White Paper

NYU Langone Health

SCITARIS
BIOPHARMA STRATEGY

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Women's Health

Introduction

For most of the history of modern drug development, the reference human was male. That choice was driven by a multitude of factors, including a cautious approach to female participation in clinical trials, the perceived inconvenience of hormonal variability in female subjects, and the under-representation of women in medicine, science, industry, and investment. This has had profound consequences for every layer of the drug development pipeline. In cardiovascular disease (CVD), which is the leading cause of death in women globally, clinical standards remain calibrated to male physiology. In autoimmunity, most research frameworks and treatment approaches are still largely sex neutral. Female-specific conditions such as endometriosis and polyendocrine metabolic ovarian syndrome (PMOS; previously known as polycystic ovary syndrome or PCOS) affect tens of millions of women but carry only meager pipelines that resemble those of rare diseases. More broadly, pipelines across female-specific diseases remain markedly underdeveloped compared with other therapeutic areas. This is compounded by a striking funding gap: women's health accounts for just ~6 percent of global private healthcare investment, and dedicated companies capture less than 1 percent of funding.[1] The consequences of this underinvestment and underrepresentation are substantial, but so is the opportunity. Women's health represents one of the largest untapped markets in modern medicine. Encouragingly, growing recognition of these disparities is driving a global shift toward more inclusive research, accompanied by increasing interest and investment in the field.

In the following paper, we focus on the data gap and its downstream consequences for drug development; provide a snapshot of the therapeutic landscape across several conditions spanning the female lifespan from reproductive years through menopause (Figure 1); and summarize the key players and events in the deal investment.

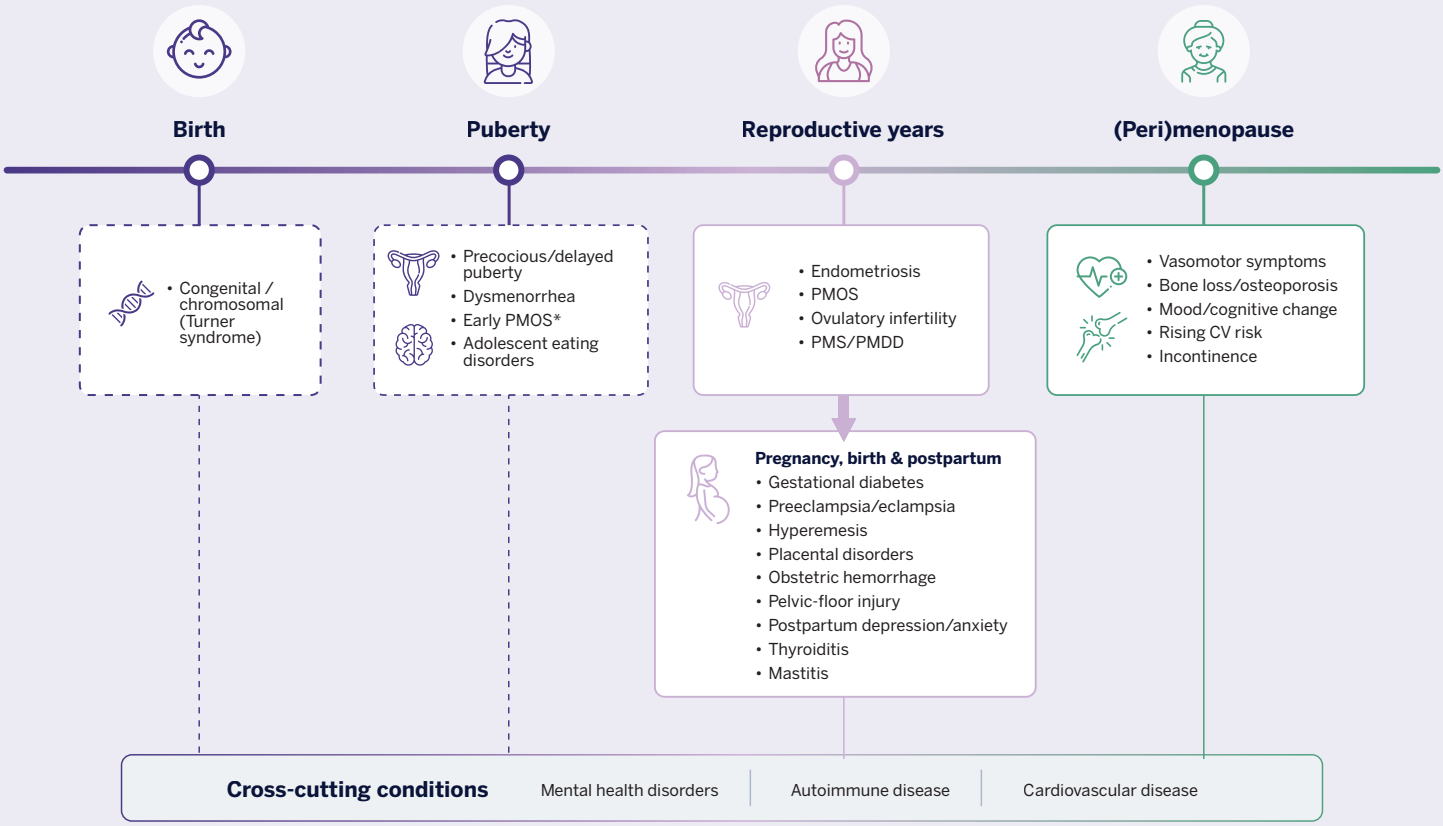


Figure 1. Conditions and Diseases Across the Female Lifespan.
Key gynecological, reproductive and hormonal conditions are mapped across four life stages: birth, puberty, reproductive years and (peri)menopause. Solid boxes indicate conditions within the scope of the present analysis. Dashed boxes indicate conditions considered out of scope. Cross-cutting conditions including mental health disorders, autoimmune disease and cardiovascular disease that span multiple life stages and are represented as a continuous bar across the lifespan. Pregnancy, birth, and postpartum conditions are shown as a distinct branch of the reproductive years. Arrows indicate the primary life stage at which each condition cluster typically manifests or becomes clinically relevant.

CV(D): cardiovascular disease, **PMDD:** premenstrual dysphoric disorder, **PMOS:** polyendocrine metabolic ovarian syndrome (previously PCOS), **PMS:** premenstrual syndrome

The Data Gap and Its Consequences

The Preclinical Blind Spot

The evidence imbalance that disadvantages women begins long before the first patient is enrolled. For decades, the male animal served as the default experimental subject because females were assumed to introduce excessive variability through hormonal cycling. Researchers believed that reliable female data required testing across all four estrous-cycle stages, increasing cost and complexity.[2] The assumption was intuitive, widely held, and wrong. It went essentially untested until a 2014 meta-analysis of 293 articles spanning roughly 10,000 biological traits found that randomly cycling female mice were no more variable than males on any measure. In fact, males were substantially more variable on several traits, an effect compounded by group housing and dominance behavior.[3] The finding was subsequently reproduced in rats and in gene-expression datasets.[4] The deeper issue was methodological asymmetry. Researchers routinely controlled for female hormonal fluctuations but rarely accounted for comparable sources of male variability, such as testosterone changes or social hierarchy effects. A concern applied to one sex but not the other, based on an assumption later shown to be false, is difficult to justify on purely scientific grounds.

In response, the National Institutes of Health (NIH) introduced its Sex as a Biological Variable (SABV) policy in 2016. The policy requires investigators to consider sex in the design, analysis, and reporting of animal and human studies. It also requires them to justify any single-sex design.[5] A decade later, inclusion has improved more than analysis. A 2026 study of 574 NIH-funded papers published between 2017 and 2024 found that, although 61 percent included both sexes as subjects, only 44 percent reported their results by sex.[6] The trend is, if anything, regressive. Among studies including both sexes, the share performing sex-disaggregated analysis fell to 42 percent in 2019 versus 50 percent in 2009.[7] In effect, SABV has not

solved the analysis part. Sex is present in experiments yet often absent from the results. That gap is now also politically exposed with NIH guidance on sex and gender removed and only partially reinstated through 2025–2026, underscoring how fragile the policy scaffolding remains.[8] The repercussions do not stay in the laboratory. A male-derived understanding of mechanism, dosing, and toxicity is built into the evidence base and carried directly into human trials.

The most significant preclinical advance may therefore be the shift away from traditional animal models. Many female-specific conditions are poorly represented in rodents, which do not menstruate and, for example, do not spontaneously develop endometriosis. Patient-derived organoids and ex vivo human-tissue systems are beginning to close this translational gap. GynQura Therapeutics has developed a biobank of tissue from more than 200 patients and over 60 endometriosis organoid lines designed to identify non-hormonal therapeutic targets.[9,10] Metri Bio is building 3D endometrial organoid models based on patient-derived samples to overcome the limitations of animal systems, an enabling step the field arguably needs before rational target discovery can replace repurposing.[11] Notably, rather than just modeling ectopic lesion biology, Metri Bio's platform was initially developed to target the implantation failure that affects an estimated 30–50 percent of endometriosis patients.[12] Similarly, San Francisco-based startup Opal Therapeutics uses menstrual blood, tissue biopsies, and advanced artificial intelligence (AI)-driven analysis to create a "uterus-in-a-dish" that aims to capture patient-specific disease phenotypes of endometriosis and other gynecological disorders.[13,14] Human-tissue platforms offer a route to generating the missing evidence without relying on limitations of animal models in the first place.

Clinical Trials: Representation and Reporting

The exclusion of women from human research has explicit regulatory roots. In the wake of the thalidomide tragedy, the Food and Drug Administration (FDA) in 1977 advised against enrolling women of childbearing potential in early-phase trials. This precaution hardened into routine exclusion and left the female body largely unstudied for a generation. The correction came in stages. The NIH began recommending the inclusion of women in the late 1980s. Female enrollment did not climb until 1993, when the agency's Revitalization Act made inclusion a matter of federal law. More recent measures have pushed further; the Food and Drug Omnibus Reform Act (FDORA) of 2022 required sponsors to file Diversity Action Plans, which set enrollment goals for underrepresented groups (i.e., sex, race, ethnicity, age), and in 2024, the FDA issued draft guidance reinforcing equitable representation in drug research.[15]

These reforms worked, but unevenly. A 2025 analysis of Phase III interventional trials registered on ClinicalTrials.gov between 2000 and 2024 found that the great majority of non-sex-specific conditions now enroll both sexes.[16] The shortfall remains upstream in Phase I, where dosing and tolerability are first established. Only 30.6 percent of Phase I participants are women, whereas 34.1 percent of early trials enroll men exclusively.[17] The barriers are partly structural and partly cultural. Women who wish to enroll must often prove repeatedly that they are not pregnant, protocols restrict participation based on "childbearing potential," and qualitative work reports that healthy female volunteers are perceived by research staff as more "difficult" participants because of their menstrual cycles.[18] The phase that sets the dose for everyone is thus the phase that contains the fewest women.

But fixing enrollment would solve only half the problem. Including women but pooling their results hides the very sex-specific effects that enrolling them was meant to reveal. Regulators have encouraged subgroup reporting, but the expectation is inconsistently enforced, and female subgroups are frequently underpowered, so sex differences in efficacy and toxicity continue to be discovered post factum rather than designed for prospectively. Inclusion without stratified analysis produces the appearance of representation without its evidentiary benefit.

PK Differences and Adverse Drug Reactions

The consequences of building drug development on male data are not merely theoretical. Women have historically been underrepresented in early pharmacological research. Sex differences in how drugs are absorbed, distributed, metabolized, and eliminated are therefore often identified only after approval, if they are detected at all. One study found that 76 of 86 commonly prescribed drugs exhibit sex-based pharmacokinetic (PK) differences, yet dosing recommendations frequently fail to reflect them. As a result, women experience adverse drug reactions at nearly twice the rate of men.[19] Using a machine-learning system (AwareDX) applied to the FDA Adverse Event Reporting System, the same researchers identified 20,817 previously unrecognized sex-specific drug risks, suggesting that known cases represent only a fraction of the true problem.[20]

Zolpidem (Ambien) provides perhaps the clearest illustration of this failure. A drug approved in 1992 was re-dosed for half the population two decades later because the relevant PK data had never been collected. Zolpidem's recommended dose for women was cut in half by the FDA in 2013 after sex differences in clearance and plasma concentration went unnoticed at

approval.[21] The same pattern appears in cardiovascular medicine, where the Digitalis Investigation Group (DIG) trial found increased mortality risk in women on digoxin, plausibly linked to higher serum concentrations that male-optimized dosing had failed to anticipate.[22] This is a reminder that a male-default research pipeline produces male-calibrated treatments, with women disproportionately bearing the consequences.

Conditions Across the Female Lifespan

Women's health needs and risks shift dramatically across the lifespan, yet medical attention has historically clustered around reproduction while other stages and systems have been systematically underserved. In this analysis, we take a lifespan perspective (Figure 1) and focus on conditions where therapeutic development is active, emerging, or has attracted meaningful strategic interest: PMOS, endometriosis, pregnancy complications, menopause, and the cross-cutting burden of depression, autoimmune disease, and cardiovascular disease in women. We deliberately exclude puberty and adolescence (precocious puberty, primary dysmenorrhea, early PMOS), which sit within pediatric regulatory frameworks, and female-specific cancers, which, as part of oncology, have followed a very different development and investment trajectory.

Figure 2 gives an overview of the current state of the drug pipeline. It covers female-specific conditions only, excluding CVD, autoimmune disease, and broader mental health indications. Within the female-specific scope, the marketed drug landscape is largely composed of established, off-patent agents: generics, reformulations and long-standing hormonal combinations with genuine first-in-class innovation concentrated in a small subset of recent new drugs, such as fezolinetant and elinzanetant (NK3 antagonists for vasomotor symptoms), zuranolone (GABA-A modulator for postpartum depression), and the newer gonadotropin-releasing hormone (GnRH) antagonists elagolix and linzagolix. The active clinical pipeline is thin. There are approximately 65 assets across all clinical stages, with only 11 in Phase III. The preclinical stage shows modestly greater breadth, with endometriosis, female infertility, and PMOS each carrying meaningful representation, but the volume of discontinued clinical programs substantially exceeds those currently active. This pattern reflects historical attrition rather than recent progress. In sum, the pipeline is one where mechanistic innovation has been limited, most approved therapies act on the same hormonal axes developed decades ago, and the gap between preclinical activity and clinical proof of concept remains wide.

Reproductive Years

Women's reproductive years have been the historical focal point of "women's health." Nonetheless, substantial gaps remain in the recognition and treatment of major gynecological conditions. Two illustrate this interplay of high burden and limited progress particularly clearly: PMOS and endometriosis.

PMOS

Until recently known as polycystic ovary syndrome (PCOS), PMOS epitomizes how a woman's health condition can be common yet suffer from fundamental misunderstanding. In 2026, a global expert panel formally adopted PMOS as the new name. The previous name fixated on ovarian cysts and neglected the broader multi-hormonal, metabolic nature of the syndrome.[23] Affecting 170 million women, PMOS is among the most prevalent female health disorders. It is diagnosed when at least two of three key features are present: (1) ovulatory dysfunction (oligo- or anovulation), (2) hyperandrogenism, and (3) polycystic ovarian morphology.[24,25]

PMOS pathophysiology is complex. The interconnection of hyperandrogenism, insulin resistance, and inflammation create self-reinforcing loops that cannot be addressed as separate problems. However, current standard treatments do exactly that. Oral contraceptive pills help regulate menstrual cycles and reduce excess hair/acne by suppressing ovarian androgens, insulin sensitizers like metformin tackle metabolic symptoms, and ovulation-induction drugs help fertility by forcing ovulation. Each therapy acts on one downstream symptom, and none are disease-modifying or curative.

Given PMOS's prevalence and impact, why has mechanistic understanding progressed so slowly? A major reason is the lack of robust preclinical models. Existing animal models capture only fragments of the phenotype, but none reproduce the full human condition. Key differences in rodent and human reproductive physiology further limit translation.[26] As a result, no model reliably predicts disease-modifying interventions, constraining drug discovery to symptom management. As discussed in the above sections, emerging human tissue and organoid platforms aim to close this gap and may eventually enable PMOS research to move from description toward therapies that alter the underlying disease.

Today's PMOS drug pipeline reflects the same gaps. Like the approved drugs, most research and development has targeted individual facets of the syndrome. GLP-1 receptor agonists (GLP-1 RAs) arguably come closest to a multi-faceted approach, given their combined metabolic and potential reproductive benefits.[27] Trials in overweight patients show meaningful improvements in weight and insulin sensitivity, though evidence for reproductive benefits remains mixed.[28,29] The more consequential question is whether these effects extend to lean PMOS patients (20–50 percent of cases), who exhibit insulin resistance independent of adiposity.[30] These patients are largely excluded from existing trials, leaving the

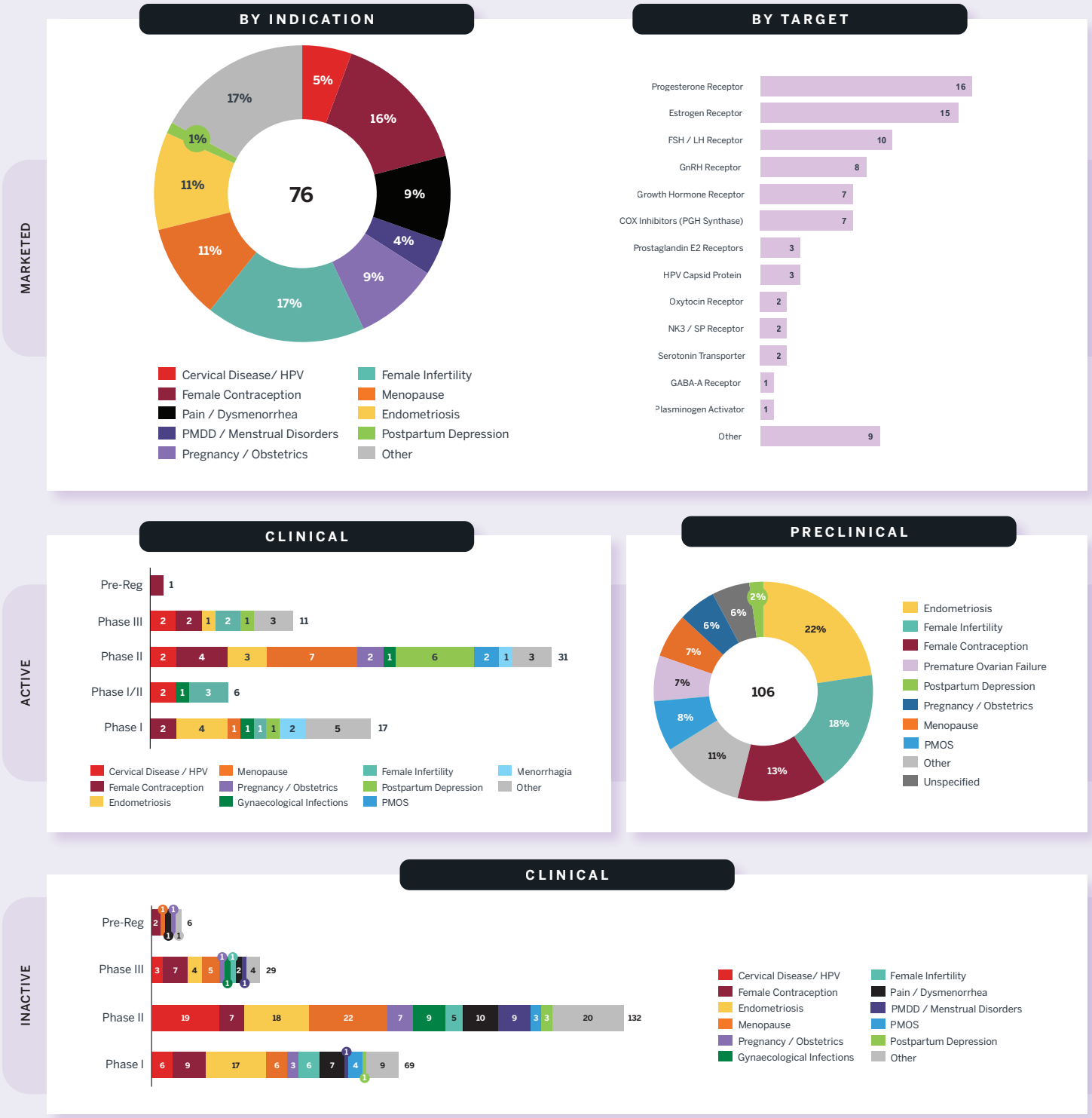


Figure 2. Developmental Pipeline in Select Indications in Women's Health
Pipeline data are sourced from GlobalData for development across the eight major markets (8MM) including China, filtered by therapeutic area (Women's Health). Only select indications are shown. The analysis captures drugs classified under the Women's Health therapeutic area descriptor and excludes assets characterised under broader parental indications such as cardiovascular disease, autoimmune disorders, osteoporosis or general mental health that fall outside GlobalData's Women's Health classification. Drugs investigated across multiple indications or acting via multiple targets, are counted once per relevant category, so total assignments may exceed total drug counts. Marketed and pre-registration stage refers to the US and Europe. Pipeline data are current as of June 2026.

Within each panel, the "Other" category includes (but is not limited to): **Marketed —** vaginal atrophy, HPV, uterine fibroids, growth disorder; **Active clinical and PreC —** PMDD, menstrual cramps, ovarian hyperstimulation syndrome, dysmenorrhea, hypothalamic amenorrhea, female sexual dysfunction, growth disorders; **Inactive clinical —** female sexual dysfunction, uterine fibroids, premature ovarian failure, major depressive disorder, miscarriage, growth disorders.

PreC: preclinical, **Pre-Reg:** pre-registration, **GnRH:** gonadotropin-releasing hormone, **FSH:** follicle-stimulating hormone, **LH:** luteinising hormone, **COX:** cyclooxygenase (prostaglandin G/H synthase 1/2), **NSAID:** non-steroidal anti-inflammatory drug, **HPV:** human papillomavirus, **PMDD:** premenstrual dysphoric disorder, **PMOS:** polyendocrine metabolic ovarian syndrome, **8MM:** eight major markets (USA, Germany, France, Italy, Spain, UK, Japan and China).

weight-independent effects of GLP-1 RAs unresolved, and with them, whether any existing therapy can correct the core multi-axis pathology of PMOS rather than managing individual symptoms.

Some ventures are also exploring less validated therapeutics modalities, such as microbiome modulation. Both gut and vaginal microbiomes appear altered in PMOS, and pilot studies suggest that normalizing gut flora can improve some PMOS metabolic and reproductive parameters.[31,32] This has sparked interest in microbiome-targeted therapies to break the inflammation–insulin–androgen feedback loop. A New York-based startup Evvy, for example, has built "the world's largest dataset on the vaginal microbiome" and is employing AI-driven analysis to find microbial biomarkers for conditions like PMOS.[33,34] Though translating such findings into therapy is still a distant prospect.

On the therapeutics front, the development in PMOS is still extremely scarce. A notable exit was Celmatix Therapeutics, which launched a program in 2023 to develop a peripherally acting melatonin receptor agonist as a novel therapy for PMOS, based on genomic links between melatonin signaling and ovarian function.[35] In March 2026, Celmatix's pipeline was acquired by Gedeon Richter, and the headline assets were in fertility and endometriosis (a FSH receptor agonist and a JNK inhibitor). Unfortunately, their PMOS-focused program apparently did not move forward as a priority.[36] Sena Therapeutics[37] is developing KISS1R binding peptide-derived molecules to restore hypothalamic-pituitary-gonadal (HPG) axis signaling upstream of androgen excess—one of the very few programs targeting PMOS biology rather than its downstream symptoms.

In short, the name change to PMOS is a positive step, but scientific and clinical progress are lagging. Women with PMOS must navigate a patchwork of partial solutions. For PMOS to truly move from a "managed" condition to one that can be cured or prevented, a more holistic research and development approach is needed. One that integrates multi-system biology, embraces patient heterogeneity, and targets upstream drivers rather than just downstream symptoms.

Endometriosis

This condition affects ~10 percent of women of reproductive age. It is defined by the presence of endometrial-like tissue outside the uterus, mostly on the ovaries, fallopian tubes, and pelvic peritoneum. Its clinical symptoms include severe dysmenorrhea, chronic pelvic pain, dyspareunia, and infertility (up to 30–50 percent of patients). Despite its prevalence, for many years, definitive diagnosis required laparoscopy, resulting in an average 7–10-year delay from symptom onset to diagnosis. In March, the American College of Obstetricians & Gynecologists released a guideline with recommendations on the clinical, imaging, and surgical evaluation of endometriosis that could allow diagnosis on the basis of symptoms, but a non-invasive biomarker has yet to achieve broad regulatory acceptance.[38–40]

Initial treatment of endometriosis typically includes non-steroidal anti-inflammatory drugs (NSAIDs) and hormonal therapy, usually estrogen-progestin or progestin-only contraceptives. Patients with an inadequate response may use alternative progestins, GnRH analogs, or an androgen. Both GnRH agonists and antagonists are used, each creating a hypoestrogenic state. Unlike GnRH agonists, antagonists are dosed orally and do not cause an initial surge in luteinizing and follicle-stimulating hormone.[41]

The pipeline has only recently begun to escape the paradigm of hormonal therapy. HMI-115, a prolactin-receptor monoclonal antibody (Hope Medicine), entered global Phase III in 2026. By operating independently of the Hypothalamic-Pituitary-Ovarian (HPO) axis, it targets intra-lesion inflammation rather than systemic estrogen suppression.[42] Phase II data showed clinically meaningful reductions in both dysmenorrhea and non-menstrual pelvic pain without menopausal effects.[43] If confirmed, HMI-115 would define a new treatment class for endometriosis. Based on GlobalData, the anticipated approval of HMI-115, as well as the commercial success of GnRH antagonists, are expected to be the primary drivers of the endometriosis market, with predicted market growth from $0.9 billion in 2024 to $2.5 billion by 2034.[44]

Earlier-stage non-hormonal programs illustrate promise, but also fragility. Organon, a spinout of Merck AG, was developing OG-6219, a small-molecule inhibitor of HSD17B1, a critical enzyme catalyzing the final step in estradiol biosynthesis.[45] It failed in Phase II in 2025, failing to demonstrate improvement in moderate-to-severe endometriosis-related overall pelvic pain compared to placebo.[46] Other companies worth noting include Gesynta Pharma with vipoglanstat (a small-molecule mPGES-1 inhibitor) that entered Phase II in 2026, targeting local prostaglandin E2 (PGE2)-driven inflammation to reduce endometriosis-related pain with endometriotic lesions evaluated as an exploratory endpoint. Originally developed for systemic sclerosis-associated Raynaud's phenomenon, where it failed to demonstrate symptomatic benefit despite confirmed target engagement, vipoglanstat's pivot to endometriosis is nonetheless mechanistically reasoned. mPGES-1 is upregulated in ectopic lesions, and PGE2 is a documented driver of lesion survival and pain sensitization.[47] However, true validation is contingent on the NOVA trial readout, with topline results expected in 2027.[48] Another example is Maipl Therapeutics advancing MA-4604, an oral prostaglandin F2α receptor antagonist in-licensed from Ferring.

This May, they reported encouraging preclinical activity across multiple translational models: uterine contractility, pain reduction, and disease modification.[49] Maipl is planning the initiation of first-in-human clinical studies in 2027.

What the entire pipeline has in common is that it targets consequences rather than causes, similarly to PMOS. GnRH antagonists suppress pain-driving estrogen, surgical excision removes lesion burden and restores pelvic anatomy, and prostaglandin-axis inhibitors reduce the inflammatory signal. None address the full biology of a disease. The question of whether removing endometriosis and restoring hormonal physiology is sufficient to reverse its effects, particularly on fertility, has a largely sobering answer. All currently available medical treatments have not been shown to substantially improve natural fertility, and the 2022 ESHRE guidelines explicitly recommend against hormonal suppression as a fertility restoration strategy.[50] Surgery may help structurally but introduces its own damage and carries an estimated recurrence rate of 20–30 percent within 1–2 years.[51] The question is whether a critical variable in endometriosis may also be the timing of intervention. If this is true, it would reframe the diagnostic gap not merely as a care delivery failure but as a biological urgency: the years between disease onset and confirmed diagnosis may be precisely the window in which the most consequential and irreversible damage accumulates.

Pregnancy and the Postpartum

Pregnancy represents a unique and high-risk physiological state, involving profound cardiovascular, metabolic, immune, and endocrine adaptations. These changes expose women to pregnancy-specific diseases for which therapeutic development has historically lagged. Not primarily due to scientific difficulty, but because pregnant women have been systematically excluded from clinical trials by default, producing a pharmacological gap.

Preeclampsia

This life-threatening complication of pregnancy—marked by hypertension and multi-organ dysfunction arising after 20 weeks' gestation—is illustrative of the problem. It originates from placental dysfunction: a stressed placenta secretes soluble fms-like tyrosine kinase 1 (sFlt-1) in excess into the maternal circulation, triggering vascular inflammation and the clinical syndrome of preeclampsia.[52] Delivery remains the only cure, frequently forcing premature birth to save the mother. Standard management is supportive: blood pressure control, seizure prophylaxis and surveillance until delivery is unavoidable. No approved drug addresses the underlying cause, but that is beginning to change. A pilot trial using apheresis to filter sFlt-1 directly from maternal blood demonstrated in 16 women with severe preterm preeclampsia that sFlt-1 removal modestly lowered blood pressure and prolonged pregnancy by approximately ten days, giving fetuses valuable extra development time.[53,54] Comanche Biopharma's CBP-4888, a siRNA engineered to accumulate in the placenta and silence sFlt-1 production at source, entered a Phase I trial in 2025, representing the first pharmacological attempt to address the root molecular cause of the disease.[55] DiaMedica's DM199 (rinvecalinase alfa), a recombinant human tissue kallikrein-1 that restores vasodilatory nitric oxide and prostacyclin to improve placental perfusion, showed reduced maternal blood pressure and improved uterine artery flow in interim Phase II data without fetal transfer.[56] These programs collectively signal a shift from managing preeclampsia's consequences to targeting its biology; a transition that has taken decades too long.

The postpartum period

Intensive antenatal monitoring drops sharply after childbirth, when half of maternal deaths occur after 42 days postpartum, a point at which women have already left obstetrical care.[57,58] This postpartum gap leaves women vulnerable to severe complications such as postpartum hemorrhage, postpartum preeclampsia, and peripartum cardiomyopathy.[59] The gap is equally evident in maternal mental health. For example, postpartum depression (PPD) affects roughly one in eight new mothers.[60] Yet historically, postpartum mental health support has been minimal. Here, too, innovation offers hope: the FDA approved brexanolone and zuranolone as the first medications specifically for PPD, and more PPD-targeted therapies are in development (Figure 2). One example is Reunion Neurosciences, which is developing psycobilin-derived therapeutics for underserved mental health disorders, including moderate-to-severe PPD, planning to initiate Phase III in 2026.[61]

Menopause and Post-Reproductive Health

If reproduction has been the focal point of women's health, the decades that follow it have been its blind spot. The menopausal transition is near-universal, and its symptoms are common; an estimated 60–80 percent of women experience vasomotor symptoms such as hot flashes and night sweats. However, it has historically been framed as a quality-of-life nuisance rather than a disease state with measurable long-term consequences.[62,63] That framing has real costs. The loss of estrogen at menopause accelerates bone loss and reshapes cardiovascular risk. Osteoporosis is the clearest example, a disease that is largely post-menopausal. The same hormonal transition that produces hot flashes also drives the decline in bone density that culminates years later in fragility fractures, linking a symptom dismissed as transient to a major source of late-life morbidity.

Therapeutic progress has been held back as much by controversy as by biology. Hormone replacement therapy (HRT) was the mainstay until the Women's Health Initiative reported elevated risk of CVD in the early 2000s, prompting a sharp and lasting decline in use. That evidence is now being actively revisited, with attention to the age at which therapy is started,

the formulations used, and the methodological limitations of the original cohort. However, the net effect of two decades of caution has been a near-total absence of innovation for the menopausal transition itself.[64] Menopause was left past the point of clinical interest.

That is beginning to change. The vasomotor space has seen its first non-hormonal approval in decades. Fezolinetant (Veozah, Astellas), a neurokinin 3 (NK3) receptor antagonist, was approved by the FDA in May 2023 as a first-in-class agent that targets the the hypothalamic KNDy-neuron circuitry (kisspeptin, neurokinin B, and dynorphin cells) governing thermoregulation rather than supplying estrogen.[65] Its arrival validates a genuinely new mechanistic class and signals renewed commercial interest in menopause beyond HRT, an interest that the market increasingly recognizes. One analysis project that effectively treating moderate-to-severe symptoms could expand the US menopause-care market roughly eightfold.[66] The pattern nonetheless mirrors what we see throughout this analysis. A single approval after decades of neglect, addressing the most visible symptom rather than the underlying endocrine transition itself. On the fertility front, Oviva Therapeutics (acquired by Granata Bio, April 2025)[67] developed OVI-586, a recombinant anti-Mullerian Hormone (AMH) designed to moderate follicle depletion and extend ovarian reserve, targeting the hormonal collapse upstream rather than managing its consequences. This asset remains preclinical, with IVF as the near-term proof-of-concept indication and menopause delay as the longer-term goal.[68]

Osteoporosis

This is the one post-reproductive condition where therapeutic development has been genuinely productive, precisely because it was reframed as a fracture-risk disease, rather than a menopausal symptom. Moreover, it affects men as well, giving it a larger, mixed-sex market that escaped the framing of a purely female condition. The standard of care is now layered by mechanism. First-line antiresorptive therapy rests on oral and intravenous bisphosphonates (alendronate, zoledronic acid), which inhibit osteoclast activity, followed by denosumab, a monoclonal antibody against RANKL that suppresses bone resorption more potently. For patients at very high fracture risk, anabolic agents that build new bone, the parathyroid hormone (PTH) analogs teriparatide and abaloparatide, and the dual-action sclerostin inhibitor romosozumab (Evenity, approved 2019), which simultaneously increases formation and reduces resorption, are now recommended as initial therapy rather than last resort.[69,70] The frontier has shifted from single agents to sequencing them. Bone formed by an anabolic agent is lost once the drug stops, and stopping denosumab triggers a rebound of rapid bone loss and multiple vertebral fractures. As a result, the field now emphasizes starting with a bone-forming or dual-action agent and consolidating the gains with an antiresorptive.

The optimal order and duration of these transitions remain an active research question.[71,72] Novel mechanisms remain comparatively limited. Cathepsin K inhibitors (odanacatib, balicatib, ONO-5334), which block the enzyme osteoclasts use to degrade bone collagen, advanced to late-stage trials, and odanacatib reduced fractures but was withdrawn over an excess stroke signal, and the class has been slow to recover. Wnt-pathway modulators beyond sclerostin, including DKK-1 inhibitors, remain largely preclinical.[73] Boston-based Skeletalis ($8 million seed, 2025)[74] is developing a bone-targeted small-molecule platform (OASIS) that concentrates drug activity at active resorption sites while minimizing systemic exposure, directly addressing the safety trade-offs that limit current agents. Primary focus is post-menopausal osteoporosis; still preclinical. The contrast of the rich pipeline compared with the rest of post-reproductive health is striking.

Osteoporosis attracted a mechanistically diverse pipeline because it was treated as a distinct, measurable disease, with a validated surrogate endpoint (bone mineral density) and a clear outcome to prevent (fracture). That combination gave developers a clear target and regulators a clear bar. On the other hand, vasomotor symptoms, genitourinary syndrome, and the metabolic shifts of menopause still lack both. Until they get them, therapeutic development is likely to remain sparse.

Cross-Cutting Conditions

Not all vital women's health challenges are uniquely female. In fact, nearly half of the global women's health burden comes from conditions that affect women disproportionately, in which female patients experience distinct biological and clinical realities too often overlooked by male-centric research. This disconnect leaves women underdiagnosed, undertreated, or suffering excessive side effects. Women are diagnosed later than men across roughly 700 diseases, and though they live longer, they spend about 25 percent more of their lives in poor health.[75] Below, we touch on three major "cross-cutting" areas: cardiovascular disease, autoimmune disease, and mental health to highlight how sex-specific differences and data gaps constrain therapeutics, and where new innovations are beginning to make a difference.

Autoimmune diseases

These conditions collectively affect 3–10 percent of the population, and women comprise roughly 80 percent of patients.[76] In the most female-predominant conditions, the disparity is striking: Sjögren's syndrome carries a female-to-male ratio of 9–22:1, systemic lupus erythematosus (SLE) approximately 7–9:1, and Hashimoto's thyroiditis approaching 19:1, while rheumatoid arthritis (RA) and multiple sclerosis sit closer to 2–3:1[77,78]—not a subtle epidemiological pattern. Nonetheless, the therapeutic development that has followed

has been largely designed as if sex were irrelevant. Here, as in clinical trials more broadly, the problem is no longer enrollment, with most autoimmune trials now including both sexes.[16] The issue is stratification. Trials analyze results in aggregate, treating the population as biologically homogeneous. In SLE, where nine out of ten patients are female, this may seem inconsequential, but it means that female-specific biology is not properly interrogated as a variable that might explain differential response, toxicity, or disease trajectory. The problem extends to biomarkers, as they might be different for the proper diagnosis of the same disease in different sexes. In SLE serology, certain markers are more frequently detected in one sex and show strong associations with sex-biased clinical phenotypes. These associations disappear when data from both sexes are pooled.[79]

The clinical consequences are visible in the post-hoc data that does exist. A systematic review and meta-analysis of 54 randomized controlled trials in psoriatic arthritis found that only 18 reported efficacy endpoints by sex, and just two reported safety outcomes by sex. Where sex-stratified data were available, treatment response differed meaningfully by drug class, pointing to a biological rather than incidental basis. In RA, post-hoc analyses consistently show higher remission rates in men than women across drug classes. In axial spondyloarthritis, women are less likely to achieve efficacy outcomes on advanced therapies. These sex differences are real and clinically meaningful, but they keep surfacing only after the fact. The underlying data are already being collected; the failure lies in whether investigators and journals choose to report them by sex.[80]

The deeper question is whether the current model, which includes women, analyzes them together and reports aggregate results, can generate the evidence base needed to develop therapeutics that truly serve the populations most affected. Given that 80 percent of autoimmune disease patients are women, and that the biological mechanisms driving their susceptibility appear to be female-specific at a genomic level, the answer is almost certainly no. Sex-stratified trial design is not just about fair representation; it is about scientific validity.

Cardiovascular diseases

Cardiovascular diseases remain the leading cause of death in US women, and the trajectory is worsening. The American Heart Association projects that nearly 60 percent of US women could have high blood pressure by 2050, and nearly one-third of women ages 22–44 may have some form of CVD, up from less than one in four currently.[81] The sex-specific biology of CVDs is not a recent discovery. Women with type 2 diabetes carry an estimated 25–50 percent greater risk of cardiovascular events than men with the same diagnosis. Blood pressure increases more rapidly with age in women than in men, and Lp(a), a highly atherogenic lipoprotein, rises at the menopausal transition in women but not in men. Women also carry a higher baseline inflammatory burden and autoimmune diseases that confer a 56 percent higher risk of incident CVD compared with matched controls. Sex-specific risk enhancers: PMOS, endometriosis, early menopause, and infertility carry their own cardiovascular signal. Yet the full picture remains incomplete. The gaps reflect decades of underinvestment in female-specific cardiovascular biology. Moreover, risk factor profiles in women change substantially across the lifespan, patterns that current clinical frameworks are poorly equipped to track or act on.[82]

Despite the biological complexity being well established, women remain structurally underrepresented in the trials that should be generating answers. A systematic review of 1,079 cardiovascular trials registered between 2017 and 2023, covering over 1.3 million participants, found that women represented just 41% overall and only 22% of ACS trial participants, despite accounting for 34% of acute coronary syndrome (ACS) prevalence. Female-to-male enrolment ratios were lowest in the indications where sex-divergent biology is most pronounced: ACS (0.32), coronary heart disease (0.39), and heart failure (0.51). No significant improvement in overall representation was observed across the study period, and industry-sponsored trials enrolled fewer women than academically led studies.[83]

Mental health

Major depressive disorder (MDD) exemplifies how sex disparities in data and biology hinder innovation in mental health. MDD is nearly twice as prevalent in women as in men, and female patients often report more severe symptoms and distinct patterns of comorbidity. There are multiple physiological and biological factors that contribute to sex-based differences in drug response and metabolism, including differences in body fat and weight distribution, liver metabolism, hormone levels, plasma volume, protein and enzyme levels, drug transport and clearance, and how individuals adhere to medication. Despite these differences, most antidepressants were developed and dosed without explicit regard to sex, reflecting historical reliance on male-dominated trials. For example, evidence suggests women generally respond better to selective serotonin reuptake inhibitors (SSRIs) and postmenopausal women show poorer antidepressant responses than younger women.[84,85] One example of biotech beginning to treat depression in women as a biologically distinct indication is Arrivo BioVentures, which advanced SP-624, an SIRT6 activator, after observing antidepressant efficacy only in women in early trials, prompting a female-focused development strategy.[86]

Rodent depression models, in particular chronic stress paradigms, were traditionally validated in male animals, and female mice show different and more variable stress responses.[87] That variability is not noise. It likely reflects genuine biological heterogeneity. The central question this raises is whether a single therapeutic framework can adequately serve a condition that manifests differently across sex, reproductive stage, and hormonal context, or whether the field needs to move toward genuinely patient-centric development. Designing female-inclusive preclinical models and clinical trials that capture disease heterogeneity is the core challenge. Without it, the next generation of therapies risks repeating the same structural blind spot.

Diagnostics First: Detour or Foundation?

Diagnostics and femtech are where a disproportionate share of innovation and capital in women's health is currently concentrating, particularly in areas where biological understanding is incomplete. Startups in the PMOS space, for example, focus primarily on diagnostics and disease management rather than novel drugs. Allara Health raised a $26 million in Series B funding[88] to scale a virtual care platform for women with PMOS, endometriosis, and related hormonal disorders, while Evvy and Solence are leveraging data and AI to improve diagnostic accuracy and care pathways. This pattern extends to women's cardiovascular health, where investment skews toward diagnostics and digital tools. The American Heart Association's "Go Red for Women" Venture Fund, managing $200 million in assets, explicitly prioritizes diagnostic differentiation and AI trained on female-specific data, with accelerator cohorts including Kelvin Health, LightHearted AI Health, and Powerful Medical.[89] Endometriosis shows a similar trajectory, with startups such as DotLab, Heranova, and Kephera Diagnostics developing blood-based tests to reduce diagnostic delays.

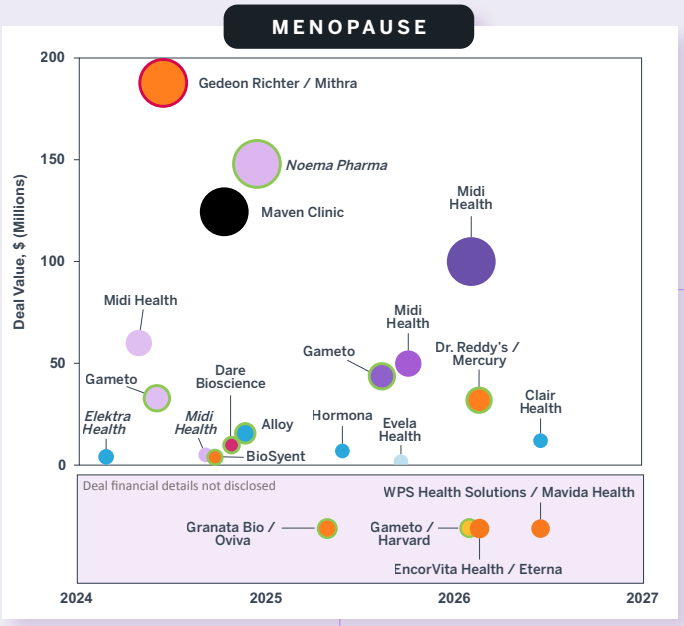
Whether the current concentration in diagnostics and digital health represents a detour from therapeutic development or a prerequisite for it remains an open question. Across most women's health indications, disease heterogeneity is insufficiently characterized to support rational drug discovery. Biomarker development, patient stratification platforms, and organoid-based disease models are the infrastructure on which targeted therapeutics will depend. The diagnostic investment accumulating now could ultimately synergize with mechanistic research to move the field away from the hormonal and symptomatic approaches that have defined most approved therapies for decades. How quickly that transition happens will depend on whether capital follows the science into the harder, slower work of target validation.

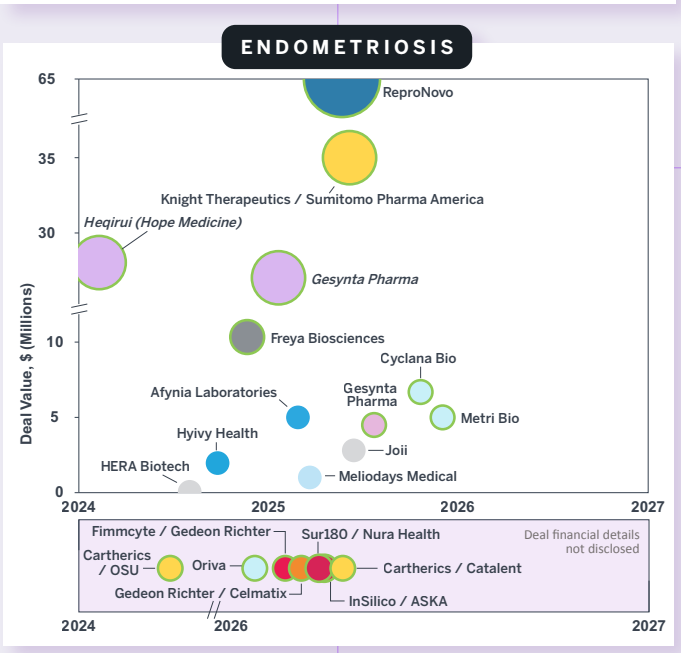
Funding Landscape

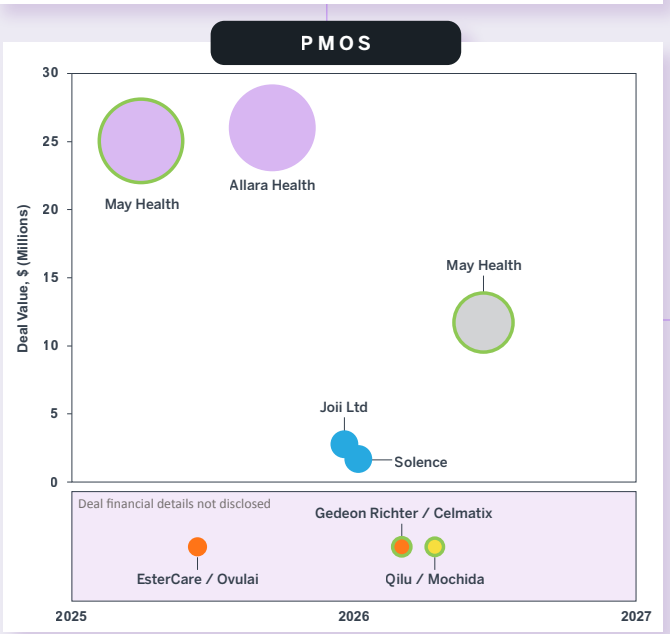
Closing the investment gap is as much a scientific challenge as a funding one. Women's health has captured just ~6 percent of private healthcare capital in recent years, and companies focused exclusively on women's health have less than one percent. Moreover, ~90 percent of that 6 percent flows to only three areas: reproductive health, maternal care, and women's cancer. This leaves glaring "white space" with conditions like endometriosis, PMOS, and menopause collectively sharing only a negligible part of investment.[1] The mismatch between disease burden and capital allocation is stark, but data indicate the commercial opportunity is vast. A Boston Consulting Group (BCG) analysis finds that better screening and care for just four prevalent women's conditions: CVD, osteoporosis, Alzheimer's, and menopause, could unlock over $100 billion in US market value by 2030. Menopause alone illustrates the scale. If all US women with moderate-to-severe symptoms received effective treatment, the menopause care market could expand eightfold to >$40 billion by 2030.[66]

So far, private capital has gravitated toward diagnostics and digital platforms. Nearly half of women's health start-ups operate in these areas, which investors perceive as more straightforward bets. That focus makes sense. Across many women's health conditions, the fundamental barrier to therapeutic breakthroughs is inadequate disease characterization, not lack of market demand. Many conditions are heterogeneous and poorly defined, clinical endpoints lack standardization, and patient subgroups are not well stratified. This creates high risk for therapeutic R&D. In this context, investing in diagnostics and enabling technologies is likely not merely a low-hanging fruit, but an essential infrastructure to de-risk drug development. Robust diagnostics, biomarkers, patient stratification platforms, and advanced disease models could reduce biological uncertainty, thereby increasing investor confidence for the next wave of capital.

In general, bridging this gap requires a shift in how capital is deployed. Historically, investors have preferred stories of breakthrough drugs. Funding foundational work in diagnostics or biology has been less appealing. But characterizing disease heterogeneity and creating the right models lays the groundwork for therapeutics by reducing scientific uncertainty. Big pharma and strategic investors are stepping in to support women's health innovation at multiple stages. For instance, Organon, Merck's 2021 spin-out dedicated to women's health, launched an accelerator in 2026 to mentor biotechs in areas like endometriosis and PMOS.[90] Eli Lilly's 2025 Grand Challenge had a track for women's health, awarding non-dilutive grants to top start-ups (e.g.,







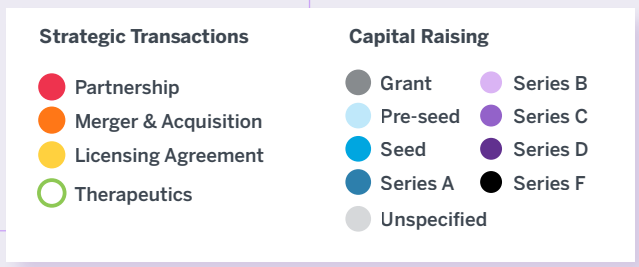


Figure 3. Recent Deal Activity in Women's Health: Menopause, Endometriosis and PMOS.
Strategic transactions and capital raising events across three women's health indication areas: menopause, endometriosis, and PMOS are shown for the period 2024–2027. Each bubble represents a single deal. Bubble size is proportional to deal value (USD millions). The x-axis indicates deal date and the y-axis indicates deal value. Deals with undisclosed financial details are plotted below the axis break. Bubble colour denotes deal type: strategic transactions include partnerships, mergers and acquisitions, licensing agreements. Capital raising events are categorised by round type (grant, pre-seed, seed, Series A through F and unspecified). Therapeutics deals are indicated by a bold green circular outline. *Venture capital extension rounds are labelled in italics.* Deal data are sourced from GlobalData and supplemented by manual review of press releases and news articles. Data are current as of July 2026.

PMOS: polyendocrine metabolic ovarian syndrome (previously PCOS)

EndoWarriors, Beech Bio, Ovo Labs) addressing women's unmet needs.[91] Mission-driven capital is also surging. In 2025, the Gates Foundation pledged $2.5 billion over five years for women's health R&D. Its largest commitment to date, targeting underfunded areas like menstrual and maternal health.[92] Similarly, the American Heart Association's "Go Red" venture fund (launched in 2025) carved out $75 million for women's cardiovascular innovations.[93]

Beyond these headline commitments, the clearest read on where the field is heading comes from the underlying deal flow. To provide a clear signal, we focused on three indications, menopause, endometriosis, and PMOS, where disease specificity to women's health is unambiguous and where deal activity most clearly reflects the field's current trajectory (Figure 3). Across these areas, the mix of early venture rounds, licensing agreements, and partnerships offers a useful proxy for relative clinical and commercial maturity. Menopause has generated the largest individual transactions to date. Endometriosis shows the broadest diversity of deal types, and PMOS remains predominantly early-stage. A small group of companies sits at the more ambitious end of this activity. These are biotechs built on female-specific biology rather than software. Gameto, for example, uses engineered ovarian support cells derived from induced pluripotent stem cell (iPSC) technology. Its lead program, Fertilo, matures eggs ex vivo to compress the two-week ovarian-stimulation phase of IVF to two or three days. It is the first iPSC-derived therapy to reach late-stage clinical development in the US. Its ARPA-H-funded Ameno program targets menopause through an implantable hormone-restoring cell therapy. Gameto has raised over $127 million in total funding, one of the largest investments in the sector.[94] Granata Bio is a less platform-oriented but legitimately venture-backed reproductive-health biopharma with clinical assets spanning four IVF product classes, having raised over $30 million across a 2024 Series A led by Google Ventures and a 2025 Series A+ backed by strategic women's-health players Gedeon Richter and CooperSurgical.[95] The contrast between the two, a category-defining iPSC platform versus a focused asset-development play, captures the range of what women's health biotech now includes.

Overall, correcting the gender investment bias is both a public health imperative and a business opportunity. To seize it, stakeholders must commit to science-first solutions. Investing in research, data and tools needed to make women's health conditions tractable, thus encouraging more traditional venture and large-cap pharma involvement. As the evidence base strengthens, perceptions of risk will diminish, unlocking greater capital flows. Coordinated efforts from pharma partnerships and accelerators to blended financing and philanthropic capital are already beginning to break the vicious cycle of underinvestment.

Closing the Gap Requires Closing the Loop

The women's health gap reflects a chain of compounding failures: a male-default research base, trials that enroll women without analyzing them by sex, and a nascent biological understanding that translates into a risk profile unpalatable to most investors. In addition, nearly every condition examined, PMOS, endometriosis, menopause, and cross-cutting diseases, is biologically heterogeneous, multi-system, and poorly stratified. In almost all cases, current therapies manage downstream symptoms rather than modify disease. Together, these patterns suggest that the standard drug-development playbook, built on homogeneous populations and single dominant targets, is a poor fit for much of women's health.

The central question is whether AI and multi-omic methods can do for women's health what biomarker stratification did for oncology: segment biologically heterogeneous conditions into subgroups homogeneous enough to run clean trials and build durable commercial franchises around, and whether those subgroups, once defined, are large enough to justify the investment. Answering that question requires exactly the biological characterization tools the field does not yet have, which is why the diagnostic and data infrastructure accumulating today is not peripheral to the therapeutic opportunity but arguably the most productive place to start. It is no coincidence that the most visible near-term progress sits in diagnostics, data platforms, and femtech. When the underlying biology is still this poorly characterized, the enabling layer flourishes first, and it is exactly the foundation the therapeutic wave depends on.

With better models, stratified trials, and validated biomarkers, the next decade of women's health will be well positioned to move from managing symptoms to changing outcomes, turning one of medicine's most neglected fields into one of its most consequential opportunities.

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Abbreviations

ARPA-H: Advanced Research Projects Agency for Health, **CVD:** cardiovascular disease, **FSH:** follicle-stimulating hormone, **GLP-1 RA:** GLP-1 receptor agonist, **GnRH:** gonadotropin-releasing hormone, **HPG:** hypothalamic-pituitary-gonadal, **HRT:** hormone replacement therapy, **MDD:** major depressive disorder, **PCOS:** polycystic ovary syndrome, **PK:** pharmacokinetic, **PMOS:** polyendocrine metabolic ovarian syndrome (previously PCOS), **PPD:** postpartum depression, **RA:** rheumatoid arthritis, **SABV:** sex as a biological variable, **sFlt-1:** fms-like tyrosine kinase 1, **SLE:** systemic lupus erythematosus

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