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COMPARATIVE EPIDEMIOLOGY AND MULTIPHYSICS MODELING OF ENDOMETRIOSIS AND POLYCYSTIC OVARY SYNDROME: LINKING REPRODUCTIVE PHENOTYPES TO MDH-EOF TRANSPORT

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Abstract

Endometriosis and polycystic ovary syndrome (PCOS) are prevalent reproductive disorders associated with pelvic pain, menstrual dysfunction, infertility, and impaired quality of life. Their overlapping epidemiological and physiological characteristics remain insufficiently understood, particularly in resource-constrained populations. This study investigates the incidence and epidemiological correlates of endometriosis and PCOS while developing a coupled magnetohydrodynamic (MHD) and electroosmotic flow framework to characterize fluid transport mechanisms relevant to female reproductive physiology. An analytical epidemiological design is proposed for reproductive-age women, incorporating demographic, reproductive, clinical, lifestyle, and biochemical variables. Multivariable logistic regression will identify independent correlates and estimate adjusted odds ratios with 95% confidence intervals. Complementarily, a mathematical model of reproductive-tract fluid transport will integrate MHD forces, electroosmotic effects, pressure gradients, viscous resistance, and physiologically relevant boundary conditions. Dimensionless transformations and analytical and numerical techniques will be employed to examine velocity, temperature, concentration, wall shear stress, and transport characteristics under varying magnetic-field and electrokinetic parameters. The epidemiological analysis is expected to identify age, menstrual irregularities, infertility, obesity-related factors, family history, and metabolic characteristics as important correlates of disease occurrence and co-occurrence. The mathematical model is expected to demonstrate that magnetic and electroosmotic forces significantly influence reproductive-fluid velocity, transport efficiency, and wall shear characteristics. Integrating epidemiological evidence with multiphysics modelling may provide a novel framework for understanding reproductive disorders and supporting predictive, personalized, and technologically informed reproductive healthcare strategies in diverse clinical settings.

Keywords: endometriosis, epidemiology, electroosmosis, magneto hydrodynamics, polycystic ovary syndrome.

Introduction

Endometriosis and polycystic ovary syndrome (PCOS) are among the most important chronic disorders affecting reproductive-age women and constitute substantial challenges to contemporary reproductive healthcare. Although clinically distinct, both conditions are characterized by complex interactions between endocrine, inflammatory, metabolic, reproductive, and environmental processes that can compromise fertility and quality of life. Endometriosis is characterized by the presence of endometrial-like tissue outside the uterine cavity and is strongly associated with chronic pelvic pain, dysmenorrhea, infertility, and inflammatory changes. PCOS, in contrast, is a heterogeneous reproductive endocrine and metabolic disorder characterized principally by hyperandrogenism, ovulatory dysfunction, and, in many women, polycystic ovarian morphology. The coexistence of reproductive dysfunction, menstrual abnormalities, infertility, and systemic consequences makes both conditions important subjects for epidemiological investigation and mechanistic modelling (World Health Organization (WHO), 2025, 2026).

Globally, endometriosis affects an estimated 10% of women of reproductive age, corresponding to approximately 190 million individuals. Its manifestations extend beyond pelvic pain to include heavy menstrual bleeding, chronic pelvic discomfort, infertility, gastrointestinal and urinary symptoms, and psychological consequences. Despite its considerable burden, diagnosis remains challenging because symptoms vary substantially among individuals and may overlap with other gynaecological conditions. The World Health Organization (WHO, 2025) emphasizes that delays in diagnosis remain common, particularly where access to specialist assessment and diagnostic technologies is limited. Contemporary evidence further indicates that endometriosis is not simply an anatomical disorder but involves interactions among oestrogen signalling, inflammation, angiogenesis, immune dysregulation, oxidative stress, and alterations of the reproductive microenvironment (Garvey, 2024; Takeuchi et al., 2024). These interconnected mechanisms may explain why apparently similar clinical presentations can produce markedly different reproductive outcomes.

PCOS represents an equally important but mechanistically heterogeneous reproductive health problem. The WHO (2026) estimates that PCOS affects approximately 10–13% of reproductive-age women worldwide, while as many as 70% of affected women may remain undiagnosed.

Its clinical manifestations include irregular or absent menstruation, anovulation, infertility, hyperandrogenic symptoms, acne, hirsutism, and metabolic abnormalities. Importantly, PCOS extends beyond reproductive dysfunction because affected women have increased risks of insulin resistance, obesity, type 2 diabetes, cardiovascular disease, sleep disorders, pregnancy complications, and psychological morbidity. Recent evidence reinforces the importance of considering PCOS as a systemic metabolic condition rather than exclusively an ovarian disorder (Helvacı & Yildiz, 2025; Lonardo et al., 2024). The heterogeneity of PCOS phenotypes also creates substantial challenges for epidemiological estimation because reported prevalence varies according to diagnostic criteria, population characteristics, age, and methodological quality.

Recent global evidence illustrates this epidemiological uncertainty. Amiri et al. (2025), in a systematic review and meta-analysis involving 88 studies and 561,287 women, reported that prevalence estimates differed substantially according to diagnostic criteria. Among high-quality studies, prevalence was approximately 10.89% using Rotterdam criteria, 10.61% using Androgen Excess and PCOS Society criteria, and 6.63% using National Institutes of Health criteria. Particularly important for resource-constrained settings, the review identified a major evidence gap in Africa, where no eligible studies were available for its high-quality regional analysis. This absence of robust epidemiological evidence highlights the need for context-specific studies capable of identifying demographic, reproductive, clinical, lifestyle, and biochemical correlates of reproductive disorders. The present study responds to this gap by positioning epidemiological assessment at the centre of its investigation while simultaneously considering the physiological mechanisms that may underlie reproductive dysfunction.

The epidemiological characteristics of endometriosis and PCOS are influenced by multiple interacting factors. Age, family history, menstrual characteristics, infertility, body composition, metabolic status, lifestyle behaviours, and hormonal characteristics may contribute to disease occurrence and clinical expression. In endometriosis, inflammatory and hormonal alterations can promote lesion establishment, angiogenesis, fibrosis, and adhesion formation, thereby modifying pelvic anatomy and reproductive function (Garvey, 2024; Takeuchi et al., 2024).

In PCOS, genetic predisposition interacts with endocrine, metabolic, environmental, and lifestyle factors, with insulin resistance, hyperandrogenism, adiposity, and chronic low-grade inflammation forming important components of the disease phenotype (Deng et al., 2024; Lonardo et al., 2024). These observations support the use of multivariable epidemiological approaches capable of distinguishing independent correlates from associations arising through confounding.

The reproductive consequences of both conditions provide an additional rationale for integrated investigation. Endometriosis-associated infertility may arise from anatomical distortion, pelvic adhesions, impaired ovarian function, altered tubal transport, inflammatory changes, oxidative stress, abnormal gamete interactions, and impaired implantation. Contemporary evidence suggests that even when severe anatomical distortion is absent, changes in the endocrine, immune, and oxidative environment may adversely affect reproductive processes (Garvey, 2024). Similarly, PCOS can disrupt follicular development and ovulation through interactions involving hyperandrogenism, altered hypothalamic-pituitary-ovarian signalling, insulin resistance, adiposity, and chronic inflammation (Lonardo et al., 2024). Consequently, reproductive-fluid movement and the microenvironment in which gametes, hormones, signalling molecules, and other biological constituents are transported deserve greater attention as potential mechanistic dimensions of reproductive health.

It is within this context that mathematical modelling provides an important complementary approach. Conventional epidemiological analyses can identify patterns of disease occurrence and statistical associations but may provide limited information about the physical mechanisms governing fluid movement within reproductive microenvironments. Mathematical models based on fluid mechanics can potentially bridge this gap by describing how pressure gradients, viscous resistance, electrical forces, magnetic forces, and boundary conditions interact to regulate biological-fluid transport. Such modelling is particularly relevant to microscale physiological systems, where electrokinetic and magnetohydrodynamic effects can influence flow behaviour. Choudhari et al. (2024) demonstrated that the integrated consideration of electroosmotic and magnetohydrodynamic mechanisms can provide useful insight into physiological peristaltic pumping and microscale biomimetic fluid transport. Their work supports the broader applicability of coupled MHD–EOF frameworks to biomedical fluid-dynamics problems.

Electroosmotic flow is generated through the interaction between an applied electric field and charged fluid interfaces, whereas magnetohydrodynamic effects arise when electrically conducting fluids interact with an externally applied magnetic field. In biological-fluid systems, these forces can modify velocity distributions, transport efficiency, wall shear characteristics, heat transfer, and concentration profiles. The simultaneous consideration of MHD and EOF is therefore potentially valuable for modelling complex reproductive-fluid environments in which electrochemical and mechanical forces may coexist. Although such modelling has increasingly been investigated in biomedical engineering and microfluidic applications, its explicit integration with epidemiological research on endometriosis and PCOS remains comparatively underdeveloped. This represents an important conceptual gap that the present study seeks to address.

The proposed integration is particularly relevant because endometriosis and PCOS involve altered biological microenvironments rather than isolated structural abnormalities. In endometriosis, inflammatory signalling, angiogenesis, fibrosis, and altered hormonal responsiveness can modify the local reproductive environment (Garvey, 2024; Takeuchi et al., 2024). In PCOS, insulin resistance, hyperandrogenism, adiposity, and chronic inflammation can influence ovarian and systemic physiology (Deng et al., 2024; Helvaci & Yildiz, 2025). A mathematical description of fluid transport may therefore provide a mechanistic layer through which changes in physical transport properties can be examined alongside epidemiological and clinical characteristics. The proposed model incorporates pressure gradients, viscous resistance, magnetic forces, electroosmotic effects, and physiologically relevant boundary conditions, thereby providing a framework for examining velocity, temperature, concentration, wall shear stress, and transport characteristics under varying physical parameters.

The need for such an interdisciplinary approach is strengthened by current developments in reproductive medicine. The 2022 European Society of Human Reproduction and Embryology (ESHRE) guideline emphasizes the complexity of diagnosing and managing endometriosis, particularly because symptoms, disease location, infertility, and long-term quality-of-life consequences vary considerably among patients (Becker et al., 2022). Similarly, the 2023 International Evidence-based Guideline for PCOS emphasizes that diagnosis and management require consideration of reproductive, metabolic, psychological, and long-term health dimensions (Teede et al., 2023).

These developments demonstrate a movement away from narrowly defined disease models toward integrated approaches that account for heterogeneous clinical phenotypes and multiple interacting biological pathways.

Against this background, the present study investigates the incidence and epidemiological correlates of endometriosis and PCOS among reproductive-age women while developing a coupled MHD–EOF framework for reproductive-tract fluid transport. The epidemiological component incorporates demographic, reproductive, clinical, lifestyle, and biochemical characteristics and uses multivariable logistic regression to identify independent correlates and estimate adjusted odds ratios with 95% confidence intervals. The mathematical component complements these analyses by examining how magnetic-field and electrokinetic parameters influence fluid velocity, transport efficiency, concentration, temperature, and wall shear characteristics. By connecting epidemiological evidence with multiphysics modelling, the study seeks to move beyond conventional descriptions of reproductive disorders toward a more integrated understanding of disease-associated reproductive microenvironments. Such an approach may contribute to improved mechanistic interpretation, predictive modelling, personalized reproductive healthcare, and future development of technologically supported approaches to reproductive medicine, particularly in populations where epidemiological and mechanistic evidence remains limited.

Figure 1 summarizes the diametric phenotypes of endometriosis and PCOS, in the context of the diverse lines of evidence described above.

Figure 2: Schematic Diagram: Endometriosis and PCOS-Simple Overview

Methods

This study was structured as an analytical, interdisciplinary evidence-synthesis study combining epidemiological comparison with a theoretical biomedical fluid-transport model. The methodological structure follows the comparative approach used by Dinsdale and Crespi (2021), who systematically organized evidence on developmental biology, endocrine physiology, reproductive physiology, morphology, life history, and epidemiology to test the diametric-disorders hypothesis.

For the epidemiological component, evidence was organized around reproductive-age women and the principal variables identified in the manuscript: age, menstrual characteristics, infertility, family history, body-composition indicators, metabolic characteristics,

hormonal profiles, and disease status. Endometriosis and PCOS were treated as distinct outcomes, with co-occurrence considered separately. Because the supplied manuscript does not contain a participant-level dataset, no new prevalence, incidence, odds ratios, p-values, or confidence intervals were calculated. Instead, quantitative findings reported in the supplied reference article were extracted and synthesized. This distinction is important because prevalence or co-occurrence estimates should not be presented as original incidence estimates without a defined population at risk and observation period (Dinsdale & Crespi, 2021).

Evidence extraction focused on developmental and morphological indicators of prenatal androgen exposure, particularly anogenital distance (AGD), second-to-fourth digit ratio (2D:4D), waist-to-hip ratio (WHR), body mass index (BMI), fat distribution, and muscle mass. These variables were selected because the source literature reports contrasting patterns between endometriosis and PCOS and relates several of them to prenatal or adult androgenic activity (Mendiola et al., 2012, 2016; Mira-Escolano et al., 2014; Peters et al., 2020).

For the endocrine component, the synthesis considered luteinizing hormone (LH), follicle-stimulating hormone (FSH), anti-Müllerian hormone (AMH), testosterone, sex hormone-binding globulin (SHBG), estradiol, β -endorphin, oxytocin, kisspeptin, activin, and inhibin. The direction of change was coded relative to unaffected controls as decreased, increased, mixed, or context-dependent, consistent with the comparative framework in the supplied reference article (Dinsdale & Crespi, 2021).

The mathematical component was formulated as a conceptual coupled magnetohydrodynamic–electroosmotic-flow (MHD–EOF) framework for reproductive-tract fluid transport. The model considers incompressible electrically conducting fluid under pressure-gradient, viscous, magnetic, and electrokinetic influences. A representative momentum balance is written as $\rho(\partial \mathbf{u} / \partial t + \mathbf{u} \cdot \nabla \mathbf{u}) = -\nabla p + \mu \nabla^2 \mathbf{u} + \rho_e \mathbf{E} + \mathbf{J} \times \mathbf{B}$, with continuity $\nabla \cdot \mathbf{u} = 0$. Here, ρ is fluid density, $\mathbf{u}$ is velocity, p is pressure, μ is dynamic viscosity, ρ_e is local charge density, $\mathbf{E}$ is electric field, $\mathbf{J}$ is electric current density, and $\mathbf{B}$ is magnetic flux density. An energy equation, species-transport equation, and wall-shear relation may subsequently be coupled to the velocity field to evaluate temperature, concentration, and transport behaviour. This modelling strategy is consistent with the use of coupled MHD and electroosmotic mechanisms in physiological and microscale transport problems described by Choudhari et al. (2024).

Analytically, the epidemiological evidence was summarized using directional comparisons and reported effect estimates, whereas the mathematical framework was intended for parameter-sensitivity analysis of magnetic-field strength, electric-field intensity, pressure gradient, viscosity, and wall-charge/electrokinetic parameters. Where the present manuscript does not provide numerical model parameters or patient-level observations, the corresponding outputs are described as theoretical/model expectations rather than empirical measurements.

Results

The evidence synthesis identified a consistent contrast between endometriosis and PCOS across developmental, morphological, endocrine, and reproductive domains. The principal pattern was that endometriosis was associated in the reviewed literature with markers consistent with relatively lower androgenic exposure or activity, whereas PCOS was

more often associated with markers consistent with higher androgenic exposure or activity. These findings reproduce the central comparative pattern reported by Dinsdale and Crespi (2021), while the present manuscript places the pattern within an epidemiological and MHD–EOF modelling framework.

Developmental and morphological correlates

Shorter AGD was repeatedly reported among women with endometriosis, whereas longer AGD was reported among women with PCOS. In the source evidence, short AGD showed strong associations with endometriosis, including reported odds ratios of 41.6, 6.0, and 2.8 across three datasets, with high specificity in two studies. PCOS was associated with longer AGD in several studies. The remaining morphological indicators showed broadly contrasting directions, with lower WHR and lower BMI more frequently reported in endometriosis and higher WHR and above-normal BMI/obesity more frequently reported in PCOS (Dinsdale & Crespi, 2021).

Trait	Relationship to hormonal biology	Endometriosis	PCOS
Anogenital distance (AGD)	Proxy positively associated with prenatal testosterone exposure	Generally shorter than controls; strong associations reported	Generally longer than controls
2D:4D digit ratio	Generally inversely related to prenatal testosterone exposure	No consistent significant difference; higher ratio linked with bleeding/dysmenorrhea related evidence	Lower ratio reported in several studies, but findings are not uniform
Waist-to-hip ratio (WHR)	Higher WHR is associated with higher testosterone in women	Generally lower than controls	Generally higher than controls
Body mass index (BMI)	Associated with testosterone and reproductive outcomes	Often lower than controls; relationship with severity is non-linear	Above-normal BMI/obesity commonly reported
Fat distribution	Androgens favour abdominal distribution; estrogen favours lower-body distribution	More gynoid/lower-body distribution reported	More abdominal adiposity reported
Muscle mass	Androgens support lean muscle development	Lower muscle mass reported	Higher muscle mass reported in comparative studies

Table 1: Comparative Developmental, Anthropometric, and Morphological Correlates of Endometriosis and Polycystic Ovary Syndrome (PCOS) in Relation to Androgenic Biology

Comparative hormonal profiles

The endocrine synthesis showed contrasting alterations in several components of the hypothalamic–pituitary–gonadal axis. Endometriosis was characterized by lower LH, higher FSH, lower AMH, lower testosterone, and higher SHBG relative to controls in the source synthesis, whereas PCOS showed the opposite direction for these variables. Additional contrasts were reported for estradiol, β -endorphin, oxytocin, kisspeptin, activin, and inhibin (Dinsdale & Crespi, 2021).

Table 2:

Hormone	Principal reproductive function	Endometriosis vs controls	PCOS vs controls
LH	Stimulates ovulation and corpus luteum development	Decreased; two surges reported	Increased; typical surge may be absent
FSH	Supports follicular maturation and estrogen secretion	Increased	Decreased
AMH	Reflects follicular pool and regulates follicular development	Decreased	Increased
Testosterone	Supports folliculogenesis and decidualization	Decreased	Increased
SHBG	Regulates sex-steroid bioavailability	Increased	Decreased
Estradiol	Regulates endometrial proliferation and reproductive development	Low-normal in serum; higher locally in lesions	Normal-high in serum; no mid-cycle peak
β -Endorphin	Modulates GnRH, pain and reproductive signalling	Decreased	Increased
Oxytocin	Contributes to uterine peristalsis	Increased	Decreased
Kisspeptin	Stimulates GnRH secretion	Mixed/limited evidence	Increased
Activin	Promotes FSH and modulates LH/testosterone	Increased	Decreased
Inhibin	Restraints FSH secretion	Decreased	Increased

Table2. *Comparative Endocrine and Reproductive Hormonal Profiles of Endometriosis and Polycystic Ovary Syndrome (PCOS) Relative to Unaffected Controls***Decidualization**

Decidualization is the coordinated biochemical and morphological transformation of endometrial stromal tissue that prepares the uterus for implantation (Okada et al., 2018). The reviewed evidence indicates that decidualization is dysregulated in both disorders, but in contrasting directions. In endometriosis, features interpreted as exaggerated or dysregulated decidualization include increased stromal-cell mobility, increased uterine natural-killer-cell activity, and altered control of endometrial invasion, enhanced angiogenesis, pro-inflammatory cytokine expression, and oxidative stress. These changes may facilitate the establishment and persistence of ectopic endometrial tissue (Dinsdale & Crespi, 2021; Gellersen & Brosens, 2014; Patel et al., 2017).

In PCOS, the evidence points toward incomplete or delayed decidualization. Endometrial stromal fibroblasts from women with PCOS have been reported to show impaired progesterone-mediated decidualization and altered cytokine profiles, while genes involved in adhesion, invasion, and tissue remodelling may be downregulated. Such abnormalities may contribute to impaired endometrial receptivity and adverse pregnancy outcomes (Piltonen et al., 2015; Younas et al., 2019). Experimental evidence further suggests that androgen excess can interfere with decidualization, while improvement in endometrial receptivity has been reported following

metformin treatment in women with PCOS (Gong et al., 2019; Palomba et al., 2006).

Within the proposed MHD–EOF framework, decidualization is best regarded as a biological boundary-condition context rather than a directly measurable fluid-dynamic variable. Changes in hormonal and inflammatory states may alter tissue properties, local fluid composition, and electrokinetic conditions; however, the supplied materials do not provide experimental measurements that would allow these biological changes to be quantitatively coupled to magnetic or electroosmotic parameters. Therefore, any such coupling should be presented as a future modelling hypothesis rather than an established empirical relationship.

Discussion

The present synthesis supports the central proposition that endometriosis and PCOS can display diametrically contrasting developmental and endocrine phenotypes. The strongest recurring contrast concerns androgenic biology: shorter AGD, lower circulating or follicular testosterone, lower AMH, lower LH relative to FSH, and lower WHR/BMI were more often associated with endometriosis, whereas longer AGD, hyperandrogenism, higher AMH, higher LH relative to FSH, and greater abdominal adiposity were more characteristic of PCOS (Dinsdale & Crespi, 2021).

These findings are biologically relevant because the HPG axis coordinates ovarian follicular development,

ovulation, menstrual cycling, and reproductive ageing. In PCOS, increased GnRH/LH activity and androgen excess contribute to follicular arrest and anovulation, while in endometriosis lower LH, altered FSH, lower testosterone, and reduced ovarian reserve are associated with a different reproductive phenotype (Dinsdale & Crespi, 2021). The comparison therefore provides a useful organizing framework for interpreting apparently overlapping clinical manifestations such as infertility and menstrual dysfunction.

The decidualization findings further strengthen the distinction between the disorders. Endometriosis is associated with an inflammatory and locally permissive endometrial environment in which altered stromal-cell behaviour, immune-cell activity, angiogenesis, and cytokine signalling may facilitate ectopic lesion establishment. PCOS, in contrast, has been associated with delayed or impaired progesterone-mediated decidualization and altered endometrial receptivity (Piltonen et al., 2015; Younas et al., 2019). These differences are important when interpreting infertility because reproductive failure may arise through different biological pathways even when the clinical endpoint is similar.

The co-occurrence evidence also warrants careful interpretation. In the supplied review, endometriosis was reported in 7.3% of women with PCOS in one comparison, compared with 19.9% among asymptomatic women without PCOS; a meta-analysis of women with PCOS undergoing ovarian drilling reported approximately 7.7% endometriosis. These estimates suggest lower co-occurrence than might be expected if the two conditions shared identical risk architecture, but the evidence is affected by differences in ascertainment and clinical selection (Dinsdale & Crespi, 2021).

The MHD–EOF component adds a mechanistic modelling layer to the epidemiological synthesis. Coupled magnetic and electroosmotic forces can alter microscale velocity fields, transport efficiency,

concentration distributions, and wall shear characteristics in electrically conducting fluids (Choudhari et al., 2024). For reproductive-fluid environments, this framework may be useful for exploring how changes in pressure, electric field, magnetic field, fluid viscosity, and surface charge could theoretically affect transport. Nevertheless, no experimental reproductive-tract MHD–EOF dataset was supplied with the manuscript; consequently, the model should presently be described as a theoretical framework rather than validated clinical evidence.

A major strength of the integrated approach is its capacity to connect epidemiological patterns with biological mechanisms while retaining the distinction between observed evidence and theoretical modelling. Its main limitation is that the supplied manuscript does not include original participant-level data, a defined sampling frame, or numerical MHD–EOF parameter values. Future empirical work should therefore collect standardized clinical and biochemical measurements, define an appropriate population denominator and observation period for incidence estimation, and validate model parameters experimentally.

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FIGURE 1.

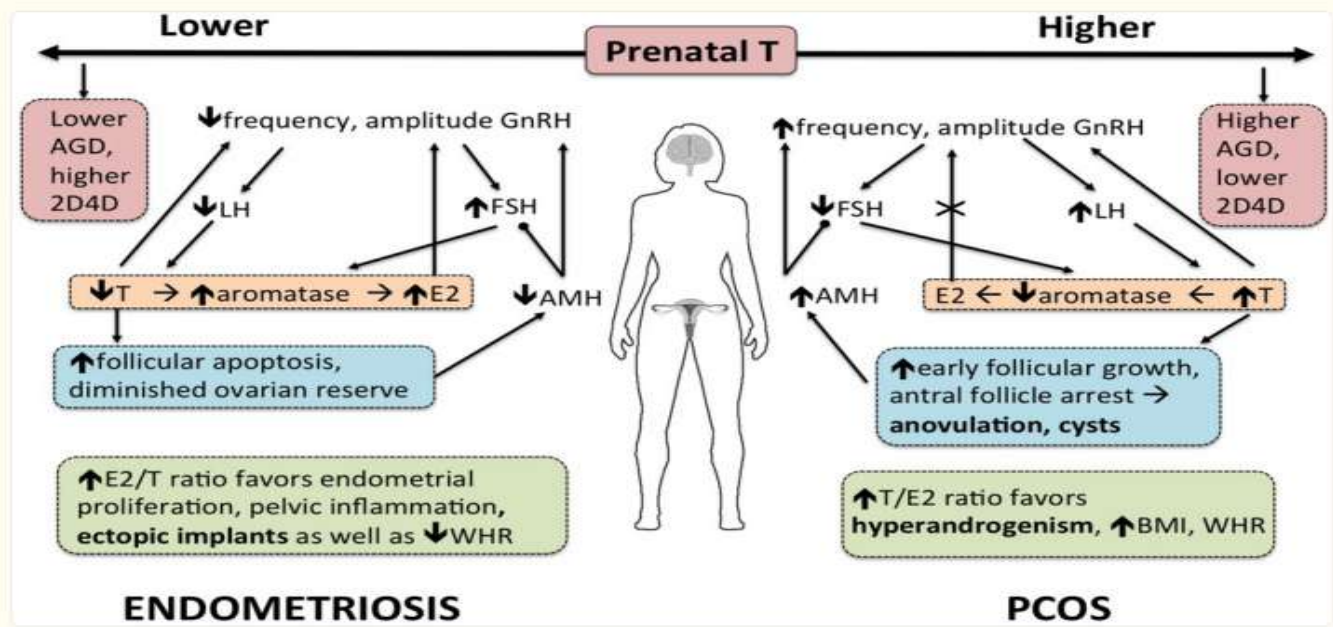


Figure 1 summarizes the diametric phenotypes of endometriosis and PCOS, in the context of the diverse lines of evidence described above.

FIGURE 2.

	ENDOMETRIOSIS		PCOS
LIFE HISTORY	Earlier	Menarche	Later
	Earlier	Menopause	Later
REPRODUCTIVE PHYSIOLOGY	Faster	Folliculogenesis	Slower
	Shorter	Menstrual cycles	Longer
MORPHOLOGY	Upregulated	Decidualization	Downregulated
	Lower	BMI	Higher
HORMONES	Lower	WHR	Higher
	Lower	Fat distribution	Android
EARLY DEVELOPMENT	Higher	Muscle mass	Higher
	Higher	LH/FSH	Higher
	Lower	AMH	Higher
	Lower	E2/T	Lower
	Higher	OT	Lower
	Lower	β	Higher
	Shorter	AGD	Longer

Figure 2: Schematic Diagram: Endometriosis and PCOS-Simple Overview

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