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Review

Optimizing women's cardiovascular health across the life course: The role of physical activity in female-specific conditions

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ABSTRACT

Cardiovascular disease is the leading cause of morbidity and mortality in women, and its development results from both traditional risk factors and female-specific determinants that emerge across the life course. This narrative review explores the role of physical activity in reducing risk associated with five major sex-specific cardiovascular determinants: hormonal contraception, polycystic ovary syndrome, endometriosis, pregnancy complications and menopause. This review synthesized up-to-date studies exploring the associations between these conditions and cardiovascular risk in women, as well as research assessing whether physical activity may help to reduce this risk through various mechanisms. Hormonal contraception, polycystic ovary syndrome, endometriosis, pregnancy complications and menopause are each associated with distinct or overlapping pathophysiological pathways that involve metabolic dysfunction, systemic inflammation, endothelial impairment, adverse vascular remodelling or prothrombotic alterations. Together, these mechanisms contribute to an elevated lifetime risk of cardiovascular disease in women. Physical activity appears to improve many of these underlying processes, although the level of evidence, the magnitude and the specificity of these benefits vary by condition. Physical activity is a central and highly relevant approach to preventing cardiovascular disease in women exposed to sex-specific risk factors. Although current evidence is encouraging, further research is needed to clarify mechanistic pathways, strengthen causal inference and refine exercise prescriptions adapted to each condition. Individualized risk assessment and the integration of physical activity into long-term preventive and clinical management strategies are essential to reduce the cardiovascular burden in women.

1. Abbreviations

CI Confidence interval
COC Combined oral contraception
CRP C-reactive protein
CVD Cardiovascular disease
HC Hormonal contraception
HR Hazard ratio
OC Oral contraception
OR Odds ratio
PA Physical activity
PCOS Polycystic ovary syndrome
RAAS Renin-angiotensin-aldosterone system
RR Relative risk

2. Background

Cardiovascular disease (CVD) is the leading cause of mortality and morbidity in women, and its rising prevalence among those aged < 65 years constitutes a major public health concern [1]. This can be explained by several risk factors. Women are exposed to the traditional well-established risk factors shared with men (e.g., hypertension, dyslipidaemia, diabetes), as well as behavioural factors, such as physical inactivity and smoking [2]. In addition, several of these shared factors exert a more adverse cardiovascular impact in women, including hypertension, smoking and type 2 diabetes [3]. Psychological determinants, such as stress, anxiety and depression, also contribute disproportionately to certain cardiovascular conditions in women [1].

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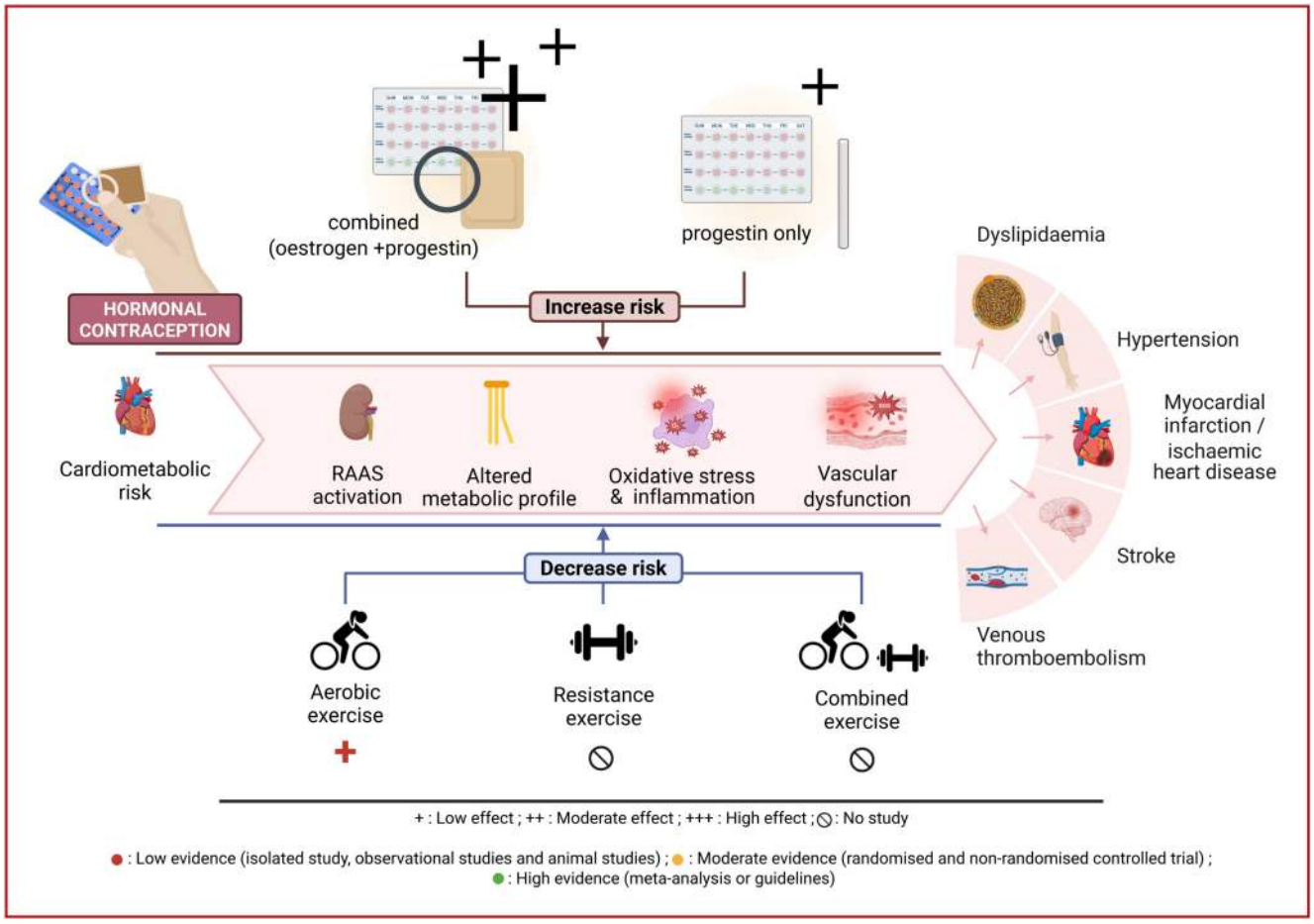


Fig. 1. The role of physical activity in reducing the cardiovascular risk associated with hormonal contraception. RAAS: renin-angiotensin-aldosterone system.

Women also face sex-specific cardiovascular risk factors across their life course. Large fluctuations in endogenous sex hormones (e.g. during pregnancy and menopause), as well as varying exposure to exogenous sex hormones through hormonal contraception (HC) or menopausal hormone therapy, influence cardiovascular physiology [4]. Hormonal and gynaecological disorders (e.g., polycystic ovary syndrome [PCOS] and endometriosis) may further contribute to inflammation and metabolic dysregulation [5]. Given this complex and evolving risk profile, adopting a physically active lifestyle represents a fundamental strategy for mitigating cardiovascular risk, with accumulating evidence indicating that women may benefit from greater cardioprotective effects of physical activity (PA) than men. For example, females achieved a coronary heart disease risk reduction of 30% (hazard ratio [HR] 0.70) with 250 minutes/week of moderate-to-vigorous PA, whereas males required 530 minutes/week for similar benefits [6]. Yet, paradoxically, women remain more susceptible to physical inactivity across all stages of life [7,8]. Because women seem more compliant with medication than men [9], physician-delivered exercise prescriptions may represent a particularly effective strategy for PA engagement, especially among those with female-specific risk factors. Despite these considerations, research specifically addressing how PA interacts with cardiovascular risk factors across the female life course remains limited [10]. This review aims to synthesize the current literature on the effects of PA on five key women-specific risk factors. These conditions were selected based on their high prevalence, distinct relevance to women and growing evidence of their association with long-term cardiovascular risk.

3. HC

HC, which is widely used by women of reproductive age, has been associated with an increased risk of arterial thrombotic events; however, the absolute risk remains very low, particularly in younger women (Fig. 1). This risk varies according to the type of contraception used (e.g., oral contraceptives, vaginal rings, intrauterine devices, implants or patches), the oestrogen dose and the type of progestin used [11], as well as other cardiovascular risk factors, such as smoking, hypertension and body mass index.

Most studies have focused on oral contraception (OC), including recent large Danish cohort studies, which reported a 1.7–1.8-fold increase in the risk of ischaemic stroke and myocardial infarction with combined OC (COC); this corresponds to a small absolute excess risk of approximately 20 and 10 additional cases per 100,000 person-years, respectively, which increases with age and only becomes statistically significant after the age of 40 (with an additional risk of up to ~25 cases per 100,000 women-years) [12,13]. Importantly, data from the WAMIF study indicated that myocardial infarction in young COC users predominantly occurs in the presence of additional cardiovascular risk factors. This suggests that the excess risk is not solely attributable to COC itself, but rather to its use by women with underlying risk factors [14]. Compared with COC, progestin-only OC appears to be associated with a lower increase in arterial events. However, this advantage is not apparent with implants or injectable formulations. Nevertheless, evidence remains limited for all progestin-only methods [11].

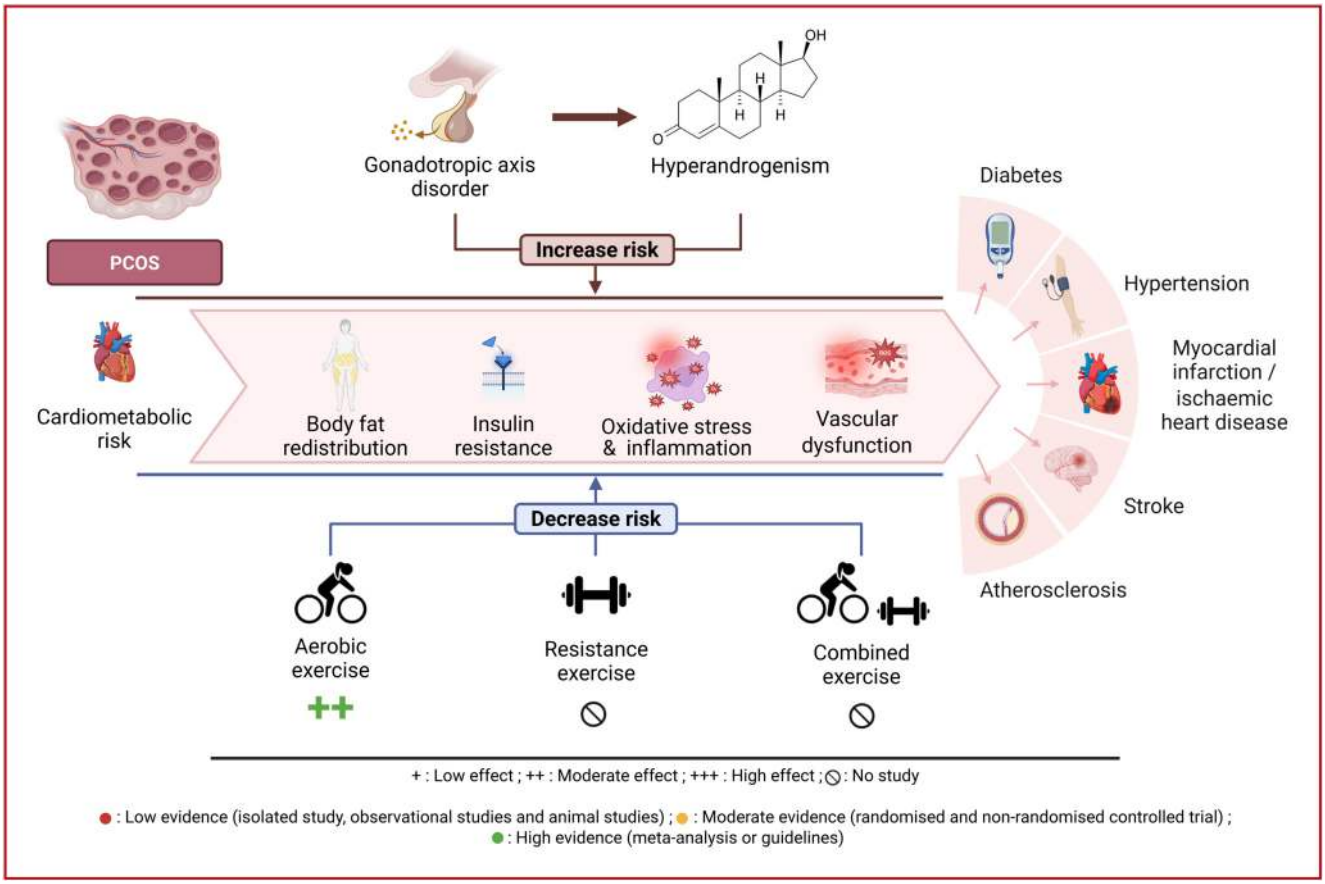


Fig. 2. The role of physical activity in reducing the cardiovascular risk associated with polycystic ovary syndrome (PCOS).

Beyond thrombotic events, OC has also been associated with the development of hypertension. A meta-analysis including 24 studies and more than 250,000 women reported a duration-dependent increase in hypertension risk, with a pooled relative risk of approximately 1.13 (95% confidence interval [CI] 1.03–1.25) per 5 years of OC use, accompanied by a modest but consistent rise in blood pressure of about 2–3 mmHg, indicating a low but epidemiologically relevant increase in hypertension risk [15]. Mechanistic studies suggest that this effect is multifactorial, involving oxidative stress, endothelial dysfunction and activation of the renin-angiotensin-aldosterone system (RAAS) [16]. Supporting this, Oliveira et al. (2020) conducted an observational study in young women using low-dose OC for at least 1 year, and found higher plasma renin concentrations compared with non-users, which correlated strongly with plasma C-reactive protein (CRP) [17]. Although these women had not reached hypertensive levels, their systolic blood pressure was higher than that of controls (Δ of 2–4 mmHg), raising the hypothesis that sustained RAAS activation might contribute to blood pressure increase over time [18]. The study also highlights the interplay between RAAS activation and inflammatory pathways, as reflected in the correlation with CRP. In addition, some studies suggest that the use of OC increases the risk of dyslipidaemia, insulin-resistance and prediabetes, whereas progestin-only contraceptives do not [19,20]. Taken together, these data promote the hypothesis that OC can influence vascular health through multiple pathways, including prothrombotic effects, inflammation, RAAS-mediated blood pressure changes and metabolic dysregulation.

Although research on this topic remains limited, several studies indicate that women using HC who are physically active tend to exhibit more favourable cardiometabolic profiles than those with an inactive lifestyle. Active women show lower triglyceride concentrations and better insulin sensitivity compared with their inactive counterparts [21]. Two months of high-intensity interval training (repeated short bouts of intense exercise interspersed with periods of rest or low-intensity activity) has been shown to reduce systemic inflammation in women using combined OC (lower CRP concentrations), although it was not associated with changes in the lipid profile [22]. Importantly, active women using OC exhibit lower arterial stiffness [23], highlighting the protective effect of regular PA on macrovascular function. Conversely, OC use has been associated with impaired oxidative balance, as women using OC showed higher markers of lipid peroxidation, and exercise-induced improvements in antioxidant capacity were markedly attenuated compared with non-users [24]. Overall, these findings suggest that while PA may not completely offset all OC-related physiological changes, it could play an important role in improving cardiovascular and metabolic health in this population. Current evidence on the interaction between exercise and HC use remains limited, and is predominantly based on cross-sectional studies, underscoring the need for well-designed longitudinal and interventional research to clarify causal relationships. Nevertheless, regular exercise remains advisable for women with additional cardiovascular risk factors, despite low-certainty evidence.

4. PCOS

PCOS is a complex endocrine disorder affecting both the gonadotropic axis and metabolism in women of childbearing age (Fig. 2). This syndrome is the result of a disorder of the female gonadotropic axis, leading to ovarian cysts, hyperandrogenism (clinical and/or biological) and fertility problems (75% of women have anovulatory cycles) [25,26]. Worldwide, its prevalence varies from 6% to 20%, depending on the diagnostic criteria used [27]. PCOS is commonly associated with metabolic disturbances promoting CVD. Central (android) obesity, dyslipidaemia, insulin resistance, type 2 diabetes and hypertension

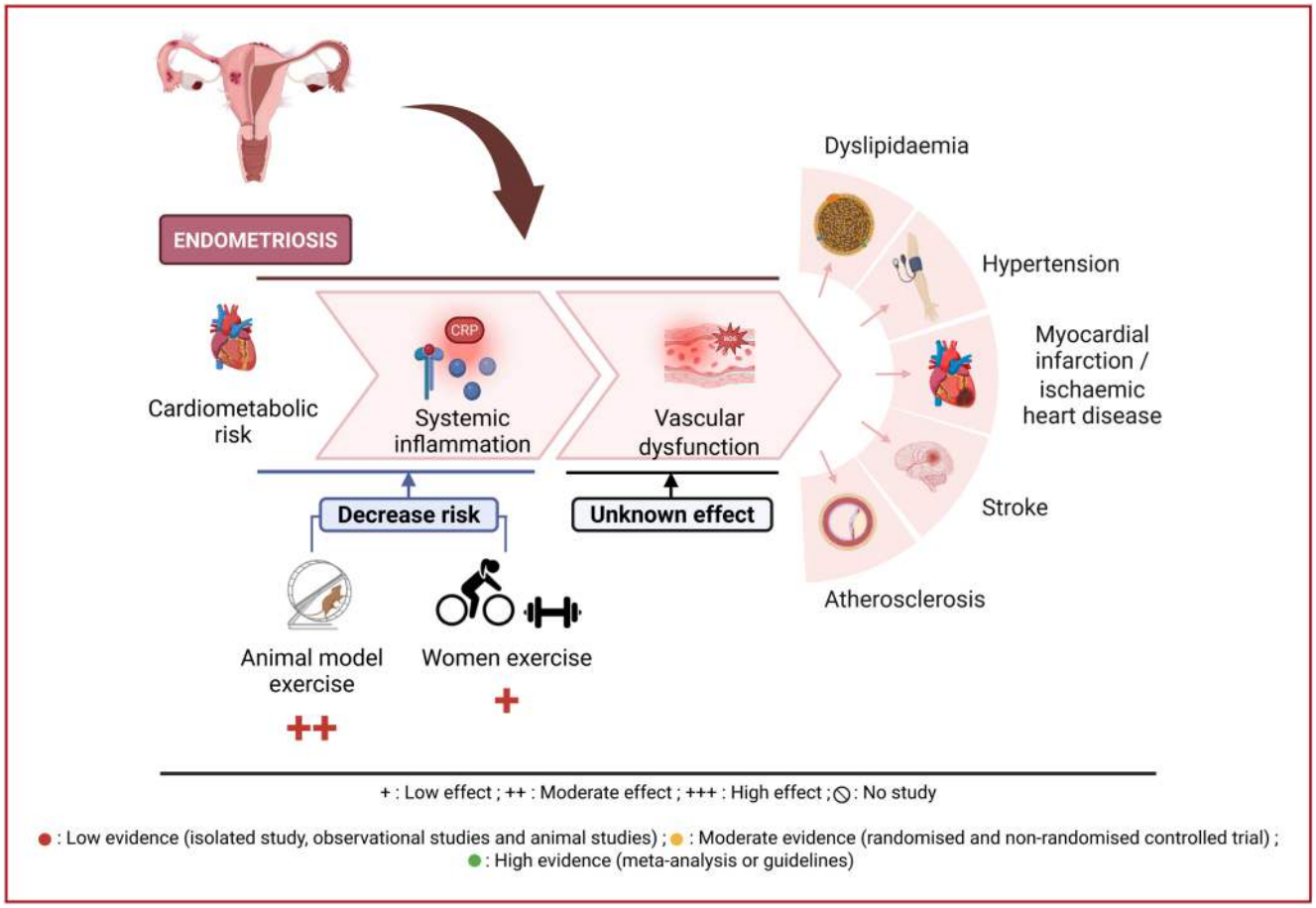


Fig. 3. The role of physical activity in reducing the cardiovascular risk associated with endometriosis.

contribute to impaired endothelial and myocardial function [28–30]. These metabolic and vascular abnormalities are accompanied by low-grade systemic inflammation, evidenced by elevated serum concentrations of inflammatory markers such as CRP, tumour necrosis factor alpha and interleukin-6, which play key roles in the pathophysiology of atherosclerosis. Consequently, women with PCOS show early signs of subclinical atherosclerosis, including increased carotid intima-media thickness, elevated pulse wave velocity and coronary calcifications, even when compared with age- and body composition-matched controls [31,32]. Collectively, these pathophysiological alterations predispose women with PCOS to overt CVD, manifesting as a higher risk of myocardial infarction, ischaemic heart disease and stroke [33]. It should also be noted that women with PCOS are at increased risk of developing pregnancy complications, such as diabetes, hypertension, preterm delivery and fetal macrosomia [34].

Treatment of PCOS is primarily based on nutritional adaptations, improving overall health, hormonal profile and quality of life [35]. Several systematic reviews have highlighted the key role of PA in the management of PCOS [36–38]. Regular exercise, performed 2–3 times per week, appears to enhance gonadotropic axis function, leading to increased menstrual regularity and ovulation. These improvements translate into better fertility outcomes, with a reported pregnancy rate of 35% in the exercise group compared with 10% in the diet-only group [39], which may be a strong source of motivation to engage in PA. From a cardiometabolic perspective, exercise interventions produce small improvements in body weight, moderate reductions in central adiposity and moderate-to-large gains in cardiorespiratory fitness and quality of life, with larger effects observed at higher training intensities [37,38,40,41]. Exercise also contributes to better carbohydrate metabolism, including improved insulin sensitivity and lower insulin concentrations, although effects on fasting glucose and insulin resistance (Homeostatic Model Assessment of Insulin Resistance) are mixed, possibly because of normal baseline values in some populations studied. Exercise may lower total and low-density lipoprotein cholesterol, with less consistent effects on high-density lipoprotein cholesterol and triglycerides, and modest reductions in systolic blood pressure have been reported, although results vary across studies [37,38,42–44]. Importantly, exercise also exerts a large anti-inflammatory effect in women with PCOS, consistently reducing key inflammatory markers while also improving anti-inflammatory cytokine profiles [45–47]. Together, these interconnected adaptations reinforce exercise as a cornerstone intervention for managing chronic inflammation and improving overall cardiometabolic health in PCOS.

In addition to its physiological benefits, exercise exerts a favourable influence on the psychological health of women with PCOS, potentially mediated by concurrent improvements in weight, self-perceived health and overall quality of life [48,49]. Current PA recommendations for women with PCOS are consistent with those for the general population [35]; however, recent evidence suggests that achieving at least 120 minutes of vigorous-intensity activity per week may be required to optimize health outcomes [38]. This target may be difficult to attain in a population that tends to exhibit lower levels of PA.

5. Endometriosis

Endometriosis is a chronic gynaecological condition affecting about 10% of women, characterized by symptoms such as dysmenorrhoea, dyspareunia, chronic pelvic pain and gastrointestinal disorders, which significantly impair women's quality of life [50] (Fig. 3). Endometriosis is also widely recognized as a chronic inflammatory disease, characterized by the presence of endometrial tissue outside the uterus, leading to

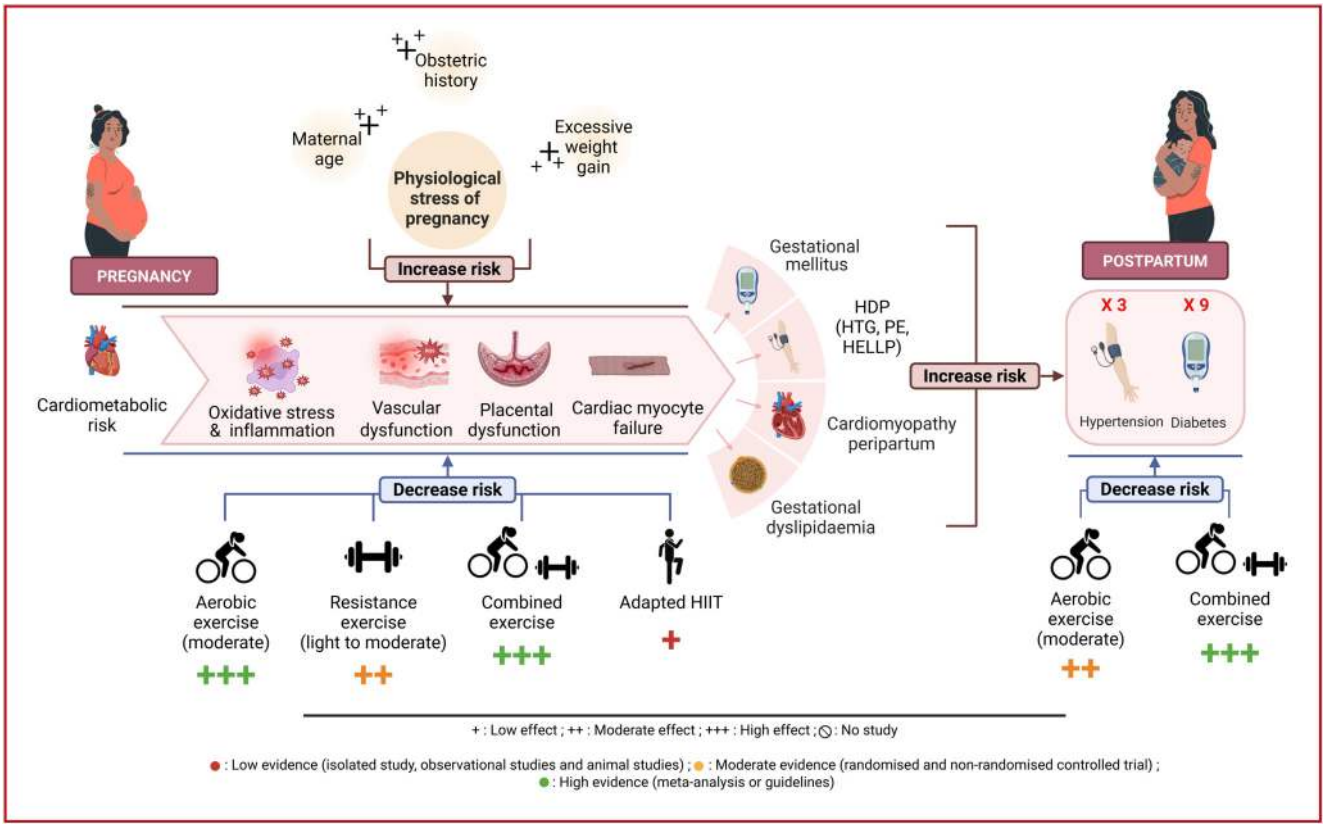


Fig. 4. The role of physical activity in reducing the cardiovascular risk associated with perinatality. DT2: type 2 diabetes; HDP: hypertensive disorders of pregnancy; HELLP: haemolysis, elevated liver enzymes and low platelets; HIIT: high-intensity interval training; HTA: arterial hypertension; HTG: gestational hypertension; PE: pulmonary embolism.

both systemic and local inflammation [51]. This inflammatory state is marked by elevated levels of proinflammatory biomarkers such as CRP, interleukin-17 and interleukin-33, which may initiate or promote atherosclerosis and thereby increase the risk of CVD [52]. Consequently, several epidemiological studies have reported an association between endometriosis and cardiovascular events and diseases, showing an increased risk of ischaemic cardiomyopathy (HR 1.43, 95% CI 1.28–1.59 to HR 1.50, 95% CI 1.37–1.65) and stroke (HR 1.17, 95% CI 1.07–1.29 to HR 1.20, 95% CI 1.11–1.30), probably related to atherosclerosis induced by chronic inflammation [53,54]. In addition, a higher risk of developing hypertension (relative risk [RR] 1.14, 95% CI 1.09–1.18) and hypercholesterolaemia (RR 1.25, 95% CI 1.21–1.30) has also been observed, possibly reflecting the effects of chronic inflammation on lipid metabolism and vascular function [55].

Therefore, PA represents a key strategy to counteract systemic inflammation, improve vascular health and ultimately reduce cardiovascular risk in women with endometriosis [56]. However, to date, very few studies have explored this non-pharmacological intervention for this purpose. Preclinical studies in rats have shown that moderate to high-intensity PA can reduce the size of endometriotic lesions, whereas high-intensity interval training associated with pentoxifylline can decrease inflammatory markers and improve the lipid profile [57–59]. In addition, data indicate that lower endometriosis severity in women is associated with higher PA levels and lower systemic inflammation, suggesting that PA may act as a potential cofactor [60]. Although several studies have shown that PA can improve quality of life and reduce pain in women with endometriosis [61], they are found to be less physically active than those without the condition [62]. These data highlight the need for targeted interventions and supportive programmes to encourage regular PA in women with endometriosis. In addition, clinical studies are needed to clarify the impact of regular PA and specific exercise modalities on disease progression, cardiovascular health and inflammatory outcomes.

6. Pregnancy complications

Pregnancy imposes significant physiological stress on the body, requiring substantial adaptative responses from the maternal cardiovascular system from the first trimester (Fig. 4). These adaptations involve structural and functional changes, such as the reduction of peripheral arterial resistance concomitant with an increase in cardiac output, aimed to ensure optimal haemodynamic conditions for fetal development [63]. Whereas healthy women adapt to these cardiovascular changes without difficulty, pregnancy can reveal pre-existing subclinical cardiovascular dysfunction. Approximately 10–15% of pregnant women develop hypertensive disorders of pregnancy, which are associated with a fourfold increase in the risk of maternal morbidity and a twofold increase in the risk of maternal mortality [64,65], with CVD being the leading cause of maternal death during the perinatal period [66]. Beyond the immediate complications, such as pre-eclampsia, HELLP syndrome (haemolysis, elevated liver enzymes and low platelets) and peripartum cardiomyopathy, there is strong evidence that women with a history of hypertensive disorders of pregnancy are at an increased risk of developing long-term CVD. In particular, their risk of developing hypertension is approximately three times higher, associated with an elevated risk of stroke [64–70]. It is also noteworthy that gestational diabetes, which affects 15% of pregnancies, increases the risk of new-onset type 2 diabetes within 15 years by up to ninefold [70]; it is further associated with a markedly elevated long-term risk of CVD (RR 1.98. 95% CI 1.57–2.50), even among women who do not develop diabetes [71].

The perinatal period (pregnancy and postpartum) is therefore an early indicator of future cardiovascular risk, highlighting the

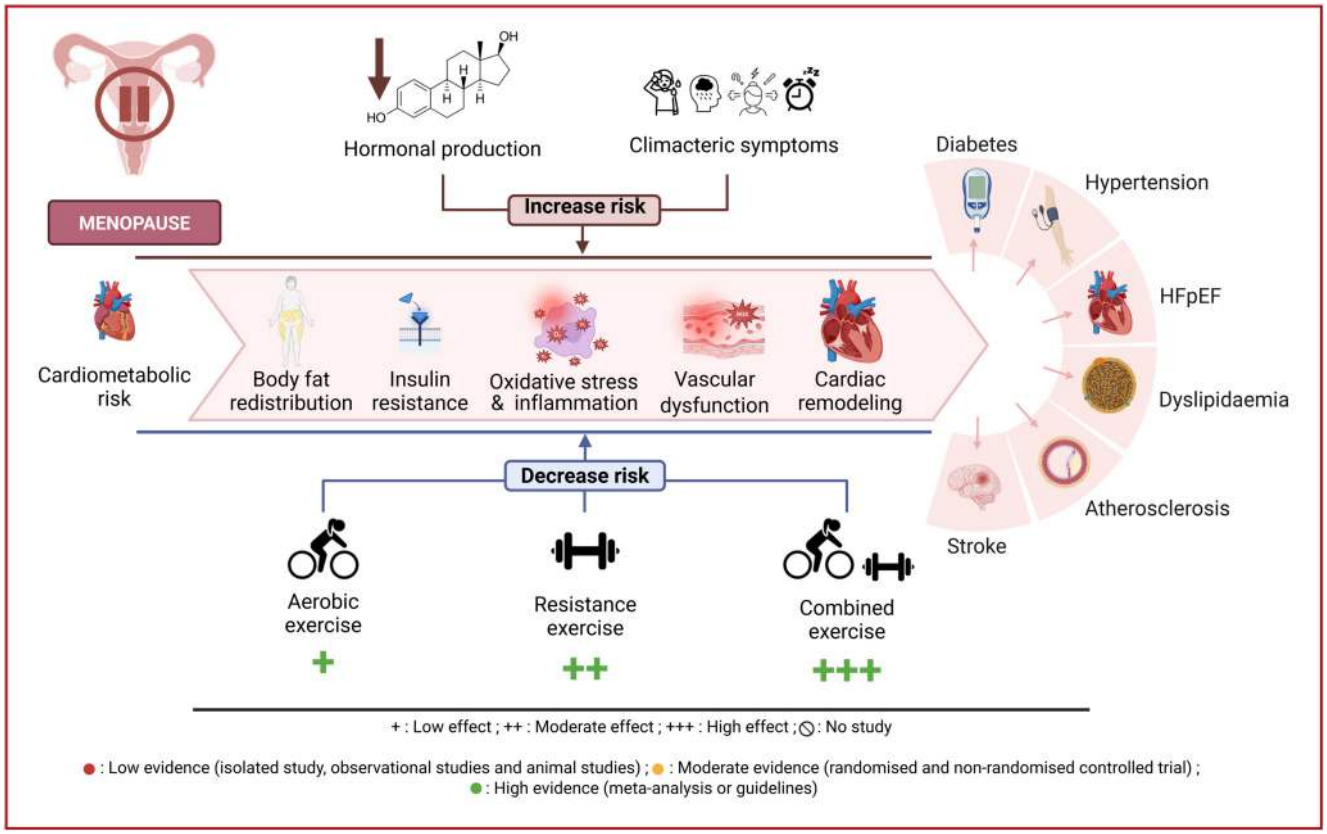


Fig. 5. The role of physical activity in reducing the cardiovascular risk associated with menopause. HFpEF: heart failure with preserved ejection fraction.

importance of appropriate screening and management, as well as the promotion of PA to support cardiovascular health. When performed in accordance with established guidelines during pregnancy, PA constitutes a safe and effective strategy for maintaining/restoring cardiometabolic health [72–74]. Indeed, PA has been shown to significantly reduce the risk of pre-eclampsia (odds ratio [OR] 0.59, 95% CI 0.37–0.94 if practiced before conception), gestational hypertension (OR 0.61, 95% CI 0.43–0.85 to OR 0.53, 95% CI 0.40–0.71) and gestational diabetes (OR 0.62, 95% CI 0.52–0.75 to OR 0.61, 95% CI 0.51–0.74), especially in women of normal weight. These effects are enhanced when practised regularly, supervised by a physical exercise professional and started early [72,75]. These clinical benefits are linked to improvement in endothelial function, optimized lipid profile, enhanced insulin sensitivity and reductions in oxidative stress and systemic inflammation [76,77]. Given these benefits, obstetrics and gynaecology societies recommend at least 150 minutes of moderate-intensity PA per week, distributed over a minimum of three sessions, and primarily consisting of aerobic activities, such as brisk walking [78]. Although most studies have focused on the effects of moderate-intensity exercise, evidence also suggests that higher-intensity activity can be safe [79]. To help women follow these guidelines safely during pregnancy, the self-administered “Get Active Questionnaire for Pregnancy”, developed by the Canadian Society for Exercise Physiology, is a simple and validated tool for identifying the rare absolute or relative contraindications to PA [80]. After childbirth, PA is particularly beneficial for women with a history of gestational complications, as it helps to reverse persistent physiological alterations by enhancing vascular function, reducing residual arterial stiffness and restoring a more balanced metabolic profile, thereby limiting the risk of progression to chronic diseases [70,81,82].

Although the safety and effectiveness of PA during pregnancy and postpartum are well established, several knowledge gaps remain to optimize intervention strategies. In particular, the effects of exercise in pregnant women with chronic hypertension remain unknown, despite its well-established benefits in other hypertensive populations [83]. Similarly, neither the risk of recurrence of hypertensive disorders of pregnancy nor the long-term evolution of cardiovascular markers in this context has been thoroughly evaluated [84].

7. Menopause

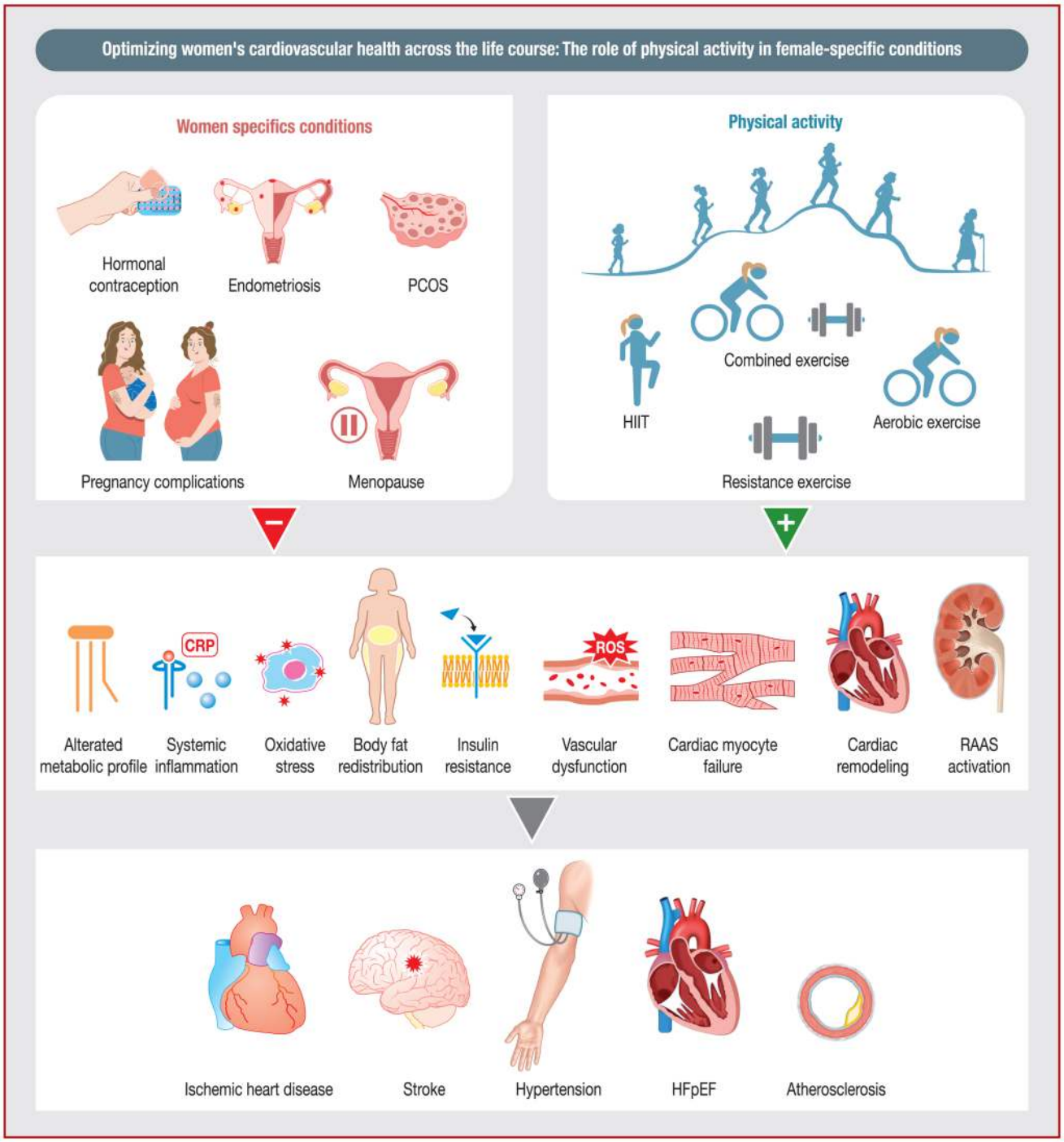
Menopause is a natural stage in a woman’s aging process, typically occurring at an average age of 50 years (range: 48–53 years), with variations influenced by ethnicity and geographic location [85] (Fig. 5). Natural menopause involves the cessation of ovarian oestrogen (17β-oestradiol) production, which leads to increased gonadotropin secretion as a result of the loss of hypothalamic-pituitary negative feedback, while ovarian testosterone production persists, significantly shifting the androgen-to-oestrogen ratio [86]. This hormonal change contributes to both physiological and psychological disturbances, including metabolic and vascular dysfunction, thereby increasing cardiovascular risk. In the initial years following menopause, a redistribution of body fat occurs, marked by a significant increase in visceral fat and a decrease in fat accumulation in the lower limbs [87]. Menopause is associated with an increased risk of metabolic alterations, including dyslipidaemia and higher insulin resistance [88], as well as vascular alterations, such as increased arterial stiffness [89], impaired endothelial function [90] and higher blood pressure levels [91]. Moreover, the decline in oestrogen promotes a proinflammatory state and increases oxidative stress, which may accelerate the development of atherosclerosis, further exacerbating endothelial dysfunction and contributing to the progression of CVD [92]. In addition, oestrogen deficiency induces adverse cardiac remodelling characterized by left ventricular hypertrophy, increased myocardial stiffness and diastolic dysfunction, thereby predisposing postmenopausal women to heart failure with preserved ejection fraction [93]. Alongside cardiometabolic alterations, menopause is characterized by climacteric symptoms affecting most women [94]. Several

studies have reported an association between vasomotor symptoms (hot flashes and night sweats) and increased risk of CVD (RR 1.54, 95% CI 1.27–1.86) and stroke (RR 1.43, 95% CI 1.07–1.92), which may be further aggravated by higher frequency and severity of vasomotor symptoms. Other symptoms, including anxiety-depressive disorders and sleep disturbances, are also linked to elevated CVD risk (RR 1.51, 95% CI 1.12–2.02) [95,96]. To alleviate climacteric symptoms, menopausal hormone therapy is recommended in women at low cardiovascular risk. Beyond symptom management, studies have suggested potential cardioprotective benefits in women aged < 60 years, within the 10 years after menopause, particularly when using the transdermal route. Nevertheless, the overall risk-benefit profile of menopausal hormone therapy is complex, and warrants individualized risk-benefit assessment [97,98].

Overall, PA should be considered as an important ally in limiting the increase in cardiovascular risk associated with menopause and managing climacteric symptoms [99]. However, the menopausal transition has been reported to be associated with a decrease in PA levels in 38% of women [100], with lower leisure-time PA participation observed in those experiencing moderate-to-severe symptoms [101]. Despite this decline, structured PA programmes have been shown to improve various cardiometabolic outcomes in postmenopausal women. Resistance, aerobic and combined training improve body composition [102], whereas high-interval aerobic training specifically decreases fat mass, including visceral fat [103]. In addition, PA reduces systolic and diastolic blood pressure, with combined or resistance training being effective, but aerobic training alone appearing less effective [104]. These reductions can be attributed to the effect of PA on arterial stiffness [105] and endothelial function [106], with combined training providing the greatest benefits. Vascular improvements are particularly observed when PA is initiated during the early postmenopausal period–a critical time window where vascular responsiveness to lifestyle interventions appears to be more favourable [107]. Moreover, several studies have highlighted the potential additive effect of menopausal hormone therapy and aerobic training on vascular health, especially in enhancing endothelial function [108]. Finally, PA further exerts beneficial effects on metabolic syndrome components, with moderate-intensity and combined training reducing blood triglyceride and glucose concentrations, and aerobic or resistance training increasing high-density lipoprotein cholesterol [109]. As a result of these physiological and metabolic benefits, regular PA has been shown to reduce cardiovascular risk, with the Women's Health Study reporting a 16–36% lower risk of CVD for ≥ 500 kcal/week of PA (RR 0.64, 95% CI 0.53–0.77) for the group at highest risk of CVD, after adjustment for other traditional risk factors [110].

8. Conclusions

This narrative review highlights the pivotal role of PA in managing cardiovascular risk in women by addressing five key female-specific risk factors: OC, PCOS, endometriosis, pregnancy complications and menopause. Evidence suggests that regular exercise favourably influences the metabolic, vascular and inflammatory pathways involved in these conditions. By acting on these key pathophysiological mechanisms, exercise emerges as a highly relevant and integrative strategy for cardiovascular prevention across the different stages of a woman's life. Nonetheless, future research should prioritize sex-specific exercise trials with cardiovascular endpoints and mechanistic biomarkers to better inform personalized prevention strategies in women (Central Illustration).



Central Illustration. Conceptual overview of how female-specific conditions across the life course influence cardiovascular health via key pathophysiological pathways (e.g., inflammation, oxidative stress, vascular dysfunction), and how physical exercise may attenuate cardiovascular disease risk. HFpEF: heart failure with preserved ejection fraction; HIIT: high-intensity interval training; PCOS: polycystic ovary syndrome; RAAS: renin-angiotensin-aldosterone system.

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Disclosure of interest

The authors declare that they have no competing interest.

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