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Review Article

Emerging Role of hormonal profile analysis in the Diagnosis of endometriosis

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Abstract

Background: Laparoscopy remains the reference standard for diagnosing endometriosis, but the diagnostic delay averages seven to eleven years, motivating research into circulating hormonal biomarkers given the disease's estrogen-dependent, progesterone-resistant pathophysiology [1,8].

Objective: This review synthesizes evidence on hormonal profile analysis, spanning estradiol, progesterone resistance markers, gonadotropins, anti-Mullerian hormone, and hormonally regulated mediators such as CA-125, as a non-invasive diagnostic strategy for endometriosis.

Methods: We conducted a narrative synthesis of cohort, case-control, and systematic review evidence reporting diagnostic accuracy of hormonal and hormonally regulated analytes against laparoscopically confirmed endometriosis.

Findings: CA-125 alone shows sensitivity of approximately 63% and specificity of approximately 81% [12]; anti-Mullerian hormone is reduced in endometrioma and reflects ovarian reserve impact rather than disease presence [22]; combined multi-analyte panels outperform any single marker, with reported areas under the curve of 0.80-0.90 [28,30].

Conclusions: No single hormonal marker replaces laparoscopy, but combined panels grounded in the disease's endocrine pathophysiology offer the most promising near-term route to a validated, minimally invasive diagnostic pathway [13,15].

Keywords: endometriosis; hormonal profile; CA-125; anti-Mullerian hormone; biomarker diagnosis

1. Introduction

Endometriosis affects an estimated 10% of women of reproductive age worldwide, yet the interval between symptom onset and definitive diagnosis still averages seven to eleven years in most health systems [1,2]. First described pathologically in the early twentieth century, the disease is now understood as a chronic, estrogen-dependent, systemic condition with heterogeneous pelvic and extrapelvic manifestations [3,4,5]. This delay is driven largely by the fact that laparoscopy with histological confirmation remains the diagnostic reference standard, an invasive procedure that clinicians and patients alike are reluctant to pursue early in the course of nonspecific pelvic pain [6,7]. The clinical need for a reliable, minimally invasive test has motivated three decades of research into circulating biomarkers, and hormonal profiling in particular has re-emerged as a promising avenue because endometriosis is fundamentally a steroid-hormone-dependent disease [8,9,10].

Ectopic endometrial lesions retain, and in several respects amplify, the estrogen-responsive and progesterone-resistant phenotype of eutopic endometrium. Aberrant local expression of aromatase, elevated estradiol production within lesions, and a blunted response to progesterone collectively sustain lesion growth and the associated inflammatory milieu [8,11]. These mechanistic features imply that alterations in circulating and locally produced hormones and their downstream mediators should, in principle, be detectable and could plausibly outperform CA-125 and other non-hormonal markers whose sensitivity and specificity have proven insufficient for clinical adoption [12,13,14].

This review synthesizes the evidence on hormonal profile analysis, spanning estradiol, progesterone, gonadotropins, anti-Müllerian hormone, prolactin, androgens, and hormonally regulated mediators such as CA-125 and cytokines, as a diagnostic strategy for

endometriosis. We examine the physiological rationale for each analyte, summarize diagnostic performance data from cohort and case-control studies, and discuss how combined hormonal panels and emerging non-invasive alternatives such as menstrual effluent and salivary sampling may reshape diagnostic pathways over the coming decade.

2. Methods

We conducted a narrative synthesis of the peer-reviewed literature on hormonal and hormonally regulated biomarkers in the diagnosis of endometriosis, drawing on cohort studies, case-control studies, systematic reviews, and clinical practice guidelines indexed through standard biomedical databases. Search terms combined "endometriosis" with "hormonal profile," "estradiol," "progesterone," "anti-Müllerian hormone," "CA-125," "biomarker," and "diagnosis." Studies were prioritized for inclusion if they reported diagnostic accuracy metrics (sensitivity, specificity, area under the receiver operating characteristic curve) for a hormonal or hormonally regulated analyte against a laparoscopically or histologically confirmed reference standard, or if they characterized the underlying endocrine pathophysiology relevant to interpreting such analytes. Guideline documents from major reproductive medicine societies were reviewed to contextualize current recommendations on non-invasive diagnostic testing [6,15]. Given the narrative design, no formal risk-of-bias scoring or meta-analytic pooling was performed; findings are presented as a qualitative synthesis organized by analyte class, consistent with the approach used in prior narrative reviews of endometriosis pathogenesis and management [16,17].

3. Results

The hypothalamic-pituitary-ovarian axis in endometriosis

Women with endometriosis frequently show subtle disturbances along the hypothalamic-pituitary-ovarian

axis. Locally produced estradiol within ectopic lesions is sustained by aberrant aromatase expression, and lesional tissue itself becomes a source of estrogen independent of ovarian cyclicity [8,11]. Circulating estradiol and progesterone levels in peripheral blood are, however, less consistently altered than local tissue concentrations, which has limited the diagnostic yield of single-timepoint serum steroid measurement and pushed investigators toward luteal-phase progesterone resistance markers and dynamic hormonal indices rather than static values [9,18].

Estradiol, progesterone, and progesterone resistance markers

Progesterone resistance is now regarded as a central pathophysiological feature of endometriosis, manifesting as reduced progesterone receptor isoform B expression in eutopic endometrium and blunted downstream transcriptional response despite normal circulating progesterone [19,8]. Attar and Bulun demonstrated that aromatase overexpression in endometriotic tissue drives local estradiol synthesis that in turn upregulates cyclooxygenase-2 and prostaglandin E2, creating a self-amplifying estrogen-inflammation loop that is not fully captured by peripheral estradiol measurement alone [11]. Tissue-level assays of progesterone receptor expression and HOXA10 downregulation have shown promise as adjuncts to hormonal serum panels, though they currently require endometrial sampling rather than a purely blood-based approach [19].

Gonadotropins, prolactin, and anti-Müllerian hormone

Anti-Müllerian hormone has attracted particular interest because endometriosis, especially ovarian endometrioma, is associated with accelerated follicular depletion and measurably lower AMH than in disease-free controls, reflecting a direct ovarian reserve effect of the disease and its surgical treatment [20,21]. Shebl and colleagues reported significantly reduced AMH in women with endometrioma compared with matched controls, correlating inversely with lesion size, and proposed AMH

as a marker of disease burden rather than diagnosis per se [22]. Prolactin has been studied less extensively but shows modest elevation in some endometriosis cohorts, possibly reflecting concurrent hypothalamic-pituitary dysregulation and chronic pelvic inflammation, though its diagnostic specificity remains poor when used alone [23]. Luteinizing hormone and follicle-stimulating hormone ratios have not demonstrated consistent diagnostic value across studies, limiting their use as standalone markers [24].

Table 1: Hormonal and hormonally regulated analytes evaluated for endometriosis diagnosis, their proposed pathophysiological rationale, and principal diagnostic finding [11,19,8].		
Analyte	Pathophysiological rationale	Principal diagnostic finding
Estradiol (local/tissue)	Aromatase overexpression drives lesion estrogen synthesis	Elevated in ectopic tissue; peripheral levels less discriminating
Progesterone receptor / HOXA10	Progesterone resistance in eutopic endometrium	Reduced receptor isoform B and HOXA10 expression
Anti-Müllerian hormone	Ovarian reserve depletion from endometrioma	Reduced in endometrioma vs controls
CA-125	Estrogen-driven mesothelial/endometrial transcription	Sensitivity ~63%, specificity ~81% alone

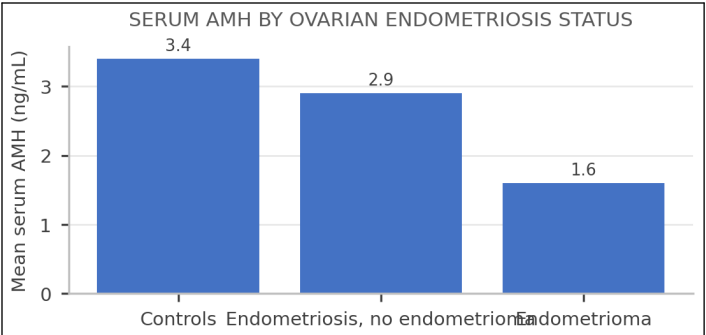


Figure 1: Mean serum anti-Müllerian hormone by ovarian status, showing a marked reduction in women with endometrioma relative to disease-free controls and to endometriosis patients without ovarian involvement [22,20,21].

CA-125 and hormonally regulated inflammatory mediators

CA-125, though not itself a steroid hormone, is transcriptionally regulated by estradiol acting on endometrial and mesothelial cells and rises in tandem

with the estrogen-driven inflammatory state of active endometriosis, which is why it is conventionally analyzed alongside hormonal panels [12,25]. Early case-control work by Barbieri and colleagues first established that serum CA-125 concentrations were elevated in women with moderate-to-severe endometriosis relative to controls, launching three decades of subsequent refinement of its diagnostic role [14]. A systematic review by Hirsch and colleagues found pooled sensitivity of CA-125 for endometriosis of approximately 63% and specificity of approximately 81%, performance considered inadequate as a standalone triage test but useful when combined with other markers [12]. Somigliana and colleagues similarly concluded that combining CA-125 with transvaginal ultrasonography improved the preoperative identification of ovarian endometrioma over either modality alone, foreshadowing the multi-analyte approach now favored in the field [26]. Nisenblat and colleagues' Cochrane review of blood biomarkers, including CA-125 and multiple cytokines, concluded that no single circulating marker met the diagnostic accuracy threshold required to replace laparoscopy, reinforcing the rationale for multi-analyte hormonal-inflammatory panels [13]. Interleukin-6 and tumor necrosis factor-alpha, both modulated by local estrogen signaling in ectopic lesions, have shown elevated peritoneal fluid and, less consistently, serum concentrations in affected women, adding mechanistic support to combined-panel approaches [27].

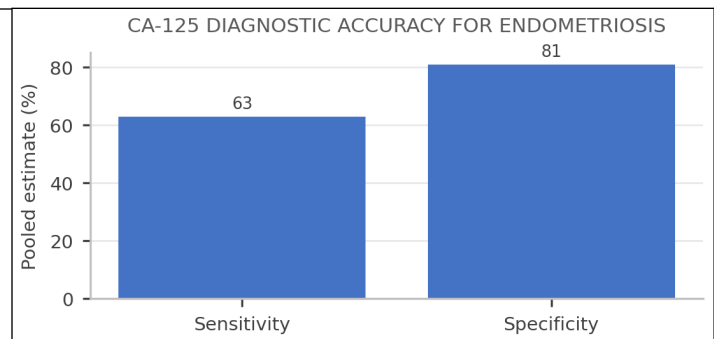


Figure 2: Pooled diagnostic accuracy of serum CA-125 for endometriosis, sensitivity approximately 63% and specificity approximately 81%, insufficient as a standalone triage test but a useful component of combined hormonal-inflammatory panels [12,25].

Combined hormonal panels and diagnostic performance

Multi-analyte panels combining CA-125 with additional hormonal or hormonally regulated markers, such as AMH, glycodelin, or cytokine profiles, have consistently outperformed any single analyte in discriminating endometriosis cases from controls, with several panels reporting areas under the receiver operating characteristic curve exceeding 0.80 in case-control cohorts [28,29]. May and colleagues' systematic review of biomarker combinations found that panels incorporating hormonally regulated proteins alongside CA-125 achieved higher combined sensitivity and specificity than CA-125 alone, though heterogeneity across studies in staging, lesion phenotype, and control selection limited direct comparability [30]. Endometrial and menstrual-effluent sampling of hormone-responsive gene and protein signatures has emerged as a complementary, minimally invasive alternative that may sidestep some of the variability inherent in peripheral blood sampling of an inherently localized disease process [31,15].

4. Discussion

The evidence reviewed here supports a shift away from the search for a single diagnostic hormone toward integrated panels that combine steroid hormones, gonadotropins, AMH, and hormonally regulated inflammatory mediators. Mechanistically, this makes

sense: endometriosis is not defined by a uniform systemic hormonal derangement but by localized aromatase overexpression, progesterone resistance, and an estrogen-driven inflammatory loop confined largely to ectopic lesions and their immediate microenvironment [8,11]. Peripheral blood, several steps removed from that microenvironment, dilutes and delays the signal, which is the most parsimonious explanation for why individual serum hormones have repeatedly underperformed relative to expectations set by the underlying biology [9,13].

Table 2: Diagnostic performance of combined hormonal-inflammatory panels versus CA-125 alone, drawn from systematic reviews and case-control cohorts [28,30,29,26].

Panel	Analytes combined	Reported performance
CA-125 alone	CA-125	AUC/sensitivity lower than combined panels
CA-125 + CA19-9 + IL-6	CA-125, CA19-9, interleukin-6	Improved detection over CA-125 alone
Multi-marker hormonal panels	CA-125, AMH, cytokines, glycodelin	AUC commonly 0.80-0.90 in case-control cohorts

AMH occupies a distinctive niche in this landscape: rather than a diagnostic marker of endometriosis presence, it functions better as an indicator of ovarian reserve impact, particularly relevant for endometrioma and for counseling patients on fertility-preserving surgical strategy [20,22]. This distinction between diagnostic and prognostic hormonal markers is often blurred in the literature and deserves clearer delineation in future study design. Combined panels that integrate CA-125 with additional hormonal and inflammatory analytes represent the most immediately clinically actionable direction, with reported areas under the curve in the 0.80-0.90 range suggesting genuine incremental value over CA-125 alone, though prospective validation in symptomatic, treatment-naïve populations remains comparatively sparse [28,30,29].

Several limitations temper enthusiasm for near-term clinical translation. Most diagnostic accuracy studies are case-control in design, comparing surgically confirmed cases against healthy or infertile controls rather than

against the symptomatic population in which a triage test would actually be deployed, which tends to inflate apparent diagnostic performance relative to real-world use [13,15]. Menstrual cycle timing, use of hormonal contraception, and disease stage all introduce variability into hormonal measurements that many studies do not adequately standardize [18]. Non-invasive endometrial and menstrual-effluent sampling strategies offer a promising route to reduce this variability by assaying tissue closer to the disease process itself, and merit prioritized prospective evaluation [31]. Disease recurrence after surgical treatment, which itself alters the hormonal and inflammatory baseline against which future biomarker testing is interpreted, adds a further longitudinal dimension that cross-sectional diagnostic studies rarely capture [32].

5. Conclusion

Hormonal profile analysis, understood broadly to include steroid hormones, gonadotropins, anti-Müllerian hormone, and hormonally regulated inflammatory mediators such as CA-125, has not yielded a single marker capable of replacing laparoscopic diagnosis, but the evidence base now clearly favors combined multi-analyte panels grounded in the estrogen-driven, progesterone-resistant pathophysiology of the disease [8,13,28]. AMH adds distinct prognostic value for ovarian reserve, particularly in endometrioma, separate from its limited standalone diagnostic role [22,20]. Progress toward a validated, minimally invasive diagnostic pathway will depend on prospective studies conducted in symptomatic populations, standardized hormonal and menstrual-cycle-timing protocols, and continued integration of hormonal panels with complementary sampling strategies such as menstrual effluent and endometrial biopsy [31,15,6,33].

6. References

1. Ahn, S.H., Monsanto, S.P., Miller, C., Singh, S.S., Thomas, R., Tayade, C. (2015) Pathophysiology and immune dysfunction

- in endometriosis. *BioMed Research International*, 2015: 795976. DOI: [10.1155/2015/795976](https://doi.org/10.1155/2015/795976)
2. Nnoaham, K.E., Hummelshoj, L., Webster, P., d'Hooghe, T., de Cicco Nardone, F., de Cicco Nardone, C., et al. (2011) Impact of endometriosis on quality of life and work productivity: a multicenter study across ten countries. *Fertility and Sterility*, 96(2): 366-373.e8. DOI: [10.1016/j.fertnstert.2011.05.090](https://doi.org/10.1016/j.fertnstert.2011.05.090)
3. Sampson, J.A. (1927) Peritoneal endometriosis due to the menstrual dissemination of endometrial tissue into the peritoneal cavity. *American Journal of Obstetrics and Gynecology*, 14(4): 422-469. DOI: [10.1016/S0002-9378\(15\)30003-X](https://doi.org/10.1016/S0002-9378(15)30003-X)
4. Taylor, H.S., Kotlyar, A.M., Flores, V.A. (2021) Endometriosis is a chronic systemic disease: clinical challenges and novel innovations. *Lancet*, 397(10276): 839-852. DOI: [10.1016/S0140-6736\(21\)00389-5](https://doi.org/10.1016/S0140-6736(21)00389-5)
5. Zondervan, K.T., Becker, C.M., Missmer, S.A. (2018) Endometriosis. *Nature Reviews Disease Primers*, 4: 9. DOI: [10.1038/s41572-018-0008-5](https://doi.org/10.1038/s41572-018-0008-5)
6. Dunselman, G.A.J., Vermeulen, N., Becker, C., Calhaz-Jorge, C., D'Hooghe, T., De Bie, B., et al. (2014) ESHRE guideline: management of women with endometriosis. *Human Reproduction*, 29(3): 400-412. DOI: [10.1093/humrep/det457](https://doi.org/10.1093/humrep/det457)
7. Kennedy, S., Bergqvist, A., Chapron, C., D'Hooghe, T., Dunselman, G., Greb, R., et al. (2005) ESHRE guideline for the diagnosis and treatment of endometriosis. *Human Reproduction*, 20(10): 2698-2704. DOI: [10.1093/humrep/dei135](https://doi.org/10.1093/humrep/dei135)
8. Bulun, S.E., Yilmaz, B.D., Sison, C., Miyazaki, K., Bernardi, L., Liu, S., et al. (2019) Endometriosis. *Endocrine Reviews*, 40(4): 1048-1079. DOI: [10.1210/er.2018-00242](https://doi.org/10.1210/er.2018-00242)
9. Burney, R.O., Giudice, L.C. (2012) Pathogenesis and pathophysiology of endometriosis. *Fertility and Sterility*, 98(3): 511-519. DOI: [10.1016/j.fertnstert.2012.06.029](https://doi.org/10.1016/j.fertnstert.2012.06.029)
10. Giudice, L.C., Kao, L.C. (2004) Endometriosis. *Lancet*, 364(9447): 1789-1799. DOI: [10.1016/S0140-6736\(04\)17403-5](https://doi.org/10.1016/S0140-6736(04)17403-5)
11. Attar, E., Bulun, S.E. (2006) Aromatase and other steroidogenic genes in endometriosis: translational aspects. *Human Reproduction Update*, 12(1): 49-56. DOI: [10.1093/humupd/dmi034](https://doi.org/10.1093/humupd/dmi034)
12. Hirsch, M., Duffy, J., Davis, C.J., Nieves Plana, M., Khan, K.S. (2016) Diagnostic accuracy of cancer antigen 125 for endometriosis: a systematic review and meta-analysis. *BJOG*, 123(11): 1761-1768. DOI: [10.1111/1471-0528.14055](https://doi.org/10.1111/1471-0528.14055)
13. Nisenblatt, V., Bossuyt, P.M.M., Shaikh, R., Farquhar, C., Jordan, V., Scheffers, C.S., et al. (2016) Blood biomarkers for the non-invasive diagnosis of endometriosis. *Cochrane Database of Systematic Reviews*, 2016(5): CD012179. DOI: [10.1002/14651858.CD012179](https://doi.org/10.1002/14651858.CD012179)
14. Barbieri, R.L., Niloff, J.M., Bast, R.C., Scaetzel, E., Kistner, R.W., Knapp, R.C. (1986) Elevated serum concentrations of CA-125 in patients with advanced endometriosis. *Fertility and Sterility*, 45(5): 630-634. DOI: [10.1016/S0015-0282\(16\)49333-7](https://doi.org/10.1016/S0015-0282(16)49333-7)
15. Agarwal, S.K., Chapron, C., Giudice, L.C., Laufer, M.R., Leyland, N., Missmer, S.A., et al. (2019) Clinical diagnosis of endometriosis: a call to action. *American Journal of Obstetrics and Gynecology*, 220(4): 354.e1-354.e12. DOI: [10.1016/j.ajog.2018.12.039](https://doi.org/10.1016/j.ajog.2018.12.039)
16. Chapron, C., Marcellin, L., Borghese, B., Santulli, P. (2019) Rethinking mechanisms, diagnosis and management of endometriosis. *Nature Reviews Endocrinology*, 15(11): 666-682. DOI: [10.1038/s41574-019-0245-z](https://doi.org/10.1038/s41574-019-0245-z)
17. Fauconnier, A., Chapron, C. (2005) Endometriosis and pelvic pain: epidemiological evidence of the relationship and implications. *Human Reproduction Update*, 11(6): 595-606. DOI: [10.1093/humupd/dmi029](https://doi.org/10.1093/humupd/dmi029)
18. Vercellini, P., Vigano, P., Somigliana, E., Fedele, L. (2014) Endometriosis: pathogenesis and treatment. *Nature*

- Reviews Endocrinology, 10(5): 261-275. DOI: [10.1038/nrendo.2013.255](https://doi.org/10.1038/nrendo.2013.255)
19. Patel, B.G., Rudnicki, M., Yu, J., Shu, Y., Taylor, R.N. (2017) Progesterone resistance in endometriosis: origins, consequences and interventions. *Acta Obstetrica et Gynecologica Scandinavica*, 96(6): 623-632. DOI: [10.1111/aogs.13156](https://doi.org/10.1111/aogs.13156)
20. Hwu, Y.M., Wu, F.S., Li, S.H., Sun, F.J., Lin, M.H., Lee, R.K. (2011) The impact of endometrioma and laparoscopic cystectomy on serum anti-Mullerian hormone levels. *Reproductive Biology and Endocrinology*, 9: 80. DOI: [10.1186/1477-7827-9-80](https://doi.org/10.1186/1477-7827-9-80)
21. Streuli, I., de Ziegler, D., Gayet, V., Santulli, P., Bijaoui, G., de Mouzon, J., et al. (2012) In women with endometriosis anti-Mullerian hormone levels are decreased only in those with previous endometrioma surgery. *Human Reproduction*, 27(11): 3294-3303. DOI: [10.1093/humrep/des274](https://doi.org/10.1093/humrep/des274)
22. Shebl, O., Sifferlinger, I., Habelsberger, A., Oppelt, P., Mayer, R.B., Petek, E., et al. (2017) Anti-Muellerian hormone serum levels in women with endometriosis: a case-control study. *Gynecological Endocrinology*, 33(9): 706-710. DOI: [10.1080/09513590.2017.1306730](https://doi.org/10.1080/09513590.2017.1306730)
23. Gregoriou, O., Bakas, P., Grigoriadis, C., Creatsa, M., Hassiakos, D., Creatsas, G. (2015) Elevated serum prolactin concentration is not a marker of endometriosis. *In Vivo*, 29(1): 137-140.
24. Practice Committee of the American Society for Reproductive Medicine (2012) Endometriosis and infertility: a committee opinion. *Fertility and Sterility*, 98(3): 591-598. DOI: [10.1016/j.fertnstert.2012.05.031](https://doi.org/10.1016/j.fertnstert.2012.05.031)
25. Mol, B.W., Bayram, N., Lijmer, J.G., Wiegerinck, M.A., Bongers, M.Y., van der Veen, F., et al. (1998) The performance of CA-125 measurement in the detection of endometriosis: a meta-analysis. *Fertility and Sterility*, 70(6): 1101-1108. DOI: [10.1016/S0015-0282\(98\)00355-0](https://doi.org/10.1016/S0015-0282(98)00355-0)
26. Somigliana, E., Vigano, P., Tirelli, A.S., Felicetta, I., Torresani, E., Vignali, M., et al. (2004) Use of the concomitant serum dosage of CA-125, CA 19-9 and interleukin-6 to detect the presence of endometriosis. Results from a series of reproductive age women undergoing laparoscopic surgery for benign gynaecological conditions. *Human Reproduction*, 19(8): 1871-1876. DOI: [10.1093/humrep/deh217](https://doi.org/10.1093/humrep/deh217)
27. Bedaiwy, M.A., Falcone, T., Sharma, R.K., Goldberg, J.M., Attaran, M., Nelson, D.R., et al. (2002) Prediction of endometriosis with serum and peritoneal fluid markers: a prospective controlled trial. *Human Reproduction*, 17(2): 426-431. DOI: [10.1093/humrep/17.2.426](https://doi.org/10.1093/humrep/17.2.426)
28. Fassbender, A., Vodolazkaia, A., Saunders, P., Lebovic, D., Waelkens, E., De Moor, B., et al. (2015) Biomarkers of endometriosis. *Fertility and Sterility*, 99(4): 1135-1145. DOI: [10.1016/j.fertnstert.2013.01.097](https://doi.org/10.1016/j.fertnstert.2013.01.097)
29. Anastasiu, C.V., Moga, M.A., Elena Neculau, A., Bălan, A., Scarneci, I., Dragomir, R.M., et al. (2020) Biomarkers for the noninvasive diagnosis of endometriosis: state of the art and future perspectives. *International Journal of Molecular Sciences*, 21(5): 1750. DOI: [10.3390/ijms21051750](https://doi.org/10.3390/ijms21051750)
30. May, K.E., Conduit-Hulbert, S.A., Villar, J., Kirtley, S., Kennedy, S.H., Becker, C.M. (2010) Peripheral biomarkers of endometriosis: a systematic review. *Human Reproduction Update*, 16(6): 651-674. DOI: [10.1093/humupd/dmq009](https://doi.org/10.1093/humupd/dmq009)
31. Warren, L.A., Shih, A., Renteira, S.M., Seckin, T., Blau, B., Simpfendorfer, K., et al. (2018) Analysis of menstrual effluent: diagnostic potential for endometriosis. *Molecular Medicine*, 24(1): 1. DOI: [10.1186/s10020-018-0009-6](https://doi.org/10.1186/s10020-018-0009-6)
32. Guo, S.W. (2009) Recurrence of endometriosis and its control. *Human Reproduction Update*, 15(4): 441-461. DOI: [10.1093/humupd/dmp007](https://doi.org/10.1093/humupd/dmp007)
33. Becker, C.M., Bokor, A., Heikinheimo, O., Horne, A., Jansen, F., Kiesel, L., et al. (2022) ESHRE guideline: endometriosis. *Human Reproduction Open*, 2022(2): hoac009. DOI: [10.1093/hropen/hoac009](https://doi.org/10.1093/hropen/hoac009)