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AHA SCIENTIFIC STATEMENT

Optimizing Prepregnancy Cardiovascular Health to Improve Outcomes in Pregnant and Postpartum Individuals and Offspring: A Scientific Statement From the American Heart Association

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ABSTRACT: This scientific statement summarizes the available preclinical, epidemiological, and clinical trial evidence that supports the contributions of prepregnancy (and interpregnancy) cardiovascular health to risk of adverse pregnancy outcomes and cardiovascular disease in birthing individuals and offspring. Unfavorable cardiovascular health, as originally defined by the American Heart Association in 2010 and revised in 2022, is prevalent in reproductive-aged individuals. Significant disparities exist in ideal cardiovascular health by race and ethnicity, socioeconomic status, and geography. Because the biological processes leading to adverse pregnancy outcomes begin before conception, interventions focused only during pregnancy may have limited impact on both the pregnant individual and offspring. Therefore, focused attention on the prepregnancy period as a critical life period for optimization of cardiovascular health is needed. This scientific statement applies a life course and intergenerational framework to measure, modify, and monitor prepregnancy cardiovascular health. All clinicians who interact with pregnancy-capable individuals can emphasize optimization of cardiovascular health beginning early in childhood. Clinical trials are needed to investigate prepregnancy interventions to comprehensively target cardiovascular health. Beyond individual-level interventions, community-level interventions must include and engage key stakeholders (eg, community leaders, birthing individuals, families) and target a broad range of antecedent psychosocial and social determinants. In addition, policy-level changes are needed to dismantle structural racism and to improve equitable and high-quality health care delivery because many reproductive-aged individuals have inadequate, fragmented health care before and after pregnancy and between pregnancies (interpregnancy). Leveraging these opportunities to target cardiovascular health has the potential to improve health across the life course and for subsequent generations.

Key Words: AHA Scientific Statements ■ cardiovascular diseases ■ pregnancy ■ pregnancy complications ■ primary prevention ■ risk factors

There is a growing burden of cardiovascular-related morbidity and mortality in pregnant and postpartum individuals in the United States.[1] Cardiovascular disease (CVD) is the leading cause of death during pregnancy and the postpartum period and represents 26.5% of pregnancy-related deaths.[2] This topic was the focus of the 2021 American Heart Association (AHA) policy statement "Call to Action: Maternal Health and Saving Mothers," which outlined multilevel opportunities aimed at improving health literacy, public awareness, cultural competency, and bias reduction in optimizing maternal cardiovascular health (CVH).[3]

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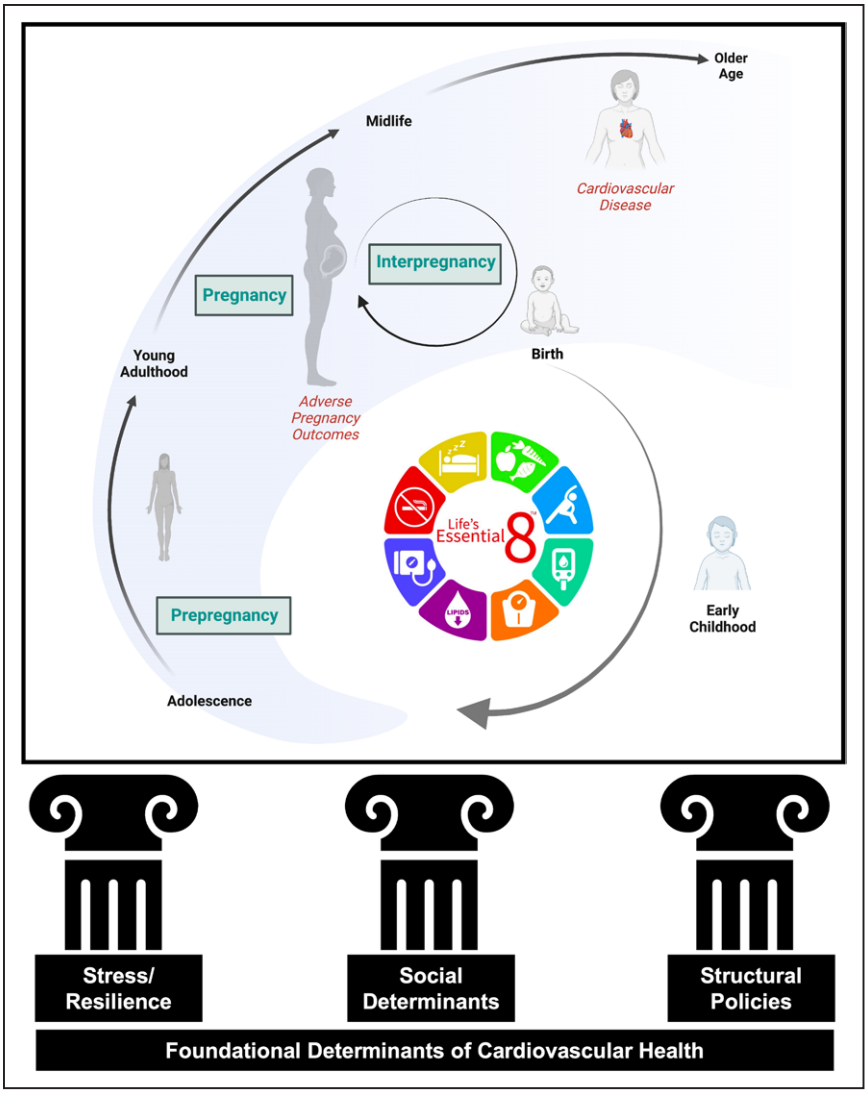

Figure. The intergenerational life cycle of cardiovascular health and its foundational determinants.
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Currently, nearly 1 in 5 births is complicated by an adverse pregnancy outcome (APO), which includes hypertensive disorders of pregnancy (HDP), preterm birth, small-for-gestational-age (SGA) birth, and gestational diabetes.[4,5] Over the past decade, rates of APOs have increased significantly, with a near doubling in rates of HDP.[4,5] There are persistent disparities, with non-Hispanic Black individuals significantly more likely to experience APOs.[6] Available data demonstrate a strong association between APOs and risk for subsequent CVD, which was detailed in the 2021 AHA scientific statement "Adverse Pregnancy Outcomes and Cardiovascular Disease Risk: Unique Opportunities for Cardiovascular Disease Prevention in Women."[7] Among individuals who experience APOs, emerging data also identify higher risk of long-term kidney disease, which is itself an important risk factor for CVD.[8] Although the pathophysiology of pregnancy-related complications is complex and likely multifactorial, emerging data suggest that these complications have, at least in part, prepregnancy origins. Thus, the prepregnancy period may be a critical window during which interventions have a great potential for benefit in birthing individuals and their offspring. In addition, interventions in the postpartum/interpregnancy period may offer a unique opportunity to target the prepregnancy period before a subsequent pregnancy.

In this AHA scientific statement, we critically review the evidence for prepregnancy CVH as a key target to improve the health of the birthing individual and offspring over the life course (Figure). We highlight the importance of a life course and intergenerational framework to assess and intervene on CVH. We offer considerations for multilevel interventions (eg, individual, community, and societal) to equitably improve prepregnancy CVH. We anchor this discussion on the AHA's construct of CVH. This was originally defined as Life's Simple 7 in 2010, which integrates 7 health factors (diet, physical activity, nonsmoking, body mass index, blood pressure, lipids, and glycemia) and has since been revised to Life's Essential 8, incorporating sleep health as the eighth metric (Table 1).[9,10] CVH is oriented on promotion

Table 1. CVH Metrics and Scoring as Originally Defined in 2010 and Revised in 2022 for Nonpregnant Adults by the AHA

CVH construct definition: 2010[10]			
	Ideal=2 points	**Intermediate=1 point**	**Poor=0 points**
Diet, Healthy Eating Index–2015 score	80–100	40–79	0–39
Physical activity, min/wk moderate to vigorous leisure-time activity	≥150	>0 but <150	0
Smoking	Never or quit >12 mo ago	Former, quit ≤12 mo ago	Current
Body mass index, kg/m^2	<25	25–29.9	≥30
Blood pressure, mm Hg	<120/<80	Systolic 120–139 or diastolic 80–89 and not on blood pressure–lowering medications	Systolic ≥140 or diastolic ≥90
Total cholesterol, mg/dL	<200 without medication	200–239 or treated to <200	≥240
Fasting glucose, mg/dL	<100 without medication	100–125 and not on glucose-lowering medications	≥126
CVH construct definition: 2022[9]			
	Ideal=100 points	**Suboptimal <100 points**	
Sleep health or average sleep per night, h	7–<9	70 points: 6–<7 20 points: 4–<5 0 points: <4	
Diet, Healthy Eating Index–2015 score or DASH (MEPA)	≥95th percentile (MEPA score 15–16)	80 points: 75th–94th percentile (MEPA score 2–14) 25 points: 25th–49th percentile (MEPA score 4–7) 0 points: 1st–24th percentile (MEPA score 0–3)	
Physical activity, min/wk moderate to vigorous leisure-time activity	≥150	80 points: 90–119 20 points: 1–29 0 points: 0	
Smoking	Never smoker and no secondhand exposure in home	75 points: former smoker, quit ≥5 y 20 points: former smoker, quit <1 y, or inhaled NDS 0 points: current smoker Subtract 20 points for living with active indoor smoker in home (unless score is 0)	
Body mass index, kg/m^2	<25	70 points: 25.0–29.9 30 points: 30.0–34.9 0 points: ≥40.0	
Blood pressure, mm Hg	<120/<80	75 points: 120–129/<80 25 points: 140–159 or 90–99 0 points: ≥160 or ≥100 Subtract 20 points if treated level	
Non-HDL cholesterol, mg/dL	<130	60 points: 130–159 20 points: 190–219 0 points: ≥220 Subtract 20 points if treated level	
Fasting glucose, mg/dL (HbA1c, %)	<100 (<5.7%) No history of diabetes	60 points: 100–125 (5.7%–6.4%) 20 points: diabetes (8.0%–8.9%) 0 points: diabetes (≥10.0%)	

AHA indicates American Heart Association; CVH, cardiovascular health; DASH, Dietary Approaches to Stop Hypertension; HbA1c, hemoglobin A1c; HDL, high-density lipoprotein; MEPA, Mediterranean Eating Pattern for Americans; and NDS, nicotine-delivery system.

of wellness, with higher CVH scores (ie, scores reflecting better health) associated with lower risk for a multitude of downstream cardiovascular and noncardiovascular outcomes in nonpregnant and pregnant individuals.[11] The clinical relevance of the CVH construct as a key target in birthing individuals was recently highlighted in a joint presidential advisory from the AHA and the American College of Obstetricians and Gynecologists that highlights the pivotal role of primary care clinicians, pediatricians, obstetricians, and cardiologists in optimizing the CVH of pregnancy-capable individuals.[12]

For the purposes of this scientific statement, we refer to individuals as females or males based on gender assigned at birth and as women or men based on presumed gender identity when these terms have been used in prior literature. There remains a dearth of data on CVH and its relationship with APOs and CVD in individuals with diverse gender identities.

CURRENT STATUS OF CVH IN BIRTHING INDIVIDUALS IN THE UNITED STATES

As of 2020, there were an estimated 64.5 million reproductive-aged (age, 15–44 years) females in the United States.[13] Approximately 3.5 to 4 million live births occur in the United States annually, and by 40 to 44 years of age, an estimated 86% of females in the United States have given birth at least once.[14]

Unfavorable CVH is prevalent in reproductive-aged young adults, with few people (<1%) having ideal levels of all CVH metrics.[11,15] According to data from the National Health and Nutrition Examination Survey (2013–2016), the prevalence of having ideal levels in ≥5 of 7 CVH metrics (using the 2010 CVH scoring system) was 45.0% among adolescents 12 to 19 years of age, 31.6% among young adults 20 to 39 years of age, and 10.6% among adults 40 to 59 years of age.[16] Gender-specific data suggest that CVH is slightly higher among females compared with males of reproductive age (eg, for adults ≥20 years of age, 21.5% of females versus 18.4% of males have ≥5 of 7 CVH metrics ideal). Similar findings were reported in data from the National Health and Nutrition Examination Survey (2013–2018) according to the revised 2022 CVH scoring system, with a mean CVH score (of 100 possible points) in women of 68.1 (SD, 0.48) compared with 63.6 (SD 0.44) in men. There are significant racial and ethnic disparities in CVH, with non-Hispanic Black females having lower mean CVH scores and worse values of most CVH metrics, including worse sleep quality, than women of other races and ethnicities.[17,18] Limited data are available for prepregnancy CVH in disaggregated Asian and Hispanic subgroups and American Indian and Alaska Native individuals. This is particularly critical given the high rates of maternal morbidity and mortality observed among American Indian and Alaska Native individuals.[19] Because race and ethnicity are social constructs, these racial and ethnic differences have been attributed to differences in upstream social factors such as education, income, and access to health care.[11] When individual CVH factors were examined among reproductive-aged females, ≈25% reported current smoking, ≈40% had obesity, 9.3% had hypertension, 4.5% had diabetes, and up to 33% had hyperlipidemia.[11,15,20–22] Lack of awareness and control of CVD risk factors is an important problem in reproductive-aged females; for example, of the 9.3% with hypertension and 4.5% with diabetes, ≈17% and 30%, respectively, were unaware of these diagnoses, and about half did not achieve optimal blood pressure or glycemic control.[22]

Maternal data on some CVH factors (prepregnancy body mass index, diabetes, hypertension, and smoking status based on a combination of self-recall and health records) are available from the National Center for Health Statistics for all live births in the United States. Fewer than half of birthing individuals have favorable prepregnancy CVH (using an abbreviated CVH defined as absence of obesity, hypertension, diabetes, and smoking).[23] Furthermore, prepregnancy CVH declined between 2011 and 2019 in all subgroups (race and ethnicity, geography, and socioeconomic status); lower CVH persisted among non-Hispanic Black females, pregnant individuals living in the South and Midwest United States, and those with Medicaid insurance during pregnancy.[23,24] With regard to specific factors, <50% of birthing individuals in 2018 had a normal prepregnancy body mass index (18.5–24.9 kg/m^2).[25,26] Levels of CVH metrics are highly correlated between the prepregnancy period and pregnancy.[15,27]

ASSOCIATIONS BETWEEN PREPREGNANCY CVH AND APOs

Prepregnancy CVH and individual CVH metrics are associated with risk of APOs in many observational studies.[11,25,28–37] According to National Center for Health Statistics data, there is a consistent and graded association between worse prepregnancy CVH and APOs (preterm birth, SGA birth, and fetal death).[28] Adjusted relative risks for preterm birth with poor levels of prepregnancy CVH metrics in 1, 2, 3, or 4 metrics (overweight or obesity, diabetes, hypertension, and smoking) were 1.15 (95% CI, 1.15–1.16), 1.62 (95% CI, 1.61–162), 2.85 (95% CI, 2.81–2.90), and 3.89 (95% CI, 3.68–4.10), respectively, compared with individuals with no poor prepregnancy CVH metrics.[28] Similar findings were observed in the multinational HAPO study (Hyperglycemia and Adverse Pregnancy Outcome), which found that lower CVH based on clinical factors at 28 weeks' gestation was associated with higher risk of APOs (preeclampsia, SGA infant).[38]

Among individual CVH metrics, prepregnancy dietary patterns are associated with risks for APOs, with healthier patterns associated with lower risk of gestational diabetes, preterm birth, SGA infant, and HDP.[39] Better prepregnancy fitness, assessed with a graded symptom-limited maximal exercise treadmill test, is associated with lower risk of gestational diabetes,[40] and greater leisure-time physical activity at the beginning of pregnancy is associated with lower risk for APOs.[41] Obesity is also associated with APOs and estimated to have a population attributable fraction resulting from HDP of between 26.5% and 30.3% in 2018 in the United States.[25] In a meta-analysis, the odds ratio was 1.31 (95% CI, 1.11–1.53) for each 1–kg/m^2 increase in body mass index from the start of one pregnancy to the next associated with HDP.[42] Prepregnancy blood pressure is associated with risk for HDP, and treatment of mild chronic hypertension starting in early pregnancy led to reduced risks for preterm birth, SGA birth, and preeclampsia in the recent CHAP trial (Chronic Hypertension and Pregnancy).[43,44] Prepregnancy lipid levels (triglycerides, high-density lipoprotein cholesterol) are associated with risk for gestational diabetes and HDP.[45] Prepregnancy glycemic status across the spectrum is associated with risk for large-for-gestational-age

birth, preterm birth, and HDP.[33,46] Poor sleep quality and duration are associated with APOs, specifically gestational diabetes and HDP.[47,48] These data, which demonstrate a similar magnitude of associations between CVH and APOs across individual CVH metrics, underscore the relevance of a strategy that comprehensively targets total CVH. Beyond the traditional CVH metrics, chronic kidney disease is an important risk factor for APOs and long-term CVD in birthing individuals.[49]

ASSOCIATIONS BETWEEN PREPREGNANCY CVH AND OFFSPRING OUTCOMES

Epidemiological studies support the association between prepregnancy CVH in the birthing person and offspring health outcomes, broadly called the developmental origins of health and disease.[50] As detailed previously, prepregnancy CVH and individual CVH metrics are associated with risk for APOs; in addition, these APOs are associated with higher risks for premature CVD among exposed offspring.[14,51–56] As an example, preterm birth is associated with 53% higher adjusted hazards for premature ischemic heart disease by 43 years of age in the offspring.[52]

Longer-term studies are emerging to provide direct evidence for links between maternal prepregnancy CVH metrics and offspring CVD risk factors and even CVD events.[54,56–60] For example, prepregnancy type 2 diabetes was associated with an adjusted hazards ratio of 1.39 (95% CI, 1.23–1.57) for offspring premature CVD by 40 years of age in a registry study.[56] No study has reported maternal prepregnancy total CVH and offspring cardiovascular outcomes. Although there are physiological changes to CVH metrics in pregnancy (eg, increase in body mass index, glucose, lipids), data demonstrate that CVD risk factor levels measured before pregnancy were highly correlated with risk factor levels during pregnancy.[27] This suggests that associations between unfavorable CVH in pregnant individuals and in offspring may stem, at least in part, from the prepregnancy period.[61] Of note, studies of maternal body mass index indicate that prepregnancy body mass index is more strongly associated with both APOs and offspring cardiovascular risk factors in adolescence compared with gestational weight gain.[34,35] However, whether the association between maternal CVH and offspring CVH is an epiphenomenon or the two are causally related requires further investigation.

POTENTIAL PATHOPHYSIOLOGICAL MECHANISMS LINKING PREPREGNANCY CVH AND APOs

Several factors shed light on the potential pathophysiological link between prepregnancy CVH and APOs. The periconceptional period before and after conception covers critical events: oocyte meiotic maturation, spermatozoa differentiation, fertilization, transition to the embryonic genome, resumption of mitotic cell cycles in the newly formed zygote, initial morphogenesis, and implantation. During this brief window, the genome is globally reprogrammed through extensive epigenetic reorganization, which determines lineage-specific gene expression—including divergence of placental and embryonic cell lineages—and establishment of metabolic controls for energy supply and growth.[50] In animal experiments, epigenetic modifications link the metabolic status of the pregnant animal to gene expression programs in the developing embryo and placenta.[62,63] As an example, in mice with obesity, the follicular fluid and oocyte are lipid enriched, resulting in endoplasmic reticulum stress, protein misfolding, and increased mitochondrial respiration and reactive oxygen species generation, with dramatic implications for energy metabolism in the oocyte and subsequently the zygote.[64–66]

Although animal and in vitro experiments provide much of the mechanistic data linking prepregnancy CVH metrics with maternal and offspring outcomes, parallel clinical observations align with these proposed periconceptional mechanisms. For example, in mice, transfer of 1-cell zygotes from diabetic dams to control recipients demonstrates that exposures around fertilization are sufficient to permanently program the postnatal phenotype. This experimental finding is mirrored by the clinical observation that in humans with diabetes, glycemic control must be achieved before pregnancy to reduce the risk of congenital anomalies and adverse neonatal outcomes.[67]

The placenta, which develops soon after fertilization and implantation occur, is a major focus of studies underlying mechanisms of APOs. Placental malperfusion is central to a cascade of vascular injury in many APOs, is secondary to inappropriate vascular remodeling of uterine spinal arteries, and begins long before clinical manifestations of APOs are apparent.[68] This abnormal placental development has been proposed to be affected by the maternal environment such as presence of prepregnancy CVD risk factors, with potential mechanisms related to angiogenesis and inflammation.[69–72] Thus, APOs may reflect the unmasking of preexisting CVD risk in response to the physiological stress of pregnancy. Indeed, markers of vascular dysfunction (eg, decreased arterial compliance, retinal microvascular constriction, diastolic dysfunction) before or in early pregnancy are associated with higher risk of APOs.[73–75] Although not conclusive, these studies add to the evidence base that unfavorable prepregnancy CVH temporally precedes and contributes to APO risk. Advancing our mechanistic understanding of the underlying pathophysiology can inform the design of potential interventions. Last, establishing whether CVH and APOs are causally related is foundationally important to

decrease the risk of APOs and future CVD by intervening on CVH.[76]

EVIDENCE FOR PREPREGNANCY AND INTERPREGNANCY CVH INTERVENTIONS

There are currently no large, randomized trials with sufficient power to test whether improving CVH before pregnancy will improve maternal and offspring outcomes (eg, reduced frequency of APOs, severe maternal morbidity, maternal mortality). Available data rely on studies that have intervened on single risk factors such as weight loss to reduce gestational diabetes risk rather than comprehensive CVH promotion.[77] Randomized controlled trials that have focused on prepregnancy behavioral interventions have improved individual CVH metrics such as diet, smoking cessation, or body mass index before pregnancy.[78–84] In a cohort study of individuals with severe obesity, bariatric surgery before pregnancy was associated with substantially lower risks of gestational diabetes (odds ratio, 0.21 [95% CI, 0.12–0.36]) and HDP (odds ratio, 0.38 [95% CI, 0.27–0.53]) but higher risks of SGA birth (odds ratio, 2.18 [95% CI, 1.41–3.38]).[85]

Data are even more limited for longer-term maternal and offspring outcomes after prepregnancy interventions. Among 2 studies with 6-year postpartum follow-up after a randomized prepregnancy lifestyle intervention for individuals with obesity and infertility before fertility care, 1 study found that mothers who successfully lost weight with the intervention had better cardiometabolic health 6 years postpartum compared with control subjects, and the other found that children of individuals who underwent the lifestyle intervention had better left ventricular structure and function.[86,87] However, data are limited by small sample sizes, attrition bias, and conflicting findings across studies for better CVH in offspring.[87,88]

Postpartum interventions, especially those that result in weight loss, have been shown to improve CVH, but the consequences of these interventions on CVD outcomes in subsequent pregnancy are limited but could inform similar prepregnancy interventions. Among women with a history of preeclampsia, small trials focused on behavior changes have demonstrated improvement in physical activity postpartum.[89–91] Among women with a history of gestational diabetes, lifestyle interventions conducted in the year after delivery that targeted overweight/obesity resulted in modest weight loss, increased physical activity, and improved glycemic measures.[92] Health care delivery strategies such as transitional clinics for postpartum care after APOs, patient navigation, and integration of maternal care at pediatric visits have been suggested as potential opportunities to improve health but have not yet been rigorously evaluated for their effects on CVH outcomes.[93–95]

A NEED FOR CLINICAL TRIALS TARGETING PREPREGNANCY CVH

The American College of Obstetricians and Gynecologists strongly advocates the assessment and promotion of preconception health behaviors and factors in individuals of reproductive age.[96] However, as just reviewed, intervening on CVH before conception remains a critical research gap. If a trial promoting CVH yielded positive results in mitigating APOs and improving maternal and offspring outcomes, it could be practice changing and provide needed impetus for clinicians and reproductive-aged individuals to be more cognizant about achieving better CVH. At the population level, primordial prevention with maintenance of ideal CVH is an overarching goal throughout the life course. Specifically, in the context of prepregnancy CVH, adolescence (before a first pregnancy) marks a critical transition in the life course when health behaviors are becoming more firmly established and distinct CVH trajectories are identifiable.[97]

Key Considerations for Trial Design Focused on Clinical Outcomes

Multiple elements need to be considered in the design of a trial that tests whether interventions initiated before pregnancy aimed at holistically promoting CVH will modify maternal and offspring outcomes. First, careful planning will be needed to recruit and retain a large, diverse sample population. Particular attention will need to be given to developing culturally cognizant strategies and to oversample individuals from groups who are underrepresented in clinical trials and who bear a disproportionate burden of unfavorable CVH, APOs, and CVD. These groups include populations that are underrepresented on the basis of racial and ethnic identity and sexual and gender identity and individuals with adverse social determinants of health. Within racial and ethnic groups, disaggregation of larger categories such as Asian (eg, Chinese, Filipina, Japanese, Korean, Vietnamese, Asian Indian) and Hispanic (eg, Mexican, Puerto Rican, Central and South American) is necessary given the heterogeneity across subgroups. Rigorous collection of self-reported race and ethnicity, sexual and gender identity, and social determinants of health, along with strategies to ensure diversity and inclusion in recruitment, will be important to understand generalizability of the treatment effect of an intervention on APOs and offspring outcomes in different populations.

Second, selection of inclusion criteria may need to focus on subsets of individuals who are capable of, open to, or actively seeking to become pregnant. To adequately power a study, it may be prudent to enrich the trial sample with a population at higher risk. For example, a trial could focus on only 1 metric such as overweight or obesity,

given that this is the most prevalent risk factor for APOs in pregnant individuals. However, risk factors often co-occur in birthing and pregnant individuals, and associations of maternal CVH with APOs and offspring CVH are not driven by any single CVH metric.[15,61] Therefore, inclusion criteria should consider selection of individuals based on unfavorable levels of multiple CVH metrics. A trial could be designed that excludes those at highest risk who already meet medical treatment thresholds for hypertension, diabetes, or hyperlipidemia, thereby focusing on the population with intermediate risk factor levels (elevated blood pressure not characterized as hypertension, prediabetes, borderline dyslipidemia) for whom intervention guidelines are especially lacking.

Third, interventions tested should include multiple components that include a focus on health behavior changes (eg, diet, exercise) with or without pharmacotherapies based on options known to be safe during pregnancy. For example, although statins have long been avoided in pregnancy, there is growing consensus that some agents (ie, hydrophilic statins such as pravastatin) may be safe and may reduce the risk of APOs.[98,99] However, it is not known whether particular components of CVH are most salient to focus on to improve pregnancy and long-term outcomes. To inform optimal trial design, foundational work, including feasibility studies to test recruitment approaches, retention strategies, and acceptability of interventions, will be needed. One hypothetical trial is outlined with the PICOTS (population, intervention, comparison, outcome, timing, setting) framework in Table 2 and is meant as a single example of what could be considered.

Table 2. Design of a Potential Clinical Trial to Test Whether Promoting CVH Before Pregnancy Improves Outcomes in Pregnant and Postpartum Individuals and Offspring Using the PICOTS Framework

Population	Pregnancy-capable individuals open to or actively seeking to become pregnant, with consideration for the following: Age: 25–44 y CVH metrics: with overweight or obesity and intermediate levels for blood pressure, glucose, and cholesterol who do not currently meet criteria for pharmacotherapy Exclusions: history of infertility Inclusive of transgender and gender-diverse individuals Oversample individuals with adverse social determinants of health (eg, household income at >200% of the federal poverty level, receiving WIC support) Oversample historically excluded racial and ethnic groups (eg, Black, Hispanic, and Asian American/Native Hawaiian/Pacific Islander individuals) Oversample individuals with prior APOs to further increase study power
Intervention	Moderate-intensity lifestyle intervention targeting multiple CVH factors (diet, physical activity, sleep) Adjunctive pharmacotherapy at cardiovascular risk factor levels below current standard of care for pharmacological intervention: BP >120/80 mm Hg Fasting blood glucose >100 mg/dL or HbA1c >5.7% Non-HDL cholesterol >160 mg/dL or total cholesterol/HDL ratio >5
Comparison	Standard of care, including provision of relevant health information on promoting CVH and referral to clinicians if any abnormal CVH metrics
Outcome	Coprimary outcomes Maternal composite outcome consisting of the following: Preeclampsia Gestational hypertension Gestational diabetes Severe maternal morbidity (eg, acute myocardial infarction, cardiac arrest, heart failure) Pregnancy-related mortality Offspring composite outcome consisting of the following: Fetal death Preterm birth SGA birth Secondary outcomes Individual components of the above composite outcomes Rigorous data collection and reporting of subgroup analyses based on sociodemographic factors (eg, race and ethnicity, gender identity)
Timing	5-y trial phase with 4 y of enrollment and an additional 1 y of follow-up (average follow-up, 3 y; range, 1–5 y)
Setting	Pragmatic trial that leverages health care systems such as federally qualified health centers, Indian Health Service clinics, HMOs, and large research networks

APO indicates adverse pregnancy outcome; BP, blood pressure; CVH, cardiovascular health; HbA1c, hemoglobin A1c; HDL, high-density lipoprotein; HMO, health maintenance organization; PICOTS, population, intervention, comparison, outcome, timing, setting; SGA, small for gestational age; and WIC, Women, Infants, and Children supplemental nutrition program.

Targeting Stress to Promote CVH

Psychological health, stress, and resilience are inextricably linked with CVH and are identified by the AHA as foundational determinants in optimizing CVH.[9] This is based on robust evidence of the association between stress and health outcomes, which include APOs and CVD.[100,101] Racism is a structural driver of disproportionate burden of psychosocial stress, and historically excluded women have different life experiences such as repeated episodes of discrimination that are associated with unfavorable CVH and risk of APOs compared with White women. Long-term exposures to stress cumulatively over the life course leads to weathering, increased allostatic load (ie, cumulative biological stress), and impaired health.[102–104] One coping mechanism among Black women, called the superwoman schema[105] because of the need to display strength in the face of long-term adversity, may also negatively affect maternal health outcomes.[106,107] Culturally responsive stress reduction and mindfulness-based interventions[108–111] that are sensitive to systemic barriers may offer a means to buffer stress and reduce maladaptive coping. Interventions targeting

stress in the prepregnancy period to improve maternal health outcomes are a critical evidence gap and should be considered in the context of multilevel policy changes that simultaneously address structural and systemic barriers to optimal health.

Digital Technologies to Promote CVH

Given that adolescents and young adults are frequent users of digital technologies (eg, smartphones, social media, mobile applications [apps]), leveraging these tools to deliver counseling could be an effective method to promote prepregnancy CVH.[112] Digital health interventions may offer increased accessibility to support individuals with low socioeconomic status, who often have more barriers to health care access and in-person visits. The integration of digital, face-to-face, and telephone interactions with health care teams may increase engagement with healthy behavior change interventions for sustainability and long-term impact.[112] In 1 study, a health information tool was used to screen for 102 health risks and to deliver tailored prepregnancy counseling to empower high-risk Black individuals to improve prepregnancy health across various domains, including emotional and mental health, nutrition and activity, and substance use.[83,84] The tool included a conversational agent, Gabby, who provided culturally sensitive and empathetic prepregnancy risk assessment and increased the proportion of individuals engaged in behavior change through person-centered decision making and goal setting. Social marketing campaigns through media outlets have shown promise in increasing awareness of the importance of prepregnancy health and existing maternal health outcomes disparities.[107,113] Further adaptation of evidence-based mobile health lifestyle interventions promoting ideal CVH[107,113] to focus on prepregnancy health should also be studied.

COMMUNITY-ENGAGED DESIGN AND IMPLEMENTATION OF PREPREGNANCY CVH INTERVENTIONS

To achieve health equity among birthing individuals, attentive design of community-based interventions is crucial and will require community-centered engagement at every stage of the research process. This is especially imperative for populations with an increased frequency of unfavorable CVH, including people of underrepresented races and ethnicities who have intersecting barriers to optimal health attributable to social determinants of health before, during, and after pregnancy.[107,114]

Innovative strategies that are culturally cognizant and recognize sociocultural and environmental contexts to optimize CVH are needed. For example, Black women are significantly more likely than White women to have unfavorable CVH with multiple prepregnancy cardiovascular risk factors (eg, obesity, diabetes), and risk is higher among individuals within lower economic strata.[115,116] Hispanic women, in particular those from certain subgroups (eg, Puerto Rican women), may experience a disproportionate burden of adverse social determinants of health such as poor health care access and quality and language barriers that contribute to disparate risk of APOs and CVD.[117] In the limited data that are available from birthing individuals from Asian subgroups, Asian Indian compared with White pregnant individuals have a higher risk of gestational diabetes. Thus, consideration of sociocultural context of individuals from various backgrounds, including consideration of nativity and acculturation along with experience of structural barriers (eg, racism, built environment, health care access), is of key importance in the design of interventions. Specifically, tailoring and adaption through user-centered and participatory approaches can bolster the effectiveness and relevance of interventions.

Effectiveness and relevance can also be optimized through engagement with community steering committees or advisory boards with key stakeholders and participants from the target population.[118–123] This will also facilitate the design of interventions that maximize strengths and resources present within communities to promote an asset-based approach that empowers disenfranchised communities. For example, the harnessing of civic engagement and community advocacy as a means to collectively address health disparities has resulted in improved cardiovascular risk factors (eg, blood pressure, physical activity) in Black women.[124,125] There is also evidence to support integration of similar models fostering volunteerism to promote favorable CVH among Hispanic females.[126] Such interventions can incorporate peer leaders (such as community health workers, or promotoras) who serve as role models and provide social support to promote CVH.[127–129] Meeting women in the community by embedding place-based interventions within their neighborhoods—at venues such as hair salons, churches, public housing, college campuses, and workplaces—is another potential strategy to promote CVH.[115,116,130] Further investigation is needed to understand how incorporating interpersonal relationships and social support through partners, friends, and community may also optimize interventions.

ADDRESSING STRUCTURAL DETERMINANTS OF CVH WITH POLICY-LEVEL INTERVENTIONS

To substantially improve prepregnancy CVH and downstream maternal and offspring outcomes, the wider influence of multifaceted structural and social determinants of health must be acknowledged and addressed. This

Table 3. Tailored Interventions to Promote CVH at Various Prepregnancy Life Stages and Across Ecological Levels

	Prepregnancy life stages		
	People with no current intention to become pregnant (includes adolescents)	**People with intention to become pregnant (prepregnancy)**	**People with intention to become pregnant again (interpregnancy)**
Individual	Lifestyle coaching Stress reduction Sleep interventions Weight loss pharmacotherapy/ bariatric surgery Text-based interventions	In addition to those for people with no current intention to become pregnant: Lifestyle-based weight loss interventions Pharmacotherapy safe during pregnancy to achieve optimal risk factor control	Lifestyle-based interventions for people with a history of APOs Pharmacotherapy among individuals after a history of APOs (eg, metformin or GLP-1RA after gestational diabetes) Cognitive behavioral therapy and pharmacologic therapy for postpartum depression
Community	Civic engagement	Peer-led support groups	Peer-led parenting groups Home visitation programs focused on stress, postpartum depression
Population	Social marketing campaigns to raise awareness about CVH promotion Built environment changes (eg, access to healthy foods, green spaces)		
Policy	Dismantled structural racism Fair housing practices Access to education, equitable employment opportunities, and paid family leave Diversity in health care workforce Continuous health insurance coverage to ensure high-quality prepregnancy counseling and to minimize interruptions in access		

APO indicates adverse pregnancy outcome; CVH, cardiovascular health; GLP-1RA, glucagon-like peptide-1 receptor agonist; SNAP, Supplemental Nutrition Assistance Program; and WIC, Women, Infants, and Children supplemental nutrition program.

requires a sincere appreciation that optimizing opportunities for CVH is not solely an individual responsibility but requires health system and society-level interventions. There is well-established evidence that structural inequities such as disenfranchised neighborhoods and physical environments, the wealth gap, inadequate access to quality health care, and food and housing insecurity are barriers for optimal CVH.[131,132] Dismantling structural racism and discriminatory policies, the root causes of disparities in CVH and APOs, is therefore critical.[131,133] Building CVH-promoting environments and contexts that support optimal CVH for all birthing individuals requires will for policy change.[134] Policy-level interventions are needed to ensure social and reproductive justice and to enable health over the life course, including during the continuum of prepregnancy and perinatal care.[135,136] Making ideal CVH the social norm in a community can be achieved with the integration of equitable opportunities to maintain healthier lifestyle practices such as increasing access to healthier and affordable foods (local supermarkets, grocery stores), greener and walkable neighborhoods, free or subsidized fitness center memberships, and safe and proximate parks and recreational facilities.[135] Enhancing economic empowerment and investment in neighborhoods through access to employment and education opportunities can profoundly influence the CVH and well-being of socioeconomically disenfranchised individuals[107,134,137] because these factors are directly linked to access to high-quality health care. The Black Maternal Health Caucus through the Black Maternal Health Momnibus has introduced legislation to address the maternal health crisis through community partnerships, diversity in the perinatal health care workforce, digital tools, and optimized health care coverage models that promote continuity and access (eg, prepregnancy and interpregnancy care) to improve quality of care and to mitigate disparities.[107] Given data that lacking preconception health insurance is associated with lower levels of pregnancy care, later initiation of prenatal care, and lower levels of postpartum care, ensuring uninterrupted access remains a critical gap, with insurance transitions or "churn" being common before and after childbirth.[138,139] Multilevel interventions will be needed that are tailored to the unique stages of the prepregnancy period. Potential examples are outlined in Table 3.

UNANSWERED QUESTIONS AND FUTURE DIRECTIONS

A growing body of evidence supports associations between CVH and APO and between APO and CVD and builds on the well-established pathways known to exist between CVH and CVD across the life course and intergenerationally. However, important knowledge gaps (a selection of which are presented in Table 4) remain in the epidemiology and pathophysiology of CVH and effective interventional strategies to promote CVH. A central question that remains is whether the associations among CVH, APOs, and CVD are epiphenomena or causally related; this question has direct consequences for the design of prevention and treatment strategies to improve CVH to decrease the frequency of APOs and the risk of CVD. Specifically, it is not known whether

Table 4. Key Knowledge Gaps in Mechanistic Pathways, Effective Interventions, and Implementation of Strategies to Equitably Promote Prepregnancy CVH

Pathophysiology	What are the pathophysiological mechanisms and pathways linking prepregnancy CVH, APOs, and CVD? Could proteomic markers assessed before pregnancy identify individuals at highest risk of APOs for whom increased surveillance or intensive risk factor modification may reduce risk of APOs? Are there mechanistic targets that can inform novel therapeutic development to improve prepregnancy CVH and pregnancy outcomes?
Interventional research	Which metrics of the CVH construct are most salient to target in a multicomponent intervention in the prepregnancy period to improve maternal and offspring outcomes? Should thresholds for treatment of cardiovascular risk factors be different in pregnancy-capable individuals with hypertension (eg, goal BP level <130/80 mm Hg vs <140/90 mm Hg before pregnancy)? How should interventions be designed to incorporate and address social determinants of health, psychological health, and stress to improve prepregnancy CVH? How should we identify individuals at highest absolute risk (eg, low CVH, biomarkers) to prioritize for testing interventions? Do interventions targeting the interpregnancy period improve outcomes of the subsequent pregnancy and offspring? Do interventions targeting prepregnancy CVH reduce the risk of long-term kidney disease?
Dissemination and implementation research	Is the proposed intervention adaptable for resource-limited populations? Is the proposed intervention culturally tailored to maximize benefit in diverse communities? Can technology-based approaches (eg, virtual reminders, mHealth) optimize delivery of interventions to improve prepregnancy CVH?
Health equity considerations	Can healthcare system–based models that provide patient navigation and peer support to address barriers that exist help to optimize prepregnancy CVH? What are optimal strategies to ensure equitable recruitment of individuals who do not have routine health care access, particularly in the prepregnancy period? What strategies are most effective in engaging key stakeholders from communities from the beginning of intervention development to process improvement in the implementation phases? Are interventions generalizable to different populations, including across race and ethnicity, socioeconomic status, and gender identity?

APO indicates adverse pregnancy outcome; BP, blood pressure; CVD, cardiovascular disease; CVH, cardiovascular health; and mHealth, mobile health.

APOs are a marker or mediator of the CVH-CVD relationship. Indeed, there are likely multiple direct and indirect pathways by which CVH may influence maternal and offspring CVH. This supports an emphasis on primordial prevention to preserve or improve CVH beginning in childhood; however, it is not known whether specific metrics in the CVH construct are most salient to focus on at different life stages (eg, before pregnancy to reduce risk of APOs). Similarly, it is not known whether strategies that reduce the frequency of APOs, by virtue of that reduction, also improve long-term health outcomes for the birthing individual and offspring. Observational evidence for several shared pathways at the CVH-APO-CVD intersection supports that the protection conferred by higher CVH for both APO and CVD risk reduction may be more than the sum of its parts.[9,38,61] However, individual-level promotion of prepregnancy CVH will be limited among individuals with unintended pregnancies, which account for nearly half of all pregnancies; unintended pregnancies are disproportionately higher among low-income individuals and people of underrepresented races and ethnicities, who are also at greater risk of poor CVH and CVD.[140] This emphasizes that population health and policy-level interventions are key, including strategies to equitably reduce unintended pregnancies (eg, access to desired long-acting reversible contraceptives, implementation of the One Key Question) and targeting CVH beginning early in the life course before the reproductive years.[141]

CONCLUSIONS

Substantial opportunity exists to improve health across the life course and generations by targeting prepregnancy CVH in the birthing individual. Numerous epidemiological studies demonstrate that CVH is a risk factor for both APOs and CVD in the birthing individual and offspring. Animal models and in vitro experiments suggest a strong link among prepregnancy CVH, APOs, and CVD. Poor prepregnancy CVH is highly prevalent in reproductive-aged individuals and disproportionately so among individuals with higher burden of adverse social factors. Identification of individuals with unfavorable CVH before pregnancy, as early in the life course as childhood and adolescence, is a necessary first step to increase awareness of risk. Clinical trial data are needed to demonstrate whether CVH interventions beginning before pregnancy will modify maternal and offspring outcomes, including APOs and CVD. Unfavorable CVH has been associated with a broad range of antecedent individual-level and structural determinants. Therefore, effective interventions should consider multilevel approaches at the individual, community, and society levels. Persistent racial, ethnic, and socioeconomic disparities illustrate the critical importance of future investigations ensuring that proposed interventions are created, implemented, and evaluated with an equity focus. The prepregnancy period offers a unique window of opportunity to address the growing public health burden of APOs and to interrupt the intergenerational transmission of poor CVH.

ARTICLE INFORMATION

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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REFERENCES

1. Petersen EE, Davis NL, Goodman D, Cox S, Mayes N, Johnston E, Syverson C, Seed K, Shapiro-Mendoza CK, Callaghan WM. Vital signs: pregnancy-related deaths, United States, 2011–2015, and strategies for prevention, 13 states, 2013–2017. *MMWR Morb Mortal Wkly Rep*. 2019;68:423–429. doi: 10.15585/mmwr.mm6818e1
2. Creanga AA, Syverson C, Seed K, Callaghan WM. Pregnancy-related mortality in the United States, 2011-2013. *Obstet Gynecol*. 2017;130:366–373. doi: 10.1097/AOG.0000000000002114
3. Mehta LS, Sharma G, Creanga AA, Hameed AB, Hollier LM, Johnson JC, Leffert L, McCullough LD, Mujahid MS, Watson K, et al; on behalf of the American Heart Association Advocacy Coordinating Committee. Call to action: maternal health and saving mothers: a policy statement from the American Heart Association. *Circulation*. 2021;144:e251–e269. doi: 10.1161/CIR.0000000000001000
4. Freaney PM, Harrington K, Molsberry R, Perak AM, Wang MC, Grobman W, Greenland P, Allen NB, Capewell S, O'Flaherty M. Temporal trends in adverse pregnancy outcomes in birthing individuals aged 15 to 44 years in the United States, 2007 to 2019. *J Am Heart Assoc*. 2022;11:e025050. doi: 10.1161/JAHA.121.025050
5. Shah NS, Wang MC, Freaney PM, Perak AM, Carnethon MR, Kandula NR, Gunderson EP, Bullard KM, Grobman WA, O'Brien MJ, et al. Trends in gestational diabetes at first live birth by race and ethnicity in the US, 2011-2019. *JAMA*. 2021;326:660–669. doi: 10.1001/jama.2021.7217
6. Grobman WA, Parker CB, Willinger M, Wing DA, Silver RM, Wapner RJ, Simhan HN, Parry S, Mercer BM, Haas DM, et al; Eunice Kennedy Shriver National Institute of Child Health and Human Development Nulliparous Pregnancy Outcomes Study: Monitoring Mothers-to-Be (nuMoM2b) Network. Racial disparities in adverse pregnancy outcomes and psychosocial stress. *Obstet Gynecol*. 2018;131:328–335. doi: 10.1097/AOG.0000000000002441
7. Parikh NI, Gonzalez JM, Anderson CAM, Judd SE, Rexrode KM, Hlatky MA, Gunderson EP, Stuart JJ, Vaidya D; on behalf of the American Heart Association Council on Epidemiology and Prevention; Council on Arteriosclerosis, Thrombosis and Vascular Biology; Council on Cardiovascular and Stroke Nursing; and the Stroke Council. Adverse pregnancy outcomes and cardiovascular disease risk: unique opportunities for cardiovascular disease prevention in women: a scientific statement from the American Heart Association. *Circulation*. 2021;143:e902–e916. doi: 10.1161/CIR.0000000000000961
8. Barrett PM, McCarthy FP, Kublickiene K, Cormican S, Judge C, Evans M, Kublickas M, Perry IJ, Stenvinkel P, Khashan AS. Adverse pregnancy outcomes and long-term maternal kidney disease: a systematic review and meta-analysis. *JAMA Netw Open*. 2020;3:e1920964. doi: 10.1001/jamanetworkopen.2019.20964
9. Lloyd-Jones DM, Allen NB, Anderson CA, Black T, Brewer LC, Foraker RE, Grandner MA, Lavretsky H, Perak AM, Sharma G. Life's Essential 8: updating and enhancing the American Heart Association's construct of cardiovascular health: a presidential advisory from the American Heart Association. *Circulation*. 2022;146:e18–e43: doi: 10.1161/CIR. 0000000000001078.
10. Lloyd-Jones DM, Hong Y, Labarthe D, Mozaffarian D, Appel LJ, Van Horn L, Greenlund K, Daniels S, Nichol G, Tomaselli GF, et al; on behalf of the American Heart Association Strategic Planning Task Force and Statistics Committee. Defining and setting national goals for cardiovascular health promotion and disease reduction: the American Heart Association's Strategic Impact Goal through 2020 and beyond. *Circulation*. 2010;121:586–613. doi: 10.1161/CIRCULATIONAHA.109.192703
11. Tsao CW, Aday AW, Almarzooq ZI, Alonso A, Beaton AZ, Bittencourt MS, Boehme AK, Buxton AE, Carson AP, Commodore-Mensah Y, et al. Heart disease and stroke statistics–2022 update: a report from the American Heart Association [published correction appears in *Circulation*. 2022;146:e141]. *Circulation*. 2022;145:e153–e639. doi: 10.1161/CIR.0000000000001052
12. Brown HL, Warner JJ, Gianos E, Gulati M, Hill AJ, Hollier LM, Rosen SE, Rosser ML, Wenger NK; on behalf of the American Heart Association and the American College of Obstetricians and Gynecologists. Promoting risk identification and reduction of cardiovascular disease in women through collaboration with obstetricians and gynecologists: a presidential advisory from the American Heart Association and the American College of Obstetricians and Gynecologists. *Circulation*. 2018;137:e843–e852. doi: 10.1161/CIR.0000000000000582
13. Colby S, Ortman JM. Projections of the size and composition of the US population: 2014 to 2060. 2015. US Census Bureau. Accessed on January 12, 2022. https://mronline.org/wp-content/uploads/2019/08/p25-1143.pdf
14. Zanetti D, Tikkanen E, Gustafsson S, Priest JR, Burgess S, Ingelsson E. Birthweight, type 2 diabetes mellitus, and cardiovascular disease: addressing the barker hypothesis with mendelian randomization. *Circ Genom Precis Med*. 2018;11:e002054. doi: 10.1161/CIRCGEN.117.002054
15. Perak AM, Ning H, Khan SS, Van Horn LV, Grobman WA, Lloyd-Jones DM. Cardiovascular health among pregnant women, aged 20 to 44 years, in the United States. *J Am Heart Assoc*. 2020;9:e015123. doi: 10.1161/JAHA.119.015123
16. Virani SS, Alonso A, Benjamin EJ, Bittencourt MS, Callaway CW, Carson AP, Chamberlain AM, Chang AR, Cheng S, Delling FN, et al; on behalf of the American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics–2020 update: a report from the American Heart Association. *Circulation*. 2020;141:e139–e596. doi: 10.1161/CIR.0000000000000757
17. Amyx M, Xiong X, Xie Y, Buekens P. Racial/ethnic differences in sleep disorders and reporting of trouble sleeping among women of childbearing age in the United States. *Matern Child Health J*. 2017;21:306–314. doi: 10.1007/s10995-016-2115-9
18. Feinstein L, McWhorter KL, Gaston SA, Troxel WM, Sharkey KM, Jackson CL. Racial/ethnic disparities in sleep duration and sleep disturbances among pregnant and non-pregnant women in the United States. *J Sleep Res*. 2020;29:e13000. doi: 10.1111/jsr.13000
19. Heck JL, Jones EJ, Bohn D, McCage S, Parker JG, Parker M, Pierce SL, Campbell J. Maternal mortality among American Indian/Alaska Native

women: a scoping review. *J Womens Health (Larchmt)*. 2021;30:220–229. doi: 10.1089/jwh.2020.8890
20. Lane-Cordova AD, Jerome GJ, Paluch AE, Bustamante EE, LaMonte MJ, Pate RR, Weaver RG, Webber-Ritchey KJ, Gibbs BB; on behalf of the Committee on Physical Activity of the American Heart Association Council on Lifestyle and Cardiometabolic Health. Supporting physical activity in patients and populations during life events and transitions: a scientific statement from the American Heart Association. *Circulation*. 2022;145:e117–e128. doi: 10.1161/CIR.0000000000001035
21. Hales CM, Carroll MD, Fryar CD, Ogden CL. Prevalence of obesity and severe obesity among adults: United States, 2017–2018. National Center for Health Statistics. NCHS Data Brief, No 360. 2020. Accessed October 18, 2022. https://stacks.cdc.gov/view/cdc/49223
22. Azeez O, Kulkarni A, Kuklina EV, Kim SY, Cox S. Hypertension and diabetes in non-pregnant women of reproductive age in the United States. *Prev Chronic Dis*. 2019;16:E146. doi: 10.5888/pcd16.190105
23. Wang MC, Freaney PM, Perak AM, Allen NB, Greenland P, Grobman WA, Phillips SM, Lloyd-Jones DM, Khan SS. Trends in prepregnancy cardiovascular health in the United States, 2011-2019. *Am J Prev Cardiol*. 2021;7:100229. doi: 10.1016/j.ajpc.2021.100229
24. Cameron NA, Freaney PM, Wang MC, Perak AM, Dolan BM, O'Brien MJ, Tandon SD, Davis MM, Grobman WA, Allen NB, et al. Geographic differences in prepregnancy cardiometabolic health in the United States, 2016 through 2019. *Circulation*. 2022;145:549–551. doi: 10.1161/CIRCULATIONAHA.121.057107
25. Wang MC, Freaney PM, Perak AM, Greenland P, Lloyd-Jones DM, Grobman WA, Khan SS. Trends in prepregnancy obesity and association with adverse pregnancy outcomes in the United States, 2013 to 2018. *J Am Heart Assoc*. 2021;10:e020717. doi: 10.1161/JAHA.120.020717
26. Singh GK, DiBari JN. Marked disparities in pre-pregnancy obesity and overweight prevalence among US women by race/ethnicity, nativity/immigrant status, and sociodemographic characteristics, 2012–2014. *J Obes*. 2019;2019:2419263. doi: 10.1155/2019/2419263
27. Harville EW, Crook CE, Bazzano LA, Woo JG, Burns TL, Raitakari O, Urbina EM, Venn A, Jacobs DR Jr, Steinberger J, et al; i3C Consortium. Cardiovascular risk factors before and during pregnancy: does pregnancy unmask or initiate risk? *J Obstet Gynaecol Res*. 2021;47:3849–3856. doi: 10.1111/jog.14971
28. Wang MC, Freaney PM, Perak AM, Allen NB, Greenland P, Grobman WA, Lloyd-Jones DM, Khan SS. Association of pre-pregnancy cardiovascular risk factor burden with adverse maternal and offspring outcomes. *Eur J Prev Cardiol*. 2022;29:e156–e158. doi: 10.1093/eurjpc/zwab121
29. Harville EW, Viikari JS, Raitakari OT. Preconception cardiovascular risk factors and pregnancy outcome. *Epidemiology*. 2011;22:724–730. doi: 10.1097/EDE.0b013e318225c960
30. Magnussen EB, Vatten LJ, Lund-Nilsen TI, Salvesen KA, Davey Smith G, Romundstad PR. Prepregnancy cardiovascular risk factors as predictors of pre-eclampsia: population based cohort study. *BMJ*. 2007;335:978. doi: 10.1136/bmj.39366.416817.BE
31. Retnakaran R, Shah BR. Divergent trajectories of cardiovascular risk factors in the years before pregnancy in women with and without gestational diabetes mellitus: a population-based study. *Diabetes Care*. 2020;43:2500–2508. doi: 10.2337/dc20-1037
32. Han ES, Krauss RM, Xu F, Sridhar SB, Ferrara A, Quesenberry CP, Hedderson MM. Prepregnancy adverse lipid profile and subsequent risk of gestational diabetes. *J Clin Endocrinol Metab*. 2016;101:2721–2727. doi: 10.1210/jc.2015-3904
33. Retnakaran R, Shah BR. The adverse cardiovascular risk factor profile of women with pre-eclampsia develops over time in the years before pregnancy. *BJOG*. 2022;129:1512–1520. doi: 10.1111/1471-0528.17084
34. Santos S, Voerman E, Amiano P, Barros H, Beilin LJ, Bergström A, Charles MA, Chatzi L, Chevrier C, Chrousos GP, et al. Impact of maternal body mass index and gestational weight gain on pregnancy complications: an individual participant data meta-analysis of European, North American and Australian cohorts. *BJOG*. 2019;126:984–995. doi: 10.1111/1471-0528.15661
35. LifeCycle Project-Maternal Obesity and Childhood Outcomes Study Group; Voerman E, Santos S, Inskip H, Amiano P, Barros H, Charles M-A, Chatzi L, Chrousos GP, Corpeleijn E, Crozier S, et al. Association of gestational weight gain with adverse maternal and infant outcomes. *JAMA*. 2019;321:1702–1715. doi: 10.1001/jama.2019.3820
36. Wei Y, Xu Q, Yang H, Yang Y, Wang L, Chen H, Anderson C, Liu X, Song G, Li Q, et al. Preconception diabetes mellitus and adverse pregnancy outcomes in over 6.4 million women: a population-based cohort study in China. *PLoS Med*. 2019;16:e1002926. doi: 10.1371/journal.pmed.1002926
37. Arvizu M, Stuart JJ, Rich-Edwards JW, Gaskins AJ, Rosner B, Chavarro JE. Prepregnancy adherence to dietary recommendations for the prevention of cardiovascular disease in relation to risk of hypertensive disorders of pregnancy. *Am J Clin Nutr*. 2020;112:1429–1437. doi: 10.1093/ajcn/nqaa214
38. Perak AM, Lancki N, Kuang A, Labarthe DR, Allen NB, Shah SH, Lowe LP, Grobman WA, Scholtens DM, Lloyd-Jones DM, et al; Hyperglycemia and Adverse Pregnancy Outcome (HAPO) Study Cooperative Research Group. Associations of gestational cardiovascular health with pregnancy outcomes: the Hyperglycemia and Adverse Pregnancy Outcome study. *Am J Obstet Gynecol*. 2021;224:210.e1–210.e17. doi: 10.1016/j.ajog.2020.07.053
39. Jarman M, Mathe N, Ramazani F, Pakseresht M, Robson PJ, Johnson ST, Bell RC; APrON and ENRICH Study Teams. Dietary patterns prior to pregnancy and associations with pregnancy complications. *Nutrients*. 2018;10:914. doi: 10.3390/nu10070914
40. Whitaker KM, Ingram KH, Appiah D, Nicholson WK, Bennett WL, Lewis CE, Reis JP, Schreiner PJ, Gunderson EP. Prepregnancy fitness and risk of gestational diabetes: a longitudinal analysis. *Med Sci Sports Exerc*. 2018;50:1613–1619. doi: 10.1249/MSS.0000000000001600
41. Catov JM, Parker CB, Gibbs BB, Bann CM, Carper B, Silver RM, Simhan HN, Parry S, Chung JH, Haas DM, et al; NICHD NuMoM2b and NHLBI NuMoM2b Heart Health Study Network. Patterns of leisure-time physical activity across pregnancy and adverse pregnancy outcomes. *Int J Behav Nutr Phys Act*. 2018;15:68. doi: 10.1186/s12966-018-0701-5
42. Martínez-Hortelano JA, Cavero-Redondo I, Álvarez-Bueno C, Sanabria-Martínez G, Poyatos-León R, Martínez-Vizcaíno V. Interpregnancy weight change and hypertension during pregnancy: a systematic review and meta-analysis. *Obstet Gynecol*. 2020;135:68–79. doi: 10.1097/AOG.0000000000003573
43. Tita AT, Szychowski JM, Boggess K, Dugoff L, Sibai B, Lawrence K, Hughes BL, Bell J, Aagaard K, Edwards RK, et al; Chronic Hypertension and Pregnancy (CHAP) Trial Consortium. Treatment for mild chronic hypertension during pregnancy. *N Engl J Med*. 2022;386:1781–1792. doi: 10.1056/NEJMoa2201295
44. Nobles CJ, Mendola P, Mumford SL, Silver RM, Kim K, Andriessen VC, Connell M, Sjaarda L, Perkins NJ, Schisterman EF. Preconception blood pressure and its change into early pregnancy: early risk factors for preeclampsia and gestational hypertension. *Hypertension*. 2020;76:922–929. doi: 10.1161/HYPERTENSIONAHA.120.14875
45. Baumfeld Y, Novack L, Wiznitzer A, Sheiner E, Henkin Y, Sherf M, Novack V. Pre-conception dyslipidemia is associated with development of preeclampsia and gestational diabetes mellitus. *PLoS One*. 2015;10:e0139164. doi: 10.1371/journal.pone.0139164
46. Kwapong YA, Boakye E, Wang G, Hong X, Lewey J, Mamas MA, Wu P, Blaha MJ, Nasir K, Hays AG, et al. Maternal glycemic spectrum and adverse pregnancy and perinatal outcomes in a multiracial US cohort. *J Cardiovasc Dev Dis*. 2022;9:179. doi: 10.3390/jcdd9060179
47. Lu Q, Zhang X, Wang Y, Li J, Xu Y, Song X, Su S, Zhu X, Vitiello MV, Shi J, et al. Sleep disturbances during pregnancy and adverse maternal and fetal outcomes: a systematic review and meta-analysis. *Sleep Med Rev*. 2021;58:101436. doi: 10.1016/j.smrv.2021.101436
48. Facco FL, Parker CB, Hunter S, Reid KJ, Zee PC, Silver RM, Haas DM, Chung JH, Pien GW, Nhan-Chang CL, et al; NICHD NuMoM2b and NHLBI NuMoM2b Heart Health Study Networks. Association of adverse pregnancy outcomes with self-reported measures of sleep duration and timing in women who are nulliparous. *J Clin Sleep Med*. 2018;14:2047–2056. doi: 10.5664/jcsm.7534
49. Al Khalaf S, Bodunde E, Maher GM, O'Reilly ÉJ, McCarthy FP, O'Shaughnessy MM, O'Neill SM, Khashan AS. Chronic kidney disease and adverse pregnancy outcomes: a systematic review and meta-analysis. *Am J Obstet Gynecol*. 2022;226:656–670.e32. doi: 10.1016/j.ajog.2021.10.037
50. Fleming TP, Watkins AJ, Velazquez MA, Mathers JC, Prentice AM, Stephenson J, Barker M, Saffery R, Yajnik CS, Eckert JJ, et al. Origins of lifetime health around the time of conception: causes and consequences. *Lancet*. 2018;391:1842–1852. doi: 10.1016/S0140-6736(18)30312-X
51. Crump C, Groves A, Sundquist J, Sundquist K. Association of preterm birth with long-term risk of heart failure into adulthood. *JAMA Pediatr*. 2021;175:689–697. doi: 10.1001/jamapediatrics.2021.0131
52. Crump C, Howell EA, Stroustrup A, McLaughlin MA, Sundquist J, Sundquist K. Association of preterm birth with risk of ischemic heart disease in adulthood. *JAMA Pediatr*. 2019;173:736–743. doi: 10.1001/jamapediatrics.2019.1327
53. Crump C, Sundquist K, Sundquist J, Winkleby MA. Gestational age at birth and mortality in young adulthood. *JAMA*. 2011;306:1233–1240. doi: 10.1001/jama.2011.1331

54. Huang C, Li J, Qin G, Liew Z, Hu J, László KD, Tao F, Obel C, Olsen J, Yu Y. Maternal hypertensive disorder of pregnancy and offspring early-onset cardiovascular disease in childhood, adolescence, and young adulthood: a national population-based cohort study. *PLoS Med*. 2021;18:e1003805. doi: 10.1371/journal.pmed.1003805
55. Kajantie E, Eriksson JG, Osmond C, Thornburg K, Barker DJ. Pre-eclampsia is associated with increased risk of stroke in the adult offspring: the Helsinki birth cohort study. *Stroke*. 2009;40:1176–1180. doi: 10.1161/STROKEAHA.108.538025
56. Yu Y, Arah OA, Liew Z, Cnattingius S, Olsen J, Sørensen HT, Qin G, Li J. Maternal diabetes during pregnancy and early onset of cardiovascular disease in offspring: population based cohort study with 40 years of follow-up. *BMJ*. 2019;367:l6398. doi: 10.1136/bmj.l6398
57. Bowers K, Ehrlich S, Dolan LM, Gupta R, Altaye M, Ollberding NJ, Szczesniak R, Catalano P, Smith E, Khoury JC. Elevated anthropometric and metabolic indicators among young adult offspring of mothers with pregestational diabetes: early results from the Transgenerational Effect on Adult Morbidity study (the TEAM study). *J Diabetes Res*. 2021;2021:6590431. doi: 10.1155/2021/6590431
58. Cox B, Luyten LJ, Dockx Y, Provost E, Madhloum N, De Boever P, Neven KY, Sassi F, Sleurs H, Vrijens K, et al. Association between maternal prepregnancy body mass index and anthropometric parameters, blood pressure, and retinal microvasculature in children age 4 to 6 years. *JAMA Netw Open*. 2020;3:e204662. doi: 10.1001/jamanetworkopen.2020.4662
59. Gaillard R, Welten M, Oddy WH, Beilin LJ, Mori TA, Jaddoe VW, Huang RC. Associations of maternal prepregnancy body mass index and gestational weight gain with cardio-metabolic risk factors in adolescent offspring: a prospective cohort study. *BJOG*. 2016;123:207–216. doi: 10.1111/1471-0528.13700
60. Mendelson MM, Lyass A, O'Donnell CJ, D'Agostino RB Sr, Levy D. Association of maternal prepregnancy dyslipidemia with adult offspring dyslipidemia in excess of anthropometric, lifestyle, and genetic factors in the Framingham Heart Study. *JAMA Cardiol*. 2016;1:26–35. doi: 10.1001/jamacardio.2015.0304
61. Perak AM, Lancki N, Kuang A, Labarthe DR, Allen NB, Shah SH, Lowe LP, Grobman WA, Lawrence JM, Lloyd-Jones DM, et al; HAPO Follow-Up Study Cooperative Research Group. Associations of maternal cardiovascular health in pregnancy with offspring cardiovascular health in early adolescence. *JAMA*. 2021;325:658–668. doi: 10.1001/jama.2021.0247
62. Catalano P, deMouzon SH. Maternal obesity and metabolic risk to the offspring: why lifestyle interventions may have not achieved the desired outcomes. *Int J Obes (Lond)*. 2015;39:642–649. doi: 10.1038/ijo.2015.15
63. Sinclair KD, Watkins AJ. Parental diet, pregnancy outcomes and offspring health: metabolic determinants in developing oocytes and embryos. *Reprod Fertil Dev*. 2013;26:99–114. doi: 10.1071/RD13290
64. Igosheva N, Abramov AY, Poston L, Eckert JJ, Fleming TP, Duchen MR, McConnell J. Maternal diet-induced obesity alters mitochondrial activity and redox status in mouse oocytes and zygotes. *PLoS One*. 2010;5:e10074. doi: 10.1371/journal.pone.0010074
65. Luzzo KM, Wang Q, Purcell SH, Chi M, Jimenez PT, Grindler N, Schedl T, Moley KH. High fat diet induced developmental defects in the mouse: oocyte meiotic aneuploidy and fetal growth retardation/brain defects. *PLoS One*. 2012;7:e49217. doi: 10.1371/journal.pone.0049217
66. Yang X, Wu LL, Chura LR, Liang X, Lane M, Norman RJ, Robker RL. Exposure to lipid-rich follicular fluid is associated with endoplasmic reticulum stress and impaired oocyte maturation in cumulus-oocyte complexes. *Fertil Steril*. 2012;97:1438–1443. doi: 10.1016/j.fertnstert.2012.02.034
67. Wyman A, Pinto AB, Sheridan R, Moley KH. One-cell zygote transfer from diabetic to nondiabetic mouse results in congenital malformations and growth retardation in offspring. *Endocrinology*. 2008;149:466–469. doi: 10.1210/en.2007-1273
68. Burton GJ, Redman CW, Roberts JM, Moffett A. Pre-eclampsia: pathophysiology and clinical implications. *BMJ*. 2019;366:l2381. doi: 10.1136/bmj.l2381
69. Huang L, Liu J, Feng L, Chen Y, Zhang J, Wang W. Maternal prepregnancy obesity is associated with higher risk of placental pathological lesions. *Placenta*. 2014;35:563–569. doi: 10.1016/j.placenta.2014.05.006
70. Huynh J, Dawson D, Roberts D, Bentley-Lewis R. A systematic review of placental pathology in maternal diabetes mellitus. *Placenta*. 2015;36:101–114. doi: 10.1016/j.placenta.2014.11.021
71. Leon-Garcia SM, Roeder HA, Nelson KK, Liao X, Pizzo DP, Laurent LC, Parast MM, LaCoursiere DY. Maternal obesity and sex-specific differences in placental pathology. *Placenta*. 2016;38:33–40. doi: 10.1016/j.placenta.2015.12.006
72. Wu FM, Quade BJ, Carreon CK, Schefter ZJ, Moses A, Lachtrupp CL, Markley JC, Gauvreau K, Valente AM, Economy KE; STORCC Investigators. Placental findings in pregnancies complicated by maternal cardiovascular disease. *JACC Advances*. 2022;1:1–11. doi: 10.1016/j.jacadv.2022.100008
73. Savvidou MD, Kaihura C, Anderson JM, Nicolaides KH. Maternal arterial stiffness in women who subsequently develop pre-eclampsia. *PLoS One*. 2011;6:e18703. doi: 10.1371/journal.pone.0018703
74. Lupton SJ, Chiu CL, Hodgson LA, Tooher J, Ogle R, Wong TY, Hennessy A, Lind JM. Changes in retinal microvascular caliber precede the clinical onset of preeclampsia. *Hypertension*. 2013;62:899–904. doi: 10.1161/HYPERTENSIONAHA.113.01890
75. Vasapollo B, Novelli GP, Gagliardi G, Farsetti D, Valensise H. Pregnancy complications in chronic hypertensive patients are linked to pre-pregnancy maternal cardiac function and structure. *Am J Obstet Gynecol*. 2020;223:425.e1–425.e13. doi: 10.1016/j.ajog.2020.02.043
76. Rich-Edwards JW, McElrath TF, Karumanchi SA, Seely EW. Breathing life into the lifecourse approach: pregnancy history and cardiovascular disease in women. *Hypertension*. 2010;56:331–334. doi: 10.1161/HYPERTENSIONAHA.110.156810
77. Hussein N, Kai J, Qureshi N. The effects of preconception interventions on improving reproductive health and pregnancy outcomes in primary care: a systematic review. *Eur J Gen Pract*. 2016;22:42–52. doi: 10.3109/13814788.2015.1099039
78. Poels M, van Stel HF, Franx A, Koster MPH. The effect of a local promotional campaign on preconceptional lifestyle changes and the use of preconception care. *Eur J Contracept Reprod Health Care*. 2018;23:38–44. doi: 10.1080/13625187.2018.1426744
79. Taylor RM, Wolfson JA, Lavelle F, Dean M, Frawley J, Hutchesson MJ, Collins CE, Shrewsbury VA. Impact of preconception, pregnancy, and postpartum culinary nutrition education interventions: a systematic review. *Nutr Rev*. 2021;79:1186–1203. doi: 10.1093/nutrit/nuaa124
80. Temel S, van Voorst SF, Jack BW, Denktaş S, Steegers EA. Evidence-based preconceptional lifestyle interventions. *Epidemiol Rev*. 2014;36:19–30. doi: 10.1093/epirev/mxt003
81. LeBlanc ES, Smith NX, Vesco KK, Paul IM, Stevens VJ. Weight loss prior to pregnancy and subsequent gestational weight gain: PREPARE, a randomized clinical trial. *Am J Obstet Gynecol*. 2021;224:99.e1–99.e14. doi: 10.1016/j.ajog.2020.07.027
82. Muirhead R, Kizirian N, Lal R, Black K, Prys-Davies A, Nassar N, Baur L, Sainsbury A, Sweeting A, Markovic T, et al. A pilot randomized controlled trial of a partial meal replacement preconception weight loss program for women with overweight and obesity. *Nutrients*. 2021;13:3200. doi: 10.3390/nu13093200
83. Jack B, Bickmore T, Hempstead M, Yinusa-Nyahkoon L, Sadikova E, Mitchell S, Gardiner P, Adigun F, Penti B, Schulman D, et al. Reducing preconception risks among African American women with conversational agent technology. *J Am Board Fam Med*. 2015;28:441–451. doi: 10.3122/jabfm.2015.04.140327
84. Jack BW, Bickmore T, Yinusa-Nyahkoon L, Reichert M, Julce C, Sidduri N, Martin-Howard J, Zhang Z, Woodhams E, Fernandez J, et al. Improving the health of young African American women in the preconception period using health information technology: a randomised controlled trial. *Lancet Digit Health*. 2020;2:e475–e485. doi: 10.1016/S2589-7500(20)30189-8
85. Kwong W, Tomlinson G, Feig DS. Maternal and neonatal outcomes after bariatric surgery; a systematic review and meta-analysis: do the benefits outweigh the risks? *Am J Obstet Gynecol*. 2018;218:573–580. doi: 10.1016/j.ajog.2018.02.003
86. Wekker V, Huvinen E, van Dammen L, Rono K, Painter RC, Zwinderman AH, van de Beek C, Sarkola T, Mol BWJ, Groen H, et al. Long-term effects of a preconception lifestyle intervention on cardiometabolic health of overweight and obese women. *Eur J Public Health*. 2019;29:308–314. doi: 10.1093/eurpub/cky222
87. den Harink T, Blom NA, Gemke RJ, Groen H, Hoek A, Mol BW, Painter RC, Kuipers IM, Roseboom TJ, van Deutekom AW. Preconception lifestyle intervention in women with obesity and echocardiographic indices of cardiovascular health in their children. *Int J Obes (Lond)*. 2022;46:1262–1270. doi: 10.1038/s41366-022-01107-1
88. Raab R, Michel S, Günther J, Hoffmann J, Stecher L, Hauner H. Associations between lifestyle interventions during pregnancy and childhood weight and growth: a systematic review and meta-analysis. *Int J Behav Nutr Phys Act*. 2021;18:8. doi: 10.1186/s12966-020-01075-7
89. Dodd JM, Deussen AR, O'Brien CM, Schoenaker DAJM, Poprzeczny A, Gordon A, Phelan S. Targeting the postpartum period to promote weight

loss: a systematic review and meta-analysis. *Nutr Rev*. 2018;76:639–654. doi: 10.1093/nutrit/nuy024
90. Rich-Edwards JW, Stuart JJ, Skurnik G, Roche AT, Tsigas E, Fitzmaurice GM, Wilkins-Haug LE, Levkoff SE, Seely EW. Randomized trial to reduce cardiovascular risk in women with recent preeclampsia. *J Womens Health (Larchmt)*. 2019;28:1493–1504. doi: 10.1089/jwh.2018.7523
91. Lewey J, Murphy S, Zhang D, Putt ME, Elovitz MA, Riis V, Patel MS, Levine LD. Effectiveness of a text-based gamification intervention to improve physical activity among postpartum individuals with hypertensive disorders of pregnancy: a randomized clinical trial. *JAMA Cardiol*. 2022;7:591–599. doi: 10.1001/jamacardio.2022.0553
92. Christiansen PK, Skjøth MM, Rothmann MJ, Vinter CA, Lamont RF, Draborg E. Lifestyle interventions to maternal weight loss after birth: a systematic review. *Syst Rev*. 2019;8:327. doi: 10.1186/s13643-019-1186-2
93. Jowell AR, Sarma AA, Gulati M, Michos ED, Vaught AJ, Natarajan P, Powe CE, Honigberg MC. Interventions to mitigate risk of cardiovascular disease after adverse pregnancy outcomes: a review. *JAMA Cardiol*. 2022;7:346–355. doi: 10.1001/jamacardio.2021.4391
94. Upadhya KK, Psoter KJ, Connor KA, Mistry KB, Levy DJ, Cheng TL. Cluster randomized trial of a pre/interconception health intervention for mothers in pediatric visits. *Acad Pediatr*. 2020;20:660–669. doi: 10.1016/j.acap.2019.10.003
95. Celi AC, Seely EW, Wang P, Thomas AM, Wilkins-Haug LE. Caring for women after hypertensive pregnancies and beyond: implementation and integration of a postpartum transition clinic. *Matern Child Health J*. 2019;23:1459–1466. doi: 10.1007/s10995-019-02768-7
96. ACOG Committee Opinion No. 762: prepregnancy counseling. *Obstet Gynecol*. 2019;133:e78–e89. doi: 10.1097/AOG.0000000000003013
97. Gooding HC, Ning H, Perak AM, Allen N, Lloyd-Jones D, Moore LL, Singer MR, de Ferranti SD. Cardiovascular health decline in adolescent girls in the NGHS cohort, 1987-1997. *Prev Med Rep*. 2020;20:101276. doi: 10.1016/j.pmedr.2020.101276
98. US Food and Drug Administration. FDA requests removal of strongest warning against using cholesterol-lowering statins during pregnancy; still advises most pregnant patients should stop taking statins. July 2021. Accessed June 1, 2022. https://www.fda.gov/drugs/drug-safety-and-availability/fda-requests-removal-strongest-warning-against-using-cholesterol-lowering-statins-during-pregnancy
99. Mauricio R, Khera A. Statin use in pregnancy: is it time for a paradigm shift? *Circulation*. 2022;145:496–498. doi: 10.1161/CIRCULATIONAHA.121.058983
100. Chinn JJ, Martin IK, Redmond N. Health equity among Black women in the United States. *J Womens Health (Larchmt)*. 2021;30:212–219. doi: 10.1089/jwh.2020.8868
101. Levine GN, Cohen BE, Commodore-Mensah Y, Fleury J, Huffman JC, Khalid U, Labarthe DR, Lavretsky H, Michos ED, Spatz ES, et al; on behalf of the American Heart Association Council on Clinical Cardiology; Council on Arteriosclerosis, Thrombosis and Vascular Biology; Council on Cardiovascular and Stroke Nursing; and Council on Lifestyle and Cardiometabolic Health. Psychological health, well-being, and the mind-heart-body connection: a scientific statement from the American Heart Association. *Circulation*. 2021;143:e763–e783. doi: 10.1161/CIR.0000000000000947
102. Barajas CB, Jones SCT, Milam AJ, Thorpe RJ Jr, Gaskin DJ, LaVeist TA, Furr-Holden CDM. Coping, discrimination, and physical health conditions among predominantly poor, urban African Americans: implications for community-level health services. *J Community Health*. 2019;44:954–962. doi: 10.1007/s10900-019-00650-9
103. Jones NL, Gilman SE, Cheng TL, Drury SS, Hill CV, Geronimus AT. Life course approaches to the causes of health disparities. *Am J Public Health*. 2019;109(suppl 1):S48–S55. doi: 10.2105/AJPH.2018.304738
104. Rodriquez EJ, Kim EN, Sumner AE, Nápoles AM, Pérez-Stable EJ. Allostatic load: importance, markers, and score determination in minority and disparity populations. *J Urban Health*. 2019;96(suppl 1):3–11. doi: 10.1007/s11524-019-00345-5
105. Bond RM, Ansong A, Albert MA. Shining a light on the superwoman schema and maternal health. *Circulation*. 2022;145:507–509. doi: 10.1161/CIRCULATIONAHA.121.058905
106. Allen AM, Wang Y, Chae DH, Price MM, Powell W, Steed TC, Rose Black A, Dhabhar FS, Marquez-Magaña L, Woods-Giscombe CL. Racial discrimination, the superwoman schema, and allostatic load: exploring an integrative stress-coping model among African American women. *Ann N Y Acad Sci*. 2019;1457:104–127. doi: 10.1111/nyas.14188
107. Bond RM, Gaither K, Nasser SA, Albert MA, Ferdinand KC, Njoroge JN, Parapid B, Hayes SN, Pegus C, Sogade B, et al; Association of Black Cardiologists. Working agenda for Black mothers: a position paper from the Association of Black Cardiologists on solutions to improving black maternal health. *Circ Cardiovasc Qual Outcomes*. 2021;14:e007643. doi: 10.1161/CIRCOUTCOMES.120.007643
108. Biggers A, Spears CA, Sanders K, Ong J, Sharp LK, Gerber BS. Promoting Mindfulness in African American Communities. *Mindfulness (N Y)*. 2020;11:2274–2282. doi: 10.1007/s12671-020-01480-w
109. Burnett-Zeigler IE, Satyshur MD, Hong S, Yang A, Moskowitz JT, Wisner KL. Mindfulness based stress reduction adapted for depressed disadvantaged women in an urban federally qualified health center. *Complement Ther Clin Pract*. 2016;25:59–67. doi: 10.1016/j.ctcp.2016.08.007
110. Lara-Cinisomo S, Clark CT, Wood J. Increasing diagnosis and treatment of perinatal depression in Latinas and African American women: addressing stigma is not enough. *Womens Health Issues*. 2018;28:201–204. doi: 10.1016/j.whi.2018.01.003
111. Watson-Singleton NN, Black AR, Spivey BN. Recommendations for a culturally-responsive mindfulness-based intervention for African Americans. *Complement Ther Clin Pract*. 2019;34:132–138. doi: 10.1016/j.ctcp.2018.11.013
112. Van Den Heuvel JF, Groenhof TK, Veerbeek JH, Van Solinge WW, Lely AT, Franx A, Bekker MN. eHealth as the next-generation perinatal care: an overview of the literature. *J Med Internet Res*. 2018;20:e9262. doi: 10.2196/jmir.9262
113. Brewer LC, Hayes SN, Jenkins SM, Lackore KA, Breitkopf CR, Cooper LA, Patten CA. Improving cardiovascular health among African-Americans through mobile health: the FAITH! app pilot study. *J Gen Intern Med*. 2019;34:1376–1378. doi: 10.1007/s11606-019-04936-5
114. Howell EA. Reducing disparities in severe maternal morbidity and mortality. *Clin Obstet Gynecol*. 2018;61:387–399. doi: 10.1097/GRF.0000000000000349
115. Ebong I, Breathett K. The cardiovascular disease epidemic in African American women: recognizing and tackling a persistent problem. *J Womens Health (Larchmt)*. 2020;29:891–893. doi: 10.1089/jwh.2019.8125
116. White BM, Rochell JK, Warren JR. Promoting cardiovascular health for African American women: an integrative review of interventions. *J Womens Health (Larchmt)*. 2020;29:952–970. doi: 10.1089/jwh.2018.7580
117. Bermúdez-Millán A, Damio G, Cruz J, D'Angelo K, Segura-Pérez S, Hromi-Fiedler A, Pérez-Escamilla R. Stress and the social determinants of maternal health among Puerto Rican women: a CBPR approach. *J Health Care Poor Underserved*. 2011;22:1315–1330. doi: 10.1353/hpu.2011.0108
118. Resnicow K, Braithwaite RL, Dilorio C, Glanz K. Applying theory to culturally diverse and unique populations. In: *Health Behavior and Health Education: Theory, Research, and Practice*. Jossey-Bass; 2002:485–509.
119. Joseph RP, Keller C, Affuso O, Ainsworth BE. Designing culturally relevant physical activity programs for African-American women: a framework for intervention development. *J Racial Ethn Health Disparities*. 2017;4:397–409. doi: 10.1007/s40615-016-0240-1
120. Brewer LC, Hayes SN, Parker MW, Balls-Berry JE, Halyard MY, Pinn VW, Radecki Breitkopf C. African American women's perceptions and attitudes regarding participation in medical research: the Mayo Clinic/The Links, Incorporated partnership. *J Womens Health (Larchmt)*. 2014;23:681–687. doi: 10.1089/jwh.2014.4751
121. Brown Speights JS, Nowakowski ACH, De Leon J, Mitchell MM, Simpson I. Engaging African American women in research: an approach to eliminate health disparities in the African American community. *Fam Pract*. 2017;34:322–329. doi: 10.1093/fampra/cmx026
122. Cooper LA, Purnell TS, Ibe CA, Halbert JP, Bone LR, Carson KA, Hickman D, Simmons M, Vachon A, Robb I, et al. Reaching for health equity and social justice in Baltimore: the evolution of an academic-community partnership and conceptual framework to address hypertension disparities. *Ethn Dis*. 2016;26:369–378. doi: 10.18865/ed.26.3.369
123. Manjunath C, Ifelayo O, Jones C, Washington M, Shanedling S, Williams J, Patten CA, Cooper LA, Brewer LC. Addressing cardiovascular health disparities in Minnesota: establishment of a community steering committee by FAITH! (Fostering African-American Improvement in Total Health). *Int J Environ Res Public Health*. 2019;16:4144. doi: 10.3390/ijerph16214144
124. Brown AG, Hudson LB, Chui K, Metayer N, Lebron-Torres N, Seguin RA, Folta SC. Improving heart health among Black/African American women using civic engagement: a pilot study. *BMC Public Health*. 2017;17:112. doi: 10.1186/s12889-016-3964-2
125. Rodriguez F, Christopher L, Johnson CE, Wang Y, Foody JM. Love Your Heart: a pilot community-based intervention to improve the cardiovascular health of African American women. *Ethn Dis*. 2012;22:416–421.

126. Estrella ML, Kelley MA, Durazo-Arvizu RA, Gallo LC, Chambers EC, Perreira KM, Zeng D, Giachello AL, Isasi CR, Wu D, et al. Volunteerism and cardiovascular health: the HCHS/SOL Sociocultural Ancillary Study. *Health Behav Policy Rev*. 2020;7:120–135. doi: 10.14485/HBPR.7.2.5
127. D'Alonzo KT, Smith BA, Dicker LH. Outcomes of a culturally tailored partially randomized patient preference controlled trial to increase physical activity among low-income immigrant Latinas. *J Transcult Nurs*. 2018;29:335–345. doi: 10.1177/1043659617723073
128. Gutiérrez Á, Young MT, Dueñas M, García A, Márquez G, Chávez ME, Ramírez S, Rico S, Bravo RL. Laboring with the heart: promotoras' transformations, professional challenges, and relationships with communities. *Fam Community Health*. 2021;44:162–170. doi: 10.1097/FCH.0000000000000286
129. Koniak-Griffin D, Brecht ML, Takayanagi S, Villegas J, Melendrez M, Balcázar H. A community health worker-led lifestyle behavior intervention for Latina (Hispanic) women: feasibility and outcomes of a randomized controlled trial. *Int J Nurs Stud*. 2015;52:75–87. doi: 10.1016/j.ijnurstu.2014.09.005
130. White BM. Barriers and facilitators to adhering to the American Heart Association's Life's Simple 7 for African American women living in public housing. *J Health Care Poor Underserved*. 2021;32:2012–2029. doi: 10.1353/hpu.2021.0179
131. Churchwell K, Elkind MSV, Benjamin RM, Carson AP, Chang EK, Lawrence W, Mills A, Odom TM, Rodriguez CJ, Rodriguez F, et al; on behalf of the American Heart Association. Call to action: structural racism as a fundamental driver of health disparities: a presidential advisory from the American Heart Association. *Circulation*. 2020;142:e454–e468. doi: 10.1161/CIR.0000000000000936
132. Sims M, Kershaw KN, Breathett K, Jackson EA, Lewis LM, Mujahid MS, Suglia SF; on behalf of the American Heart Association Council on Epidemiology and Prevention and Council on Quality of Care and Outcomes Research. Importance of housing and cardiovascular health and well-being: a scientific statement from the American Heart Association. *Circ Cardiovasc Qual Outcomes*. 2020;13:e000089. doi: 10.1161/HCQ.0000000000000089
133. Mannoh I, Hussien M, Commodore-Mensah Y, Michos ED. Impact of social determinants of health on cardiovascular disease prevention. *Curr Opin Cardiol*. 2021;36:572–579. doi: 10.1097/HCO.0000000000000893
134. Lindley KJ, Aggarwal NR, Briller JE, Davis MB, Douglass P, Epps KC, Fleg JL, Hayes S, Itchhaporia D, Mahmoud Z, et al; American College of Cardiology Cardiovascular Disease in Women Committee and the American College of Cardiology Health Equity Taskforce. Socioeconomic determinants of health and cardiovascular outcomes in women: *JACC* review topic of the week. *J Am Coll Cardiol*. 2021;78:1919–1929. doi: 10.1016/j.jacc.2021.09.011
135. Barker M, Dombrowski SU, Colbourn T, Fall CHD, Kriznik NM, Lawrence WT, Norris SA, Ngaiza G, Patel D, Skordis-Worrall J, et al. Intervention strategies to improve nutrition and health behaviours before conception. *Lancet*. 2018;391:1853–1864. doi: 10.1016/S0140-6736(18)30313-1
136. George AS, Amin A, de Abreu Lopes CM, Ravindran TS. Structural determinants of gender inequality: why they matter for adolescent girls' sexual and reproductive health. *BMJ*. 2020;368:l6985. doi: 10.1136/bmj.l6985
137. O'Neil A, Thompson K, Russell JD, Norton R. Inequalities and deteriorations in cardiovascular health in premenopausal US women, 1990-2016. *Am J Public Health*. 2020;110:1175–1181. doi: 10.2105/AJPH.2020.305702
138. Daw JR, Sommers BD. The Affordable Care Act and access to care for reproductive-aged and pregnant women in the United States, 2010-2016. *Am J Public Health*. 2019;109:565–571. doi: 10.2105/AJPH.2018.304928
139. Daw JR, Hatfield LA, Swartz K, Sommers BD. Women in the United States experience high rates of coverage "churn" in months before and after childbirth. *Health Aff (Millwood)*. 2017;36:598–606. doi: 10.1377/hlthaff.2016.1241
140. Finer LB, Zolna MR. Declines in unintended pregnancy in the United States, 2008–2011. *N Engl J Med*. 2016;374:843–852. doi: 10.1056/NEJMsa1506575
141. Allen D, Hunter MS, Wood S, Beeson T. One Key Question®: first things first in reproductive health. *Matern Child Health J*. 2017;21:387–392. doi: 10.1007/s10995-017-2283-2