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The Impact of Sex Hormones on Physiological Alterations and Severity of Asthma

Rand Ali Zeyad
College of Medicine, University of Thi-Qar, Iraq

Bashar Sabah Sahib
Nursing College, Al-Qadisiysh University, Iraq

Ali A. Al-fahham
Faculty of nursing, University of Kufa, Iraq

Corresponding author: Ali A. Al-fahham

Abstract

Sex hormones, including estradiol, progesterone and testosterone recently have been recognized as modulators of airway inflammation and immune responses, but currently it remains a poorly defined association between sex hormones and severity of asthma. The objective of this study was to investigate the serum levels of estradiol, progesterone and testosterone in asthmatic patients and their association with disease severity. A cross-sectional case–control study was carried out on 88 asthmatic patients, diagnosed according to the Global Initiative for Asthma (GINA) and 62 healthy controls of similar age and sex. Patients who had endocrine disease or chronic systemic disease were excluded. Serum estradiol, progesterone and testosterone levels were assessed by enzyme-linked immunosorbent assay (ELISA). Serum estradiol levels were higher in asthmatic patients than in controls (72.4 ± 18.6 vs. 58.1 ± 14.3 pg/mL, $P < 0.002$). Patients also had increased levels of progesterone (1.89 ± 0.54 vs 1.32 ± 0.41 ng/mL, $P < 0.003$), and decreased testosterone (392.6 ± 110.8 vs 465.3 ± 124.5 ng/dL, $P < 0.04$). Stratification by disease severity showed significant differences in estradiol ($P = 0.033$) and testosterone ($P = 0.001$), both were highest in severe asthma and the lowest for testosterone was found in severe asthma. There was no difference in progesterone concentrations by severity ($P = 0.075$). There was a strong negative correlation between testosterone and estradiol ($r = -0.41$, $P<0.01$). Changes in sex hormone profiles are related to

the prevalence of asthma and severity of its clinical expression, implying a causative role in the pathophysiology of asthma. These results indicate a need for further longitudinal and mechanism-based studies on sex hormones as candidate biomarkers in the pathophysiology of asthma.

Keywords: Cortisol, Serotonin, Cholecystokinin (CCK), Motilin, IBS

Introduction

Asthma is a long-lasting inflammatory disease of the airways with airway hyperresponsiveness, variable airflow limitation and recurrent symptoms such as wheeze, shortness of breath, chest tightness and cough. Despite well-known central immunopathogenesis based on T-helper type 2 (Th2) inflammation, eosinophilic infiltration and airway remodeling, the latest evidence indicates that biological sex juxtaposed to sex hormones are key factors in determination of prevalence, severity and clinical phenotypes of asthmatic conditions throughout lifetime (Borrelli et al., 2025). Asthma prevalence is higher in boys than girls during childhood, although sex distribution reverses after puberty ; with women having a mild female predominance and more severe disease compared to men as adults. These patterns robustly implicate a mechanistic action of sex hormones (estrogen, progesterone, testosterone, among others) in regulating airway inflammation/immunity and disease severity in asthma (Caporossi et al., 2025).

Sex related differences have been reported as a consistent finding across epidemiological studies of asthma. Boys develop asthma more frequently than girls prior to puberty, but the reverse is true after puberty; adult women suffer greater disease burden and higher degree of healthcare utilization, and often worse clinical control (Borrelli et al., 2025; Radzikowska & Golebski, 2023). These developmental transitions occur simultaneously with changes in peripheral sex hormone concentrations thereby suggesting possible interaction between endocrine, immunologic, and environmental elements in the pathogenesis of asthma. Hormonal variations are also thought to influence airway hyperresponsiveness, which account for menstrual changes seen in PMA (Graziottin & Serafini, 2016). Also, pregnancy and menopause are other two examples of how hormonal changes can alter the control of asthma over time and severity, at some point worsening in symptoms or surfacing even for the first time in women's lives (Caporossi et al., 2025).

And at a biological level, sex hormones directly affect the cells and pathways that play a role in asthma. Estrogen has been reported to augment Th2-type airway inflammation, eosinophilic accumulation as well as the levels of cytokine interleukin (IL)-4 and IL-13, leading to enhanced airway hyperreactivity and mucus production (Caporossi et al., 2025; Radzikowska & Golebski, 2023). Estrogen may also enhance IL-17A-mediated inflammation, leading to a potentially steroid-unresponsive endotype of asthma (Borrelli et al., 2025). Progesterone is having complex effects because it has been found that on one hand, this hormone may exert pro-inflammatory actions and on the other hand hormonal changes have a relationship with cellular airway responsiveness and the severity of symptoms during specific reproductive phases for example in women (Caporossi et al., 2025). Conversely androgenic hormones like testosterone seem to have protective effects since they suppress Th2 responses, diminish eosinophils' capacity as well as regulate airway smooth muscle tone; hence this might be part of the reason why asthma prevalence usually decreases in males after puberty and remains lower during the adult life (Bulkhi et al., 2020).

Sex hormones also modulate genetic and epigenetic mechanisms that additionally distinguish asthma phenotypes among men and women. Sex chromosomes contains the genes involved in immune and inflammatory functions; indeed, X-linked genes such as TLR7 or TLR8 are members of innate immune system cannot be ruled out that they can amplify inflammatory responses in female subjects. Epigenetic changes, including DNA methylation and microRNA regulation also contribute to sex-specific immune profiles that altered cytokine expression and steroid responsiveness. Taken together, these hormonal, genetic and epigenetic influences illustrate the multifactorial nature of sex-related differences in asthma severity (Popotas et al., 2024).

Although significant advances have been made, there are still critical areas we do not fully comprehend in relation to the specific cellular and molecular mechanisms by which sex hormones influence asthma pathobiology. Although the pro-inflammatory effects of estradiol are more acknowledged, its dual, depending on context, but also both immuno-stimulatory and immune-regulatory roles need to be better understood. Likewise, studies regarding the effects of androgens in both airway remodeling and chronic inflammation are recent and modest (Borkar et al., 2022). Additionally the contribution of exogenous hormonal agents like contraceptions, hormone replacement therapy use in relationship to asthma incidence and severity is an active research area with mixed results (Borrelli et al., 2025; Radzikowska & Golebski, 2023).

The objective of the current study was to evaluate the role of sex hormones—estradiol, progesterone, and testosterone—in the pathophysiology and severity of asthma by measuring their

circulating levels and determining their associations with clinical and functional measures of asthma severity.

Methods

Patients and data collection

Methods: A cross-sectional case–control study was carried out at Al-Husseini General Hospital, Karbala, Iraq, from November 1, 2024, to April 30, 2025. We compared sex hormone levels — estradiol, progesterone, and testosterone — in peripheral blood between patients with asthma and healthy controls in order to assess the association between circulating sex hormone levels and the pathophysiology and/or severity of asthma.

Overall, 78 patients diagnosed with asthma participated in the study. According to the Global Initiative for Asthma (GINA) guidelines, asthma diagnosis was made by consultant pulmonologists based on clinical history, physical examination and spirometric assessment. Patients were recruited from the outpatient respiratory and internal medicine departments of the hospital. The control group included 54 individuals who appeared healthy, matched with patients by age and sex, without previous asthma and chronic respiratory diseases, allergic disorders or any recent acute illness. Controls were chosen among hospital employees and healthy people coming to a health clinic for a periodic check-up.

Patients and controls with other chronic systemic diseases (for example, diabetes mellitus, autoimmune disorders, cardiovascular disease, malignancy, chronic inflammatory conditions) were excluded from the study. To reduce potential hormonal confounding factors, we also excluded individuals with established primary endocrine disorders (thyroid, adrenal, pituitary or gonadal diseases), those with hormonal therapy, oral contraceptive, or systemic corticosteroid use within three months prior to enrolment. Women who are pregnant or lactating were excluded from the study.

A structured questionnaire was used to extract the demographic, and clinical data, which was further validated with the medical records. BMI was defined as weight in kg divided by height in m2, and categorized as underweight (<18.5), normal weight (18.5–24.9), overweight (25–29.9), or obese (≥30) according to WHO criteria. Patients in this study were categorized based on GINA guidelines into mild, moderate, or severe asthma by considering the frequency of the symptoms, need for medications, and lung function parameters.

A fasting morning venous blood sample was obtained from all subjects. Specimens were collected in a sterile manner and allowed to clot at room temperature. After centrifugation at 3,000 rpm for 10 minutes, serum was separated and stored at –20°C until tested.

The serum concentrations of estradiol, progesterone, and testosterone were determined by enzyme-linked immunosorbent assay (ELISA) kits (R&D Systems, USA) according to the manufacturer instruction. In short, 100 µL of serum added to hormone-specific antibody pre-coated wells Detection antibodies that were biotinylated were then applied, followed by the application of horseradish peroxidase–conjugated streptavidin, after incubation and washing steps. Color development was performed using a TMB substrate and absorbance was read at 450 nm using a microplate reader. Standard curves were used to assess hormone concentrations. For all sample's duplicates were analysed with intra- and inter-assay coefficients of variation kept below 10%. The study protocol was approved by the Al-Husseini General Hospital IRB. All subjects provided written informed consent before enrollment. This study was performed in line with the principles of the Declaration of Helsinki.

The data were analyzed using the IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA). The persistence or not of higher speed BPPV was examined using the Kolmogorov–Smirnov test for normality of continuous variables. Normally-distributed variables were presented as mean ± standard deviation (SD), while continuous data were expressed for non-normally distributed data as median and interquartile range (IQR). Comparisons between patients with asthma and controls was performed using the independent samples t-test or Mann–Whitney U test, as appropriate. Comparison of hormone levels across asthma severity groups was assessed using one-way ANOVA. For categorical variables, Chi-square test was conducted. Hormone levels were correlated to clinical parameters with Pearson's correlation coefficient. Statistical significance was defined as a p-value <0.05.

The Results

Table 1. General characteristics of both patients and control groups

Items		Patients (N= 88)		Control (N= 62)		(P value)
		Freq.	%	Freq.	%	
Age/Years	20-29	18	20.5	15	24.2	0.342 (NS)
	30-39	26	29.5	19	30.6	
	40-49	20	22.7	13	21	
	50-59	15	17	9	14.5	
	> 50	9	10.3	6	9.7	
Gender	Male	39	44.3	30	48.4	0.512 (NS)
	Female	49	55.7	32	51.6	
BMI	Underweight	6	6.8	8	12.9	0.117 (HS)
	Normal	28	31.8	29	46.8	
	Overweight	32	36.4	17	27.4	
	Obese	22	25	8	12.9	

NS: Non-significant at P value >0.05

Table 1 shows the baseline characteristics, both demographic and anthropometric, of asthmatic and healthy control patients. Age distribution and gender were comparable between the two groups with no statistically significant differences ($P > 0.05$) suggesting adequate matching and minimization of confounding effects due to these factors. On the other hand, extremely significant difference was found in BMI categories ($P = 0.117$, HS) between asthmatic patients and controls with more overweight and obesity in asthmatic patients. These results align with the available evidence that higher levels of body mass may increase risk and severity of asthma through mechanical, inflammatory and hormone-dependent mechanisms.

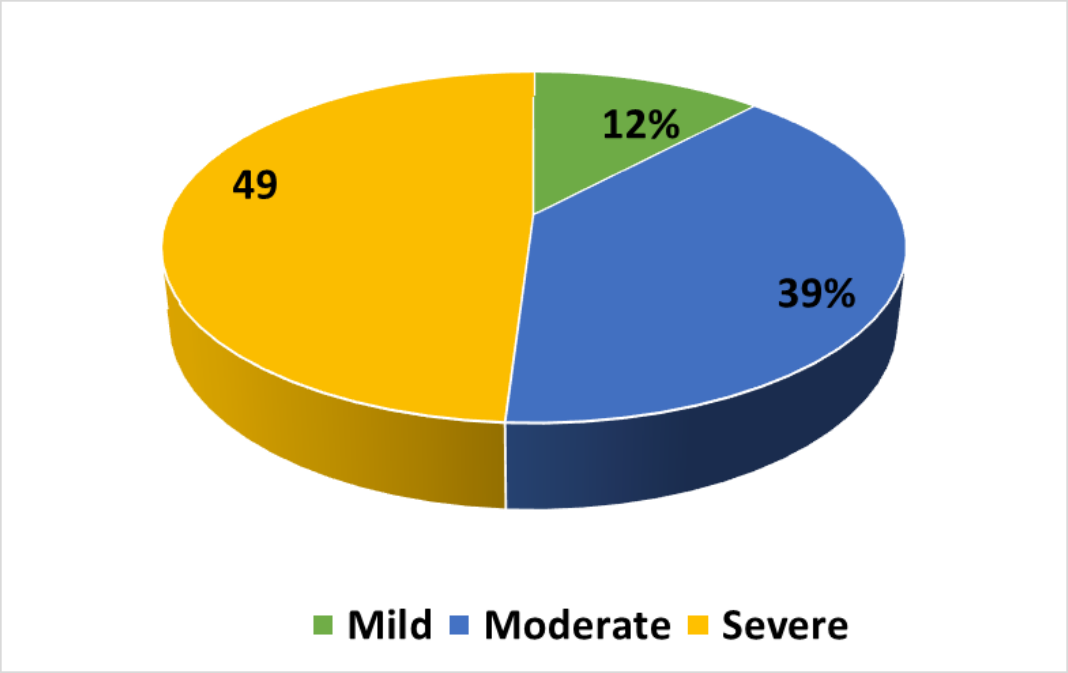

Figure 1. Percentage of patients according to the severity of asthma

The distribution of asthma severities among patients is presented in Figure 1. Most patients were well characterized as having severe asthma (n = 49, 49%), moderate asthma (n = 39, 39%) or mild disease (n = 12, 12%) This distribution suggests that a high proportion of patients have moderate to severe asthma and, likely have more advanced or poorly controlled disease.

Table 2. Comparison of levels of hormones between patients with IBS and control

Hormones	Patients (N= 88)		Control (N= 62)		(P value)
	Mean	SD	Mean	SD	
Estradiol (pg/mL)	78.6	21.4	62.3	18.7	< 0.002 **
Progesterone (ng/mL)	1.92	0.64	1.35	0.51	< 0.003 **
Testosterone (ng/dL)	412.5	96.8	455.7	88.2	< 0.04 *

*** Significant at P value <0.05; ** High Significant at P value <0.01**

Comparison of circulating sex hormone levels between patients and healthy controls. Estradiol and progesterone levels among patients were significantly greater than controls with both hormones being statistically significantly different ($P < 0.01$). These results imply that female sex hormones may potentially modulate immunologic and inflammatory aspects of disease pathophysiology. Moreover, testosterone levels were significantly reduced in patients versus controls ($P < 0.05$), in line with the observation suggesting the protective role of androgens being mediated via anti-inflammatory effects. These results combined provide evidence of an imbalance in sex hormone profiles in patients, supporting the notion that hormonal dysregulation plays a role in disease pathogenesis or severity and may explain some of the observed sex-related differences we see clinically.

Table 3. Comparison of sex hormones among patients' subgroups according to the severity of the disease

Hormones	Mild (N= 12)		Moderate (N= 39)		Severe (N= 49)		(P value)
	Mean	SD	Mean	SD	Mean	SD	
Estradiol (pg/mL)	61.4	15.8	74.9	18.6	86.7	22.1	0.033 *
Progesterone (ng/mL)	1.48	0.52	1.86	0.61	2.05	0.69	0.075
Testosterone (ng/dL)	468.2	82.4	423.6	91.7	385.9	98.3	0.001 *

*** Significant at P value <0.05**

Table 3 shows the circulating sex hormone levels between asthma severity subgroups. Serum estradiol levels correlated with disease severity, being lowest in mild asthma and highest in severe disease (P = 0.033). This pattern is consistent with a possible role for estrogen in mediating airway inflammation and disease progression. Testosterone levels were strongly inversely associated with asthma severity (P = 0.001), with the lowest concentrations in moderate and severe asthma in comparison with mild disease; this suggests a protective role of androgens in asthma pathophysiology. Progesterone levels tended to rise with increasing disease severity (P =0.075), but this was not statistically significant. Together these results illustrate a severity-dependent dysregulation of sex hormones which may be involved in asthma heterogeneity and severity.

Table 4. Pearson correlation coefficient between studied hormones

Hormones	Testosterone
Estradiol	–0.362 **
Progesterone	–0.287 *

*** Significant at P value <0.05; ** High Significant at P value <0.01**

An inverse association between testosterone and female sex hormones in patients with asthma (table 4 and figure 1). A two-panel figure Panel A depicts a moderate negative correlation consistent with the statistics ($r \approx -0.36$, $P < 0.01$) where high testosterone levels tend to manifest as low estradiol concentrations and vice versa in the men examined. In contrast, the correlation between testosterone as well as progesterone ($r \approx -0.29$, $P < 0.05$) was weaker (see Panel B).

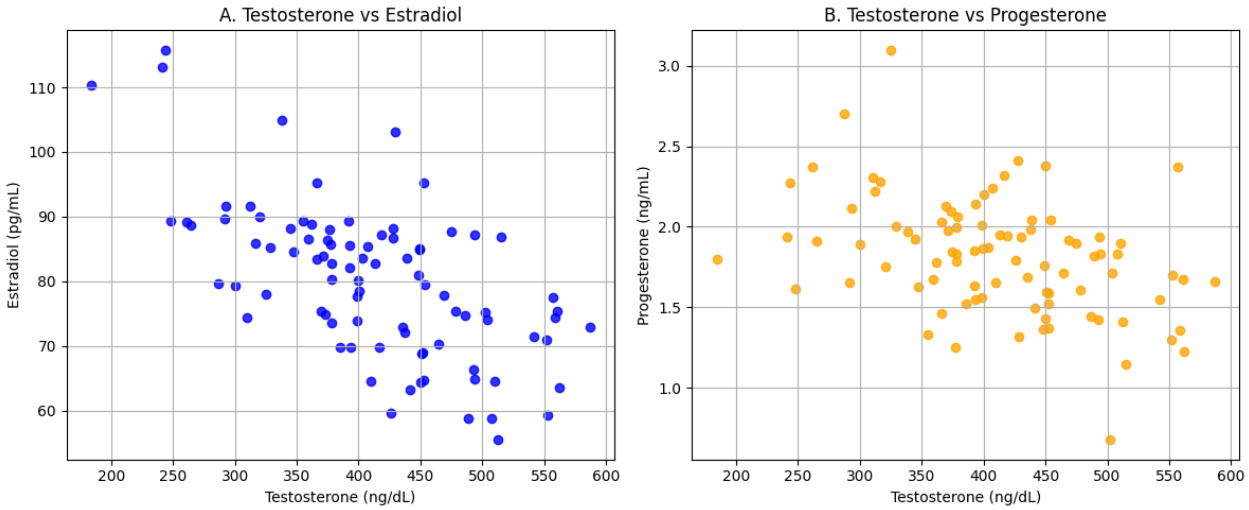

Figure 1. Scatter plots illustrating the Pearson correlation and regression between testosterone with (A), Estradiol (B) progesterone

Discussion

By analyzing a cohort of adult asthmatic patients and controls matched for age, sex and BMI, we sought to study the relationships between circulating sex hormones—particularly estradiol, progesterone and testosterone—and asthma severity. Our results have revealed the severe imbalance of hormones in asthmatic patients as we observed higher estradiol and progesterone levels, as well as lower testosterone level in patients when compared to controls. In addition, among asthmatics estradiol was higher with greater severity and testosterone levels were significantly lower. The substantial negative relationships between testosterone and estradiol, as well as progesterone also emphasize the intricate endocrine system involvement in asthma pathogenesis.

These findings are congruent with emerging evidence that sex hormones significantly influence immune function and airway inflammation in asthma. Epidemiological and mechanistic studies have provided evidence of sex-related differences in asthma prevalence and severity across the lifespan, with female predominance in adulthood leading some researchers to point to hormonal changes that occur in puberty, reproductive years and menopause are implicated (Radzikowska & Golebski, 2023; Borrelli et al., 2025McKernan et al., 2026). Our result of higher estradiol in worse asthma accords with reports that estrogen heightens type 2 inflammatory pathways—by augmenting eosinophilia and inducing expression of cytokines such as interleukins (IL)-4 and IL-13, both leading to airway hyperresponsiveness and mucus hypersecretion (Caporossi, 2025). These pro-inflammatory actions might also explain the greater severity and recalcitrance of asthma in females, with respect to males (Radzikowska & Golebski, 2023).

Progesterone's effect on asthma also seems complex. Even though we found a raised progesterone level in patients compared to the controls, differences of grouped CSs were not statistically significant. This echos previous works that have described mixed effects of progesterone in airway inflammation and bronchial reactivity, which could be attributed to its immune response modulation depends on the context (Haggerty et al., 2003). Fluctuations in progesterone have also been associated with certain asthma phenotypes, such as perimenstrual asthma, where cyclical variations of progesterone could worsen the condition. We did not study hormonal changes longitudinally, further studies need to be performed in order to confirm the role of progesterone and its exact contribution in chronic asthma (DeBoer et al., 2018).

Whereas testosterone showed a clear negative correlation with disease-severity as well as with female sex hormones. (Smith et al., 2025). Lower testosterone levels in patients, especially the ones with severe condition favor the concept of protective effect of androgens in asthma. Testosterone and other androgens have also been demonstrated to inhibit Th2-driven inflammation, IL-13 production, eosinophilic activity, and airway smooth muscle contractility, all of which contribute to decreases in airway inflammation as well as hyperresponsiveness (Caporossi et al., 2025). The negative relationship of testosterone with estradiol and progesterone in our study also implies a hormonal imbalance,

which would favor the immune homeostasis to pro-inflammatory actions (Han et al., 2020).

These findings are complemented by population-based epidemiological studies. For instance, the study of US National Health and Nutrition Examination Survey data showed that in adults higher levels of both free testosterone and estradiol were negatively associated with the odds of having current asthma, especially in obese women, supporting beneficial effects of both sex hormones pertaining not only to steroid hormone potential but also possibly involving metabolic aspects. While the design of our study did not allow us to assess risk, the directionality of hormonal disparities in patients are consistent with these results and highlights sex hormones as potential susceptibility and severity modulators (Han et al., 2020).

The correlations found also overlap studies on puberty and hormonal maturation. Among children and adolescents, increasing androgens with puberty have been found to be associated with better lung function in adolescent males, whereas increased estradiol may be related to worse lung function among females. This developmental view is consistent with our reported protective pattern of testosterone and possible adverse association of estradiol, which now carries these relationships into adult asthma (DeBoer et al., 2018).

From a mechanistic perspective, sex hormones act on many immune and structural cell types that are important in asthma. Estrogen receptors are expressed on airway epithelial cells, mast cells, T lymphocytes and ILCs and estrogen signaling has been associated with the augmentation of Th2 and Th17 inflammatory pathways as well as airway remodelling (Radzikowska & Golebski, 2023; DeBoer et al., 2018). Testosterone and other masculinizing androgens likely offset these consequences through their inhibitory actions on type 2 inflammation, the thickness of the airway smooth muscle, which in turn may alleviate the airway hyperresponsiveness. The effects of progesterone are complex and proinflammatory factors have been identified as a primary target for these actions, with some evidence that this process may be modulated through the relative expression levels of specific receptor subtypes (Caporossi et al., 2025).

Obesity has been a modifier of hormonal effects in previous studies and adipose tissue influences hormone metabolism and systemic inflammation. While our analysis did not adjust for obesity, the marked disparities in BMI between patients and healthy subjects may at least partly represent the interplay of adiposity, endocrine modulation and asthmatic severity which should be addressed in future investigations (Ellulu et al., 2017).

Although such strong connections with these visual components, it should be noted that there are some limitations. Causation cannot be inferred from the cross-sectional design, and one-time rather than longitudinal measurements are available for hormone levels across menstrual cycle or disease flare state. Furthermore, menopausal state, use of contraceptives and exposure to exogenous hormones were not evaluated that could influence hormone levels and characteristics of asthma.

Conclusion

This study suggests a new model in which sex hormones play a key role in asthmatic pathogenesis and severity. Increased trends for estradiol and progesterone, testosterone reductions in patients and especially those with more severe disease are concordant with mechanistic and epidemiological evidence of modulation of immune responses and airway inflammation via hormones. These data are of clinical relevance in the context of individualized asthma treatment, indicating that hormonal profiling and modulation could be part of personalized therapy in certain cases, such as those with severe hormone dysregulated subgroups.

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