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DENTAL CARIES AND PERIODONTAL DISEASES: A NARRATIVE REVIEW OF SHARED RISK FACTORS, PATHOGENESIS, INTERRELATIONSHIPS AND PREVENTIVE STRATEGIES

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ABSTRACT

Background: Dental caries and periodontal diseases are the two most prevalent oral diseases worldwide and remain leading causes of pain, tooth loss, reduced quality of life and avoidable health expenditure. Although traditionally studied as separate entities, they share an ecological, behavioural and socio-economic foundation that has important implications for prevention and clinical care.

Objective: To provide an integrated narrative review of the epidemiology, pathogenesis, shared risk factors, classification, prevention and management of dental caries and periodontal diseases, and to consider the implications for routine clinical practice and public oral health.

Methods: A narrative review was conducted using PubMed/MEDLINE and Google Scholar, supplemented by hand-searching of consensus reports, Cochrane reviews and World Health Organization publications. English-language systematic reviews, consensus statements, guidelines and key primary studies were prioritised. Reporting was guided by the SANRA quality criteria for narrative reviews.

Results and Discussion: Both conditions are biofilm-mediated, non-communicable diseases driven by dysbiosis of the oral microbiome in response to environmental pressures, principally frequent free-sugar intake in caries and host inflammatory dysregulation in periodontitis. Tobacco use, poor plaque control, socio-economic disadvantage and systemic conditions such as diabetes act as common risk factors. Evidence supports fluoride delivery, sugar reduction, sealants and minimally invasive caries management for caries, and mechanical biofilm control, risk-factor modification and staged periodontal therapy for periodontal diseases. A common risk factor approach, delivered through patient-centred behaviour change and integrated care pathways, offers the most efficient route to reducing the combined burden.

Conclusion: Caries and periodontal diseases should be managed within a single preventive framework that targets the shared determinants of oral health. Strengthening prevention in primary dental care, particularly in resource-limited settings such as South Africa, is essential for reducing the burden of these largely preventable diseases.

Keywords: Dental caries; Periodontal diseases; Periodontitis; Oral biofilm; Risk factors; Preventive dentistry

1 INTRODUCTION

Oral health is more than the absence of disease: it has been defined as a state of the mouth, teeth and orofacial structures that enables essential functions such as eating, speaking and smiling, and that contributes to physical, mental and social wellbeing [1]. Yet oral diseases remain among the most widespread non-communicable diseases globally. The World Health Organization estimates that oral diseases affect close to 3.5 billion people, with the burden concentrated in low- and middle-income countries [2,3]. Untreated caries of permanent teeth and severe periodontitis consistently rank among the most prevalent health conditions in the Global Burden of Disease analyses [4,5].

Dental caries and periodontal diseases have historically been taught, researched and treated as distinct disciplines. However, both arise from the interaction between the oral microbial biofilm and the host under the influence of behavioural, social and systemic factors. They share modifiable determinants including diet, oral hygiene behaviour, tobacco use and access to preventive care, which is the rationale for the common risk factor approach to oral health promotion [6,7]. Clinicians in general practice encounter both conditions in the same patient, often at the same visit, and treatment planning that addresses them in isolation risks duplicating effort and missing the underlying drivers of disease.

The research problem addressed by this review is the fragmented way in which caries and periodontal diseases are frequently presented in the literature and managed in practice, despite their common aetiological foundations. The objective of this review is to synthesise current evidence on the epidemiology, pathogenesis, shared risk factors, classification, prevention and management of these two disease groups, and to outline the implications for clinical practice and public oral health, with particular reference to resource-limited settings.

2 MATERIALS AND METHODS

2.1 Study design

This study is a narrative review. A narrative design was selected because the aim was to integrate evidence from epidemiology, microbiology, clinical trials, consensus reports and public health policy, rather than to answer a single focused intervention question that would be better suited to a systematic review or meta-analysis. The structure and reporting of the review were guided by the SANRA scale for the quality assessment of narrative review articles [8].

2.2 Literature search

PubMed/MEDLINE and Google Scholar were searched for English-language publications using combinations of the terms "dental caries", "periodontal diseases", "periodontitis", "gingivitis", "oral biofilm", "dysbiosis", "risk factors", "common risk factor approach", "prevention" and "fluoride". Reference lists of key publications were hand-searched, and documents from the World Health Organization and consensus reports from professional bodies, including the European Federation of Periodontology (EFP), the Organisation for Caries Research (ORCA) and the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions, were reviewed directly. The search was restricted to the period from 1980 to 2026, with priority given to publications from the last decade and to seminal earlier papers that established key concepts.

2.3 Selection and synthesis

Priority was given to systematic reviews, Cochrane reviews, clinical practice guidelines, consensus reports, major epidemiological analyses and landmark primary studies. Case reports, non-peer-reviewed opinion pieces and studies without an accessible full text were excluded. Findings were synthesised thematically under the headings of epidemiology, pathogenesis, shared risk factors, classification and diagnosis, prevention and management, and public health implications. No formal risk-of-bias assessment or quantitative pooling was performed.

2.4 Ethical considerations

This review is based exclusively on published literature and did not involve human participants, patient data or animals. Ethical approval and informed consent were therefore not required.

3 RESULTS AND DISCUSSION

3.1 Epidemiology and burden of disease

Dental caries is a sugar-driven, multifactorial, biofilm-mediated and dynamic disease resulting in net mineral loss from dental hard tissues [9]. It is the most common chronic disease of childhood and, when untreated in permanent teeth,

has been estimated to affect approximately two billion people worldwide, while roughly half a billion children are affected by caries of the primary dentition [2,4]. Its consequences extend beyond the tooth: pain, infection, disrupted sleep, school absenteeism and impaired growth and quality of life are well documented, particularly where access to restorative care is limited [3,10].

Periodontal diseases range from reversible gingivitis to destructive periodontitis. Severe periodontitis affects approximately 11% of the global population and is a major cause of tooth loss in adults, with prevalence rising with age [4,11]. Beyond its oral effects, periodontitis reduces masticatory function, nutritional intake and quality of life, and it generates substantial direct and indirect economic costs [11,12].

Both conditions display marked social gradients. Disease burden is higher among socio-economically disadvantaged groups, who also have poorer access to fluoride, preventive services and health education [3,7]. In South Africa, oral disease burden and access to dental services are unevenly distributed, with limited public-sector capacity, a predominance of urgent and extraction-focused care and a strong case for prevention-oriented, primary-care based strategies [2,13].

3.2 Pathogenesis of dental caries

Caries develops when the balance between demineralisation and remineralisation at the tooth-biofilm interface is tipped towards mineral loss. Acids produced by plaque bacteria from fermentable carbohydrates lower the local pH; when the pH remains below the critical value for enamel for sufficient time, net demineralisation occurs, whereas saliva, fluoride and buffering capacity promote remineralisation [9,14]. Caries is therefore best understood as a continuum, from subclinical mineral changes to cavitation, with early lesions remaining reversible [14,15].

The classical view that a limited number of specific bacteria, notably *Streptococcus mutans*, cause caries [16] has been refined by the ecological plaque hypothesis. In this model, frequent sugar exposure and repeated acidification select for acidogenic and aciduric species, shifting a health-associated community towards a cariogenic one, a process of dysbiosis rather than infection by a single pathogen [17,18]. The plaque is thus viewed as a structured microbial community whose composition responds to environmental change [19,20]. This has practical consequences: the most effective way to prevent caries is to alter the environment, chiefly by reducing sugar frequency and increasing fluoride exposure, rather than to attempt to eliminate specific organisms.

Free sugars are the dominant dietary determinant. Evidence across life stages supports a dose-response relationship between free-sugar intake and caries, and the World Health Organization recommends that free sugars supply less than 10% of total energy intake, with a further reduction below 5% suggested for additional benefit [21-23].

3.3 Pathogenesis of periodontal diseases

Gingivitis is a reversible inflammatory response to plaque accumulation at the gingival margin, and it precedes but does not always progress to periodontitis [24,25]. Periodontitis is a chronic multifactorial inflammatory disease associated with dysbiotic plaque biofilms and characterised by progressive destruction of the tooth-supporting apparatus [12,26]. Susceptibility varies markedly between individuals, a concept established in the classic model of periodontitis pathogenesis in which the host response, modified by genetic, environmental and acquired factors, determines whether and how quickly tissue destruction occurs [27].

Early work identified structured subgingival microbial complexes associated with disease severity [28]. Current understanding emphasises polymicrobial synergy and dysbiosis: keystone pathogens, present at low abundance, can remodel the microbial community and subvert host immunity, thereby promoting inflammatory destruction [29]. The resulting dysregulated immune-inflammatory response, rather than bacterial toxins alone, is responsible for most of the connective tissue and alveolar bone loss, and it also provides a plausible route for systemic inflammatory effects [30].

Longitudinal data support the value of long-term control. In a 40-year natural history study, oral hygiene and regular preventive care were associated with markedly better tooth retention, whereas untreated disease progressed with a high risk of tooth loss [31].

3.4 Shared risk factors and the interrelationship between the diseases

The two diseases share a set of upstream determinants. Poor plaque control, socio-economic disadvantage, limited health literacy and irregular dental attendance increase risk for both. Tobacco use is a major modifiable risk factor for periodontitis and is also associated with adverse caries outcomes, while high-frequency sugar intake predominantly drives caries but contributes to a pro-inflammatory metabolic state relevant to the periodontium [7,12]. Systemic disease is an important modifier: diabetes mellitus and periodontitis have a well-established two-way relationship, with

poorly controlled diabetes increasing periodontitis risk and severity and periodontal inflammation adversely affecting glycaemic control [7,32]. Associations between periodontitis and cardiovascular disease are supported by consensus, although the evidence for causality and for a cardiovascular benefit from periodontal treatment remains under investigation [33].

Table 1 compares the main features of the two disease groups. Although the mechanisms differ, the recurring theme is that both are driven by biofilm dysbiosis under the influence of modifiable exposures, which is why an integrated, common risk factor strategy is logical [6,34]. Clinically, the diseases also interact locally: gingival recession and periodontal attachment loss expose root surfaces to root caries, particularly in older adults, while carious or overhanging restorative margins impede plaque control and can worsen periodontal inflammation [7].

Table 1: Comparison of the main features of dental caries and periodontal diseases.

Feature	Dental caries	Periodontal diseases
Nature of disease	Dynamic, sugar-driven, biofilm-mediated demineralisation of dental hard tissues	Chronic inflammatory disease of the gingiva and supporting tissues, from reversible gingivitis to periodontitis
Microbial mechanism	Selection of acidogenic and aciduric species by frequent sugar exposure	Dysbiotic, polymicrobial subgingival biofilm with keystone pathogens and host immune dysregulation
Principal modifiable risks	Free-sugar frequency, low fluoride exposure, poor plaque control, reduced salivary flow	Plaque accumulation, tobacco use, poorly controlled diabetes, socio-economic factors
Reversibility	Early (non-cavitated) lesions reversible; cavitation is not	Gingivitis reversible; attachment and bone loss are not, but progression can be arrested
Classification framework	Staging by lesion depth and activity (e.g. caries terminology consensus)	2017 World Workshop classification: stages I-IV and grades A-C
Key preventive measures	Fluoride, sugar reduction, sealants, early lesion arrest	Mechanical plaque control, tobacco cessation, glycaemic control, supportive periodontal care

3.5 Classification and diagnosis

Contemporary caries management relies on a shared vocabulary of lesion detection, severity and activity assessment, and it distinguishes between non-cavitated and cavitated lesions to guide non-operative versus operative care [9,15]. Periodontal diagnosis follows the 2018 classification, which recognises periodontal health, gingivitis and periodontitis, with periodontitis characterised by stage (severity and complexity, I to IV) and grade (rate of progression and risk, A to C) [26,35,36]. Grading incorporates risk modifiers such as smoking and diabetes, reinforcing the link between systemic and behavioural factors and periodontal outcomes.

3.6 Prevention and management of dental caries

Fluoride remains the cornerstone of caries prevention. A Cochrane review found that fluoride toothpaste prevents caries in children and adolescents compared with placebo, with a clear dose-response relationship and greater benefit at higher concentrations [37]. Community water fluoridation has also been associated with a higher proportion of caries-free children and lower caries experience, although the evidence base is largely from studies conducted before widespread use of fluoride toothpaste [38]. Pit and fissure sealants reduce occlusal caries in the permanent molars of children and adolescents compared with no sealant [39].

Modern management prioritises non-operative and minimally invasive approaches: controlling the biofilm and diet, remineralising early lesions, and arresting or selectively removing carious tissue, with restorations reserved for lesions that cannot be controlled otherwise [9,15]. Topical silver diamine fluoride has shown efficacy in arresting caries in the primary dentition, offering a low-cost option that is particularly relevant where access to operative services is restricted [40]. Dietary counselling targeting sugar frequency should be part of every caries risk assessment [21,23].

3.7 Prevention and management of periodontal diseases

Prevention of periodontitis depends on the control of gingivitis. Consistent, effective mechanical plaque removal by the patient, supported by professional prophylaxis, is the primary strategy, with adjunctive chemical agents considered

where mechanical control is inadequate [25,34]. Risk factor control, including tobacco cessation and optimised glycaemic control, is integral rather than optional [7,32].

For established periodontitis, the EFP S3-level clinical practice guideline recommends a stepwise approach. The first step introduces behaviour change, supragingival biofilm control and risk-factor modification; the second step provides subgingival instrumentation; the third addresses non-responding sites; and the fourth is supportive periodontal care to maintain stability [41]. Long-term supportive care is critical, because outcomes depend on the maintenance of low plaque levels and regular recall [31,41].

3.8 Implications for clinical practice and public oral health

Several practical implications follow from the shared basis of these diseases. First, risk assessment should be comprehensive: the same appointment can address sugar intake, plaque control, tobacco use, fluoride exposure and medical history including diabetes [7,34]. Second, behaviour change support, such as personalised oral hygiene coaching and dietary counselling, should be treated as core clinical care and not as an adjunct to operative treatment [34,41]. Third, patients should be enrolled in risk-based recall intervals that provide long-term preventive and supportive care rather than episodic, symptom-driven visits [31,34].

At the population level, upstream policies, including fluoride availability, restrictions on marketing and taxation of sugar-sweetened products, tobacco control and integration of oral health into general non-communicable disease programmes, are likely to deliver larger and more equitable gains than clinical services alone [2,3,6]. For South Africa and comparable settings, this argues for strengthening prevention within primary care, using low-cost evidence-based interventions and task-sharing where appropriate [2,13].

3.9 Limitations

This review has several limitations. As a narrative review, it is subject to selection bias, and no formal risk-of-bias assessment or meta-analysis was undertaken. Restriction to English-language publications may have omitted relevant regional evidence. Much of the available evidence on the interaction between caries and periodontal diseases is observational, which limits causal inference, and South African data specific to both conditions are limited. Future research should evaluate integrated preventive pathways in primary care and public health settings, and the cost-effectiveness of common risk factor interventions in low-resource contexts.

4 CONCLUSION

Dental caries and periodontal diseases are highly prevalent, largely preventable, biofilm-mediated diseases that share behavioural, social and systemic determinants. Advances in understanding oral dysbiosis have moved emphasis from eliminating specific organisms towards modifying the environmental conditions that drive disease. Effective care therefore combines fluoride use, sugar reduction, meticulous biofilm control, tobacco cessation, management of systemic risk factors, and minimally invasive treatment, delivered within long-term preventive relationships. Adopting an integrated common risk factor approach in daily practice and health policy offers the greatest potential to reduce the burden of both conditions, especially in under-resourced communities.

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