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Article

Androgens and Sexual Health of Women in Postmenopause: From The Historical Concept of "Androgen Deficiency" to Evidence-Based Personalized Therapy

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Abstract: Historically, androgens were regarded mainly as male sex hormones, although testosterone production by the ovaries and adrenal glands, and its peripheral conversion, play a physiological role in women. Since the late 1930s, attempts have been made to use androgens to treat gynecological and sexual complaints, but early interventions were based mainly on clinical observations. The 2002 Princeton consensus formulated the concept of female androgen insufficiency, whereas data accumulated later showed the absence of a reproducible biochemical threshold for such a syndrome. The aim of this review is to trace the evolution of ideas about androgens in peri- and postmenopause and to critically assess the diagnostic value of hormonal measurements and questionnaires, as well as the efficacy and safety of interventions, taking into account the 2025 recommendations of the International Menopause Society, a 2026 systematic review, and the scientific programme of the 20th World Congress on Menopause (Rio de Janeiro, 2026). The review addresses the physiology of ovarian–adrenal androgens, HSDD, the limitations of laboratory methods and psychometric instruments, the results of randomized trials, and the prospects of phenotyping. At present, the indication for a trial of transdermal testosterone with a convincing evidence base is clinically confirmed hypoactive sexual desire disorder in postmenopausal women after a biopsychosocial assessment; an isolated low testosterone level does not establish the diagnosis. Standardization of measurement methods, cultural adaptation of scales, and study of predictors of response and long-term safety are needed.

Keywords: menopause; postmenopause; testosterone; androgens; SHBG; hypoactive sexual desire disorder; HSDD; FSFI; DSDS; hormone replacement therapy; sexual dysfunction.

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1. Introduction

Female sexual health is shaped at the intersection of neuroendocrine, vascular, genitourinary, psychological, and interpersonal mechanisms. In peri- and postmenopause, changes in sexual desire, pain during intercourse, and disturbances of arousal and satisfaction are possible, but these phenomena are not inevitable consequences of aging. The presence of a symptom, its clinical significance, and individually experienced distress are three distinct diagnostic levels. The traditional tendency to attribute the whole complex of complaints to "decreased testosterone" has

led to overdiagnosis of a condition that has not been established by laboratory testing. The modern approach involves defining a specific clinical syndrome, assessing potentially correctable factors, and only then discussing specific treatment[1].

This shift is especially important for endocrinology: even a highly accurate quantitative assessment of androgens does not answer the question of whether a particular patient experiences clinically significant distress related to sexual desire. Consequently, the analytical validity of a biomarker is not equivalent to diagnostic or prognostic usefulness. For clinical practice, what matters is not only the association of hormone concentrations with scales, but also the added value of measuring hormones after accounting for age, comorbidity, medications, and relationship circumstances[2].

2. Materials and Methods

This is a critical narrative review, not a PRISMA systematic review: an exhaustive search, independent double screening, and formal risk-of-bias assessment of each publication are not claimed. In the final version for the journal, it is necessary to specify the databases, search strings, search dates, selection criteria, the procedure for verifying congress abstracts, and author contributions. Numerical results are given only for identified publications; quantitative conclusions do not extend to congress presentations without full text.

3. Results

A historical review published in 2009 traces the use of testosterone in women with gynecological and sexual complaints from the late 1930s. These interventions preceded the modern criteria for HSDD, sensitive immunoassay methods, and standardized scales. Clinical reports from that period should be evaluated taking into account the absence of randomization, uniform dosing, and systematic monitoring of androgenic adverse effects. It is not possible to reliably name a single “discoverer” of androgen therapy for female sexual dysfunction: clinical practice developed gradually across several disciplines[3].

By the middle of the 20th century, studies in endocrine physiology had shown that testosterone is also found in women and that its source is not limited to the ovaries. In 1976, Greenblatt and co-authors discussed changes in estrogens and androgens with aging, reflecting the contemporary search for hormonal explanations of menopausal symptoms. However, the small size of early groups and analytical limitations do not allow these conclusions to be directly transferred to the modern population.

The real transition from empirical therapy to a testable hypothesis began with placebo-controlled studies of transdermal forms. In the study by Shifren and co-authors, published in the New England Journal of Medicine in 2000, 75 women after oophorectomy who were receiving estrogens used placebo and testosterone patches of 150 and 300 μg/day sequentially. A positive effect was demonstrated on individual measures of sexual function, primarily at the higher dose studied. Importantly, the study concerned surgical menopause and could not automatically confirm a testosterone deficiency syndrome after natural menopause[4].

In 2005, larger RCTs in surgical menopause were published, including 533 patients in the study by Buster and co-authors. In 2008, the APHRODITE study in 814 women showed that a 300 μg/day patch can improve sexual function even without mandatory concomitant estrogen use, while the authors explicitly pointed to uncertainty about long-term safety, including risks to the breast. The historical significance of these works lies not in confirming a universal “deficiency” but in identifying a therapeutically responsive clinical phenotype[5].

The Princeton expert group proposed defining female androgen insufficiency as a combination of typical clinical symptoms and reduced bioavailable testosterone with sufficient estrogen status. The authors already stated at that time that test systems had insufficient sensitivity in the low female range. This concept contributed to the recognition of androgenic mechanisms in women's health, but its vulnerability lay in the absence of a

reliable normal limit and of specific symptoms. Reduced energy, mood, or libido is not pathognomonic of testosterone deficiency[6].

The 2019 Global Consensus, endorsed by several international professional societies, formulated two statements: no level of circulating androgens can be used to diagnose HSDD, and the only evidence-based indication for systemic testosterone in women is postmenopausal HSDD. The 2021 ISSWSH guideline translated these statements into a practical algorithm for selection, dosing, and monitoring. The IMS recommendations, published in December 2025, additionally emphasized the age-related dynamics of testosterone, the errors of immunological measurements, and the importance of a comprehensive biopsychosocial assessment.

Table 1. Historical Evolution of Scientific Understanding and Clinical Approaches to Testosterone Therapy in Women (1930s–2026)

Period	Historical milestone	How understanding changed
Late 1930s – 1970s	Clinical use of androgens, physiological observations	Sexual complaints were viewed through a hormonal hypothesis; little evidence
2000	RCT by Shifren et al., NEJM	A testable therapeutic effect emerges in a specific group
2002	Princeton consensus	Attempt to define the syndrome of female androgen insufficiency
2005–2008	Large RCTs of patches	Focus shifts to HSDD and clinical outcomes
2019	Global Consensus, JCEM	No valid threshold of androgen deficiency; the proven indication is HSDD
2021	ISSWSH	Clinical algorithm, physiological doses, monitoring
2025–2026	IMS and new reviews	Personalized biopsychosocial care; criticism of the expansion of indications

In women, circulating androgens originate from direct secretion by the ovaries and adrenal glands and from peripheral conversion of precursors. Total testosterone is the sum of the bound and unbound fractions; a significant portion is transported with sex hormone-binding globulin (SHBG). Therefore, the same total testosterone concentration at different SHBG levels can be combined with a different calculated free fraction. However, this does not mean that the free testosterone index has a validated threshold for sexual dysfunction[7].

Current longitudinal data do not support the notion of a sharp drop in testosterone at the moment of natural menopause. According to IMS 2025, testosterone concentration declines mainly during the reproductive years, does not show an obligatory abrupt break at natural menopause, and may rise at older ages; DHEA gradually declines with age. This fact explains why the term “menopausal androgen deficiency” should not serve as an automatic diagnosis. After bilateral oophorectomy, hormonal dynamics differ, so clinical studies of surgical and natural menopause cannot be mechanically pooled.

The pathways of androgen influence on sexual function presumably include central mechanisms of motivation and reward, as well as peripheral interactions with genital tissues. However, the observed clinical associations are mediated by estrogen status, pain, emotional state, the severity of chronic diseases, and relationship context. An associative

increase or decrease in testosterone should not be declared a direct causal mechanism of sexual desire[8].

Clinical Phenotype: Hsdd, Distress, And Differential Diagnosis

HSDD implies a persistent or recurrent decrease in sexual desire that is significant to the patient herself, with associated distress, and is not better explained by another disorder, the relationship, or the effects of medications. In the diagnostic interview, it is necessary to clarify whether the decrease was acquired or lifelong, generalized or situational, and whether it is related to new pain, dryness, conflict, or the absence of a sexual partner. A low frequency of sexual activity is not in itself equivalent to a desire disorder[9].

Changes in diagnostic classifications require transparency: DSM-5 uses the category female sexual interest/arousal disorder, which combines several aspects of interest and arousal, whereas the clinical literature and therapy studies widely use HSDD. When synthesizing studies, one cannot assume complete identity of these constructs. In clinical work, it is advisable to fix the operational case definition in advance and to specify the criteria by which the diagnosis was made.

Genitourinary syndrome of menopause (GSM), dyspareunia, recurrent urogenital symptoms, sleep disturbance, depression, anxiety, fatigue, and medication-induced decrease in libido should be considered before prescribing androgens. Oral estrogen therapy can raise SHBG, reducing the free fraction of testosterone; nevertheless, elevated SHBG alone does not confirm the need for androgen therapy. Individual assessment is preferable to a universal hormonal algorithm[10].

Hormonal Diagnostics: Why An Accurate Test Does Not Always Yield A Diagnosis

Low testosterone values in women lie in a range where many common direct immunoassays have limited sensitivity, accuracy, and comparability. In scientific studies, liquid chromatography–mass spectrometry (LC–MS/MS) is preferable if a validated method is available. Baseline measurement of total testosterone before therapy serves primarily to exclude high concentrations and to monitor supraphysiological exposure, rather than to search for a diagnostic threshold for HSDD.

Measurement of SHBG is important for interpreting total testosterone, especially in women with obesity, insulin resistance, thyroid pathology, and different estrogen therapy regimens. Calculating free testosterone requires knowledge of albumin and of the equation used; direct analog immunoassays for free testosterone are insufficiently reliable. DHEA-S may reflect the adrenal component of androgen metabolism, but an isolated low concentration also does not establish HSDD. TSH, prolactin, glycemic indices, and other tests are ordered according to clinical need, not as an unlimited panel for every woman[11].

For a scientific model, three hypotheses must be tested separately: the association of concentration with the symptom, the additional prognostic value of the biomarker beyond available clinical data, and the ability of the biomarker to predict specifically the response to testosterone. The absence of an answer to the first hypothesis does not exclude a pharmacological effect of the drug, and a positive statistical association does not establish a treatment threshold.

The Decreased Sexual Desire Screener (DSDS) consists of five questions and was developed for simplified identification of acquired generalized HSDD. In the prospective multicenter validation by Clayton and co-authors (2009), 263 women from 27 North American centers participated. Compared with an expert clinical interview, sensitivity was 83.6%, specificity 87.8%, and diagnostic agreement 85.2%. These are figures from the original sample, not guaranteed accuracy among all older women, in other languages, and in other cultural settings. The DSDS does not measure biochemical androgen deficiency. The Female Sexual Function Index (FSFI), presented by Rosen and co-authors in 2000, contains 19 items grouped into six domains: desire, arousal, lubrication, orgasm, satisfaction, and pain. In the cross-validation study by Wiegel and co-authors, a total score threshold of 26.55 optimally separated participants with and without sexual dysfunction in the population studied. However, the accuracy of this threshold depends on the composition of the sample. Values associated with the absence of activity can lower the

total score in women who do not have a sexual disorder; therefore, mechanical application of a single cutoff for postmenopause is a methodological error[12-13].

The Female Sexual Distress Scale–Revised (FSDS-R) is specifically designed to assess subjective distress associated with sexual dysfunction. The 2008 work by DeRogatis and co-authors confirmed its discriminant validity in a sample of 296 women aged 18–50 years. Transferring the threshold and psychometric characteristics to women aged 60–70 without local verification is unjustified. It is advisable to use the FSDS-R together with an interview and a function scale, rather than as the sole diagnostic test.

MENQOL measures menopause-related quality of life but does not establish HSDD; PHQ-9 helps to quantify depressive symptoms; scales of relationship satisfaction and GSM assessment are useful for eliminating confounding. For use in Uzbek, permission to use the instrument, forward and back translation, cognitive interviewing, and testing of reliability, factor structure, reproducibility, and diagnostic characteristics relative to a clinical interview are required. A self-translated questionnaire must not be declared "validated" without a separate study[14].

Table 2. Validated Clinical Assessment Instruments for Evaluating Female Sexual Function, HSDD, Menopausal Quality of Life, and Associated Psychological Factors

Instrument	Purpose	Confirmed data	Main limitation
DSDS, 5 questions	Screening for acquired generalized HSDD	n=263; Se 83.6%; Sp 87.8%; 2009 [10]	Clinical confirmation required; cultural transferability unknown
FSFI-19	Six domains of sexual function	Threshold 26.55 in a 2005 study [14]	Not a specific test for HSDD; influence of sexual activity
FSDS-R	Distress due to sexual problems	Original validation n=296, aged 18–50 years	Not a stand-alone criterion for the cause of the symptom
MENQOL	Menopausal quality of life	Widely used to assess outcomes	Does not diagnose HSDD or "androgen deficiency"
PHQ-9	Depressive symptoms	Screening measure of possible confounding	Does not replace psychiatric diagnosis

The most informative quantitative synthesis was performed by Islam and co-authors in The Lancet Diabetes & Endocrinology : 46 publications on 36 double-blind randomized trials included 8,480 participants. In postmenopausal women compared with control, an increase in the number of satisfying sexual events was noted: mean difference 0.85 (95% CI 0.52–1.18); sexual desire improved with a standardized mean difference of 0.36 (95% CI 0.22–0.50), and distress decreased with a standardized mean difference of −0.27 (95% CI −0.36 to −0.17). Clinically, this effect is moderate, and the average result does not predict an individual response. Differences between drugs, routes of administration, and baseline populations limit direct comparability.

The pooled evidence base is most consistent for physiological doses of transdermal testosterone in women with documented HSDD. At the same time, studies of testosterone for low energy, cognitive impairment, decline in overall performance, sarcopenia, or osteoporosis have not provided evidence sufficient for routine prescription. The absence of an established laboratory threshold means that treatment cannot be optimized by achieving a "desired" testosterone level[15].

In 2026, Furlan and co-authors published another systematic review on the use of testosterone in female sexual dysfunction, covering premenopausal and postmenopausal

populations. Its existence increases the need for subsequent authors to move away from simple repeated retelling of efficacy: it is more important to study effect modifiers, completeness of safety assessment, analytical methods, and the transferability of conclusions to different phenotypes. Any new review should not be perceived as automatically cancelling the limitations of the original RCTs[16].

Table 3. Overview of Key Clinical Trials and Systematic Reviews on the Efficacy of Testosterone Therapy in Women with Hypoactive Sexual Desire Disorder (HSDD)

Source	Design/sample size	Key finding	Limitation
Shifren et al., 2000	Crossover study, 75, surgical menopause	Positive clinical signal of transdermal testosterone	Cannot be directly transferred to natural menopause
Buster et al., 2005	RCT, 533	Effect on sexual desire confirmed	Surgical menopause, concomitant estrogens
Davis et al., 2008	RCT, 814	Moderate effect of 300 μg/day without mandatory estrogens	Uncertainty about long-term safety
Islam et al., 2019	36 RCTs, 8,480	Improvement in desire, satisfying events, and distress	Heterogeneity, limited long-term follow-up
Furlan et al., 2026	Systematic review	New pooled data on female sexual dysfunction	Does not replace assessment of the quality and applicability of each study

The ISSWSH guideline proposes considering transdermal testosterone for women with HSDD after careful assessment of contraindications and informed consent; in the absence of a licensed female product, doses of the male formulation should be drastically reduced, usually to approximately one-tenth of the standard male dose of the specific 1% gel, taking into account the mass of the applied product and the characteristics of the formulation. This is not a universal instruction for self-administration. The goal of monitoring is to avoid concentrations above the physiological range of premenopausal women. Injectable preparations, implants with a risk of supraphysiological levels, and non-standardized compounded mixtures are not preferred alternatives[17].

Before prescribing, total testosterone, SHBG, lipid profile, liver function tests, the presence of acne, hirsutism, alopecia, and breast disease should be assessed. Total testosterone is usually monitored 3–6 weeks after the start of therapy, after a dose change, and subsequently at an interval set by the clinical protocol. If there is no clinically significant effect within six months, treatment is discontinued or reconsidered. Long-term safety regarding cardiovascular outcomes, the breast, and the endometrium remains insufficiently characterized, especially in high-risk women, who were often excluded from RCTs.

The most discussed adverse reactions of physiological doses are acne and local increase in hair growth; significant virilization when physiological concentrations are maintained is rare but requires attention. It is dangerous to extrapolate the results of short-term RCTs to long-term use of high doses or to patients with contraindications. Safety results should be presented separately for the route of administration and exposure, rather than as a general effect of "testosterone."

4. Discussion

4.1 Other Strategies: Mht, Local Therapy, Flibanserin, And Non-Drug Care

Menopausal hormone therapy (MHT) has established indications, primarily the treatment of vasomotor and certain genitourinary symptoms, but is not a specific universal treatment for HSDD. In GSM and dyspareunia, treatment with local estrogens and other indicated agents can improve comfort and sexual function by reducing pain, without requiring the effect to be explained by androgen insufficiency. Intravaginal prasterone (DHEA) has an evidence base in dyspareunia due to vulvovaginal atrophy, but systemic DHEA is not recommended as a treatment for decreased sexual desire.

In December 2025, the US prescribing information for flibanserin (Addyi) was changed: the indication extends to women under 65 years of age with acquired generalized HSDD regardless of menopausal status, provided the diagnostic conditions are met. This is important news of 2026, but it does not mean that the drug is approved and equally available in all countries. The agent is associated with risks of hypotension, syncope, drowsiness, and drug interactions; it is contraindicated in hepatic impairment and in the presence of moderate or strong CYP3A4 inhibitors, and requires specific instructions regarding alcohol. If there is no effect after eight weeks, use is discontinued according to the US labeling[18].

Psychoeducation, individual psychosexual therapy, and work on relationships are substantive components of care. Their presence does not indicate an "absence of a biological component": the biopsychosocial model assumes an interaction of mechanisms, not competition between hormonal and psychological explanations. When designing studies, it is important to standardize non-drug care across all groups; otherwise the effect of the drug will be confounded with differences in counseling.

4.2 What exactly was presented at the 2026 congress in rio de janeiro

The 20th World Congress of the International Menopause Society took place in Rio de Janeiro in autumn 2026; the official scientific programme included a lecture by Jan Shifren, "Testosterone for Sexual Health in Women: What does the evidence show?". Of particular scientific value is the very choice of topics: besides standard therapy questions, the programme announced work on symptom improvement independent of baseline testosterone concentration (P.114), preliminary results of a randomized comparison of estradiol versus an estradiol–testosterone combination in subcutaneous implants (P.206), a systematic review of the effect of testosterone on cognitive outcomes, and an RCT on prevention of bone loss.

These presentations and abstracts cannot be interpreted as a change in recommendations until the final full-text results are published and study quality is appraised. In particular, the implant data do not remove earlier cautions regarding supraphysiological concentrations, and the presentation of an RCT on bone tissue is not evidence of osteoporosis prevention. Similarly, the report of a symptomatic response independent of starting testosterone needs verification of the design, patient selection, and statistical interaction. Thus, the 2026 congress confirmed the relevance of the question of predictors of response but did not create a new diagnostic criterion for female hypoandrogenism.

The first gap is the lack of prospective cohorts with accurate LC–MS/MS analytics and simultaneous clinical phenotyping. It is necessary to determine whether total or calculated free testosterone adds diagnostic information after accounting for SHBG, metabolic disorders, medications, depressive symptoms, and GSM. The second is the absence of a confirmed predictor of response to testosterone: it is impossible to promise efficacy in advance on the basis of a laboratory value. The third is the limited transferability of international questionnaires across languages and cultural settings.

For Central Asia, of particular interest are culturally adapted DSDS, FSFI, and FSDS-R scales with confirmed validity, population characteristics of clinically significant sexual distress, differences in obesity and insulin resistance, and a description of help-seeking

practices. Studies should avoid selection bias, in which only sexually active women or only those presenting with particular complaints are included in the analysis. Prevalence should be assessed in a consecutive or representative sample, and the case–control design should be applied separately to search for risk factors.

A prognostic model may include age, duration of postmenopause, pain syndrome, PHQ-9, antidepressant use, relationship indicators, and SHBG; biomarkers are added sequentially, comparing calibration, Brier score, and discrimination. To test the effect of testosterone, a randomized trial with a uniform psychosexual support protocol and a predefined clinically significant endpoint is best. Without assessing the interaction between baseline testosterone level and treatment, personalization on the basis of hormone level cannot be claimed.

The optimal sequence includes: (1) respectful collection of the sexual history and documentation of the complaint, (2) DSDS screening with assessment of distress, (3) confirmation of clinical HSDD and differential diagnosis, (4) treatment of pain-related, genitourinary, psychiatric, and medication-related causes, (5) discussion of specific methods if HSDD persists, (6) obtaining informed consent, laboratory monitoring, and regular assessment of clinical effect. In this model, testosterone is one of the possible therapeutic tools for a strictly defined condition, but not a marker of an obligatory replacement need in every menopausal woman.

5. Conclusion

Over almost nine decades, ideas about testosterone in women have progressed from empirical use in gynecology to the development of the concept of female androgen insufficiency and then to its methodological revision. The key achievement of modern science is the separation of hormonal testing from clinical diagnosis: low testosterone by itself does not define HSDD, whereas physiological transdermal therapy can moderately improve individual sexual outcomes in carefully selected postmenopausal women. The IMS 2025 recommendations and the Rio 2026 scientific programme strengthen the focus on interdisciplinary assessment, long-term safety, the cultural validity of questionnaires, and the search for reproducible phenotypes of treatment response. The next stage of research should shift from the question “is there a testosterone deficiency?” to the questions “what is the mechanism of symptoms in a particular patient?”, “who will benefit from a given therapy?”, and “at what cost to long-term safety?”

REFERENCES

[1] “Testosterone therapy in women with gynecological and sexual disorders: A triumph of clinical endocrinology from 1938 to 2008,” Journal of Sexual Medicine, 2009. PMID: 19138368. https://pubmed.ncbi.nlm.nih.gov/19138368/

[2] R. B. Greenblatt et al., “Estrogen–androgen levels in aging men and women: Therapeutic considerations,” Journal of the American Geriatrics Society, 1976. https://doi.org/10.1111/j.1532-5415.1976.tb04294.x

[3] J. L. Shifren et al., “Transdermal testosterone treatment in women with impaired sexual function after oophorectomy,” New England Journal of Medicine, vol. 343, pp. 682–688, 2000. https://doi.org/10.1056/NEJM200009073431002

[4] J. E. Buster et al., “Testosterone patch for low sexual desire in surgically menopausal women: A randomized trial,” Obstetrics & Gynecology, vol. 105, pp. 944–952, 2005. https://doi.org/10.1097/01.AOG.0000158103.27672.0d

[5] S. R. Davis et al., “Testosterone for low libido in postmenopausal women not taking estrogen,” New England Journal of Medicine, vol. 359, pp. 2005–2017, 2008. https://doi.org/10.1056/NEJMoa0707302

[6] G. Bachmann et al., “Female androgen insufficiency: The Princeton consensus statement on definition, classification, and assessment,” Fertility and Sterility, vol. 77, pp. 660–665, 2002. https://doi.org/10.1016/S0015-0282(02)02969-2

[7] R. M. Islam et al., “Safety and efficacy of testosterone for women: A systematic review and meta-analysis of randomised controlled trial data,” The Lancet Diabetes & Endocrinology, vol. 7, pp. 754–766, 2019. https://doi.org/10.1016/S2213-8587(19)30189-5

[8] S. R. Davis et al., “Global Consensus Position Statement on the Use of Testosterone Therapy for Women,” The Journal of Clinical Endocrinology & Metabolism, vol. 104, pp. 4660–4666, 2019. https://doi.org/10.1210/jc.2019-01603

[9] S. J. Parish et al., “International Society for the Study of Women’s Sexual Health Clinical Practice Guideline for the Use of Systemic Testosterone for Hypoactive Sexual Desire Disorder in Women,” Journal of Sexual Medicine, vol. 18, pp. 849–867, 2021. https://doi.org/10.1016/j.jsxm.2020.10.009
[10] A. H. Clayton et al., “Validation of the decreased sexual desire screener,” Journal of Sexual Medicine, vol. 6, pp. 730–738, 2009. https://doi.org/10.1111/j.1743-6109.2008.01153.x
[11] G. D. Braunstein et al., “Safety and efficacy of a testosterone patch for the treatment of HSDD in surgically menopausal women,” Archives of Internal Medicine, vol. 165, pp. 1582–1589, 2005. https://doi.org/10.1001/archinte.165.14.1 582
[12] N. Panay et al., “International Menopause Society recommendations and key messages on women’s midlife health and menopause,” Climacteric, vol. 28, pp. 634–656, 2026. https://doi.org/10.1080/13697137.2025.2585487.
[13] R. Rosen et al., “The Female Sexual Function Index (FSFI): A multidimensional self-report instrument,” Journal of Sex & Marital Therapy, vol. 26, pp. 191–208, 2000. https://doi.org/10.1080/009262300278597
[14] M. Wiegel, C. Meston, and R. Rosen, “The Female Sexual Function Index: Cross-validation and development of clinical cutoff scores,” Journal of Sex & Marital Therapy, vol. 31, pp. 1–20, 2005. https://doi.org/10.1080/00926230590475206
[15] L. DeRogatis et al., “Validation of the Female Sexual Distress Scale–Revised,” Journal of Sexual Medicine, vol. 5, pp. 357–364, 2008. https://doi.org/10.1111/j.1743-6109.2007.00672.x
[16] V. A. Furlan et al., “Testosterone therapy for female sexual dysfunction: A systematic review of the literature demonstrating outcomes in premenopausal and postmenopausal women,” Journal of Sexual Medicine, vol. 23, no. 8, 2026. https://doi.org/10.1093/jsxmed/qdag206
[17] U.S. DailyMed, “ADDYI (flibanserin): Prescribing information,” revised Dec. 2025. https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=3819daf3-e935-2c53-c527-e1d57922f394
[18] International Menopause Society, “20th World Congress on Menopause, Rio de Janeiro 2026: Official scientific programme.” https://imsrio2026.com/scientific-program/