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Expression of Hormonal Receptors (FSHR, ER-α, and PR) in Women with Pregnancy-Associated Periodontal Disease: A Molecular PCR-Based Study

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Annotation: Background: The response of gingival tissues and the susceptibility to inflammation are THUS1170 EX8524 MODERATED BY HORMONAL CHANGES. Detection of hormonal receptor (follicle-stimulating hormone receptor (FSHR), estrogen receptor alpha (ERα), and progesterone receptor (PR) genes in gingival tissues could be a crucial factor in the pathogenesis of periodontal disease. Objective To investigate molecular detection of follicle-stimulating hormone receptor (FSHR), estrogen receptor alpha (ER-α), and progesterone receptor (PR) genes among pregnant women with and without periodontal disease and compared to healthy non-pregnant women.

Methodology: Gingival tissue samples were collected from three study groups: pregnant women with periodontal disease, healthy pregnant women, and non-pregnant control women. The presence of FSHR, ERα, and PR genes was analyzed using conventional PCR.

Results: Altered detection patterns of hormonal receptors were observed in pregnant women with periodontal disease. Increased detection rates of ERα and PR were associated with higher inflammatory status, whereas FSHR detection demonstrated variable patterns among the studied groups.

Conclusion: Pregnancy-associated

periodontal disease is linked to differential detection of sex hormone receptors in gingival tissues. These molecular alterations may contribute to increased susceptibility to inflammation and periodontal tissue breakdown during pregnancy.

Keywords: Hormone receptors, FSHR, Estrogen receptor, Progesterone receptor, PCR, Periodontal.

1. Introduction

Estrogen plays a major role in uterine growth, increasing bone mineral density, and the development and maintenance of primary and secondary sexual characteristics. Estradiol is the most potent naturally occurring estrogen, functioning through specific receptors in target cells known as ERα and ERβ (Hewitt & Korach, 2002). Also, the progesterone is another essential female hormone, belongs to the class of steroid hormones called progestins. It is derived from pregnane, a 21-carbon saturated steroid hydrocarbon. Similar to estrogen, progesterone exerts its effects via specific progesterone receptors. Two isoforms of these receptors, PR-A and PR-B, have been identified in most rodents and humans (Sathish *et al.,* 2022). To be effective in an organ system, a hormone must be able to induce genetic changes; for this to happen, it needs to locate its target cells that possess the appropriate receptors and metabolizing factors within that system. Estrogen and progesterone receptors are found in gingival tissues, periosteal fibroblasts, lamina propria fibroblasts, ligament fibroblasts, and osteoblasts (Ramamurthy, 2015). Although sex steroid hormone receptors are not widely distributed, they are highly concentrated in target tissues responsive to hormones. Since human gingiva contains specific estrogen and progesterone receptors, the periodontium is considered one of these target tissues (Singh *et al.*, 2013). Hormonal changes, together with the presence of dental plaque, are believed to contribute to the alterations observed in periodontal tissues during pregnancy. The localization of estrogen receptors (ER) and progesterone receptors (PgR) in the human periodontium confirms that it is one of the target tissues for these hormones (Jawed & Jawed, 2025). The rising levels of estrogen and progesterone during pregnancy are thought to be responsible for the exaggerated inflammatory response to plaque, leading to the progression of gingivitis (Kshirsagar & Balamurugan, 2018). Estrogen and progesterone are responsible for various physiological changes in women. Specifically, estrogens influence the cytodifferentiation of stratified squamous epithelium and contribute to the synthesis and maintenance of fibrous collagen (Bhardwaj & Bhardwaj, 2012). Estrogen receptors identified in osteoblast-like cells provide a mechanism for the hormone's direct action on bone. These receptors have also been located in periosteal fibroblasts, scattered fibroblasts within the lamina propria, and fibroblasts of the periodontal ligament (PDL), supporting the concept of a direct action of sex hormones on multiple periodontal tissues (Al-Sherbini *et al.,* 2014). Thus, the estrogen and progesterone receptors are also present in various periodontal components, particularly the gingiva and periodontal ligaments. Estrogen has been shown to exert multiple effects on periodontal tissues. By regulating epithelial glycogen production, estrogen reduces gingival keratinization, which compromises the integrity of the epithelial barrier. It also down regulates T-lymphocyte activity, contributing to an increased incidence of gingival inflammation, even in the absence of elevated dental plaque levels. Estrogen also plays a role in fibroblast proliferation and extracellular matrix synthesis and maturation of gingival connective tissues. This suppresses leukocyte formation in the bone marrow, consequently decreasing release of pro-inflammatory cytokines from marrow-deprived cells (Peruga et al., 2023). In females, the follicle-stimulating hormone

receptor (FSHR) mediates follicular development and estrogen production, being expressed on the granulose cells of the ovary in response to the FSH from the pituitary. Thus, FSH was historically thought to be involved in maintaining pregnancy only through events that occurred in the ovarian follicular phase, contributing little to progression beyond ovulation (Stilley & Segaloff, 2018). Yet, beyond the ovary, there is an extra gonadal expression of FSHR, which points to additional physiological roles for FSH [7]. FSHR polymorphisms have been implicated in female reproductive dysfunctions, including polycystic ovary syndrome (PCOS) and amenorrhea (Zilaitiene et al., 2018). FSH receptor (FSHR)—first identified to be involved in gonadal function—has recently been found to be expressed in a wide range of extra gonadal tissues such as the placenta, umbilical vein, various types of vascular smooth muscle cells, placental endothelial cells, myometrium, fallopian tube, liver, bone osteoclasts, monocytes, endometrial stromal and glandular epithelial cells (Chen et al., 2025). These results provide evidence that specific polymorphisms of gonadotropin receptors are particularly relevant to ovarian function and to the response to exogenous gonadotropins (Chrusciel et al., 2019). Antioxidants are particularly important during pregnancy, particularly to oppose increased oxidative stress and to preserve maternal and fetal health. Oxidative stress is considered pathophysiological mechanism for a number of pregnancy complications, such as preeclampsia, gestational diabetes mellitus, premature birth and stunted fetal growth (Sies et al, 2017). The relationship between ROS and antioxidants represents an important physiological equilibrium, where ROS cell signalling is counteracted by the antioxidant properties of antioxidants to prevent cellular damage resulting from overactivity of ROS (Obeagu et al., 2024). By contrast, a reduction of antioxidant homeostasis as a result of low levels of non-enzymatic antioxidants (such as glutathione, vitamins C and E) and/or enzymatic ones (such as superoxide dismutase, glutathione peroxidase, and catalase) exposes cells to oxidative stress [7]. Disruption of this balance gives rise to oxidative stress (Poston et al., 2011), even in physiological conditions, where redox balance is maintained by strict regulation of reactive oxygen species (ROS) production and antioxidant defenses.

2. Methodology

This study was conducted through cooperation between the Department of Biology, College of Science, Wasit University, and several medical and laboratory centers in Wasit Province, Iraq. All clinical examinations were carried out under the supervision of a qualified dentist. The study population consisted of women aged 16–40 years who were clinically and laboratorially evaluated for periodontal status. Participants were divided into three groups. The first group included fifty (50) pregnant women diagnosed with gingivitis following clinical periodontal examination. These participants exhibited one or more clinical signs, including gingival redness, swelling, tenderness, bleeding during brushing or flossing, plaque and calculus accumulation, periodontal pocket formation, halitosis, and dental caries. Group two (15) women with clinically healthy gingiva and healthy pregnancy. The third group consisted of fifteen (15) healthy non-pregnant women without periodontal and systemic diseases that served as controls. Individuals were excluded if they were women with systemic diseases that may have affected the oral environment or had received antibiotics, anti-inflammatory drugs in the period preceding the examination, systemic diseases, those who were currently taking other medications affecting periodontal condition, and those who were obese. All control participants were in good general health and showed no clinical signs of periodontal disease.

2.1 Blood Sample Collection

Genomic DNA was extracted from whole blood samples using standard DNA isolation protocols. Venous blood samples (5 mL) were collected from the antecubital vein of each participant using sterile disposable syringes. The collected blood was divided into two portions: one portion was transferred into EDTA tubes for genomic DNA extraction, while the second portion was transferred into gel tubes and centrifuged at 5000 rpm for 10 minutes to obtain

serum. The separated serum was stored at −20°C until further biochemical and immunological analyses were performed.

2.2 Molecular and Biochemical Analysis

Molecular and oxidative stress analyses were performed for all study participants. Molecular analysis was conducted to detect the presence of follicle-stimulating hormone receptor (FSHR), estrogen receptor alpha (ER-α), and progesterone receptor (PgR) using conventional polymerase chain reaction (PCR). Genomic DNA was extracted from whole blood samples using standard DNA isolation protocols, and all reagents were used according to the manufacturers' instructions' In addition, serum levels of oxidative stress parameters were evaluated. The concentrations of total oxidant status (TOS) and total antioxidant status (TAS) were measured using standard colorimetric assay methods according to the manufacturers' protocols.

2.3 Statistical analysis

Data was collected, summarized, analyzed, and presented using the statistical package for social sciences (SPSS) version 26. The numerical data were presented as (mean ± standard deviation) after performing the Kolmogorov-Smirnov normality test to determine the distribution pattern. Chi-square test as well as a one-way ANOVA tests were applied, followed by Duncan's multiple range test (DMRTs) as a post hoc analysis to assess differences among groups. Besides, Pearson's correlation was used to evaluate the relationship between two numerical variables. The P-value of < 0.05 was considered statistically significant (Daniel & Cross, 2018).

3. Results

The detection of the FSH receptor gene was performed in the study groups using specific PCR primers for molecular analysis. In current study, the FSH receptor gene (345 bp product) was detected in 27 (54.0%) of pregnant women with periodontal disease (PD), 9 (60.0%) of healthy pregnant women, and all 15 (100.0%) healthy non-pregnant women, as shown in figure 1-1. The difference between the groups was statistically significant ($p = 0.005$).

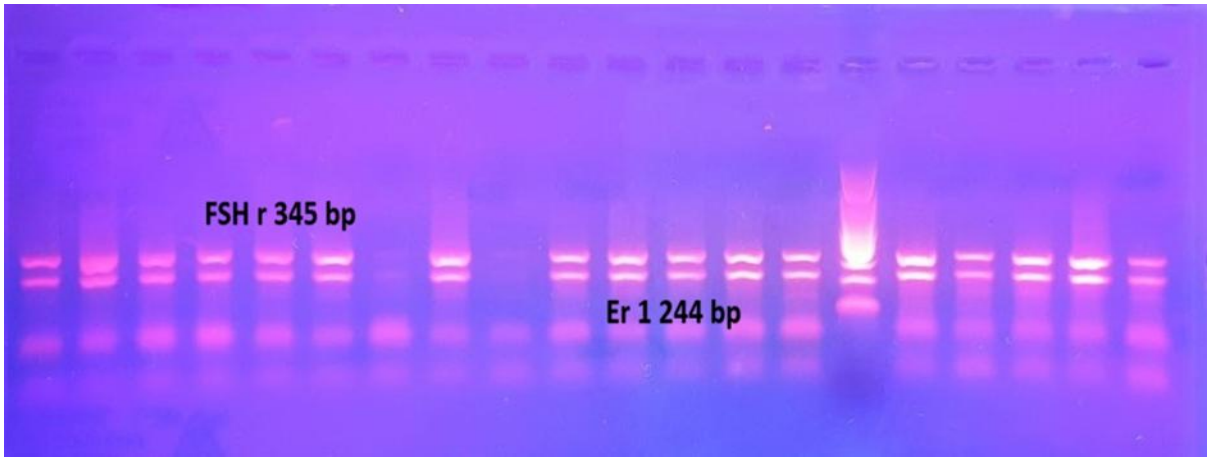

Figure (1-1): An agarose gel electrophoresis image that shows the PCR product analysis of the FSH gene

A specific PCR primer was used to detect the presence of the E2-α receptor gene (244 bp product) among the study groups. The gene was identified in 46 out of 50 (92.0%) of pregnant women with periodontal disease (PD), 9 out of 15 (60.0%) of healthy pregnant women, and all 15 (100.0%) of healthy non-pregnant women, as shown in figure 1-2. The difference in detection rates among the groups was statistically significant ($p = 0.001$), suggesting a possible association between periodontal inflammation and altered expression or presence of the E2-α receptor gene during pregnancy.

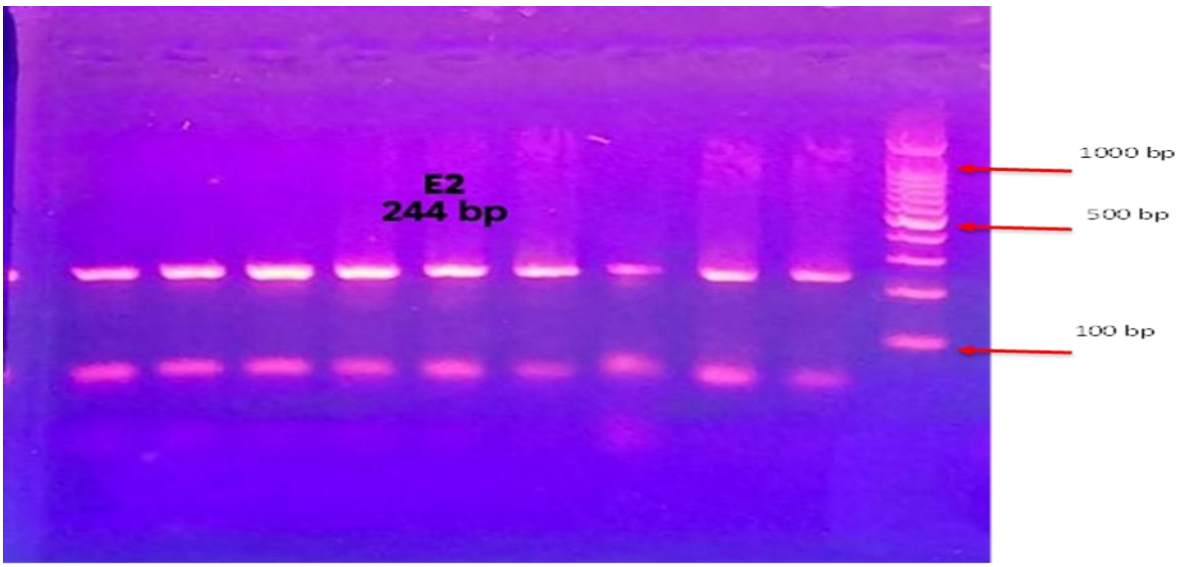

Figure (1-2): An agarose gel electrophoresis image that shows the PCR product analysis of the E2-α gene

A specific PCR primer was used to detect the presence of the progesterone receptor (PR) gene (833 bp product in both patients and controls) in the study groups. The gene was identified in 6 out of 15 (40.0%) healthy non-pregnant women, 11 out of 50 (22.0%) of pregnant women with periodontal disease (PD), and in none of the healthy pregnant women (0%), as shown in figure 1-3. The difference in detection rates among the groups was statistically significant ($p = 0.027$), suggesting a potential association between pregnancy status, periodontal inflammation, and the expression of the progesterone receptor gene.

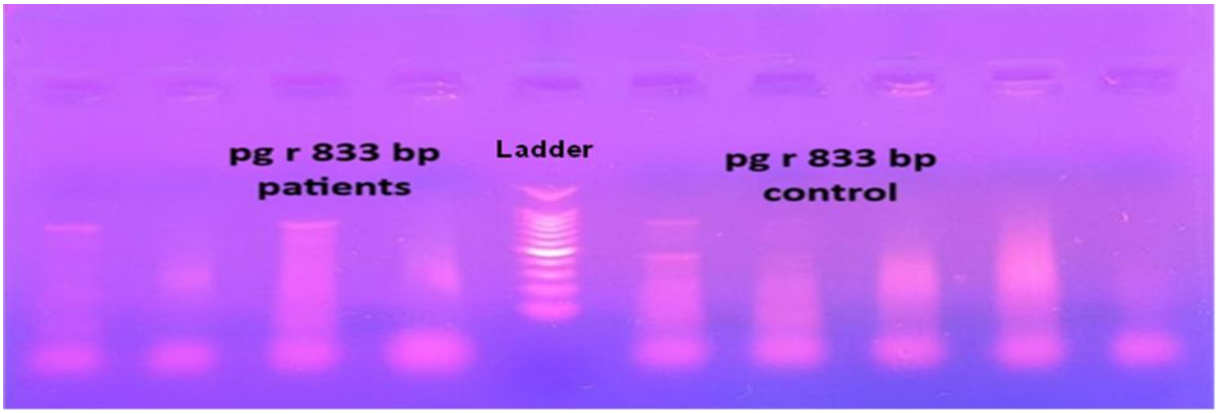

Figure (1-3): An agarose gel electrophoresis image that shows the PCR product analysis of the progesterone gene

The serum total oxidant status (TOS) among pregnant women with PD, healthy pregnant women, and non-pregnant controls was evaluated. Mean serum TOS levels were increased significantly ($P < 0.001$) in both groups of pregnant women respectively compared to healthy non-pregnant women. However, there was no significant difference ($P > 0.05$) between the two pregnant groups (with PD and healthy), as shown in Table (1-1).

Table (1-1): Total oxidant capacity in pregnant with PD, healthy pregnant and non-pregnant women participants groups

Groups		Total Oxidation Capacity (U/mL)
Pregnant with PD	Mean ± SD	0.32 ± 0.06^{A}
	Range	0.21-0.67
Healthy Pregnant	Mean ± SD	0.33 ± 0.05^{A}
	Range	0.20-0.40

Healthy non-Pregnant	Mean ± SD	0.23 ± 0.04^{B}
	Range	0.17-0.34
p-value		0.001** †
Means followed by different superscript letters are significantly different according to Duncan's multiple range comparisons (DMRTs). Means followed by the same superscript letter are not significantly different.		

The total antioxidant capacity (T-AOC) in pregnant women with PD, healthy pregnant and non-pregnant women. The serum TAOC levels were decreased significantly ($P < 0.001$) in both groups of pregnant women compared to healthy non-pregnant women. However, there was no significant difference ($P > 0.05$) between pregnant women with periodontal disease and healthy pregnant women shown in Table (1-2).

Table (1-2): Total antioxidants capacity in pregnant with PD, healthy pregnant and non-pregnant women participants groups

Groups		Total Antioxidants Capacity U/mL
Pregnant with PD	Mean ± SD	0.177 ± 0.05^{A}
	Range	0.07-0.26
Healthy Pregnant	Mean ± SD	0.164 ± 0.04^{A}
	Range	0.07-0.22
Healthy non-Pregnant	Mean ± SD	0.552 ± 0.11^{B}
	Range	0.34-0.78
p-value		0.001** †
Means followed by different superscript letters are significantly different according to Duncan's multiple range comparisons (DMRTs). Means followed by the same superscript letter are not significantly different.		

4. Discussion

These findings suggest that periodontal inflammation during pregnancy may influence ER-α gene expression, potentially reflecting either an amplified inflammatory response or a compensatory mechanism in response to tissue damage. The dramatic fluctuations in estrogen levels during pregnancy are reflected by simultaneous influences on the expression of estrogen receptors (ER)—in particular ER–α—in many tissues (gingival). Inflammation can also exert modulation over ER expression, presumably to allow for some sort of counterbalance to maintain tissue homeostasis (Gilliver, 2010). Estrogens can affect immune functions by modulating pathways involved in inflammation and cytokine production. They can have pro-inflammatory as well as anti-inflammatory effects through their receptors depending on the physiological background (Straub, 2007). The full expression observed in healthy non-pregnant women (100%) may indicate normal or basal ER-α expression under stable physiological conditions, in the absence of pregnancy-related hormonal fluctuations or inflammatory stress. These data confirm that ER-α are expressed in a variety of tissues, even in the absence of their ligands, where ER-α may participate in maintaining homeostasis, regulating physiological functions within the cell (Kovats, 2015). These results indicate that PR gene expression may be regulated by not only pregnancy status, but also by local oral microenvironmental factor(s), specifically interactions with periodontopathogens including Porphyromonas gingivalis. Peters et al. found that in healthy non-pregnant women, increased P. gingivalis counts associated with higher progesterone were able to sperate natural from pathological dla, suggesting that progesterone can modulate the gingival environment and trigger expression of receptor-related genes even in non-pregnant situations (Peters et al., 2017). Such a complexity may explain high PR gene expression in non-pregnant controls suggesting a biological interplay, not merely

confined to inflammation or endocrine activity (Massoni et al., 2019).

Moreover, in some pregnant women, "functional progesterone withdrawal appears to take place because of inflammatory signaling that interferes with the receptor, causing suppression of receptor activity despite high levels of hormone." IL-1 β stimulation has been found to suppress PR activity, exemplifying the interplay of gingival microbiota, inflammation and PR signaling (Lee et al., 2012). Chronic inflammation in periodontal disease could decrease FSHR expression levels during pregnancy. There is a dramatic increase in circulating reproductive hormones (tested in pregnant women: progesterone: +17,000% or estrogen: +1000%) during pregnancy which has the potential to alter the gene expression of hormone receptors including FSHR. Hormonal changes may decrease the sensitivity of tissues and/or decrease receptor expression (Smith & Johnson, 2022). The chronic inflammation characteristics of the periodontal disease appears to interfere with epigenetic regulation of tunable genes to develop functional suppression of FSHR expression. This can lead to long-lasting changes in gene expression, hormonal response, and reproductiveility (Liu et al., 2010). The marked increase in total oxidant status (TOS) in serum of both group of pregnant women irrespective of their periodontal status, as compared to healthy apparently non-pregnant women, is a well recognised feature of pregnancy; confirming the known state of increased oxidative stress during this physiological condition. Pregnancy is associated with considerable metabolic and physiologic changes, such as increased placental uptake of oxygen, increased mitochondrial respiration, and increased oxygen consumption that leads to overproduction of reactive oxygen species (ROS) and increased oxidative stress status (Akalın et al., 2009).

Higher TOS and lower T-AOC have, consistently, been ascribed to placental metabolism and vascular remodelling with increased oxidants production and relative oxidants excess [19,20,21] during pregnancy (Tóthová & Celec, 2017). Even more support exists for this oxidative shift in the form of evidence that markers of lipid peroxidation like 8-iso-prostaglandin F2α increase progressively over pregnancy and the postpartum period (in concert with a decrease in major antioxidant defenses such as α-tocopherol) (Ishihara et al., 2004). Pregnancy is accompanied by several complex physiological modifications, such as increased metabolism of the placenta, which leads to the excessive generation of reactive oxygen species (ROS) and free oxygen radicals [7]. Such disbalance raises oxidative stress and utilizes available antioxidant reserves leading to significantly lower T–AOCs (Burton & Jauniaux, 2011). Placental ROS production, and nitric oxide synthase activity, continue to rise, contributing to further oxidative stress, which then perpetuates a cycle of increased oxidative stress if not buffered appropriately by antioxidants leading to impaired placental function and poor pregnancy outcome (Myatt & Cui, 2004). Regarding the obstetric relevance of the mentioned biochemistry, the reduction in antioxidant capacity in the future mothers is relevant as it leads to other complications (such as preeclampsia, preterm birth and intrauterine growth restriction [15]) which are more frequent in this population than in other reproductive group of women. Oxidative stress further triggers inflammatory pathways, which directly leads to vascular dysfunction and poor function of pregnancy-related tissues (Chapple & Matthews, 2007). Conversely, the levels of T-AOC were higher in non-pregnant women, which may suggest a better equilibrium between ROS generation and antioxidant defense mechanisms. This finding support the view of pregnancy as a natrual biological stressor who put on strong machine the maternal oxidative system. In addition, despite some compensatory mechanisms being activated, these are usually unable to compensate for the heightened oxidative burden of pregnancy (Agarwal et al., 2012). Also, periodontal disease aggravates oxidative stress by further increasing local inflammation and inducing the cytokine release, leading to free radical overproduction (Mohideen et al., 2023). This may be the case for reduced salivary and gingival antioxidant defenses thus the depletion of systemic antioxidant capacity may translate to a greater susceptibility to and more severe gingival inflammation be evident during pregnancy (Chapple, 2006).

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