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“Women Are Not Small Men”: The Difference Is Real, the Prescription Is Not

> **An evidence audit of 32 claims about female physiology, training and nutrition**

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Why this document exists

Dr Sims has done a great deal for women. She has addressed the shortcomings in the research. She has advocated heavy strength training for women, against an overwhelming perception that women should be lifting light pink weights. She has made the case that women can do high intensity sprint training, and that women can eat higher protein diets. All of that public awareness is to be welcomed, encouraged and acknowledged.

That aside, there are questions about the evidence base used to make some of the claims, and at times the claims themselves run counter to what current science and best practice coaching would suggest.

Dr Sims has said of the phrase women are not small men that it "became a concise way to remind people to check whether the underlying data actually applies to women, rather than treating women as simply smaller versions of men" (Sims 2026). With that spirit and that idea in mind, this document looks at what the best human science actually says about women's health and fitness, specifically in relation to her claims. Each claim is set out with its evidence and its verdict, so you can weigh it yourself. This is a review of the claims, not of the person who made them. It is a tool for seeing where what is being said and what the human research shows line up, and where they do not. This audit is written in the same spirit Dr Sims champions: getting the science right for women.

Executive summary

Dr Stacy Sims argues that female physiology is categorically different from male physiology and therefore demands sex-specific training, fuelling and weight-management strategies across the lifespan. Assessed against the human interventional evidence, the picture is mixed rather than uniform. Of the thirty-two claims made by Dr Sims and examined here, **five are Supported, twenty-two are Overstated, four are Contradicted and one is Untested**. Several of Dr Sims' positions are sound and are credited as such: that resistance training and higher-intensity work can benefit women, that protein can sit in the athlete range, that creatine and iron are commonly under-used, that low energy availability and RED-S are real and often under-recognised, and that women have been historically under-represented in exercise research, although the current figures are better than the rhetoric implies, with 81.5% of studies in the leading journals now including female participants. **The issue is not the headline encouragement to lift, train hard and eat enough; it is the specific mechanistic claims layered on top of it.**

Three recurring patterns explain almost every Overstated or Contradicted verdict, and the audit flags the first two wherever they occur.

First, a claimed female-specific effect that is not. Time and again, a real or supposed sex difference in physiology (oestrogen, muscle fibre type, mitochondrial density, lactate handling or fuel use during exercise) is used to argue that women must train or fuel differently, **when head-to-**

head human studies show both sexes responding the same way. This underlies the claims about fasted training, moderate loads, Zone 2 versus high-intensity work, protein needs, the exercise-cortisol response and post-menopausal muscle-building. Where a claim of this kind appears, it is flagged **Not uniquely female**.

Second, a confident claim built on rodent data that human evidence contradicts. Several of the most assertive claims rest on animal studies and are then contradicted by human trials. The fear that fasting shuts down the menstrual cycle through kisspeptin traces to a study in juvenile rats, while year-long human trials show no adverse effect on sex hormones; the idea of a distinct female mitochondrial response derives from mouse-liver studies, while human muscle data show no sex difference; and the reproductive-axis argument against time-restricted eating comes from a fasting study in female mice, while human trials show no adverse hormonal effect in women. The case against Zone 2 for women is similar, leaning on dated and largely animal physiology of lactate transporters and an oestrogen mechanism, while human training data show no sex difference in mitochondrial or lactate-handling adaptation. These rows are flagged **Rodent-based claims, contradicted by human data**.

A premise drawn from a mixed and contested literature. Much of the 'women-are-naturally-endurance-adapted' framing (more slow-twitch fibres, higher mitochondrial density, greater fatigue resistance) rests on an older body of work that is inconsistent and that modern fibre-level methods have not reproduced; the best current human comparison finds no sex difference in mitochondrial content or capillarisation. Claims built on this premise, including the proposal to shorten women's rest between intervals, are weaker than they are usually presented to be.

Purpose. This document takes each claim made by Dr Stacy Sims and weighs it against the strongest available human interventional evidence, assigning a considered verdict to each. The claims are drawn from her own words: exact quotations from her books ROAR (revised edition, 2024) and Next Level (2022), together with positions she has set out in long-form interviews and in her own published articles. The revised edition of ROAR is used throughout, so the audit addresses her current published positions rather than guidance she has since updated. Where she cites a source in support of a claim, that source has been read and is assessed on what it reports rather than on how it is described. It is meant as a fair audit, not a blanket rebuttal: several of her claims are well supported by current science and are marked as such, and the value of the exercise lies in showing precisely where the human evidence agrees with her and where it diverges. A point-by-point reading of this kind is both more useful and harder to dismiss than a uniform acceptance or rejection.

How to read the verdicts:

Supported means the claim aligns with current human evidence.

Overstated means the claim has a valid core but is pushed beyond what the human data support, or is framed as female-specific when it is sex-neutral.

Contradicted means the best available human evidence points the other way.

Untested means the specific claim has not been directly studied, so the current human evidence can neither support nor refute it; the burden sits with whoever asserts it.

Method and standing rule. Where possible the verdict rests on randomised trials, meta-analyses or consensus statements in humans, **consistent with the principle that recommendations for women should rest on human interventional research rather than on rodent data or theoretical sex differences.** The recurring pattern in the contradicted and overstated rows is the same: a real physiological sex difference, or a finding from animal or mechanistic work, is extrapolated into a training or nutrition prescription that human trials do not bear out.

Citations are given in author-year form; full details are in the References. Each claim is colour-coded by its verdict: green for Supported, amber for Overstated, red for Contradicted, and grey for Untested.

Summary of claims, verdicts and certainty

#	CLAIM	VERDICT	CERTAINTY	KEY CITATIONS
1	Women are not small men (male-default; categorically different)	OVERSTATED	Moderate	Ose 2025; Refalo 2025; Hunter 2023
2	Women built for endurance (more type I fibres / mitochondria)	OVERSTATED	Moderate	Bailleul 2025; Hunter 2023
3	Cycle drives programming (substrate, HRV, thermo, laxity)	OVERSTATED	Moderate	McNulty 2020; Schlie 2025
4	Track cycle and program by phase	CONTRADICTED	Moderate	D'Souza 2026; Mikkonen 2025
5	Oral contraceptives blunt adaptation / lean mass	OVERSTATED	Low	Elliott-Sale 2020; Nolan 2024
6	Prefer a hormonal IUD over the pill	UNTESTED	Very low	no head-to-head study
7	Pill masks the RED-S warning sign	SUPPORTED	Moderate	Mountjoy 2023
8	Women need more protein, timed (luteal / menopause)	OVERSTATED	Moderate	Morton 2018; Colenso-Semple 2025
9	Carbs not the enemy; keto contraindicated for women	OVERSTATED	Moderate	Vargas-Molina 2024; Leaf 2024
10	IF / fasted training contraindicated for women	OVERSTATED	Moderate	Varady 2024; Tan 2021
11	Low energy availability is the primary threat	SUPPORTED	High	Mountjoy 2023; Gallant 2024
12	Creatine benefits women (wrongly framed as male)	SUPPORTED	Low (female data)	Smith-Ryan 2025; Naddafha 2026
13	Iron deficiency common in women; screen	SUPPORTED	High	Sim 2019; Pompano 2019

#	CLAIM	VERDICT	CERTAINTY	KEY CITATIONS
14	Vitamin D / calcium + adaptogens	OVERSTATED	Mixed	Bai 2025; Coope 2026
15	Women better fat-burners; men lose fat more easily	OVERSTATED	Moderate	Astorino 2026; Hunter 2023
16	Lift heavy: heavy RT the most important modality	OVERSTATED	Moderate	Refalo 2025; Kistler-Fischbacher 2021
17	Heavy loads necessary (acidosis impairs growth)	CONTRADICTED	High	Schoenfeld & Grgic 2021; Morton 2016
18	Strength via neural adaptation, not hypertrophy	OVERSTATED	Moderate	Refalo 2025; Hunter 2023
19	HIIT more effective for women; SIT low-cortisol	OVERSTATED	Moderate	Lock 2024; Mølmen 2025
20	Zone 2 built on male data; less optimal for women	CONTRADICTED	Moderate	Mølmen 2025; Diaz-Canestro 2019
21	Long moderate work spikes cortisol; cap at 30 min	OVERSTATED	Moderate	Dote-Montero 2021; Lang 2024
22	Women recover faster; shorter inter-interval rest	OVERSTATED	Premise mod. / prescription very low	Hunter 2014; Ansdell 2020
23	Calorie restriction alone fails (cortisol)	OVERSTATED	Moderate	Fontana 2015; Josse 2011; Longland 2016
24	Timing of eating matters more than total calories	CONTRADICTED	High	Schoenfeld, Aragon & Krieger 2013
25	Many women under-eating; eat more to lose fat	OVERSTATED	Moderate	Müller 2016
26	Menopause removes satellite-cell muscle stimulus	OVERSTATED	Moderate	Dam 2023; Svensen 2025
27	Perimenopause: eat-less-move-more counterproductive	OVERSTATED	Moderate	McCarthy 2021; Anekwe 2025
28	Post-menopause: muscle and bone are the priority	SUPPORTED	High	Watson 2018; Kistler-Fischbacher 2021
29	Gut microbiome shifts at menopause	OVERSTATED	Low	Lim 2026; Honda 2024
30	Female heart adapts differently to training	OVERSTATED	Moderate	Cotie 2025; Hedge 2026
31	Hydration: body water / sodium / luteal / post-menopause	OVERSTATED	Low	Sims 2007a/b; Giersch 2020
32	Women "running an experiment on themselves"	OVERSTATED	Moderate	Kraemer 2025; Hunter 2023

Verdicts: Supported (aligns with current human evidence), Overstated (valid core pushed beyond the data, or framed as female-specific when sex-neutral), Contradicted (best human evidence points

the other way), Untested (not directly studied). Five claims bundle more than one proposition and carry sub-verdicts in their entries below.

1. The foundational claim: "Women Are Not Small Men"

> **Claim 1. Exercise science was built on a male-default model; female physiology is categorically different and male-derived guidelines are fundamentally flawed, so women need sex-specific training, fuelling and weight management.**

OVERSTATED

Certainty of evidence: moderate. Sub-verdicts: under-representation/sampling claim: Supported; the inference that female physiology is categorically different and therefore requires categorically different training, fuelling and weight management: Overstated.

Not uniquely female. Two separable claims sit inside one. The sampling claim has a real core, and the field does still lean male (Niazi et al. 2026). But the current data do not describe a field that ignores women: in the most recent audit of the three leading sports medicine journals (1,441 studies, 2021 to 2023), 81.5% of studies included female participants and women were 43.95% of all participants, up from 39% a decade earlier, with 18.5% of studies male-only (Ose et al. 2025). That participant total is itself dragged down by two outsized epidemiological studies; set those aside and women were 52.3% of participants. What remains scarce is female-only work (7.2%) and menstrual-status control (5.6%), which is a matter of sampling and methodological design rather than proof of categorically different physiology (Ose et al. 2025), and an audit of exercise-vascular research likewise found few studies achieved best-practice control of ovarian hormones (Thompson et al. 2024).

The physiological inference does not follow. When men and women complete the same program, the relative response is the same: a 2025 Bayesian meta-analysis (29 studies, 1,278 male and 1,537 female data points) found relative muscle growth essentially identical between sexes (exponentiated percentage change 0.69%, 95% HDI -1.50 to 2.88), with only a small absolute advantage to men that is explained by their larger starting size (Refalo et al. 2025). A direct comparison of 14 eumenorrheic women and 13 men, all untrained, found no sex difference in fibre-specific muscle mitochondrial content or capillarisation, nor in the protein content of the beta-oxidation enzyme beta-HAD or the electron-transport-chain subunits (Bailleul et al. 2025), and post-exercise muscle protein synthesis and signalling are similar between sexes (West et al. 2012; Dreyer et al. 2010).

The same holds into older age: two separate meta-analyses of resistance training in older adults found men and women make similar relative gains in muscle size, with relative strength gains equal to or greater in women and only an absolute edge to men (Jones et al. 2021; Hawley et al. 2023), and a meta-analysis dedicated to resistance training in women found large dynamic-strength gains and clear hypertrophy (Hagstrom et al. 2020). Refalo and colleagues conclude resistance training

may be prescribed similarly for both sexes, with differences driven by goals, not sex; a 2025 review of cycle-based programming takes as its starting point that there is no convincing evidence that strength training approaches in women should markedly differ from those in men, with adaptations in strength and hypertrophy relatively similar under similar training, while noting that gains over months or years of high-volume training may remain somewhat smaller in women and that more research is needed (Mikkonen & Häkkinen 2025), and the ACSM and NSCA statements and Kraemer's review reach the same verdict that the same training principles apply to both sexes (Hunter et al. 2023; Dabbs et al. 2026b; Kraemer et al. 2025).

The accurate conclusion from the literature is narrower: women have been under-represented in samples, and the studies behind training guidelines and vascular research have often lacked the authorship diversity and hormonal control to fully account for it, not that male-derived training adaptations fail to apply. None of this denies that genuine physiological sex differences exist. The hormonal milieu, the absolute strength ceiling and average muscle fibre size do differ, as the 2026 NSCA position statement details. The 2023 ACSM consensus statement makes the strongest respectable version of the opposing case, that biological sex is a primary determinant of athletic performance, with males outperforming females by roughly 10 to 30 per cent in events relying on strength, power or endurance, driven by the pubertal rise in testosterone (Hunter et al. 2023); this audit does not dispute that, and does not claim the sexes perform identically.

The claim under examination is the further one, that these performance differences mandate categorically different training, fuelling and weight management, and on that question the same consensus is on the audit's side, reporting that males and females show similar relative magnitudes of hypertrophy, strength, power and cardiovascular adaptation to equivalent training, with the absolute differences tracking larger male starting size rather than a different adaptive response (Hunter et al. 2023). The point is narrower: these differences alter the rate and absolute magnitude of adaptation, not the principles of programming, and the NSCA is explicit that the basic principles of strength training apply to all humans and that shortfalls in the provision and delivery of programs for women are often wrongly attributed to their biology (Dabbs et al. 2026b).

> **Claim 2. Women are built for endurance: they have a higher proportion of type I (slow-twitch) fibres and greater muscle mitochondrial density than men, so they are naturally suited to steady-state work and need different training and fuelling.**

OVERSTATED

> *Certainty of evidence: moderate.*

Not uniquely female. The premise is really two claims, and they fare differently, which is why a flat contradiction overshoots. The fibre-type and fatigue-resistance half is real on average and should be conceded: females carry a greater proportional area of type I fibres than males and are more fatigue-resistant during isometric and slow-to-moderate dynamic contractions normalised to maximal strength, a difference the 2023 ACSM consensus statement treats as reflecting that greater proportional type I area (Hunter et al. 2023), and which the 2026 NSCA position statement likewise

recognises, linking it to the greater proportion of low-threshold motor units in female athletes (Dabbs et al. 2026b).

The largest synthesis of the question sets the size of that difference in context. A meta-analysis of 156 studies and 6,222 healthy adults found that females do carry a greater proportion of type I fibres, but the margin is 3.1 percentage points, 51.4 against 48.3 per cent, which the authors classify as a small effect (standardised mean difference -0.24), alongside a greater type I proportional area of 51.8 against 44.9 per cent (standardised mean difference -0.60) (James et al. 2025). Both sexes therefore sit close to an even split on fibre number, which is some distance from the categorical endurance phenotype the claim describes. The same dataset indicates why the area figure is the larger of the two. Male type II fibres are 40.9 per cent larger in cross-sectional area (standardised mean difference 1.17) while male type I fibres are 17.3 per cent larger, and the authors conclude that the sex differences in proportional area are driven by the large sex differences in cross-sectional area rather than by fibre type distribution (James et al. 2025). A greater female type I proportional area is therefore in part a statement about how much space male type II fibres occupy, rather than evidence of a distinct female oxidative phenotype. The same analysis reports that the fibre type distribution difference was present in young adults but not in the midlife or older groups, where the absolute difference fell to roughly 0.1 to 0.2 percentage points, which bears on a claim most often addressed to women at midlife and beyond. A separate meta-analysis reaches the same pattern (Nuzzo 2024).

The audit's own key citation cuts the same way: alongside its finding of no difference in mitochondrial content, Bailleul found a greater proportional type I fibre area in the women (44 versus 31 per cent, $p = 0.03$), which must be reported here (Bailleul et al. 2025). What does not follow is the prescription. Fibre-type proportion is highly individual and muscle-specific, and it does not dictate how a person should train: high-effort training recruits the full motor-unit pool, including type II fibres, regardless of load, so a real average difference in fibre-type mix is not a basis for training women differently. The greater-mitochondrial-density half of the claim, by contrast, is not supported.

The best direct human comparison finds no sex difference in mitochondrial content or capillarisation of type I or type II fibres in untrained adults (Bailleul et al. 2025). This matters beyond the present claim, because the assertion that females have better mitochondrial respiration and density than men is the premise Sims uses elsewhere to argue that women need less low-intensity endurance work, and the source she gives for it is a study of liver mitochondria in mice (Von Schulze et al. 2018), which is addressed under Claim 20.

The broader synthesis points the same way. A multivariate meta-analysis of 50 studies, 2,258 participants and 39 separate measures of mitochondrial biology found only two aggregate sex differences across the whole field: higher mitochondrial content in the white adipose tissue and isolated leukocyte subpopulations of women, and higher reactive oxygen species production in the skeletal muscle of men (Junker et al. 2022). Skeletal muscle mitochondrial content was not among them. The tissues in which a female mitochondrial advantage does appear are therefore not the tissue the claim is about.

Returning to the human muscle data: the ACSM consensus notes greater whole-muscle oxidative capacity in females, consistent with their larger type I area, but finds the capillary-density data few and contrasting, giving no clear picture of a large sex difference in the muscle's ability to use oxygen, with capillary-density differences small and contrasting (Hunter et al. 2023); and the few direct comparisons of mitochondrial respiratory function are small and conflicting, some reporting higher intrinsic respiration in women and others lower ADP sensitivity and lower absolute respiration at physiological ADP (Cardinale et al. 2018; Miotto et al. 2018).

A more recent comparison suggests that part of that disagreement is methodological. When 13 females and 13 males were matched for peak oxygen uptake normalised to fat-free mass rather than to body mass, there was no effect of sex on mass-specific or intrinsic mitochondrial respiration supported by complex I or by complexes I and II, in the presence or absence of creatine, and no effect of sex on total, intermyofibrillar or subsarcolemmal mitochondrial volume density, or on electron transport chain subunit protein content (Ferguson et al. 2025). The authors note that females typically carry a higher body fat percentage, so normalising peak oxygen uptake to body mass leaves the two groups unmatched for aerobic fitness, and that matching to fat-free mass is the appropriate control. The sample is small, as in the studies it is set against, but it is the comparison in which the fitness matching is handled most carefully, and it reports no sex difference.

The often-cited claim that women are categorically better fat-burners is more limited than it first appears.

A real average difference does exist at moderate intensity: a meta-analysis found men rely more on carbohydrate and women more on lipid during moderate aerobic exercise, though the lipid-oxidation difference was absent in athletes (Cano et al. 2021). But the difference is context-dependent rather than a fixed female trait. The largest synthesis to date (36 studies, 8,990 participants) found that although absolute maximal fat oxidation is lower in women, there is no significant sex difference in maximal fat oxidation expressed relative to lean or fat-free mass, or in the intensity at which it peaks, with cardiorespiratory fitness, body-mass index and exercise modality the main moderators in both sexes and body-mass index and age the factors that shape the sex difference itself, leading the authors to question the assumption that women consistently oxidise more fat than men (Ferri-Marini et al. 2026).

Consistent with this, in a direct comparison of active men and women, fat oxidation per kilogram of fat-free mass did not differ between the sexes whether exercise was prescribed by percentage of maximal heart rate or relative to the ventilatory threshold (Astorino et al. 2026), and the higher-fat-oxidation advantage is in any case confined to low work rates, roughly 41 to 61 per cent of peak aerobic capacity (Hunter et al. 2023). Substrate use during exercise is therefore not, by itself, a determinant of how training should be programmed. Aerobic training can itself induce muscle hypertrophy (Konopka & Harber 2014). The verdict is therefore narrower than a flat contradiction: the fibre-type and fatigue-resistance premise is real on average but does not license a sex-specific training prescription, while the higher-mitochondrial-density premise the claim leans on is not borne out in humans.

2. The menstrual cycle as a training variable

> **Claim 3. Cyclical oestrogen and progesterone produce measurable, predictable changes in substrate metabolism, autonomic recovery (HRV), thermoregulation and connective-tissue laxity that should drive programming.**

OVERSTATED

> *Certainty of evidence: moderate (large, consistent syntheses built on mostly low-quality primary studies).*

The best available synthesis finds cycle phase has at most a trivial and highly variable effect on performance, with performance slightly lower in the early follicular phase, on low-quality evidence (McNulty et al. 2020: 78 studies, only 8% rated high quality, 68% low or very low, concluding that general phase-based guidelines cannot be formed). Meta-analyses continue to find cycle-phase effects trivial and highly variable: a meta-analysis of strength measures across the cycle found trivial changes amid high bias (Blagrove et al. 2020), and a multimethod systems-physiology review judged the impact of the cycle and oral contraceptives on performance, substrate oxidation and hypertrophy to be small or nonexistent (D'Souza et al. 2023); a 2025 systematic review restricted to studies that verified cycle phase by serum hormones likewise found no consistent or robust phase effect on performance, with 58% of its 19 studies reporting an effect on at least one outcome but in no consistent direction or magnitude (Schlie et al. 2025), and a 2025 multi-author methodological position paper argues that studies relying on assumed or estimated cycle phases cannot support phase-based conclusions in the first place (Elliott-Sale et al. 2025).

The Norwegian FENDURA project in endurance-trained women reached the same conclusion: there is no point in the cycle where all women are reliably fitter or less fit, and performance-determining variables do not track cycle phase (Taylor et al. 2024), and acute physiological and perceptual responses to low-intensity and interval sessions show only small, inconsistent phase effects (Taylor et al. 2026). A controlled study using LH-confirmed phasing found force, velocity and power in the half-squat unchanged across the early follicular, late follicular and mid-luteal phases at every load from 20 to 80% of 1RM (Romero-Moraleda et al. 2019). A separate repeated-measures study using ovulation-confirmed phasing across two tracked cycles found peak power, mean power, anaerobic capacity, fatigue index and perceived exertion on a 30-second Wingate test unchanged across the early follicular, late follicular and mid-luteal phases in eumenorrheic women (Lambright, Bunn & Newmire 2026).

Core temperature does rise modestly in the luteal phase, but the performance impact in trained women is small. More recent work fits the same pattern once the size of the effects is read honestly: muscle desaturation during interval work was statistically greater in the low-hormone phase, but by about 1.5 percentage points of tissue saturation, a difference the authors read as a real phase effect and this audit judges small against the training-induced changes in the same measure (Baker et al. 2026); and the circulating extracellular-vesicle and metabolite response to resistance exercise was present in all groups, minimally affected by group among metabolites and

not strongly influenced by hormonal-contraceptive use, although follicular-phase women showed distinct baseline and exercise-responsive microRNA profiles (Kargl et al. 2026).

Phase effects are real in principle and trivial, inconsistent and individual in practice. This position is now echoed by the 2026 NSCA position statement on female athletes, which concludes there is only a trivial impact of cycle phase on performance, finds no systematic relationship between cycle phase and performance measures, and states there is not sufficient evidence to support modifying resistance training prescription by cycle phase for most active female athletes (Dabbs et al. 2026b).

> **Claim 4. Women should track their cycle for at least three months and program by phase: push hard in the follicular phase, prioritise recovery and lower intensity in the late luteal phase.**

CONTRADICTED

> *Certainty of evidence: moderate.*

The single best-designed test examined this question: a within-participant randomised controlled trial randomised each leg of 24 eumenorrheic women to two of four conditions (non-exercising control, evenly loaded, high volume in the follicular phase, high volume in the luteal phase) across three full menstrual cycles, and found no difference in thigh lean mass, vastus lateralis cross-sectional area or 1RM strength between follicular-loaded, luteal-loaded and evenly-loaded conditions; total volume-load, not cycle phase, drove the gains (D'Souza et al. 2026). A 2025 critical review in the *Strength and Conditioning Journal* reaches the same verdict on the wider literature: only five training studies have ever tested cycle-based strength programming (78 participants, mostly untrained, mostly single-limb designs, none in a hormonally verified eumenorrheic cohort, and none compared against ordinary periodisation), and only one endurance study exists, in which a cycle-tailored program and a standard polarised program improved performance equally (Mikkonen & Häkkinen 2025).

The studies often cited to claim a follicular-phase advantage are confounded by cross-education and temperature-only phase estimation in single-limb designs (Sung et al. 2014), by measurement-timing and taper artefacts, and by unsupervised training and the mixing of naturally cycling women with contraceptive users (Wikström-Frisén et al. 2017), so the apparent advantage cannot be attributed to hormones. An umbrella review separately concluded it is premature to claim reproductive hormones appreciably influence performance or adaptations, and that because most women do not have textbook 28-day cycles, phase-based prescription is just arbitrary undulating periodisation (Colenso-Semple et al. 2023).

The most fully worked mechanistic case for phase-based fuelling and training is Oosthuyse and colleagues' review, which argues from oestradiol and progesterone effects on substrate metabolism and satellite-cell function that follicular-phase training and phase-specific fuelling should be advantageous (Oosthuyse et al. 2023); engaged on its own terms, that prediction is not borne out by the human training and performance trials above. The approach is also irrelevant to the hundreds of millions of women on hormonal contraceptives and to peri- and post-menopausal

women, who have no such phases. Tracking for symptom awareness is harmless; programming by phase is not evidence-based. The 2026 NSCA position statement reaches the same conclusion, finding insufficient evidence to support modifying resistance training prescription by menstrual cycle phase for most active female athletes, while noting that tracking the cycle remains useful for monitoring symptoms and recovery (Dabbs et al. 2026b).

> **Claim 5. Oral contraceptives suppress natural hormone fluctuations, blunt training adaptations and particularly impair lean-mass accretion, so contraceptive status changes all recommendations.**

OVERSTATED

> *Certainty of evidence: low (sparse, mostly low-quality trials; an absence of evidence, not a demonstrated absence of an effect).*

A meta-analysis of 42 studies found oral contraceptive users may show slightly inferior but trivial performance compared with non-users, on low-quality evidence (Elliott-Sale et al. 2020), and reviews find no convincing evidence that contraceptives meaningfully blunt lean-mass gains (D'Souza et al. 2023; Nolan et al. 2024). A dedicated meta-analysis of hormonal contraceptive use and resistance training adaptations reached the same conclusion, finding no clear impairment of hypertrophy, power or strength gains in users versus non-users (Nolan et al. 2024). A systematic review dedicated to the cycle and contraceptives in resistance training found the evidence conflicting and methodologically weak across 17 studies and 418 participants, and while it suggested hormones may affect responses, the subsequent meta-analysis found no such impairment (Thompson et al. 2020; Nolan et al. 2024).

The same applies to aerobic capacity: the pooled performance data show only trivial, highly heterogeneous average effects, so the specific claim of a reduced peak aerobic capacity is not borne out at the group level (Elliott-Sale et al. 2020), and no pooled data on aerobic training adaptation in contraceptive users exist to support the claim of a blunted ability to adapt to high-intensity training. The strong version of the claim rests on isolated studies and on a mechanism, progestin competing at anabolic-hormone receptors, that is presented as if it were a measured training outcome rather than a hypothesis; the pooled human evidence does not show the predicted impairment.

The 2023 ACSM consensus statement reaches the same reading, judging combined oral contraceptives to have at most a minor and variable effect on performance, of unclear practical significance, and noting that although the training-adaptation results are discordant, the limited number of studies collectively show oral contraceptive use does not markedly impair or enhance adaptations to exercise training (Hunter et al. 2023), a reading the resistance-training meta-analysis bears out (Nolan et al. 2024). The valid core is narrower: contraceptive type and dose do shift the hormonal milieu, but the claim that adaptations, especially muscle gain, are meaningfully suppressed is not supported.

> **Claim 6. Athletes who need contraception should prefer a hormonal IUD over the pill, because it delivers a localised dose of progestin without the systemic oestrogen and progesterone of oral contraceptives.**

UNTESTED

Certainty of evidence: very low (no head-to-head study exists).

No study has compared a hormonal IUD against the oral contraceptive pill for any athletic performance or training-adaptation outcome, so the recommendation can be neither supported nor refuted on direct evidence: this is an untested comparison, an empty cell rather than a demonstrated result. It also rests on a premise the evidence does not support: the pooled data show oral contraceptive effects on performance and muscle adaptation are trivial or absent (Elliott-Sale et al. 2020; Nolan et al. 2024), so there is no established performance deficit for an IUD to circumvent. A hormonal IUD may well be a reasonable individual choice on other grounds, such as side-effect profile, bleeding control or convenience, but as performance guidance the claim of categorical superiority is untested, and the burden sits with whoever asserts it.

> **Claim 7. Because the pill produces a withdrawal bleed rather than a true period, it masks the menstrual irregularities that are an early warning sign of low energy availability or RED-S.**

SUPPORTED

Certainty of evidence: moderate.

This is sound and is one of the stronger points in this area. Withdrawal bleeding on combined oral contraceptives can conceal the loss of natural menstruation that signals functional hypothalamic suppression, removing a visible early indicator of low energy availability, which the IOC consensus identifies as a key clinical warning sign for RED-S (Mountjoy et al. 2023). The caution is a legitimate monitoring point rather than an argument for or against contraception as such.

3. Nutrition

> **Claim 8. Women need more protein, timed strategically: about 1.4 to 2.2 g/kg/day toward the upper end in the luteal phase and menopause, a 0.32 to 0.38 g/kg post-exercise bolus, and about 10 g essential amino acids near exercise to overcome anabolic resistance.**

OVERSTATED

Certainty of evidence: moderate. Sub-verdicts: protein amount in the athlete range: Supported (sex-neutral); luteal-phase and menopause up-titration and the female-specific post-exercise timing/bolus: Overstated, with the luteal claim directly contradicted.

Not uniquely female. The claimed 1.4 to 2.2 g/kg band brackets the standard athlete recommendation of 1.6 to 2.2 g/kg, in which a post hoc check found no apparent sex difference, on limited female data (Morton et al. 2018), so the numbers themselves are reasonable. Total daily protein, not timing, is the dominant predictor of hypertrophy and strength, and the narrow post-exercise window is refuted (Schoenfeld, Aragon & Krieger 2013). The luteal-phase protein claim is directly contradicted: cycle phase does not alter muscle protein synthesis or whole-body myofibrillar breakdown after resistance exercise (Colenso-Semple et al. 2025). Acute post-exercise spikes in anabolic hormones do not meaningfully drive muscle protein synthesis or hypertrophy (Van Every, D'Souza & Phillips 2024), which further undercuts the case for a female-specific post-exercise protein window.

Anabolic resistance and higher protein needs are real in older adults of both sexes, not a female-specific phenomenon, and postmenopausal women retain a normal response to feeding and loading: a single 35 g dose of whey maximally stimulates myofibrillar protein synthesis even during energy restriction (Larsen et al. 2023), and post-exercise whey shifted the timing rather than the four-hour cumulative synthetic response in post-menopausal women, a pattern consistent with age-related rather than oestrogen-driven anabolic resistance (McKenna et al. 2024). Human work is consistent with sex-neutral drivers, though not decisive on the point: in energy deficit a bout of resistance exercise restores muscle protein synthesis, and post-exercise protein raises it further in a dose-dependent manner (Areta et al. 2014, a small mixed-sex study of eight men and seven women without a sex-stratified analysis).

A 2025 review of protein requirements for women across the lifespan, written by researchers who study protein in female cohorts, concluded that any sex-based difference in protein needs is minimal or non-existent, that needs do not change across the menstrual cycle or with oral contraceptives, that age-related anabolic resistance is driven by ageing itself rather than by oestrogen loss, and that active peri- and post-menopausal women require no different protein target from premenopausal women; it also notes that in an energy deficit the primary driver of lean-mass retention is resistance training, not protein intake (D'Souza et al. 2025, an industry-published review that is not peer reviewed). The careful case for a hormonal influence on muscle and tendon protein metabolism has been made and is worth acknowledging, but it rests on sensitivity to loading rather than a large change in resting synthesis and stops well short of a female-specific protein-timing prescription (Hansen 2018). The numbers are reasonable; the female-specific timing and luteal framing are not.

> **Claim 9. Carbohydrates are not the enemy; low-carb and ketogenic diets are contraindicated for active women, carbohydrate should be increased in the luteal phase, and pre-exercise carbohydrate matters more for women.**

OVERSTATED

> *Certainty of evidence: moderate.*

The luteal-phase carbohydrate claim inherits the same problem as the luteal-phase protein claim: a randomised controlled trial found that menstrual cycle phase does not influence training-induced muscle hypertrophy or strength (D'Souza et al. 2026). On ketogenic diets specifically, the female and sex-comparative evidence likewise does not support a female-specific contraindication. They reliably aid fat loss and, when resistance training and protein are adequate, preserve muscle: in trained women, eight weeks of training on a ketogenic diet reduced fat mass and maintained fat-free mass, although only the normal-diet group gained strength and jump performance and neither group significantly changed fat-free mass, so keto preserves rather than maximises gains (Vargas-Molina et al. 2020), and a comparable fat-loss-with-lean-preserved pattern appears in overweight women (Jabekk et al. 2010).

The performance cost is real but concentrated in high-intensity work and is not sex-specific: in a mixed cohort of trained women and men, four days of a ketogenic diet lowered Wingate peak power by about 7% and intermittent high-intensity running distance by 15% (Wroble et al. 2019), and in a five-athlete pilot case study time to exhaustion fell by about two minutes while VO2max did not change significantly (Zinn et al. 2017). Maximal strength, by contrast, is largely spared: in competitive powerlifters and weightlifters including women, three months on a ketogenic diet reduced body and lean mass but left 1RM lifting performance unchanged (Greene et al. 2018), and pooled meta-analyses across both sexes find no significant difference in maximal bench-press or squat strength between ketogenic and higher-carbohydrate diets (Vargas-Molina et al. 2024).

The clearer caveat is body composition without training: in a four-week controlled feeding crossover in 17 normal-weight women without a training intervention, a ketogenic diet reduced lean mass more than their fat mass at matched energy intake, so protein and resistance training matter (Burén et al. 2024). None of this singles women out. Where the sexes were compared directly the shift toward fat oxidation was larger in the men, and the women already oxidised more fat at baseline (Durkalec-Michalski et al. 2019), and an ISSN position stand co-authored by Sims concludes there is insufficient evidence that ketogenic diets affect men and women differently, while allowing a plausible mechanistic basis for one (Leaf et al. 2024).

The one suggestive exception is a single-arm study in which women showed a larger fall in peak aerobic capacity than men, though without a control group (Durkalec-Michalski et al. 2021). The accurate reading is that ketogenic diets are a poor fit for high-intensity and explosive performance in either sex and can still support fat loss, and even preserve maximal strength, when protein and resistance training are adequate; a blanket female-specific contraindication outruns the data, even as the question of subtle sex differences remains open.

In addition to the core point being sex-neutral, the add-on claims (luteal-phase carbohydrate loading, carbohydrate being more important pre-exercise for women specifically) rest on small and inconsistent data and are not established. The International Society of Sports Nutrition position stand on the female athlete, on which Sims is first author, does recommend a luteal-phase carbohydrate emphasis, though it stops short of a female-specific low-carbohydrate ban (Sims et al. 2023), and a 2025 systematic review of 71 human intervention studies found carbohydrate

strategies understudied in women and offered no basis for a cycle-phase carbohydrate prescription (Larrosa et al. 2025).

This point has already been contested in the peer-reviewed literature rather than only in public commentary: a formal reply published in the same journal argued specifically that advising women to raise carbohydrate intake for small luteal-phase fluctuations runs counter to general public health messaging and is not warranted for most active women (Charkoudian et al. 2023). That reply is framed as a difference of perspective rather than a censure, and is treated here as the ordinary scholarly exchange it is; the relevant point for this audit is simply that the recommendation is disputed by researchers in the field, in print, and not only by this document. On the constructive side, part of carbohydrate's benefit in events under an hour, when glycogen is far from depleted, is expectancy, since telling cyclists they had received carbohydrate improved 40 km time-trial power by about 4% whether or not they had (Clark et al. 2000), and a carbohydrate mouth rinse improves performance through a mouth-to-brain signal in both fasted and fed states (Carter et al. 2004; Deng et al. 2025), a practical tool that reinforces the point that carbohydrate is an ally rather than an enemy.

> **Claim 10. Intermittent fasting and fasted training are contraindicated for active women: fasted training causes greater cortisol and stress-hormone response, promotes muscle catabolism and visceral fat storage, and chronically suppresses the HPO axis.**

OVERSTATED

> *Certainty of evidence: moderate.*

Not uniquely female. Rodent-based claim, contradicted by human data. Direct male-female comparisons report little to no difference in training adaptation in both the fed and fasted state, which undercuts the female-specific harm claim. The specific mechanism (fasted training drives greater cortisol and visceral fat gain in women) is not established in human trials, and the trials that exist point the other way. Longer-term evidence also contradicts this rodent-derived cortisol fear: two years of calorie restriction (prescribed at 25%, achieved at about 19.5% then 9.1%) in 218 adults did not lower IGF-1 or cause a sustained cortisol rise, though cortisol rose transiently at one year before returning to baseline (Fontana et al. 2015). In overweight women, interval training performed fed or fasted improved body composition and muscle oxidative capacity equally (Gillen et al. 2013).

Fasted versus fed aerobic exercise produced no meaningful difference in body composition (Schoenfeld, Aragon et al. 2014). A randomised clinical trial found overnight-fasted and fed resistance training produced comparable adaptations (Vieira et al. 2025a), and nutrient timing did not materially change adaptations in resistance-trained women (Pihoker et al. 2019). Six weeks of sprint-interval training performed fasted or fed improved VO2peak and knee-flexion strength equally in a mixed cohort of 35 adults, 22 of them women, with no penalty to the fasted group (Tan et al. 2021). A pooled analysis of three alternate-day-fasting studies (n = 75) similarly found weight, fat, lean mass, insulin and blood pressure fell equally in pre-menopausal women, post-menopausal

women and men, though the authors note the small male subsample (n = 10) limited power to detect sex differences (Lin et al. 2020).

Time-restricted eating trials in men and in pre- and post-menopausal women, including a 12-month randomised trial, produced weight loss with no change in testosterone or sex hormone binding globulin in men or women, and no change in oestrogens or progesterone in the post-menopausal women in whom they were measured, with only a modest fall in DHEA (Lin et al. 2024; Kalam et al. 2023), and a review of intermittent-fasting trials found muscle mass and strength were preserved even where androgens dipped (Cienfuegos et al. 2022). Set against claims of superior fasting-based weight loss, a one-year randomised trial found alternate-day fasting no better than daily calorie restriction, with higher dropout (Trepanowski et al. 2017), and the TREAT trial found 16:8 time-restricted eating no more effective than regular eating, with some lean-mass loss (Lowe et al. 2020), an outlier that the Varady group's review of the TRE trials attributes to reduced protein intake or uncontrolled hydration at the DXA scan, since across the other trials the weight lost on time-restricted eating is mainly fat (Ezpeleta et al. 2024).

The wider evidence agrees. A meta-analysis of 24 randomised trials (1,768 participants) found intermittent fasting and daily calorie restriction produce the same weight loss, a difference of 0.26 kg (Elortegui Pascual et al. 2023); an umbrella review of 23 meta-analyses of fasting trials found high-certainty reductions in waist circumference, fat mass, fasting insulin and LDL cholesterol in adults with overweight, with fat-free mass higher, not lower, than under usual eating (Sun et al. 2024); a 12-month randomised trial in 90 adults with obesity, mostly Black and Hispanic women, found time-restricted eating and calorie restriction lost 4.6 and 5.4 kg against control with no loss of lean mass or bone mineral density in either (Lin et al. 2023); and in 80 adults with obesity and fatty liver disease, 81 per cent of them women, three months of alternate-day fasting combined with five aerobic sessions a week cut liver fat and fat mass without any difference in lean mass between fasting, exercise, combined and control groups (Ezpeleta et al. 2023).

None of these trials reported a female-specific harm, undercutting the idea that fasting protocols carry a particular hormonal or lean-mass penalty for women. Measured over a full 24 hours in a metabolic chamber, exercising before breakfast actually raised whole-day fat oxidation in women, the opposite of the harm claim (Iwayama et al. 2017).

The female-specific hormonal-harm argument leans on animal studies rather than trials in active women: the fear that fasting disrupts the menstrual cycle traces largely to a 2013 experiment in juvenile rats (Kumar & Kaur 2013), whereas a review of human trials found intermittent fasting does not affect oestrogen, gonadotropins or prolactin in women, and where androgens fell, in premenopausal women with obesity, the authors regarded the change as potentially beneficial rather than harmful (Cienfuegos et al. 2022), a reading the same group has carried forward from its 2022 Nature Reviews Endocrinology review, which noted that reproductive hormones had then been assessed in only two fasting trials, with androgens, sex hormone binding globulin and prolactin unchanged after 24 weeks of 5:2 fasting in premenopausal women, and that fasting spares lean mass no better and no worse than daily calorie restriction (Varady et al. 2022), through its 2024 commentary (Varady et al. 2024) and the 12-month trial data (Lin et al. 2024).

A 2025 narrative review of the 23 trials that have combined the 5:2 diet, alternate-day fasting or time-restricted eating with exercise for more than four weeks, from the same Chicago group, concluded that fasting does not impair adaptation to exercise training and may improve explosive strength, endurance and maximal oxygen consumption, while its effect on fat-free mass depends on weight status and exercise dose (Gabel et al. 2025). Time-restricted eating itself preserves or increases lean mass when combined with concurrent training in a mostly female overweight cohort (Kotarsky et al. 2021), a meta-analysis of intermittent fasting or calorie restriction combined with exercise found most performance measures unimpaired, at the cost of some fat-free mass (Kazeminasab et al. 2025b), and in an 8-week randomised trial combining 16:8 eating with Zone 2 training (52 randomised, 36 analysed, 28 of them women, with no sex-specific analysis), it affected mood, affect and eating behaviour no differently from training fed or from eating freely (Hofstätter et al. 2025); the kisspeptin and reproductive-axis concern again traces to a 24-hour fasting study in female mice (da Silva Mansano et al. 2023).

The newest trial from the same group points the same way in the women most often warned off fasting: in 76 premenopausal women with polycystic ovary syndrome, six months of time-restricted eating (a 1 pm to 7 pm window) and daily calorie counting produced the same weight loss of about 4.5 kg and both lowered testosterone, with time-restricted eating reducing the free androgen index and HbA1c more, and neither approach worsening menstrual regularity (Corapi et al. 2026). The pooled resistance-training evidence points the same way once the fasting type is separated out. A systematic review of four trials and 114 participants found no difference between fasted and fed states in fat-free mass (SMD 0.529, 95% CI -0.801 to 1.859), muscle hypertrophy (SMD 0.645, -0.753 to 2.042) or strength (SMD 0.415, -0.399 to 1.228), with an overall body-fat result favouring the fasted state (SMD -1.001, -1.970 to -0.032).

The subgroup analysis is the informative part and it cuts both ways: in the overnight-fasting studies, which are the scenario actually at issue, neither fat-free mass (SMD -0.489, -1.216 to 0.237) nor body fat (SMD -0.206, -0.724 to 0.313) differed, and both apparent overall effects came from the Ramadan studies, where fluid restriction and shifted daily intake are additional variables. Three of the four trials were at high risk of bias, only two included women, and no result is reported by sex, so the review supports the absence of a general effect rather than settling the female-specific question, and its authors call for work in women (Vieira et al. 2025b).

There is a valid concern, and Sims states a narrower version of it than the popular one: in her own writing she confines the warning to prolonged windows rather than an overnight fast, and exempts women with polycystic ovary syndrome, obesity or inactivity, for whom she regards intermittent fasting as potentially useful. On that narrower reading the disagreement is smaller than it appears. Chronic under-fuelling can suppress the hypothalamic-pituitary-ovarian axis, but that is the low energy availability pathway, not a property of acute fasted training, and a 2026 review of fasting and reproductive function draws the same line, finding benefit in metabolically compromised women and advising caution only in young, lean or energy-deficient individuals (La Vignera & Condorelli 2026). The same formal reply, to the ISSN position stand on which Sims is first author, warned more generally that its framings of hydration, heat, the cycle and carbohydrate could lead readers to

wrongly conclude women are at greater risk from ordinary exercise training (Charkoudian et al. 2023). The blanket contraindication outruns the human data.

> **Claim 11. Low energy availability (RED-S), not caloric excess, is the primary nutritional threat to female athletes, and it is far more prevalent than commonly recognised.**

SUPPORTED

> *Certainty of evidence: high.*

Read as a claim about female athletes, which is how it is stated, this is correct and strongly supported, so it is marked Supported; the qualification that follows is a note on scope, not a deduction against the claim. The threat is real and under-recognised. Low energy availability is the central mechanism of Relative Energy Deficiency in Sport, which drives a syndrome of impaired bone, reproductive, metabolic, immune and performance outcomes, and under-fuelling relative to training load is a major, under-recognised nutritional threat (Mountjoy et al. 2023). The current consensus is also explicit that REDs affects athletes of both sexes: the 2023 IOC statement and a 2024 review in Endocrine Reviews both describe the syndrome and its endocrine consequences in men as well as women, with male-specific manifestations including suppressed testosterone and impaired bone health (Mountjoy et al. 2023; Angelidi et al. 2024).

The specific numbers attached to the claim do not carry the weight placed on them. The figure of 30 kcal/kg of lean body mass per day comes from a five-day laboratory study in 29 habitually sedentary, regularly menstruating young women, in which energy availability was set at 45 and either 10, 20 or 30 kcal/kg while participants expended 15 kcal/kg per day in supervised exercise; luteinising hormone pulse frequency fell and amplitude rose only below 30, and the authors describe the disruption as occurring deep into negative energy balance (Loucks & Thuma 2003).

The same author later showed the response is not general to adult women: across the same protocol, low energy availability reduced luteinising hormone pulse frequency in adolescents but not in adults ($P = 0.39$), and suppressed 24-hour mean luteinising hormone in adolescents but not in adults ($P = 0.72$), the effect disappearing by about 14 years of gynaecological age (Loucks 2006). A critical review by the group that ran the largest downstream trial concludes that the proposed threshold is not a cut-off below which menstrual disturbances occur but represents an increased risk of experiencing them, noting that disturbances occurred both above and below it and that changes in luteinising hormone pulse frequency predicted only luteal phase defects rather than anovulation or oligomenorrhoea (Salamunes, Williams & De Souza 2024).

The paired male figure of 15 kcal/kg is not a measured threshold at all. It is the low energy availability condition that investigators imposed on six exercising men, in whom it reduced leptin by 53 to 56 per cent and insulin by 34 to 38 per cent, whether or not exercise was used to create the deficit, while leaving the other metabolic hormones unchanged (Koehler et al. 2016). Presenting 15 and 30 as two measured tipping points, with the gap between them explaining a categorical sex difference in vulnerability, therefore compares an age-dependent laboratory threshold in sedentary

women, since reframed by its own investigators as a risk gradient, against an experimental dose given to six men.

The underlying claim that low energy availability is the primary nutritional threat does not depend on those numbers and is not weakened by their removal. Framing low energy availability as primarily a female-athlete problem is therefore a historical artefact of the condition having first been characterised through the female athlete triad, not a reflection of who is at risk. The position is established across a decade of consensus documents, including the ACSM Female Athlete Triad position stand (Nattiv et al. 2007), the 2014 Female Athlete Triad Coalition statement (De Souza et al. 2014), the IOC RED-S 2018 update (Mountjoy et al. 2018) and the 2025 Female Athlete Triad clinical guidelines (Williams et al. 2025), and a 2024 systematic review and meta-analysis of 59 studies confirms the high prevalence of low energy availability, approaching 45% of athletes (Gallant et al. 2024).

Emerging work refines this further: persistent within-day energy deficits are associated with a lower resting metabolic rate and higher cortisol even when 24-hour intake looks adequate (Fahrenholtz et al. 2018; Torstveit et al. 2018), and a 2025 hypothesis paper proposes low T3 as the link between low carbohydrate availability, low energy availability and REDs (Hackney et al. 2025), which supports fuelling around training rather than skewing intake to the evening. Energy availability is also hard to measure reliably in the field, which complicates individual diagnosis. In short, that low energy availability rather than caloric excess is the primary nutritional threat, and is more prevalent than commonly recognised, is accurate and is one of Sims' strongest points; the only caveat is one of scope rather than correctness, since the syndrome is not sex-specific and men are equally susceptible, a fact the female-athlete-triad history tends to obscure. The 2026 NSCA position statement reinforces this scope: it describes RED-S as coined to identify health and performance consequences affecting all athletes, and reports symptom data from mixed male and female cohorts (Dabbs et al. 2026a).

> **Claim 12. Creatine: women have lower baseline creatine stores and benefit for strength, power, cognition and brain health, especially peri- and post-menopause; it has been wrongly framed as a male supplement.**

SUPPORTED

> *Certainty of evidence: low for the female-specific claims (small, underpowered female subgroups); high for the general benefit of creatine.*

Creatine does benefit women, and framing it as a male supplement is wrong: supplementation supports strength, power, body composition and, with resistance training, bone, with emerging benefits for mood and cognition, including after menopause, though no trial has yet tested perimenopausal women specifically (Smith-Ryan et al. 2025). The premise usually offered for it, however, does not survive checking, and the audit reports this plainly because the claim is repeated widely and because earlier drafts of this audit carried it too. The figure behind "women have lower creatine stores" traces to a single primary source, which estimated rates of creatine synthesis of

7.7, 5.6 and 3.7 mmol/day in men aged 20 to 39, 40 to 59 and 60 and over, and stated that the rates for women were about 70 to 80 per cent of those in men (Brosnan & Brosnan 2007).

That is a modelled synthesis rate derived from national food-consumption and creatinine-excretion data, expressed in absolute terms rather than per unit of lean mass; since more than 90 per cent of body creatine sits in skeletal muscle, a lower absolute rate is what lower muscle mass and lower food intake alone would predict. In the restatement the figure is inverted. A lifespan review reports that females exhibit 70 to 80 per cent lower endogenous creatine stores than males, citing Brosnan (Smith-Ryan et al. 2021), which turns women being at 70 to 80 per cent of men into women being 70 to 80 per cent below them, and turns a synthesis rate into a store. The ISSN position stand on the female athlete carries the inverted figure forward and narrows it again, to 70 to 80 per cent lower endogenous intramuscular phosphocreatine stores, citing the same source (Sims et al. 2023).

The same lead author restates the figure correctly four years later as 20 per cent lower synthesis rates alongside a 30 to 40 per cent lower dietary intake, again citing Brosnan (Smith-Ryan et al. 2025), without noting the discrepancy. The direct measurement points the other way. In the one human muscle-biopsy study of the question, 50 healthy adults sampled by percutaneous needle biopsy, total muscle creatine per kilogram of fat-free solids was 145 plus or minus 10 mmol/kg in the women against 132 plus or minus 10 in the men, which the authors describe as much lower in the male subjects than in the female subjects (Forsberg et al. 1991). The ISSN position stand cites that same biopsy study in the same sentence, for women having roughly 10 per cent higher resting intramuscular creatine concentrations, so a large deficit and a small surplus in the same tissue are asserted together (Sims et al. 2023).

None of this weakens the case for creatine in women, which rests on trial evidence rather than on a deficiency. The point is narrower: the deficiency premise is not supported, and the recommendation does not need it. A 2026 meta-analysis of seven randomised trials adds detail here, finding that creatine at 5 g/day or more with resistance training produced small but meaningful lean-mass and strength gains in post-menopausal women specifically, though bone effects remain unclear (Naddafha et al. 2026). The mood evidence most often cited is real but singular: in 52 women with major depressive disorder, eight weeks of escitalopram plus creatine 5 g/day produced significantly greater improvement in Hamilton Depression Rating Scale score than escitalopram plus placebo, evident by week 2 and maintained at weeks 4 and 8 (Lyoo et al. 2012).

That is one trial, at 5 g/day, as augmentation in diagnosed depression, which is narrower than the plural randomised trials often invoked for creatine as a general mood aid in healthy women. The hair-loss concern has now been tested directly rather than merely lacking evidence: a 12-week randomised controlled trial found no group-by-time interaction for any hormonal or hair-related outcome, although its participants were required to be biological males, so it addresses the mechanism rather than women specifically (Lak et al. 2025). The one qualifier is certainty, not direction: the female-specific evidence base is thin, so a confident claim of an established female-specific benefit runs ahead of what has been demonstrated, but that is an absence of strong evidence rather than evidence of absence.

A broader meta-analysis confirms creatine's lower-limb strength benefit independent of population characteristics (Lanhers et al. 2015), and where sex-stratified analyses find the effect smaller or non-significant in women, the female subgroups are strikingly underpowered: the analysis reporting a significant strength gain in males but not females in adults under 50 rested on roughly 460 male against 49 female participants, with 20 of its 23 studies male-only and the sex difference itself only a non-significant trend ($p = 0.067$) (Wang et al. 2024); lean mass rose by about 1.46 kg in males versus a non-significant 0.29 kg in females (Delpino et al. 2022); and a 2025 meta-analysis found both sexes improve on some lifts, men more consistently across the squat, vertical jump and peak power, though its one significant female bench-press effect was about 0.15 kg, real but of no practical size (Kazeminasab et al. 2025a).

Smith-Ryan's review concludes women most likely respond much as men do (Smith-Ryan et al. 2025), and the thin female evidence base is itself an under-study artefact that vindicates Sims' point that women have been under-researched. The claim is therefore supported: the benefit is real and the male-only framing is wrong; only the strength of the female-specific evidence, not its direction, is limited, and reading these underpowered subgroups, which simply failed to find an effect, as showing that creatine does not work in women would invert what they actually mean.

> **Claim 13. Iron: female athletes have a high prevalence of iron deficiency, including non-anaemic deficiency, which impairs endurance and recovery; routine screening is warranted.**

SUPPORTED

> *Certainty of evidence: high.*

Supported and mainstream. Iron deficiency, including non-anaemic iron deficiency, is common in female athletes and impairs performance, and periodic screening is a reasonable practice (narrative review: Sim et al. 2019). A randomised controlled trial provides the causal evidence, albeit in sedentary rather than athletic women: iron supplementation in 73 iron-depleted, non-anaemic, untrained young women improved endurance performance at submaximal and near-maximal intensities, without raising estimated maximal oxygen uptake (Pompano & Haas 2019).

> **Claim 14. Vitamin D and calcium are important for bone health as oestrogen declines; adaptogens such as ashwagandha help manage cortisol during high-stress training.**

OVERSTATED

> *Certainty of evidence: moderate for the bone claim, very low for the adaptogen claim. Sub-verdicts: vitamin D and calcium for bone health: Supported; ashwagandha/adaptogens for cortisol and training outcomes: Untested.*

Vitamin D and calcium for bone health are mainstream and supported, endorsed by the NIH consensus statement (NIH Consensus Development Panel 2001), although the American College of Physicians guideline rates their fracture-prevention effect as uncertain (Qaseem et al. 2017), and a 2025 meta-analysis of 13 randomised trials shows that adding exercise to calcium and vitamin D

further improves spine and femoral-neck density in post-menopausal women (Bai et al. 2025). The adaptogen claim is weaker: evidence for ashwagandha and cortisol or training outcomes is preliminary and not established for the proposed context. A 2026 randomised trial reported that 600 mg/day ashwagandha for six weeks stabilised salivary cortisol and perceived recovery in female team-sport athletes and improved jump performance in the males (Coope et al. 2026), but this remains early and mixed rather than confirmatory. Taken together, the bone-health portion is solid while the adaptogen portion is not, so the claim as a whole is overstated.

> **Claim 15. Sex differences in substrate metabolism mean women are better fat burners and men lose fat more easily, so women should fuel and train to suit their fat-burning physiology.**

OVERSTATED

> *Certainty of evidence: moderate.*

Not uniquely female. Overstated and in part contradicted. Real average differences in substrate use exist: a meta-analysis confirmed men rely more on carbohydrate and women more on lipid during moderate aerobic exercise, but the lipid-oxidation difference was absent in athletes (Cano et al. 2021). Yet the headline that women are better fat burners does not survive scrutiny. The largest synthesis to date (36 studies, 8,990 participants) found that although absolute maximal fat oxidation is lower in women, there is no significant sex difference once it is expressed relative to lean or fat-free mass, or in the intensity at which fat oxidation peaks, with cardiorespiratory fitness, body-mass index and exercise modality the main moderators and body-mass index and age the factors that shape the sex difference itself (Ferri-Marini et al. 2026); and in a direct comparison of active men and women, fat oxidation per kilogram of fat-free mass did not differ between the sexes whether exercise was prescribed by percentage of maximal heart rate or relative to the ventilatory threshold (Astorino et al. 2026).

The 2023 ACSM consensus statement frames it the same way: women do on average oxidise more fat than men during endurance exercise, but that advantage is confined to low work rates, roughly 41 to 61 per cent of peak aerobic capacity, and is unlikely to translate into an advantage at the higher intensities most events demand (Hunter et al. 2023). The sex difference in substrate mix does not, by itself, determine performance or dictate a fat-loss strategy. Fat loss is governed by 24-hour energy balance, not by which fuel is burned within a session, and the short post-exercise respiratory-exchange measurements often cited are misleading, because fed-state training simply shifts fuel use within the day with a later catch-up. The claim that men lose fat more easily is not supported when energy balance and training status are matched. The accurate statement is that substrate-use differences are real but do not translate into a sex-specific fat-loss strategy.

4. Training

> **Claim 16. Lift heavy: resistance training with heavy loads is the most important modality for women across the lifespan, and women will not bulk up because of lower testosterone.**

OVERSTATED

Certainty of evidence: moderate.

Not uniquely female. There is a valid core: resistance training is a valuable modality for women across the lifespan, and lower testosterone means large involuntary muscle gain is unlikely. Relative hypertrophy is essentially identical between sexes and upper-body strength gains can favour women (Refalo et al. 2025; Roberts, Nuckols & Krieger 2020). The overreach is the word heavy and the framing as the single most important modality. **It also implies a female-specific prescription where none exists: resistance training adaptations are not sex-dependent, so nothing here is unique to women, contrary to the implication of the framing** (Refalo et al. 2025; Roberts, Nuckols & Krieger 2020; Hunter et al. 2023).

The prescription the human evidence supports is resistance training, not heavy resistance training specifically: for muscle, a broad range of loads taken close to failure produces similar hypertrophy (Schoenfeld, Grgic, Ogborn & Krieger 2017), and for bone, low and moderate loading both produce significant gains; high-intensity loading gave a larger point estimate at the lumbar spine that was significantly greater than low intensity but not statistically distinguishable from moderate ($p = 0.07$), showed no effect at the femoral neck, and meta-regression found no relationship between percentage of 1RM and the bone response (Kistler-Fischbacher, Weeks & Beck 2021). Consistent with a valued-but-not-uniquely-female modality, advanced or heavier-technique loading confers no special advantage: breakdown-set training produced no greater muscular adaptation than conventional sets (Fisher et al. 2016). Heavy loading is one effective and well-tolerated route, and it retains a clear edge only for maximal 1RM strength, which is itself sex-neutral. The 2023 ACSM consensus statement concurs that the relative magnitude of hypertrophy, strength and power gains from resistance training is similar between the sexes (Hunter et al. 2023).

> **Claim 17. Maximal or heavy loads are necessary to build muscle mass, because moderate loads cause fatigue and metabolic acidosis that impair hypertrophy.**

CONTRADICTED

Certainty of evidence: high.

Not uniquely female. Contradicted, and the stated mechanism runs backwards. For muscle size specifically, load is not the lever: when sets are taken close to failure, a wide spectrum of loads from roughly 30 to 85% of 1RM produces similar hypertrophy (Schoenfeld, Grgic, Ogborn & Krieger 2017), and this was already evident in an early meta-analysis finding loads at or below 60% of 1RM

produced substantial hypertrophy in untrained participants, with only a non-significant trend toward heavy-load superiority (effect-size difference 0.43, p = 0.076) (Schoenfeld et al. 2016). The metabolic-acidosis premise is the opposite of a problem: muscle fibre activation is unaffected by load when training is taken to task failure (Morton et al. 2019), and Type I and Type II fibres grow to the same extent under light and heavy loads (Morton et al. 2016), so lighter and moderate loads recruit and grow the full fibre pool.

The reason load is not the lever is mechanistic: hypertrophy is driven by mechanical tension sensed locally within the working muscle, not by the systemic hormonal surge that heavier or larger-muscle-mass sessions produce. In mice, mechanical loading activates mTOR signalling through a local diacylglycerol-kinase and phosphatidic acid pathway within the fibre (You et al. 2014), and the human trials that manipulated the hormonal environment directly confirm its independence from systemic hormones: training one arm under a high post-exercise testosterone, growth hormone and IGF-1 response and the other under a basal response produced identical hypertrophy and strength (West et al. 2009), and across a trial of 49 resistance-trained men no post-exercise rise in any anabolic hormone correlated with muscle or strength gains (Morton et al. 2016). This directly undercuts any model in which hypertrophy must be chased through hormonal levers rather than mechanical loading.

The wider belief that heavier is better rests on a misapplication of the size principle that was already discredited in 2008 (Carpinelli 2008). The metabolic acidosis Sims invokes is in fact associated with light, high-repetition loads rather than moderate ones, and the leading reviewers of this literature treat that discomfort as an adherence issue, not a barrier to growth, identifying moderate loads as the efficient sweet spot for hypertrophy and confirming there is no single hypertrophy zone: similar growth occurs across loads at or above roughly 30 percent of 1RM taken near failure, independent of age and training status (Schoenfeld et al. 2021; Weakley et al. 2023). The volume-matched evidence shows heavier loads have an edge only for maximal 1RM strength, not for hypertrophy (Carvalho et al. 2022), and a network meta-analysis of 28 studies and 747 adults reached the same split, finding hypertrophy load-independent while strength favoured moderate and high loads over low (Lopez et al. 2021).

This same pattern holds even when the load gap is pushed to its extreme: a meta-analysis of high-load versus low-load blood-flow-restriction training found similar hypertrophy despite much lighter loads, while maximal strength again favoured high load (Lixandrão et al. 2018). A within-participant trial settles the practical question directly: in 14 trained adults (11 men, 3 women), one leg trained with heavy low-rep sets (3 sets of 3 to 5RM) and the other with light high-rep sets (3 sets of 20 to 25RM) achieved similar muscle growth, with strength gains diverging by test: leg press similar, leg-extension 1RM favouring the heavy leg and local endurance the light leg (Cumming et al. 2025), consistent with the low-versus-heavy (30 versus 80% 1RM) hypertrophy equivalence first shown in young men in Phillips' laboratory (Mitchell CJ et al. 2012).

The female-specific and older-female evidence is decisive: across 126 studies and more than 4000 women, resistance training improved strength and body composition with effect sizes near-identical before and after menopause, so general guidelines apply (Isenmann et al. 2026). In

postmenopausal and elderly women, a meta-analysis of 26 mostly uncontrolled pre-post studies (745 women) found resistance training increases lean body mass (Thomas et al. 2021), and a meta-analysis of 101 studies and 5697 postmenopausal women found exercise increased muscle mass, cross-sectional area and fat-free mass, with resistance and combined training best for muscle gain (Khalafi et al. 2023).

A systematic review and meta-analysis in Menopause found benefits for functional capacity, hot-flash frequency and fat mass in this group, at low certainty (Sa et al. 2023). The point is reinforced by head-to-head intensity trials in exactly the women Sims has in mind: high-intensity and low-intensity resistance training produced comparable musculoskeletal gains in early postmenopausal women (Bemben & Bemben 2000), and high- and low-intensity training both increased type I fibre area with no change in thigh tissue composition in elderly women, with strength gains numerically larger and femoral density preserved only under the higher load (Taaffe et al. 1996); in mixed-sex untrained older adults, even strength gains were similar from 6RM to 15RM over 18 weeks (Harris et al. 2004).

A 2026 randomised crossover trial in 27 women in their late sixties adds a within-person test: eight weeks of whole-body training at 10RM and at 15RM, each set taken to failure, produced comparable gains in DXA-estimated skeletal muscle mass (about 0.4 to 0.6 kg), and when each woman was classified as a responder or non-responder her status was largely the same under both loads, so the heavier scheme did not rescue those who responded poorly to the lighter one; the trial is short and small, and residual gains persisted through the washout, but the direction is the same as every other load comparison in this population (Ribeiro et al. 2026). Equipment is not the magic variable either: free weights and machines yield similar hypertrophy in mixed-sex adult cohorts (Haugen et al. 2023). Critically, where load and volume have been separated in this population, it is training volume, not maximal load, that drives whole-body muscle hypertrophy: low-volume training produced small gains and higher-volume training moderate gains (Nunes et al. 2024). Heavy loading is one valid route to muscle, not a requirement, and moderate loads are not disqualified by fatigue or acidosis.

> **Claim 18. Women gain strength mainly through neural adaptations rather than hypertrophy, so they can get much stronger without much muscle gain.**

OVERSTATED

> *Certainty of evidence: moderate.*

Both sexes rely on neural adaptation early in training, and the most recent Bayesian meta-analysis (29 studies) found relative muscle growth essentially identical between sexes (exponentiated percentage change 0.69%) and Type II fibre hypertrophy similar, while an earlier meta-analysis found upper-body strength gains actually favouring women (Refalo et al. 2025; Roberts, Nuckols & Krieger 2020). The 2023 ACSM consensus statement adds that adult males and females generate similar, near-maximal neural drive during maximal voluntary contractions, so the maximal-strength difference between the sexes is explained by muscle mass rather than by any sex difference in the

nervous system's ability to activate muscle (Hunter et al. 2023). There is no good basis for the claim that women depend on neural adaptation more than hypertrophy as a sex-specific feature.

> **Claim 19. HIIT is more effective for women than steady-state work for fat loss and metabolic and cardiovascular health, and sprint interval training is a particularly potent stimulus that minimises cortisol exposure.**

OVERSTATED

> *Certainty of evidence: moderate.*

Not uniquely female. HIIT and SIT are effective for everyone but are not uniquely superior for women. The strongest case for the opposing view should be stated first, because it rests on real data. Two studies from the same Swedish group report genuine sex differences in the acute response to repeated 30-second sprints: serum insulin rose more in women than men, while blood lactate and ammonia accumulated more in men (Esbjörnsson et al. 2009), and basal plasma and muscle leucine were about 40 per cent higher in males, who also showed the greater fall in plasma leucine and rise in muscle ammonia, with a compensating glutamine rise in the women (Esbjörnsson et al. 2012). These are careful studies and the audit accepts their findings.

Three limits govern how far they reach. They are small, at eight women and ten men, and eight women and six men respectively. The growth hormone result is narrower than its usual restatement: growth hormone rose in both sexes to similar peak levels, and what differed was the timing, peaking after the first sprint in women and the third in men (Esbjörnsson et al. 2009), so this is a difference in time course rather than in magnitude. And they measure acute hormonal and metabolic markers rather than training outcomes, which matters because acute post-exercise hormone responses do not meaningfully drive hypertrophy in either sex (Van Every, D'Souza & Phillips 2024), as set out under Claims 8 and 26. The cortisol half of the claim is not supported and is contradicted by the same source.

In that study serum cortisol rose by approximately 10 to 15 per cent after sprint exercise independent of sex (Esbjörnsson et al. 2009). The broader framing offered in support, that regular endurance training raises basal cortisol while regular high-intensity interval training lowers it, is drawn from a review of the endocrine stress response to exercise which states it as general physiology rather than as a female-specific finding, and which treats the endurance-related rise as an adaptive response rather than a harm (Athanasiou et al. 2023). Presenting it as evidence about women specifically is the recurring move this audit flags. The largest human meta-regression found mitochondrial and VO2max gains were similar across low and moderate continuous (Zone 2), HIIT and SIT, and were not influenced by sex or menopause (Mølmen et al. 2025).

Direct training data point the same way: the 2023 ACSM consensus statement reports that males and females show similar relative adaptations to short-term (6 to 12 week) high-resistance and endurance training, and adapted similarly in relative terms to twelve weeks of high-intensity interval training on the bike, with comparable muscle hypertrophy of about 5 per cent and gains in fatigue resistance of about 4.8 per cent, while the 2026 NSCA statement notes that aerobic

adaptations differ in magnitude between the sexes and that with the same program women may show greater relative improvements in aerobic fitness and strength, with men starting and finishing higher in absolute terms (Hunter et al. 2023; Dabbs et al. 2026b).

The most direct test of whether HIIT favours women is a 2024 systematic review and meta-analysis of 28 trials (462 women, 503 men) that found identical effect sizes for maximal oxygen uptake in women and men (g = 0.57 in both), identical gains in threshold power (g = 0.38 in both), and no sex difference on any primary outcome; only well-trained women showed a larger peak-power-output effect than well-trained men, and baseline training status and intervention length, not sex, explained most of the variability (Lock et al. 2024). The strongest form of the opposing view, that female muscle is more fatigue-resistant at equivalent high-intensity dosages and that acute differences may translate into different chronic adaptations, is set out by Ansdell and colleagues (Ansdell et al. 2020); it is a serious argument, but the head-to-head interval-training adaptation data do not yet show a sex difference in the training outcome.

SIT produces faster early gains that plateau, which is often missed in the public message. Claims that sprint interval training is uniquely the best method for peri- and post-menopausal women rest on a small set of confounded trials, including studies with uncontrolled diet and sedentary do-nothing control groups, which cannot establish superiority over other training (Boutcher et al. 2019); much of what is marketed as supramaximal sprinting is in fact short micro-interval work by the accepted definition (Buchheit et al. 2013). The related belief that interval training is anabolic while continuous training is catabolic is also unsupported: a two-week within-participant unilateral-cycling trial in ten young men found myofibrillar protein synthesis did not differ between HIIT and work-matched continuous exercise (Trommelen et al. 2025), and endurance-training intensity has minimal influence on changes in fat-free mass (Steele et al. 2021).

> **Claim 20. Zone 2 (long, slow, low-intensity) training is built on male data and is less optimal for women, who benefit more from polarised training weighted toward high intensity.**

CONTRADICTED

> *Certainty of evidence: moderate (large mitochondrial dataset; the direction of VO2max trainability by sex is still contested).*

Not uniquely female. Rodent-based claim, contradicted by human data. Contradicted by the largest human dataset to date. Across 5,973 participants, percentage gains in mitochondrial content and capillarisation were similar for low-to-moderate-intensity continuous training, HIIT and SIT and were not influenced by age, sex or disease, while percentage gains in maximal oxygen uptake were greater in women than in men (p = 0.008), with absolute gains identical at about 3.8 mL/kg/min; higher training frequency produced larger gains, and sprint intervals were the most time-efficient stimulus per hour of training (Mølmen et al. 2025). Menopause status was not measured directly: the authors classified women under 35 as premenopausal and over 55 as postmenopausal by assumption, found no menopause effect on mitochondrial or capillary

adaptation, and noted a tendency to smaller percentage gains in maximal oxygen uptake in the older women, which limits inference about post-menopausal trainability.

The evidence on maximal-oxygen-uptake trainability by sex is not unanimous: an earlier meta-analysis reported greater gains in men (Diaz-Canestro & Montero 2019), a conclusion subsequently contested for baseline body-size and composition confounding, whereas this larger and more recent dataset finds no female disadvantage; either way, sex is not a sound basis for steering women away from the lower-intensity volume that builds mitochondrial content. A 2025 review likewise states that there is no evidence that endurance training in women should drastically differ from men's, even allowing for the known sex difference in maximal aerobic performance, and reports that the single trial of cycle-tailored endurance programming found it no better than a standard polarised plan (Mikkonen & Häkkinen 2025).

The sources behind the claim can be identified precisely, because Sims sets them out with citations in two articles published in October 2024, and the audit engages them directly rather than at second hand. The argument has three parts and each rests on a named source. The premise is that females have better mitochondrial respiration and mitochondrial density than men, and so do not need long Zone 2 work. The source given for it is a study of hepatic mitochondria in male and female C57BL/6J mice, wild type and two knockout lines, given voluntary running wheels for four weeks at six to ten animals per group (Von Schulze et al. 2018); a companion strand of the same argument rests on sex-hormone regulation of monocarboxylate transporter expression in rat liver (Cao et al. 2017). The inference therefore runs from mouse to human, from liver to skeletal muscle, and from a mechanistic observation to a training prescription.

In human skeletal muscle the direct comparison finds no sex difference in fibre-specific mitochondrial content or capillarisation (Bailleul et al. 2025). The second part is that low-intensity training produces minimal increases in MCT1 or MCT4 expression in women. The source given is a cross-sectional laboratory study of the MCT1 T1470A polymorphism and venous blood lactate during three circuit weight-training protocols at 70 per cent of the 15-repetition maximum, in 14 women and 15 men (Cupeiro et al. 2012). It contains no training intervention, so it cannot show what training of any intensity does; it measures blood lactate rather than transporter expression; the exercise studied is circuit weight training rather than low-intensity work; and its finding in women is that lactate did not differ across the AA, TA and TT genotypes, which is an absence of a genotype effect rather than an absence of a training response.

Its authors conclude that the polymorphism influences lactate transport in males. The third part is that sprint-interval training does increase MCT1 expression in women regardless of the recovery taken between intervals. This is cited accurately. Four weeks of work-matched sprint-interval training in 14 active women increased MCT1, NHE1 and carbonic anhydrase XIV protein abundance, and rest interval duration did not influence that response (McGinley & Bishop 2017). Every participant, however, performed sprint-interval training, and the trial has no low-intensity or other-intensity arm.

The consequence is that neither cited training source compares low intensity against sprint intervals in women. One involves no training and no transporter measurement; the other has a

single intensity. The intensity-by-sex comparison the prescription depends on is not present in either, and the premise beneath both is a study of mouse liver. Older mechanistic work on MCT1 and MCT4 and on an oestrogen-driven transporter mechanism, much of it animal work spanning the 1980s to the 2010s, is offered in support, but in humans no training study shows a sex-dependent lactate-transporter adaptation, and the mitochondrial adaptations the transporter argument is meant to explain do not differ by sex (Mølmen et al. 2025).

The competing premise sometimes used the other way, that women are endurance-adapted with more slow-twitch fibres and more mitochondria, is only half right in humans: women do carry a larger proportional type I fibre area, but fibre-specific mitochondrial content and capillarisation do not differ by sex (Bailleul et al. 2025), so sex is not a basis for steering women toward or away from any intensity band. Mechanistically, training volume is the main driver of mitochondrial content while higher intensity drives mitochondrial respiratory function through a distinct stress response, so the intensities produce divergent but complementary gains rather than one being superior (Botella et al. 2025, a mechanistic study in 28 young men whose authors call for it to be repeated in women and older adults). Because lower-intensity work is, for most people, the most practical way to accumulate the training frequency and volume that build mitochondrial content, reducing it is counterproductive.

> **Claim 21. Long or sustained moderate-intensity workouts spike cortisol, which is already elevated in menopause and signals the body to store fat, so menopausal women should keep workouts under about 30 minutes and avoid sustained moderate intensity.**

OVERSTATED

> *Certainty of evidence: moderate.*

Not uniquely female. The cortisol response to exercise is acute and self-limiting: it rises during and immediately after hard exercise and returns to baseline within roughly 24 hours, which is normal endocrine signalling rather than harm (Dote-Montero et al. 2021). In the Seattle Midlife Women's Health Study, overnight urinary cortisol rose with menopause-related hormone changes and with nocturnal catecholamines, and that study did not assess exercise, so it says nothing for or against a training effect (Woods et al. 2009). Higher aerobic fitness blunts the cortisol response to stress in older women, and exercise and weight-loss trials in postmenopausal women show no adverse cortisol change.

It should be recorded that Sims argues much the same in print. In an article published in October 2024 she describes cortisol as necessary rather than harmful, holds that short stressors such as exercise build stress resilience, states that cortisol should not prevent women from doing anything more strenuous than walking, and criticises cortisol-based restrictions on women's training as misinformation. She also notes that the intensity above which the cortisol response scales rises as fitness improves, so that the same absolute workload provokes a smaller response after some months of training. On this claim the audit and her own written position agree, and the restriction attributed to her sits awkwardly with both.

Set against this, cardiorespiratory fitness is among the strongest predictors of healthy ageing and lower mortality (Lang et al. 2024), and vigorous training raises fitness more than moderate work. In young adults a single vigorous bout blunted the cortisol response to a subsequent laboratory stressor in a dose-dependent way (Caplin et al. 2021), and sustained or increasing activity across adulthood lowers all-cause mortality by roughly 20 to 40 per cent (Yu R et al. 2025). Capping training to avoid a transient, normal hormonal response trades a large, well-established benefit for a speculative harm. The valid core, that some menopausal women carry elevated sympathetic and cortisol tone, is real; the training restriction drawn from it is not.

> **Claim 22. Women recover faster between high-intensity intervals than men, so their rest-between-intervals prescription should differ, with shorter recoveries.**

OVERSTATED

> *Certainty of evidence: the premise is moderate, the prescription very low. Sub-verdicts: greater female fatigue resistance and faster inter-effort recovery: plausible and reasonably supported; a distinct shorter-rest prescription by sex: Untested.*

This row is best handled in two parts, because the premise is on firmer ground than the prescription. That women are more fatigue-resistant and recover faster during repeated efforts is reasonably well supported: the 2023 ACSM consensus statement reports greater fatigue resistance in females during isometric and slow-to-moderate dynamic contractions normalised to maximal strength, and faster recovery from fatiguing dynamic and multiple-sprint exercise, attributing both partly to the greater proportional type I fibre area, and the 2026 NSCA position statement likewise recognises greater female fatigue resistance (Hunter 2014; Ansdell et al. 2020; Hunter et al. 2023; Dabbs et al. 2026b).

The qualifiers matter and belong here too: the same consensus notes no sex difference during fast dynamic contractions and no difference in repetitions to failure hours after resistance exercise, and it flags that the recovery advantage may last less than a few hours and remains an area for further study (Hunter et al. 2023). What is untested is the prescription, not the premise: recovery between intervals is governed mainly by work intensity, interval duration and the training goal, and no trial has shown that prescribing shorter rest by sex improves any outcome.

The one trial that has manipulated rest interval duration in women points away from the prescription rather than toward it. In 14 active women randomised to one minute or five minutes of rest between sprints across four weeks of work-matched sprint-interval training, rest interval duration did not influence adaptations in acid/base transport protein content, and where a difference did emerge it favoured the longer rest, with greater improvement in total work during a repeated-sprint test for the five-minute group (effect size 0.51, 90 per cent confidence limits plus or minus 0.37) and similarly modest gains in peak aerobic capacity in both (McGinley & Bishop 2017). The trial is small and was not designed as a comparison between the sexes, so it does not settle the question; it does mean the shorter-rest prescription is not merely untested but is unsupported by

the only direct test in women, and it is a trial Sims cites herself. This suggests a reasonably supported premise attached to an untested prescription.

5. Weight loss

> **Claim 23. Calorie restriction alone does not work for active women, because chronic restriction triggers a cortisol-driven stress response that promotes abdominal fat retention and muscle loss.**

OVERSTATED

> *Certainty of evidence: moderate.*

Energy balance remains the primary determinant of body-mass change in both sexes. Chronic severe restriction does provoke adaptive responses, but the claim that restriction causes net fat retention through cortisol in women is not supported. The full picture includes human data partly supporting the cortisol argument: a 2x2 randomised experiment in 115 women (99 completers by the paper's Methods, though its abstract reports 121 randomised) found restricting calories over three weeks raised total cortisol output (Tomiyama et al. 2010), and in 51 overweight women, 12 weeks of energy restriction enhanced the dinner-meal cortisol response and flattened the diurnal rhythm, an effect prevented by adequate dairy intake (Witbracht et al. 2012).

But longer, better-controlled trials do not support the downstream harm: 24 weeks of 25% restriction, by diet or diet plus exercise, did not alter salivary cortisol in 35 overweight men and women (Tam et al. 2013), and two years of prescribed 25% restriction in 218 non-obese men and premenopausal women aged 21 to 50, about 70 per cent of them women (Ravussin et al. 2015), produced only a small, transient rise at one year that had resolved by two years, with no fall in IGF-1 (Fontana et al. 2015). Neither of these cohorts was athletic, which is a limit on how far they reach, but they are the longest and best-controlled human restriction data available and they do not show the sustained stress response the claim requires.

A moderate energy deficit does work, and what governs the composition of the weight lost is training and protein, not sex. The trial most often cited for this was in men: 40 overweight young men held to a 40% energy deficit for four weeks while training six days a week gained lean mass and lost fat, the higher-protein group (2.4 g/kg/day) gaining about 1.2 kg of lean mass and losing 4.8 kg of fat against 0.1 kg and 3.5 kg on 1.2 g/kg/day (Longland et al. 2016).

The same laboratory had already shown the same thing in women. In 90 overweight and obese premenopausal women, 16 weeks of a 500 kcal/day dietary deficit with daily aerobic exercise and twice-weekly supervised resistance training produced about 4.3 kg of weight loss in every group; the women eating 30% of energy as protein (about 1.3 g/kg/day) gained 0.7 kg of lean mass while losing more total, trunk and visceral fat than those on 15%, who held or lost lean mass, and visceral adipose tissue fell in all three groups (Josse et al. 2011). That is the opposite of restriction driving

abdominal fat retention through cortisol: under a sustained deficit, active women lost visceral fat and, given enough protein and resistance training, built muscle.

The pattern holds after menopause. During five days of energy restriction of roughly 800 kcal/day, a single 35 g dose of whey after resistance exercise maximally stimulated myofibrillar protein synthesis in 40 overweight postmenopausal women, so the anabolic response to feeding and loading is intact in a deficit at the age Sims is most concerned about (Larsen et al. 2023); and in 54 overweight postmenopausal women, 12 weeks of energy restriction cost only about 1 kg of fat-free mass whether protein was set at 0.8 or 1.5 g/kg/day, with performance on the Short Physical Performance Battery improving even as handgrip strength fell slightly (Englert et al. 2021), which fits the point made under Claim 8 that resistance training, rather than protein alone, is the main lever for keeping lean mass in a deficit.

In overweight premenopausal women, intermittent and continuous restriction produce similar weight and fat loss (Harvie et al. 2010; Harvie et al. 2013), though a meta-analysis shows deficits beyond about 500 kcal/day begin to impair lean-mass gains while sparing strength (Murphy et al. 2021). In healthy men, five days of severe energy deficit with aerobic exercise shifted the muscle proteome toward a more oxidative phenotype, although fat-free mass fell by about 2 kg; comparable data in women are lacking (Nishimura et al. 2025). The valid core is that under-fuelling relative to training load is harmful (the low energy availability pathway), which is an argument against excessive restriction in athletes, not against energy balance itself. The 2026 NSCA position statement concurs that resistance training in a caloric deficit has a lean-mass sparing effect, and that combined with impact loading it helps maintain bone density (Dabbs et al. 2026a).

> **Claim 24. Timing of eating matters more than total calories for body composition: a pre-workout snack, a recovery meal within 30 to 45 minutes, and avoiding large gaps around training.**

CONTRADICTED

> *Certainty of evidence: high.*

Contradicted. Total energy intake, and for muscle outcomes total daily protein, is the dominant lever, while nutrient timing and the narrow post-exercise window have minor effects (Schoenfeld, Aragon & Krieger 2013), and the anabolic window is likely several hours wide (Aragon & Schoenfeld 2013). Timing is a supporting actor, not the primary driver, and the broader meal-timing literature bears this out. Requiring habitual breakfast-skippers (49 women) to eat breakfast added about 266 kcal/day and led to weight gain with no compensation (LeCheminant et al. 2017), yet a meta-analysis of randomised trials found breakfast skipping led to modestly greater short-term weight loss and no adverse body-composition effect, though skippers showed higher LDL (Bonnet et al. 2020). In a small trial of 40 overweight women under the same deficit, meal frequency did not affect weight loss, although the higher-frequency group lost somewhat more body fat (Ötüken Köroğlu & Öztürk 2024).

Where timing does matter its effects are real but secondary: in a tightly controlled crossover of 16 adults with overweight, late isocaloric eating doubled the odds of hunger and lowered waking energy expenditure (Vujović et al. 2022), and front-loading calories to breakfast produced markedly greater weight loss in one 12-week trial of overweight women with metabolic syndrome, a single-trial result not consistently replicated (Jakubowicz et al. 2013). Taken together, these findings reinforce that such secondary factors modulate but do not override the primacy of total energy and, for muscle, total protein intake.

> **Claim 25. Many women who struggle to lose fat are under-eating, not over-eating; eating more, but strategically, restores metabolic rate and supports recomposition.**

OVERSTATED

> *Certainty of evidence: moderate.*

Metabolic adaptation to restriction is real but modest and does not reverse a true energy deficit, so as general weight-loss advice "eat more to lose fat" is not supported. Metabolic-adaptation research supports this modest-but-real framing: adaptive thermogenesis during weight loss is limited, tied early on to reduced insulin secretion and fluid shifts in the re-analysis of the Minnesota Starvation Experiment in 32 men, and to low leptin during weight maintenance, rather than a mechanism that reverses an energy deficit (Müller et al. 2015; Müller et al. 2016). It holds only in the specific case of an athlete recovering from genuine low energy availability, where restoring intake is appropriate.

6. Perimenopause and menopause

> **Claim 26. Menopause removes the oestrogen-driven, satellite-cell-mediated muscle-building stimulus, so it becomes much harder for women to build and keep muscle after menopause.**

OVERSTATED

> *Certainty of evidence: moderate.*

Not uniquely female. The direction is right, but a flat contradiction overshoots what the consensus literature will bear. Mechanical tension from training, not circulating hormones, is the primary driver of muscle protein synthesis and hypertrophy, and the anabolic signalling response to resistance exercise is similar between sexes (Van Every et al. 2024), with a resistance-exercise-specific growth pathway now identified in human muscle of both sexes (Zhu et al. 2025). The strong mechanistic form of the oestrogen and satellite-cell claim rests largely on rodent studies (for example oestrogen-receptor-alpha signalling in mice, with preliminary human biopsy data; Collins et al. 2019).

But the defensible position is not that oestrogen is irrelevant: the 2023 ACSM consensus statement holds that oestrogen is thought to have a role in the maintenance and repair of skeletal muscle in females, and possibly in the muscle-growth response to resistance training in both young and post-menopausal women, even though it has no direct effect on muscle protein synthesis at rest, and it notes that satellite-cell content is similar between the sexes for type I fibres (Hunter et al. 2023). The claim therefore has a real, if hedged, mechanistic basis; what it lacks is a demonstrated effect on the training outcome.

The strongest direct test is human rather than rodent: a randomised trial in early post-menopausal women found that transdermal oestrogen therapy prevented the training-associated decline in satellite-cell number seen under placebo, a genuine effect on the satellite-cell compartment, yet the molecular markers of hypertrophy changed similarly with and without oestrogen (Dam et al. 2023), so the mechanism is real at the cellular level while the training outcome is not oestrogen-dependent, which is exactly the distinction this row turns on. And the functional evidence runs against the pessimistic conclusion: a 2025 study found lower-limb strength, muscle thickness, lean mass and balance improved over 12 weeks of resistance training with no difference between pre-, peri- and post-menopausal women not taking hormone therapy, evidence that the menopause transition does not blunt the response to a given training stimulus (Svensen et al. 2025).

Not every trial agrees: in a 20-week study, strength rose in both groups but fat-free mass and muscle thickness increased only in the pre-menopausal women, whatever the load, which the authors read as post-menopausal women needing more volume rather than a different kind of training (Isenmann et al. 2023). Across the literature, relative gains from resistance training are similar between sexes, including in adults over 50 and over 60 (Roberts, Nuckols & Krieger 2020; Jones et al. 2021; Hawley et al. 2023), with no evidence that women lose muscle faster than men with age (Mitchell WK et al. 2012). Post-menopausal women build muscle in response to ordinary training; the strong hormonal-pessimism framing is overstated rather than flatly contradicted.

> **Claim 27. Perimenopause requires a proactive recalibration; the conventional advice to eat less and move more is particularly counterproductive, and resistance training and HIIT become more important.**

OVERSTATED

> *Certainty of evidence: moderate. Sub-verdicts: the greater importance of resistance training and adequate protein through the transition: Supported; the categorical claim that energy-balance principles invert and that eat-less-move-more is counterproductive: Overstated.*

Resistance training and adequate protein do matter through this transition (supported), and recent work reinforces these valid components: adequate protein protects against muscle-mass loss during weight loss (McCarthy et al. 2021), lifestyle-medicine reviews support resistance training and diet through the transition (Anekwe et al. 2025), and leucine supplementation adds no fat-free-mass or strength benefit beyond resistance training itself in midlife women, with only minor adipokine differences in a follow-up analysis (Funderburk et al. 2019; Chapman-Lopez et al. 2023). But the categorical claim that energy-balance principles invert in perimenopause is not supported.

Training importance increasing is reasonable; "eat less, move more is counterproductive" is an overstatement.

> **Claim 28. Post-menopause, muscle and bone are the priority: higher protein, heavy resistance training, impact exercise, and HRT/MHT considered with a physician.**

SUPPORTED

> *Certainty of evidence: high.*

Supported, and one of Sims' strongest positions. Resistance training, impact and loading exercise, adequate protein, and informed HRT discussion are mainstream, evidence-based strategies for musculoskeletal health after menopause (Anekwe et al. 2025; Dabbs et al. 2026a). Over one year, resistance training changed spine bone mineral density by +0.43 per cent, against -0.66 per cent for hormone replacement therapy without exercise, +0.70 per cent for the two combined and -3.6 per cent for controls, so training matched or bettered HRT for preserving spine density, with no added benefit from combining them; because HRT was self-selected and only the training allocation randomised, this is a quasi-experimental comparison (Maddalozzo et al. 2007).

Loading and impact exercise do improve bone in this group, and high-intensity work is a valid and well-tolerated route: the LIFTMOR trial showed twice-weekly high-intensity resistance and impact training (5 sets of 5 at over 85% of 1RM plus jumping) improved lumbar-spine density and preserved femoral-neck density relative to a low-intensity control, and improved physical function, safely in postmenopausal women with low bone mass (Watson et al. 2018). Similarly, maximal-strength training improved bone mineral content at the lumbar spine and femoral neck in post-menopausal women with osteopenia or osteoporosis, alongside large strength gains, although areal bone mineral density itself was unchanged, which the authors attribute to a DXA bone-area measurement artefact, in a small trial with eight per group at follow-up (Mosti et al. 2013).

What the human evidence shows is a site-specific picture rather than a heavy-load requirement. The most comprehensive meta-analysis to date (53 trials, 63 interventions) found moderate-intensity loading (65 to 80% 1RM) significantly improved bone density at the lumbar spine, femoral neck and total hip; high-intensity loading, tested in only four interventions, produced the largest spine point estimate (0.031 versus 0.012 g/cm2 for moderate), which was significantly greater than low intensity ($p = 0.002$) but not statistically distinguishable from moderate ($p = 0.07$), showed no effect at the femoral neck, and was too sparsely studied to assess at the total hip, and meta-regression found no relationship between resistance-training intensity as a percentage of 1RM and the change in spine or femoral-neck density (Kistler-Fischbacher, Weeks & Beck 2021).

Heavy loading may therefore carry an edge at the spine, on four trials, but it is not required for benefit at either site. A 2025 network meta-analysis of 49 trials in 3,360 postmenopausal women ranked combined aerobic and resistance training first for both lumbar-spine and femoral-neck density, with resistance training alone also effective at both sites, and did not identify load intensity as the deciding variable (Li et al. 2025). For muscle mass a wide range of loads taken close to failure

produces similar hypertrophy (Schoenfeld, Grgic, Ogborn & Krieger 2017), so heavy loading is one effective route rather than a strict requirement, and heavier loads retain a clear edge only for maximal strength, which is itself sex-neutral.

Observational data across the whole adult lifespan point the same way on the priority itself: in 251 adults aged 18 to 85, maximal leg-press strength and lower-body lean mass were positively associated with hip bone density independent of age, sex and visceral adiposity, with strength retaining explanatory value beyond lean mass alone; hip density declined with age in both sexes while spine density declined only in women, a divergence the authors attribute mainly to degenerative changes inflating spine DXA readings in older men rather than to a female-specific vulnerability (Kraemer et al. 2026).

The design is cross-sectional, and neither menopausal status nor training history was recorded, so it cannot show that building strength builds bone, but it supports treating lower-body strength as a priority marker of skeletal health at every age; the authors' suggestion that heavier loading and rate coding may carry extra skeletal benefit is offered as a hypothesis for future trials, not a result, and does not alter the site-specific conclusion above. Resistance training improves strength in middle-aged women whatever their menopausal status, although in one 20-week trial body-composition gains were seen only before menopause, suggesting post-menopausal women may need more volume (Isenmann et al. 2023). Its benefits extend beyond bone and muscle too: a meta-analysis of randomised trials found resistance training improves the lipid profile in post-menopausal women (He et al. 2023). This is endorsed by the 2026 NSCA position statement, which recommends that resistance training at menopause focus on maintaining or improving muscle mass and bone mineral density (Dabbs et al. 2026a).

> **Claim 29. The gut microbiome shifts significantly at menopause as oestrogen declines, affecting metabolism, immunity and brain health; diversity, fibre and probiotic foods help.**

OVERSTATED

> *Certainty of evidence: low (emerging mechanism; claimed outcomes not established by intervention trials).*

Overstated. The oestrogen-microbiome interaction (the estrobolome) is a genuine but still emerging research area, and it is now somewhat better characterised: reviews link diet, the gut microbiome and oestrogen physiology in menopause (Lim et al. 2026; Marano et al. 2026), and a probiotic with beta-glucuronidase activity has been shown to modestly modulate serum oestrogen in peri- and post-menopausal women (Honda et al. 2024). General dietary diversity and fibre advice is sound, and whole-grain intake has shown metabolic benefits in overweight adults (Schutte et al. 2018), but the specific claimed outcomes are not yet established by intervention trials. The core biology is genuine; the strength of the claim outruns the evidence.

> **Claim 30. The female heart adapts differently to training, and women's cardiovascular disease risk is under-recognised because it presents differently, so prescriptions should account for this.**

OVERSTATED

Certainty of evidence: moderate.

Overstated. The claim bundles a real point with an unsupported one. Women's cardiovascular disease is under-recognised and can present differently, and baseline cardiac structure does differ between the sexes, so the valid core is real. What does not follow is that the heart adapts differently to training, or that this warrants a sex-specific exercise prescription. A 2025 systematic review and meta-analysis of cardiac adaptations to aerobic training in healthy adults found the response largely sex-neutral, with no sex difference in left ventricular mass, end-systolic volume or ejection fraction, and the single exception that end-diastolic volume increased somewhat more in men (Cotie et al. 2025). This mirrors findings in clinical populations: a pooled analysis of prospective randomised trials in coronary-heart-disease patients found women and men had a similar cardiopulmonary and echocardiographic response to aerobic training, with a similar proportion of responders (women n = 19, men n = 64; Trachsel et al. 2020).

What the evidence does show is a difference in the magnitude and time-course of cardiac adaptation rather than in kind: the 2023 ACSM consensus statement notes that short-term relative gains in cardiovascular function are similar between the sexes, but that longer-term cardiac adaptations may be larger in males: in young adults, gains in stroke volume, arteriovenous oxygen difference and maximal oxygen uptake were similar between the sexes at three months of endurance training, after which males kept improving maximal oxygen uptake for up to nine months and left-ventricular mass for up to six months while females plateaued at three months, with both sexes reaching athlete-level ventricular mass after a year; males also have an enhanced ability to use the Frank-Starling mechanism, a baseline difference rather than a training adaptation, while the regulation of heart rate and the cardiac-output response for a given metabolic demand are essentially identical between the sexes (Hunter et al. 2023); the 2026 NSCA position statement similarly describes aerobic adaptations as differing in magnitude between male and female athletes (Dabbs et al. 2026b).

The most direct test of that distinction comes from the same laboratory whose earlier trial produced the blunted-female-response finding the consensus relies on (Howden et al. 2015). Twenty-eight previously sedentary middle-aged adults, 15 of them women, mean age 54 and spanning pre- and post-menopause, completed two years of endurance training, and by cumulative training impulse the women trained at least as hard as the men. The absolute gain in peak oxygen uptake was about half as large in the women (0.27 versus 0.53 L/min), and the difference survived scaling to body mass, but it disappeared once scaled to fat-free mass (sex by time p = 0.113); in percentage terms the gap was modest, 20.1 against 16.6 per cent.

The mechanism was a smaller rise in peak stroke volume and cardiac output, not a failure of the heart to remodel: resting left-ventricular end-diastolic volume index rose independently of sex (p =

0.888), and in each sex it was the participants with the smallest ventricle before training who gained the most (r = -0.65 in men, r = -0.78 in women). The authors' stated conclusion is that the improvement in cardiorespiratory fitness with endurance training is influenced by sex, but that the degree of cardiac remodelling in previously sedentary adults is not different between the sexes, nor impaired by a smaller pre-training ventricle; and because their cohort spanned the menopause transition while almost every woman increased her stroke volume index, they judge that menopause alone does not explain the blunted central adaptation earlier work reported in older women (Hedge et al. 2026).

Two caveats belong here. The trial is small, and the women's ventricles remained stiffer than the men's after two years, their post-training compliance curve sitting where the men's had been before training, which is a real sex difference in the ageing heart rather than in its response to training. The separation the claim turns on nonetheless holds: the size of the fitness gain differs, the capacity to remodel does not. The defensible reading is therefore narrower than a flat sex-neutral claim: differences in the magnitude of cardiac adaptation are real, but they do not establish that the heart adapts differently in kind, nor do they license a distinct, sex-specific exercise prescription.

7. Hydration

> **Claim 31. Women have less body water; pre-exercise sodium loading improves fluid balance and endurance in the heat, especially in the luteal phase; post-menopausal women clear water more slowly (hyponatraemia risk); standard recommendations underestimate women's luteal heat stress (Sims et al. 2007a)**

OVERSTATED

> *Certainty of evidence: low (limited, largely single-group data). Sub-verdicts: lower total body water in women: Supported; the luteal sodium-loading, post-menopausal water-clearance and luteal-heat-stress prescriptions: Overstated.*

Lower total body water in women is true. The luteal-phase sodium and heat-stress claims and the post-menopause water-excretion claims rest on limited data, some of it from Sims' own group, and have not been confirmed by independent trials. The general point (individualise hydration) is fine; the strong sex- and phase-specific prescriptions are not well-supported. The benefit of pre-exercise sodium loading for fluid balance and endurance in the heat is not obviously female-specific: the 2007 studies from this group used comparable protocols in men and in women and both reported benefits, with greater plasma-volume expansion and reduced physiological strain in the men (Sims et al. 2007a; Sims et al. 2007b), and a further study found pre-exercise sodium increased voluntary fluid intake, reduced dehydration and improved time-trial performance in the heat (Morris et al. 2015).

On the specific luteal heat-stress claim, a meta-analysis co-authored by Sims found the luteal rise in resting core temperature to be about 0.2°C, shrinking to roughly 0.05°C after exercise and becoming negligible in hot conditions (Giersch et al. 2020); and while women may need more heat-acclimation sessions than men to reach comparable adaptation, that is a difference in time-course rather than a categorically different hydration requirement (Wickham et al. 2021). Individual responses vary with sweat rate and acclimatisation, so hydration is best individualised rather than prescribed by sex or cycle phase.

8. The research-gap meta-claim

> **Claim 32. Women are historically under-represented in research, studies often fail to report cycle, contraceptive or menopausal status, and women have been "running an experiment on themselves" by following male-derived guidelines.**

OVERSTATED

> *Certainty of evidence: moderate.*

The descriptive part is true: women are under-represented in samples and reproductive status is often unreported (Cowley et al. 2021; Costello et al. 2014), and calling for better reporting is reasonable: an audit found the participant pool behind governing-body strength-training guidelines is about 70% male for adult guidelines and 54% for older-adult guidelines (Pandit et al. 2023), a figure the Mikkonen and Häkkinen review also uses to call for more female-specific training research (Mikkonen & Häkkinen 2025). The conclusion is where it breaks. That women have been "running an experiment on themselves" by following male-derived guidelines is a non sequitur: under-representation in samples does not establish that the resulting guidance is wrong for women, and it can only be shown wrong by evidence that women following it were harmed or under-served, which the male-female comparison trials do not show.

The strongest version of the counter-argument is not the one usually made, and it is worth conceding: under-representation widens the confidence intervals around estimates for women, and acting on wide intervals is a judgement call rather than a demonstrated error. Granting that still leaves the conclusion unsupported, and it has to be met on its best ground. The 2023 ACSM consensus statement is the most authoritative recent argument that biological sex is a primary determinant of athletic performance, and it makes that case plainly while also calling, as Sims does, for sex to be included as a biological variable in exercise research and for studies powered to characterise the female response (Hunter et al. 2023).

The audit's position is compatible with both halves of that statement: performance differences are real and women remain under-researched, yet the same consensus reports that relative adaptations to short-term resistance and endurance training are similar between the sexes, with the main documented exception being larger long-term cardiac remodelling in males (Hunter et al.

2023), which is the specific point on which the charge that women have been "running an experiment on themselves" fails. Under-representation is a reason to do, and to fund, more and better research in women; it is not evidence that the training principles derived so far are wrong for them. Where the question has been studied directly, the evidence points the other way.

Resistance training is the clearest test case, because it is the modality studied most extensively in women. It may surprise many that women were included, both for comparison with men and in studies of their own, as far back as 1974 (Wilmore 1974; Brown & Wilmore 1974; Mayhew & Gross 1974). The conclusion has been stable for over three decades: the first NSCA position stand on women and resistance training (1989) held that women should train around the same sound principles as men, and a review of the female response thirty years later found that basic conclusion still held, that males and females should train for strength in the same basic way with similar methodologies, programs and exercises.

William Kraemer, who organised the NSCA task force behind that 1989 position stand and has studied resistance training in women for four decades, concludes in a 2025 review that recommendations for men and women are highly similar and goal-dependent, that training principles for the two sexes remain virtually the same with a few caveats around hypertrophy, substrate use, fatigability and recovery, and that a recent meta-analysis found no effect of sex on strength or hypertrophy gains (Kraemer et al. 2025). Far from having followed harmful male-derived advice, women who trained on these shared principles obtained the same benefits, and for all-cause mortality the benefit appears larger in women than in men, a 19 per cent reduction against 11 per cent in the largest population cohort, alongside consistent reductions in depressive symptoms (Kraemer et al. 2025). The under-representation and under-reporting are real and worth correcting; the leap from there to women having been experimented on, or to a need for categorically sex-specific programming, is not supported and is in fact contradicted by the researcher who has looked hardest at the question.

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