ORIGINAL ARTICLE

Annals of Agricultural and Environmental Medicine
www.aaem.pl

Toxicological profile of sodium lauryl sulfate – insights into cytotoxic, cardiotoxic, developmental and antimicrobial effects across biological models

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Stachurski P, Magryś A, Rudkowska M, Boguszewska-Czubara A, Żelazowska R, Sobieszczański J, Waligórska M, Mieszawska S. Toxicological profile of sodium lauryl sulfate – insights into cytotoxic, cardiotoxic, developmental and antimicrobial effects across Biological models. Ann Agric Environ Med. doi:10.26444/aaem/221032

Abstract

Introduction and Objective. Sodium lauryl sulfate (SLS) is a widely used surfactant present in many personal care products. Despite its broad application, increasing evidence suggests potential cytotoxic and ecotoxic effects. The aim of the study is to evaluate SLS toxicity using *in vivo, in vitro* and microbiological models to assess risks to health and the environment.

Materials and Method. Toxic effects were assessed in zebrafish (Danio rerio) embryos (OECD 236) and mammalian cell lines: human fibroblasts (BJ) and rat cardiomyoblasts (H9C2). Cell viability was measured by MTT assay after exposure to SLS (1–200 µg/mL). Zebrafish embryos were observed for mortality, developmental changes and hatching rate after 96-hour exposure. Antimicrobial and antibiofilm activities were tested against *Streptococcus mutans* and *Enterococcus faecalis* using microdilution and biofilm assays.

Results. SLS showed marked cytotoxic and cardiotoxic activity (LC_{50}: 38.46 µg/mL for BJ and 36.97 µg/mL for H9C2 cells). In zebrafish, developmental delay and morphological alterations occurred with LC_{50} = 2.654 mM, indicating moderate acute toxicity. No significant changes in heart rate or locomotor activity were detected at sublethal doses. Antimicrobial effects were strain-dependent: *S. mutans* showed moderate sensitivity (MIC = 50 µg/mL), while *E. faecalis* remained resistant. Biofilm assays confirmed limited antibiofilm potential.

Conclusions. SLS exhibits considerable cytotoxicity and moderate developmental toxicity, implying potential health and environmental risks. Limited antimicrobial and antibiofilm effects question its safety and efficacy in oral hygiene formulations, supporting the need for stricter evaluation of its use.

Key words

cytotoxicity, biofilm, cardiotoxicity, zebrafish, antimicrobial activity, ecotoxicology, sodium lauryl sulfate

INTRODUCTION

Sodium lauryl sulfate (SLS) is one of the most ubiquitous surfactants used in a wide array of industrial and consumer products, including detergents, cosmetics, and personal care items. Its effectiveness as a detergent stems from its amphiphilic nature, allowing it to interact with both water and oil, thereby breaking down and removing dirt and grease [1]. Chemically, SLS belongs to the class of sodium alkyl sulfates and is primarily composed of dodecyl sulfate, a 12-carbon chain connected to a sulfate group (Fig. 1). This structure is central to its role as a surfactant.

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Received: 04.11.2025; accepted: 23.04.2026; first published: 28.05.2026

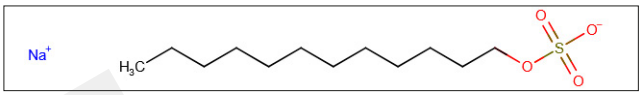

Figure 1. Chemical structure of sodium lauryl sulfate

The production of SLS involves the sulfation of lauryl alcohol, which is commonly obtained through the hydrolysis and hydrogenation of natural oils such as coconut oil or palm kernel oil. The sulfation process typically uses chlorosulfonic acid or sulfur trioxide, followed by neutralization with sodium hydroxide or sodium carbonate to yield the final product [2]. Commercially available samples of SLS often include a mixture of alkyl sulfates, with dodecyl sulfate being the dominant component due to the synthesis process. SLS is available in various forms, including powders, pellets, and crystals, and it is characterized by its white or pale yellow appearance, soapy texture, and slightly bitter taste [3–5].

Despite its widespread use, SLS has been a subject of considerable scrutiny due to its potential toxic effects. SLS is known to be a moderately toxic substance, with acute and chronic exposure posing risks to human health. The primary mechanism of toxicity is linked to its surfactant properties, which can disrupt cell membranes, leading to cellular damage and inflammation [6]. SLS can also induce the release of cytokines and cause conformational changes in proteins, further contributing to its toxic effects [7]. Additionally, its ability to damage beneficial bacteria that reside in various parts of the body raises concerns about its overall impact on human health.

One of the most significant concerns regarding SLS is its potential to cause skin toxicity [8, 9]. The skin, being the largest organ of the body, serves as the first line of defence against environmental stressors, including harmful chemicals. SLS, when used in high concentrations or over prolonged periods, has been shown to cause irritation and damage to the skin. This is particularly concerning in products like cosmetics and soaps, where SLS is often a key ingredient.

The irritation caused by SLS is not limited to the skin. It can also affect the eyes, mucous membranes, and the upper respiratory tract. Prolonged exposure to SLS has been linked to more severe health issues, including chronic inflammation and possibly carcinogenic effects, although the latter remains a topic of ongoing research. The toxic effects of SLS are dose-dependent, with higher concentrations posing a greater risk. However, even at lower concentrations, the cumulative effect of long-term exposure can lead to significant health issues [3, 4].

SLS also presents challenges in the context of environmental toxicity, Its widespread use and eventual release into water systems have raised concerns about its impact on aquatic life. SLS can cause harm to marine organism by disrupting their cell membranes, similar to its effects on human cells [10–12]. The biodegradability of anionic surfactants in the environment is another critical factor, as incomplete degradation can lead to the accumulation of harmful byproducts that further exacerbate their toxic effects on ecosystems [13].

Given the potential health and environmental risks associated with SLS, it is crucial to understand its toxicological profile comprehensively. To evaluate the toxicological profile of SLS, it is essential to employ robust and relevant biological models. Zebrafish (*Danio rerio*) and cell lines such as BJ (human fibroblasts), H9C2 (rat cardiomyoblasts) offer distinct and complementary advantages in assessing the mechanisms of SLS toxicity, dose-response relationships, and long-term effects of exposure.

Zebrafish are a widely accepted model organism in toxicology due to their high genetic similarity to humans, rapid development, and transparency during early life stages, which allow for real-time observation of physiological and developmental changes. They are particularly valuable for studying the effects of chemicals on organ development, neurotoxicity, and systemic responses due to their well-characterized organ systems and ease of genetic manipulation. Zebrafish also facilitate high-throughput screening, making them ideal for evaluating the toxicological impact of various concentrations of SLS over different exposure periods. This model provides insights into whole-organism effects, including developmental and reproductive toxicity, which are critical for assessing the broader ecological and health implications of SLS exposure [14–17].

BJ cells, derived from human skin fibroblasts, and H9C2 cells, derived from rat heart myoblasts, serve as relevant *in vitro* models to investigate the cellular and molecular mechanisms of SLS toxicity in specific tissues. BJ cells are particularly useful for studying dermal toxicity, given the widespread use of SLS in personal care products that come into direct contact with human skin. Evaluating cytotoxicity and inflammatory responses studies can provide valuable data on potential skin irritation and damage mechanisms [18, 19].

On the other hand, H9C2 cells provide a model for assessing cardiotoxicity, which is crucial given emerging concerns about the broader systemic effects of SLS, including its impact on cardiac cells. These cells allow for detailed examination of the cellular effects of SLS, including alterations in cell viability, mitochondrial function, and apoptotic pathways, which are important for understanding the risks associated with systemic absorption and chronic exposure [20].

In addition to its effects on eukaryotic cells, SLS can also influence the growth and viability of microorganisms such as *Streptococcus mutans* and *Enterococcus faecalis*. These bacteria are of particular interest because they are common inhabitants of the human oral cavity and are implicated in dental caries and endodontic infections, respectively. Evaluating how SLS affects their viability and biofilm formation can provide valuable insights into its antimicrobial potential and its dual role as both a cytotoxic and antibacterial agent, depending on concentration and exposure time.

OBJECTIVE

The aim of the study is to provide a comprehensive evaluation of the ecotoxicity and general toxicity of SLS. The combined use of zebrafish and BJ and H9C2 cell lines allows for a comprehensive evaluation of SLS toxicity from both organism and cellular perspectives. In addition, the microbiological aspect of the study focuses on evaluating the antibacterial potential of SLS against *Streptococcus mutans* and *Enterococcus faecalis*, including the determination of minimum inhibitory concentration (MIC), as well as the assessment of its anti-biofilm properties. This integrative approach enhances the understanding of SLS's toxicological mechanisms, supports the identification of specific dose-response relationships, and provides insights into its antimicrobial potential and the potential long-term health and ecological effects associated with exposure.

MATERIALS AND METHODS

Drugs. Sodium lauryl sulfate (SLS, CAS 151–21–3) was purchased from Sigma-Aldrich.

Culture of BJ and H9C2 Cells. Human fibroblast BJ cells (CRL-2522) and rat H9C2(2–1) cells (CRL-1446) were obtained from the American Type Culture Collection (ATCC; Manassas, VA, USA). The cells were maintained at 37 °C in a humidified incubator with 5% CO2. BJ cells were grown in Eagle's Minimum Essential Medium (EMEM), while H9C2 cells were cultured in Dulbecco's Modified Eagle's Medium (DMEM). Both culture media were supplemented with

10% heat-inactivated foetal bovine serum (FBS), penicillin (100 U/mL), and streptomycin (100 µg/mL) to prepare the complete growth medium.

MTT assay for cell viability. BJ and H9C2 cells were seeded separately in 96-well plates at a density of 5×10^4 cells per well in their respective EMEM and DMEM media, containing 10% FBS and 1X antibiotic solution. The cells were incubated at 37˚C with 5% CO2. After washing the cells with 100 µL of 1X PBS, they were treated with varying concentrations (1–200 µg/mL) of SLS and incubated again under the same conditions for 24 hours. Following the treatment, the medium was removed, and the cells were incubated with MTT (0.5 mg/mL in 1X PBS) at 37˚C for 4 hours in a CO2 incubator. After incubation, the MTT solution was removed, and the cells were washed with 100 µL of PBS. The resulting formazan crystals were dissolved in 100 µL of DMSO and thoroughly mixed. The formazan dye, which turns a purple-blue colour, was measured for absorbance at 570 nm using a microplate reader.

For statistical analysis, non-linear regression was applied to the dose-response data, using a 4-parameter sigmoidal curve to calculate the LC_{50}. The analysis was conducted with Prism v10.0.0 from GraphPad software.

Zebrafish study. Stock solutions of the tested drugs were prepared in a zebrafish medium – medium E3 (pH 7.1–7.3), containing 5.0 mM NaCl, 0.17 mM KCl, 0.33 mM CaCl2, and 0.33 mM MgSO4. The solutions were diluted to the desired test concentrations immediately before use in the experiments. The zebrafish of the AB strain were kept at the Experimental Medicine Centre of the Medical University in Lublin, Poland.

Acute toxicity assay. The zebrafish embryo assay followed a modified version of the OECD Guidelines for Testing of Chemicals (OECD, 2013). For each treatment group, at least 40 embryos were randomly selected and transferred to a 6-well plate containing 4 mL of the tested concentrations, as well as a control solution, no later than 90 minutes post-fertilization. The embryos were examined under a light microscope (Stemi 508, Zeiss), and 20 viable, fertilized embryos were chosen and moved to 96-well plates within 3 hours post-fertilization (hpf). The embryos were individually incubated in 200 µL of the tested concentrations or control solutions (medium E3). The drug solutions were gently refreshed every 24 hours with freshly prepared solutions. The embryos were exposed to either the control or treatment solutions for a total of 96 hours. The plates were kept in an incubator at 28.5 ± 0.5 °C with a 14:10 hour light/dark cycle.

The embryos were observed every 24 hours under a stereomicroscope, with survival, hatching rate, and developmental abnormalities recorded. At 96 hours post-fertilization, the number of dead embryos was counted, and the concentration lethal to 50% of the embryos (LC_{50} value) was calculated. Death was determined by the coagulation of embryos or the absence of a heartbeat, observed over 60 seconds. Additionally, at 96 hpf, the heart rate was measured. The larvae were allowed to equilibrate to room temperature for 30 minutes before counting the heartbeats under a stereomicroscope for 20 seconds, and these values were multiplied by 3 to obtain the beats per minute (bpm).

Locomotor activity. For assessing locomotor activity, a test was conducted on 5-day post-fertilization (dpf) larvae, with one larva placed in each well of a 96-well plate. The larvae were incubated in 200 µL of E3 medium or tested concentrations of SLS for 30 minutes prior to the test. Locomotor activity was evaluated using EthoVision XT video tracking software (Noldus), measuring the distance moved by the zebrafish larvae in 10 minutes under light conditions. After the experiment, the larvae were euthanized by immersion in a 15 µM solution of tricaine methanesulfonate (Sigma Aldrich).

Bacterial strains and culture conditions. Two reference bacterial strains were used in this study: *Streptococcus mutans* (ATCC 35668) and *Enterococcus faecalis* (ATCC 700221). Strains were cultured aerobically on Columbia agar supplemented with 5% sheep blood (Becton Dickinson) and incubated at 37 °C for 24–48 hours prior to testing.

Determination of the minimum inhibitory concentration (MIC) of SLS. The antimicrobial activity of SLS was evaluated using the broth microdilution method in accordance with EUCAST guidelines. Bacterial suspensions were prepared in sterile saline and adjusted to a 0.5 McFarland standard (approximately 1.2×10^8 CFU/mL), then diluted to 5×10^5 CFU/mL in Brain Heart Infusion (BHI) broth. SLS was serially diluted in 96-well plates, and 20 µL of bacterial suspension was added to each well, resulting in final concentrations ranging from 0- 200 µg/mL. Plates were incubated at 35 °C for 18 hours. The MIC was defined as the lowest concentration with no visible growth or metabolic activity as confirmed by MTT (0.2 mg/mL) conversion after 30 minutes of incubation. All samples were tested in triplicate.

ANTI-BIOFILM ACTIVITY ASSESSMENT

Biofilm formation and treatment. Bacterial suspensions (5×10^6 CFU/mL) were prepared in BHI supplemented with 1% sucrose. In 96-well plates, 180 µL of SLS (from a 500 µg/mL stock) and 20 µL of bacterial suspension were combined. Control wells without SLS provided growth controls, and wells with only 200 µL of broth provided sterility controls. Plates were incubated at 37 °C for 24 hours. After incubation, wells were washed twice with PBS to remove planktonic cells.

MTT Assay (determination of bacterial cell viability in biofilm). Fresh BHI and 40 µL of MTT (1 mg/mL) were added to each well and incubated at 37 °C for 3 hours. Absorbance at 570 nm was measured. Bacterial cells' viability percentage was assessed by comparing the percentage of treated cells to controls and calculated as:

$$MTT\ \%\ cell\ viability = \frac{A570\ of\ treated\ bacterial\ cells}{A570\ of\ control\ bacterial\ cells} \times 100\%$$

The minimum biofilm inhibitory concentration (MBIC) was defined as the lowest concentration showing ≥90% inhibition of biofilm viability.

Crystal violet staining (determination of biofilm biomass). After PBS washes, biofilms were heat-fixed (65 °C, 1 hour), stained with 0.1% crystal violet for 15 minutes, and rinsed. Stain was eluted with 96% ethanol, and absorbance at 570 nm

was recorded. Biofilm biomass was interpreted relative to control wells. The cut-off OD value (ODc) was calculated as: **ODc = mean OD of negative control + (3 × SD).** Biofilm formation was categorized as follows:

- **Non-adherent**: OD ≤ ODc.
- **Weak biofilm producer**: ODc < OD ≤ 2×ODc.
- **Moderate**: 2×ODc < OD ≤ 4×ODc.
- **Strong**: OD > 4×ODc.

Statistical analysis. For statistical analysis, non-linear regression was applied to the dose-response mortality data. A 4-parameter sigmoidal curve was used to calculate the LC_{50}. Data were analyzed using one-way ANOVA, followed by Tukey's *post-hoc* test, respectively. A confidence limit of $p < 0.05$ was considered statistically significant. The analysis was performed using GraphPad Prism v8.3.1 software. Approval of the Local Ethical Commission was not required for experiments involving larvae up to 120 hpf (5 dpf),.

RESULTS

Cytotoxicity. Firstly, evaluation of the toxicological effect caused by exposure to human fibroblasts (BJ) by SLS was performed to evaluate general cytotoxicity. SLS elicited toxicity resulting in IC_{50} of 38.46 µg/mL (Fig. 2).

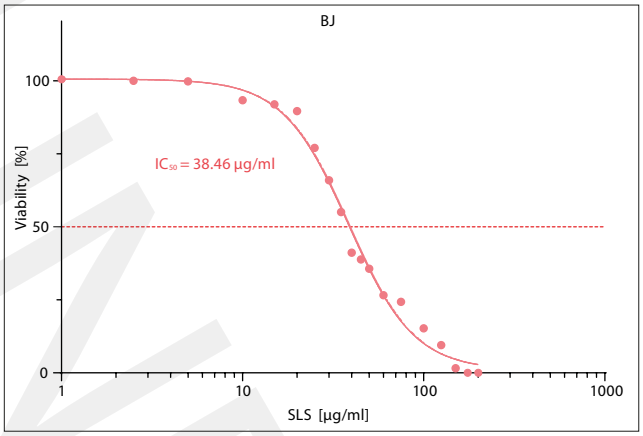

Figure 2. Cytotoxic effects of SLS on human fibroblasts (BJ cells). The viability of BJ cells was assessed following 24 hours treatment with increasing concentrations of SLS (0–200 µg/mL). The inhibitory concentration 50 (IC50) was determined to be 38.46 µg/mL

Cardiotoxicity. Then, rat myoblasts were used to evaluated the toxicological effect caused by exposure to SLS using rat myoblasts H9C2 to establish cardiotoxicity. SLS elicited cardiotoxicity resulting in IC_{50}=36.97 µg/mL (Fig. 3).

***In vivo* toxicity.** First, the toxicological effect caused by exposure to SLS using the modified Fish Embryo Acute Toxicity (FET) Test [21] was evaluated. SLS elicited low toxicity, resulting in LC_{50} of 765 µg/mL (Fig. 4A). The delay of hatching rate was observed at 96 hpf at the concentrations of 721 µg/mL (Fig. 4B), and a similar effect was observed at 72 hpf (data not shown). SLS displayed no effect on heart rate [F (3, 28) = 2.547, P = 0.0761] (Fig. 4C), nor distance swam by larvae [F(3, 28) = 0.575, P = 0,6362] (Fig. 4D). No changes in the development of pigment (PIG) were observed. Morphological alterations were observed for SLS

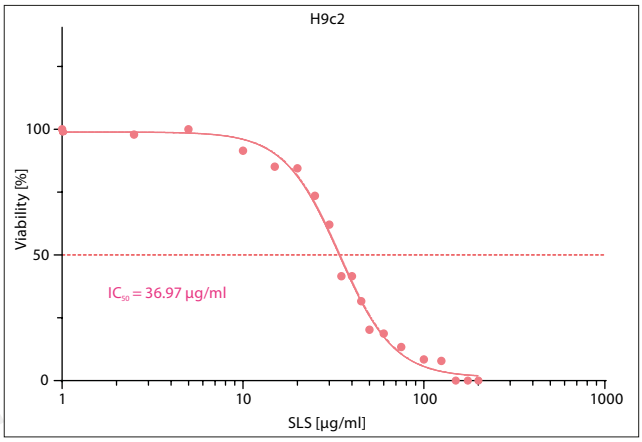

Figure 3. Cardiotoxic effects of SLS on rat myoblast H9c2 cells. The viability of H9c2 cells was assessed following 24 hours treatment with increasing concentrations of SLS (0–200 µg/mL). The inhibitory concentration 50 (IC50) was determined to be 36.97 µg/mL

at concentrations of 288 µg/mL (10 %) and 721 µg/mL (20 %) (Fig. 5).

Antibacterial activity of SLS. The minimum inhibitory concentration (MIC) of SLS was determined for 2 bacterial strains: *Enterococcus faecalis* and *Streptococcus mutans*. In the current study, SLS exhibited MIC values of 200 µg/mL for *E. faecalis* and 50 µg/mL for *S. mutans*, indicating a strain-dependent antimicrobial effect. While SLS showed moderate inhibitory activity against *S. mutans*, the high MIC observed for *E. faecalis* suggests that this compound alone may not be sufficiently potent against more resilient or biofilm-forming Gram-positive bacteria (Tab. 1). The Table shows the concentrations of SLS that inhibited (MIC) the tested organisms. The values are given as the highest value out of 3 replicates.

Table 1. MIC values (µg/mL) of SLS for different bacterial strains

	Bacterial strains	
	Enterococcus faecalis	*Streptococcus mutans*
SLS MIC (µg/mL)	200	50

MIC – Minimum inhibitory concentration: the lowest concentration of a compound that inhibits bacterial growth

Anti-biofilm activity of SLS. The minimum biofilm inhibitory concentration (MBIC) was evaluated to determine the effectiveness of SLS in preventing biofilm formation across the tested bacterial strains. SLS did not achieved >90% inhibition of biofilm metabolic activity, with MBIC values estimated as >200 µg/mL for both *E. faecalis* and *S. mutans*, confirming its limited anti-biofilm activity. (Figure 6, Table 2).

It should be noted that the MTT assay used measurements of metabolic activity in biofilm-embedded cells, and reductions in metabolic activity did not necessarily correspond to complete biofilm eradication.

The minimum biofilm inhibition concentration (MBIC) was expressed as the lowest concentration that shows 90% inhibition of biofilm formation.

Effect of SLS on biofilm biomass. Crystal violet assays demonstrated robust biofilm formation in control conditions.

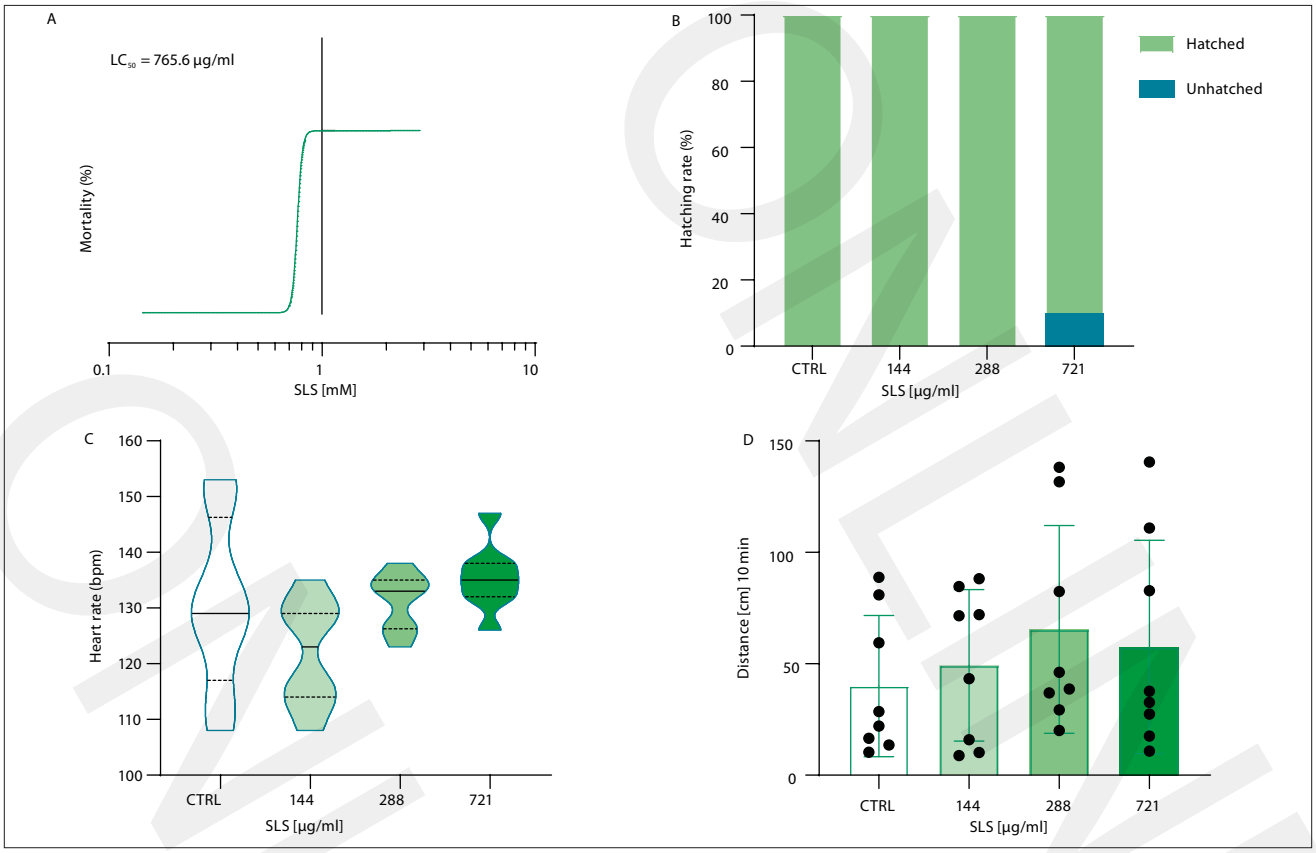

Figure 4. Toxicological profile of SLS in larval zebrafish model. LC_{50}, n = 20 (A); hatching rate, n = 20 (B), and heart rate, n = 8 (C) were calculated at the 96-hour time point of the FET test. Locomotor activity (D) was investigated in a different batch of larvae (<120 hpf) after 30 min exposure to SLS in 10 min light period. Data are presented as means ± SEM; one-way ANOVA followed by Tukey's test

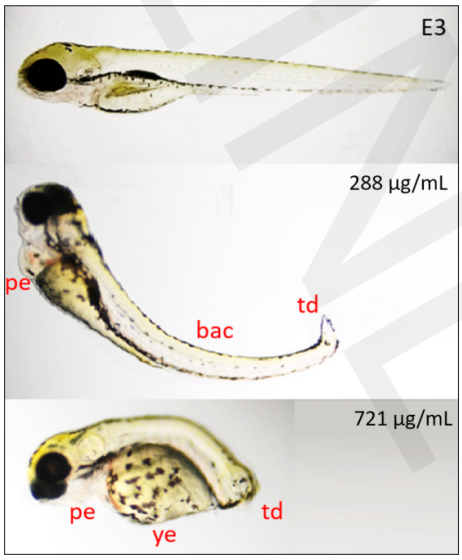

Figure 5. Morphological changes induced by SLS at the 96-hour time point of observations in FET test; bac – body axis curvature, td – tail deformation, pe – pericardial edema, ye – yolk sac oedema

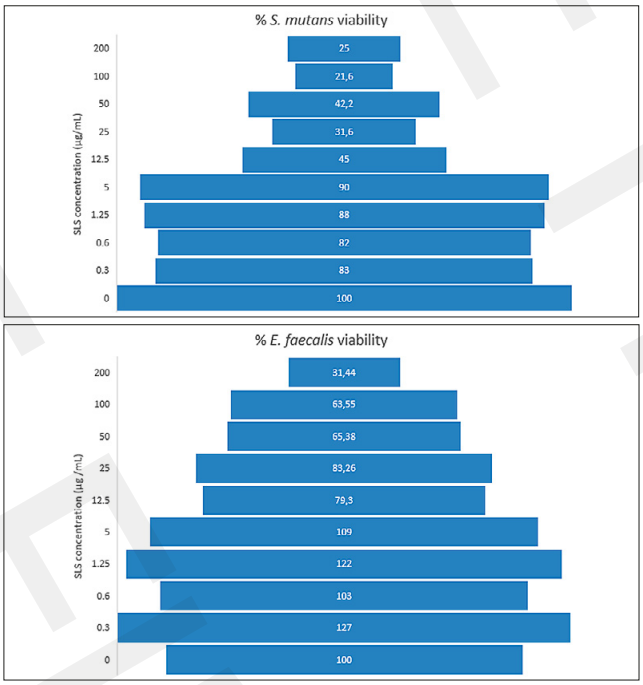

Figure 6. Anti-biofilm activity of sodium lauryl sulfate (SLS) against *Enterococcus faecalis* and *Streptococcus mutans*. Biofilms were treated with increasing concentrations of SLS, and bacterial metabolic activity was assessed using the MTT assay. MBIC (Minimum Biofilm Inhibitory Concentration) was defined as the lowest concentration resulting in ≥90% inhibition of metabolic activity and was not achieved within the tested range (≥200 µg/mL). The MTT assay reflects the metabolic activity of biofilm-embedded cells, and reductions in metabolic activity do not necessarily correspond to complete biofilm eradication. Data are presented as means (n = 3)

Table 2. Minimum concentration of SLS preventing biofilm development against tested bacterial strains, expressed as Minimum Biofilm Inhibitory Concentration (MBIC)

	MBIC (µg/mL) of SLS
Enterococcus faecalis	> 200
Streptococcus mutans	> 200

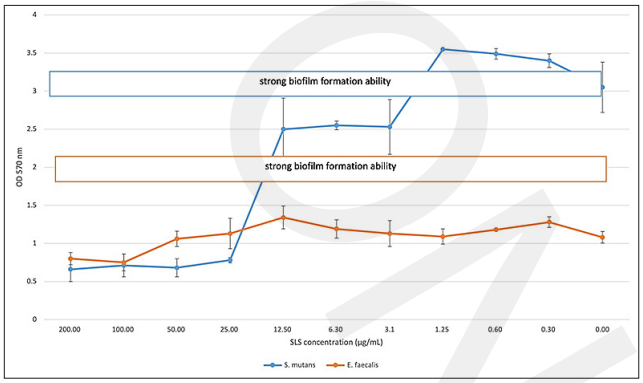

Figure 7. Comparison of the effectiveness of sodium lauryl sulfate (SLS) in reducing the biofilm of *Streptococcus mutans* and *Enterococcus faecalis* using crystal violet staining. Data are presented as means of 3 independent experiments ± SEM

Despite SLS treatment, the reduction in biomass was minimal across all concentrations. Based on OD classification, all tested concentrations of SLS failed to reduce biofilm biomass below the threshold for strong biofilm producers.

A cut-off OD (ODc) of 0.052 was established. All OD values for SLS-treated biofilms were significantly above this threshold (e.g., OD > 0.6 at 200 µg/mL), confirming strong biofilm formation despite SLS exposure (Fig. 7).

DISCUSSION

The presented findings on the toxicity of SLS add to the growing body of evidence highlighting the potential risks associated with this widely-used surfactant. SLS is commonly found in a variety of personal care products, including toothpaste, shampoos, and soaps, due to its effectiveness as a detergent, emulsifier, and foaming agent. Despite its widespread use, concerns about the safety of SLS have persisted, particularly regarding its effects on soft tissues and its potential to exacerbate existing health conditions.

The results of the current study provide valuable insights into the toxicological effects of SLS across different biological systems, highlighting both its cytotoxic and cardiotoxic properties, as well as its impact on embryonic development in zebrafish.

The cytotoxicity observed in human fibroblasts (BJ) with an LC_{50} of 38.46 µg/ml underscores the significant risk that SLS poses to human cells at relatively low concentrations. This aligns with previous studies suggesting that SLS can damage soft tissues, potentially leading to conditions such as gingival recession and periodontal disease [21]. The findings suggest that SLS can cause epithelial cell necrosis, which could have serious implications for oral health, especially with prolonged use of SLS-containing products.

The cardiotoxicity data, with an LC_{50} of 36.97 µg/ml in rat myoblasts (H9C2), reveal that SLS not only affects general cellular viability, but also has specific toxic effects on cardiac muscle cells. This finding is particularly concerning as it suggests that SLS, when absorbed systemically, could have detrimental effects on the heart, potentially leading to cardiotoxicity. While SLS is primarily used for its surfactant properties, its impact on cardiac cells highlights the need for further investigation into its long-term cardiovascular effects, especially in individuals who might be exposed to high levels of SLS over extended periods.

FET test revealed that SLS has a comparatively lower toxicity in zebrafish embryos, with an LC_{50} of 765 µg/ml. This suggests that the embryonic stages of zebrafish are less sensitive to SLS compared to mammalian cells. However, the delayed hatching observed at concentrations of 721 µg/ml and the morphological alterations at 288 and 721 µg/ml indicate that SLS still poses developmental risks. The lack of significant effects on heart rhythm and locomotor activity in zebrafish larvae suggests that the impact of SLS may vary across different biological processes, and that the developmental stage of the organism plays a critical role in its susceptibility to SLS toxicity.

The variation in sensitivity between human cells, rat myoblasts, and zebrafish embryos highlights the complex nature of SLS toxicity. While zebrafish embryos show lower sensitivity, the significant toxic effects observed in mammalian cells suggest that the impact of SLS might be more pronounced in higher organisms, especially in terms of cellular and cardiac health.

The obtained results highlight the need for caution in the use of SLS, particularly in everyday products that come into direct contact with soft tissues. Although SLS is an effective cleaning agent, its toxicity at both the cellular and organismal levels suggests the importance of seeking safer alternatives or developing strategies to minimize the risks associated with its use. Additionally, the presented study underscores the need for further research into the long-term effects of SLS on human health and ecosystems to fully understand the consequences of its widespread use.

Although SLS has been widely reported to possess antimicrobial and anti-biofilm properties due to its surfactant activity and ability to disrupt lipid membranes [22], the current findings demonstrate that this effect is strain-dependent and limited in scope. In the study, SLS exhibited a MIC of 50 µg/mL against *S. mutans*, indicating moderate inhibitory activity, whereas *E. faecalis* showed resistance up to 200 µg/mL, the highest tested concentration. These results suggest that while SLS may have some bacteriostatic potential against specific oral pathogens, its efficacy does not extend broadly across clinically relevant Gram-positive species.

The variability in MIC values may stem from fundamental differences in cell wall structure, membrane composition, and tolerance mechanisms between the 2 species. *E. faecalis*, for instance, is known for its robust stress response systems, intrinsic resistance to detergents, and high biofilm-forming capacity which, together, likely contribute to its relative insensitivity to SLS [13, 23]. In contrast, *S. mutans* may be more susceptible due to a thinner peptidoglycan layer and less efficient efflux systems.

Despite partial inhibition of planktonic growth in *S. mutans*, our biofilm assays revealed that SLS failed to significantly reduce metabolic activity (MTT assay) or total biomass (crystal violet staining) in biofilms of either strain. These observations indicate that the bacteriostatic effect of SLS does not translate into effective biofilm disruption, even at concentrations approaching cytotoxic levels for eukaryotic cells. This lack of antibiofilm efficacy is likely due to multiple protective features of mature biofilms, including the extracellular polymeric substance (EPS) matrix, reduced growth rates of embedded bacteria, and altered gene expression related to stress resistance [24]. SLS, while amphiphilic and capable of disrupting lipid bilayers in isolated cells, may be inactivated or sequestered by proteins

and polysaccharides within the biofilm, thereby limiting its penetration and interaction with target membranes. Notably, previous studies that reported strong antibiofilm effects of SLS or SDS often employed early-stage adhesion models, continuous-flow systems, or included synergistic compounds in formulations [22, 25]. The current findings, based on a static mono-species biofilm model in protein-rich media, provide a more stringent test of SLS efficacy under conditions that better reflect clinical biofilm environments.

Moreover, it is worth emphasizing that while the antimicrobial activity of SLS appears modest and strain-specific, its cytotoxic (LC_{50} = 38.46 µg/mL in BJ fibroblasts) and cardiotoxic (LC_{50} = 36.97 µg/mL in H9C2 myoblasts) effects are much more pronounced. This narrow therapeutic margin further limits its potential application as a stand-alone antimicrobial agent in formulations intended for mucosal or systemic exposure.

Taken together, the results obtained indicate that SLS exhibits selective antimicrobial activity against *S. mutans*, but lacks the potency to effectively target biofilm-embedded bacteria. These findings are clinically relevant given the persistence of *E. faecalis* in endodontic infections and the increasing demand for agents capable of biofilm eradication.

CONCLUSIONS

Despite the fact that SLS is effective as a surfactant, its use in consumer products should be carefully monitored and regulated due to its potential cytotoxic and cardiotoxic effects. The relatively low LC_{50} values determined for human and rat cells indicate that SLS can be harmful even at low concentrations, raising concerns about its safety in everyday products. Furthermore, the developmental toxicity observed in zebrafish embryos points to the potential environmental risks posed by SLS, especially in aquatic ecosystems.

From a microbiological perspective, SLS demonstrated limited antimicrobial and anti-biofilm activity, with only moderate inhibitory effects observed against *Streptococcus mutans* (MIC = 50 µg/mL) and no significant inhibition of *Enterococcus faecalis* at concentrations up to 200 µg/mL. Additionally, SLS failed to reduce biofilm biomass or metabolic activity in mature biofilms of either species. These results indicate that SLS, while capable of disrupting bacterial membranes under ideal conditions, is not effective as a stand-alone antimicrobial or anti-biofilm agent, particularly in protein-rich or biofilm-associated environments.

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