Chapter

Periodontitis, Its Associations, and Prevention

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Abstract

The ultimate goal of studying associations with diseases is to plan, implement, and evaluate preventive strategies. Today, after reviewing the body of evidence, one needs to ask: What has not been learned yet regarding periodontitis associations and its prevention? Current recommendations to prevent periodontitis are mostly limited to individual patient care while population-based approaches are nearly absent. Current strategies are not only time-consuming and costly but can be also ineffective to combat disease burden in populations. To initiate and sustain successful outcomes, prevention needs to be applied at multiple levels. Interventions need to target unhealthy behaviors along with their associated social and physical environmental constraints. The chapter presents highlights from current research on associations with periodontitis, its limitations, and the need to understand pathways linking periodontitis with its exposures over the life course. Finally, a suggested multilevel strategy for periodontitis prevention was outlined.

Keywords: periodontitis, prevention, associations, pathways, life course, social determinants of health

1. Introduction

A common approach in our clinical care is to advice patients to adopt healthy behaviors including smoking cessation, oral self-care (e.g., tooth brushing and use of interdental aids), healthy dietary habits, and physical activity [1]. While plenty of theories have been suggested trying to explain or change health behaviors, such approach can be limited without considering social and physical environmental exposures [2, 3]. For example, using different strategies with patients to quit smoking including referral to smoking cessation clinics could have limited benefits [4–6]. In contrast, implementing sin tax and age restrictions on tobacco products by policymakers can make these products less affordable and accessible. Consequently, more cost-effective and radical change may occur at population level; people may quit smoking or refrain from smoking in first place. The social and environmental exposures can be collectively called social determinants of health, which include socioeconomic status, housing, residential neighborhood, environmental safety, social support network, social norms, structural racism, and discrimination [4, 5, 7–9]. Although targeting the social determinants of health can be effective in prevention of

diseases along with their related behaviors, they could be more effective when applied as multilevel strategies including clinical care.

In Geoffrey Rose's seminal paper "*sick individuals and sick populations*", key concepts of studying disease etiology and its prevention were discussed [10]. Rose highlighted the importance of considering different ecological levels when studying the disease etiology. When an exposure is uniformly distributed within a population, it could hamper its detection, which necessitates studying its variations among populations. Besides Rose's remarks, lack of variations in distribution could be also indicative of methodological limitations in measuring, summarizing, or analyzing the disease and its exposures [11]. During periodontal indices' era, it was believed that gingivitis leads to periodontitis and consequently results in tooth loss in virtually everyone [12, 13]. These notions of lack of variations in susceptibility to periodontitis were attributed to several methodological limitations. Periodontal disease was measured using indices including Russell's Periodontal Index [14] which was based on the presence or absence of pockets without precise measurements. Thus, it did not properly differentiate between gingivitis and periodontitis [12]. The gingivitis and periodontitis were measured as continuous process using gradually increasing scores, summarized for as individual's mean index score, then summarized for a population as mean score based on individual's mean scores. Consequently, variations within and between individuals were lost [12, 13]. The periodontal indices, which measure and summarize periodontal disease as mean score, were abandoned by the end of 1980s and replaced by site-specific measurements [15, 16]. The improvement of periodontal measurement methods allowed detection of variations at multilevels (site-level, tooth-level, individual level, and between populations) [17–22]; consequently, it motivated research on host susceptibility and associations with periodontitis [23–25].

Since the periodontal indices were abandoned, periodontitis is summarized in population-based studies using prevalence, extent, and severity [12, 26]. Prevalence can be defined as the proportion of individuals with periodontitis, extent can be defined as mean proportion of sites or teeth with periodontitis, and severity can be defined as mean clinical attachment loss (CAL) or mean periodontal probing depth (PPD). Among these summaries, periodontitis severity is calculated as a mean score for population; such summary does not properly account for the multilevel nature of the measurements and its variations at site level, tooth level, and individual level before it is summarized for the population [11, 27]. The summary also averages diseased and non-diseased sites in individuals with or without periodontitis. Similar issues are encountered when periodontitis progression is calculated as mean annual score in longitudinal studies [28]. This summary does not provide proper insights regarding the variations at different periods of observation in addition to the limitations abovementioned for periodontitis severity. Therefore, it appears that methodological limitations of the twentieth century still apply to some current research approaches. Other limitations of studying associations with periodontitis were discussed in the following sections.

2. Associations with periodontitis

Among dominant notions in the twentieth century was dental plaque considered the cause of periodontal diseases; it was thought that unless plaque is prevented, gingivitis invariably leads to periodontitis and subsequently tooth loss [13, 29]. Russell, who was considered the father of periodontal disease epidemiology, analyzed two population-based data and concluded that plaque and age alone explain around

90.3% of variations in periodontal disease regardless of which population being studied [30]. Similarly, an experimental gingivitis study was used to support the evidence that plaque is the cause of periodontal disease [31]. Consequently, clinical practice of that era almost solely focused on plaque control [32–34]. Though, these findings reflect the abovementioned methodological limitations of periodontal and plaque indices. By 1980s, studies on untreated populations used site-specific measurements, unveiled variations in susceptibility to periodontitis despite accumulations of uniformly high levels of plaque and calculus [19, 22, 35–39]. Variations in susceptibility to periodontitis were observed among individuals, population groups, and teeth sites. Thus, it was no longer accepted that plaque is the only cause of periodontal disease. Several theories evolved trying to explain the role of dental plaque including nonspecific and specific plaque theories [40]. Currently, there is a general agreement that dysbiosis of oral biofilm may induce an inflammatory response in susceptible hosts to cause periodontitis [41]. Dysbiosis is a shift in the species within the oral microbiome that results in loss of homeostasis and may impact the human health; it can result due to an expansion of pathogenic microorganisms, altered metabolic capacity, decreased beneficial microorganisms, or reduced species diversity. Thus, the etiology of periodontitis can be attributed to "*sufficient cause*" where a set of components (exposures) can be sufficient for causing the disease that requires dysbiotic biofilm as a necessary component [42–44]. However, the biofilm alone is not sufficient cause of periodontitis. Hypothetical sets of sufficient causes for periodontitis are depicted in **Figure 1**.

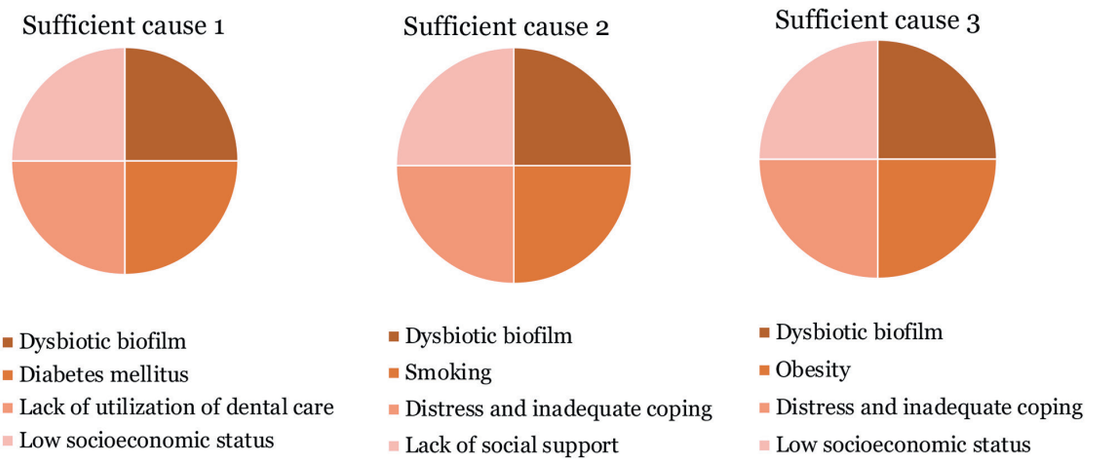

Figure 1.
Hypothetical sets of sufficient causes for periodontitis using sufficient cause component model (Rothman's pies) [45]. Dysbiotic biofilm is a necessary component but insufficient for causation [42, 44].

Periodontitis and other chronic diseases including atherosclerotic cardiovascular disease, obesity, and diabetes mellitus are associated with increased low-grade systemic inflammation and oxidative stress [46–49]. Evidence suggests that aggregation of chronic diseases within individuals is associated with higher level of inflammation and oxidative stress compared to having a single condition [46, 48, 49]. Chronic diseases also have common risk factors with periodontitis including underlying exposures and health behaviors [50, 51]. Underlying exposures can be either non-modifiable including age, sex, ethnicity, and genetics or modifiable such as the social determinants of health. Underlying exposures are mostly associated

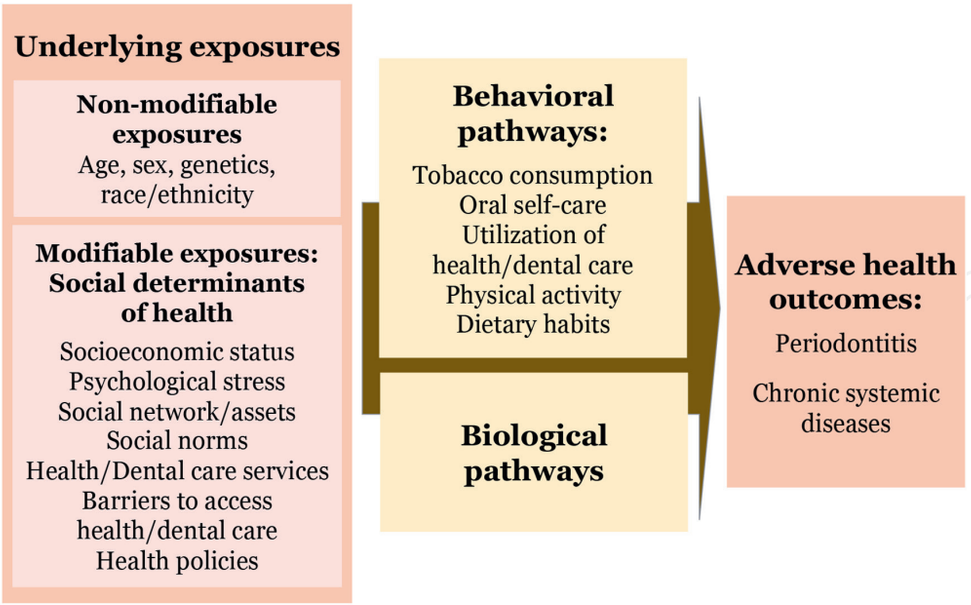

Figure 2.
Behavioral and biological pathways that could link the underlying exposures with the adverse health outcomes including periodontitis.

with periodontitis or other chronic diseases mainly through behavioral or biological pathways (**Figure 2**).

Several models were proposed to conceptualize disease causation including sufficient component cause model (**Figure 1**), web of causation, and structural equation models [44, 45, 52]. Nancy Krieger criticized the use of sufficient component cause model and web of causation for lacking accountability for unjust disparity in disease distribution when proposed her ecosocial theory [53, 54]. Krieger's theory is not a method of disease causation since it is concerned with individuals and their communities between the past and present. "*Shared observations of disparities in health do not necessarily translate to common understandings of cause; it is for this reason theory is key*" [53]. The term "*embodiment*" was introduced to refer to interactions between society and biology. The theory also focused on pathways of these interactions at multiple ecological levels over the life course while considering the accountability for injustice. Ecological levels can be conceptualized at multiple levels and domains including home, school, and work. In contrast to causal models that can be used for testing specific hypotheses, Krieger's theory can be useful for identifying knowledge gaps, generating research questions, articulating increments of evidence from separate studies to understand disease etiology, and taking the theory into action [54].

Two epidemiological concepts relevant to disease prevention that need to be distinguished are the life course approach and the natural course of disease. The life course epidemiology is concerned with understanding psychosocial and physical environmental exposures and when they can impact health behaviors or become biologically embedded over the life course (intrauterine life, childhood, adolescence, early adulthood, late adulthood, or across generations) [55–57]. While earlier approaches for life course focused on diseases as its end points, recent approaches shifted to study multiple aspects of physiological functions [55]. The exposures are studied for their impact independently, cumulatively, or for their interactions. In contrast, the natural course of disease is a disease specific concept and based on different stages of disease development (susceptibility, asymptomatic, symptomatic,

and terminal stages) in the absence of treatment [11, 58, 59]. Recruitment of study population that is either never treated or all treated similarly for periodontal conditions can control for treatment status. However, disregarding impacts of treatment can lead to erroneous interpretations of study findings [11, 13]. This can be especially true when considering that periodontal treatment (non-surgical or surgical) has non-negligible impacts on the periodontitis severity, extent, and progression [11]. Unfortunately, studying the periodontitis associations while disregarding the treatment impacts is a common approach in periodontal research.

2.1 Impact of age on periodontitis: What has not been learned yet?

Based on cross-sectional surveys, periodontitis increased in severity, extent, and prevalence with age [11, 60–63]. When periodontitis defined using CAL, its association with age was more consistent, whereas PPD had less clear pattern [11, 15, 60, 64, 65]. When periodontitis included non-severe thresholds (CAL ≤4 mm, PPD ≤6 mm, or ≤ 1/3 bone loss), the prevalence steeply increased after 20 years through 30s until it reaches its peak between 35 and 45 years [11, 38, 60, 66, 67]. The prevalence before 20 years, although less frequent, widely varied within and among populations; estimates from different populations were between 1.0% and 69.2% [11, 60, 62, 66, 68–70]. Similar patterns were observed when periodontitis incidence was studied in longitudinal surveys [67, 71, 72]. In contrast, when periodontitis was defined at severe thresholds, majority of its variations, within populations, were mostly confined to adults [38, 64, 66, 73, 74]. Despite that clinical classifications consider molar-incisor pattern suggestive of localized aggressive periodontitis in young age groups [75–78], evidence from epidemiological surveys indicates that such pattern is common in all age groups [17, 18, 22, 29, 79]. Similar pattern was observed in a study on untreated population; most impacted teeth were upper molars and lower incisors of different age cohorts (**Figure 3**) [11].

There is general agreement that age association with periodontitis is due to accumulative effect of exposures and disease experience over time rather than having an increased susceptibility to periodontitis at specific age [15, 80]. However, it worth noting that such association remains robust after adjustment for different exposures including sex, social, behavioral, and medical exposures in cross-sectional or longitudinal studies [11, 60–62, 81, 82]. In a study in untreated Sri Lankan tea workers, periodontitis progression had an independent association with both age and follow-up time [83]. These age-related findings may suggest that effect of age on periodontitis cannot be completely explained as function of accumulation of exposures and disease experience over time. Among the difficulties of fully understanding the effect of age, in periodontal literature, is that majority of studies either focus on younger or older age groups excluding direct age-related comparisons [11]. Such approach could hide age-related patterns of periodontitis distribution and its associated exposures. Another limitation is that periodontitis progression is often studied as mean annual CAL or PPD for comparisons among age groups [28]. As previously stated, such summary does not properly account for variations in the rate of progression within or between individuals, subgroups, and at different periods.

An ideal study design to better understand the role of age on susceptibility to periodontitis would be a life course approach. Such approach can identify if there are sensitive periods in development, which can have behavioral or biological impacts [55]. Sensitive periods can be psychologically, socially, or physiologically defined [84]. Behaviors including smoking, unhealthy dietary habits, and inadequate coping with social adversity are often acquired during adolescence. This makes interventions

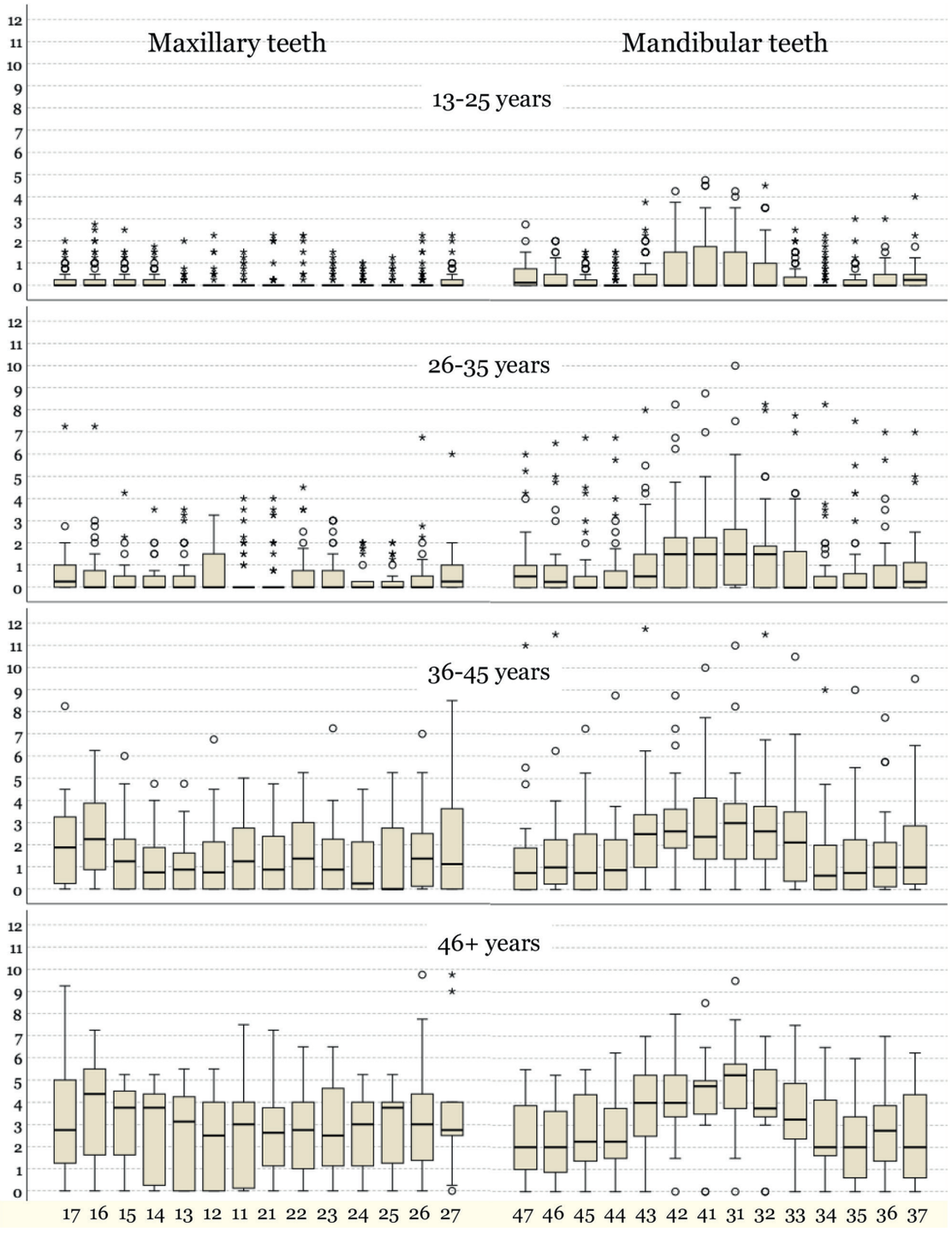

Figure 3.
Periodontitis severity for maxillary and mandibular teeth in an untreated population [11, 60]. The most impacted teeth were the lower incisors and upper molars across different age cohorts.

to combat certain behaviors such as smoking most effective at that stage [84]. At older age, compromised manual dexterity and physical disability could limit oral self-care and utilization of dental care. The life course study design is mostly limited due to its feasibility especially when considering the lengthy latency periods between exposures and periodontitis incidence.

2.2 Sex

Males almost consistently had higher periodontitis prevalence, extent, and progression compared with females in epidemiological studies [60, 61, 81, 85–87]. In contrast to suggestions that sex-related differences can be attributed to better oral health behaviors in females [80, 88], studies on populations with uniformly high levels of plaque and minimal access to dental care also found an increased periodontitis prevalence, extent, and progression in males [11, 60, 86, 87]. National surveys from Brazil, France, and the United States reported higher periodontitis prevalence in males despite the adjustment for behaviors including patterns of dental visits, smoking, and alcohol consumption [61, 81, 89, 90]. In contrast, a recent systematic review found almost no sex-related differences in terms of periodontitis progression rates [28]. This finding was based on a subgroup meta-analysis of two studies only. However, subgroup meta-analysis can be only conducted when there are minimally 10 available studies [91]. In addition, the sex-related differences were compared based on mean annual progression, which does not properly account for multilevel variations. In a study Brazilian population, males had an unadjusted risk ratio (RR) of 1.24 (95% confidence interval [CI]: 1.09, 1.42). After adjustment for smoking, males had an adjusted RR of 1.22 (95% CI: 1.08, 1.39). This may be interpreted as having small effect of smoking on sex. However, the study found an interaction between smoked packs per year and sex; females who smoked 30+ packs had higher RR of periodontitis progression after adjustments for age and level of education.

Sex dimorphism in immunological responses was suggested as plausible biological mechanism that may explain the differences in susceptibility to diseases [85]. Circulating levels of sex steroids could alter as a function of age mainly during puberty and menopause. However, such findings were mostly based on responses to acute infections. Although the use of life course approach would have been ideal to understand the sex dimorphism in susceptibility to periodontitis, cross-sectional surveys with wide age ranges may provide indirect evidence to identify these differences as function of age. This approach may narrow down the required observation time before conducting longitudinal studies. For example, in a study on untreated population, the sex-related differences in periodontitis prevalence and extent (**Figures 4** and **5**) were mainly observed after 20 years [26, 60]. Similarly, periodontitis had no association with sex in 15–19-year adolescents from several Latin American nations [70]. These findings may suggest the need to study the sex-related differences in exposures prior to 20 years.

2.3 Race/ethnicity

Studies from the United States almost consistently presented an association between race/ethnicity and adverse health outcomes including periodontitis [61, 94– 96]. In the first National Health and Nutrition Examination Survey (NHANES), higher periodontitis prevalence was found in black Americans compared with whites with an adjusted odds ratio (OR) of 1.31 (95% CI: 0.78, 2.19) [94]. In NHANES III, the adjusted OR in Blacks increased to 2.09 (95% CI: 1.68, 2.60); the adjustments in both surveys were done for age, socioeconomic indicators, smoking, diabetes, and time elapsed since last dental visits. In NHNANES (2009–2012), both Hispanics and non-Hispanic blacks had higher periodontitis prevalence than

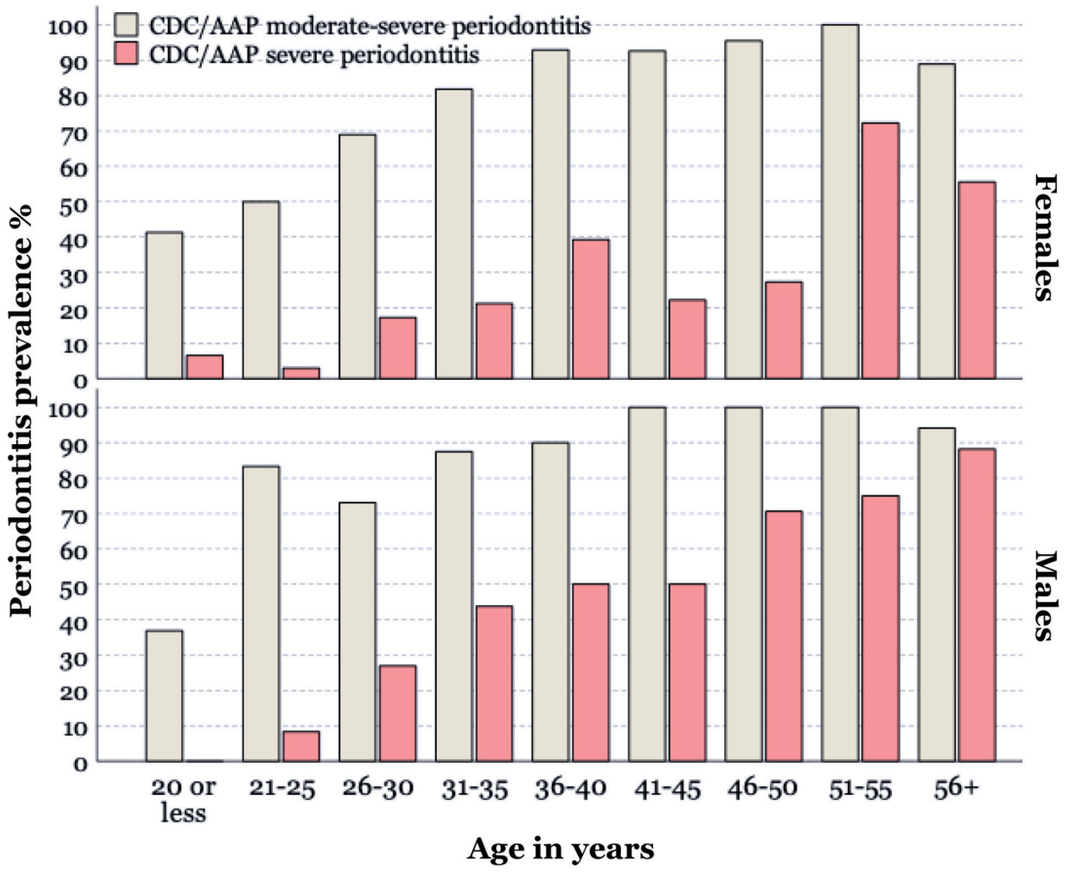

Figure 4.
Periodontitis prevalence per age and sex groups in an untreated population [11, 60]. Periodontitis defined based on criteria by the Centers for Disease Control and Prevention and American Academy of Periodontology (CDC/AAP) [92, 93].

whites [61]. Ethnicity-related associations were not confirmed in Brazilian population [81, 82]. Disparities in health outcomes based on race/ethnicity could not be explained by genetic differences but could rather reflect differences in social, cultural, and behavioral factors between the past and the present [53, 94, 96]. Thus, ethnicity is mainly considered as a social construct, and more information needs to be provided to explain its role within a broader context.

Borrell *et al*., suggested that disparity in periodontitis prevalence between African Americans and Caucasians in the United States can be attributed to discrimination, racism, and residential segregation [94]. Residential segregation for example may impact health outcomes by potentially impacting exposures to environmental hazards, quality of education, employment opportunities, and influencing certain behaviors such as increased tobacco consumption is some neighborhoods. In a study that analyzed the association between ethnicity and periodontitis based on different socioeconomic status (SES), black Americans with high income had the highest periodontitis prevalence compared to both high-income whites and low-income blacks. This finding illustrates how increased income may not directly improve health outcomes. Possible explanations include increased income may occurred short term after development of periodontitis or was not accompanied by skills in managing resources. Social epidemiologists suggested that income increase in short term (e.g., winning lottery) can be associated with unhealthy behaviors such as increased smoking [84]. Another possible explanation is that high-income blacks may experience

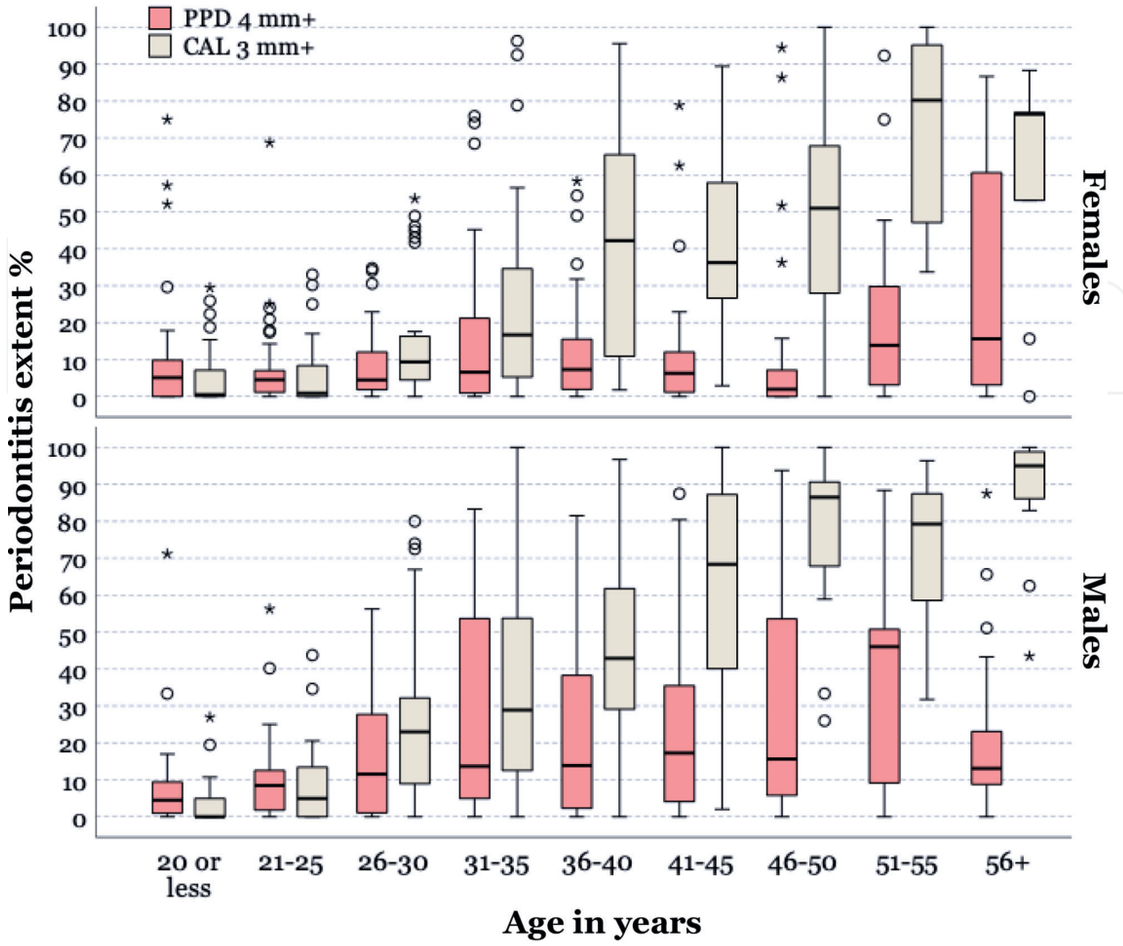

Figure 5.
Periodontitis extent per age and sex groups in an untreated population [11, 60]. PPD: Periodontal probing depth, CAL: Clinical attachment loss.

stressful conditions and increased social isolation, which adversely impact their health. Therefore, information about structural racism and residential segregation within a historical context provides better insights about the social and physical environmental constrains rather than "*blaming the victim*" for their behaviors and health conditions [53, 54, 97].

2.4 Socioeconomic status, its pathways, and embedment over the life course

SES is a term describes the social standing and power in society, and it is often measured using indicators such as level of education, income, and occupation. Social status may also be determined by the level of power "*In the context of workplace, there are those who occupy positions of supervisory authority over subordinates versus those who take orders from above*" [84]. Lower education was associated with periodontitis with a pooled OR of 1.86 (95% CI: 1.66, 2.10) from 18 studies [98]. The association between periodontitis and SES is almost consistently positive in the literature when using level of education and income as indicators [60, 61, 88, 95, 99]. However, such approach can be limited without studying this association within a broader context that could explain the link between the SES indicators and periodontitis. It has been suggested in the literature that income mainly acts as a mediator for the association between education and disparities in health outcomes; thus, some researcher would elect to report

only one of these two indicators. Though, both education and income demonstrated independent association with periodontitis after adjustments in multivariate models [99]. The neighborhood SES also had an independent association with periodontitis after the adjustment for individual level SES, age, sex, marital status, ethnicity, smoking, and diabetes [100]. Low and medium neighborhood SES had an adjusted OR of 1.73 (95% CI: 1.29, 2.32) and 1.63 (95% CI: 1.23, 2.17), respectively, compared to high SES.

SES can be linked to disparities in health outcomes through interrelated pathways (**Figure 6**) where the association can be mediated by psychosocial exposures, material factors (resources), and health behaviors [9, 95, 99, 101]. In neo-material pathway, effect of education can be mediated by income, occupation's working conditions, related resources such as health insurance; consequently, it can improve housing, residential neighborhood, affordability of hygiene products, and access to health/dental care. In psychosocial pathway, higher educational attainment can be associated with having broader social network, which can provide social support, enhancement of control beliefs, problem-solving, and coping skills; consequently, improve response to stressors. In behavioral pathway, education can increase the chance of being surrounded by well-educated people, change the social norms, and increase health literacy, which can improve health behaviors.

SES needs to be conceptualized as a dynamic approach over the life course; income and occupation could fluctuate (increase or decrease) over time while education either remains stable or increases. A dynamic approach for SES also implies reciprocities where increased SES could improve health and vice versa. Some relevant questions to be asked regarding life course approach include when is the sensitive period in development where SES could impact behaviors or can be biologically embedded?

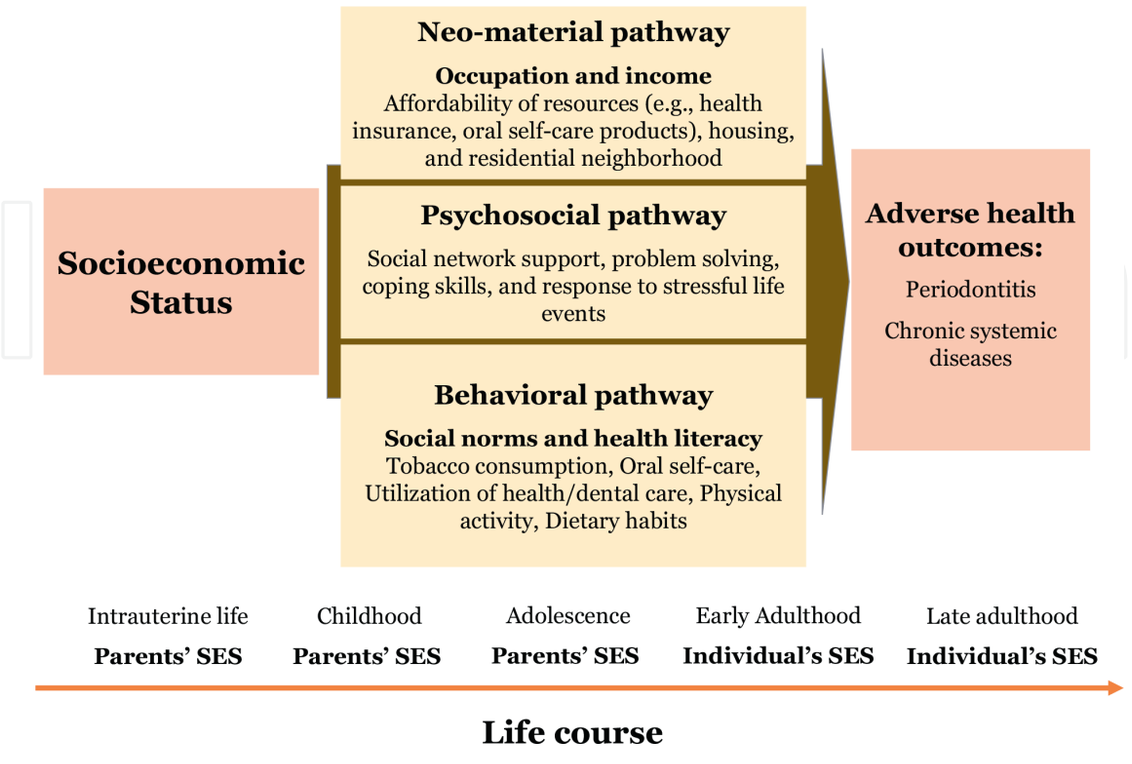

Figure 6.
Suggested pathways that could link socioeconomic status (SES) to periodontitis or other chronic systemic diseases over the life course.

What is the latency period between exposure to SES and having behavioral or biological impacts? Does the SES have accumulative impacts throughout life? Is the individual SES during adulthood or the parents' SES during childhood more relevant to periodontal health? Are childhood SES and adulthood SES correlated or independent? What is the impact of social mobility (transition to higher or lower SES)? It worth mentioning that in populations with rapid changes, social mobility needs to be assessed at smaller intervals over the life course rather than limiting the comparisons between childhood and adulthood.

Suggested models for dynamic approach for studying SES include [84]:

1. Critical (sensitive) period model assumes that Low SES could impact health at sensitive period in development; SES changes at later period cannot attenuate or reverse the impact already occurred.

2. Accumulation of risk model assumes that low SES's health impact increases incrementally.

3. Chain of risk (trajectory) model assumes that low SES in early life results in low SES later. However, impacts on health are only embedded later in life.

The importance of understanding the impact of SES on health and its related behaviors is to know when to intervene to achieve the best health outcomes. Based on a recent systematic review on seven longitudinal studies, early life SES had positive association with periodontitis [102]. However, the included studies had heterogeneous study designs and definitions of both SES and periodontitis; thus, made it difficult to arrive at a more specific conclusion. In one study, the parents' SES had no significant association with periodontal status in 15–16 years individuals; though, the parents' SES (income) was predictive for utilization of dental care [103]. In another study, early SES measured as parents' occupational status was associated with periodontitis in 26 years individuals, whereas the individual's SES in adulthood was not significant [104]. In contrast, the adult SES was more relevant to smoking and depression. The role of social mobility was also evaluated; highest periodontitis proportion was found in those with persistent low SES, followed by upward mobility group, then downward mobility group [104]. The lowest periodontitis proportion was found in those with persistent high SES. In another study that assessed the periodontal health at 15 years (baseline) up to 31 years, childhood SES was associated with periodontitis in adults, which was not mediated by adulthood SES or behaviors; RR was 1.85 (95% CI: 1.06, 3.24) [105]. So, studies that used the life course approach suggested that early SES during childhood can be more predictive of periodontitis rather than adulthood SES. These findings support the critical period model for impact of SES on periodontal health. However, the longest follow-up was up to 38 years, and the possible accumulative effect of SES cannot be excluded.

2.5 Psychosocial exposures, their pathways, and interactions

When negative life events occur, they undergo an appraisal process, if the stressors exceed the individual's coping ability, they can be perceived (appraised) as stressful [106, 107]. The physiological responses to stressors can be mediated by negative emotions including anxiety, depression, anger, or mixed emotions [107]. The "*allostatic load*" was a term introduced by McEwen to refer to wear and tear from chronic

over- or underactivity of protective body systems against stressors including the autonomic nervous system, hypothalamic–pituitary–adrenal (HPA) axis, metabolic, immune, and cardiovascular systems [108]. The perceived stress can be associated with health conditions (including periodontitis) through interrelated behavioral and pathophysiological pathways (**Figure** 7) [109, 110].

Different psychological exposures had an association with periodontitis [112–114]. In one study, psychosocial stress was not associated with periodontitis but had positive association with oral health behaviors (smoking and oral self-care) in 65–74 years women [115]. Among different daily strains, only financial strain was associated with higher CAL and bone loss after adjustment for age, sex, and smoking [113]. Intrinsic mechanisms that could regulate and the stresses include coping [116]. Inadequate coping (high emotion-focused and low problem-focused coping) was associated with higher CAL and bone loss regardless of the level of financial strain [113]. Problem-focused coping includes reappraisal of stressful events as challenging events and applying strategies that focus on solving the problems [116]. In contrast, emotion-focused coping includes applying strategies that deals with the emotions rather than the problem; it can include positive strategies such as regulations of emotions, meditation, spirituality, distracting oneself, and seeking support from social network. However, it can be also negative when denying the event, avoiding dealing with it, or adopting negative health behaviors such as unhealthy dietary habits, smoking, consuming alcohol, and drugs. Thus, emotion focused coping can be an inadequate coping strategy with stresses. Other behaviors related to inadequate coping include negligence of oral self-care and lack of sleep [111, 117, 118]. In contrast to coping, social network support and positive life events could buffer the impact of stressors through external pathways [106, 119]. Social support can be received (actual support) or perceived, which refers to the subjective beliefs about the availability of support; the perceived social support can be more effective in buffering the impact of stressors (**Figure** 7) [106].

When impact of social network was studied for association with periodontitis, marital status had no significant association with periodontitis prevalence or progression rate [61, 82, 114, 120]. However, having fewer friends and being

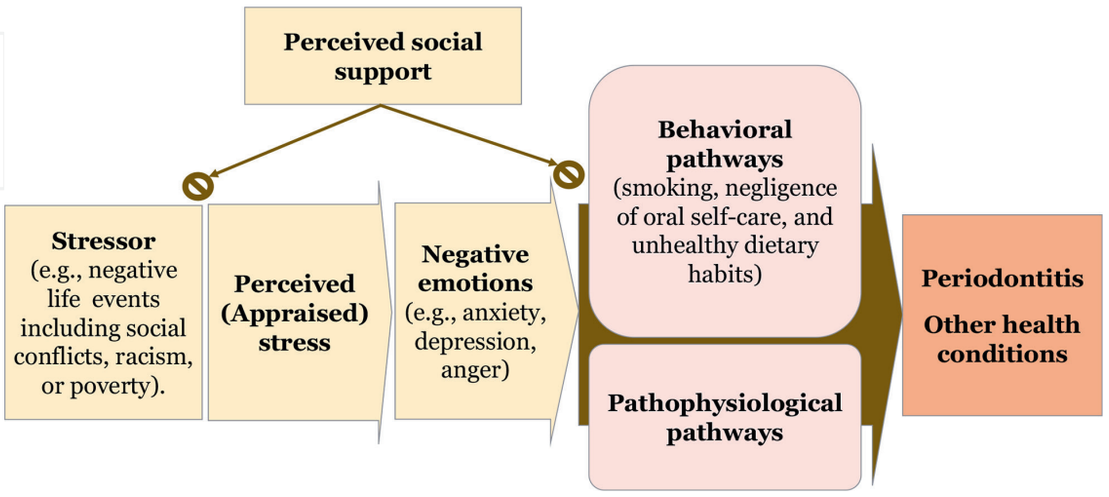

Figure 7.
Illustration of psychosocial interactions. Stressors can have adverse health impacts after being perceived (appraised) as stressful [106]. Negative emotions could be mediators of perceived stress and their impact on health could occur through interrelated pathophysiological pathways and behavioral pathways [109–111]. The perceived social support may buffer the impact of psychological stresses either before or after perceiving the event as stressful [106].

widowed were associated with higher periodontitis extent [120]. In a prospective study on healthcare professional men, having at least one friend was associated with a lower risk of periodontitis; RR was 0.70 (95% CI: 0.51, 0.96) after adjustments for age, marital status, smoking, body mass index (BMI), alcohol consumption, and diabetes mellitus [114]. Furthermore, participation in religious meetings had a lower risk of periodontitis and the adjusted RR was 0.73 (95% CI: 0.64, 0.83). In a cross-sectional study by Zini *et al*., religiosity had protective association with periodontitis through spirituality (intrinsic pathway) and social support (extrinsic pathway) [121]. Other studies did not find an association between social support and periodontitis [60, 122].

Possible mechanisms of how social network positively impact health (**Figures 6** and 7) is by buffering impact of stressors, shaping norms for health behaviors, sharing information can increase health literacy and awareness, helping in problem-solving, sharing funds and resources, and improving overall wellbeing [119, 123, 124]. In a recent systematic review, social support had positive association with oral health literacy, quality of life, and behaviors including utilization of dental care in immigrants and ethnic minorities [125]. It is important to keep in mind, however, that impact of psychosocial exposures can be either positive or negative [11]. For example, perceived stress manifested as anxiety can stimulate sympathetic nervous system and HPA axis; consequently, help people to meet certain deadlines and get the job done [107]. The allostatic load, up to a certain limit, can be within the body's reparative capabilities before it can be associated with pathological changes. Similarly, social network could have positive influence on behaviors (e.g., oral self-care), negative influence (e.g., smoking), enable certain behaviors (e.g., utilization of dental care), or constrain such behaviors [123]. Social network could also be a source of conflicts, stressors, or transmission of diseases. Thus, measurement of social network in terms of its size and range (structural indicators) alone can be deficient without considering specific functions [106, 119] or social norms [11]. Among different social support functions, emotional and informational support was the most responsive to buffer wide range of stressors [106, 119]. However, the overall research on mechanisms, pathways, and interactions of psychosocial exposures is still limited in periodontal literature.

2.6 Oral health behaviors

Health behaviors can be largely shaped by social environment including social adversity, social norms, social support, and socioeconomic status [3, 101, 113, 120, 123]. Health behaviors appear to cluster within individuals. For example, those who are physically active can also follow healthy dietary habits, regularly utilize dental/health care, regularly practice oral self-care, and vice versa. Unhealthy behaviors often aggregate in individuals with low SES [84]. In addition, behaviors such as smoking, negligence of oral self-care, and lack of utilization of dental care could be indicative of inadequate coping with social adversity [111, 117, 118].

Impacts of smoking on periodontium were well documented, and evidence was considered adequate to conclude that smoking is a cause of periodontitis by the office of surgeon general in 2004 [126]. The frequency and duration of smoking had a dose response association with periodontitis prevalence and progression [82, 127]. Smoking also had positive association with periodontitis in adolescents (15–19 years) with an adjusted OR of 1.6 (p-value: 0.017) after adjustments for demographics, plaque scores, and bleeding on probing [70]. Number of years since quitting of smoking also had a gradient reduction in susceptibility to periodontitis. Based on a systematic review on 14 prospective studies, periodontitis incidence in smokers had an RR of

1.85 (95% CI: 1.5, 2.2) [128]. Smokers also had poor periodontal treatment outcomes (non-surgical and surgical) compared to never smokers [129–131].

Despite that oral self-care had clear association with gingivitis [31, 80], its association with periodontitis was not consistent. Based on systematic review on 14 studies, tooth brushing had an OR of 1.44 (95% CI: 1.21, 1.71) [132]. Infrequent oral self-care and presence of calculus had positive association with periodontitis in young populations (14–29 years) [62, 69]. Good oral self-care had protective association with overall periodontal treatment need in Portuguese population [133]. Other studies did not confirm the association of oral self-care with periodontitis [82, 134]. It worth noting here that self-reported oral self-care can be subject to social desirability bias, that is, study participants could report daily brushing and use of interdental aids because they are socially desirable behaviors [11]. Therefore, measurements of full mouth plaque scores can be used to validate such self-reported measures.

Several models were proposed to conceptualize the utilization of health/dental services [135–140]. Access is a term refers to potential access to health service based on enabling resources while utilization is the actual use of service [136]. Barriers to utilize health services can be structural such as accessibility (transportation), availability of resources, and accommodation (availability of appointments); financial barriers include affordability; personal barriers include acceptability (including trust in dental care providers), psychosocial factors, and awareness [135, 138, 140]. Populations with no (or minimal) access to dental care had an overall higher periodontitis prevalence, extent, severity, and progression compared to estimates from general populations [11, 17, 18, 22, 35, 37, 38, 60, 65]. Frequency of dental visits had positive associations with periodontitis in adults [81, 82] and young population (15 to 21 years) [69].

3. Periodontitis prevention: are we there yet?

The ultimate goals of studying the etiology of diseases are to plan, implement, and evaluate interventions to combat disease burden [141]. Clinical interventions, mostly based on behavioral theories, had positive periodontal outcomes for smoking cessation, diabetes control, dietary habits modifications, weight control, and increased physical activity [142]. The study periods ranged from few weeks to 24 months. Despite the positive outcomes for changing behaviors by clinicians, such strategies can be potentially challenging, costly, time-consuming, and have temporary or limited effectiveness if not implemented as multilevel strategies [2, 3, 10]. Economic burden of periodontitis in 2018 was estimated to be ≥150 billion dollars in the United States and ≥ 150 billion euros in Europe [143]. However, up to most recent recommendations to prevent periodontitis were mostly confined to individual clinical care [142, 144–146]. A recent review also indicated that preventive strategies for periodontitis at population level are almost non-existent [51].

Among several approaches of defining periodontitis cases that evolved over history, current classification system by the AAP and European Federation of Periodontology (EFP) seemingly has resolved several longstanding issues [75, 147]. Periodontitis is mainly defined under single category rather than relying on age. Such approach may help overcoming issues of discontinuity in reporting findings for younger and older individuals to better understand the effect of age on periodontitis [11]. Periodontitis staging follows the clinical stages of natural course of disease, and its practical implications were explicitly outlined [75]. Stage I and II were classified

separately, though, their practical implications were similarly confined to non-surgical periodontal treatment. Stage III and IV mainly require surgical periodontal treatment and the latest additionally requires functional rehabilitations. The grading aims were to identify the progression rate, responsiveness to therapy, and possible impact on systemic health. The grading is assigned based on rate of progression and clinical phenotype then modified based on two risk factors: glycemic control and cigarette smoking. In contrast to staging, the practical implications of periodontitis grading were not explicitly suggested. Staging can be readily applied in population-based studies while grading's application can be limited due to relying on radiographic examinations, could fluctuate over time, and limited to two risk factors (diabetes mellitus and smoking) [11]. However, other exposures could also have direct impact on patient care including barriers to utilize dental care (e.g., anxiety, lack of trust, lack of awareness, lack of perceived need for care) which may impede patients from seeking clinical care or complying with supportive periodontal therapy. In addition, the staging/grading framework is limited to the latest stages of the natural course of disease; therefore, its implications are confined to individual patient care.

While most of the periodontitis exposures can have practical implications for different stakeholders, recommendations based on too many exposures related to periodontitis alone can be exhaustive and impractical [11]. The common risk factor approach to integrate prevention of several chronic diseases (e.g., cardiovascular disease, diabetes mellitus, and obesity) has been suggested [50, 51]. This approach simultaneously targets risk factors/indicators of several chronic diseases such as tobacco consumption, unhealthy diet, sedentary lifestyle, lack of glycemic control, and psychological stress when planning population-based strategies. The common risk factor approach might also be more manageable at the population level rather than suggesting disease specific preventive approaches. Also, duplication of efforts in combatting diseases can be avoided [50]. This can be especially important when considering that periodontitis is not a life-threatening disease and may not be perceived as a priority by policymakers compared with other chronic diseases (e.g., cardiovascular diseases). It is important to consider, however, that common risk factor approach can achieve its benefits the most when applied as a multilevel strategy. When considering common risk factor approach in clinical care, there is a possible need for integrated dental and medical care. Dentists may routinely refer their patients for medical care, but more help is required from medical care providers to refer their patients to dentists once they are diagnosed with chronic medical conditions such as diabetes mellitus [11]. Patients may prioritize medical over dental care either due to having more serious consequences, dental anxiety, or limited dental insurance coverage [11, 60].

3.1 Periodontitis prevention as a multilevel strategy

A framework suggested by the author for applying periodontitis prevention at multilevel was outlined in **Table 1** [11]. Periodontitis diagnoses integrated early stages (susceptibility stages) of the natural course of disease with the AAP/EFP staging criteria [75]. The diagnostic criteria follow the assumption that staging is irreversible; once a diagnosis is assigned, it can only be upgraded [75]. The common risk factor was a recommended approach for all levels of prevention along the natural course of disease. The framework may also enhance knowledge translation where evidence from clinical/epidemiological research can be applied in clinical, population, and public health practice and vice versa.

	Susceptibility stages (Pre-periodontitis)		Periodontitis stages		
	Stage of underlying exposures	**Stage 0 (Periodontitis risk factors/indicators)**	**Stage I + II**	**Stage III**	**Stage IV (Terminal stage)**
Staging diagnostic criteria	Underlying exposures (risk determinants). Non-modifiable: aging, males, and ethnicity. Modifiable: Social determinants of health (social and environmental constraints).	Underlying exposures. Periodontitis exposures/risk factors/risk indicators. Health behaviors: smoking, lack of utilization of dental care, unhealthy diet, and sedentary lifestyle. Medical exposures: diabetes mellitus, obesity, other chronic diseases.	Underlying exposures. Periodontitis exposures/ risk factors. CAL: 1–4 mm. No indications for surgical periodontal treatment.	Underlying exposures. Periodontitis exposures/ risk factors. CAL ≥5 mm. Indications for periodontal surgery: PPD ≥6 mm, osseous defects (Vertical bone loss, craters), class II, or III furcation involvements.	Underlying exposures. Periodontitis exposures/risk factors. CAL ≥5 mm. Indications for periodontal surgery: (PPD ≥6 mm, osseous defects (Vertical bone loss, craters), class II, or III furcation involvements. Loss of masticatory functional stability, excessive teeth mobility, drifting, and teeth loss due to periodontitis (≥5 teeth).
Level of prevention	Primordial	Primary	Secondary	Tertiary	
Stakeholders	Mainly policy makers and governments.	Mainly public health professionals but also includes dentists/ hygienists and medical professionals.	Dentists/ hygienists, and medical professionals.	Periodontists and medical professionals.	Periodontists, other dental specialities, and medical professionals.
Aims of prevention	Targeting the underlying exposures to prevent the development of periodontitis risk factors/ exposures.	Targeting the underlying and periodontitis risk factors/ exposures to prevent periodontitis incidence.	Targeting underlying and periodontitis risk factors/ exposures to prevent disease progression.	Targeting underlying and periodontitis risk factors/ exposures to prevent disease progression and complications.	Targeting underlying and periodontitis risk factors/ exposures to prevent further disease deteriorations.

	Susceptibility stages (Pre-periodontitis)		Periodontitis stages		
	Stage of underlying exposures	**Stage 0 (Periodontitis risk factors/indicators)**	**Stage I + II**	**Stage III**	**Stage IV (Terminal stage)**
Prevention strategies	Population-based prevention strategy: Use of common risk factors approach for combatting chronic diseases such as mass awareness campaigns regarding oral health and general health, imposing sin tax on tobacco and added sugar containing products, applying strategies for increasing resources for population with low SES such as facilitating dental and health insurance, improve access to dental and medical care, and increase outdoor/indoor spaces for physical activity.	High-risk prevention strategy: use of common risk factors for combatting chronic diseases for high risk individuals: Increasing awareness campaigns for promoting periodontal health and general health in susceptible individuals/ population groups including advocating for healthy diet, regular dental visits, regular medical visits, smoking cessations, increase physical activity, management of anxiety/ stress reduction protocols, and prophylaxis (removal of plaque, and calculus) by dental care provider.	Prevention by clinical care: Applying common risk factor approach for patients including advocating for healthy diet, regular dental visits, regular medical visits, smoking cessations, increase physical activity, and glycemic control by regularly taking medications, management of anxiety/ stress reduction protocols, promotion of oral-self-care, and prophylaxis (removal of plaque, and calculus) by dental care provider.	Prevention by clinical care: Applying common risk factor approach for patients including advocating for healthy diet, regular dental visits, regular medical visits, smoking cessations, increase physical activity, and glycemic control by regularly taking medications, management of anxiety/ stress reduction protocols, promotion of oral-self-care, prophylaxis (removal of plaque, and calculus) by dental care provider, and surgical periodontal treatment.	Prevention by clinical care: Applying common risk factor approach for patients including advocating for healthy diet, regular dental visits, regular medical visits, smoking cessations, increase physical activity, and glycemic control by regularly taking medications, management of anxiety/stress reduction protocols, promotion of oral-self-care, prophylaxis (removal of plaque, and calculus) by dental care provider, surgical periodontal treatment, and multidisciplinary functional rehabilitation.

Underlying exposures include non-modifiable exposures including age, sex, and genetics or modifiable exposures such as the social determinants of health. Periodontitis risk factors/exposures include medical and behavioral exposures that could have more direct association with periodontitis. CAL: Clinical Attachment Loss, PPD: Periodontal Probing Depth, SES: Socioeconomic status.

Table 1.
A multilevel prevention strategy for periodontitis with integrated use for individual clinical care and population/public health [11].

3.1.1 Susceptibility stages

In *stage of underlying exposures*, primordial prevention is applied to the population, and it aims to reduce development of disease specific risk factors including adopting unhealthy behaviors [11]. This level of prevention mainly benefits the population at an early stage in development, that is, during childhood and teenage [148, 149]. The strategies can be mainly applied by governments and policy makers to target the social determinants of health and take into consideration the non-modifiable exposures. Examples of preventive strategies include imposing sin tax on tobacco- and added sugar containing products, increase safe public places for indoor or outdoor physical activities, and reduce barriers to access dental and health care such as facilitating health and dental insurance for low profile jobs. The latest can be accompanied by conditional strategies that enforce regular attendance to dental/medical care.

In *stage 0*, primary level of prevention can be applied to individuals or population subgroups who developed periodontitis specific risk factors/indicators [11]. It aims to reduce periodontitis incidence and strategies can be mainly applied by public health practitioners. Examples include health awareness campaigns and use of behavioral theories to apply public health interventions for smoking cessation, glycemic control, modification of dietary habits, and weight loss.

3.1.2 Periodontitis stages

Periodontitis stages have similar diagnostic criteria proposed for periodontitis staging by the AAP/EFP [75]. Exposures of susceptibility stage still apply to periodontitis stages based on the current understanding that periodontitis develops in a susceptible host. The grading criteria were removed since their application may not be suitable for population-based studies due to the abovementioned limitations [11]. Periodontitis stages are mainly managed by clinicians following the secondary and tertiary levels of prevention. An additional strategy is to apply the common risk factor approach in clinical stages including the need for integrated dental and medical care. Medical professionals need to refer their patients once diagnosed with chronic systemic disease. Stage I and II were combined since they are managed similarly by non-surgical periodontal treatment. Diagnosis of stage I can be challenging when assessments are conducted by non-periodontists due to the use of low disease threshold. Population-based surveys commonly consider CAL ≥3 mm as minimum threshold to avoid misclassifying cases [92, 93, 150]. However, diagnosing periodontitis cases at lower thresholds allows early interventions, which can be particularly important in younger population [11].

3.2 Additional considerations for the multilevel prevention framework

Despite that gingivitis precedes periodontitis, it was not included in the framework because pristine gingival health may not exist even under optimal oral self-care [151–154]. Also, gingivitis can be reversible or self-limited. However, periodontitis staging is assigned with a purpose of being irreversible. Similarly, plaque per se is a universal finding even in individuals following stringent plaque control strategies [34, 155–157]. Plaque needs to be controlled in all members of the population. In addition, plaque is a reversible exposure, which makes it unsuitable for periodontitis staging.

When applying multilevel preventive strategies, it is recommended to consider outlining a specific plan for each exposure at a time [2]. For example, strategies for combatting tobacco consumption at population level could include imposing sin tax, age restrictions, and increase smoke-free spaces [6]; for smokers population subgroup, smoking cessation interventions and awareness campaigns can be implemented; for periodontitis patients, smoking cessation strategies including referral to smoking cessation clinics can be applied.

4. Concluding remarks

Key messages that need to be emphasized include:

- Variation in distribution is a key concept for studying diseases and their exposures. Therefore, using mean scores while disregarding the multilevel variations in periodontitis severity or annual progression rates makes these population-based summaries have questionable validity.

- Evidence suggests that majority of variations in periodontitis distribution in populations occur between 20 and 40 years. Thus, studies on associations with periodontitis need to take this age range into consideration.

- A major gap in knowledge regarding periodontitis and its associations is understanding the transition between adolescence and adulthood. Limited evidence suggests that exposures such as socioeconomic status may have its major impact during early life. However, the focus of current research on associations is mostly confined to adulthood.

- The use of life course approach is useful for understanding the pathways of associations with periodontitis, the nature of their impacts, their latency period, their impacts on health behaviors, and biology. However, since feasibility is a major limitation for such study designs, evidence from cross-sectional studies using wide age ranges may help in generating hypotheses and identifying the most appropriate assessment periods.

- The use of ecosocial theory by Krieger can be useful for generating hypotheses for mapping pathways of associations with periodontitis while considering the broader context over the life course [54].

- When studying the psychosocial exposures, it is important to consider both of their protective and harmful impacts.

- The periodontal treatment status needs to be considered in studies on associations with periodontitis. Consequently, the impacts and limitations of current periodontal treatment strategies can be evaluated at population-level.

- A multilevel periodontitis prevention framework based on common risk factor approach was suggested as an alternative to current approaches, which are limited to individual patient care.

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