CLINICAL MONOGRAPH · HORMONE OPTIMIZATION

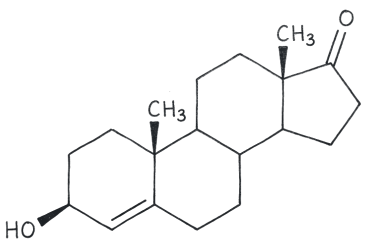
DHEA

Adrenal-derived androgen precursor

DHEA (dehydroepiandrosterone) is a steroid hormone your adrenal glands make in large amounts during early adulthood [orentreich1984]. It peaks around age 20, 25 and then declines roughly 2% per year, so an 80-year-old has about 10, 20% of the DHEA they had at 25. The body uses it as a starting material to make small amounts of testosterone and estrogen inside specific tissues, a process called intracrinology [labrie1991intracrinology].

Only one FDA-approved DHEA product exists: Intrarosa, a vaginal insert (prasterone 6.5 mg) approved in 2016 for painful intercourse in postmenopausal women caused by vaginal tissue changes [fda_label_intrarosa]. Oral DHEA, by contrast, is sold over-the-counter as a dietary supplement, unusual for a hormone precursor [parasrampuria1998]. Studies have shown OTC products often don't contain the dose stated on the label.

RonanRx compounds DHEA when the OTC supplement market or Intrarosa cannot meet a documented clinical need, for example, very low doses (10, 25 mg) that aren't commercially available, sublingual or topical forms, or formulations made to USP pharmacy standards for identity and purity. The evidence for systemic DHEA is mixed: it helps women with adrenal insufficiency (Addison's disease), is being studied in IVF poor responders, and has shown only modest or null effects in healthy older adults for cognition, mood, or body composition [arlt1999nejm; nair2006nejm].



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EVIDENCE POSTURE FDA APPROVED EMERGING REVIEWED **2026-05-11**

State-licensed 503A	Pharmacist reviewed	Doctor led	Cold-chain ready	Patient choice preserved

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Contents

Click any section to jump there. Page numbers update on render.

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FOR CLINICIANS

DHEA is an adrenal C19 steroid synthesized primarily in the zona reticularis under regulation by ACTH and intra-adrenal factors [gurnell2008longterm]. Circulating concentrations are dominated by the sulfate ester DHEA-S, which serves as a long-half-life reservoir for tissue-level desulfation and downstream conversion to androstenedione, testosterone, and estradiol via tissue-specific intracrine steroidogenesis [labrie1991intracrinology, miller2011steroidogenesis]. Serum DHEA-S peaks in the third decade and declines linearly with age (adrenopause), reaching 10, 20% of young-adult levels by the eighth decade [orentreich1984, orentreich1992].

The only FDA-approved DHEA product is Intrarosa (prasterone 6.5 mg vaginal insert, approved November 2016) for moderate-to-severe dyspareunia in postmenopausal women with vulvovaginal atrophy / genitourinary syndrome of menopause [fda_label_intrarosa]. The pivotal evidence was generated by Labrie and colleagues across phase III trials [labrie2011dyspareunia, archer2015dyspareunia, labrie2017menocombined] demonstrating improvement in vaginal pH, vaginal cell maturation index, and most bothersome symptom (pain at sexual activity) without clinically significant elevation of serum estrogens or androgens beyond the postmenopausal reference range [labrie2013local, labrie2017lowdose].

Off-label systemic uses with the strongest evidence are adrenal-insufficiency androgen replacement [arlt1999nejm]. Trials of DHEA for cognition, mood, and body composition in eugonadal older adults have been largely null or modest [nair2006nejm] [hunt2000; corona2013meta]. DHEA for IVF poor responders [wiser2010ivf] has been broadly adopted in fertility practice but the Cochrane review [nagels2015cochrane] concludes that the benefit is uncertain [yeung2014ivf].

Compounded DHEA under 503A addresses needs the OTC supplement market and Intrarosa cannot meet: sub-OTC dose individualization (particularly 10, 25 mg for women, where OTC capsules commonly start at 25, 50 mg), sublingual or topical formulation, excipient-sensitive patients, and USP <795>-compliant identity and purity [alkatib2009meta; parasrampuria1998; fda_essentially_a_copy]. Because oral DHEA is available OTC, compounded oral DHEA is approached with the FDA 'essentially-a-copy' framework in mind, and is reserved for documented patient-specific clinical needs [percheron2003; usp_795].

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Why Personalized DHEA

The FDA reviewed DHEA in exactly one form: Intrarosa, a 6.5 mg vaginal insert dosed once daily for postmenopausal dyspareunia. That clearance does not speak to the patient sitting in front of an endocrinologist with Addison's disease, the woman whose serum DHEA-S has been in the bottom decile for her age since her forties, or the IVF poor responder whose reproductive endocrinologist wants 75 mg a day for ten weeks. It also does not speak to the more pedestrian reality that women generally need 10 to 25 mg a day for replacement and men 25 to 50 mg, while the off-the-shelf supplement market starts at 25 mg and climbs to 100 mg. Baseline DHEA-S, sex, age, free testosterone, SHBG, and tolerance for androgenization (acne, oily skin, hair thinning) all move the right dose around.

Compounding lets the prescriber pick the dose, not the supplement aisle. A 10 mg or 15 mg capsule for a woman whose DHEA-S is creeping toward the young-adult reference range without overshooting it. A sublingual troche when oral first-pass sulfation is producing more DHEA-S than free DHEA than the prescriber wants. A topical cream for a patient who cannot tolerate oral dosing. Identity, potency, and excipient profile are verified to USP <795> standards, which addresses the documented variability in OTC supplement content (the Parasrampuria 1998 JAMA paper that found commercial DHEA capsules often missed their labeled dose by wide margins). For postmenopausal dyspareunia specifically, Intrarosa is the right first call; compounded vaginal DHEA exists for documented patient-specific reasons like excipient sensitivity, not as a substitute.

This is what pharmacy looked like before mass manufacturing arrived. A prescriber wrote the order, a pharmacist prepared it, and the bottle had one patient's name on it. Compounded DHEA is that older arrangement, kept honest by modern oversight.

Quick Facts About DHEA

Category: Adrenal androgen precursor (C19 steroid); intracrine substrate for tissue-specific conversion to testosterone and estradiol

Active ingredient: Dehydroepiandrosterone (DHEA), also known as prasterone, bioidentical to the endogenous adrenal-derived hormone; circulates predominantly as the sulfate ester DHEA-S

FDA-approved branded product: Intrarosa (prasterone 6.5 mg vaginal insert), approved November 2016 for moderate-to-severe dyspareunia in postmenopausal women with vulvovaginal atrophy / genitourinary syndrome of menopause

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OTC status: Oral DHEA is sold OTC in the US as a dietary supplement under DSHEA 1994, unusual for a sex-steroid prohormone. Commercial supplement DHEA content has been shown to vary substantially from labeled potency [parasrampuria1998]. Compounded DHEA under 503A addresses dose, route, and quality needs that the OTC supplement market does not meet.

Routes studied in humans: Oral (capsule, sublingual, micronized), intravaginal (Intrarosa, compounded), topical/transdermal (limited)

Evidence posture: FDA-approved for intravaginal prasterone in postmenopausal dyspareunia (Labrie, Archer phase III). Systemic oral DHEA evidence is mixed: positive in adrenal insufficiency replacement (Arlt 1999, Hunt 2000, Gurnell 2008, Alkatib 2009 meta-analysis); null or modest for aging, cognition, and body composition in eugonadal elderly (Nair 2006 NEJM, Percheron 2003); controversial in IVF poor responders (Wiser 2010, Cochrane Nagels 2015).

Compounded under: 503A, patient-specific prescription only, where the OTC supplement or FDA-approved Intrarosa product does not meet documented clinical need (dose, route, excipient sensitivity, or USP-grade purity)

Important compounding context: Because oral DHEA is available OTC as a dietary supplement, compounded oral DHEA is held to the FDA 503A 'essentially-a-copy' threshold even though there is no FDA-approved oral DHEA product to compare against. RonanRx compounds DHEA when the prescriber documents that OTC supplements cannot meet the patient's clinical need, typically for sub-OTC-strength dosing in women (10, 25 mg), sublingual or topical formulation, or USP <795>-compliant identity and purity assurance.

Doping status: Prohibited in-competition and out-of-competition by the World Anti-Doping Agency (WADA) as an anabolic androgenic steroid

SPECIALS: PATIENT-SPECIFIC PRESCRIPTION ONLY

DHEA described in this monograph is a 503A compounded preparation. Every dose is made on a prescription, for a named patient, by a licensed pharmacist. It is not a stocked, mass-manufactured product.

- **Made to order, not off a shelf.** No batch sits in a warehouse waiting for buyers. Your prescription triggers the prep.
- **Named-patient label.** The bottle carries one patient's name. The batch records carry one prescription.
- **Dose, strength, and route chosen for the patient.** A prescriber decides what gets compounded, not a manufacturer who set the strength for a trial population.
- **Licensed pharmacist on the hook.** A real person, with a license that can be pulled, signs off on every prep. State inspectors check the facility.
- **Compounded drugs are not FDA-approved.** They should not be evaluated using branded-drug trial data alone. Availability varies by state and prescribed medication.

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How This Differs from a Research-Use-Only Website

A research-use-only website ships a vial from a warehouse. There is no prescription, no pharmacist, no facility inspection, and no way to recall the product if something is wrong with it. If the vial is mislabeled, contaminated, or under-potent, there is nobody whose license is at stake.

A 503A compounding pharmacy is the other thing. The doctor writes the prescription. A licensed pharmacist, whose name is on the label, prepares the medicine in a facility the state inspects. If something goes wrong, there is a person and a license on the hook, and a documented chain of custody on every lot. That accountability is what makes it safe.

What is DHEA?

Dehydroepiandrosterone (DHEA; 3β-hydroxy-5-androsten-17-one), also known by the international nonproprietary name prasterone, is a 19-carbon (C19) steroid synthesized predominantly in the zona reticularis of the adrenal cortex. In circulation it exists in two interconverting forms: free DHEA and the sulfate ester DHEA-S. DHEA-S is the most abundant steroid in human blood, quantitatively orders of magnitude higher than testosterone, and serves as a long-half-life reservoir from which tissues regenerate free DHEA for local conversion to active sex steroids [labrie1991intracrinology, miller2011steroidogenesis].

DHEA was first isolated and characterized in 1934. Its physiological role as the substrate for adrenarche, the prepubertal rise in adrenal androgens around age 6, 8, was clarified across the 1980s and 1990s [havelock2004adrenarche]. Serum DHEA-S follows a distinctive lifespan trajectory: low in childhood, rising at adrenarche, peaking in the third decade, then declining approximately 1, 2% per year, a pattern sometimes called 'adrenopause' [orentreich1984, orentreich1992].

In the United States, oral DHEA is sold over-the-counter as a dietary supplement under the Dietary Supplement Health and Education Act of 1994, a regulatory anomaly for a sex-steroid prohormone. The only FDA-approved DHEA prescription product is Intrarosa, a 6.5 mg prasterone vaginal insert approved in November 2016 for moderate-to-severe dyspareunia in postmenopausal women [fda_label_intrarosa].

How DHEA Works

DHEA is best understood not as a hormone with direct receptor agonism but as an intracrine substrate. Labrie's 1991 'intracrinology' framework [labrie1991intracrinology] described how peripheral tissues, vagina, skin, bone, brain, adipose, express the steroidogenic enzymes (steroid sulfatase, 3β-HSD, 17β-HSD, aromatase, 5α-reductase) to convert circulating DHEA and DHEA-S locally into testosterone, dihydrotestosterone, or estradiol, producing tissue-specific androgen and estrogen action without raising serum levels of those active hormones.

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After adrenarche, DHEA-S accounts for the bulk of adrenal androgen output. In postmenopausal women, with ovarian estrogen and androgen synthesis having ceased, essentially all sex-steroid action in peripheral tissues derives from intracrine conversion of circulating DHEA and DHEA-S [labrie1991intracrinology, burger2002androgen]. This is the mechanistic basis for intravaginal prasterone (Intrarosa): locally delivered DHEA is converted by vaginal epithelial enzymes to estradiol and testosterone, restoring vaginal trophic effects while serum sex-steroid levels remain in the postmenopausal range [labrie2013local, labrie2017lowdose, ke2015serum].

DHEA also has direct, AR/ER-independent actions at low affinity on neurosteroid receptors including GABA-A (negative allosteric modulator) and NMDA, and binds the σ-1 receptor, pharmacologically positioning DHEA as a neurosteroid distinct from its role as an androgen/estrogen precursor [baulieu2000dheage]. The clinical relevance of these neurosteroid actions for mood, cognition, and well-being remains an active research question with mixed clinical results.

Biological Role of DHEA

DHEA-S is, by mass, the most abundant steroid hormone in the human circulation in young adults. Its biological role is to serve as a circulating reservoir for tissue-level intracrine generation of androgens and estrogens [labrie1991intracrinology]. This intracrine system is the principal source of sex-steroid action in postmenopausal women and contributes substantially to total androgen exposure in adult men beyond what is provided by testicular testosterone.

Adrenarche, the prepubertal rise in adrenal androgens around ages 6, 8, precedes gonadal puberty by several years and produces the first appearance of pubic and axillary hair, mild acne, and adult-type body odor. The molecular trigger for adrenarche remains incompletely understood but involves remodeling of the adrenal cortex with expansion of the zona reticularis and a shift in CYP17A1 catalytic activity toward 17,20-lyase function [havelock2004adrenarche, miller2012lyase].

Adrenopause, the age-related decline of DHEA and DHEA-S, was documented by Orentreich and colleagues across cross-sectional [orentreich1984] and longitudinal [orentreich1992] studies in the 1980s and early 1990s. The decline is roughly linear at 1, 2% per year from the peak in the third decade. Whether replacing DHEA in aging adults provides clinical benefit beyond restoring laboratory values has been the subject of three decades of trials with largely modest or null results in eugonadal aging populations [nair2006nejm, percheron2003, corona2013meta].

Detailed Mechanism of DHEA

Adrenal biosynthesis. DHEA is synthesized in the zona reticularis from pregnenolone by the 17,20-lyase activity of CYP17A1, with the cofactor cytochrome b5 enhancing lyase versus hydroxylase activity. The reticularis is unique in its high b5 expression and low 3β-HSD activity, which channels precursors toward

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DHEA rather than into the glucocorticoid or mineralocorticoid pathways. DHEA is sulfated by SULT2A1 to DHEA-S; the sulfate ester is the predominant circulating form and has a much longer half-life (hours to a day) than free DHEA (minutes to ~1 hour). Steroid sulfatase (STS) regenerates free DHEA from DHEA-S at peripheral tissues for local conversion [miller2011steroidogenesis, miller2012lyase].

Intracrine conversion. Labrie's intracrinology framework [labrie1991intracrinology] holds that, after the adolescent gonadal surge wanes (in women, after menopause; in older men, as testicular output declines), peripheral tissues meet their sex-steroid needs by local conversion of circulating DHEA and DHEA-S. The vaginal epithelium expresses STS, 3β-HSD, 17β-HSD, and aromatase, producing local estradiol and testosterone from intravaginal DHEA without raising systemic concentrations beyond the postmenopausal range, the mechanistic basis for Intrarosa [labrie2013local, labrie2017lowdose, ke2015serum]. Bone, brain, adipose, and skin show comparable but tissue-specific enzyme repertoires.

Adrenarche and adrenopause. The lifespan trajectory of DHEA-S is well characterized. Orentreich's cross-sectional [orentreich1984] and longitudinal [orentreich1992] studies in J Clin Endocrinol Metab quantified the rise of DHEA-S at adrenarche (ages 6, 8), its peak in the third decade, and its decline at approximately 1, 2% per year thereafter. By the seventh and eighth decades, serum DHEA-S is 10, 20% of young-adult values, an order-of-magnitude decline that distinguishes adrenopause from the comparatively modest fall in testosterone with age. The biological significance of this decline is contested: it correlates with multiple aspects of aging biology but causality is not established, and DHEA replacement trials in eugonadal older adults have been largely null on hard endpoints [nair2006nejm, corona2013meta].

Pharmacokinetics of oral and intravaginal DHEA. Oral DHEA undergoes first-pass sulfation to DHEA-S and rapidly raises serum DHEA-S; in young women given dexamethasone-suppressed baseline, single-dose oral DHEA produced predictable serum DHEA, DHEA-S, androstenedione, testosterone, and estradiol elevations dose-proportionally [arlt1998pk]. In healthy elderly subjects, daily oral DHEA 50 mg produced steady-state DHEA-S elevations within the young-adult reference range and a smaller increase in testosterone (more pronounced in women than men) [legrain2000pk]. Long-term daily oral DHEA in young men was characterized by Acacio 2004 [acacio2004pk]. Intravaginal prasterone produces tissue-level local action with serum DHEA, DHEA-S, testosterone, and estradiol concentrations remaining within postmenopausal reference ranges [labrie2013local, labrie2017lowdose, ke2015serum]. Sublingual and topical compounded routes have less published PK data and produce concentration profiles that depend strongly on excipient formulation.

Neurosteroid actions. DHEA and DHEA-S act at low affinity on GABA-A receptors (where DHEA-S is a negative allosteric modulator, contrasting with positive allosteric modulation by allopregnanolone), NMDA receptors, and the sigma-1 receptor [baulieu2000dheage]. These actions are pharmacologically distinct from androgen/estrogen-receptor-mediated effects and have been hypothesized to underlie reported mood and cognitive effects, though the clinical-trial signal for systemic DHEA on mood and cognition in healthy older adults is weak to absent.

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WADA / doping. The World Anti-Doping Agency lists DHEA as a prohibited anabolic agent under section S1.1b of the Prohibited List, both in- and out-of-competition. Oral DHEA reliably raises urinary testosterone and androsterone glucuronide metabolites and can produce positive testosterone:epitestosterone ratios and IRMS abnormalities used in anti-doping testing. Compounded DHEA preparations for individual clinical use are not exempted from sport-specific bans.

DHEA Research History

DHEA was isolated in 1934 by Butenandt from male urine and chemically characterized over the following decade. Its physiology as an adrenal C19 steroid was established by the 1950s, but its function, beyond being a precursor, remained obscure for decades. The age-related decline of DHEA-S was first quantified at scale by Orentreich and colleagues in cross-sectional [orentreich1984] and longitudinal [orentreich1992] studies in J Clin Endocrinol Metab, establishing the trajectory now called adrenopause.

Labrie's 1991 'intracrinology' paper in Mol Cell Endocrinol [labrie1991intracrinology] reframed DHEA as a substrate for tissue-specific local sex-steroid synthesis rather than a hormone with direct receptor agonism. This framework became the mechanistic basis for both the systemic and the local (intravaginal) DHEA literature. The first randomized replacement trial in eugonadal aging adults was Morales 1994 [morales1994], 50 mg/day oral DHEA for 3 months in men and women aged 40, 70 produced rises in DHEA-S to young-adult levels and improved subjective well-being, but the study was small (n=30) and end points were patient-reported.

The clearest clinical use case emerged in adrenal-insufficiency replacement. Arlt and Allolio's 1999 NEJM trial [arlt1999nejm] randomized 24 women with adrenal insufficiency to 50 mg/day DHEA versus placebo for 4 months and reported improvements in mood, fatigue, sexuality, and well-being scores, the first rigorous demonstration of a DHEA-replacement indication. Hunt 2000 [hunt2000] in JCEM reproduced the mood and fatigue findings in 39 Addison's patients. The DHEAge study by Baulieu and colleagues [percheron2003] [baulieu2000dheage, percheron2003] tested 50 mg/day for 1 year in 280 older adults and reported skin and libido improvements in women over age 70 but no benefit on muscle function. Nair 2006 NEJM [nair2006nejm], the definitive Mayo Clinic trial in 87 elderly men and 57 elderly women, reported no benefit of DHEA (or low-dose testosterone in men) on body composition, physical performance, insulin sensitivity, or quality of life at 2 years.

Bone and body-composition evidence accumulated through the 2000s. Villareal 2004 JAMA [villareal2004] showed reduced abdominal visceral fat and improved insulin sensitivity at 6 months in older adults on 50 mg/day. Jankowski's 2006 and 2008 JCEM papers [jankowski2006bone, jankowski2008estrogen] demonstrated bone-mineral-density gains in older adults with DHEA replacement, mediated by aromatization to estrogens. Weiss 2009 [weiss2009bone] extended the bone findings to 2 years. Gurnell 2008 [gurnell2008longterm] in JCEM reported the longest randomized DHEA trial, 12 months in primary adrenal insufficiency, with sustained subjective benefit. The Alkatib 2009 meta-

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analysis [alkatib2009meta] in JCEM integrated 10 randomized trials in adrenal insufficiency and concluded that DHEA produced a small but statistically significant improvement in health-related quality of life. The Endocrine Society's 2016 clinical practice guideline on primary adrenal insufficiency (Bornstein) [bornstein2016endo] acknowledges this evidence but stops short of routinely recommending DHEA replacement, citing variable response.

Female sexual function and menopause symptoms have produced mixed results for systemic DHEA. The Genazzani 2003, 2011 [genazzani2003neuroactive, genazzani2011climacteric] studies reported endocrine and climacteric-symptom effects of low-dose oral DHEA in postmenopausal women. Panjari 2009 J Sex Med [panjari2009sexual] randomized 93 postmenopausal women with low libido to 50 mg/day oral DHEA versus placebo for 6 months and found no significant improvement in the primary sexual-function endpoint. The 2015 Cochrane review (Scheffers) [scheffers2015cochrane] of 28 trials concluded that there is no clinically significant effect of DHEA on most menopausal symptoms; the women's androgen-therapy Endocrine Society guideline [wierman2014women] [wierman2014women] does not recommend systemic DHEA for sexual dysfunction outside of adrenal insufficiency. Davis 2011 JCEM [davis2011review] provides a contemporary clinical review of DHEA replacement in postmenopausal women.

The intravaginal prasterone program, Labrie's principal late-career work, generated the only successful FDA-approval pathway. Labrie 2011 Climacteric [labrie2011dyspareunia] was the first published phase III demonstration of intravaginal DHEA for dyspareunia. Subsequent phase III evidence including Archer 2015 Menopause [archer2015dyspareunia] confirmed efficacy on pain at sexual activity and vaginal trophic endpoints without clinically meaningful elevation of serum estrogens or androgens [labrie2013local, ke2015serum, labrie2017lowdose]. FDA approval of Intrarosa followed in November 2016 [fda_label_intrarosa]. Labrie's 2017 combined analysis in Menopause [labrie2017menocombined] integrated the program's evidence base.

IVF poor-responder protocols using DHEA were pioneered by Gleicher and Barad at the Center for Human Reproduction. The Wiser 2010 randomized trial [wiser2010ivf] in 33 poor responders reported improved live birth rates with 75 mg/day DHEA pre-stimulation. Yeung 2014 [yeung2014ivf] was a larger pilot RCT (n=72) that showed no significant benefit on the primary ovarian-response endpoint. The Cochrane review [nagels2015cochrane] [nagels2015cochrane] of androgens including DHEA in poor responders concluded that 'androgen pre-treatment may improve live birth rate' but evidence is low quality. The Zhang 2020 Hum Reprod Update network meta-analysis [zhang2020nma] integrated this evidence into broader poor-responder treatment strategies. DHEA for IVF remains broadly adopted in reproductive endocrinology practice despite the uncertain Cochrane verdict, a tension that should be communicated honestly to patients.

Lupus (SLE) and other autoimmune indications. Prasterone (Genelabs' GL-701) was tested in multiple phase III SLE trials, with Petri 2002 [petri2002sle] (steroid-sparing) and Petri 2004 [petri2004sleactive] (disease activity) showing modest signals on lupus activity in subgroups. The FDA did not ultimately approve prasterone for SLE; the development program closed without an approved indication. The

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mechanism rationale rests on the consistently low DHEA-S levels in patients with active SLE [verthelyi2001sle].

Pharmacokinetics and oral-route characterization spans Arlt 1998 [arlt1998pk], Legrain 2000 [legrain2000pk], and Acacio 2004 [acacio2004pk]. Quality-control studies of the commercial OTC DHEA supplement market, Parasrampuria 1998 in JAMA [parasrampuria1998], found that several over-the-counter products contained DHEA at concentrations substantially different from the labeled potency. The 503A compounding pathway, governed by USP <795> for nonsterile preparations [usp_795], addresses these identity and potency concerns when supplements cannot meet documented clinical need.

DHEA Timeline

1934 • Butenandt isolates DHEA from male urine

1984 • Orentreich et al [orentreich1984]. (JCEM) characterize age changes and sex differences in serum DHEA-S concentrations across adulthood, establishes the adrenopause trajectory

1991 • Labrie publishes 'Intracrinology' in Mol Cell Endocrinol, reframes DHEA as substrate for tissue-specific local sex-steroid synthesis [labrie1991intracrinology]

1992 • Orentreich's longitudinal DHEA-S study (JCEM) confirms the within-subject decline over time, distinct from cohort effects [orentreich1992]

1994 • DSHEA enacted in the US, oral DHEA classified as a dietary supplement and sold OTC

1994 • Morales et al [morales1994]. (JCEM), first randomized DHEA replacement trial in advancing-age men and women, small (n=30), positive on subjective well-being

1998 • Arlt et al [arlt1998pk]. (JCEM) characterize oral DHEA pharmacokinetics and peripheral conversion to androgens and estrogens

1998 • Parasrampuria et al [parasrampuria1998]. (JAMA) document substantial variability in DHEA content of commercial OTC supplement products

1999 • Arlt et al [arlt1999nejm]. (NEJM), DHEA replacement in women with adrenal insufficiency improves mood, fatigue, sexuality, and well-being scores in a 4-month placebo-controlled crossover trial

2000 • Hunt et al [hunt2000]. (JCEM) reproduce mood and fatigue findings in 39 patients with Addison's disease on DHEA replacement

2000 • Legrain et al [legrain2000pk]. (JCEM), pharmacokinetic and pharmacodynamic study of DHEA in healthy elderly subjects

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- **2000** • Baulieu et al [baulieu2000dheage]. (PNAS) publish DHEAge study results, sociobiomedical synthesis of DHEA aging trial in 280 older adults
- **2002** • Petri et al [petri2002sle]. (Arthritis Rheum) report prasterone steroid-sparing effects in women with SLE
- **2003** • Percheron et al [percheron2003]. (Arch Intern Med), DHEAge 1-year follow-up shows no effect on muscle function or cross-sectional area in older adults
- **2004** • Villareal & Holloszy (JAMA), DHEA reduces abdominal visceral fat and improves insulin action in elderly women and men over 6 months [villareal2004]
- **2004** • Acacio et al [acacio2004pk]. (Fertil Steril), long-term daily oral DHEA pharmacokinetics in healthy young men
- **2004** • Petri et al [petri2004sleactive]. (Arthritis Rheum), prasterone effects on SLE disease activity and symptoms in a second phase 3 trial
- **2005** • Dhatariya et al [dhatariya2005]. (Diabetes), DHEA replacement improves insulin sensitivity and lipids in hypoadrenal women
- **2006** • Nair et al [nair2006nejm]. (NEJM), definitive Mayo Clinic 2-year RCT in 87 men and 57 women aged 60+: no benefit of DHEA on body composition, performance, insulin sensitivity, or quality of life
- **2006** • Jankowski et al [jankowski2006bone]. (JCEM), DHEA replacement improves bone mineral density in older adults
- **2008** • Gurnell et al [gurnell2008longterm]. (JCEM), long-term (12-month) DHEA replacement RCT in primary adrenal insufficiency, sustained subjective benefit
- **2008** • Jankowski et al [jankowski2008estrogen]. (JCEM), DHEA-driven BMD increases mediated by aromatization to estrogens
- **2009** • Alkatib et al [alkatib2009meta]. (JCEM), systematic review and meta-analysis of 10 RCTs concludes DHEA produces a small but statistically significant improvement in health-related quality of life in adrenal insufficiency
- **2009** • Panjari et al [panjari2009sexual]. (J Sex Med), RCT of 50 mg/day oral DHEA in postmenopausal women with low libido: no significant effect on primary sexual-function endpoint
- **2009** • Weiss et al [weiss2009bone]. (Am J Clin Nutr), 1- and 2-year effects of DHEA on bone in older adults
- **2010** • Wiser et al [wiser2010ivf]. (Hum Reprod), randomized prospective trial: addition of DHEA to poor-responder IVF improves pregnancy rate

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2011 • Labrie et al [labrie2011dyspareunia]. (Climacteric), first phase III RCT of intravaginal prasterone for dyspareunia

2011 • Davis (JCEM) clinical review, DHEA replacement for postmenopausal women [davis2011review]

2013 • Labrie et al [labrie2013local]. (J Steroid Biochem Mol Biol), intravaginal prasterone provides local action without clinically significant change in serum estrogens or androgens

2013 • Corona et al [corona2013meta]. (JCEM), meta-analysis of placebo-controlled trials of DHEA in elderly men: only small effects

2014 • Yeung et al [yeung2014ivf]. (Fertil Steril), randomized controlled pilot trial of DHEA in poor responders: no significant effect on primary ovarian-response endpoint

2014 • Wierman et al [wierman2014women]. (JCEM), Endocrine Society guideline on androgen therapy in women, does not recommend systemic DHEA outside of adrenal insufficiency

2015 • Scheffers et al [scheffers2015cochrane]. (Cochrane), systematic review of 28 RCTs concludes no clinically significant effect of systemic DHEA on most menopausal symptoms

2015 • Nagels et al [nagels2015cochrane]. (Cochrane), androgens (DHEA or testosterone) for women undergoing assisted reproduction: low-quality evidence of possible live-birth-rate improvement

2015 • Archer et al [archer2015dyspareunia]. (Menopause), phase III RCT of intravaginal prasterone for pain at sexual activity (dyspareunia)

2015 • Ke et al [ke2015serum]. (J Steroid Biochem Mol Biol), serum sex steroid measurements after 12 weeks of intravaginal 0.50% DHEA: levels remain in postmenopausal range

2016 • Bornstein et al [bornstein2016endo]. (JCEM), Endocrine Society guideline on primary adrenal insufficiency acknowledges DHEA evidence but does not routinely recommend

2016 • FDA approves Intrarosa (prasterone 6.5 mg vaginal insert) for moderate-to-severe dyspareunia in postmenopausal women with vulvovaginal atrophy (November 17, 2016) [fda_label_intrarosa]

2017 • Labrie et al [labrie2017menocombined]. (Menopause), combined data of intravaginal prasterone against vulvovaginal atrophy of menopause

2017 • Labrie & Martel (Horm Mol Biol Clin Investig), confirm strictly local action of low-dose (6.5 mg) intravaginal DHEA [labrie2017lowdose]

2020 • Zhang et al [zhang2020nma]. (Hum Reprod Update), network meta-analysis of adjuvant treatment strategies for poor responders in IVF, including DHEA

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Clinical Contexts for DHEA

Moderate-to-severe dyspareunia in postmenopausal women with vulvovaginal atrophy

FDA APPROVED

FDA-approved indication for manufactured Intrarosa.

Intrarosa (prasterone 6.5 mg vaginal insert) is FDA-approved (November 2016) for moderate-to-severe dyspareunia, a symptom of vulvovaginal atrophy due to menopause [fda_label_intrarosa]. Pivotal phase III evidence by Labrie 2011 [labrie2011dyspareunia] and Archer 2015 [archer2015dyspareunia] demonstrated improvement in vaginal pH, percentage of parabasal and superficial cells, and most bothersome symptom (pain at sexual activity) compared with placebo over 12 weeks. PK and serum endocrine data [labrie2013local, ke2015serum, labrie2017lowdose] confirm that local action is achieved without clinically significant elevation of serum estradiol, testosterone, or DHEA-S beyond postmenopausal reference ranges. The combined-data analysis [labrie2017menocombined] integrates the program evidence.

Branded product: Intrarosa (prasterone vaginal insert, originally Endoceutics, marketed by Millicent Pharma)

Adrenal-androgen replacement in primary or secondary adrenal insufficiency

EMERGING

Off-label; supported by multiple randomized trials and a meta-analysis but not routinely recommended by the Endocrine Society 2016 guideline.

The Arlt 1999 NEJM trial [arlt1999nejm] in 24 women with adrenal insufficiency randomized to 50 mg/day oral DHEA versus placebo for 4 months reported improvements in mood, fatigue, sexuality, and overall well-being. Hunt 2000 [hunt2000] reproduced these findings in 39 Addison's patients. The Callies 2001 follow-on [callies2001women] examined body composition, leptin, bone turnover, and exercise capacity. Gurnell 2008 [gurnell2008longterm] extended the evidence to 12 months. The Alkatib 2009 meta-analysis [alkatib2009meta] of 10 RCTs concluded that DHEA in adrenal insufficiency produces a small but statistically significant improvement in health-related quality of life. Dhatariya 2005 [dhatariya2005] reported insulin-sensitivity and lipid effects in hypoadrenal women. The Endocrine Society 2016 guideline [bornstein2016endo] notes that DHEA replacement may be considered in adrenally-insufficient women with low libido or persistent symptoms despite optimal glucocorticoid and mineralocorticoid replacement, but stops short of routine recommendation. Brooke 2006 [brooke2006hypopit] reported reduction in GH dose requirement in hypopituitary women on DHEA replacement.

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IVF poor-responder pre-stimulation EMERGING

Off-label; widely adopted in fertility practice but supporting evidence is low quality per Cochrane.

DHEA pre-stimulation in women with diminished ovarian reserve was popularized by Gleicher and Barad at the Center for Human Reproduction in the 2000s. Wiser 2010 [wiser2010ivf] was a randomized prospective trial in 33 poor responders showing improved pregnancy rates with 75 mg/day DHEA pre-stimulation. Yeung 2014 [yeung2014ivf] was a larger pilot (n=72) that showed no significant effect on the primary ovarian-response endpoint. The Cochrane 2015 review by Nagels [nagels2015cochrane] concluded that androgen pre-treatment 'may improve live birth rate' but evidence is low quality. The Zhang 2020 Hum Reprod Update network meta-analysis [zhang2020nma] integrated this evidence across poor-responder strategies. DHEA for IVF remains broadly adopted in reproductive endocrinology practice despite the uncertain Cochrane verdict, patients should be counseled that benefit is plausible but unproven.

Systemic lupus erythematosus EMERGING

Off-label; phase III evidence (prasterone, Genelabs) showed modest signals but FDA approval was not granted; further development was discontinued.

Patients with active SLE have consistently low circulating DHEA-S [verthelyi2001sle]. Petri 2002 [petri2002sle] reported steroid-sparing effects of prasterone in women with SLE; Petri 2004 [petri2004sleactive] examined effects on disease activity and symptoms. The Genelabs prasterone development program (GL-701) did not produce an FDA-approved SLE indication. Use for SLE remains investigational with modest published evidence.

Bone mineral density in older adults EMERGING

Off-label; supported by randomized trials with effect sizes that are statistically significant but smaller than those of bisphosphonates or estrogen replacement.

Jankowski 2006 [jankowski2006bone] and 2008 [jankowski2008estrogen] reported significant gains in lumbar spine and hip BMD with 12-month oral DHEA 50 mg/day in older adults, with mediation by aromatization to estrogens. Weiss 2009 [weiss2009bone] extended findings to 2 years. The Nair 2006 NEJM trial [nair2006nejm] did not find body-composition or strength benefit at 2 years but did not power for BMD. The Endocrine Society women's androgen guideline [wierman2014women] does not recommend systemic DHEA for osteoporosis prevention; FDA-approved bone-density agents remain first-line.

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Postmenopausal sexual function and well-being EMERGING

Off-label; systematic-review evidence does not support routine systemic DHEA for menopausal symptoms.

The Panjari 2009 J Sex Med trial [panjari2009sexual] randomized 93 postmenopausal women with low libido to oral DHEA 50 mg/day versus placebo for 6 months and found no significant effect on the primary sexual-function endpoint. The 2015 Cochrane review by Scheffers [scheffers2015cochrane] of 28 RCTs concluded that DHEA does not produce a clinically significant effect on most menopausal symptoms. Davis 2011 [davis2011review] and Genazzani 2011 [genazzani2011climacteric] reviewed the field and reported small effects in subgroups; Genazzani 2003 [genazzani2003neuroactive] examined endocrine and neuroactive steroid effects. The Endocrine Society 2014 women's androgen guideline [wierman2014women] does not recommend systemic DHEA for sexual dysfunction outside of adrenal insufficiency.

Cognition, mood, and aging in eugonadal elderly EMERGING

Off-label; randomized evidence in healthy older adults is largely null or modest.

The Nair 2006 NEJM trial [nair2006nejm], 87 men and 57 women aged 60, 88 randomized to DHEA, low-dose testosterone (men), or placebo for 2 years, found no benefit on body composition, physical performance, insulin sensitivity, or quality of life. The DHEAge study [baulieu2000dheage] reported small skin and libido improvements in women over 70 but no muscle-function benefit. Schmidt 2005 [schmidt2005depression] reported antidepressant effects of DHEA monotherapy in midlife-onset major and minor depression, one of the few positive mood signals in the systemic-DHEA literature. Wolkowitz 1997 [wolkowitz1997depression] reported an earlier signal in major depression. Corona 2013 [corona2013meta], meta-analysis of placebo-controlled trials in elderly men, concluded effects are small and inconsistent [percheron2003]. The Rabijewski 2020 Polish Menopause Society position statement [rabijewski2020] reviews the European clinical landscape.

Off-Label Uses of DHEA

Adrenal-androgen replacement in primary or secondary adrenal insufficiency EMERGING

Off-label; consistent randomized evidence and meta-analytic support, but Endocrine Society does not routinely recommend.

Multiple RCTs [arlt1999nejm, hunt2000, gurnell2008longterm] and a meta-analysis [alkatib2009meta] support DHEA replacement for symptomatic women with adrenal insufficiency who remain symptomatic despite optimal glucocorticoid and mineralocorticoid replacement. The Endocrine Society 2016 guideline [bornstein2016endo] permits but does not routinely recommend.

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Diminished ovarian reserve / IVF poor-responder protocols EMERGING

Off-label; broadly adopted in reproductive endocrinology, supporting evidence is low quality per Cochrane.

Empiric pre-stimulation with oral DHEA 25 mg three times daily (or 75 mg/day) for 6, 12 weeks is widely used in poor-responder IVF cycles [zhang2020nma]. The Wiser 2010 RCT [wiser2010ivf] was positive on pregnancy rate; the Yeung 2014 [yeung2014ivf] pilot was null on the primary endpoint; the Cochrane 2015 review [nagels2015cochrane] concluded evidence is low quality.

Systemic lupus erythematosus EMERGING

Off-label; phase III evidence did not lead to FDA approval; development discontinued.

Petri 2002 and 2004 [petri2002sle, petri2004sleactive] reported modest steroid-sparing and symptom signals; the Genelabs prasterone development program did not produce approval [verthelyi2001sle]. Use is investigational.

FDA-Approved Uses of DHEA

Brand	Indication	Year	Route
Intrarosa (prasterone)	Moderate-to-severe dyspareunia, a symptom of vulvovaginal atrophy, due to menopause	2016	Intravaginal insert

Intrarosa is a prasterone (DHEA) 6.5 mg vaginal insert FDA-approved on November 16, 2016, for moderate-to-severe dyspareunia, a symptom of vulvovaginal atrophy, due to menopause [fda_label_intrarosa]. Approval was based on phase III evidence by Labrie 2011 [labrie2011dyspareunia] and Archer 2015 [archer2015dyspareunia] demonstrating improvement in the co-primary endpoints (vaginal pH, percentage of parabasal cells, percentage of superficial cells, and most bothersome symptom of pain at sexual activity) compared with placebo over 12 weeks. Serum sex-steroid measurements during treatment [labrie2013local, ke2015serum, labrie2017lowdose] remained within postmenopausal reference ranges, supporting the mechanistic claim of intracrine local action.

Intrarosa is the only FDA-approved DHEA product. Oral DHEA is sold OTC as a dietary supplement and is not FDA-approved for any indication. Compounded oral DHEA, intravaginal DHEA, and topical DHEA preparations are not FDA-approved products.

Compounded DHEA (503A)

Compounded DHEA under 503A is dispensed only when the prescribing clinician documents a patient-specific clinical need that neither the FDA-approved Intrarosa product nor the OTC dietary-supplement

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market can meet [fda_label_intrarosa]. DHEA occupies an unusual regulatory position: oral DHEA is sold OTC as a dietary supplement under DSHEA 1994, while the only FDA-approved DHEA prescription product is intravaginal Intrarosa. This means compounded oral DHEA is approached with the FDA 'essentially-a-copy' framework in mind, even though there is no FDA-approved oral comparator [fda_essentially_a_copy].

Documented patient-specific clinical needs typically fall into four categories: (1) sub-OTC dose individualization, particularly for women, where typical OTC capsule strengths (25 mg, 50 mg, 100 mg) exceed the 10, 25 mg doses commonly appropriate for female replacement based on the published trials [arlt1999nejm, hunt2000, gurnell2008longterm]; (2) sublingual or topical formulation when oral first-pass sulfation must be reduced or when the patient cannot tolerate oral dosing; (3) excipient sensitivity to components of OTC supplements (titanium dioxide, magnesium stearate, common allergens); and (4) USP <795>-compliant identity, potency, and purity assurance, addressing the well-documented variability of commercial OTC DHEA content [parasrampuria1998, usp_795] [fda_label_intrarosa].

Compounded preparations are not bioequivalent to Intrarosa and are not bioequivalent to any specific OTC product [fda_label_intrarosa]. PK characteristics depend on the route, excipient base, and concentration. Sublingual compounded DHEA is hypothesized to bypass first-pass sulfation and produce a different DHEA-to-DHEA-S ratio than oral capsules, published PK data for sublingual DHEA are limited [acacio2004pk, arlt1998pk, legrain2000pk] and individualized monitoring is appropriate.

DHEA is a WADA-prohibited anabolic agent in- and out-of-competition for athletes subject to anti-doping testing [fda_label_intrarosa]. Compounded preparations do not exempt the user from sport-specific bans. Patients who are competitive athletes must be informed before dispensing.

RonanRx does not fill DHEA prescriptions that read as routine substitution for an OTC supplement without documented clinical rationale, and is particularly attentive to the 'essentially-a-copy' framing for oral DHEA. Pharmacist review confirms a documented clinical reason that the OTC market or Intrarosa cannot meet the patient's need [fda_label_intrarosa].

DHEA Formulations and Routes

Form	Concentration	Description
Compounded oral capsule (micronized DHEA)	Custom, typically 5, 10, 15, 20, 25, or 50 mg per capsule; women's replacement doses commonly 10, 25 mg, men's commonly 25, 50 mg	Nonsterile oral capsule compounded under USP <795> standards on patient-specific prescription. Micronized API supports more reproducible dissolution and absorption than coarser supplement-grade material.
	Custom, typically 5, 25 mg	Sublingual delivery is hypothesized to partially bypass first-pass hepatic sulfation, altering the DHEA-to-

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Form	Concentration	Description
Compounded sublingual troche or lozenge		DHEA-S ratio relative to oral capsules. Published PK data specific to sublingual DHEA are limited.
Compounded topical cream or gel	Custom, commonly 5, 10 mg per dose	Topical/transdermal delivery for selected patients with intolerance to oral routes. Systemic exposure depends on excipient base and skin site; pharmacokinetics are not well characterized in the published literature.
FDA-approved intravaginal insert (reference product)	6.5 mg prasterone per insert	Intrarosa is the only FDA-approved DHEA product, supplied as 6.5 mg prasterone vaginal inserts for once-daily use in postmenopausal dyspareunia.
OTC oral capsule (dietary supplement, not FDA-approved)	Commonly 25 mg, 50 mg, or 100 mg	Sold OTC under DSHEA 1994. Independent analyses have documented substantial variability between labeled and assayed DHEA content in commercial supplement products [parasrampuria1998]. Not subject to FDA pre-market efficacy or potency review.

Routes used in published literature: oral, sublingual, topical, transdermal, vaginal.

DHEA Dosing

Route	Population	Range	Duration	Study type
Intravaginal	Postmenopausal women with moderate-to-severe dyspareunia from vulvovaginal atrophy (Intrarosa labeled regimen)	6.5 mg prasterone vaginal insert once daily at bedtime	Indefinite while clinically beneficial	FDA-approved labeled regimen
Oral	Women with primary or secondary adrenal insufficiency	25, 50 mg once daily in the morning; titrated to symptom response and to DHEA-S in young-adult female reference range	Indefinite while clinically beneficial	Randomized trial-derived (off-label)
Oral	IVF poor-responder pre-stimulation (off-label)	25 mg three times daily (75 mg/day total) for 6, 12 weeks prior to ovarian stimulation	6, 12 weeks pre-cycle, often continued through stimulation	Randomized trial-derived (off-label, low-quality evidence per Cochrane)

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Route	Population	Range	Duration	Study type
Oral	Aging adults / postmenopausal women (research dosing)	25, 50 mg once daily; most trials used 50 mg/day	3, 24 months in published RCTs	Randomized trial-derived (largely null on hard endpoints in eugonadal aging populations)
Oral	Midlife-onset major or minor depression (off-label)	Titrated 90, 450 mg/day across 6 weeks in the Schmidt 2005 protocol	6 weeks in the published RCT	Randomized trial-derived (off-label)

Doctor-prescribed and titrated. Female replacement doses are commonly 10, 25 mg/day, lower than typical OTC capsule strengths and one rationale for compounding. Male replacement (where indicated, typically only in primary adrenal insufficiency) is commonly 25, 50 mg/day [arlt1999nejm; hunt2000; gurnell2008longterm]. Intravaginal Intrarosa is dosed at 6.5 mg once daily per label and should not be substituted with compounded oral DHEA for the dyspareunia indication [fda_label_intrarosa].

Target DHEA-S to the young-adult sex- and age-specific reference range. In women, free testosterone, total testosterone, SHBG, and estradiol should be monitored periodically, androgenization (acne, oily skin, hirsutism, scalp hair thinning) is the most common dose-limiting effect and prompts dose reduction. Higher doses (>50 mg/day in women) are commonly associated with supraphysiologic testosterone elevations [nagels2015cochrane].

IVF poor-responder pre-stimulation protocols typically use 75 mg/day for 6, 12 weeks. The Cochrane review notes evidence is low quality; patients should be counseled accordingly. Compounded DHEA mirrors the supplement-derived dose strategy rather than introducing novel regimens [wiser2010ivf].

DHEA Safety

The dominant safety concerns with systemic DHEA are dose-dependent androgenic effects, particularly in women: acne, oily skin, hirsutism, scalp hair thinning, and (less commonly) deepening of the voice. These reflect peripheral conversion of DHEA to testosterone and DHT, and they are the most common reason for dose reduction or discontinuation in clinical trials [10][26][30]. Acne and oily skin are reported in 10, 30% of women on 50 mg/day in the published trials and resolve with dose reduction.

Estrogenic effects from peripheral aromatization (breast tenderness, vaginal spotting, endometrial stimulation) are uncommon at typical replacement doses but have been reported. Long-term safety data on endometrial effects of systemic DHEA in postmenopausal women are limited; the Endocrine Society 2014 women's androgen-therapy guideline [40] does not recommend long-term systemic DHEA outside of adrenal insufficiency in part because of these uncertainties.

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Intravaginal prasterone (Intrarosa) has a distinct safety profile dominated by application-site effects (vaginal discharge most commonly reported) rather than systemic androgenic effects, consistent with the intracrine local mechanism and the absence of clinically meaningful elevations in serum sex steroids [3][8][43][47]. The Intrarosa label warns against use in women with undiagnosed abnormal genital bleeding or with a history of, or active or suspected, breast cancer.

Hormone-sensitive cancer considerations: because DHEA is converted to estradiol and testosterone in peripheral tissues, it is contraindicated by most clinicians in patients with hormone-sensitive cancers (breast, prostate, endometrial) regardless of route. The FDA Intrarosa label includes this contraindication for the vaginal product [51]. There are no high-quality long-term safety data on breast or prostate cancer outcomes with systemic DHEA in the general population; the Endocrine Society 2014 women's guideline notes this gap [40].

Other notable considerations: insomnia or mood activation has been reported at higher doses; lipid effects are modest and inconsistent (small reductions in HDL in some trials of women); insulin sensitivity effects are inconsistent [19]. DHEA is a WADA-prohibited anabolic agent, athletes subject to anti-doping testing should not use DHEA in any route or formulation. OTC supplement quality concerns documented by Parasrampuria 1998 [9] are an additional safety consideration favoring compounded preparations or FDA-approved Intrarosa when clinically indicated.

Contraindications

DHEA is contraindicated in patients with known or suspected hormone-sensitive cancers, breast, endometrial, prostate, or other malignancies that may be stimulated by androgens or estrogens. The FDA Intrarosa label specifically contraindicates use in women with undiagnosed abnormal genital bleeding or with known, suspected, or history of breast cancer [51] [40].

Pregnancy and lactation: DHEA is contraindicated in pregnancy. Conversion to estrogens and androgens makes systemic DHEA inappropriate during pregnancy. The Intrarosa label states the product is not indicated for use in women of reproductive potential [40].

Caution in patients with active liver disease (hepatic sulfation is the primary pharmacokinetic determinant of DHEA-S levels), polycystic ovary syndrome (DHEA can worsen hyperandrogenism), and uncontrolled cardiovascular disease (estrogenic effects are theoretically a concern at higher systemic doses, though clinical-trial data do not show a cardiovascular signal at typical replacement doses) [40].

Athletes subject to WADA, NCAA, or sport-specific anti-doping testing should not use DHEA in any route or formulation, DHEA is a prohibited anabolic agent in- and out-of-competition [40].

Drug interactions

DHEA is metabolized via sulfation (SULT2A1), oxidation (3β-HSD, 17β-HSD), and aromatization (CYP19A1). Drug interactions through these pathways are clinically modest at typical replacement doses [51]. Important pharmacologic considerations include the additive effects with concurrent androgen or estrogen

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therapy (testosterone, estradiol, progestins), combination use raises the risk of androgenization or estrogenization beyond what monotherapy would produce.

Carbamazepine, phenytoin, and other inducers of hepatic metabolism may lower DHEA and DHEA-S levels. Conversely, oral contraceptives and exogenous estrogens raise SHBG and may alter the free testosterone produced from DHEA conversion. Anastrozole and other aromatase inhibitors block peripheral aromatization, blunting the estrogenic arm of DHEA action and altering the androgen-to-estrogen ratio.

Glucocorticoids suppress adrenal DHEA-S production and can be expected to interact with DHEA replacement; in adrenal insufficiency, DHEA replacement is added to glucocorticoid replacement rather than substituted for it [10][28].

Adverse events

Across the published systemic DHEA trials in women, the most common adverse events are androgenic: acne (10, 30% on 50 mg/day in 4-month to 12-month trials), oily skin, mild hirsutism, scalp hair thinning, and rarely deepening of the voice [10][11][30]. These are dose-dependent and reversible with dose reduction. Women at typical replacement doses of 10, 25 mg/day report fewer androgenic events than at the 50 mg/day commonly used in research protocols.

Estrogenic adverse events are uncommon at typical replacement doses but include breast tenderness, vaginal spotting (in postmenopausal women), and theoretical concerns about endometrial stimulation. Long-term endometrial safety data are limited.

Insomnia and mood activation have been reported at higher doses (>100 mg/day) and are characteristic of the morning-dosing regimen. Lipid effects are modest and inconsistent across trials; small reductions in HDL cholesterol have been reported in some women's studies.

Insulin sensitivity effects are inconsistent: Villareal 2004 [19] reported improved insulin action over 6 months; Nair 2006 [26] reported no change at 2 years; Dhatariya 2005 [20] reported improved insulin sensitivity in hypoadrenal women specifically.

Intravaginal prasterone (Intrarosa) adverse events in the phase III program were predominantly application-site related; vaginal discharge was the most common event, occurring in approximately 6% of treated participants in the pivotal trials [33][42]. Discontinuation rates were low. Systemic adverse events were uncommon, consistent with the lack of clinically meaningful elevation in serum sex steroids [38][43].

Long-term cancer outcome data on systemic DHEA are limited; this is the principal reason the Endocrine Society women's androgen-therapy guideline [40] does not recommend systemic DHEA outside of adrenal insufficiency despite the symptom-relief evidence base.

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Monitoring DHEA Therapy

Baseline assessment: serum DHEA-S, total and free testosterone, SHBG, estradiol, lipid panel, hepatic function, and clinical androgenization survey [bornstein2016endo]. In women of menopausal age, mammographic screening and endometrial assessment per general menopause-care guidelines. Pregnancy status confirmed in patients of reproductive potential.

On therapy: serum DHEA-S targeted to young-adult sex-specific reference range; free testosterone monitored in women to detect supraphysiologic exposure; androgenization survey at each visit (acne, oily skin, hirsutism, voice changes, scalp hair) [wierman2014women] [labrie2013local]. Lipid panel annually. Endometrial assessment if abnormal uterine bleeding develops in postmenopausal women [fda_label_intrarosa].

Intravaginal Intrarosa does not require serum sex-steroid monitoring at the labeled dose, as serum levels remain in the postmenopausal range [wierman2014women] [ke2015serum; labrie2017lowdose]. Application-site assessment is appropriate at each visit.

DHEA in Special Populations

DHEA Evidence Quality

Evidence for intravaginal prasterone (Intrarosa) is the strongest in the DHEA literature: multiple phase III randomized double-blind placebo-controlled trials [labrie2011dyspareunia, archer2015dyspareunia, labrie2017menocombined] demonstrated effects on co-primary endpoints (vaginal pH, parabasal and superficial cell percentages, most bothersome symptom of pain at sexual activity) at 12 weeks, with mechanistic PK and serum-endocrine data [labrie2013local, ke2015serum, labrie2017lowdose] supporting local intracrine action without clinically meaningful systemic estrogen or androgen elevation [fda_label_intrarosa]. FDA approval followed in November 2016.

Evidence for systemic oral DHEA in adrenal insufficiency is moderate: consistent positive randomized signals from Arlt 1999 [arlt1999nejm], Hunt 2000 [hunt2000], Gurnell 2008 [gurnell2008longterm], and Dhatariya 2005 [dhatariya2005], integrated by the Alkatib 2009 meta-analysis [alkatib2009meta] [fda_label_intrarosa]. Effect sizes on quality-of-life endpoints are small but statistically robust. The Endocrine Society 2016 guideline [bornstein2016endo] acknowledges this evidence but does not routinely recommend, citing variable response and the absence of long-term outcome data.

Evidence for systemic DHEA in eugonadal aging adults is largely null or modest: Nair 2006 NEJM [nair2006nejm], Percheron 2003 [percheron2003] (DHEAge), and Corona 2013 meta-analysis

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[corona2013meta] do not support DHEA replacement for body composition, physical performance, insulin sensitivity, or general aging endpoints [fda_label_intrarosa]. Modest signals exist for bone mineral density [jankowski2006bone, jankowski2008estrogen, weiss2009bone] mediated by aromatization to estrogens, but effects are smaller than FDA-approved bone-density agents.

Evidence for IVF poor-responder pre-stimulation is low quality per the Cochrane review [nagels2015cochrane] integrating Wiser 2010 [wiser2010ivf] (positive on pregnancy rate, n=33), Yeung 2014 [yeung2014ivf] (null on primary endpoint, n=72), and other small trials [fda_label_intrarosa]. The Zhang 2020 network meta-analysis [zhang2020nma] places DHEA in the context of broader adjuvant strategies. Practice adoption exceeds the evidence base; patient counseling should reflect this.

Evidence for SLE is insufficient. Two Petri-led phase III trials [petri2002sle, petri2004sleactive] reported modest signals but did not produce an FDA-approved indication; the prasterone development program (GL-701) was discontinued [fda_label_intrarosa].

Evidence for mood and depression is limited but includes one positive monotherapy RCT in midlife-onset depression [schmidt2005depression] and earlier signals [wolkowitz1997depression] [fda_label_intrarosa]. Use for psychiatric indications is investigational.

Critically, evidence for compounded DHEA specifically does not exist as a separate program, compounded preparations rely on the manufactured (Intrarosa) and supplement-derived (oral systemic) PK and clinical evidence base, with the caveat that compounded preparations may differ in concentration, excipient profile, and route [fda_label_intrarosa]. The Parasrampuria 1998 JAMA quality study [parasrampuria1998] documents the gap that compounded preparations under USP <795> [usp_795] are positioned to address relative to the unregulated OTC supplement market.

Major DHEA Clinical Studies

Study	Design	Participants	Duration	Finding
Arlt et al. (1999, NEJM), DHEA in adrenal insufficiency	Randomized double-blind placebo-controlled crossover trial of oral DHEA 50 mg/day in women with adrenal insufficiency	24	4 months per arm	Improved mood, fatigue, sexuality, and overall well-being scores vs placebo; landmark trial establishing DHEA replacement as a clinical option in adrenal insufficiency [arlt1999nejm]
Hunt et al. (2000, JCEM), DHEA in Addison's disease	Randomized double-blind placebo-controlled trial of oral DHEA in patients with Addison's disease	39	12 weeks	Improvements in mood and fatigue scores with DHEA replacement, reproducing the Arlt 1999 signal [hunt2000]

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Study	Design	Participants	Duration	Finding
Gurnell et al. (2008, JCEM), long-term DHEA in primary adrenal insufficiency	Randomized double-blind placebo-controlled trial of oral DHEA 50 mg/day in primary adrenal insufficiency	—	12 months	Sustained improvement in subjective well-being and quality of life over the longest randomized DHEA trial in adrenal insufficiency [gurnell2008longterm]
Callies et al. (2001, JCEM), body composition in adrenal insufficiency	Randomized placebo-controlled DHEA trial in women with adrenal insufficiency examining body composition, leptin, bone turnover, and exercise capacity	—	4 months	Modest effects on body composition and exercise capacity; supports the Arlt 1999 mechanism case [callies2001women]
Alkatib et al. (2009, JCEM), meta-analysis of DHEA in adrenal insufficiency	Systematic review and meta-analysis of 10 randomized placebo-controlled trials	Pooled approximately 500 women	—	Small but statistically significant improvement in health-related quality of life with DHEA in adrenal insufficiency; effect sizes modest [alkatib2009meta]
Nair et al. (2006, NEJM), DHEA in elderly women, DHEA or testosterone in elderly men	Randomized double-blind placebo-controlled trial of DHEA 75 mg/day in men and 50 mg/day in women (plus low-dose testosterone arm in men)	144	2 years	No benefit of DHEA on body composition, physical performance, insulin sensitivity, or quality of life, definitive negative trial in eugonadal aging adults [nair2006nejm]
Percheron et al. (2003, Arch Intern Med), DHEAge muscle function	Double-blind placebo-controlled trial of 1-year oral DHEA 50 mg/day in 60, 80-year-old adults	280	12 months	No effect on muscle function or cross-sectional area; small skin and libido improvements reported in women over 70 in the broader DHEAge program [percheron2003; baulieu2000dheage]
Villareal & Holloszy (2004, JAMA), abdominal fat and insulin action	Randomized double-blind placebo-controlled trial of DHEA 50 mg/day in	56	6 months	Reduced abdominal visceral fat and improved insulin action with DHEA, one of the few positive systemic-DHEA trials on

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Study	Design	Participants	Duration	Finding
	elderly men and women			metabolic endpoints [villareal2004]
Jankowski et al. (2006, JCEM), bone mineral density	Randomized double-blind placebo-controlled trial of DHEA 50 mg/day in older adults	—	12 months	Significant gains in lumbar spine and hip BMD with DHEA, mediated by aromatization to estrogens (companion 2008 paper [jankowski2008estrogen]) [jankowski2006bone]
Weiss et al. (2009, Am J Clin Nutr), 1- and 2-year bone effects	Extension of the Jankowski older-adult DHEA RCT to 2 years	—	24 months	Sustained bone-density gains with DHEA replacement over 2 years [weiss2009bone]
Labrie et al. (2011, Climacteric), first phase III intravaginal prasterone for dyspareunia	Phase III randomized double-blind placebo-controlled trial of intravaginal prasterone (DHEA) in postmenopausal women with vulvovaginal atrophy	—	12 weeks	Significant improvement in vaginal pH, percentage of parabasal and superficial cells, and pain at sexual activity vs placebo, pivotal evidence for Intrarosa approval pathway [labrie2011dyspareunia]
Archer et al. (2015, Menopause), phase III prasterone for dyspareunia	Phase III randomized double-blind placebo-controlled trial of intravaginal prasterone 6.5 mg daily in postmenopausal women with moderate-to-severe dyspareunia	—	12 weeks	Significant improvement in co-primary endpoints (vaginal pH, parabasal cells, superficial cells, pain at sexual activity); supported FDA approval of Intrarosa in 2016 [archer2015dyspareunia]
Labrie et al. (2017, Menopause), combined intravaginal prasterone data	Integrated combined-data analysis of the Intrarosa phase III program against vulvovaginal atrophy of menopause	—	—	Reproducible effect on dyspareunia and vaginal trophic endpoints across trials; supports the local intracrine mechanism [labrie2017menocombined]
Wiser et al. (2010, Hum Reprod),	Randomized prospective trial of	33		Improved pregnancy rate vs placebo, small study,

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Study	Design	Participants	Duration	Finding
DHEA in IVF poor responders	DHEA 75 mg/day for 6+ weeks pre-stimulation in poor-responder IVF		Pre-cycle treatment + IVF cycle	foundational for DHEA-in-IVF practice adoption [wiser2010ivf]
Yeung et al. (2014, Fertil Steril), DHEA in poor-responder IVF	Randomized controlled pilot trial of DHEA pre-stimulation in poor responders	72	16 weeks pre-stimulation + IVF cycle	No significant effect on primary ovarian-response endpoint; tempers the Wiser 2010 enthusiasm [yeung2014ivf]
Nagels et al. (2015, Cochrane), androgens for poor-responder IVF	Cochrane systematic review of DHEA or testosterone pre-treatment in women undergoing assisted reproduction	—	—	Low-quality evidence suggests androgen pre-treatment may improve live birth rate; further high-quality trials needed [nagels2015cochrane]
Petri et al. (2002, Arthritis Rheum), prasterone steroid-sparing in SLE	Phase III randomized double-blind placebo-controlled trial of prasterone 200 mg/day in women with SLE	—	7, 9 months	Modest steroid-sparing effect with prasterone; evidence was insufficient for FDA approval as Genelabs GL-701 [petri2002sle]
Petri et al. (2004, Arthritis Rheum), prasterone for SLE activity	Phase III randomized double-blind placebo-controlled trial of prasterone in women with active SLE	—	—	Modest signals on disease activity and symptoms in subgroups; not sufficient for FDA approval of prasterone for SLE [petri2004sleactive]
Scheffers et al. (2015, Cochrane), DHEA for peri- and postmenopausal women	Cochrane systematic review of 28 RCTs of DHEA in peri- or postmenopausal women	—	—	No clinically significant effect of systemic DHEA on most menopausal symptoms; cited by Endocrine Society guidance [scheffers2015cochrane]
Panjari et al. (2009, J Sex Med), DHEA for postmenopausal low libido	Randomized double-blind placebo-controlled trial of oral DHEA 50 mg/day in postmenopausal women with low libido	93	6 months	No significant improvement in primary sexual-function endpoint; tempers enthusiasm for systemic DHEA in HSDD outside adrenal insufficiency [panjari2009sexual]

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Study	Design	Participants	Duration	Finding
Corona et al. (2013, JCEM), meta-analysis in elderly men	Meta-analysis of placebo-controlled trials of DHEA supplementation in elderly men	—	—	Small effects on body composition and quality of life; effect sizes modest and inconsistent across trials [corona2013meta]
Schmidt et al. (2005, Arch Gen Psychiatry), DHEA for midlife depression	Randomized double-blind placebo-controlled trial of DHEA monotherapy (titrated to 450 mg/day) in midlife-onset major and minor depression	—	6 weeks	Significant antidepressant effects vs placebo, one of the few positive systemic-DHEA mood signals [schmidt2005depression]
Morales et al. (1994, JCEM), first DHEA replacement trial	Randomized placebo-controlled trial of DHEA 50 mg/day in advancing-age men and women	30	3 months	Restoration of DHEA-S to young-adult levels; improved subjective well-being, small landmark trial that initiated the systemic-DHEA replacement field [morales1994]
Arlt et al. (1998, JCEM), oral DHEA pharmacokinetics	PK study of oral DHEA after dexamethasone suppression in young healthy women	—	—	Characterized peripheral conversion to androstenedione, testosterone, and estrogens; established the dose-response relationship for compounded oral dosing [arlt1998pk]
Legrain et al. (2000, JCEM), DHEA PK/PD in elderly subjects	PK and PD study of daily oral DHEA in healthy elderly men and women	—	—	Daily 50 mg restored serum DHEA-S to young-adult levels; small testosterone rise (greater in women); androstenedione and estradiol increases observed [legrain2000pk]
Acacio et al. (2004, Fertil Steril), long-term DHEA PK in young men	Pharmacokinetics of long-term daily oral DHEA in healthy young men	—	—	Characterized steady-state DHEA, DHEA-S, androstenedione, testosterone, and estrogen profiles with chronic dosing [acacio2004pk]
Parasrampuria et al. (1998, JAMA), OTC	Independent assay of commercial OTC	—	—	Substantial variability between labeled and assayed DHEA content; demonstrates a quality

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Study	Design	Participants	Duration	Finding
DHEA supplement quality	DHEA dietary-supplement products			gap in the OTC supplement market that compounded preparations under USP <795> can address [parasrampuria1998]
Labrie & Martel (2017, Horm Mol Biol Clin Investig), low-dose intravaginal DHEA serum profile	Serum endocrine assessment during 6.5 mg/day intravaginal prasterone	—	—	Serum estrogens, androgens, and metabolites remained within postmenopausal reference ranges, supports strictly local action of Intrarosa [labrie2017lowdose]
Orentreich et al. (1984, JCEM), DHEA-S age trajectory	Cross-sectional measurement of serum DHEA-S across adulthood	—	—	Established the age-related decline of DHEA-S, the adrenopause trajectory [orentreich1984]

DHEA Pharmacokinetics & Pharmacodynamics

Pharmacokinetics

Oral DHEA undergoes rapid hepatic and intestinal sulfation by SULT2A1 to DHEA-S, which is the predominant circulating form (half-life ~7, 10 hours). Free DHEA has a much shorter half-life (~1 hour). After dexamethasone suppression of endogenous adrenal production, single-dose oral DHEA 50 mg in young women produced serum DHEA, DHEA-S, androstenedione, testosterone, and estradiol rises dose-proportionally [arlt1998pk]. Daily oral DHEA 50 mg in healthy elderly subjects restored DHEA-S to young-adult reference values, with smaller increases in testosterone (more pronounced in women than men) [legrain2000pk]. Long-term daily oral DHEA in young men produced steady-state DHEA-S elevations with concurrent androstenedione and testosterone rises [acacio2004pk].

Intravaginal prasterone (Intrarosa 6.5 mg daily) produced local tissue action, restored vaginal pH and cell maturation, without clinically significant elevation of serum DHEA, DHEA-S, testosterone, or estradiol beyond the postmenopausal reference range [labrie2013local, ke2015serum, labrie2017lowdose]. This PK profile is the mechanistic basis for the FDA approval of Intrarosa and distinguishes intravaginal from oral systemic dosing.

Compounded sublingual and topical preparations have less published PK data. Sublingual delivery is hypothesized to partially bypass first-pass sulfation, producing a higher DHEA-to-DHEA-S ratio than oral capsules, but published PK studies are limited and excipient-dependent. Compounded preparations are not bioequivalent to OTC supplement products or to Intrarosa.

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Pharmacodynamics

DHEA functions predominantly as an intracrine substrate [arlt1999nejm]. Pharmacodynamic effects depend on tissue-specific expression of steroid sulfatase, 3β-HSD, 17β-HSD, aromatase, and 5α-reductase, each tissue converts circulating DHEA and DHEA-S to the active androgen or estrogen profile appropriate to that tissue [labrie1991intracrinology, labrie2013local].

Measured clinical effects in randomized trials include: serum DHEA-S elevation to young-adult ranges; modest testosterone rise (greater in women than men); small estradiol rise; vaginal pH normalization and cell maturation index improvement with intravaginal dosing; improved mood, fatigue, and well-being scores in adrenal insufficiency; modest bone-density gains in older adults mediated by aromatization to estrogens; and dose-dependent androgenization (acne, oily skin, hirsutism) particularly in women [arlt1999nejm; jankowski2008estrogen].

Effects on body composition, physical performance, cognition, and metabolic endpoints in eugonadal aging adults are largely null or modest [nair2006nejm, percheron2003, corona2013meta] [jankowski2008estrogen].

↓↑ Comparing DHEA Formulations

The FDA-approved Intrarosa vaginal insert (6.5 mg prasterone) is the only DHEA product with FDA pre-market efficacy and quality review [labrie2013local; fda_label_intrarosa]. It is the appropriate choice for postmenopausal dyspareunia from vulvovaginal atrophy. Serum sex-steroid levels remain in the postmenopausal range during use [ke2015serum].

OTC oral DHEA capsules (commonly 25, 50, or 100 mg) are sold as dietary supplements without FDA pre-market quality or efficacy review. Independent assays have shown substantial variability between labeled and assayed content [parasrampuria1998]. For patients in whom OTC supplements are clinically appropriate, the lowest commercially available dose is typically 25 mg, which exceeds the 10, 25 mg dose appropriate for many women.

Compounded oral DHEA capsules and sublingual troches under USP <795> [usp_795] address dose individualization (5, 10, 15, 20 mg increments not commercially available), excipient sensitivity, and identity/potency assurance, areas where the OTC supplement market has documented variability [parasrampuria1998] [labrie2017lowdose]. Compounded topical DHEA is used in selected patients but has minimal published PK characterization.

Compounded preparations are not bioequivalent to Intrarosa and are not bioequivalent to any specific OTC product. Switching between routes should be accompanied by reassessment of DHEA-S and clinical response [ke2015serum].

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DHEA Storage and Handling

Intrarosa is stored at controlled room temperature (20, 25°C; 68, 77°F) with excursions permitted between 15, 30°C (59, 86°F) per the FDA label [fda_label_intrarosa] [usp_795]. Inserts are individually packaged and used with the supplied applicator.

Compounded oral DHEA capsules and sublingual troches are stored at controlled room temperature per the pharmacy's stability and beyond-use date assignment under USP <795>. DHEA active pharmaceutical ingredient is light-sensitive; compounded preparations are typically dispensed in amber or opaque containers [usp_795].

DHEA Compounding & Operations

503A compounding

Compounded DHEA is prepared under 503A on patient-specific prescriptions in state-licensed compounding pharmacies. RonanRx prepares nonsterile DHEA preparations (oral capsules, sublingual troches, topical creams) per USP General Chapter <795>, the official compendial standard for nonsterile pharmaceutical compounding [usp_795] [fda503a]. Documented active ingredient sourcing, gravimetric and analytical verification, identity testing (e.g., HPLC or USP-monograph methods), and lot traceability are standard.

Beyond-use dating, ingredient identity verification, and stability assessment follow USP <795> requirements [fda503a]. Each compounded batch is documented per state board of pharmacy retention rules with full traceability from API lot through dispensing. The Parasrampuria 1998 JAMA study [parasrampuria1998] documenting variability in OTC supplement DHEA content is one of the principal historical references motivating USP-compliant compounded preparations as a quality-assurance alternative when patient-specific clinical need is documented.

Pharmacist review

Each prescription for compounded DHEA undergoes pharmacist review prior to dispensing. The review confirms: a documented patient-specific clinical reason that the OTC supplement market or FDA-approved Intrarosa product cannot meet the patient's clinical need (sub-OTC dose, sublingual/topical formulation, excipient sensitivity, or USP <795> identity/potency assurance); absence of contraindications (hormone-sensitive cancer, undiagnosed abnormal genital bleeding, pregnancy, lactation); appropriate concomitant medication review (concurrent androgen, estrogen, or aromatase inhibitor therapy); and a prescribed regimen consistent with the published trial dosing in the relevant indication [fda_label_intrarosa].

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RonanRx is particularly attentive to the FDA 'essentially-a-copy' framework for oral DHEA [fda_essentially_a_copy], even though there is no FDA-approved oral DHEA comparator, the existence of an OTC supplement market raises analogous questions, and prescriptions are reviewed for clinical rationale that an OTC product cannot satisfy [parasrampuria1998]. For postmenopausal dyspareunia, the Intrarosa product is the appropriate first-line choice; compounded intravaginal DHEA is reserved for documented patient-specific reasons (excipient sensitivity, dose individualization) [fda_label_intrarosa; usp_795].

Quality and traceability

Active pharmaceutical ingredients are sourced from FDA-registered facilities with documented certificates of analysis (CoA) including identity, assay, residual solvents, and heavy-metals testing. Each batch is recorded with lot numbers traceable to API source, compounding date, beyond-use date, identity verification (HPLC or USP-monograph method), and dispensing pharmacist of record [usp_795]. Finished product lot records are retained per state board of pharmacy retention requirements. The supplement-market quality gap documented by Parasrampuria 1998 [parasrampuria1998] is one of the principal motivations for the USP <795>-compliant traceability standard applied to compounded DHEA.

Frequently Asked Questions About DHEA

Is DHEA the same as testosterone or estrogen?

No. DHEA is a precursor that the body converts to small amounts of testosterone and estrogen inside specific tissues. It is not testosterone or estrogen itself, and its effects depend on each tissue's enzymes for converting it, a process called intracrinology [labrie1991intracrinology].

Why is oral DHEA available over the counter?

DHEA is sold OTC as a dietary supplement in the US under the Dietary Supplement Health and Education Act of 1994. This is unusual for a sex-steroid prohormone, and it means OTC products are not subject to FDA pre-market quality or efficacy review [parasrampuria1998]. Independent analyses have documented substantial variability between labeled and actual DHEA content in commercial supplements.

What is Intrarosa?

Intrarosa is the only FDA-approved DHEA product, a 6.5 mg prasterone vaginal insert approved in November 2016 for moderate-to-severe painful intercourse in postmenopausal women caused by vulvovaginal atrophy [fda_label_intrarosa]. The pivotal trials (Labrie 2011, Archer 2015) showed improvement in vaginal pH, cell maturation, and pain without clinically significant elevation of serum estrogens or androgens [labrie2011dyspareunia; archer2015dyspareunia].

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When is a compounded DHEA preparation appropriate?

Per FDA 503A standards, compounded preparations are appropriate when the prescriber documents a patient-specific clinical need that neither the OTC supplement market nor the FDA-approved Intrarosa product can meet [fda_essentially_a_copy; parasrampuria1998; usp_795]. Common reasons: sub-OTC doses (10, 25 mg, common for women); sublingual or topical formulation; excipient sensitivity to OTC product fillers; or USP <795>-compliant identity and potency. Cost or preference alone does not qualify.

Does DHEA work for anti-aging?

Evidence in healthy older adults is largely null. The definitive Mayo Clinic trial (Nair 2006, NEJM) of 87 men and 57 women aged 60, 88 on DHEA for 2 years found no benefit on body composition, physical performance, insulin sensitivity, or quality of life [nair2006nejm]. The earlier DHEAge study (Percheron 2003) reported small skin and libido improvements in women over 70 but no muscle benefit [percheron2003]. Compounding pharmacies should not market DHEA for anti-aging.

Does DHEA help with adrenal insufficiency?

Yes, this is the strongest off-label use case. Multiple randomized trials (Arlt 1999 NEJM; Hunt 2000; Gurnell 2008) and a meta-analysis (Alkatib 2009) show that DHEA 25, 50 mg/day improves mood, fatigue, and well-being scores in women with adrenal insufficiency who remain symptomatic on optimal glucocorticoid and mineralocorticoid replacement [arlt1999nejm; hunt2000; gurnell2008longterm]. The Endocrine Society 2016 guideline permits but does not routinely recommend [bornstein2016endo] [alkatib2009meta].

Does DHEA help with IVF?

It is widely used in poor-responder IVF protocols (75 mg/day for 6, 12 weeks pre-stimulation), but the supporting evidence is low quality. The Cochrane 2015 review found that androgen pre-treatment 'may improve live birth rate' but high-quality data are lacking. The Wiser 2010 trial was positive (n=33); the Yeung 2014 pilot was null on the primary endpoint (n=72) [wiser2010ivf; yeung2014ivf]. Patients should be counseled that benefit is plausible but unproven [nagels2015cochrane].

What are the most common side effects?

Androgenic effects in women, acne, oily skin, mild hirsutism, scalp hair thinning, are the most common and are dose-dependent. They occur in 10, 30% of women on 50 mg/day in research protocols and are typically reversible with dose reduction [arlt1999nejm; panjari2009sexual]. Estrogenic effects (breast tenderness, vaginal spotting) are less common. Intravaginal Intrarosa has a distinct profile dominated by application-site events (vaginal discharge) rather than systemic effects [labrie2011dyspareunia].

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Who should not take DHEA?

Patients with known or suspected hormone-sensitive cancers (breast, endometrial, prostate), undiagnosed abnormal genital bleeding, pregnancy, lactation, or active liver disease. Competitive athletes subject to WADA, NCAA, or other anti-doping testing should not use DHEA, it is a prohibited anabolic agent in- and out-of-competition [fda_label_intrarosa; wierman2014women].

Does RonanRx sell DHEA directly to patients?

No. Compounded DHEA requires a patient-specific prescription written by a licensed prescriber for an identified patient with a documented clinical reason that OTC supplements or the FDA-approved Intrarosa product cannot meet the patient's need, plus pharmacist review before dispensing [fda_essentially_a_copy]. RonanRx is not a direct-to-consumer storefront [fda503a].

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How to Access DHEA

Compounded DHEA is dispensed under 503A on a patient-specific prescription. Depending on your role, the next step looks different.

FOR PRESCRIBING CLINICIANS

Offer this medication

A pharmacist will follow up within two business days. We'll cover state availability, supported formulations, and what integration looks like for your clinic.

ronanrx.com/request-partnership-call

PATIENT WITH A DOCTOR

Receive your prescription

If your doctor has prescribed DHEA, sign up so we can prepare and ship your medication. The signup wizard collects intake and connects you to the prescribing workflow.

ronanrx.com/patients

PATIENT WITHOUT A DOCTOR

Find a partner clinic

RonanRx prescribes through partner clinics — we don't initiate prescriptions on this site. Read how the referral process works and how to find a partner clinic in your state.

ronanrx.com/find-clinic

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Other compounds RonanRx makes

This monograph is one of many in the RonanRx formulary. Every compound below is prepared under 503A on a patient-specific prescription. Browse the full catalog at ronanrx.com/medications and ronanrx.com/peptides, or scan the codes at right for each index.

Medications

Peptides

MEDICATIONS (40)

Alpha-Lipoic Acid (ALA) — Antioxidant & mitochondrial
Coenzyme Q10 (CoQ10) — Antioxidant & mitochondrial
Glutathione — Antioxidant & mitochondrial
NAD+ / NMN — Antioxidant & mitochondrial
Compounded Topical Anesthetics (BLT, LET) — Dermatology
Topical Minoxidil — Dermatology
Topical Tretinoin — Dermatology
Compounded Magnesium — Energy & nutritional
Cyanocobalamin — Energy & nutritional
High-Dose Vitamin D — Energy & nutritional
Hydroxocobalamin — Energy & nutritional
Iron (Compounded) — Energy & nutritional
L-Carnitine — Energy & nutritional
Methylcobalamin (B12) — Energy & nutritional
Methylfolate — Energy & nutritional
Anastrozole — Hormone optimization
Clomiphene & Enclomiphene — Hormone optimization
DHEA — Hormone optimization
Estradiol — Hormone optimization
Estriol — Hormone optimization
Human Chorionic Gonadotropin (HCG) — Hormone optimization
Pregnenolone — Hormone optimization
Progesterone — Hormone optimization
Testosterone — Hormone optimization
Compounded Metformin — Metabolic & weight
Compounded Semaglutide — Metabolic & weight
Compounded Tirzepatide — Metabolic & weight
Lipotropic Injection (MIC, MICC) — Metabolic & weight
Low-Dose Naltrexone (LDN) — Metabolic & weight
Naltrexone-Bupropion Combination — Metabolic & weight
Topiramate — Metabolic & weight
Bremelanotide / PT-141 — Sexual health
Compounded Sildenafil — Sexual health
Compounded Tadalafil — Sexual health
Trimix Injection — Sexual health
Compounded Gabapentin — Sleep & recovery
Compounded Melatonin — Sleep & recovery
Compounded T3 (Liothyronine) — Thyroid
Compounded T3/T4 Combinations — Thyroid
Compounded T4 (Levothyroxine) — Thyroid

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PEPTIDES (21)

Sermorelin — Available now

Tesamorelin — Available now

AOD-9604 — Growth-hormone axis (under FDA review)

CJC-1295 — Growth-hormone axis (under FDA review)

GHRP-2 / GHRP-6 — Growth-hormone axis (under FDA review)

Hexarelin — Growth-hormone axis (under FDA review)

Ipamorelin — Growth-hormone axis (under FDA review)

MK-677 / Ibutamoren — Growth-hormone axis (under FDA review)

5-Amino 1MQ — Metabolic & longevity (under FDA review)

Epitalon / Epithalon — Metabolic & longevity (under FDA review)

MOTS-C — Metabolic & longevity (under FDA review)

Thymosin Alpha-1 / Thymalin — Metabolic & longevity (under FDA review)

DSIP, Delta Sleep-Inducing Peptide — Neuro & cognitive (under FDA review)

Selank — Neuro & cognitive (under FDA review)

Semax — Neuro & cognitive (under FDA review)

Vasoactive Intestinal Peptide (VIP) — Neuro & cognitive (under FDA review)

BPC-157 — Tissue repair (under FDA review)

KPV — Tissue repair (under FDA review)

LL-37 — Tissue repair (under FDA review)

Pentadeca Arginate (PDA) — Tissue repair (under FDA review)

TB-500 / Thymosin Beta-4 — Tissue repair (under FDA review)

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