The primary etiologic factor for periodontal diseases is “Dental plaque.” Although pathogenic bacteria in dental plaque are required for the incidence of periodontal disease, a susceptible host is also very important. The susceptibility of the host can be modified by many systemic factors with hormones level being one. The periodontium shows an exaggerated inflammatory response to plaque modified by female sex hormones during puberty, pregnancy, in women taking oral contraceptives, and at the postmenopausal stage. This review provides an in detail analysis of how periodontium is influenced by the fluctuation in sex steroid hormones of females during different phases of their lifetime and to discuss how much the same hormone at different ages and stages shows an exaggerated gingival response to plaque.

**KEYWORDS:** Female sex hormones, menopause, periodontium, pregnancy, puberty

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**ABSTRACT**

The primary etiologic factor for periodontal diseases is “Dental plaque.” Although pathogenic bacteria in dental plaque are required for the incidence of periodontal disease, a susceptible host is also very important. The susceptibility of the host can be modified by many systemic factors with hormones level being one. The periodontium shows an exaggerated inflammatory response to plaque modified by female sex hormones during puberty, pregnancy, in women taking oral contraceptives, and at the postmenopausal stage. This review provides an in detail analysis of how periodontium is influenced by the fluctuation in sex steroid hormones of females during different phases of their lifetime and to discuss how much the same hormone at different ages and stages shows an exaggerated gingival response to plaque.

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**INRODUCTION**

The periodontium, defined as the tissues that surround and support the teeth, includes the gingivae, bone, cementum, and periodontal ligament. Periodontitis is the result of an inflammatory response by the host to a bacterial infection of periodontal tissues. Numerous species of pathogenic bacteria present in subgingival plaque can interrupt the routine homeostatic processes, attack the surfaces of the tooth, infiltrate periodontal tissues, and organize themselves in the form of biofilm.[1]

The host’s immune response, triggered by microorganisms and their metabolites, stimulates the synthesis and release of cytokines, inflammation mediators, and metalloproteinases of the matrix, leading to the damage of the tissues.[2] The disease progression and severity depend on the aggressiveness of the subgingival biofilm which is countered by the host’s immune response[3,4] and further modulated by the genetic and epigenetic context and also environmental factors such as gender, age, smoking, and oral hygiene.[5,6] It is clearer that, due to bacteremia and the systemic release of endotoxins, the presence of pathogenic bacteria in periodontal lesions is connected to various systemic disorders, including cancer, diabetes mellitus, rheumatoid arthritis, cardiovascular disease, infertility, and adverse birth outcomes.[7] Female periodontium undergoes developmental changes beginning at puberty and progressing through menstruation, pregnancy, and menopause depending on the levels of sex hormones in the body.

**SEX HORMONES AND PERIODONTAL CHANGES**

Ovaries secreting estrogen is responsible for the maintenance of secondary sex characteristic and uterine growth while progesterone influences the second half of the menstrual cycle and pregnancy. Progesterone influences the vascularity of the gingival and periodontal tissues through the receptors present in them.[8,9]

This leads to the production of prostaglandins and the movement of polymorphonuclear leukocytes further leading to increases inflammation while estrogen increases the inflammatory component of gingiva without deposition of bacterial plaque.[9]

Raised hormonal content is act as a feeder and growth factor for black-pigmented bacteria including...
Bacteroides, Prevotella intermedia, and the Capnocytophaga species. Studies supporting this hypothesis have shown that preeclamptic women are infected in higher proportion as compared to nonpreeclamptic or normal pregnant women.\textsuperscript{[10]}

Hormonal fluctuations during a female’s lifetime may produce an exaggerated inflammatory response to dental plaque, resulting in gingivitis. Dental plaque deposition and high inflammatory gingiva (bleeding and redness) are typical features of hormone-associated gingivitis. Symptoms vary according to individuals’ responses to hormonal changes. Susceptibility to infections (e.g., periodontal infection) increases due to variations in the immune system and can be elucidated by the hormonal changes and suppression of T-cell activity, decreased neutrophil chemotaxis and phagocytosis, altered lymphocyte response and depressed antibody production, chronic maternal stress, and even nutritional deficiency concomitant with increased nutritional demand by both the mother and the fetus. The amount of dental plaque and inflammation is not dose dependent; thus, even in a small quantity of irritants can induce heightened response depending on the individuals’ immune reaction.

Hormone-associated gingivitis is reversible following puberty or pregnancy, and no radiographic bone loss is observed.\textsuperscript{[11]} The incidence of periodontitis can be as high as 23\% in adult women, but half of the women with periodontitis above the age group of 55 years still have healthy teeth.\textsuperscript{[12]}

As a woman’s health changes throughout her lifetime from prepuberty to puberty to postmenopausal, the body undergoes continuous changes in hormonal levels throughout the life cycle, thus leading to change in the body environment with specific oral health implications. Furthermore, menopausal and postmenopausal women have low levels of sex hormone levels, which also have some oral health problems associated with gingival.\textsuperscript{[13]}

**Puberty**

Puberty is associated with increased levels of progesterone and estrogen levels. These hormones act as a growth factor for the periodontis causing bacteria to grow and dwell in this specific age group; thus, periodontis is more common in the pubertal age group as compared to children in their prepubertal stage. Gingivitis associated with hormonal presence is highly sensitive to their presence and they return to normal condition in the circumpubertal stage. Most females with healthy gingivae likely will not develop significant periodontal changes. A higher population of bacteria in subgingival pockets is present during puberty, which may selectively accumulate estradiol and progesterone.\textsuperscript{[9]} Meticulous oral home care consisting of regular brushing and flossing and routine dental visits may help prevent the development of gingivitis.

**Menstruation**

Many women report an increase in gingival inflammation and discomfort associated with their menstrual cycle. “Gingival inflammation was lower during menstruation than during ovulation and premenstruation. “This may be attributed to the hormone known as serum estradiol, which is a natural form of estrogen that peaks and drops during ovulation and premenstruation. During the luteal phase of the menstrual cycle, levels of progesterone peak leading to elevated inflammatory changes in gingiva and periodontium. It is at its peak at the beginning of the cycle which eventually subsides with time. This can be hypothesized due to changes in bacterial flora.\textsuperscript{[14]}

**Pregnancy**

Maternal periodontal disease strongly influences pregnancy outcome is a well-known fact.\textsuperscript{[15]} Researchers consider that hematogenous transport of bacteria and/or pro-inflammatory mediators from sites of periodontal infection into the placenta, fetal membranes, and amniotic cavity induces pathological processes that lead to these adverse outcomes. An exaggerated gingival inflammatory response to dental plaque is the primary cause of gingivitis, which usually starts around the 2\textsuperscript{nd} month of pregnancy and generally resolves following parturition. In addition, pregnancy may accelerate the development of periodontitis (deep pockets and bone loss around teeth).\textsuperscript{[16]} There may also be a link between periodontitis and adverse pregnancy outcomes, including preterm delivery and low birth weight babies.\textsuperscript{[17]}

Periodontal treatment of the pregnant patient has been controversial. Findings from one clinical study reported that periodontal treatment during the second and third trimesters of pregnancy is safe, but the risk of adverse effects, such as preterm delivery (birth occurring before 37 weeks of pregnancy), low birth weight, fetal growth restriction, or preeclampsia, is not reduced.\textsuperscript{[18,19]} On the other hand, data from four clinical trials found that periodontal treatment in pregnant women may lower the incidence of preterm delivery and low birth weight babies. Maternal and fetal exposures to Gram-negative periodontal bacteria may trigger inflammatory events in both the mother and the fetus, which may stimulate the early rupture of membranes and parturition.\textsuperscript{[20]}

**Use of Oral Contraceptives**

Oral contraceptives enhance periodontal break down reducing the resistance to dental plaque and can
induce gingival enlargement in otherwise healthy females.

The long-term use of oral contraceptives may cause clinical attachment loss, increased gingival inflammation, and gingival enlargement. Therefore, it is expected that the same gingival changes seen during pregnancy will also be seen in women taking oral contraceptives. Women using contraceptives have poor periodontal and gingival health. Currently, there are newer oral contraceptive formulations in the market which contain a lower concentration of hormones, resulting in a milder inflammatory response of the gingival to dental plaque.

**Menopause**

Menopause is associated with important systemic and oral changes. The sudden decrease in estrogen levels that happens in menopause is considered to be the main cause of primary osteoporosis, which also affects jawbones. It has been suggested that this reduction in bone mineral density could contribute to the progression of periodontal disease. Besides their effect on bone, estrogens also interfere with other periodontal tissues (gingiva and periodontal ligament) and influence host immune-inflammatory responses. Change in salivary viscosity, especially in postmenopausal women, also leads to a change in the microbial environment of the periodontium.

The relationship between menopause and periodontal disease is difficult to establish due to the multitude of factors involved. If any relationship is found, it will always be less significant comparing to other well-known risk factors of periodontal disease.

Bisphosphonate-associated osteonecrosis of the jaws is characterized by pain or swelling in the affected jaw, an irregular mucosal ulceration with exposed bone in the mandible or maxilla, and infection. The pharmacist and dentist need to be aware that the patient will be or currently is taking a bisphosphonate. Commonly prescribed bisphosphonates include zoledronic acid (Zometa), pamidronate (Aredia), alendronate (Fosamax), ibandronate (Boniva), risedronate (Actonel), tiludronate (Skelid), clodronate (Bonefos), and etidronate (Didronel).

Additional oral symptoms experienced by postmenopausal women include burning oral sensations – especially on the tongue – altered taste sensations, and a decrease in salivary flow. If xerostomia is present, dental plaque may accumulate secondary to reduced salivary function/flow, leading to caries and, possibly, gingivitis.

**Conclusion**

Female sex hormone influences the bacterial flora further leading to onset and progression of the gingivitis and periodontal disease during puberty, menstruation, and pregnancy.

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**Conflicts of interest**

There are no conflicts of interest.

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