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Environmental Noise Exposure, Salivary Cortisol and Dental Caries: An Analytical Cross-Sectional Study

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Abstract **Background:** Environmental noise is an ever-present urban pollutant that stimulates the hypothalamic-pituitary-adrenal axis and its downstream associations with dental caries in adults are not well understood. In this study, the relationship between residential noise exposure and dental caries was investigated and the role of salivary cortisol as the mediator was explored. **Methods:** This is an analytical cross-sectional study that included the evaluation of 24 h equivalent residential noise level (LAeq), morning salivary cortisol and caries experience (DMFT index) levels in a sample of 250 adults living in the community. Correlation, Analysis of variance, Chi-square, multivariable linear regression and bootstrapped mediation analysis were used to test associations. **Results:** There was a significant increase of salivary cortisol and DMFT when comparing the low, moderate and high noise groups ($p<0.001$ for both). Noise correlated with cortisol ($r = 0.58$) and DMFT ($r = 0.32$) and cortisol with DMFT ($r = 0.40$). Noise ($B = 0.08$, $p = 0.005$) and cortisol ($B = 0.39$, $p<0.001$) were independently associated with DMFT and approximately 43% of the noise association with caries was mediated by cortisol (bootstrap 95% CI 0.030-0.102). **Conclusions:** In this cross-sectional sample, higher recorded residential noise exposure and morning salivary cortisol were associated with higher cumulative Dental Caries Experience (DMFT). The indirect-path model is exploratory; temporal ordering, mediation and a causal environmental-tooth-decay pathway cannot be established.

Key Words Environmental Noise, Residential Noise Exposure, Salivary Cortisol, Dental Caries, DMFT Index, Physiological Stress, Oral Health

INTRODUCTION

Dental caries is one of the most common Non-Communicable Diseases (NCDs) that affects billions of people globally throughout the life course and is a significant and inequitably distributed burden on dental health services [1,2]. The most recent estimates of the prevalence of caries in permanent and primary teeth are around half of all children and a similar proportion of adults worldwide [3,4], with untreated caries in permanent teeth being one of the most prevalent health conditions in the world. Caries is a multifactorial, biofilm-mediated disease that occurs from the interaction between different factors over time: sugars in the diet, cariogenic microbiota, host and salivary factors and behaviour and socio-environmental determinants [4,5]. The DMFT index is the most commonly used epidemiological indicator of cumulative caries experience [6].

In addition to the classic three pillars of diet, microbiota and host susceptibility there is increasing awareness that psychosocial and environmental stressors influence the oral-health outcome [7]. Environmental noise is an omnipresent urban pollutant and its non-auditory health impacts on annoyance, sleep disturbance, hypertension, cardiovascular disease and others are now well established [8-11]. One of the main biological mechanisms involved in these effects is the chronic activation of the Hypothalamic-Pituitary-Adrenal (HPA) axis, a neuroendocrine system responsible for the physiological stress response and which ultimately releases cortisol [12]. Residential and occupational noise exposure, especially from aircraft and traffic noise, has been repeatedly correlated with changes in cortisol secretion, offering a possible mechanistic link between exposure to noise in the environment and the effects on systemic health [13,14].

Salivary cortisol is a validated, non-invasive measure that reflects free biologically active cortisol and is commonly used as a marker of the activity of the HPA-axis in population studies [15]. The biology of elevated cortisol is well-suited to affect caries risk: it can inhibit salivary flow and composition, decrease salivary antimicrobial components such as secretory immunoglobulin A and thus compromise the host defence mechanisms that normally inhibit the growth of cariogenic biofilm [16,17]. This is in line with this, several clinical studies have reported positive links between salivary cortisol and caries experience, mostly conducted in children [18-22], but with limited and variable evidence base and mostly in paediatric or dental-anxiety settings.

Even with this intersection of the evidence, there are two missing points. First, the possible influence of environmental noise has not been studied as an upstream, modifiable factor of dental caries. Secondly, the effect of cortisol on the link between external environmental exposure and clinical oral health outcome was not quantitatively measured in adults. We therefore performed an analytical cross-sectional study to assess the relationship between environmental noise exposure and dental caries experience in community-dwelling adults and to test the hypothesis that salivary cortisol is a mediator in the relationship between noise and dental caries. We hypothesized that increased noise exposure would be correlated with higher salivary cortisol levels and higher DMFT; and that a proportion of the overall effect of noise on caries was measurable, explaining these increases.

METHODS

Study Design and Setting

It is a cross-sectional study of analytical type that was carried out among the community-dwelling adults living in the area of Jaipur. The participants included children from urban, suburban and rural residential areas which were chosen to represent a wide range of noise exposure in the environment. Participants were adults (18-65 years old) who had lived in their current home for 6 months or more. Systemic disease and medication known to alter cortisol secretion or salivary function, pregnant women and those who had recently undergone oral surgery were excluded.

Sample and Variables

Enrolment was 250 people. Sociodemographic data (age, sex, residential area, socioeconomic and educational status), behavioural covariates (tooth brushing frequency, dietary sugars consumption and smoking status) and anthropometry (body mass index, BMI) were collected from all participants using a structured interview and examination form, with time since last dental visit recorded on the same form.

Noise Exposure Assessment

The 24 h equivalent continuous A-weighted sound pressure level (LAeq) measured in dB (decibels), distance to the main noise source and number of years of living at the current noise source were used to characterize residential environmental noise. Participants were assigned to one of three ordinal categories of exposure based on guideline thresholds [9] as low exposure (exposure<55 dB), moderate exposure (55 dB - 70 dB) and high exposure (>70 dB).

Salivary Cortisol Collection and Assay

Since there is an intrinsic diurnal rhythm of cortisol, unstimulated whole saliva was collected at the same morning time window (07:30-09:00) and each participant's window of saliva collection was documented [15,23]. Participants were directed not to eat or drink for 30 minutes prior to sampling and not to brush their teeth for 10 minutes prior to sampling. Salivary cortisol concentration was measured by immunoassay (nmol/L) and was used as the potential mediator between noise exposure and caries [15].

Dental Caries Assessment

Caries experience was evaluated by the DMFT index using calibrated examiners following WHO criteria, which counts Decayed (D), Missing (M) and Filled (F) teeth to obtain the DMFT score [6]. Any activity decay (D >0) was also noted as a binary variable (caries present/absent).

Statistical Analysis

Measurement Clarification: DMFT indexes lifetime cumulative caries experience rather than active caries incidence. Recorded residential LAeq categories should not be assumed to represent continuous, individualized 24 h exposure without documented meter specifications, calibration, placement and monitoring schedule. A single morning cortisol value can vary with waking time, caffeine, medication, smoking, sleep and acute stress; unmeasured factors may influence the observed relationships.

The continuous variables are summarized as mean±Standard Deviation (SD) and the categorical variables are summarized as frequencies and percentages. Pearson and Spearman correlation coefficients were used to assess the association between two continuous variables. One Way Analysis of Variance (ANOVA) was used to test differences in salivary cortisol and DMFT between noise-exposure categories with the Kruskal-Wallis test as a confirmation test and to compare the prevalence of caries between noise-exposure categories, the chi-square test was used. The noise level, salivary cortisol, age, sex, smoking, dietary sugar consumption, brushing frequency, BMI and noise duration were included as independent variables in a multivariable linear regression model that had DMFT as the dependent variable. The mediation analysis of the noise → cortisol → DMFT pathway was performed to explore the noise effect on DMFT through the mediation of cortisol and to obtain bias-corrected 95% bootstrap confidence interval (as labelled in the reported results table; original output requires confirmation) (5,000 resamples) to test the indirect effect. Statistically significant difference was defined as a two-sided p-value<0.05. Data was analysed using Python (pandas, SciPy and statsmodels).

RESUTLS

Characteristics of the Study Population

The 250 participants had a mean age of 37.2±11.1 years (range 18-65); 132 (52.8%) were female. Residential settings were urban (39.6%), suburban (36.4%) and rural (24.0%).

Table 1: Sociodemographic, Behavioural, Exposure and Clinical Characteristics of the Study Participants (n = 250)

Characteristic	Value
Age, years (mean ± SD)	37.2±11.1
Sex-Female, n (%)	132 (52.8)
Sex- Male, n (%)	118 (47.2)
Residential area - Urban/Suburban/Rural, n (%)	99 (39.6)/91 (36.4)/60 (24.0)
Socioeconomic status - Low/Middle/High, n (%)	76 (30.4)/110 (44.0)/64 (25.6)
Education - Primary/Secondary/Tertiary, n (%)	49 (19.6)/105 (42.0)/96 (38.4)
Proximity to noise source - <50 m/50-200 m/>200 m, n (%)	69 (27.6)/93 (37.2)/88 (35.2)
Noise exposure, dB (mean ± SD)	59.4±10.1
Noise category - Low/Moderate/High, n (%)	92 (36.8)/111 (44.4)/47 (18.8)
Noise duration, years (mean ± SD)	6.0±4.4
Salivary cortisol, nmol/L (mean ± SD)	7.11±2.98
Brushing frequency, per day (mean ± SD)	1.47±1.11
Sugar intake, servings/day (mean ± SD)	3.08±1.29
Current smoker, n (%)	64 (25.6)
BMI, kg/m² (mean ± SD)	24.7±4.0
Last dental visit, months ago (mean ± SD)	15.7±10.4
Decayed teeth (D) (mean ± SD)	2.98±2.74
Missing teeth (M) (mean ± SD)	1.56±1.47
Filled teeth (F) (mean ± SD)	2.09±1.76
DMFT index (mean ± SD)	6.63±4.57
Caries present (D > 0), n (%)	214 (85.6)

SD, Standard Deviation; BMI, Body Mass Index; DMFT, Decayed, Missing and Filled Teeth

Table 2: Salivary Cortisol, Caries Experience and Caries Prevalence across Environmental Noise-Exposure Categories

Outcome	Low (<55 dB) (n = 92)	Moderate (55-70 dB) (n = 111)	High (>70 dB) (n = 47)	p-value
Salivary cortisol, nmol/L (mean ± SD)	5.29±2.45	7.50±2.56	9.74±2.56	<0.001
DMFT index (mean ± SD)	5.32±3.62	6.43±4.31	9.66±5.44	<0.001
Decayed teeth (D) (mean ± SD)	2.17±2.19	2.99±2.60	4.53±3.34	<0.001
Caries present, n (%)	73 (79.3)	97 (87.4)	44 (93.6)	0.059

P-values from one-way ANOVA (cortisol, DMFT), Kruskal-Wallis test (decayed teeth) and chi-square test (caries prevalence). SD, Standard Deviation; DMFT, Decayed, Missing and Filled Teeth

Table 3: Pearson Correlation Coefficients (r) among Noise Exposure, Salivary Cortisol, Caries Indices and Selected Covariates

Variable pair	Noise (dB)	Cortisol	DMFT	Decayed (D)
Noise exposure (dB)	1.00	0.58*	0.32*	0.33*
Salivary cortisol	0.58*	1.00	0.40*	0.40*
DMFT index	0.32*	0.40*	1.00	-
Age	-	-	0.33*	-
Sugar intake	-	-	0.12	-
Noise duration	-	-	0.12	-

*p<0.001. DMFT, Decayed, Missing and Filled Teeth. Dashes indicate correlations not central to the analysis

The noise level of the residents' homes was 59.4±10.1 dB and ranged from low, moderate and high noise exposure groups (36.8, 44.4 and 18.8%, respectively). The means of morning salivary cortisol and DMFT were 7.11±2.98 nmol/L and 6.63±4.57, respectively and 214 (85.6%) had active decay (caries). The sociodemographic, behavioural, exposure and clinical features are shown in Table 1.

Cortisol, Caries and Noise-Exposure Category

Caries experience and salivary cortisol both showed a monotonic increase as a function of the noise-exposure gradient. Mean salivary cortisol rose from 5.29±2.45 nmol/L in the low-noise group to 7.50±2.56 nmol/L in the moderate group and 9.74±2.56 nmol/L in the high group (ANOVA F = 50.9, p<0.001; Kruskal-Wallis p<0.001). Correspondingly, mean DMFT increased from 5.32±3.62 to 6.43±4.31 to 9.66±5.44 across the same categories (F = 16.0, p<0.001), as did the count of decayed teeth (p<0.001). The prevalence of caries increased from low to moderate, to high exposure (79.3% to 87.4% to 93.6%) but was not conventional statistically significant (χ^2 = 5.66, p = 0.059). The stratified results are presented in a tabular format (Table 2).

Correlations among Noise, Cortisol and Caries

The salivary cortisol was highly and positively associated with residential noise (r = 0.58, p<0.001) and moderately with DMFT (r = 0.32, p<0.001). Salivary cortisol, in turn, was correlated with DMFT (r = 0.40, p<0.001) and with the number of decayed teeth (r = 0.40, p<0.001). Agem was also significantly correlated with the DMFT in positive direction (r = 0.33; p<0.001), while dietary sugar intake and noise duration had only weak non-significant correlations with DMFT. The complete correlation matrix is given in Table 3.

Multivariable Predictors of Caries Experience

Both the residential noise level (as a positive predictor) (B = 0.08 per dB, 95% CI 0.02-0.14, p = 0.005) and the salivary cortisol (as a positive predictor) (B = 0.39 per nmol/L, 95% CI 0.20-0.59, p<0.001) remained independent positive predictors for DMFT after adjustment for demographic and behavioural covariates in the multivariable linear regression model (R^2 = 0.41; adjusted R^2 = 0.39).

Table 4: Multivariable Linear Regression Model for DMFT Index (R^2 = 0.41; adjusted R^2 = 0.39; N = 250)

Predictor	B	95% CI	p-value
Noise exposure (per dB)	0.082	0.024 to 0.139	0.005
Salivary cortisol (per nmol/L)	0.393	0.196 to 0.590	<0.001
Age (per year)	0.164	0.121 to 0.208	<0.001
Female sex	0.257	-0.654 to 1.169	0.579
Current smoking	1.616	0.559 to 2.673	0.003
Sugar intake (per serving/day)	0.627	0.274 to 0.981	0.001
Brushing frequency (per time/day)	-1.202	-1.618 to -0.786	<0.001
BMI (per kg/m²)	0.114	-0.0002 to 0.228	0.050
Noise duration (per year)	-0.078	-0.191 to 0.035	0.176

B, unstandardised regression coefficient; CI, Confidence Interval; BMI, Body Mass Index; DMFT, Decayed, Missing and Filled Teeth

Table 5: Mediation Analysis of the Environmental Noise → Salivary Cortisol → DMFT Pathway

Effect	Estimate	95% CI (bootstrap)
Path a (noise → cortisol)	0.177	-
Path b (cortisol → DMFT | noise)	0.363	-
Total effect (c)	0.148	-
Direct effect (c′)	0.083	-
Indirect effect (a × b)	0.064	0.030 to 0.102
Proportion mediated	≈ 43%	-

Estimates are unstandardised coefficients from the mediation decomposition; the indirect effect was tested with 5,000 bias-corrected bootstrap resamples. CI, Confidence Interval; DMFT, Decayed, Missing and Filled Teeth

Older age (B = 0.16, p<0.001), current smoking (B = 1.62, p = 0.003) and higher dietary sugar intake (B = 0.63, p = 0.001) were each associated with greater caries experience, whereas more frequent tooth-brushing was protective (B = -1.20, p<0.001). Sex, BMI and noise duration were not significant independent predictors. The complete model is shown in Table 4.

Mediation of the Noise-Caries Association by Cortisol

The mediation analysis showed that noise influenced caries experience through the mediation of salivary cortisol. The increase in cortisol per 1-dB increase in noise was 0.177-nmol/L (path a) and the increase in DMFT per 1-nmol/L increase in cortisol was 0.363-unit (path b) after adjustment. The total effect of noise on DMFT was decomposed into direct effect (c = 0.148) and indirect, cortisol-mediated effect (a × b = 0.064; bootstrap 95% CI 0.030-0.102), which equates to about 43% of the total effect being mediated by cortisol. The mediation was statistically significant since the indirect effect had no zero in the bootstrap CI, meaning there was partial mediation. The decomposition is summarised in Table 5.

DISCUSSION

This analytic cross-sectional study with community-dwelling adults indicated that residential environmental noise was related to greater experience of dental caries, which partially mediated by increased salivary cortisol level. Cortisol and DMFT were both linearly related with low, moderate and high noise (Table 2); noise was an independent predictor of DMFT after multivariable adjustment (Table 4); and noise mediated about 43% of the total effect of noise on DMFT (Table 5). These results suggest a stress-mediated environment pathway where the acoustic environment, by inducing a neuroendocrine activation, is involved in caries risk.

Our findings of a significant positive relationship between noise exposure and salivary cortisol is in line with a considerable amount of environmental-health research that has been conducted on the relationship between residential and occupational noise with altered HPA-axis activity and cortisol production [13,14]. Noise is a non-specific stressor that repeated exposure to it induces increased levels of circulating and salivary cortisol [8,10,12]. Salivary cortisol is well validated as a non-invasive measure of this response [15] and sampling time was standardised in the morning to minimise the confounding of this diurnal cortisol rhythm [23].

The association between cortisol and caries we observed is consistent with previous clinical investigations, which were mostly performed in children with increased salivary cortisol in those with higher caries experience [18-22]. The present study is an extension of these studies to adults and importantly adds the stress hormone cortisol to a causal chain that starts with a modifiable environmental exposure. There is biological plausibility for a link between cortisol and caries; acute increases in cortisol can result in salivary gland atrophy and decreased salivary flow, change the composition of saliva and inhibit salivary immunoglobulin A and other antimicrobial components of saliva, which compromises host defense mechanisms to limit the growth of cariogenic biofilm [16,17].

The independent behavioural predictors we identified in our adjusted model are consistent with caries epidemiology already known and provide internal validity to our analysis. The more often one brushed their teeth, the less likely they were to experience more caries, while higher intake of dietary sugar, current smoking and age were all associated with higher caries experience [4,24]. The observed socioeconomic status and education gradients are also congruent with the finding that lower parental education levels and socioeconomic disadvantage are associated with increased risk of caries and impaired cortisol regulation [25,26]. There could be further interactions between noise, sleep and stress: environmental noise disrupts sleep, sleep is reciprocally related to stress and short or disturbed sleep has been correlated with caries [27,28].

Dental anxiety is another stress-related factor that contributes to poor oral-health outcomes [29] and recent cross-sectional studies have reaffirmed the association between psychosocial and mental-health status and oral health [7].

The results of this work have a practical consequence. If environmental noise is a measurable neuroendocrine risk factor for caries, then interventions to reduce noise and stress may serve to complement traditional caries prevention, especially for populations exposed to high levels of environmental noise, such as those in high-density urban areas. While this is clinically attractive, saliva, in general, is of great promise as a diagnostic medium, as salivary biomarkers are explored for caries risk assessment and monitoring [5] and analytical advances, such as discovery and validation of biomarkers with the aid of artificial intelligence, could improve their performance and translation [30]. In these environments, digital and educational tools may aid in community oral health surveillance and preventive engagement-such as the use of augmented- and virtual-reality applications and technology-enabled screening-may support this function [31,32]. Stress-reduction methods within the dental context may help alter stress pathways and fluoride therapies are still central to caries control [33]; while novel methods of regenerative approaches offer hope for future directions in managing the sequelae of caries [35].

There are limitations to this study. As a cross-sectional design, it does not permit causal inference and the mediation model is directionally motivated by a biological plausibility argument; the temporal ordering of variables of oral disease, stress and cortisol cannot be determined from one time point; reverse causalities as well as bidirectional relationships between oral disease and stress are not ruled out by the design or the data presented herein. While cortisol is standardised, it is not a complete picture of its diurnal profile, nor of chronic cortisol output, as there will be better tools to show this using repeated sampling or hair cortisol. There is the possibility of residual confounding by unmeasured factors. There is a need to confirm the direction and magnitude of the pathway suggested here with well-designed longitudinal studies that employ repeated exposure and biomarker measurements.

Consistency audit: The displayed mediation table gives an indirect estimate of 0.064 and a total effect of 0.148, implying approximately 43.2% as a descriptive ratio (0.064/0.148), not 20%. The reported bootstrap interval is labelled 95% in the table and must use that level consistently throughout. Because exposure, cortisol and cumulative DMFT were measured concurrently, this statistical decomposition should not be interpreted as causal mediation.

CONCLUSION

The reported data show associations of residential noise category and salivary cortisol with DMFT in 250 sampled adults, but the cross-sectional design cannot establish whether noise caused stress, whether cortisol influenced caries, or the temporal direction of these relationships. The findings justify prospective environmental exposure monitoring and longitudinal dental assessments, not a demonstrated mechanistic pathway.

Author Contributions

Conceptualization, S.K.G. and A.V.; Methodology, S.K.G., V.R. and Sh.V.; Formal analysis, A.V. and S.S.K.; Investigation, S.K.G., V.R., S.M. and Sh.G.; Data curation, S.M. and S.S.; Writing-original draft preparation, S.K.G. and S.S.; Writing-review and editing, all authors; supervision, A.V. and Sh.V. All authors have read and agreed to the published version of the manuscript.

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Informed Consent Statement

Written informed consent was obtained from all participants involved in the study.

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Conflicts of Interest

The authors declare no conflicts of interest.

REFERENCES

[1] WHO. “Oral health.” World Health Organization, 2024. Available online: https://www.who.int/news-room/fact-sheets/detail/oral-health (accessed 23 August 2026).

[2] Gupta, A. *et al.* “Prevalence and association of emotional eating with dental caries in Indian college students: A cross-sectional study.” *Oral Sphere Journal of Dental and Health Sciences*, vol. 1, no. 2, 2025, pp. 56-62. DOI:10.63150/osjdhs.2025.2.

[3] Kazeminia, M. *et al.* “Dental caries in primary and permanent teeth in children worldwide, 1995 to 2019: A systematic review and meta-analysis.” *Head and Face Medicine*, vol. 16, no. 1, 2020, article 22. DOI:10.1186/s13005-020-00237-z.

[4] Karande, V. *et al.* “AI-assisted versus traditional radiographic interpretation for early caries detection.” *Bioinformation*, vol. 22, no. 4, 2026, pp. 2278-2282. DOI:10.6026/973206300222278.

[5] Alamoudi, A. *et al.* “Role of salivary biomarkers in diagnosis and detection of dental caries: A systematic review.” *Diagnostics*, vol. 12, no. 12, 2022, article 3080. DOI:10.3390/diagnostics12123080.

[6] World Health Organization. Oral Health Surveys: Basic Methods. 5th Edn., World Health Organization, 2013.

[7] Ahmad, F.N. *et al.* “Problematic gaming behavior, mental health status and its effect on oral health among BDS undergraduate students in an urban setting: A cross-sectional study.” *Oral Sphere Journal of Dental and Health Sciences*, vol. 2, no. 3, 2026. DOI:10.63150/osjdhs.2026.25.

[8] Basner, M. *et al.* “Auditory and non-auditory effects of noise on health.” *The Lancet*, vol. 383, no. 9925, 2014, pp. 1325-1332. DOI:10.1016/S0140-6736(13)61613-X.

[9] World Health Organization Regional Office for Europe. Environmental noise guidelines for the European Region. WHO Regional Office for Europe, 2018.

[10] Stansfeld, S.A. and M.P. Matheson. "Noise pollution: Non-auditory effects on health." *British Medical Bulletin*, vol. 68, 2003, pp. 243-257. DOI:10.1093/bmb/ldg033.

[11] Munzel, T. *et al.* "Transportation noise pollution and cardiovascular disease." *Nature Reviews Cardiology*, vol. 18, no. 9, 2021, pp. 619-636. DOI:10.1038/s41569-021-00532-5.

[12] Chrousos, G.P. "Stress and disorders of the stress system." *Nature Reviews Endocrinology*, vol. 5, no. 7, 2009, pp. 374-381. DOI:10.1038/nrendo.2009.106.

[13] Selander, J. *et al.* "Saliva cortisol and exposure to aircraft noise in six European countries." *Environmental Health Perspectives*, vol. 117, no. 11, 2009, pp. 1713-1717. DOI:10.1289/ehp.0900933.

[14] Baudin, C. *et al.* "Saliva cortisol in relation to aircraft noise exposure: Pooled-analysis results from seven European countries." *Environmental Health*, vol. 18, no. 1, 2019, article 102. DOI:10.1186/s12940-019-0540-0.

[15] Hellhammer, D.H. *et al.* "Salivary cortisol as a biomarker in stress research." *Psychoneuroendocrinology*, vol. 34, no. 2, 2009, pp. 163-171. DOI:10.1016/j.psyneuen.2008.10.026.

[16] Marcotte, H. and Lavoie, M.C. "Oral microbial ecology and the role of salivary immunoglobulin A." *Microbiology and Molecular Biology Reviews*, vol. 62, no. 1, 1998, pp. 71-109. DOI:10.1128/MMBR.62.1.71-109.1998.

[17] Magacz, M. *et al.* "The significance of lactoperoxidase system in oral health: Application and efficacy in oral hygiene products." *International Journal of Molecular Sciences*, vol. 20, no. 6, 2019, article 1443. DOI:10.3390/ijms20061443.

[18] Hussein, Y.A. *et al.* "Association between environmental stress factors, salivary cortisol level and dental caries in Egyptian preschool children: A case-control study." *Scientific Reports*, vol. 15, 2025, article 11063. DOI:10.1038/s41598-025-94327-0.

[19] Padmanabhan, V. *et al.* "Association between salivary cortisol levels, dental anxiety and dental caries in children: A cross-sectional study." *Dentistry Journal*, vol. 11, no. 9, 2023, article 205. DOI:10.3390/dj11090205.

[20] Caruso, S. *et al.* "Association between salivary cortisol level and caries in early childhood." *European Journal of Paediatric Dentistry*, vol. 19, no. 1, 2018, pp. 10-15. DOI:10.23804/ejpd.2018.19.01.02.

[21] Rai, K. *et al.* "Estimation of salivary cortisol in children with rampant caries." *Journal of Clinical Pediatric Dentistry*, vol. 34, no. 3, 2010, pp. 249-252. DOI:10.17796/jcpd.34.3.l858480k80031jn2.

[22] Pani, S.C. *et al.* "Salivary cortisol as a biomarker to explore the role of maternal stress in early childhood caries." *International Journal of Dentistry*, vol. 2013, 2013, article 565102. DOI:10.1155/2013/565102.

[23] Kelly, S.J. *et al.* "Levels and confounders of morning cortisol collected from adolescents in a naturalistic (school) setting." *Psychoneuroendocrinology*, vol. 33, no. 9, 2008, pp. 1257-1268. DOI:10.1016/j.psyneuen.2008.06.010.

[24] Shenkin, J.D. *et al.* "The association between environmental tobacco smoke and primary tooth caries." *Journal of Public Health Dentistry*, vol. 64, no. 3, 2004, pp. 184-186. DOI:10.1111/j.1752-7325.2004.tb02750.x.

[25] Tarullo, A.R. *et al.* "Cortisol and socioeconomic status in early childhood: A multidimensional assessment." *Development and Psychopathology*, vol. 32, no. 5, 2020, pp. 1876-1887. DOI:10.1017/S0954579420001315.

[26] Dumitrescu, R. *et al.* "The impact of parental education on schoolchildren's oral health: A multicenter cross-sectional study in Romania." *International Journal of Environmental Research and Public Health*, vol. 19, no. 17, 2022, article 11102. DOI:10.3390/ijerph191711102.

[27] Nollet, M. *et al.* "Sleep deprivation and stress: A reciprocal relationship." *Interface Focus*, vol. 10, no. 3, 2020, article 20190092. DOI:10.1098/rsfs.2019.0092.

[28] Chen, H. *et al.* "Insufficient sleep and incidence of dental caries in deciduous teeth among children in Japan: A population-based cohort study." *The Journal of Pediatrics*, vol. 198, 2018, pp. 279-286. DOI:10.1016/j.jpeds.2018.03.033.

[29] Coxon, J.D. *et al.* "The impact of dental anxiety on the oral health of children aged 5 and 8 years: A regression analysis of the Child Dental Health Survey 2013." *British Dental Journal*, vol. 227, no. 9, 2019, pp. 818-822. DOI:10.1038/s41415-019-0853-y.

[30] Adeoye, J. and Su, Y.X. "Artificial intelligence in salivary biomarker discovery and validation for oral diseases." *Oral Diseases*, vol. 30, no. 1, 2024, pp. 23-37. DOI:10.1111/odi.14641.

[31] Kashwani, R. *et al.* "Assessing knowledge, attitudes and practices of augmented reality technology in dentistry: A cross-sectional survey." *Oral Sphere Journal of Dental and Health Sciences*, vol. 1, no. 1, 2025. DOI:10.63150/osjdhs.2025.30.

[32] Kashwani, R. *et al.* "Virtual Vs augmented reality in the field of dentistry." *Community Practitioner*, vol. 21, no. 3, 2024, pp. 597-605.

[33] Lashkar, R.M. and N.O. Binmuhana. "Fluoride therapies in caries prevention: An updated evidence review." *Oral Sphere Journal of Dental and Health Sciences*, vol. 2, no. 3, 2026. DOI:10.63150/osjdhs.2026.28.

[34] Wankhade, A. *et al.* "Effect of lavender and orange aromatherapy on dental anxiety and pain perception in children undergoing tooth extraction: A randomized controlled trial." *Oral Sphere Journal of Dental and Health Sciences*, vol. 2, no. 3, 2026. DOI:10.63150/osjdhs.2026.22.

[35] Gajdhar, S.K. "USAG-1 inhibition: Paving the way for lab-grown tooth regeneration in modern dentistry." *Oral Sphere Journal of Dental and Health Sciences*, vol. 1, no. 4, 2025. DOI:10.63150/osjdhs.2025.29