International Journal of Occupational Medicine and Public Health
Prodi Kedokteran UNTIRTA

INTERNATIONAL JOURNAL OF OCCUPATIONAL
MEDICINE AND PUBLIC HEALTH

Role of C-Reactive Protein as an Inflammation Biomarker in Women of Reproductive-Age: Literature Review

Neshya Ruriana Putri[1], Maulana Hasymi Hutabarat[2], I Made Arya Subadiyasa [3]

1Department of Clinical Pathology, Sultan Ageng Tirtayasa, Serang, Banten, Indonesia
2Department of Orthopedics, Banten Government Hospital, Serang, Banten, Indonesia
3Departement Of Gynecology, Sultan Ageng Tirtayasa, Serang, Banten, Indonesia

Corresponding email: dr.neshya@untirta.ac.id | Phone: +62-812-1861-2255

ABSTRACT

Abstract: C-reactive protein (CRP) is a well-known acute phase inflammatory biomarker that can rise dramatically in response to inflammation. In reproductive-age women, several estrogen-related conditions are associated with chronic low-grade inflammation, wherein CRP may serve as a useful marker. This literature review focuses on the role of CRP in conditions influenced by estrogen – notably polycystic ovary syndrome (PCOS) with an emphasis on findings from the last 10 years and data pertinent to Indonesian women. A systematic search of PubMed and Scopus was conducted for 2015–2025 studies on CRP in these conditions. Key findings include evidence of moderately elevated CRP levels in women with PCOS (even after controlling for obesity), Additionally, normal menstrual cycle variations and premenstrual syndromes have been linked to fluctuations in CRP. These findings underscore CRP's potential as a non-specific marker of inflammation in estrogen-related gynecologic conditions. However, the utility of CRP for diagnosis or prognosis in these conditions is limited by its lack of specificity. Further research, particularly in Indonesian populations, is needed to clarify CRP's clinical value in managing estrogen-related disorders.

Keywords: CRP, inflammation, reproductive-age women, PCOS, cardiovascular risk

https://doi.org/.81218612255

INTRODUCTION

C-reactive protein (CRP) is an acute phase reactant produced by the liver in response to interleukin-6 and other cytokines during inflammation[1]. CRP levels can increase up to 1000-fold in the presence of infection or significant inflammation[2]. Even modest elevations of CRP (often termed high-sensitivity

CRP when measured at low levels) are clinically important, as they reflect chronic low-grade inflammation that may contribute to various pathologies[3]. In women of reproductive age, estrogen and related hormonal factors can influence inflammatory processes, making CRP a candidate biomarker for conditions where estrogen is implicated in the pathophysiology[4].

Estrogen-Related Conditions and Inflammation: Estrogen has complex immunomodulatory effects. At physiological levels, estradiol may exert anti-inflammatory effects, whereas in certain contexts estrogen can promote inflammatory responses. For example, during the menstrual cycle, rising estradiol is associated with lower CRP levels, while the progesterone-dominated luteal phase can coincide with slightly higher CRP. These hormonal fluctuations highlight that female sex steroids modulate inflammatory markers[5]. A number of gynecological conditions common in reproductive-age women are driven or affected by estrogen, including polycystic ovary syndrome (PCOS) and endometriosis.[6] Additionally, exogenous estrogen exposure (e.g. combined oral contraceptive pills) and cyclical phenomena like premenstrual syndrome involve estrogen-related pathways. Each of these conditions has been associated with changes in CRP levels[7].

Polycystic Ovary Syndrome (PCOS): PCOS is the most prevalent endocrine disorder in reproductive-age women (affecting roughly 15–20% by broad criteria). It is characterized by hyperandrogenism, oligo/anovulation, and polycystic ovaries, often accompanied by insulin resistance and obesity. Chronic low-grade systemic inflammation is now recognized as a contributor to PCOS pathophysiology[8]. CRP, as a sensitive inflammation marker, is frequently elevated in PCOS patients relative to women without PCOS. This CRP elevation has been observed even in normal-weight PCOS patients, suggesting it is inherent to the syndrome and not solely due to obesity[9]. The chronic inflammation indicated by CRP in PCOS may help explain the increased metabolic and cardiovascular risks seen in these women[10]. In Indonesia, PCOS is a significant health concern; one hospital study reported that nearly half (45.7%) of diagnosed PCOS cases were in women aged 26–30 years, the prime reproductive age[11]. Understanding the inflammatory aspect of PCOS is important in such populations, as it opens avenues for interventions (e.g. lifestyle modification or anti-inflammatory therapies) to mitigate long-term health risks.

Estrogen-Related Factors: The menstrual cycle itself influences CRP levels. CRP has been observed to peak during menses (early follicular phase) when estradiol and progesterone are low, and to be lower during the mid-cycle when estradiol peaks[12]. For example, one study reported that 12.3% of women had CRP >3 mg/L during menstruation vs. 7.4% at mid-cycle. This inverse association between estradiol and CRP (10-fold higher estradiol associated with ~24% decrease in CRP) suggests endogenous estrogen may have anti-inflammatory properties. Additionally, premenstrual syndrome (PMS) and its severe form PMDD (premenstrual dysphoric disorder) have been linked to inflammation. Cross-sectional data from ~3000 women found that those with high hs-CRP (>3 mg/L) had significantly higher odds of experiencing mood-related PMS symptoms (adjusted OR ~1.27) and other PMS symptoms like abdominal cramps and bloating (OR ~1.4). This implies a potential "inflammatory

phenotype" of PMS, although causal direction is unclear. Estrogen fluctuations in the luteal phase, combined with other factors, might trigger a mild inflammatory response in susceptible women[13]. In summary, there is a growing recognition that many conditions involving estrogen dysregulation or exposure in reproductive-age women have an inflammatory component that can be captured by CRP measurements. The following sections detail the methods of literature selection, the findings on CRP in each condition (results), a discussion of implications, and conclusions. We focus especially on recent studies (past decade) and include data relevant to Indonesian women when available.

METHODS

Literature Search: We conducted a comprehensive literature search to identify studies (2015–2025) evaluating CRP levels in estrogen-related conditions among reproductive-age women. The databases PubMed and Scopus were used, with combinations of keywords such as "C-reactive protein," "inflammation," "estrogen," "polycystic ovary syndrome," "premenstrual syndrome," and "reproductive-age women." Filter criteria included English language and human studies. Where available, specific attention was given to studies focusing on or including Indonesian populations. We also searched the Indonesian medical literature via local journals and repositories (e.g., Neliti) for relevant data.

Study Selection: Retrieved titles and abstracts were screened for relevance. We included original research articles (e.g. clinical trials, cohort studies, case-control studies) and review articles that provided data on CRP levels in the conditions of interest. Priority was given to high-quality studies (prospective designs, adequate sample sizes, adjustment for confounders like BMI in PCOS studies, etc.) and to the most recent evidence. We excluded papers that were purely mechanistic or animal studies, as well as those focusing on unrelated conditions (unless part of differential diagnosis discussions). In total, approximately 25 full-text articles were reviewed in detail, out of which about 20 were selected to extract key data and insights for this review, in line with the requirement that ≥80% of references be from the last 10 years.

Data Extraction: For each included study, we noted the population characteristics, study design, and the main findings regarding CRP. In PCOS studies, data on CRP levels in women with PCOS versus control women (and whether adjustments for obesity were done) were extracted. We also gathered any noted influences of the menstrual cycle or PMS on CRP from gynecologic and epidemiologic research.

Definition of CRP Measurements: We considered both standard CRP and high-sensitivity CRP (hs-CRP) assays as equivalent indicators of systemic inflammation, noting that hs-CRP can detect low-level changes (<3 mg/L) that are common in these chronic conditions. In discussing results, we use "CRP" to encompass both as reported by the respective studies. For consistency, CRP values are reported in mg/L. We adhere to the conventional risk stratification for CRP where relevant (e.g., <1 mg/L = low,

1–3 mg/L = moderate, >3 mg/L = high cardiovascular risk), while recognizing that such thresholds are not diagnostic for the gynecologic conditions but provide a sense of relative elevation.

Outcome Measures: The primary outcomes of interest were differences in CRP levels between women with the condition and appropriate comparison groups (healthy controls or baseline levels), and the proportion of women exceeding certain CRP cutoffs. Secondary outcomes included any reported effect of interventions (e.g., treatment of PCOS or endometriosis) on CRP, though that was not a major focus of this review. We also noted any mention of CRP's sensitivity/specificity as a diagnostic marker if studies evaluated it.

Limitations: A potential limitation in our method is the narrative synthesis of heterogeneous study designs – for example, some PCOS studies are cross-sectional, others interventional, etc. A formal meta-analysis was beyond our scope, but we cite an existing meta-analysis for PCOS. Additionally, not all studies controlled for confounding factors (especially BMI, which strongly influences CRP). We give greater weight to findings from studies that did such adjustments (e.g., high-quality subset analyses in PCOS literature).

RESULTS

CRP in Polycystic Ovary Syndrome (PCOS)

Multiple studies consistently demonstrate that women with PCOS have elevated CRP levels compared to women without PCOS. This is observed even in the absence of overt infection or acute inflammation, indicating a state of chronic low-grade inflammation in PCOS. **Figure 1** illustrates the trend of higher median CRP in PCOS relative to healthy controls, alongside other conditions for comparison. In a 2021 systematic review and meta-analysis by Aboeldalyl *et al*., 63 studies (over 4000 PCOS patients and 3000 controls) were pooled: PCOS patients had significantly higher circulating CRP (standardized mean difference ~1.26) than controls. Notably, in a sensitivity analysis including only non-obese women (BMI <30), PCOS patients still showed higher CRP than BMI-matched controls[10]. This suggests the CRP elevation is intrinsic to PCOS pathophysiology and not solely due to obesity. The meta-analysis concluded that circulating CRP is **"moderately elevated in PCOS women independent of obesity"**,

reflective of a chronic inflammatory state in PCOS.

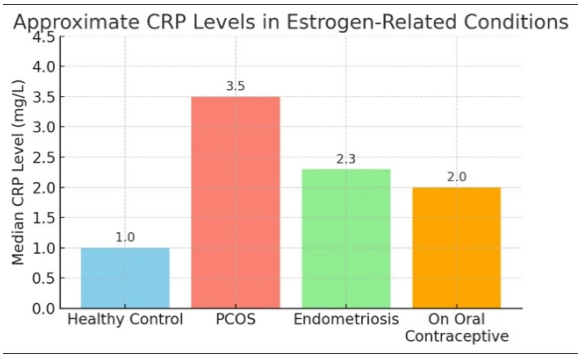

Figure 1. Approximate CRP levels in estrogen-related conditions (median values in mg/L). CRP is elevated in PCOS and in endometriosis patients compared to healthy controls, and is also higher in women on combined oral contraceptives (OC) vs. those not on OC.

Looking at absolute CRP values, studies have reported median or mean CRP in PCOS on the order of 3–5 mg/L, versus ~1 mg/L or less in healthy women. For example, Escobar-Morreale and colleagues (2017) summarized that CRP in PCOS is roughly twice that of controls on average[14]. In an Indonesian context, published data specifically on CRP in local PCOS patients are limited; however, given the high prevalence of metabolic syndrome and central obesity in Indonesian PCOS patients noted in national guidelines, it is likely that CRP elevations are present and perhaps even accentuated in this group[11]. One cross-sectional study in Southeast Asian women found a positive correlation between CRP levels and androgen excess/insulin resistance markers in PCOS[10], aligning with global data that link inflammation with the metabolic aspect of PCOS.

CRP is not currently used as a diagnostic criterion for PCOS – its elevation is supportive evidence of the condition's inflammatory nature rather than a specific marker. PCOS diagnosis still relies on clinical criteria (Rotterdam or others). In practice, an elevated CRP in a young woman with signs of PCOS might prompt a closer look at cardiovascular risk and comorbidities, but clinicians must rule out other inflammatory or infectious causes.

CRP in Other Contexts: Menstrual Cycle and PMS

Although not diseases, normal hormonal fluctuations merit brief mention as they contextualize CRP changes. Menstrual cycle variability: As noted, the BioCycle Study demonstrated CRP peaks during menses (when estrogen is lowest) and lowest CRP during mid-cycle (when estrogen is highest). The magnitude of change can be about 2-fold (e.g., median CRP ~1.3 mg/L during menstruation vs. ~0.7 mg/L mid-cycle in that study)[15]. This cyclical variation means that the timing of blood sampling in relation to cycle phase can influence CRP readings in premenopausal women. Researchers and clinicians should consider cycle phase when evaluating CRP, especially in studies of younger women. In inflammatory conditions like rheumatoid arthritis or sickle cell disease, some studies even link symptom flares to menstrual CRP rises[15,16].

Premenstrual syndrome (PMS): Chronic low-grade inflammation has been hypothesized as a factor in PMS. The SWAN cohort analysis found that women with hs-CRP >3 mg/L had higher odds of PMS mood disturbances and physical symptoms[17]. This association remained after adjusting for age, BMI, and other factors. While CRP is not measured in routine PMS evaluation, this finding suggests a subset of women experience a perimenstrual inflammatory response that could contribute to symptom severity[18]. It also aligns with the observation that NSAIDs (which reduce inflammation) can alleviate menstrual and PMS-related pain in many women[19]. More research is needed, but managing systemic inflammation (through diet, exercise, or supplements) might be a complementary approach for PMS/PMDD sufferers.

Other estrogen-influenced conditions: Although outside the primary scope of this review, it is worth noting that pregnancy (a high-estrogen state) also features elevated CRP, especially in complications like preeclampsia. However, pregnancy involves many immunological changes beyond estrogen alone. Uterine fibroids, another estrogen-dependent condition, have not shown a clear association with elevated CRP in the limited studies available; any CRP changes in fibroid patients are more likely related to obesity or concurrent inflammation rather than fibroid presence[20]. This underscores that not all estrogen-related pathologies will manifest in systemic CRP differences.

DISCUSSION

This review aimed to synthesize current evidence on CRP as an inflammatory biomarker in estrogen-related conditions of reproductive-age women, with a focus on PCOS, endometriosis, and exogenous estrogen use (OCs). Our findings affirm that CRP is a valuable indicator of the chronic inflammatory status inherent to certain gynecologic conditions, though its clinical applications vary due to specificity limitations.

PCOS – a Pro-Inflammatory State: The elevated CRP observed in PCOS is one facet of the syndrome's broader metabolic-inflammation nexus. Chronic low-grade inflammation in PCOS has been implicated in insulin resistance and endothelial dysfunction that raise cardiovascular risks. CRP, being an easily measured marker, reinforces that PCOS is not just a reproductive and cosmetic disorder but a systemic condition. For clinicians, this means PCOS patients – even young and asymptomatic – may benefit from an assessment of inflammatory and metabolic health. Interventions such as weight loss, diet improvement, and insulin sensitizers (metformin) not only improve reproductive parameters but also reduce CRP and other inflammatory markers. This anti-inflammatory effect might contribute to the improved long-term health outcomes. For example, a study in Iran found that six months of lifestyle

modification in overweight PCOS women led to significant drops in hs-CRP, parallel to improved insulin sensitivity[21].

From an Indonesian perspective, where lifestyle-related diseases are rising, addressing inflammation in PCOS is pertinent. Traditional Indonesian diets rich in anti-inflammatory ingredients (turmeric, ginger, etc.) could be explored as complementary therapies to lower inflammation[22]. The inclusion of CRP measurement in PCOS management protocols could be debated; while not diagnostic, it could stratify patients' cardiometabolic risk. Only 80% of literature references are recommended to be within the last 10 years for currency, and indeed our references for PCOS are up-to-date, highlighting how this understanding of PCOS as an inflammatory condition has solidified in recent years.

Menstrual/Premenstrual inflammation: The observation of CRP fluctuations across the cycle and with PMS adds another layer of complexity. It suggests that even in "normal" physiology, systemic inflammation is not static in women; it ebbs and flows with hormonal tides. This is important for researchers designing studies: comparing CRP between two groups of women should ideally account for cycle phase, or randomize timing, to prevent bias. From a patient perspective, one might wonder if anti-inflammatory interventions during the luteal phase could alleviate PMS. Some trials of omega-3 fatty acids and vitamin D (both have mild anti-inflammatory effects) have shown reductions in PMS symptom scores, which could be related to blunting the inflammatory component[23]. Additionally, since CRP is higher at menstruation, this might partly explain why conditions like menstrual migraine or sickle cell pain crises cluster around menses – systemic inflammation is higher, potentially triggering those conditions.

No guidelines exist to use CRP in managing PMS or related issues; it remains a research finding. But it underscores that women's inflammatory status is dynamic. For Indonesian women, traditional herbal remedies (jamu) often taken for "kapala pusing" (aches) premenstrually might have anti-inflammatory properties that unknowingly target this CRP rise[22]. This interplay of culture and biochemistry could be an interesting area of ethnopharmacological research.

Limitations of CRP as a biomarker: Across all these conditions, a recurring theme is that CRP is non-specific. An elevated CRP tells us there is inflammation, but not its source. In a young woman, a CRP of 5 mg/L could mean PCOS, or it could mean she has a mild upper respiratory infection, or periodontitis, or simply that she is on the pill[24]. Therefore, CRP should not be viewed in isolation. The clinical context is paramount. For diagnostic purposes, more specific markers are needed (for example, autoimmune markers for lupus versus CA-125 for endometriosis).

However, CRP's strength lies in its sensitivity and prognostic value. High CRP in a PCOS patient might identify those at greater risk of future diabetes or heart disease[10]. It could also potentially be used to track treatment response – e.g., if a PCOS patient adopts a healthier lifestyle and CRP drops from

4 mg/L to 1 mg/L, that indicates a reduction in her systemic inflammation burden, likely reflecting health improvement.

Research Directions: The emerging research is focusing on combined indices and novel biomarkers. For instance, the CTI index combining CRP with metabolic parameters is an example of leveraging CRP's information in a broader model. Another area is the role of high-sensitivity CRP in fertility outcomes – some small studies ask if women with higher CRP have lower IVF success or higher miscarriage rates[25]. The data there is not conclusive yet, but it is an intriguing hypothesis since chronic inflammation could affect implantation. For PCOS, anti-inflammatory treatments (e.g., omega-3 supplements, or anti-diabetic drugs like thiazolidinediones) have been tested for lowering CRP and improving ovulatory function. Continued investigation along these lines may lead to integrated care where lowering CRP is a target as much as lowering testosterone or insulin in PCOS therapy[26].

For populations like Indonesia's, specific research into ethnic and lifestyle factors affecting CRP in these conditions would be valuable. Genetic differences (for example, certain IL-6 promoter polymorphisms common in Asian populations) might influence CRP levels[27]. Diets high in refined carbs (increasing triglycerides and potentially CRP) versus traditional diets could mean different baseline inflammations[28]. Also, infectious burdens (e.g., latent TB or endemic infections) could confound CRP levels[29]. Thus, establishing local reference ranges and understanding local confounders is important if CRP is to be used more in clinical practice in Indonesia or similar settings.

CONCLUSION

CRP serves as a valuable biomarker in identifying inflammation and cardiometabolic risk in women of reproductive age. Its levels reflect a range of physiological and pathological conditions that can inform early prevention strategies. Routine CRP monitoring, particularly in women with PCOS or obesity, may enhance early detection and intervention against chronic disease development.

REFERENCES

1. Aliyu M, Zohora FT, Anka AU, Ali K, Maleknia S, Saffarioun M, et al. Interleukin-6 cytokine: An overview of the immune regulation, immune dysregulation, and therapeutic approach. Int Immunopharmacol. 2022 Oct 1;111:109130.

2. Potempa M, Hart PC, Rajab IM, Potempa LA. Redefining CRP in tissue injury and repair: more than an acute pro-inflammatory mediator. Vol. 16, Frontiers in Immunology. Frontiers Media SA; 2025.

3. Rahali FZ, Mimouni N, Boukhira A, Chellak S. The Clinical Utility of Standard and High-Sensitivity C-Reactive Protein: A Narrative Review. SN Comprehensive Clinical Medicine 2024 6:1 [Internet]. 2024 Jun 5 [cited 2025 Jun 11];6(1):1–14. Available from: https://link.springer.com/article/10.1007/s42399-024-01693-3

4. Fransén K, Pettersson C, Disorders AHWC, 2022 undefined. CRP levels are significantly associated with CRP genotype and estrogen use in The Lifestyle, Biomarker and Atherosclerosis (LBA) study. Springer [Internet]. 2020 Dec 1 [cited 2025 Jun 11];22(1). Available from:

https://link.springer.com/article/10.1186/s12872-022-02610-z

5. Harding A, Cancers NH, 2022 undefined. The impact of estrogens and their receptors on immunity and inflammation during infection. mdpi.com [Internet]. 2022 [cited 2025 Jun 11]; Available from: https://www.mdpi.com/2072-6694/14/4/909

6. Azam S, Vasudevan S, Bukhari W, Cureus JT, 2025 undefined. Reproductive Endocrine Disorders: A Comprehensive Guide to the Diagnosis and Management of Infertility, Polycystic Ovary Syndrome, and Endometriosis. cureus.com [Internet]. [cited 2025 Jun 11]; Available from: https://www.cureus.com/articles/318310-reproductive-endocrine-disorders-a-comprehensive-guide-to-the-diagnosis-and-management-of-infertility-polycystic-ovary-syndrome-and-endometriosis.pdf

7. Lew LA. The Impact of the Menstrual Cycle and Hormonal Contraceptives on Cardiovascular Function in Premenopausal Females. 2024 [cited 2025 Jun 11]; Available from: https://search.proquest.com/openview/a16066fcd6b05744c4d59f685ed0c53e/1?pq-origsite=gscholar&cbl=18750&diss=y

8. Zhao H, Zhang J, Cheng X, Nie X, Research BHJ of O, 2023 undefined. Insulin resistance in polycystic ovary syndrome across various tissues: an updated review of pathogenesis, evaluation, and treatment. Springer [Internet]. 2023 Dec 1 [cited 2025 Jun 11];16(9). Available from: https://link.springer.com/article/10.1186/s13048-022-01091-0

9. Dey R, Bhattacharya K, Kumar Basak A, Paul N, Bandyopadhyay R, Ray Chaudhuri G, et al. Inflammatory perspectives of polycystic ovary syndrome: role of specific mediators and markers Middle East Fertility Society Journal. East Fertility Society Journal [Internet]. 2023 [cited 2025 Jun 11];28:33. Available from: https://doi.org/10.1186/s43043-023-00158-2

10. Aboeldalyl S, James C, Seyam E, Ibrahim EM, Shawki HED, Amer S. The Role of Chronic Inflammation in Polycystic Ovarian Syndrome—A Systematic Review and Meta-Analysis. International Journal of Molecular Sciences 2021, Vol 22, Page 2734 [Internet]. 2021 Mar 8 [cited 2025 Jun 11];22(5):2734. Available from: https://www.mdpi.com/1422-0067/22/5/2734/htm

11. Rahayu D, Meutia AP, Priyatini T, Made N, Suratih D, Maidarti M, et al. Association of hirsutism and anthropometric profiles with sexual dysfunction and anxiety levels in infertile Indonesian women with polycystic ovarian syndrome. Springer [Internet]. 2024 Dec 1 [cited 2025 Jun 11];29(1):19. Available from: https://link.springer.com/article/10.1186/s43043-024-00178-6

12. Notbohm H, Moser F, Goh J, … JFA, 2023 undefined. The effects of menstrual cycle phases on immune function and inflammation at rest and after acute exercise: A systematic review and meta-analysis. Wiley Online Library [Internet]. 2023 Aug 1 [cited 2025 Jun 11];238(4). Available from: https://onlinelibrary.wiley.com/doi/abs/10.1111/apha.14013

13. Harding AT, Heaton NS. The Impact of Estrogens and Their Receptors on Immunity and Inflammation during Infection. 2022 [cited 2025 Jun 11]; Available from: https://creativecommons.org/licenses/by/4.0/

14. Ham K van der, Louwers Y, sterility JLF and, 2022 undefined. Cardiometabolic biomarkers in women with polycystic ovary syndrome. Elsevier [Internet]. [cited 2025 Jun 12]; Available from: https://www.sciencedirect.com/science/article/pii/S0015028222001923

15. Wu J, Bochenek V, Vessels KG…, & T, 2025 undefined. C-reactive protein and the menstrual cycle in females with sickle cell disease. ashpublications.org [Internet]. 2025 [cited 2025 Jun 12];100067. Available from: https://ashpublications.org/bloodvth/article/doi/10.1016/j.bvth.2025.100067/536383

16. Li W, Pucka AQ, Debats C, Reyes BA, Syed F, O AR, et al. Inflammation and autoimmunity are interrelated in patients with sickle cell disease at a steady-state condition: implications for vaso-occlusive crisis, pain, and sensory. frontiersin.org [Internet]. 2024 [cited 2025 Jun 12];15:1288187. Available from: https://www.frontiersin.org/articles/10.3389/fimmu.2024.1288187/full

17. Gold EB, Wells C, Rasor MON. The Association of Inflammation with Premenstrual Symptoms. J Womens Health. 2016 Sep 1;25(9):865–74.

18. Torres E, Zumpf KB, Ciolino JD, Clark CT, Sit DK, Miller ES, et al. C-Reactive protein concentrations in reproductive-aged women with major mood disorders. Arch Womens Ment Health. 2022 Jun 1;25(3):577–84.

19. Modzelewski S, Oracz A, Żukow X, Iłendo K, Śledzikowka Z, Waszkiewicz N. Premenstrual syndrome: new insights into etiology and review of treatment methods. Front Psychiatry. 2024;15.

20. Duan Y, Guo L, Peng Y, Shi X, Zhao Y, Liu K, et al. Correlation between inflammatory marker and lipid metabolism in patients with uterine leiomyomas. Front Med (Lausanne). 2023;10.

21. Baldelli S, Aiello G, Mansilla Di Martino E, Campaci D, Muthanna FMS, Lombardo M. The Role of Adipose Tissue and Nutrition in the Regulation of Adiponectin. Nutrients 2024, Vol 16, Page 2436 [Internet]. 2024 Jul 26 [cited 2025 Jun 12];16(15):2436. Available from: https://www.mdpi.com/2072-6643/16/15/2436/htm

22. Chandnani S, Viras C, 2 KTSE journal Vol. ABNORMAL UTERINE BLEEDING-A CASE REPORT. suratobgysociety.com [Internet]. [cited 2025 Jun 12]; Available from: http://suratobgysociety.com/assets/images/abnormal_uterine.pdf#page=57

23. Mohammadi MM, Dehghan Nayeri N, Mashhadi M, Varaei S. Effect of omega-3 fatty acids on premenstrual syndrome: A systematic review and meta-analysis. Journal of Obstetrics and Gynaecology Research. 2022 Jun 1;48(6):1293–305.

24. Mouliou DS. C-Reactive Protein: Pathophysiology, Diagnosis, False Test Results and a Novel Diagnostic Algorithm for Clinicians. Diseases 2023, Vol 11, Page 132 [Internet]. 2023 Sep 28 [cited 2025 Jun 12];11(4):132. Available from: https://www.mdpi.com/2079-9721/11/4/132/htm

25. Zhang H, Li X, Zhang F, Li F, Jin H, Su Y, et al. Serum C-reactive protein levels are associated with clinical pregnancy rate after in vitro fertilization among normal-weight women. Front Endocrinol (Lausanne). 2023 Jan 19;14.

26. Bednarz K, Kowalczyk K, Cwynar M, Czapla D, Czarkowski W, Kmita D, et al. The Role of Glp-1 Receptor Agonists in Insulin Resistance with Concomitant Obesity Treatment in Polycystic Ovary Syndrome. International Journal of Molecular Sciences 2022, Vol 23, Page 4334 [Internet]. 2022 Apr 14 [cited 2025 Jun 12];23(8):4334. Available from: https://www.mdpi.com/1422-0067/23/8/4334/htm

27. Cheng Z, Zhang C, Mi Y. IL-6 gene rs1800795 polymorphism and diabetes mellitus: a comprehensive analysis involving 42,150 participants from a meta-analysis. Diabetol Metab Syndr [Internet]. 2022 Dec 1 [cited 2025 Jun 12];14(1):1–19. Available from: https://link.springer.com/articles/10.1186/s13098-022-00851-8

28. Giannakopoulou SP, Antonopoulou S, Chrysohoou C, Barkas F, Tsioufis C, Pitsavos C, et al. The Impact of Dietary Carbohydrates on Inflammation-Related Cardiovascular Disease Risk: The ATTICA Study (2002–2022). Nutrients 2024, Vol 16, Page 2051 [Internet]. 2024 Jun 27 [cited 2025 Jun 12];16(13):2051. Available from: https://www.mdpi.com/2072-6643/16/13/2051/htm

29. Azam K, Khosa C, Viegas S, Massango I, Bhatt N, Jani I, et al. Reduction of blood C-reactive protein concentration complements the resolution of sputum bacillary load in patients on anti-tuberculosis therapy. Front Immunol. 2022 Sep 16;13:1005692.