



## **Oral Health: A Guide for Your Health as a Periodontist's Point of View**

**Vinay H. Vadvadgi<sup>1</sup>, Amit Mani<sup>1</sup>, Neeta S. Padmawar<sup>2\*</sup>  
and Lingraj Harihar<sup>3</sup>**

<sup>1</sup>Department of Periodontology, Rural Dental College, Pravara Institute of Medical Sciences (Deemed to be University), India.

<sup>2</sup>Department of Pediatric & Preventive Dentistry, Rural Dental College, Pravara Institute of Medical Sciences (Deemed to be University), India.

<sup>3</sup>Department of Oral Medicine, Diagnosis & Radiology, P.M.N.M Dental College & Hospital, Bagalkot, India.

### **Authors' contributions**

*This work was carried out in collaboration among all authors. Author VHV managed the literature searches and wrote the first draft of the manuscript. Author AM guided during designing manuscript. Author NSP managed the literature searches and reviewed of final draft of the manuscript. Author LH managed the literature searches. All authors read and approved the final manuscript.*

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### **ABSTRACT**

Dental caries and Periodontitis are the most commonly reported dental diseases. These can lead to loss of tooth structure and compromising the functions of teeth like mastication and thus affecting the overall health. Periodontitis is inflammation of periodontium resulting in loss of periodontal ligament attachment, bone destruction, tooth mobility and ultimately tooth loss. This is caused by the microorganisms present in the oral biofilm. One cubic millimeter of dental plaque contains about 100 million bacteria. At present almost more than 500-600 different varieties of bacteria have been identified in the oral cavity. Key perio-pathogens are the group of perio-pathogens that are responsible for the commencement and progression of periodontal disease as well as failed periodontal therapy. *A. actinomycetemcomitans*, *Tannerella forsythia* and *Porphyromonas gingivalis* are the established key-pathogens in the various periodontal diseases.

\*Corresponding author: E-mail: [opneeta23@gmail.com](mailto:opneeta23@gmail.com);

Through blood stream, these micro-organisms can be transported to various organs or system in the human body and causing and affecting overall health negatively. Endotoxins produced by these key perio-pathogens are associated with the non-oral diseases. It is a proven fact that periodontal health plays an important role in general health status in mankind. Periodontal pathogens can affect the systemic diseases and conditions adversely and can lead to unfavourable outcomes. Patients with cardiovascular diseases showed pathogens having same DNA as periodontal pathogens. In periodontitis patients, inflammatory mediators produced can trigger the hyperglycaemia. In pregnant women, premature birth and low birth weight is found linked with poor periodontal health. This paper highlights the role of periodontal health in various systemic diseases and conditions for better treatment planning and prevention of the adverse outcomes.

*Keywords: Periodontitis; keypathogens; systemic health; adverse effect.*

## 1. INTRODUCTION

Dental caries and Periodontitis are the most commonly reported dental diseases. These can lead to loss of tooth structure and compromising the functions of teeth like mastication and thus affecting the overall health.

Periodontitis is inflammation of periodontium resulting in loss of periodontal ligament attachment, bone destruction, tooth mobility and ultimately tooth loss. This is caused by the microorganisms present in the oral biofilm. The destruction of the periodontium is associated with the presence of gram-negative anaerobic bacteria localized in the subgingival region, and include typically *Porphyromonas gingivalis* (Pg), *Prevotella intermedia* (Pi), *Actinobacillus actinomycetemcomitans* (Aa), and *Bacteroides forsythus* (Bf) are the initiator for periodontal diseases and *Aggregatibacter actinomycetemcomitans*, *Fusobacterium nucleatum*, *Prevotella intermedia*, *Campylobacter rectus*, *Peptostreptococcus migros*, *Eikenellacorrodens* are required for the progression of the disease [1].

Periodontitis can be mild, moderate or severe type depending upon the clinical and radiographic presentations. According to a survey in 2013, the prevalence of mild periodontitis was found in 35% and moderate to severe periodontitis in 11% of study population [2].

In severe periodontitis cases, along with severe clinical manifestations; elevated levels of inflammatory mediators like CRP, hyperfibrinogenemia, moderate leukocytosis and IL-1 and IL-6 were found as compare to healthy counterparts [3,4,5,6,7].

In 1900 a British Doctor William Hunter based upon his clinical experience postulated a concept of "focal infection" in which he emphasized on the there is a link between oral pyemia and systemic health. He further said that if infected tooth is extracted or removed, general health of an individual improved [8].

Through blood stream, micro-organisms causing periodontitis can be transported to various organs or system in the human body and causing and affecting overall health [9]. Endotoxins produced by gram negative anaerobic micro-organisms are associated with the non-oral diseases [10].

### 1.1 Periodontal Pathogens

One cubic millimeter of dental plaque contains about 100 million bacteria .At present almost more than 500-600 different varieties of bacteria have been identified in the oral cavity [11]. These bacteria grow on the tooth surface, gingival margin and subgingival margin. Among these bacteria, only small amount of bacteria can cause periodontal disease [12]. These bacterias can be anaerobes, aerobes, capnophiles and microaerophiles; but most of periodontal pathogens are anaerobes and this contribution depends on environment of the periodontal pocket. These possible etiological pathogens should have ability to grow subgingivally, produce enzymes and toxins and or antigens and inflammatory mediators which will commence inflammatory reaction leading to injury or destruction of periodontal tissue [13,14]. key perio-pathogens are the group of perio-pathogens that are responsible for the commencement and progression of periodontal disease as well as failed periodontal therapy. *A. actinomycetemcomitans*, *Tannerella forsythia*

and Porphyromonas gingivalis are the established key pathogens in the various periodontal diseases [15]. Other than these species, subgingival species like Prevotella intermedia, Prevotella nigrescens (formerly P. intermedia), Bacteroides forsythus, Fusobacterium nucleatum, Campylobacter rectus, Eikenellacorrodens, Treponema denticola, Micromonas micros (formerly Peptostreptococcus micros) and some other species are also present subgingivally.

This article gives a glimpse of the relation between periodontal pathogens and various systemic conditions like cardiovascular disease, diabetes mellitus, osteoporosis, colon cancer, premature deliveries etc.

## 2. MECHANISM OF EFFECT OF PERIODONTAL PATHOGENS ON SYSTEMIC HEALTH

Exact Pathophysiology of impact of periodontal diseases on system health is explained through following two mechanisms-

1. Direct Mechanism: During the progression of periodontitis of chronic type, the epithelium becomes inflamed and ulcerated creating the first hand access for the periodontal pathogens into the blood stream. Thus these systemically circulating bacteria having direct contact with some organs and causing or affecting the systemic outcome [16].  
For example (eg) Periodontal bacteria have been detected in thrombi of acute myocardial infarction.
2. Indirect mechanism: Periodontal pathogens or the chemicals produced by them begins inflammatory changes which may show systemic consequences indirectly. Now it is proven fact inflammation is among triggering factors which can cause or initiate systemic illness like cardiovascular disease, diabetes type 2, rheumatoid arthritis. Thus chronic inflammatory responses caused by periodontitis may influence the pathogenesis of inflammatory based diseases [17,18].  
For eg. Level of C-reactive protein (CRP) is an indicator for systemic inflammation and CRP values are increased in individuals with periodontitis.

## 3. PERIODONTITIS & ITS CONNECTIONS WITH VARIOUS SYSTEMIC DISEASES AND CONDITIONS:

### 3.1 Periodontitis & Cardiovascular Diseases

Globally main cause of death is cardiovascular diseases [19]. Main etiology of these cardiovascular diseases is thickening of arteries called as atherosclerosis. It occurs due to deposition of calcium and fatty materials resulting in plaque formation thus causing hardening and stiffening of the arteries. Complications of this atherosclerosis depends upon location and amount of deposits. Some most common complications are angina, myocardial infarction, stroke, or aneurysm [20].

The association between periodontal disease and atherosclerotic cardiovascular disease is independent of other known confounding risk factors [21]. The link between the periodontal disease and atherosclerotic cardiovascular disease is direct and is not related to other associated risk factors. Periodontal disease plays direct role in pathophysiology of atherosclerosis (ATH) by causing thromboembolic events and furnishing systemic needs through liposachharides and inflammatory cytokines. Streptococcus sanguis and Porphyromona gingivalis caused platelet aggregation and activation by the expression of collagen-like platelet aggregation-associated proteins. These aggregated proteins play a role in atheroma formation and thromboembolic events directly or indirectly [22].

Bahekar et al. [23] carried of a meta-analysis of five studies which included 86092 persons and concluded that persons with periodontitis had higher chances of developing coronary heart disease than healthy persons. They also found that this relation is not depend of confounding risk factors.

Another case control study, which included 1423 individuals, showed more than double the risk of frequency of occurrence of cardiovascular disease in individuals with periodontitis [23].

Some studies evaluated the contents like bacterial DNA, antibodies in atheromatous plaque samples. They confirmed the presence of DNA of bacterias namely P. gingivalis most frequently followed by A. actinomycetemcomitans, T. forsythia,

Eikenellacorrodens, Fusobacterium nucleatum and Campylobacter rectus [24,25]. Haraszthy *et al.* identified periodontal pathogens in human carotid atheromas. They found 26% for *P. gingivalis*, 18% for *Aggregatibacter actinomycetemcomitans*, and 14% for *P. intermedia*. They also found that more than 40% of atherosclerosis have more than one periodontal pathogen [26].

This finding suggest that these micro-organisms can travel from oral cavity to distant organs in the body [25,26].

In individuals having high plasma levels of fibrinogen & Tumour Necrosis Factor-alpha(TNF-alpha) and having periodontal disease, they showed increased thickness of carotid intima-media thickness (IMT). Thus increasing the chances of atherosclerosis [27,28,29,30].

Further studies required to evaluate the impact of the improved periodontal health and condition on the cardiovascular condition.

### 3.2 Periodontal Disease & Diabetes Mellitus

Diabetes mellitus is a metabolic disorder which is marked by increase in blood levels of sugar known as hyperglycemia. This occurs due to faulty or defective secretion or activity of insulin [31].

Diabetes mellitus is classified into three types depending upon signs & symptoms as-type 1, type 2 and gestational. Type 1 Diabetes Mellitus is caused by wasting or destruction of beta-cells within the islets of Langerhans of the pancreas causing complete insulin deficiency. Type 2 diabetes mellitus starts due to insulin resistance and slowly progressing towards pancreatic beta-cell failure whereas in gestational type of diabetes mellitus results due to glucose intolerance during pregnancy [32].

Pathophysiology of periodontal disease and diabetes mellitus is considerably alike, that both the diseases are inflammatory in origin and in both increased levels of AGEs cause marked destruction. Thus both the conditions results in production of inflammatory mediators like IL-18 and CRP or IL-18 and IL-6 [33,34].

In diabetic patients, hyperglycemia is caused due to increased levels of advanced glycation endproducts (AGEs) in the serum [35]. These

AGEs causes production of inflammatory mediators by activation of endothelial cells and monocytes. If AGEs gets deposited/accumulated in the gingiva, they cause increased vascular permeability, increased disintegration of collagen fibers and faster impairment of nonmineralized connective tissue as well as of bone [36]. Similar observations were done by many researchers stating that severe periodontal destruction resulting in tooth loss occurs in patients with uncontrolled diabetes [37,38,39].

In patients of severe periodontitis with diabetes mellitus, due to production of inflammatory mediator's sugar level may get affected.

Thus periodontitis and diabetes mellitus share dual relationship and their prognosis is interlinked but more studies are required to prove and understand the progression and therapeutical outcomes.

### 3.3 Periodontitis & Pneumonia

Pneumonia is an infection of the lungs. Etiology of this infection can be bacteria, mycoplasma, viruses, fungi, or parasites. Bacterial pneumonia can be classified as community-acquired pneumonia and hospital-acquired (nosocomial) pneumonia. Nosocomial pneumonia, generally occurs within 48–72 hours of admission to a hospital or nursing home, can be subdivided into two subtypes: ventilator-associated pneumonia (VAP) and non-VAP.

Many oral pathogens have been identified in lung infection. Some of these are including *A. actinomycetemcomitans*, *Actinomyces israelii*, *Capnocytophaga* spp, *Chlamydia pneumoniae*, *E. corrodens*, *F. nucleatum*, *Fusobacterium necrophorum*, *P. gingivalis*, *P. intermedia* and *Streptococcus constellatus*. Saliva and dental plaque in patients with periodontal disease are the major source for spreading these pathogens to the lower airway [40,41,42].

When the pathogens in patients admitted in intensive care unit were compared, it was observed that genetic structure of the pathogens from dental plaque had similarity to the genetic structure of pathogens isolated from bronchoalveolar lavage fluid. Thus it can be concluded that the dental plaque can act as a stock for respiratory pathogens [43].

Many authors suggested that oral and respiratory bacteria from the dental plaque lean out into the

saliva and then travel to the lower respiratory tract and lungs provoking infection [44,45].

Second mechanism suggested was cytokines and enzymes produced due to periodontal inflammation in the oral biofilm may get transferred into the lungs triggering local inflammatory response in the lung and encouraging colonization of pathogens and the actual lung infection [44,45].

Another possible way of pulmonary infection is through entry of airborne micro-organisms through respiration or circulation of microorganisms through bacteria from local infection.

Individuals with low salivary flow, diminished cough reflex, swallowing disorders, with poor oral hygiene or poor oral health care habits or physical disabilities are at higher risk of developing lung infections as compare to healthy persons [44].

Gomes-Filho et al observed that the patients with periodontal diseases are three time more susceptible to the nosocomial pneumonia as compare to individuals with healthy periodontium [46].

Porto AN et al examined forty individuals who had orotracheal intubation. And observed that pathogens like *A.actinomycetemcomitans*, *P. gingivalis* and *T. forsythia* were present in large quantity. In this study both dentulous and edentulous individuals were included. Thus it can be concluded that oral cavity has favourable conditions for accumulation and growth of pathogens [47].

Poor oral health, dental plaque, or oropharyngeal bacterial colonization have been associated with the occurrence of pneumonia in hospitalized or ICU patients [45,48,49].

In nursing care home for elderly, individuals with high plaque scores, bacterial presence in saliva and colonization in oropharynx was found to be associated with pneumonia [50,51].

Dentulous individuals have higher risk of developing pneumonia and respiratory tract infections than edentulous individuals [50,51].

Use of chlorhexidine topically in ventilated patients reduces the chances of development of pneumonia and diminishes the doses of systemically administered antibiotics thus shorten the time period for mechanical ventilation

in ICU patients. Early use of topical chlorhexidine in intubated patients can reduce the colonized oral bacterial count and postpone the onset of VAP [52,53,54,55,56].

Recently in a study it was observed that use of povidone iodine in combination of mechanical oral health care in ventilated patients reduces the risk of pneumonia [57].

Thus it can be concluded that oral health status can affect the lungs negatively but with proper oral health care severity of adverse outcomes can be reduced.

### **3.4 Periodontal Disease And Osteoporosis**

Osteoporosis is a skeletal disease which is characterized by reduced bone density and bone quality resulting in decreased bone strength, making it vulnerable for fracture [58].

Both osteoporosis and periodontal disease has similarity that both results in bone resorption. Age, estrogen deficiency and smoking are the common threats for the systemic as well as oral osteopenia [59,60,61].

Increased bone resorption can be due to surge in systemic / local osteoclastic activity or due to cellular or cytokine effects in both diseases [62].

In periodontitis, gingiva shows thick layer of infiltration by mononuclear leukocytes, T lymphocytes and monocytes or osteoclast like progenitor cells [63]. In periodontal infection, osteoclast formation occurs due to interaction between T cells and monocyte/lymphocyte progenitor cells. This is the most important event in periodontal infection [64].

When gene sequencing was examined in advanced periodontitis patients, it was found that RANKL mRNA was increased whereas osteoprotegerin (OPG) mRNA was downregulate/decreased in the gingival [64].

Nagasawa et al observed that increased OPG mRNA was due to LPS from *P. gingivalis* and *A. actinomycetemcomitans*, thus it can be correlated that LPS-stimulated OPG may be responsible for the osteoclast formation in periodontitis [65].

Gram negative bacteria associated with periodontitis may aggravate the RANKL activity

and thus causing osteoclast activation hence initiating the osteoporosis in periodontitis patients. This was observed by Teng YTA et al and Jiang Y et al in their studies also [66,67].

In women, Estrogen deficiency was main etiological factor associated with osteoporosis [68].

Estrogen regulates cytokines production that is essential for bone metabolism as well as modulation of host response to inflammation through factors like as IL-1 alpha, IL-1 beta, TNF-alpha, and macrophage colony-stimulating factor (M-CSF). Hence estrogen deficiency results in increased osteoclasts population thus resulting disparity in bone metabolism leading to reduced bone mineral density (BMD) [69].

In Periodontitis host's proinflammatory response is activated through engagement of cytokines and prostanooids causing activation of osteoclasts and resulting in bone resorption. In gingivitis, elevated levels of IL-1 beta, TNF-alpha, IL-6, and IL-8 were associated with bone resorption. Thus proving interlink between periodontal disease and estrogen deficiency [70,71].

Wactawski-Wende et al. [110] determined a strong and consistent association between alveolar crestal height (ACH) and osteoporosis through measurements of bone density and ACH in postmenopausal women [72].

Stefen Renvert et al observed that 61.1% patients with Rheumatoid arthritis showed signs and symptoms of periodontitis as compare to control group-33.7% [73].

Lozano et al in their case control study observed significant prevalence of periodontal disease in rheumatoid arthritis patients as compare to control group. They also observed that in rheumatoid arthritis group, severity of clinical signs and symptoms were more as compare to control group [74].

Still the question remains unclear whether periodontitis causes osteoporosis or vice versa. Further studies are required to evaluate the exact role of periodontitis in osteoporosis occurring in Rheumatoid arthritis.

### 3.5 Oral and Colorectal Cancer

Cancer is the cause of death for every fourth person, which adds to emotional and financial burden.

In 1990, *Helicobacter pylori* was the first pathogen to be identified as the one of the etiological factor in human gastric cancer [75]. Thus it was the first bacterial pathogen to be associated with cancer in humans [76,77].

Yao QW et al in their study of 3183 patients, found that the individuals with periodontitis had higher risk of developing oral cancer [78].

Michaud DS et al found positive relationship in patients with periodontitis and cancer of pancreas, head, lung and neck [76].

Wen BW et al examined one million Taiwanese individuals and concluded that individuals who had periodontitis have higher risk of developing cancer than individuals with gingivitis [79].

*P. gingivalis* pathogen responsible for the periodontal disease was found in higher level in patients of oral squamous cell carcinoma (OSCC) and esophagus squamous cell carcinoma as compare to healthy patients [77,80].

In animal study model, Binder Gallimidi A et al evaluated possible role of periodontal pathogens in the instate of the oral cancer and they observed that periodontal pathogens *P. gingivalis* & *P. nucleatum* trigger tumorigenesis through unmediated linkage with oral epithelium [81].

In OSCC, *P. gingivalis* causes invasion and metastasis of oral squamous cell through matrix metalloproteinase 9 (pro-MMP9) expression. Periodontal pathogen *F. nucleatum* does not play any role [80].

Ha NH et al. observed that the aggressiveness of oral cancer is affected by *P. gingivalis* positively. They also observed that chronic & recurrent exposure of *P. gingivalis* causes augmentation of aggressiveness of oral cancer by triggering epithelial mesenchymal transition-like changes in the cells [82].

Colorectal carcinoma (CRC) is the fourth most common carcinoma diagnosed and cause of deaths in cancer patients. In CRC patients, high numbers of *F. nucleatum* and *Clostridium difficile* were found in intestinal microbiota. [83]

Warren RL evaluated 130 specimens of CRC patients and specimen showed presence of Gram-negative anaerobic oral pathogens such as *Fusobacterium*, *Leptotrichia* and *Campylobacter* [84].

When human colonic adenoma site and surrounding healthy tissue in CRC patients and healthy patients were evaluated, it was found that *F. nucleatum* colonizes were more in number in CRC patients. Even stools of CRC patients showed high number of *F. nucleatum* as compare to healthy patients [85,86].

*F. nucleatum* not only migrate to human intestinal tract but also colonize their triggering severe inflammation [83,87].

In a mouse study of CRC, it was found that *F. nucleatum* not only affects the constitution of lumen microbiota and moderate the secretion of cytokines and activate tumorigenesis-related pathway [86].

These all finding suggest that *F. nucleatum* causes changes in microenvironment which favours the advancement of CRC.

In summarization, it can be concluded that *P. gingivalis* and *F. nucleatum* have positive correlation in development of various carcinoma like oscc, crc etc. and this relationship can be used for early detection and better prognosis. To evaluate this further studies are required.

### 3.6 Alzheimer's Disease

Alzheimer's disease (AD) is a neurological disorder which causes progressive and irreversible.

Alzheimer's disease develops due to different response of brain to inflammatory process and this occurs through complement activation and cytokine and chemokine expression [88].

This occurs due to emergence of synaptotoxic amyloid plaques and hyperphosphorylated tau proteins in the area of the brain designated to the advanced cognitive functions [89].

AD is emerged by the development of extracellular amyloid  $\beta$ -peptide (A $\beta$ P) plaques and intraneuronal neurofibrillary tangles (NFTs) of hyperphosphorylated tau protein which results into loss of neuronal synapses gradually and ultimately neuronal degeneration with diminution of essential neurotransmitters [88].

Inflammation is the common factor associated with periodontitis and Alzheimer's disease. In

AD, activated glial cells causes crucial increase in inflammatory cytokines [90].

AD can show early or late onset. Early onset AD is due to genetic linkage; whereas late onset or sporadic AD is caused due to combination of genetic and environmental factors. Age, education, high fat diet, hypertension, diabetes, history of head trauma, and susceptibility genes such as apolipoprotein E (APOE) and periodontitis are the elements which influence AD negatively.

The linkage between periodontitis and AD can be explained by two mechanism-

- i) Micro-organisms associated with periodontitis causes increased levels of proinflammatory cytokines as host response. This procedure causes production of various cytokines and pro-inflammatory mediators in systemic circulation, resulting in systemic or peripheral inflammation. These pro-inflammatory mediators can cross the BBB and entering the cerebral regions. This results in activation of microglial cells and adverse reactions causing neuronal damage.
- ii) According to second mechanism, microorganisms associated with the dental plaque biofilm directly enters in the brain through blood stream or via peripheral nerves. These micro-organisms illicit inflammatory response in central nervous system. This response is initiated due to interplay between neurons and glial cells. This interaction results in release of different cytokines like IL family, TNF- $\alpha$ , transforming growth factor- $\beta$ , and chemokines (monocyte chemotactic protein, IL-8, macrophage migration inhibitory factor, and monokine induced by  $\gamma$ -interferon) which are identified as biomarkers for AD [91].

Cytokines like TNF-alpha plays major role in neurodegenerative disease. TNF- $\alpha$  aggravates the inflammatory process, causing in gliosis, demyelination, blood brain barrier (BBB) deterioration, and cell death [92,93].

Seulggie Choi MD et al in their study found that individuals with Chronic periodontitis have higher risk of developing dementia and AD [94].

Increased levels of antibodies for *A. actinomycetemcomitans*, *P. gingivalis*, *T. forsythia*, *F. nucleatum* and *P. intermedia* were detected in individuals with AD compared with healthy persons [90,95].

It can be summarized that poor oral hygiene specially periodontitis can be a risk factor for AD but still question arises that what comes first and further longitudinal studies required for the evaluation of the relationship between periodontitis and AD.

### 3.7 Adverse Pregnancy Outcomes

Females with maternal infection are at higher risk of unfavourable pregnancy outcomes like preterm labour, preterm premature rupture of the membranes, pre-eclampsia, miscarriage, intrauterine growth retardation, low birthweight, stillbirth, and neonatal sepsis [96].

Hormonal changes occurring during pregnancy increases the risk of developing to gingivitis and periodontitis by 40% in pregnant women than non-pregnant women [96].

The association between oral health status and unfavourable pregnancy outcome is explained by two mechanisms-

- i) Oral pathogens directly move from unhealthy mouth or oral cavity and crossing the placenta ,reaches in the intra-amniotic fluid and fetal circulation [97].
- ii) Second mechanism can be systemic circulation of endotoxins or inflammatory mediators released in periodontal disease can strike adversely on development of the fetus or cause spontaneous abortion [98].

*F. nucleatum* is the frequently occurred oral pathogen in placental and fetal tissues [99].

Han YW et al reported a case of stillbirth where they found *F. nucleatum* was moved and travelled to the uterus from the mouth when mother suffered from respiratory infection during pregnancy [100]. In many studies, *F. nucleatum* was frequently identified in amniotic fluid and cord blood from of preterm birth and neonatal sepsis cases [101,102].

*F. nucleatum*, *P. gingivalis* and other oral species were oftenly detected in intrauterine infection,

thus proving that these micro-organisms can migrate from oral cavity [102,103].

But in mice model study, it was found that of *P. gingivalis* has negative impact on pregnancy, LPS from *P. gingivalis* confined placental development and fetal growth. And when antibodies against *Pgingivalis* administered ,it caused fetal loss [104,105].

The maternal-fetal interconnect hosts the immune tolerance to the fetus as well as produce strong host defence against infections. Placenta produces Toll-like receptors(TLRs) during normal pregnancy as a part of innate immunity. Increase in TLRs levels suggest that innate immunity activation against the periodontal pathogens like *T. denticola* and *P. gingivalis* [106,107,108,19].

Although more studies are required to evaluate the exact relationship and effect of the periodontal diseases and pregnancy outcomes. Results will help to combat and prepare better oral health care protocols during pregnancy.

### 4. CONCLUSION

Periodontal health has an impact on overall health directly or indirectly. Individuals with periodontal disease are at higher risk for developing systemic diseases than their healthy counterparts, this relationship can be dual. Present literature includes epidemiological , animal and clinical studies which reinforce the link between the periodontal pathogens, inflammation and bacteraemia in the systemic diseases. Further studies are required to explicate the exact role of periodontal health in various systemic diseases and it's role in aggravating present systemic condition are required. But proper oral hygiene care and timely management of periodontal disease can have positive impact on the overall health leading to favouring or assuring outcomes.

### CONSENT

Not applicable.

### ETHICAL APPROVAL

Not applicable.

### COMPETING INTERESTS

Authors have declared that no competing interests exist.



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