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## **Menopause and exercise: hormonal influences on cardiovascular regulation: a narrative review**

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**Abstract**

The menopausal transition is associated with hormonal changes that adversely affect cardiovascular regulation and contribute to increased cardiovascular disease risk in women. Declining estrogen availability is linked to alterations in vascular function, blood pressure regulation, autonomic balance, and metabolic health that extend beyond the effects of chronological aging alone. This narrative review summarizes current evidence on menopause-related cardiovascular changes and examines the effects of physical activity and structured exercise training on vascular, hemodynamic, autonomic, and cardiorespiratory outcomes in peri- and postmenopausal women. Available evidence indicates that menopause is associated with increased arterial stiffness, altered blood pressure regulation, and a shift toward sympathetic predominance. Exercise training, particularly aerobic exercise, is consistently associated with improvements in arterial function, blood pressure control, cardiorespiratory fitness, and autonomic regulation. Resistance-based training shows more variable cardiovascular effects but supports broader functional and metabolic adaptations. Notably, many exercise-related benefits occur independently of significant weight loss, underscoring the importance of physiological rather than purely anthropometric mechanisms. Overall, regular exercise emerges as an effective systems-level strategy for attenuating menopause-related

cardiovascular dysregulation and supporting cardiovascular health during and after the menopausal transition.

**Keywords:** menopause; cardiovascular adaptations; exercise benefits; endothelial function; autonomic regulation; cardiorespiratory fitness

## INTRODUCTION

Cardiovascular disease (CVD) remains the leading cause of morbidity and mortality among women worldwide, with a marked acceleration in risk observed during midlife. Beyond chronological aging, the menopausal transition represents a critical biological period characterized by profound hormonal changes that influence cardiovascular regulation. Accumulating evidence indicates that the increase in cardiovascular risk observed in women around menopause cannot be fully explained by age alone, but rather reflects complex interactions between declining ovarian hormone levels and alterations in vascular, autonomic, and metabolic function [1].

The loss of estrogen during the menopausal transition has been implicated in multiple pathways relevant to cardiovascular health. Estrogen plays a key role in maintaining endothelial function, modulating arterial compliance, and regulating blood pressure through both direct vascular effects and interactions with the autonomic nervous system. Consequently, estrogen deficiency has been associated with endothelial dysfunction, increased arterial stiffness, altered blood pressure regulation and unfavorable changes in cardiometabolic risk profiles. These physiological alterations provide a mechanistic basis for the observed rise in cardiovascular risk during and after menopause [2,3].

Importantly, menopause is increasingly recognized not only as a reproductive milestone, but as a systemic transition with implications for cardiovascular regulation under both resting and stress conditions. Alterations in autonomic control, including increased sympathetic activity and reduced parasympathetic modulation, have been reported during the menopausal transition and are thought to contribute to impaired blood pressure control and heightened cardiovascular strain [4]. Such changes may also influence cardiovascular responses to physical stressors,

including exercise, thereby modifying both acute responses and chronic adaptations to physical activity in postmenopausal women [5].

Regular physical activity plays a central role in cardiovascular disease prevention throughout adulthood. Exercise training has been shown to improve endothelial function, reduce arterial stiffness, lower blood pressure, and enhance cardiorespiratory fitness, all of which are directly relevant to cardiovascular risk reduction [6]. However, the majority of exercise physiology literature has historically been derived from male or mixed-sex cohorts, with limited consideration of menopause-specific physiology. As a result, whether and how menopausal hormonal changes modify cardiovascular responses and adaptations to exercise remains incompletely understood [7].

Given the convergence of hormonal alterations, vascular dysfunction, and autonomic dysregulation during menopause, exercise represents a particularly relevant intervention. Exercise constitutes a controlled physiological stress that challenges cardiovascular regulation and promotes adaptive remodeling of vascular and autonomic function. Understanding how menopausal status influences these responses is essential for optimizing exercise prescription and maximizing cardiovascular benefits in midlife and older women [6,8].

Therefore, the purpose of this narrative review is to synthesize current evidence on the interplay between menopause, hormonal influences on cardiovascular regulation, and the effects of physical activity and exercise training. Specifically, this review aims to summarize menopause-related changes in cardiovascular and autonomic regulation, examine the effects of exercise training on key cardiovascular outcomes in postmenopausal women, and discuss the potential mechanisms by which exercise may reduce cardiovascular risk during menopause [1,6].

## METHODS

This narrative review was conducted to synthesise and contextualise current evidence examining menopause-related changes in cardiovascular and autonomic regulation, as well as the effects of physical activity and structured exercise interventions on cardiovascular outcomes in peri- and postmenopausal women. The methodological approach followed established principles of narrative synthesis, enabling the integration of findings from heterogeneous study designs and facilitating a physiological and mechanistic interpretation of the available evidence. A broad, iterative literature search was carried out in major scientific databases, including PubMed, Scopus and Web of Science. Search strategies combined MeSH terms and free-text keywords related to menopause (e.g., “menopause,” “postmenopause,” “menopausal

transition”), cardiovascular regulation (e.g., “arterial stiffness,” “endothelial function,” “blood pressure,” “autonomic regulation,” “heart rate variability”), and physical activity or exercise (e.g., “physical activity,” “exercise training,” “aerobic exercise,” “resistance training,” “combined training”). Additional searches targeted cardiovascular risk-related outcomes (e.g., “cardiorespiratory fitness,” “vascular function,” “cardiometabolic risk”). Reference lists of relevant articles were also screened to identify additional studies of potential relevance.

No restrictions were placed on study design, allowing inclusion of systematic reviews, meta-analyses, randomised controlled trials, non-randomised interventional studies, observational cohort studies and cross-sectional analyses. Priority was given to publications from the past 10–15 years, with particular emphasis on recent high-quality reviews and meta-analyses, as well as key original studies that provided mechanistic or physiological insights.

Studies were considered eligible for inclusion if they met the following criteria:

- 1) involved peri- or postmenopausal women,
- 2) examined cardiovascular, vascular or autonomic outcomes relevant to cardiovascular regulation or risk, and
- 3) investigated associations with physical activity, structured exercise interventions, or provided mechanistic insights relevant to menopause-related cardiovascular changes.

Exclusion criteria were intentionally limited to reflect the exploratory and integrative nature of narrative synthesis. Studies were excluded if they were non-original publications (e.g., editorials, commentaries), did not involve human participants, focused exclusively on premenopausal populations without menopause-specific analyses, or were not available in English.

Evidence was analysed thematically, with findings organised into key conceptual domains, including menopause-related vascular and autonomic changes, cardiovascular responses and adaptations to exercise training, and potential mechanistic pathways linking hormonal changes with exercise-induced cardiovascular regulation. This approach enabled integration of results across heterogeneous methodologies and outcomes, highlighting areas of consistency, divergence and emerging concepts relevant to cardiovascular health in menopausal women.

As this review was intended to provide an integrative and interpretative overview rather than a quantitative synthesis, no formal meta-analysis or structured risk-of-bias assessment was undertaken. Instead, the focus was placed on interpreting the available evidence within its physiological and methodological context.

## RESULTS

### 1. Menopause-related cardiovascular and autonomic changes

Evidence from observational studies and mechanistic research consistently indicates that the menopausal transition is accompanied by meaningful changes in cardiovascular regulation. The decline in ovarian estrogen production during this period is associated with alterations in vascular structure and function, as well as changes in autonomic cardiovascular control, which together contribute to a less favorable cardiovascular profile compared with premenopausal women [9].

Across studies, menopause has been associated with impaired vascular function, particularly at the level of the endothelium. Reduced estrogen exposure is linked to diminished nitric oxide bioavailability and attenuated vasodilatory responses to shear stress. In parallel, structural and functional changes within the arterial wall have been reported, resulting in increased arterial stiffness and reduced arterial compliance. These changes are reflected by higher pulse wave velocity and altered pressure wave reflections, which are considered early markers of cardiovascular risk [7,10].

Alterations in blood pressure regulation have also been consistently described during and after the menopausal transition. Rather than a uniform increase in resting blood pressure, the literature indicates a shift toward altered blood pressure regulation, including higher average values, increased variability, and exaggerated responses to physiological stressors. Importantly, these features have been observed even in women without clinically diagnosed hypertension, suggesting that menopause represents a period of subclinical cardiovascular dysregulation [6].

In addition to vascular changes, evidence points to menopause-related alterations in autonomic cardiovascular control. Studies have reported increased sympathetic activity alongside reduced parasympathetic modulation during the menopausal transition. This autonomic imbalance has been associated with impaired baroreflex sensitivity and altered heart rate dynamics, which may contribute to dysregulated blood pressure control and heightened cardiovascular reactivity to stress [11].

The literature further suggests that vascular and autonomic changes during menopause are closely interrelated. Increased arterial stiffness may blunt baroreceptor signaling, while heightened sympathetic tone can exacerbate vasoconstriction and elevate vascular resistance. Together, these processes appear to reinforce one another, leading to integrated disturbances in cardiovascular regulation. These findings provide important context for understanding

cardiovascular responses to physical stressors, including exercise, in postmenopausal women [12,13].

## **2. Effects of exercise training on vascular and hemodynamic outcomes**

Evidence from systematic reviews, meta-analyses, and interventional studies indicates that exercise training exerts beneficial effects on vascular function and blood pressure regulation in postmenopausal women. Across the literature, structured exercise interventions have been consistently associated with improvements in arterial stiffness and hemodynamic profiles, although the magnitude of these effects varies depending on training modality, intensity, and baseline cardiovascular status [14].

### **2.1 Arterial stiffness**

Arterial stiffness has emerged as a key vascular outcome in studies examining the effects of exercise training in postmenopausal women. Measures such as pulse wave velocity are frequently used as surrogate markers of arterial health and are strongly linked to cardiovascular risk. Evidence suggests that aerobic exercise training is associated with reductions in arterial stiffness, particularly when interventions are of sufficient duration and intensity [14]. Moderate-intensity continuous training programs lasting several weeks to months have been shown to improve arterial compliance, likely reflecting both functional and structural adaptations within the arterial wall [15].

Resistance training and combined aerobic–resistance programs have also been investigated, although findings are more heterogeneous. Some studies report modest improvements in arterial stiffness following resistance-based interventions, while others show neutral effects. Differences in training volume, load prescription, and participant characteristics may partly explain these discrepancies. Overall, the literature suggests that aerobic components are particularly important for inducing favorable changes in arterial stiffness in postmenopausal women, whereas resistance training alone may produce more variable vascular responses [16,17].

### **2.2 Blood pressure**

Exercise training has also been associated with improvements in blood pressure regulation in postmenopausal women. Across studies, regular aerobic exercise is linked to reductions in resting systolic and diastolic blood pressure, as well as improvements in blood pressure responses to physiological stress. Importantly, these benefits have been observed not only in women with elevated baseline blood pressure but also in those with values within the normotensive range, supporting a role for exercise in early cardiovascular risk modification [8].

Beyond resting blood pressure, exercise training appears to influence dynamic aspects of blood pressure regulation. Some studies report attenuated blood pressure responses during submaximal exercise and improved post-exercise blood pressure recovery following training. These effects may reflect adaptations in vascular function, autonomic control, or both. Resistance and combined training programs have demonstrated variable effects on blood pressure, with outcomes likely influenced by training intensity, progression, and participant baseline characteristics [6].

### **2.3 Integrated vascular and hemodynamic adaptations**

Taken together, the evidence indicates that exercise training induces integrated vascular and hemodynamic adaptations in postmenopausal women. Improvements in arterial stiffness and blood pressure regulation appear to be interrelated, as reduced arterial stiffness may contribute to lower systolic load and improved pressure buffering. Although heterogeneity exists across studies, the overall pattern supports a beneficial role of regular exercise training in counteracting menopause-related vascular and hemodynamic alterations [8].

## **3. Exercise and cardiorespiratory fitness**

Cardiorespiratory fitness is a central marker of cardiovascular health and a strong predictor of cardiovascular morbidity and mortality. In postmenopausal women, lower levels of cardiorespiratory fitness have been consistently reported compared with premenopausal counterparts, highlighting its relevance as a modifiable outcome in this population. The available literature indicates that exercise training is an effective strategy for improving cardiorespiratory fitness in postmenopausal women, although the magnitude of improvement varies across studies [18].

Across systematic reviews and interventional studies, aerobic exercise training has been most consistently associated with improvements in cardiorespiratory fitness, typically assessed using measures such as peak oxygen uptake or maximal exercise capacity. Interventions involving moderate-intensity continuous training performed several times per week over periods of weeks to months commonly report significant gains in fitness. These improvements appear to occur across a wide range of baseline fitness levels, suggesting that postmenopausal women retain a substantial capacity for aerobic adaptation [19].

High-intensity interval training has also been examined in a smaller number of studies, with evidence suggesting that it may elicit comparable or greater improvements in cardiorespiratory fitness over shorter timeframes. However, heterogeneity in protocol design, intensity prescription, and participant characteristics limits direct comparison with moderate-intensity

approaches. As a result, while interval-based training appears promising, the literature does not yet allow firm conclusions regarding its superiority in postmenopausal populations [20].

Resistance training alone has generally produced smaller or more variable effects on cardiorespiratory fitness. While resistance-based programs contribute to improvements in muscular strength and functional capacity, their impact on aerobic fitness outcomes is less consistent. Combined aerobic–resistance training programs have been shown to improve cardiorespiratory fitness, largely reflecting the aerobic component of the intervention, although such programs may offer additional benefits for overall physical function [15].

Importantly, improvements in cardiorespiratory fitness have been observed even in the absence of substantial changes in body mass or body composition, indicating that training-induced adaptations are not solely dependent on weight loss. This finding underscores the relevance of exercise training as a cardiovascular intervention during and after the menopausal transition, independent of changes in adiposity [21].

Overall, the evidence indicates that exercise training, particularly when it includes an aerobic component, is effective in improving cardiorespiratory fitness in postmenopausal women. These findings complement reported improvements in vascular and hemodynamic outcomes and support the role of regular exercise as a key strategy for enhancing cardiovascular health in this population [15].

#### **4. Exercise and autonomic regulation**

Alterations in autonomic cardiovascular regulation are increasingly recognized as an important feature of the menopausal transition and a potential contributor to cardiovascular risk. As described earlier, menopause is associated with a shift toward increased sympathetic activity and reduced parasympathetic modulation. Against this background, exercise training has been investigated as a non-pharmacological strategy capable of modulating autonomic function in postmenopausal women [5].

Evidence from interventional studies suggests that regular exercise training can induce favorable changes in autonomic regulation, most commonly assessed using indices of heart rate variability. Aerobic exercise interventions performed over several weeks to months have been associated with increases in parasympathetic markers and improvements in overall autonomic balance. These adaptations are generally interpreted as reflecting enhanced vagal modulation and reduced sympathetic dominance, although the magnitude of reported effects varies across studies [22].

The effects of exercise on autonomic regulation appear to depend on training characteristics and baseline autonomic status. Moderate-intensity aerobic training has been most consistently

associated with improvements in heart rate variability, whereas evidence for resistance-based interventions is more limited and heterogeneous. Combined training programs may also improve autonomic indices, although it remains difficult to disentangle the specific contributions of aerobic and resistance components due to variability in study design [23,24]. Beyond resting measures, some studies have examined exercise-induced changes in dynamic autonomic responses, such as heart rate recovery following exercise or autonomic responses during submaximal workloads. Improvements in post-exercise heart rate recovery have been reported following training, suggesting enhanced parasympathetic reactivation. These findings are particularly relevant given the established association between impaired heart rate recovery and increased cardiovascular risk [25].

Despite these encouraging findings, the literature on exercise and autonomic regulation in postmenopausal women remains relatively limited compared with evidence for vascular and hemodynamic outcomes. Differences in assessment methods, small sample sizes, and heterogeneity in training protocols contribute to variability across studies. Nevertheless, the available evidence supports the concept that exercise training has the potential to partially counteract menopause-related autonomic dysregulation, complementing its effects on vascular function and cardiorespiratory fitness.

## DISCUSSION

### 1. Integrative mechanistic interpretation

The findings synthesized in this review indicate that menopause-related cardiovascular changes arise from the interaction of vascular, autonomic, and metabolic alterations rather than from a single isolated mechanism. Declining estrogen availability during the menopausal transition contributes to impaired endothelial function, unfavorable changes in large artery properties, and a shift in autonomic balance toward sympathetic predominance. Together, these alterations compromise cardiovascular regulation and may increase vulnerability to adverse hemodynamic responses, particularly during physiological stress [9,14].

Exercise training appears to counteract several of these menopause-related changes through multiple, interconnected pathways. Improvements in vascular function and blood pressure regulation are likely driven, at least in part, by repeated increases in shear stress during aerobic exercise, which stimulate endothelial adaptations and nitric oxide availability. At the same time, exercise-induced modulation of autonomic control, reflected by enhanced parasympathetic

activity and improved autonomic balance, may contribute to more stable cardiovascular regulation both at rest and during dynamic conditions [22,23].

Metabolic factors represent an additional and important layer within this integrative framework. Menopause is associated with adverse metabolic changes, including increased insulin resistance, greater visceral adiposity, and reduced metabolic flexibility, all of which have been linked to endothelial dysfunction, increased vascular load, and heightened sympathetic activity. Exercise training improves metabolic health by enhancing insulin sensitivity, reducing visceral fat, and increasing skeletal muscle oxidative capacity, often without substantial changes in body mass. These metabolic adaptations may therefore mediate part of the beneficial influence of exercise on vascular and autonomic regulation in peri- and postmenopausal women [24].

Importantly, the relative contribution of these mechanisms likely varies across individuals and exercise modalities. Aerobic exercise appears to provide a particularly potent stimulus for coordinated vascular and autonomic adaptations, whereas resistance-based training may exert more variable effects through distinct mechanical and metabolic pathways [15,16]. This heterogeneity highlights the importance of considering hormonal status, baseline cardiometabolic health, and training characteristics when interpreting cardiovascular responses to exercise during and after the menopausal transition.

Overall, the available evidence supports a model in which exercise mitigates menopause-associated cardiovascular risk through the combined influence of vascular, autonomic, and metabolic adaptations. Rather than acting through a single pathway, regular physical activity exerts broad regulatory effects that may partially offset the physiological changes accompanying menopause [7].

## **2. Practical implications for exercise programming**

The evidence summarized in this review supports the use of regular physical activity and structured exercise training as a central strategy for maintaining cardiovascular health during and after the menopausal transition. While menopause-related vascular, autonomic, and metabolic changes may increase cardiovascular vulnerability, the available data indicate that these alterations do not preclude meaningful cardiovascular adaptations to exercise [7].

From a practical perspective, exercise programs for peri- and postmenopausal women should prioritize activities that provide a sufficient cardiovascular stimulus to promote vascular and hemodynamic adaptations. Aerobic exercise performed at moderate intensity on a regular basis appears particularly effective in improving vascular function, blood pressure regulation, and cardiorespiratory fitness. Importantly, these benefits have been reported even in women without

overt cardiovascular disease or hypertension, underscoring the value of exercise as a preventive strategy rather than solely a therapeutic intervention [14,15].

Resistance training, although associated with more variable cardiovascular responses, remains an important component of comprehensive exercise programming. Improvements in muscular strength, functional capacity, and metabolic health may indirectly support cardiovascular regulation and overall physical independence [16,24]. Combined aerobic–resistance programs may therefore offer a balanced approach, addressing multiple physiological systems simultaneously, even if vascular adaptations are primarily driven by the aerobic component.

The heterogeneity observed across exercise studies highlights the need for individualized exercise prescription. Baseline cardiometabolic health, fitness level, menopausal stage, and tolerance to different training modalities should be considered when designing programs. Gradual progression, appropriate intensity selection, and long-term adherence are likely more important determinants of cardiovascular benefit than the choice of a specific exercise modality [26].

Overall, the findings of this review support current public health recommendations encouraging regular physical activity across adulthood and reinforce the notion that the menopausal transition represents a critical window during which exercise may help attenuate emerging cardiovascular risk.

## CONCLUSIONS

This narrative review highlights that menopause-related cardiovascular changes arise from the combined influence of vascular, autonomic, and metabolic alterations that extend beyond the effects of chronological aging alone. The menopausal transition is characterized by declining estrogen availability, which contributes to impaired cardiovascular regulation and increased vulnerability to adverse hemodynamic responses. Importantly, the evidence synthesized in this review indicates that these changes do not represent a loss of physiological adaptability, but rather a shift in regulatory balance that remains responsive to lifestyle interventions.

Exercise training emerges as a central modulator of cardiovascular health in peri- and postmenopausal women. Across the literature, regular physical activity particularly when it includes an aerobic component is consistently associated with improvements in vascular function, blood pressure regulation, cardiorespiratory fitness, and autonomic balance. These adaptations appear to occur through coordinated mechanisms involving endothelial function,

neural cardiovascular control, and metabolic regulation, reinforcing the concept of exercise as a systems-level intervention rather than a single-pathway strategy.

The findings further suggest that metabolic adaptations induced by exercise, such as improved insulin sensitivity and enhanced skeletal muscle oxidative capacity, may indirectly support cardiovascular regulation, even in the absence of significant weight loss. This underscores the importance of focusing on functional and physiological outcomes rather than body mass alone when evaluating the cardiovascular benefits of exercise during and after the menopausal transition.

Collectively, the available evidence supports regular exercise as a key strategy for mitigating menopause-associated cardiovascular risk and preserving cardiovascular health across midlife and beyond. While the current literature is weighted toward postmenopausal populations, emerging data indicate that earlier engagement in physical activity during the menopausal transition may be particularly beneficial. Future research integrating menopausal stage, cardiometabolic health, and exercise characteristics will be essential for refining exercise-based approaches aimed at optimizing cardiovascular regulation in women during this critical period of life.

## **Disclosure**

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