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Clinical review: Menopause hormone therapy in weight management

Courtney Younglove

Heartland Weight Loss, 14205 Metcalf Avenue, Overland Park, KS, 66223, USA

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ABSTRACT

Background: This document provides clinicians with an evidence-based overview of menopause hormone therapy (MHT) and its role in weight management for perimenopausal and postmenopausal women with obesity or overweight.

Methods: This clinical review was developed using a structured, evidence-informed process designed to synthesize current knowledge about menopause hormone therapy (MHT) and its relationship to weight regulation in perimenopausal and postmenopausal women with obesity or overweight. The methodology incorporated a targeted literature review and expert clinical interpretation of the author.

Results: The menopausal transition is associated with significant changes in body composition - including increased total body fat, visceral adipose tissue accumulation, and decreased lean body mass that occurs independent of chronological age. While these changes are primarily driven by estrogen deficiency and evidence from randomized trials and meta-analyses demonstrates that MHT may attenuate central fat accumulation and preserve favorable body composition, it is not indicated as a primary weight loss intervention. Furthermore, although recent observational data suggest potential synergistic effects when MHT is combined with obesity medications (OMs), these findings are limited by small sample sizes, retrospective design, and potential confounding, requiring validation through rigorous clinical trials.

Conclusion: Following individualized risk-benefit assessment, MHT should be prescribed based on established clinical indications: moderate to severe vasomotor symptoms, prevention of osteoporosis, treatment of hypoestrogenism, or treatment of vulvovaginal symptoms. While MHT may provide indirect benefits for weight management through improvements in cardiometabolic health and improvement of menopausal symptoms that interfere with lifestyle interventions, it should not be marketed or prescribed for weight loss or obesity treatment. Comprehensive obesity treatment should focus on the four pillars: nutrition, physical activity, behavioral modification, medical interventions, including metabolic and bariatric surgery and appropriate use of FDA-approved obesity medications when indicated.

1. Introduction

This clinical review addresses the intersection of menopause hormone therapy and weight management, a topic of increasing clinical relevance given the aging population and expanding treatment options for obesity.

Each year, approximately 1.3 million women in the United States enter the menopausal transition - a physiological shift with well-documented consequences affecting body composition, cardiometabolic health, and quality of life. Evidence-based society guidelines, including the comprehensive hormone therapy position statement of The Menopause Society [1], provide clinicians with a rigorous framework for evaluating menopause hormone therapy (MHT) in appropriately selected women. Yet despite this robust scientific foundation, MHT has become one of the most commercially distorted topics in women's health. Direct-to-consumer platforms, medical spas, and social media influencers have promoted hormone therapies with claims that extend beyond the current evidence base - most prominently, the assertion that MHT causes meaningful weight loss. The result is a clinical environment in which both patients and clinicians struggle to separate legitimate therapeutic indications from marketing-driven mythology. This clinical review addresses that gap; synthesizing the current evidence on MHT and weight management to provide clinicians with a clear, evidence-grounded framework for counseling perimenopausal and postmenopausal women with obesity or overweight.

As illustrated in Fig. 1, body composition changes attributable to the menopause transition increase risks of developing obesity. Treatment of obesity and treatment of hypoestrogenemia are different but may work

E-mail address: courtney@heartlandweightloss.com.

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synergistically.

The purpose of this statement is to provide evidence-based guidance on the appropriate role of MHT in the comprehensive management of perimenopausal and postmenopausal women with obesity or overweight.

2. Body composition changes during the menopausal transition

Weight gain in midlife women is often attributed to aging alone - and longitudinal data confirm this concern is valid. Among American women followed from young adulthood through midlife, 92% were on upward weight trajectories, generally moving into a higher BMI category by middle age [2]. A 2022 cross-sectional NHANES analysis found that women gained an average of 12 lbs. over the preceding 10-year period – approximately twice the 6 lbs. gained by men in the same timeframe [3]. However, total body weight itself does not appear to accelerate at the menopausal transition.

Analysis of DXA-measured body composition data from the Study of Women's Health Across the Nation (SWAN) - a longitudinal observational study of 3302 midlife women across 7 sites followed from 1996 — demonstrated that weight climbed linearly during premenopause without acceleration at the menopausal transition, and its trajectory flattened after the final menstrual period [4].

What changes profoundly, however, is body composition. At the onset of the menopausal transition, the rate of fat gain doubles while lean mass simultaneously declines - changes that persist until approximately 2 years after the final menstrual period, after which both trajectories decelerate to near zero slope [4]. Concurrent with these changes in fat and lean mass, estrogen deficiency drives a redistribution of adipose tissue from a gynoid pattern (subcutaneous, gluteofemoral) to an android pattern (central, abdominal) - a shift that occurs independently of chronological aging and has been extensively documented across multiple large-scale studies [4–9]. It is this redistribution, rather than weight gain per se, that carries the greatest implications for cardiometabolic risk in postmenopausal women.

In addition, skeletal muscle mass exhibits a steady decline after the fourth decade of life, and the rate accelerates with age. This decline is more pronounced in women than men and further exacerbates adverse changes in body composition. Most women begin adulthood with less muscle mass than men and, in general, women do less to maintain their muscle mass as they age. This loss of muscle mass is often concealed by an unaltered or even increasing body mass index, resulting in sarcopenic obesity. Additionally, the menopausal transition is compounded by the more sedentary lifestyle that many women adopt as they age, with decreased time performing daily chores and increased time in sedentary behaviors [4,7,8].

2.1. Pathophysiology driving body composition changes

As noted in Table 1, The reason for these shifts reflects multiple converging mechanisms and is incompletely understood.

2.1.1. Relative hyperandrogenemia

Testosterone is the most abundant sex hormone in the female body after menarche. Following peak levels around age thirty, ovarian production of testosterone tends to decline slowly and gradually over the course of a woman's lifetime. During the menopause transition, as levels of estrogen drop, the ratio of testosterone to estrogen increases, resulting

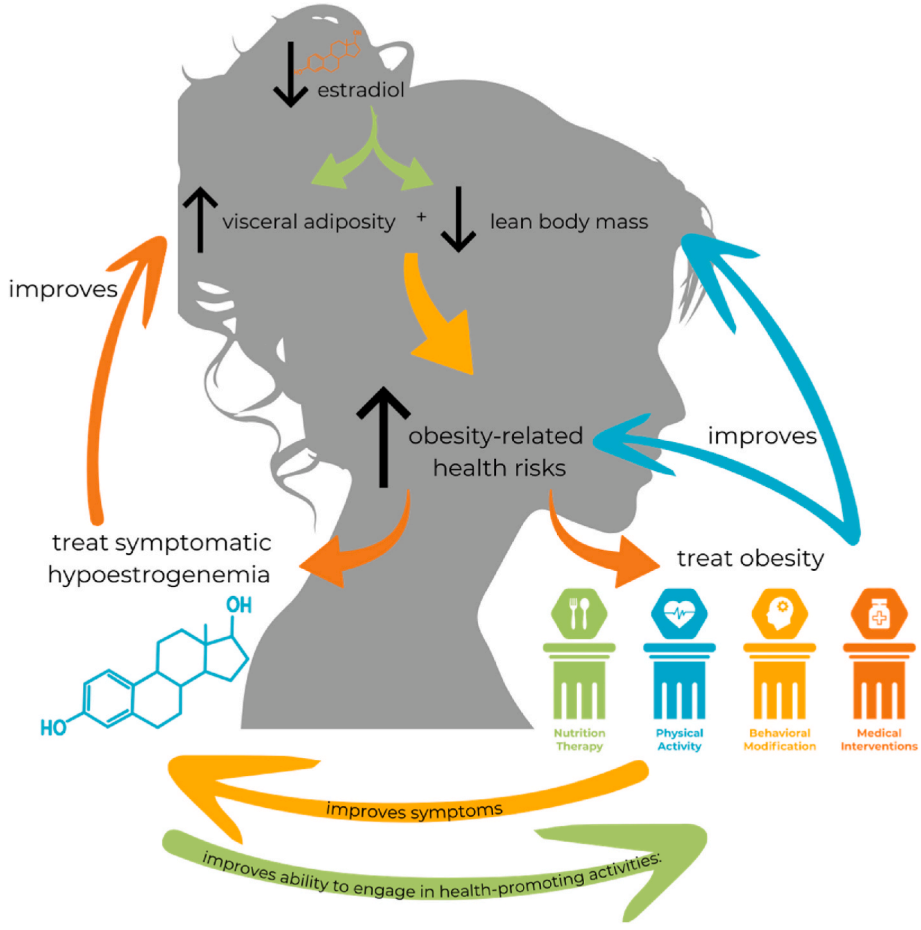

Fig. 1. Body composition changes attributable to the menopause transition increase risks of developing obesity. Treatment of obesity and treatment of hypoestrogenemia are different but may work synergistically. Original figure drafted by the author.

Table 1
Possible mechanisms driving body composition changes during the menopausal transition.

Mechanism	Effect	Clinical Significance
Relative Hyperandrogenemia	↑ testosterone-to-estrogen ratio	Promotes central fat distribution
Estrogen & Appetite Regulation	↓ POMC neuron activity ↑ NPY activity ↓ Leptin sensitivity ↑ Ghrelin activity	Increased hunger signals Altered energy balance
Adipocyte Regulation	Loss of ERα signaling	Shift from subcutaneous to visceral fat accumulation
Bone Marrow-Derived Adipocytes	↑ BMDA production in VAT	Enhanced central adiposity
Follicle-Stimulating Hormone	Elevated FSH levels	Direct effects on fat mass and energy expenditure (independent of estradiol)
Metabolic Alterations	↓ Beta-oxidation gene expression ↑ Visceral fat lipolysis	Impaired fatty acid utilization ↑ Free fatty acids → insulin resistance
Lean Body Mass Loss	Progressive sarcopenia	↓ Basal metabolic rate Risk of sarcopenic obesity

POMC = pro-opiomelanocortin; NPY = neuropeptide Y; ERα = estrogen receptor alpha; BMDA = bone marrow-derived adipocytes; VAT = visceral adipose tissue, FSH = follicle-stimulating hormone.

in a state of relative hyperandrogenemia [10].

2.1.2. Estrogen's role in appetite regulation

Estrogen plays a critical role in appetite suppression through multiple pathways. Estrogen stimulates pro-opiomelanocortin neurons, inhibits neuropeptide Y activity, reduces ghrelin activity, and potentiates leptin signaling by increasing expression and sensitivity of leptin receptors. Loss of these regulatory effects during the menopausal transition contributes to increased hunger signals and altered energy balance [4,11].

2.1.3. Adipocyte regulation

Estrogen promotes subcutaneous fat accumulation through estrogen receptor alpha (ERα) signaling. Loss of estrogen signaling results in preferential accumulation of central body fat deposits [11].

2.1.4. Bone marrow-derived adipocytes

Estrogen deficiency shifts the differentiation of bone marrow mesenchymal stem cells away from the osteogenic lineage and toward adipogenesis, resulting in expansion of bone marrow adipose tissue (BMAT) [12]. This shift is clinically relevant beyond its skeletal effects: BMAT is an endocrine-active depot that secretes adipokines including adiponectin and leptin and contributes to systemic metabolic signaling. Expansion of BMAT following estrogen withdrawal has been directly documented in humans, with higher FSH quartiles independently associated with greater vertebral marrow adiposity [13]. These changes may compound visceral adiposity by altering the bone marrow microenvironment and contributing to sarcopenic changes in the axial skeleton, though direct trafficking of BMDAs into peripheral visceral depots remains an area of emerging research.

2.1.5. Increase in follicle-stimulating hormone (FSH)

Rising FSH levels during the menopausal transition may have independent effects on body composition and energy homeostasis beyond their role as a marker of ovarian aging. In preclinical models, administration of FSH antibodies decreased fat mass and increased energy expenditure independently of estradiol levels, and FSH receptors have been identified in visceral adipocytes, suggesting a direct adipogenic role [14]. Human observational data, not surprisingly, present a more complex picture. Large cross-sectional cohort studies have found that elevated serum FSH is associated with higher bone marrow adiposity but is inversely correlated with visceral fat mass, body fat percentage, HOMA-IR, and metabolic syndrome in postmenopausal women, suggesting that postmenopausally, lower FSH may actually serve as a marker of greater metabolic dysfunction, reflecting more profound estrogen deficiency rather than a direct causal pathway [13,15–17]. Longitudinal studies adequately powered to isolate the independent contribution of FSH from estrogen decline are needed before causal conclusions can be drawn.

2.1.6. Metabolic alterations

Estradiol loss downregulates genes involved in beta-oxidation, resulting in impaired fatty acid utilization. Simultaneously, enhanced lipolysis in visceral adipose tissue (VAT) produces excess free fatty acids, promoting insulin resistance and metabolic dysfunction [18,19].

2.2. Clinical implications

The cardiovascular and metabolic consequences of the body composition changes that occur during the menopause transition have far-reaching short- and long-term consequences. As summarized in Table 2, the primary cardiometabolic changes are.

2.2.1. Alterations in basal metabolic rate (BMR)

Recent landmark research using doubly labeled water measurements demonstrates that adult energy expenditure, adjusted for fat-free mass, remains remarkably stable from ages 20–60 years, even during pregnancy. However, the proportion of lean mass is positively associated with basal metabolic rate. The loss of lean body mass during menopause therefore causes a decline in BMR. This change is often concealed by an unaltered or even increasing body mass index, resulting in sarcopenic obesity [20].

2.2.2. Increase in VAT

The accumulation of VAT during menopause has significant health implications. VAT is metabolically active tissue that releases free fatty acids, inflammatory cytokines, and adipokines that promote insulin resistance, dyslipidemia, and cardiovascular disease. Postmenopausal women experience accelerated cardiovascular risk, with rates of coronary heart disease approaching those of age-matched men within 10 years after menopause. It has been estimated that postmenopausal women have an approximately 5-fold higher risk of developing abdominal obesity compared to premenopausal women, though this estimate derives from a single cross-sectional study and should be interpreted with caution [21].

Importantly, this relationship is bidirectional. While estrogen deficiency drives adverse changes in body composition and cardiometabolic risk, the metabolic health a woman carries into the menopausal

Table 2
Cardiovascular and metabolic consequences of menopausal body composition changes.

Change	Metabolic Consequence	Clinical Impact
↑ Visceral Adipose Tissue	• Release of free fatty acids • ↑ Inflammatory cytokines • ↑ Adipokines	• Insulin resistance • Dyslipidemia • ↑ CVD risk
↓ Lean Body Mass	• ↓ Basal metabolic rate • ↓ Insulin sensitivity	• Sarcopenic obesity • Functional decline • ↑ Frailty risk
Fat Mass Redistribution	• Android (central) vs gynoid (peripheral) pattern	• ~5-fold ↑ risk of abdominal obesity • Accelerated CVD risk approaching age-matched men within 10 years

CVD = cardiovascular disease.

transition independently shapes how severely those changes manifest. Women with well-managed weight and insulin sensitivity prior to menopause experience fewer and less severe vasomotor symptoms, accumulate visceral adipose tissue at lower rates, and carry a more favorable baseline risk profile when MHT is clinically indicated [4,5,21,22]. This bidirectionality has a practical clinical implication that extends beyond the menopausal transition itself: weight management in the perimenopausal years is not a separate agenda from menopausal care - it is foundational to it.

3. Effects of menopause hormone therapy on body weight, composition, and metabolic health

Multiple randomized controlled trials and meta-analyses have examined the effects of MHT on body weight and composition, with generally consistent findings. These are summarized in Table 3.

3.1. Total body weight

A 2023 systematic review and meta-analysis of RCTs published from 2005 onwards found no statistically significant weight gain with most MHT regimens compared to non-users [23]. A comprehensive Cochrane review of 28 RCTs involving 28,559 women found no evidence that unopposed estrogen or combined estrogen and progestogen influence body weight additional to that usually gained at the time of menopause, and no evidence that hormone therapy prevents weight gain experienced at menopause [24].

The Women's Health, Osteoporosis, Progestin, Estrogen (Women's HOPE) study, a randomized, double-blind, placebo-controlled trial of 2673 postmenopausal women (1–4 years post-menopause), concluded that lower- and standard-dose regimens of conjugated estrogen (CE) or CE combined with medroxyprogesterone acetate (MPA) are not associated with greater weight gain than placebo. Body mass index does not influence response to treatment, nor does body weight change with lower doses of these medications in early postmenopausal women [25].

The Massachusetts Women's Health Study provided longitudinal data on 418 women aged 50–60 and concluded that use of MHT was not significantly related to weight. Behavioral factors - particularly exercise and ethanol consumption - were more strongly related to weight than the menopause transition [6].

The Postmenopausal Estrogen/Progestin Interventions (PEPI) Trial, a 3-year, multicenter, randomized, double-blinded, placebo-controlled clinical trial of 847 healthy postmenopausal women aged 45–64 years, found that women using estrogen with or without progestogen weighed, on average, 1 kg less than placebo recipients at the end of the 3-year trial (secondary outcome) [26].

3.2. Body composition

While total body weight may remain relatively stable, MHT demonstrates favorable effects on body composition. Studies using dual-energy X-ray absorptiometry and computed tomography consistently show that MHT.

- Attenuates the increase in total body fat mass that occurs during the menopausal transition
- Reduces visceral adipose tissue accumulation
- Preserves or increases fat-free (lean) mass
- Prevents the shift toward central fat distribution (decreased waist-to-hip ratio)

A 2019 meta-analysis that included 201 studies (11 longitudinal) involving 1,049,919 women found evidence of a potential protective role of hormone therapy in preventing or reducing trunk fat deposition, but not in preventing overall fat mass gain. As summarized in Table 4, fat mass significantly increased between premenopausal and postmenopausal women in the following measures: BMI, body weight, body fat percentage, waist circumference, waist-hip ratio, and visceral fat. The exception was total leg fat percentage, which significantly decreased [7].

A prospective study of postmenopausal women receiving combined MHT (17β-estradiol transdermal 50 μg plus medroxyprogesterone acetate) demonstrated fat loss of 2.1 ± 0.2 kg and decreased waist-to-hip ratio after 3 months, alongside increased lipid oxidation and energy expenditure [27].

The OsteoLaus cohort study, involving over 1000 women aged 50–80 years, found that MHT users had significantly lower VAT and android fat mass compared to never users, with effects persisting even after MHT discontinuation [28].

The Women's HOPE trial specifically examined body composition during treatment with conjugated estrogens with and without medroxyprogesterone acetate and provided additional evidence supporting favorable body composition effects independent of total weight change [25].

Importantly, these favorable body composition effects were observed even in studies using older formulations such as CEE and MPA - regimens that, as discussed below, likely underestimate the metabolic benefits achievable with contemporary bioidentical preparations.

3.3. Confounding variables and study heterogeneity

These findings are encouraging, but their clinical interpretation

Table 4
Meta-Analysis of Body Composition Changes: Premenopausal to Postmenopausal Women (Ambikairajah et al., 2019).

Measure	Mean Change	95% CI	Significance
BMI	+1.14 kg/m^2	0.95–1.32	Significant ↑
Body Weight	+1.00 kg	0.44–1.57	Significant ↑
Body Fat %	+2.88%	2.13–3.63	Significant ↑
Waist Circumference	+4.62 cm	3.90–5.35	Significant ↑
Waist-Hip Ratio	+0.04	0.03–0.05	Significant ↑
Visceral Fat	+26.90 cm^2	13.12–40.68	Significant ↑
Total Leg Fat %	−3.19%	−5.98 to −0.41	Significant ↓

Table 3
Evidence summary: Body weight change in individual trials of MHT.

Study	Design	N	Intervention	Duration	Key Findings
Women's HOPE	RCT, DB, PC	2673	CEE 0.3–0.625 mg daily with or without MPA 1.5–2.5 mg daily	2 years	No greater weight gain vs placebo; BMI does not influence response
PEPI Trial	RCT, DB, PC, MC	847	CEE 0.625 mg daily alone or combined with MPA 2.5–10 mg or micronized progesterone 200 mg daily	3 years	−1 kg vs placebo, favorable body composition
Massachusetts Women's Health Study	Longitudinal	418	Estrogen with or without progestogen, specific regimens not reported	N/A	MHT is not associated with weight gain. Exercise and alcohol more strongly related to weight than menopause or MHT

RCT = randomized controlled trial; DB = double-blind; PC = placebo-controlled; MC = multicenter; CEE = conjugated equine estrogens; MPA = medroxyprogesterone acetate; BMI = body mass index, MHT = menopause hormone therapy.

Table 5
Four pillars of obesity management in menopausal women.

Pillar	Key Components	Menopausal-Specific Considerations
NUTRITION	• Whole, minimally processed foods • Minimize postprandial glucose excursions • Adequate protein • Limit fructose • Moderate/eliminate alcohol • Adequate fiber • Minimize ultra-processed foods & NNS	• Protein especially critical given accelerated lean mass loss • Gut microbiome support during metabolic transition
PHYSICAL ACTIVITY	Aerobic: 150–300 min/wk moderate or 75–150 min/wk vigorous Resistance: 2–3 sessions/wk all major muscle groups Flexibility & Balance: Fall prevention NEAT: ↓ Sedentary time	• Resistance training priority to combat sarcopenia • Regular exercise → milder VMS, shorter duration • Address estrogen-related tissue stiffness • Stability training for functional capacity
BEHAVIORAL & PSYCHOLOGICAL	• Self-monitoring • Goal setting • Stimulus control • Problem-solving • Stress management • Professional counseling when indicated	• Recognize biopsychosocial complexity of midlife • Multiple competing demands (caregiving, work, identity shifts) • Stress → ↑ cortisol → ↑ VAT and insulin resistance • Address broader life context, not just "motivation"
MEDICAL INTERVENTIONS	OMs: BMI ≥30 or ≥27 with comorbidities Bariatric Surgery: BMI ≥40 or ≥35 with significant comorbidities	• Consider MHT OM interactions in shared decision-making for women with established MHT indications • Do NOT initiate MHT to enhance OM efficacy

NNS = non-nutritive sweeteners, NEAT = non-exercise activity thermogenesis, BMI = body mass index, VMS = vasomotor symptoms, VAT = visceral adipose tissue, MHT = menopause hormone therapy, OM = obesity medication.

requires an important caveat: MHT is not a single intervention. The studies contributing to this literature vary substantially in the type of estrogen and progestogen used-route of administration, timing of initiation, and baseline characteristics of enrolled populations — differences that carry meaningful biological consequences and that pooled analyses frequently obscure. Treating these pharmacologically distinct regimens as equivalent exposures produces attenuated or misleading estimates of metabolic effect, and likely accounts for much of the variability observed across trials. Understanding these sources of heterogeneity is therefore essential to translating the evidence into clinical practice.

3.3.1. *Route of administration*

Oral and transdermal estrogen preparations are pharmacologically distinct. Oral estrogen undergoes first-pass hepatic metabolism, stimulating hepatic production of VLDL and increasing triglycerides, while transdermal estradiol bypasses this pathway entirely, producing neutral-to-favorable effects on triglycerides and demonstrating greater reductions in insulin resistance in head-to-head comparisons [29,30].

3.3.2. *Formulation and regimen*

The specific hormonal components of MHT are a frequently overlooked source of heterogeneity. Conjugated equine estrogen (CEE), a mixture of multiple estrogen compounds derived from equine sources, differs meaningfully from 17β-estradiol, which is identical in molecular structure to endogenous human estrogen. These are not pharmacologically interchangeable and differ in their binding affinity at estrogen receptor subtypes, their hepatic metabolic profiles, and their downstream effects on lipid parameters, coagulation factors, and insulin sensitivity.

Synthetic progestogens, particularly medroxyprogesterone acetate (MPA), appear to blunt the favorable lipid and metabolic effects of estrogen to a greater degree than micronized progesterone, which more closely mirrors the molecular structure and receptor binding profile of endogenous progesterone. As demonstrated in a 2025 systematic review and meta-analysis of 17 RCTs, progestogen co-administration attenuates the insulin-sensitizing effects of estrogen alone, with combined estrogen-progestogen therapy producing significantly smaller reductions in HOMA-IR than estrogen alone [29].

It has been hypothesized that much of the adverse signal in the landmark WHI trial is attributable specifically to CEE combined with medroxyprogesterone acetate - a regimen that may not be representative of the metabolic effects of bioidentical 17β-estradiol combined with micronized progesterone, yet the two are routinely pooled in meta-analyses as equivalent exposures.

Regimen structure, whether continuous combined, sequential cyclic, or estrogen-alone, introduces additional variability that is rarely accounted for in pooled analyses.

Studies that treat MHT as a generalized exposure without accounting for estrogen type, progestogen type, and regimen structure are therefore likely to produce attenuated or misleading estimates of metabolic effect.

3.3.3. *Timing and duration*

Consistent with the timing hypothesis established in the cardiovascular literature, the metabolic benefits of MHT may similarly be contingent on proximity to menopause onset. Estrogen receptors in adipose tissue, skeletal muscle, and the hypothalamic appetite-regulatory centers may become progressively less responsive with prolonged estrogen deprivation - a concept supported mechanistically by animal models and indirectly by the observation that late initiators in RCTs consistently demonstrate attenuated metabolic responses compared with early initiators [31,32]. Evidence specific to weight and body composition outcomes stratified by timing of initiation and duration of therapy remains sparse, representing a significant gap in the literature.

3.3.4. *Population heterogeneity*

Studies evaluating MHT and body composition enroll populations that differ substantially in baseline characteristics known to independently modify treatment response; including BMI, insulin sensitivity, menopausal stage, race and ethnicity, and physical activity level. Until studies consistently stratify by baseline metabolic phenotype, pooled estimates of MHT's effect on body composition and metabolic outcomes should be interpreted with considerable caution.

3.4. *Appropriate indications for MHT*

Despite potential favorable effects on body composition, it is critical to emphasize that MHT is not indicated for weight loss or obesity treatment. The effects of MHT on total body weight are minimal and clinically insignificant - far below the 5–10% body weight reduction recommended for clinically meaningful health benefits in obesity management.

The North American Menopause Society (renamed The Menopause Society in 2023) [1], the American College of Obstetricians and Gynecologists [33], and The Endocrine Society [34] all emphasize that MHT should be prescribed based on validated clinical indications following individualized assessment. None recommend MHT as a primary treatment for obesity or overweight.

MHT is only FDA-approved for the following established clinical indications.

- Treatment of moderate to severe vasomotor symptoms
- Prevention of osteoporosis in postmenopausal women

- Treatment of hypoestrogenism caused by hypogonadism, bilateral oophorectomy or premature ovarian insufficiency
- Treatment of moderate to severe vulvovaginal symptoms

3.5. Effects of MHT on menopausal symptoms and quality of life

While MHT should not be prescribed solely for weight loss, it may provide indirect benefits that support comprehensive obesity treatment in appropriately selected patients.

3.5.1. Improvement in vasomotor symptoms

Vasomotor symptoms affect up to 80% of US women, with a median duration of 7.4 years [35,36]. These symptoms consist of episodes of flushing accompanied by the sensation of increased heat production, typically accompanied by sweating and lasting an average of 1–5 min. Peripheral vasodilation results in an increase in skin temperature up to 7 °C and can be especially pronounced in the fingers and toes. It may take up to 30 min to return to normal and may be followed by a decrease in core body temperature, which then causes chills [37].

Nearly 25% of women experience enough discomfort to seek help from a healthcare professional [38]. Bothersome vasomotor symptoms can significantly interfere with lifestyle modifications essential for weight reduction.

Vasomotor symptoms have important associations with weight status. Women with overweight and obesity report increased frequency and, through impaired thermoregulation, potentially severity of vasomotor symptoms, possibly due to a reduced thermoneutral zone. Adipose tissue acts as an insulator; therefore, more frequent and severe events may be required to achieve adequate heat loss in women with excess adipose tissue [22].

In addition to being more frequent in women with obesity, duration and intensity of vasomotor symptoms are typically worse in Black women, cancer survivors, smokers, women with anxiety, and women of lower socioeconomic status [35,36].

Understanding the relationship between weight loss and vasomotor symptoms is important, as data are mixed. A secondary analysis of the WHI Dietary Modification trial, concluded that postmenopausal women with vasomotor symptoms at baseline who lost $\geq$10% of their body weight within one year were significantly more likely to achieve symptom elimination compared with weight-stable controls - an effect attenuated for moderate-to-severe symptoms, although only losses exceeding 22 lbs. were associated with meaningful reduction [39]. Conversely, in a small RCT, Thurston found that weight loss was significantly correlated with reductions in questionnaire-reported hot flashes, with the magnitude of weight loss correlating with degree of symptom reduction [40].

Longitudinal data from the Study of Women's Health Across the Nation (SWAN) showed mixed results by menopausal stage [5] and the Midlife Women's Health Study found no significant association between weight change and hot flash frequency or severity, with investigators suggesting that lifestyle factors such as smoking may be more important determinants of vasomotor symptom burden than weight change per se [41]. Taken together, these findings suggest that while clinically meaningful weight loss may reduce vasomotor symptoms in some postmenopausal women, this relationship is neither consistent nor generalizable across all populations or menopausal stages.

It is important to note that actively gaining weight during perimenopause is associated with increased frequency and severity of vasomotor symptoms - regardless of a woman's starting weight [5].

3.5.2. Improvements in sleep

Sleep disturbances are among the most prevalent and clinically consequential symptoms of the menopausal transition, reported by 40–60% of perimenopausal and postmenopausal women [42]. Nighttime vasomotor symptoms directly fragment sleep architecture through repeated awakenings, and vasomotor symptom burden is the most consistently identified predictor of perceived sleep disturbances during the menopause transition [43].

The metabolic consequences of disrupted sleep are well-established: sleep deprivation produces measurable dysregulation of appetite-regulating hormones, specifically decreased leptin and elevated ghrelin, resulting in increased hunger and preferential consumption of energy-dense foods [44]. Chronically poor sleep quality is independently associated with metabolic syndrome, obesity, and cardiovascular risk factors – which is particularly relevant during the menopausal transition given the concurrent vulnerability to visceral adiposity and insulin resistance [45].

It is important to note that while vasomotor symptoms are the most common contributor to sleep disruption in midlife women, they are not the only one. Obstructive sleep apnea and restless leg syndrome are both significantly more prevalent in postmenopausal women than in younger women and are frequently underdiagnosed in this population - in part because their symptoms overlap with VMS-related sleep disruption [46]. Stress, alcohol, and caffeine sensitivity also commonly contribute to sleep fragmentation during this life stage and warrant clinical assessment independent of VMS management.

By effectively reducing the vasomotor symptom burden, MHT may improve sleep – and, by extension, improve overall well-being and support healthy lifestyle behaviors. A 2022 systematic review and meta-analysis confirmed that multiple MHT regimens significantly improve subjective sleep quality compared with placebo, with effects most pronounced in women with moderate-to-severe vasomotor symptoms [47].

3.5.3. Improvements in mood

The menopausal transition is independently associated with increased risk of depressive symptoms and diagnosed depression, with perimenopause representing the highest-risk stage across the reproductive lifespan [48]. Prior psychiatric history is the strongest individual predictor of mood disturbance during this period; women with a history of depression are significantly more likely to experience recurrence during the menopausal transition, suggesting that menopause frequently triggers exacerbation of existing psychological vulnerability rather than causing de novo mood disorders in most women [49]. Sleep disruption compounds this vulnerability: the same nocturnal vasomotor symptoms, musculoskeletal pain, urinary urgency, and other physical sequelae of estrogen deficiency that fragment sleep also contribute to emotional lability and impaired cognitive function; creating a bidirectional cycle in which mood disturbance worsens sleep and sleep deprivation worsens mood. Mood disturbances, in turn, can impair motivation for lifestyle change, increase stress-related eating, and reduce physical activity - each of which independently undermines weight management efforts.

Estrogen supplementation, particularly transdermal estradiol, has been associated with modest reductions in depressive symptoms, with effects most pronounced in perimenopausal women [34].

3.5.4. Improvements in physical function

Hypoestrogenism exerts direct effects on musculoskeletal tissues through the loss of estrogen receptor signaling in synovium, cartilage, and subchondral bone, producing a clinical syndrome of arthralgias, periarticular stiffness, and declining functional range of motion. These changes are clinically significant; decreased activity tolerance and pain-limited mobility represent independent contributors to sedentary behavior and physical deconditioning in perimenopausal and postmenopausal women. This phenotype has been formally characterized as the musculoskeletal syndrome of menopause [50] - a construct that usefully frames these symptoms as a coherent, estrogen-mediated pathophysiological entity rather than incidental age-related complaints.

Similarly, the genitourinary syndrome of menopause, encompassing genital dryness, dyspareunia, urinary urgency, and recurrent urinary tract infections, can substantially impair quality of life and limit engagement in physical activity [51].

By addressing musculoskeletal and/or genitourinary symptoms,

MHT may remove significant barriers to exercise participation and support maintenance of functional capacity - creating conditions under which behavioral weight management interventions are more likely to succeed.

3.6. Effects of MHT on visceral adiposity and cardiometabolic health

The visceral fat accumulation and cardiometabolic consequences that characterize the menopausal transition represent a distinct target through which MHT may provide clinically meaningful benefits beyond symptom relief.

3.6.1. Improvements in visceral adiposity

Estrogen deficiency drives the preferential accumulation of visceral adipose tissue that characterizes the menopausal transition [4] - a shift with well-documented consequences for insulin sensitivity, lipid metabolism, and cardiovascular risk. Evidence from randomized trials and observational cohorts suggests that MHT may attenuate several of these cardiometabolic changes, representing a mechanistically distinct pathway through which hormone therapy may support metabolic health in appropriately selected postmenopausal women [7,28].

3.6.2. Improvements in insulin sensitivity

A 2006 meta-analysis of 107 RCTs demonstrated that MHT significantly reduced insulin resistance across postmenopausal women, with reductions in HOMA-IR of 12.9% in non-diabetic women and 35.8% in women with established diabetes compared with controls, suggesting a clinically meaningful effect that may be amplified in the context of existing metabolic dysfunction. MHT was also associated with a 30% reduction in new-onset type 2 diabetes (RR 0.70, 95% CI 0.6–0.9) and a significant reduction in abdominal fat (−6.8%, 95% CI −11.8 to −1.9%) [52]. These findings have been corroborated by more recent evidence: a systematic review and meta-analysis of 17 RCTs involving 5772 postmenopausal women without diabetes confirmed that hormone therapy significantly reduced HOMA-IR compared with placebo. Notably, estrogen-alone regimens demonstrated substantially greater reductions in insulin resistance than combined estrogen-progestogen therapy, suggesting that progestogen co-administration attenuates the favorable metabolic effects of estrogen - a finding with potential implications for regimen selection in women with or at risk for insulin resistance [29].

3.6.3. Improvements in lipid parameters

The effects of MHT on lipid profiles appear to be route- and regimen-dependent. Oral estrogen preparations consistently reduce LDL cholesterol through accelerated LDL catabolism and raise HDL cholesterol when used as estrogen alone, though progestogen co-administration substantially attenuates the HDL benefit [53]. Oral preparations also significantly increase triglycerides via first-pass hepatic stimulation of VLDL production - an effect not observed with transdermal estradiol, which produces minimal changes in LDL and HDL but significantly reduces triglyceride levels [30]. Both oral and transdermal estrogen preparations reduce lipoprotein(a) concentrations, with a pooled mean reduction of approximately 20% across 47 studies, with oral estrogen demonstrating greater reductions than transdermal preparations [54]. The effect of progestogen co-administration on Lp(a) remains unsettled with some studies showing no significant attenuation with combined therapy, and others reporting that estrogen-plus-progestogen regimens are associated with higher Lp(a) compared with estrogen alone - a discordance that warrants caution in drawing firm conclusions about regimen-specific Lp(a) effects [30,54].

3.6.4. Cardiovascular risk modification

The cardiovascular effects of MHT appear to be dependent on timing of initiation relative to menopause onset. The ELITE trial - the only RCT specifically designed to test the timing hypothesis - demonstrated that oral estradiol slowed subclinical atherosclerosis progression when initiated within 6 years of menopause but had no effect when initiated more than 10 years after menopause [31]. A 2024 meta-analysis of 33 RCTs involving 44,639 postmenopausal women found no significant cardiovascular benefit in the overall population, but subgroup analysis revealed lower all-cause mortality and cardiovascular events in women who initiated MHT within 10 years of menopause compared with later initiators [55]. This discordance is consistent with the timing hypothesis: RCTs have historically enrolled women well beyond the critical window during which cardiovascular benefit appears most likely, attenuating any protective signal in pooled analyses [31,32,55]. These findings have achieved mainstream cardiovascular society endorsement, with the American Heart Association concluding that beneficial effects on CVD outcomes and all-cause mortality are specific to women younger than 60 years or fewer than 10 years from menopause onset, while null or harmful effects predominate with later initiation [32]. Stroke risk warrants specific consideration. Although transdermal estradiol has not been shown to increase the risk of ischemic stroke, oral preparations have [56]. The increased relative risk of ischemic stroke ranges from 11% with cyclic combined regimens to 29% with continuous combined regimens, while estrogen-only oral therapy confers an intermediate risk of approximately 18% [56]. This risk appears timing-dependent as well: early initiation of MHT was not associated with increased stroke risk regardless of route or formulation, while late initiation - particularly with oral conjugated equine estrogen - conferred significantly elevated risk [57]. Direct RCT comparisons by route of administration are lacking.

3.6.5. Preservation of lean body mass

The relationship between lean body mass and cardiometabolic health is well-established: skeletal muscle is the primary site of insulin-mediated glucose disposal, and its loss during the menopausal transition directly reduces resting metabolic rate while increasing cardiometabolic risk [58]. MHT may attenuate this process through estrogen receptor-mediated effects on muscle protein synthesis, with observational and mechanistic evidence supporting this pathway. A 2019 meta-analysis of 12 RCTs involving 4474 postmenopausal women found a trend toward lean mass preservation with MHT use; however, the difference did not reach statistical significance [59]. This effect may be particularly relevant when initiated early in the menopausal transition and combined with resistance training, but further study is warranted. Nonetheless, preservation of skeletal muscle mass remains a meaningful target for reducing cardiometabolic risk and supporting long-term weight management capacity in this population.

4. Risk stratification

The symptomatic and cardiometabolic benefits of MHT must be interpreted within the context of a well-characterized risk profile that not only takes into account formulation, route, and timing, but also considers the underlying health status of each patient. MHT is not appropriate for all patients. Clinical enthusiasm for its potential benefits should be matched by equally rigorous individualized risk stratification.

4.1. Cardiovascular risk

As established by the timing hypothesis, underlying cardiovascular health significantly influences the risk-benefit ratio of MHT. Women with obesity may have subclinical or established cardiovascular disease at younger ages than the general population, introducing a degree of individual cardiovascular risk that existing trial data, drawn largely from healthier populations, cannot fully quantify. This does not preclude MHT use in women with obesity, but it reinforces the necessity of thorough cardiovascular assessment prior to initiation.

4.2. Venous thromboembolism (VTE)

Oral estrogen increases VTE risk through first-pass hepatic activation of coagulation factors and acquired protein C resistance, approximately doubling baseline risk. Transdermal estradiol at standard doses does not significantly elevate VTE risk, including in women with obesity and other thrombotic risk factors [60]. Progestogen type further modulates thrombotic risk: synthetic progestogens, particularly MPA, carry a higher thrombotic burden than micronized progesterone [61]. In women with obesity, who carry independently elevated baseline VTE risk, these distinctions in route and formulation are of particular clinical relevance.

4.3. Malignancy

Public perception of MHT and cancer risk has been disproportionately shaped by early reporting of the Women's Health Initiative, which conflated the risks of a single regimen, conjugated equine estrogen combined with medroxyprogesterone acetate, with hormone therapy broadly. A more complete reading of the evidence tells a considerably more nuanced story. Estrogen-alone therapy, used in women without a uterus, is associated with a reduction in breast cancer incidence and mortality over long-term follow-up - a finding that directly contradicts the widely held perception that MHT uniformly increases breast cancer risk [62].

Combined estrogen-progestogen therapy is associated with a small duration-dependent increase in breast cancer risk, estimated at fewer than one additional case per 1000 women per year of use - a magnitude comparable to the risk conferred by 2 alcoholic beverages per day, obesity, or physical inactivity [1].

Endometrial cancer risk is effectively mitigated by appropriate progestogen co-administration in women with an intact uterus, and ovarian cancer risk, while statistically detectable, is small in absolute terms - approximately one additional case per 1000 users over 5 years [63].

5. Emerging data: MHT and highly effective obesity medications

Observational data published between 2024 and 2026 have suggested potential synergistic effects when MHT is combined with highly effective obesity medications, particularly GLP-1 receptor agonists. The evidence base remains small and methodologically limited, and the findings require careful interpretation before any clinical conclusions can be drawn.

5.1. Current evidence

5.1.1. Tirzepatide and MHT

A retrospective observational study evaluated 120 women treated with tirzepatide; 40 taking MHT vs. 80 not taking MHT. At a median follow-up of 18 months, postmenopausal women using MHT achieved 17% total body weight loss compared to 14% in those not using MHT ($p = 0.01$) - representing 35% greater absolute weight loss in the MHT group. Additionally, 45% of MHT users achieved $\geq$20% total body weight loss, compared to 18% of non-users [64].

5.1.2. semaglutide and MHT

A 2024 study in Menopause found that postmenopausal women on hormone therapy had higher weight loss at 12 months with semaglutide: 16% versus 12% ($p = 0.04$). This association remained significant after adjusting for confounders, although it was also a retrospective study and only 16 women were on hormone therapy versus 90 who were not [65].

5.2. Potential mechanisms

Current evidence cannot establish causation. The following mechanisms are proposed as biological hypotheses that would make a true interaction plausible, pending validation through randomized trials.

5.2.1. Biological synergy

Preclinical studies in rodents suggest potential synergistic interactions between estrogen and GLP-1 signaling pathways. Estrogen may enhance GLP-1 effects on appetite suppression, energy expenditure, and glucose metabolism through overlapping central nervous system pathways controlling satiety.

5.2.2. Metabolic priming

By preventing visceral fat accumulation and preserving insulin sensitivity, MHT may create a more favorable metabolic environment for obesity medication efficacy.

5.2.3. Healthy user bias

Women using MHT may be more engaged in overall health behaviors, more adherent to medications, and more likely to participate in complementary lifestyle interventions. The mitigation of vasomotor symptoms by MHT may facilitate exercise and dietary adherence. This explanation alone may account for the observed differences without any direct pharmacological interaction.

5.3. Critical limitations and cautions

The existing data, while hypothesis-generating, carry substantial methodological constraints that preclude clinical conclusions.

5.3.1. Study design and selection bias

Current evidence derives exclusively from retrospective, observational studies subject to multiple confounders. Selection bias, healthy user effect, and unmeasured confounding limit causal inference.

5.3.2. Lack of randomization

Without randomized assignment to MHT, it is impossible to determine whether observed differences reflect direct MHT effects or characteristics of women who choose to use MHT.

5.3.3. Small sample sizes

Most published analyses involve relatively small cohorts from single centers. For example, the study evaluating tirzepatide and MHT screened 15,639 patients, of whom 259 met inclusion criteria; after 1:2 propensity score matching on age, BMI, menopausal characteristics, prior obesity medication use, and diabetes status, the final analyzable cohort comprised only 40 women in the MHT group versus 80 without MHT, limiting statistical power to detect differential effects across subgroups or assess rare adverse events [64].

5.3.4. Publication bias

Positive findings are more likely to be presented at conferences and published, potentially skewing the evidence base.

6. Comprehensive obesity management in perimenopausal and menopausal women

Effective obesity management in perimenopausal and postmenopausal women requires a comprehensive, individualized approach addressing all four pillars outlined in the Obesity Medicine Association's Obesity Algorithm: nutrition, physical activity, behavioral modification, and medical intervention including pharmacotherapy and metabolic and bariatric surgery when indicated [66]. As summarized in Table 5, These evidence-based pillars are necessary and complementary - no single intervention is sufficient, and pharmacotherapy or MHT should not be considered a substitute for foundational lifestyle intervention.

6.1. Nutrition

A metabolic approach to nutrition during the menopause transition and beyond focuses on minimizing postprandial glucose excursions, reducing insulin stimulation, and attenuating chronic low-grade inflammation - the same pathways driving visceral adiposity and insulin resistance discussed throughout this review. This framework prioritizes whole, minimally processed foods; adequate protein to preserve muscle during a period of accelerated lean mass loss; limitation of dietary fructose, which drives hepatic de novo lipogenesis and insulin resistance; moderation or elimination of alcohol; and adequate soluble and insoluble fiber to support gut microbiome diversity [66]. Minimizing ultra-processed foods and non-nutritive sweeteners (NNSs) is particularly important during this life stage, as both alter gut microbiome composition and gut barrier integrity in ways that may compound the metabolic vulnerability of estrogen deficiency.

6.2. Physical activity

A comprehensive exercise program for menopausal women must address both cardiovascular fitness and preservation of lean body mass - the two most clinically consequential physical domains affected by the menopausal transition. The well-established benefits of regular physical activity, including improvements in insulin sensitivity, cardiovascular risk, bone density, mood, sleep, and cognitive function, are not unique to menopausal women but are particularly important during this life stage given the concurrent hormonal and metabolic vulnerabilities.

6.2.1. Aerobic exercise

150–300 min per week of moderate intensity aerobic exercise or 75–150 min per week of vigorous intensity aerobic exercise addresses cardiovascular fitness, cortisol regulation, and insulin sensitivity [66,67].

6.2.2. Resistance training

Skeletal muscle mass decline begins as early as the third decade of life and accelerates with menopause, reducing resting metabolic rate and perpetuating a vicious cycle of reduced energy expenditure and increased adiposity. 2–3 sessions of resistance exercise per week targeting all major muscle groups is of particular priority during the menopausal transition. Sarcopenic obesity, the coexistence of excess adiposity with insufficient muscle mass, poses compounded health risks beyond those of either condition alone, including accelerated functional decline, insulin resistance, frailty, and increased cardiometabolic mortality [58]. Preservation of muscle mass should be treated as a metabolic priority, not merely a fitness goal.

6.2.3. Flexibility, balance, and stability

Estrogen receptor-mediated collagen synthesis maintains the structural integrity and elasticity of muscle, ligament, and tendon tissues. Estrogen deficiency progressively compromises these properties, increasing stiffness and reducing range of motion in ways that directly impair balance, stability, and functional capacity for activities of daily living [50]. Targeted flexibility, balance, and stability training attenuate these changes and reduce fall risk - a clinical priority that becomes increasingly relevant as the menopausal transition advances. Reducing sedentary time and increasing non-exercise activity thermogenesis (NEAT) complement these efforts and round out a comprehensive movement strategy [66].

6.3. Behavioral and psychological considerations

The menopausal transition does not occur in a vacuum. It unfolds during what may be the most psychosocially complex chapter of a woman's life, in which physiologic change, identity reconfiguration, and extraordinary external demands converge simultaneously [68,69]. Understanding this context is central to personalized obesity treatment.

Women in midlife occupy a uniquely demanding position in the social architecture of American life. Nearly half of adults in their 40s and 50s are simultaneously raising or financially supporting children while caring for aging parents - the so-called sandwich generation - and women bear a disproportionate share of this burden [70]. These women are, in every meaningful sense, the load-bearing infrastructure of their families, workplaces, and communities - often at direct cost to their own health.

This caregiving burden intersects with the menopausal transition to create compounding metabolic risk. Chronic stress, generated by caregiving demands, professional responsibilities, relationship changes, and identity shifts occurring simultaneously [69], activates the hypothalamic-pituitary-adrenal axis, elevating cortisol and directly promoting visceral fat accumulation and insulin resistance. Stress management in this population is a metabolic intervention.

Midlife also frequently represents a period of significant identity reconfiguration - a time when women may seek to align their lives more closely with their authentic values and aspirations rather than continuing to fulfill roles defined by external expectations [70]. This process can be disorienting and emotionally taxing even in the absence of physiologic stressors, and its intersection with hormonal change, sleep disruption, mood instability, and physical symptoms creates a complexity that standard behavioral interventions often miss.

Standard evidence-based behavioral strategies - self-monitoring, goal setting, stimulus control, and problem-solving - remain foundational tools in obesity management and should be implemented as part of comprehensive care. However, their effectiveness depends on clinicians recognizing and, where possible, addressing the extraordinary competing demands under which midlife women are functioning.

When psychological barriers are prominent, evidence-based referral pathways include cognitive behavioral therapy, professional counseling, and peer support from women navigating similar life transitions.

It bears explicit acknowledgment that MHT, while capable of alleviating the physiologic contributors to mood instability, sleep disruption, and fatigue, does not resolve challenges related to relationships, career satisfaction, identity, or caregiving responsibilities - domains that require their own appropriate support and intervention.

Behavioral intervention in this population must be contextualized within the broader biopsychosocial demands of midlife. The physiologic changes of the menopausal transition do not occur in isolation - they unfold alongside caregiving responsibilities, occupational demands, relationship transitions, and identity shifts that independently tax the cognitive and emotional resources on which behavioral change depends. Treatment plans that fail to account for this context address only a fraction of the barriers these patients face.

6.4. Medical interventions

FDA-approved obesity pharmacotherapy should be considered for women with BMI $\geq$30 kg/m^2 or BMI $\geq$27 kg/m^2 with weight-related comorbidities, consistent with standard obesity medicine practice. For women with BMI $\geq$40 kg/m^2 or BMI $\geq$35 kg/m^2 with comorbidities, metabolic and bariatric surgery should be discussed as an option.

7. Limitations

This clinical review has several limitations that readers should consider.

7.1. Evidence base

Most of the evidence regarding MHT effects on body composition derives from studies conducted with older MHT formulations (primarily conjugated equine estrogens with or without medroxyprogesterone acetate). Contemporary bioidentical hormone preparations and

alternative routes of administration may have different effects that are not fully captured in this review.

7.2. Emerging MHT-OM data

The observational studies examining potential synergistic effects of MHT combined with obesity medications are limited by small sample sizes, retrospective design, lack of randomization, and potential for confounding. These preliminary findings require validation through adequately powered randomized controlled trials before clinical practice changes can be recommended.

7.3. Population diversity

Much of the evidence base for MHT effects on body composition comes from studies with limited racial and ethnic diversity. Body composition changes during menopause and responses to MHT may differ across populations, and generalizability of findings to all women requires caution.

7.4. Clinical heterogeneity

Women with obesity represent a heterogeneous population with varying degrees of metabolic dysfunction, cardiovascular risk, and menopausal symptom severity. This review provides general guidance but cannot capture all individual variations in risk-benefit profiles that influence clinical decision-making.

7.5. Rapidly evolving field

The landscape of obesity pharmacotherapy is advancing rapidly, with new anti-obesity medications and formulations emerging. As this field evolves, the interactions between MHT and novel OMs will require ongoing study and may alter clinical recommendations.

7.6. Testosterone therapy in women

This review addresses menopause hormone therapy as conventionally defined; estrogen therapy with or without progestogen co-administration. Testosterone therapy in women, while a topic of growing clinical and research interest particularly in the context of sexual function, mood, and body composition, is outside the scope of this review. The evidence base for testosterone's role in weight management and metabolic health in postmenopausal women remains limited and heterogeneous, and its relationship to obesity treatment represents a distinct and emerging area warranting dedicated review as the literature matures.

8. Conclusion

The menopausal transition represents a critical and often underappreciated inflection point in women's cardiometabolic health. Estrogen deficiency drives meaningful changes in body composition - including visceral fat accumulation, loss of lean body mass, and shifts in insulin sensitivity and lipid metabolism - that increase obesity-related health risks independent of chronological aging. While MHT provides well-established benefits for vasomotor symptoms, bone health, and quality of life, current evidence does not support its use as a primary or adjunctive weight loss intervention. Marketing claims suggesting otherwise are inconsistent with the evidence and with the positions of major professional societies.

Preliminary observational data suggest potential synergistic effects when MHT is combined with highly effective obesity medications in appropriately selected postmenopausal women; however, these findings are limited by small sample sizes, retrospective design, and potential confounding, and require validation through adequately powered randomized controlled trials before influencing clinical practice.

Obesity management in perimenopausal and postmenopausal women should be grounded in the four pillars of obesity treatment: nutrition, physical activity, behavioral modification, and medical interventions - with MHT prescribed only when established clinical indications exist following individualized risk-benefit assessment. Menopausal symptoms that impair adherence to lifestyle interventions deserve clinical recognition and treatment in their own right, not as a strategy for weight loss but because undertreated symptoms represent a meaningful and modifiable barrier to the comprehensive care these patients deserve. As the fields of menopausal medicine and obesity pharmacotherapy continue to advance rapidly and often in parallel, the intersection of these two disciplines represents a research priority with significant implications for women's health.

The relationship between metabolic health and the menopausal transition is bidirectional. Estrogen deficiency reshapes body composition, insulin sensitivity, and cardiovascular risk - but the severity of that reshaping is meaningfully influenced by the metabolic health a woman carries into the transition. Women with well-managed weight and cardiometabolic risk factors experience fewer and less severe menopausal symptoms, carry a more favorable baseline risk profile for MHT when it is indicated, and respond more robustly to comprehensive obesity treatment. Weight management in the years preceding menopause is therefore not a separate clinical agenda from menopausal care - it is foundational to it.

Key takeaways

- Menopause hormone therapy and obesity treatment address distinct but potentially synergistic physiological consequences of estrogen deficiency. Conflating them - by either prescribing MHT as a weight loss intervention or failing to recognize how undertreated menopausal symptoms undermine comprehensive obesity management - represents a missed opportunity for both. MHT should be prescribed for established clinical indications following individualized risk-benefit assessment; obesity should be treated with the full four-pillar framework regardless of MHT status.
- The indirect benefits of MHT in the context of obesity management - encompassing symptomatic relief, improvements in sleep and mood, preservation of physical function, and favorable cardiometabolic effects - are clinically meaningful but highly dependent on patient selection, timing of initiation, route of administration, and formulation. Preliminary observational data suggesting enhanced efficacy when MHT is combined with obesity medications require validation through randomized controlled trials before influencing clinical practice.
- The relationship between metabolic health and the menopausal transition is bidirectional: estrogen deficiency reshapes body composition and cardiometabolic risk, but the severity of that reshaping is meaningfully influenced by the weight and metabolic health a woman carries into the transition. Weight management in the years preceding menopause reduces symptom burden, improves the risk-benefit profile of MHT when it is indicated, and enhances the likelihood of successful obesity treatment - making it one of the most evidence-informed and underutilized strategies at the intersection of obesity medicine and women's health.

Author contributions

Courtney Younglove conceived the manuscript topic and structure, conducted the literature review, wrote the initial draft, and incorporated revisions.

Artificial intelligence declaration

During the preparation of this work, the author used ChatGPT and

Claude to assist with formatting, grammar and spelling verification, generation of initial table structures, verification of citation accuracy and statistical claims against primary sources, identification of relevant literature published after the initial manuscript preparation, and refinement of phrasing and sentence-level editing. The central argument, clinical interpretation, evidence synthesis, and overall structure of the manuscript represent the original intellectual work of the author. After using these tools, the author reviewed and edited all content and takes full responsibility for the content of the publication.

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Courtney Younglove has no financial or personal relationships with other people or organizations that could inappropriately influence this work.

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