*Applied Chemical Engineering (2025) Volume 9 Issue 1* ISSN: 2578-2010 (O)
doi: 10.59429/ace.v9i1.5894

ORIGINAL ARTICLE

# Functional Role of Female Sex Hormones and Receptors in Cervical Carcinoma

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**ARTICLE INFO**

Received: 23 January 2026
Accepted: 06 February 2026
Available online: 27 February 2026



## ABSTRACT

Cervical carcinoma remains one of the most prevalent malignancies affecting women worldwide. While human papillomavirus (HPV) infection is a well-established etiological factor, growing evidence suggests that female sex hormones and their receptors may significantly influence the pathogenesis and progression of cervical cancer. Hormonal imbalances, particularly involving estrogen and progesterone, may impact tumor growth and immune modulation within the tumor microenvironment. This study investigates the functional role of selected female sex hormones and their corresponding receptors in Iraqi women diagnosed with cervical carcinoma. A total of 60 female participants were enrolled from gynecology and oncology departments in Baghdad and Karbala, Iraq. Thirty women with histologically confirmed cervical carcinoma were assigned to the patient group, while thirty age-matched healthy volunteers served as controls. Blood samples were collected under fasting conditions, and serum levels of sex hormone-binding globulin (SHBG), estradiol (E2), dehydroepiandrosterone sulfate (DHEAS), progesterone, estrogen receptor alpha (ERα), and progesterone receptor (PR) were quantified using standardized ELISA kits. Statistical comparisons were conducted using SPSS with significance set at $p < 0.05$. Significant hormonal differences were observed between patient and control groups. Estradiol levels were markedly lower in postmenopausal patients than in premenopausal patients, while DHEAS levels were significantly higher in the same comparison. SHBG and DHEAS levels were also elevated in patients compared to controls. Importantly, ERα levels were significantly decreased, while PR levels were significantly increased in cervical cancer patients relative to controls. A strong association was observed between cervical cancer diagnosis and positive family history (73.3% in patients vs. 13% in controls). Our findings suggest that imbalances in circulating sex hormones and alterations in receptor expression—particularly reduced ERα and elevated PR, may contribute to cervical carcinogenesis. These hormonal and biochemical signatures, alongside potential hereditary patterns, highlight the importance of incorporating hormonal profiling in the clinical evaluation of cervical cancer. Further research is warranted to explore the therapeutic

potential of targeting hormone receptor pathways in cervical cancer management.

***Keywords:*** Cervical carcinoma; estrogen receptor alpha; progesterone receptor; sex hormone; SHBG, DHEAS; estradiol

# 1. Introduction

Cervical carcinoma remains one of the most prevalent cancers among women globally, ranking as the fourth most common cancer in terms of both incidence and mortality [1]. Despite major advances in screening and vaccination programs, cervical cancer continues to pose a significant public health burden, particularly in low- and middle-income countries, where access to preventive care remains limited. In Iraq and across many Middle Eastern nations, the epidemiological landscape of cervical cancer is poorly characterized, and studies examining its molecular and hormonal underpinnings are scarce. This reality underlines the urgent need for context-specific research that not only explores known viral and environmental risk factors but also integrates emerging molecular insights—including those related to sex hormones and their receptors [2].

While persistent infection with high-risk human papillomavirus (HPV) types is widely accepted as the primary etiological factor, it is becoming increasingly evident that additional cofactors may influence the initiation and progression of cervical cancer. Among these, hormonal regulation—particularly through estrogen and progesterone—has garnered increasing attention in recent years [3]. Cervical carcinoma is now considered a hormone-responsive malignancy, although the specific endocrine pathways involved remain poorly understood. Several observational and mechanistic studies have proposed that hormonal fluctuations across a woman's reproductive lifespan, including those linked to menopause, oral contraceptive use, and pregnancy, may contribute to changes in cervical epithelial biology and immune surveillance, thereby modifying cancer risk [3,4].

Estrogen, the principal female sex hormone, has been shown to play a multifaceted role in oncogenesis. Its biological effects are primarily mediated through nuclear hormone receptors, estrogen receptor alpha (ERα) and estrogen receptor beta (ERβ). Upon ligand binding, these receptors dimerize and translocate to the nucleus, where they regulate the transcription of genes involved in cell proliferation, differentiation, apoptosis, and angiogenesis [3]. In cervical cancer models, estrogen signaling has been found to drive tumor cell proliferation and disrupt apoptotic pathways, especially through ERα-dependent gene activation [4]. Furthermore, estrogen is increasingly implicated in modulating the tumor microenvironment by affecting immune cell infiltration, suppressing cytotoxic T-cell responses, and promoting a pro-tumorigenic milieu through its immunosuppressive effects [4].

However, in contrast to hormone-dependent malignancies such as breast and endometrial cancers, where the role of estrogen signaling is well-defined and integrated into therapeutic strategies, the involvement of sex hormones in cervical cancer remains less clearly delineated [5]. One area of particular ambiguity is the regulation of 17β-estradiol (E2) and its interaction with estrogen receptors in cervical epithelial and tumor cells. Although E2 is the most potent form of circulating estrogen, the extent to which it contributes to cervical carcinogenesis, either independently or synergistically with HPV oncoproteins, is not yet fully understood [6]. Moreover, differences in hormone receptor expression between premenopausal and postmenopausal women may further complicate disease behavior and therapeutic outcomes.

In addition to estrogen, progesterone is another critical sex hormone with emerging relevance in cervical cancer biology. Progesterone exerts its actions via the progesterone receptor (PR), a ligand-activated transcription factor that can influence gene expression, immune cell recruitment, and stromal remodeling. Preclinical studies have demonstrated that the synthetic progestin medroxyprogesterone acetate (MPA) can suppress cervical cancer cell growth through mechanisms involving p14ARF suppression, p53 hyperphosphorylation, and reduced expression of matrix metalloproteinases [7]. These findings suggest a

possible protective role for progesterone in cervical carcinogenesis, although the literature remains divided on the consistency and clinical relevance of PR expression in tumor tissues [8].

Despite the suggestive evidence linking sex hormones and their receptors to cervical cancer development, there remains a striking lack of clinical studies assessing these biomarkers in diverse patient populations, particularly from Middle Eastern or Arab regions. Most existing studies have been conducted in Western or East Asian populations, limiting their generalizability. To address this gap, the present study investigates the serum levels of sex hormone binding globulin (SHBG), estradiol (E2), dehydroepiandrosterone sulfate (DHEAS), progesterone, ERα, and PR among Iraqi women diagnosed with cervical carcinoma. By stratifying the analysis across premenopausal and postmenopausal groups and comparing them with healthy controls, this study aims to elucidate the endocrine signatures associated with cervical cancer in this understudied population.

The findings of this research have the potential to contribute to a better understanding of hormonal involvement in cervical cancer pathogenesis and to identify candidate biomarkers that could inform early detection strategies or hormonal interventions. Moreover, exploring hormone-receptor profiles in relation to menopausal status and family history may uncover important genetic or endocrine patterns that have not yet been sufficiently investigated in regional or global cancer literature.

# 2. Materials and methods

## 2.1. Study population

This cross-sectional, observational study was conducted at the Iraqi National Center for Cancer Research in Baghdad, in collaboration with the College of Medicine, University of Karbala. The research was carried out between July 2023 and March 2024. Ethical approval was obtained from the Institutional Review Board (IRB) of the University of Baghdad, and all study procedures complied with the ethical principles outlined in the Declaration of Helsinki. Written informed consent was obtained from all participants after they were provided with detailed information regarding the study objectives, procedures, confidentiality assurances, and their right to withdraw at any time without consequence.

## 2.2. Sample collection

A total of 60 adult female participants were recruited for this study using a purposive sampling strategy. Thirty patients with histologically confirmed cervical carcinoma (of any clinical stage) were enrolled from the gynecology and oncology departments and formed the patient group. The control group consisted of 30 age-matched healthy women without any history of cancer, selected from hospital visitors and outpatient clinics.

Participants were eligible if they met the following inclusion criteria: (1) aged 25 years or older, (2) not currently receiving hormone replacement therapy or oral contraceptives within the past two months, (3) no prior history of other malignancies, and (4) willingness to provide informed consent. Women were excluded if they had other active cancers, endocrine disorders, autoimmune diseases, recent infections, or were pregnant at the time of data collection.

## 2.3. Blood sample collection and processing

Blood samples (5–10 mL) were collected from participants via venipuncture in the early morning following an overnight fast (8–10 hours) to control for diurnal and dietary hormonal fluctuations. Samples were drawn using sterile needles and transferred to serum-separating tubes. After clotting at room temperature, the samples were centrifuged at 3000 rpm for 10 minutes. The resulting serum was aliquoted into labeled tubes and stored at −20°C until laboratory analysis.

### 2.4. Biochemical analysis

Serum concentrations of sex hormone-binding globulin (SHBG), estradiol (E2), dehydroepiandrosterone sulfate (DHEAS), progesterone, estrogen receptor alpha (ERα), and progesterone receptor (PR) were measured using commercially available enzyme-linked immunosorbent assay (ELISA) kits (Elabscience Biotechnology Inc., USA), following the manufacturer's protocols. All hormone assays were performed in duplicate to ensure accuracy and reproducibility. The sensitivity of each assay, along with intra-assay and inter-assay coefficients of variation, were within acceptable limits as reported by the manufacturer, thereby ensuring the reliability of the quantitative results.

### 2.5. Family history and menopausal status

Sociodemographic and clinical data were collected using a structured questionnaire and medical records. Variables recorded included age, menopausal status, smoking history, use of hormonal contraceptives, and family history of cancer. Menopausal status was classified as premenopausal or postmenopausal based on self-reported menstrual history corroborated by clinical examination.

### 2.6. Statistical analysis

All statistical analyses were conducted using IBM SPSS Statistics (version XX, IBM Corp., Armonk, NY). Descriptive statistics were used to summarize participant characteristics. Continuous variables were expressed as mean ± standard deviation (SD) or median with interquartile range (IQR) as appropriate. The Shapiro–Wilk test was used to assess normality of distribution.

Comparisons between groups (patients vs. controls; premenopausal vs. postmenopausal) were performed using independent samples t-tests for normally distributed variables and Mann–Whitney U tests for non-parametric data. Categorical variables were analyzed using chi-square or Fisher's exact tests. A p-value of less than 0.05 was considered statistically significant.

## 3. Results

As showed in table 1, and figure 1 the age of the participants patients had a mean age of 44.4 ± 9.8 years, while the control group had a mean age of 43.3 ± 6.9 years.

**Table 1.** Age range in study groups

| Variables | Controls<br>Mean ± SD | Patients<br>Mean ± SD |
|---|---|---|
| Age (years) | 43.3 + 6.9 | 44.4 + 9.8 |
| p-value | 0.06 | |

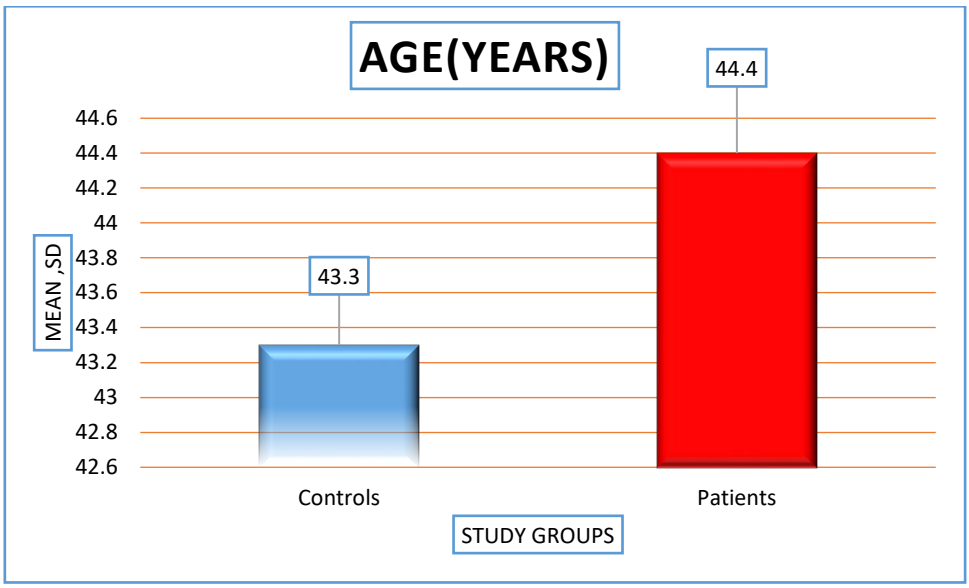


**Figure 1.** Age range in study groups.

Family history was noted in the study population, with 73.3% of patients reporting a family history, compared to 13% in the control group. Regarding smoking status and menstrual status, among the patients, 60% (18 individuals) were premenopausal, and 40% (12 individuals) were postmenopausal. In the control group, 53.3% (16 individuals) were premenopausal, and 46.6% (14 individuals) were postmenopausal, as seen in table 2, and figure 2.

**Table 2.** Sociodemographic data of the study groups.

| Variables | Controls | Patients |
|---|---|---|
| Family history | 13% | 73.3% |
| Smoking | 43.3% | 56.6% |
| Menstrual status | | |
| Premenopausal | 16 (53.3%) | 18(60%) |
| Postmenopausal | 14(46.6%) | 12(40%) |

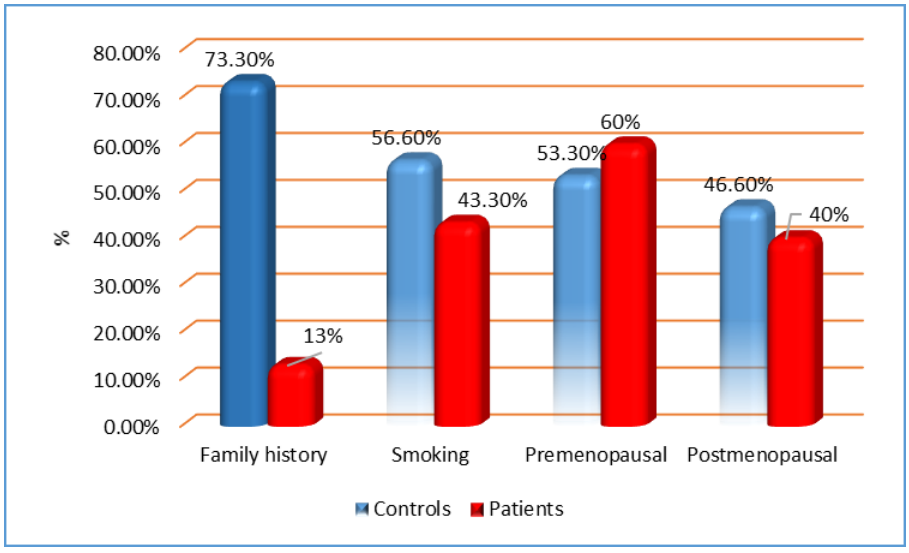


**Figure 2.** ociodemographic data of the study groups.

Table 3 shows the median level of hormones in pre and postmenopausal women participants. In the study, the median SHBG levels in postmenopausal women had a median of 77.5 (21.3), premenopausal women had a median of 81.4 (21.5), the control group for postmenopausal women had a median of 80 (13.2), and the control group for premenopausal women had a median of 71.1 (8.46). The median estradiol levels with interquartile ranges (IQR) showed that postmenopausal women had a median of 35.3 (10.46), premenopausal women had a median of 102.2 (27.9), the control group for postmenopausal women had a median of 43 (7.32), and the control group for premenopausal women had a median of 92.5 (9.87). The DHEAS in postmenopausal women had a median of 1102 (30136), and the control group had a median of 677 (81.31). There was a significant difference in all of the hormones levels in the postmenopausal women; however, there was a significant differences in the levels of the SHBG and DHEAS.

**Table 3.** The median level of hormones in pre and postmenopausal women participants

| | Control | | Patients | |
|---|---|---|---|---|
| **Hormones** | **Premenopausal** | **postmenopausal** | **Premenopausal** | **Postmenopausal** |
| | | Median (IQR) | | |
| SHBG(nmol/L) | 8.46)(71.1 | 13.2)(80 | 81.4(21.5)* | 77.5(21.3)* |
| Oestradio(ng/L) | 10.6)(87.4 | 13.2)(7 | 88.7(24.08) | 6.3(19.02)* |
| DHEAS(ng/mL) | 92.5 (9.87) | 43 (7.32) | 102.2 (27.9)* | 35.3 (10.46)* |
| Progesterone(ng/ml) | 677 (81.31) | | 1102 (301,36) | |

*IQR ¼ interquartile range, * there is a significant differences at P>0.05.*

The levels of Estrogen receptor alpha (ERα) were documented with means and standard deviations: patients had levels of 53.87 ± 8.6, while the control group had levels of 234 ± 45.78. The levels of Progesterone receptor (PR) were reported with means and standard deviations: patients had levels of 33.78 ± 9.8, and the control group had levels of 0.26 ± 0.05. There was a significant decrease in the level of the estrogen receptor alpha, however, there was a significant increase in the levels of the progesterone receptor, P value >0.05.

**Table 4.** Hormones receptors in women participants.

| **Steroid hormone receptor** | **Controls Mean ± SD** | **Patients Mean ± SD** |
|---|---|---|
| Estrogen receptor alpha (ERα) | 234±45.78 | 53.87 ±8.6* |
| Progesterone receptor (PR) | 0.26±0.05 | 33.78±9.8* |

** There is a significant differences at P>0.05.*

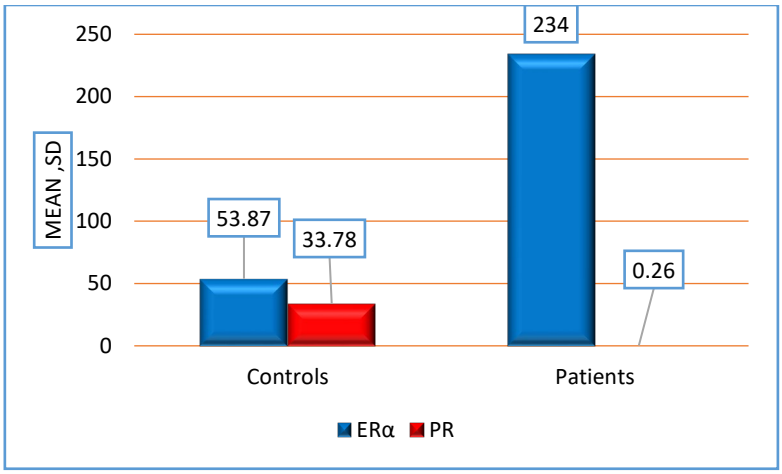


**Figure 3.** Hormones receptors in women participants.

## 4. Discussion

Estrogen (17β-estradiol, E2) and progesterone (P4) are the two primary female sex hormones, playing critical roles in regulating cervical physiology. The cervical stroma is comprising squamous epithelial layers, glandular components, dense collagen, and stromal fibroblasts, is highly responsive to hormonal changes throughout the reproductive life cycle [9, 10]. These hormones exert their effects through a range of mechanisms, including modulation of cellular proliferation, differentiation, inflammation, apoptosis, and tissue remodeling via autocrine and paracrine pathways [11,12]. Disruption in these finely tuned hormonal dynamics may contribute to abnormal cellular behavior, creating a permissive environment for carcinogenesis.

Our findings highlight several clinically relevant hormonal alterations in patients with cervical carcinoma. A striking observation was the significantly higher proportion of patients with a positive family history of cancer (73.3%) compared to the control group. This supports previous research by Muñoz et al. (2006), who emphasized the role of genetic predisposition and shared environmental exposures in increasing cervical cancer risk, particularly in familial clusters [13].

With respect to menopausal status, we observed a near-even distribution among premenopausal and postmenopausal women in both groups, consistent with a hormonally diverse cohort. Although smoking data were not fully recorded for all participants, previous work by Plummer et al. (2003) has established a robust association between smoking and increased risk of cervical intraepithelial neoplasia and invasive cervical cancer, likely due to the carcinogenic and immunosuppressive effects of tobacco compounds on cervical epithelium [14]. Moreover, hormonal changes across menopausal transitions may further influence susceptibility, as suggested by Roura et al. (2016), who reported a differential cancer risk based on reproductive and hormonal factors [15].

In our biochemical analysis, estradiol levels were significantly reduced in postmenopausal patients compared to both premenopausal patients and healthy controls, reflecting expected physiological declines. Interestingly, premenopausal patients demonstrated elevated estradiol compared to controls, suggesting a potential hyperestrogenic state in disease. This aligns with Stanczyk et al. (2006), who proposed that aberrant estrogen exposure, especially in the context of persistent HPV infection, may act as a cofactor in cervical cancer progression [16].

Notably, levels of dehydroepiandrosterone sulfate (DHEAS) were significantly elevated in patients, particularly among postmenopausal women. As an adrenal androgen and precursor to estrogen, elevated DHEAS may contribute to increased peripheral estrogen production in extra-gonadal tissues. Key et al. (2002) previously hypothesized that adrenal androgens may play a role in hormone-sensitive cancers, especially in postmenopausal women with lower ovarian estrogen output [17].

One of the most compelling findings of our study was the significant downregulation of estrogen receptor alpha (ERα) and concurrent upregulation of progesterone receptor (PR) in cervical cancer patients compared to controls. Reduced ERα levels may indicate impaired estrogen signaling or a compensatory downregulation in response to chronic stimulation, findings echoed in Cheng et al. (2016), who reported that reduced ERα expression is associated with poorer prognosis in cervical cancer [18]. On the other hand, elevated PR expression may suggest an adaptive or compensatory mechanism, or a possible role in modifying tumor behavior. This hypothesis is supported by De Vivo et al. (2002), who emphasized that hormone receptor expression levels can influence tumor progression, treatment response, and prognosis in hormone-sensitive cancers [19].

While hormonal modulation remains a promising therapeutic avenue, the literature reflects substantial heterogeneity in hormone receptor expression and function in cervical cancer. As noted in prior studies, not all cervical carcinomas express progesterone receptors, and when present, PR levels may vary with estrogen status and tumor differentiation [20, 10]. In our study, the presence of PR in both normal and cancerous cervical

tissues suggests potential biological relevance; however, we observed a trend of lower PR positivity in tumors compared to healthy tissue. This observation raises the possibility that an imbalance between estrogen and progesterone signaling, such as high estrogen, low progesterone activity, may disrupt normal gene regulation and promote invasive phenotypes. Supporting this, earlier work demonstrated that progesterone receptor signaling inversely correlates with lymph node metastasis and collagen deposition, pointing to a protective role in limiting tumor invasion and angiogenesis [21, 10].

Collectively, our findings support the hypothesis that disruptions in hormone-receptor dynamics, manifesting as elevated estradiol or DHEAS, reduced ERα, and altered PR expression, may contribute to the hormonal microenvironment favoring cervical carcinogenesis. These disruptions likely interact with HPV oncogene activity and immune dysregulation to drive malignant transformation. The observed hormone-receptor profiles suggest that cervical cancer may share mechanistic parallels with other hormone-responsive cancers, such as breast and endometrial cancer, where receptor-targeted therapies have already shown clinical utility [22].

Understanding these hormonal imbalances and their receptor-mediated effects could guide future strategies in early detection, risk stratification, and treatment planning. Hormone receptor modulators—already used in other cancers, may represent a novel adjunct in cervical cancer management, particularly in tumors exhibiting defined receptor profiles.

**Limitation**

While this study provides valuable insights into the hormonal and receptor-related alterations in cervical carcinoma, several limitations should be acknowledged. First, the sample size was relatively small, with only 60 participants, which may limit the generalizability of the findings and reduce statistical power for subgroup analyses. Future studies involving larger, multicenter cohorts are needed to validate and extend these observations. Second, this was a cross-sectional study; therefore, causal relationships between hormone levels, receptor expression, and cervical cancer development cannot be definitively established. Longitudinal studies tracking hormonal changes over time in relation to disease onset and progression would provide more robust evidence. Third, although hormone levels were measured using validated ELISA kits, we did not assess tissue-level receptor expression through immunohistochemistry (IHC), which could have provided more precise localization and quantification of ERα and PR in cervical tissues. Additionally, factors such as body mass index (BMI), nutritional status, and stress, known to influence hormonal profiles, were not controlled for in this analysis. Lastly, HPV status, a key etiological factor in cervical cancer, was not documented for all participants, which limits the ability to evaluate interactions between viral oncogenesis and hormonal dysregulation. Despite these limitations, the study offers important preliminary evidence on the endocrine milieu associated with cervical cancer in an understudied population and serves as a foundation for future research.

## 5. Conclusion

This study highlights significant hormonal and receptor-based differences between women diagnosed with cervical carcinoma and healthy controls, with particular emphasis on menopausal status. Notably, estradiol levels were markedly lower in postmenopausal patients compared to premenopausal patients, while DHEAS levels were significantly elevated, suggesting altered steroid metabolism in disease states. Additionally, both SHBG and DHEAS levels were higher in patients than in controls, underscoring systemic endocrine alterations associated with cervical cancer. Importantly, cervical cancer patients demonstrated lower expression of estrogen receptor alpha (ERα) and elevated levels of progesterone receptor (PR), indicating possible dysregulation of hormone-receptor signaling pathways. These receptor imbalances may play a contributory role in tumor development and progression. Furthermore, the high proportion of patients reporting

a positive family history supports the presence of a potential hereditary or genetic susceptibility component. Despite matched age distributions and no significant differences in menstrual status between groups, the observed biochemical and receptor-related disparities emphasize the influence of hormonal factors beyond chronological aging. These findings underscore the importance of incorporating hormonal profiling into cervical cancer research and may inform future strategies for risk assessment, early detection, and targeted hormonal intervention.

## Author contributions

AH works on Conceptualization, Funding acquisition, Methodology, Project administration, Investigation, and Validation. HS,KN and NM work on Visualization, Software, Formal analysis, Writing—original draft, Writing—review and editing.

All authors have read and agreed to the published version of the manuscript.


## Funding

Not applicable.

## Acknowledgments

The authors gratefully thank the national leading center of cancer research, Baghdad University, for providing lab facilities and instruments. The authors are also thankful to Al-Rayan company for their role in providing some of the instruments to examine the samples.


## Conflict of interest

The authors declare no conflict of interest.

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