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Oral Health Indices, Salivary pH, And Bacterial Load In Relation To Systolic Blood Pressure: A Comparative Cross-Sectional Study

Sreedevi Adikay[1*], Sirisha Chowdary G[1]

[1*]Institute of Pharmaceutical Technology, Sri Padmavati Mahila Visvavidyalayam,Tirupati, Andhra Pradesh, India.

[1]Institute of Pharmaceutical Technology, Sri Padmavati Mahila Visvavidyalayam, Tirupati, Andhra Pradesh, India

[*]Corresponding author: Sreedevi Adikay,

[*]Mailing address: Prof.A.Sreedevi, IPT, Sri Padmavati Mahila Visvavidyalayam, Tirupati, Andhra Pradesh, India ,

Email: sreedevi.professor@gmail.com.

ABSTRACT

Objective Oral health is closely linked to overall systemic health, yet this connection is often neglected in routine medical care and remains unfamiliar to many people.This study examined the association between oral health indicators, salivary pathogenic bacterial loads, and elevated systolic blood pressure (SBP) in healthy adults with no history of hypertension or other systemic diseases.

Methods In this comparative cross-sectional study, 60 adults aged 30–40 years were enrolled. Demographic and oral hygiene practice (OHP) data were collected using a validated questionnaire.Study participants were stratified by SBP into Group A (120–129 mmHg; n=30) and Group B (>129 mmHg; n=30). Assessments included oral health parameters such as halitosis (organoleptic, 0–5), salivary pH, plaque index (PI), gingival index (GI), and bleeding index (BI). Unstimulated saliva was cultured to quantify Streptococcus mutans(S. mutans) and Porphyromonas gingivalis (P. gingivalis) (colony-forming units per ml, CFU/ml). Independent t-test and Pearson correlation were used (two-sided α=0.05).

Results Groups were demographically comparable. As expected by design, SBP was higher in Group B (138.7±2.43 vs 127.5±1.87 mmHg; $p<0.0001$). Group B also showed lower salivary pH (6.79±0.14 vs 7.05±0.11) and with higher mean scores of halitosis (3.16±0.38 vs 2.12±0.28), PI (2.04±0.34 vs 1.10±0.28), GI (1.71±0.34 vs 0.57±0.22), and BI (1.00±0.25 vs 0.56±0.22); all $p<0.0001$. Salivary bacterial burden (S. mutans 87,000 vs 15,860 CFU/mL; $p<0.0001$ and P. gingivalis 19.34 vs 10.08 CFU/mL; $p<0.0001$) was higher in Group B. SBP correlated positively with oral-health indices (range r=0.62–0.90) and inversely with salivary pH (Group A: r=−0.77; Group B: r=−0.83). In Group B elevated SBP is associated with sub optimal oral health.

Conclusion These findings support considering oral health assessment in cardiovascular risk discussions and motivate longitudinal and interventional studies to test whether improving oral health favorably influences blood-pressure trajectories.

KEYWORDS: *Systolic blood pressure, Halitosis, Plaque index (PI), Gingival index (GI), Bleeding index (BI), Streptococcus mutans, Porphyromonas gingivalis.*

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1. INTRODUCTION

Poor status of oral health is one of the self neglected public health problems affecting especially among older and middle aged individuals[1]. Mechanical cleaning of teeth by using tooth brush, is a simple and very cost effective oral care practice for regular oral health maintenance[2]. By maintaining consistent and appropriate oral hygiene habits, dental plaque can be effectively eliminated, thereby helping to prevent periodontal diseases[3]. “Caries and periodontal conditions can affect

several systemic disorders in addition to their local effects on the dentition and tissues that support the teeth" [4].

Chronic oral infection and inflammation are thought to contribute to systemic vascular changes. For example, hypertensive patients have been found to harbor distinct oral microbiota profiles. one study noted a higher relative abundance of the periodontopathic bacterium *Porphyromonas gingivalis* alongside reduced overall microbial diversity in hypertensive individuals (accompanied by elevated circulating pro-inflammatory cytokines). One mechanistic pathway connecting oral microbes to blood pressure is the nitric oxide (NO) signaling axis. Commensal oral bacteria play a key role in the enterosalivary nitrate–nitrite–NO pathway that helps in regulating vascular tone [5]. In a healthy oral ecosystem, nitrate-reducing bacteria (such as *Neisseria* and *Rothia* species) convert dietary nitrate into nitrite, which is swallowed and eventually reduced to nitric oxide (NO), a potent vasodilator, in the blood circulation. Disruption of this pathway can lead to loss of NO bioavailability and higher blood pressure. Notably, the use of antiseptic mouthwash or broad-spectrum antibiotics, which can deplete oral nitrate-reducing flora, has been shown to cause an acute drop in salivary and plasma nitrite levels and a concomitant rise in SBP by approximately 2–3 mmHg [6,7]. Conversely, individuals with a healthier oral microbiome tend to generate more salivary nitrite and maintain better blood pressure. A case-control study reported that normotensive adults had three-fold higher salivary NO concentrations than age-matched hypertensives, and they also exhibited greater abundance of *Neisseria* (a beneficial nitrate-reducer) in their saliva [8].

In addition to loss of beneficial microbes, overgrowth of pathogenic oral bacteria may contribute to hypertension via systemic inflammation. *Porphyromonas gingivalis*, a keystone pathogen in periodontitis, is of particular interest. This anaerobe can invade periodontal tissues and enter the bloodstream through ulcerated gums or during routine activities (chewing, brushing), disseminating to distant sites. *P. gingivalis* DNA has been frequently detected within atherosclerotic plaques, and experiments in animal models confirm that chronic *P. gingivalis* infection accelerates atherogenesis [9]. Mechanistically, *P. gingivalis* appears to induce endothelial dysfunction. In endothelial cell studies, *P. gingivalis* exposure upregulates inducible nitric oxide synthase (iNOS) while downregulating endothelial NOS, perturbing the normal production of NO and promoting nitrosative stress [10]. *Streptococcus mutans*, classically known for its role in dental caries, has also emerged as a contributor to cardiovascular pathology. During episodes of transient bacteremia (for instance, during dental procedures or even vigorous brushing), *S. mutans* can adhere to endothelial surfaces, especially strains expressing collagen-binding adhesins. Collagen-binding *S. mutans* (e.g., Cnm-positive strains) have been shown to invade vascular endothelial cells and activate matrix metalloproteinase-9, weakening the endothelial basement membrane [11]. Consistently, *S. mutans* is frequently identified in excised heart valves and arterial plaque specimens. Importantly, the presence of the Cnm collagen-binding protein is a prerequisite for *S. mutans* to colonize blood vessels and instigate such cardiovascular damage [12]. These findings underscore that pathogenic oral bacteria (*P. gingivalis* and *S. mutans*) can instigate systemic inflammation, endothelial disruption, and ultimately contribute to the development of hypertension and cardiovascular disease. Against this backdrop, the present study aimed to evaluate the relationship between oral health and blood pressure in healthy adults. Specifically, we assessed a spectrum of oral health indicators,including clinical indices of plaque accumulation and gingival inflammation, salivary pH, halitosis scores, and salivary loads of *S. mutans* and *P. gingivalis* – in individuals with optimal vs. elevated (pre hypertensive) SBP.

2. MATERIALS AND METHODS

Study Design and Setting

We conducted an observational, comparative study during a community health camp organized by Sri Padmavathi School of Pharmacy, Tirupati, India. The report follows STROBE recommendations for observational studies.

Ethics

The protocol was approved by the Institutional Ethics Committee (SPSP/2024-2025/PH. D/01). All participants were informed about the study procedures and provided written informed consent before enrollment.

Participants

Adults aged 30–40 years attending the camp were approached (n = 140); 60 (42.9%) consented and were enrolled. Inclusion criteria: medically healthy at screening; no prior diagnosis of hypertension and any systemic diseases; ≥20 natural permanent teeth; body mass index (BMI) 20.0–30.0 kg/m²; and signed consent. Exclusion criteria: ongoing orthodontic treatment; antibiotic use within the prior 3 months; deleterious habits (smoking, alcohol, paan chewing, habitual mouth-breathing); and oral defects/diseases interfering with mastication.

Group Definitions and Sampling

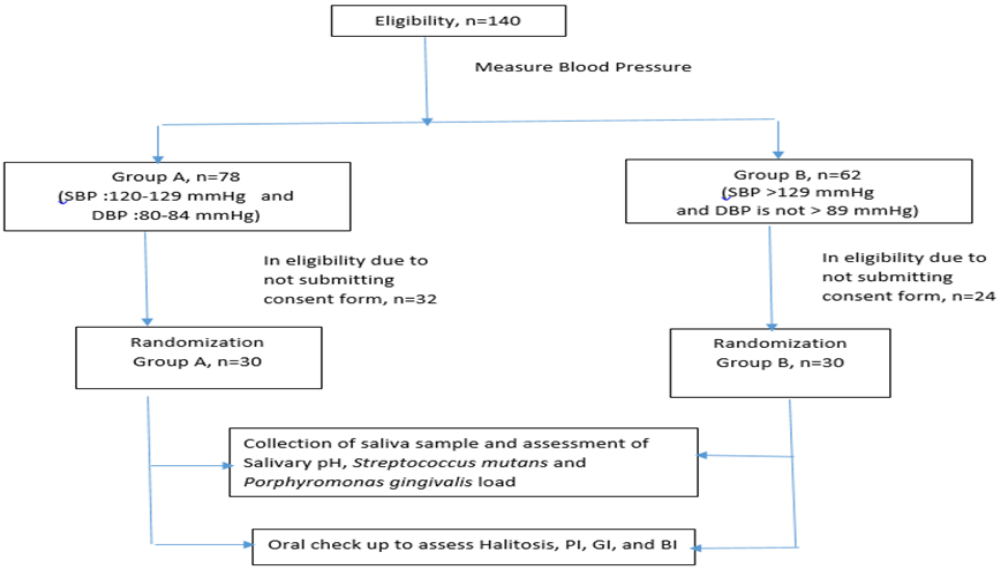

Figure 1: Study groups selection

A total of 140 healthy adults attending the health camp were screened against the study's inclusion and exclusion criteria. All potential participants received detailed information about the study procedures, informed consent, and the study questionnaire. Based on blood pressure thresholds, participants were classified into Group A (SBP 120–129 mmHg; DBP 80–84 mmHg; n=78) and Group B (SBP >129 mmHg; DBP ≤89 mmHg; n=62). Prior to random selection, exclusions were applied for non-submission of informed consent and for food intake within 1 hour before saliva collection, resulting in 32 and 24 exclusions from Groups A and B, respectively (Figure 1). From the remaining eligible pool, 30 participants were randomly selected from each group. Post hoc power analysis indicated >99% power at α=0.05, confirming that the final sample size was adequate to detect between-group differences.

Clinical Measurements

Body weight and height were measured using calibrated instruments, and BMI was calculated (kg/m²). Blood pressure was obtained with a validated automated oscillometric sphygmomanometer, following standard guidelines: participants were seated with back support and feet flat, an appropriately sized cuff was placed on the non-dominant arm at heart level, and three readings were taken after ≥5 minutes of rest; the mean SBP/DBP was used for group classification and analysis.

Saliva Collection and Handling

Unstimulated whole saliva was collected between 09:00 and 10:00 hours in the morning. Participants abstained from food and beverages for ≥1 hour prior to sampling. Approximately 5–10 ml of saliva was expectorated into sterile, leak-proof polypropylene containers, immediately stored in an insulated ice box (<4 °C), and transported without delay for same-day analysis at Pathgene Health Care Pvt. Ltd., Tirupati. Salivary pH was measured promptly using a calibrated digital pH meter (accuracy ±0.1).

Oral Health Assessments

Oral hygiene practices were captured using the WHO Oral Health Questionnaire for Adults. Halitosis was assessed by three trained examiners using an organoleptic method (Rosenberg scale, 0–5) after 2 minutes of mouth closure; participants exhaled through a 10-cm tube according to standardized procedures, and examiners—masked to group assignment—scored odor using a standardized guide [13,14]. Calibration sessions for halitosis scoring and clinical indices were conducted before data collection to standardize assessments. Dental plaque was disclosed with erythrosine and recorded using the Quigley–Hein Index as modified by Turesky, Gilmore, and Glickman [15]. Gingival inflammation and bleeding were assessed using the Löe–Silness Gingival Index [16] and the sulcus bleeding index, respectively [17,18].

Microbiological Procedures

Target organisms, Salivary *Streptococcus mutans* and *Porphyromonas gingivalis* were quantified as colony-forming units per milliliter (CFU/m).

Sample preparation and serial dilutions. Saliva was vortexed for homogenization. A 1:10 dilution in sterile double-distilled

water was prepared, followed by serial 10-fold dilutions. From appropriate dilutions, 100 µL aliquots were plated for enumeration.

S. mutans aliquots were spread onto mitis salivarius bacitracin (MSB) agar. Plates were incubated at 37 °C in 5% CO_2 for >48 hours. Colonies with morphology characteristic of *S. mutans* on MSB were counted on plates yielding 10–300 colonies and multiplied by the reciprocal dilution to derive CFU/ml [19].

For selective recovery of *P. gingivalis*, aliquots were streaked onto an enriched anaerobic blood agar (e.g: Trypticase soy agar supplemented with 5% defibrinated sheep blood, 5 µg/ml hemin, and 1 µg/ml vitamin K_1). Plates were incubated at 37 °C for 48–72 hours under anaerobic conditions (e.g., anaerobic jar with gas-generating packs; ~85% N_2, 10% H_2, 5% CO_2). Black pigmented colonies with typical morphology were counted on countable plates (10–300 colonies) and expressed as CFU/ml after applying the dilution factor [20]. Where confluent growth occurred, counts were taken from the nearest countable dilution. All culture procedures were performed on the day of collection to minimize pre-analytic variability.

Quantification rules

When multiple dilutions were countable, the plate within the 10–300 range closest to 100 colonies was used. If no plate fell in this range, counts from the nearest dilution were recorded with notation. Replicate plates were used as needed to confirm counts.

Blinding, Calibration, and Quality Assurance

Examiners conducting halitosis and clinical index scoring were masked to group status. Equipment (pH meter, weighing scale, stadiometer, sphygmomanometer) was maintained and calibrated per manufacturer recommendations. Microbiological assays were conducted by trained personnel using standardized SOPs, with same day processing of specimens.

Statistical Analysis

Analyses were performed in GraphPad Prism 8.0.1. Continuous variables were summarized as mean ± SD. Between-group comparisons used independent-samples t-tests; when normality or variance homogeneity assumptions were questionable, parametric, unpaired 2-tailed t-test, or non-parametric Mann-Whitney u test alternatives were considered. Pearson correlation coefficients quantified associations between SBP and oral health indicators with 95% confidence intervals; for ordinal or non-normally distributed variables, Spearman's rho was considered. Two-sided $p < 0.05$ was considered statistically significant.

3. RESULTS

Table 1: Baseline comparability characteristics of the patients

Characteristic	Group A mean (S.D)	Group B mean (S.D)	p-Value
Age (years)	35.47 (2.89)	35.20 (2.76)	0.72[a]
Body Mass Index (kg/m^2)	26.52 (1.56)	26.43 (1.49)	0.82[a]
Oral Hygiene Practice	2.10 (0.31)	2.03 (0.18)	0.61[b]
Systolic Blood Pressure (mmHg)	127.5 (1.87)	80.13 (0.78)	<0.0001 [a]
Diastolic Blood Pressure (mmHg)	138.7 (2.43)	80.97 (2.43)	0.0786 [a]

[a] Parametric, unpaired 2-tailed t-test, [b] Non-parametric Mann-Whitney u test

Groups did not differ by age (p=0.72), BMI (p=0.82), or oral-hygiene practice score (p=0.61). As per grouping, systolic blood pressure differed significantly between groups (p<0.0001), while diastolic blood pressure did not (p=0.0786) (Table 1).

Table 2: Comparison of core oral health indicators

Characteristic	Group A mean (S.D)	Group B mean (S.D)	p-Value
Halitosis	2.12 (0.28)	3.16 (0.38)	<0.0001[a]
Salivary pH	7.05 (0.11)	6.79 (0.14)	<0.0001[a]
PI [b]	1.10 (0.28)	2.04 (0.34)	<0.0001[a]
GI [c]	0.57 (0.22)	1.71 (0.34)	<0.0001[a]
BI [d]	0.56 (0.22)	1.00 (0.25)	<0.0001[a]

[a]Non-parametric Mann-Whitney u test, [b] Plaque Index, [c] Gingival Index, [d] Bleeding Index

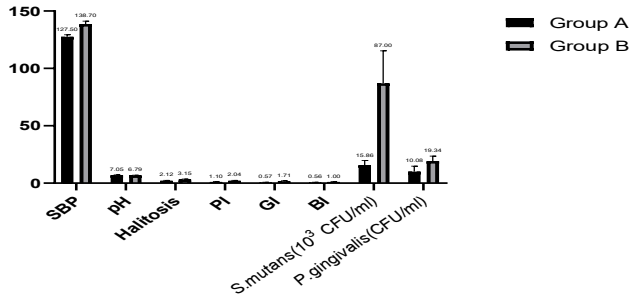

Fig.1: Inter group comparision of SBP and oral health parameters in Group A and B

Compared with Group A, Group B showed worse clinical indices and more malodor: higher halitosis (3.16 vs 2.12), higher plaque index (2.04 vs 1.10), higher gingival index (1.71 vs 0.57), higher bleeding index (1.00 vs 0.56), and lower salivary pH (6.79 vs 7.05); all p<0.0001 (Table 2,Fig.1).

Table 3: Salivary microbial load between groups

Microorganism	Group A Mean (S.D)	Group B Mean (S.D)	p-value
***S. mutans* (10³ CFU/ml)**	15.86 (3.9)	87.00 (2.82)	<0.0001[a]
***P. gingivalis* (CFU/ml)**	10.08 (4.72)	19.34 (4.19)	<0.0001[b]

[a] Non-parametric Mann-Whitney U test, [b] Parametric, unpaired-two-tailed-test

Group B had markedly greater bacterial burden: *S. mutans* 87.00×10³ vs 15.86×10³ CFU/ml (p<0.0001) and *P. gingivalis* 19.34 vs 10.08 CFU/ml (p<0.0001) (Table 3).

Table 4: Correlation between SBP and oral health indicators in group A

Characteristic	Pearson r	R-squared	p-Value
Halitosis	0.667	0.445	<0.0001
Salivary pH	-0.770	0.594	<0.0001
PI	0.768	0.591	<0.0001
GI	0.727	0.529	<0.0001
BI	0.731	0.535	<0.0001
S. mutans	0.802	0.643	<0.0001
P. gingivalis	0.624	0.390	<0.0001

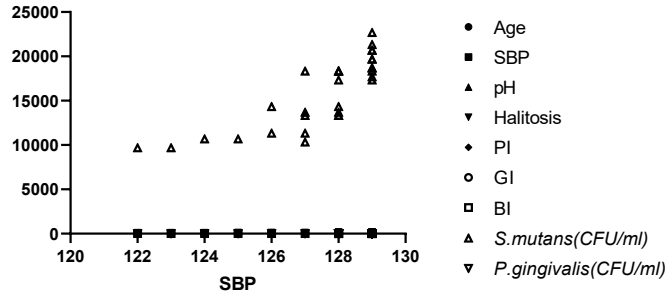

Fig. 2: Correlation of SBP with oral health parameters in group A

SBP correlated negatively with salivary pH (r=–0.770, p<0.0001) and positively with halitosis, PI, GI, BI (r=0.667–0.768; all p<0.0001). SBP also correlated with *S. mutans* (r=0.802) and *P. gingivalis* (r=0.624; both p<0.0001) (Table 4, Fig.2).

Patterns were directionally consistent and slightly stronger in places: SBP vs pH (r=–0.833, p<0.0001); positive correlations with halitosis, PI, GI, BI (r=0.703–0.815; all p<0.0001), *S. mutans* (r=0.741), and *P. gingivalis* (r=0.669; both p<0.0001) (Table 5, Fig.3).

Table 5: Correlation between SBP and oral health indicators in group B

Characteristic	Pearson r	R-squared	p-Value
Halitosis	0.745	0.555	<0.0001
Salivary pH	-0.833	0.694	<0.0001
PI	0.815	0.665	<0.0001
GI	0.703	0.495	<0.0001
BI	0.706	0.498	<0.0001
S. mutans	0.741	0.550	<0.0001
P. gingivalis	0.669	0.447	<0.0001

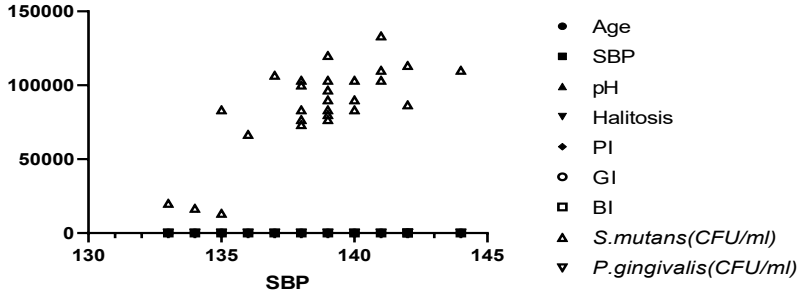

Fig. 3: Correlation of SBP with oral health parameters in group B

Table 6: Inter-correlations among oral parameters in group A

	Salivary pH	Halitosis	PI	GI	BI	*S. mutans*	*P. gingivalis*
pH	1	-0.7865	-0.9116	-0.8369	-0.8943	-0.8316	-0.7860
Halitosis	-0.7865	1	0.7576	0.6971	0.6482	0.6219	0.6893
PI	-0.9116	0.7576	1	0.7964	0.8355	0.7612	0.6849
GI	-0.8369	0.6971	0.7964	1	0.8008	0.7250	0.6931
BI	-0.8943	0.6482	0.8355	0.8008	1	0.7744	0.7642
S. mutans	-0.8316	0.6219	0.7612	0.7250	0.7744	1	0.8641
P. gingivalis	-0.7860	0.6893	0.6849	0.6931	0.7642	0.8641	1

Salivary pH showed strong inverse relationships with clinical indices and microbial loads in both groups (e.g., Group A: pH–PI r=–0.912; Group B: pH–*S. mutans* r=–0.970). Clinical indices co-varied positively, and *S. mutans* and *P. gingivalis* were strongly correlated (Group A r=0.864; Group B r=0.776). (Table 6 and Table 7).

Table 7: Inter-correlations among oral parameters in group B

	Salivary pH	Halitosis	PI	GI	BI	*S. mutans*	*P. gingivalis*
Salivary pH	1	-0.8608	-0.9117	-0.8292	-0.8372	-0.9696	-0.7614
Halitosis	-0.8608	1	0.7560	0.7496	0.7712	0.8124	0.6852
PI	-0.9117	0.7560	1	0.8115	0.7962	0.9016	0.6487
GI	-0.8292	0.7496	0.8115	1	0.9782	0.7958	0.5698
BI	-0.8372	0.7712	0.7962	0.9782	1	0.8131	0.6081
S. mutans	-0.9696	0.8124	0.9016	0.7958	0.8131	1	0.7764
P. gingivalis	-0.7614	0.6852	0.6487	0.5698	0.6081	0.7764	1

4. DISCUSSION

Consistent with previous reports, poor oral health showed a positive association with cardiovascular disease, suggesting that preventive oral care and education could reduce both dental burden and cardiovascular risk[21]. In Group B, poor oral-health indicators notably lower salivary pH, increased plaque, gingival inflammation, and higher microbial load were

strongly interrelated. All oral-health indices and microbial loads were significantly interrelated, indicating that increases in plaque and gingival inflammation coincide with higher bacterial burden and greater halitosis. The microbial burden appeared more pronounced in this group than in Group A, plausibly contributing to elevated systolic blood pressure (SBP).

Salivary *Streptococcus mutans* and *Porphyromonas gingivalis* represent dental caries and periodontitis markers, respectively[12,22]. *S.mutans* ability to initiate and sustain biofilm structures influences the overall microbial balance, creating a supportive environment that facilitates diverse bacterial activities [23]. *S.mutans*, a Gram-positive facultative anaerobe, initiates the caries process [24]. *P. gingivalis*, a Gram-negative anaerobe, triggers host inflammatory responses that affect the periodontium and is a key contributor to chronic periodontitis[25-27]. Nitric oxide (NO) plays a central role in hypertensive conditions, with normotensive individuals exhibiting higher NO levels; modification of the oral microbiota by decreasing salivary *S. mutans* and *P. gingivalis* may increase NO concentrations and be associated with blood pressure reduction [28]. Evidence from human and animal studies indicates that oral microbiota and NO influence cellular mechanisms—immune, hormonal, and metabolic, that can either protect against or promote hypertension [29].

Multiple studies link dental disease and poor oral hygiene, including periodontitis, to dysregulated blood pressure and hypertension. Poor oral health has been identified as a prevalent factor in hypertension, even among young adults [30]. Consistent with these observations, lower salivary loads of *S. mutans* and *P. gingivalis* were observed in Group A with normal SBP than in Group B with elevated SBP.

Stable salivary pH (approximately ≥7.0) is maintained by salivary buffering through electrolytes, bicarbonates, phosphates, and proteins [31]. A strong inverse association between salivary pH and halitosis was observed in both groups (Group A: –0.7865; Group B: –0.8608), in agreement with prior work showing that lower salivary pH correlates with higher organoleptic scores ($r = –0.35$, $p < 0.001$) [32]. A salivary pH below 7.0 denotes an acidic oral environment associated with increased risks of tooth decay, halitosis, and periodontal disease; sustained acidity may also contribute to broader systemic conditions [33]. Periodontal pathogens tend to thrive in slightly acidic environments.

In the study, Koppolu P et al,2022 strong and significant correlations between salivary pH and periodontal indices (PI, GI, and BI) have been reported [34]. In the present study, salivary pH was a strong inverse predictor of periodontal status across both groups: lower pH tracked with higher plaque accumulation, greater gingival inflammation, and increased bleeding tendency. The near identical strength of correlations particularly for PI ($r \approx –0.91$) indicates that acidic oral environments consistently foster plaque formation and periodontal breakdown. In Group B, these relationships may be amplified by systemic factors linked to elevated SBP, suggesting an oral–systemic interaction in which poor control of oral acidity exacerbates periodontal disease and may contribute to higher blood pressure.

Salivary pH correlates negatively with *S. mutans* load, consistent with the organism's acidophilic ecology [35]. The strong inverse relationship between pH and *S. mutans* across both groups indicates that acidity is a key ecological determinant for this cariogenic bacterium; the more pronounced association in Group B suggests a greater propensity toward oral dysbiosis in individuals with poorly regulated blood pressure, potentially reflecting shared inflammatory or dietary risks. Maintaining a neutral-to-alkaline oral environment may suppress pathogenic bacterial growth and support oral and systemic health.

Only a few studies have explored the association between periodontitis related bacteria and cardiovascular diseases in animal models; however, one study found that systemic exposure to *P. gingivalis* markedly accelerated atherogenic plaque formation in mice [36]. *P. gingivalis* has been reported to trigger platelet aggregation and foam cell formation, key events in the progression of atheromatous plaque [37].The strong inverse correlations between salivary pH and *P. gingivalis* in both groups support the view that acidic conditions favour the proliferation of this periodontal pathogen. Given the role of *P. gingivalis* in periodontitis progression and potential systemic inflammatory effects, maintaining oral pH balance emerges as a pragmatic preventive strategy applicable to normotensive and hypertensive individuals.

These findings indicate that salivary pH is a critical ecological regulator of oral microbial balance. Lower pH facilitates the proliferation of both *S. mutans* and *P. gingivalis*, organisms linked to the onset of dental caries and periodontal disease. The stronger correlation with *P. gingivalis* in Group B indicates a more hostile oral environment in which bacterial colonisation and host inflammation may be amplified.

Oral nitrate-reducing bacteria may inhibit proliferation of acidogenic *S. mutans*, offering protection against dental caries; predominant anaerobic nitrate-reducers on the human tongue include *Veillonella dispar* and *Veillonella atypica.* Modifications in the oral microbiome may increase NO salivary concentration, which is associated with a decrease in blood pressure [38]. The elevated *S. mutans* load observed here may coincide with reduced nitrate-reducing taxa; further investigations are warranted to quantify these communities in individuals with uncontrolled hypertension. Established associations between periodontal disease and systemic conditions such as hypertension suggest that maintaining a neutral-

to-alkaline salivary pH may benefit oral health and cardiovascular risk profiles. These results underscore the interconnectedness of oral microbial ecology, salivary chemistry, and blood pressure regulation.

5. CONCLUSION

The study found significant associations between poor oral health indicators and elevated systolic blood pressure (SBP). Lower salivary pH and higher salivary loads of *Streptococcus mutans* and *Porphyromonas gingivalis* co-occurred with greater plaque accumulation, gingival inflammation, and halitosis, particularly among individuals with elevated SBP. These patterns are consistent with oral microbial dysbiosis in an acidic milieu and suggest a potential contributory role in hypertension pathophysiology. The results suggest that routine oral health screening may serve as an adjunctive tool for cardiovascular risk assessment, and integrating evidence based oral hygiene practices targeting oral microbial balance and pH regulation could contribute to hypertension management. Further prospective and mechanistic studies evaluating oral microbial balance in hypertensive populations are recommended and also needed to explore the specific effects of *S.mutans* and *P.gingivalis* on nitrate-reducing bacteria, considering the intricate microbial interactions within the oral cavity.

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CONFLICT OF INTEREST

The authors do not have any conflict of interest.

ETHICS STATEMENT

This research involved human participants with the ethical permission of Sri Padmavathi School of Pharmacy Institutional Ethical Committee ,bearing the proposal number SPSP/2024-2025/PH.D/01.

INFORMED CONSENT STATEMENT

This study involved human participants, so each study participant received complete information regarding the study and voluntarily agreed and participated in the study by signing an informed consent form.

AUTHOR CONTRIBUTIONS

Sreedevi A: Study supervision,Project Admininstration,Funding Acquisition,Resources
Sirisha Chowdary G: Data Collection, Analysis,Write up.

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