Review Article

The Nexus of Mouthwash, Oral Dysbiosis, and Cardiovascular Health: A Review

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ABSTRACT

The human oral cavity is home to a diverse microbial community consisting of bacteria, fungi, archaea, protozoa, and viruses that thrive on various surfaces. These bacteria influence oral hygiene and also play a vital role in cardiovascular homeostasis. Oral Hygiene has become one of the most important factors of overall health and a crucial determinant of social health. The mouthwash and oral hygiene market is estimated to include 202.58 million consumers in 2024. Regular use of mouthwashes has been shown to improve oral and dental hygiene; however, mouthwash-induced oral dysbiosis has become a concern. It is found to induce a proinflammatory state that promotes atherosclerosis, worsens insulin resistance and hypertension, and increases the risk of cardiovascular disease. The guidelines for determining the use of mouthwash that takes into account both the benefits and harm associated with its use are not clear and are a topic of further research. The complex interplay between oral dysbiosis, oral hygiene, and cardiovascular outcomes highlights the need for a comprehensive understanding of these relationships to improve oral and cardiovascular health. This review focuses on the pathophysiology of mouthwash-induced oral dysbiosis and cardiovascular function and discusses various evidence to compare the pros and cons of mouthwash usage.

Key words: Mouthwash, Cardiovascular disease, oral microbiota, Chlorhexidine

The oral cavity provides one of the best-suited ecosystems in the human body, which supports communities of aerobic and anaerobic bacteria, fungi, archaea, protozoa, and viruses. The various surfaces of the mouth, like subgingival crevice, dorsum of the tongue, hard palate, tonsils, teeth, and dental implants, act as a setting for these microorganisms to flourish [1]. The colonization of oral bacteria begins immediately after birth and occurs in two phases: an early phase before dental eruption and later with the emergence of primary teeth. The early colonization is a foundation for later communities to develop and is also influenced by maternal microbiomes [2]. The oral microbiome is also disrupted by several lifestyle habits like alcohol consumption, smoking, vaping, and oral hygiene practices [1].

Oral hygiene involves cleaning both the hard and soft tissues within the mouth, including the teeth, gums, tongue, fixed dental prosthetics, oral devices, and dentures [3]. Proper oral hygiene enhances oral health, affects social interactions, and, according to many studies, is closely linked to overall health and quality of life [4, 5]. To enhance oral and dental well-being, using oral rinse and mouthwash has become a crucial component in oral hygiene practices. Mouthwashes can be classified into multiple categories based on the predominant chemical ingredient in their formulation. The oldest known formulation is an isotonic solution of sodium bicarbonate, which can neutralize acids, moisturize, and decrease oral commensals [6]**.** Other commonly available mouthwashes contain essential oils like menthol, fluorine compounds like sodium fluoride, glycerol, and ethyl alcohol [7].

Individuals use mouthwashes for various purposes, like refreshing breath or as a component of a healthy routine for plaque removal, aiming to decrease oral bacteria and enhance oral well-being. Another essential aspect of mouthwashes is their ingestion as a surrogate alcohol when other alcoholic beverages are not available. Mouthwash ingestion has been seen in restricted settings such as hospitals and prisons, and particularly in teenagers who are unable to purchase alcohol in

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stores [8, 9]. Most of these mouthwashes have ethanol contents that are higher than the strength of beer or wine, usually ranging from 20% to 30% by volume [8].

Mouthwash is widely accessible in numerous countries through various brand names such as Listerine, Crest, and Colgate, and is frequently recommended by dental professionals to aid in addressing oral issues [5, 10]. In 2018, Listerine dominated the mouthwash market in the United States with sales surpassing 354 million U.S. dollars, while private label products followed as the second highest-selling category with sales reaching 199.2 million U.S. dollars [11]. According to the U.S. Census information and the Simmons National Consumer Survey (NHCS), approximately 199.56 million individuals in the United States utilized mouthwash or dental rinse in 2020, and projections suggest this number will climb to 202.58 million by 2024 [12]. In a study conducted by Macfarlane et al., mouthwash usage rates declined with age, dropping from 56% among those aged 25 to 34 to 36.4% among individuals aged 65 to 74 ($P < 0.001$). In the same study, women exhibited a higher tendency to use mouthwash compared to men, with rates of 47.2% and 41.5%, respectively ($P = 0.004$) [13].

Suboptimal oral health is linked to adverse consequences because of heightened chances of systemic and respiratory issues [5]. Compelling evidence specifically links periodontal bacteria and tooth loss to various systemic conditions, notably cardiovascular diseases like high BP [14]. For individuals with periodontal disease (PD), the risk of developing cardiovascular disease (CVD) doubles. Inflammatory conditions like periodontal diseases trigger the production of inflammatory substances like tumor necrosis factor α (TNF-α), interleukins (IL-1, IL-6, IL-8), and C-reactive protein (CRP), which are associated with the formation of atherosclerosis. Moreover, specific bacteria present in the oral flora, such as Porphyromonas gingivalis (Pg), Aggregatibacter actinomycetemcomitans (Aa), and Streptococcus sanguis (Ss), have been detected in atherosclerotic plaques, likely due to transient bacteremia by crossing through the dental sulcus [15].

Regular utilization of mouthwashes containing chlorhexidine, essential oils, and cetylpyridinium chloride, along with regular tongue brushing, can impact the levels of oral bacteria associated with nitric oxide reduction. Antiseptic mouthwash disrupts the nitrate-nitrite-NO pathway through mechanisms that impact the reactions to L-arginine, which could have implications for individuals with cardiovascular conditions [16]. Reduced generation or function of nitric oxide, caused by endothelial dysfunction, contributes to the development of numerous cardiovascular conditions, such as atherosclerosis and cardiovascular diseases like hypertension and coronary artery disease [14]. Considering that oral hygiene is associated with cardiovascular health, in this article, we will review the effect of the use of mouthwash on the risk of cardiovascular diseases.

Pathophysiology

Oral dysbiosis is characterized by an imbalance in the composition, diversity, or function of the oral microbiome, leading to detrimental host-microbial interactions. This imbalance arises due to factors like antimicrobial mouthwash use, smoking, poor oral hygiene, or systemic diseases. [9] Oral dysbiosis induces local inflammation and increases mucosal permeability, facilitating systemic translocation of pathogenic microbes and inflammatory mediators. Consequently, these systemic inflammatory responses can adversely impact cardiovascular health by promoting endothelial dysfunction, vascular inflammation, oxidative stress, and metabolic disruptions, thus serving as a significant risk factor for cardiovascular conditions such as atherosclerosis, heart failure, and hypertension. [9, 10]

The oral cavity is home to a vast variety of microorganisms, such as bacteria, fungi, viruses, and bacteriophages, making it one of the most significant microbial reservoirs in the human body [17]. Mouthwashes may induce "dysbiosis," a condition in which some bacteria are eliminated and others—sometimes undesirable—take over [1]. Dysbiosis is a term wherein the composition or distribution, or metabolic activities of the microbiota have tipped in such a way that it results in harmful host-microbial interaction. While in vitro studies show that mouthwashes are effective at targeting and killing harmful bacteria [18], thereby treating and preventing disorders such as dental plaque, gingivitis, and periodontitis [19]. They have, however, been found to affect the composition and diversity of the oral microbiome. Much of the studies on the effect of mouthwash on oral microbiota have been done with chlorhexidine, and it has been shown to inhibit even the beneficial microbiota in healthy subjects [1].

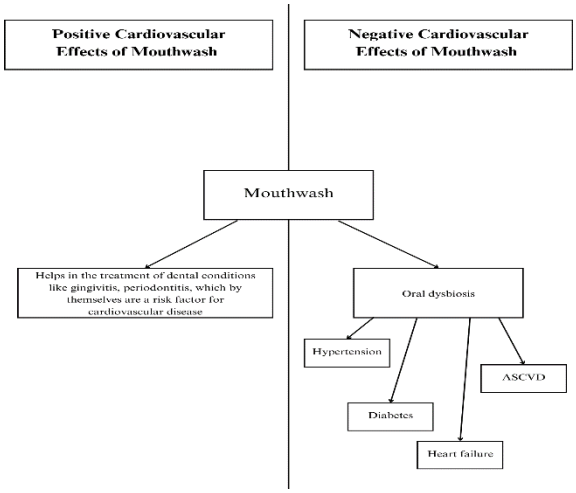

Figure 1 - Effects of mouthwash on the Cardiovascular system.

Effect of Oral Dysbiosis on Atherosclerosis

Several independent mechanisms have been described explaining the role of oral dysbiosis in ASCVD. One of the mechanisms points to the effect of Oral dysbiosis on chronic low-grade systemic inflammation. The local inflammation and the resultant increase in the local vascular permeability have been shown to cause seeding of the oral bacteria and their components, like the lipopolysaccharide, flagellin, and teichoic acid, into the systemic circulation [21]. These bacteria and their components then activate the innate immune system, inducing a generalized Chronic low-grade inflammation with resultant release of pro-inflammatory cytokines like the TNF alpha and the interleukins. These pro-inflammatory cytokines cause endothelial dysfunction, which is an early marker of atherosclerosis [22]. In another mechanism, the occurrence of oral microbiota within atherosclerotic plaques serves as evidence that bacteremia may contribute to inflammation within these plaques [23].

It also adversely affects the lipid profile, increasing the LDL and the triglycerides, another risk factor for atherosclerotic plaque progression [24]. Moreover, Oral dysbiosis has also been shown to increase the risk of autoantibody formation, a mechanism called molecular mimicry. These antibodies bind to host proteins in the atherosclerotic plaque and increase the subsequent macrophage recruitment, which causes the progression of atherosclerosis. Oral dysbiosis is also a risk factor for periodontitis, and the role of periodontitis in cardiovascular disease is a well-known association [25]. Mechanisms, including molecular mimicry and bacteremia, have been linked to increased systemic inflammation [26] and progression of heart failure and diabetes in patients with periodontitis.

Effect of oral dysbiosis on Heart failure

Heart failure (HF) is a complex clinical syndrome caused by structural and/or functional cardiac abnormalities that result in the impairment of ventricular filling and/or ejection. Inflammation has been recognized as an important pathophysiological factor throughout the spectrum of heart failure, including HFrEF, HFpEF, and the ADHF [27]. Chronic systemic inflammation from oral dysbiosis has been shown to activate the cardiac fibroblasts, leading to cardiac fibrosis and worsening heart failure [28]. This is mediated by TGF-beta which is released by the macrophages that have infiltrated the heart in response to the pro-inflammatory signaling [29] Another similar mechanism has been described wherein the acid resistant oral microbiota could travel to the gut to cause gut dysbiosis with resultant increased gut permeability ultimately leading to endotoxemia and bacteremia contributing to the chronic systemic inflammation with subsequent effects on heart failure, atherosclerosis and insulin resistance[30].

Effect of oral dysbiosis on hypertension

Mouthwash like Chlorhexidine, due to its non-specific antimicrobial effects, has been shown to suppress even beneficial microbes like *Veillonella*, *Actinomyces*, *Haemophilus*, *Rothia*, and *Neisseria*, which are nitrate-converting bacteria [20]. Nitric oxide (NO) is a vasodilator and plays a vital role in controlling BP (BP). The body primarily obtains NO from the exogenous nitrate–nitrite–NO pathway and the endogenous L–arginine–NOS pathway [31, 32]. The endogenous pathway generates NO from the precursor amino acid L-arginine. The NO produced thereby is quickly oxidized to nitrite and nitrate, and together with nitrates present in food, is absorbed into the salivary gland. The deep clefts of the dorsal surface of the tongue harbor nitrate-reducing anaerobic bacteria, which eventually reduce nitrates to nitrites through the enzyme nitrate reductase. Once swallowed, these nitrites are quickly absorbed into blood and, along with nitrites formed from the endogenous pathway, exhibit their vasodilatory effect [31, 33]. Oral dysbiosis has been shown to increase both the systolic and diastolic BP by 2-3.5 mm Hg [34].

DISCUSSION

Mouthwash and effects on BP and Hypertension

Mouthwash is used to maintain oral hygiene, improve the reduction of plaque biofilm, and kill bacteria in areas difficult to reach with routine oral care [35]. Recently, due to the demonstrated importance of the oral microbiome in regulating BP by maintaining a functional nitrate-nitrite-nitric oxide pathway and reducing insulin resistance, it has become essential to strike a delicate balance in eradicating pathogenic bacteria without affecting beneficial oral commensals [36, 37]. Chlorhexidine mouthwash has been shown to have a detrimental effect on such oral flora; it decreases the number of nitrate-reducing bacteria, reducing the production of nitrite, a vasodilator, thus affecting the cardiometabolic health [38].

A crossover study was done to investigate whether the suppression of oral microflora by mouthwash use affected the BP of individuals. The study was conducted on 19 healthy volunteers for a 7-day control and a 7-day testing period with chlorhexidine mouthwash. It was seen that the antiseptic mouthwash use increased both the systolic and diastolic BP by 2-3.5 mm Hg, along with a decrease in circulating nitrite concentrations ($r^2 = 0.56$, $p = 0.002$). Oral nitrite production was decreased by 90% ($p<0.001$) and plasma nitrite levels by 25% ($p=0.001$) [39]. Additionally, a three-year long follow-up longitudinal study done including 40- to 65-year-old overweight/obese individuals noted that incidence of hypertension was higher in people who used mouthwash twice/day or more compared to less frequent users (Incidence Rate Ratio=1.85, 95% confidence interval: 1.17, 2.94), and compared to non-users (IRR= 2.17, 95% confidence interval: 1.27, 3.71). The study also highlighted that since most of the

mouthwashes had some level of antibacterial properties, the detrimental effect of mouthwash on BP can be seen with almost all types and brands of mouthwashes [40].

A randomized controlled crossover study done on patients treated for hypertension showed that the use of antibacterial mouthwash could have a detrimental effect on BP. For these patients, a 3-day use of antibacterial mouthwash resulted in a noticeable rise in home systolic BP (2.3 mmHg, p = 0.01) [41]. Although the increase in systolic BP is rather small (about 2 - 3.5 mmHg), according to estimates, the mortality rate from ischemic heart disease and stroke increases by 7 and 10%, respectively, with every 2 mmHg increase in systolic BP [42]. On the flip side, a single-blinded crossover study including healthy vegetarians and healthy omnivores aged between 18 and 45 years was conducted to detect the differences in systolic BP after using antibacterial mouthwash. It was found that though antibacterial mouthwash significantly reduced the abundance of nitrate-reducing oral bacteria in both vegetarians and omnivores, this did not correspond to an increase in BP and resting metabolic rates in both groups ($p > 0.05$) [43].

Mouthwash and effects on CVD Mortality

A recent long-term study in Finland conducted for approximately 19 years showed that good oral hygiene encompassing both brushing and flossing significantly lowered the risk of CVD mortality. Even the patients who had CAD at baseline experienced some benefit in CVD mortality with good oral hygiene ($p = 0.07$). Additionally, the study also revealed that the effect of independent usage of routine chlorhexidine mouthwash on CVD mortality was not meaningful (HR 0.95, p=0.89). Thus, stating that mouthwash use did not have any long-term benefit or harm on CVD mortality [44]. Hence, due to conflicting evidence and lack of proper associations, we are still in need of more double-blinded, randomized control trials to conclude this case.

As the risk factors of hypertension and diabetes coincide and the reduction in production of NO has been linked to increased insulin resistance, it is important to explore the links between mouthwash use and acquiring pre-diabetes/diabetes [45]. A three-year longitudinal study recruited overweight/obese individuals aged 40-65 years who were free from diabetes and any major cardiovascular diseases. The study suggested that the people who used mouthwash more than twice per day had more than double the risk of developing pre-diabetes or diabetes (multivariate IRR= 1.55, 95% CI: 1.21- 1.99). However, non-frequent users did not show a significant association with the risk of developing pre-diabetes/ diabetes. The study controlled for the majority of risk factors associated with diabetes [10]. Thus, suggesting that judicious use of mouthwash should be more encouraged to prevent any potential harmful effects of indiscriminate use of mouthwash.

Oral care with chlorhexidine mouthwash is the norm in critically ill patients, including those on mechanical ventilation. Several studies have evaluated the hospital mortality among such patients associated with their use of mouthwash. A per-protocol study consisting of 254 patients, 9.84% of the patients had adverse events like CHX-induced mucositis. This side effect was seen in patients using 2% CHX oral gel and was strongly and independently associated with death (RR= 2.42 [1.64 - 3.56], 95% CI). It also prolonged the length of ICU stay, mechanical ventilation, and longer periods of antimicrobial therapy. This indicates that the disruption of oral mucosa integrity due to the chlorhexidine mouthwash helps in dislocation of the bacteria into the bloodstream and serves as an independent factor in increasing morbidity and mortality [46].

An updated review of evidence done in 2014 that included 16 RCTs and 3630 patients showed that routine oral care with chlorhexidine prevents nosocomial pneumonia in cardiac surgery patients but may have no significant effect in reducing ventilator-associated pneumonia risk in non-cardiac surgery patients. This can be because cardiac surgery patients are extubated within a day, while non-cardiac surgery patients usually take 1 to 2 weeks before being extubated. The endotracheal tube can act as a reservoir for infections, and chlorhexidine oral care might remain insufficient to overcome those hazards [47].

While considering the above-mentioned facts, we should also keep in mind that dental infection is one of the most commonly encountered infections. The process of dental caries entails the dissemination of infection from the dentine to the periapical bone tissues and vascular dental pulp, followed by the involvement of nearby soft tissues and propagation of sepsis. Periodontitis is directly correlated with higher cardiovascular, cerebrovascular, and peripheral risk according to several case-control, cross-sectional, and cohort studies [48]. A systematic review done including 826 subjects aging from 16 to 62 years and 19 articles comparing the effects of using essential oil mouthwash (EOMW) and chlorhexidine mouthwash stated that – chlorhexidine (CHX) mouthwash was significantly better at reducing plaque formation than EOMW mouthwash in both short (<4 weeks) and long term (>4 weeks) studies. However, when it came to controlling gingival inflammation in the long term, CHX and EOMW mouthwashes were not different from each other [49].

Although CHX mouthwashes are the most relied upon for preventing plaque buildup, some of the side effects of using CHX mouthwashes include brownish discoloration of teeth, alteration in taste sensation, and an increase in calculus formation [50]. A randomized, double blind, placebo-controlled parallel group of 42 patients with cardiovascular disease and type 1, type 2 diabetes was done with the aim of evaluating the effects of probiotic mouthwash in these patients in terms of plaque control and bleeding on probing. The study

revealed that probiotic mouthwash treatment is efficient in reducing plaque control record and bleeding on probing. However, the study was limited in terms of only one week of observation of the participants [51].

Thus, despite multiple studies being undertaken on the effects of various types of mouthwash, there is a lack of causation and a scarcity of analytical data required to reach a definitive conclusion about its long-term consequences. It is a topic for future research, thus, numerous large, long-term, randomized double-blind control trials would be required to analyze and compare the systemic effects of various formulations/types/brands of over-the-counter mouthwash in different populations [40]. Trials investigating the effects of various antibacterial agents in mouthwash, as well as the consequences of using mouthwash in populations with increased cardiovascular risk, would also contribute to a better knowledge of the effects of mouthwash on the cardiovascular system [41].

Table 1 - Recent studies of Mouthwash's effects on the cardiovascular system.

Author Year of Study	Study	Results	Study limitations/ Implications for future research
Kapil V et al [39] 2013	A crossover study with 19 healthy non-smoking volunteers in 18-45 years of age and BMI of 18-40 kg/m2, no systemic conditions, no self-reported use of mouthwash, or recent treatment with oral antibiotics was included.	After 7-day use of twice-daily antiseptic mouthwash, salivary nitrate levels were decreased by 90% ($p<0.001$) and plasma nitrite by 25% ($p=0.001$). BP rise was significantly related to the decrease in plasma nitrite concentrations ($r^2= 0.56$, $p=0.002$).	The study was non-randomized and non-placebo controlled. Small-scale study with 19 patients; hence, the larger-scale study would help generalize the findings.
Joshipura K et al [40] 2020	San Juan Overweight Adults Longitudinal Study (SOALS) recruited 40-65 years overweight/obese individuals. Out of the 1351 participants, 630 were excluded because of high BP at baseline and 176 did not complete the study, and 5 were excluded for missing data; thus, 540 participants were included in the final study.	12% of the participants developed hypertension. Frequent users had more risk of developing hypertension on follow-up than less frequent users (IRR= 1.85, 95% CI: 1.17, 2.94) and compared to nonusers (IRR= 2.17, 95% CI: 1.27, 3.71).	This observational study cannot establish causality. It did not assess oral nitrate-reducing activity. Large, long-term, randomized clinical trials would be more helpful in determining the causality.
Bondonno CP [41] 2014	A randomized control trial with fifteen men and women aged 50-70 years, who did not use antibacterial mouthwash, BMI between 20-35 kg/m2, with SBP between 120-159 mmHg and DBP less than 100 mmHg, taking between one and three antihypertensive medications, was recruited.	Compared to control, 3-day use of mouthwash resulted in a significant reduction in salivary nitrite (95% CI: 72, 150, P=0.01). There was a significant increase in home systolic BP (2.3 mmHg, 95% CI: 0.5, 4.0; P=0.01). However, no significant differences were noted in plasma and saliva nitrate and nitrite, and cGMP.	Further studies were recommended to study the effect of different antibacterial agents in mouthwash, and the consequences of using mouthwash in populations with higher cardiovascular risk.
Ashworth A et al [43] 2019	22 Healthy vegetarians and omnivores aged between 18 and 45 years who were non-smokers, did not have pre-existing medical conditions like hypertension, diabetes, or any dental condition like gingivitis, were included.	This single-blinded, non-randomized, crossover study revealed that after using antibacterial mouthwash, both plasma and salivary concentrations of nitrite were significantly lower in both groups. However, there were no significant differences noted in SBP, DBP, mean arterial BP, and resting metabolic rate ($p>0.05$).	This study did not randomize treatment and may have missed smaller effects on BP due to the smaller sample size. Variability in dietary nitrate estimates also has limited accuracy.
Janket SJ et al [44] 2023	Using Cox regression analysis, the link between OHS and CVD mortality was evaluated among 354 dentate subjects from the Kuopio Oral Health and Heart study. The study was controlled for age, sex, smoking, dyslipidemia, diabetes, hypertension, and education were taken into consideration.	In the multivariate-adjusted models, the study revealed that good oral health hygiene, which included brushing and flossing, was associated with a 51% reduction in CVD mortality (HR=0.49; p=0.07). There was a slight benefit seen for those who had CAD at baseline (0.50; p=0.07). Thus, indicating that there are no additional benefits or harm for using mouthwash.	This study may carry risk amplification from its original case-control design however, robust analysis mitigates this risk. Furthermore, oral hygiene frequency was assumed to reflect efficacy, which requires further validation.

Joshipura KJ et al [10] 2017	1206 overweight/obese individuals, aged 40-65 years, free from diabetes and major cardiovascular diseases, were recruited by the San Juan Overweight Adults Longitudinal Study (SOALS), among whom 945 completed the study and were included in the final analysis.	Participant who used mouthwash more or equal to twice per day had a significantly increased risk for acquiring pre-diabetes/diabetes compared to those who used it once daily (IRR=1.55, 95% CI: 1.21-199) and those who do not use mouthwash (IRR= 1.49, 95% CI: 1.13-1.95).	The study was limited in not evaluating nitrate/nitrate levels and nitrate-reducing oral bacteria. Did not collect data on the type of mouthwash and the reason for use.
Bellissimo-Rodrigues WT 2019 [46] 2019	A post hoc analysis of a randomized controlled trial included adult patients who were admitted to an ICU setting for at least 2 days between January 1, 2011, and August 8, 2013. The experimental group was provided dental care by a dentist, plus routine oral care, while the control group was provided with only routine oral care. Both groups used 0.12% CHX oral solution, if fully conscious, and 2% CHX gel if conscious, thrice a day for their ICU stay.	Out of the 254 patients as per the "per protocol" study, 25 of them developed adverse events related to oral care, most commonly CHX-induced mucositis. This adverse event was exclusively associated with patients who were exposed to 2% CHX oral gel (p=0.006).	The study did not assess whether CHX application increased the mortality of patients or not as all of them were exposed to it.
Klompas M et al [47] 2014	Randomized controlled trials that compared chlorhexidine and placebo in adults on mechanical ventilation were selected. 16 studies, which included 3630 patients, fit into the inclusion criteria.	Fewer respiratory infections were noted in cardiac surgery patients randomized to chlorhexidine (RR=0.56 [95% CI: 0.41-0.77]), whereas there was no significant difference in ventilator-associated pneumonia risk in non-cardiac patients (RR=0.88 [95% CI: 0.25- 2.14]).	The possible association between chlorhexidine use and increased mortality is not significant. Analysis was limited by wide confidence intervals and significant variability across the studies.
Neely AL et al [49] 2012	The systematic review included short-term (<4 weeks) and long-term (>4 weeks) studies comparing the effects of essential oil mouthwash (EOMW) with chlorhexidine (CHX) on plaque and calculus accumulation, tooth staining, and gingival inflammation. The final study included 19 articles with a total of 826 subjects, aged 16 to 62 years, who completed all trials.	The authors concluded that CHX is the first choice when plaque control is the main goal of the treatment. However, it also suggested that EOMW could be a reliable option in controlling gingival inflammation, as CHX and EOMW were not different from each other in the long-term control of gingival inflammation.	The study was limited by variability in study design, patient population, and treatment protocols, which affect the generalizability of results.
Bollero P et al 2017	The study included 42 participants with either type 1 or type 2 or cardiovascular disease (CVD). In the intervention group (IG) and control group (CG), plaque control record (PCR) and bleeding on probing (BOP) were measured at baseline and two weeks after probiotic mouthwash (PM) or positive control treatment.	Reduction in PCR was noted in all treatments and samples (p<0.01). Bleeding on probing was also significantly reduced in all the samples except for IG in the CVD sample (p=0.15).	The study was limited by small sample size, short duration, and lack of a control group, which affects the long-term validity of the findings.

CONCLUSION

Oral health, particularly periodontal conditions, has been intricately linked to systemic diseases, notably cardiovascular diseases like atherosclerosis and heart failure, through mechanisms including chronic inflammation, bacteremia, lipid profile alterations, and autoantibody formation. Mouthwash usage, a pivotal component of oral hygiene routines, varies widely in its chemical formulations and purposes, impacting overall oral health and well-being. The popularity and accessibility of mouthwashes underscore their significance in dental care, with a considerable number of individuals utilizing them worldwide. The use of mouthwashes has also been shown to have detrimental effects on the cardiovascular system, involving hypertension, atherosclerosis, and heart failure by inducing oral dysbiosis. However, there is still a lack of definitive causality and substantial analytical data to draw firm conclusions regarding the long-term effects of mouthwash, and further research is needed to fully understand the effects of mouthwash on the cardiovascular system.

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