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Biological And Social Factors That Influence Dental Caries-A Review

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Abstract

Dental caries is one of the most widespread diseases globally. Despite being highly preventable, its prevalence has significantly increased over the past 30 years. It is a complex condition arising from disruptions in the microbial balance of the dental biofilm, influenced by factors such as saliva flow and composition, fluoride exposure, dietary sugar consumption, and oral hygiene habits. Biological and social factors, such as sex, age, the oral microbiome, genetic predisposition, socioeconomic status, and diet further influence the progression and severity of dental caries. Electronic databases such as PubMed, ScienceDirect, and Google Scholar were used to extract articles for the review. The keywords used included dental caries, socioeconomic status and caries, age and caries, gender and caries, diet and caries, oral microbiome and caries, and fluoride and caries. Relevant articles were selected. This review examines these factors and their influence on the development and management of dental caries.

Keywords

Dental caries, fluoride, microbiome, age, diet

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INTRODUCTION

Dental caries continues to pose a significant public health concern worldwide, especially in lower- and middle-income nations. Its prevalence is often associated with broader social and economic inequalities, including access to dental care, underdeveloped healthcare systems, poor dietary habits, and limited awareness of effective oral hygiene routines. According to the World Health Organization, oral health issues affect around 3.5 billion people across the globe, with the majority, approximately 75%, living in middle-income nations. Caries in permanent teeth impact an estimated 2 billion individuals, while tooth decay in primary teeth affects roughly 514 million children [1]. In India, dental caries affects over half the population, with an overall prevalence of 54.16%. The condition appears more frequently in adults over 18 years, where the rate reaches 62%, compared to 52% among individuals aged 3 to 18 years [2]. Among the WHO-designated index age groups in India, the average prevalence of dental caries was 49% in children aged 5–12, 60% at age 15, 78% for those between 35–44, and 84% in the 65–74 age group. Between January 2000 and April 2016, the mean deft/dmft scores were reported as 2.36 (age 5), 1.95 (age 12), 3.31 (age 15), and 7.01 (ages 65–74), indicating a rising trend with age [3].

Dental caries is a slow-developing condition marked by the breakdown of tooth enamel due to acids produced when bacteria in the mouth ferment dietary sugars. This process reflects an imbalance in the oral environment, where biofilm activity disrupts the natural remineralization of dental tissues [4].

Biofilms exhibit dynamic microbial activity that leads to shifts in plaque pH levels. These changes stem from acid production by bacteria, which are typically moderated by the buffering capacity of saliva and the mineral content of nearby tooth structures. This creates a constant balance between demineralization and remineralization at the tooth surface. However, when pH levels fall below a critical point, the enamel, dentine, or cementum begins to lose minerals, initiating the caries process. Conversely, remineralization occurs when the pH rises. These demineralization and remineralization processes are continuous and happen frequently throughout the day. Over time, this dynamic process determines whether caries lesions develop or existing lesions are repaired and reversed [5]. It is essential to implement key prophylactic measures, including teaching proper brushing and flossing techniques, applying fluoride, reducing sugar consumption, and enhancing health education for primary caregivers. By identifying and addressing risk factors for caries development in children from lower-middle-income groups (LMICs) at an early stage, a significant multiplier effect can be achieved, leading to improved oral health outcomes.

SOCIOECONOMIC STATUS AND DENTAL CARIES

Various sociodemographic factors including age, ethnicity, gender, education level, oral health awareness, dietary patterns, parental income, and availability of dental care have been identified as significant contributors to both the prevalence of dental caries and the extent to which children access dental services. In their systematic review and meta-analysis, Costa et al. examined the link between socioeconomic status and dental caries, revealing a strong correlation between factors such as education, income, occupation, economic standing, and community index with a higher prevalence of dental caries. Adult dental caries was more severe among populations with the largest percentages of individuals with poor socioeconomic status [6]. Tanaka et al. found that a higher education level of 15 years or more for either parent was strongly associated with a reduced risk of dental caries in their children. However, no significant correlation was observed between the risk of dental caries in children and the mother's occupation or household income [7]. According to Yousaf et al. the risk of dental caries was greater in low-middle-income countries (LMIC), primarily due to high sugar content in diet, limited maternal education, and varying socioeconomic status (SES). To decrease the incidence of dental caries in these groups, they recommended measures such as reducing sugar intake, improving oral health awareness, introducing nationwide fluoride programs, and addressing sociodemographic factors [8]. Children with tooth decay had 81% lower odds of participating in oral health promotion programs (OHPP), which effectively decreased the financial burden in 97 out of 100 OHPPs. Children under 6 years of age received the maximum benefit from OHPP, resulting in decreased decayed, missing, and filled teeth scores (dmft) [9]. Children belonging to racial minority groups exhibited a greater mean difference in dmft score than children from the privileged community. The dmft scores of children belonging to minority ethnic groups in Australia were greater, followed by those from New Zealand, Britain, and the USA. The average prevalence of caries-infected teeth was 23 % greater among racially minoritized children [10]. Studies on dental caries among ethnic minority children in China have shown that cavities are more common in primary teeth than in permanent ones. However, among larger ethnic minority groups with populations exceeding one million, the median prevalence of caries in permanent teeth was found to be higher [11]. Shi *et al*. found significant differences in children's oral health across ethnic groups. Compared to White populations, minority groups such as Filipinos, Arabs, and Indigenous children were more likely to experience poorer oral health. Filipino children had nearly five times the likelihood of suffering from severe, untreated dental issues compared to White children [12].

Among the children from the Indigenous Australian community aged 5 to 10 years, the average number of decayed, missing, and filled surfaces (DMFS) was 6.4, compared to an average DMFS of 2.9 in their non-Indigenous peers [13]. Racial disparities were often associated with lower socioeconomic status and "health-damaging" cultural characteristics, including care-seeking behaviours, eating habits, and oral hygiene practices [14]. In North Carolina, 30.4% of White pupils, 39.0% of Black students, and 51.7% of Spanish students had dental caries. In schools where less than 75% of children participated in the National School Lunch Program (NSLP), a statistically significant disparity in adjusted caries experience was observed between Black and White students [15]. A cross-sectional study conducted in the United States from 2016 to 2019 examining three socioeconomic factors found that children lacking a medical home had the highest rates and risk of dental caries and untreated tooth decay. Additionally, the likelihood of children from the lowest household income experiencing tooth decay was 50% higher than those from higher income categories. Uninsured children were nearly twice as likely to miss

dental treatment as those covered by public, private, or a combination of both insurance types [16]. Younger children with an established medical home were more likely to receive preventive dental care [17].

Conditions like loss of teeth, the requirement for dental fixers, and periodontal disease were used as markers to assess oral disease burden (ODB). This was present in 86.9% of 17,560 individuals screened in Brazil, which included adolescents (31.7%), adults (34.5%), and older adults (33.9%) [18]. In Sardinian children in Italy, greater levels of socioeconomic deprivation were linked to higher rates and severity of dental caries. Caries experience was significantly higher in children aged 6–11 years than 3–5 years, indicating that caries is a cumulative condition that worsens with age. The study highlighted geographical disparities in caries prevalence across different municipalities in northern Sardinia, with areas of higher deprivation showing worse oral health outcomes. The findings emphasized the importance of a focused educational and preventive strategy to mitigate oral health disparities among Sardinian children, particularly in schools and high-deprivation areas [19]. A notable association was identified in China between socioeconomic status (SES) and both the prevalence of dental caries and the dmft index. Children from households with lower income levels and less parental education exhibited higher dmft scores compared to those from more affluent and better-educated families. Disparities in dental caries were more evident in urban settings in relation to household income, while in rural areas, parental educational background played a more significant role [20]. In China, parents with higher educational attainment are more likely to take their children to the dentist regularly, not just for addressing dental pain but also for preventive care and to gain information on maintaining oral health [21]. Preschool children exhibiting severe caries during follow-up evaluations and those not receiving dental therapy were more likely to experience a decline or a severe decline in their oral health-related quality of life. Additionally, an increase in the number of children in the home has also contributed to the significant deterioration in oral health [22].

AGE AND CARIES

Early childhood caries (ECCL) affects one or more primary teeth in children as young as 72 months. It can lead to gravitated or non-gravitated lesions, caries-related tooth loss, or restorations on teeth. ECCL continues to pose a major challenge to public health. Severe early childhood caries (S-ECCL) affects children below the age of three and is recognized as a widespread concern, with global prevalence rates estimated between 60% and 90% [23]. Among Iranian children aged 3 to 6 years, the average decay-missing-filled (DMF) index for primary teeth was recorded at 1.7. For permanent teeth, the index was 0.2 in children aged 6 to 9 years, increased to between 0.9 and 1.5 by age 12, and ranged from 3.3 to 4.8 in 9-year-olds [24]. Children of 12 years are considered the target group for the DMD index, which serves as an appropriate metric for identifying dental caries in society [25]. In a retrospective study, Moca *et al*. analysed 400 panoramic radiographs of Romanian children aged 6 to 14 to investigate the association between age and dental caries. The findings revealed that a higher number of superficial carious lesions on the first permanent molars was linked to younger chronological age. Conversely, an increased number of medium and deep carious lesions was associated with older chronological or dental age. These results suggest that the development of caries in the first permanent molars is influenced by the child's age [26].

Kale *et al*. performed a meta-analysis to assess the prevalence of dental caries among 5 to 15-year-old children in the East Mediterranean region. A pooled prevalence of 65% and 61% was reported among 5 and 12-year-olds, respectively. It was linked to refined food consumption, low parental education, low socioeconomic status, and limited access to dental care. The prevalence in the 15-year age group was 70% and was associated with poor dental hygiene habits, limited socioeconomic resources, and a diet rich in starch and sugar [27]. The incidence of dental caries in primary dentition was 80.95% in the Gulf Cooperation Council [28]. Doumit *et al.* reported a mean dmft (decayed, missing, and filled teeth) of 0.6 for ages 6 to 8, 3.42 for 12 years, and 5.44 for 15 years. DDS (decayed, filled surfaces) of > 0 was found in 1% of 1-year-olds, 4% of 2-year-olds, 12% at 3 years, 23% at 5 years, and 43% in the seven-year age group [29]. In the United States, dmft decreased from 17.96 to 1.16 in 2007. The decline in the dmft index was primarily attributed to a reduction in the number of missing teeth, despite an increase in the number of filled teeth. FT (filled teeth) and MT (missing teeth) were more prevalent in 75 years and older age group compared to those aged 65 to 74 years. Lower FT in the older population above 65 years indicated issues with dental service accessibility. Caries experience varied among ethnicities; dmft was higher in American whites because of filled teeth, while Blacks had a higher dmft due to increased MT. As income levels declined, both the number of filled teeth (FT) and missing

teeth (MT) increased, although the proportion of filled teeth showed a decrease [30].

In the US during 2011-2012, 91% of adults aged 20–64 had dental caries, and 27% had untreated tooth decay. The prevalence of caries was 94%–97 % among those aged 35-64 and 82% among those aged 20-34. Among Hispanic adults aged 20-64, the prevalence was 85%; for African Americans of non-Spanish origin, it was 86%; for Asians of non–Hispanic descent, it was 85%; and for non-Hispanic white adults, it was 94%. The prevalence of untreated dental caries was 42% among non-Hispanic black adults, 22% among non-Hispanic white adults, and 17% among Asian adults. Compared to Hispanic adults (36%), untreated tooth decay was less common among non-Hispanic white and Asian adults. The percentage of edentulous individuals was higher among non-Hispanic black adults aged 65 (29%), compared to 17% for non-Hispanic white adults and 15% for Hispanic adults [31].

GENETIC FACTORS AND CARIES SUSCEPTIBILITY

Even among individuals with similar tooth brushing frequencies or dietary patterns, the rates of dental caries can differ. In other words, individuals who brush their teeth twice or rinse their mouths after every meal are still susceptible to caries. Heredity has been linked to caries incidence since 1899 [32]. Mansbridge, in his study involving 96 monozygotic twins and 128 dizygotic twins, explained that caries experience showed greater similarity among monozygotic twins than dizygotic twins. Beyond genetic similarity, he emphasized the importance of environmental factors in caries development [33]. Goodman *et al.* reported similarities in oral microbe heritability, flow rate of saliva, pH, and activity of amylase enzyme, among 38 twins [34]. According to Lovelina *et al*. monozygotic twin pairs showed stronger correlations for dental caries, periodontal disease, and malocclusion than dizygotic twin pairs [35]. Monozygotic twins raised separately, and exposed to diverse domestic conditions, dietary patterns, and oral health practices, demonstrated 45-67% similarity in teeth retention, restored tooth structures, and presence of dental caries, providing evidence that genetic components contribute to dental status and caries experience [36]. Similarities in the oral microbiome of twins with and without caries confirm the role of familial factors in microbial colonization. The heritable nature of surface-based caries prevalence, lesion severity, and preference for sweetness among 115 pairs of twins was reported by Bret *et al*. in 2006. He also noted a genetic predisposition for the occurrence and intensity of dental caries among 388 twin pairs [37].

Genetic mutations can lead to the production of abnormal proteins or a reduced quantity of proteins during tooth development, resulting in impaired mineralization. This, in turn, may compromise enamel resistance to acidic environments or promote bacterial adhesion, thereby increasing the risk of dental caries [38] Full genome screening for dental caries aimed at identifying associated genetic loci responsible for tooth decay was conducted in 1,305 US children aged 3-12. The study revealed several loci—ACTN2, MTR, EDARADD, MPPED2, and LPO with plausible biological roles in dental caries [39]. The gene encoding Human Leukocyte Antigen (HLA) molecules is highly polymorphic and may influence individual variations in immune responses to oral microbial colonization, thereby affecting susceptibility to dental caries [38]. McCarlie *et al*. reported that individuals positive for HLA-DR4 showed reduced immune responses to *Streptococcus mutans*, including lower reactivity to antigen I/II, decreased specific secretory IgA activity relative to total IgA, and diminished reactivity to whole cells of *S. mutans* UA159, suggesting a potential link between HLA-DR4 and increased caries susceptibility [40]. According to Valarini *et al.* individuals positive for the HLA-DR2 allele were less susceptible to caries [41]. The mutant genotype of the mannose-binding lectin (MBL2) gene was observed more frequently in individuals with high caries prevalence compared to those with low caries experience [42]. Improved insight into the genetic factors influencing caries development can aid in prevention by mitigating risk factors. This, in turn, will reduce patient suffering and save time and money.

SALIVA AND CARIES

Saliva is a multifaceted fluid that contains electrolytes, proteins, organic compounds, metabolites, and debris from oral microorganisms. It serves several functions, including moistening oral tissues, cleaning the gums and teeth, and assisting with speaking and swallowing. Additionally, saliva plays key roles in buffering the oral cavity, remineralizing teeth, providing antimicrobial protection, supporting tissue repair, and aiding in taste and digestion [43]. The composition of saliva is influenced by age, sex, health, and duration of the day. It protects the teeth by diluting and eliminating sugars, utilizing its buffering capacity, balancing demineralization/remineralization, and exerting its antimicrobial action [44].

Increased dental caries in older adults over 65 years was linked to a reduced stimulated salivary flow rate (≤ 0.6 milliliters/minute). Conversely, decreased caries lesion in children and adolescents was

associated with low buffering capacity and thick, sticky, or frothy salivary consistency. Stimulating salivary flow by chewing sugar-free gum after meals reduced caries prevalence [45]. Mucins make up approximately 20–30% of total salivary proteins and are classified into two main types: MG-1 (MUC5) and MG-2 (MUC7). People with high susceptibility to caries showed an increase in MG-1, while MG-2 was noted in patients with low caries intensity [46]. The role of salivary IL-17, TGF-β, and IgA in caries was studied by Nawaz *et al* [47]. IgA quantity in saliva was elevated in caries patients, whereas IL-17 and TGF-β levels were decreased compared to healthy individuals. Salivary alpha-amylase hydrolyses the α bonds in huge insoluble polysaccharides into soluble forms, with maltose as an end product. It has binding sites for the enamel surface of teeth as well as oral microorganisms. Monea *et al*. observed an increased quantity of salivary alpha-amylase in children with active dental caries. [48]. Picco *et al*. found higher salivary carbonic anhydrase VI activity in caries affected children [49].

Pyati *et al*. investigated various salivary parameters including flow rate, pH, buffering capacity, total protein, malondialdehyde (MDA), and total antioxidant capacity (TAC)—in children with and without caries. It was revealed that caries-active children exhibited significantly lower salivary flow rate, pH, and buffering capacity, while showing elevated levels of total protein, MDA, and TAC compared to their caries-free counterparts. The IgA antibody in saliva neutralizes toxins and viruses and prevents the growth and colonization of microorganisms on tooth surface [50]. Soesilawati *et al*. reported significantly higher salivary IgA levels in children with low caries activity compared to those with active caries. In adults, the presence of salivary proline-rich phosphoproteins-1/2 (APRP-1/2) and *Lactobacillus* species was examined in individuals with severe and minimal caries. APRP-1/2 plays a role in binding oral bacteria and facilitating their colonization on dental surfaces. Interestingly, *Lactobacillus* spp. was absent in approximately one-third of individuals from both the severe and minimal caries groups [51] Salivary APRP-1/2 protein content was double in the severe caries group compared to the minimal caries group, as noted by Szkaradkiewicz-Karpinska *et al* [52].

ORAL MICROBIOME AND CARIES

Being the gateway to the digestive system, the buccal cavity plays a significant role in regulating human health due to its exposure to diet and external stress. Factors such as oxidative stress, acidic pH, and variations in nutrition can alter the interactions among bacteria in the oral cavity. This dysbiosis can be exacerbated by poor dental health and unfavourable host genetics. A recent analysis of the oral microbiome identified a total of 1,591 microbial species, encompassing bacteria, fungi, archaea, viruses, and protozoa [53]. The diversity of the oral microbiome is ranked second only to that of the colon. In healthy individuals, the oral microbiome remains stable for seven years. Dental caries can develop from a disruption in the oral microbiota, driven by complex interactions among the host, diet, and microbial community. Among various factors, fermentable carbohydrates in the diet are key contributors to dental caries. When carbohydrate levels rise in the oral cavity, the pH of the dental biofilm can drop to 4 or lower. This acidic environment promotes enamel demineralization, resulting in mineral loss, the formation of white spot lesions, cavities, pulp infections, and potentially tooth loss [55]. Following birth, bacteria from the genera *Rothia*, *Neisseria*, and *Haemophilus* are among the initial colonizers of a healthy oral cavity. In contrast, children with dental caries often harbor *Prevotella* spp., *Streptococcus mutans*, and the Epstein-Barr virus [55]. According to Chen *et al*. microorganisms associated with childhood caries include *Veillonella parvula*, *Fusobacterium nucleatum*, *Prevotella denticola*, and *Leptotrichia wadei*. Metagenomic analyses have further identified caries-associated species such as *Leptotrichia buccalis*, *V. parvula*, *Streptococcus gordonii*, *Actinomyces gerencseriae*, *Parascardovia denticolens*, *Hallella multisaccharivorax*, and *Propionibacterium acidifaciens*. *Streptococcus sobrinus* and *S. mutans* are commonly recognized as the primary cariogenic agents. In contrast to *S. mutans, S. sobrinus* exhibits greater acidogenicity and aciduricity, but it is less adaptable to the biofilm environment [56].

Candida species represent a significant secondary cariogenic agent. They synthesize short-chain carboxylic acids and proteinases, adhere to non-living surfaces, and form biofilms, as noted by Sivamaruthi [57]. Among the various microbes associated with caries, *S. mutans* has been extensively studied. *S. mutans* is at the core of the three-dimensional (3D) spherical biofilm found in caries lesions involving primary teeth, while other bacteria form the outer layer. The virulence factors of *S. mutans* can be classified into four major categories: extracellular polymeric substance (EPS) synthesis, adhesion, acid generation, and acid resistance. A significant factor in the pathogenicity of *S. mutans* is its ability to produce extracellular polymeric substances (EPS), which serve as the

foundation for the biofilm. EPS consists of glucans, lipoteichoic acid, extracellular proteins, and DNA [58]. Glucan functions as a binding site for bacteria. EPS acts as a chemical and physical barrier, aiding in evading immune responses, antimicrobial activity, fluid stress, complement activity, and immune cell invasion. The sucrose-independent pathway is essential for initiating adhesion, which is then enhanced by the sucrose-dependent pathway that stimulates glucan synthesis, ultimately promoting biofilm formation [59]. EPS maintains a low pH in the oral cavity, which fosters a caries-inducing biofilm community. Positively charged chlorhexidine has poor penetration into the biofilm. Acid resistance is mediated by the agmatine deiminase enzyme of *S. mutans*, which produces an alkali that counteracts acid stress. Proton accumulation on bacterial surfaces enveloped by EPS is vital for acid resistance [60].

Yama *et al.* explored the recurrence of oral disease following dental treatment and linked it to an imbalance in the oral microbiome, which persists even after therapy. Even after treatment, the salivary microbiome of the diseased group remained distinct from that of healthy individuals [61]. Probiotics are viable microbes that offer beneficial impacts on host health when consumed in adequate quantities. While their beneficial role in the gut is established, their impact on dental caries, oral mucositis, and halitosis is being investigated [62]. Probiotics such as *Lacticaseibacillus paracasei* and *Lactiplantibacillus plantarum*, contribute to microbial balance in the oral cavity and decrease the quantity of *S. mutans* in dental plaque and saliva [63]. *Limosilactobacillus reuteri* offers strong acid-base stability, while Bifidobacteria spp. act as effective probiotics in the prevention and treatment of dental caries. Yogurt containing Bifidobacterium DN-173010 has been shown to significantly reduce the levels of Streptococcus mutans [64].

DIET AND CARIES

Sugars in the diet play a crucial role in the initiation of dental caries. When carbohydrates are broken down by microorganisms in the mouth, they produce acids that decrease the pH level to below 5.5. This drop in pH facilitates enamel demineralization, which can eventually result in tooth decay. Among the different types of sugars found in foods, sucrose is recognized as the most cariogenic, posing a greater risk than other sugars such as glucose, fructose, lactose, or maltose. Natural sugars rich in polyphenols, calcium, fiber, and water are less cariogenic than added sugars. Natural sugars are those found in fresh fruits, vegetables, milk, and other dairy products. The high calcium and casein content in cow's milk protects teeth from dental caries [65].

A study conducted among 3-year-old school children from low socioeconomic backgrounds who consumed sugar 4 to 5 times per day, was 4.7 times more prone to caries over one year than children with low sugar consumption of once per day. In a cross-sectional study, young adults with high caries activity exhibited poor dietary habits, characterized by a high consumption of sweet foods and drinks, along with inadequate consumption of fruits, vegetables, and dietary fiber [66]. Chaffee *et al*. reported the highest prevalence of ECC and dmft in children who consumed the sugary items at 6 and 12 months of age [67].

A diet high in sugar promotes the colonization of *Streptococcus mutans*, a key bacterial species strongly associated with an increased risk of developing dental caries in the future. Sucrose fermentation by oral microbes leads to the formation of a polysaccharide matrix, and acid production results in an acidic pH that Favors biofilm formation and excessive demineralization over remineralization. Longitudinal studies proved an association between sugar intake during the first year of life and colonization by cariogenic microbiota, as well as the occurrence of dental caries in subsequent years [68]. Dental caries and the mean dmft index were higher among high-sugar consumers in different age groups of 4, 15, and 18 years studied than among low-sugar consumers [69].

Ahmed *et al*. conducted a study in Egypt to explore the relationship between poor dietary habits and the occurrence of dental caries in children. They utilized the dmft index to evaluate caries in permanent teeth, the dmft index for primary teeth, and the deft index where *d* stands for decayed teeth requiring fillings, *e* for decayed teeth recommended for extraction, and *f* for filled teeth for those with mixed dentition. The study revealed that 93.7% of the participants had dental caries, and 66.9% were classified as overweight. Most of the children with a dmft score above 4 were obese males who followed an unhealthy lifestyle, consuming foods such as meat, sweetened cereals and milk, ice cream, sugary drinks, granulated sugar, Halawa or honey, desserts, fast food, and caffeinated beverages. Interestingly, the group with a dmft score higher than 4 included a greater proportion of obese females. They consumed similar foods to the dmft >4 group [70].

Snacking at night should be avoided, since saliva production decreases at night. Caries-preventive products should be prioritized in the diet over those that induce caries. One such product is the protein

found in cow's milk. The protein in cow's milk reduces the attachment of caries-inducing bacteria to hydroxyapatite, which in turn lowers sugar metabolism and decreases plaque mass. Additionally, lactose in milk is fermented more slowly than sucrose, preventing pH from dropping to levels that promote caries. The calcium and phosphate in milk aid in remineralization [71]. Hard cheese is another caries-protective food that provides a rich source of calcium, supporting enamel remineralization. Foods high in arginine, including nuts, soy, tuna, and some vegetables, possess the ability to increase pH levels. Polyphenols in vegetables, fruits, coffee, and tea inhibit glucosyltransferase activity, reduce bacterial adherence and acid production, and impair salivary amylase activity, making glucose less accessible to oral bacteria [72]. In a two-year follow-up study involving children aged 3 to 5 years, Lim *et al*. found a strong association between the intake of soft drinks and the emergence of new cavitated dental lesions. To promote oral health and lower the risk of caries, it is advisable to reduce consumption of sugars and organic acids, follow a balanced, fibre rich diet, and avoid frequent snacking between meals [73]

FLUORIDE AND CARIES

The biomineralization of teeth is an intricate process that continues throughout a person's lifetime. This process is affected by various factors, including sugar intake, plaque formation, buffering activity and saliva flow rate, patient education, antibacterial agents, and fluoride. Fluoride reaches the teeth in two main forms: systemic and topical. Systemic form is an ingestible form obtained through the fluoridation of water, diet, or fluoride supplements. Systemic fluorides integrate with tooth elements and structures during tooth formation before eruption. Topical fluorides are available as varnish, gels, mouth rinses, and toothpaste. Both forms of fluoride convert hydroxyapatite into fluorapatite, which is resistant to caries.

Fluoride varnishes (FVs) are applied by dental professionals to the teeth two to four times annually depending on a child's caries risk assessment. They consist of resin, alcohol, and sodium fluoride. Varnish adheres to tooth surfaces for an extended period, releasing fluoride efficiently and effectively. Light-curable fluoride varnish (LCFV), which serves as a protective coating for both dentin and enamel surfaces, has demonstrated greater durability and longer-lasting effects compared to traditional fluoride varnishes [74]. Additionally, light-curable resin-modified glass ionomer (RMGI) varnish has been used to manage tooth sensitivity and to seal newly erupted or partially erupted teeth. It released calcium and phosphate for remineralization and was superior to FV [75]. In a two-year follow-up study, RMGI outperformed FV in preventing occlusal caries in children [76]. Applying resin sealant together with fluoride varnish was found to be more effective in preventing occlusal caries than using fluoride varnish alone [77].

For primary prevention of caries, the most effective approach was to use normal-strength fluoride toothpaste for up to 8 years, followed by 5000 ppm fluoride toothpaste with FV twice a year. Silver diamine fluoride contained double the fluoride concentration found in FV, in addition to silver ions and ammonia. Its fluoride diffused more effectively into enamel and dentine; silver acted as an antibacterial agent, while ammonia served as an antiseptic [79]. A meta-analysis involving 133 trials revealed a 26% reduction in caries due to topical fluoride application on permanent dentition, while five studies on primary dentition showed a 33% reduction in dmft [80]. A Cochrane meta-analysis of 107 studies conducted in the USA reported that water fluoridation led to a 35% decrease in caries occurrence in primary teeth and a 26% reduction in permanent teeth [81]. In Taiwan, the government implemented Professionally Applied Topical Fluoride Applications (PTFA) with the assistance of dentists for children aged 0 to 14 years, administered twice a year from 2008 to 2021. The implementation of PTFA led to a reduction in outpatient visits per patient for dental caries. Among children aged 0–4 years, the average visits dropped from 2.66 in 2008 to 1.90 in 2021. In the 5–9 age group, visits decreased from 2.32 to 2.23, while among those aged 10–14, the rate declined from 1.85 to 1.70 over the same period [82]. Melough *et al*. found that although dietary added sugars were associated with an increased risk of dental caries, exposure to optimal fluoride levels in household water moderated this effect, leading to a reduced incidence of caries in children's primary teeth [83].

GENDER AND CARIES

Similar to several other health conditions, females tend to be more vulnerable to dental caries than males, owing to both behavioral and biological factors. These include variations in saliva composition and flow, elevated estrogen levels, and hormonal fluctuations. As reported by Aslant Ceylan, girls exhibited a higher prevalence of caries in permanent teeth, whereas boys had a greater incidence in primary (milk) teeth [84]. Women with elevated estrogen levels are more prone to caries than men with low estrogen levels [85]. Elevated

estrogen levels during pregnancy have been linked to an increased risk of dental caries. Fluctuations in estrogen can affect the composition of saliva and diminish its buffering capacity, making it less effective at neutralizing the bacteria responsible for caries [86]. Research on skeletal remains from medieval cemeteries in London revealed a significant correlation between the prevalence of dental caries and sex, with females exhibiting a higher frequency of caries across all age groups. Sex determination in these studies was primarily based on distinct skeletal features of the pelvis and skull, which are considered the most reliable indicators. The presence of at least one carious lesion on a tooth, regardless of severity, was scored as positive for caries [87]. According to Wang *et al.* the prevalence of caries and caries scores were found to be comparable between males and females in the age groups of 0–6 years, 14–18 years, and those over 18. However, within the mixed dentition group, males exhibited higher caries scores in their primary teeth compared to females [88]. Dye *et al*. reported only a slight difference in the prevalence of edentulism, with 18% of men and 19% of women affected [31].

CONCLUSION

This review supports that dental caries is influenced by a range of factors including socioeconomic status, age, genetic constitution, saliva features, nature of oral microbiome, diet composition, fluoride exposure, and gender. A deeper understanding of how these factors influence the development and progression of dental caries can contribute to more effective prevention strategies and improved disease management.

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