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Maternal Periodontal Disease and Adverse Pregnancy Outcomes: A Critical Review of Current Evidence

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Abstract

Background: Pregnancy is a complex physiological state involving hormonal, immunological, and vascular changes that may predispose women to oral diseases such as periodontal disease (PD). In recent years, maternal PD has emerged as a possible risk factor for adverse pregnancy outcomes (APOs) including preterm birth (PTB), low birth weight (LBW), preeclampsia (PE), and gestational diabetes mellitus (GDM). This critical review aims to assess current evidence linking maternal periodontal disease with pregnancy complications and highlights its relevance as a public health concern.

Methods: A comprehensive literature search was conducted across PubMed, Scopus, Web of Science, and Google Scholar for studies published between 2015 and 2025. Relevant keywords included "Periodontal disease," "Pregnancy complications," "Preterm birth," "Low birth weight," "Preeclampsia," and "Maternal oral health." Eligible studies included case-control, cohort, interventional, experimental, and systematic reviews.

Results: Numerous studies report statistically significant associations between maternal PD and APOs, particularly PTB, LBW, and PE. Epidemiological data, animal models, and biological mechanisms—including systemic inflammation and microbial translocation—support this link. However, inconsistencies in diagnostic criteria, sample sizes, study designs, and confounding factors limit causal inference. While some interventional studies demonstrate benefit from periodontal treatment, others show inconclusive results.

Conclusion: Although definitive causality remains unproven, maternal PD appears to be a plausible and potentially modifiable risk factor for adverse pregnancy outcomes. Emphasising oral healthcare during pregnancy

may offer benefits for both maternal and fetal health. Further multicentric, standardised research is essential to validate these associations and guide public health policy.

Keywords: Maternal Periodontitis, Preterm Birth, Low Birth weight, Preeclampsia, Spontaneous Abortion.

Introduction

Are mothers' smiles an indicator of her babies' future?

Pregnancy is a unique and dynamic physiological process that involves profound hormonal, vascular, and immunological changes necessary for fetal development. Although these alterations are necessary for pregnancy, they may also make mothers more vulnerable to other illnesses, such as periodontal disease.[1] In recent decades, maternal oral health has gained attention as a systemic health indicator, particularly in relation to adverse pregnancy outcomes (APOs) including preterm birth (PTB), low birth weight (LBW), preeclampsia (PE), and gestational diabetes mellitus (GDM). Increasing evidence suggests that poor periodontal health could be a modifiable risk factor during pregnancy.

The term "periodontal disease" refers to a group of chronic inflammatory disorders affecting the gingiva, periodontal ligament, and alveolar bone. The condition typically begins with gingivitis, a reversible gum inflammation triggered by microbial plaque. If left untreated, gingivitis can progress to periodontitis, characterized by attachment loss, alveolar bone resorption, deep periodontal pockets, and eventual tooth loss.[2] Gram-negative, anaerobic, or microaerophilic bacteria, such as Porphyromonas gingivalis, that invade the subgingival environment are the main cause of periodontitis.[3,4] However, a complex interaction of microbiological, genetic, behavioural, and environmental factors—such as stress, tobacco use, systemic disorders, and immune response variability—influences the disease's progression and severity.

Hormonal changes during pregnancy, particularly increased estrogen and progesterone—modify the maternal immune response and raise gingival vascular permeability. This enhances the inflammatory response to dental plaque, leading to increased severity of periodontal inflammation. Epidemiological studies reveal that a substantial proportion of pregnant women globally suffer from periodontal disease, with prevalence influenced by geographic, socioeconomic, and healthcare access factors.[5]

The idea that the effects of periodontal disease can go beyond the oral cavity and affect systemic health and pregnancy outcomes has been the subject of much investigation in recent decades. This connection has been explained by a number of biological mechanisms. Periodontal disease may adversely affect pregnancy outcomes through both direct microbial invasion and indirect systemic inflammatory responses.[6] Periodontal pockets harbour Gram-negative anaerobic bacteria and virulence factors such as lipopolysaccharide (LPS), prostaglandin E2 (PGE2), and tumour necrosis factor-alpha (TNF-α), which can enter the bloodstream due to the periodontium’s high vascularity.[7] These components may reach the fetal-placental unit, triggering chorioamnionic and trophoblastic cells to release pro-inflammatory cytokines like IL-1β and PGE2, promoting uterine contractions and preterm labour. Alternatively, local periodontal inflammation may result in systemic release of fetotoxic mediators. A poor maternal immune response can permit bacterial translocation into circulation, amplifying inflammation via hepatic acute-phase reactants like C-reactive protein.[8] These inflammatory cascades may impair placental function, suppress fetal growth factors, and lead to outcomes such

as premature rupture of membranes, preeclampsia, or low birth weight.[9] Although interventions targeting periodontal disease have not consistently reduced adverse outcomes, epidemiological data support its pathogenic link, particularly among socioeconomically disadvantaged populations. Thus, maternal periodontal infections represent a potential systemic threat to fetal development.[10]

Numerous studies have identified significant correlations between maternal periodontal disease and APOs. For example, strong associations have been observed between periodontitis and PTB, LBW, and GDM, although some studies acknowledge methodological limitations and potential biases, such as exposure misclassification and publication bias.[11] Experimental studies in animal models further support the plausibility of this link, demonstrating that systemic inflammation induced by P. gingivalis can lead to reduced birth weights and placental inflammation, even in the absence of bacterial colonisation of fetal tissues.[12]

Despite these insights, a causal relationship between periodontal disease and APOs remains inconclusive. Interventional trials aimed at improving pregnancy outcomes through periodontal therapy have shown mixed results, often limited by variability in study design, timing of treatment, and population characteristics.[9] Nevertheless, considering the high prevalence of periodontal disease and its systemic implications, maintaining oral health during pregnancy may be an important, though often overlooked, element of prenatal care. This review critically explores the current evidence that links maternal periodontal health to adverse pregnancy outcomes and highlights its relevance as a public health concern.

Materials & Methods

To conduct this critical review titled "Maternal Periodontal Disease and Adverse Pregnancy Outcomes: A Critical Review of Current Evidence", relevant literature was systematically retrieved from PubMed, Scopus, Web of Science, and Google Scholar. The search focused on publications from 2015 to 2025 to ensure the inclusion of recent and clinically significant evidence. The review aimed to provide a comprehensive overview of current knowledge on the association between maternal periodontal disease and adverse pregnancy outcomes. The search terms included: "Periodontal disease," "Pregnancy complications," "Preterm birth," "Low birth weight," "Preeclampsia," "Spontaneous abortion," "Intrauterine growth restriction," "Maternal oral health," and "Adverse pregnancy outcomes." The selected studies encompassed case reports, laboratory investigations, cohort studies, case-control studies, interventional trials, and systematic reviews, offering a broad perspective on the potential implications of maternal periodontal status during pregnancy.

Discussion

Maternal Periodontal Disease and Preterm Birth (PTB):

Preterm birth (PTB), defined as delivery before 37 completed weeks of gestation, and low birth weight (LBW, defined as <2,500g), are significant contributors to neonatal morbidity and mortality worldwide, posing a significant public health challenge, even in industrialised nations.[7] While LBW is independent of gestational age, it often correlates with prematurity. Globally, PTB accounts for nearly six million perinatal deaths annually, highlighting the urgency to identify modifiable risk factors.[9]

Emerging evidence suggests that maternal periodontal disease (PD) may be a potential risk factor for adverse

pregnancy outcomes (APOs), particularly PTB and LBW. A meta-analysis involving 22 studies and 17,053 subjects reported a wide prevalence range of PTB (0.03% to 57.3%) among women with PD.[10] Strong associations between PD and three major APOs—PTB, LBW, and GDM—were identified in a synthesis of 43 systematic reviews[11]. An analysis of 33 cohort and case-control studies including 13,098 women determined that pregnant women with PD had a 1.10-fold increased risk of delivering preterm.[13] Additionally, 60% of studies showed a positive correlation between PD and APOs, with low heterogeneity.[14]

Epidemiological studies across various populations further support the association. Women with periodontitis had twice the odds of delivering preterm compared to periodontally healthy women. [15] First-time mothers with PD were found to be 12 times more likely to experience preterm labour.[16] A 3.46-fold increased risk of preterm birth (PTB) was reported in women with severe periodontitis.[17] In a case-control study involving 555 postpartum women, a six-fold increase in PTB risk was observed among those with PD.[18] Among Ivorian women, the risk of PTB was 3.62 times higher in those with PD.[19] Periodontitis was found in 82% of preterm cases versus 46% in term cases, with an odds ratio of 5.35.[20] A dose-response relationship was indicated, as 80.5% of mothers without PD delivered at term, compared to only 58% of those with severe PD.[21] A significant severity-based correlation between periodontitis and PTB was also reported.[22] Large population-based data involving 748,792 deliveries showed that 22.09% of women with PD experienced adverse pregnancy outcomes (APOs), compared to 18.58% among those without, with spontaneous abortion, PTB (14.46%), and LBW (5.92%) being the most common complications. PD showed the strongest association with spontaneous abortion.[23]

Despite some inconsistencies, interventional studies lend further credibility to the association. Participants who underwent scaling and root planing (SRP) had a substantially reduced risk of PTB—only 3 of 22 PTBs occurred in the intervention group compared to 19 in controls. LBW incidence was also lower in the treated group.[24] Likewise, a systematic review of 24 reviews concluded that periodontal treatment significantly reduced the risk of PTB and LBW, especially when administered during pregnancy.[25]

Pathophysiologically, PD is characterised by elevated systemic inflammatory markers that are also implicated in the initiation of labour. Higher levels of prostaglandin E2 (PGE2), IL-1, and TNF-α were found in women who delivered preterm compared to term controls—mediators known to trigger labour.[26] Animal studies further support this biological plausibility; inoculation of Fusobacterium nucleatum and Porphyromonas gingivalis in pregnant mice induced adverse outcomes, including intrauterine growth restriction, fetal death, and PTB, likely due to hematogenous migration of bacteria to the placenta.[27]

However, not all studies corroborate a direct association. PD alone may not significantly raise the risk of preterm low birth weight infants unless accompanied by behavioural, genetic, or maternal comorbidities.[28] Studying 70 puerperal women, no significant link was found between periodontitis or periodontopathogens and PTB, reinforcing the multifactorial nature of preterm delivery.[29] A Mendelian randomisation study further challenged the causality hypothesis, finding no genetic evidence to support a direct causal relationship between PD and APOs such as PTB, LBW, stillbirth, miscarriage, or gestational hypertension.[30]

Maternal Periodontitis and Low birth Weight (LBW):

Numerous studies have investigated the link between PD and low birth weight (LBW), with varying results. Offenbacher et al. (1996) were among the first to identify periodontitis as a previously unrecognized but clinically important risk factor for preterm low birth weight (PLBW), attributing 18.2% of PLBW cases to maternal periodontal infection.[7] In a review of ten studies encompassing 1,349 LBW cases and 4,344 controls, a wide LBW prevalence range of 0.04% to 40.2% was reported, suggesting potential variability in populations and diagnostic criteria.[10] Supporting these findings, data from 16 cohort and case-control studies involving 3,575 women found that those with PD had a 2.19-fold increased risk of delivering LBW infants.[13] Similarly, an earlier study demonstrated that women with PD during their first pregnancy were 2.3 times more likely to deliver LBW infants.[16] This is echoed by another study that reported a 2.6-fold increased LBW risk among women with PD at delivery in northern Tanzania.[31] Volgyesi et al. (2023) also found a significant association between LBW and elevated bleeding on probing (BOP) and sulcus bleeding index (SBI) among 111 healthy primigravida women, highlighting the importance of periodontal health assessment during pregnancy.[32]

Large-scale population data further reinforce this association. In a study involving 748,792 pregnant women, it was found that 5.92% of deliveries among mothers with PD resulted in LBW, as opposed to lower rates in those without PD.[23] A meta-analysis, which included over 2.5 million women, confirmed a moderate correlation between PD and LBW, though findings on preterm birth were less conclusive due to heterogeneity and publication bias.[33] However, other studies challenge the consistency and causality of this association. For instance, a case-control study conducted in Brazil with 951 participants, found no significant difference in periodontitis prevalence between mothers of LBW and normal-birth-weight infants, even after adjusting for confounders such as maternal age, BMI, prenatal care, and smoking.[34]

Overall, while many studies indicate a significant association between PD and LBW, inconsistencies remain, suggesting the need for further high-quality, controlled research to clarify causal relationships and guide preventive strategies.

Dual Burden: Maternal Periodontitis and the Risk of Preterm Low Birth Weight (PLBW)

Among the most studied consequences are PTB and LBW, both major contributors to neonatal morbidity and mortality worldwide. Emerging research suggests that maternal periodontitis, a chronic inflammatory condition of the gums, may act as a modifiable risk factor for these adverse outcomes, though the evidence remains mixed. Biologically, this association is supported by findings that mothers of PLBW infants exhibit elevated levels of periodontal pathogens such as Bacteroides forsythus, Porphyromonas gingivalis, Actinobacillus actinomycetemcomitans, and Treponema denticola, all of which are associated with mature plaque and chronic periodontitis.[35] Experimental studies using animal models further substantiate this connection; it was demonstrated that infection with P. gingivalis in rats resulted in PTB and LBW offspring via inflammatory pathways.[36]

Several systematic reviews and meta-analyses provide compelling support for this association. Through 10 studies involving 1,349 LBW cases and 4,344 controls, a prevalence of PTB and LBW ranging from 0.01% to 5.7% was reported.[10] Strong associations between PD and PTB, LBW, and gestational diabetes mellitus were found in an analysis of 43 systematic reviews.[11] A two to threefold increased risk of PTB and LBW in pregnant

women with PD was noted after synthesising findings from 15 systematic reviews.[37] Significant associations between PD and both PTB and LBW were confirmed using fixed-effect meta-analysis on over 15,000 participants.[38] In a systematic review of 10 studies, nine studies identified a positive link between maternal PD and PTB/LBW.[39] Likewise, statistically significant associations between PD and PTB (63.96%), LBW (71.87%), PLBW (59.18%), preeclampsia (68.89%), and prelabor rupture of membranes (65.38%) were found in an extensive meta-analysis of 232 articles (n = 119,774).[40]

Further clinical studies echo these findings. All moderate-to-severe PD cases, and a significant number of mild ones, were associated with PTB and LBW, showing an inverse relationship between periodontal severity and both birth weight and gestational age.[41] PTB (62.5%) and LBW (68.7%) were the most common adverse outcomes linked to PD, attributed to bacterial translocation into placental tissues, as noted in a review of 16 studies.[42]

Despite the above findings, some studies failed to find statistically significant associations. A case-control study with 296 controls and 148 cases found no significant correlation between maternal PD and PTB/LBW.[43] Similarly, no significant differences in periodontal parameters between groups with PLBW and those with term normal-weight deliveries were found in a study of 287 pregnant women.[44] Another animal study reported no significant differences in prematurity, fetal weight, or birth weight between periodontitis-induced and control rats, although a lower fertility rate was noted in the PD group.[45] In a Libyan cohort, a 14% PTB and 10.7% LBW rate was observed, but no significant correlation between PD and these outcomes was found.[46] No association was also reported in a cohort of 98 women.[47] Similar conclusions were echoed, attributing findings to the lack of standardized PD definitions and insufficient pregnancy duration to detect the full impact of chronic oral infections.[48]

Preeclampsia and Periodontal Disease: A Hidden Inflammatory Pathway?

A maternal multi-organ disease, preeclampsia (PE) is characterised by hypertension (systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg) and proteinuria in the second half of pregnancy. These symptoms are frequently accompanied by major organ dysfunction, including liver, kidney, and brain involvement. It is among the leading causes of maternal and fetal morbidity and mortality.[49] Several studies suggest that maternal periodontitis may adversely affect pregnancy outcomes, including PE, PTB, vulvovaginitis, and LBW.[50] The mechanism by which maternal PD causes PE involves periodontal bacteria and their virulence factors stimulating the production of inflammatory cytokines (IL-1, PGE_2, TNF-α, etc.) and antibodies against the bacteria. These cytokines, in conjunction with the bacteria, cause structural damage to the placenta, which can alter the mother's blood pressure and disrupt normal fetal blood flow, ultimately leading to PE.[8]

Multiple interventional studies, case-control, and cohort studies have found an association between PD and PE. A woman who has periodontitis at delivery has 3.4 times higher odds of going on to develop PE[31]. In one study of 200 patients, 46% had periodontitis, and 67 had both conditions.[49] These findings indicate a positive association, supporting periodontitis as a risk factor for PE, consistent with reports that maternal PD increases PE risk.[51]

In a study, preeclamptic women with chronic PD had higher levels of periodontal markers such as Plaque Index (PI), Periodontal Pocket Depth (PPD), Bleeding on

Probing (BOP), and Clinical Attachment Loss (CAL) than the other groups. Additionally, they had the highest levels of viruses, periodontal pathogens, and expression of mir155, providing compelling evidence that maternal PD can increase the risk of PE, especially when combined with microbial dysbiosis and genetic inflammatory markers like mir155.[52] This association is further supported by an experimental study, which found that the significance of PD as a potential cause of preeclampsia is evidenced by the fact that, after 15 days of ligature placement and P. gingivalis inoculation, the animals displayed typical symptoms of PE, including altered blood pressure, proteinuria, and changes in litter size and pup weight compared to the control group.[53]

Additional observational studies provide further evidence of the association between PD and preeclampsia. Among 273 pregnant women, 4.3% experienced preeclampsia, and while PD was not significantly associated with PTB, it was significantly linked to preeclampsia, even after adjusting for confounders.[54] Similarly, in a retrospective analysis of 165 mothers, those with severe periodontitis had significantly higher rates of preeclampsia, uterine fibroids, and chronic hypertension, as well as an increased risk of very and extremely preterm births compared to those with mild or moderate periodontitis.[55]

A systematic review involving 30 studies (24 case-control and 6 cohort) confirmed a significant association between periodontitis and PE, particularly in cohort studies, and highlighted its potential importance in lower-middle-income countries.[56] Women with periodontitis had a 3.85-fold higher risk of developing preeclampsia.[57] Supporting these findings, an 8.7% prevalence of preeclampsia was reported among pregnant women with PD.[58]

While many studies support an association between periodontitis and preeclampsia, some findings refute it. A genome-wide association study (GWAS) involving individuals of European ancestry used genetic variants linked to aggressive periodontitis, chronic periodontitis, and preeclampsia as instrumental variables in Mendelian randomisation (MR) analysis. No bidirectional causal relationship was found, suggesting neither condition alters the other's risk.[59] Similarly, in 200 pregnant women, significant statistical differences in periodontal markers (PD, CAL, BOP, PCR) between hypertensive and normotensive groups were clinically negligible (<0.3 mm). Periodontal disease was present in 98% of preeclamptic and 93% of normotensive women (P = 0.17). The study concluded there is no strong clinical evidence linking the two conditions.[60]

Spontaneous Abortion, and Other Adverse Outcomes: Is There a Role for Periodontal Disease?

A growing body of evidence suggests that maternal periodontal disease (PD) may contribute to a range of adverse pregnancy outcomes beyond the well-documented associations with preterm birth (PTB) and low birth weight (LBW). These include spontaneous abortion (SAB), intrauterine growth restriction (IUGR), preterm premature rupture of membranes (PROM), congenital anomalies, and even gestational diabetes mellitus (GDM). While the precise mechanisms remain under investigation, the systemic inflammatory burden and translocation of periodontal pathogens are hypothesised to play key roles.

The link between maternal PD and spontaneous abortion has been highlighted in several studies. According to a comprehensive study of 748,792 expectant mothers, 2.22% of PD patients had SAB, which is a far greater percentage than that of non-PD patients.[23] Similarly, a marked difference in periodontitis prevalence between women with SAB (50.6%) and controls (21.2%) in a matched case-control study was demonstrated.[61] Using

data from a North American preconception cohort, a history of tooth mobility—a clinical indicator of PD—was reported to be positively associated with SAB risk, even though prior PD diagnosis or treatment alone did not show strong associations.[62] This suggests that chronic, progressive disease may be more relevant to adverse outcomes than past treatment history.

Periodontal disease has also been associated with intrauterine growth restriction. In a case-control study, significantly greater probing depth among pregnant women with ultrasound-confirmed IUGR compared to healthy controls was reported, indicating more severe periodontal inflammation.[63] Women who received periodontal therapy postpartum—implying untreated periodontitis during pregnancy—had significantly higher odds of IUGR (6.3% prevalence), emphasising the detrimental effects of active inflammation during gestation.[64] These findings support the hypothesis that untreated or active maternal PD may compromise fetal growth, potentially through systemic inflammatory mediators or microbial translocation.

Beyond growth restriction and pregnancy loss, maternal PD has also been proposed as a potential contributor to congenital anomalies. Pregnant mice injected with sonicated Porphyromonas gingivalis exhibited a 12.5% incidence of cleft palate (CP) in offspring, significantly higher than in controls.[65] Although the precise pathogenesis remains to be elucidated, this study highlights a possible role of periodontal pathogens in congenital anomalies, possibly via hematogenous dissemination or interference with fetal morphogenesis.

Several studies have explored the role of PD in membrane rupture during pregnancy. Elevated levels of gingival crevicular fluid (GCF) cytokines such as IL-10 were observed in women with PROM, suggesting that periodontal inflammation may influence anti-inflammatory and pro-inflammatory signalling in pregnancy.[66] Longitudinal differences in cytokine profiles between women with PROM and those with uncomplicated pregnancies were reported, implying that inflammation may originate in the periodontal tissues and disseminate to distant maternal compartments, including the amniotic sac.[67]

Clinical data support these findings. A strong association between periodontal pockets and PROM, PTB, and LBW was reported. Although no significant link with preeclampsia was noted, the authors emphasised that chronic infection in periodontal tissues could independently contribute to PROM.[68] Similarly, women with PD were found to be significantly more likely to experience PROM and associated infections like vulvovaginitis.[69] PROM prevalence was 32% in women with PD versus lower rates in those without. Although stillbirth and IUGR rates were not significantly impacted, the findings support a consistent correlation between PD and adverse membrane outcomes.[58]

In addition to structural and inflammatory complications, PD may also influence metabolic regulation during pregnancy. Periodontal disease has also been implicated in the pathogenesis of gestational diabetes. A systematic analysis, which included 12 cohort and case-control studies (n = 6636), showed that pregnant women with PD had a 1.39-fold increased risk of developing GDM compared to those without PD. This association may be mediated by the systemic inflammatory burden and insulin resistance linked to chronic periodontal inflammation.[13]

Conclusion

This review underscores the growing evidence suggesting that maternal periodontal disease (PD) may be a plausible, though not definitively proven, risk factor for adverse pregnancy outcomes (APOs) such as preterm

birth (PTB), low birth weight (LBW), preeclampsia, and spontaneous abortion. Several clinical, observational, and experimental studies support this association through mechanisms involving systemic inflammation and microbial translocation. However, inconsistencies in findings, methodological limitations, and uncontrolled confounders make it difficult to establish a direct causal link. Study heterogeneity—including variations in design, diagnostic criteria, population demographics, and outcome measures—further weakens the strength of evidence and complicates meta-analytical synthesis. Small sample sizes, single-centre settings, and potential selection or reporting biases also limit the generalisability of results. To better clarify this relationship, future research should prioritise standardised periodontal diagnostic criteria, multicentric and longitudinal study designs, and well-controlled prospective or interventional trials. Establishing PD as a modifiable prenatal risk factor could provide a cost-effective strategy to improve maternal and neonatal health outcomes. Until stronger evidence emerges, promoting and maintaining optimal periodontal health during pregnancy remains a prudent public health recommendation that may contribute to better pregnancy outcomes.

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