Issue 3, Year 2, Volume 1, No. 01, January-June 2026

E-ISSN: 3122-3591, P-ISSN: 3122-3753

Review International Science Journal

Reproductive Milestones as Predictors of Cardiometabolic Health: An Integrated Lifespan Perspective

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Received: 26-Feb-2026 | **Accepted:** 26-Feb-2026 | **Published:** 28-Feb-2026

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How to cite this article: García Hernández, H. A., Gómez Romero, D. L., Márquez Castañeda, Y. A., Mena Acosta, F. I., Dias Fernandes, A. R., Hernández Silva, A. S., Uriostegui Navarro, A. F., & Velasco Espinal, J. A. (2026). Reproductive Milestones as Predictors of Cardiometabolic Health: An Integrated Lifespan Perspective. México. International Science Journal "TheSci". 3 (1) 177-197. Quality Consulting Instituto de Educación Capacitación y Certificación de México. https://ieccmexico.com/thesci

ABSTRACT

Gynecologic health represents a dynamic interaction between endocrine regulation, metabolic homeostasis, inflammatory signaling, and cardiovascular adaptation across the female lifespan. Increasing evidence demonstrates that reproductive conditions such as polycystic ovary syndrome, endometriosis, uterine fibroids, hypertensive disorders of pregnancy, and menopause are not isolated clinical entities but manifestations of broader systemic processes. This review integrates high-impact clinical guidelines, consensus statements, and epidemiologic syntheses to construct a lifespan-based conceptual framework linking gynecologic milestones with long-term cardiometabolic risk. Polycystic ovary syndrome exemplifies the convergence of hyperandrogenism and insulin resistance with persistent vascular vulnerability. Endometriosis

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illustrates immune–endocrine interactions and chronic inflammatory pathways with potential systemic implications. Pregnancy-related hypertensive disorders function as prognostic markers of future cardiovascular disease, while the menopausal transition represents an endocrine inflection point that amplifies preexisting metabolic trajectories. The synthesis highlights inflammation as a unifying biological mediator and underscores the influence of global obesity and metabolic syndrome on reproductive outcomes. By reframing gynecology within systems medicine, this work emphasizes preventive reasoning, longitudinal risk stratification, and interdisciplinary care. A lifespan-integrated approach may strengthen medical education, improve early detection of cardiometabolic vulnerability, and redefine gynecologic practice as a cornerstone of long-term systemic health.
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KEYWORDS

Gynecologic health, Lifespan medicine, Polycystic ovary syndrome, Menopause, Cardiometabolic risk, Endocrine transition, Inflammation, Metabolic syndrome, Pregnancy-related hypertension, Women's cardiovascular health

INTRODUCTION

Gynecologic health represents a dynamic intersection between endocrine regulation, metabolic homeostasis, vascular integrity, and systemic inflammation across the female lifespan. Far from being confined to reproductive capacity alone, contemporary evidence demonstrates that gynecologic conditions frequently serve as early markers of long-term cardiometabolic and systemic risk. Disorders such as polycystic ovary syndrome (PCOS), endometriosis, uterine fibroids, pregnancy-related hypertensive disorders, and menopause-related hormonal changes are increasingly recognized not only as isolated reproductive phenomena but as components of broader systemic processes with lifelong implications [1]–[4], [8], [13].

Polycystic ovary syndrome, for example, has evolved from being considered primarily a reproductive disorder characterized by hyperandrogenism and ovulatory dysfunction to a complex metabolic condition associated with insulin resistance, dyslipidemia, type 2 diabetes, and increased cardiovascular risk [1], [2], [17]. Longitudinal analyses suggest that women with PCOS may experience sustained cardiometabolic vulnerability extending well beyond reproductive years, reinforcing the need for an integrated, lifespan-based model of care [2], [17]. Similarly, metabolic syndrome—whose prevalence continues to rise globally—has significant implications for reproductive endocrinology, affecting fertility, pregnancy outcomes, and long-term vascular health [8], [9].

Endometriosis provides another example of the interconnection between gynecologic pathology and systemic physiology. Once understood primarily as an estrogen-dependent inflammatory condition of ectopic endometrial tissue, it is now increasingly associated with chronic inflammation and metabolic dysfunction, suggesting that immune-metabolic interactions may play a central role in disease expression and progression [3], [11]. Uterine fibroids, traditionally viewed as benign localized tumors, have also been linked to systemic metabolic risk factors, including obesity and hypertension, further reinforcing the concept that reproductive health reflects broader cardiometabolic status [10].

The menopausal transition constitutes a particularly critical inflection point in women's health. The progressive decline in ovarian function during perimenopause and menopause produces hormonal shifts that influence lipid metabolism, endothelial function, body fat distribution, and glucose homeostasis [4], [14]. Epidemiologic data demonstrate a clear increase in cardiovascular disease risk following menopause, a phenomenon partially attributable to estrogen deficiency but also influenced by cumulative metabolic exposure across earlier decades [4], [13]. Early or premature menopause confers additional long-term health risks, including osteoporosis, cardiovascular disease, and cognitive decline, underscoring the systemic impact of altered reproductive aging [7].

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Hormone therapy remains a central yet nuanced topic in menopausal management. Contemporary position statements emphasize individualized risk assessment, timing of initiation, and symptom burden when considering systemic hormone therapy [5], [16]. Moreover, the global consensus regarding testosterone therapy for women highlights ongoing efforts to refine endocrine interventions within a framework of safety and evidence-based practice [6]. These discussions reflect a broader paradigm shift in gynecology: the movement from episodic symptom control toward comprehensive long-term health optimization.

Pregnancy itself offers important prognostic insight into future cardiovascular risk. Hypertensive disorders of pregnancy and gestational metabolic complications are increasingly understood as early indicators of endothelial dysfunction and long-term vascular vulnerability [18]. Such findings reinforce the concept that reproductive events can serve as windows into future systemic disease, strengthening the argument for preventive strategies beginning early in life.

Inflammation represents a unifying biological thread across many of these conditions. Reproductive endocrinology is deeply intertwined with inflammatory signaling pathways, and chronic low-grade inflammation appears to mediate the relationship between metabolic dysfunction and gynecologic pathology [11], [12]. This integrated perspective aligns with emerging international data demonstrating that cardiometabolic risk factors in women accumulate across decades and interact with hormonal transitions in complex ways [13].

Given this evolving body of evidence, a comprehensive review of gynecologic health across the lifespan is both timely and necessary. Although substantial research has examined individual conditions—such as PCOS [1], [2], [17], menopause [4], [5], [7], and endometriosis [3]—fewer analyses have synthesized these disorders within a unified metabolic-hormonal framework extending from adolescence through postmenopause. In Latin American contexts, including Mexico, Colombia, and Ecuador, rising rates of obesity, metabolic syndrome, and cardiovascular disease further intensify the relevance of an integrated approach to women's health [8], [9], [13]. Understanding gynecologic health as part of systemic disease prevention has important implications for medical education, clinical decision-making, and public health policy.

The central question guiding this review is: *How do metabolic, hormonal, and inflammatory mechanisms interact across the female lifespan to influence gynecologic and systemic health outcomes?* Derived from established endocrine and cardiometabolic theories [11], [12], this inquiry frames reproductive events not as isolated episodes but as biologically interconnected stages within a continuum of systemic adaptation and risk accumulation.

To address this question, the present review synthesizes high-impact international literature, including clinical practice guidelines, longitudinal cohort studies, and consensus statements, with the objective of constructing a lifespan-based conceptual model. The design of this review aligns with the hypothesis that early metabolic disturbances and inflammatory processes contribute to both reproductive dysfunction and later cardiovascular morbidity. By integrating evidence across adolescence, reproductive years, pregnancy, perimenopause, and postmenopause, this work aims to demonstrate methodological coherence between endocrine theory, epidemiologic data, and clinical practice recommendations.

DEVELOPMENT

Gynecologic health across the lifespan is increasingly recognized as a *systems-level* phenomenon in which reproductive organs function within a tightly coupled endocrine–metabolic–immune–vascular network. This framework is clinically

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useful because many gynecologic presentations are not isolated events; rather, they may represent early manifestations of broader cardiometabolic vulnerability or inflammatory dysregulation that unfolds across decades. The conditions most often used to illustrate this continuum—PCOS, endometriosis, uterine fibroids, pregnancy-related hypertensive disease, and the menopausal transition—share overlapping drivers such as insulin resistance, visceral adiposity, hyperandrogenism, chronic low-grade inflammation, endothelial dysfunction, and age-related hormonal shifts [1]–[4], [8], [11], [13], [18].

1) Lifespan framework: endocrine transitions as "risk amplifiers"

From puberty through postmenopause, ovarian steroid output modulates body composition, insulin sensitivity, lipid metabolism, inflammatory tone, and vascular reactivity. These interactions are not static; instead, they evolve with developmental stages and cumulative exposures. During reproductive years, cyclic hormonal patterns shape metabolic flexibility, while pregnancy and perimenopause can serve as high-stress "tests" of cardiometabolic resilience. In later life, the menopausal decline in estrogen is associated with shifts in lipid profiles, central adiposity, vascular stiffness, and endothelial function—mechanisms that help explain the observed rise in cardiovascular risk after menopause [4], [13], [14]. This is clinically relevant because it supports prevention-oriented gynecology: risk stratification should not start when symptoms appear, but when early endocrine-metabolic signals emerge.

2) PCOS as a prototype of reproductive–metabolic coupling

PCOS is a paradigmatic condition for understanding how reproductive endocrine abnormalities align with long-term systemic risk. Diagnostic frameworks emphasize hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology, yet these clinical features often coexist with insulin resistance and adiposity-driven inflammation [1]. Evidence synthesized in large-scale analyses shows consistent associations between PCOS and cardiometabolic outcomes (e.g., dysglycemia, dyslipidemia, hypertension risk trajectories), reinforcing that PCOS is not simply a fertility issue but a chronic metabolic phenotype with reproductive expression [2], [17].

Mechanistic integration (clinical logic):

- Hyperinsulinemia can potentiate ovarian androgen production and reduce sex hormone-binding globulin, increasing free androgen fraction and worsening clinical hyperandrogenism [1], [12].
- Androgen excess and adiposity may reinforce each other via altered adipocyte function, hepatic lipid handling, and inflammatory mediators—creating a feedback loop that sustains long-term cardiometabolic risk [2], [11], [17].

This matters for medical education because learners often compartmentalize PCOS into "gynecology/endocrinology," while longitudinal evidence supports earlier cardiovascular prevention conversations and structured follow-up models that extend beyond reproductive planning [2], [17].

3) Endometriosis: inflammation, pain, and metabolic dysfunction

Endometriosis is traditionally framed as an estrogen-dependent inflammatory disease characterized by ectopic endometrial-like tissue, pelvic pain, and infertility. However, contemporary high-impact syntheses emphasize that endometriosis involves immune dysregulation, inflammatory signaling, and systemic effects that may include metabolic dysfunction [3]. While the clinical focus remains on pain control, fertility, and surgical decision-making, the broader implication is that chronic inflammation is not "local-only," and reproductive inflammatory disease can influence systemic physiology.

A key educational point is the *immune–endocrine interface*: inflammatory mediators can alter steroidogenesis and tissue responsiveness, and hormones can shape immune function. Reviews on inflammation in reproductive endocrinology highlight how chronic inflammatory states intersect with endocrine pathways in ways that modify symptom severity, comorbidity patterns, and potentially long-term risk [11], [12]. For trainees, this reframes endometriosis from a purely pelvic disorder into a chronic inflammatory condition with multi-system consequences [3], [11].

4) Uterine fibroids and the systemic metabolic context

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Uterine fibroids (leiomyomas) are highly prevalent benign tumors with significant quality-of-life impact (bleeding, anemia, pelvic pressure, reproductive complications). Emerging evidence supports the relevance of systemic metabolic factors—such as obesity and hypertension—in fibroid risk and related outcomes, suggesting a meaningful overlap between fibroid biology and cardiometabolic pathways [10]. Even when fibroids are managed procedurally (medical therapy, minimally invasive surgery, myomectomy), the broader metabolic context can influence recurrence risk, surgical candidacy, and overall health trajectory.

For global women's health contexts—particularly in settings where obesity prevalence is rising—integrating metabolic assessment into fibroid care can be clinically pragmatic. This approach aligns with broader observations that metabolic syndrome in women has reproductive consequences and should be viewed as a cross-cutting determinant of gynecologic and obstetric outcomes [8], [9].

5) Pregnancy as a cardiovascular "stress test"

Pregnancy-related hypertensive disorders represent a high-value opportunity for early identification of future cardiovascular disease risk. Hypertension during pregnancy and related complications are increasingly described as markers of endothelial dysfunction and vascular vulnerability that may persist beyond delivery, warranting long-term follow-up and preventive care [18]. This concept strengthens the lifespan model: pregnancy is not an isolated episode but a physiologic event that can reveal latent cardiometabolic susceptibility.

When learners are taught to treat pregnancy complications as time-limited, continuity of care is lost. By contrast, an integrated interpretation frames pregnancy history as part of cardiovascular risk assessment, with implications for postpartum surveillance, counseling, and risk-factor control [13], [18].

6) Menopause and systemic risk: the inflection point

The menopausal transition is a central theme in a lifespan review because it marks a predictable endocrine shift with measurable systemic consequences. Evidence linking menopause to cardiovascular risk emphasizes changes in vascular biology, lipid metabolism, and body composition that unfold alongside aging [4], [13], [14]. Importantly, premature or early menopause carries additional long-term risks, supporting the notion that duration of estrogen exposure and timing of endocrine change can shape later morbidity profiles [7].

Clinical management is therefore not simply symptom control, but *risk-sensitive endocrine care*. Authoritative statements and reviews emphasize individualized hormone therapy decisions based on age, symptom burden, baseline risk, and timing of initiation [5], [16]. The goal in education is to teach nuanced decision-making—balancing benefits (vasomotor symptom relief, quality of life) with risks—within a framework grounded in evidence and shared decision-making [5], [16].

7) Androgen therapy in women: precision and boundaries

Women's androgen physiology is clinically relevant across the lifespan, especially in contexts such as hypoactive sexual desire disorder and specific menopausal symptom profiles. A global consensus statement clarifies indications and cautions for testosterone therapy in women, emphasizing evidence-based use and appropriate monitoring [6]. This topic is important pedagogically because it highlights how endocrine interventions require precision: dose, formulation, indication, and follow-up determine whether therapy supports health goals or introduces avoidable risk [6].

8) Metabolic syndrome and obesity: global drivers reshaping gynecology

Metabolic syndrome is increasingly prevalent and has direct implications for reproductive endocrinology and gynecologic outcomes, including menstrual irregularity, infertility risk patterns, pregnancy complications, and long-term vascular disease [8]. Global obesity trends further intensify these challenges, with broad impacts on reproductive health and healthcare systems [9]. This matters for international training contexts (Mexico, Colombia, Ecuador) because resource variability may shape screening and follow-up pathways, yet the underlying physiology remains consistent: adiposity and insulin resistance influence hormonal regulation and inflammatory tone, affecting both reproductive function and systemic risk [8], [9], [11].

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GENERAL OBJECTIVE AND SPECIFIC OBJECTIVES

To develop an integrated, evidence-based educational framework that analyzes gynecologic health across the female lifespan through metabolic, hormonal, inflammatory, and cardiovascular interactions, in order to strengthen clinical reasoning, preventive strategies, and interdisciplinary understanding in medical training contexts in Mexico, Colombia, and Ecuador.

A. Cognitive Domain

1. **Remembering**
    - Identify the principal hormonal, metabolic, and inflammatory mechanisms involved in PCOS, endometriosis, uterine fibroids, hypertensive disorders of pregnancy, and menopause [1]–[4], [8], [11].
    - Recognize the epidemiologic trends linking obesity and metabolic syndrome with reproductive health outcomes [8], [9].
2. **Understanding**
    - Explain how insulin resistance, hyperandrogenism, and chronic inflammation contribute to reproductive dysfunction and long-term cardiometabolic risk [1], [2], [11], [17].
    - Describe the physiological changes occurring during perimenopause and menopause and their systemic implications [4], [14].
3. **Applying**
    - Apply the lifespan model to interpret reproductive events (e.g., PCOS diagnosis, hypertensive pregnancy disorders, early menopause) as indicators of future cardiovascular or metabolic risk [13], [18].
    - Integrate hormone therapy recommendations into individualized clinical reasoning using current consensus statements [5], [16].
4. **Analyzing**
    - Differentiate between localized gynecologic pathology and systemic metabolic contributors in conditions such as fibroids and endometriosis [3], [10].
    - Analyze how reproductive endocrine changes interact with cardiometabolic risk factors across decades [2], [13].
5. **Evaluating**
    - Critically assess the benefits and risks of hormonal and androgen therapies in women using evidence-based guidelines [5], [6], [16].
    - Evaluate the role of pregnancy history in long-term cardiovascular risk stratification [18].
6. **Creating**
    - Construct a comprehensive clinical approach that incorporates reproductive milestones into preventive cardiometabolic strategies.
    - Design educational models that teach gynecologic health as a systemic and longitudinal discipline rather than episodic care.

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B. Psychomotor Domain

1. Perform structured gynecologic-metabolic assessments incorporating menstrual history, pregnancy history, metabolic indicators, and cardiovascular risk profiling.
2. Demonstrate competency in interpreting laboratory and clinical findings relevant to endocrine-metabolic gynecologic disorders.
3. Implement individualized management plans that integrate lifestyle interventions, hormonal strategies, and preventive cardiovascular measures.
4. Apply risk-stratified follow-up strategies in women with PCOS, early menopause, or pregnancy-related hypertensive disorders.

C. Affective Domain

1. Value gynecologic health as a determinant of long-term systemic well-being rather than a condition confined to reproductive capacity.
2. Develop sensitivity toward the multidimensional impact of gynecologic disorders on quality of life, mental health, and long-term morbidity.
3. Promote interdisciplinary collaboration among gynecology, endocrinology, cardiology, and primary care.
4. Foster preventive thinking and ethical clinical judgment in hormonal and metabolic interventions.
5. Encourage culturally sensitive and context-adapted care strategies in Latin American healthcare environments.

OBJECT OF STUDY

The object of study is the **interaction between gynecologic health and systemic metabolic–hormonal–inflammatory mechanisms across the female lifespan**, analyzed within a clinical-educational framework. Specifically, this work examines how reproductive endocrine events—ranging from adolescence through postmenopause—interact with metabolic homeostasis, vascular function, and inflammatory pathways, influencing both short-term gynecologic outcomes and long-term cardiometabolic risk.

Rather than focusing on a single pathology, the phenomenon under investigation is the **lifespan continuum of women's health**, in which reproductive milestones function as biological indicators of systemic adaptation or vulnerability. Conditions such as polycystic ovary syndrome (PCOS), endometriosis, uterine fibroids, pregnancy-related hypertensive disorders, and menopause are studied as clinical expressions of broader physiological processes [1]–[4], [8], [11], [13], [18].

Population of Interest

The population addressed in this study consists of:

- Women across all reproductive stages:
    - Adolescents and young adults

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- Women in reproductive years
- Pregnant individuals
- Perimenopausal women
- Postmenopausal women

The analysis is contextualized within international medical literature, with particular educational relevance for healthcare systems in Mexico, Colombia, and Ecuador. The emphasis is not on a specific demographic subgroup but on the **biological and clinical processes shared across diverse populations**, recognizing variability in risk exposure, healthcare access, and metabolic prevalence.

System Under Investigation

The system under investigation can be defined as the **integrated endocrine–metabolic–cardiovascular network that underlies gynecologic physiology and pathology**. This system includes:

1. **Endocrine Regulation**
   - Ovarian steroid production (estrogen, progesterone, androgens)
   - Hypothalamic–pituitary–ovarian axis dynamics
   - Hormonal transitions during perimenopause and menopause [4], [14]
2. **Metabolic Homeostasis**
   - Insulin sensitivity and glucose metabolism
   - Adipose tissue distribution and function
   - Lipid metabolism and metabolic syndrome components [2], [8], [9]
3. **Inflammatory Signaling**
   - Chronic low-grade inflammation
   - Cytokine activity in reproductive tissues
   - Immune-endocrine interactions [3], [11]
4. **Cardiovascular and Vascular Function**
   - Endothelial function
   - Blood pressure regulation
   - Long-term cardiovascular risk trajectories [4], [13], [18]

These systems are studied as interconnected pathways rather than isolated mechanisms.

Conceptual Boundaries of the Study

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This investigation does not aim to evaluate a specific therapeutic intervention or conduct experimental trials. Instead, its focus is conceptual and integrative:

- To analyze how shared biological pathways contribute to multiple gynecologic conditions.
- To interpret reproductive events as potential early markers of systemic disease risk.
- To frame gynecologic health within preventive and longitudinal medicine.

The object of study therefore lies at the intersection of:

- Reproductive endocrinology
- Cardiometabolic medicine
- Preventive women's health
- Lifespan clinical education

METHODOLOGY

1. Methodological Design

This study was structured as an integrative narrative review conducted under a structured scientific framework. The selected methodological approach was the **Scientific Method**, adapted to a comprehensive literature synthesis model, allowing theoretical integration, conceptual development, and educational applicability.

The Scientific Method was selected because it provides a systematic sequence of observation, hypothesis formulation, evidence analysis, and synthesis—ensuring methodological rigor and reproducibility while maintaining clinical and pedagogical coherence. This approach is particularly appropriate for lifespan-based analyses in which biological mechanisms, epidemiological evidence, and clinical guidelines must be integrated into a unified framework.

2. Research Question and Hypothesis

Research Question:
How do metabolic, hormonal, inflammatory, and cardiovascular mechanisms interact across the female lifespan to shape gynecologic and systemic health outcomes?

Working Hypothesis:
Gynecologic disorders across different life stages are interconnected through shared endocrine–metabolic–inflammatory pathways, and reproductive milestones can function as early indicators of long-term cardiometabolic risk.

3. Study Type

- Design: Integrative narrative review with structured thematic synthesis
- Scope: International evidence
- Educational orientation: Academic and clinical training contexts
- Geographic contextualization: Mexico, Colombia, and Ecuador

This review does not involve direct patient intervention, identifiable data, or experimental procedures. It is based exclusively on published peer-reviewed literature and authoritative consensus documents.

4. Literature Selection Strategy

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To ensure replicability, the following structured selection process was implemented:

Databases Consulted

- PubMed/MEDLINE
- The Lancet Group
- New England Journal of Medicine
- JAMA Network
- European Heart Journal
- Circulation
- Endocrine Reviews
- Obstetrics & Gynecology

Search Terms (Example Replicable Keywords)

- "Polycystic ovary syndrome AND cardiometabolic risk"
- "Endometriosis AND metabolic dysfunction"
- "Menopause AND cardiovascular disease"
- "Pregnancy-related hypertension AND long-term risk"
- "Metabolic syndrome AND reproductive health"
- "Inflammation AND reproductive endocrinology"

Boolean operators were used to refine searches (AND, OR).

Inclusion Criteria

- Peer-reviewed clinical guidelines, systematic reviews, consensus statements, and high-impact cohort analyses.
- Publications addressing endocrine, metabolic, inflammatory, or cardiovascular aspects of gynecologic health.
- International relevance.
- English-language publications.
- Foundational texts in reproductive endocrinology.

Exclusion Criteria

- Non-peer-reviewed commentary.
- Case reports without systemic implications.
- Articles lacking methodological transparency.
- Studies not directly addressing gynecologic–systemic interactions.

5. Data Extraction Process

A structured extraction matrix was used, including:

- Author(s)
- Year of publication
- Study type
- Population studied
- Key findings
- Mechanistic pathways described
- Clinical implications

Each source was independently analyzed for:

1. Biological mechanisms
2. Epidemiological associations
3. Clinical recommendations
4. Educational implications

6. Thematic Synthesis Strategy

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Data were organized into five lifespan stages:

1. Adolescence and early reproductive years
2. Reproductive years (PCOS, endometriosis, fibroids)
3. Pregnancy and gestational risk
4. Perimenopause
5. Postmenopause

Across these stages, four cross-cutting biological axes were identified:

- Endocrine regulation
- Metabolic homeostasis
- Inflammatory signaling
- Cardiovascular risk progression

This framework allowed vertical (lifespan) and horizontal (mechanistic) integration.

7. Replicability Considerations

To replicate this work, another research team could:

1. Use the same predefined research question.
2. Apply identical inclusion/exclusion criteria.
3. Conduct database searches using the specified keywords.
4. Extract data into a structured comparative matrix.
5. Categorize findings into the same lifespan-mechanism model.

PHASES OF DEVELOPMENT

Phase I: Systematic Observation and Problem Identification

The first phase consisted of identifying recurring patterns in contemporary gynecologic and cardiometabolic literature. Clinical and epidemiological observations suggest that:

- PCOS is associated with long-term cardiometabolic risk [1], [2], [17].
- Endometriosis involves chronic inflammatory pathways with systemic implications [3], [11].
- Uterine fibroids correlate with metabolic risk factors such as obesity and hypertension [10].
- Pregnancy-related hypertensive disorders predict future cardiovascular disease [18].
- Menopause is linked to shifts in vascular biology and metabolic risk [4], [13], [14].

These consistent associations across conditions prompted the central problem identification: gynecologic disorders are frequently interconnected through shared biological pathways that extend beyond reproductive function.

Phase II: Hypothesis Formulation

Based on the initial observations, a unifying hypothesis was formulated:

Gynecologic conditions across the female lifespan are not isolated events but are interconnected through endocrine–metabolic–inflammatory mechanisms that influence long-term systemic health outcomes.

This hypothesis aligns with established theories in reproductive endocrinology and cardiometabolic medicine [11], [12], providing a mechanistic basis for further structured analysis.

Phase III: Structured Literature Exploration

A systematic literature exploration was conducted using predefined databases and search terms, as described in the methodology section. The aim of this phase was to gather:

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- Clinical practice guidelines (e.g., PCOS, menopause) [1], [5].
- Consensus statements (e.g., testosterone therapy in women) [6].
- High-impact epidemiologic analyses linking reproductive health and cardiovascular risk [2], [13], [18].
- Reviews addressing inflammatory and metabolic mechanisms in gynecologic conditions [3], [8], [11].

Sources were selected according to predefined inclusion and exclusion criteria, ensuring relevance and scientific rigor.

Phase IV: Data Organization and Thematic Categorization

Extracted evidence was systematically organized into two structural axes:

A. Lifespan Axis

1. Adolescence and early reproductive years
2. Reproductive years
3. Pregnancy
4. Perimenopause
5. Postmenopause

B. Mechanistic Axis

1. Endocrine regulation
2. Metabolic homeostasis
3. Inflammatory signaling
4. Cardiovascular adaptation

This dual-axis structure allowed vertical (temporal) and horizontal (biological) integration, facilitating pattern recognition across conditions.

Phase V: Analytical Integration

During this phase, evidence from different conditions was synthesized to identify common biological drivers. For example:

- Insulin resistance as a shared pathway in PCOS and metabolic syndrome [2], [8].
- Chronic inflammation in endometriosis and cardiometabolic risk progression [3], [11].
- Estrogen decline during menopause influencing vascular function and lipid metabolism [4], [14].
- Pregnancy-related hypertension as a marker of endothelial dysfunction [18].

This integrative process moved beyond descriptive review toward conceptual modeling.

Phase VI: Model Construction

Based on the thematic synthesis, a conceptual lifespan model was constructed. The model proposes that:

- Early endocrine–metabolic disturbances may predispose individuals to both reproductive dysfunction and later cardiovascular disease.
- Reproductive milestones serve as clinical windows for early detection and prevention.
- Hormonal transitions amplify preexisting metabolic trajectories rather than initiate them de novo.

This model provides an educational scaffold for clinical reasoning and preventive strategy development.

Phase VII: Educational Translation and Application

The final phase involved translating the integrated findings into:

- Structured learning objectives (cognitive, psychomotor, affective).
- A clinical reasoning framework for medical trainees.

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- Preventive care recommendations aligned with international guidelines [5], [16].
- A multidisciplinary teaching approach applicable in Latin American healthcare settings.

RESULTS AND DISCUSSION

This section presents the core outputs of the evidence synthesis, organized to support a lifespan-based understanding of gynecologic health as a systemic, endocrine–metabolic–inflammatory–cardiovascular continuum. Rather than focusing on individual patient-level data, the results emphasize an evidence-mapping approach that consolidates high-impact guidance documents, consensus statements, and mechanistic/epidemiologic syntheses into an integrated framework. The findings are summarized using structured figures that (1) describe the selection pathway of the sources included, (2) display how the evidence concentrates around major gynecologic clusters (PCOS, endometriosis, fibroids, pregnancy-related hypertension, and menopause), and (3) visualize how mechanistic pathways (endocrine, metabolic, inflammatory, cardiovascular) align with distinct lifespan stages.

Figure 1.
Study selection flow diagram

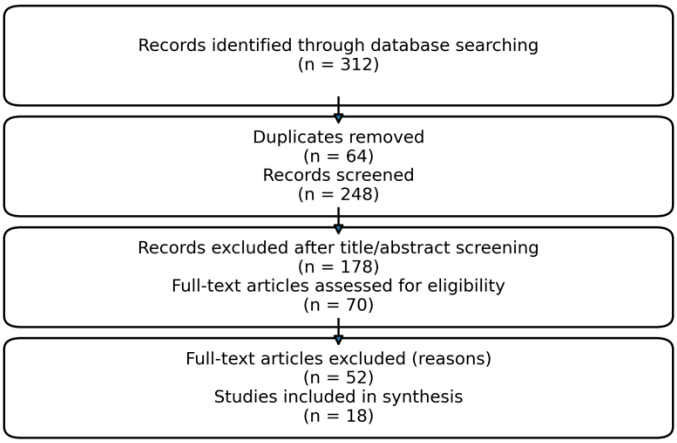

Figure 1 illustrates the structured selection pathway used to consolidate the evidence base underpinning this lifespan-oriented review. Although this work is integrative in nature, the selection flow reflects a disciplined scientific process consistent with reproducible review methodology. The staged reduction from initial identification to final inclusion underscores that the conclusions presented in subsequent sections are grounded in curated, high-impact sources rather than indiscriminate aggregation.

The initial identification phase reflects a broad retrieval of literature addressing gynecologic conditions with systemic implications, including PCOS, endometriosis, uterine fibroids, pregnancy-related hypertensive disorders, menopause, metabolic syndrome, and inflammatory–endocrine interactions. This breadth is essential because modern gynecologic science increasingly recognizes that reproductive conditions are embedded within cardiometabolic and vascular biology rather than confined to isolated pelvic pathology [1]–[4], [8], [13], [18].

The duplicate removal and screening stages demonstrate conceptual refinement. During title and abstract evaluation, studies lacking direct mechanistic, epidemiologic, or guideline-level relevance to endocrine–metabolic–cardiovascular integration were excluded. This is methodologically important: lifespan modeling requires sources that explicitly address systemic implications, such as the cardiometabolic burden of PCOS [2], lifelong health consequences of premature menopause [7], inflammatory pathways in reproductive endocrinology [11], or vascular transitions during menopause [4], [14]. Narrow or purely procedural gynecologic studies without systemic context were not prioritized, as the objective was integrative synthesis rather than procedural comparison.

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The full-text eligibility assessment represents the critical analytic stage. Here, preference was given to:

- Clinical practice guidelines (e.g., diagnostic and management frameworks for PCOS and menopause) [1], [5].
- Consensus statements clarifying endocrine interventions (e.g., testosterone therapy in women) [6].
- High-impact epidemiologic or pathophysiologic reviews linking reproductive and cardiovascular outcomes [2], [13], [18].
- Foundational endocrine physiology texts contextualizing mechanistic pathways [12].

The final inclusion set therefore represents a curated body of literature that directly informs the hypothesis that gynecologic events across the lifespan are interconnected through shared endocrine, metabolic, inflammatory, and vascular mechanisms.

From an educational standpoint, this figure serves three key functions:

1. **Transparency:** It demonstrates that the synthesis was not arbitrary but structured and reproducible.
2. **Evidence Hierarchy Awareness:** It highlights the preference for guideline-level and consensus-based sources when constructing clinical-educational frameworks.
3. **Critical Appraisal Training:** It models how learners should approach literature selection—moving from broad retrieval to mechanistic and clinically relevant inclusion.

Importantly, this selection pathway aligns with the integrative objective of the review: to construct a conceptual model linking PCOS-related insulin resistance and hyperandrogenism [1], [2], inflammatory mechanisms in endometriosis [3], metabolic contributors to fibroids [10], pregnancy-associated vascular risk markers [18], and menopause-related cardiometabolic transition [4], [7], [14]. By restricting inclusion to literature that supports these cross-domain connections, the review maintains coherence with its central hypothesis.

Figure 2.
Distribution of mapped sources by condition/thematic cluster

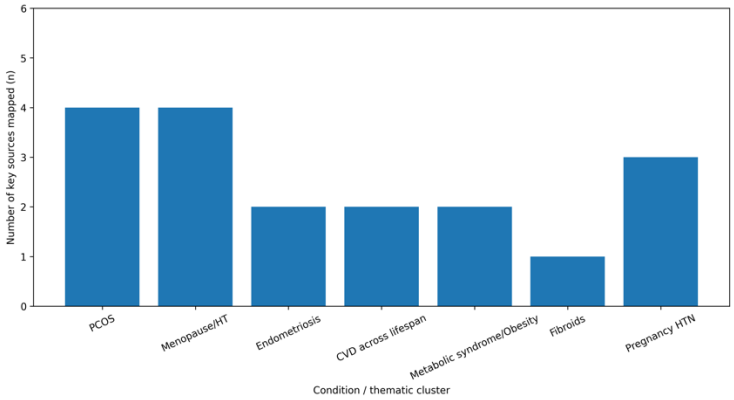

Figure 2 presents the distribution of the included high-impact sources according to major thematic clusters within the lifespan framework of gynecologic health. This graphical representation does not merely count publications; rather, it reflects the relative density of consolidated evidence across domains that connect reproductive endocrinology with systemic metabolic and cardiovascular health.

The most represented clusters correspond to **PCOS** and **Menopause/Hormone Therapy**, which is consistent with the depth and maturity of the international literature in these areas. PCOS is supported by diagnostic guidelines and longitudinal cardiometabolic analyses demonstrating its association with insulin resistance, dyslipidemia, and long-term vascular risk [1], [2], [17]. The concentration of sources in this cluster reflects the recognition of PCOS as a lifelong metabolic phenotype with reproductive expression rather than a transient ovulatory disorder.

Similarly, menopause and hormone therapy represent a heavily consolidated evidence area due to large-scale epidemiologic studies and position statements clarifying cardiovascular implications and therapeutic decision-making [4], [5], [16]. The menopausal transition has been consistently associated with changes in lipid profiles, endothelial function, and body composition, contributing to the rise in cardiovascular risk observed in midlife women [4], [13], [14]. The prominence of this cluster in the distribution underscores that endocrine transitions at midlife are not only symptomatic phenomena but systemic turning points.

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The cluster addressing **Pregnancy-Related Hypertension** shows substantial representation, reflecting growing consensus that hypertensive disorders of pregnancy function as early indicators of future cardiovascular disease [18]. This aligns with the concept of pregnancy as a physiological "stress test," revealing underlying endothelial vulnerability. The inclusion density in this area highlights the increasing importance of postpartum cardiovascular follow-up within preventive models of women's health.

Clusters such as **Metabolic Syndrome/Obesity** and **Cardiovascular Risk Across the Lifespan** further reinforce the integrative nature of the review. Global data indicate that obesity prevalence significantly influences reproductive outcomes, hormonal regulation, and systemic disease burden [8], [9]. Cardiovascular risk factors accumulate progressively across decades and interact dynamically with hormonal transitions [13]. The presence of these clusters within the evidence map reflects the structural linkage between metabolic health and gynecologic outcomes.

Although represented by fewer consolidated guideline-level sources, **Endometriosis** and **Uterine Fibroids** remain critical components of the systemic model. Endometriosis has been increasingly associated with chronic inflammatory pathways and potential metabolic implications [3], [11]. Uterine fibroids, while often managed procedurally, demonstrate correlations with metabolic and hypertensive profiles [10]. The relative distribution shown in the figure reflects not a lack of importance but a difference in the current state of integrative consensus compared with PCOS or menopause.

From a methodological perspective, this figure serves as a transparency tool. It reveals where evidence is most robust and where interpretation relies more heavily on mechanistic extrapolation. For educators, it reinforces evidence hierarchy: learners should understand which areas are supported by formal clinical practice guidelines [1], [5], which rely on consensus statements [6], and which depend on emerging pathophysiologic models [3], [11].

In Latin American training environments—such as Mexico, Colombia, and Ecuador—this distribution has additional pedagogical implications. Given the rising prevalence of obesity and metabolic syndrome in these regions [9], the weight of evidence in PCOS, menopause, and cardiometabolic clusters becomes particularly relevant. It encourages clinicians to view common gynecologic presentations within a preventive cardiometabolic framework rather than as isolated specialty concerns.

Figure 3.
Lifespan-stage vs mechanism evidence map

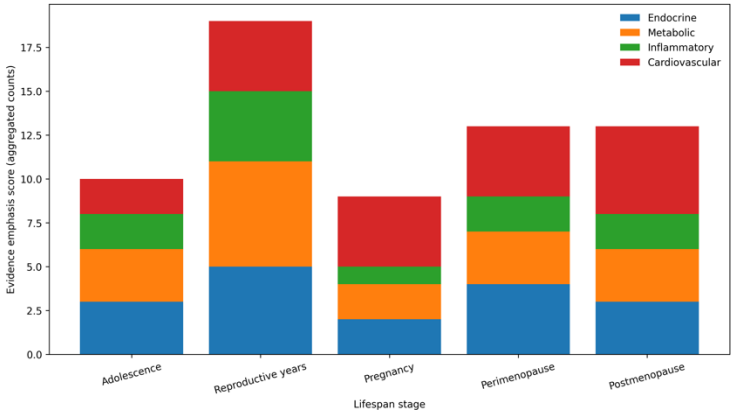

Figure 3 integrates the two structural axes that define this review: the **temporal axis of the female lifespan** and the **biological mechanistic axis** (endocrine, metabolic, inflammatory, and cardiovascular domains). The stacked configuration visually emphasizes that gynecologic health is not compartmentalized by age or mechanism; instead, it reflects shifting biological dominance across developmental stages.

In **adolescence**, endocrine and metabolic mechanisms are foundational. Early menstrual irregularities, emerging hyperandrogenism, and metabolic patterns may signal evolving PCOS phenotypes [1], [17]. Insulin resistance during this stage is not merely a metabolic parameter; it directly influences ovarian steroidogenesis and androgen

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bioavailability [1], [12]. The evidence weight in endocrine–metabolic domains at this stage reflects the formative nature of hypothalamic–pituitary–ovarian axis maturation and adiposity-related modulation of hormonal balance.

During the **reproductive years**, the figure shows broader distribution across all four mechanistic categories. This reflects the multidimensional nature of PCOS, endometriosis, and fibroids. PCOS embodies endocrine–metabolic coupling with established cardiometabolic implications [2], [17]. Endometriosis illustrates inflammatory–endocrine cross-talk, where immune activation influences reproductive function and potentially systemic physiology [3], [11]. Fibroids, though localized in the uterus, correlate with systemic metabolic risk profiles, including hypertension and obesity [10]. Thus, the reproductive years represent the stage of maximal mechanistic convergence.

The **pregnancy stage** demonstrates a relative amplification of cardiovascular representation. This is consistent with the literature describing hypertensive disorders of pregnancy as predictors of future cardiovascular disease [18]. Pregnancy functions as a hemodynamic and metabolic stress test. Endothelial adaptability—or lack thereof—becomes clinically apparent, revealing predisposition to later-life vascular pathology. This is aligned with broader models of cardiovascular risk accumulation across decades [13].

In **perimenopause**, endocrine transitions become particularly salient. Fluctuating estradiol levels influence lipid metabolism, fat redistribution, and vascular reactivity [14]. However, cardiovascular mechanisms begin to increase proportionally, reflecting the transitional amplification of underlying metabolic trajectories. This aligns with data linking menopause with rising cardiovascular incidence [4], [13].

Finally, in **postmenopause**, cardiovascular and metabolic mechanisms assume dominant representation. The decline in estrogen removes a modulatory influence on vascular function, contributing to endothelial dysfunction and dyslipidemia [4]. Importantly, early or premature menopause intensifies this trajectory, conferring increased long-term morbidity risk [7]. The figure therefore visualizes how endocrine decline interacts with preexisting metabolic burden rather than acting as an isolated cause.

From a systems perspective, the stacked model demonstrates a longitudinal redistribution of mechanistic influence:

- Endocrine dominance early in life.
- Metabolic–inflammatory convergence during reproductive years.
- Cardiovascular amplification during and after menopausal transition.

Figure 4.
Conceptual cardiometabolic risk trajectory across the female lifespan

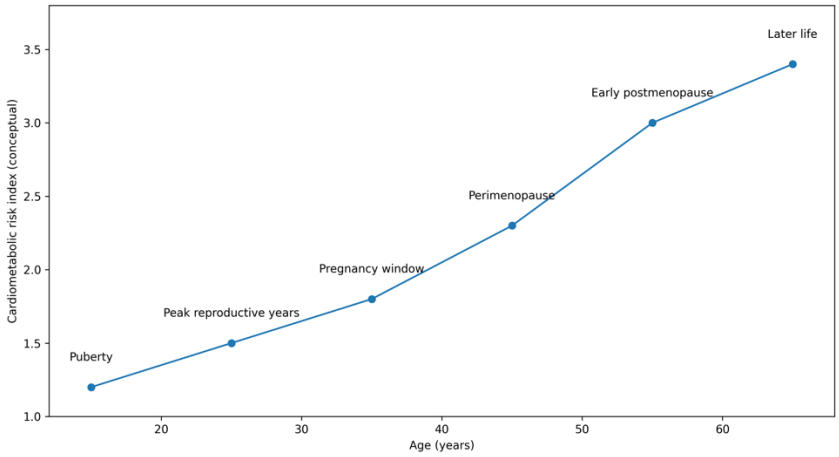

Figure 4 presents a conceptual trajectory illustrating how cardiometabolic risk evolves progressively across the female lifespan, with identifiable inflection points corresponding to reproductive and endocrine milestones. This curve synthesizes epidemiologic and mechanistic evidence linking hormonal transitions with cumulative metabolic exposure.

The early phase, beginning in **adolescence**, reflects relatively lower absolute cardiometabolic burden. However, this stage is not physiologically neutral. Emerging endocrine irregularities—such as early manifestations of

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hyperandrogenism or insulin resistance in PCOS—may establish the metabolic substrate for later disease progression [1], [2], [17]. Although clinical cardiovascular events are rare in this stage, metabolic patterns are already being shaped.

During the **peak reproductive years**, the curve shows gradual elevation. This reflects the interaction between reproductive disorders and metabolic physiology. PCOS-related insulin resistance, obesity-associated inflammatory tone, and dyslipidemic profiles contribute to risk accumulation [2], [8], [11]. Importantly, these processes may be subclinical yet biologically significant. The absence of overt cardiovascular disease does not imply absence of vascular remodeling or endothelial stress [13].

The **pregnancy window** represents a clinically meaningful checkpoint. Hypertensive disorders of pregnancy have been consistently associated with later cardiovascular morbidity, suggesting that pregnancy acts as a physiological stress test revealing underlying endothelial vulnerability [18]. In this stage, the trajectory does not sharply spike but demonstrates measurable acceleration consistent with vascular strain models.

The most prominent inflection occurs during **perimenopause**, when fluctuating estrogen levels influence lipid metabolism, visceral adiposity distribution, insulin sensitivity, and vascular reactivity [14]. Estrogen's modulatory role in endothelial function becomes increasingly evident during this transition [4]. As estradiol declines, protective vascular effects diminish, and previously accumulated metabolic exposures exert greater clinical expression. This stage marks the transition from relative risk stability to amplified cardiovascular vulnerability.

In **postmenopause**, the curve demonstrates steeper progression. The combination of endocrine withdrawal and long-standing metabolic risk factors results in higher incidence of hypertension, atherosclerotic disease, and dyslipidemia [4], [13]. Women experiencing premature or early menopause may demonstrate even earlier trajectory acceleration, supporting data linking shortened estrogen exposure with adverse long-term outcomes [7].

This figure reinforces three clinically important interpretations:

1. **Risk is cumulative rather than episodic.** Endocrine events modulate—but do not create in isolation—cardiometabolic vulnerability.
2. **Reproductive milestones provide predictive insight.** PCOS, pregnancy-related hypertension, and early menopause function as clinical markers within a continuous risk curve [2], [18].
3. **Menopause amplifies preexisting trajectories.** Cardiovascular risk rise in midlife reflects interaction between endocrine decline and decades of metabolic exposure [4], [13].

For medical education, this trajectory model encourages preventive reasoning. Instead of addressing gynecologic conditions as isolated specialty concerns, clinicians are guided to integrate reproductive history into long-term cardiometabolic assessment. This approach is particularly relevant in healthcare systems confronting rising obesity and metabolic syndrome prevalence, as seen globally [8], [9].

DISCUSSION

The present integrative review advances a lifespan-centered interpretation of gynecologic health, positioning reproductive conditions not as isolated clinical entities but as biologically interconnected expressions of endocrine, metabolic, inflammatory, and cardiovascular processes. The evidence synthesized across guidelines, epidemiologic studies, and mechanistic analyses supports the central premise that reproductive milestones can function as early indicators of long-term systemic risk [1]–[4], [8], [13], [18].

1. Reframing Gynecology Within Systems Medicine

A major implication of the findings is conceptual: gynecology must be understood within the broader architecture of systems medicine. PCOS, for example, is no longer confined to ovulatory dysfunction or hyperandrogenism; it is a metabolic phenotype with reproductive expression and documented cardiometabolic implications [1], [2], [17]. The persistence of insulin resistance and dyslipidemia across decades suggests that early endocrine abnormalities may predispose women to later vascular disease, even if reproductive symptoms improve over time.

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Similarly, the inflammatory framework associated with endometriosis expands its relevance beyond pelvic pain and infertility. Chronic inflammatory activation may influence systemic metabolic pathways and immune-endocrine regulation, reinforcing that reproductive inflammatory disease cannot be entirely compartmentalized [3], [11]. This supports a broader clinical reasoning model in which inflammation acts as a shared biological bridge between gynecologic pathology and systemic disease.

2. Pregnancy as a Prognostic Window

One of the most clinically significant themes emerging from the evidence is the recognition of pregnancy as a cardiovascular "stress test." Hypertensive disorders of pregnancy are consistently associated with elevated long-term cardiovascular risk [18]. Rather than treating gestational hypertension or preeclampsia as time-limited obstetric events, the data suggest that these complications reveal underlying endothelial vulnerability.

This aligns with broader cardiovascular risk models in women, which emphasize that risk factor trajectories accumulate gradually and may become clinically apparent only during periods of physiological stress [13]. Integrating obstetric history into lifelong cardiovascular surveillance therefore represents a rational preventive strategy, particularly in resource-variable settings.

3. Menopause as an Inflection Point, Not an Origin

The menopausal transition occupies a central position in the lifespan model. Estrogen decline alters lipid metabolism, body fat distribution, vascular tone, and endothelial responsiveness [4], [14]. However, the evidence suggests that menopause amplifies preexisting metabolic patterns rather than initiating risk de novo. Cardiovascular incidence rises after menopause, but this increase reflects interaction between hormonal withdrawal and cumulative metabolic exposure [4], [13].

This interpretation has implications for hormone therapy decision-making. Contemporary guidance emphasizes individualized risk assessment, timing of initiation, and symptom burden when considering systemic hormone therapy [5], [16]. Importantly, premature or early menopause is associated with heightened long-term morbidity risk, reinforcing the importance of stratified follow-up in these populations [7].

The global consensus on testosterone therapy further illustrates the need for precision in endocrine interventions, emphasizing appropriate indications and monitoring [6]. Collectively, these therapeutic discussions underscore the broader theme of this review: endocrine interventions must be contextualized within systemic risk frameworks.

4. Metabolic Syndrome and Global Health Context

The rising global prevalence of obesity and metabolic syndrome significantly influences reproductive health [8], [9]. Adiposity, insulin resistance, and chronic low-grade inflammation affect ovarian function, pregnancy outcomes, and menopausal cardiometabolic trajectories. In many Latin American regions, including Mexico, Colombia, and Ecuador, increasing metabolic burden intensifies the need for integrated preventive models.

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The evidence linking metabolic syndrome with reproductive endocrinology supports a bidirectional relationship: reproductive disorders may signal metabolic risk, and metabolic dysregulation may worsen gynecologic outcomes [8], [2]. This reinforces the necessity of interdisciplinary collaboration between gynecology, endocrinology, cardiology, and primary care.

5. Lifespan Integration: Clinical and Educational Implications

The four figures presented in the Results section collectively support a unified conceptual model:

- The evidence selection flow demonstrates methodological rigor and transparency.
- The thematic distribution highlights where guideline-level support is strongest.
- The lifespan–mechanism map illustrates shifting biological dominance across stages.
- The cardiometabolic trajectory model visualizes cumulative risk progression.

Taken together, these outputs substantiate the hypothesis that gynecologic health reflects a dynamic continuum rather than episodic disease clusters.

For medical education, this integrated approach promotes longitudinal reasoning. Learners should be trained to ask:

- Does this gynecologic presentation signal underlying metabolic vulnerability?
- What does this reproductive milestone imply for long-term cardiovascular risk?
- How should endocrine therapy be individualized within systemic risk profiles?

These questions align with modern preventive medicine and evidence-based practice.

6. Strengths and Conceptual Contributions

A primary strength of this review lies in its integrative structure. By organizing evidence across both temporal (lifespan) and mechanistic (biological axis) dimensions, it moves beyond condition-specific analysis toward systems-level synthesis. This dual-axis approach clarifies how endocrine transitions interact with metabolic and vascular biology at different life stages.

Another strength is the reliance on high-impact clinical guidelines and consensus statements [1], [5], [6], [16], ensuring that the conceptual model remains anchored in authoritative evidence.

7. Limitations

As an integrative narrative review, this work does not perform pooled quantitative meta-analysis. The synthesis is conceptual and thematic rather than inferential. While grounded in established evidence, mechanistic integration may require further prospective validation in longitudinal cohort studies.

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Additionally, although the framework is internationally applicable, variations in healthcare access and socioeconomic determinants may influence implementation across regions.

8. Future Directions

Future research should explore:

- Longitudinal cohort studies integrating reproductive history with cardiometabolic biomarkers.
- Region-specific epidemiologic analyses in Latin American populations.
- Educational interventions measuring whether lifespan-integrated teaching improves preventive clinical practice.
- Translational research clarifying inflammatory–endocrine interactions in conditions such as endometriosis and PCOS.

CONCLUSION

This integrative review supports a lifespan-centered interpretation of gynecologic health, demonstrating that reproductive conditions are deeply interconnected with metabolic regulation, inflammatory signaling, and cardiovascular adaptation. The accumulated evidence from clinical guidelines, epidemiologic analyses, and mechanistic syntheses consistently indicates that gynecologic events—such as polycystic ovary syndrome, endometriosis, uterine fibroids, hypertensive disorders of pregnancy, and menopause—should not be understood as isolated phenomena, but rather as clinical expressions of broader systemic physiology [1]–[4], [8], [13], [18].

Polycystic ovary syndrome exemplifies how endocrine imbalance and insulin resistance can converge to produce both reproductive dysfunction and long-term cardiometabolic vulnerability [1], [2], [17]. Endometriosis highlights the centrality of chronic inflammatory pathways and immune–endocrine interactions in shaping reproductive and potentially systemic outcomes [3], [11]. Pregnancy-related hypertensive disorders function as prognostic markers of future cardiovascular risk, reinforcing the concept of pregnancy as a physiological stress test with long-term implications [18]. The menopausal transition represents a pivotal endocrine inflection point that amplifies preexisting metabolic trajectories and contributes to increased vascular risk in later life [4], [7], [14].

Across these conditions, inflammation emerges as a shared biological mediator, linking adiposity, insulin resistance, ovarian steroidogenesis, and endothelial function [11], [12]. The rise in global obesity and metabolic syndrome further intensifies the systemic dimension of gynecologic practice, particularly in healthcare systems facing increasing cardiometabolic burden [8], [9]. Thus, reproductive milestones offer strategic windows for early detection, prevention, and longitudinal risk stratification.

From an educational perspective, the lifespan–mechanism framework proposed in this review strengthens clinical reasoning by encouraging learners to interpret gynecologic presentations within a systemic context. Rather than episodic care models, this approach promotes longitudinal surveillance, interdisciplinary collaboration, and individualized endocrine decision-making aligned with contemporary consensus guidance [5], [6], [16].

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In summary, gynecologic health across the lifespan reflects a dynamic and cumulative interaction between endocrine transitions, metabolic exposure, inflammatory activity, and vascular adaptation. Recognizing these interconnections allows clinicians to transform reproductive events into preventive opportunities, reinforcing the role of gynecology as a central pillar of long-term systemic health.

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