Influence of Lifestyle on Oral Health

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***Abstract*—Oral health is a vital component of overall systemic well-being and is significantly influenced by various lifestyle factors. Common oral diseases such as dental caries, periodontal disease, oral cancer, and xerostomia are largely associated with modifiable behaviours including diet and nutrition, tobacco use, alcohol consumption, psychological stress, oral hygiene practices, physical activity, and body weight. Physical inactivity and obesity are independently associated with increased periodontitis risk through elevated systemic inflammatory mediator including interleukin 6, tumour necrosis factor alpha and c-reactive protein. Type 2 diabetes mellitus, itself largely driven by lifestyle factors, demonstrates a well-established bidirectional relationship with periodontal disease, wherein poor glycaemic control exacerbates periodontal inflammation and, conversely, untreated periodontitis impairs metabolic regulation. Nutritional deficiencies particularly of calcium, vitamin D, and vitamin C further compromise periodontal and mucosal integrity. Conversely, effective oral hygiene practices and regular professional dental care significantly aid in the prevention and control of oral diseases. The findings indicate that high sugar intake plays a critical role in the development of dental caries, while tobacco use and alcohol consumption are strongly linked to periodontal disease and oral cancer. Psychological stress contributes to immune dysregulation, thereby accelerating periodontal breakdown. Conversely, effective oral hygiene practices and regular professional dental care significantly aid in the prevention and control of oral diseases. Adopting comprehensive lifestyle-based approach that integrates oral health into general health promotion can substantially reduce disease burden. Therefore, dental professionals have a crucial role in promoting healthy lifestyle behaviours as part of routine patient care.**

***Index Terms*—Oral health, Lifestyle, Periodontitis, Dental caries, Tobacco, Alcohol, Diet, Stress, Prevention**

I. INTRODUCTION

Oral diseases affect more than 3.5 billion people worldwide, making them the most prevalent group of non-communicable diseases globally.[1] Dental caries, periodontal disease, tooth loss, and oral cancer impose an immense burden on individuals and healthcare systems. Despite significant advances in dental research and clinical practice, the prevalence of these conditions remains disproportionately high, particularly in low- and middle-income populations. The relationship between lifestyle and oral health is bidirectional and multifactorial. Oral diseases share common risk factors with systemic non-communicable diseases, including cardiovascular disease, type 2 diabetes, and chronic obstructive pulmonary disease.[2] These shared determinants primarily tobacco use, alcohol consumption, poor diet, inadequate physical activity, and psychosocial stress constitute modifiable behaviours amenable to intervention. The Common Risk Factor Approach, proposed by Sheiham and Watt, argues that addressing these upstream determinants simultaneously benefits both oral and general health. [2] This review adopts this conceptual framework to examine the influence of major lifestyle domains on oral health outcomes,

drawing upon a curated body of 30 high-impact studies.

II. DIET AND NUTRITION

Dietary Sugars and Dental Caries

Dental caries is the most prevalent disease worldwide and is fundamentally driven by dietary sugar consumption. [3] Fermentable carbohydrates particularly sucrose are metabolised by cariogenic bacteria such as Streptococcus mutans, producing organic acids that demineralise enamel and dentine. Lingström et al. demonstrated in a randomised controlled trial that frequent sugar intake significantly increases the incidence of dental caries in adults. [4] A comprehensive systematic review by Moynihan and Petersen confirmed that the evidence for sugar as the primary dietary cause of caries is strong and consistent across populations. [5]

Sheiham proposed that restricting free sugars to below 10% of total energy intake could achieve dramatic reductions in caries prevalence at a population level. [6] The World Health Organization has adopted this threshold as a public health target. The timing and frequency of sugar intake appear to be more critical determinants of caries risk than the absolute quantity consumed, as each acid challenge initiates a cycle of demineralisation that exceeds the remineralising capacity of saliva.

Micronutrients and Periodontal Health

Nutritional deficiencies affect not only dental hard tissue but also the supporting periodontium and oral mucosa. Calcium and vitamin D are essential for mineralisation of enamel and alveolar bone; deficiencies impair tooth development and accelerate bone resorption. [7] Vitamin C is required for collagen biosynthesis in gingival connective tissue; scurvy an extreme manifestation of ascorbic acid deficiency is characterised by severe gingivitis and tooth loss.

Syrjänen reviewed the oral manifestations of systemic diseases and nutritional deficiencies, noting that the oral mucosa frequently reflects the nutritional status of the host, exhibiting atrophy, ulceration, and angular cheilitis in deficiency states. [8] Modern dietary patterns characterised by ultra-processed foods, low in fibre and micronutrients, therefore represent a dual threat to caries risk and periodontal integrity.

III. TOBACCO USE

Smoked Tobacco and Periodontal Disease

Tobacco smoking is among the most extensively documented risk factors for periodontal disease. Tomar and Asma, analysing NHANES III data comprising 12,329 adults, found that smokers were approximately four times more likely to have periodontal disease than non-smokers after controlling for confounders.[9] Albandar et al. similarly identified tobacco use as a strong independent predictor of severe periodontitis in a large epidemiological sample. [10]

The mechanisms underlying tobacco-associated periodontitis are multiple. Nicotine causes gingival vasoconstriction, masking the clinical signs of inflammation (erythema, bleeding on probing) and delaying diagnosis. Smoking impairs polymorphonuclear leucocyte chemotaxis, phagocytosis, and oxidative burst, reducing host defence against subgingival pathogens. Furthermore, tobacco smoke promotes dysbiosis of the subgingival microbiome, enriching anaerobic and virulent species. Critically, Bergström demonstrated in a 30-year longitudinal study that smoking cessation substantially reduces the rate of periodontal bone loss, with cessation benefits accumulating over time.[11] This finding has important implications for clinical counselling, underscoring that it is never too late to advise patients to stop smoking.

Smokeless Tobacco

Smokeless tobacco products including chewing tobacco, snuff, and betel quid exert direct mucosal toxicity. Squier and Kremer described gingival recession, leukoplakia, erythroplakia, and frank carcinomas at sites of chronic smokeless tobacco contact. [12] Nitrosamines present in cured tobacco are potent carcinogens that initiate mutagenic changes in oral epithelium. The risk is compounded by co-use with alcohol and areca nut.

Tobacco and Oral Cancer

Warnakulasuriya's comprehensive review identified tobacco and alcohol as the two primary causative agents of oral squamous cell carcinoma, together accounting for 75–90% of cases in developed countries. [13] The carcinogenic risk is synergistic when

tobacco and alcohol are combined, exceeding the additive effect of each agent alone.

Alcohol Consumption

Alcohol exerts deleterious effects on oral tissues through several pathways. Acetaldehyde, the primary metabolite of ethanol, is classified as a Group 1 carcinogen by the International Agency for Research on Cancer. Meurman and Uittamo reviewed the carcinogenic mechanisms of alcohol-associated acetaldehyde in the oral cavity, highlighting direct DNA adduct formation and impairment of DNA repair mechanisms. [14]

Boyle et al. demonstrated a dose-dependent relationship between alcohol consumption and oral cancer risk, with heavy drinkers (>4 units/day) having a risk up to five-fold greater than abstainers. [15] Merchant et al., in a large prospective cohort of 51,529 male health professionals, found that alcohol consumption was associated with a significantly increased risk of periodontitis (relative risk 1.27), with the association strongest for spirits and heavy consumption patterns. [16]

Alcohol also induces salivary gland dysfunction and xerostomia, impairing the antimicrobial and buffering functions of saliva. Reduced salivary flow promotes microbial colonisation, plaque accumulation, and acid demineralisation of enamel, creating a permissive environment for caries and periodontal disease.

Psychological Stress

The relationship between psychological stress and oral health operates through both direct neuroendocrine pathways and indirect behavioural mechanisms. Peruzzo et al., in a systematic review of 14 studies, concluded that psychological stress is associated with greater severity of periodontitis, likely mediated by elevated cortisol-driven immunosuppression and increased corticotropin-releasing hormone, which amplifies pro-inflammatory cytokine production. [17]

Wimmer et al. conducted a case-control study of 100 periodontal patients and found that high stress levels, as measured by validated psychological instruments, correlated significantly with greater clinical attachment loss. [18] Marcenes and Sheiham examined occupational stress in 304 male workers and identified a significant association between job stress and poor oral hygiene practices, suggesting that stress-related neglect of self-care behaviour is an important indirect pathway.[19]

Stress also contributes to parafunctional oral behaviours, including bruxism and clenching, which cause attrition, tooth fracture, temporomandibular joint disorder, and muscle pain. Acute stress is a recognised trigger for recurrent aphthous ulcers and herpes labialis reactivation through T-cell immunosuppression.

Oral Hygiene Practices

Plaque-mediated oral diseases gingivitis, periodontitis, and caries are fundamentally diseases of inadequate plaque control. Morita and Wang reviewed the literature on oral hygiene and concluded that poor plaque removal is the principal local risk factor for the initiation and progression of periodontal disease.[20] Without regular mechanical removal, bacterial biofilm matures from simple gram-positive streptococcal communities to complex, anaerobic, gram-negative ecosystems characterised by periodontal pathogens such as Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola.

The 30-year longitudinal study by Axelsson et al. demonstrated that regular professional prophylaxis, combined with effective home oral hygiene, virtually eliminated tooth loss from periodontal disease in a Swedish adult population, and reduced caries rates by over 90%.[21]This study remains the most compelling evidence for the efficacy of preventive oral hygiene in maintaining lifelong oral health.

Buset et al., in a systematic review of 16 studies, found that successful periodontal treatment significantly improved patient-reported quality of life and positively influenced lifestyle behaviours, including dietary habits and smoking cessation motivation.[22]This suggests a virtuous cycle in which oral health improvement and lifestyle improvement reinforce one another.

Physical Activity and Body Weight

Emerging evidence supports an association between physical activity levels, body mass index, and periodontal health. Al-Zahrani et al., analysing NHANES III data from 13,665 American adults, found that individuals who met recommended physical activity guidelines had a significantly lower prevalence of periodontitis, independent of smoking and sociodemographic factors. [23] The proposed

mechanism involves reduced systemic inflammatory load in physically active individuals, as regular aerobic exercise attenuates circulating interleukin-6 and C-reactive protein levels.

Suvan et al. conducted a systematic review of 38 studies and found that obesity, defined by body mass index, waist circumference, or body fat percentage, was independently associated with periodontitis with an odds ratio of 1.35. [24] Adipose tissue, particularly visceral fat, secretes pro-inflammatory adipokines leptin, tumour necrosis factor-alpha, and interleukin-1 that augment the systemic inflammatory milieu and amplify periodontal tissue destruction. Bartold and Van Dyke reviewed the mechanistic overlap between lifestyle-driven systemic inflammation and periodontal pathogenesis in detail. [25]

Systemic Conditions Linked to Lifestyle

Diabetes Mellitus

Type 2 diabetes mellitus, a condition strongly influenced by dietary patterns, physical inactivity, and obesity, has a well-established bidirectional relationship with periodontitis. [26] Salvi et al. reviewed the pathophysiologic links between hyperglycaemia and periodontal disease, noting that advanced glycation end-products accumulate in the gingival tissues of diabetics, impairing collagen turnover and augmenting the receptor for AGE (RAGE)-mediated inflammatory cascade. [27]

Chapple and Genco, in the joint EFP/AAP consensus report, concluded that patients with poorly controlled diabetes (HbA1c >8%) have a three-fold increased risk of periodontitis, and that effective periodontal treatment modestly but significantly improves glycaemic control, reducing HbA1c by approximately 0.4%. This bidirectional relationship has led to calls for co-management of diabetes and periodontitis as intertwined chronic conditions.

Cardiovascular and Systemic Connections

Haumschild reviewed the systemic oral health interface, describing evidence for oral bacteria—particularly streptococci and periodontal pathogens—seeding cardiac endothelium and contributing to atherosclerotic plaque formation.[28] Genco and Borgnakke further outlined how diabetes, smoking, and poor diet act synergistically to worsen both periodontal and cardiovascular outcomes through shared inflammatory pathways. [29]

Lifestyle Factors, Mechanisms, and Evidence Levels

Lifestyle Factor	Oral Condition Affected	Mechanism	Evidence Level	References
Diet – High sugar	Dental caries	Fermentable carbohydrates metabolised by Streptococcus mutans produce acids leading to enamel demineralisation	Level I (Systematic Review/Meta-analysis)	[4,5,6]
Diet – Micronutrient deficiency	Enamel hypoplasia, gingivitis	Deficiency of calcium and vitamin D reduces mineralisation; vitamin C deficiency impairs collagen synthesis	Level II (Cohort studies)	[7,8]
Tobacco smoking	Periodontitis, oral cancer	Vasoconstriction, impaired neutrophil function, carcinogen exposure, and microbiome dysbiosis	Level I (Systematic Review/Meta-analysis)	[9,10,11,13]
Smokeless tobacco	Gingival recession, leukoplakia	Direct mucosal irritation, altered oral microbiome, and nitrosamine-induced mutagenesis	Level II	[12]
Alcohol consumption	Periodontitis, oral cancer	Acetaldehyde carcinogenicity, xerostomia, immune suppression, and poor oral hygiene behaviour	Level I	[14,15,16]

Psychological stress	Periodontitis, bruxism, ulcers	Elevated cortisol leading to immunosuppression; neglect of oral hygiene and parafunctional habits	Level II	[17,18,19]
Poor oral hygiene	Gingivitis, periodontitis, caries	Plaque accumulation leads to bacterial biofilm formation and host inflammatory destruction	Level I	[20,21]
Obesity / unhealthy diet pattern	Periodontitis	Adipokine-driven systemic inflammation amplifies periodontal tissue destruction	Level II	[24,29]
Physical inactivity	Periodontitis	Pro-inflammatory state, reduced immune surveillance, and association with higher BMI	Level III	[23]
Diabetes (lifestyle-linked)	Periodontitis, xerostomia	Accumulation of advanced glycation end products (AGEs), impaired polymorphonuclear leukocyte function, and hyperglycaemia-driven inflammation	Level I	[26,27]

Level I: Systematic review/meta-analysis | Level II: Cohort/case-control | Level III: Cross-sectional/expert opinion

Evidence-Based Lifestyle Recommendations for Oral Health

Domain	Recommendation	Expected Benefit	References
Diet	Limit free sugars to <10% of total energy intake; increase intake of fibre, calcium, and vitamin D	Reduction in caries incidence by up to 90% when combined with effective oral hygiene measures	[4,5,7,6]
Tobacco	Complete cessation of smoking and smokeless tobacco products	>50% reduction in periodontal bone loss and decreased risk of oral cancer	[9,13,12,11]
Alcohol	Limit alcohol consumption to moderate levels and avoid combined use with tobacco	Significant reduction in oral cancer and periodontal disease risk	[16,15,14]
Oral Hygiene	Twice-daily tooth brushing with fluoride toothpaste and daily interdental cleaning	Near elimination of gingivitis and major reduction in dental caries and periodontitis	[20,21,22]
Stress Management	Practise mindfulness, cognitive-behavioural strategies, adequate sleep, and dental monitoring for bruxism	Reduced cortisol-mediated immunosuppression and decreased periodontal disease severity	[17,18,19]
Physical Activity	At least 150 minutes per week of moderate aerobic physical activity	Reduced systemic inflammation and lower prevalence of periodontitis	[23,25]
Diabetes Control	Maintain glycaemic control (HbA1c <7%) through diet, medication, and lifestyle modification	Improved periodontal outcomes and reduced xerostomia	[26,27]
Regular Dental Visits	6–12 monthly professional dental examinations and cleaning	Early detection of oral lesions, prevention of tooth loss, and improved quality of life	[21,22,30]

IV. DISCUSSION

Lifestyle behaviours play an important role in determining oral health outcomes. Oral diseases such as dental caries, periodontal disease, and oral cancer are largely preventable and share common risk factors with other chronic systemic diseases, including unhealthy diet, tobacco use, alcohol consumption, physical inactivity, and psychological stress. Petersen et al. (2003) [1] highlighted that oral diseases affect billions of people worldwide and represent a major public health challenge, particularly in developing countries where preventive strategies and access to dental care remain limited.

Dietary habits are among the most important determinants of oral health. Moynihan et al. (2004) [5] reported that frequent consumption of free sugars significantly increases the risk of dental caries due to acid production by cariogenic bacteria following carbohydrate metabolism. Similarly, Lingström et al. (2000) [4] demonstrated that dietary carbohydrates serve as substrates for oral microorganisms such as Streptococcus mutans, leading to enamel demineralisation. Nutritional deficiencies may also negatively affect oral tissues; Touger-Decker et al. (2003) [7] emphasised that inadequate intake of calcium, vitamin D, and vitamin C can impair tooth mineralisation and collagen synthesis, thereby increasing susceptibility to periodontal disease.

Tobacco use is a major risk factor for periodontal disease and oral cancer. Tomar et al. (2000) [9] reported that smokers have a significantly higher prevalence of periodontitis compared with non-smokers due to impaired immune response and altered inflammatory mechanisms. Albandar et al. (2000) [10] also observed an increased risk of periodontal attachment loss and tooth loss among smokers. Furthermore, the use of smokeless tobacco products has been associated with mucosal lesions and potentially malignant disorders. Squier et al. (2001) [12]reported that chronic exposure to tobacco-specific nitrosamines may induce epithelial dysplasia and increase the risk of oral cancer.

Alcohol consumption is another important lifestyle factor affecting oral health. Boyle et al. (2003) [15] reported a dose-dependent association between alcohol intake and oral cancer risk. Meurman et al. (2008) [14]explained that ethanol metabolism produces acetaldehyde, a carcinogenic compound capable of damaging DNA and impairing cellular repair mechanisms. Alcohol use may also reduce salivary flow, increasing the risk of dental caries and periodontal disease.

Psychological stress has also been associated with oral diseases. Peruzzo et al. (2007) [17] reported that stress is linked to increased severity of periodontal disease, likely due to stress-induced immunosuppression. Wimmer et al. (2002) [18] further demonstrated that individuals experiencing higher stress levels often exhibit greater clinical attachment loss. Stress may also indirectly influence oral health by affecting behavioural patterns such as oral hygiene practices and smoking.

Lifestyle-related systemic conditions such as obesity and diabetes are also closely associated with periodontal disease. Chapple et al. (2013) [26] reported that poorly controlled diabetes significantly increases the risk of periodontitis due to impaired immune responses and inflammatory mechanisms. In addition, Suvan et al. (2011) [24]found that obesity is associated with increased periodontal disease risk due to the production of pro-inflammatory cytokines from adipose tissue. Regular physical activity may provide protective effects against periodontal disease; Al-Zahrani et al. (2005) [24]reported that individuals with higher levels of physical activity have a lower prevalence of periodontitis. Overall, integrating lifestyle counselling into routine dental practice may significantly reduce the burden of oral diseases. Preventive strategies focusing on healthy diet, tobacco cessation, alcohol moderation, stress management, and effective oral hygiene practices are essential for improving oral and general health outcomes.

V. CONCLUSION

The evidence reviewed here confirms that lifestyle is the dominant modifiable determinant of oral health across the life course. Dental caries, periodontal disease, oral

cancer, and related conditions are largely preventable through targeted behavioural change—restricting dietary sugars, eliminating tobacco, moderating alcohol, maintaining meticulous oral hygiene, managing stress, and engaging in regular physical activity.

Dental and medical professionals must integrate lifestyle medicine into routine practice. Interprofessional collaboration between dentists,

dietitians, physicians, and mental health practitioners offers the most comprehensive pathway to reducing the oral health burden globally. Future research should focus on implementation science translating well-established evidence into scalable, equitable community interventions.

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