Journal of Communicable Diseases
Volume 58, Issue 1 - 2026, Pg. No. 18-25
Peer Reviewed & Open Access Journal

Research Article

Association between Reproductive Hormones and Inflammatory Cytokines in Iraqi COVID-19 Patients

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DOI: https://doi.org/10.24321/0019.5138.202603

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How to cite this article:
Ahmed H S, Association between Reproductive Hormones and Inflammatory Cytokines in Iraqi COVID-19 Patients. J Commun Dis. 2026;58(1):18-25.

Date of Submission: 2026-01-15
Date of Acceptance: 2026-02-24

A B S T R A C T

Introduction: Due to the COVID-19 pandemic and the lack of well-defined factors to control the disease, various indicators, such as reproductive hormones and inflammatory markers, have been explored. This study investigates the relationships among serum levels of luteinising hormone (LH), follicle-stimulating hormone (FSH), testosterone, and key inflammatory cytokines. Methods: This study included 100 patients aged 25 to 40 years with COVID-19 at the Baghdad Teaching Hospital, Medical City, in Baghdad, Iraq, from October 2022 to April 2023. Reverse transcriptase polymerase chain reaction testing confirmed the infection in nasal swab samples from all eligible individuals. They were compared with 100 healthy individuals who served as the control group. Results: COVID-19 patients showed significantly elevated levels of interleukin-1, -6, -18, tumour necrosis factor-α, and C-reactive protein compared with controls. They also exhibited higher serum LH and FSH levels, and markedly lower testosterone levels. Within the patient group, males had significantly lower LH and FSH levels but higher testosterone levels than females. In the control group, females showed higher LH and FSH levels and significantly lower testosterone levels. Immunoglobulin G and M levels were significantly increased in COVID-19 patients.

Conclusion: Immunological responses to COVID-19 are closely linked to the regulation and activity of sex hormones, and sex-related variations in cytokine levels may influence vaccine-induced immunity.

Keywords: Coronavirus Disease-19, C-Reactive Protein, Cytokines, Immunoglobulins, Sex Hormones, Tumour Necrosis Factor-α

Introduction

Coronavirus disease 2019 (COVID-19), initiated by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), triggered an epidemic, the severity of which can be characterised by a cytokine storm (CS), a hyperinflammatory process.[1] Cytokines play a crucial role in immunological modulation. However, CS can result from severe inflammation, causing tissue damage, organ failure, and other serious complications that may ultimately lead to death.[2] Interleukins (ILs) and tumour necrosis factor-alpha (TNF-α) are two examples of the diverse range of immunoreactive molecules proposed to influence CS development.[3] Anti-inflammatory cytokines exacerbate the pro-/ anti-inflammatory ratio in COVID-19, as tissue

damage and immunological invasions are anticipated to result from the drop in pro-inflammatory cytokine levels.[4] In the early phases of viral infection, macrophages produce IL-18, which promotes the synthesis of IL-6 and interferon-gamma (IFN-γ), both of which can be essential for optimal host viral defence.[5] Significantly elevated blood IL-18 levels have been associated with mortality and illness severity in a few viral infections categorised by CS, such as the dengue virus and avian influenza. However, aberrant IL-18 production can also result in chronic pathology.[6]

COVID-19 incidence and mortality vary based on sex, with males experiencing poorer outcomes.[7] Factors such as behavioural determinants, immune system alterations, and sex hormones, including luteinising hormone (LH), follicle-stimulating hormone (FSH), and total testosterone, can contribute to these disparities. Sex stratification is crucial for disease progression and treatment.[8] Understanding how reproductive hormones, including LH, FSH, testosterone, and inflammatory cytokines, contribute to the onset and spread of COVID-19 can impact the planning of clinical research and surveillance programmes for disease prevention and management.[9]

SARS-CoV-2 infection induces exuberant inflammatory responses by secreting multiple cytokines, including immunoglobulin G (IgG) and immunoglobulin M (IgM). IgG makes up to 10–20% of plasma proteins, making it one of the most prevalent proteins in human serum. Among the five Ig classes in humans, it is the principal class.[10] It is produced primarily during the secondary immune response to pathogens, can activate the classical complement pathway, and provides strong protection. IgGs are monomeric antibodies (Abs) in serum and are crucial for protecting memory of immune responses and immunity over time following infection. IgG is often detected eight days after infection.[11] IgM, a pentameric Ab, has a molecular weight of 970 kDa and an average serum concentration of 1.5 mg/mL. It is mostly generated in response to infectious pathogens or antigens during the first immune response.[12] Due to the polyvalent nature of IgMs, they may exhibit higher avidity for antigen (Ag) than bivalents IgGs. In addition to neutralising pathogens, IgM Abs are highly effective at activating complement to lyse target cells and pathogens. It is detectable after three to six days of the infection.[13] In COVID-19, the first line of immune defence is represented by IgM Ab, which can be detected after the onset of symptoms. It peaks within two to three weeks, then declines, while the IgG Ab continues to increase and remains elevated beyond seven weeks.[14] Hence, this study aims to investigate the correlation between certain inflammatory cytokines and the levels of these reproductive hormones in Iraqi COVID-19 patients.

Materials and Methods

Study Design and Participants

This case–control study included 100 patients diagnosed with COVID-19 (55 males and 45 females), aged 25–40 years, who were admitted to the Baghdad Teaching Hospital and Medical City in Baghdad, Iraq, between October 2022 and April 2023. They were examined and diagnosed by an endocrinologist. Eligible individuals had a positive SARS-CoV-2 reverse transcription polymerase chain reaction (RT-PCR) nasal swab result. They were compared with 100 healthy individuals (50 men and 50 women) who served as the control group.

Inclusion and Exclusion Criteria

Patients with COVID-19 aged 25–40 years, with varying disease onset, were included in this study. Seronegative patients and controls were vaccinated. Patients with diabetes, a previous history of kidney or liver disease, thyroid disorders, autoimmune diseases, pregnant women, and patients with malignant tumours were excluded from the study. The study's flowchart is shown in Figure 1.

Anthropometric and clinical traits were assessed for all participants. The questionnaire included data on age, height, weight, blood pressure (BP), disease onset, family history, smoking, and current medications. The Institutional Scientific Committee at Al-Karkh University for Science approved this study in accordance with the Declaration of Helsinki for human studies and the instructions of the Iraqi Ministry of Health and Environment (No. 19, on 15/1/2023).

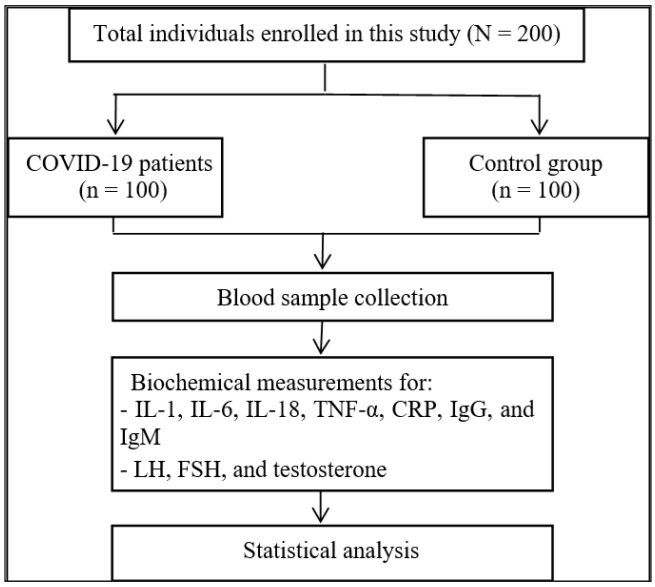

Figure 1. Flowchart of the Study

Sex, age, systolic and diastolic blood pressure (SBP and DBP) were measured for each person. A person's body

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202603

mass index (BMI) was determined by dividing their weight (kg) by their height squared (m^2). After collecting 5 mL of blood from each patient and control, the samples were transferred into clean tubes, left at room temperature for 15 min to clot, and centrifuged for 15 min at 1000 × g. Before testing, the patient's serum was isolated and stored at -20°C. Serum IL-1 (Ref. E-EL-H0052), IL-6 (Ref. E-EL-H6140), IL-18 (Ref. E-EL-H0233), and TNF-α (Ref. E-EL-H0102) levels were measured using sandwich ELISA kits from Elabscience Biotech (USA). Sex hormone levels, including LH (Ref. 30406), FSH (Ref. 30407), total testosterone (Ref. 414320), in addition to C-reactive protein (CRP) (Ref. 6K26-30), were assessed using an autoanalyser (MiniVidas, Biomerieux, France). Serological tests were used to evaluate the presence of IgG and IgM in serum by ELISA using Biorex diagnostic kits (UK; Ref. BXE0689A and Ref. BXE0690A, respectively). Every process was carried out in accordance with the manufacturer's instructions.

Statistical Analysis

Data were statistically analysed using the Statistical Package for the Social Sciences (SPSS) v26. The means of the two independent groups were compared using a t-test. P-values less than 0.05 were deemed statistically significant, and the results were displayed as means ± standard deviation (SD). Pearson correlation coefficients (r) between cytokine and hormone levels were calculated for the patient group.

Results

Among the 100 patients; 65% had fever, 35% had dyspnoea and cough, 21% had diarrhoea, 28% had chest pain, 35% had acute respiratory distress syndrome (ARDS), 28% had fatigue symptoms, and 30% had nausea and vomiting, as shown in Table 1. Table 2 shows that the values of anthropometric and clinical features in patients were considerably higher ($p < 0.05$) than in the healthy group, except for age, which did not differ significantly between the two groups.

COVID-19 patients exhibited significantly higher levels of IL-1, IL-6, IL-18, and TNF-α than controls ($p = 0.0001$). Additionally, compared to controls, COVID-19 patients had significantly higher serum CRP levels, as shown in Table 3.

Serum LH and FSH levels were considerably increased ($p = 0.04$) in COVID-19 patients compared to controls. Testosterone levels were significantly lower ($p = 0.0001$) in patients than in controls, as illustrated in Table 4. In the patient group, LH and FSH levels were substantially lower in males than in females ($p = 0.03$). In contrast, males in the patient group had higher testosterone levels than females. Females in the control group had high levels of LH ($p = 0.07$) and FSH ($p = 0.04$), and lower levels of testosterone ($p = 0.0001$), as shown in Table 5. Additionally, Figure 2 represents IgG and IgM levels in patients and controls. Patients had IgG levels of 11.54 ± 2.90 vs 1.11 ± 1.16 IU/L in controls and IgM levels of 1.95 ± 0.41 vs 0.038 ± 0.57 IU/L in controls ($p = 0.0001$).

Furthermore, the current findings indicated a strong positive correlation between LH and FSH levels and other parameters, comprising IL-1, IL-6, IL-18, TNF-α, and CRP. Table 6 reveals strong negative correlations between testosterone levels and IL-1, IL-6, IL-18, TNF-α, CRP, IgG, and IgM.

Table 1.Signs and Symptoms of COVID-19 in the Participants

Symptoms	n (%)
Fever	65 (65)
Dyspnoea	35 (35)
Cough	35 (35)
Diarrhoea	21 (21)
Chest pain	28 (28)
ARDS	35 (35)
Fatigue	28 (28)
Nausea and vomiting	30 (30)

Table 2.Anthropometric, Clinical, and Demographic Features of the Study Groups

Parameters	Mean ± SD		p Value
	Patients	Control	
Number	100	100	-
Male/female number	55/45	50/50	-
Age (years)	33.45 ± 7.31	30.42 ± 5.30	0.0600

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202603

BMI (kg/m^2)	32.52 ± 2.16	25.85 ± 1.22	0.0010
SBP (mmHg)	155.30 ± 3.18	120.41 ± 0.32	0.0001
DBP (mmHg)	90.32 ± 1.96	80.02 ± 1.15	0.0010

p < 0.05: Significant, BMI: Body mass index;DBP:Dystolic blood prusser; SBP:Systolic blood

Table 3.Cytokine and CRP Levels in the Study Groups

Parameters	Mean ± SD		p Value
	Patients (n = 100)	Control (n = 100)	
IL-1 (ρg/mL)	28.50 ± 5.72	1.76 ± 2.81	0.0001
IL-6 (ρg/mL)	45.38 ± 6.21	5.23 ± 0.62	0.0001
IL-18 (ρg/mL)	298.25 ± 12.63	50.10 ± 8.44	0.0001
TNF-α (ρg/mL)	75.85 ± 10.14	0.63 ± 1.30	0.0001
CRP (mg/L)	15.30 ± 125.52	18. 0±1.32	0.0001

p < 0.05: Significant; IL: Interlukins; CRP:C-reactive protein; TNF- α: Tumor necrosis factor

Table 4.Hormone Levels in the Study Groups

Parameters	Mean ± SD		p Value
	Patients (n = 100)	Control (n = 100)	
LH (IU/L)	8.64 ± 3.52	3.82 ± 0.31	0.0400
FSH (IU/L)	9.78 ± 1.23	4.28 ± 0.25	0.0400
Testosterone (ng/dL)	1.92 ± 1.12	122.60 ± 14.10	0.0001

p < 0.05: Significant; LH: Lutenizing hormone; FSH:Folical stimulating hormone

Table 5.Sex-Wise Comparison of Hormone Levels in the Study Groups

Parameters	Mean ± SD			
	Patients		Control	
	Male (n = 45)	Female (n = 35)	Male (n = 50)	Female (n = 50)
LH (IU/L)	6.64 ± 0.20	0.15 ± 27.10	3.12 ± 0.05	4.25 ± 0.72
	p = 0.0400		p = 0.0700	
FSH (IU/L)	6.83 ± 0.21	35.0 ± 12.42	2.53 ± 0.02	6.62 ± 0.23
	p = 0.0300		p = 0.0400	
Testosterone (ng/dL)	2.54 ± 1.30	1.28 ± 0.64	180.10 ± 15.93	65.10 ± 12.27
	p = 0.0600		p = 0.0001	

p < 0.05: Significant

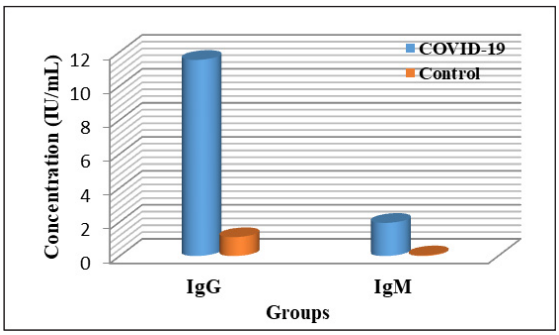

Figure 1. Serum IgG and IgM Levels in the Study Groups

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202603

Table 6.Correlation Coefficient between Hormone Levels and Other Parameters in COVID-19 Patients

Parameters	LH (IU/L)		FSH (IU/L)		Testosterone (ng/dL)	
	r	p	r	p	r	p
IL-1 (ρg/mL)	0.540	0.001	0.350	0.035	-0.462	0.003
IL-6 (ρg/mL)	0.145	0.040	0.138	0.060	-0.385	0.004
IL-18 (ρg/mL)	0.358	0.005	0.410	0.004	-0.602	0.001
TNF-α (ρg/mL)	0.650	0.001	0.618	0.010	-0.584	0.001
CRP (mg/L)	0.752	0.001	0.486	0.001	-0.490	0.001
IgG (IU/mL)	0.820	0.001	0.780	0.001	-0.652	0.001
IgM (IU/mL)	0.760	0.001	0.692	0.001	-0.575	0.001

$p < 0.05$: Significant; IgG: ImmunoglobulinG; IgM: ImmunoglobulinM

Discussion

SARS-CoV-2 causes COVID-19, a highly contagious disease that has affected more than 200 countries.[15] Its severity and prognosis are strongly correlated with elevated cytokine levels, leading to a dysregulated response and pro-inflammatory state. In the present investigation, elevated levels of pro-inflammatory cytokines, including IL-1, IL-6, IL-18, and TNF-α, were observed during the acute phase of SARS-CoV-2 infection compared with healthy controls.[16] These cytokine concentrations typically reach their peak during the acute stage of the disease and subsequently decline gradually during recovery.[17] Moreover, increased cytokine levels have been strongly associated with disease severity and adverse clinical outcomes in COVID-19 patients.

Cytokines, including IL-6, TNF-α, interferons, and monocyte chemoattractant protein-1 are produced either locally or systemically during viral infection due to inflammation caused by the virus. Testicular cells are harmed by these cytokines; IL-6, for instance, prevents Leydig cell differentiation. Furthermore, it has been demonstrated that IFN-γ inhibits testosterone production by suppressing the expression of the rate-limiting enzyme, steroidogenic acute regulatory protein (StAR). The hypothalamohypophyseal axis may be affected by pain and emotional, physical, or psychological strains caused by infections.[18] Furthermore, it has been claimed that COVID-19 affects the central nervous system, leading to an increase in the release of antidiuretic hormone. It has been proposed that the two kinds of IL-1, α and β, are implicated at distinct phases of chronic illness. Changes in the rhythm of LH secretion and anomalies in the hypothalamic-pituitary axis could therefore be contributory causes. The two isoforms of IL-1, namely IL-1α and IL-1β, have been implicated in different stages of chronic inflammatory diseases. IL-1α, which is primarily produced by endothelial and epithelial cells, functions as an alarmin and can activate the NOD-like receptor pyrin domain-containing protein 3 (NLRP3) inflammasome.[19]

As shown in severe COVID-19, excessive inflammasome activation leads to a significant release of IL-1β and consequent systemic hyperinflammation, which exacerbates lung damage and may also cause endotheliopathy and coagulopathy, ultimately resulting in extensive organ damage. Higher levels of IL-6 and IL-18 beyond the typical ranges (IL-6: 0–43.5 ρg/mL; IL-18: 37–215 ρg/mL) were observed in the patient group, as documented in previous studies.[20] It has been suggested that TNF-α, an inducer cytokine produced by immune cells in response to inflammation, increases IL-6 production. This suggests that IL-6 is a downstream effector of TNF-α. However, by promoting follicular helper T cell development, inhibiting the antiviral helper T cell 1 (Th1) axis, and promoting Th2 cell differentiation via IL-4 and IFN-γ, the immune response can be enhanced. IL-6 has also been found to raise the production of both TNF-α and IL-8.[21]

The pro-inflammatory cytokine IL-18, a member of the IL-1 family, has been linked to several inflammatory diseases and lung infections. It is commonly known that mitochondrial dysfunction controls IL-18-dependent inflammasome signalling. By targeting damaged mitochondria for lysosomal breakdown, mitophagy, a selective autophagic process, maintains mitochondrial homeostasis and quality control.[22]

The increased production of pro-inflammatory cytokines in patients with COVID-19 is consistent with the elevated TNF-α levels seen in the current investigation.[23]

Several pro-inflammatory cytokines, comprising TNF-α, IL-1, and IL-6, play a crucial role in the early immune response to COVID-19 infection.[23] The current findings show that patients with severe COVID-19 pneumonia had significantly reduced testosterone levels. Therefore, based on pathophysiological considerations, all cells expressing angiotensin-converting enzyme 2 (ACE2) receptors are susceptible to COVID-19 infection. It has previously been shown that the infection can cause complications beyond the respiratory system[24], as ACE2 is expressed in the urinary, gastrointestinal, cardiovascular, and respiratory systems.[25,26] A previous study found a difference of 130.20

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202603

ng/dL in the testosterone levels between the patient and the control group.[27] The current findings revealed that COVID-19 patients had higher LH and FSH levels than individuals without COVID-19. COVID-19 damages sperm characteristics, consistent with a previous study.[28] Thus, the study contributes to the clinical evidence demonstrating the effect of COVID-19 on reproductive health. This study, however, could not confirm that female hormones were protective against SARS-CoV-2 infection. Contrary to the present study's findings, a recent study reported lower LH and FSH levels in COVID-19 patients than in the control group.[29]

In this study, all males, regardless of age, had noticeably reduced testosterone levels, which may contribute to a higher risk of adverse outcomes.[30] A comprehensive analysis of sex hormone levels revealed that the total testosterone levels dramatically dropped in severe COVID-19 cases, and individuals with lower testosterone levels experienced more severe illness and higher mortality, which is consistent with the previous findings.[31] A prior study revealed that individuals with severe COVID-19 had reduced testosterone levels compared to those with mild or moderate disease, which is consistent with the current findings.[32]

Furthermore, treatments, such as corticosteroids, anticoagulants, and antiviral medications, including remdesivir, administered to hospitalised patients may have altered the sex hormone gene expression.[33] In addition, women seem to have stronger immune systems, reduced cytokine-dependent inflammatory responses, and lower levels of ACE2, which is critical for SARS-CoV-2 cell entry.[34,35] Oestrogen appears to be a significant protective factor in this situation. As people mature, their oestrogen levels change: they rise during the prepubescent stage and fall during the postpubescent stage.[36] Therefore, the increased vulnerability and severe development of COVID-19 in older people may be explained by an age-related decrease in oestradiol levels.[37] The significant male disadvantage in COVID-19-related mortality, especially before vaccination, likely reflects men's relatively weaker immune systems and increased susceptibility to new infections, consistent with their heightened susceptibility during prior health emergencies.[38]

COVID-19 patients showed significantly elevated serum IgG and IgM levels compared to controls, reflecting the humoral immune response, which can be triggered in two ways: first, after exposure to the infecting microorganism; and second, through vaccination. It has been observed in the development of neutralizing antibodies (NAbs) in response to vaccination and COVID-19 infection.[39] Vaccination dramatically reduces mortality from COVID-19 infection, particularly among individuals with comorbidities, even though both conditions elicit protective antibodies against the illness. Reduced illness severity and transmission rates may also be associated with lower mortality. This study has limitations, including a small sample size, a single-gender analysis, and insufficient quantification of variables affecting the hypothalamic–pituitary–gonadal axis. The study's retrospective design, along with the potential influence of questionnaire-induced symptom reporting, may affect the accuracy of patients' perceptions.

Conclusion

Males and females may experience COVID-19 differently. In addition to influencing immune responses to COVID-19, sex chromosome biology is also associated with other important factors, including pre-existing chronic diseases and physiological hormonal fluctuations throughout the lifespan. Differences in the sex genome may alter immune responses to COVID-19 throughout the lifetime and could also affect how individuals with COVID-19 respond to medications and vaccines. Sex-specific COVID-19 treatment approaches may improve patient outcomes. The sex-linked differences in disease manifestation are also reflected in this.

Ethical Approval

Before being included in the study, each subject gave their informed consent. Every technique was carried out in compliance with the Declaration of Helsinki and the institutional research committee's ethical guidelines.

Authors' Contribution:

The study's conceptualization and design, data collection, analysis, and interpretation of the findings, and paper writing fell under the purview of the author.

Declaration of Generative AI and AI Assisted Technologies in the Writing Process: This manuscript was written, analyzed, and prepared without the assistance of generative AI or AI-assisted technology.

Source of Funding: None.

Conflict of Interest:

The author states that there are no conflicts of interest with relation to this manuscript's publication.

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ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202603