °C and can grow at a temperature range from 7 °C to 48 °C. The production of enterotoxins is also temperature-dependent. Research indicates that enterotoxin gene expression peaks at temperatures conducive to bacterial growth (~ 37 °C). The minimum temperature at which this microorga-

nism was isolated from bacon is 5 °C (Farrell & Upton, 1978; Hudson, Olsen & Cook, 2011) while growth was recorded in UHT milk at 7 °C (Medved'ova et al., 2009). The maximum temperature at which *S. aureus* can grow in skimmed milk is 48.9 °C. The minimum and maximum temperatures at which *S. aureus* can produce enterotoxins are 10 °C and 45 °C, with an optimal temperature of 35-40 °C (Bergdoll & Lee Wong, 2006).

The thermal stability of enterotoxins is influenced by factors such as the type of food, pH, sodium chloride content, and the specific type of enterotoxin. These factors determine how well the toxins can withstand heat and whether they can remain active under different conditions. SEA is more resistant to heat at higher pH values (> pH 6.0) than at lower pH values (pH 4.5-5.5). This means that when the food is more alkaline, SEA is less likely to be inactivated by heat, making it more stable in certain food products under those conditions. On the other hand, SED shows greater stability in the more acidic range (pH 4.5-5.5) and is more susceptible to heat at higher pH values (~ pH 6.0) (Wallin-Carlquist et al., 2010).

In situations where the enterotoxins are not fully inactivated by heat during cooking, there is a possibility of reactivation. This reactivation can occur during further stages of food processing, such as storage or incubation. If the conditions, such as temperature or pH, change during these stages, the enterotoxins may regain their activity and cause foodborne illness even if they were initially weakened or partially deactivated by cooking. Tsutsuura and Murata (2012) examined the synthesis of SEA toxins in different types of food such as milk, cream, eggs and mushrooms at different incubation temperatures. As the temperature increased, so did the concentration of SEA toxins.

They investigated the effect of incubation temperatures at 10, 15, 20 and 37 °C on SEA synthesis, using different strains of *S. aureus* that produce SEA toxin. The maximum obtained concentration of SEA at different incubation temperatures did not change significantly, which indicates that in addition to temperature, other factors affect the synthesis of SEA toxins. These results indicate that the synthesis of SEA toxin is strongly influenced by the strain of *S. aureus* - per se, as well as environmental factors such as temperature and nutrients, i.e. the matrix in which it is found.

Most *S. aureus* isolates grow in the pH range of 4.2 to 9.3, with optimal growth at pH 7-7.5 (Kadariya, Smith & Thapaliya, 2014). The optimum pH value for enterotoxin synthesis in food is between 7 and 8 and may be in the pH range of 5 - 9.6 (Grispoldi et al. 2021). *S. aureus* is capable of growing in NaCl (sodium chloride) concentrations as high as 15% (Kadariya et al. 2014), but enterotoxin synthesis for most *S. aureus* strains is lower in foods containing more NaCl (Etter, Ukowitz, Eicher, Tasara & Johler, 2022). One study (Schelin et al., 2011) showed that lactic acid has a specific effect on blocking the production of certain toxins, especially those regulated by *the agr* system in *S. aureus* such as the toxins SEB, SEC and SED. Wallin-Carlquist et al. (2010) investigated that mild acetic acid stress (pH 5.5 - 7) can lead to the activation of its genetic material (prophage), leading to increased production of SEA, in food products such as ham. However, when the pH is lower (4.5 - 5), SEA production is minimal or absent.

Bacterial growth under anaerobic conditions is considerably slower, and even after several days, the growth does not match the rate observed under aerobic conditions. Consequently, the production of SEB is ten times more intense in the presence of oxygen compared to an atmosphere with 95% N2 and 5% CO2. Although SEA synthesis is reduced in anaerobic conditions, it was still detected after 120 minutes of incubation in both aerobic and anaerobic environments (Belay & Rasooly, 2002). Carbon sources play a role in the production of enterotoxins (Smith, Buchanan & Palumbo, 1983). The addition of glucose was found to inhibit the production of SEA, SEB, and SEC toxins, likely due to the decrease in pH resulting from the metabolism of the sugar. The presence of amino acids affects the production of SEB toxins. Oxygen exposure, either during milk collection after milking or during the cheese-making process, may promote the growth of *S. aureus. In contrast,* microanaerobic conditions within the cheese are less conducive to the production of enterotoxins (Cretenet et al., 2011).

In addition to the physicochemical factors that influence the production of staphylococcal enterotoxins (SE) in milk and dairy products, *S. aureus* encounters a microbial environment that can limit its growth. The presence of other microorganisms, especially lactic acid bacteria

(LAB), is known to limit the growth of *S. aureus* (Mossel & Van Netten, 1990). Some studies have shown that LAB inhibits the synthesis of SE during cheese production, although the exact mechanisms behind this antagonism remain unclear (Cretenet et al., 2011; Even et al., 2009). Most studies have been conducted in controlled laboratory settings, but it is believed that in milk, LAB's antagonism towards *S. aureus* likely involves mechanisms such as nutritional competition or the production of inhibitory metabolites (bacteriocins or hydrogen peroxide) (Cotter, Hill & Ross, 2005; Ross, Morgan & Hill, 2002; Ito et al., 2003; Cretenet et al., 2011). Pitt, Harden and Hull (2000) investigated the growth patterns of *S. aureus* in both raw and pasteurized milk at 37 °C. They found that the growth rates were identical during the first 16 hours of incubation. However, after reaching the peak population density, *S. aureus* declined more rapidly in raw milk. After 72 hours, the bacterial count in raw milk was approximately 2 log cfu/mL lower compared to pasteurized milk.

This data indicates that the growth in raw and pasteurized milk exhibits similar intensity in the early stages of cheese production. In cheeses made from raw milk, LAB, as well as competitive microflora, show an inhibitory effect on pathogenic microorganisms.

PRESENCE OF *S. AUREUS* ENTEROTOXIN GENES IN MILK AND MILK PRODUCTS

There is significant variation in the reported amount of enterotoxin required to cause symptoms of food poisoning. Mossel, Corry, Struijk and Baird (1995) suggest that to develop symptoms of poisoning in adults, a dose of 10-20 µg of staphylococcal enterotoxin per kilogram of body weight must be ingested. Other researchers, however, believe that even less than 1 µg of enterotoxin can trigger symptoms in individuals who are particularly sensitive to it. In contrast, Balaban and Rasooly (2000), as well as Omoe et al. (2002), argue that the minimum toxic dose for enterotoxin A (SEA) is just 100 ng. In an outbreak caused by chocolate milk contaminated with enterotoxin A (SEA), the toxin concentration was reported to be as low as 0.5 ng/mL (Pinchuk, Beswick & Reyes, 2010).

Research by Li et al. (2011) indicates that TSST-1 toxin is highly resistant to heat treatment, as well as pepsin and trypsin during digestion, retaining its superantigenic activity. After treatment with pepsin or trypsin, TSST-1 is cleaved into smaller fragments but still retains its superantigen and shock-inducing properties, unlike SEA toxin. This suggests that the different levels of resistance between SEA and TSST-1 toxins contribute to their distinct pathogenic effects, particularly in food poisoning. Enterotoxins can cause food poisoning in humans, and TSST-1 modulates the host's immune response (Ote, Taminiau, Duprez, Dizier & Mainil, 2011). The release of TSST-1 into the bloodstream can result in severe medical conditions, including toxic shock syndrome, sudden infant death syndrome (SIDS), and Kawasaki disease. These complications are associated with systemic inflammation and organ dysfunction caused by the toxin's effects (Deurenberg et al. 2005). Farahmand, Ahmadi. Dastmalchi and Anassori (2013) conducted a study on *S. aureus* isolates collected from nine dairy farms with mastitis in East and West Azerbaijan (Iran). They used PCR to test for the presence of the *tst* gene. The *S. aureus* isolates were identified based on their growth in culture, biochemical properties, and the amplification of the *aroA* gene, which is specific to *S. aureus*. Out of 58 total isolates, 9 (15.5%) tested positive for the 350 base pair of *tst* gene. On the other hand (Baniardalan, Mohammadzadeh, Pajohi-Alamoti, Mahmoodi & Sadeghinasab, 2017), there are studies that found only one *S. aureus* isolate (1.3%) carried the *tst* gene out of 76 *S. aureus* isolates obtained from milk samples collected from 7 dairies in Hamedan, Iran. The identification of the isolates was based on biochemical and molecular methods, including PCR amplification of the *femA* gene. These findings suggest that *S. aureus* strains responsible for mastitis in dairy cows may harbour the *tst* gene, which could potentially lead to toxic shock syndrome in consumers. In Spain, they tested 53 *S. aureus* isolates from food for the presence of TSST-1 and proved only one food sample that was in contact with the hands of workers. Fueyo et al. (2005) examined 269 *S. aureus* isolates from food that had been in contact with the hands of workers in Spain, testing for the presence of genes associated with enterotoxin synthesis and toxic shock syndrome. The results revealed that 57 isolates produced at least one of the four enterotoxins (SEA, SEB, SEC, SED), 10 isolates produced only the TSST-1 toxin, and 10 isolates produced both enterotoxins and TSST-1.

In Italy, a case of food poisoning was linked to the consumption of Primosale sheep's cheese from Sicily. *S. aureus* was isolated from the cheese and found to produce enterotoxin C (SEC), along with the genes for enterotoxin C (*sec*) and toxic shock syndrome toxin (*tst*). Following this, a large-scale study was conducted on 971 food samples, including raw milk, meat, cheese, and other products. *S. aureus* was found in 102 samples, and 42% of the *S. aureus* strains carried the *tst* gene, either alone or alongside other toxin genes. Enterotoxin C was the most commonly detected enterotoxin, primarily found in dairy products. Among the *S. aureus* isolates, six carried only the *sea* gene, two carried both the *sea* and *seb* genes, and one carried both the *sea* and *sec* genes (Vitale et al. 2015). Some studies evaluated the expression of the *tst* gene from *S. aureus* in different types of milk at different temperatures and storage conditions. The study focused on pasteurized and UHT (ultra-high temperature) milk, both of which were inoculated with a monotoxic *S. aureus* strain. The results showed that the growth rate of *S. aureus* and the up-regulation of the *tst* gene were influenced by both storage temperature and milk type. Specifically, when pasteurized and UHT milk was stored at 15 °C and 22 °C, *S. aureus* exhibited a significant increase in its virulent potential over time. This effect was more pronounced in UHT milk, which was used as a "proof of concept". The researchers suggested that the presence of LAB in pasteurized milk could have a down-regulatory effect on the *tst* gene, potentially explaining the slightly lower up-regulation of the toxin in pasteurized milk compared to UHT milk (Babić et al., 2018). Also, the results showed that factors like milk type, storage time, and temperature all influenced the growth rate of *S. aureus*, and expression of the *sea* gene. Specifically, storing pasteurized milk at 22 °C for 24 hours led to a significant increase in *sea* gene expression compared to storage at 8 °C, which correlated with an increase in *S. aureus* numbers (up to 105 CFU/mL) and the detected SEA levels. Similarly, UHT-pasteurized milk stored at 22 °C for 24 hours and at 15 °C for 48 hours also showed significantly higher *sea* gene expression compared to milk stored at 8 °C, which corresponded with increased *S. aureus* growth and SEA synthesis (Babić et al., 2019). It has been reported that out of 220 milk and dairy product samples tested, *S. aureus* was present in 58 samples (26.36%), of which the -*sea* gene was present in 25% of *S. aureus* isolates from milk and in 18% of cheese isolates (Hassani, Doust & Mobarez, 2014). Bianchi et al. (2014) analyzed 1245 samples, including 848 raw milk samples and 397 dairy product samples, to detect 11 staphylococcal enterotoxins and enterotoxin-like toxins (SEA, SEB, SED, SEE, SEG, SEH, SEI, SER, SElJ, and SElP) between January 2010 and July 2011. Of the total number of samples, 481 samples were positive for *S. aureus*, whereas 255 (53%) were positive for one or more toxin genes, revealing 35 different enterotoxin-producing gene profiles. While the *seb* and *sec* genes were not detected, the *ser* gene was most commonly found, present in 134 (28%) isolates, followed by *sed* (25%) and *selj* (25%).

CONCLUSIONS

The expression of *sea* and *tst*-1 genes in *Staphylococcus aureus* presents significant health risks in milk and dairy products. Continuous monitoring and stringent control measures are essential to ensure food safety in the dairy industry. The growth and production of *S. aureus* enterotoxins are significantly impacted by environmental factors, such as water activity, temperature, pH, and others. Understanding these factors is crucial for developing effective food safety measures to prevent SFP.

Monitoring conditions during food processing and storage can help mitigate the risks associated with this bacterium. Understanding how temperature affects the growth of *S. aureus* and the expression of its enterotoxin genes in milk and milk products is crucial for food safety and quality control. When milk is stored above 8 °C for extended periods, the risk of *S. aureus* growth increases significantly, along with the likelihood of enterotoxin production. Maintaining proper refrigeration and monitoring temperature can reduce the risk of contamination and toxin production in milk and milk products.

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PRISUSTVO GENA ZA ENTEROTOKSINE *STAPHYLOCOCCUS AUREUS* U MLEKU I PROIZVODIMA OD MLEKA-PREGLED

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Sažetak: *Staphylococcus aureus* je veoma prilagodljiv mikroorganizam koji se može naći u različitim sredinama, kao što je ljudska kože, sluzokoža, u vazduhu, zemljištu i vodi. Poznat je po tome što poseduje širok spektar faktora virulencije, koji mu omogućava da izazove bolest. U ovom pregledu najviše pažnje biće posvećeno prisustvu gena za enterotoksin A i TSST-1 u mleku i proizvodima od mleka. *S. aureus* izolovan iz mleka može da sintetiše ili nosi gene za klasične enterotoksine, bilo pojedinačno ili u kombinaciji. Ekspresija mnogih gena *S. aureus* povezanih sa virulencijom reguliše system *agr* (accessory gene regulator), dvokomponentni system signalizacije koji smanjuje površinske proteine dok povećava sekretorne proteine, uključujući različite enterotoksine kao što su SEB, SEC, SED i TSST-1. Stafilokokno trovanje hranom (SFP) je obično povezano sa rastom stafilokoka u hrani bogatoj proteinima poput mesa, mleka i mlečnih proizvoda. Na rast *S. aureus* i proizvodnju enterotoksina u velikoj meri utiče složeni sastav hrane, uključujući faktore kao što su sadržaj soli, pH, dostupnost hranljivih materija, nivo kiseonika i temperatura. Kontinuirano praćenje temperature i stroge mere kontrole proizvodnje mleka su od suštinskog značaja da bi se osigurala bezbednost hrane u mlekarskoj industriji. Kada se mleko duže čuva pri temperaturi iznad 8°C, rizik od rasta *S. aureus* značajno se povećava, kao i verovatnoća proizvodnje enterotoksina.

Ključne reči: *bezbednost hrane, enterotoksin A, toksin-1 toksičnog šok sindroma, mleko*

Received: *13 January 2025/* ***Received in revised form:*** *06 March 2025/* ***Accepted:*** *07 March 2025*

Available online: *March 2025*