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AHA SCIENTIFIC STATEMENT

Consensus Guidance for the Female-Specific Prevention and Management of Cardiovascular Disease: A Scientific Statement From the American Heart Association, American Academy of Pediatrics, American College of Obstetricians and Gynecologists, and Society for Maternal-Fetal Medicine

Endorsed by the Society for Maternal-Fetal Medicine (SMFM) and should be considered official SMFM guidance
Endorsed by the American Academy of Pediatrics
In Collaboration with the American College of Obstetricians and Gynecologists (ACOG)

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ABSTRACT: There is growing recognition that the optimal prevention and management of cardiovascular disease in girls and women requires attention to sex-specific considerations relevant to the interconnected domains of hormonal, psychosocial, and cardiovascular health across the life course. Dysregulation in these health domains tends to co-occur and is associated with increased risk of cardiovascular disease. However, healthcare for girls and women is uniquely fragmented across clinical specialties, including pediatrics, adolescent medicine, primary care, obstetrics, maternal-fetal medicine, gynecology, cardiology, endocrinology, hematology, and neurology. Furthermore, inconsistent alignment across society guidelines contributes to missed opportunities to optimize cardiovascular health. Therefore, the American Heart Association, American Academy of Pediatrics, American College of Obstetricians and Gynecologists, and Society for Maternal-Fetal Medicine convened a multisociety science advisory group and developed the Female Life Stage construct. This construct defines 4 distinct and clinically meaningful stages—perimenarche (S1), ovarian cycling (S2), perimenopause (S3), and postmenopause (S4)—to enable female-specific screening, prevention, and treatment considerations. In addition, this construct incorporates pregnancy as a unique modifying life event when cardiovascular risk may emerge or be unmasked (eg, adverse pregnancy outcomes). This scientific statement crosswalks major society guidelines, synthesizes the evidence, and provides consensus-based clinical guidance for each Female Life Stage and interconnected health domain (ie, hormonal, psychosocial, and cardiovascular health). We conclude with a call to action to improve clinical workflows, enhance clinician education, and address scientific and implementation gaps to improve cardiovascular health in girls and women.

Key Words: AHA Scientific Statements ■ cardiovascular diseases ■ child ■ female ■ gynecology ■ pregnancy ■ primary prevention

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Cardiovascular disease (CVD) is the leading cause of morbidity and mortality among women and men, accounting for 1 in 3 deaths in the United States and worldwide.[1] However, clinical recommendations to optimize prevention and management of CVD in girls and women are distributed across multiple professional societies' practice guidelines. Therefore, girls and women experience unique aspects of care fragmentation, given the involvement of pediatrics, primary care, obstetrics and gynecology, maternal-fetal medicine, neurology, endocrinology, and cardiology. In addition, there is limited cross-disciplinary harmonization across specialties, which contributes to suboptimal care in girls and women.[2–4] Moreover, despite advances in the understanding of biologic sex differences and their direct and indirect influence on cardiovascular risk, translation of this knowledge into routine clinical practice remains incomplete.[5,6] As such, a female-specific framework that integrates interconnected domains of hormonal, psychosocial, and cardiovascular health is needed to inform a unifying approach for clinical care across the life course.

It is well recognized that cardiovascular health trajectories are shaped by cumulative exposures across the life course and, in girls and women, are additionally influenced by dynamic changes in sex hormones, as well as pregnancy (Supplemental Table 1).[7,8] Even when individual professional societies provide evidence-based recommendations for assessment and management of hormonal, psychosocial, and cardiovascular health, the interconnectedness among these domains is not always recognized, precluding a truly unified approach to clinical care.[2,9] Moreover, gaps in knowledge persist related to sex differences in pathophysiology, CVD phenotype heterogeneity, and benefits and adverse effects of evidence-based therapies.[10]

Building on the conceptual framework for the Female Life Stage construct, which is established in the accompanying presidential advisory[10a] as comprising four clinically meaningful stages, this joint scientific statement on behalf of the American Heart Association (AHA), American Academy of Pediatrics (AAP), American College of Obstetricians and Gynecologists (ACOG), and Society for Maternal-Fetal Medicine (SMFM) presents stage-specific clinical guidance across the interconnected domains of hormonal, psychosocial, and cardiovascular health. The presidential advisory[10a] and this scientific statement are accompanied by a third document[10b] in the compendium, which addresses how systems can be structured to optimize care, including through the creation and maintenance of Comprehensive Centers of Excellence for Women's Cardiovascular Care that are anchored on interdisciplinary care models. A list of abbreviations used in this compendium is presented in Supplemental Table 2.

Together, these 3 documents aim to transform the current siloed approach to cardiovascular care for girls and women into a coordinated, evidence-based, and stage-specific approach (Figure 1). By embedding a life course lens and promoting harmonization across clinical specialties, this framework represents a paradigm shift in how care is conceptualized, organized, and delivered. It addresses the distinct and recognizable stages in the female life course—perimenarche (S1), ovarian cycling (S2), perimenopause (S3), and postmenopause (S4)—that are driven by dynamic hormonal changes and acknowledges the interconnectedness among hormonal, psychosocial, and cardiovascular health.[11] This scientific statement uses a crosswalk of major society guidelines, in addition to a review of the scientific literature and expert consensus discussion, to provide consensus-based clinical guidance (Figure 2) and identify fundamental gaps related to the science, screening, prevention, and management of CVD for girls and women. All clinical considerations detailed in this scientific statement are anchored in the principles of person-centered decision-making (also known as shared decision-making).[12]

Of note, this document uses the words girls and women to refer to individuals who had female sex assigned at birth, and not to indicate gender identity, which represents a social construct. Correspondingly, this document does not address cardiovascular issues that are specifically relevant in the provision of transgender care. Although the document integrates guidance across multiple societies within an overarching life course framework, the goal of the clinical guidance is predicated on cardiovascular impact, which includes health behaviors, health factors, and risk of CVD. Therefore, other areas of health in women that are important for overall well-being (eg, bone health, cancer screening, vaccination, alcohol and substance use) are not covered. Moreover, detailed care of the pregnant woman with CVD or management of a pregnant woman at high risk of CVD is not covered, as these approaches are incorporated into existing clinical practice guidelines for heart failure (HF),[13] valvular heart disease,[14] and congenital heart disease.[15]

SEX HORMONES AND CARDIOVASCULAR BIOLOGY

Sex differences in risk of CVD, onset of overt CVD, clinical CVD phenotypes, and CVD outcomes are due to the complex interplay of multiple biologic determinants (including sex chromosomes, sex hormones [both endogenous and exogenous exposure], and cellular metabolism) and nonbiologic factors, including gender norms and social drivers of health (SDOH).[10,16] The intersection of sex and gender, along with other intersectional identities and SDOH, plays a fundamental role in health and disease across the life course, and is discussed in further detail in the accompanying presidential advisory and in related scientific statements and society documents.[17–21]

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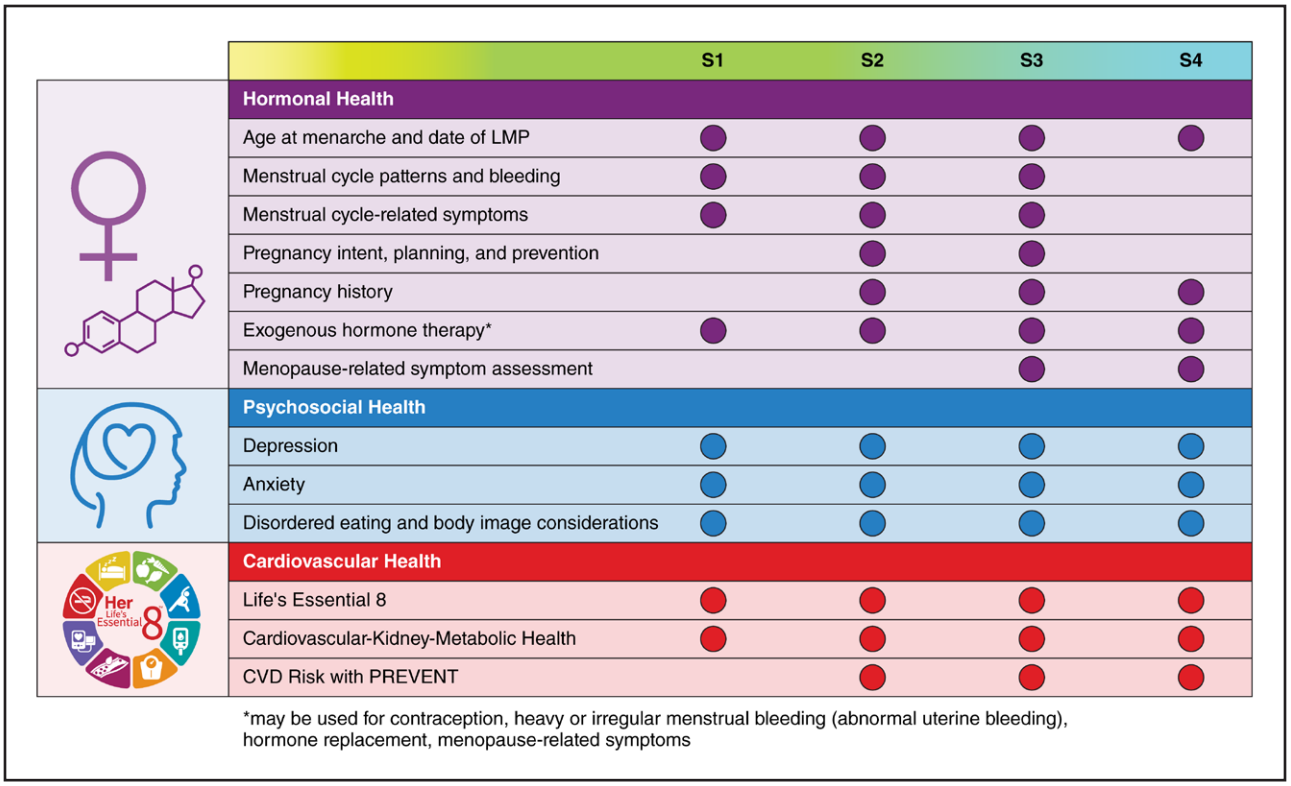


Figure 1. Female Life Stage chart assessing interconnected domains of hormonal, psychosocial, and cardiovascular health. A representative well-visit chart integrating hormonal, psychosocial, and cardiovascular health across the Female Life Stages, highlighting stage-specific assessments and core evaluations within each interconnected domain. S1 represents perimenarche; S2, ovarian cycling; S3, perimenopause; and S4, postmenopause. CVD indicates cardiovascular disease; LMP, last menstrual period; and PREVENT, Predicting Risk of CVD Events.

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Endogenous sex hormones, coordinated through the hypothalamic-pituitary-ovarian axis (estrogens, progesterone, androgens, and gonadotropins [follicle-stimulating hormone and luteinizing hormone]), are among the most dynamic drivers of sex differences across the female life course. Additional regulators, such as inhibin, anti-Müllerian hormone, and sex hormone binding globulin, further modulate hypothalamic-pituitary-gonadal axis feedback, follicular dynamics, and sex steroid bioavailability, respectively.[22,23] Circulating estradiol exerts effects on cells through multiple molecular pathways, including activation of estrogen receptor α on endothelial cells, which stimulates nitric oxide production.[22] In addition, estradiol promotes vasodilation by stimulating endothelial prostacyclin,[24] and more broadly, estradiol has antioxidant and anti-inflammatory properties that enhance nitric oxide bioavailability in the premenopausal years.[25,26] Estrogen modulates lipid metabolism through upregulation of hepatic low-density lipoprotein receptors. In preclinical models, aromatase-knockout mice, which cannot synthesize endogenous estrogen, have more adiposity, less lean body mass, and higher cholesterol levels than wild-type mice, supporting the causal role of estrogen on adiposity, metabolic dysregulation, and cardiovascular health.[27] This evidence cumulatively illustrates that endogenous sex hormones are fundamental modifiers of cardiovascular health across the life span of girls and women. It is important to note that exogenous hormone therapy does not automatically confer the same protective benefits observed with endogenous sex hormones. Rather, hormone therapy may result in either no difference in or increased CVD risk depending on age, timing, formulation, and individual antecedent patient risk.[28]

CLINICAL RATIONALE FOR THE FEMALE LIFE STAGE CONSTRUCT

The clinical rationale to anchor the Female Life Stage framework on changes in endogenous sex hormones builds upon the biologic rationale and is based on 3 key considerations. First, endogenous sex hormones are dynamic and fluctuate across the life course, and resultant physiologic changes are readily accessible without laboratory or imaging measurements (eg, menstrual cycle patterns). Second, life stages due to hormonal transitions (eg, perimenarche, perimenopause, peripregnancy) represent periods of increased vulnerability to different psychosocial stressors and CVD risk and mark key times for earlier prevention and intervention efforts. Third, this framework makes explicit the interconnectedness of hormonal, psychosocial, and cardiovascular health.

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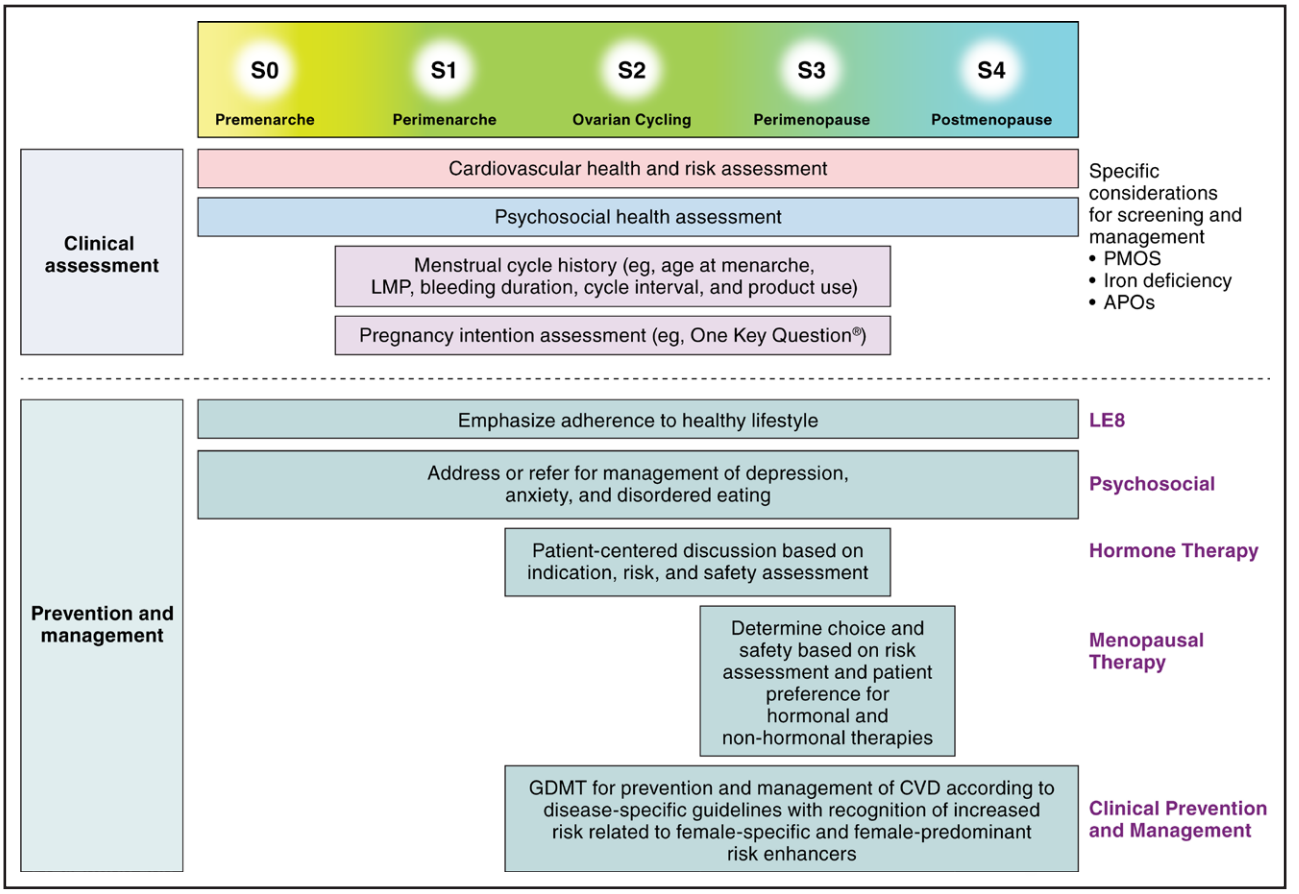


Figure 2. Clinical assessment, prevention, and management of cardiovascular disease in girls and women across Female Life Stages.

Overview of a practical clinical algorithm that begins with female life staging followed by clinical assessment and prevention and management of cardiovascular disease. This approach supports patient-centered, stage-informed cardiovascular care. APO indicates adverse pregnancy outcome; CVD, cardiovascular disease; GDMT, guideline-directed medical therapy; LE8, Life's Essential 8; LMP, last menstrual period; and PMOS, polyendocrine metabolic ovarian syndrome.

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In addition, there are female-specific conditions that confer increased cardiovascular risk across the life span; some examples include adverse pregnancy outcomes (APOs), irregular menstrual cycles, and polyendocrine metabolic ovarian syndrome (PMOS; formerly known as polycystic ovary syndrome).[29] Higher CVD risk may also be conferred by female-specific (eg, premature ovarian insufficiency) or female-predominant (eg, autoimmune disorders) conditions, as well as by both surgical (eg, premenopausal bilateral oophorectomy) or medical (eg, hormone therapies, such as for contraception, fertility treatment, or menopausal hormone therapy) changes to the hormonal milieu. Changes in bleeding patterns may be caused by oligo-ovulation or anovulation manifesting as oligomenorrhea or amenorrhea (eg, functional hypothalamic amenorrhea resulting in hypoestrogenism).[30] Therefore, irregular menstrual cycles or abnormal uterine bleeding may contribute to CVD risk in multiple ways.

Beyond female-specific cardiovascular risk factors, female-specific differences in symptom presentation, clinical phenotypes, and outcomes exist as well. For example, there are some CVD conditions that occur only in women, such as those related to pregnancy (eg, peripartum cardiomyopathy, hypertensive disorders of pregnancy [HDPs]). Some CVD conditions may occur in both sexes, but are more common in female patients, such as Takotsubo cardiomyopathy, spontaneous coronary artery dissection, and cardiomyopathy due to chemotherapy. Because Takotsubo cardiomyopathy occurs predominantly in postmenopausal women, clinicians should maintain heightened awareness in S4.[31] Spontaneous coronary artery dissection typically occurs in S3 or S4 but can also occur in S2, particularly as a common cause of pregnancy-associated myocardial infarction.[32] In breast cancer, the most common cancer in women, chemotherapy, and surgical oophorectomy in S2 or S3 contribute to premature and abrupt onset of menopause, which is associated with heightened risk of CVD. Cardiotoxicity due to chemotherapy should be evaluated and monitored across all life stages.[33]

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GUIDELINE CROSSWALK FOR FEMALE-SPECIFIC RECOMMENDATIONS

To provide harmonized, evidence-based clinical guidance for the prevention and management of CVD in girls and women, the AHA convened a Science Advisory Group with broad multidisciplinary expertise in collaboration with AAP, ACOG, and SMFM. The Science Advisory Group comprised representatives from multiple clinical specialties, including endocrinology, cardiology, hematology, maternal-fetal medicine, neurology, nursing, obstetrics and gynecology, pediatrics, adolescent medicine, and primary care; individuals with multiple areas of research expertise, including epidemiology, vascular biology, outcomes research, and implementation science; and patient advocates, who shared their experiences as women with CVD.

Harmonization Across Multiple Societies' Guidelines

First, we systematically identified and compiled available guideline recommendations from the AHA (Supplemental Table 3; led by the AHA/American College of Cardiology [ACC] Joint Committee on Clinical Practice Guidelines with engagement of other subspecialty organizations), AAP, ACOG, and SMFM, as well as selected guidelines from other organizations (eg, American Psychiatric Association, International PCOS Network, The Menopause Society [formerly known as North American Menopause Society], American Society of Hematology, Women's Preventive Services Initiative, Society for Adolescent Health and Medicine). This approach is consistent with established methods for structured review and comparison of clinical practice guidelines.[34,35] Female-specific guideline recommendations relevant to cardiovascular care in girls and women were extracted and organized by clinical domain and Female Life Stage (Table 1) with specific attention to female-specific CVD risk enhancers (Supplemental Table 4). The following sections detail the evidence-based, stage-specific guidance as determined from this process across each domain of hormonal, psychosocial, and cardiovascular health. An overview of the subsequent integrative approach detailed in the following sections is provided in Supplemental Figure 1 with validated screening tools for each domain described in Supplemental Table 5. These sections build on foundational clinical guidance established in practice guidelines that is similar for both male and female patients (eg, optimizing cardiovascular health; Supplemental Table 6) and address how health behaviors and factors may be influenced by female-specific anatomy and physiology, warranting specific clinical considerations for girls and women across Female Life Stages.

Review of Sex-Specific Language in Existing Guidelines and Society Documents

To characterize the current state of sex-inclusive evidence within the literature cited in guideline documents, we also performed an in-depth review of the latest AHA/ACC guidelines and AHA scientific statements. Full-text documents were reviewed for sex-specific terminology using a classification framework developed for this analysis (Supplemental Methods). All clinical trial references (defined by PubMed clinical trial type or ClinicalTrials.gov identifier) were extracted (n=3361 unique clinical trial references). Across 338 documents examined, only 68% cited at least 1 clinical trial containing any sex-related term and only 19% cited at least 1 trial with interaction testing by sex or sex-stratified results. The substantial gaps in sex-inclusive evidence, reporting, and integration reflect upstream factors such as sex- and gender-based bias and discrimination, the relative underfunding of women's health, underrepresentation of women in CVD clinical trials, and inconsistent consideration of sex as a biologic variable in analyses.

HORMONAL HEALTH ACROSS FEMALE LIFE STAGES

Across the life course, stage-specific assessment of menstrual history should be conducted (Table 2 and Supplemental Figure 2) to comprehensively evaluate cardiovascular health. In menstruating patients (which may include S1–S3), this should include a detailed menstrual history, including age at menarche, menstrual cycle patterns and regularity, amount of menstrual bleeding, and menstrual-related symptoms. ACOG designates the menstrual cycle as a vital sign.[36] Aligned with this principle, ACOG recommends monitoring menstrual patterns at every preventive or comprehensive visit, which includes visits focused on preventive cardiovascular care. Onset of menses also introduces prescribing considerations for cardiovascular medications (eg, antiplatelets, anticoagulants) that may have adverse effects on menstrual cycle bleeding. Factors during the perimenopause (S3) and postmenopause (S4) stages, such as age at menopause, cause of menopause (ie, natural or iatrogenic), and vasomotor symptom burden, are also associated with CVD risk.[37] As covered in greater detail in the following sections, pregnancy may unmask cardiovascular risk. Therefore, cardiovascular health should be assessed before pregnancy, during pregnancy, and postpartum. As such, clinicians providing cardiovascular care need information on hormonal health to personalize screening and preventive efforts, and to coordinate interdisciplinary care and subspecialty referrals, as needed.

Onset of Menarche

Beginning at age 8 years, clinicians should monitor for pubertal progression with anticipatory guidance for

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Table 1. Clinical Topics and Selected Guideline or Society Document Crosswalk by Female Life Stage

Female Life Stages	Female-specific clinical topics	Selected guideline or society documents for clinical guidance or considerations*
Evaluation and management of menstrual disorders		
S1–S4	Early (<10 y) menarche	ACOG Committee Opinion No. 651 (2015; reaffirmed 2025) AAP clinical report on early puberty (2016) AAP Bright Futures (4th edition)
S1–S3	Screening for or prevention of abnormal uterine bleeding (eg, heavy or irregular menstrual bleeding)	ACOG Committee Opinion No. 785 (2019; reaffirmed 2023) ACOG Practice Bulletin No. 206 (2019) ACOG Committee Opinion No. 557 (2013; reaffirmed 2026)
S1–S3	Use of dual antiplatelet or anticoagulation therapy in menstruating patients	2026 AHA/ACC pulmonary embolism guideline ASH Education Program (DeLoughery and Bannow; 2022)
S1–S4	PMOS (formerly known as PCOS)	2023 PCOS International Guideline ACOG Practice Bulletin No. 194 (2018; reaffirmed 2025) AAP Clinical Practice Guideline on Obesity (2023)
S1–S3	Dysmenorrhea and endometriosis	ACOG Clinical Practice Guideline No. 11 (2026)
S1–S4; peripregnancy	Evaluation and management of iron deficiency	ACOG Committee Opinion No. 785 (2019; reaffirmed 2023) ACOG Committee Opinion No. 557 (2013; reaffirmed 2026) ASH Education Program (Ning and Zeller; 2019) 2025 *Lancet* Haematology expert consensus
Screening and management of menopause-related symptoms or disorders		
S3–S4	Screening and management of vasomotor symptoms	2022 North American Menopause Society Hormone Therapy Position Statement 2023 North American Menopause Society Nonhormone Therapy Position Statement ACOG Practice Bulletin No. 141 (2014; reaffirmed 2024) International Menopause Society Recommendations (2025) 2020 AHA scientific statement on menopause
S3–S4	Premature ovarian insufficiency (either spontaneous or iatrogenic due to surgical or medical intervention) and early menopause (either spontaneous or iatrogenic due to surgical or medical intervention)	ACOG Committee Opinion No. 698 (2017; reaffirmed 2025) 2019 ACC/AHA primary prevention guideline 2020 AHA scientific statement on menopause ACOG Committee Opinion No. 605 (2014; reaffirmed 2025) ESHRE/ASRM/CREWHIRL/IMS guideline on premature ovarian insufficiency (2025)
Evaluation and management of psychosocial health		
S1	Depression and anxiety screening	ACOG Clinical Practice Guideline No. 4 (2023) ACOG Clinical Practice Guideline No. 5 (2023) AAP clinical report (Weitzman et al; 2025) AAP Endorsed Clinical Practice Guideline (2018; reaffirmed 2025) AAP Bright Futures (2025 Periodicity Schedule) WPSI Well Women Chart (2026) 2021 AHA scientific statement on psychosocial health
S1	Disordered eating behaviors, eating disorders, and body image	AAP clinical report (Hornberger and Lane; 2021) AAP clinical practice guideline on obesity (2023) AAP Bright Futures (4th Edition) ACOG Committee Opinion No. 740 (2018; reaffirmed 2025) APA guideline for eating disorders (2023)
S2	Premenstrual syndrome/premenstrual dysphoric disorder	ACOG Clinical Practice Guideline No. 7 (2023)
Pregnancy and postpartum	Peripartum depression, sleep disturbance	ACOG Clinical Practice Guideline No. 4 (2023) AAP Policy Statement (Earls et al; 2019) 2025 AHA scientific statement on psychological health

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Table 1. Continued

Female Life Stages	Female-specific clinical topics	Selected guideline or society documents for clinical guidance or considerations*
Cardiovascular implications of exogenous hormone therapy in female patients		
S1–S3	Combined hormone therapy	CDC US Medical Eligibility Criteria for Contraceptive Use (2024) ACOG Practice Bulletin No. 206 (2019) AAP Contraception for Adolescents: Policy Statement and Clinical Report (2025) 2025 AHA/ACC/multisociety high BP guideline
S3–S4	Menopausal hormone therapy or hormone replacement therapy	2022 North American Menopause Society Hormone Therapy Position Statement ACOG Practice Bulletin No. 141 (2014; reaffirmed 2024) European Society of Endocrinology Guideline for Menopause (2025)
Female-specific considerations for the peripregnancy period		
Prepregnancy and pregnancy	Assessment for and avoidance of teratogenic medication if intending pregnancy or during pregnancy	ACOG Committee Opinion No. 762 (2019; reaffirmed 2024) ACOG Practice Bulletin No. 212 (2019; reaffirmed 2025) ACOG Practice Bulletin No. 203 (2019; reaffirmed 2024)
Postpartum	Assessment of lactation safety for cardiovascular medications in postpartum women	SMFM Statement Checklist for Postpartum Discharge for HDP (2020; reaffirmed 2025) ACOG Practice Bulletin No. 212 (2019; reaffirmed 2025)
Prepregnancy and pregnancy	Chronic hypertension in pregnancy	ACOG Practice Bulletin No. 203 (2019; reaffirmed 2024) ACOG Practice Bulletin No. 212 (2019; reaffirmed 2025) 2025 AHA/ACC/multisociety high BP guideline
Pregnancy and postpartum	New-onset hypertensive disorders of pregnancy	ACOG Practice Bulletin No. 212 (2019; reaffirmed 2025) ACOG Practice Bulletin No. 222 (2020; reaffirmed 2026) SMFM Statement Checklist for Postpartum Discharge for HDP (2020; reaffirmed 2025) 2025 AHA/ACC/multisociety high BP guideline 2026 AHA/ACC/multisociety dyslipidemia guideline ACOG Committee Opinion No. 743 (2018; reaffirmed 2023)
Pregnancy and postpartum	Gestational diabetes	ACOG Practice Bulletin No. 190 (2018; reaffirmed 2026) ADA 2025 Standards of Care
Pregnancy and postpartum	Other APOs	ACOG Practice Bulletin No. 212 (2019; reaffirmed 2025) ACOG Committee Opinion No. 736 (2018; reaffirmed 2025) SMFM Special Statement on Postpartum Visit Checklists (Morgan et al; 2022) 2019 AHA/ACC primary prevention guideline 2021 AHA scientific statement on APOs 2026 AHA/ACC/multisociety dyslipidemia guideline
Pregnancy and postpartum	VTE	ASH Guidelines for Management of VTE in the Context of Pregnancy (2018) 2026 AHA/ACC pulmonary embolism guideline
Care transitions and interdisciplinary care for girls and women across the life course		
S1–S2	Adolescence to young adult transitions	AAP/AAFP/ACP clinical report (2018) AAP report on transitions (2025) WPSI Well-Woman Chart (2026)
S1–S3	Pregnancy intention, prepregnancy planning, and counseling to promote cardiovascular health	ACOG Committee Opinion No. 762 (2019; reaffirmed 2024) ACOG Practice Bulletin No. 201 (2018; reaffirmed 2025) ACOG Practice Bulletin No. 230 (2021; reaffirmed 2025) 2023 AHA Scientific Statement on optimizing prepregnancy cardiovascular health
Pregnancy	Multidisciplinary cardio-obstetrics team–based care with prepregnancy CVD	ACOG Committee Opinion No. 813 (2020; reaffirmed 2024) ACOG Practice Bulletin No. 212 (2019; reaffirmed 2025) ESC guideline for the management of CVD and pregnancy (2025)
Postpartum	Structured postpartum CVD transition and interpregnancy planning	ACOG Committee Opinion No. 736: (2018; reaffirmed 2025) SMFM Special Statement on Postpartum Visit Checklists (Morgan et al; 2022) ACOG/SMFM Obstetric Care Consensus No. 8 (2019; reaffirmed 2025)

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Table 1. Continued

Female Life Stages	Female-specific clinical topics	Selected guideline or society documents for clinical guidance or considerations*
S1–S4	Interdisciplinary care coordination	2018 AHA/ACOG presidential advisory (Brown et al) 2024 AHA scientific statement on patient-centered adult cardiovascular care 2026 AHA science advisory on Comprehensive Centers of Excellence of Women's Cardiovascular Care

AAFP indicates American Academy of Family Physicians; AAP, American Academy of Pediatrics; ACC, American College of Cardiology; ACOG, American College of Obstetricians and Gynecologists; ACP, American College of Physicians; ADA, American Diabetes Association; AHA, American Heart Association; APA, American Psychiatric Association; APO, adverse pregnancy outcome; ASH, American Society of Hematology; ASRM, American Society for Reproductive Medicine; BP, blood pressure; CDC, Centers for Disease Control and Prevention; CVD, cardiovascular disease; ESC, European Society of Cardiology; ESHRE, European Society of Human Reproduction and Embryology; HDP, hypertensive disorder of pregnancy; IMS, International Menopause Society; PCOS, polycystic ovary syndrome; PMOS, polyendocrine metabolic ovarian syndrome; SMFM, Society for Maternal-Fetal Medicine; VTE, venous thromboembolism; and WPSI, Women's Preventive Services Initiative.

*See Supplemental Table 12 for the expanded titles and citations of each included society document.

girls and parents regarding onset of menarche. Age at onset of menarche varies according to demographic, social, and clinical factors, with earlier age at onset associated with higher body mass index (BMI).[38] The onset of menarche should trigger ongoing assessment of menstrual cycle regularity and menstrual bleeding heaviness (period duration and volume based on product use) and their impact on cardiovascular health behaviors (eg, physical activity, sleep). In people with Müllerian agenesis (eg, absent uterus), which affects ≈1 in 5000 female individuals, intact hypothalamic-pituitary-gonadal signaling with physiologic ovarian cycling occurs in the absence of menses (ie, primary amenorrhea).[39]

Age at onset of menarche itself carries prognostic cardiovascular information, with early menarche associated with modestly increased CVD risk that is partially mediated by excess adiposity.[40,41] Consequently, early menarche is best framed as a qualitative risk enhancer that reflects an early signal of increased cardiovascular risk. Notably, current pediatric statements and guidelines on cardiovascular risk factors from the National Heart, Lung, and Blood Institute,[42] AAP,[43] AHA,[44] and American Diabetes Association[45] make no reference to menstrual health. However, the latest 2026 ACC/AHA/multisociety dyslipidemia guideline[46] incorporates early menarche (defined as age <10 years) as a CVD risk enhancer in adulthood when assessing risk for women 30 to 79 years of age.

Menstrual History Assessment

The menstrual cycle length and bleeding amount typically stabilize in the first 1 to 3 years after menarche.[36] Menstrual cycle assessment is well-established in adolescent preventive care, but the cardiovascular significance of that assessment is unevenly recognized across clinical disciplines. Ongoing assessment of menstrual cycle regularity allows identification of conditions with cardiometabolic consequences, such as hypothalamic amenorrhea, PMOS, or premature ovarian insufficiency.[47] In S1 through S3, it is also important to estimate blood loss from menstrual bleeding as part of the menstrual history, and tools are available to assess for bleeding quantity (eg, pictorial blood loss assessment chart, menstrual bleeding questionnaire).[48,49] It is important to note that menstrual patterns may be modified with use of hormone therapy, which is detailed in the following sections.

Irregular or Heavy Menstrual Bleeding

Abnormal uterine bleeding encompasses variations of menstrual bleeding patterns, including irregular menstrual bleeding or heavy menstrual bleeding (HMB).[50] Irregular menstrual bleeding is defined as variations in cycle intervals (<21 days or >35 days), which includes oligomenorrhea (infrequent, irregular cycles, defined as <8 periods/year) and secondary amenorrhea (>3 consecutive months of amenorrhea in the absence of hormone therapy). Signs of HMB can include menstrual flow for >7 days, changing soaked period products (eg, tampons, pads) more frequently than every 1 to 2 hours, or requiring >6 regular pads or tampons every 24 hours. The International Federation of Gynecology and Obstetrics defines HMB as excessive menstrual blood loss that interferes with physical, emotional, social, or material quality of life.[51]

Quantifying menstrual bleeding is recommended by ACOG and is relevant to cardiovascular health in several ways.[52] First, patterns of abnormal uterine bleeding can be due to ovulatory dysfunction (eg, oligo-ovulation or anovulation); can have physiologic, pathologic, or iatrogenic causes; and may be associated with increased CVD risk.[53] Second, cumulative blood losses from menstruation can lead to iron deficiency with or without anemia, which can limit physical activity and has been linked to increased CVD risk. Iron deficiency is highly prevalent in S1 through S3, affecting ≈38% of menstruating patients.[54,55] As such, assessment for anemia and iron deficiency is recommended, at a minimum, among female patients with HMB or symptoms of iron deficiency (eg, fatigue, lightheadedness, increased heart rate, reduced exercise tolerance, shortness of breath, hair loss, pica, restless legs).[55] For those with iron deficiency, iron supplementation and appropriate management (eg, with hormone therapy or tranexamic acid if it is related to menstrual bleeding) are recommended, whether or not anemia is present. In addition, evaluation to identify the source of iron deficiency

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Table 2. Clinical Considerations for Menstrual History and Cardiovascular Implications: Hormonal Health Assessment

Approximate age range, y	Female Life Stages	Specific clinical considerations
All	All	In all girls and women, assess and document Female Life Stage at every clinical encounter to inform cardiovascular risk assessment, medication safety, and preventive efforts (see Supplemental Figure 1 for overview figure)
<10	S0	In girls in S0, assess for onset of menarche and document age at menarche at every comprehensive or preventive visit, beginning at age 8 y until menarche occurs
10–49	S1–S3	In girls and women in S1–S3, obtain a structured menstrual history at least annually, including age at menarche, date of last menstrual period, cycle regularity, cycle length, bleeding duration, and bleeding heaviness (flow duration and volume by product use)
10–49	S1–S3	In girls and women in S1–S3 with menstrual cycle irregularity (eg, amenorrhea, oligomenorrhea) or clinical features of hyperandrogenism, evaluate for PMOS (formerly known as PCOS) and other underlying causes
10–49	S1–S3	In girls and women with a diagnosis of PMOS (formerly known as PCOS), management for PMOS should include assessment and management of hormonal, psychosocial, and cardiovascular-kidney-metabolic health
10–49	S1–S3	In girls and women with a diagnosis of PMOS (formerly known as PCOS), screen for cardiovascular-kidney-metabolic health factors (eg, body mass index, BP, lipids, hemoglobin A1c), at least annually
10–49	S1–S3	In girls and women in S1–S3 with heavy menstrual bleeding, assess for iron deficiency and anemia and replace iron, if indicated, to promote cardiovascular health
10–49	S1–S3	In girls and women in S1–S3 with heavy menstrual bleeding, assess impact of symptoms on physical activity, sleep, daily functioning, and psychosocial health
10–49	S1–S3	In girls and women in S1–S3 with heavy menstrual bleeding, discuss pharmacotherapeutic options to address symptoms, including hormone therapy (eg, progestins or combined hormonal contraceptives) and antifibrinolytics, when indicated
10–49	S1–S3	In girls and women in S1–S3 receiving dual antiplatelet therapy or anticoagulant therapy, assess for heavy menstrual bleeding and provide anticipatory counseling about risk of heavy menstrual bleeding and consider treatment to minimize menstrual bleeding
All	All	In girls and women, routinely incorporate dysregulation of hormonal health (eg, early menarche, PMOS, premature ovarian insufficiency, early menopause) into cardiovascular risk assessment as risk enhancers to inform patient-centered decision-making on preventive therapies

PCOS indicates polycystic ovary syndrome; and PMOS, polyendocrine metabolic ovarian syndrome.

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should be performed. Third, the degree of menstrual bleeding can be exacerbated by evidence-based therapies, such as anticoagulant or antiplatelet therapies for treatment of venous thromboembolism (VTE [ie, deep vein thrombosis or pulmonary embolism]), stroke, coronary heart disease, or peripheral artery disease.[56] The risk of HMB varies by anticoagulation or antiplatelet regimen. Up to 70% of menstruating patients receiving anticoagulation experience abnormal menstrual bleeding or HMB.[57] Notably, rivaroxaban is associated with higher rates of HMB compared with warfarin, whereas apixaban appears to have similar rates to low-molecular-weight heparin.[58–60] However, data on changes in menstrual bleeding for antiplatelet therapies are sparse, with dual antiplatelet therapy estimated to have a greater increased risk than single antiplatelet therapy.[61] Thus, initiation of anticoagulation or antiplatelet therapy in women in S1 through S3 should be accompanied by anticipatory guidance regarding potential changes to menstrual bleeding and options for minimizing bleeding to avoid interruption of evidence-based therapy.[62]

Dysmenorrhea

Menstrual cycle symptoms and related disorders may affect mood, health behaviors (eg, sleep, physical activity), and CVD risk. Dysmenorrhea, defined as cramping pain in the lower abdomen, is common, affecting the majority of adolescent girls (up to ≈70%), approximately doubling the odds of insomnia and poor sleep quality.[7,63,64] Therefore, clinicians should specifically ask about menstrual-related sleep disruption. Although it is not known whether treating dysmenorrhea improves sleep and other cardiovascular health metrics within the Life's Essential 8 (LE8) construct, managing symptoms is integral to addressing overall wellbeing. Dysmenorrhea may also be caused by underlying disorders, and evaluation to determine its underlying cause should be undertaken. For example, endometriosis, a cause of dysmenorrhea, has been associated with increased stroke risk and is recognized in the 2024 AHA/American Stroke Association stroke prevention guideline as a female-specific stroke risk factor.[65]

Premenstrual disorders (eg, premenstrual syndrome [PMS], premenstrual dysphoric disorder) are also relatively common: ≈25% of menstruating patients have PMS and ≈2% to 5% have premenstrual dysphoric disorder, with prevalence estimates higher in adolescents. These conditions are characterized by psychosocial distress, functional impairment,[66] and sleep disturbance, and are associated with increased CVD risk.[66–69]

Polyendocrine Metabolic Ovarian Syndrome

PMOS, formerly known as polycystic ovary syndrome, is one of the most common but most underrecognized

endocrine disorders in girls and women.[29] PMOS is diagnosed by the presence of 2 of 3 features: hyperandrogenism (clinical or biochemical), ovulatory dysfunction, and either polycystic ovarian morphology or elevated anti-Müllerian hormone; it is commonly associated with metabolic abnormalities. Clinical markers may include menstrual cycle irregularities (ie, amenorrhea, oligomenorrhea), acne, hirsutism, acanthosis nigricans, and insulin resistance. PMOS can also result in anovulation and infertility and, as a result, is often first diagnosed in S2. Identification of women with potential PMOS should prompt referral to a clinician with expertise in the condition (eg, gynecologist, endocrinologist, adolescent medicine) and cardiovascular risk assessment. The 2023 international evidence-based guideline for the assessment and management of polycystic ovary syndrome details stage-specific diagnostic criteria for S1 versus S2 and recommends screening for CVD risk at diagnosis regardless of BMI.[47,70] Oral glucose tolerance testing is recommended as the most accurate test for assessment of glycemic status, but fasting plasma glucose or glycated hemoglobin may be considered as alternatives. Data remain limited on how continuous glucose monitoring may be incorporated into screening and management of women with PMOS.

Available behavioral (eg, mood management, weight loss) and pharmaceutical treatments for PMOS that target hormonal health (eg, combined hormonal contraceptives [CHCs]) and cardiometabolic health (eg, metformin, glucagon-like peptide-1 [GLP-1]–based therapies) have cardiovascular implications.[71,72] Prevention of diabetes, optimizing cardiometabolic health, and prevention of CVD are key modifiable goals in women with PMOS, including through lifestyle interventions and approaches such as the Diabetes Prevention Program.[73] If pregnancy is planned, monitoring and prevention for APOs is also recommended, as PMOS is associated with a higher risk of gestational diabetes and HDPs.[74] Pediatric cardiovascular guidelines have not historically listed PMOS as a risk enhancer,[42] but the most recent 2026 ACC/AHA/multisociety Dyslipidemia Guideline[46] and 2026 AHA/ACC/multisociety Cardiovascular Kidney Metabolic Guideline[75] both included it as a risk enhancer in adults.[71]

Menopausal Symptom Screening

The onset of menopause-related symptoms typically occurs in perimenopause (S3), which is a key inflection point for cardiovascular health, with many women having ongoing menopause-related symptoms into early S4. There is substantial and irregular fluctuation in estradiol levels. In addition, the follicle-stimulating hormone level increases, and, in S4, there is permanent cessation of ovarian function. The beginning of perimenopause is marked by menstrual cycle irregularities (persistent ≥7-day variation in consecutive cycle length).[76] The median age at physiologic menopause is ≈51 years in the United States, and 90% of women experience menopause between ages 45 and 56 years.[77] However, early menopause (ie, 40–44 years) and premature menopause (ie, <40 years; also known as premature or primary ovarian insufficiency) affect ≈8% to 12% and 1% to 3.5% of women in the United States, respectively.[37] Menopause occurring <45 years of age is identified as a cardiovascular risk enhancer, underscoring the importance of assessing and documenting age at menopause.[78]

The diagnosis of perimenopause is made based on clinical presentation, not on hormone levels, and it can begin up to 10 years before the final menstrual period. Menstrual cycle irregularity in the absence of hormone therapy, particularly in the presence of PMS-like or menopause-related symptoms, can help identify onset of perimenopause (S3). Several validated instruments for menopause symptoms have been used in research studies (eg, Menopause Rating Scale, Menopause-Specific Quality of Life Questionnaire, Greene Climacteric Scale, Kupperman Index). Routine measurement of estradiol and follicle-stimulating hormone levels is not recommended for diagnosis of perimenopause, in large part due to their variability in this stage. Given that the frequency and severity of vasomotor symptoms (eg, hot flashes, night sweats, and palpitations, which reflect hormonally mediated thermoregulatory dysfunction), poor sleep, depressed mood, and anxiety have cardiovascular implications, systematic screening for menopause-related symptoms should be incorporated into cardiovascular care and may be considered beginning at age 40 years.[79] However, clinicians and patients should be aware that menopause-related symptoms can begin <40 years of age and should be appropriately recognized and managed. In addition, appropriate recognition of menopause due to iatrogenic causes, such as chemotherapy or surgical bilateral oophorectomy, is important to inform cardiovascular risk assessment and management.

PSYCHOSOCIAL HEALTH ACROSS FEMALE LIFE STAGES

Depression, anxiety, and eating disorders are particularly relevant to female-specific cardiovascular care because they are more prevalent among girls and women, beginning at menarche, and are associated with increased CVD risk (Table 3). Moreover, the distinct stages that represent transition periods in the female life course (ie, perimenarche, peripartum, perimenopause) represent potential for dynamic changes in psychosocial health and wellbeing. As such, clinicians providing cardiovascu-

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Table 3. Clinical Considerations for Female-Specific Psychosocial Health Assessment

Approximate age range, y	Female Life Stages	Guidance
≥13	S1–S4	In girls and women age ≥13 y, screen for anxiety using a validated screening instrument, such as GAD-2, and perform further evaluation with GAD-7 in those with a positive screen with follow-up management or referral, as indicated
≥12	S1–S4	In girls and women age ≥12 y, screen for depression using a validated screening instrument, such as PHQ-2, and perform further evaluation with PHQ-9 in those with a positive screen with follow-up management or referral, as indicated; in adolescents, PHQ-A may be used as an alternate instrument
≥12	S1–S3	In pregnant and postpartum patients, screen for anxiety and depression symptoms using validated perinatal screening instruments with appropriate evaluation, follow-up, and treatment, when indicated (eg, Edinburgh Postnatal Depression Scale)
≥11	S1–S4	In girls and women age ≥11 y, screen for eating disorders or disordered eating behaviors, including dietary quality, meal frequency, eating patterns, and body image concerns, at comprehensive or preventive encounters
≥40	S3–S4	In women beginning at age 40 y, assess for menopause-related symptoms, including psychosocial symptoms, vasomotor symptoms, sleep disturbance, and quality of life impairment; counsel on therapeutic options to address symptoms (including pharmacologic hormonal and nonhormonal options as well as behavioral strategies [eg, cognitive behavioral therapy])

GAD-2 indicates Generalized Anxiety Disorder 2-item scale; GAD-7, Generalized Anxiety Disorder 7-item scale; PHQ-2, Patient Health Questionnaire 2-item scale; PHQ-9, Patient Health Questionnaire 9-item scale; and PHQ-A, Patient Health Questionnaire for adolescents.

lar care across various care settings (eg, primary care, pediatrics, gynecology, cardiology) should incorporate routine screening with validated measures, such as the Patient Health Questionnaire 2-item scale and Generalized Anxiety Disorder 2-item scale, as an initial assessment, followed by screening with the full Patient Health Questionnaire 9-item scale and Generalized Anxiety Disorder 7-item scale for those who screen positive on the short form, and referral for mental healthcare based on screening evaluations, as indicated.[80,81] Adverse psychosocial health is more common and may be exacerbated in the context of adverse SDOH.

Depression and Anxiety

Major professional societies endorse routine psychosocial health screening beginning in adolescence for all female patients. The AAP, ACOG, and Women's Preventive Services Initiative recommend screening for depression in female patients 12 years or older and screening for anxiety in those 13 years or older, and the AAP recommends evaluation for anxiety beginning at 8 years of age.[80,82–85] There is frequent co-occurrence of these disorders. However, optimal screening intervals are unknown. Validated tools for screening (Supplemental Tables 7 and 8) include the Patient Health Questionnaire 9-item scale for depression (ages ≥12 years), SCARED (Screen for Child Anxiety Related Emotional Disorders; ages 8–18 years) or the Generalized Anxiety Disorder 7-item scale (ages ≥13 years) for anxiety, and the Patient Health Questionnaire 4-item scale (ages ≥18 years) for efficient combined initial screening, with reflexive full assessment following a positive screen.[86] Co-occurrence of mental health conditions with CVD can raise concerns for delayed diagnosis when cardiovascular symptoms are inappropriately attributed to psychological causes without evaluation.[87] Screening for psychosocial symptoms does not obviate the need to evaluate cardiovascular causes for symptoms that may commonly overlap with anxiety (eg, palpitations, chest pain); evidence suggests that women are more likely to be misdiagnosed with anxiety when presenting with acute coronary syndromes.[88]

Eating Disorders, Disordered Eating Behaviors, and Body Image Considerations

Annual assessment for eating disorders and disordered eating behaviors is advised by several professional societies and has important implications for cardiovascular care.[89,90] The AAP recommends annual screening for disordered eating and body image disorders in all female individuals ≥11 years of age,[91] whereas ACOG recommends evaluating patients who are identified to be at risk for eating disorders in S1 through S3.[92] Menstrual dysregulation often occurs in the setting of an eating disorder or energy imbalance and should also prompt disordered eating screening. Providing body-positive education, using sensitive and nonstigmatizing language, and engaging in motivational interviewing are encouraged by both the AAP and the AHA, given their positive impact on cardiovascular health behaviors.[93,94]

Clinicians should screen for disordered eating behaviors before praising weight loss and assess for body image dissatisfaction before providing lifestyle counseling. Simple questions (eg, "How do you feel about your weight?") may reveal concerns regarding weight stigma; clinicians should explore the patient's own perspective given cultural variation in body size perceptions. All professional societies emphasize systems-level support for managing positive screens. For adolescents, the AAP has developed implementation resources for assessing body image (Supplemental Table 9)[95]; for adults, the American Academy of Family Physicians recommends the SCOFF questionnaire[96] to screen for eating disorders. ACOG recommends simple screening questions. Of note, some have expressed concern that the increasing use of GLP-1–based therapies will drive an increasing

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prevalence of disordered eating and body image disorders.[97]

Social Factors

There is also broad agreement across major professional societies for standardized screening of SDOH. Several validated tools exist that are not sex-specific (eg, Health Leads). However, US women compared with men are more likely to experience poverty, food insecurity, and single parenthood, and to be caregivers, all of which affect health.[98–100] In addition, a trauma-informed approach is recommended when screening for adverse childhood experiences, particularly sexual abuse, which are more common in girls and contribute to poor psychosocial and cardiovascular health.[101,102] Intimate partner violence affects 25% of US women and is associated with increased CVD risk.[103] Posttraumatic stress disorder is particularly common among female Veterans, a high-risk population that warrants personalized care.[104] All professional societies emphasize the importance of practice-level and systems-level support for managing positive screens and highlight potential clinical and ethical risks of screening without the ability to assess, triage, refer, and follow-up an individual with a positive screen.[105] Ultimately, addressing cardiovascular health in women will require integration of clinical, social, and structural solutions.

CARDIOVASCULAR HEALTH ACROSS FEMALE LIFE STAGES

Sex-agnostic screening, prevention, and management of CVD is well-detailed in specific AHA/ACC guidelines focused on cardiovascular risk factors (eg, blood pressure [BP], dyslipidemia), primary prevention, and specific cardiovascular conditions (eg, HF, acute coronary syndromes, valvular heart disease). The preceding sections addressed hormonal health (eg, menstrual disorders, menopause-related symptoms) and psychosocial health (eg, depression, anxiety, disordered eating), both of which directly influence cardiovascular health behaviors, health factors, and CVD risk. This section addresses optimizing cardiovascular health, assessing CVD risk, and promoting CVD prevention across the female life course (Table 4).

Cardiovascular health assessment should include the LE8 components, which include health behaviors (ie, diet, physical activity, nicotine exposure, sleep) and health factors (ie, BMI, BP, lipid levels, glucose levels). LE8 is relevant in all stages, although different stages warrant a different emphasis for cardiovascular care. In S1, the focus is on establishing healthy behaviors and identifying emerging risk factors; in S2 and S3, CVD risk stratification (using tools such as the PREVENT equations [Predicting Risk of CVD Events]) and attention to female-specific risk enhancers become increasingly relevant; in S4, when clinical CVD typically emerges, disease management joins prevention as a priority for atherosclerotic CVD (ASCVD, which includes coronary heart disease, peripheral artery disease, stroke) and HF. The following subsections address female-specific considerations at each stage, emphasizing when sex-specific factors should inform screening interpretation, risk assessment, treatment decisions, and care transitions.

Female Life Stage 1 (S1)

Cardiovascular health assessment in S1 should include the LE8 components, with female-specific considerations in timing of assessment and interpretation of results. Current pediatric guidelines (eg, National Heart, Lung, and Blood Institute, AAP, AHA/ACC) recommend universal assessment for BMI and BP annually, lipid testing at ages 9 through 11 years and 17 through 21 years (and selectively at other ages based on risk), and glycemic status based on risk.[46] Sex is incorporated into interpretation for BMI and BP with sex-specific percentiles. However, lipid and glucose thresholds are defined by sex-agnostic levels.[43,93] Recommended timing of lipid testing at ages 9 through 11 years and 17 through 21 years is intended to account for physiologic changes of puberty on lipid levels. However, pubertal staging itself is not incorporated to define thresholds for elevated lipid levels despite evidence that pubertal stage can vary for the same chronologic age. Derivation studies for sex- and pubertal-stage-specific thresholds linked to outcomes are needed.

The transition from pediatric to adult care represents a high-risk period for loss of information related to female-specific preventive care. Existing transition frameworks (eg, Got Transition [AAP]) do not address female-specific care considerations, such as menstrual history or disorders, as a required element of the transition medical summary.[106] The transition process for adolescent female patients should include anticipatory guidance on adult care model options (eg, gynecology, primary care, preventive cardiology) with education that CVD risk factor screening must continue and is relevant to preconception health; menstrual history (eg, menarche timing, cycle regularity) as a required element of the transition summary; and clear delineation of psychosocial concerns (eg, body image issues, evolving eating or exercise patterns, mood symptoms). Empowering the adolescent to understand her own cardiovascular risk profile, including the relevance of hormonal and psychosocial health to cardiovascular outcomes, and advocate for appropriate care is a core element of anticipatory guidance and is a principle that applies across the life course and all domains of cardiovascular health.

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Table 4. Clinical Considerations for Female-Specific Cardiovascular Health Assessment

Approximate age range, y	Female Life Stages	Guidance
≥10	S1–S4	In girls and women age ≥10 y in S1–S4, assess the influence of hormonal health (eg, menstrual disorders) and psychosocial health (eg, depression, anxiety, disordered eating) factors on cardiovascular health behaviors (eg, physical activity, diet, sleep) in the LE8
≥30	S2–S4	In women age ≥30 y in S2–S4 without established cardiovascular disease, use the female-specific PREVENT equations to estimate 10-y and 30-y risk to guide patient-centered decision-making for antihypertensive drug therapy, lipid-lowering therapy, and CKM therapies
≥30	S3–S4	In women with menopause-related symptoms interfering with daily life, incorporate 10-y risk of atherosclerotic cardiovascular disease with the PREVENT-ASCVD equations as part of a risk-based approach to determine preventive therapies for hypertension, dyslipidemia, and CKM therapies in addition to exogenous hormone therapy to address menopause-related symptoms
≥45	S3–S4	In women age ≥45 y with borderline (3%–<5%) or intermediate 10-y risk (5%–<10%) of ASCVD, consider coronary artery calcium scoring if further risk refinement is needed to determine preventive therapies or exogenous hormone therapy, if indicated

ASCVD indicates atherosclerotic cardiovascular disease; CKM, cardiovascular-kidney-metabolic; LE8, Life's Essential 8; and PREVENT, Predicting Risk of CVD Events.

Female Life Stage 2 (S2)

Cardiovascular health assessment in S2 should include assessment of LE8 components. Whereas cardiovascular health screening and scoring is not sex-specific, the estimation of short- and long-term CVD risk are based on sex-specific equations. The 2025 AHA/ACC/multisociety high BP guideline,[107] the 2026 ACC/AHA/multisociety dyslipidemia guideline,[46] and the 2026 AHA/ACC/multisociety cardiovascular-kidney-metabolic guideline[75] recommend assessment of risk of CVD, ASCVD, and HF with the sex-specific PREVENT equations for 10-year risk in adults ages 30 through 79 years of age and 30-year risk in adults 30 through 59 years of age.[108,109] In addition, at least annual screening for BMI and BP and every 4 to 6 years for lipids in healthy individuals from ages 20 through 79 years is recommended, with increased frequency if abnormalities are identified.[46,107] The estimation of 30-year risk and risk percentiles is particularly useful for women who may have low short-term risk but high long-term risk. In these women, not including 30-year risk assessment leads to the misperception that women are not at risk for CVD. In addition, qualitative risk enhancers, including early menarche, PMOS, irregular menstrual cycles, and APOs, should be incorporated into risk assessment. The 2026 ACC/AHA/multisociety dyslipidemia guideline[46] formally incorporated a selected set of sex-specific risk enhancers (ie, APOs [eg, HDPs, gestational diabetes, preterm birth], early menarche [<10 years], early menopause [<45 years], PMOS, irregular menses).[46]

Observational data also suggest that women with a history of infertility, particularly when infertility is due to PMOS, have an increased risk of coronary heart disease.[72,110] However, whether infertility serves as an early marker of CVD risk only when anovulatory disorders are present or reflects shared pathophysiologic pathways, similar to other female-specific risk enhancers (eg, APOs, early menopause), is unclear.[111] Despite this uncertainty, integration of risk enhancers offers an opportunity for more counseling, frequent screening, motivation for lifestyle optimization, and earlier consideration of drug therapy. It may be reasonable to prompt consideration of earlier initiation of preventive therapy or more intensive treatment even when 10-year risk is low for a broader set of female-specific risk enhancers, including endometriosis, infertility, and pregnancy loss.[46]

Female Life Stages 3 and 4 (S3 and S4)

The menopausal transition represents a key stage when CVD risk may accelerate due to metabolic and vascular changes that are driven by ovarian aging and the concomitant change in the hormonal milieu, including the progressive decline of estradiol, in addition to chronologic aging. The incidence of CVD <50 years of age in women is low, but the onset of perimenopause is associated with marked worsening in lipid levels (eg, increases in total and low-density lipoprotein cholesterol, ApoB [apolipoprotein B], and lipoprotein[a]), body composition (eg, central adiposity accumulation, decreased lean mass), vascular stiffening, and atherosclerosis.[37,112,113]

Cardiovascular health assessment in S3 and S4 should include assessment of LE8 components. CVD risk assessment in S3 and S4 follows the same established guidelines as discussed for S2 and should begin with calculation of 10-year risk of CVD, ASCVD, and HF with the PREVENT equations in patients 30 to 79 years of age. In addition, in patients 30 to 59 years of age, 30-year risk should also be calculated. Menopause-related symptoms that may represent cardiovascular risk enhancers (eg, frequent, persistent, or severe vasomotor symptoms; poor sleep; depressed mood; anxiety), as well as female-specific risk enhancers identified in S1 and S2, should be considered. If more information is needed to guide treatment decisions, use of coronary artery calcium scoring may be useful to guide decision-making regarding lipid-lowering therapy as well as to refine risk assessment when hormone therapy for menopause-related symptoms is being considered (Supplemental Figure 3). Given higher prevalence of HF with preserved ejection fraction in women, risk assessment of HF is particularly important, and biomarkers (eg, brain natriuretic peptide)

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or echocardiography, or both, may be useful to guide preventive efforts.[114]

Management of CVD should follow established guidelines. The most common subtypes of CVD in the postmenopausal stage (S4) are ASCVD (ie, coronary heart disease, stroke, peripheral artery disease), HF, and atrial fibrillation.[13,115–117] For CVD conditions that occur exclusively in women (eg, peripartum cardiomyopathy) or predominantly in women (eg, spontaneous coronary artery dissection, chemotherapy-associated cardiomyopathy, ischemia with nonobstructive coronary arteries, myocardial infarction with nonobstructive coronary arteries), current scientific statements and guidelines focused on these conditions are available and management considerations are not repeated here.[61,118] Sex-specific thresholds for valvular heart disease should be considered because women, compared with men, may benefit from intervention at lower absolute chamber dimensions.[119]

Peripregnancy Period

Pregnancy can be conceptualized as a "stress test" or a modifying life event that is associated with marked elevations in endogenous sex hormones along with hemodynamic and metabolic adaptations to support the fetus. Estradiol exposure in pregnancy substantially exceeds that of routine menstrual cycles or from hormone therapy. There is heightened risk for perinatal depression and anxiety, which not only affect ≈10% to 20% of pregnant and postpartum women, but also increase the chance of having a cardiovascular complication.[120] APOs, including new-onset HDPs, preterm birth, small for gestational age birth, gestational diabetes, and stillbirth are all associated with increased risk of incident CVD risk-factor development and CVD in the short and long term.[121,122] In addition, occurrence of an APO is associated with higher risk of APO recurrence in future pregnancies.

Therefore, assessment of pregnancy intent to assist with pregnancy planning or prevention, in addition to a detailed pregnancy history, should be incorporated by all clinicians providing care to women (Tables 5 and 6). This can be facilitated by discussing pregnancy intent with the One Key Question ("Would you like to become pregnant in the next year?"; Supplemental Figures 4 and 5). Approximately half of pregnancies in the United States are not planned, and thus the possibility of pregnancy, regardless of whether intent is expressed or contraception is being used, in women who are capable of pregnancy and are or may be sexually active with men, should be considered in provision of anticipatory guidance and counseling, such as with regard to medication safety with pregnancy.

Early recognition of poor cardiovascular health factors can prompt attempts at optimization before pregnancy, trigger increased surveillance during pregnancy and postpartum, and raise awareness of the potential to improve maternal (both short- and long-term) and offspring outcomes, particularly because this life period represents a time when interactions with the healthcare system are frequent. Given the growing recognition that CVD is a leading cause of maternal deaths and most maternal deaths due to CVD occur in women without a known diagnosis of CVD, the Centers for Medicare and Medicaid Services introduced a process-of-care quality measure (MIPS#496) to promote CVD risk assessment in all pregnant patients to direct further diagnostic workup and referral to primary or specialty care (eg, maternal-fetal medicine, cardiology), as indicated.[123] This measure is endorsed by the Partnership for Quality Measurement.

Prepregnancy Optimization of Cardiovascular Health

Epidemiologic data suggest that 80% of APOs may be preventable if prepregnancy cardiovascular health were optimized.[124] Therefore, greater awareness of the importance of cardiovascular health assessment and counseling in the prepregnancy period may have a considerable effect on health of women and their offspring; this topic was the focus of a previous scientific statement[125] and is highlighted in ACOG and SMFM guidelines, all of which underscore the importance of preconception consultation, particularly for women with CVD risk factors.

Prepregnancy cardiovascular health is highly relevant as it is associated with pregnancy-related outcomes and should include assessment of health behaviors and health factors (eg, LE8).[126] For example, among women with stage 2 hypertension before pregnancy, 1 in 3 will experience a pregnancy complication, and lowering BP to <140/90 mm Hg is recommended during pregnancy to improve maternal and fetal outcomes.[127] As noted in the presidential advisory, aligning and using consistent terminology of stage 2 hypertension, which is the same as chronic hypertension in pregnancy (≥140/90 mm Hg), is important to minimize confusion among clinicians and patients navigating transitions from primary care to obstetric care and back to primary care or subspecialty care. The 2025 AHA/ACC/multisociety high BP guideline promotes anticipatory guidance regarding low-dose aspirin for women with stage 2 hypertension who intend to become pregnant or who may become pregnant.[107] However, current trends in optimal BP management and low-dose aspirin for HDP prophylaxis, as well as the factors associated with adherence, are not well understood. In addition, screening, prevention, and management of obesity, diabetes, and hyperlipidemia, as well as assessment of cardiovascular health behaviors, are important for optimizing prepregnancy cardiovascular health.

Stroke in Pregnancy and the Postpartum Period

Maternal stroke, which was the focus of a recent scientific statement,[128] accounts for at least half of preeclampsia-

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Table 5. Cardiovascular Considerations for Pregnancy Planning, Pregnancy Prevention, and Safety of Medications in the Peripregnancy Period

Approximate age range, y	Female Life Stages	Specific clinical considerations
18–49	S2–S3	In all pregnancy-capable women in S2–S3, assess pregnancy intention at every clinical encounter using a standardized tool, One Key Question ("Would you like to become pregnant in the next year?"), to inform counseling on pregnancy planning, pregnancy prevention, medication safety, and cardiovascular health
10–49	S1–S2	In female patients not intending pregnancy, discuss pregnancy prevention plan and counsel on safe and effective contraception choices (ie, contraception type, hormonal content, route), accounting for cardiovascular and VTE risk based on age, risk factors, and 30-y risk calculation of ASCVD with the PREVENT equations (when indicated)
10–49	S1–S2	In female patients initiated on combined hormonal therapy for contraception, acne, heavy menstrual bleeding, PMOS, or other indication, perform routine follow-up BP assessment within 3 mo of initiation followed by regular monitoring
10–49	S1–S2	In female patients, estrogen-containing contraception (eg, ethinyl estradiol, estradiol valerate, estetrol) should be avoided for women with history of ASCVD or at high risk for ASCVD (≥10%), peripartum cardiomyopathy, valvular heart disease, VTE with ≥1 factor for recurrence, or migraine with aura; progestogen-only contraceptives are possible alternatives for pregnancy prevention or other indications for hormone therapy
18–49	S2–S3	In women considering or intending pregnancy, assess and optimize prepregnancy cardiovascular health at a pregnancy planning visit with the LE8 score; counsel regarding initiation of prophylactic aspirin for prevention of preeclampsia once pregnant, if indicated, optimally <16 wk of gestation (between 12 and 28 wk of gestation in those at risk [ie, stage 2 hypertension, at increased risk for preeclampsia*])
18–49	S1–S3	In women who intend to become pregnant in the near future, who are actively attempting to become pregnant, or who are pregnant, assess safety of current cardiovascular medications, if any, and modify, if indicated, to avoid fetal teratogenic exposure (see Supplemental Table 10 for common cardiovascular medications with teratogenic potential)
18–49	S1–S3	In pregnant women with stage 2 hypertension (≥140/90 mm Hg), use preferred first-line agents in pregnancy (eg, labetalol, extended-release nifedipine) to target a BP <140/90 mm Hg and monitor for worsening BP (eg, acute severe hypertension, superimposed preeclampsia)
18–49	S1–S3	In lactating women, assess safety of cardiovascular medications and modify, if needed, to optimize benefit with evidence-based therapy and mitigate adverse effects in infants related to excretion in breast milk
10–44	S1–S3	In all pregnant and postpartum individuals, screen for perinatal depression and anxiety symptoms with validated instruments (eg, Edinburgh Postnatal Depression Scale), at least at the initial prenatal visit, near or in the third trimester, and at the 6-wk postpartum visit

ASCVD indicates atherosclerotic cardiovascular disease; BP, blood pressure; LE8, Life's Essential 8; PMOS, polyendocrine metabolic ovarian syndrome; PREVENT, Predicting Risk of CVD Events; and VTE, venous thromboembolism.

*According to the US Preventive Services Task Force, American College of Obstetricians and Gynecologists, and Society for Maternal-Fetal Medicine, any one of the following high-risk factors (history of preeclampsia, multifetal gestation, stage 2 hypertension, type 1 or type 2 diabetes, kidney disease, autoimmune disease) or ≥2 moderate-risk factors (nulliparity, age ≥35 y, obesity, family history of preeclampsia, sociodemographic factors, personal history factors).

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associated fatalities; the majority of pregnancy-related strokes are due to intracerebral hemorrhage and occur postpartum. However, current guidance for stroke prevention in pregnancy and postpartum relies on low levels of evidence due to the rarity of stroke events and the exclusion of pregnant and postpartum women from stroke prevention trials. The 2024 AHA/American Stroke Association guideline for primary stroke prevention, consistent with ACOG and SMFM guidelines, recommended immediate treatment for systolic BP ≥160 mm Hg or diastolic BP ≥110 mm Hg in pregnant or postpartum women to prevent hemorrhagic stroke.[65] Whereas the AHA/American Stroke Association guideline also recommends targeting a BP goal of <140/90 mm Hg to reduce the risk of pregnancy-associated stroke for all women with HDP, regardless of whether hypertension was chronic or new-onset during pregnancy, this recommendation is based on limited observational data. High-quality data to support management of women with new-onset HDPs (ie, gestational hypertension, preeclampsia) are needed. For example, the optimal BP target and whether BP should be treated at levels <140/90 mm Hg for women with new-onset HDPs remains an important evidence gap.

VTE in Pregnancy and the Postpartum Period

VTE during pregnancy and the postpartum period is estimated to occur in 1 to 2 women per 1000 deliveries and is responsible for ≈10% of maternal deaths in the United States.[129] Compared with nonpregnant women of a similar age, the relative risk of VTE is increased 4- to 5-fold during pregnancy and to an even higher degree postpartum.[130] The VTE risk is highest in the first 1 to 3 weeks postpartum and remains elevated for up to 12 weeks postpartum; approximately half of all pregnancy-associated VTE events occur postpartum, with most occurring within the first 6 weeks of delivery.[4,131] Although cerebral venous thrombosis is a relatively rare cause of stroke, the greatest risk occurs in the postpartum period, and it accounts for 2% of pregnancy-associated strokes.[128] These data highlight the need for patient education and clinician awareness regarding VTE throughout pregnancy and the postpartum period.

Table 6. Clinical Considerations for Cardiovascular Implications of Adverse Pregnancy Outcomes: Prevention, Screening, and Follow-up of Adverse Pregnancy Outcomes

Approximate age range, y	Female Life Stages	Specific clinical considerations
13–49	S1–S3	In pregnant patients, use consistent terminology for defining hypertension before, during, and after pregnancy (stage 2 hypertension defined as ≥140/90 mm Hg, which is aligned with chronic hypertension in pregnancy)
13–49	S1–S3	In pregnant patients, assess risk of hypertensive disorders of pregnancy* and use prophylactic aspirin in those at increased risk for prevention of preeclampsia, optimally >16 wk of gestation (between 12 and 28 wk of gestation)
13–49	S1–S3	In all pregnant patients, perform a structured assessment of cardiovascular health with the LE8 score, at least at the first prenatal visit
13–49	S1–S3	In all pregnant patients, assess and optimize cardiovascular health at each prenatal visit and during the postpartum period
13–49	S1–S3	In all pregnant patients, screen for gestational diabetes at 24–28 wk of gestation; the timing and type of screening test may vary according to patient's risk factors and clinician and patient preferences
13–49	S1–S3	In female patients with hypertensive disorders of pregnancy, measure BP in the early postpartum period, with timing based on severity (within 72 h after discharge for those with severe hypertension or 7–10 d postpartum); measure follow-up BP at least at 6–12 wk postpartum and annually thereafter with more frequent monitoring according to severity, control, and antihypertensive treatment
13–49	S1–S3	In postpartum women with prepregnancy hypertension or de novo hypertensive disorders of pregnancy, use home BP monitoring (with or without telehealth or remote monitoring) to monitor and optimize BP control
18–49	S1–S3	In women with a history of gestational diabetes, assess glycemic status at 4–12 wk postpartum; diagnostic approach and timing of testing may vary according to clinician and patient preferences and may include either HbA1c or OGTT with ongoing screening every 1–3 y
≥13	S1–S4	In all female patients, obtain and document a structured pregnancy history, including adverse pregnancy outcomes (eg, hypertensive disorders of pregnancy, gestational diabetes, preterm birth, small-for-gestational age infant), and incorporate these as risk enhancers to guide patient-centered decision-making for therapies

BP indicates blood pressure; HbA1c, hemoglobin A1c; LE8, Life's Essential 8; and OGTT, oral glucose tolerance test.

*According to the US Preventive Services Task Force, American College of Obstetricians and Gynecologists, and Society for Maternal-Fetal Medicine, any one of the following high-risk factors (history of preeclampsia, multifetal gestation, chronic hypertension, type 1 or type 2 diabetes, kidney disease, autoimmune disease) or ≥2 moderate-risk factors (nulliparity, age ≥35 y, obesity, family history of preeclampsia, sociodemographic factors, personal history factors).

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Cardiovascular Health in the Postpartum Period

The postpartum period represents another window of opportunity to assess and modify cardiovascular risk through approaches including lifestyle counseling and specialty referrals for interdisciplinary care, when indicated. Postpartum screening for LE8 health behaviors and health factors, which should be conducted within 12 months of delivery for all women and sooner after an APO, was the focus of a recent scientific statement.[75,122] For patients with HDPs, postpartum BP monitoring is an important area of focus for improving maternal health, which was the focus of a recent scientific statement.[122] Optimizing BP control across the peripartum period was the focus of a 2026 National Academies of Science, Engineering, and Medicine multigroup report titled *Protecting Maternal Heart Health:* Prevention and Care Before, During, and After Pregnancy[132] and may be supported by multidisciplinary postpartum hypertension clinics.[133] Among patients with gestational diabetes, testing for diabetes is recommended with oral glucose tolerance testing 4 to 12 weeks postpartum. However, suboptimal completion rates may result from the complexity and burden required of this test. Therefore, alternative approaches (eg, oral glucose tolerance testing before discharge after delivery) or different types of screening approaches (eg, glycated hemoglobin, fasting glucose, continuous glucose monitoring) and at different follow-up times (eg, delivery admission inpatient, early postpartum) should be considered. In addition to benefits of breastmilk for infants, recent evidence supports benefits of lactation for maternal cardiovascular health. However, many barriers exist to lactation, including lack of paid maternity leave, limited insurance coverage or access to lactation consultants, and high costs of pumping supplies.[134]

The transition from pregnancy care to primary care presents an opportunity to establish care pathways to measure, monitor, and modify cardiovascular health in the short and long term. The AHA/American Stroke Association guidelines highlight the importance of risk enhancers, such as APOs, but do not specify optimal frequency of screening, prevention, and management after an APO. Given that APOs are associated with CVD, in large part through modifiable CVD risk factors, the presence of these factors represents key targets to prompt more frequent screening and focused intensive preventive efforts. In addition, emerging data that show an increased risk of early-onset HF, particularly HF with preserved ejection fraction, after HDPs support the potential utility of the PREVENT-HF equations along with the PREVENT-ASCVD equations in this population.

Risk Profile of Cardiovascular Medications

Management of CVD during pregnancy and in lactating women requires specific considerations of safety and benefit of medications (Supplemental Table 10). Use of online support tools such as LactMed, REPROTOX, and other resources can identify pertinent considerations for medications before, during, and after pregnancy.

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CARDIOVASCULAR RISK ASSESSMENT TO GUIDE HORMONE THERAPY

Hormone therapy use is common in women and may be recommended for various indications. Uses include regulation of heavy or irregular menstrual bleeding, dysmenorrhea, PMS or premenstrual dysphoric disorder, suppression of ovulation, contraception, relief of menopausal symptoms, and assisted reproductive technology (ART). In S3 and S4, uses include management of hormone insufficiency or menopause-related symptoms or to reduce fracture risk. Despite the cardiovascular benefits conferred by endogenous sex hormones, this protection is not consistently conferred by exogenous hormone therapy. In fact, hormone therapy may be associated with increased risk of CVD or VTE when taken at higher doses, particularly in older or at-risk women.[22]

Current guidelines often apply categorical contraindications to guide decision-making for hormone therapies that fail to account for the heterogeneity of baseline risk among women (eg, not all women with diabetes are at similarly increased risk of ASCVD). Conversely, clinical decision-making may often prioritize relative risk increases when certain conditions are present (eg, hypertension) without contextualizing the magnitude of absolute risk change (eg, although the relative risk increase may be moderate, if the incidence of the event is low, the absolute risk is still low). Therefore, integration of quantitative risk assessment can inform person-centered decision-making and absolute risk change, which is dependent on the baseline risk, with any given therapy. Thus, a risk-based framework for use of hormone therapy can incorporate symptom burden, absolute risk increase, and patient autonomy, and builds upon the analogous AHA/ACC approach to risk-based prevention of CVD.[107,114,135]

Combined Hormonal Contraception

Most commonly used in S1 through S3, CHCs may be considered for various indications, and must balance efficacy, safety, and patient preference, which may differ by CHC dose, formulation, and route of administration. The 2017 ACC/AHA high BP guideline[136] revised BP standards and definitions to designate lower thresholds for a diagnosis of hypertension (≥130/80 mm Hg) and define 2 categories of hypertension: stage 1 (systolic BP 130–139 mm Hg or diastolic BP 80–89 mm Hg) and stage 2 (≥140/90 mm Hg). The US medical eligibility criteria published by the Centers for Disease Control and Prevention, however, were developed based on data collected during periods when previous definitions of hypertension were used, and little data exist to delineate risk of CHC when used in an individual with stage 1 compared with stage 2 hypertension.[137] The current US medical eligibility criteria recommendations state that CHCs are relatively contraindicated in patients with stage 2 hypertension and absolutely contraindicated in those with BP ≥160/100 mm Hg. Other cardiovascular contraindications to CHCs include history of myocardial infarction, stroke, or VTE. In addition, migraine with aura is considered a US medical eligibility criteria category 4 and migraine without aura is considered US medical eligibility criteria category 2. However, risk factors, risk-mitigating strategies (eg, prophylactic anticoagulation), and patient preferences should be integrated into person-centered decision-making for CHCs.

Assisted Reproductive Technology

Hormone therapy used in ART and CVD risk was the focus of a recent scientific statement.[138] The use of ART is increasing in the United States, with ≈2.1% of US births conceived with ART. Hormone therapy results in ovarian hyperstimulation in 0.1% to 6% of ART cycles and a 2.5- to 3-fold increased risk of VTE during the first trimester. Data are limited, but preliminary studies suggest no significant increase in long-term risk of CVD due to ART.[139]

Menopausal Symptom Therapy

Menopause-related or menopausal symptoms in S3 and S4 can be managed with hormone therapies (ie, CHCs, progestogens, menopausal hormone therapy) or nonhormonal therapies, including behavioral and pharmacologic options.[37] Menopause-related symptoms should be carefully assessed, and person-centered decision-making should focus on symptom management in parallel with risks and benefits of menopausal hormone therapy, which differ by patient age, dose, formulation, and route of administration.[77] Menopausal symptoms can be well managed with strategies tailored to the clinical context. Progestogen therapy can manage abnormal or irregular menstrual bleeding. Low-dose CHCs (defined by US medical eligibility criteria as those containing ≤35 µg ethinyl estradiol) are effective for attenuating the widely fluctuating hormonal levels in the perimenopausal stage and providing contraception, which is an approach endorsed by both ACOG and The Menopause Society until age 55 years in the absence of contraindications.[140] Once ovulation has largely ceased and contraception is no longer needed, CHCs can be transitioned to menopausal hormone therapy, if needed, to manage menopause-related symptoms.[141]

Hormone therapy is approved by the US Food and Drug Administration for moderate to severe postmenopausal vasomotor symptoms; treatment of hypoestrogenism caused by hypogonadism, bilateral oophorectomy, or premature ovarian insufficiency; prevention of postmenopausal osteoporosis and to reduce fracture risk; and treatment of genitourinary symptoms.[140,142] Systemic hormone therapy for menopausal symptoms includes estrogen

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alone or in combination with a progestogen or bazedoxifene (for women with a uterus to prevent the proliferative effects of estrogen on the endometrium).[76,143] When menopause occurs <45 years of age, hormone replacement therapy is recommended in doses to approximate physiologic premenopausal levels until 51 years of age, which is higher than hormone therapy doses commonly used for menopausal symptoms.[144] Similarly, if surgical menopause occurs before the final menstrual period, with the resultant abrupt withdrawal of ovarian hormones, hormone therapy is recommended until age 51 years. Observational data suggest that women who experienced surgical menopause <50 years of age and received hormone replacement therapy had lower risk of CVD.[145]

To address menopause-related symptoms, person-centered decision-making should include symptom burden, contraindications, and risks and benefits of therapeutic options.[144] Although the absolute risk increases associated with hormonal therapies for menopausal symptoms (CHCs or systemic menopausal hormone therapy) are generally low, increased risks include VTE, stroke, and breast cancer (for estrogen plus progestogen).[140] Estimation of absolute risk of ASCVD with the PREVENT-ASCVD equations can personalize treatment considerations according to each patient's risks and benefits (Figure 3 and Supplemental Figures 6 and 7), with nonhormonal therapies preferred for high 10-year risk of ASCVD, defined as PREVENT-ASCVD ≥10%.

In addition to clinical factors, risks are dependent on the hormone therapy used (eg, dose, formulation, administration route). Nonoral formulations avoid the first-pass hepatic effect, resulting in lower risk, although direct comparisons for clinical CVD outcomes are lacking.[28,146] Topical vaginal estrogen is efficacious for the management of genitourinary symptoms, has limited systemic absorption, and is not associated with increased CVD risk.

Available data from randomized controlled trials in postmenopausal women demonstrate no evidence for substantial cardiovascular harm with hormone therapy among women between the ages of 50 and 59 years or within 10 years of menopause. In the Women's Health Initiative, post hoc analyses suggested that women age 50 to 59 years or within 10 years of menopause had no significant increase in ASCVD events with hormone therapy. A follow-up secondary analysis in the subset of women age 50 to 59 years with moderate to severe vasomotor symptoms demonstrated consistent findings of no significant increase in ASCVD with hormone therapy. These data underpin the "timing hypothesis" that CVD effects of hormone therapy may depend on the timing of initiation in relation to the onset of S4.[144] ELITE (Early versus Late Intervention Trial With Estradiol; URL: https://www.clinicaltrials.gov; Unique identifier: NCT00114517) demonstrated reduced progression of carotid intima-media thickness with oral estradiol in women within 6 years of menopause (compared with >10 years after menopause); however, computed tomographic measures (coronary artery calcium, total stenosis, and plaque) did not demonstrate any difference.[147] Overall, data on risks and benefits of hormone therapy in perimenopausal patients before the final menstrual period are limited. Beyond addressing symptoms in perimenopause or postmenopause, ACOG, The Menopause Society, the International Menopause Society, and the US Preventive Services Task Force all explicitly state that hormone therapy is not recommended for primary or secondary prevention of CVD.[148]

The risk of VTE in all age groups appears to be increased by ≈2-fold with oral estrogen plus progestogen compared with placebo when used as postmenopausal hormone therapy.[149,150] Similar to ASCVD, an individual's absolute risk of VTE, and the absolute risk increase associated with hormone therapy, is based on personal risk factors (eg, age, obesity, personal history of VTE, family history of VTE, thrombophilia), such that those with higher baseline risk have a higher absolute risk increase from hormone therapy. The risk of VTE returns to baseline after stopping hormone therapy. Transdermal hormone therapy has been associated with no increased VTE risk in observational studies.[151,152] The International Menopause Society supports transdermal hormone therapy as an option in women at high risk for VTE, such as those with additional VTE risk factors, inherited high-risk thrombophilia, or history of VTE.

For women who choose not to use hormone therapy or for whom it is contraindicated, 3 nonhormonal US Food and Drug Administration–approved treatments are available to address vasomotor symptoms: 2 neurokinin receptor antagonists (fezolinetant and elinzanetant) and a selective serotonin reuptake inhibitor (paroxetine mesylate).[153] Other medications (eg, other selective serotonin reuptake inhibitors, serotonin-norepinephrine reuptake inhibitors, gabapentin, oxybutynin) and behavioral therapies (ie, cognitive behavioral therapy, clinical hypnosis) have demonstrated efficacy in managing menopause-related symptoms, which include vasomotor symptoms, sleep disturbance, and symptoms of depression or anxiety, or both.[154–156]

Testosterone therapy is increasingly being used in perimenopause and postmenopause to address a broad range of symptoms.[157] However, there are no US Food and Drug Administration–approved testosterone formulations for women. The 2019 Global consensus position statement on testosterone therapy for women[158] recommended that if testosterone therapy is preferred for management of sexual dysfunction, only transdermal formulations should be used, with appropriate dosage considerations, but data on long-term CVD safety with event end points are limited. Oral testosterone in postmenopausal women is not recommended for menopausal symptom management and has been shown to negatively affect cholesterol levels.[158]

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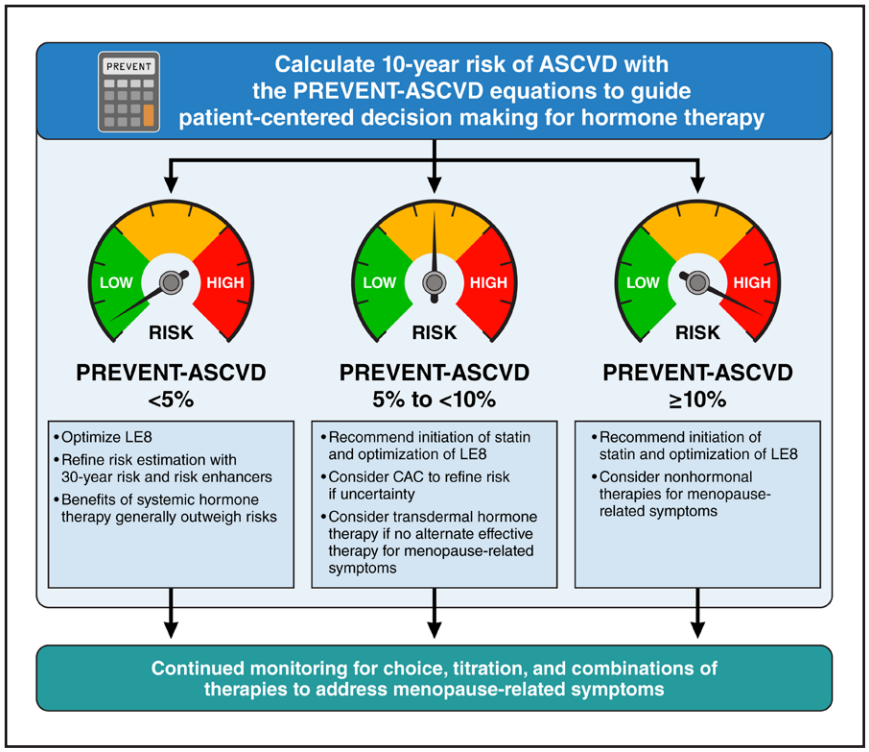


Figure 3. Patient-centered decision-making according to PREVENT-ASCVD for hormone therapy to address menopause-related symptoms in S3 and S4.
A risk-based framework for guiding decisions about hormone therapy for menopause-related symptoms (vasomotor symptoms, sleep disturbance, depressed mood, and anxiety) based on cardiovascular disease risk assessment as well as patient age and timing of menopause onset. This algorithm integrates assessment and modification of atherosclerotic cardiovascular disease (ASCVD) risk based on the PREVENT-ASCVD (Predicting Risk of CVD Events–ASCVD) equations. CAC indicates coronary artery calcium; and LE8, Life's Essential 8.

A CALL TO ACTION FOR PATIENT-CENTERED AND EVIDENCE-BASED CARE IN GIRLS AND WOMEN

Optimizing prevention and management of CVD in girls and women across the life course requires a multipronged, coordinated, and person-centered approach (Figure 4). Within clinical care models, the Female Life Stage construct can be incorporated within the electronic health record and prompt screening. In addition, a standardized form could be included as a previsit checklist for all female patients (eg, Supplemental Table 11). Healthcare systems have the opportunity to leverage technology to overcome medical silos and provide integrated, interdisciplinary patient care with routine incorporation of validated screening tools for hormonal, psychosocial, and cardiovascular health. This care should incorporate documentation of menstrual history, including key life transitions in female patients (including age at menarche and age at menopause) and pregnancy history in structured fields. Among women with CVD risk factors, on cardiovascular medications, or with CVD, anticipatory counseling for pregnancy planning and prevention, if indicated, as well as automated review by the electronic health record of medications for potential teratogenicity should be prompted in each encounter. In addition, prescription of anticoagulation medications among menstruating patients (eg, S2 or S3) should automate a prompt for anticipatory counseling about the potential impact on menstrual bleeding. These clinical decision support tools should be complemented with clinician education that can be supported with case-based approaches, artificial intelligence to evaluate competencies, and opportunities for certification.

Evidence gaps continue to inhibit the provision of best care due to both persistent underrepresentation of women in clinical cardiovascular trials and underfunding of clinical trials focused on women's health. Therefore, this document serves as a call to action to address gaps through increased representation of girls and women in clinical trials. A commitment to increased funding for research focused on optimizing health in women is necessary. Research efforts can also be promoted through clinical trials networks, such as can be developed through Comprehensive Centers of Excellence for Women's Cardiovascular Care. In parallel, empowering girls and women to advocate for female-specific issues is needed to improve outcomes, and should include a focus on health literacy to address improved understanding and use of health information for personal well-being. In aggregate, the multiple components of a call to action include clinical care pathways, education, research, digital health and systems, community and policy, and Comprehensive Centers of Excellence, which are detailed in the accompanying science advisory (Figure 5).

UNANSWERED QUESTIONS AND FUTURE DIRECTIONS

The Female Life Stage construct described in this and the accompanying presidential advisory highlights numerous areas with evidence gaps. The following section presents a selected list of key evidence gaps that

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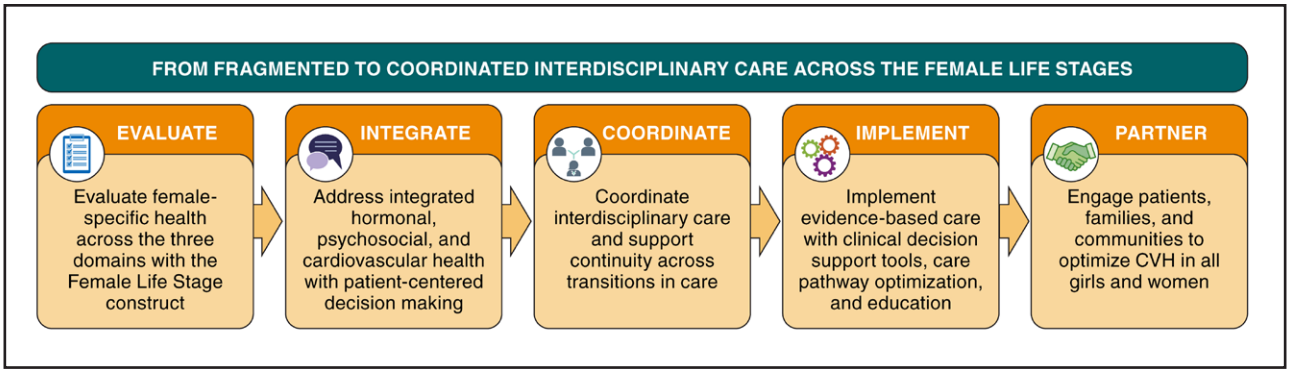


Figure 4. From fragmented to coordinated interdisciplinary care: multipronged, coordinated, and patient-centered care.
A stepwise pathway to transform the current fragmented approach to cardiovascular care in women toward multipronged, coordinated, and patient-centered care. Key steps include evaluating, integrating, coordinating, implementing, and partnering to improve cardiovascular health (CVH) in girls and women with clinical-, system-, and community-level integration.

are deemed high priority for future investigation. This list is not meant to be comprehensive. Additional gaps specific to individual conditions, populations, and clinical scenarios are noted throughout the preceding sections.

Exogenous Hormone Therapy and CVD Risk

There are multiple uses of hormone therapy across S1 through S4, including for management of PMOS, HMB, and perimenopause. Specifically, key scientific gaps exist regarding how best to address menopause-related symptoms and CVD risk. These include (1) the effect of ovulation blocking hormones and menopausal hormone therapy (when used in contemporary oral or transdermal formulations) on clinical CVD outcomes, and whether this effect differs by age at initiation (<60 years) or in relation to the final menstrual period (particularly in perimenopausal women in S3 who have not yet had a final menstrual period); (2) the safety and efficacy of postmenopausal hormone therapy for women at intermediate risk of CVD based on coronary artery calcium but who are on optimal preventive therapies, such as intensive lipid-lowering and BP-lowering; (3) whether incorporation of menopause-specific risk factors improves ASCVD risk assessment and stratification (eg, premature or early age at menopause, vasomotor symptom frequency or burden); and (4) the best lifestyle or therapeutic interventions to mitigate menopause-related worsening of CVD risk factors.

Female-Specific Interventions to Optimize Psychosocial and Cardiovascular Health

Despite robust epidemiologic evidence of the association of psychosocial factors and CVD risk and broad agreement that psychosocial factors meaningfully influence cardiovascular health, clinical guidance on how frequently screening should be implemented in cardiovascular care settings and what specific interventions improve cardiovascular outcomes are lacking. Several evidence gaps related to psychosocial challenges warrant further study: (1) whether implementation of clinic-based screening and counseling improves psychosocial health and cardiovascular health trajectories (ie, with pragmatic hybrid implementation effectiveness trials); (2) how best to structure interventions for disordered eating behaviors to optimize psychosocial and cardiovascular health; and (3) how to design female-specific lifestyle strategies that address menstrual cycle–related symptoms of PMS or premenstrual dysphoric disorder that improve cardiovascular health.

Female-Specific Considerations in Weight Management to Optimize Cardiovascular Health

Achieving and maintaining a healthy weight across the life course is a key component of cardiovascular health, along with optimizing cardiovascular health behaviors. Appropriate gestational weight gain is physiologic in pregnancy, but excess gestational weight gain and postpartum weight retention are risk factors for obesity.[122] In addition, the menopausal transition is associated with changes in body composition, which include increases in visceral adipose tissue. Therefore, future studies should evaluate intensive lifestyle interventions with or without digital health technology at key life transitions. Use of GLP-1–based therapies is rapidly increasing, particularly among women. GLP-1 receptor agonists are being used frequently in patients with PMOS, after pregnancy, or in the perimenopausal period to address weight management and metabolic health, but there is limited clinical trial evidence to support their long-term benefit when used for these reasons. Moreover, short-term use of GLP-1 receptor agonists around the time of pregnancy to improve fertility, for weight loss, or for diabetes may be complicated by rebound weight gain that may

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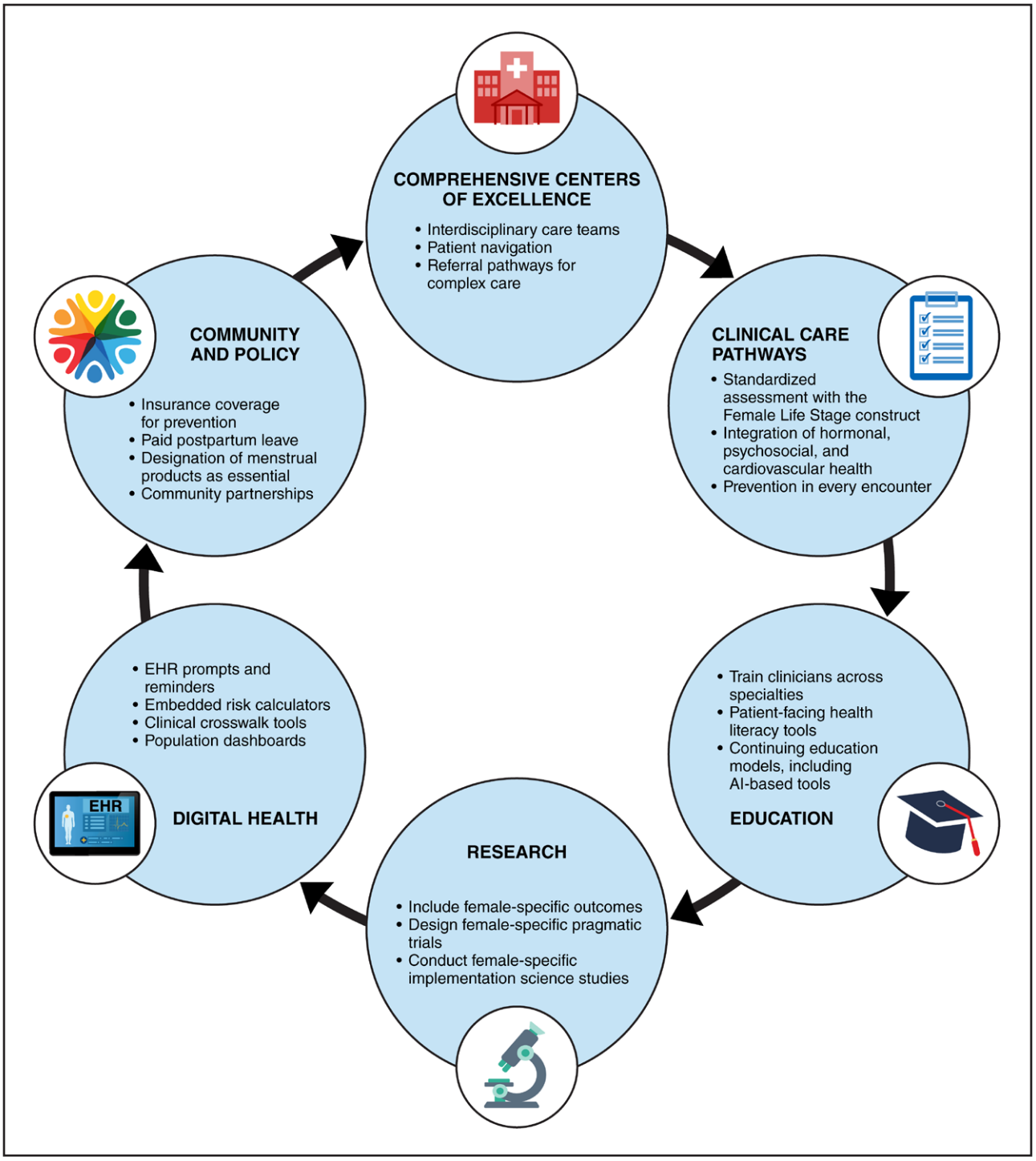


Figure 5. A call to action for optimizing cardiovascular health in girls and women across the life course.
Optimizing cardiovascular health in girls and women across the life course requires clinical care pathways, education, research, digital health and systems, community and policy, and development of comprehensive centers of excellence. Together, these components form an integrated ecosystem to improve cardiovascular outcomes across the life course and at scale. AI indicates artificial intelligence; and EHR, electronic health record.

result in a higher risk of gestational weight gain and other APOs.[159] Best practices for use of these agents for these selected indications and the optimal timing of discontinuation when pregnancy is intended remains uncertain.

Peripregnancy Period and Cardiovascular Health

Better LE8 health behaviors and health factors are associated with lower risk of maternal and neonatal

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adverse outcomes based on observational evidence, but the best prepregnancy interventions to enhance cardiovascular health as a means to improving pregnancy-related outcomes remain unknown. Currently, a cluster-randomized trial enrolling high-risk pregnant women receiving home visiting services is evaluating whether an LE8 intervention initiated during pregnancy is associated with better postpartum cardiovascular health and offspring cardiovascular health.[160] After an APO, close monitoring for development of cardiovascular risk factors is recommended, but evidence remains lacking regarding the frequency of monitoring, the optimal antihypertensive medication class, and the optimal treatment targets (eg, BP target during the first year postpartum). Evaluation of postpartum glycemic status after a diagnosis of gestational diabetes with continuous glucose monitoring may offer a useful alternative to oral glucose tolerance testing, but further study is needed.

CONCLUSION

CVD risk in girls and women is shaped by biologic, clinical, psychosocial, and structural factors that vary across the life course, with the earliest determinants emerging well before overt clinical symptoms. Current care is fragmented across specialties, and female-specific and female-predominant factors are inconsistently integrated into comprehensive care. This multisociety scientific statement provides the first harmonized, life stage–based clinical guidance, integrating considerations across interconnected domains of hormonal, psychosocial, and cardiovascular health. The crosswalk of major society guidelines and statements reveals meaningful concordance and advances a unified vision: recognizable clinical stages that offer distinct opportunities to identify risk earlier and intervene more effectively. Important evidence gaps exist and mechanistic studies, randomized clinical trials, and implementation science approaches are needed to determine best equitable models for care delivery. By aligning science, clinical care, and policy through a life course lens, this joint multisociety compendium, on behalf of the AHA, AAP, ACOG, and SMFM, calls for coordinated, comprehensive, and person-centered care for girls and women, recognizing that every clinical encounter is an opportunity to improve cardiovascular health.

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The American Heart Association makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest.

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*Modest.

†Significant.

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Jennifer Thompson	Vanderbilt University Medical Center	None	None	None	None	None	None	None
Debra B. Waldron	American Academy of Pediatrics	CDC (funding goes to the AAP)†; PCORI (funding goes to the AAP)†	None	None	None	None	None	None

This table represents the relationships of reviewers that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all reviewers are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives ≥$5000 during any 12-month period, or ≥5% of the person's gross income; or (b) the person owns ≥5% of the voting stock or share of the entity, or owns ≥$5000 of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

†Significant.

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