

TOP TAKEAWAYS

Consensus Guidance for the Female-Specific Prevention and Management of Cardiovascular Disease

A Deep Dive on the 2026 AHA/AAP/ACOG/SMFM Scientific Statement

SCIENTIFIC STATEMENT | WOMEN'S CARDIOVASCULAR HEALTH

SECTION A – PURPOSE, CRITICAL GAP, AND FEMALE LIFE STAGE FRAMEWORK

- **Purpose of the Scientific Statement.** This joint Scientific Statement on behalf of AHA, AAP, ACOG, and SMFM presents stage-specific clinical guidance across the interconnected domains of hormonal health, psychosocial health, and CV health across the female life course. It uses a crosswalk of major society guidelines, evidence synthesis, and expert consensus.
- **Critical gap it fills.** Currently, clinical recommendations for prevention and management of CVD in girls and women are distributed across multiple societies. As a result, girls and women experience unique fragmentation in their clinical care across pediatrics, adolescent medicine, primary care, OB/GYN, maternal-fetal medicine, endocrinology, cardiology, hematology and neurology. Cross-disciplinary harmonization is needed. Despite advances in sex-based biology, translation into routine practice remains incomplete.
- **The Female Life Stage construct.** Four clinically meaningful stages defined by changes in endogenous sex hormones leading to key life events and transitions: S1 perimenarche, S2 ovarian cycling, S3 perimenopause, S4 postmenopause. Pregnancy is a CV modifier and life event when latent CV risk may emerge (e.g., APOs). Peripregnancy spans prepregnancy (12 mo), pregnancy (~40 weeks), postpartum (12 mo; first 12 weeks = fourth trimester).
- **Three anchoring principles.** (1) Endogenous sex hormones are dynamic and produce physiologic changes accessible without labs. (2) Hormonal transitions (perimenarche, perimenopause, peripregnancy) are windows of opportunity that reflect changing vulnerability to psychosocial stressors and CVD risk. (3) The framework makes explicit the interconnectedness of hormonal, psychosocial, and CV health.
- **Sex hormones as CV modulators.** Estradiol acts via ER α on endothelial cells to stimulate NO production, promotes vasodilation via endothelial prostacyclin, and has antioxidant/anti-inflammatory effects. Modulates lipid metabolism via hepatic LDL receptor upregulation. Exogenous HT does NOT automatically confer endogenous protection and may increase CVD risk depending on age, timing, formulation, and baseline risk.
- **Female-specific CV risk enhancers.** APOs, irregular menstrual cycles, PMOS (formerly PCOS), premature ovarian insufficiency (POI), autoimmune disorders, and both surgical (premenopausal bilateral oophorectomy) or medical (exogenous HT) changes to the hormonal milieu.
- **Female-predominant CVD phenotypes.** Peripartum cardiomyopathy and HDP occur only in women. Takotsubo cardiomyopathy, SCAD, and chemotherapy-related cardiomyopathy are

more common in women. Takotsubo predominates in S4; SCAD typically S3-S4 but may occur in S2 (a common cause of pregnancy-associated MI).

SECTION B – METHODS AND GUIDELINE CROSSWALK

- **Multi-society Science Advisory Group.** The AHA convened the Science Advisory Group with representatives from pediatrics, adolescent medicine, primary care, obstetrics, maternal-fetal medicine, cardiology, endocrinology, hematology, neurology, and nursing alongside researchers in epidemiology, vascular biology, outcomes, and implementation science, plus patient advocates.
- **Systematic guideline harmonization.** Compiled recommendations from AHA/ACC, AAP, ACOG, SMFM, and selected guidelines from APA, International PCOS Network, Menopause Society, ASH, WPSI, and Society for Adolescent Health and Medicine. Organized by clinical domain and Female Life Stage anchored in relevance to cardiovascular health.
- **Sex-inclusive language review.** From the 338 AHA/ACC Guidelines and AHA statements reviewed for the compendium, there are clear gaps in the available evidence and clinical practice recommendations, *female-specific* clinical guidance, as well as in the implementation of the available guidance.

SECTION C – STAGE-SPECIFIC CLINICAL GUIDANCE

STAGE S1 – PERIMENARCHE

Hormonal Health

- **Anticipatory guidance and assessment of menarche onset.** Beginning at age 8 y, monitor pubertal progression. Assess cycle regularity, bleeding heaviness, and impact on CV health behaviors.
- **Early menarche as risk enhancer.** Early menarche (<10 y) is associated with modestly increased CVD risk, partially mediated by excess adiposity. Recognized as a risk enhancer in the 2026 AHA/ACC/Multi-Society Dyslipidemia Guideline.
- **Menstrual cycle as a vital sign.** ACOG designates the menstrual cycle a vital sign. Ongoing assessment identifies conditions that may have cardiometabolic consequences. Cycle length and bleeding typically stabilize 1–3 y post-menarche.

Psychosocial Health

- **Depression, anxiety, and eating disorder screening.** AAP, ACOG, and WPSI endorse routine screening from adolescence. Depression: PHQ-2 → PHQ-9 (≥12 y). Anxiety: GAD-2 → GAD-7 (≥13 y); AAP anxiety evaluation from age 8. Eating disorders: annual screening ≥11 y. Use body-positive, non-stigmatizing language.

Cardiovascular Health

- **Sex-specific pediatric assessment.** Universal BMI and BP screening yearly using age- and sex-specific percentiles. Lipid testing ages 9–11 and 17–21 y; glycemic screening based on risk. Pubertal staging is NOT currently incorporated into thresholds.
- **Transition from pediatric to adult care.** High-risk period for loss of female-specific preventive information. Include: anticipatory guidance on adult care options; menstrual history as a required element of the transition summary; delineation of psychosocial concerns.

STAGE S2 - OVARIAN CYCLING

Hormonal Health

- **Structured menstrual history.** Assess age at menarche, LMP, cycle regularity, cycle length, bleeding duration, bleeding heaviness. Secondary amenorrhea or irregularity ≥ 3 months (without exogenous HT).
- **Heavy or irregular menstrual bleeding.** HMB: flow > 7 days; changing soaked products more frequently than every 1–2 hours; > 6 pads/tampons per 24 hours. The International Federation of Gynecology and Obstetrics (FIGO) defines HMB as excessive blood loss interfering with quality of life.
- **Iron deficiency is common and CV-relevant.** Affects $\sim 38\%$ of menstruating females. Assess in HMB or symptoms (fatigue, dyspnea, exercise intolerance, pica, restless legs). Treat regardless of anemia and evaluate source.
- **Anticoagulation and antiplatelet considerations.** Up to 70% of anticoagulated menstruating women experience abnormal/heavy bleeding. Rivaroxaban is associated with higher rates of heavy menstrual bleeding than warfarin; apixaban similar to LMWH. Provide anticipatory counseling.
- **Dysmenorrhea and PMS/PMDD.** Dysmenorrhea affects up to $\sim 70\%$ of adolescent girls and $\sim$ doubles insomnia odds. Endometriosis-related dysmenorrhea is a stroke risk factor (2024 AHA/ASA). Rates in women with menstrual cycles: PMS $\sim 25\%$; PMDD 2–5%
- **PMOS (formerly PCOS).** Recognize symptoms and refer: hyperandrogenism (clinical/biochemical), ovulatory dysfunction, polycystic morphology or elevated AMH. Screen for CVD risk at diagnosis regardless of BMI. OGTT preferred; HbA1c/fasting glucose alternatives. Manage with lifestyle, CHCs, metformin, GLP-1. Recognized risk enhancer in 2026 Dyslipidemia and CKM Guidelines.

Psychosocial Health

- **Continue routine screening.** Continue PHQ-9 and GAD-7. Watch for onset of perinatal mood disorders during peripregnancy.

Cardiovascular Health

- **Sex-specific PREVENT.** Use sex-specific PREVENT: 10-y risk in 30–79 y; 30-y risk in 30–59 y. 30-year risk addresses misperception that women are not at risk.
- **Risk enhancers to incorporate.** Include early menarche (< 10 y), PMOS, irregular menstrual cycles, APOs (HDP, GDM, preterm birth), early menopause (< 45 y) — formally incorporated in the 2026 Dyslipidemia Guideline. Consider endometriosis, infertility, pregnancy loss for individualized decisions.

- **Female-specific CVD phenotypes.** Consider SCAD, MINOCA, INOCA, Takotsubo. In women presenting with chest pain, recognize risk for underdiagnosis and consider cardiac causes.

STAGE S3 - PERIMENOPAUSE

Hormonal Health

- **Diagnose by clinical presentation.** Median onset ~47 y; median menopause ~51 y. Persistent ≥ 7 -day variation in cycle length marks onset. Diagnosis is clinical. Routine estradiol/FSH measurement NOT recommended due to variability.
- **Symptom burden.** Vasomotor symptoms (hot flashes, night sweats, palpitations), poor sleep, depressed mood, anxiety, carry CV implications. Not limited to just about estrogen deficiency but the variability and changes in other neurotransmitters too. Systematic screening for menopause-related symptoms may be considered beginning at age 40 y.
- **Early and premature menopause.** Early menopause 40–44 y (~8–12% of US women). Premature menopause / POI <40 y (~1–3.5%). Both are CV risk enhancers. Recognize iatrogenic causes.

Psychosocial Health

- **Screen and address mood, sleep, and stress.** Depression and anxiety intensify. Screen with PHQ-9 and GAD-7. Sleep disturbance, caregiving burden, economic stress compound risk.

Cardiovascular Health

- **Metabolic and vascular shifts.** LDL-C, apoB, and Lp(a) rise; visceral adiposity increases; vascular stiffening and atherosclerosis progress — independent of chronologic aging.
- **Risk assessment.** Calculate 10-y PREVENT (30–79 y) and 30-y PREVENT (30–59 y). Consider menopause-related symptoms and female-specific enhancers. CAC scoring refines risk when needed.
- **HF risk.** Given higher HFpEF prevalence in women, HF risk assessment matters with PREVENT-HF. Biomarkers (e.g., BNP), echocardiography, or both may be useful to guide preventive efforts.

STAGE S4 - POSTMENOPAUSE

Hormonal Health

- **Sustained low estradiol.** Postmenopause begins after 12 months of amenorrhea (or after surgical/medical intervention) and represents ~40% of a woman's lifespan. Symptoms most common in first 3–6 years with hormonal and non-hormonal treatment options. Genitourinary syndrome of menopause common and can be easily addressed.

Psychosocial Health

- **Age-related psychosocial stressors.** Depression, anxiety, and insomnia remain prevalent. Widowhood, isolation, and caregiving compound risk. Continue routine screening.

Cardiovascular Health

- **Overt CVD emerges.** Most common CVD subtypes: ASCVD, HF, atrial fibrillation. Follow established guidelines with heightened awareness that CVD and its risk factors are most likely to develop in this stage. Sex-specific thresholds for valvular heart disease apply.

- **Brain health integration.** Stroke and dementia are #3 and #4 causes of death in US women. Menstrual disorders (PMOS, endometriosis), APOs, premature ovarian insufficiency or POI/early menopause, high vasomotor symptom burden associate with elevated stroke and dementia risk.
- **Cross-cutting risk enhancers.** POI: ~1–3.5%; underdiagnosed; physiologic hormone replacement underutilized.
 - Autoimmune diseases: predominantly female.
 - Breast and ovarian cancer therapies: anthracyclines, trastuzumab, radiation, SERMs (VTE), aromatase inhibitors (hypoestrogenism) require integrated management across treatment and survivorship.

SECTION D - HORMONE THERAPY

- **Hormone therapy.** Different hormone formulations are available and can be used for a variety of indications including hormonal regulation, suppression of ovulation, contraception, heavy menstrual bleeding, PMOS (formerly PCOS), fertility treatment, and menopause-related symptoms.
- **Combined hormonal contraception.** USMEC: relatively contraindicated in Stage 2 hypertension; absolutely contraindicated if BP $\geq 160/100$ mm Hg. Other CV contraindications: history of MI, stroke, VTE.
- **Menopausal HT - timing and route. Not equivalent to endogenous protection in all cases.** May confer no protection or increased CVD risk depending on age, timing, formulation, and baseline risk. WHI post-hoc: no significant increase in ASCVD events with HT among women 50–59 y or within 10 y of menopause. Non-oral formulations may have more favorable thrombotic profile. Topical vaginal estrogen: efficacious for GU symptoms, limited systemic absorption, no clear CVD risk. Oral estrogen + progestogen ~2-fold VTE risk vs. placebo.
- **HT is NOT for CV prevention.** ACOG, Menopause Society, IMS, USPSTF concur. Nonhormonal therapies are preferred for menopause-related symptoms when 10-y PREVENT-ASCVD $\geq 10\%$.

SECTION E - CALL TO ACTION

- **Optimize clinical workflows.** Incorporate the Female Life Stage construct into EHRs. Document age at menarche, pregnancy history, and date of menopause. Automate teratogenicity review in prepregnancy counseling and anticoagulation-related uterine bleeding counseling.
- **Address evidence gaps.** Increase representation of girls and women in clinical trials; expand research funding; leverage Comprehensive Centers of Excellence.

Bottom line. This Scientific Statement provides the first harmonized, life stage-based clinical guidance. The multi-society compendium (PA + SS + SA) calls for coordinated, comprehensive, person-centered female-specific care across the life course.