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Excess dietary sugar and its impact on periodontal inflammation: a narrative review

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INTRODUCTION: Sugar is omnipresent in the current food environment and sugar consumption has drastically risen over the past century. Extensive evidence highlights the negative health consequences of consuming excess dietary sugars, leading the World Health Organization (WHO) and the American Heart Association (AHA) to devise guidelines to restrict sugar intake. According to the WHO's Global Oral Health Status Report of 2022, oral diseases and severe periodontitis are a massive public health problem, and dietary sugars are a modifiable risk factor.

METHODS: We conducted a literature review using key databases to summarise the health effects of excessive sugar consumption and their potential role in periodontal inflammation.

RESULTS AND CONCLUSION: Available evidence suggests that excess dietary fructose and sucrose can cause low-grade systemic inflammation; and induce dysbiosis in both gut and the oral microbiota. Also, dietary sugar is potentially addictive and hypercaloric and its overconsumption can lead to obesity, metabolic syndrome, and other risk factors for periodontal inflammation. Hence, an unbalanced diet with excess dietary sugars holds the potential to initiate and aggravate periodontal inflammation. In the modern food environment that enables and facilitates a high-sugar diet, adopting a diverse diet and restricting sugar intake according to WHO and AHA guidelines seem beneficial to systemic and periodontal health. Since clinical evidence is limited, future research should study the effectiveness of dietary interventions that control sugar consumption in preventing and managing the global public health problem of periodontal inflammation.

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INTRODUCTION

Optimal nutrition through a diverse and well-balanced diet is vital to human health. The chronic non-communicable disease (NCD) burden of modern times is largely linked to suboptimal diet and nutrition [1]. Carbohydrates are one of the major macronutrients integral to an optimal diet and their impact on health and disease has been discussed extensively in the scientific literature [2, 3]. Dietary sugars—a widely consumed and important source of carbohydrates—have recently received attention for their impact on human health and disease.

Sugars are simple carbohydrates and can be monosaccharides, disaccharides, or sugar alcohols [4]. Glucose and fructose are the most common monosaccharides. Both glucose and fructose have the same chemistry— $C_6H_{12}O_6$ —but differ in structure [5, 6]. These monosaccharides naturally occur in fruits in high quantities. Often, fructose is considered the sweetest of the simple sugars (depending on the form of consumption) with a higher relative sweetness over other mono and disaccharides [4, 7–9]. Sucrose is the most common disaccharide predominant in cane and beet sugar and is broken down to its constituent monosaccharides—glucose and fructose—in the intestinal mucosa [10]. Glucose can be readily used by all cells for energy. Dietary fructose is metabolised in the liver, which is either converted to lactate (for oxidation in tissues) or glucose (for use or storage) [11, 12].

In prehistoric times, natural sweetness was only found in energy and nutrient-rich food sources (fruit, dates, and honey). Hence, the pleasant taste of sweetness means an easily digestible, instant source of energy and an absence of harmful toxins. This led to a ubiquitous preference for sweet taste among humans [13, 14]. Sugar was first extracted from cane juice as granular crystals in India around 500 BC [15]. The Industrial Revolution improved the purity of sugar and the efficiency of sugar production, making it a household commodity. Since then, the worldwide consumption of sugar has skyrocketed and continues in the 21st century [16]. In the modern world, sugar is omnipresent in the form of ultra-processed food and sugar-sweetened beverages [17]. They are widely used to enhance the palatability of processed and packaged food and extend its shelf life. An average American consumes 17 teaspoons (approximately 68 grams) of added sugar daily [18, 19]. India is the highest sugar consumer in the world where an average person consumes approximately 18 kilograms of sugar per year [20, 21]. A 2019 study found that in approximately 60% of the population of the United Kingdom, the total energy from free-sugar intake was 12.4% [22].

Consumption of a diet high in sugars is linked to systemic inflammation, gut dysbiosis, metabolic syndrome, and numerous chronic non-communicable diseases (NCDs) of the modern era [23–27]. The American Heart Association (AHA) recommends restricting the consumption of added sugars (sugars added during

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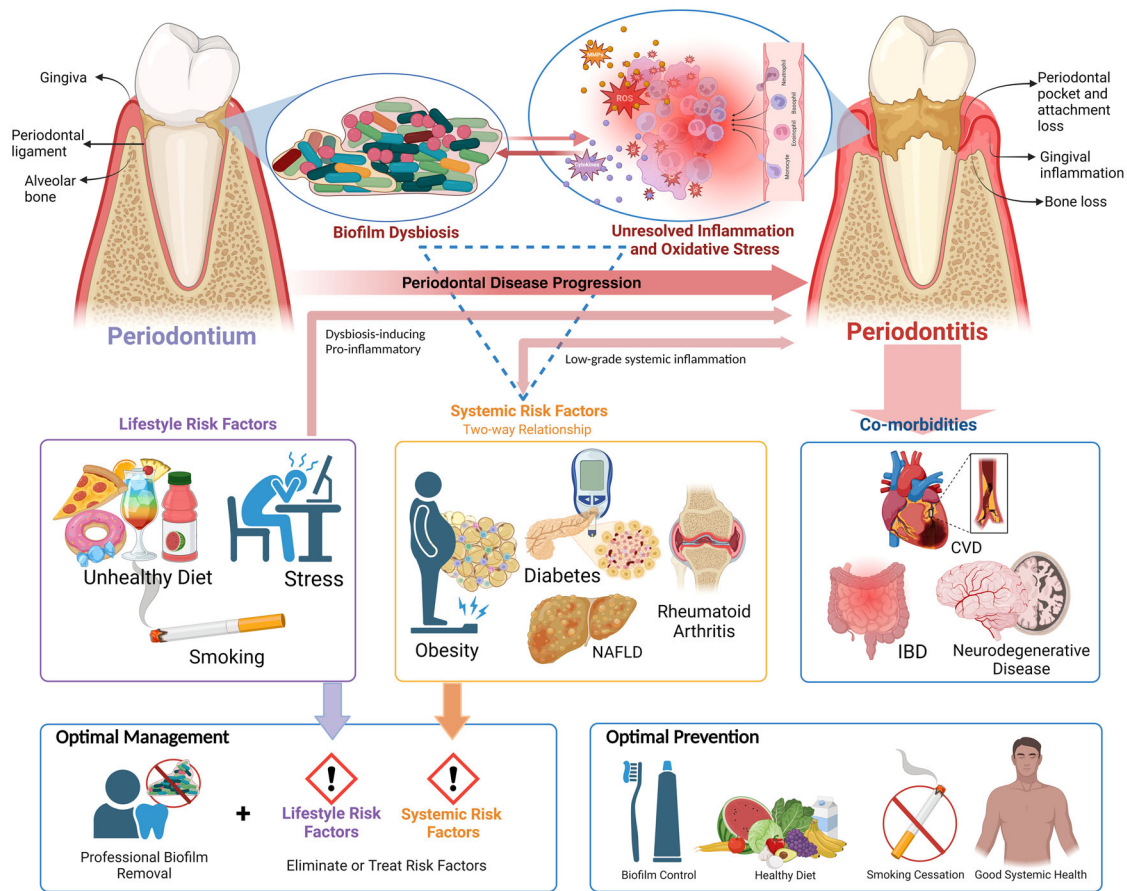


Fig. 1 Periodontal inflammation: an overview. The periodontium is the structural apparatus that provides support and anchorage to the tooth. Dysbiosis of the biofilm that accumulates on the tooth elicits an altered immune response in the form of a non-resolving hyperinflammation. Prolonged inflammation and oxidative stress (due to neutrophil oxidative killing) in the periodontium leads to tissue damage and destruction that manifests as periodontitis (pocket formation, attachment loss, and bone loss); gradually the tooth loses support and if inflammation is untreated, results in tooth loss. Periodontitis is influenced by dysbiosis-inducing and pro-inflammatory lifestyle risk factors (unhealthy diet, stress, smoking) and systemic risk factors like obesity, diabetes, rheumatoid arthritis, and non-alcoholic fatty liver disease (NAFLD); periodontitis and systemic risk factors have a two-way relationship through low-grade systemic inflammation. Periodontitis also results in extra-oral co-morbidities such as cardiovascular disease (CVD), inflammatory bowel disease (IBD), and neurodegenerative disease. The optimal management of periodontitis should focus on the removal of dysbiotic biofilm as well as the elimination of dysbiosis-inducing and pro-inflammatory lifestyle and systemic risk factors. Along with daily biofilm control through brushing and flossing, the optimal prevention strategy should also advocate for a healthy diet, smoking cessation, and a healthy lifestyle with good systemic health. (Created in BioRender. Biorender, P. (2024) BioRender.com/x62x671).

processing and preparation of food) to less than 36 grams/day for men and to less than 25 grams/day for women [4, 28]. The 2015 World Health Organization (WHO) guideline recommends limiting the intake of free sugars (i.e. total added sugars plus the inherent natural sugars of the food) throughout life and limiting free sugars to less than 10% of the total energy consumption [4, 29].

Oral diseases (a collective term representing tooth decay, periodontitis, tooth loss, and oral cancer) are the most common diseases that affect humans and a global public health problem affecting around 3.5 billion people across the world [30, 31]. The WHO's Global Oral Health Status Report of 2022 puts the gravity of the problem into perspective; the global prevalence of oral diseases is greater than the combined global prevalence of the five major NCDs (neurodegenerative diseases, type-2 diabetes mellitus, cardiovascular disease, chronic respiratory diseases, and cancer) by a billion cases [31]. The highest number of cases of oral diseases (excluding cancers) are in the Southeast Asian region of the world which is the highest consumer of sugar globally [31, 32]. High sugar consumption, along with tobacco and alcohol abuse, are key modifiable risk factors for preventing oral diseases [30, 31].

Oral diseases and chronic NCDs share several common risk factors and sugar consumption is of particular concern [30, 31]. It has been proposed to redefine the current non-communicable disease framework by including oral disease as an NCD and sugar as a common risk factor for NCDs [24]; the proposed 6×6 approach consists of six NCDs (oral diseases, mental disorders and conditions, cardiovascular disease, diabetes, cancer, and chronic respiratory diseases) with six common risk factors (sugars, air pollution, tobacco, alcohol, unhealthy diet, and sedentary lifestyle) [24].

Severe periodontitis has a global prevalence of 19%, with a billion cases worldwide in 2019 [31]. There is mounting evidence that a high-sugar, ultra-processed diet increases the risk of periodontal inflammation [33–35]. Periodontitis is the inflammation of tooth-anchoring tissues (the periodontium) due to dysbiosis of the periodontal microbiota, leading to the gradual loss of tooth support (Fig. 1). Periodontal health is inextricably linked to systemic health and several lifestyle factors play an important yet underestimated role in periodontitis [36–38]. Among them, the role of dietary sugars in periodontal inflammation has not been discussed extensively in the literature. Hence,

this review aims to summarise the effects of consuming excess dietary sugars (fructose and sucrose) on periodontal inflammation and its relevance in preventing and managing the global public health problem of periodontitis.

METHODS

We adopted a non-systematic, narrative approach to review the existing literature on the health consequences of consuming excess dietary sugars and their potential role in periodontal inflammation. We used the PubMed and SCOPUS databases to search with the keywords “Sugar” OR “Fructose” OR “Sucrose” AND “Inflammation” OR “Periodontal Inflammation” OR “Dysbiosis” OR “Periodontitis” OR “Health”. We summarised the plausible mechanisms relating to excess dietary sugars and systemic and periodontal inflammation using relevant articles.

Sugar and inflammation

Inflammation is a host defence response against a wide range of noxious stimuli. It is mounted by the immune system, which holds a diverse army of cells and proteins intricately connected in a complex network of physiological and biochemical processes, functioning to protect the host [39, 40]. Unregulated or unresolved chronic inflammation can potentially lead to pathology, making inflammation a double-edged sword [41]. Low-grade systemic chronic inflammation is a common factor in several of the most prevalent NCDs like cardiovascular disease (CVD), diabetes mellitus (DM), neurodegenerative diseases, cancer, and metabolic syndrome [42]. Diet, lifestyle, and microbiome dysbiosis are major contributors to systemic chronic inflammation [42]. Current evidence indicates that excess dietary fructose can induce chronic low-grade systemic inflammation through several mechanisms [23, 43].

Inflammation due to gut barrier dysfunction. Permeability is a normal homeostatic feature of the intestinal mucosal barrier (epithelium and the mucus layer). An increased intestinal permeability disrupts homeostasis and leads to the transfer of bacteria and their products into the host causing endotoxemia and inflammation [44]. Consuming excess sucrose and fructose increases intestinal permeability and causes endotoxemia and inflammation [45–49]. The consumed fructose in the intestine is absorbed into the hepatic portal vein and reaches the liver for metabolism. Most of the fructose is absorbed into the intestinal epithelial cells through GLUT5 (fructose transporter type 5) and is metabolised by ketohexokinase. This process causes stress in the endoplasmic reticulum and leads to an increased intestinal permeability due to the downregulation of tight junction proteins [43]. Also, a portion of unabsorbed fructose in the small intestine influences the gut microbiota; excess fructose causes an increase in gram-negative bacteria and lipopolysaccharide (LPS) levels in the intestinal environment. LPS activates the macrophages to release pro-inflammatory cytokines that affect the tight junction proteins to cause increased intestinal permeability leading to the entry of endotoxins into the portal vein [45, 50]. The fructose-induced disruption of nitric oxide homeostasis in the intestinal epithelium also plays a role in the loss of tight junctions and barrier dysfunction [50].

Gut barrier dysfunction causes the bacterial endotoxin to translocate to the liver, activating toll-like receptor 4 (TLR-4) of the Kupffer cells (resident liver macrophages). The activated Kupffer cells, through the nuclear factor kappa-B (NF- κ B) pathway, produce pro-inflammatory cytokines like interleukin-1 β (IL-1 β) and tumour necrosis factor- α (TNF- α). This cascade causes hepatic insulin resistance and the formation and accumulation of fat in the liver leading to hepatic steatosis and non-alcoholic fatty liver disease (NAFLD) [45, 46, 49–52]. Hepatic steatosis and NAFLD increase circulating pro-inflammatory cytokines like IL-1 β , TNF- α ,

IL-6, and the acute phase C-reactive protein (CRP), leading to chronic low-grade systemic inflammation. The pro-inflammatory state of NAFLD has system-wide effects and is associated with metabolic syndrome, type-2 diabetes mellitus (T2DM), and CVD [53–55]. This pro-inflammatory state triggers the adipose tissue to produce adipokines that further exacerbate NAFLD and systemic inflammation thereby initiating a vicious cycle [53, 54]. Hence, excess dietary sugars can drive inflammation by activating TLR-4 and initiating NAFLD [45, 46, 49–52, 56, 57].

Inflammation and oxidative stress due to immune dysfunction. Excessive consumption of fructose is shown to cause inflammation by directly affecting the immune system; a sugar-rich diet increases the production of pro-inflammatory cytokines from macrophages through TLR-4 activation, increases tissue infiltration of macrophages and neutrophils, increases the Th17/Treg cell ratio and promotes inflammation by augmenting Th17 cell-mediated responses [23, 58–60]. Fructose, through AGE-RAGE interactions, causes a pro-inflammatory state [61]; high levels of fructose cause the accumulation of advanced glycation end-products (AGEs) that activate the receptor for advanced glycation end-products (RAGE) and lead to pro-inflammatory dendritic cells that produce IL-1 β and IL-6 [62]. Also, high fructose consumption led to systemic oxidative stress in rats; there was an increase in reactive oxygen species (ROS) in blood mononuclear cells and elevated IL-12 and IL-6 in the serum following high fructose consumption for 12 weeks [63].

Metabolites of fructose activate the immune system and elicit pro-inflammatory responses [43, 64]. Uric acid, a byproduct of fructose metabolism, activates nicotinamide adenine dinucleotide oxidase (NADPH oxidase) and increases the production of ROS leading to mitochondrial oxidative stress [43, 65]. This oxidative stress activates the NLRP3 inflammasome which recruits caspase-1 and increases the production of IL-1 β [43, 66]. Uric acid also activates the NF- κ B pathway to produce pro-inflammatory cytokines [43]. Lactate is another metabolite that accumulates during a high-fructose diet and promotes chronic inflammation [43]. Lactate promotes the formation of the Th17 subset, induces the production of IL-17, and interferes with the killing capacity of CD8⁺ T lymphocytes [43, 67, 68]. High fructose consumption caused the accumulation of NF- κ B and IL-1 β and reduced the antioxidant capacity in female rats [69]. Overall, high dietary fructose intake induces oxidative stress and low-grade systemic inflammation and puts the body in a pro-inflammatory state [23, 43, 58, 64]. It is important to note that chronic low-grade systemic inflammation and periodontitis have a bi-directional relationship [70, 71].

Sugar and periodontal inflammation. A diet high in sugars is associated with periodontal inflammation [33, 34, 72–74]. An observational study found that an excessive intake of added sugars was associated with periodontal disease in adolescents [33]. A recent systematic review concluded that excessive consumption of free sugars was positively associated with periodontal disease, but also reported that the evidence is limited due to the study designs [72]. An analysis of NHANES data (1988–1994) showed that frequent consumption of added sugars was positively associated with periodontal disease [34]. Consuming added sugars beyond the WHO and AHA recommended limit was associated with a higher prevalence of dental decay and inflammatory periodontal disease in Brazilian adolescents [73, 74]. A 2023 systematic review concluded that sugar-sweetened beverage consumption increases the risk of gingival and periodontal inflammation [75]. Another 2023 meta-analysis concluded that restricting free-sugar intake reduces gingival inflammation [76]. Hence, high dietary sugar intake can exacerbate periodontal inflammation and detrimentally affect periodontal health, possibly through low-grade systemic inflammation induced by gut barrier

dysfunction, NAFLD and immune dysfunction stemming from a high-sugar diet. Also, gut barrier dysfunction has a bi-directional relationship with periodontal inflammation through the oral-gut axis [77, 78].

Sugar-induced microbial dysbiosis

The human microbiome is critical to health and disease [79]. “Microbiome” is a term that represents all the microbes that reside in the human body (microbiota) along with their genomic content [80]. Humans have evolved to co-exist with microorganisms symbiotically and live in a finely balanced relationship with the microbiota. The most diverse microbiomes in the human body are in the gut and the oral cavity [81, 82]. A change in the composition and the diversity of the microbiota that leads to the disruption of their symbiotic balance with the host is called dysbiosis [83]. Diet and lifestyle, among several other factors, contribute to the dysbiosis of the microbiota leading to pathological states [84].

Gut microbial dysbiosis. The gut microbiota mainly comprises the phyla: *Firmicutes*, *Proteobacteria*, *Bacteroidetes*, *Actinobacteria*, and *Verrucomicrobia* [26, 45]. The gut microbiota produces beneficial short-chain fatty acids (SCFAs) like butyrate and propionate that play an important role in gut barrier integrity and mucosal immunity [85]. Dysbiosis of the gut microbiota has numerous local and systemic consequences and is tied to several systemic diseases [86]. A high-sugar diet induces gut dysbiosis by increasing the abundance of *Bacteroidetes* and *Proteobacteria* and decreasing the abundance of *Firmicutes* and SCFA-producing bacteria [26, 43, 45, 85]. This alteration increases intestinal permeability, endotoxin translocation and inflammation [43, 85, 87]. In rats, 10% fructose feeding for four weeks showed a decrease in *Firmicutes*, an increase in *Bacteroidetes* and *Proteobacteria*, and a decrease in SCFA-producing bacteria [88]. Rats on a sucrose-rich diet showed an increase in the *Bacteroidetes/Firmicutes* ratio and a decrease in α diversity. There was also a decrease in SCFAs along with lipid accumulation in the blood and liver [89]. High fructose consumption in rats (10.5g/kg/day) increased the abundance of *Lachnospira*, *Parasutterella*, and *Blantia* with no change in the diversity of the microbiota; increased uric acid levels; increased IL-6 and TNF- α ; decreased IL-10; decreased the expression of intestinal tight junction proteins; led to increased intestinal permeability and caused lipid accumulation in the liver [90]. Study participants consuming high-fructose syrup (100g/day) showed increased *Bacteroidetes*, decreased *Firmicutes*, and reduced SCFA-producing bacteria [91]. Interestingly, consuming similar levels of fructose (100g/day) entirely through fruits positively affected the gut microbiota [91].

Oral microbial dysbiosis. The oral microbiome is a diverse community of around 700 different species of bacteria, predominantly of the phyla: *Actinobacteria*, *Bacteroidetes*, *Firmicutes*, *Fusobacteria*, *Proteobacteria*, *Saccharibacteria*, and *Spirochaetota* [82]. Emerging evidence suggests that excessive intake of dietary sugars can push the oral microbiome towards dysbiosis. Short-term, 10% sucrose rinses (14 days) caused a loss of α -diversity and increased the abundance of *Actinomyces* and *Corynebacterium* in the supragingival microbiota [92]. An in-vitro study showed that 2% sucrose treatment could disrupt the balance between alkali and acid-producing bacteria to cause dysbiosis in a multi-species biofilm model [93]. Also, sucrose incubation reduced oral microbiota diversity and increased the abundance of *Streptococcus* species [94]. Higher sucrose intake and high-glycaemic load food items decreased the oral microbiota diversity in post-menopausal women [95]. A recent systematic review concluded that excessive sugar intake significantly reduced the diversity and caused dysbiosis of the oral microbiota [96]. However, the quality of the systematic review was reportedly questionable [97]. Since the

evidence regarding sugars and dysbiosis of the oral microbiota is not robust, future research needs to study further the microbiological consequences of high-sugar intake in the oral cavity.

The addictive and hypercaloric sugar

Addiction potential of sugar. Sweet taste is one of the most intense sensory experiences of humans and current evidence indicates that excess dietary sugars are potentially addictive [98]. Addiction—also known as substance use disorder—is diagnosed using a set of eleven criteria given by the Diagnostic and Statistical Manual of Mental Disorders-5 (DSM-5) [99]. Animal models of sugar addiction display several diagnostic criteria for substance use disorder like bingeing, craving, tolerance, withdrawal, and hazardous use [98, 100, 101]. Also, several parallels exist between sugar and drugs of abuse (cocaine, heroin, amphetamine, nicotine etc); both involve similar cross-sensitization phenomena and neurochemical pathways—Intermittent sugar intake causes the release of dopamine in the nucleus accumbens (the pleasure centre of the brain) that drives a sense of reward (liking; reinforcement and learning; and motivation and wanting) while also delaying the release of satiety-inducing acetylcholine in the nucleus accumbens [98, 100, 101]. Cross-sensitization is when one drug of abuse elicits the same hyperactive locomotory behaviour of another drug of abuse; rats sensitised to amphetamine showed similar hyperactivity when fed with a 10% sucrose solution [102]. Sugar addiction also involves the endogenous opioid system. Highly palatable food rich in sugar releases endogenous opioids in the limbic system and causes dependence; injection of naloxone (an opioid antagonist) causes signs of opioid withdrawal symptoms in sugar-dependent rats [103]. Overall, there is significant evidence to indicate that sugar behaves like a drug and could potentially lead to addiction [98, 100, 101, 104].

Overconsumption of sugar. Some evidence indicates that sugars are no different from other calories and that only when sugar consumption results in a positive energy balance (higher energy intake compared to energy expenditure), it leads to obesity, metabolic syndrome and other related co-morbidities [105–111]. However, sugar can be hypercaloric as it is easy to overconsume for several reasons: consuming sugar is potentially addictive; sugar is widely used in most ultra-processed, packaged food that is aggressively and attractively advertised and marketed; sugar and sugar-containing ultra-processed food are hyper-palatable, easily accessible, readily available and relatively inexpensive; sugar and sweets are widely accepted to the extent that it is even associated with celebratory occasions; and unlike nicotine or alcohol, there is neither taboo nor governmental restriction policies on sugar. Another factor that can lead to overconsumption is the lack of adequate satiety after consuming sugar-sweetened beverages [111, 112]. Consuming added sugars can also promote nutrient and energy deficits through various mechanisms leading to overconsumption and obesity [113]. These factors predispose an individual to overconsume added sugars through an unbalanced diet and disrupt the overall energy balance. This paradigm is evident with the global increase in the prevalence of obesity and metabolic syndrome in parallel with the global rise in sugar consumption [114–116]. Extensive evidence in scientific literature shows that sugar is associated with several NCDs [117]. A 2023 umbrella review concluded that high sugar intake is associated with obesity, metabolic syndrome, NAFLD, T2DM, CVD, and cancer [118]. It is important to note that obesity, metabolic syndrome, T2DM, and NAFLD are well-known risk factors with a bi-directional relationship to periodontal inflammation [71, 119–125].

Clinical relevance

There are three critical factors in the initiation, development, and progression of periodontal inflammation: dysbiosis of biofilm communities that initiate periodontal inflammation, unresolved

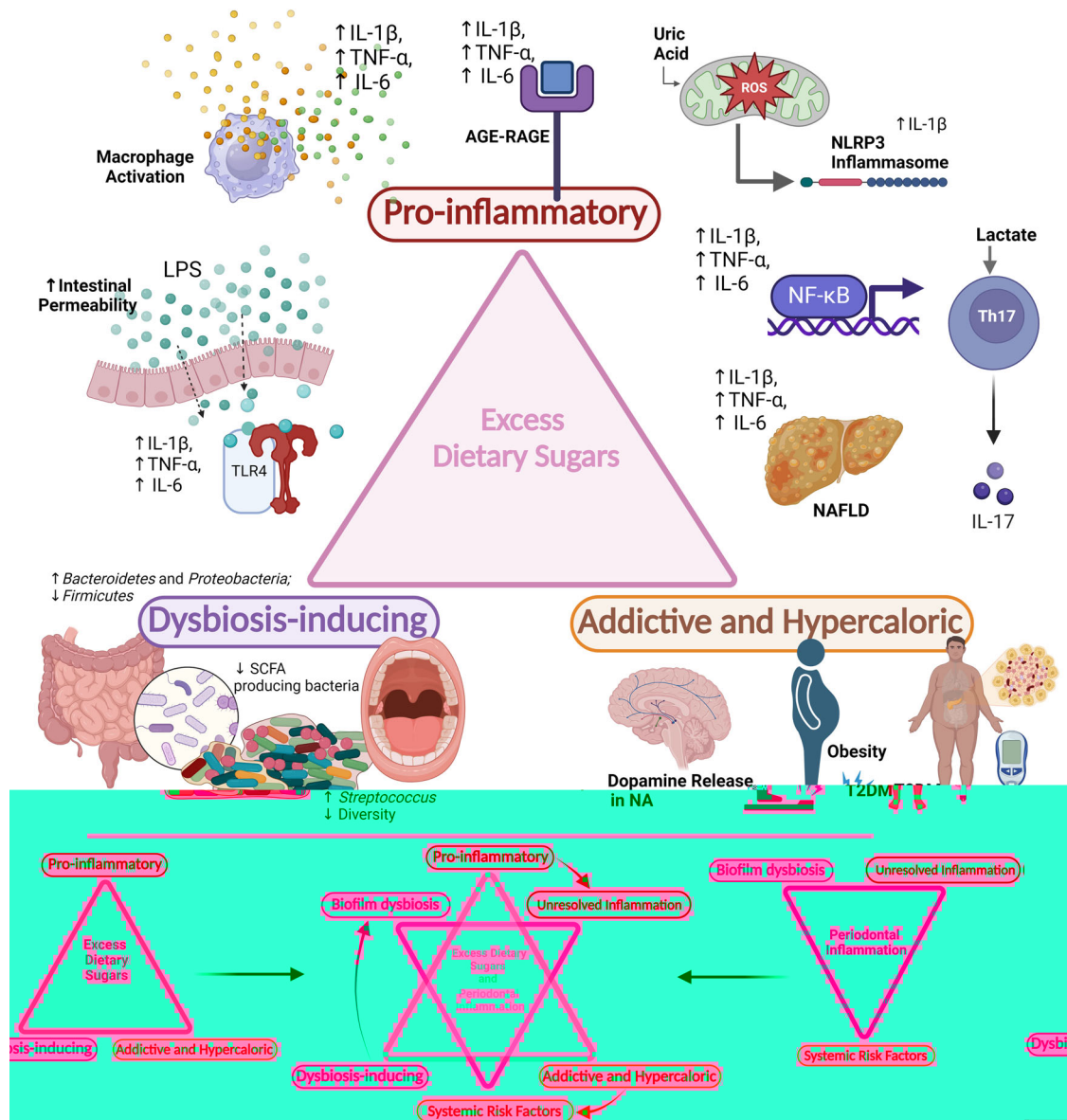


Fig. 2 Excess dietary sugars and periodontal inflammation. Excess dietary sugars are potentially pro-inflammatory, dysbiosis-inducing, addictive, and hypercaloric and can initiate and aggravate periodontal inflammation. (AGE- advanced glycation end-products; RAGE- receptor for advanced glycation end-products; ROS- reactive oxygen species; LPS- lipopolysaccharide; TLR-4- toll-like receptor-4; NAFLD- Non-alcoholic fatty liver disease; NF- κ B- nuclear factor kappa-B; SCFA- short-chain fatty acids; NA- Nucleus accumbens; T2DM- type-2 diabetes mellitus; IL- interleukin; TNF- α - tumour necrosis factor- α ; NLRP3- NOD-, LRR- and pyrin domain-containing protein 3) (Created in BioRender. Biorender, P. (2024) BioRender.com/h79u248).

and exaggerated host inflammatory response that causes tissue damage, and systemic diseases and conditions that aggravate the periodontal host inflammatory response [126, 127]. Excessive intake of dietary sugars (fructose and sucrose) can negatively influence all three factors and can play an important role in the initiation, development and progression of periodontal inflammation (Fig. 2)—excess sugars disrupt the eubiotic balance of the oral microbiota to cause dysbiosis; excess dietary fructose is pro-inflammatory and cause low-grade systemic inflammation and oxidative stress that aggravates periodontitis; excess dietary sugars are addictive and hypercaloric which leads to systemic risk factors (obesity, metabolic syndrome, T2DM, and NAFLD) that further aggravate periodontal inflammation.

At present, the main strategies to prevent (daily brushing of teeth) and manage (professional biofilm removal) periodontal inflammation are unidimensional and only aim to control and

remove the dysbiotic biofilm on the teeth. Along with the current convention, we need to adopt multidimensional strategies to modify lifestyle factors that induce dysbiosis and a systemic pro-inflammatory state for optimal prevention and management of periodontal inflammation (Fig. 1) [36]. Excess consumption of sugar through an unhealthy diet facilitated by the current food environment seems to be deleterious to systemic and periodontal health and is a modifiable lifestyle risk factor that can be used to prevent and manage periodontal inflammation.

Recently, several dietary and nutritional interventions have been discussed and investigated against periodontal inflammation [128]—a micronutrient-rich diet low in sugar and processed carbohydrates significantly reduced gingival and periodontal inflammation [129, 130]; a similar type of diet also reduced the abundance of periodontitis-associated bacterial species in the supragingival biofilm [131]. A more recent secondary analysis of

four studies found that avoiding sugar, processed food, and sweetened beverages for four weeks with a focus on whole food rich in dietary fibre and micronutrients, significantly reduced the inflammation in the gingiva [132].

There is limited research in the literature regarding the effects of sugar and a sugar-rich diet on periodontal health and disease. This lack of a robust body of evidence sets the stage for future sugar and periodontal health research. Also, with the current genomic technologies, future nutrigenomics research could explore the association between sugar and periodontal inflammation in greater detail by studying the effect of excess dietary sugars on gene expression [133].

Concluding remarks

Available evidence suggests that excessive consumption of sugar is potentially pro-inflammatory and dysbiosis-inducing. Sugar is also potentially addictive, and several factors facilitate its over-consumption leading to systemic risk factors for periodontal inflammation. Current research, although limited, indicates that excessive dietary sugar intake is associated with periodontal inflammation and can be viewed as a modifiable lifestyle risk factor. Lifestyle modification strategies through dietary and nutritional interventions that control sugar and ultra-processed food consumption seem beneficial to periodontal health and need further investigation. Advocating for conscious eating with moderation in dietary sugar intake could serve as a simple, cost-effective, and practical public health strategy for preventing and managing periodontal inflammation.

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SS conceived the title, the design and structure of the paper, wrote the main text and designed the original figures in the manuscript. SK contributed to the main text and reviewed and revised the manuscript.

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ADDITIONAL INFORMATION

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