International Journal of Women's Health

Dovepress
Taylor & Francis Group

Open Access Full Text Article

REVIEW

A Review of Traditional and Non-Traditional Cardiovascular Risk Factors in Women

Sheran Vasanthakumar[1,2], Sarang Paleri[1], Emma Aldous[1], Shaun Khanna[2,3], Rhea Nandurkar[1], Esther F Davis[1], Andrew Lin[1,2], Nitesh Nerlekar[1,2]

[1]Department of Cardiology, Victorian Heart Hospital, Clayton, VIC, Australia; [2]Department of Cardiology, Baker Heart and Diabetes Institute, Melbourne, VIC, Australia; [3]Cardiovascular Program, The George Institute for Global Health, UNSW, Sydney, NSW, Australia

Correspondence: Sheran Vasanthakumar, Victorian Heart Hospital, 631 Blackburn Road, Clayton, VIC, 3168, Australia, Tel +61400894500, Email sheran.vasanthakumar@monash.edu

Abstract: Cardiovascular disease (CVD) remains a significant cause of morbidity and mortality amongst women. While our general understanding of evaluation and treatment of CVD has greatly improved over the past decades, there is a gap in the knowledge of female specific CVD risk factors and cardiac health. Young women in particular are often unaware of their cardiovascular risk and have experienced a smaller reduction in CVD risk over time as compared to other demographics. In recent years, the variation in traditional risk factors and the identification of non-traditional risk factors have been studied as a potential avenue to identify and better address this risk. Non-traditional risk factors such as pregnancy-related hypertensive and glucose disorders have been implicated in CVD risk and may benefit from assessment. Furthermore, novel risk factors such as breast arterial calcification may provide new avenues in which to evaluate patients. While a number of variables have been identified as potential female-specific CVD risk factors, it is still unclear how this can aid risk prediction and therefore our management of CVD in women. Our review aims to summarize the current knowledge of traditional and non-traditional CVD risk factors in women and how this may implicate future management of CVD in women.

Keywords: cardiovascular diseases, women's health, sex factors, risk factors, pregnancy complications, risk assessment

Introduction

Cardiovascular disease (CVD) is a leading cause of mortality worldwide. Despite advances in the identification and management of at-risk individuals, there is limited awareness, particularly among healthcare providers and young women, of sex-specific risk factors and atypical presentations in female patients. Women often exhibit distinct symptom profiles and pathophysiological patterns of CVD in comparison to men, yet these differences are not adequately captured by widely used risk prediction models.

Typical risk models incorporate traditional risk factors such as diabetes, hypertension, hyperlipidemia, smoking status and age. However, these variables manifest and impact women differently across the lifespan. In addition, cardiovascular risk in women is influenced by factors that fall outside of conventional models that can be broadly categorized as non-traditional factors. Female-specific risk factors represent a subset of non-traditional risk factors that arise from sex-specific biology and include conditions such as pre-eclampsia, polycystic ovarian syndrome and breast arterial calcification (Figure 1). The discrepancy between traditional and non-traditional risk factors is especially evident in younger women who may have elevated risk in the absence of conventional risk factors.

Recent years have seen growing interest in refinement of cardiovascular risk assessment to better reflect the nuances of sex-specific risk in women. In this review, we examine the current evidence on both traditional and non-traditional risk factors as they relate to women and how this evolving area informs clinical risk prediction and prevention strategies.

https://doi.org/10.2147/IJWH.S574244
Received: 14 October 2025
Accepted: 28 January 2026
Published: 28 February 2026

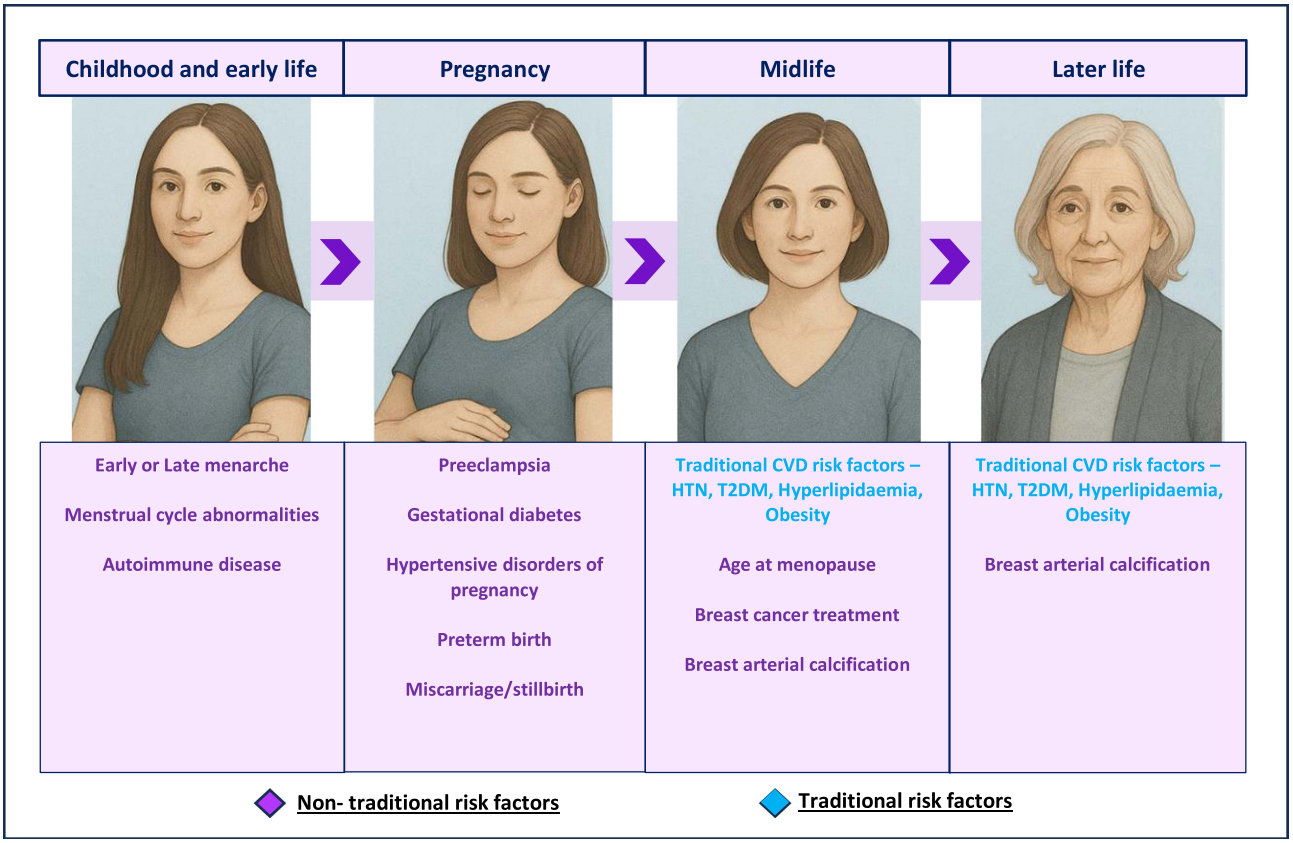

Figure 1 Visualization of traditional and non-traditional risk factors across the lifespan for women. Non-traditional risk factors, on average, are evident at an earlier age and precede a change in cardiometabolic phenotype, making them ideal as a risk marker for early identification and intervention of cardiovascular risk.

Scope of the Issue

Cardiovascular disease, particularly coronary heart disease, remains one of the major contributors to both mortality and morbidity in women. While men tend to experience CVD earlier in life, women often exhibit a delayed onset with incidence rates rising substantially in midlife and beyond.[1] A 2021 global commission reported a general decline in the prevalence of CVD since 1990, although the rate of decline has begun to slow in recent years. Notably, several regions – including parts of Asia, Africa and Oceania – have seen an increase in CVD prevalence during the same period.[2]

In high-income countries, age-stratified data reveals a concerning trend among women. A Canadian cohort study found that while overall CVD rates have declined, reduction in young women, aged 20 to 49, has remained the slowest.[3] Furthermore, younger women with ST elevation myocardial infarction (STEMI) experience higher mortality rates than their male counterparts.[4] This paradox of lower prevalence of traditional risk factors in young women but poorer outcomes raises important questions about the adequacy of current risk prediction in this population.

A key contributing factor may be limited awareness of sex-specific presentation and risk factors for CVD, both among the general public and healthcare professionals. Despite several public health campaigns focused on CVD in women, recent data shows that awareness of heart disease as the leading cause of death in women has declined over the past decade. Furthermore, one-third of post-graduate medical trainees report minimal or no training in sex-specific medicine and fewer than half of cardiologists feel adequately prepared to assess cardiovascular risk in women.[5] Bridging these gaps in awareness, education and clinical assessment is essential to improving cardiovascular outcomes and achieving equity in prevention and treatment strategies.

Traditional Risk Factors

Diabetes

Diabetes is a well-established independent risk factor for both cardiovascular disease and cardiovascular related mortality.[6] Importantly, its impact appears to be disproportionately greater in women, associated with both an elevated mortality risk and an earlier onset of cardiovascular disease.[7–9] One of the largest pooled analyses to date by Wang et al, encompassing 49 studies and over five million patients found that compared to men, women with diabetes had a 58% increased risk of coronary heart disease mortality and a 13% increased risk of all-cause mortality (Table 1). [10–24] They identified a pooled adjusted relative risk ratio for cardiovascular disease mortality in women as compared to men of 1.30 (95% CI 1.13–1.49, $p < 0.001$) (Figure 2).[10]

Table 1 Meta Analysis and Pooled Risk Ratios for Specific Cardiovascular Risk Factors and Cardiovascular Outcomes in Women

Study	N	Risk Factor	Risk ratio	Adjustment
*Wang et. al (2019)[12]	1,148,188	Diabetes	CHD – 2.79 (2.25–3.46) CVD mortality – 2.75 (1.88–4.01)	Partial: CVD risk factors
*Wang et al (2019)[10]	5,162,654	Diabetes	CHD mortality – 3.16 (2.61–3.82) CVD mortality – 2.42 (2.10–2.78)	Full: Age Partial: smoking, HTN, BMI
*Peters et al (2014)[20]	858,507	Diabetes	CHD – 2.63 (2.27–3.06) CHD mortality – 2.83 (2.25–3.54)	Full: Age Partial: CVD risk factors
**Lee et al (2000)[21]	75,000	Diabetes	CHD mortality – 2.58 (2.05–3.26)	Full: Age Partial: CVD risk factors
*Mongraw-Chaffin et al (2015)[25]	1,219,187	Per 1 kg/m2 increase in body mass index	CHD – 1.04 (1.05–1.05)	
*Peters et al (2016)[26]	1,022,276	Per 1 mmol/L increase in cholesterol	CHD – 1.20 (1.16–1.24)	Partial: Maternal age, socioeconomic status, obstetric history and chronic diseases
*Lin et al (2022)[27]	535,263	Per 10mmHg increase in systolic blood pressure	CVD – 1.19 (1.13–1.26)	Partial: CVD risk factors
*Huxley et al (2011)[11]	3,912,809	Smoking	CHD – 1.92 (1.66–2.23)	Partial: CVD risk factors
Kramer et al (2019)[18]	5,390,591	GDM	CVD – 1.98 (1.57–2.50)	Partial: CVD risk factors
***Li et al (2018)[15]	3,417,020	GDM	CHD – 1.59 (1.30–2.08) CVD – 1.95 (1.83–2.08)	Full: Mean age at pregnancy, data collection methods, sample size, GDM diagnostic criteria
Wekker et al (2020)[22]	286,754	PCOS	Non-fatal CHD – 1.78 (0.99–3.23) CHD mortality – 14.48 (0.14–15,83) CVD mortality – 1.30 (0.62–2.74)	Partial: CVD risk factors
***Tay et al (2023)[28]	1,062,280	PCOS	CVD – 1.68 (1.26–2.23) CHD composite – 1.48 (1.07–2.05) CVD mortality – 1.19 (0.53–2.69)	Partial: CVD risk factors, ethnicity
Wan et al (2024)[29]	74,824,979	PCOS	CVD – 1.51 (1.36–1.91)	Partial: CVD risk factors
Wu et al (2017)[17]	6,456,379	Preeclampsia	CHD – 2.50 (1.43–4.37) CHD mortality – 2.10 (1.25–3.51) CVD mortality – 2.21 (1.83–2.66)	Partial: CVD risk factors and obstetric history
Mcdonald et al (2008)[23]	2,375,751	Preeclampsia	CVD mortality – 2.29 (1.73 −3.04)	Full: Age Partial: CVD risk factors
Brown et al (2013)[24]	2,010,656	Preeclampsia	CVD – 2.28 (1.87–2.77)	Not mentioned
Bellamy et al (2007)[16]	3,488,160	Preeclampsia	CHD – 2.16 (1.86–2.52)	Partial: Socioeconomic status, CVD risk factors

(*Continued*)

Table 1 (Continued).

Study	N	Risk Factor	Risk ratio	Adjustment
Inversetti et al *(2024)*[30]	13,162,030	Preeclampsia	CVD mortality – 2.08 (1.70–2.54) CHD – 2.04 (1.76–2.38)	Partial: Socioeconomic factors, CVD risk factors
Galimzhanov et al *(2023)*[19]	836,201	Breast cancer	CVD mortality – 1.09 (1.07–1.11) CHD – 0.97 (0.90–1.02)	Partial: Age, socio economic factors, CVD risk factors
***Osman* et al *(2022)*[13]	33,494	BAC	CHD – 2.39 (1.68–3.41)	Partial: Age, CVD risk factors
Lo et al *(2020)*[14]	3,601,192	GHTN	CHD – 1.83 (1.33–2.51) CVD – 1.81 (1.41–2.32)	Partial: CVD risk factors

Notes: Compilation of meta-analyses of specific CVD risk factors in women and CVD/CHD event rate or mortality. Adjusted confounders for each study are given as full, where included in all studies or partial, where included in part. Adjustment for CVD risk factors refers to one or more traditional CVD risk factor.[10–24] * – Cohort included male and female participants (matched or unmatched), ** – Approximate cohort number (exact not available), *** – Odds ratio used as a surrogate for relative risk in setting of low event rate in population.
Abbreviations: CHD, coronary heart disease; CVD, cardiovascular disease.

Several mechanisms have been proposed to explain this sex disparity. Earlier studies suggested that women develop diabetes at a more advanced stage of metabolic dysregulation, presenting with a higher burden of cardiometabolic risk factors than men.[6] Additionally, the relative cardio-protection observed in premenopausal women without diabetes appears to be lost upon diabetes onset.[8] Treatment disparities may also play a role; women with diabetes have been shown to receive less aggressive risk factor modification and achieve poorer glycemic, lipid and blood pressure control.[31–33] This raises a second possibility, that the difference is attributable to less aggressive treatment of diabetes and metabolic syndrome in women. These findings suggest that both biological differences and clinical inertia may contribute to the observed sex difference in risk.

Given its major role in the pathogenesis of coronary artery disease, diabetes should be rigorously screened for and aggressively managed in all women.

Body Mass Index

Body mass index (BMI) and body composition, while appearing simple on the surface, in reality has a complex relationship with cardiovascular disease. Elevated BMI has been linked to increased CVD outcomes. A meta-analysis of over 1 million participants demonstrated each increment of 1 kg/m^2 in BMI associated with a hazard ratio of 1.04 (1.03–1.05) of incident coronary heart disease in women.[25] This would advocate the hypothesis that increase weight and/or body fat is associated with CVD. However, body composition appears to have an interesting impact on CVD risk in women – high muscle and high fat mass is associated with a lower CVD mortality, while high muscle and low-fat mass is not. In comparison, men demonstrate a lower CVD mortality with high muscle mass irrespective of fat mass.[34] The distribution of body fat itself is also predictive, with higher truncal fat in women being associated with elevated CVD risk and higher leg fat associated with reduced CVD risk.[35] Cardiometabolic disease correlates with elevated visceral adipose tissue and the elevated risk from truncal adiposity may be a reflection of this. In premenopausal women, estrogen drives lower body rather than truncal and visceral fat deposition with a reversal in this pattern post menopause.[36] While obesity is considered a marker of cardiovascular health, there are sex-based differences and perhaps a different optimal phenotype to aim for.

Smoking

Smoking represents one of the most significant and modifiable risk factors for cardiovascular disease.[37] While smoking prevalence among women has declined in many high-income countries, it continues to contribute substantially to preventable morbidity and morbidity worldwide, particularly in younger women and in low and middle income regions.[38,39]

A meta-analysis involving over two million participants reported that women who smoke have a 25% greater relative risk of coronary heart disease events compared to their male counterparts.[11] Beyond cardiovascular disease, smoking

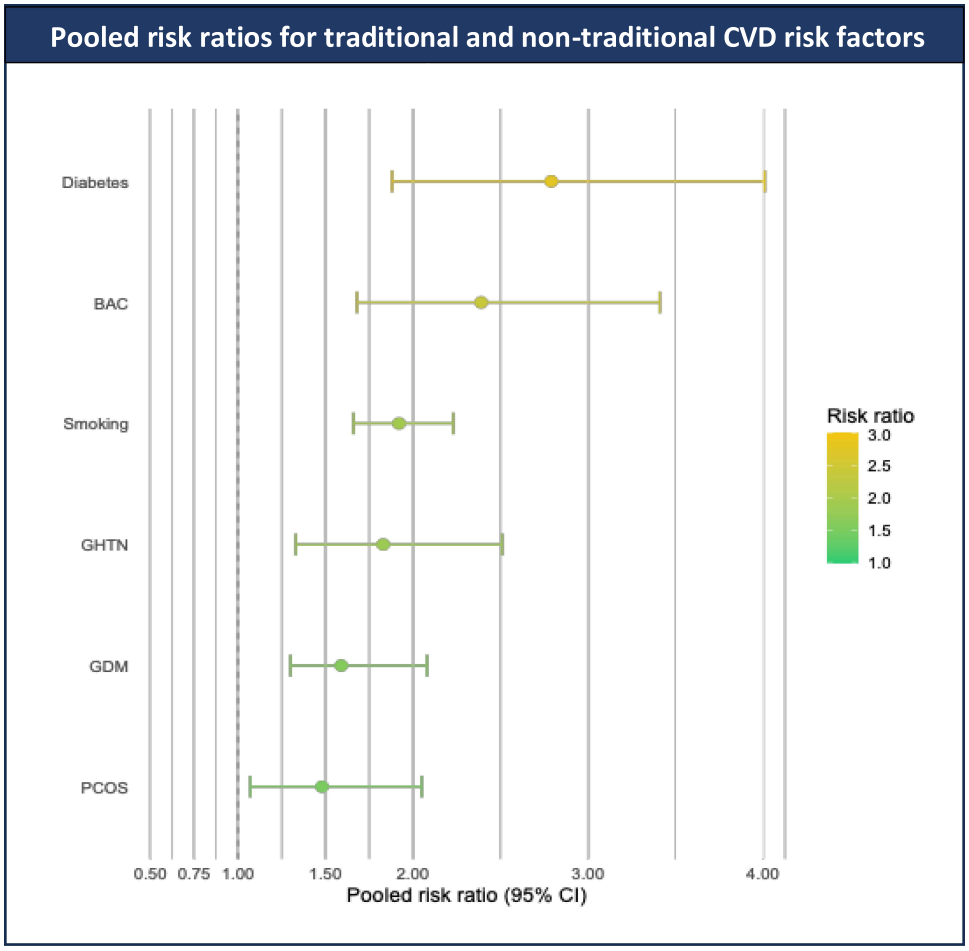

Figure 2 Risk ratios for traditional and non-traditional risk factors, demonstrating low (green), moderate (yellow), high (Orange) and red (very high) increased risk of CHD events. All results are pooled risk ratios from large meta-analyses (see. Table 1). Note the end point for PCOS was a composite of CHD events that included elective admissions and may result in an elevated risk.[11–15,28]

Abbreviations: CHD, coronary heart disease; CVD, cardiovascular disease; BAC, breast arterial calcification; GHTN, gestational hypertension; GDM, gestational diabetes mellitus; PCOS, polycystic ovarian syndrome.

increases the risk of several cancers and is associated with adverse pregnancy outcomes, including fetal growth restriction and preterm birth, enforcing the importance of encouraging smoking cessation.

One proposed explanation for the heightened cardiovascular risk in women is the anti-estrogen effect of smoking. Estrogen is known to exert a protective effect on vascular endothelium and lipid metabolism. Smoking appears to disrupt estrogen bioactivity and elevate blood androgen levels, potentially accelerating vascular damage and attenuating the benefits of estrogen in premenopausal women.[40–42]

Hypertension

Hypertension is a prevalent risk factor for cardiovascular disease (CVD) in both men and women, however, emerging evidence suggests that blood pressure impacts cardiovascular risk differently across sexes.[27] In a moderate-sized Iranian cohort of 7710 participants, predictors of hypertension at diagnosis differed by sex, with age common to both, while contraceptive use, pre-hypertension and kidney stones significant only in women.[43] The INTERHEART study showed that hypertension was amongst a group of risk factors that elevated the risk of myocardial infarction more significantly in younger women than in older women.[9] In one of the largest cohorts, with over 27,000 participants, examining sex differences in blood pressure, Ji et al observed that while hypertension increased CVD risk in both sexes, women exhibited elevated risk at a lower systolic blood pressure threshold.[44] Additionally, research indicates that while

premenopausal women have a lower average blood pressure than men, this gap diminishes significantly post menopause.[45]

Currently, hypertension guidelines are not sex-specific. However, given the observed differences in blood pressure patterns and associated cardiovascular risks between sexes, there is a compelling need for research to inform potential sex-specific recommendations and therapeutic targets. Kringeland et al found that in individuals in their early forties, stage 1 diastolic hypertension was more strongly linked to the development of acute coronary syndrome in women compared to men, suggesting that young women may benefit from more aggressive treatment of diastolic hypertension.[46] Amongst older women, perimenopausal women exhibit significant blood pressure variability and postmenopausal women are more prone to non-dipping nocturnal blood pressure patterns, both of which are associated with increased cardiovascular risk.[47] These observations underscore the necessity for sex-specific research to understand the underlying mechanisms of blood pressure regulation in women and to develop tailored management strategies.

Hyperlipidemia

Dyslipidemia is a widely recognized risk factor for cardiovascular disease and a key therapeutic target. On average, a 1-mmol/L increase in total cholesterol corresponds to a similar risk in women (1.20 RR, 1.16–1.24 95% CI) as compared to men (1.24 RR, 1.20–1.28) of coronary heart disease.[26] However, in women, lipid profiles vary considerably across life stages, influenced by hormonal fluctuations and should be a consideration in interpreting and managing lipid disorders. In reproductive-age women, total cholesterol and low-density lipoprotein cholesterol (LDL-C) levels tend to rise during the follicular phase and fall in the luteal phase, while high-density lipoprotein cholesterol (HDL-C) peaks during ovulation. The fall in LDL from follicular to luteal phase varies between 5% and 10%, while the fluctuation in HDL has been noted anywhere between 11% and 49%.[48] Early menarche has been associated with adverse lipid profiles, such as elevation of both triglycerides and LDL-C.[48] Oral contraceptives can further modify lipid metabolism, raising triglycerides and HDL-C while reducing LDL-C, depending on the estrogen potency of the pill.[49] Pregnancy also induces a physiologic rise in cholesterol and triglycerides, though its long term cardiovascular implications remain unclear.[49]

Evidence suggests sex disparities in the management of dyslipidemia. Women with familial hypercholesterolemia are diagnosed later, receive less intensive lipid lowering therapy and were less likely to achieve guidelines recommended lipid targets.[50] Despite almost equal efficacy of lipid-lowering therapy in reducing cardiovascular events in both sexes, women are less likely to be offered therapy.[51] Statin discontinuation is also more common in women, which may be related to both clinician- or patient-related factors.[52,53] Potential reasons include a lack of awareness of CVD risk in women, both from the patient and practitioner, and an elevated rate of drug-related adverse events or intolerance. This is suggested by the literature which demonstrates a high rate of musculoskeletal side effects cited by women as a reason for cessation of lipid-lowering therapy.[54]

In recent years, lipoprotein(a) has emerged as a novel biomarker of atherosclerotic risk, particularly for premature coronary artery disease. Elevated Lp(a) levels are independently associated with increased CVD risk across both general and high-risk populations.[55] Some studies have shown a correlation between Lp(a) levels and coronary artery disease amongst women.[56–58] However, in a pooled analysis of the Women's Health Study, Women's Health Initiative and JUPITER trials, the incremental value of Lp(a) in risk prediction for women was limited.[59] Ongoing research is needed to clarify its role in sex-specific CVD risk assessment.

Age

Cardiovascular disease has traditionally been regarded as a condition of aging and remains an independent predictor of cardiovascular events.[60] The contribution of age may in part reflect the cumulative prolonged exposure to risk factors such as hypertension and dyslipidemia.[61,62] Notably, in women, the rise in cardiovascular risk with age appears steeper than in men. A Finnish cohort study of 14,000 men and women found that age-related increases in coronary heart disease incidence and mortality were more pronounced in women. The role of CVD risk factors in increasing CHD risk was similar in both sexes, and the overall risk factor level was lower in women. However, risk began to plateau at 45 to 50 in men, whereas in women risk continues to sharply increase until 60 to 65.[63]

The disparity is particularly evident during menopause, when withdrawal of estrogen is associated with a sudden increase in cardiovascular risk. Estrogen has well-documented vasoprotective effects, including favorable modulation of lipids and vascular function. Although hormone replacement therapy (HRT) was initially hypothesized to mitigate CVD risk during the perimenopausal period, large randomized trials such as those in the Women's Health Initiative (WHI) demonstrated contradictory results to previous observational studies by showing an increase in vascular events with HRT.[64,65] This discordance is still a matter for debate; however, it may be related to a higher proportion of younger women in early menopause who enrolled in observational studies. Recent evidence suggests that there is a potential benefit in a younger subgroup but without a large enough signal to advocate change in clinical practice.[66,67]

The transition from low to elevated risk following menopause may contribute to lower awareness and under-recognition of cardiovascular disease and risk factors in women that are pre-menopausal. This underscores the importance of assessing cardiovascular risk throughout the lifespan in women.

Non-Traditional Risk Factors

While many non-traditional risk factors for cardiovascular disease in women are supported primarily by observational evidence, emerging data suggests their cardiovascular effects may converge on shared biological pathways. These include endothelial dysfunction, chronic low-grade inflammation, metabolic dysregulation, vascular remodeling and microvascular disease. These are all processes that may be differentially modulated by sex hormone exposure.[68] Importantly, for several female-specific risk factors, mechanistic evidence remains incomplete and association should be interpreted as markers of heightened susceptibility rather than direct causal pathways.

Pregnancy

Several complications during pregnancy are now recognized as sex-specific risk enhancers for long-term maternal cardiovascular disease.[69] Risk enhancers, as defined in the American College of Cardiology (ACC) blood cholesterol management guidelines, are clinical features that complement the established risk models to inform decisions regarding preventative strategies.[70] The most commonly described are preeclampsia, gestational hypertension (GHT) and gestational diabetes (GDM).[16,71,72]

Preeclampsia is theorized to induce metabolic, endothelial and vascular dysfunction and has been associated with a higher frequency of spiral arterial lipid deposition.[73,74] Even after adjustment for traditional risk factors, it remains strongly associated with elevated long-term risk of cardiovascular death, coronary artery disease, heart failure and stroke.[17,30] Large population-based studies have also demonstrated a higher prevalence and earlier onset of subclinical atherosclerosis. Women with a history of preeclampsia exhibit premature and increased development of coronary artery calcification at ages 45 to 50 compared to women without.[75] In addition to this independent risk, affected women are also more likely to develop cardiometabolic risk factors – including hypertension, elevated body mass index and insulin resistance, suggesting a prolonged trajectory of metabolic dysfunction.[76]

GDM and GHTN have both been implicated in the development of chronic diseases such as renal dysfunction, liver disease and cardiovascular diseases.[77–81] A 2019 meta-analysis demonstrated a two-fold increased CVD risk in women with prior GDM, even in those who do not progress to overt type 2 diabetes mellitus (T2DM).[18] In the absence of GDM, hyperglycemia also predicts future CVD and may be part of the spectrum of disease.[79,82] Hypertensive disorders during pregnancy other than preeclampsia are also a significant consideration for future CVD risk given an incidence of 10%.[83] Women with hypertension in a first pregnancy have an increased risk of future hypertension, stroke and ischemic heart disease.[17,84–88] However, the risk associated with GHTN appears to be less pronounced than that of preeclampsia.[14] This observation aligns with the concept that preeclampsia represents a more severe phenotype along the spectrum of hypertensive disorders of pregnancy, characterized by systemic vascular and end-organ involvement. Furthermore, while the elevated CVD risk in both conditions is partially mediated through co-existing cardiovascular risk factors, preeclampsia shows a greater relative risk and a higher proportion of that risk which is not attributable to this.[89]

Other pregnancy outcomes, such as miscarriage, stillbirth and preterm birth, are increasingly recognized as potential predictors of future cardiovascular events. A recent meta-analysis demonstrates that stillbirth and recurrent miscarriage are significantly associated with increased risk of coronary artery disease.[90] Less commonly studied factors, such as

parity and fertility therapy, have shown heterogenous results related to CVD over the past two decades.[91–93] Recent data however, leans towards an absence of a relationship and safety of fertility therapy from a cardiovascular perspective. A cohort of 27,000 women demonstrated an adjusted relative risk of 0.93 (0.82 to 1.05 95% CI) of CVD hospitalization with a history of fertility treatment.[94] In regard to parity, recent long-term outcomes from the WHI did not demonstrate any excess risk at differing parity levels. These results appear to conflict with previous evidence demonstrating an increased risk; however, this may be explained by the age of the first pregnancy. Parity of three to five was associated increased CVD events, but this association was not significant when adjusted for age at first child birth. Furthermore, an age of less than 20 and 20 to 24 at first child birth was associated with a 32.3% and 11.4% elevated risk of CVD, respectively, compared to women who first gave birth at ages 25 to 29.[95] This data suggests that age during first pregnancy rather than parity may be a more relevant CVD risk factor.

In a 2023 Swedish cohort study, of over 2 million women, participants were evaluated for pregnancy-related complications and CVD over a median follow-up period of 22.7 years. Adjusted hazard ratios for cardiovascular mortality were elevated for several pregnancy-related conditions: preeclampsia (HR 2.10; CI 1.47–2.99), gestational hypertension (HR 1.79; CI 1.20–2.66), gestational diabetes (HR 3.03; CI 1.49–6.16), preterm birth (HR 1.84; CI 1.38–2.44), small for gestational age (HR 1.77; CI 1.19–2.64) and stillbirth (HR 3.14; CI 1.81–5.44). Notably, while these results were adjusted for multiple confounders, they did not include dyslipidemia. Nevertheless, these findings underscore the importance of incorporating pregnancy history into cardiovascular risk assessment and better post-natal screening.[96]

Menstrual Cycle

Menstrual cycle abnormalities have recently gained recognition as a sex-specific risk factor for CVD. Menopause has been the most studied area, with estrogen deficiency hypothesized to increase CVD risk.[97,98] However, as noted previously, randomized controlled trials – including the Women's Health Initiative – found that hormone replacement therapy (HRT) does not reduce cardiovascular events. Consequently, current guidelines recommend HRT for symptomatic relief, not for CVD prevention.[99,100]

Menstrual cycle irregularity is increasingly recognized for its links to metabolic dysfunction and is associated with insulin resistance, low-grade inflammation and cardiometabolic syndrome.[101–103] Although results from cohort studies are variable, overall evidence supports a relationship between menstrual irregularity and future cardiometabolic risk.[104] A recent large cohort study demonstrated that both short (<21 days) and long (>35 days) cycle lengths were associated with increased risk of coronary heart disease and atrial fibrillation.[105] History of early or late menarche has also been shown to be associated with CVD risk albeit with a small strength of association. Estrogen exposure does not appear to account for this although there does appear to be a correlation with markers of inflammation.[106]

PCOS

Polycystic ovarian syndrome (PCOS) is a common endocrine disorder in women and is associated with cardiometabolic conditions including insulin resistance, dyslipidemia, obesity and type 2 diabetes.[107] The association between PCOS and CVD varies amongst studies often attributed to the confounding from traditional CVD risk factors. Given its increasing prevalence, PCOS is an important consideration in women's long-term cardiovascular health.[108,109] Three separate meta-analyses have now confirmed an increased risk of both cardiovascular disease and cardiovascular events among women with PCOS, though no clear excess in cardiovascular mortality was observed.[28,29,110] Longitudinal studies have demonstrated that this risk emerges as early as young adulthood and may be independent of body mass index (BMI).[111] Whether PCOS independently contributes to CVD or does so primarily through metabolic dysfunction remains an area of active investigation.

Management of PCOS includes a combination of pharmacological and lifestyle interventions often involving use of combined oral contraceptives (COC).[112] Early studies raised concern for an increased thrombotic risk with COC use and caused caution regarding usage.[113] Over the last decade, studies have had conflicting results regarding the magnitude of thrombotic and CVD risk associated with COC.[114] Recent large-scale cohort data from the United Kingdom found the

use of COC was not associated with an increased risk of CVD and may potentially be protective in patients with PCOS.[115] These findings offer reassurance in the continued use of COC for symptom control when clinically appropriate.

Metformin is also frequently used in PCOS, particularly in women with insulin resistance or metabolic syndrome.[112] Clinical trial evidence of metformin use in patients with PCOS suggests a significant reduction in diastolic blood pressure and total cholesterol.[116] Given the known association between metabolic diseases and PCOS highlights metformin as a therapy of interest outside of traditional indications.

Autoimmune Disease

Although autoimmune diseases (AIDs) are not inherently sex-specific, they are significantly more prevalent in women, comprising up to 80% of diagnosed cases and they represent an important sex-specific cardiovascular risk modifier.[117] These conditions are associated with accelerated atherosclerosis and microvascular dysfunction, likely mediated by chronic inflammation, immune cell activation and subsequent endothelial injury.[118] Cardiovascular events are a known phenomenon in autoimmune disease and often present at a younger age compared to the general population. In a recent cohort study, Restivo et al reported an increased relative risk of symptomatic cardiovascular events with lupus (1.98, 95% CI 1.18–3.31) and rheumatoid arthritis (1.55, 95% CI 1.18–2.02).[119] Systemic sclerosis has also been associated with a fourfold increased CVD risk, potentially driven by vascular fibrosis and a high incidence of arrhythmias.[120] Pooled odds ratios demonstrate an elevated risk in patients with autoimmune disease and hypertension (1.67, 95% CI 1.58–1.76) and ischemic heart disease (1.38, 1.21–1.57).[121] The burden of multiple autoimmune diseases compound this risk, with hazard ratios rising from 1.41 for a single condition to 2.73 for two or more.[122] Although autoimmune-related cardiovascular risk may be lower than that conferred by hypertension or diabetes, it is now recognized as a clinically relevant non-traditional risk enhancer, particularly in young women.

AIDs are among few non-traditional female-specific risk factors currently integrated into cardiovascular risk prediction models. For instance, rheumatoid arthritis (RA) is incorporated in the QRISK-2 calculator, while high sensitivity C-reactive protein (hsCRP) is included in the Reynolds Risk Score.[123,124] The Expanded-Risk-Score in RA and the Trans-Atlantic Cardiovascular Consortium for RA Score are two smaller but RA specific risk models that account for more extensive disease features; however, they have not proven incremental benefit beyond traditional scores.[125,126]

Breast Cancer

While not female specific, breast cancer is a condition that predominantly impacts women and therefore should be a sex-specific consideration. Long-term cardiovascular outcomes following breast cancer have become a growing concern, particularly due to the cardiotoxic effects of anthracycline-based chemotherapy, HER-2 targeted therapy and thoracic radiotherapy. Survivors of breast cancer are at an increased risk of CVD with HR of 1.32 in the first 10–15 years although they have no increased risk beyond 15 years.[127] A 2019 meta-analysis further identified a modest rise in cardiovascular mortality during the first year post-treatment (HR 1.09).[19] Furthermore, women with advanced breast cancer stage at diagnosis are more likely to have prevalent cardiovascular disease.[128] Interestingly, modification of cardiovascular health also improves cancer-related mortality outcomes, suggesting a dual benefit for CVD risk monitoring in these patients.[129]

Although this excess risk is less pronounced than that associated with traditional risk factors, it is clinically relevant given the lifetime incidence of breast cancer in women, estimated at 12.4%.[130] Contemporary cardio-oncology guidelines now recommend early cardiovascular risk assessment and proactive monitoring of cardiac function in women undergoing breast cancer treatment, particularly in the early years following diagnosis.[131]

Breast Arterial Calcification

Breast arterial calcification (BAC) identified on screening mammography, is a novel marker of vascular calcification that is increasingly recognized for its role in cardiovascular risk stratification in women. Unlike coronary artery disease, which involves intimal plaque deposition, BAC presents as linear or sheet-like calcification in the medial layer of arteries, similar to cerebrovascular and peripheral arterial disease.[132,133] Despite these histological differences, numerous studies have shown an association with cardiovascular disease and certain cardiometabolic risk factors. A 2015 meta-analysis reported an adjusted hazard ratio of 1.32 to 1.44 for the association between BAC and coronary heart disease,

with BAC also associated with age, diabetes and chronic kidney disease.[134] While some smaller studies have found no association, the majority of studies in the past two decades have supported the relationship.[135]

Mammography is slowly becoming a potential tool for cardiovascular screening with the combination of BAC and low breast density (BD), which correlates with cardiometabolic disease. Combination of BAC and BD has an independent association with coronary artery disease and has utility by improving standard coronary risk prediction models.[136] Artificial-intelligence-based quantification of BAC is also possible and research demonstrates an independent and incremental association between BAC scores with mortality and CVD. Importantly, BAC is detectable on routine screening mammograms, offering a non-invasive, low risk and cost-effective opportunity for cardiovascular assessment at a population level.[135] BAC holds promise as an imaging specific personalized risk marker and its additive value to traditional and non-traditional risk factors is a subject of ongoing exploration.

These non-traditional risk factors are discussed as discrete entities for clarity; however, cardiovascular risk in women is a cumulative and dynamic process across the life span. Female-specific exposures frequently coexist with each other and traditional cardiometabolic risk factors. Pregnancy-related complications, for example, may unmask underlying metabolic or vascular vulnerability that subsequently predisposes to risk of obesity, hypertension or diabetes which accelerates cardiovascular disease later in life. Recognition of this interaction is important in understanding that both female-specific risk factors are not used in isolation but as an adjunct to traditional risk modelling.

Risk Modelling

Although multiple predictive models exist for cardiovascular risk assessment, their accuracy in women remains limited due to sex-specific variation in disease presentation and pathophysiology. A 2019 systematic review identified 160 risk models tailored for women, however, only two incorporated female-specific risk factors such as preeclampsia or gestational diabetes (Table 2).[137] Research evaluating the utility of current, commonly used CVD risk models for women demonstrated limited applicability, particularly in younger women.[138] This is in part due to the lack of any women below the age of 40 included in cohorts from which scores such as SCORE2 and the Pooled Cohort Equation were derived.[139,140] Furthermore, CVD is often seen as a "male" disease and awareness is suboptimal in both patients and clinicians.[141] This may result in reduced CVD screening and undiagnosed CVD risk factors, which are the cornerstone of such risk models.

Table 2 Studies Assessing Risk Prediction Models with Female Specific Risk Factors

Study	Risk factors assessed	Results
Parikh et al *(2016)*[142]	Number of live births, age at menarche, menstrual irregularity, age at first birth, stillbirths, miscarriages, infertility, breastfeeding	C statistics 0.726 increase to 0.730 with addition of non-traditional risk factors (**No significant improvement**)
van der Meer et al *(2016)*[143]	Age at menarche, menopausal age, HRT use, Number of live births, GHTN, GDM	No change in C statistic with non-traditional risk factors (**No significant improvement)**
Stuart et al *(2018)*[144]	Hypertensive disorders of pregnancy and parity	C statistic change from 0.691 to 0.693 (**No significant improvement)**
Ukah et al *(2020)*[145]	Hypertensive disorders of pregnancy, caesarean delivery, stillbirth, neonatal death, previous pregnancy complications, gestational age at delivery, GDM	C statistic of 0.66 – no comparison (**N/A**)
Doust et al *(2024)*[146]	Early menarche (<11 years), endometriosis, excessive, frequent or irregular menstruation, miscarriage, number of miscarriages, number of stillbirths, infertility, preeclampsia or eclampsia, gestational diabetes (without subsequent type 2 diabetes), premature menopause (<40 years), early menopause (<45 years), and natural or surgical early menopause (menopause <45 years or timing of menopause reported as unknown and oophorectomy reported at age <45)	C statistic with traditional RFs – (0.710, 0.713, 0.718) C statistic with non-traditional + traditional RFs – (0.712, 0.715, 0.720) **(No significant improvement)**

Notes: List of studies assessing utility of female specific non-traditional cardiovascular risk factors in CVD risk prediction models.[142–145]
Abbreviations: CVD, cardiovascular disease; HRT, hormone replacement therapy; GHTN, gestational hypertension; GDM, gestational diabetes mellitus; RF, risk factors.

The largest prospective study to date, including over 135,000 women, evaluated reproductive factors such as early menarche, infertility, miscarriage, preeclampsia, gestational diabetes and early menopause. When used individually or cumulatively, these factors only yielded marginal improvements in risk model prediction, increasing the C-statistic from 0.726 to 0.730. However, mean age in the study was reported as 57.5, limiting the applicability to younger women.[146] Similarly, a 2016 study incorporating menarche, breastfeeding and fertility indicators reported only minor improvement, mainly through improved reclassification in women without cardiovascular events (Table 2).[142–145] Conversely, other studies have found some improvement with pregnancy-related condition, especially hypertensive disorders, when integrated into 10-year predictive models.[145] Evaluations of combined models, incorporating traditional and non-traditional risk factors, demonstrated no change in 10-year risk prediction, however, once again within predominant focus on middle age to elderly women.[143]

The future utility of female-specific risk factors with risk prediction models may lie in early identification of women at risk of developing modifiable cardiometabolic conditions, especially given the suboptimal awareness. These exposures often occur in early life stages, well before the emergence of most traditional risk factors, offering a critical window for preventative intervention. Risk factors such as pregnancy-related complications and breast arterial calcification may act as a marker of predisposition to cardiometabolic disease and a sign to instigate earlier screening.

Current Recommendations – Non-Traditional Risk Factors

Preventing cardiovascular disease through risk-guided therapy remains a central objective in cardiovascular medicine and women's health, yet challenges persist due to sex-specific variation in risk and historical underrepresentation of women in clinical research. Central to these efforts is accurate risk stratification and proactive management of cardiovascular risk factors. Statin therapy has long served as the cornerstone of primary prevention in individuals at elevated risk, supported by large-scale randomized controlled trials.[147]

However, data on women are limited and in recent years, increased recognition of this evidence gap has led to evolving perspectives on non-traditional and sex-specific risk factors in CVD prevention.[148]

Pregnancy Related Risk Factors

Several pregnancy-related complications – including hypertensive disorders of pregnancy, gestational diabetes, intrauterine growth restriction, preterm birth and placental abruption – are linked to long-term cardiovascular risk and have gained traction as an actionable risk marker.[17,73,74] The 2020 ACC/AHA summary recommendations, in alignment with guidance from the American College of Obstetricians and Gynecologists, recommend that women with such complications undergo CVD risk assessment within three months post-partum.[149] A Postpartum cardiovascular intervention clinic has demonstrated feasibility with consumers and may be an option to integrate this recommendation at a population level.[150]

The 2024 American Heart Association (AHA) statement on the postpartum cardiovascular care further expands upon these recommendations. Reflecting the elevated risk of diabetes and hypertension portended by gestational diabetes and hypertensive disorders of pregnancy respectively, the AHA advises intensified cardiometabolic screening in the first-year postpartum. In addition, they recommend routine cardiovascular risk factor screening for all women – regardless of pregnancy history – during the first 12 months after delivery. This approach seeks to address the current barrier of low attendance and participation in postpartum care, with only 18% to 25% of women with complications of pregnancy seeing their general practitioner within the first six months. The AHA also considers it reasonable to perform lipid screening during this period, given the historically low rates of lipid testing among young women.[151]

Breast Cancer

As long-term breast cancer survival rates continue to improve, cardiovascular disease has emerged as a leading cause of morbidity and mortality in this population.[152] This concern is heightened by shared risk factors – such as obesity, age and smoking – as well as the cardiotoxic effect of many cancer therapies.[153] Cardioprotective agents such as beta blockers and angiotensin converting enzyme inhibitors have shown promise in small cohort studies for mitigating chemotherapy-induced cardiac dysfunction; however, results have been inconsistent and do not support routine use of these agents in

breast cancer patients.[154] The use of aspirin and statins also remains investigational outside of established cardiovascular indications. A 2015 meta-analysis suggested that statin use may have some prognostic benefit for cancer survival, however there has been little assessing utility in CVD prevention.[155]

Breast Arterial Calcification

Current clinical guidelines are limited on the use of breast arterial calcification detected on mammography for CVD risk assessment. The Canadian Society of Breast Imaging advise that BAC could be useful for CVD risk stratification and has proposed a 4-stage model for BAC grading. Reported awareness of BAC and CVD risk is 17% amongst Canadian physicians, however, over half agree that BAC grading/guidelines would be beneficial and that BAC reporting would prompt further investigation.[156] The role of BAC may be as an incidental finding during cancer screening that prompts CVD screening and is additive to CVD risk assessment.

Despite the advances, standard CVD prevention strategies remain the cornerstone of management for women, including those with AIDs.[157] Hypertension, dyslipidemia and diabetes remain underrecognized and undertreated, highlighting the need for awareness of cardiovascular risk for women – in particular among young women and women from ethnically diverse backgrounds.[2] Moreover, few international studies have specifically evaluated lifestyle or pharmacologic strategies for CVD risk reduction in women with non-traditional risk factors, highlighting a gap and need for focus research in this area.[158,159]

Future Direction

Over the past decade, there has been substantial growth in research examining cardiovascular disease in women; however, how best to translate sex-based differences in risk stratification and management strategies remains unclear. A central objective of ongoing research is to improve identification of women at increased CVD risk who are not adequately captured by current guideline-based risk models.

While traditional risk factors remain fundamental to prevention strategies, increasing attention has been directed towards non-traditional and female-specific risk factors. At present, the evidence supporting many of these factors is largely observational, with limited interventional data. As such, their clinical utility lies not in defining treatment pathways alone, but in enhancing risk recognition and refining risk estimation, particularly in younger women and those classified low risk by conventional tools.

Non-traditional risk factors such as breast arterial calcification, pregnancy-related complications and early menopause offer important insight into sex-specific pathways of cardiovascular pathology. Incorporating these variables into future risk prediction frameworks, alongside traditional risk factors, has the potential to enable earlier identification of at-risk women and support more individualized preventative strategies. For optimal cardiovascular risk assessment in women, clinicians should account for sex-specific differences in the impact of traditional risk factors and recognize non-traditional risk factors that may indicate elevated cardiovascular risk even in the absence of conventional high-risk features.

Conclusion

Despite decades of progress in cardiovascular medicine, our understanding of risk in women remains incomplete. While traditional risk factors are well established, their interpretation and clinical impact often differ in women, especially when considered alongside sex-specific non-traditional risk factors such as pregnancy complications, autoimmune diseases and early reproductive transitions. Many of these factors emerge early in life, long before routine cardiovascular screening begins.

What emerges from this synthesis is not a lack of data but a lack of integration. Risk models remain narrowly focused and clinical guidelines seldom address the complexity of female cardiovascular risk across the lifespan. The opportunity now lies in reframing prevention – not simply through adapting existing tools but by redefining how we measure and intervene on risk in women. Doing so will require investment into longitudinal research, improved representation in clinical trials and a shift towards personalized, sex-specific care.

Ethics

No human participants/research was involved in the production of this article.

Acknowledgments

We note the use of AI only in generating part of the non-text images for Figure 1 via model GPT-4.5

Funding

There is no funding to report.

Disclosure

The authors report no conflicts of interest in this work.

References

1. Woodward M. Cardiovascular disease and the female disadvantage. *Int J Environ Res Public Health*. 2019;16(7):1165. doi:10.3390/ijerph16071165
2. Vogel B, Acevedo M, Appelman Y, et al. The Lancet women and cardiovascular disease commission: reducing the global burden by 2030. *Lancet*. 2021;397(10292):2385–2438. doi:10.1016/s0140-6736(21)00684-x
3. Tu JV, Khan AM, Ng K, Chu A. Recent temporal changes in atherosclerotic cardiovascular diseases in ontario: clinical and health systems impact. *Can J Cardiol*. 2017;33(3):378–384. doi:10.1016/j.cjca.2016.11.009
4. Champney KP, Frederick PD, Bueno H, et al. The joint contribution of sex, age and type of myocardial infarction on hospital mortality following acute myocardial infarction. *Heart*. 2009;95(11):895–899. doi:10.1136/hrt.2008.155804
5. Wenger NK, Lloyd-Jones DM, Elkind MSV, et al. Call to action for cardiovascular disease in women: epidemiology, awareness, access, and delivery of equitable health care: a presidential advisory from the American heart association. *Circulation*. 2022;145(23):e1059–e1071. doi:10.1161/cir.0000000000001071
6. Norhammar A, Schenck-Gustafsson K. Type 2 diabetes and cardiovascular disease in women. *Diabetologia*. 2013;56(1):1–9. doi:10.1007/s00125-012-2694-y
7. Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet*. 2004;364(9438):937–952. doi:10.1016/s0140-6736(04)17018-9
8. Huxley R, Barzi F, Woodward M. Excess risk of fatal coronary heart disease associated with diabetes in men and women: meta-analysis of 37 prospective cohort studies. *BMJ*. 2006;332(7533):73–8. doi:10.1136/bmj.38678.389583.7C
9. Anand SS, Islam S, Rosengren A, et al. Risk factors for myocardial infarction in women and men: insights from the INTERHEART study. *Eur Heart J*. 2008;29(7):932–940. doi:10.1093/eurheartj/ehn018
10. Wang Y, O'Neil A, Jiao Y, et al. Sex differences in the association between diabetes and risk of cardiovascular disease, cancer, and all-cause and cause-specific mortality: a systematic review and meta-analysis of 5,162,654 participants. *BMC Med*. 2019;17(1):136. doi:10.1186/s12916-019-1355-0
11. Huxley RR, Woodward M. Cigarette smoking as a risk factor for coronary heart disease in women compared with men: a systematic review and meta-analysis of prospective cohort studies. *Lancet*. 2011;378(9799):1297–1305. doi:10.1016/s0140-6736(11)60781-2
12. Wang H, Ba Y, Cai RC, Xing Q. Association between diabetes mellitus and the risk for major cardiovascular outcomes and all-cause mortality in women compared with men: a meta-analysis of prospective cohort studies. *BMJ Open*. 2019;9(7):e024935. doi:10.1136/bmjopen-2018-024935
13. Osman M, Regner S, Osman K, et al. Association between breast arterial calcification on mammography and coronary artery disease: a systematic review and meta-analysis. *J Womens Health*. 2022;31(12):1719–1726. doi:10.1089/jwh.2020.8733
14. Lo CCW, Lo ACQ, Leow SH, et al. Future cardiovascular disease risk for women with gestational hypertension: a systematic review and meta-analysis. *J Am Heart Assoc*. 2020;9(13):e013991. doi:10.1161/jaha.119.013991
15. Li J, Song C, Li C, Liu P, Sun Z, Yang X. Increased risk of cardiovascular disease in women with prior gestational diabetes: a systematic review and meta-analysis. *Diabet Res Clin Pract*. 2018;140:324–338. doi:10.1016/j.diabres.2018.03.054
16. Bellamy L, Casas JP, Hingorani AD, Williams DJ. Pre-eclampsia and risk of cardiovascular disease and cancer in later life: systematic review and meta-analysis. *BMJ*. 2007;335(7627):974. doi:10.1136/bmj.39335.385301.BE
17. Wu P, Haththotuwa R, Kwok CS, et al. Preeclampsia and future cardiovascular health: a systematic review and meta-analysis. *Circ Cardiovasc Qual Outcomes*. 2017;10(2). doi:10.1161/circoutcomes.116.003497
18. Kramer CK, Campbell S, Retnakaran R. Gestational diabetes and the risk of cardiovascular disease in women: a systematic review and meta-analysis. *Diabetologia*. 2019;62(6):905–914. doi:10.1007/s00125-019-4840-2
19. Galimzhanov A, Istanbuly S, Tun HN, et al. Cardiovascular outcomes in breast cancer survivors: a systematic review and meta-analysis. *Eur J Prev Cardiol*. 2023;30(18):2018–2031. doi:10.1093/eurjpc/zwad243
20. Peters SA, Huxley RR, Woodward M. Diabetes as risk factor for incident coronary heart disease in women compared with men: a systematic review and meta-analysis of 64 cohorts including 858,507 individuals and 28,203 coronary events. *Diabetologia*. 2014;57(8):1542–1551. doi:10.1007/s00125-014-3260-6
21. Lee WL, Cheung AM, Cape D, Zinman B. Impact of diabetes on coronary artery disease in women and men: a meta-analysis of prospective studies. *Diabetes Care*. 2000;23(7):962–968. doi:10.2337/diacare.23.7.962

22. Wekker V, van Dammen L, Koning A, et al. Long-term cardiometabolic disease risk in women with PCOS: a systematic review and meta-analysis. *Hum Reprod Update*. 2020;26(6):942–960. doi:10.1093/humupd/dmaa029
23. McDonald SD, Malinowski A, Zhou Q, Yusuf S, Devereaux PJ. Cardiovascular sequelae of preeclampsia/eclampsia: a systematic review and meta-analyses. *Am Heart J*. 2008;156(5):918–930. doi:10.1016/j.ahj.2008.06.042
24. Brown MC, Best KE, Pearce MS, Waugh J, Robson SC, Bell R. Cardiovascular disease risk in women with pre-eclampsia: systematic review and meta-analysis. *Eur J Epidemiol*. 2013;28(1):1–19. doi:10.1007/s10654-013-9762-6
25. Mongraw-Chaffin ML, Peters SAE, Huxley RR, Woodward M. The sex-specific association between BMI and coronary heart disease: a systematic review and meta-analysis of 95 cohorts with 1·2 million participants. *Lancet Diabetes Endocrinol*. 2015;3(6):437–449. doi:10.1016/s2213-8587(15)00086-8
26. Peters SA, Singhateh Y, Mackay D, Huxley RR, Woodward M. Total cholesterol as a risk factor for coronary heart disease and stroke in women compared with men: a systematic review and meta-analysis. *Atherosclerosis*. 2016;248:123–131. doi:10.1016/j.atherosclerosis.2016.03.016
27. Lin YT, Chen YR, Wei YC. Evaluating sex differences in the effect of increased systolic blood pressure on the risk of cardiovascular disease in Asian populations: a systematic review and meta-analysis. *Glob Heart*. 2022;17(1):70. doi:10.5334/gh.1159
28. Tay CT, Mousa A, Vyas A, Pattuwage L, Tehrani FR, Teede H. 2023 international evidence-based polycystic ovary syndrome guideline update: insights from a systematic review and meta-analysis on elevated clinical cardiovascular disease in polycystic ovary syndrome. *J Am Heart Assoc*. 2024;13(16):e033572. doi:10.1161/jaha.123.033572
29. Wan Z, Zhao J, Ye Y, et al. Risk and incidence of cardiovascular disease associated with polycystic ovary syndrome. *Eur J Prev Cardiol*. 2024;31(13):1560–1570. doi:10.1093/eurjpc/zwae066
30. Inversetti A, Pivato CA, Cristodoro M, et al. Update on long-term cardiovascular risk after pre-eclampsia: a systematic review and meta-analysis. *Eur Heart J Qual Care Clin Outcomes*. 2024;10(1):4–13. doi:10.1093/ehjqcco/qcad065
31. Kautzky-Willer A, Kamyar MR, Gerhat D, et al. Sex-specific differences in metabolic control, cardiovascular risk, and interventions in patients with type 2 diabetes mellitus. *Gend Med*. 2010;7(6):571–583. doi:10.1016/j.genm.2010.12.001
32. Gouni-Berthold I, Berthold HK, Mantzoros CS, Böhm M, Krone W. Sex disparities in the treatment and control of cardiovascular risk factors in type 2 diabetes. *Diabetes Care*. 2008;31(7):1389–1391. doi:10.2337/dc08-0194
33. Franch-Nadal J, Mata-Cases M, Vinagre I, et al. Differences in the Cardiometabolic control in type 2 diabetes according to gender and the presence of cardiovascular disease: results from the eControl study. *Int J Endocrinol*. 2014;2014:131709. doi:10.1155/2014/131709
34. Srikanthan P, Horwich TB, Calfon Press M, Gornbein J, Watson KE. Sex differences in the association of body composition and cardiovascular mortality. *J Am Heart Assoc*. 2021;10(5):e017511. doi:10.1161/jaha.120.017511
35. Chen GC, Arthur R, Iyengar NM, et al. Association between regional body fat and cardiovascular disease risk among postmenopausal women with normal body mass index. *Eur Heart J*. 2019;40(34):2849–2855. doi:10.1093/eurheartj/ehz391
36. Frank AP, de Souza Santos R, Palmer BF, Clegg DJ. Determinants of body fat distribution in humans may provide insight about obesity-related health risks. *J Lipid Res*. 2019;60(10):1710–1719. doi:10.1194/jlr.R086975
37. World Health Organization. *WHO Report on the Global Tobacco Epidemic 2017: Monitoring Tobacco Use and Prevention Policies*. 2017.
38. Jafari A, Rajabi A, Gholian-Aval M, Peyman N, Mahdizadeh M, Tehrani H. National, regional, and global prevalence of cigarette smoking among women/females in the general population: a systematic review and meta-analysis. *Environ Health Prev Med*. 2021;26(1):5. doi:10.1186/s12199-020-00924-y
39. World Health Organization. *WHO Global Report on Trends in Prevalence of Tobacco Use 2000-2025*. 2021.
40. Berta L, Frairia R, Fortunati N, Fazzari A, Gaidano G. Smoking effects on the hormonal balance of fertile women. *Horm Res*. 1992;37(1–2):45–48. doi:10.1159/000182280
41. Mendelsohn ME, Karas RH. The protective effects of estrogen on the cardiovascular system. *N Engl J Med*. 1999;340(23):1801–1811. doi:10.1056/nejm199906103402306
42. Ihenacho U, Sriprasert I, Mack WJ, et al. A systematic review and meta-analysis of smoking and circulating sex hormone levels among premenopausal women. *Nicotine Tob Res*. 2022;24(11):1705–1713. doi:10.1093/ntr/ntac066
43. Rezaianzadeh A, Johari MG, Baeradeh N, Seif M, Hosseini SV. Sex differences in hypertension incidence and risk factors: a population-based cohort study in Southern Iran. *BMC Public Health*. 2024;24(1):3575. doi:10.1186/s12889-024-21082-8
44. Ji H, Niiranen TJ, Rader F, et al. Sex differences in blood pressure associations with cardiovascular outcomes. *Circulation*. 2021;143(7):761–763. doi:10.1161/circulationaha.120.049360
45. Reckelhoff JF. Gender differences in the regulation of blood pressure. *Hypertension*. 2001;37(5):1199–1208. doi:10.1161/01.hyp.37.5.1199
46. Kringeland E, Tell GS, Midtbø H, Igland J, Haugsgjerd TR, Gerdts E. Stage 1 hypertension, sex, and acute coronary syndromes during midlife: the Hordaland health study. *Eur J Prev Cardiol*. 2022;29(1):147–154. doi:10.1093/eurjpc/zwab068
47. Beth L, Abramson KS, Leslie L, Davis NP, Parapid B. Women and hypertension: beyond the 2017 guideline for prevention, detection, evaluation, and management of high blood pressure in adults. *Am Coll Cardiol*. 2018
48. Mumford SL, Dasharathy S, Pollack AZ, Schisterman EF. Variations in lipid levels according to menstrual cycle phase: clinical implications. *Clin Lipidol*. 2011;6(2):225–234. doi:10.2217/clp.11.9
49. Jacobson TA, Maki KC, Orringer CE, et al. National lipid association recommendations for patient-centered management of dyslipidemia: part 2. *J Clin Lipidol*. 2015;9(6 Suppl):S1–122.e1. doi:10.1016/j.jacl.2015.09.002
50. Ryzhaya N, Cermakova L, Trinder M, et al. Sex differences in the presentation, treatment, and outcome of patients with familial hypercholesterolemia. *J Am Heart Assoc*. 2021;10(11):e019286. doi:10.1161/jaha.120.019286
51. Zimodro JM, Mucha M, Berthold HK, Gouni-Berthold I. Lipoprotein metabolism, dyslipidemia, and lipid-lowering therapy in women: a comprehensive review. *Pharmaceuticals*. 2024;17(7):913. doi:10.3390/ph17070913
52. Nanna MG, Wang TY, Xiang Q, et al. Sex differences in the use of statins in community practice. *Circ Cardiovasc Qual Outcomes*. 2019;12(8):e005562. doi:10.1161/circoutcomes.118.005562
53. Bradley CK, Wang TY, Li S, et al. Patient-Reported Reasons for Declining or Discontinuing Statin Therapy: insights From the PALM Registry. *J Am Heart Assoc*. 2019;8(7):e011765. doi:10.1161/jaha.118.011765
54. Karalis DG, Wild RA, Maki KC, et al. Gender differences in side effects and attitudes regarding statin use in the understanding statin use in america and gaps in patient education (USAGE) study. *J Clin Lipidol*. 2016;10(4):833–841. doi:10.1016/j.jacl.2016.02.016

55. Forbes CA, Quek RG, Deshpande S, et al. The relationship between Lp(a) and CVD outcomes: a systematic review. *Lipids Health Dis*. 2016;15:95. doi:10.1186/s12944-016-0258-8
56. Costello BT, Silverman ER, Doukky R, et al. Lipoprotein(a) and Increased Cardiovascular Risk in Women. *Clin Cardiol*. 2016;39(2):96–102. doi:10.1002/clc.22500
57. Orth-Gomér K, Mittleman MA, Schenck-Gustafsson K, et al. Lipoprotein(a) as a determinant of coronary heart disease in young women. *Circulation*. 1997;95(2):329–334. doi:10.1161/01.cir.95.2.329
58. Foody JM, Milberg JA, Pearce GL, Sprecher DL. Lipoprotein(a) associated with coronary artery disease in older women: age and gender analysis. *Atherosclerosis*. 2000;153(2):445–451. doi:10.1016/s0021-9150(00)00427-5
59. Cook NR, Mora S, Ridker PM. Lipoprotein(a) and cardiovascular risk prediction among women. *J Am Coll Cardiol*. 2018;72(3):287–296. doi:10.1016/j.jacc.2018.04.060
60. Dhingra R, Vasan RS. Age as a risk factor. *Med Clin North Am*. 2012;96(1):87–91. doi:10.1016/j.mcna.2011.11.003
61. Sniderman AD, Furberg CD. Age as a modifiable risk factor for cardiovascular disease. *Lancet*. 2008;371(9623):1547–1549. doi:10.1016/s0140-6736(08)60313-x
62. Kannel WB, Vasan RS. Is age really a non-modifiable cardiovascular risk factor? *Am J Cardiol*. 2009;104(9):1307–1310. doi:10.1016/j.amjcard.2009.06.051
63. Jousilahti P, Vartiainen E, Tuomilehto J, Puska P. Sex, age, cardiovascular risk factors, and coronary heart disease: a prospective follow-up study of 14 786 middle-aged men and women in Finland. *Circulation*. 1999;99(9):1165–1172. doi:10.1161/01.cir.99.9.1165
64. Baker L, Meldrum KK, Wang M, et al. The role of estrogen in cardiovascular disease. *J Surg Res*. 2003;115(2):325–344. doi:10.1016/s0022-4804(03)00215-4
65. Costello BT, Sprung K, Coulter SA. The rise and fall of estrogen therapy: is testosterone for "manopause. *Next? Tex Heart Inst J*. 2017;44(5):338–340. doi:10.14503/thij-17-6360
66. Hodis HN, Mack WJ. Menopausal hormone replacement therapy and reduction of all-cause mortality and cardiovascular disease: it is about time and timing. *Cancer J*. 2022;28(3):208–223. doi:10.1097/ppo.0000000000000591
67. LaMonte MJ, Manson JE, Anderson GL, et al. Contributions of the women's health initiative to cardiovascular research: JACC state-of-the-art review. *J Am Coll Cardiol*. 2022;80(3):256–275. doi:10.1016/j.jacc.2022.05.016
68. Geraghty L, Figtree GA, Schutte AE, Patel S, Woodward M, Arnott C. Cardiovascular disease in women: from pathophysiology to novel and emerging risk factors. *Heart Lung Circ*. 2021;30(1):9–17. doi:10.1016/j.hlc.2020.05.108
69. Mosca L, Benjamin EJ, Berra K, et al. Effectiveness-based guidelines for the prevention of cardiovascular disease in women--2011 update: a guideline from the American heart association. *Circulation*. 2011;123(11):1243–1262. doi:10.1161/CIR.0b013e31820faaf8
70. Grundy SM, Stone NJ, Bailey AL, et al. 2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Blood Cholesterol: a Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Circulation*. 2019;139(25):e1082–e1143. doi:10.1161/CIR.0000000000000625
71. Mongraw-Chaffin ML, Cirillo PM, Cohn BA. Preeclampsia and cardiovascular disease death: prospective evidence from the child health and development studies cohort. *Hypertension*. 2010;56(1):166–171. doi:10.1161/hypertensionaha.110.150078
72. Lykke JA, Langhoff-Roos J, Sibai BM, Funai EF, Triche EW, Paidas MJ. Hypertensive pregnancy disorders and subsequent cardiovascular morbidity and type 2 diabetes mellitus in the mother. *Hypertension*. 2009;53(6):944–951. doi:10.1161/hypertensionaha.109.130765
73. Ahmed R, Dunford J, Mehran R, Robson S, Kunadian V. Pre-eclampsia and future cardiovascular risk among women: a review. *J Am Coll Cardiol*. 2014;63(18):1815–1822. doi:10.1016/j.jacc.2014.02.529
74. Staff AC, Dechend R, Pijnenborg R. Learning from the placenta: acute atherosis and vascular remodeling in preeclampsia-novel aspects for atherosclerosis and future cardiovascular health. *Hypertension*. 2010;56(6):1026–1034. doi:10.1161/hypertensionaha.110.157743
75. Margaret Krasne AW, Park K, Davis M, Blumenthal RS, Sharma G. Preeclampsia is an independent risk factor for early onset coronary artery calcification: a call for risk assessment and modification. American College of Cardiology; 2021. Available from: https://www.acc.org/Latest-in-Cardiology/Articles/2021/02/09/19/44/Preeclampsia-is-an-Independent-Risk-Factor-for-Early-Onset-CAC#:~:text=Importantly%2C%20women%20with%20a%20history,to%20women%20with%20prior%20preeclampsia. Accessed February 04, 2026.
76. Henry A, Mangos G, Roberts LM, et al. Preeclampsia-associated cardiovascular risk factors 6 months and 2 years after pregnancy: the P4 study. *Hypertension*. 2024;81(4):851–860. doi:10.1161/hypertensionaha.123.21890
77. Fu J, Retnakaran R. The life course perspective of gestational diabetes: an opportunity for the prevention of diabetes and heart disease in women. *EClinicalMedicine*. 2022;45:101294. doi:10.1016/j.eclinm.2022.101294
78. Retnakaran R, Shah BR. Role of type 2 diabetes in determining retinal, renal, and cardiovascular outcomes in women with previous gestational diabetes mellitus. *Diabetes Care*. 2017;40(1):101–108. doi:10.2337/dc16-1400
79. Retnakaran R. Hyperglycemia in pregnancy and its implications for a woman's future risk of cardiovascular disease. *Diabet Res Clin Pract*. 2018;145:193–199. doi:10.1016/j.diabres.2018.04.008
80. Retnakaran R, Luo J, Shah BR. Gestational diabetes in young women predicts future risk of serious liver disease. *Diabetologia*. 2019;62(2):306–310. doi:10.1007/s00125-018-4775-z
81. Rawal S, Olsen SF, Grunnet LG, et al. Gestational diabetes mellitus and renal function: a prospective study with 9- to 16-year follow-up after pregnancy. *Diabetes Care*. 2018;41(7):1378–1384. doi:10.2337/dc17-2629
82. Retnakaran R, Shah BR. Glucose screening in pregnancy and future risk of cardiovascular disease in women: a retrospective, population-based cohort study. *Lancet Diabetes Endocrinol*. 2019;7(5):378–384. doi:10.1016/s2213-8587(19)30077-4
83. National Institute for Health and Care Excellence: Guidelines. *Hypertension in Pregnancy: Diagnosis and Management*. Vol. 2019. National Institute for Health and Care Excellence (NICE) Copyright © NICE; 2019.
84. Regitz-Zagrosek V, Roos-Hesselink JW, Bauersachs J, et al. 2018 ESC Guidelines for the management of cardiovascular diseases during pregnancy. *Eur Heart J*. 2018;39(34):3165–3241. doi:10.1093/eurheartj/ehy340
85. Grandi SM, Filion KB, Yoon S, et al. Cardiovascular disease-related morbidity and mortality in women with a history of pregnancy complications. *Circulation*. 2019;139(8):1069–1079. doi:10.1161/circulationaha.118.036748
86. Wang YX, Arvizu M, Rich-Edwards JW, et al. Hypertensive disorders of pregnancy and subsequent risk of premature mortality. *J Am Coll Cardiol*. 2021;77(10):1302–1312. doi:10.1016/j.jacc.2021.01.018

87. Honigberg MC, Zekavat SM, Aragam K, et al. Long-Term cardiovascular risk in women with hypertension during pregnancy. *J Am Coll Cardiol*. 2019;74(22):2743–2754. doi:10.1016/j.jacc.2019.09.052
88. Unger T, Borghi C, Charchar F, et al. 2020 international society of hypertension global hypertension practice guidelines. *Hypertension*. 2020;75(6):1334–1357. doi:10.1161/hypertensionaha.120.15026
89. Stuart JJ, Tanz LJ, Rimm EB, et al. Cardiovascular risk factors mediate the long-term maternal risk associated with hypertensive disorders of pregnancy. *J Am Coll Cardiol*. 2022;79(19):1901–1913. doi:10.1016/j.jacc.2022.03.335
90. Kyriacou H, Al-Mohammad A, Muehlschlegel C, et al. The risk of cardiovascular diseases after miscarriage, stillbirth, and induced abortion: a systematic review and meta-analysis. *Eur Heart J Open*. 2022;2(5):oeac065. doi:10.1093/ehjopen/oeac065
91. Gao L, Bowe SJ, Moodie M. Preterm Delivery and increased risk of recurrent cardiovascular events in australian women. *J Womens Health*. 2022;31(12):1773–1781. doi:10.1089/jwh.2022.0091
92. Li W, Ruan W, Lu Z, Wang D. Parity and risk of maternal cardiovascular disease: a dose-response meta-analysis of cohort studies. *Eur J Prev Cardiol*. 2019;26(6):592–602. doi:10.1177/2047487318818265
93. Udell JA, Lu H, Redelmeier DA. Failure of fertility therapy and subsequent adverse cardiovascular events. *Cmaj*. 2017;189(10):E391–e397. doi:10.1503/cmaj.160744
94. Tomic D, Rombauts L, Da Silva Costa F, et al. Cardiovascular disease hospitalizations among women who undergo fertility treatment. *Reprod Biomed Online*. 2024;49(1):103812. doi:10.1016/j.rbmo.2024.103812
95. Xing Z, Kirby R, Chen H, Cain M, Alman A. Parity, age at childbirth, and later-life maternal cardiovascular disease in postmenopausal women in the women's health initiative study. *O&G Open*. 2025;2e063. doi:10.1097/og9.0000000000000063
96. Täufer Cederlöf E, Lundgren M, Lindahl B, Christersson C. Pregnancy complications and risk of cardiovascular disease later in life: a nationwide cohort study. *J Am Heart Assoc*. 2022;11(2):e023079. doi:10.1161/jaha.121.023079
97. Hulley S, Grady D, Bush T, et al. Randomized trial of estrogen plus progestin for secondary prevention of coronary heart disease in postmenopausal women. Heart and Estrogen/progestin Replacement Study (HERS) research group. *JAMA*. 1998;280(7):605–613. doi:10.1001/jama.280.7.605
98. Rossouw JE, Anderson GL, Prentice RL, et al. Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results from the women's health initiative randomized controlled trial. *JAMA*. 2002;288(3):321–333. doi:10.1001/jama.288.3.321
99. Stampfer MJ, Colditz GA, Willett WC, et al. Postmenopausal estrogen therapy and cardiovascular disease. Ten-year follow-up from the nurses' health study. *N Engl J Med*. 1991;325(11):756–762. doi:10.1056/nejm199109123251102
100. Stuenkel CA, Davis SR, Gompel A, et al. Treatment of symptoms of the menopause: an endocrine society clinical practice guideline. *J Clin Endocrinol Metab*. 2015;100(11):3975–4011. doi:10.1210/jc.2015-2236
101. Wei S, Schmidt MD, Dwyer T, Norman RJ, Venn AJ. Obesity and menstrual irregularity: associations with SHBG, testosterone, and insulin. *Obesity*. 2009;17(5):1070–1076. doi:10.1038/oby.2008.641
102. Duleba AJ, Dokras A. Is PCOS an inflammatory process? *Fertil Steril*. 2012;97(1):7–12. doi:10.1016/j.fertnstert.2011.11.023
103. Daan NMP, Jaspers L, Koster MPH, et al. Androgen levels in women with various forms of ovarian dysfunction: associations with cardiometabolic features. *Hum Reprod*. 2015;30(10):2376–2386. doi:10.1093/humrep/dev195
104. Okoth K, Smith WP, Thomas GN, Nirantharakumar K, Adderley NJ. The association between menstrual cycle characteristics and cardiometabolic outcomes in later life: a retrospective matched cohort study of 704,743 women from the UK. *BMC Med*. 2023;21(1):104. doi:10.1186/s12916-023-02794-x
105. Huang C, Lin B, Yuan Y, et al. Associations of menstrual cycle regularity and length with cardiovascular diseases: a prospective study from UK biobank. *J Am Heart Assoc*. 2023;12(11):e029020. doi:10.1161/jaha.122.029020
106. Lee JJ, Cook-Wiens G, Johnson BD, et al. Age at menarche and risk of cardiovascular disease outcomes: findings from the national heart lung and blood institute-sponsored women's ischemia syndrome evaluation. *J Am Heart Assoc*. 2019;8(12):e012406. doi:10.1161/jaha.119.012406
107. Bedaiwy MA, Abdel-Rahman MY, Tan J, et al. Clinical, hormonal, and metabolic parameters in women with subclinical hypothyroidism and polycystic ovary syndrome: a cross-sectional study. *J Womens Health*. 2018;27(5):659–664. doi:10.1089/jwh.2017.6584
108. Azziz R, Woods KS, Reyna R, Key TJ, Knochenhauer ES, Yildiz BO. The prevalence and features of the polycystic ovary syndrome in an unselected population. *J Clin Endocrinol Metab*. 2004;89(6):2745–2749. doi:10.1210/jc.2003-032046
109. Yildiz BO, Bozdag G, Yapici Z, Esinler I, Yarali H. Prevalence, phenotype and cardiometabolic risk of polycystic ovary syndrome under different diagnostic criteria. *Hum Reprod*. 2012;27(10):3067–3073. doi:10.1093/humrep/des232
110. Zhang J, Xu JH, Qu QQ, Zhong GQ. Risk of cardiovascular and cerebrovascular events in polycystic ovarian syndrome women: a meta-analysis of cohort studies. *Front Cardiovasc Med*. 2020;7:552421. doi:10.3389/fcvm.2020.552421
111. Ollila MM, Arffman RK, Korhonen E, et al. Women with PCOS have an increased risk for cardiovascular disease regardless of diagnostic criteria-a prospective population-based cohort study. *Eur J Endocrinol*. 2023;189(1):96–105. doi:10.1093/ejendo/lvad077
112. Akre S, Sharma K, Chakole S, Wanjari MB. Recent advances in the management of polycystic ovary syndrome: a review article. *Cureus*. 2022;14(8):e27689. doi:10.7759/cureus.27689
113. Kaminski P, Szpotanska-Sikorska M, Wielgos M. Cardiovascular risk and the use of oral contraceptives. *Neuro Endocrinol Lett*. 2013;34(7):587–589.
114. Horton LG, Simmons KB, Curtis KM. Combined hormonal contraceptive use among obese women and risk for cardiovascular events: a systematic review. *Contraception*. 2016;94(6):590–604. doi:10.1016/j.contraception.2016.05.014
115. Dou W, Huang Y, Liu X, et al. Associations of oral contraceptive use with cardiovascular disease and all-cause death: evidence from the UK biobank cohort study. *J Am Heart Assoc*. 2023;12(16):e030105. doi:10.1161/jaha.123.030105
116. Soldat-Stankovic V, Popovic Pejicic S, Stankovic S, et al. The effect of myoinositol and metformin on cardiovascular risk factors in women with polycystic ovary syndrome: a randomized controlled trial. *Acta Endocrinol*. 2021;17(2):241–247. doi:10.4183/aeb.2021.241
117. Angum F, Khan T, Kaler J, Siddiqui L, Hussain A. The prevalence of autoimmune disorders in women: a narrative review. *Cureus*. 2020;12(5):e8094. doi:10.7759/cureus.8094
118. Mehta PK, Levit RD, Wood MJ, et al. Chronic rheumatologic disorders and cardiovascular disease risk in women. *Am Heart J Plus*. 2023;27:100267. doi:10.1016/j.ahjo.2023.100267

119. Restivo V, Candiloro S, Daidone M, et al. Systematic review and meta-analysis of cardiovascular risk in rheumatological disease: symptomatic and non-symptomatic events in rheumatoid arthritis and systemic lupus erythematosus. *Autoimmun Rev*. 2022;21(1):102925. doi:10.1016/j.autrev.2021.102925
120. Cafaro G, Perricone C, Gerli R, Bartoloni E. Cardiovascular risk in systemic autoimmune diseases. *Lancet*. 2023;401(10370):21–22. doi:10.1016/s0140-6736(22)02477-1
121. Barozet M, Le Tilly O, Bejan-Angoulvant T, Fesler P, Roubille C. Hypertension and cardiovascular outcomes in inflammatory and autoimmune diseases: a systematic review and meta-analysis. *Curr Hypertens Rep*. 2024;26(11):419–429. doi:10.1007/s11906-024-01311-6
122. Conrad N, Verbeke G, Molenberghs G, et al. Autoimmune diseases and cardiovascular risk: a population-based study on 19 autoimmune diseases and 12 cardiovascular diseases in 22 million individuals in the UK. *Lancet*. 2022;400(10354):733–743. doi:10.1016/s0140-6736(22)01349-6
123. Hippisley-Cox J, Coupland C, Vinogradova Y, Robson J, Brindle P. Performance of the QRISK cardiovascular risk prediction algorithm in an independent UK sample of patients from general practice: a validation study. *Heart*. 2008;94(1):34–39. doi:10.1136/hrt.2007.134890
124. Ridker PM, Buring JE, Rifai N, Cook NR. Development and validation of improved algorithms for the assessment of global cardiovascular risk in women: the Reynolds Risk Score. *JAMA*. 2007;297(6):611–619. doi:10.1001/jama.297.6.611
125. Solomon DH, Greenberg J, Curtis JR, et al. Derivation and internal validation of an expanded cardiovascular risk prediction score for rheumatoid arthritis: a consortium of rheumatology researchers of north america registry study. *Arthritis Rheumatol*. 2015;67(8):1995–2003. doi:10.1002/art.39195
126. Crowson CS, Gabriel SE, Semb AG, et al. Rheumatoid arthritis-specific cardiovascular risk scores are not superior to general risk scores: a validation analysis of patients from seven countries. *Rheumatology*. 2017;56(7):1102–1110. doi:10.1093/rheumatology/kex038
127. Koric A, Chang CP, Mark B, et al. Cardiovascular disease risk in long-term breast cancer survivors: a population-based cohort study. *Cancer*. 2022;128(14):2826–2835. doi:10.1002/cncr.34224
128. Angelov I, Haas AM, Brock E, et al. Cardiovascular disease and breast cancer stage at diagnosis. *JAMA Network Open*. 2025;8(1):e2452890. doi:10.1001/jamanetworkopen.2024.52890
129. Lin L, Hu Y, Lei F, et al. Cardiovascular health and cancer mortality: evidence from US NHANES and UK biobank cohort studies. *BMC Med*. 2024;22(1):368. doi:10.1186/s12916-024-03553-2
130. Cancer stat facts: female breast cancer. vol. 2023. Available from: https://seer.cancer.gov/statfacts/html/breast.html. Accessed October 15, 2023.
131. Mehta LS, Watson KE, Barac A, et al. Cardiovascular disease and breast cancer: where these entities intersect: a scientific statement from the American heart association. *Circulation*. 2018;137(8):e30–e66. doi:10.1161/cir.0000000000000556
132. Amann K. Media calcification and intima calcification are distinct entities in chronic kidney disease. *Clin J Am Soc Nephrol*. 2008;3(6):1599–1605. doi:10.2215/cjn.02120508
133. Crystal P, Zelingher J, Crystal E. Breast arterial calcifications as a cardiovascular risk marker in women. *Expert Rev Cardiovasc Ther*. 2004;2(5):753–760. doi:10.1586/14779072.2.5.753
134. Hendriks EJ, de Jong PA, van der Graaf Y, Mali WP, van der Schouw YT, Beulens JW. Breast arterial calcifications: a systematic review and meta-analysis of their determinants and their association with cardiovascular events. *Atherosclerosis*. 2015;239(1):11–20. doi:10.1016/j.atherosclerosis.2014.12.035
135. Bui QM, Daniels LB. A review of the role of breast arterial calcification for cardiovascular risk stratification in women. *Circulation*. 2019;139(8):1094–1101. doi:10.1161/circulationaha.118.038092
136. Aldous E, Goel V, Cameron W, et al. Combined mammographic breast density and breast arterial calcification as an incremental predictor of coronary artery disease. *J Womens Health*. 2025. doi:10.1089/jwh.2024.0966
137. Baart SJ, Dam V, Scheres LJJ, et al. Cardiovascular risk prediction models for women in the general population: a systematic review. *PLoS One*. 2019;14(1):e0210329. doi:10.1371/journal.pone.0210329
138. Goh LG, Dhaliwal SS, Lee AH, Bertolatti D, Della PR. Utility of established cardiovascular disease risk score models for the 10-year prediction of disease outcomes in women. *Expert Rev Cardiovasc Ther*. 2013;11(4):425–435. doi:10.1586/erc.13.26
139. de Vries TI, Cooney MT, Selmer RM. SCORE2-OP risk prediction algorithms: estimating incident cardiovascular event risk in older persons in four geographical risk regions. *Eur Heart J*. 2021;42(25):2455–2467. doi:10.1093/eurheartj/ehab312
140. Goff DC Jr, Lloyd-Jones DM, Bennett G, et al. 2013 ACC/AHA guideline on the assessment of cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2014;129(25 Suppl 2):S49–73. doi:10.1161/01.cir.0000437741.48606.98
141. Bairey Merz CN, Andersen H, Sprague E, et al. Knowledge, attitudes, and beliefs regarding cardiovascular disease in women: the women's heart alliance. *J Am Coll Cardiol*. 2017;70(2):123–132. doi:10.1016/j.jacc.2017.05.024
142. Parikh NI, Jeppson RP, Berger JS, et al. Reproductive risk factors and coronary heart disease in the women's health initiative observational study. *Circulation*. 2016;133(22):2149–2158. doi:10.1161/circulationaha.115.017854
143. van der Meer MG, van der Graaf Y, Schuit E, et al. Added value of female-specific factors beyond traditional predictors for future cardiovascular disease. *J Am Coll Cardiol*. 2016;67(17):2084–2086. doi:10.1016/j.jacc.2016.02.031
144. Stuart JJ, Tanz LJ, Cook NR, et al. Hypertensive disorders of pregnancy and 10-year cardiovascular risk prediction. *J Am Coll Cardiol*. 2018;72(11):1252–1263. doi:10.1016/j.jacc.2018.05.077
145. Ukah UV, Dayan N, Auger N, He S, Platt RW. Development and Internal validation of a model predicting premature cardiovascular disease among women with hypertensive disorders of pregnancy: a population-based study in Quebec, Canada. *J Am Heart Assoc*. 2020;9(20):e017328. doi:10.1161/jaha.120.017328
146. Doust J, Baneshi MR, Chung HF, Wilson LF, Mishra GD. assessing the accuracy of cardiovascular disease prediction using female-specific risk factors in women aged 45 to 69 years in the UK biobank study. *Circ Cardiovasc Qual Outcomes*. 2024;17(12):e010842. doi:10.1161/circoutcomes.123.010842
147. Arnett DK, Blumenthal RS, Albert MA, et al. 2019 ACC/AHA Guideline on the Primary Prevention of Cardiovascular Disease: a Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Circulation*. 2019;140(11):e596–e646. doi:10.1161/cir.0000000000000678

148. Jin X, Chandramouli C, Allocco B, Gong E, Lam CSP, Yan LL. Women's participation in cardiovascular clinical trials from 2010 to 2017. *Circulation*. 2020;141(7):540–548. doi:10.1161/circulationaha.119.043594
149. Cho L, Davis M, Elgendy I, et al. Summary of updated recommendations for primary prevention of cardiovascular disease in women: JACC state-of-the-art review. *J Am Coll Cardiol*. 2020;75(20):2602–2618. doi:10.1016/j.jacc.2020.03.060
150. Manthorpe T, Arstall M, Andraweera PH, Aldridge E. Patient experiences of a postpartum cardiovascular disease intervention clinic for pregnancy complications. *Matern Child Health J*. 2025;29(3):310–321. doi:10.1007/s10995-025-04047-0
151. Lewey J, Beckie TM, Brown HL, et al. Opportunities in the postpartum period to reduce cardiovascular disease risk after adverse pregnancy outcomes: a scientific statement from the American heart association. *Circulation*. 2024;149(7):e330–e346. doi:10.1161/cir.0000000000001212
152. Sakr RA, Nasr AA, Zineldin EI, Gouda MA. Long-term survival in patients with cancers: surveillance, epidemiology and end results-based analysis. *Sultan Qaboos Univ Med J*. 2023;23(3):344–350. doi:10.18295/squmj.1.2023.002
153. Ma Y, Grootaert MOJ, Sewduth RN. Cardiotoxicity of chemotherapy: a multi-OMIC perspective. *J Xenobiot*. 2025;15(1):9. doi:10.3390/jox15010009
154. Elghazawy H, Venkatesulu BP, Verma V, et al. The role of cardio-protective agents in cardio-preservation in breast cancer patients receiving Anthracyclines ± Trastuzumab: a Meta-analysis of clinical studies. *Crit Rev Oncol Hematol*. 2020;153:103006. doi:10.1016/j.critrevonc.2020.103006
155. Wu QJ, Tu C, Li YY, et al. Statin use and breast cancer survival and risk: a systematic review and meta-analysis. *Oncotarget*. 2015;6(40):42988–43004. doi:10.18632/oncotarget.5557
156. Zaki-Metias KM, McKee H, Applewhaite C, et al. Breast Arterial Calcifications on Mammography: awareness and Reporting Preferences Amongst Referring Physicians in Canada. *Can Assoc Radiol J*. 2025;76(1):94–104. doi:10.1177/08465371241262292
157. McCauley KM. Modifying women's risk for cardiovascular disease. *J Obstet Gynecol Neonatal Nurs*. 2007;36(2):116–124. doi:10.1111/j.1552-6909.2007.00125.x
158. Rich-Edwards JW, Stuart JJ, Skurnik G, et al. Randomized trial to reduce cardiovascular risk in women with recent preeclampsia. *J Womens Health*. 2019;28(11):1493–1504. doi:10.1089/jwh.2018.7523
159. Hutchesson MJ, Taylor R, Shrewsbury VA, et al. Be healthe for your heart: a pilot randomized controlled trial evaluating a web-based behavioral intervention to improve the cardiovascular health of women with a history of preeclampsia. *Int J Environ Res Public Health*. 2020;17(16):5779. doi:10.3390/ijerph17165779