Rethinking Women's Longevity

Estrogen, Hormone Optimization, & the Science of Healthspan + Sexspan

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The Woman in Front of Us

CASE ANCHOR

49 years old. She wants relief now — and protection for the life she is still building.

Her Presenting Symptoms

- Irregular cycles · Hot flashes
- Fragmented sleep · Brain fog
- Vaginal discomfort · Lower desire

Her Clinical Profile

- Intact uterus · Migraine without aura
- No prior VTE or hormone-sensitive cancer

When **sleep**, **cognition**, **comfort**, and **sexual function** improve, a woman regains the capacity to train, work, connect, and sustain the behaviors that protect her **healthspan**. Her symptoms are the entry point; her long-term wellbeing is the destination.

THE PROBLEM

The Conversation Women Inherited

For decades, women heard what **estrogen** might do *to* them. Fear shaped clinical practice — and too often, it replaced nuance. Women deserve a fuller picture.

What Estrogen Does

Its role across **multiple organ systems** throughout a woman's life

What Its Loss Means

The **multisystem consequences** of estrogen deprivation across the decades ahead

What the Evidence Supports

Which **treatment options** are backed by rigorous, correctly interpreted data

Not fear. Education and options.

Women's Risk Conversation Is Out of Proportion

Incidence, **mortality**, and **individual risk** are three separate conversations.
Women deserve all three — with accurate data, not amplified fear.

❤️ Heart Disease

The **leading cause of death** in U.S. women. **304,970 deaths in 2023** — approximately 1 in 5 female deaths. Often under-recognized and under-treated.

🎗️ Breast Cancer

The **most commonly diagnosed cancer** in U.S. women (excluding skin cancer). **321,910 invasive diagnoses** and **42,140 deaths** estimated in 2026.

Lung Cancer

The **leading cause of cancer death** in U.S. women. **61,950 deaths** estimated in 2026 — exceeding breast cancer mortality by a wide margin.

Sources: CDC, Women and Heart Disease, 2024; American Cancer Society, Cancer Facts & Figures 2026

The 2026 FDA Labeling Reset

✅ Removed from the Boxed Warning

- Cardiovascular disease
- Breast cancer
- Probable dementia
- Blanket "lowest dose for shortest duration" instruction

⚠️ Still Required

- Individual risk assessment for every patient
- Systemic risk information in labeling
- Endometrial protection when the uterus is present

Source: U.S. Food and Drug Administration, February 12, 2026

2026: The Regulatory Conversation Changed

Two landmark updates expose the same problem: women need **formulation-specific evidence** and appropriate products — not blanket fear or therapeutic neglect.

February 2026 — Estrogen Labeling Reset

FDA-approved labeling changes *removed* cardiovascular disease, breast cancer, and probable dementia from the boxed warning on affected menopausal hormone products. Endometrial safety and individualized risk assessment remain.

September 17, 2026 — Testosterone Product Gap

FDA convened a public workshop on testosterone use in menopausal women. **The United States still has no female-specific approved product**; Australia has AndroFeme 1% for postmenopausal HSDD.

FDA menopausal hormone labeling update, Feb 12, 2026; FDA public workshop, Sep 17, 2026; Australian TGA AndroFeme 1 PI.

Estrogen Biology

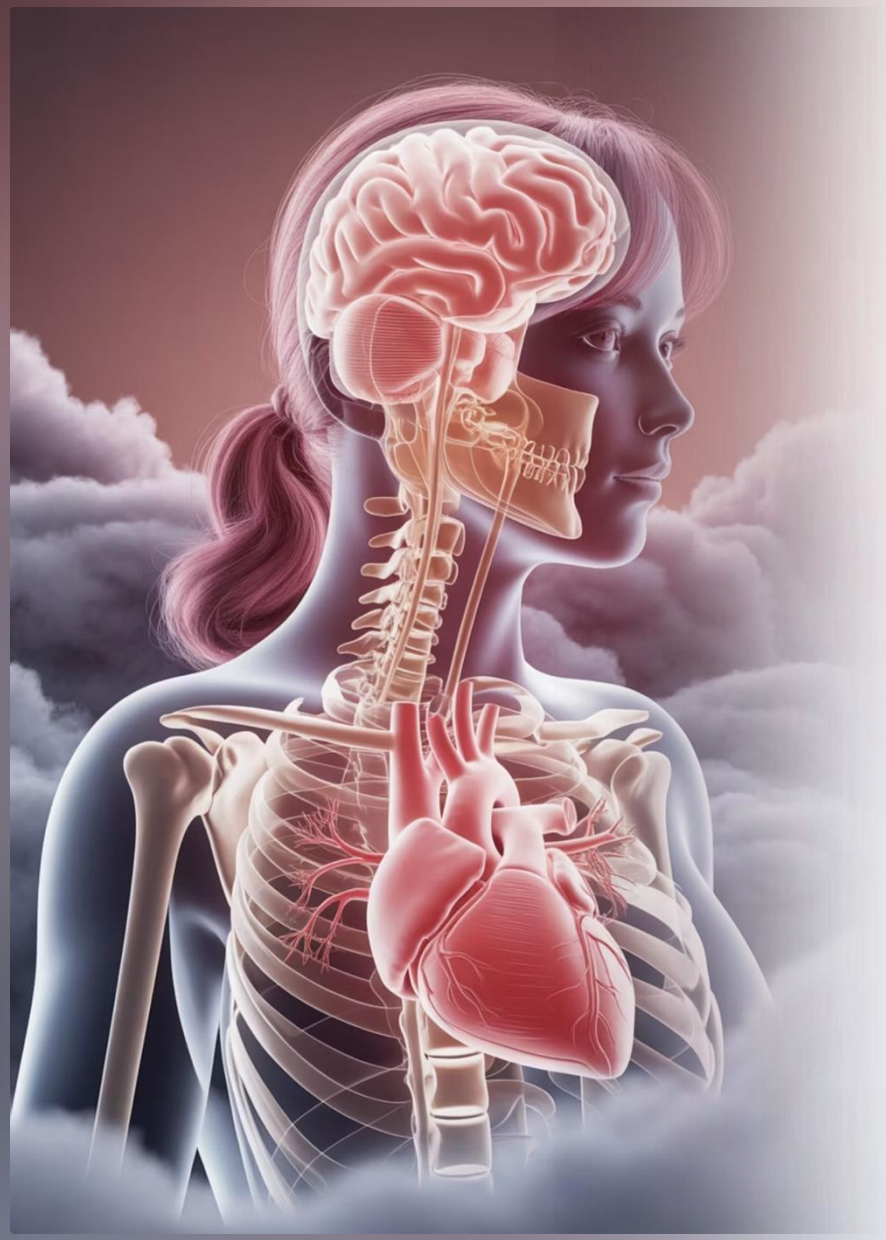
Hormone receptors are everywhere — understanding their reach explains why estrogen loss is a multisystem event.

Hormone Receptors Are Everywhere

Sex steroid receptors — **estrogen (ERα, ERβ)**, **progesterone (PR)**, and **androgen (AR)** — are expressed in nearly every organ system. They don't just modulate reproduction.

- Bone density and muscle mass
- Vascular tone and endothelial repair
- Gene transcription and protein synthesis
- Skin elasticity and collagen synthesis
- Eye health, tear film
- Mitochondrial efficiency and energy production
- Neurotransmitter production and receptor sensitivity
- Inflammatory modulation and immune function
- Cognitive processing and emotional regulation

This explains why hormone therapy produces **systemic benefits** — from brain fog resolution to improved cardiovascular risk profiles. Symptoms multiply across domains when estrogen declines.



Peri/Menopause Is a Multisystem Endocrine Transition

Symptoms are the visible part of a broader **physiologic shift** — one that reaches far beyond the reproductive system.

Brain & Thermoregulation

Hot flashes, cognitive shifts, and hypothalamic dysregulation reflect estrogen's central role in temperature and neural signaling.

Sleep & Mood

Disrupted sleep architecture, mood variability, and heightened anxiety emerge as estrogen modulates serotonin and circadian rhythms.

Vascular & Metabolic

Shifts in lipid profiles, insulin sensitivity, and vascular tone increase cardiometabolic risk in the postmenopausal years.

Bone, Muscle & Body Composition

Accelerated bone loss, reduced lean mass, and increasing central adiposity mark the body's response to declining estrogen.

Genitourinary Tissue

Vaginal atrophy, urinary urgency, and tissue thinning result from estrogen-dependent changes in the genitourinary tract.

Sexual Function

Changes in desire, arousal, and comfort often reflect a combination of hormonal, physical, and psychological factors.

Timing and Context of Hormone Loss Matter

Perimenopause is part of the treatment window, and not every loss of ovarian function is the same clinical event.

Perimenopause

Symptoms can begin years before the final menstrual period. Diagnosis after age 45 is usually clinical — and treatment can begin here.

Premature or Early Loss

POI, oophorectomy, and treatment-induced menopause create longer exposure to hormone deprivation with meaningful cardiometabolic and skeletal consequences.

Clinical Safeguards

Address contraception when relevant, and evaluate abnormal bleeding rather than attributing it automatically to menopausal transition.

The Menopause Society 2022 position statement; ACOG guidance on menopause and POI.

Estrogen Therapy Is Not One Exposure

Menopausal estradiol, hormonal contraception, and low-dose vaginal estriol or estradiol are **not interchangeable exposures** — clinically or biologically.
Route, molecule, dose, and pattern each shape distinct pharmacokinetic profiles.

Route

Oral, injectable, transdermal, intravaginal ring, or **local vaginal** — each produces a distinct pharmacokinetic profile and tissue exposure pattern.

Molecule

17β-estradiol, conjugated equine estrogen, and **ethinyl estradiol** differ meaningfully in receptor affinity, metabolism, and hepatic effects.

Dose and Pattern

Continuous, cyclic, or **endpoint-specific** dosing strategies produce different bleeding patterns and endometrial exposures.

What Is Proven — and What Remains a Healthspan Question

Treat indications confidently. Frame broader longevity hypotheses without turning mechanism into a promise.

Established Clinical Outcomes

- Vasomotor symptom relief
- GSM treatment
- Prevention of bone loss and fracture during use
- Replacement for premature or early hypoestrogenism when appropriate
- Decreased Cardiovascular Risk (initiated within 10-year window)

Important — But Not Prevention Claims

Dementia, breast cancer, metabolic aging, and biological age require **outcome-specific evidence**.
Timing, route, and formulation may modify risk — but do not justify guarantees.

The Menopause Society 2022; Cochrane 2026 randomized-evidence update.

Cardiometabolic & Skeletal Health

Menopause is an inflection point — not just a reproductive milestone.
Some changes accelerate specifically around the final menstrual period.

Menopause Is a Cardiometabolic Inflection Point

Some changes reflect aging. Several accelerate specifically around the **final menstrual period** — making timing clinically relevant, not just theoretically interesting.

Lipid Shifts

LDL-C and **apolipoprotein B** rise during the late transition. A higher HDL-C does not automatically confer protection — particle composition and function also change.

Visceral Fat Accelerates

Central adiposity accelerates approximately **two years before the final menstrual period**. Fat gain rises and lean mass declines even when scale weight changes little.

Compounding Risk Factors

Blood pressure, insulin resistance, sleep disruption, and vasomotor burden can compound cardiovascular risk simultaneously.

Treat symptoms and cardiometabolic risk in parallel — not sequentially.

Sources: El Khoudary et al., Circulation, 2020; Greendale et al., JCI Insight, 2019; Samargandy et al., Menopause, 2021

A Woman's Reproductive History Is Part of Her Cardiovascular History

It can reveal cardiovascular risk **before** a conventional calculator does — and it belongs in the intake interview.

Polycystic Ovary Syndrome

Longstanding metabolic and androgen-related risk that persists well beyond reproductive years

Hypertensive Disorders of Pregnancy

Independently associated with elevated future cardiovascular risk, including stroke

Gestational Diabetes

A strong predictor of future type 2 diabetes and metabolic disease

Preterm Delivery & Placental Complications

Markers of placental vascular dysfunction that can signal systemic risk

POI, Early Menopause, or Bilateral Oophorectomy

More years of estrogen deprivation with meaningful cardiovascular and bone implications

Autoimmune Disease & Treatment-Induced Menopause

Adds inflammatory burden and may constrain hormone options

Sources: Parikh et al., Circulation, 2021; ACC/AHA Primary Prevention Guideline, 2019

Bone Loss Accelerates Around the Final Menstrual Period

In **SWAN**, average annual bone loss accelerated sharply during the **transmenopause window** — approximately one year before through two years after the final menstrual period. The rate slowed after transition, but bone already lost was **not automatically restored**.

2–3%

Lumbar Spine

Average annual bone loss per year during transmenopause

1–2%

Femoral Neck

Average annual bone loss per year during transmenopause

The fracture prevention conversation belongs in perimenopause — not only at age 65. These losses compound over years; a woman entering her 50s without bone health awareness may reach screening age already carrying significant deficit.

Source: Greendale et al., Journal of Bone and Mineral Research, 2012

MHT Protects Bone — Osteoporosis Still Needs Its Own Plan

MHT prevents bone loss and reduces fractures while it is being used — but it is not a complete osteoporosis management strategy on its own.
Estrogen therapy carries an FDA indication for osteopenia to prevent osteoporosis.

01

Screen Appropriately

Order DXA early — do **not** wait until a woman is 65+ years old. Perimenopause is the time to establish a baseline, if not sooner!!

02

Treat the Fracture Risk

A hip or vertebral fragility fracture, an osteoporotic T-score, or high FRAX risk may require **osteoporosis-specific therapy** — not MHT alone.

03

Plan Beyond the Prescription

Bone protection decreases after MHT stops. Fracture prevention preserves mobility, independence, and the ability to participate fully in the decades ahead.

USPSTF Osteoporosis Screening Recommendation, 2025; LeBoff et al., Osteoporosis International, 2022; Bofill Rodriguez et al., Cochrane, 2026

Estradiol Exposure, Absorption, and Bone Health

A 2026 review proposes an emerging reference point — use it as clinical context, not a universal prescription target.

≈60

pg/mL serum estradiol

Emerging reference point — not a universal target

What the Paper Suggests

Across gel trials and KEEPS/ELITE data, approximately **60 pg/mL** was associated with symptom and skeletal benefit.

What It Does Not Prove

No prospective trial established one serum target for every woman, route, assay, or clinical outcome.

How to Use It

Interpret with dose, route, application, lab timing, symptoms, DEXA, fracture risk, and adherence.

Piette PCM, Simon JA. Gynecol Endocrinol. 2026; British Menopause Society guidance.

The Brain in Transition

Perimenopause is not merely hormonal — it is profoundly neurological.
Estrogen acts as brain fuel, and its erratic decline creates a measurable energy deficit.

The Female Brain in Perimenopause: A Neuroendocrine Transition

Neurotransmitter Disruption

Fluctuating estrogen destabilizes serotonin and dopamine synthesis, producing mood instability, irritability, and cognitive fog.

Hypothalamic Instability

Temperature regulation falters, generating vasomotor symptoms (hot flashes) and fragmented sleep architecture.

Metabolic Shift

The brain transitions from glucose-dependent to ketone-preferring metabolism — a temporary energy crisis requiring support.

Early hormone support and ketogenic nutrition can stabilize this transition, **reducing** symptom severity and preserving cognitive resilience. *(Mosconi, 2024)*

The Menopausal Brain: Structural & Metabolic Changes

These changes are adaptive, not purely degenerative — the brain is learning to function on alternative fuel. With proper hormonal, nutritional, and lifestyle support, metabolic resilience can be restored.

Quantifiable Neural Changes

- **2–6% gray matter volume loss** post-menopause (Mosconi et al., Sci Rep 2021)
- **10–15% reduction** in brain glucose metabolism, particularly in hippocampus and prefrontal cortex
- **White matter integrity decline** affecting neural connectivity (Rehkopf et al., 2022)
- **Most affected regions:** hippocampus (memory), hypothalamus (regulation), amygdala (emotion), prefrontal cortex (executive function)

Hormone replacement therapy *may* attenuate volume loss and metabolic decline if initiated within the critical **5-year window** after the final menstrual period.

Mood & Mental Health in Midlife: A Critical Window

35%

Peri/Menopausal Women

Experience clinical depression **during** the transition

40%

Increase

In **antidepressant use** among midlife women

Suicide risk peaks between ages 45–55 (CDC 2024)

- Hormonal turbulence intersects with caregiving burden, career stress, and identity shifts
- Many women are prescribed polypharmacy when root-cause, physiological treatment could be beneficial

Midlife represents the **perfect storm of biological and psychosocial stressors**. Addressing neurochemistry through **hormones**, **nutrients**, and **lifestyle** can reduce reliance on psychiatric medications and restores women's sense of agency.

Supporting Brain Health in Midlife Women

Hormonal Support

Estradiol and progesterone improve cognition and mood. **Testosterone** enhances energy, focus, and assertiveness. Timing matters — early initiation offers neuroprotection.

Nutritional Optimization

MIND diet rich in leafy greens, berries, nuts, and fatty fish. **Omega-3 DHA** supports membrane integrity. **Magnesium** stabilizes neurotransmission. **Vitamin D3** modulates receptor function.

Social Connection & Sleep

Sleep restores glymphatic clearance of metabolic waste. **Social bonding** elevates oxytocin. **Purpose and meaning** stabilize limbic function and reduce cortisol.

Physical Movement

Exercise increases BDNF, promoting neuroplasticity. **Resistance training** boosts testosterone. **Cardiovascular activity** enhances blood flow and mitochondrial biogenesis.

Genitourinary Syndrome of Menopause

GSM is chronic, progressive if untreated, and often requires ongoing therapy — yet it remains under-diagnosed and undertreated. It affects quality of life, sexual health, and urinary function simultaneously.

Hallmark Symptoms

- Vaginal dryness, burning, and tearing
- Dyspareunia — entry and contact pain
- Urinary urgency and recurrent UTI
- Reduced tissue elasticity, blood flow, and sensation

Treatment Principles

Low-dose vaginal estrogen can be used **without systemic estrogen** and does not require concurrent progestogen.

Local therapy is a **fundamentally different exposure** — it can be offered to women across life stages and for a broad range of clinical contexts, including preveention!!!

Sexual Health & the Sexspan

Sexual wellness is a window into systemic health — not a peripheral concern.

Restoring it requires treating physiological causes, not just symptoms.

Sexual Dysfunction: "The Canary in the Coalmine"

1 Damaging Beliefs

- Normal part of aging
- Caused by lack of attraction
- General disinterest in sex
- Purely psychological ("in your head")
- Permanent and untreatable
- Women lack libido compared to men

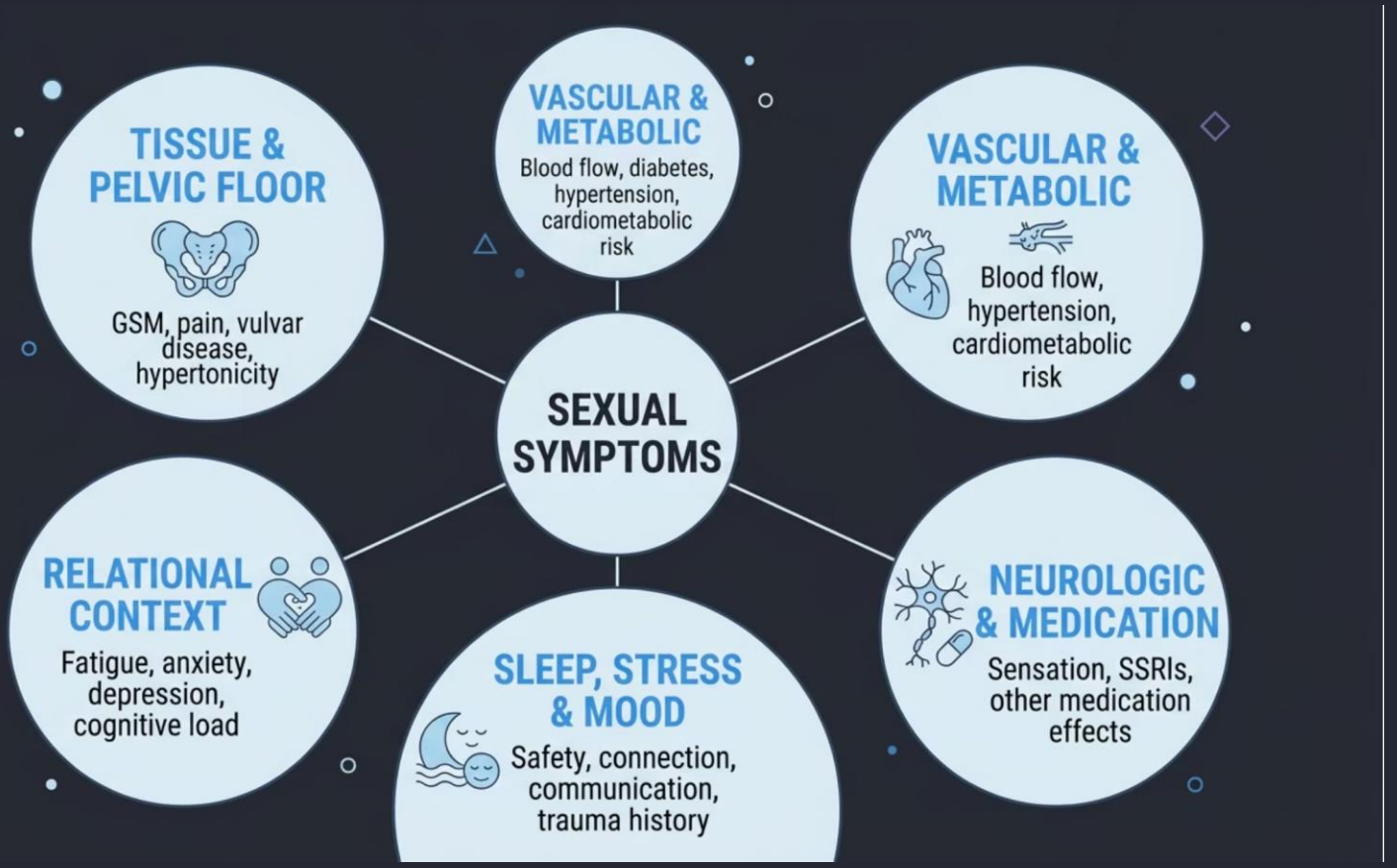
2 Reality: Early **Warning** Signs

Sexual dysfunction often signals **underlying systemic health issues**:

- Insulin resistance and diabetes
- Cardiovascular disease
- Hormonal imbalances · Thyroid dysfunction
- Depression and anxiety · Sleep disorders
- HPA axis dysfunction
- Neurodegenerative disease

Sexual Function as a Window into Healthspan

A new or worsening sexual concern can reveal changes across several systems. It is a reason to widen the clinical lens — not narrow it to a single prescription.



Clinical framing adapted from ACOG Practice Bulletin No. 213 and The Menopause Society sexual-health resources.

The Menopausal Brain & Sexual Wellness

Mood, Motivation & Connection

Estrogen Decline

Disrupts serotonin and dopamine synthesis, reducing motivation, curiosity, and emotional warmth

Structural Changes

Hippocampal atrophy and prefrontal alterations produce emotional flatness and cognitive distance

Reduced Bonding

Lower oxytocin signaling diminishes empathy, relational presence, and responsive desire

Testosterone Deficiency

Loss of confidence, assertiveness, and initiation — the "spark" that drives proactive intimacy

> "Women often describe feeling detached from partners and themselves. That's neuroendocrine, not psychological. Restoring estrogen, testosterone, and oxytocin reawakens vitality and intimacy."

Benefits of Orgasms

Regularly achieving orgasm delivers measurable physiologic benefits — each one a clinical argument for taking women's sexual health seriously.

Immune Support

Strengthens immune system function through neuroendocrine activation

Pain Management

Natural pain relief through endorphin and enkephalin release

Stress Reduction

Decreases stress and anxiety levels naturally through cortisol regulation

Better Sleep

Promotes deep relaxation and improved sleep quality via oxytocin and prolactin release

Cognitive Health

Promotes neuron growth in hippocampus, supporting memory and learning

Intimacy & Longevity

Increases partner connection through oxytocin release; associated with increased lifespan and vitality

Desire **Does Not** Always Arrive First

Spontaneous and responsive desire are both typical patterns. Context can determine whether interest emerges — and understanding this distinction prevents misdiagnosis.

Spontaneous Desire

Interest occurs **before** stimulation or sexual activity. Novelty, anticipation, or mental preoccupation may create the first signal. More common in early relationships and in younger women.

Responsive Desire

Interest emerges **after** connection, stimulation, or arousal begins. This pattern is common — particularly in long-term partnerships and after midlife — and does not automatically represent dysfunction.

> *"A laboratory value can tell me something about her physiology. It cannot tell me whether she feels safe, connected, rested, attracted, stimulated, or whether sex is pleasurable."*
> — Ashley Madsen, PA-C, HHC, ABAAHP

Basson R. J Sex Marital Ther. 2000; ACOG Practice Bulletin No. 213. 2019.

Female Arousal Is a Neurovascular Event

1

Brain

Attention, desire, reward, medication effects

2

Autonomic State

Safety, stress response, capacity to receive stimulation

3

Blood Flow

Clitoral engorgement, lubrication, vascular responsiveness

4

Tissue & Pelvic Floor

Elasticity, sensation, estrogen exposure, GSM, relaxation, and pain response

Arousal depends on **central processing**, **autonomic state**, **genital blood flow**, **tissue health**, and **pelvic-floor response** — not desire alone.

⊗ **When sex hurts, treating desire alone is the wrong sequence.**

O'Connell HE, et al. J Urol. 2005; ACOG Practice Bulletin No. 213. 2019.

Sexual Symptoms Are Not One Symptom

A comprehensive assessment requires separating distinct **drivers of sexual dysfunction** — and treating overlapping contributors in parallel. Constipation, IBS, and pelvic-floor dysfunction can coexist with dyspareunia.

Tissue

Dryness, burning, tearing, and bleeding from genitourinary atrophy

Pain

Entry pain, deep pain, pelvic-floor guarding, vulvar disease, and bowel-related pelvic pain

Arousal

Lubrication, genital sensation, engorgement, and blood flow response

Desire & Orgasm

Responsive desire, distress threshold, latency, intensity, and medication effects

Context

Sleep, stress, safety, body image, relationship quality, and trauma history

The Hormones: Big Three

Estrogen, progesterone, and testosterone are not interchangeable — each plays a distinct and essential role in women's physiology.

Estrogen, Progesterone & Testosterone: The Big Three

Estrogen (E2) — The Igniter

Upregulates serotonin receptors, enhances nitric oxide synthesis, maintains bone density and skin elasticity. Critical for mood stability, vascular health, and vaginal tissue integrity. **Neuroprotective when initiated during the menopausal transition window.**

Progesterone (P4) — The Soother

Converts to allopregnanolone, a potent positive allosteric modulator of GABA-A receptors. Promotes calm, deep sleep, and emotional regulation. Balances estrogen's proliferative effects on endometrium and breast tissue.

Testosterone (T) — The Sustainer

Drives motivation, assertiveness, erotic imagination, confidence, and libido. Enhances dopamine synthesis, mitochondrial function, and muscle mass. Essential for cognitive sharpness, energy, and sexual initiation — not a "male" hormone but a **human hormone**.

Estrogen lights the fire, progesterone soothes it, testosterone sustains it.

Testosterone Is a Women's Hormone

Its physiology extends far beyond libido. Treatment evidence is strongest — and boundaries clearest — for postmenopausal HSDD.

Brain and Motivation

Reward, sexual motivation, and initiation; treatment outcomes remain evolving

Muscle and Metabolism

Androgen signaling supports physiology; clinical outcome claims are not yet established

Bone

Androgen-receptor signaling and aromatization contribute; testosterone is not an osteoporosis treatment

Breast

Complex androgen-receptor biology; short-term physiologic-dose data reassuring, long-term data limited

Sexual Function

Best-supported use: postmenopausal HSDD

Global Consensus Position Statement, 2019; ISSWSH Clinical Practice Guideline, 2021; FDA workshop, 2026.

Treating the Woman In Front of You

Route, molecule, dose are clinical decisions — not defaults.

Match the prescription to the patient in front of you.

"Am I Too Old to Start Hormones?"

Age alone does not answer this question. The indication, vascular age, route, formulation, and absolute risk matter far more than the number of candles on a birthday cake.

Starting Systemic MHT

Benefit-risk profile is generally most favorable **before age 60 or within 10 years** of menopause onset — but this is a starting point, not a cutoff. **Don't make your patients wait until menopause.**

Starting Later in Life

Vascular age, route, formulation, and absolute risk require careful individual assessment. Later initiation is a more nuanced conversation — not an automatic contraindication.

Local Vaginal Estrogen

Every woman. GSM can be treated regardless of age. Local therapy represents a fundamentally different exposure than systemic MHT.

Continuing Therapy

No automatic stopping age exists. Reassess benefit and evolving risk regularly — in true partnership with the patient.

She has not **aged out** of care. She may have aged into a **more nuanced conversation**.

Physiologic Hormone Therapy: The Core Regimen

Transdermal Estradiol

Bioidentical 17β-estradiol via patch, gel, or cream. Dose titrated to symptom relief and serum levels (E2 60–100 pg/mL). **Avoids first-pass hepatic metabolism**, reducing thrombotic risk. May add vaginal estriol or estradiol for genitourinary symptoms.

Micronized Progesterone

Oral bioidentical P4, 100–200 mg at bedtime. Provides endometrial protection and GABAergic sleep support. Converts to allopregnanolone, reducing anxiety. **Avoid synthetic progestins when possible, however progestin-eluding IUD popular option too especially in perimenopause for BC.** Intravaginal or rectal route available for oral intolerance.

Transdermal Testosterone

Compounded cream/ Androgel applied daily to smooth skin (behind the knee, inner thigh, or vulvar region). Monitor total and free testosterone, DHT; target mid- to high normal female range. Restores libido, energy, and cognitive clarity within **4–8 weeks**.

Localized Vaginal Hormones (Next Slide)

Intravaginal nightly. Metabolized locally reversing vulvovaginal atrophy **without systemic absorption**. FDA-approved formulations for dyspareunia.

Vaginal Hormone Prescribing

Match the formulation to the affected tissue, symptom pattern, and likelihood of adherence.

Option	Typical prescription	Best clinical fit
Estradiol softgel insert	4 or 10 mcg nightly for 2 weeks, then twice weekly	Ultra-low dose, applicator-free and minimal mess
Estradiol vaginal tablet	10 mcg nightly for 2 weeks, then twice weekly	Familiar, generic option for internal vaginal symptoms
Estradiol ring	Releases 7.5 mcg/day; replace every 90 days	Excellent adherence and low maintenance
Estradiol 0.01% cream	Individualize induction; 0.5 g two to three nights weekly for maintenance	Best when the introitus, vestibule or vulva also requires direct treatment
Prasterone vaginal insert	6.5 mg nightly	FDA-approved for moderate to severe dyspareunia associated with menopause

Prescribing pearls

- Inserts, tablets, and rings primarily treat internal vaginal tissue.
- Cream offers the most flexibility when external tissues are involved. Prescribe the dose in **grams** and explain placement.
- Progestogen is generally not required with low-dose local vaginal estrogen.
- GSM is chronic. Treatment may continue.
- Evaluate any postmenopausal bleeding.

Addressing Libido: Treatment Options for Women

Multiple pathways exist — choose based on the clinical phenotype, not a one-size-fits-all approach.

Vyleesi (Bremelanotide)

Melanocortin receptor agonist that works centrally to increase desire and motivation. FDA-approved for premenopausal acquired, generalized HSDD.

Addyi (Flibanserin)

Daily medication that modulates neurotransmitters to enhance desire. For women under 65 with acquired, generalized HSDD.

Testosterone

Improves both sexual desire and physical arousal. Best-supported for postmenopausal HSDD.

Estrogen/Estradiol

Supports mood, influences nitric oxide production, maintains vaginal tissue health, lubrication, and elasticity.

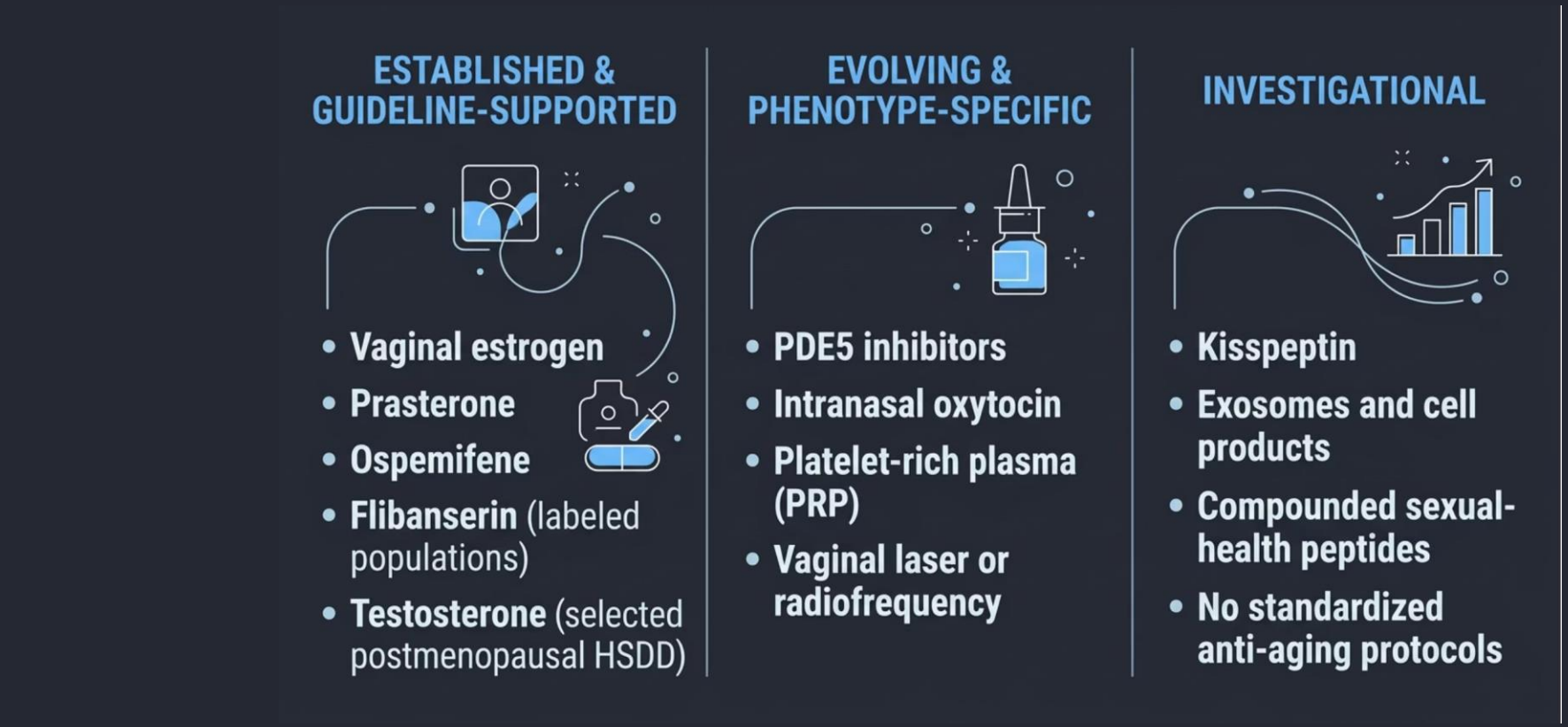
Oxytocin

Available as troche, nasal spray, or transdermal to enhance bonding and pleasure. Supports responsive desire.

Kisspeptin

Stimulates GnRH leading to increased LH and FSH production, enhancing hormone production and menstrual regularity.

The Next Frontier in Women's Sexspan



ISSWSH 2021; FDA prescribing information; sham-controlled device literature; emerging therapies remain investigational.

Micronutrients & Mitochondria

No hormone functions in a nutrient vacuum. Every prescription should begin with biochemical repletion — **foundations before pharmaceuticals**.

Magnesium

Relaxes vascular smooth muscle, supports GABA signaling, stabilizes HPA axis. **300–600 mg glycinate or L-threonate** daily.

Zinc

Cofactor for testosterone synthesis and aromatase regulation. **15–30 mg daily** with copper balance.

Iron

Required for hemoglobin, mitochondrial enzymes, and dopamine synthesis. Monitor ferritin; **target >50 ng/mL**.

Omega-3 Fatty Acids

DHA supports neuronal membrane integrity and serotonin receptor function. **2–4 g EPA+DHA daily**.

Vitamin D3 + K2

Modulates hormone receptor expression and immune function. **Target 25-OH Vitamin D: 60–90 ng/mL**.

B Vitamins

B6, B9, B12 support methylation, neurotransmitter synthesis, and energy metabolism. Essential for homocysteine clearance.

Muscle and Metabolic Reserve Determine the Quality of Added Years

Menopause care should preserve strength, power, insulin sensitivity, and the capacity to recover — not just manage symptoms.

Progressive Resistance

Train strength and power, not only steps or cardio. Muscle mass is a longevity asset.

Protein and Energy

Support muscle protein synthesis and avoid chronic under-fueling, which impairs recovery and hormonal health.

Sleep and Recovery

Treat vasomotor disruption, apnea risk, and excessive training load — all of which undermine adaptation.

Metabolic Treatment

Address insulin resistance and obesity with evidence-based therapy tailored to the individual.

MHT is not a weight-loss drug. Testosterone is not a general muscle-building or metabolic prescription for women.

Returning to the Patient: A Clinical Framework

Treat hormone therapy as a **defined clinical experiment**: a clear indication, measurable outcomes, and planned reassessment.

Clarify Her Symptoms, Goals, and Priorities

What matters most to her? What does success look like in her daily life?

Identify Contraindications and Establish Baseline Risk

Reproductive history, vascular markers, bone density, metabolic labs

Match the Route and Regimen to Her Physiology

Route, molecule, dose, and progestogen type are clinical decisions — not defaults

Protect the Endometrium; Treat Persistent GSM Locally

Required when the uterus is intact; local therapy is always available regardless of systemic MHT status

Evaluate Sexual Concerns Biopsychosocially

Tissue, desire, arousal, context — assess each domain before prescribing

Reassess Symptoms, Bleeding, Tolerability, and Evolving Risk

No automatic stopping age — reassess benefit and risk in partnership with the patient

The New Clinical Standard

Women do not need another reflexive "yes" or "no" about hormones. Women deserve **evidence**, **context**, and the **agency to make an informed decision** — and access to clinicians equipped to provide all three.

Ask Earlier

Begin the menopause and bone health conversation in **perimenopause** — before symptoms become burdensome and before bone is already lost.

Interpret Evidence Precisely

Read population, formulation, timing, and outcome correctly. Resist both the original overcorrection and the emerging counter-overcorrection.

Individualize the Regimen

Route, molecule, dose, and progestogen type are clinical decisions — match the prescription to the patient in front of you.

Measure What Matters to Her

Define success by her symptoms, function, and quality of life — not just a laboratory value or a calendar date.

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Thank you for joining me in rethinking women's hormone health and longevity. Every woman deserves a clinician who asks better questions, interprets the evidence with nuance, and provides individualized care—so she can age with greater health, vitality, and agency.

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