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Review Article

Association Between Obesity and Dental Caries: A Narrative Review of Shared Etiological Factors

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Abstract:
Obesity and dental caries are two of the most common chronic health problems affecting people of all age groups across the world. In recent years, increasing attention has been given to the possible relationship between body weight and oral health. Although obesity and dental caries are distinct conditions, they share several common risk factors, including unhealthy dietary habits, frequent consumption of sugary foods and beverages, physical inactivity, and poor lifestyle practices. Obesity may further compromise oral health by altering salivary composition, increasing inflammation, and negatively influencing routine health behaviours. Many studies have explored this association; however, the findings remain controversial, with some reporting a positive relationship while others show little or no association. This review article aims to discuss the link between obesity and dental caries, explain the possible mechanisms linking the two conditions, and emphasise preventive strategies based on a common risk-factor approach.
Keywords: Obesity, Dental Caries, Sugar Consumption, Oral Health, Body Mass Index, Common Risk Factor Approach**.**

Introduction

Obesity has evolved into a major global health threat, reaching epidemic proportions across both developed and developing nations (1). Obesity is defined as the accumulation of fat in adipose tissue to such an extent that it deteriorates the quality of life of the affected individual. Childhood obesity has shown a positive association with general physical,

dental, and mental health. It is also associated with many other comorbidities (2, 3). Recognised by the World Health Organisation (WHO) as a major public health concern, obesity is currently considered one of the leading causes of mortality worldwide (4,5). Obesity is a multifactorial disease associated with dietary habits, sedentary lifestyles, socio-economic factors, and genetic and environmental factors (6,7). Since diet is the only modifiable factor for both obesity and dental caries, efforts have been taken to use it for intervention and control of both obesity and dental caries. Childhood obesity is very likely to progress into adulthood, and parameters such as Body Mass Index (BMI), Bioelectric Impedance Analysis (BIA), and anthropometric measurements such as waist circumference and body fat ratio are used in the analysis. BMI has been extensively used in screening and epidemiological surveys and has yielded reliable results for categorising obesity. BMI is defined as a body fat measurement based on an individual's height and weight (8). BMI calculators are readily available on online platforms and can be accessed freely. Nowadays, body fat percentage is considered more reliable for categorising males and females as obese or non-obese (9).

Chronic conditions like diabetes and cardiovascular diseases, which are closely related to obesity, are the leading causes of death worldwide. (10-14). Type II diabetes is significantly related to obesity, and both are serious public health threats according to the latest reports by the WHO in 2021 & 2022. (15). Metabolic disorders, hyperlipidemia, insulin resistance, certain types of cancers, infertility issues, osteoarthritis, along with dental caries and xerostomia, are closely related to obesity. (16-19). Salivary glands are severely affected by obesity, compromising salivary flow, and enzymes alter the oral microflora, thereby leading to increased dental caries due to reduced salivary flow and increased microflora (20-23). In obese people, decreased salivary gland weight reduces the concentrations of sialic acids, phosphoperoxidases, and even salivary immunoglobulins, which are positively correlated with increased dental caries. (24)

It was in 1634 that the term dental caries was first used in the literature. The term dental caries is a symbolic term for holes in the teeth (25). Dental caries is a prime cause of oral pain. It is the destruction and dissolution of dental hard tissues due to a disruption of the equilibrium between acidogenic bacteria and sugar substitutes, mainly sucrose. It can initially appear as a white spot lesion, which can progress to deep cavitation and cause severe oral and dental pain.

The primary causative agent of dental caries is Streptococcus mutans, a gram-positive coccus and facultative anaerobe. The cariogenic streptococcal strains are not found when there are no primary teeth, nor after the extraction of all the permanent teeth.

The primary causative agents of dental caries are the Streptococcus group of microorganisms and Lactobacillus, except Lactobacillus fermentum and Lactobacillus lactis. The major streptococcal strains implicated in dental caries are Streptococcus salivarius, Streptococcus mitis, Streptococcus oralis, and Streptococcus sanguis.

Table 1. Localisation of Cariogenic Microflora

Type of Caries	**Predominant Microorganisms**
Pit and fissure caries	Streptococcus mutans, Streptococcus sanguinis, Lactobacillus spp., Actinomyces spp.
Smooth surface caries	Streptococcus mutans, Streptococcus salivarius
Root caries	Actinomyces viscosus, Actinomyces naeslundii, Streptococcus mutans, Streptococcus sanguinis
Deep dentinal caries	Lactobacillus spp., Actinomyces naeslundii

Table 2. Types of Dental Caries

Type of Caries	Description
Incipient caries / Primary caries	A carious lesion is present on a tooth surface with no previous history of decay or restoration.
Recurrent / Secondary caries	Caries occurring adjacent to an existing restoration or previously treated tooth surface.
Arrested caries	A non-progressive lesion in which demineralisation has ceased due to remineralisation.

Table 3. Classification of Dental Caries Based on Location (G.V. Black)

Classification	Description
Class I	Caries involving the pits and fissures of teeth.
Class II	Carious lesions involving the proximal surfaces of posterior teeth.
Class III	Caries involving the proximal surfaces of anterior teeth without extension to the incisal edge.
Class IV	Caries involving the proximal surfaces of anterior teeth with extension to the incisal edge.
Class V	Lesions located on the cervical (gingival) third of the facial or lingual surfaces of teeth.
Class VI	Lesions involving the incisal edges of anterior teeth or cusp tips of posterior teeth.

According to the World Health Organisation (WHO) 2024 report, obesity has emerged as a major global public health concern. In 2022, approximately one out of every eight individuals worldwide was affected by obesity. The prevalence of obesity among adults has doubled over the years, while adolescent obesity has increased nearly fourfold. Globally, around 43% of adults aged 18 years and above were classified as overweight, of which nearly 16% were obese. In addition, a significant proportion of children were affected, with approximately 30% of children under 5 years of age reported to be overweight worldwide.

Table 4. WHO Classification of Obesity

BMI Category	Body Mass Index (kg/m²)	Risk Level
Underweight	< 18.5	Low
Healthy weight	18.5–24.9	Average
Overweight	≥ 25.0	Increased
Pre-obesity	25.0–29.9	Increased
Obesity Class I	30.0–34.9	Moderate
Obesity Class II	35.0–39.9	Severe
Obesity Class III	≥ 40.0	Very severe

Note: BMI = Body Mass Index; kg/m² = kilograms per square meter.

Etiopathology of dental caries

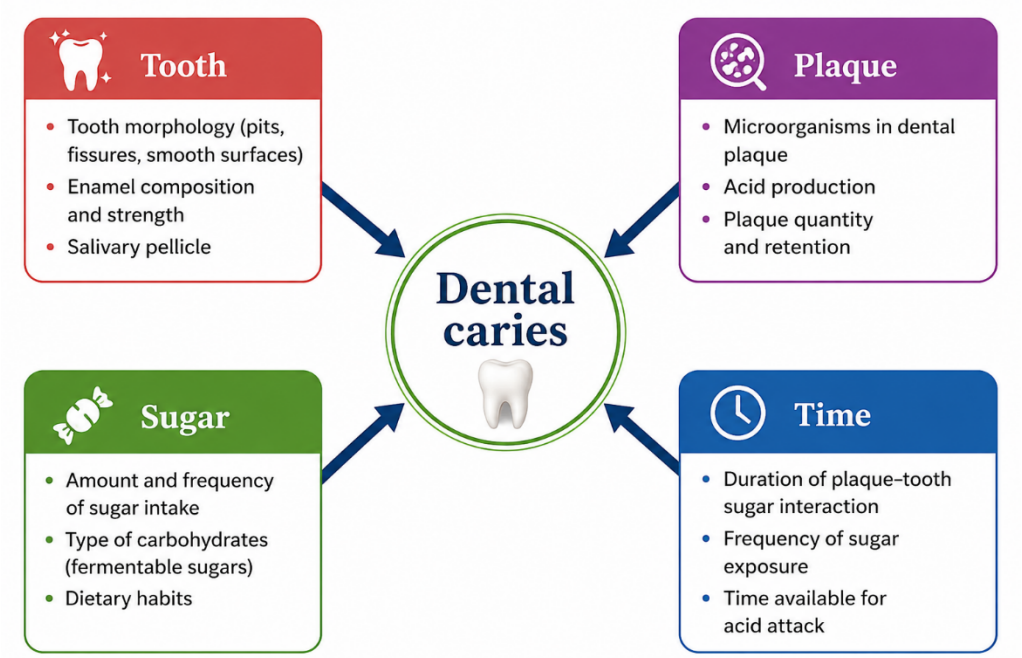

Figure 1. Dental caries is a multifactorial disease of the oral cavity that affects almost everyone at some point in their lifetime.

Dental Plaque

Dental plaque is the primary niche for cariogenic microorganisms and forms a structured biofilm on the tooth surface, harbouring numerous microbial species. It has been reported that one gram of dental plaque may contain approximately 10^{11} bacteria (30). In addition to microorganisms, dental plaque also contains food debris and viruses (31,32). Frequent consumption of sticky and sugary foods promotes plaque accumulation and increases bacterial concentration, which may subsequently mineralise into calculus that is difficult to remove by routine brushing. Increased sugar intake enhances bacterial growth, as oral microorganisms utilise fermentable carbohydrates for energy production. Along with Streptococcus spp., other bacteria such as Prevotella spp. and Veillonella spp. have also been associated with the cariogenic process (33–36). However, certain studies have suggested that dental caries may progress even in the absence of Streptococcus mutans (37).

Deep pits and fissures on tooth surfaces favour the retention of food particles and microorganisms, making plaque removal difficult and thereby accelerating the cariogenic process. Newly erupted teeth are particularly susceptible to dental caries because of incomplete enamel maturation and reduced mineralisation.

Frequent consumption of fermentable carbohydrates, particularly sucrose, is considered a major etiological factor in dental caries. Repeated intake of sugary foods and beverages, especially between meals, lowers the oral pH and increases the duration of acid attacks on the tooth surface, as explained by Stephan's curve. This acidic environment promotes enamel demineralisation and enhances cariogenic activity.

The duration and frequency of exposure to sticky fermentable carbohydrates, along with the continuous interaction between tooth surfaces, microorganisms, and dietary sugars, contribute to the progression of dental caries. Over time, repeated cycles of demineralisation may lead to the transition of non-cavitated lesions into cavitated lesions, resulting in irreversible tooth destruction.

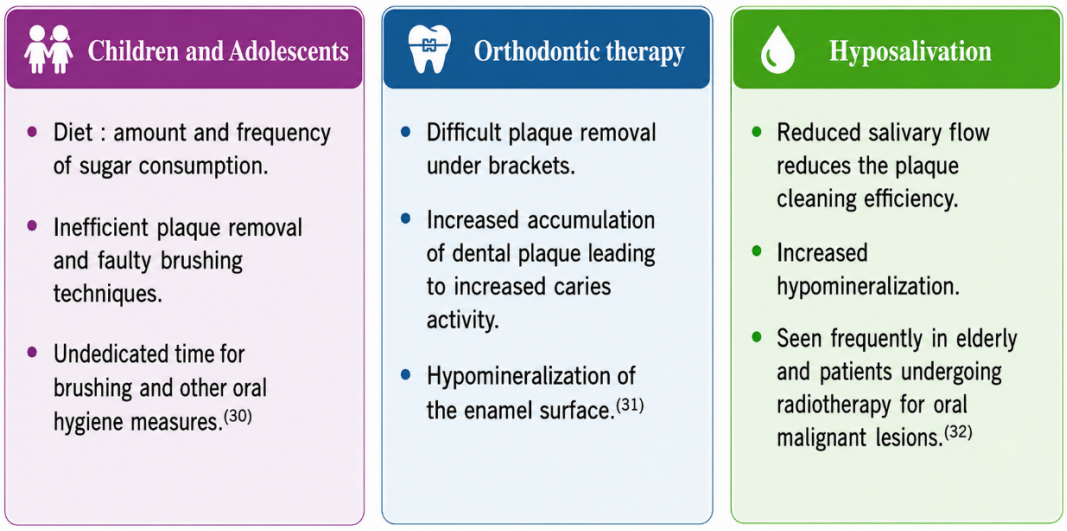

Figure 2. Factors contributing to dental caries include sugar intake, orthodontic plaque retention, and hyposalivation.

Etiopathology of Obesity

The pathogenesis of obesity is a complex, multifactorial process involving interactions among genetic, environmental, behavioural, and psychosocial factors (38). According to the World Health Organisation, 2025 data, more than 160 million children worldwide are affected by obesity (39). In India, the prevalence of obesity has increased considerably due to rapid urbanisation, increased consumption of junk and street foods, reduced physical activity, and sedentary lifestyles.

Genetic Factors: Obesity may result from several genetic influences and is broadly classified into monogenic, polygenic, and syndromic obesity. Monogenic obesity results from mutations or defects in a single gene. It is often associated with excessive appetite and obesity that begin at an early age, usually between 3 and 5 years: polygenic obesity, the most common type, results from the interaction of multiple genes with environmental factors. Syndromic obesity is associated with developmental and genetic disorders such as Prader–Willi syndrome.

Leptin–Melanocortin Pathway: Leptin is an appetite-regulating hormone secreted primarily by adipose tissue. In obesity, excessive leptin secretion may lead to leptin resistance, impairing satiety signalling. Consequently, affected individuals continue to experience hunger despite adequate energy stores, leading to excessive food intake and weight gain.

Hypothalamic Obesity: Structural or functional alterations involving the ventromedial and arcuate nuclei of the hypothalamus may contribute to hypothalamic obesity. Hypothalamic tumours and their treatment can disrupt appetite regulation, energy expenditure, and metabolic control, thereby promoting obesity.

Physical Inactivity: Physical inactivity is a major contributor to obesity. Prolonged sitting, dependence on passive modes of transport, excessive caloric intake, and lack of regular exercise contribute to metabolic dysfunction, increased insulin resistance, chronic low-grade inflammation, and reduced mitochondrial activity in muscle tissue, ultimately resulting in weight gain (40).

Drug-Induced Obesity: Certain medications, including antidepressants, antidiabetic drugs, antipsychotics, and corticosteroids, may contribute to obesity. These drugs can alter dopamine and serotonin levels, increase insulin secretion, enhance glucose absorption, and disturb metabolic regulation, thereby promoting weight gain.

Socioeconomic Status: Obesity has been observed more frequently among individuals belonging to higher socioeconomic groups compared to lower-income populations. Factors such as sedentary lifestyle, increased consumption of high-calorie diets, and reduced physical activity contribute significantly to this trend (41).

Correlation Between Obesity and Dental Caries

Dietary Mechanism: High consumption of fermentable carbohydrates increases the availability of substrates for cariogenic microorganisms such as Streptococcus mutans and Lactobacillus spp. These microorganisms produce acids that lower the pH of the oral environment and promote enamel demineralisation. Simultaneously, diets rich in carbohydrates, sugars, and fats contribute to positive energy balance and adipose tissue accumulation, thereby increasing the risk of obesity.

Altered Salivary Flow: Childhood obesity has been associated with reduced stimulated salivary flow, which is strongly linked to dental caries (42). Saliva normally protects the oral cavity by buffering acids, maintaining oral pH, and inhibiting microbial growth. However, obese children often exhibit hyposalivation, lower salivary pH, and reduced buffering capacity (43). Obesity-induced systemic inflammation and pro-inflammatory cytokines may alter the hypothalamic–pituitary–adrenal axis, thereby affecting salivary gland function and increasing susceptibility to dental caries.

Inflammatory Response: Individuals with obesity commonly exhibit persistent low-grade systemic inflammation. Elevated levels of inflammatory mediators, particularly Tumour Necrosis Factor-alpha (TNF-α), have been strongly associated with obesity. Studies have shown that increased salivary TNF-α levels correlate with a greater number of decayed tooth surfaces, suggesting that obesity may intensify the inflammatory response associated with early childhood caries and worsen oral disease progression when both conditions coexist (44,45).

Prevention and Health Promotion

The Common Risk Factor Approach (CRFA), proposed by Aubrey Sheiham and Richard G. Watt in 2000, emphasises the importance of targeting shared behavioural and environmental determinants to prevent multiple chronic diseases simultaneously. Since obesity and dental caries share several risk factors, this integrated strategy provides an effective framework for improving both general and oral health outcomes. The approach focuses on reducing free sugar consumption, promoting healthy dietary habits, encouraging regular physical activity, improving oral hygiene practices, increasing oral health awareness, and scheduling routine dental consultations. By addressing these modifiable risk factors collectively, the CRFA can significantly reduce the burden of both obesity and dental caries at the individual and community levels (48).

Dietary counselling plays an important role in the prevention and management of dental caries and obesity. It involves limiting sugary snacks and beverages, encouraging fruit and vegetable intake, promoting balanced nutritional habits, and avoiding frequent snacking between meals. These measures help reduce acid production by cariogenic microorganisms and support overall oral and systemic health. Before initiating dietary counselling, effective control of contributing factors such as dental plaque is essential, as it enhances the overall effectiveness of preventive strategies and improves oral health outcomes (46).

Table 5. Preventive Strategies for Dental Caries

Target	Intervention
Plaque control	• Use of fluoridated toothpaste • Maintenance of regular brushing and flossing habits • Use of antimicrobial mouth rinses
Dietary habits / Sugar consumption	• Limiting the intake of sugary foods and beverages • Reduction in the frequency of sugar exposure • Stimulation of salivary flow
Tooth morphology and enamel protection	• Application of remineralising agents • Topical fluoride therapy • Professional fluoride varnish application • Placement of pit and fissure sealants

Physical Activity: The World Health Organisation recommends that children and adolescents engage in at least 60 minutes of moderate-to-vigorous physical activity daily. In comparison, adults should perform at least 150 minutes of moderate physical activity per week. Regular physical activity plays an important role in maintaining overall health, preventing obesity, and improving quality of life. Recommended activities include walking, jogging, cycling, swimming, outdoor sports, yoga, stretching exercises, and school-based physical activities (47).

Oral Health Education: Effective oral health education also plays a vital role in preventing dental diseases and promoting lifelong healthy habits. Important oral hygiene practices include brushing teeth twice daily with fluoridated toothpaste, using proper toothbrushing techniques, daily use of dental floss or other interdental cleaning aids, routine tongue cleaning, and rinsing the mouth after meals to reduce food debris accumulation. In addition, regular dental check-ups and professional oral prophylaxis are essential for maintaining optimal oral health and preventing the progression of dental diseases.

Conclusion

The rapidly increasing prevalence of obesity and dental caries highlights the urgent need to adopt a comprehensive approach that integrates general and oral health promotion. The growing dependence on processed foods and sugar-sweetened beverages has contributed significantly to the rise of both obesity and dental caries, especially among children and adolescents. Prevention of these conditions should focus on promoting healthier lifestyles from an early age. Limiting sugary foods and beverages, encouraging balanced nutrition, promoting physical activity, maintaining good oral hygiene, and attending regular dental check-ups can help reduce the risk of both obesity and dental caries. In addition, oral health education initiatives in schools and communities can play an important role in raising awareness of healthy dietary behaviours and preventive oral care practices.

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