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Staphylococcus epidermidis SdrH, *SdrG*, and *SdrF* Expression in Commensal and Ocular Infection Isolates

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Abstract

Background: *Staphylococcus epidermidis* contaminates medical devices and produces biofilm, complicating antibiotic treatment for its elimination and causing a nosocomial health issue. The *sdr* genes (*sdrG*, *sdrF*, and *sdrH*) are involved in bacterial adhesion to the surface of medical devices for biofilm formation.

Objectives: To compare the presence and expression of the *sdr* gene in *S. epidermidis* from infectious and commensal isolates to propose them as a therapeutic target.

Methods: This is a descriptive, observational, and retrospective study. Infected ocular (n = 64), healthy conjunctiva (n = 46), and healthy skin (n = 53) isolates were genotyped using the Staphylococcal Chromosome *mec* (SCC*mec*) cassette to avoid clonality. Different genotypes representative of each isolation source selected isolates. In the selected isolates, *sdr* genes were determined by PCR, and RT-qPCR determined their expression.

Results: The *sdrG*, *sdrF*, and *sdrH* genes were present in the genome of all selected isolates. The expression level of the *sdr* genes was low in all isolates and there was no significant difference between different isolation sources.

Conclusions: The presence and expression of *sdrG*, *sdrF*, and *sdrH* genes are independent of the genotype and isolation source; this suggests that these proteins could be therapeutic targets to prevent the contamination of medical devices by *S. epidermidis*.

Keywords: *Staphylococcus epidermidis*, Infections, Health, Conjunctiva, Skin

1. Background

Staphylococcus epidermidis is a skin commensal ubiquitous in healthy humans with two lifestyles, one harmless and the other as an opportunistic pathogen. In recent years, *S. epidermidis* has become relevant to healthcare because it has been identified in clinical specimens as the sole causative agent of certain infections. *S. epidermidis* can cause bacteremia (1), sepsis (2), endocarditis, meningitis, and toxic shock syndrome (3), as well as superficial skin infections, ocular infections (4), and infections associated with indwelling medical devices such as shunts (5), catheters (6), and prosthetic joints (7). The majority of these infections result from the use of contaminated medical devices and, therefore, usually occur in hospitals, with high costs to the public health system. The ubiquity and persistence of *S. epidermidis* on human skin is a high-risk factor for the contamination of medical devices, which can come from hospital staff or patients and pose a serious health problem. For example, one study reported that of 24,179 cases of nosocomial bloodstream infections caused by contaminated devices in 49 hospitals in the USA, coagulase-negative staphylococci (including *S. epidermidis*) were responsible for 31% of the cases (8). The cost of hospital-acquired infections is a serious problem for the public health system; the cost associated with vascular catheter-associated bloodstream infections caused by *S. epidermidis* is estimated to be 2 billion annually in the USA (9). The scenario is complicated by the ever-expanding use of medical devices in recent years and the excessive use of antibiotics. The ability of this bacterium to form biofilms has led *S. epidermidis* to become an essential nosocomial pathogen; therefore, the search for new

therapeutic targets to prevent infections caused by this bacterium is of great medical interest.

A biofilm is an organized microbial ecosystem consisting of one or more microorganisms attached to a living or inert surface and enveloped in a self-produced extracellular matrix. Biofilm development occurs in three steps: Adherence, cellular aggregation, and degradation. In the adherence step, planktonic bacteria adhere to a biotic or abiotic surface; the adhered bacteria subsequently grow and secrete various compounds including polysaccharides, proteins, and extracellular DNA (eDNA). Biofilm maturation is achieved as these compounds form an extracellular matrix. Finally, the components of the biofilm matrix age, degrade and disassemble (10). *Staphylococcus epidermidis* can produce biofilm, which acts as a biological barrier to prevent the penetration of immune cells and antibodies. It contributes to its invasion, persistence, multidrug resistance, and host immune system evasion (11).

Staphylococcus epidermidis expresses proteins on its cell surface with biotic or abiotic adhesion characteristics during the biofilm adhesion step. One such protein is the serine-aspartate repeat (*sdr*) protein, which recognizes components of the host tissue extracellular matrix and binds to them to initiate biofilm formation (12). *S. epidermidis* expresses three *sdr* proteins (*sdrG*, *sdrF*, and *sdrH*), each with a specific affinity for extracellular matrix proteins of host tissues: *sdrG* binds specifically to fibrinogen and *sdrF* to collagen. However, the ligand for *SdrH* is currently unknown. *Staphylococcus epidermidis sdr* expression is a crucial factor in biofilm formation, an initial step in the contamination of medical devices or the infection process (13). In patients infected with *S. epidermidis*, *sdrG* is necessary to promote fibrinogen adherence. Moreover, *sdrG* expression increases, and as a patient response, antibodies against *sdrG* are present in the serum of infected individuals (12, 14). Similarly, *sdrF* is essential for establishing intracardiac valve infections, and infected patients produce antibodies against this protein (15). The production of high antibody levels against *sdrG* and *sdrF* in patients infected with *S. epidermidis* indicates the involvement of these proteins in the infection process. Therefore, *sdr* proteins can be considered potential therapeutic targets for controlling infections by this bacterium. Thus, blocking *sdr* proteins would help prevent *S. epidermidis* adhesion to the surface of medical devices or the host tissue extracellular matrix, reducing biofilm formation and preventing infections caused by this bacterium.

2. Objectives

Bacteria residing on the skin, such as *S. epidermidis*, may be potential causes of medical device contamination leading to infection. *S. epidermidis Sdr* proteins play an essential role in biotic and abiotic adhesion. However, it is currently unknown whether *Sdr* proteins are ubiquitous and differentially expressed in commensal and clinical isolates.

This study aimed to investigate the presence and expression of *sdr* genes in clinical isolates (from ocular infections) and commensal isolates (from healthy conjunctiva and skin) to propose them as therapeutic targets for preventing *S. epidermidis* infections.

3. Methods

3.1. Strains

We conducted a descriptive, observational, and retrospective study. A collection of *S. epidermidis* isolates was obtained in 2014, as previously reported (4), and this bacterial collection was donated for the present study. The bacterial collection comprises a total of 163 strains, distributed as follows: Isolates from ocular infections (n = 64), healthy conjunctiva (n = 46), and healthy skin (n = 53). Multilocus sequence typing (MLST) of ocular infection and healthy conjunctiva isolates was performed by Flores-Paez et al. (4); MLST of healthy skin isolates was performed separately with the same procedure as described (4).

The protocol for this bacterial study was approved by the Ethics Committee of the "Secretaría de Investigación y Posgrado (SIP)" of Instituto Politécnico Nacional (IPN), Mexico, under grant SIP-20240492.

Since the study used a collection of *S. epidermidis* isolates previously reported (4), patient-informed consent was not required for protocol approval. There was no direct interaction with patients, and the study did not impact their treatment.

3.2. DNA Extraction

Strains were cultured in trypticase soy broth (TSB, Sigma-Aldrich, Mexico) and incubated for 24 h at 37°C. They were then centrifuged at 21,633 × g for 1 minute.

Cells were resuspended in 200 µL of saline solution, and glass beads (≤ 100 µm, Sigma-Aldrich) were added. Tubes were shaken in a cell disruptor (Genie, USA) for 5 min. Two hundreds µL of Winston's solution (2% triton × -100, 1% SDS, 100 mM NaCl, 10 mM Tris-base, pH 8, 1 mM EDTA) and 400 µL of phenol-chloroform-isoamyl alcohol solution (25:24:1) were then added. The mixture was centrifuged at 13,845 × g for 15 min. The aqueous phase was separated, and the DNA was precipitated with isopropanol at double the original volume for 10 min. The mixture was centrifuged at 13,845 × g for 15 min, washed twice with 70% ethanol, and resuspended in sterile distilled water (16).

3.3. Genotyping

The determination of the Staphylococcal Chromosome *mec* (SCC*mec*) cassette consisted of multiplex PCR (17) with primers for the *mec*A gene and SCC*mec* types I, II, III, and V. Subsequently, another PCR with primers for type IV was performed. Genotyping by MLST was performed as described in (4).

3.4. PCR for sdr Genes

A reaction mixture of 25 µL was prepared containing 5 µL of 5x PCR buffer (Bioline; TN, USA), 0.5 µL of forward primer (10 µM), 0.5 µL of reverse primer (10 µM) (Table 1), 0.2 µL of Taq DNA polymerase (5U/µL) (Bioline), and 1 µL of genomic DNA. The conditions were 94°C for 2 min, followed by 35 cycles of 94°C for 30 sec, 55°C for 30 sec, and 72°C for 30 sec, with a final extension at 72°C for 5 min (16).

Table 1. Primers for *sdr* Genes

Genes	Primers Sequence
sdrG	F: ACACGACGATACAGCAAACTG
	R: CACCTTGTCCTTGACCTGAAC
sdrF	F: CACAGTTGAGTTTGAGACACC
	R: ATTGTGAGATTATCAGCTCCG
sdrH	F: AGTGGAGATGGTGCGCCTTT
	R: AGCACCCGTGGCTAATTGTC

Abbreviations: F, forward; R, reverse.

3.5. Total RNA Extraction

Cells were harvested from 3 mL of bacterial culture grown in TSB medium at 37°C for 24 h by centrifugation at 15,000 × g for 3 min. Subsequently, 200 µL of TE buffer and glass beads (≤ 100 µm) were added, and cell lysis was performed using a cell disruptor (Genie) at 865 × g for 7 min. Afterward, 1 mL of Trisure (Bioline) and 200 µL of chloroform were added. The mixture was incubated at room temperature for 10 min, centrifuged at 15,000 × g for 10 min, and the aqueous phase was collected. The volume obtained was doubled with isopropanol and incubated at -20°C for 12 h. The mixture was then centrifuged at 15,000 × g for 15 min, washed twice with 70% ethanol, and finally, the RNA was resuspended in 20 µL of sterile distilled water. In addition to extraction, total RNA was treated with DNase I (Bioline), using 2 µL of DNase I buffer (10×) and 1 µL of DNase I (10,000 units), and incubated at 37°C for 45 min. The enzyme was then inactivated at 75°C for 10 min (18).

3.6. cDNA Synthesis

For each sample, 3 µg of total RNA and 1 µL of Random Hexamer (Bioline) were used and adjusted to 10 µL with sterile distilled water. It was heated at 75°C for 10 min and immediately placed on ice. Subsequently, 4 µL of buffer (5×), 2 µL of DTT (0.1M Dithiothreitol; Bioline), 1 µL of dNTPs (10 mM, Bioline), and 2 µL of water were added and incubated for 2 min at room temperature. Then, 1 µL of M-MLV reverse transcriptase was added, and the mixture was incubated at room temperature for 8 min, incubated at 37°C for 1 hour, and finally at 65°C for 10 min (19).

3.7. Real-time PCR

The PCR mixture was prepared using the following reagents: Two µL of cDNA, 10 µL of master mix (SYBR green one master; Thermo Scientific, USA), 0.2 µL of each primer (Table 1), and 8.6 µL of water for each tube. PCR conditions were 94°C for 5 minutes, followed by 40 cycles of 94°C for 30 sec and 60°C for 40 sec. Relative expression was calculated using the $2^{-\Delta\Delta Ct}$ method, using the 16S rRNA gene as the reference (19).

3.8. Statistical Analysis

For analysis of genotyping by SCC*mec* types, the chi-squared test was used. For the presence of the *sdr* genes, a contingency table of presence and absence was formulated, and Fisher's exact test was used to compare the proportions. Relative expression of the *sdr* genes was compared using two-way ANOVA with Tukey's test for

Table 2. Genotype Percentage of Staphylococcal Chromosome *mec* Type

Variables	SCC*mec* Type				
Isolation source	**I**	**II**	**III**	**IV**	***mec*A Gen**
Ocular infection (n = 64) [a]	15 (23.3)	16 (25)	8 (12.5)	14 (21.8)	48 (75)
Healthy conjunctiva (n = 46) [b]	8 (17.3)	14 (30.4)	9 (19.5)	12 (26)	32 (69.5)
Healthy skin (n = 53) [c]	10 (18.8)	20 (37.7) [d]	11 (20.7)	9 (16.9) [d]	36 (67.9)

Abbreviation: *SCCmec*, Staphylococcal chromosome cassette *mec*.

[a] In 11 isolates were not found the SSC*mec* type.

[b] In 3 isolates were not found the SSC*mec* type.

[c] In 3 isolates were not found the SSC*mec* type.

[d] Significant difference ($P < 0.05$) with the chi-squared test.

between-group comparison. A value of $P < 0.05$ was considered significant for all tests.

4. Results

4.1. Staphylococcal Chromosome mec Genotyping and Multilocus Sequence Typing

The SCC*mec* types for each isolate were analyzed, and it was found that healthy skin and conjunctiva isolates predominantly exhibited type II SCC*mec*, with frequencies of 37.7% and 30.4%, respectively; the *mec*A gene was expressed in these samples with frequencies of 67.9% and 69.5%, respectively. In ocular infection isolates, type II SCC*mec* was the most prevalent (25%), followed by type I with a frequency of 23.3%. The frequency of the *mec*A gene in ocular infection isolates was 75% (Table 2). Type V SCC*mec* was not detected in any of the isolates studied. There was no significant difference between types of SCC*mec* for each isolate group ($P > 0.05$, chi-squared test), except for healthy skin isolates for types II and IV ($P < 0.05$, chi-squared test; Table 2).

The SCC*mec* type frequencies of each isolate were compared with MLST as previously reported by Flores-Paez et al., (4) and with those obtained in this work for healthy skin isolates. As shown in Tables 3 - 5, the isolates from each source were selected based on their ST and all four SCC*mec* types, with the aim of genotypically diverse isolates (non-clonal). These selected isolates were used for the additional determinations.

4.2. Genomic Detection of sdr Genes

Strains corresponding to healthy skin showed the presence of *sdrG*, *sdrF*, and *sdrH* genes with frequencies of 100%, 84.6%, and 100%, respectively. In strains corresponding to healthy conjunctiva, the gene frequencies were 92.8% for *sdrG*, 92.8% for *sdrF*, and 92.8% for *sdrH*. Finally, *sdrG*, *sdrF*, and *sdrH* genes were found for ocular infection strains with frequencies of 100%, 88.8%, and 100%, respectively. No significant difference was observed in the presence of *sdr* genes based on the isolation source ($P > 0.05$, Fisher's exact test).

4.3. Relative mRNA Expression of sdr Genes

The relative expression of *sdrG*, *sdrF*, and *sdrH* genes from the ocular infection strain group showed similar values ranging from 0.7 to 0.8 (Table 3), with no significant difference observed among the strains ($P > 0.05$; two-way ANOVA test). The same pattern was observed for the healthy conjunctiva and skin strain groups, with relative mRNA expression values ranging from 0.6 to 0.8 and no significant difference found among the strains ($P > 0.05$; two-way ANOVA test; Tables 4 and 5). Relative values obtained were less than 1, indicating that expression was lower than that of the reference gene 16S rRNA, suggesting weak expression of the *sdr* genes.

5. Discussion

The level of expression of *Sdr* proteins in clinical and commensal isolates is previously unestablished. In this study, we compared the presence and expression of mRNA from *sdr* genes between isolates from ocular infections and healthy samples of conjunctiva and skin.

The presence of *sdr* genes was high in both ocular infection and commensal strains, with no difference between the isolation sources, suggesting that *sdr* genes are essential for the adhesion of *S. epidermidis* regardless of virulence capacity. This indicates that in medical

Table 3. The Relative Expression of *sdr* Genes from Ocular Infection [a]

Ocular Infection and Strains	ST	SCC*mec*	mRNA Relative Expression		
			sdrG	*sdrF*	*sdrH*
35	2	I	0.9 ± 0.07	0.7 ± 0.09	0.7 ± 0.06
168	2	II	0.7 ± 0.02	0.8 ± 0.05	0.7 ± 0.04
213	2	III	0.8 ± 0.03	0.7 ± 0.08	0.8 ± 0.07
53	2	IV	0.7 ± 0.04	0.6 ± 0.08	0.6 ± 0.06
50	9	II	0.7 ± 0.04	0.8 ± 0.08	0.8 ± 0.07
2050	9	III	0.9 ± 0.01	0.8 ± 0.06	0.8 ± 0.03
58	9	IV	0.7 ± 0.02	0.8 ± 0.05	0.7 ± 0.07
96	10	I	0.8 ± 0.05	0.6 ± 0.08	0.5 ± 0.07
1700	23	I	0.7 ± 0.05	0.8 ± 0.06	0.7 ± 0.05
54	23	II	0.8 ± 0.03	0.9 ± 0.07	0.6 ± 0.08
199	23	IV	0.9 ± 0.01	0.8 ± 0.08	0.7 ± 0.06
98	38	I	0.9 ± 0.04	0.8 ± 0.09	0.9 ± 0.02
1948	46	IV	0.9 ± 0.04	0.7 ± 0.09	0.8 ± 0.04
1654	71	III	0.8 ± 0.05	0.7 ± 0.03	0.6 ± 0.05
90	87	I	0.8 ± 0.07	0.8 ± 0.03	0.7 ± 0.06

Abbreviations: ST, sequence type; SCC*mec*, Staphylococcal chromosome cassette *mec*.

[a] No significant difference P > 0.05 with two-way ANOVA test was found.

devices contaminated with *S. epidermidis* and used in hospital patients, the bacterium has the potential to adhere to the extracellular matrix of the host tissue. In surgeries for medical implants, *S. epidermidis* isolates from orthopedic and catheter-associated infections have been found to have the *sdrG* gene present in 78% to 91% of cases (20). These results are very similar to those obtained in our study. Arrecubieta et al. indicate that *sdr* genes are present in most species of the Staphylococcus genus (15); with our data, this suggests that these genes are a fundamental component of the *S. epidermidis* genome and the potential for tissue attachment. Additionally, they could be considered therapeutic targets to reduce this bacterium's contamination of medical devices.

We did not find significant differences in *sdr* gene expression among the three isolation sources. This indicates that any individual (hospital staff or patient) may carry *S. epidermidis* on their skin with the potential to express *Sdr*s at a low level. This could result in contamination of medical devices and subsequent infection. Therefore, the expression of the *sdr* gene, regardless of the source of isolation, suggests a potential risk in the initial adhesion of *S. epidermidis* to host tissue and the establishment of infection. As a result, using strategies to block or inhibit the *Sdr* proteins could help prevent infection.

Several strategies to block *Sdr* proteins have been documented for controlling *S. epidermidis* infections. One of these is using antibodies against *SdrG*, which has been shown to prevent *S. epidermidis* adherence to catheters (21). Another related approach is immunization with a subdomain of *SdrG* to induce antibody production; this has been shown to prevent *S. epidermidis* infection in a mouse model (14). Regarding medical devices, an effective strategy for combating infection is using biomaterials coated with antimicrobial compounds specific to *S. epidermidis*. One approach is to coat catheters with osteopontin under shear flow, which prevents early bacterial adhesion and subsequent biofilm formation (22). Another is to use polymer blends of polyvinyl chloride (PVC) and polystyrene-ethylene-butylene-styrene (SEBS) in catheter tips (23). Another option is using silver nanoparticles in chitosan nanogel (24), which inhibits *S. epidermidis* infection.

Another option is to change the type of material used in medical devices. Materials with highly hydrophobic surfaces, such as dacron or polystyrene, increase *sdrF* gene expression, resulting in a notable enhancement in bacterial adhesion (15). Consequently, contaminated

Table 4. The Relative Expression of *sdr* Genes From Healthy Conjunctiva [a]

Healthy Conjunctiva and Strains	ST	SCC*mec*	mRNA Relative Expression		
			sdrG	*SdrF*	*SdrH*
139	2	IV	0.7 ± 0.08	0.8 ± 0.01	0.8 ± 0.01
114	4	I	0.7 ± 0.04	0.8 ± 0.03	0.7 ± 0.04
92	4	II	0.7 ± 0.03	0.8 ± 0.05	0.7 ± 0.09
10	5	I	0.6 ± 0.08	0.7 ± 0.04	0.6 ± 0.04
106	5	II	0.6 ± 0.08	0.6 ± 0.06	0.6 ± 0.05
14	5	III	0.7 ± 0.03	0.7 ± 0.01	0.7 ± 0.07
8	5	IV	0.8 ± 0.04	0.7 ± 0.03	0.8 ± 0.09
48	9	II	0.7 ± 0.06	0.8 ± 0.06	0.7 ± 0.03
30	9	III	0.8 ± 0.03	0.7 ± 0.04	0.7 ± 0.05
137	10	I	0.7 ± 0.06	0.8 ± 0.06	0.7 ± 0.07
9	10	II	0.8 ± 0.04	0.7 ± 0.05	0.7 ± 0.03
97	10	III	0.7 ± 0.03	0.8 ± 0.03	0.8 ± 0.04
22	10	IV	0.8 ± 0.04	0.7 ± 0.02	0.7 ± 0.06
35	23	II	0.8 ± 0.06	0.7 ± 0.07	0.6 ± 0.05
13	26	II	0.8 ± 0.06	0.7 ± 0.06	0.8 ± 0.06
31	43	IV	0.7 ± 0.02	0.6 ± 0.09	0.8 ± 0.01
103	48	III	0.6 ± 0.08	0.7 ± 0.07	0.6 ± 0.06
128	118	I	0.7 ± 0.03	0.8 ± 0.05	0.8 ± 0.04
114	118	II	0.6 ± 0.03	0.6 ± 0.05	0.7 ± 0.01
126	118	IV	0.8 ± 0.02	0.08 ± 0.01	0.7 ± 0.06
17	135	IV	0.9 ± 0.06	0.8 ± 0.09	0.7 ± 0.05
131	173	II	0.8 ± 0.01	0.7 ± 0.08	0.7 ± 0.09
94	238	III	0.8 ± 0.06	0.9 ± 0.03	0.8 ± 0.05
29	494	II	0.5 ± 0.07	0.7 ± 0.03	0.6 ± 0.09

Abbreviations: ST, sequence type; SCC*mec*, Staphylococcal chromosome cassette *mec*.

[a] No significant difference P > 0.05 with two-way ANOVA test was found.

Table 5. The Relative Expression of *sdr* Genes From Healthy Skin [a]

Healthy skin and Strains	ST	SCC*mec*	mRNA Relative Expression		
			sdrG	*sdrF*	*sdrH*
17	2	I	0.8 ± 0.08	0.8 ± 0.03	0.7 ± 0.03
22	2	II	0.9 ± 0.03	0.8 ± 0.08	0.9 ± 0.01
3	2	III	0.8 ± 0.02	0.9 ± 0.02	0.8 ± 0.07
60	2	IV	0.6 ± 0.01	0.7 ± 0.04	0.6 ± 0.03
52	9	I	0.7 ± 0.03	0.7 ± 0.08	0.7 ± 0.02
57	23	I	0.7 ± 0.05	0.7 ± 0.04	0.8 ± 0.02
58	23	II	0.7 ± 0.07	0.8 ± 0.01	0.7 ± 0.08
37	189	I	0.8 ± 0.03	0.8 ± 0.04	0.7 ± 0.02
7	189	III	0.7 ± 0.04	0.6 ± 0.09	0.8 ± 0.06
47	370	II	0.7 ± 0.03	0.7 ± 0.08	0.7 ± 0.07
51	370	II	0.7 ± 0.03	0.8 ± 0.02	0.7 ± 0.09
9	481	II	0.9 ± 0.01	0.8 ± 0.03	0.8 ± 0.06

Abbreviations: ST, sequence type; SCC*mec*, Staphylococcal chromosome cassette *mec*.

[a] No significant difference P > 0.05 with two-way ANOVA test was found.

medical devices made with these materials may promote bacterial adhesion and contribute to infection. These measures to reduce *S. epidermidis* infections in hospitals are currently being developed. It is anticipated that these strategies will help resolve the issues associated with *S. epidermidis* infection in the near future.

Although this work focused on ocular infection isolates, the results suggest that it may also occur in other *S. epidermidis* infections related to the use of

medical devices such as shunts (5), catheters (6), and prosthetic joints (7). In these cases, the presence and expression of *sdr*s genes will also be involved in the initial adhesion process for subsequent biofilm formation.

5.1. Conclusions

The relative mRNA expression of the *sdrG*, *sdrF*, and *sdrH* genes in ocular infection and commensal isolates of *S. epidermidis* is homogeneous and weak in each case. This suggests that, in human health, the skin of healthy individuals carries *S. epidermidis* with the potential to infect the eye, as it presents and expresses *Sdr* proteins (which are involved in adhesion to host tissue). Moreover, healthy individuals are also a source of *S. epidermidis* contamination for medical devices. Introducing these devices into the patient can cause infections due to the bacteria's adhesion via *Sdr* proteins and its difficult removal due to biofilm formation. Therefore, from a public health perspective, it is important to avoid hand-eye contact, especially in immunocompromised individuals. Additionally, medical staff should be educated about the risks of contact between medical devices and their skin.

On the other hand, *Sdr* proteins could be therapeutic targets to prevent the contamination of medical devices by *S. epidermidis* through the use of catheters coated with anti-adhesion molecules. Immunization with *Sdr* antigens may also be an alternative prevention strategy for diseases caused by *S. epidermidis*.

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Footnotes

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