Plaque-Suspension pH following a Cariogenic Challenge on Silver Diamine Fluoride-Treated Enamel Blocks: An Exploratory *in Situ* Study

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Abstract
Background: Silver Diamine Fluoride (SDF) has garnered considerable interest because of its ability to arrest caries, along with its excellent antibacterial and anti-plaque activity. However, scientific literature evaluating the effect of SDF on plaque pH remains limited.
Aim: To evaluate the effect of 38% SDF on plaque pH after a cariogenic challenge in a pilot in situ model.
Methods and Materials: A pilot *in situ* study was conducted among 20 healthy volunteers aged 20-30 years. Participants wore mandibular Hawley's appliances embedded with enamel blocks (5 × 5 mm). Following overnight wear, baseline plaque samples were collected (T0). The enamel blocks were treated with 38% SDF extra-orally, re-inserted, and subsequently subjected to an intra-oral cariogenic challenge (fruit juice, pH ≈ 3.72) after 2 hours. Plaque samples were subsequently collected at 2 (T_1), 6 (T_2), 12 (T_3), and 24 hours (T4) to measure plaque suspension pH. Statistical analysis was performed using repeated-measures ANOVA with Bonferroni post-hoc tests ($\alpha = 0.05$).
Results: The mean plaque pH decreased from 7.466 ± 0.125 at baseline to 7.041 ± 0.156 at 24 hours, with a statistically significant overall reduction over time ($P < 0.001$). However, at all measured intervals, all mean values remained within the neutral range (pH > 7.0).
Conclusion: Within the limitations of this pilot study, even after a cariogenic exposure, SDF-treated enamel blocks maintained plaque pH within the neutral range. These findings suggest that SDF may influence plaque acidogenicity. Controlled split-mouth studies are required to validate these findings against the natural buffering capacity of saliva.
Keywords: Silver Diamine Fluoride, dental caries prevention, plaque pH, cariogenic challenge
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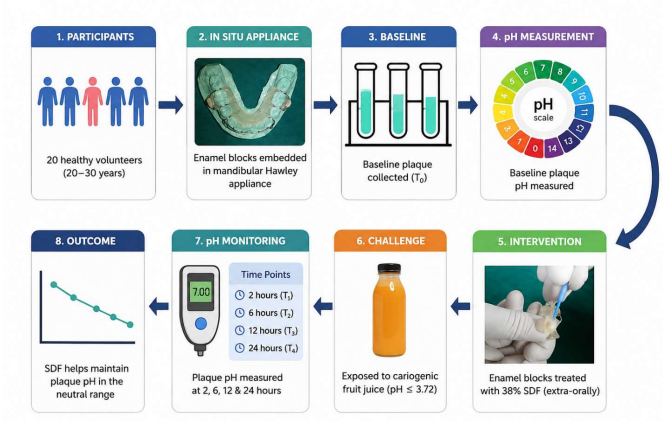

Graphical Abstract

1. Introduction

Untreated dental caries among children remains one of the most common challenges faced by pediatric dentists [1]. Untreated decay affects not only the child's oral health but also the child's overall physical and psychological well-being [2]. Therefore, it is essential to identify effective, low-cost treatment alternatives for children at high caries risk and for populations with limited access to dental care [3]. Recently, Silver Diamine Fluoride (SDF) has gained considerable attention due to its ability to arrest dental caries [4]. Clinical studies have demonstrated that topical application of 38% SDF is highly effective in arresting and preventing dental caries. SDF promotes remineralization of demineralized dentin and also exhibits antibacterial and possible antiplaque properties [5].

Stephen reported that the fall in plaque pH following sugar exposure is greater in caries-active individuals compared to caries-free individuals [6]. Studies have shown that dental caries is associated with increased proportions of acidogenic and aciduric bacteria that are capable of demineralizing enamel [7]. These microorganisms grow and metabolize in acidic pH and become more competitive under these conditions. In contrast, bacteria associated with enamel health are unable to thrive in acidic environments. There exists a homeostatic mechanism that maintains a balance between the host and the resident oral microflora. Disruption of this balance leads to plaque-mediated diseases such as dental caries [8]. The cariogenic potential of dental plaque largely depends on plaque pH, which reflects the interaction between the microbial communities associated with health and disease. If SDF possesses significant antiplaque properties, it may also influence plaque pH. However, limited literature exists evaluating the effect of SDF on plaque pH. Therefore, the present study was undertaken to evaluate the effect of SDF on plaque pH following a cariogenic challenge at different time intervals [9, 10].

Exploring the effectiveness of SDF in reducing the burden of untreated dental decay in pediatric cohorts presents compliance and standardization challenges. Consequently, involving adult volunteers in the study provides an excellent, highly standardized in situ biological model. The findings of this exploratory pilot study provide preliminary insights into the potential role of SDF in maintaining plaque homeostasis. However, the results cannot be directly generalized to children.

2. Materials and methods

A pilot in situ study was conducted in the Faculty of Dental Sciences, Ramaiah University of Applied Sciences. Ethical clearance was obtained from the Institutional Ethical Committee [EC-19/19-FDS]. Informed consent was obtained from all participants before the study commenced. Since this was an exploratory pilot study, an *in-situ* appliance model was used to monitor short-term plaque dynamics. Clinical trial registration was not done, as the study does not evaluate long-term or clinical patient outcomes. G*Power software (version 3.1) was used to estimate sample size. An effect size (f) of 0.40, alpha error of 5%, power of 80%, and five repeated measurements were assumed based on a previous study evaluating pH changes following application of 38% SDF [11]. Although the calculation indicated that at least 9 participants were needed, to improve precision and account for potential attrition, 20 participants were included in this study. Given the exploratory nature of the design, the study is presented as a pilot *in situ* investigation.

Inclusion and Exclusion Criteria

This study included healthy individuals aged 20-30 years with a stimulated salivary flow rate greater than 1 ml/min and an unstimulated salivary flow rate above 0.25 ml/min. Participants were excluded if they had undergone fluoride application within one week prior to the study, had a known allergy to silver, were medically compromised, or were on medications known to affect salivary flow.

2.1 Saliva Collection from the Participant

Volunteers were selected with appropriate salivary flow by estimating both stimulated and unstimulated salivary flow rates. To estimate unstimulated salivary flow rate [12], the subjects were instructed to sit upright with the head inclined forward. The saliva collected on the floor of the mouth was collected in a graduated test tube for 10 minutes, then expressed in milliliters per minute. In the estimation of stimulated salivary flow rate [13], subjects were asked to chew a flavored gum (Mentos® Pure Fresh Sugarfree Spearmint Gum, Perfetti Van Melle, India) for 2 minutes. The chewing gum was utilized exclusively during the baseline screening phase. The saliva was expectorated into a graduated test tube, and the volume was measured by subtracting the weight of the test tube from the total weight of the test tube and the saliva, expressed in milliliters per minute. Volunteers with normal flow as per **Table 1** were selected.

2.2 Sample Preparation of Enamel Sections

Freshly extracted permanent teeth were obtained from the Department of Oral and Maxillofacial Surgery, FDS, RUAS. The coronal portion was sectioned along the long axis into buccal and lingual segments using a high-speed diamond-tipped disc. The buccal and lingual segments were prepared as enamel slabs measuring 5×5 mm using a diamond disc. The enamel sections were then sterilized with 3% ethylene oxide for 16 hours in the Department of Central Sterile Supply, Ramaiah Memorial Hospital, Bengaluru.

2.3 Fabrication of Appliance

Maxillary and mandibular alginate impressions were made of the 20 volunteers. Casts were poured in dental stone, following which mandibular Hawley appliances with buccal extensions on both sides were fabricated. A 6×6 mm window was created in the buccal extensions on both the right and left sides of the appliance using acrylic trimming burs. The enamel blocks were incorporated into this window with cyanoacrylate adhesive [14]. The cyanoacrylate adhesive was precisely applied exclusively to the adhesive interface of the enamel block to ensure that the experimental dental surface remained entirely uncontaminated and open to natural biofilm coating.

2.4 Measurement of plaque pH

The participants were asked to use non-fluoridated toothpaste for 1 week prior to wearing the Hawley appliance. Once the Hawley appliances were delivered to the participants, they were instructed to wear them overnight before applying SDF. Following overnight wear of the appliance, the plaque samples were collected from the embedded enamel sections in a 0.5 mL Eppendorf tube, and the pH was recorded. This represented the baseline plaque pH prior to SDF application (T0).

2.5 Exposure of Enamel Sample Sections to 38% silver diamine fluoride

SDF applications for all the enamel sections were done outside the oral cavity, and the appliance was inserted back into the subject's oral cavity following the application. The SDF application was carried out by a single operator. Enamel sections were first dried with a gentle flow of compressed air. One drop of 38% silver diamine fluoride (Fagamin®, Tedequim S.R.L., Córdoba, Argentina) was dispensed on the dappen dish provided by the manufacturer. A microbrush was dipped into the dappen dish and gently dabbed against the side to remove excess solution before application. It was then applied directly onto the enamel sections on both sides of the appliance. The sections were dried with a gentle flow of compressed air for at least 1 minute, until the liquid became dry. Any excess liquid was removed with gauze or a cotton pellet. The appliance was inserted back into the oral cavity, and the site was isolated for 3 minutes with cotton rolls [15-17].

After SDF application, participants were instructed not to eat or drink anything for 1 hour. After 2 hours, the enamel sections were exposed to a cariogenic challenge. The subjects were given a sugar-containing fruit juice (Minute Maid® Mixed Fruit Juice, pH ≈ 3.72, containing 11 g/100 ml of simple carbohydrates, as detailed by the manufacturer) and were asked to gently swish 25 ml of the juice around the mouth for 5 minutes. Subsequently, a plaque sample was collected for pH measurement. This constituted a plaque sample 2 hours after the application of SDF and immediately after a cariogenic exposure (T1). Plaque samples were again collected at 6-hour intervals (T2), 12-hour intervals (T3), and 24-hour intervals (T4). Following the plaque collection, the samples were transported to the laboratory within 1 hour for sample preparation and pH measurement [18, 19].

2.6 Evaluation of Plaque pH

Dental plaque samples taken in 0.5 mL Eppendorf tubes were briefly centrifuged at 1000 rpm for 5 minutes. Samples were suspended in 0.5 mL distilled water and vortexed for 20 seconds at 2500 rpm. Plaque suspension was diluted in distilled water at a 1:10 weight-to-volume ratio in 15 mL Falcon tubes to obtain a homogeneous suspension for accurate pH measurement with the electrode [20, 21]. A pH meter (EuTech India) was calibrated with standard buffers of pH 4.0, 7.0, and 10.0. The electrode was immersed in plaque suspension, and pH readings were recorded.

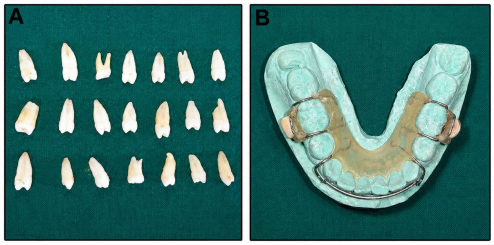

Figure 1: Preparation of the in situ appliance model: (A) Extracted permanent teeth selected for preparation of enamel blocks before sectioning; (B) Representative mandibular Hawley appliance fabricated with bilateral buccal acrylic extensions containing embedded enamel blocks

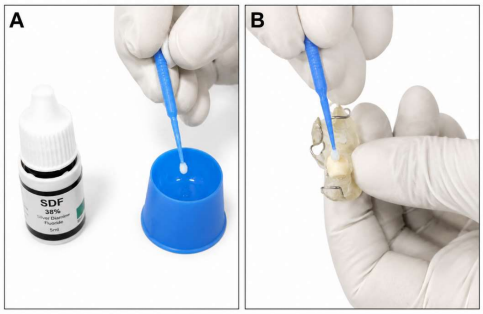

Figure 2: Figure 2. Extraoral application of 38% silver diamine fluoride (SDF) to the enamel blocks: (A) A disposable microbrush was used to collect the SDF solution from the dispensing dish/cup; (B) SDF was applied directly to the exposed enamel surface of the block mounted in the Hawley appliance before reinsertion of the appliance into the participant's oral cavity

2.7 Statistical Analysis

Statistical analysis was performed using SPSS version 23 (IBM Corp., Armonk, NY, USA). Shapiro-Wilk

test was used to assess the normality of plaque pH values at each time point. Plaque pH across five time intervals was compared with repeated-measures ANOVA. The assumption of sphericity was evaluated using Mauchly's test. Bonferroni-adjusted pairwise comparisons were performed for post hoc analysis. Effect size was reported using partial eta squared (η^2). Statistical significance was set at $P < 0.05$.

3. Results

The results of the present study showed a progressive reduction in mean plaque pH over time following SDF application. This reduction was statistically significant, as assessed by repeated measures ANOVA ($P < 0.001$). The mean plaque pH decreased from 7.466 ± 0.125 at baseline to 7.041 ± 0.156 at 24 hours (Table 2, Figure 3). The plaque pH remained within the neutral range across all measured time intervals, even after exposure to a cariogenic challenge, although it gradually declined. Pairwise comparisons using Bonferroni post hoc analysis revealed that the difference between baseline and 2 hours was not statistically significant ($P = 0.34$). However, the mean plaque pH at baseline was significantly higher than at 6 hours ($P = 0.002$), 12 hours ($P < 0.001$), and 24 hours ($P < 0.001$). The plaque pH at 2 hours was also significantly higher than at 12 hours ($P = 0.02$) and 24 hours ($P < 0.001$), whereas the difference between 2 and 6 hours was not statistically significant ($P = 0.06$). Additionally, the plaque pH at 6 hours was significantly higher than at 24 hours ($P = 0.003$). However, no statistically significant differences were observed between 6 and 12 hours ($P = 0.29$) and between 12 and 24 hours ($P = 1.00$). Shapiro–Wilk tests showed that plaque pH values at baseline ($P = 0.892$), 6 hours ($P = 0.738$), and 12 hours ($P = 0.434$) were normally distributed, whereas values at 2 hours ($P = 0.003$) and 24 hours ($P = 0.033$) were not. Mauchly's test indicated that the assumption of sphericity was not violated ($W = 0.421$, $\chi^2 = 15.064$, $df = 9$, $P = 0.091$); therefore, Greenhouse-Geisser correction was not required. Repeated-measures ANOVA showed a significant effect of time on plaque pH ($F(4,76) = 21.556$, $P < 0.001$), with a large effect size (partial $\eta^2 = 0.532$).

Table 1: Reference ranges for unstimulated and stimulated salivary flow rates (mL/min).

Flow rate	Unstimulated saliva	Stimulated saliva
Normal	>0.25	>1.0
Low	0.10–0.25	0.70–1.0
Very Low	<0.10	<0.70

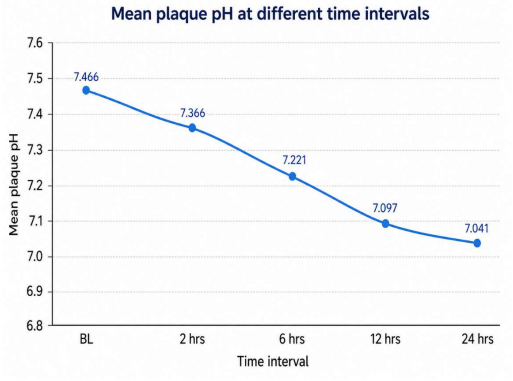

Figure 3: Mean plaque pH values at different time intervals

Table 2: Plaque-suspension pH at each assessment time and overall repeated-measures ANOVA

Time	N	Mean	SD	Min	Max	P-Value
Baseline	20	7.466	0.125	7.23	7.69	
2 hrs	20	7.366	0.101	7.04	7.47	
6 hrs	20	7.221	0.162	6.88	7.47	<0.001*
12 hrs	20	7.097	0.285	6.54	7.52	
24 hrs	20	7.041	0.156	6.68	7.26	

Overall time effect, $F(4, 76) = 21.556$, $P < 0.001$; partial $\eta^2 = 0.532$. Values are mean ± SD. Group means remained above 7.0, but individual minima were below 7.0 at 6, 12, and 24 hours.

Table 3: Multiple comparison of mean difference in pH levels between different time intervals using Bonferroni's post hoc Test

(I) Time	(J) Assessment Time	Mean Diff.	95% CI for the Differences		
	(J)Time	(I-J)	Lower	Upper	Adjusted P-Value
Baseline	2 h	0.101	-0.039	0.24	0.34
	6 h	0.245	0.077	0.413	0.002*
	12 h	0.369	0.151	0.587	<0.001*
	24 h	0.426	0.27	0.581	<0.001*
2 h	6 h	0.144	-0.001	0.29	0.06

	12 h	0.268	0.031	0.506	0.02*
	24 h	0.325	0.183	0.467	<0.001*
6 h	12 h	0.124	-0.043	0.291	0.29
	24 h	0.18	0.052	0.309	0.003*
12 h	24 h	0.056	-0.135	0.248	1

*Statistically significant after Bonferroni adjustment

4. Discussion

Dental caries is a multifactorial disease driven by a dysbiotic shift in the oral microbiome, in which the persistent fermentation of dietary carbohydrates by acidogenic and aciduric bacteria leads to local pH drops and subsequent enamel demineralization [2, 22, 23]. While early investigations focused on targeting isolated pathogens such as *Streptococcus mutans*, modern cariology recognizes that plaque pH ultimately reflects the collective metabolic output of the entire biofilm community [24, 25]. The microenvironment plays a major role in determining biofilm virulence. Plaque formed in a high cariogenic environment produces strong organic acids, whereas that formed in a low cariogenic environment produces alkali and can actively buffer acid challenges and maintain plaque homeostasis [7, 8]. Hence, plaque pH fluctuations following the use of a preventive agent reflect its metabolic-modulating efficacy.

Conventional topical fluoride agents, such as varnishes and gels, have been shown to have excellent potential for remineralization, while their ability to modulate biofilm acidogenicity remains limited [26, 27]. On the other hand, multiple studies have demonstrated the superior efficacy of 38% SDF in long-term arrest of caries [28, 29]. Results of the present study show that SDF maintains plaque pH above the critical threshold for demineralization, thereby maintaining homeostasis between the host and resident oral microflora. This demonstrates the strong influence of SDF on plaque pH kinetics and the prevention of ecological shifts towards cavitation.

In the current study, the pH of the plaque suspension was measured at various time points after SDF application and subsequent exposure to a cariogenic challenge. The measured pH values were maintained within the neutral range without falling to the critical acidic level. Commercially available 38% SDF is an ammoniacal silver fluoride solution that contains silver and fluoride ions within a stable diamine silver complex, thereby suppressing biofilm acidity through multiple pathways [30]. The silver ions (Ag^+) in SDF exhibit a high affinity for bacterial cellular components. They bind to bacterial cell walls and cytoplasmic membranes, inducing structural leakage and cell lysis. Upon entering the cell, silver ions also denature vital microbial enzymes and disrupt the metabolic pathways involved in glycolysis, thereby halting the fermentation of dietary sugars into organic acids [31, 32]. The fluoride ions (F^-) in SDF synergistically act with silver ions to inhibit the intracellular bacterial enzymes specifically enolase, which is a key enzyme in the glycolytic pathway [33, 34].

Furthermore, SDF impairs the synthesis of extracellular polysaccharides (EPS), which are the essential sticky matrix of dental plaque formed by oral streptococci that traps acids against the enamel surface [30, 35]. SDF enables caries arrest by suppressing acidogenic bacteria, particularly Streptococcus mutans and Lactobacillus species, which are vital for caries initiation and progression. Mei et al. reported that application of 38% SDF maintained higher pH values at the plaque-tooth interface by considerably reducing lactic acid production by S. mutans [31]. In a similar study, Knight et al. demonstrated that SDF reduced the fall in plaque pH following a sucrose exposure by suppressing bacterial metabolism [36]. In the present study, despite the statistically significant decline in plaque pH over 24 hours, the pH values remained within the neutral range. While evaluating the sustained neutral pH in the study, an important aspect to consider is the study sample included. The study included a cohort of caries-free participants, which can be a confounding factor influencing plaque pH. The biochemical quality of plaque and efficiency of the bicarbonate (HCO_3^-) buffering system are superior in caries-free individuals. These factors likely provide a defense against cariogenic challenges [37]. The bicarbonate buffering system of saliva effectively neutralizes plaque acids and clears acidic substrates formed from dietary carbohydrates [38, 39]. Plaque formed in a non-cariogenic environment contains commensal bacteria that form ammonia as a metabolic by-product, making the plaque alkaline [40, 41]. Hence, the neutrality of pH sustained across various time intervals of baseline, 2, 6, 12 and 24 hours may be attributed to various factors like plaque microenvironment, salivary buffering capacity and host defense working in concert with the inhibitory effect of SDF on acidogenic bacteria [37, 42].

An important aspect of the present findings is the distinction between statistical and clinical significance of the decline in plaque pH. The repeated-measures analysis demonstrated a statistically significant gradual decline in plaque pH from baseline (7.466) to 24 hours (7.041). The critical pH at which enamel demineralization is initiated is 5.5. The lowest pH reading recorded in the current study is 6.5. This makes the plaque microenvironment safe from structural dissolution. Hence, the gradual fall in pH

over 24 hours has no negative clinical impact.

Some methodological limitations should be considered while evaluating the results of this study. Firstly, as a pilot exploratory study, it lacked a control group, which limits causal inference. Controlled split-mouth trials should be conducted to isolate the effect of SDF from natural salivary buffering capacity. Since this study evaluates pH measurements over the duration of 2, 6, 12, and 24 hours following SDF application and a cariogenic challenge, a critical window of pH fall may have been missed. As demonstrated by Stephan's curve, following a sugar exposure, the plaque pH falls sharply within minutes, reaching the lowest point in 5-10 minutes and then gradually returns towards baseline in 30-60 minutes [43]. The present study evaluates the long-term effect of SDF on plaque pH; hence, the initial pH changes as per Stephan's curve were not recorded. As a result, the data reflect the long-term effect of SDF in maintaining plaque stability following a cariogenic challenge. Future studies should address this limitation by evaluating pH changes immediately after sugar exposure (at 5, 10, 20, 30, and 60 minutes) and over the long term. Also, the gradual decline in plaque pH, even though it remains within the neutral range, suggests that repeated application of SDF may be helpful in achieving long-term effects.

Another methodological consideration is the fluoride washout period used in the study. In standard plaque studies, in the case of prior fluoride applications, a 2-4-week washout period is considered ideal. As this was a pilot, in situ study, a 1-week salivary clearance period was utilized, especially to improve participant compliance [44, 45]. As the study uses external enamel blocks, this duration was considered sufficient for initial evaluation. Longer washout protocols are recommended for future trials. The present study employs an *in situ* model using a mandibular Hawley's appliance with embedded enamel blocks. This in situ model helps standardize the experimental parameters while allowing plaque accumulation under natural oral conditions. In comparison with pure in vitro plaque studies, in situ models have the advantage of enabling direct interaction of dental plaque with saliva, oral microflora, and fermentable carbohydrates within the oral environment. They also allow better control of experimental variables and eliminate the risk of discoloration of natural teeth compared to in vivo studies. Therefore, an *in situ* model serves as a reliable method for evaluating the effect of preventive agents such as SDF on plaque pH.

SDF was applied to the enamel blocks outside the oral cavity to prevent staining of hard and soft tissues and also to standardize the quantity and method of application. In extraoral application of SDF, many intraoral factors that may influence the clinical outcomes, such as salivary flow, plaque accumulation, and soft tissue contact, are not accounted for. Therefore, future studies should consider in vivo application protocols followed in clinical practice to improve data quality. In our study design, enamel blocks were secured into the acrylic window using cyanoacrylate adhesive. Cyanoacrylate adhesives polymerize to form a chemically inert, smooth barrier with less microbial retention. The surface energy and texture of cyanoacrylate are different from enamel and dentin; hence, its application was confined to the adhesive interface without extending to the exposed plaque-accumulating surface [46, 47]. This makes any potential influence of adhesive on plaque accumulation and bacterial adhesion negligible. Nevertheless, the difference in the substrate surface properties is acknowledged as a limitation, as it could theoretically affect biofilm behavior.

Even though the cariogenic challenge solution (Minute Maid® Mixed Fruit Juice, pH ≈ 3.72, 11 g/100 ml simple carbohydrates) used in the study is acidic at baseline, the plaque pH still remained within the neutral range in the 24-hour monitoring period. This suggests that the salivary buffering capacity, as well as the alkalizing effect of SDF, may have counteracted the direct acidogenic potential of the juice. Commercial fruit juices vary in composition. Sugar content and buffering capacity. This may influence plaque pH independent of bacterial acid production. Use of a 10% standardized sucrose solution should be considered in future studies to isolate bacterial acid production from substrate acidity.

A final methodological limitation of the present study is that the plaque suspension was diluted in distilled water prior to pH estimation. A standard dilution method according to the principle of plaque suspension in water introduced by Stephan (1940) and refined in later plaque studies was employed here [43]. Dilution ensures reproducibility and electrode stability and also eliminates interference from undiluted plaque mass. At the same time, dilution of plaque can potentially shift the actual pH to neutrality, which in turn underestimates the initial pH fall. To address this limitation, future studies should consider using micro-pH electrodes, which directly assess the pH from the plaque mass without dilution.

Many factors that influence caries susceptibility, such as salivary buffering capacity, plaque quantity, diet, and oral hygiene practices of the participants, were not standardized in the present study. Therefore, in vivo studies with larger sample sizes, including individuals with high caries susceptibility, should be conducted to validate the current findings of the study.

Conclusion

Within the limitations of this *in situ* pilot study, 38%

SDF- treated enamel blocks maintained plaque pH within the neutral range following cariogenic exposure. These findings suggest a possible role of SDF in regulating plaque acidogenicity. However, controlled split-mouth studies with standardized cariogenic challenge protocols, short-term pH monitoring, and larger sample sizes, including caries-active participants, are required to validate the findings.

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