ORIGINAL RESEARCH PAPER

Dental Sciences

ORAL DISEASES-THE SILENT EPIDEMIC- AN ORIGINAL REVIEW ARTICLE

KEY WORDS: Oral health, Systemic health, Periodontal disease, Cardiovascular disease, Diabetes mellitus

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ABSTRACT

Oral diseases are among the most prevalent chronic conditions worldwide and represent a significant public health burden. Conditions such as dental caries, periodontal diseases, oral cancer, and oral infections not only affect the oral cavity but also influence systemic health. Increasing scientific evidence supports a bidirectional relationship between oral diseases and systemic conditions including cardiovascular diseases, diabetes mellitus, adverse pregnancy outcomes, respiratory diseases, and autoimmune disorders. Chronic oral inflammation, microbial dissemination, and immune–inflammatory pathways act as biological mediators linking oral and systemic health. This paper explores the conceptual framework, available literature, clinical implications, and future research directions concerning the relationship between oral diseases and overall body health.

INTRODUCTION

Oral health is an integral component of general health and well-being. According to the World Health Organization, oral health is defined as the absence of chronic mouth and facial pain, oral and throat cancer, oral sores, birth defects such as cleft lip and palate, periodontal disease, tooth decay, and tooth loss.

Globally, oral diseases affect nearly 3.5 billion people. Among these, periodontal disease and untreated dental caries are the most common. The mouth acts as a gateway to the body, serving as a reservoir of microorganisms capable of systemic dissemination. Over the past three decades, research has highlighted strong associations between oral diseases and systemic disorders, leading to the emergence of "periodontal medicine" as a specialized area of study.

Fig 1 SHOWS THE PERIODONTAL DISEASE AFFECTING GUMS

REVIEW OF LITERATURE

Y Li et al (2001) conducted to determine the prevalence of bone fracture, including hip fracture, in six Chinese populations with water fluoride concentrations ranging from 0.25 to 7.97 parts per million (ppm). A total of 8266 male and female subjects > or =50 years of age were enrolled. Parameters evaluated included fluoride exposure, prevalence of bone fractures, demographics, medical history, physical activity, cigarette smoking, and alcohol consumption. The results confirmed that drinking water was the only major source of fluoride exposure in the study populations. A U-shaped pattern was detected for the relationship between the prevalence of bone fracture and water fluoride level. The prevalence of overall bone fracture was lowest in the population of 1.00-1.06 ppm fluoride in drinking water, which was significantly lower ($p < 0.05$) than that of the groups exposed to water fluoride levels > or =4.32 and < or =0.34 ppm. The prevalence of hip fractures was highest in the group with the highest water fluoride (4.32-7.97 ppm). The value is significantly higher than the population with 1.00-1.06 ppm water fluoride, which had the lowest prevalence rate. It is concluded that long-term fluoride exposure from drinking water containing > or =4.32 ppm increases the risk of overall fractures as well as hip fractures. Water fluoride levels at 1.00-1.06 ppm decrease the risk of overall fractures relative to negligible fluoride in water; however, there does not appear to be similar protective benefits for the risk of hip fractures.

Riccardo Mazzoli et al (2025) conducted a systematic review and dose-response meta-analysis by searching literature in online databases (MEDLINE/PubMed, Embase and Web of Science) up to August 26, 2025, and by pooling results of studies examining fluoride exposure and its associations with bone health outcomes, namely fracture risk and BMD. They also conducted stratified analyses by sex, age, and fracture site. This study is registered in PROSPERO (CRD42022321899). included 37 studies published between 1945 and 2024. 10 pertained to bone density only; 19 to bone fractures only; and eight to both outcomes. They found a non-linear, positive relation between fluoride exposure and fracture risk, with an indication of an approximate threshold around 1.5 mg/L of fluoride in drinking water and a nearly linear increasing fracture risk above that concentration (risk ratios of 1.06, 1.19 and 1.35 at 2.0, 3.0 and 4.0 mg/L, respectively, compared to null exposure). Sex-specific dose-response analyses, available only for fragility fractures, indicated an effect in females but little or nothing in males. The association between fluoride and BMD was inconsistent, showing opposite trends for different bone sites (hip and spine) and amounts of exposure, and by sex. Among females aged over 50 years, an association of drinking water fluoride with fragility fracture risk started as early as around 0.5 mg/L (risk ratio of 1.26 at 1.0 mg/L). This study indicates increased risk of fractures with fluoride exposure > 1.5 mg/L in drinking water, with an enhanced susceptibility in postmenopausal females. These findings, which appear consistent with those yielded by recent pooled analysis related to other endpoints in children, may be helpful in assessing the risk-benefit profile for fluoride exposure.

Eida Medjedovic et al (2015) conducted a study to determine the effects of improving the health status of teeth after six months treatment with the use of topical fluoridation 0.5% NaF, and the level and quality of the impact of treatment with chemical 0.5% NaF on the dental health of children at age from 8 to 15 years, in relation to gender and chronological age. This study included school children aged 8 to 15 years who visited health and dental services dependent in Mostar. It is obvious that after the implementation of treatment with 5% NaF by the method of topical fluoridation, health status of subjects from the experimental group significantly improved, so that at the final review 89.71% or 61 subjects of the experimental group had healthy (cured teeth), tooth with dental caries only 5.88% or 4 respondents tooth with dental caries and filling 4.41% or 3 respondents, extracted baby tooth 14.71% or 10 respondents, while for 13.24% of respondents was identified state with still unerupted teeth. Our findings are indirectly confirmed that the six-month treatment of fluoridation with 5% NaF, contributed to statistically significant improvement in overall oral health of the experimental group compared to the control group which was not treated by any dental treatment. It can be concluded that there is a statistically significant difference in the evaluated parameters of oral health of children in the control group compared to the studied parameters of oral health the experimental group of children at the final dental examination.

Xin Hai Yin et al (2015) conducted fourteen observational studies involving thirteen cohort studies and one case-control study were included in the meta-analysis. Exposure to fluoride in drinking water does not significantly increase the incidence of hip fracture (RRs, 1.05; 95% CIs, 0.96–1.15). Sensitivity analyses based on adjustment for covariates, effect measure, country, sex, sample size, quality of Newcastle–Ottawa Scale scores, and follow-up period validated the strength of the results. Meta-regression showed that country, gender, quality of Newcastle–Ottawa Scale scores, adjustment for covariates and sample size were not sources of heterogeneity. Little evidence of publication bias was observed. The present meta-analysis suggests that chronic fluoride exposure from drinking water does not significantly increase the risk of hip fracture. Given the potential confounding factors and exposure misclassification, further large-scale, high-quality studies are needed to evaluate the association between exposure to fluoride in drinking water and hip fracture risk.

Yurong Leng et al (2023) conducted twenty-six studies were included. In patients with periodontal disease, the prevalence of CVD was 7.2% [9 studies; 95% confidence interval (CI): 2.7-13.6%], and prevalence for CHD, hypertension, stroke, and heart failure was 6.6, 25.3, 1, and 1.1%, respectively. There was a significant association between periodontal disease and CVD in men [odds ratio (OR) = 1.22; 95% CI: 1.12-1.34] and women (OR = 1.11; 95% CI: 1.05-1.17), with no significant sex difference ($P > 0.05$). Cardiovascular disease is relatively common in patients with periodontal disease, and an increased risk of CVD is associated with periodontal disease independent of sex. Interventions targeting periodontal disease may be beneficial for CVD.

Naif Alwithanani et al (2023) calculated the combined impact of diabetes-related hyperglycemia on the start or progression of periodontitis using meta-analysis. To look into possible sources of study heterogeneity, subgroup analyses and meta-regression were used. With 49,262 participants from 13 studies that met the inclusion criteria, 3197 of whom had been diagnosed with diabetes. Diabetes augmented the likelihood of developing or progressing into periodontitis by 86%, according to meta-analyses of adjusted estimates (RR 1.86; 95% CI 1.3-2.8). On the association between periodontitis and diabetes, there is little data, nonetheless. This study provides proof that persons with diabetes have an increased risk of developing periodontitis. Methodological limitations mentioned in this study should be overcome in upcoming prospective longitudinal investigations.

Andrea Butera et al (2023) conducted to analyze studies focused on periodontitis and adverse pregnancy outcomes. 16 articles have been included. Most of the studies showed adverse outcomes, like preterm birth and the low weight of the unborn child, are among the most frequent consequences (respectively 62.5% and 68.7% of articles); pre-eclampsia is also linked to this condition (12.5% of articles); and perinatal mortality (12.5% of articles). Periodontal disease appears to be associated with adverse events in pregnancy due to the transport of biofilm bacteria into the bloodstream and into placental tissue; what would cause adverse events is the body's immune response to infection.

Issac Suzart Gomes Filho et al (2020) conduct a systematic review and meta-analysis to evaluate the recent scientific literature addressing the association between periodontitis and asthma, chronic obstructive pulmonary disease (COPD), and pneumonia. A total of 3,234 records were identified in the database search, with only 13 studies meeting the eligibility criteria and 10 studies contributed data for meta-analysis. Using a random effects models periodontitis was associated with asthma: ORadjusted: 3.54 (95% CI: 2.47-5.07), $I^2 = 0\%$; with COPD: OR adjusted: 1.78 (95% CI: 1.04-3.05), $I^2 = 37.9\%$; and with pneumonia: OR adjusted: 3.21 (95% CI: 1.997-5.17), $I^2 = 0\%$. The main findings of this systematic review validated an association between periodontitis and asthma, COPD and pneumonia.

Yan Ma et al (2024) conducted a thorough search of electronic databases (EMBASE, PubMed, Web of Science, and Cochrane Library) up to September 2023. The Newcastle-Ottawa Scale (NOS) was applied to assess study quality. To evaluate potential publication bias, both a funnel plot and Egger's test were employed. Additionally, a sensitivity analysis was conducted to explore the source of heterogeneity when the I2 statistic exceeded 50%. This systematic review encompassed 16 studies, involving a total of 6,032 OC patients and 7,432 healthy controls. Our meta-analysis, incorporating data from nine studies, revealed a significant correlation between periodontitis and the risk of OC (OR [odds ratio] = 2.94, 95% CI [confidence interval] (2.13, 4.07); five studies, 6,927 participants; low certainty of evidence). Findings also suggested that individuals with more than 15 missing teeth may have a heightened risk of OC (OR = 1.91, 95% CI (1.01, 3.62)). Furthermore, clinical attachment loss (CAL) and decayed, missing, and filled teeth (DMFT) in OC patients were more pronounced compared to the control group (CAL, SMD = 1.94, 95% CI (0.22, 3.66); DMFT, SMD = 0.65, 95% CI (0.12, 1.18)). Periodontitis may serve as a potential risk factor for OC. However, caution is warranted in interpreting these findings due to the substantial level of heterogeneity.

Daniel Diehl (2026) conducted the oral-systemic health connection refers to the bidirectional relationship between oral health and overall systemic health. Emerging research has shown that periodontal diseases, such as gingivitis and periodontitis, are linked to a range of systemic conditions, including cardiovascular disease (CVD), diabetes mellitus, adverse pregnancy outcomes (APOs), and cancer. The pathogenesis of periodontitis involves complex host-microbe interactions, which can lead to periodontal tissue destruction and systemic inflammation. Bacterial translocation and the systemic spread of pro-inflammatory mediators, such as lipopolysaccharides and cytokines, are explored as potential pathways linking oral to systemic health.In this chapter, the evidence from epidemiological and clinical studies supporting these associations is reviewed, addressing the limitations and controversies in existing research and emphasizing the mechanistic evidence for a possible causal relationship. Additionally, it discusses the impact of improving periodontal health on managing systemic conditions, presenting periodontal treatment as a potential adjunctive therapy to reduce systemic disease risk and severity.

Sara Camanes Gonzalvo et al (2024) conducted across four databases, encompassing potentially relevant studies published up to April 2024 related to the PECO question: "Is there a differentiation in oral microbial composition between adult patients diagnosed with colorectal cancer compared to healthy patients?". The Newcastle-Ottawa Scale was used to evaluate the quality of the studies included. The level of evidence was assessed through the GRADE (Grading of Recommendations, Assessment, Development and Evaluation) tool. Sixteen studies fulfilled the eligibility criteria. Based on low to moderate evidence profile, high levels of certain subspecies within Firmicutes (such as Streptococcus anginosus, Peptostreptococcus stomatis, S. koreensis, and S. gallolyticus), Prevotella intermedia, Fusobacterium nucleatum, and Neisseria oralis were found to be associated with colorectal cancer. Conversely, certain bacteria (e.g., Lachnospiraceae, F. periodonticum, and P. melaninogenica) could exert a symbiotic protective effect against colorectal cancer. Based on existing evidence, it appears that variations in oral microbiota composition exist among individuals with and without colorectal cancer. However, further research is necessary to determine the mechanisms of oral dysbiosis in colorectal carcinogenesis.

Ryota Nomura et al (2020) evaluated the virulence of *S. mutans* for IE development using rats with concurrent severe dental caries and heart valve injury. Dental caries was induced in rats through the combination of a caries-inducing diet and the administration of *S. mutans* into the oral cavity. Then, the heart valves of a subset of rats were injured using a sterile catheter and wire under general anesthesia. The rats were euthanized at various times with various stages of dental caries. The number of teeth affected by dental caries with pulp exposure was increased in the rats in a time-dependent manner. *S. mutans* was recovered from injured heart tissue, which was mainly observed in rats with higher number of *S. mutans* bacteria in mandibular bone and a larger number of teeth in which caries extended to pulp. Dental caries was more severe in rats with heart injury than in rats without heart injury. Sequencing analysis targeting 16S rRNA revealed that specific oral bacteria appeared only in rats with heart injury, which may be related to the development of dental caries. Our findings suggest that dental caries caused by the combination of *S. mutans* infection and sucrose intake may contribute to *S. mutans* colonization in injured heart tissue.

Fig 2 SHOWS THE INFECTIVE ENDOCARDITIS

CONCEPTUAL SECTIONS

1. Biological Mechanisms Linking Oral and Systemic Health

- Inflammatory Pathway: Chronic oral inflammation increases systemic inflammatory markers.
- Bacteremia: Oral bacteria enter systemic circulation through ulcerated periodontal pockets
- Immune Modulation: Altered host immune response contributes to systemic complications.
- Molecular Mimicry: Antibodies against periodontal pathogens may cross-react with host tissues.

2. Common Risk Factor Approach (CRFA)

Many oral and systemic diseases share common risk factors:

- Tobacco us
- Alcohol consumptio
- Poor diet
- Stress
- Socioeconomic determinants

This approach integrates oral health into general health promotion strategies.

3. Psychosocial Impact

Oral diseases affect quality of life, nutrition, speech, and social interactions, thereby indirectly influencing systemic health and mental well-being.

Fig 3 SHOWS THE LOW BIRTH WEIGHT BABY

CRITICAL ANALYSIS SECTIONS

Although numerous observational studies show associations between oral and systemic diseases, establishing causality remains challenging.

Strengths of Existing Evidence:

- Large epidemiological studies.
- Biological plausibility supported by laboratory research.
- Intervention trials showing improvement in systemic markers after periodontal therapy.

Limitations:

- Confounding variables such as smoking and socioeconomic status.
- Variation in diagnostic criteria
- Lack of long-term randomized controlled trials.
- Modest magnitude of systemic improvement after dental treatment.

Current evidence supports association but not definitive causation in many systemic conditions.

Fig 4 SHOWS THE PATIENT SUFFERING FROM COPD (Chronic Obstructive Pulmonary Disease)

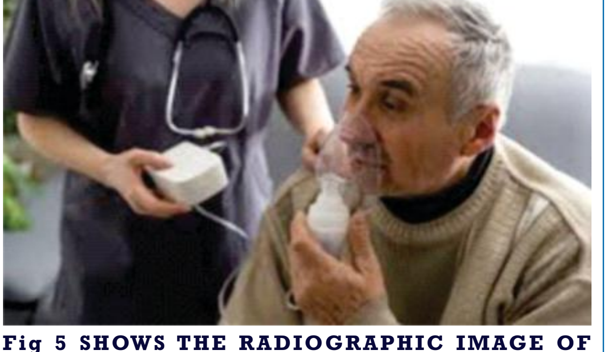


Fig 5 SHOWS THE RADIOGRAPHIC IMAGE OF PNEUMONIA

Fig 6 SHOWS THE DENTAL FLUOROSIS

Fig 7 SHOWS THE COLON CANCER

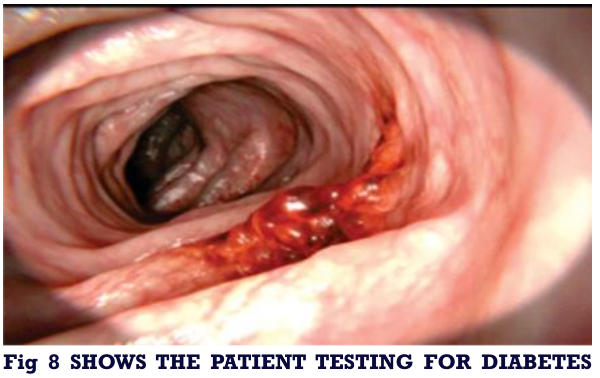


Fig 8 SHOWS THE PATIENT TESTING FOR DIABETES MELLITUS

CLINICAL IMPLICATIONS-

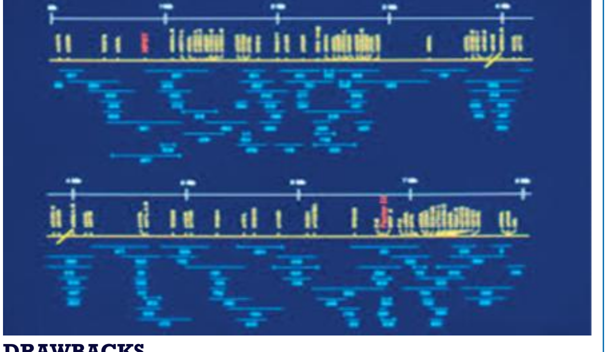
- Interdisciplinary Collaboration-Integration between dentists and physicians is essential for holistic patient care.
- Screening and Early Detection- Dental professionals can help identify undiagnosed diabetes or hypertension through chairside screening.
- Preventive Strategies-Emphasis on oral hygiene education as part of chronic disease prevention
- Hospital Oral Care Protocols-Implementation of structured oral hygiene programs to prevent hospital-acquired infections.
- Patient Education- Patients must understand that oral health is inseparable from general health.

Fig 9 SHOWS THE HOSPITAL ORAL CARE PROTOCOLS

FUTURE RESEARCH DIRECTIONS-

- Long-term randomized controlled trials to establish causality
- Genomic and microbiome studies exploring host–microbe interactions.
- Biomarker development for early detection of systemic complications.
- Evaluation of cost-effectiveness of integrated oral-systemic health models.
- Artificial intelligence-based risk prediction tools.
- Public health intervention trials incorporating oral health into NCD control programs.

Fig 10 SHOWS THE GENOME MAPPING



DRAWBACKS-

- Limited awareness among healthcare professionals.
- Fragmented healthcare delivery systems.
- Inadequate insurance coverage for dental care.
- Lack of standardized periodontal diagnostic criteria.
- High global burden with limited resources in developing countries.
- Difficulty in isolating oral disease as an independent risk factor.

CONCLUSION-

Oral diseases significantly influence systemic health through inflammatory, microbial, and immunological pathways. The relationship is often bidirectional, particularly in conditions such as diabetes and cardiovascular diseases. While strong associations exist, further research is required to establish definitive causal links. Integrating oral healthcare into primary healthcare systems is crucial for comprehensive disease prevention and health promotion. Recognizing oral health as an essential component of general health will improve overall quality of life and reduce the global burden of chronic diseases. Good Oral health can help control or lessen risk associated with systemic health condition and it also improves self-esteem

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