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Rener Health PRODUCTS

RENER HEALTH EXPO 2026

Women's Vitality Across the Lifespan:

A Clinical Framework for Hormonal, Metabolic and Microbiome Health



Presented by: Amie Skilton, Functional Medicine Practitioner & Educator

About the Presenter | Amie Skilton



Functional Medicine Practitioner & Educator

I've had nearly two decades as a functional medicine practitioner and educator in naturopathic medicine, addressing audiences from conference stages to television and digital platforms.

My personal diagnosis of Chronic Inflammatory Response Syndrome (CIRS) in 2017 reshaped my clinical focus – now extending naturopathic practice to include environmental health hazards alongside conventional treatment.

My approach integrates environmental factors with traditional health practices, emphasising the harmonious relationship between individuals and their surroundings for optimal health.

Today's Agenda

1. **The Four-Phase Framework**
   A review of the map of the menopausal transition and where each intervention fits.
2. **Foundations & Testing**
   Ovulation-first principles, lifestyle levers, and pathology across the four phases.
3. **Hormonal Health in Menopause**
4. **Metabolic Health in Menopause**
5. **Microbiome Health**
6. **Clinical Prescribing Summary**
   Key ingredients at a glance – dose, mechanism, evidence.

The Four-Phase Framework Overview

HPA / HPO axis support across the menopausal transition.

Phase	Clinical Picture	Key Markers	Today's Focus
Phase 1 **On-ramp**	Shifting cycles, heavier bleeding, breast pain, PMS, reduced stress tolerance	FSH < 40 IU/L Mid-luteal P4 < 50 nmol/L	CDG · Vitex Nicotinamide Riboside
Phase 2 **Crossing**	Anovulatory / skipped cycles, hot flashes, cognitive shift, up to 3x oestrogen	Loss of 1:1 FSH:LH Mid-luteal P4 < 35 nmol/L	Myo-Inositol Creatine
Phase 3 **Corridor**	No cycle 60+ days, escalating hot flashes, insomnia, insulin resistance	Rising FSH > 40 IU/L Mid-luteal P4 < 20 nmol/L	Shatavari Peony
Phase 4 **Chapter**	New hormonal cadence, relative androgen rise, body composition priority	12 months amenorrhoea Low oestrogen & P4, ↓SHBG	Creatine (bone & tissue mass)

Foundational Goals Across All Phases

1

Prolong ovulation

Keep ovulating as long as possible – it's the primary route to endogenous progesterone production.

2

Protect active tissue mass

Maintain, and preferably build, lean mass to reduce sarcopenia risk through the transition.

3

Optimise daily rhythm

Nutrition, circadian biology, nervous-system support and sleep are the substrate every intervention sits on.

4

Intervene early

Address poor stress and sleep patterns, and unresolved menstrual irregularity, before they compound.

Optimising Ovulation: The Foundations

Every prescription in this talk sits on top of these levers – addressed and continued through all phases.

Lever	Why it matters	Practical target
Nutrition	Adequate protein & carbohydrate stabilise the LH surge and support progesterone synthesis; fats provide cholesterol substrate.	Protein ~1.8–2.4 g/kg LBM; carbs 100–300 g/day per activity level.
Movement	Overtraining raises cortisol and suppresses the HPO axis; under-training loses metabolic and mood benefits.	Moderate resistance + aerobic; replace high-intensity work in luteal phase if stressed.
Sleep	Poor sleep drives next-day cortisol/pregnenolone steal and erratic FSH/LH output.	7.5–9 hours; consistent sleep/wake times.
Circadian rhythm	Light timing entrains the HPA/HPO clock via the SCN.	Morning light within 30 min of waking; dim/blue-light-free evenings.
Stress physiology	Chronic stress suppresses GnRH pulsatility via cortisol.	Daily parasympathetic 'white space'; hard boundary after dinner.

Testing Across the Four Phases Overview

A single elevated FSH does not confirm menopause – interpret markers within the full clinical picture.

Marker	Phase 1 On-ramp	Phase 2 Crossing	Phase 3 Corridor	Phase 4 Chapter
FSH	Usually < 40 IU/L	Loss of 1:1 FSH:LH (CD3)	Rising, < 40 IU/L	Confirmed at 12 mo amenorrhoea
Mid-luteal progesterone	< 50 nmol/L	< 35 nmol/L	< 20 nmol/L	Sustained low
Other markers	BBT rise ≥0.4°C confirms ovulation; DEXA	Possible leptin rise	Slight relative androgen rise	↓SHBG, ↑free testosterone; insulin & bone monitoring



HPO

Hormonal Health

Nutritional, lifestyle and herbal support for optimal hormone support during menopause.

Hormonal Patterns

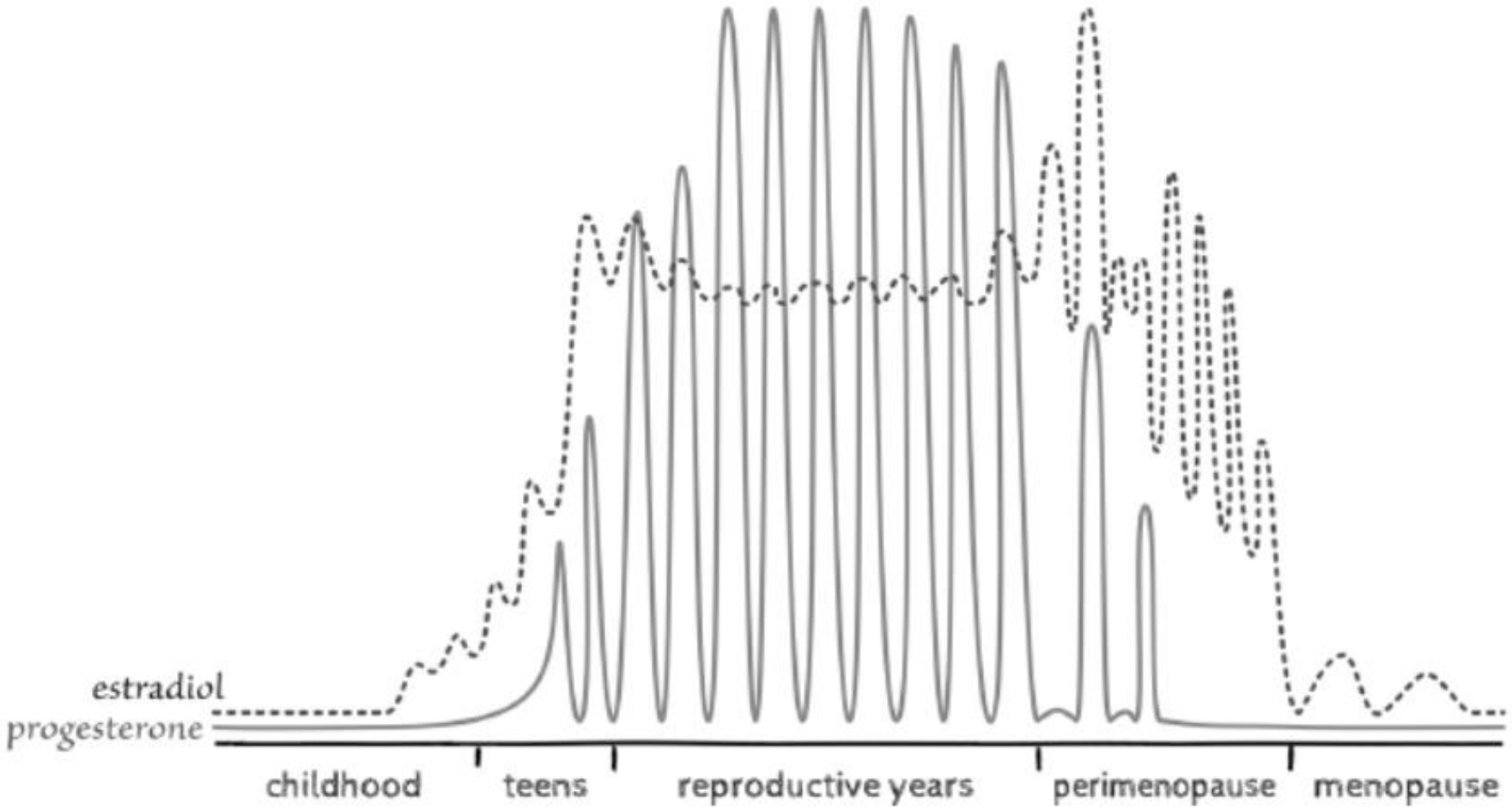


Image adapted from JC Prior, 'Perimenopause lost – reframing the end of menstruation', Journal of Reproductive and Infant Psychology, 2006, vol. 24, pp. 323–35.



CDG

Calcium-D-Glucarate

Managing the estrobolome.

Calcium-D-Glucarate

CDG

EVIDENCE: MECHANISTIC

PHASE 1 · Oestrogen Clearance

Symptom picture	Heavier bleeding, breast tenderness, PMS, migraines, irritability – an oestrogen-dominant picture as progesterone falls.
Treatment goal	Lower circulating active oestrogen load by supporting Phase II hepatic clearance.
Mechanism of action	Supports glucuronidation (Phase II conjugation) of oestrogen; its metabolite D-glucaro-1,4-lactone inhibits gut bacterial β-glucuronidase, blocking deconjugation/reactivation of oestrogen in the gut and reducing enterohepatic recirculation of active hormone.
Dose	200–500mg, 2–3×/day with food; up to 1,500–3,000mg/day divided for higher oestrogen-load presentations.

Calcium-D-G

Science / Eviden

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Clinical outcome

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Metabolism, uptake, and excretion of a D-glucaric acid salt and its potential use in cancer prevention

Z Walaszek [1], J Szemraj, M Narog, A K Adams, J Kilgore, U Sherman, M Hanausek

Affiliations + expand

PMID: 9101079

Abstract

D-Glucaric acid (GA) is a nontoxic, natural compound. One of its derivatives is the potent beta-glucuronidase inhibitor D-glucaro-1,4-lactone (1,4-GL). The goal of this study was to demonstrate the in vivo formation of 1,4-GL from a D-glucarate salt and determine its metabolism, uptake by selected organs, and excretion following oral administration of potassium hydrogen D-[14C]glucarate to male and female Sprague-Dawley rats. 1,4-GL increases detoxification of carcinogens and tumor promoters/progressors by inhibiting beta-glucuronidase and preventing hydrolysis of their glucuronides. 1,4-GL and its precursors, such as potassium hydrogen D-glucarate and calcium D-glucarate, may exert their anticancer action, in part, through alterations in steroidogenesis accompanied by changes in the hormonal environment and the proliferative status of the target organ. Thus, GA derivatives may be useful as new or adjuvant cancer preventive and therapeutic agents. In our study, 1,4-GL was found to be formed from the D-glucarate salt in the stomach of rats. It was apparently absorbed from the gastrointestinal tract, transported with the blood to different internal organs, and excreted in the urine and to a lesser extent in bile. There were no significant differences in the metabolism of PHG between male and female rats. Thus, formation of 1,4-GL from D-glucaric acid derivatives may be prerequisite for their inhibition of chemical carcinogenesis in rodents and prevention of breast, prostate, and colon cancer in humans.

Calcium-D-Glucarate — Evidence & Prescribing

Science / Evidence

- Walaszek et al. 1997, Cancer Detection and Prevention — male & female Sprague-Dawley rats given oral potassium hydrogen D-glucarate to characterise metabolism, tissue uptake and excretion, focused on conversion to D-glucaro-1,4-lactone, the active β-glucuronidase-inhibiting metabolite.
- High-dose dietary glucarate (~10% of total diet, roughly 1,000mg/kg bodyweight) reduced serum oestrogen by 23% vs control, attributed to enhanced glucuronidation and reduced β-glucuronidase-mediated reactivation of conjugated oestrogens.
- Supporting mechanistic context: the gut 'estrobolome' concept (Ervin, Lim & Roberts 2019, J Biol Chem) and oestrogen glucuronidation pathway (Raftogianis et al. 2000, JNCI Monographs).

Clinical outcome / Timeframe

Mechanism validated at high dose in animal models; use clinically as an adjunct within a broader Phase 1 protocol, assessing alongside cycle and symptom tracking over 8–12 weeks for reduction in oestrogen-excess symptoms (prolonged use at practitioner discretion).



PROG

Vitex agnus-castus

Supporting ovulation and progesterone production.

Vitex agnus-castus

Chaste Tree

EVIDENCE: STRONG

PHASE 1 · Luteal / Ovulation Support

Symptom picture	Premenstrual tension, breast tenderness, irritability, cycle irregularity, dysmenorrhoea – a pattern of relative luteal insufficiency and/or long follicular phase (can also be low oestrogen).
Treatment goal	Support LH-driven ovulation and corpus luteum function to improve progesterone production and luteal phase length.
Mechanism of action	Dopaminergic activity lowers elevated prolactin (which can inhibit ovulation); modulates pituitary LH secretion, supporting corpus luteum function and a longer, more robust luteal phase. Contraindicated in high-LH or high-oestradiol states.
Dose	20mg/day standardised extract – established as the clinically optimal dose in a randomised dose-finding trial, single morning dose, taken continuously.

Vitex agnus-cast

Science / Evidence

- Schellenberg et al. 2012, Phytome
  DSM-diagnosed PMS, 3 cycles): 8r
  (p<0.0001); 30mg added no furthe
- Berger et al. 2000 — prospective,
  PMS severity, with improvement i
- Schellenberg et al. 2001, BMJ — r
  significantly improved symptoms
- Across all three trials, standardise

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Clinical outcome / Tim

Symptom improvement typically buil
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Planta Med. 2013 May;79(7):562-75. doi: 10.1055/s-0032-1327831. Epub 2012 Nov 7.

Vitex agnus-castus extracts for female reproductive disorders: a systematic review of clinical trials

M Diana van Die [1], Henry G Burger, Helena J Teede, Kerry M Bone

Affiliations + expand

PMID: 23136064 DOI: 10.1055/s-0032-1327831

Free article

Abstract

Vitex agnus-castus L. (chaste tree; chasteberry) is a popular herbal treatment, predominantly used for a range of female reproductive conditions in Anglo-American and European practice. The objective of this systematic review was to evaluate the evidence for the efficacy and safety of Vitex extracts from randomised, controlled trials investigating women's health.Eight databases were searched using Latin and common names for Vitex and phytotherapeutic preparations of the herb as a sole agent, together with filters for randomised, controlled trials or clinical trials. Methodological quality was assessed according to the Cochrane risk of bias and Jadad scales, as well as the proposed elaboration of CONSORT for reporting trials on herbal interventions.Thirteen randomised, controlled trials were identified and twelve are included in this review, of which eight investigated premenstrual syndrome, two premenstrual dysphoric disorder, and two latent hyperprolactinaemia. For premenstrual syndrome, seven of eight trials found Vitex extracts to be superior to placebo (5 of 6 studies), pyridoxine (1), and magnesium oxide (1). In premenstrual dysphoric disorder, one study reported Vitex to be equivalent to fluoxetine, while in the other, fluoxetine outperformed Vitex. In latent hyperprolactinaemia, one trial reported it to be superior to placebo for reducing TRH-stimulated prolactin secretion, normalising a shortened luteal phase, increasing mid-luteal progesterone and 17β-oestradiol levels, while the other found Vitex comparable to bromocriptine for reducing serum prolactin levels and ameliorating cyclic mastalgia. Adverse events with Vitex were mild and generally infrequent. The methodological quality of the included studies varied, but was generally moderate-to-high. Limitations include small sample sizes in some studies, heterogeneity of conditions being treated, and a range of reference treatments.Despite some methodological limitations, the results from randomised, controlled trials to date suggest benefits for Vitex extracts in the treatment of premenstrual syndrome, premenstrual dysphoric disorder and latent hyperprolactinaemia. Further research is recommended, and greater transparency in reporting for future trials.

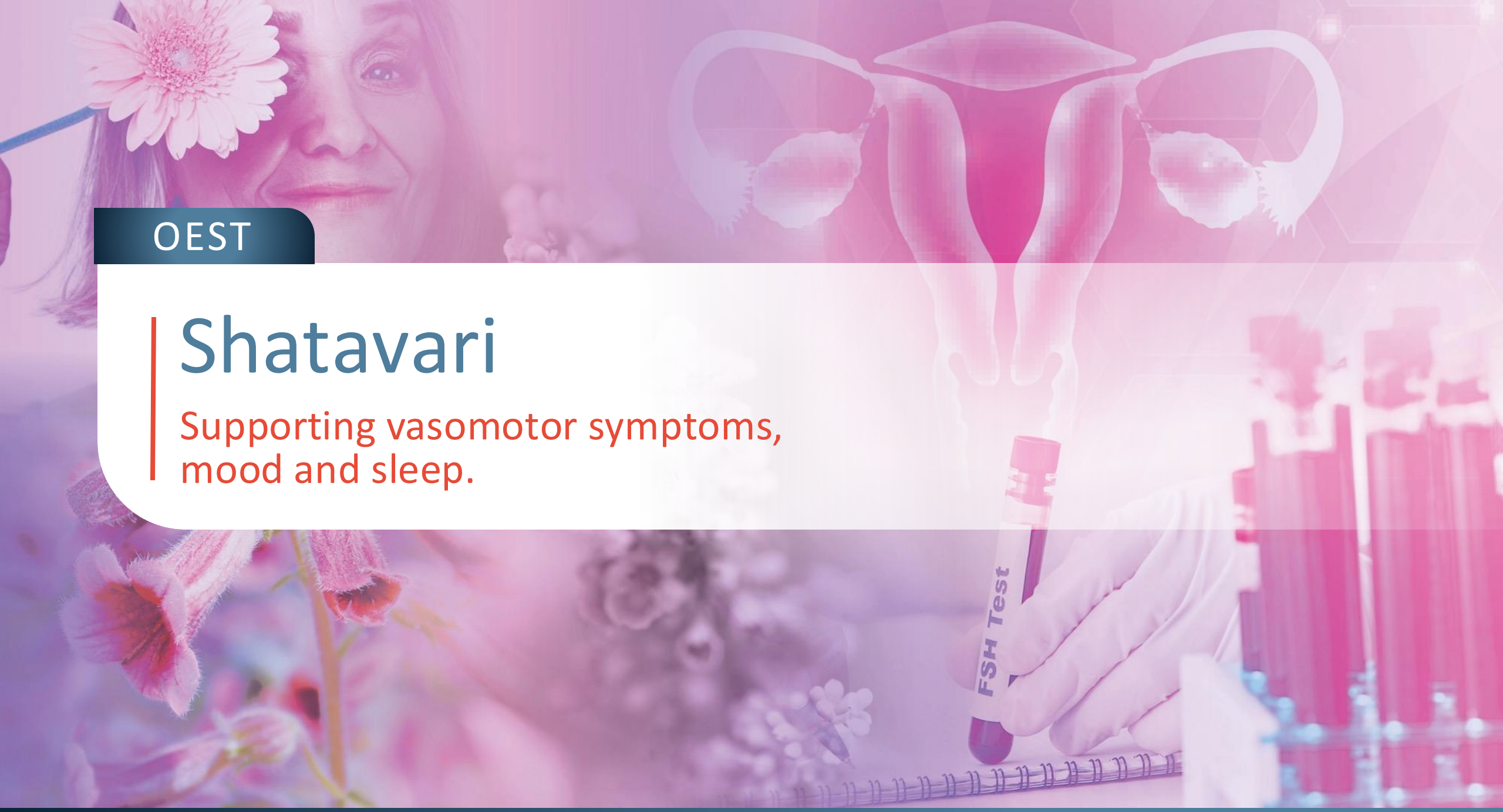
Vitex agnus-castus – Evidence & Prescribing

Science / Evidence

- Schellenberg et al. 2012, Phytomedicine – multicentre, randomised, double-blind, placebo-controlled dose-finding trial (n=162, DSM-diagnosed PMS, 3 cycles): 8mg, 20mg or 30mg Ze 440/day vs placebo. 20mg significantly superior to placebo and to 8mg ($p<0.0001$); 30mg added no further benefit – 20mg is the clinically optimal dose; ~81% responder rate.
- Berger et al. 2000 – prospective, multicentre, RCT (n=50, DSM-III PMS, 8-cycle design): 20mg/day significantly reduced overall PMS severity, with improvement in irritability, mood swings and breast tenderness.
- Schellenberg et al. 2001, BMJ – randomised, double-blind, placebo-controlled trial (n=170, community-based PMS): 20mg/day significantly improved symptoms vs placebo ($p<0.001$); 52% responder rate vs 24% on placebo.
- Across all three trials, standardised extract was well tolerated with only mild adverse events reported.

Clinical outcome / Timeframe

Symptom improvement typically builds over 2–3 menstrual cycles (~8–12 weeks) of continuous use; reassess luteal phase length, ovulation window, basal body temperature chart and PMS severity at that point.



OEST

Shatavari

Supporting vasomotor symptoms, mood and sleep.

Shatavari

Asparagus racemosus

EVIDENCE: STRONG

PHASE 2–3 · Vasomotor / HPO Balance

Symptom picture	Hot flashes, night sweats, fatigue, mood disturbance, sleep disruption and vaginal dryness through the Menopausal Corridor.
Treatment goal	Support HPO axis communication and buffer vasomotor and mood symptoms as oestrogen falls.
Mechanism of action	Steroidal saponins (shatavarins) – the primary bioactive constituents – exert an adaptogenic, SERM-like balancing action on hormonal signalling: blunting excess oestrogenic effects while providing mild oestrogenic support when levels are low.
Dose	250mg standardised extract (≥5% shatavarins), twice daily (500mg/day total).

Shatavari – Ev

Science / Evidence

- Gudise, Dasari & Kuricheti 2
  peri/post-menopausal wom
  cellulose), 35 per arm
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  domains — depression, anx
- Insomnia (Regensburg Inso
- Hot flash frequency fell fro
  significant adverse events

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s/day; well tolerated with no

Clinical outcome /

Vasomotor and quality-of-life

in a peer-reviewed RCT.

Cureus. 2024 Apr 8;16(4):e57879. doi: 10.7759/cureus.57879. eCollection 2024 Apr.

Efficacy and Safety of Shatavari Root Extract for the Management of Menopausal Symptoms: A Double-Blind, Multicenter, Randomized Controlled Trial

Vani S Gudise [1], Meenakshi P Dasari [2], Siva Sai K Kuricheti [3]

Affiliations + expand

PMID: 38725785 PMCID: PMC11079574 DOI: 10.7759/cureus.57879

Abstract

Background Menopause is a physiological state that occurs in all women and refers to the halt of the reproductive phase. The cessation of the reproductive phase occurs through various stages and presents different symptoms such as hot flashes, night sweats, insomnia, anxiety, depression, and irritability. Such pre- and post-menopausal symptoms may affect the daily activities and production capacities of women, impacting the quality of life (QoL) of women. Hormone replacement therapy (HRT) is primarily used to manage menopausal symptoms. However, various side effects have been reported related to HRT. Therefore, women are choosing alternative medicine such as Ayurveda that can benefit them with less or no adverse effects. Shatavari (*Asparagus racemosus*) is known in Ayurveda as an effective medicinal plant source for various women's health remedies since ancient times. This study aimed to evaluate the safety and efficacy of the Ayurvedic Shatavari formulation on menopausal symptoms compared to the placebo. Methodology This is a randomized, double-blinded, multicenter, placebo-controlled, clinical study. Altogether, 70 patients were randomized to two groups, i.e., the test group (active group) and the placebo group (microcrystalline cellulose), with 35 participants in each group. Results The study outcomes showed a positive and significant effect of the active test ingredient over the placebo in terms of reduction in hot flashes, night sweats, insomnia, anxiety, nervousness, vaginal dryness, and loss of libido. The Utian QoL improved significantly in the test group compared to the placebo group. No significant adverse events were recorded in the test group, suggesting the safety of this formulation. Conclusions The test compound could be a safe alternative to modern drugs. The findings of this study support the traditional use of Shatavari. Further clinical and pharmacological studies with longer duration and larger and more diverse sample sizes are required to understand the generalized effect of Shatavari root extract in menopausal women.

Shatavari – Evidence & Prescribing

Science / Evidence

- Gudise, Dasari & Kuricheti 2024, Cureus 16(4):e57879 — randomised, double-blind, multicentre, placebo-controlled RCT: 70 peri/post-menopausal women aged 40–65, 8 weeks, 250mg standardised extract twice daily vs placebo (microcrystalline cellulose), 35 per arm.
- Utian Quality of Life score improved 35% vs placebo at day 60 ($p<0.0001$); DASS-21 showed significant reductions across all three domains — depression, anxiety and stress ($p<0.0001$).
- Insomnia (Regensburg Insomnia Scale) improved 41% relative to placebo ($p<0.0001$).
- Hot flash frequency fell from 1.97 to 0.14 episodes/day; night sweats fell from 0.77 to 0.06 episodes/day; well tolerated with no significant adverse events.

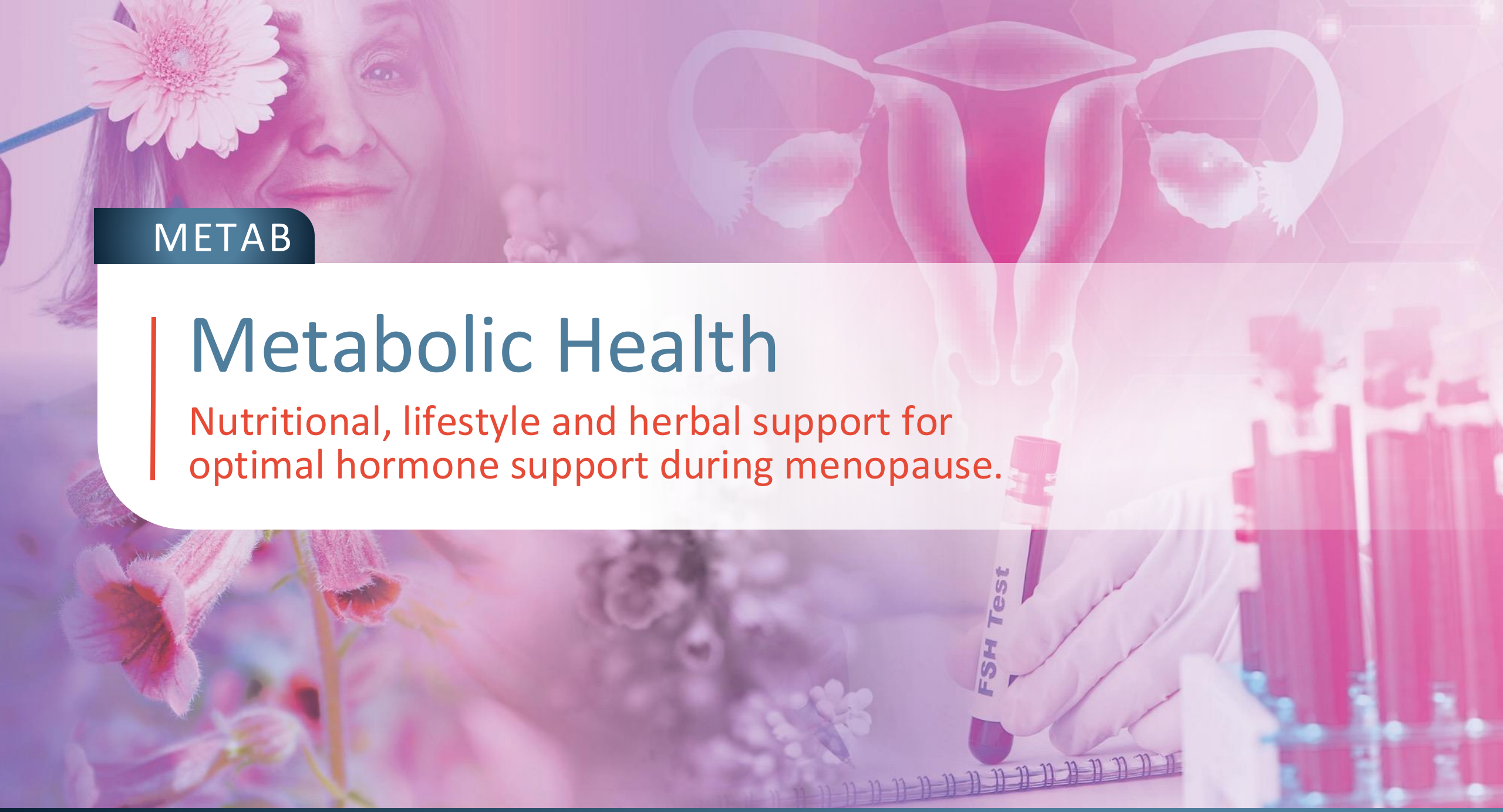
Clinical outcome / Timeframe

Vasomotor and quality-of-life benefit demonstrated by day 60 (8 weeks) of continuous twice-daily use in a peer-reviewed RCT.

METAB

Metabolic Health

Nutritional, lifestyle and herbal support for optimal hormone support during menopause.



Mitochondria in the Ovary: The Body's Most Energy-Hungry Cell

Science / Evidence

- Mature human oocytes carry several hundred thousand copies of mitochondrial DNA – more than any other human cell type – making the oocyte "the richest cell of the body in mitochondria" (May-Panloup et al. 2016, Hum Reprod Update).
- This mitochondrial mass powers meiotic spindle formation, chromosome segregation and fertilisation competence, and provisions every cell of the early embryo, since mtDNA does not replicate until after implantation (May-Panloup et al. 2016).
- Follicular development requires a minimum of ~2 picomoles of ATP; oocyte mitochondria cluster around the spindle and germinal vesicle to meet this localised demand (Ju et al. 2024, Front Endocrinol).
- The "mitochondrial bottleneck": primordial germ cells start with only ~100 mtDNA copies, later amplified during oogenesis – a quality-control checkpoint thought to purge defective mitochondrial genomes (May-Panloup et al. 2016).

What Happens to Ovarian Mitochondria in Perimenopause & Menopause

Science / Evidence

- mtDNA copy number and mitochondrial membrane potential decline with reproductive age in both oocytes and granulosa cells, alongside visible mitochondrial swelling and cristae loss (May-Panloup et al. 2016).
- Human ovarian tissue and oocyte analysis shows a significant, age-correlated rise in oxidative protein and lipid damage in primordial follicles, alongside falling NAD+, purine and pyrimidine pools and compensatory glycolytic/glutamine metabolism as oxidative phosphorylation falters (Smits et al. 2023, Hum Reprod).
- Mitochondrial biogenesis signalling (PGC-1α, NRF1/2, SIRT1/3) and mitophagy (PINK1/Parkin-mediated clearance) are both downregulated with age, so damaged mitochondria accumulate rather than being replaced or cleared (Ju et al. 2024, Front Endocrinol).
- mtDNA mutation rates of 28–50.5% have been reported in aged oocytes and up to 66% in cumulus cells, compounding the decline (Ju et al. 2024).

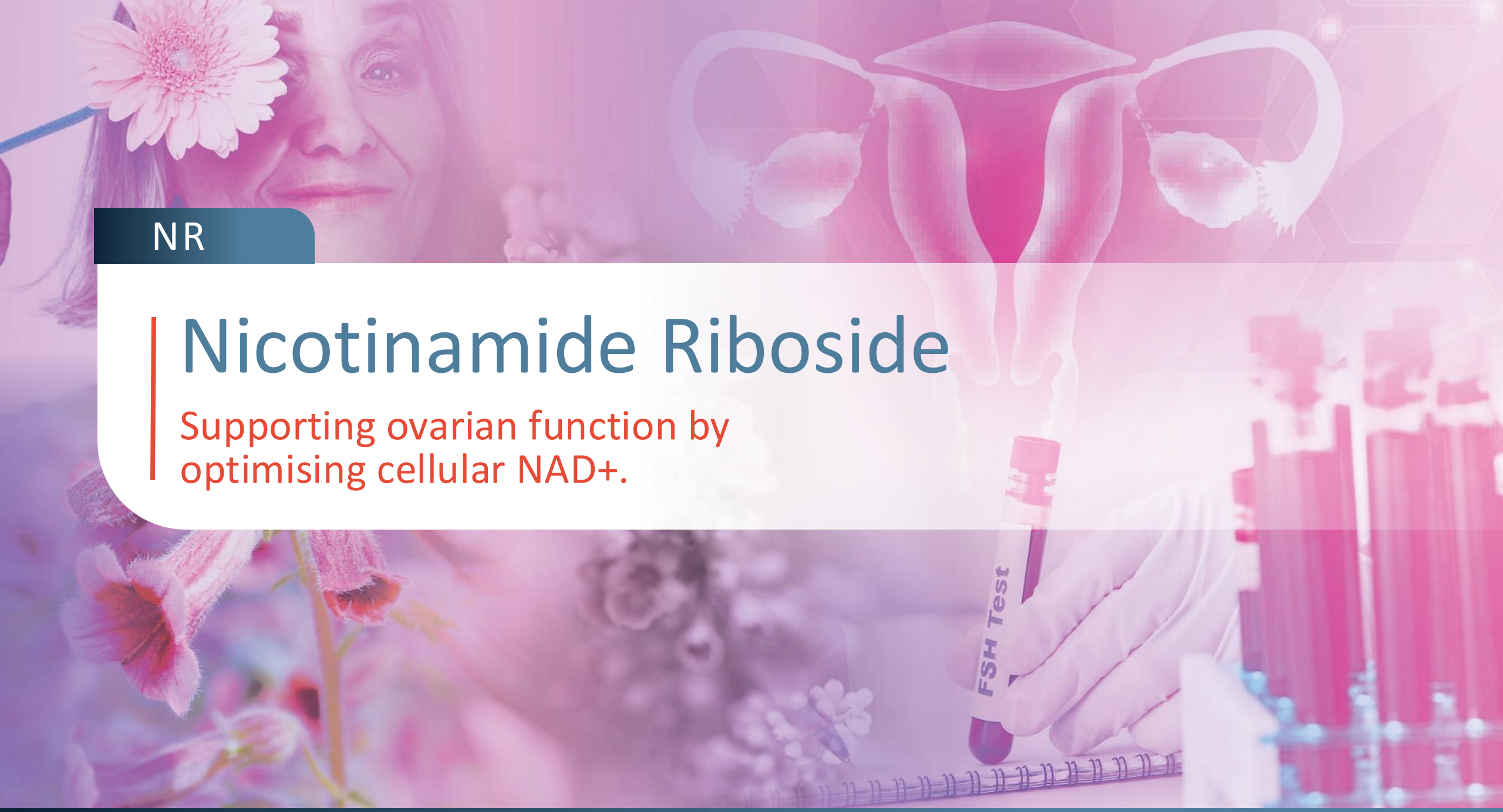
Clinical Relevance

Net effect: fewer, more damaged mitochondria per follicle – a direct mechanistic driver of declining oocyte competence, shortening cycles, and the eventual transition to menopause; mitochondrial nutrient support is upstream, not just symptomatic care.

Beyond the Ovary: Systemic Impact of Declining Mitochondrial Function

Science / Evidence

- Oestrogen is a direct transcriptional regulator of mitochondrial biogenesis, coordinating nuclear-encoded respiratory genes and mitochondrial DNA replication via oestrogen receptor signalling — so declining oestrogen removes a key driver of mitochondrial renewal well beyond the ovary (Klinge 2020, Redox Biol).
- PET imaging shows reduced brain glucose metabolism across the menopause transition, in regions overlapping mood and memory circuits — a neuroenergetic signature that tracks with the cognitive and affective symptoms many women report in perimenopause (Mosconi et al. 2021, Sci Rep).
- In aged mice, restoring NAD+ via NR or NMN improved mitochondrial biogenesis, autophagy and TCA cycle function and partially rescued ovarian reserve — proof-of-concept that the same pathway supporting oocyte energetics is therapeutically accessible (Ju et al. 2024, Front Endocrinol).



NR

Nicotinamide Riboside

Supporting ovarian function by optimising cellular NAD+.

Nicotinamide Riboside

NR · NAD+ precursor

EVIDENCE: EMERGING

PHASE 1 · Mitochondrial / Ovarian Energy

Symptom picture	Declining ovarian reserve markers and oocyte quality, fatigue and low cellular energy reserve compounding Phase 1 ovulatory decline.
Treatment goal	Replenish the NAD+ pool to support mitochondrial ATP production in oocytes and granulosa cells, buffering age- and stress-related oxidative decline in ovarian function.
Mechanism of action	NR is phosphorylated via the NRK1/2 pathway to nicotinamide mononucleotide (NMN), then converted to NAD+ by NMNAT. NAD+ is an essential coenzyme for ~200 enzymes including mitochondrial dehydrogenases and sirtuins, and is required for ATP production via oxidative phosphorylation. NAD+-dependent sirtuins have emerging roles in gamete mitochondrial function and protection against oxidative stress in reproductive ageing.
Dose	Studied clinical dose 500mg NR chloride twice daily (1,000mg/day); standard capsule format is 300mg – reaching the studied dose requires multiple capsules/day at practitioner discretion.

Nicotinamid

Science / Eviden

- Martens et al. 2018, Na
  aged/older adults: 500
  tolerated, with 8–10mr
- Conze et al. 2019, Scier
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- Trammell et al. 2016, N
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Clinical outcome

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Randomized Controlled Trial > Nat Commun. 2018 Mar 29;9(1):1286.
doi: 10.1038/s41467-018-03421-7.

Chronic nicotinamide riboside supplementation is well-tolerated and elevates NAD$^+$ in healthy middle-aged and older adults

Christopher R Martens [1], Blair A Denman [2], Melissa R Mazzo [2], Michael L Armstrong [3], Nichole Reisdorph [3], Matthew B McQueen [2], Michel Chonchol [4], Douglas R Seals [2]

Affiliations + expand

PMID: 29599478 PMCID: PMC5876407 DOI: 10.1038/s41467-018-03421-7

Abstract

Nicotinamide adenine dinucleotide (NAD$^+$) has emerged as a critical co-substrate for enzymes involved in the beneficial effects of regular calorie restriction on healthspan. As such, the use of NAD$^+$ precursors to augment NAD$^+$ bioavailability has been proposed as a strategy for improving cardiovascular and other physiological functions with aging in humans. Here we provide the evidence in a 2 × 6-week randomized, double-blind, placebo-controlled, crossover clinical trial that chronic supplementation with the NAD$^+$ precursor vitamin, nicotinamide riboside (NR), is well tolerated and effectively stimulates NAD$^+$ metabolism in healthy middle-aged and older adults. Our results also provide initial insight into the effects of chronic NR supplementation on physiological function in humans, and suggest that, in particular, future clinical trials should further assess the potential benefits of NR for reducing blood pressure and arterial stiffness in this group.

PubMed Disclaimer

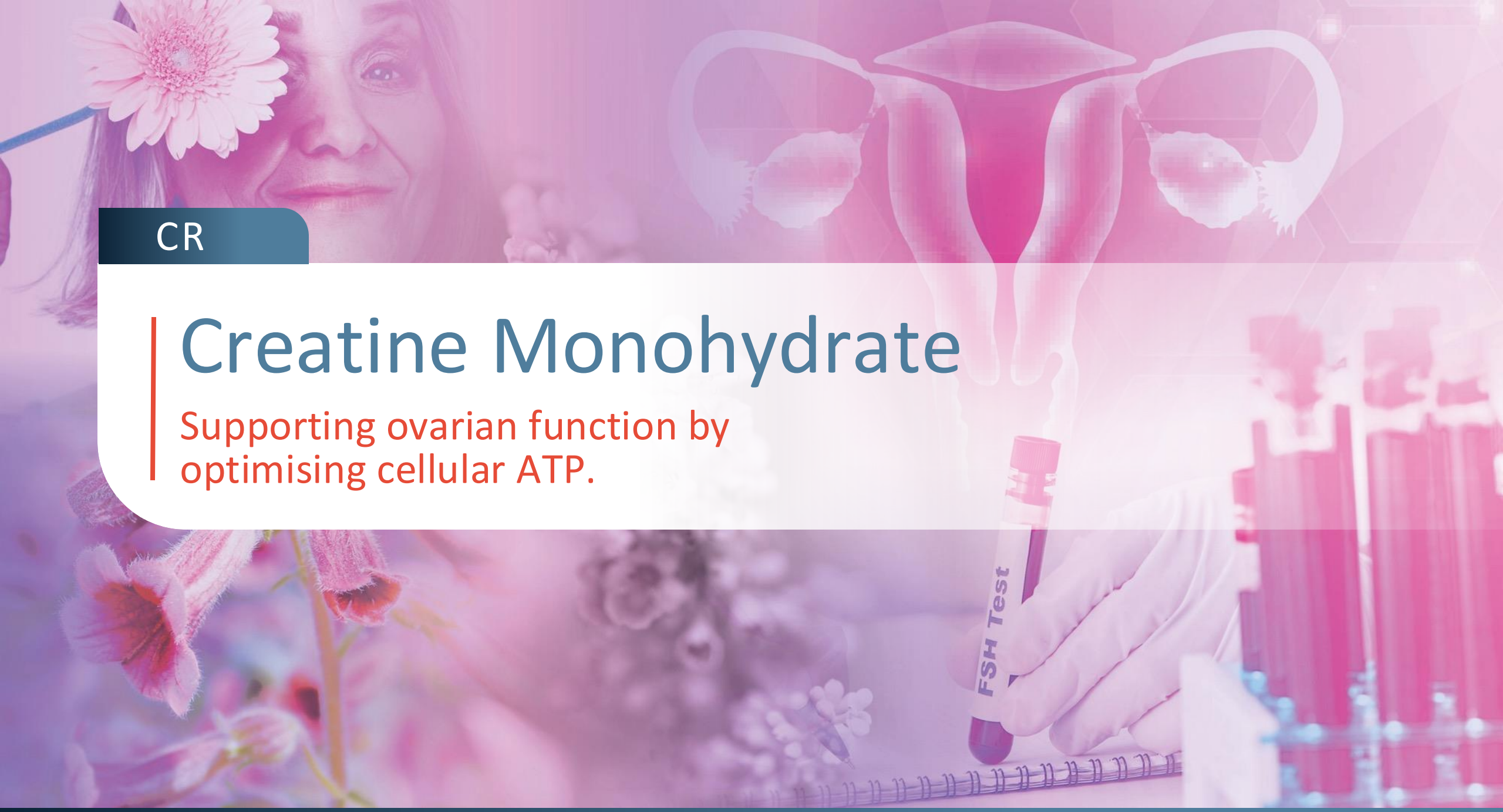
Nicotinamide Riboside – Evidence & Prescribing

Science / Evidence

- Martens et al. 2018, Nature Communications – randomised, double-blind, placebo-controlled crossover trial in healthy middle-aged/older adults: 500mg NR chloride twice daily (1,000mg/day) for 6 weeks raised blood NAD+ by ~60% over baseline; well tolerated, with 8–10mmHg reduction in systolic BP and reduced arterial stiffness in those with elevated baseline BP.
- Conze et al. 2019, Scientific Reports – long-term randomised, double-blind, placebo-controlled trial of NR chloride in healthy overweight adults confirmed safety and tolerability of sustained supplementation.
- Trammell et al. 2016, Nature Communications – established NR as uniquely and orally bioavailable in mice and humans, the pharmacokinetic basis for oral NAD+ repletion.
- Ovarian relevance is currently mechanistic: Tatone et al. 2018 (Hum Reprod Update) and 2015 (Oxid Med Cell Longev) review sirtuin/NAD+-dependent pathways in gamete mitochondrial function and reproductive ageing — direct human RCT data on NR for oocyte quality or ovulatory outcomes is not yet available.

Clinical outcome / Timeframe

General NAD+ repletion demonstrated within 6 weeks in adults. Ovarian-specific functional outcomes haven't yet been directly trialled – this is a mechanistic adjunct within Phase 1 mitochondrial/energy support, and reassess alongside broader ovulation markers rather than in isolation.



CR

Creatine Monohydrate

Supporting ovarian function by optimising cellular ATP.

Creatine Monohydrate

Cognition & Active Tissue Mass

EVIDENCE: STRONG

PHASE 2 · Energy Availability

Symptom picture	Mental fatigue, brain fog, declining strength and lean mass, reduced resilience under the high cognitive and physical demand of perimenopause.
Treatment goal	Support cellular energy availability for brain and muscle; preserve lean mass and cognitive resilience through the transition.
Mechanism of action	Stored as phosphocreatine in muscle and brain, where it rapidly regenerates ATP during short, high-intensity or high-demand effort. Supplementation increases phosphocreatine stores by roughly 10–40%, boosting power output, training capacity and cellular energy availability in neurons under stress or sleep loss.
Dose	5g/day, no loading phase required.

Creatine M

Science / Evid

- Rae et al. 2003, Proc
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- Smith-Ryan 2025, J I
- Xu et al. 2024, Front
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Clinical Trial > Proc Biol Sci. 2003 Oct 22;270(1529):2147-50. doi: 10.1098/rspb.2003.2492.

Oral creatine monohydrate supplementation improves brain performance: a double-blind, placebo-controlled, cross-over trial

Caroline Rae [1], Alison L Digney, Sally R McEwan, Timothy C Bates

Affiliations + expand

PMID: 14561278 PMCID: PMC1691485 DOI: 10.1098/rspb.2003.2492

Abstract

Creatine supplementation is in widespread use to enhance sports-fitness performance, and has been trialled successfully in the treatment of neurological, neuromuscular and atherosclerotic disease. Creatine plays a pivotal role in brain energy homeostasis, being a temporal and spatial buffer for cytosolic and mitochondrial pools of the cellular energy currency, adenosine triphosphate and its regulator, adenosine diphosphate. In this work, we tested the hypothesis that oral creatine supplementation (5 g d(-1) for six weeks) would enhance intelligence test scores and working memory performance in 45 young adult, vegetarian subjects in a double-blind, placebo-controlled, cross-over design. Creatine supplementation had a significant positive effect ($p < 0.0001$) on both working memory (backward digit span) and intelligence (Raven's Advanced Progressive Matrices), both tasks that require speed of processing. These findings underline a dynamic and significant role of brain energy capacity in influencing brain performance.

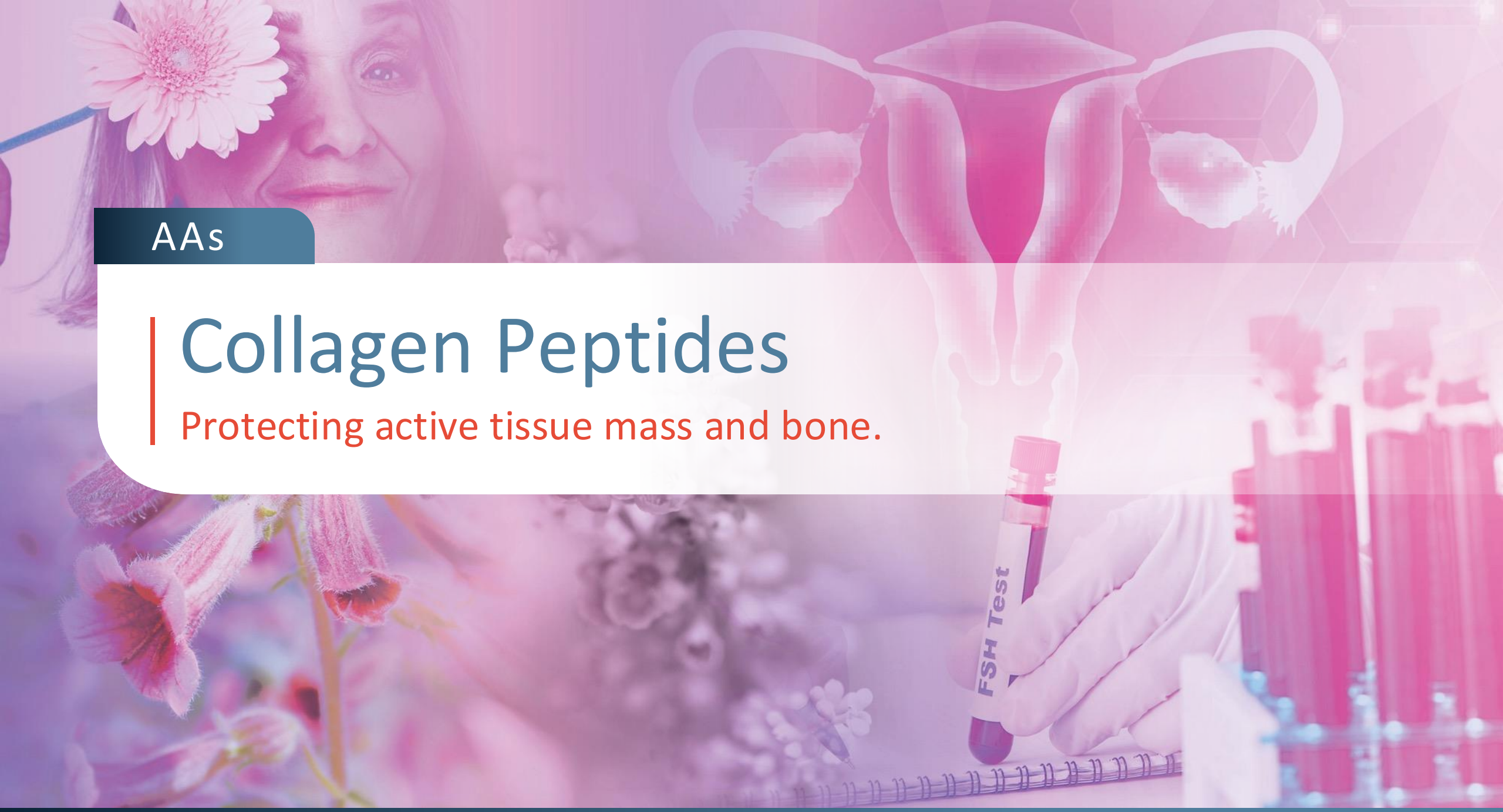
Creatine Monohydrate — Evidence & Prescribing

Science / Evidence

- Rae et al. 2003, Proc R Soc Lond B – double-blind, placebo-controlled crossover trial in 45 young vegetarian adults: 5g/day for 6 weeks (matches the standard non-loading dose) significantly improved working memory (backward digit span) and fluid intelligence (Raven's APM), both $p<0.0001$.
- Smith-Ryan 2025, J Int Soc Sports Nutr – narrative review: creatine's role across menstruation, pregnancy and menopause.
- Xu et al. 2024, Frontiers in Nutrition and Prokopidis et al. 2023, Nutr Rev – systematic reviews/meta-analyses confirming cognitive and memory benefits, particularly under fatigue or sleep deprivation.
- Chilibeck et al. 2023, Med Sci Sports Exerc – 2-year RCT: creatine + resistance training improved bone health in postmenopausal women (see also Phase 4).

Clinical outcome / Timeframe

Cognitive benefits (working memory, processing speed) demonstrated within 6 weeks; strength and lean-mass gains with resistance training typically emerge over 12+ weeks.



AAs

Collagen Peptides

Protecting active tissue mass and bone.

Collagen Peptides

Hydrolysed bovine collagen

EVIDENCE: MODERATE

PHASE 2–4 · Active Tissue Mass / Bone

Symptom picture	Loss of strength and lean mass, slower recovery, joint aches, thinning skin and declining bone density – as falling oestrogen reduces collagen formation through perimenopause.
Treatment goal	Preserve – and preferably build – active tissue mass and bone, alongside resistance training and adequate total protein intake.
Mechanism of action	Supplies glycine, proline and hydroxyproline for muscle repair and connective tissue. Specific bioactive peptides are partly absorbed intact and signal target cells – upregulating anabolic (mTOR, PI3K-Akt) pathways in muscle, chondrocytes in cartilage and osteoblasts in bone.
Dose	12.5g/day (1 scoop Whole Body Collagen™: 5g FORTIGEL®, 5g FORTIBONE®, 2.5g VERISOL®); muscle trials used 15g/day with resistance training 3×/week. Heat-stable – add to hot or cold drinks.

Collagen

Science / Ev

- Jendricke et al. 2
- Zdzieblik et al. 2
- Centner et al. 20
- König et al. 2018

tides with resistance training 3×/wee
han placebo.
ining for 12 weeks increased fat-fre
c signalling pathways (PI3K-Akt, MAPK
nonths increased spine and femor
l. 2021).

Randomized Controlled Trial Nutrients. 2019;11(4):892.
doi: 10.3390/nu11040892

Specific collagen peptides in combination with resistance training improve body composition and regional muscle strength in premenopausal women: a randomized controlled trial

Jendricke P, Centner C, Zdzieblik D, Gollhofer A, König D

Key findings

- **Design:** prospective, randomised, double-blind, placebo-controlled; 77 untrained premenopausal women.
- **Intervention:** 15 g/day specific collagen peptides with resistance training three times a week for 12 weeks.
- **Lean mass:** significantly greater increase in fat-free mass than placebo.
- **Fat mass:** significantly greater reduction in fat mass.
- **Strength:** greater gains in hand-grip and regional muscle strength.
- **Take-home:** collagen peptides amplify the body-composition benefits of resistance training in women.

Clinical outcome / Timeframe

Strength and lean-mass gains seen over 12 weeks with resistance training; bone density benefits need 12 months. Muscle trials used 15g of a specific muscle-targeted peptide – pair with training and adequate protein.

Collagen Peptides – Evidence & Prescribing

Science / Evidence

- Jendricke et al. 2019, Nutrients – RCT in 77 untrained premenopausal women: 15g/day specific collagen peptides with resistance training 3×/week for 12 weeks increased fat-free mass and hand-grip strength and reduced fat mass more than placebo.
- Zdzieblik et al. 2015, British Journal of Nutrition – 53 older men with sarcopenia: 15g/day with resistance training for 12 weeks increased fat-free mass (+4.2 vs +2.9kg) and quadriceps strength more than placebo, with greater fat loss.
- Centner et al. 2022, Frontiers in Physiology – a single 15g dose after resistance exercise upregulated anabolic signalling pathways (PI3K-Akt, MAPK) in skeletal muscle vs placebo.
- König et al. 2018, Nutrients – 131 postmenopausal women with reduced BMD: 5g/day FORTIBONE® for 12 months increased spine and femoral neck BMD and P1NP vs placebo; gains were maintained at 4-year follow-up (Zdzieblik et al. 2021).

Clinical outcome / Timeframe

Strength and lean-mass gains seen over 12 weeks with resistance training; bone density benefits need 12 months. Muscle trials used 15g of a specific muscle-targeted peptide – pair with training and adequate protein.



MI

Myo-inositol

Managing blood sugar and insulin challenges.

Myo-Inositol

MI · ‘Vitamin B8’

EVIDENCE: STRONG

PHASE 2 · Insulin-Mediated Ovulation

Symptom picture	Anovulatory or irregular cycles, insulin resistance features, PCOS-type ovarian dysfunction, sub-fertility or poor oocyte quality.
Treatment goal	Restore insulin-mediated ovarian signalling and improve oocyte quality and ovulatory consistency.
Mechanism of action	Acts as a second messenger for insulin and FSH signalling in the ovary. In ovarian dysfunction, follicular fluid MI:DCI ratio collapses from a healthy ~100:1 to as low as 0.2:1 — correcting this ratio restores calcium signalling, oocyte maturation and meiotic competence.
Dose	1.1–4g/day (clinical trials commonly use 2g twice daily); often paired with folate.

Myo-Inositol

Science / Eviden

- Unfer, Facchinetti, Orrù,
  active vs 249 control, MI
- Significant decreases in f
  reduced testosterone
- Serum SHBG rose signifi
  improves the metabolic
- Zeng & Yang 2017 (Endo
  efficacy comparable to r

Clinical outcome

Improvements in insulin se
consistent supplementatio

> Endocr Connect. 2017 Nov;6(8):647-658. doi: 10.1530/EC-17-0243.

Myo-inositol effects in women with PCOS: a meta-analysis of randomized controlled trials

Vittorio Unfer [1], Fabio Facchinetti [2], Beatrice Orrù [3], Barbara Giordani [3], John Nestler [4]

Affiliations + expand

PMID: 29042448 PMCID: PMC5655679 DOI: 10.1530/EC-17-0243

Abstract

Myo-inositol (MI) supplementation in women with polycystic ovary syndrome (PCOS) has been evaluated over the last years. Many hormonal and reproductive impairments associated with this disorder seem relieved by the supplement. The objective of the meta-analysis was to assess the effects of MI alone or combined with d-chiro-inositol (DCI) on the endocrine and metabolic abnormalities of women with PCOS. Literature was retrieved from selected databases, MEDLINE, EMBASE, PubMed and Research Gate (up to November 2016). Only randomized controlled trials (RCTs) investigating the effects of MI alone or combined with DCI were reviewed. Nine RCTs involving 247 cases and 249 controls were included. Significant decreases in fasting insulin (SMD = -1.021 µU/mL, 95% CI: -1.791 to -0.251, *P* = 0.009) and homeostasis model assessment (HOMA) index (SMD = -0.585, 95% CI: -1.145 to -0.025, *P* = 0.041) were identified after MI supplementation. The trial sequential analysis of insulin meta-analysis illustrates that the cumulative *z*-curve crossed the monitoring boundary, providing firm evidence of the intervention effect. A slight trend toward a reduction of testosterone concentration by MI with respect to controls was found (SMD = -0.49, 95% CI: -1.072 to 0.092, *P* = 0.099), whereas androstenedione levels remained unaffected. Throughout a subgroup's meta-analysis, a significant increase in serum SHBG was observed only in those studies where MI was administered for at least 24 weeks (SMD = 0.425 nmol/L, 95% CI: 0.050-0.801, *P* = 0.026). These results highlight the beneficial effect of MI in improving the metabolic profile of women with PCOS, concomitantly reducing their hyperandrogenism.

Myo-Inositol – Evidence & Prescribing

Science / Evidence

- Unfer, Facchinetti, Orrù, Giordani & Nestler 2017, Endocrine Connections – meta-analysis of 9 RCTs in women with PCOS (247 active vs 249 control, MEDLINE/EMBASE/PubMed/ResearchGate); doses 1.1–4g/day.
- Significant decreases in fasting insulin and HOMA-IR vs control – a genuine improvement in insulin sensitivity; trend toward reduced testosterone.
- Serum SHBG rose significantly, but only in the subgroup of studies where MI was taken for ≥24 weeks – authors concluded MI improves the metabolic profile while reducing hyperandrogenism.
- Zeng & Yang 2017 (Endocrine) and Bodepudi et al. 2023 (Cureus) corroborate: MI improves ovulation and LH:FSH ratio, with efficacy comparable to metformin but a markedly better side-effect profile.

Clinical outcome / Timeframe

Improvements in insulin sensitivity typically measurable within 12 weeks; SHBG and androgen benefits require ≥24 weeks of consistent supplementation to reach significance.



OEA

Oleoylethanolamide

Supporting satiety, GLP-1 signalling and metabolic health.

Oleoylethanolamide

OEA · PPAR-α agonist

EVIDENCE: MODERATE

PHASE 2–4 · Appetite / Metabolic Health

Symptom picture	Rising appetite and cravings, central weight gain, climbing triglycerides and insulin resistance through the transition – often alongside low-grade inflammation and gut dysbiosis.
Treatment goal	Restore satiety signalling and metabolic flexibility – supporting appetite control, lipid handling, glycaemic regulation and inflammatory tone via the gut–brain axis.
Mechanism of action	Gut-derived lipid messenger (does not bind cannabinoid receptors). Activates PPAR-α (fatty acid uptake and β-oxidation), GPR119 on intestinal L-cells (↑ GLP-1, with DPP-4 suppression) and TRPV1 (vagal satiety signalling); dampens NF-κB, preserves tight junctions and increases Akkermansia muciniphila.
Dose	125mg twice daily (250mg/day), 20–30 minutes before meals; Trpti OEA™ with LipiSperse® for bioavailability. Avoid in pregnancy and lactation; use caution alongside hypoglycaemic or lipid-lowering medicines.

Oleoyleth

Science / Ev

- Payahoo et al. 2
- Payahoo et al. 2
- Tutunchi et al. 2
- Shivyari et al. 20

-α expression and reduced weight,

IL-6 and TNF-α, reduced energy

on for 12 weeks: ↓ weight, waist, fa

nsulin, HOMA-IR, TNF-α, CRP and

Randomized Controlled Trial Appetite. 2018;128:44–49.
doi: 10.1016/j.appet.2018.05.129

Oleoylethanolamide increases the expression of PPAR-α and reduces appetite and body weight in obese people: a clinical trial

Payahoo L, Khajebishak Y, Barzegari A, Alipour S, Asghari Jafarabadi M, Farrin N, Ostadrahimi A

Key findings

- **Design:** randomised, placebo-controlled trial in 60 adults with obesity.
- **Intervention:** OEA 125 mg twice daily (250 mg/day) for 8 weeks vs placebo.
- **Mechanism:** PPAR-α gene expression increased significantly in the OEA group.
- **Body composition:** reductions in weight, BMI, waist circumference and fat mass.
- **Appetite:** less hunger, desire to eat and sweet cravings, with greater fullness.
- **Safety:** no adverse effects reported.

Clinical outcome / Timeframe

Appetite, body composition and metabolic markers improve over 8–12 weeks at 250mg/day, with no serious adverse events.
Trials were not menopause-specific – reassess waist, appetite, triglycerides and HOMA-IR at 12 weeks.

Oleoylethanolamide – Evidence & Prescribing

Science / Evidence

- Payahoo et al. 2018, Appetite – RCT in 60 adults with obesity: 125mg twice daily for 8 weeks increased PPAR-α expression and reduced weight, BMI, waist circumference and fat mass, with less hunger and fewer sweet cravings.
- Payahoo et al. 2018 (Endocr Metab Immune Disord Drug Targets) and 2019 (Appetite) – same dose lowered IL-6 and TNF-α, reduced energy and carbohydrate intake, and increased Akkermansia muciniphila vs placebo.
- Tutunchi et al. 2020, Pharmacological Research – obese adults with NAFLD, 250mg/day with calorie restriction for 12 weeks: ↓ weight, waist, fasting glucose, insulin and HOMA-IR; ↑ PPAR-α, UCP1 and UCP2 expression.
- Shivyari et al. 2024, Journal of Ovarian Research – PCOS (n=90), 125mg/day for 8 weeks: ↓ fasting glucose, insulin, HOMA-IR, TNF-α, CRP and AMH vs placebo; triglycerides also fell in obese adults (Ostadrahimi et al. 2024).

Clinical outcome / Timeframe

Appetite, body composition and metabolic markers improve over 8–12 weeks at 250mg/day, with no serious adverse events.
Trials were not menopause-specific – reassess waist, appetite, triglycerides and HOMA-IR at 12 weeks.



GSM

Microbiome Health

Strategies for genitourinary syndrome of menopause.



SB

Saccharomyces boulardii

Driving secretory IgA for mucosal immunity.

Saccharomyces boulardii

Probiotic yeast · sIgA support

EVIDENCE: MODERATE

PHASE 2–4 · Mucosal Immunity / Microbiome

Symptom picture	Recurrent gut, urogenital or respiratory infections, food reactivity, loose stools or post-antibiotic dysbiosis – often with low faecal sIgA – as mucosal barriers thin with declining oestrogen.
Treatment goal	Restore the mucosal ‘first line of defence’: raise secretory IgA, calm mucosal inflammation and support a stable, protective microbial biofilm along the gut lining.
Mechanism of action	Increases secretory IgA and its transporter (secretory component) in the small intestine. sIgA neutralises pathogens and toxins without triggering inflammation ('immune exclusion') and anchors commensals in the mucus layer, supporting healthy biofilm formation. Also degrades C. difficile toxins and dampens NF-κB inflammatory signalling.
Dose	250–500mg (5–10 billion CFU) daily, commonly 500mg twice daily. As a yeast it is unaffected by antibiotics, so can be taken alongside them. Avoid with central venous catheters or severe immunocompromise.

Saccharo

Science / Ev

- Buts et al. 1990, 56.9% and secre mucosal immun
- Qamar et al. 200 specific anti-C. d
- Bollinger et al. 2 formation along
- Szajewska & Koł halved the risk o the gut ecosyste

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: S. boulardii roughly dence that it protects

Animal Study Dig Dis Sci. 1990;35(2):251–256.
doi: 10.1007/BF01536771

Stimulation of secretory IgA and secretory component of immunoglobulins in small intestine of rats treated with Saccharomyces boulardii

Buts JP, Bernasconi P, Vaerman JP, Dive C

Key findings

- **Design:** growing rats given oral S. boulardii (0.5 mg/g body weight, three times daily) for 8 days vs saline or ovalbumin control.
- **Secretory IgA:** duodenal fluid s-IgA concentration rose 56.9% vs saline (P<0.01).
- **Secretory component:** up 62.8% in duodenal fluid and 48.5% in jejunal crypt cells – the receptor that transports IgA across the epithelium.
- **Specificity:** no change in mucosal mass or epithelial turnover, indicating a direct effect on mucosal immune secretion.
- **Authors' conclusion:** S. boulardii's protective effect may involve stimulation of intestinal s-IgA and secretory component.

Clinical outc

The sIgA mechanism is established in animal models; human sIgA data remain limited, while clinical protection of the gut ecosystem is well supported. Trial for 8–12 weeks and reassess symptoms and, where available, faecal sIgA and calprotectin.

Saccharomyces boulardii – Evidence & Prescribing

Science / Evidence

- Buts et al. 1990, Digestive Diseases and Sciences – growing rats given oral S. boulardii for 8 days: duodenal secretory IgA rose 56.9% and secretory component 62.8% vs control ($p<0.01$), with no change in mucosal turnover – a direct trophic effect on mucosal immunity.
- Qamar et al. 2001, Infection and Immunity – mice fed S. boulardii had ~1.9-fold higher total intestinal IgA and a 4.4-fold rise in specific anti-C. difficile toxin A IgA ($p<0.001$), showing it amplifies antigen-specific mucosal defence.
- Bollinger et al. 2003 (Immunology) and Mantis, Rol & Corthésy 2011 (Mucosal Immunology) – sIgA supports commensal biofilm formation along the gut epithelium and neutralises threats without inflammation, underpinning innate barrier defence.
- Szajewska & Kołodziej 2015, Alimentary Pharmacology & Therapeutics – meta-analysis of 21 RCTs (n=4,780): S. boulardii roughly halved the risk of antibiotic-associated diarrhoea (RR 0.47) in adults and children – the strongest human evidence that it protects the gut ecosystem.

Clinical outcome / Timeframe

The sIgA mechanism is established in animal models; human sIgA data remain limited, while clinical protection of the gut ecosystem is well supported. Trial for 8–12 weeks and reassess symptoms and, where available, faecal sIgA and calprotectin.



SBO

Sea Buckthorn Oil

Non-hormonal support for vaginal dryness.

Sea Buckthorn Oil

Hippophae rhamnoides · Omega 7

EVIDENCE: MODERATE

PHASE 2–4 · GSM / Vaginal Dryness

Symptom picture	Vaginal dryness, itching, burning and dyspareunia, often with dry eyes, mouth and skin – the 'dry mucous membrane' picture of oestrogen decline, including where vaginal oestrogen is contraindicated or declined.
Treatment goal	Support regeneration, hydration and integrity of the vaginal epithelium and other mucous membranes through non-hormonal pathways.
Mechanism of action	Supercritical CO2-extracted seed and pulp oil supplies omega-7 (palmitoleic acid, up to 50% of pulp oil) plus omega-3, -6 and -9, carotenoids, tocopherols/tocotrienols and plant sterols – building blocks for epithelial membranes and barrier lipids, with antioxidant and anti-inflammatory activity.
Dose	3g/day (6 × 500mg capsules, 3 taken twice daily) for at least 3 months – the dose used in the vaginal atrophy RCT; 1–2g/day studied for skin and dry eye. Mild GI upset possible, as with any oil supplement.

Sea Buck

Science / Ev

- Larmo et al. 201 oil for 3 months toward a better
- Chan et al. 2024 improvement in significantly vs p
- Larmo et al. 201 osmolarity and e
- Yang et al. 2009, elasticity (p<0.0

3g/day sea buckthorn –8.95), with a trend

very satisfied with 05), though not

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ed skin hydration and

Randomized Controlled Trial Maturitas. 2014;79(3):316–321.
doi: 10.1016/j.maturitas.2014.07.010

Effects of sea buckthorn oil intake on vaginal atrophy in postmenopausal women: a randomized, double-blind, placebo-controlled study

Larmo PS, Yang B, Hyssälä J, Kallio HP, Erkkola R

Abstract

Background: Vaginal atrophy, the thinning and drying of vaginal mucosa, is associated with menopause. The standard estrogen treatment is not suitable for all women. **Method:** A total of 116 postmenopausal women experiencing symptoms of vaginal dryness, itching or burning were randomized to this placebo-controlled, double-blind study. Ninety-eight participants completed the intervention of three months, during which they consumed 3 g of SB or placebo oil daily. **Results:** Compared to placebo, there was a significantly better rate of improvement in the integrity of vaginal epithelium in the SB group when both compliant and noncompliant participants were included (OR = 3.1, 95% CI 1.11–8.95). There was a tendency (P = 0.08) toward better improvement of vaginal health index in the SB group. **Conclusions:** SB oil showed beneficial effects on vaginal health, indicating it is a potential alternative for mucosal integrity for those women not able to use estrogen treatment for vaginal atrophy.

Clinical outc

Improvement in vaginal epithelial integrity shown after 3 months of continuous use; reassess dryness, dyspareunia and comfort at 12 weeks.

Sea Buckthorn Oil – Evidence & Prescribing

Science / Evidence

- Larmo et al. 2014, Maturitas – RCT in 116 postmenopausal women with vaginal dryness, itching or burning: 3g/day sea buckthorn oil for 3 months improved vaginal epithelial integrity vs placebo (36% vs 20% improved; OR 3.1, 95% CI 1.11–8.95), with a trend toward a better vaginal health index (p=0.08).
- Chan et al. 2024, Journal of Functional Foods – 12-week RCT in 40 women over 45 (1g/day): 65% satisfied or very satisfied with improvement in vaginal dryness; itching, redness, dryness, odour and dysuria improved from baseline ($p<0.05$), though not significantly vs placebo; HDL-C +14.8%.
- Larmo et al. 2010, Journal of Nutrition – RCT in 100 adults with dry eye: 2g/day for 3 months attenuated the rise in tear film osmolarity and eased redness and burning – consistent with a systemic mucous-membrane effect.
- Yang et al. 2009, J Appl Cosmetol – 60 women aged 50–70: 2g/day oral oil for 3 months significantly improved skin hydration and elasticity ($p<0.001$); Erkkola & Yang 2003 review its traditional and clinical use for mucosal health.

Clinical outcome / Timeframe

Improvement in vaginal epithelial integrity shown after 3 months of continuous use; reassess dryness, dyspareunia and comfort at 12 weeks.



4 PHASES

A Comprehensive Protocol

Nutraceutical support across phase 1-4 of menopause.

Clinical Prescribing Summary

Key ingredients at a glance:

Ingredient	Phase / Indication	Dose	Mechanism (snapshot)	Evidence
Calcium-D-Glucarate	P1 + 2 · Oestrogen clearance	200–500mg 2–3×/day	↑ Glucuronidation, ↓ β-glucuronidase recycling of oestrogen	Mechanistic
Vitex agnus-castus	P1 + 2 · Luteal / ovulation support	20mg/day std. extract, AM	↓ Prolactin, modulates LH → supports corpus luteum	Strong
Nicotinamide Riboside	P1 + 2 · Mitochondrial / ovarian energy	300mg/day label (1,000mg studied)	NAD+ precursor → ATP regeneration & sirtuin activity	Emerging
Myo-Inositol	P2 · Insulin-mediated ovulation	1.1–4g/day (2g BID common)	Insulin/FSH second messenger; restores MI:DCI ratio	Strong
Creatine Monohydrate	P2–P4 · Cognition, tissue mass, bone	5g/day no loading	Phosphocreatine → rapid ATP regeneration, muscle & brain	Strong
Shatavari	P3 · Vasomotor / HPO balance	250mg BID std. extract	Shatavarins – SERM-like adaptogenic HPO modulation	Strong
Peony (+ liquorice)	P3 · Dysmenorrhoea / androgen support	3–9g/day dried root	Antispasmodic (uterine tension); ↓ androgen synthesis in combo formulae	Mechanistic
Sea Buckthorn Oil	P2–P4 · GSM / vaginal dryness	3g/day for min. 3 months	Omega-7 rich; supports vaginal epithelial integrity (non-hormonal)	Moderate

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