HHS Public Access

Author manuscript

Circulation. Author manuscript; available in PMC 2024 June 24.

Published in final edited form as:

Circulation. 2023 May 09; 147(19): 1471–1487. doi:10.1161/CIR.0000000000001139.

Cardiovascular Disease Risk Factors in Women: The Impact of Race and Ethnicity: A Scientific Statement From the American Heart Association

Laxmi S. Mehta, MD, FAHA [Chair],
The Ohio State University

Gladys P. Velarde, MD, FAHA,
University of Florida–Jacksonville

Jennifer Lewey, MD, MPH,
University of Pennsylvania, Perelman School of Medicine Perelman Center for Advanced Medicine

Garima Sharma, MD,
Ciccarone Center for the Prevention of Cardiovascular Disease, Johns Hopkins University School of Medicine

Rachel M. Bond, MD,
Chandler Regional Medical Center, Dignity Health

Ana Navas-Acien, MD, PhD,
Columbia University

Amanda M. Fretts, MPH, PhD,
University of Washington

Gayenell S. Magwood, PhD, RN, FAHA,
University of South Carolina

Eugene Yang, MD,
University of Washington Medical Center

Roger S. Blumenthal, MD, FAHA,
Johns Hopkins University

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.

This statement was approved by the American Heart Association Science Advisory and Coordinating Committee on January 26, 2023, and the American Heart Association Executive Committee on February 22, 2023. A copy of the document is available at https://professional.heart.org/statements by using either "Search for Guidelines & Statements" or the "Browse by Topic" area. To purchase additional reprints, call 215–356-2721 or email Meredith.Edelman@wolterskluwer.com.

Rachel-Maria Brown, MD,
Northwell Health

Jennifer H. Mieres, MD, FAHA [Vice Chair]
Northwell Health

American Heart Association Cardiovascular Disease and Stroke in Women and Underrepresented Populations Committee of the Council on Clinical Cardiology; Council on Cardiovascular and Stroke Nursing; Council on Hypertension; Council on Lifelong Congenital Heart Disease and Heart Health in the Young; Council on Lifestyle and Cardiometabolic Health; Council on Peripheral Vascular Disease; and Stroke Council

Abstract

Cardiovascular disease is the leading cause of death in women, yet differences exist among certain racial and ethnic groups. Aside from traditional risk factors, behavioral and environmental factors and social determinants of health affect cardiovascular health and risk in women. Language barriers, discrimination, acculturation, and health care access disproportionately affect women of underrepresented races and ethnicities. These factors result in a higher prevalence of cardiovascular disease and significant challenges in the diagnosis and treatment of cardiovascular conditions. Culturally sensitive, peer-led community and health care professional education is a necessary step in the prevention of cardiovascular disease. Equitable access to evidence-based cardiovascular preventive health care should be available for all women regardless of race and ethnicity; however, these guidelines are not equally incorporated into clinical practice. This scientific statement reviews the current evidence on racial and ethnic differences in cardiovascular risk factors and current cardiovascular preventive therapies for women in the United States.

Keywords

AHA Scientific Statements; cardiovascular diseases; ethnicity; primary prevention; racial groups; risk factors; women

Cardiovascular disease (CVD) remains the number one cause of death for women in the United States.[1] The focused research on women at risk for CVD has clarified our understanding of some of the sex-specific factors that are important in the detection of atherosclerosis and reduction in death. The 2010 landmark Institute of Medicine report, *Women's Health Research: Progress, Pitfalls and Promise*, underscored the fact that although major progress was made in reducing cardiovascular death, disparities existed in disease burden among subgroups of women, particularly among those who were socially disenfranchised because of race, ethnicity, educational attainment, and income level.[2] Women who are at high risk for the development of CVD and its associated risk factors face even greater risk when they are members of certain racial and ethnic groups. Expanding the cardiovascular risk assessment to include the social determinants of health (SDOH) and nonbiological variables that affect cardiovascular risk and outcomes is essential for achieving the American Heart Association's (AHA's) 2024 Impact Goal of advancing cardiovascular health for all by identifying and removing barriers to health care access and quality.[3]

Racial, ethnic, and sex disparities in cardiovascular care are well documented. This scientific statement aims to highlight the need for an expanded approach to risk factors and primary prevention strategies for CVD for women of underrepresented races and ethnicities who are particularly vulnerable to health disparities. It is important to acknowledge that there are gaps in current knowledge and data specific to each race and ethnicity, all of which warrant further investigation and are beyond the scope of this document.

METHODS

The writing group members were nominated by the AHA Manuscript Oversight Committee and have a broad range of expertise on CVD in women and the relationship to race and ethnicity. A general framework outlined by the committee chair was used to conduct a comprehensive literature review to summarize existing evidence and to indicate gaps in current knowledge. Only English-language studies were reviewed, with PubMed/MEDLINE as our primary resource, as well as the Cochrane Library Reviews, Centers for Disease Control and Prevention, and US Census data as secondary resources.

CARDIOVASCULAR RISK FACTORS

Traditional risk factors of CVD are well defined: diabetes, hypertension, dyslipidemia, family history of heart disease, smoking, physical inactivity, poor diet, and obesity.

Clinical risk stratification tools and risk scores have been developed for stratifying risk and tailoring preventive intervention. The Pooled Cohort Risk equations were recommended in the American College of Cardiology/AHA cholesterol and hypertension guidelines, and although they include sex and Black or White race status, limitations remain in risk calibration for other racial and ethnic groups.[4,5] Given the limited race and ethnicity data available for women living in the United States, we focus our discussion on the traditional CVD risk factors of hypertension, dyslipidemia, diabetes, obesity, and tobacco use among non-Hispanic Black, Hispanic/Latina, American Indian/Alaska Native, and Asian women, as well as the current limitations of using traditional risk factor screening models in these populations.

Limitations of Traditional Risk Factors and Consideration of Nontraditional Risk Factors

There are many limitations to traditional risk factors and their ability to comprehensively estimate a person’s risk for CVD. For example, body mass index (BMI) is often used as a means to identify obesity; however, it is not an ideal metric in people of underrepresented races and ethnicities such as Asian individuals.[6] To date, risk assessment models do not take into account sex-specific biological risk factors: pregnancy-related factors (preeclampsia, gestational diabetes or hypertension, miscarriages, preterm delivery), menstrual cycle history (age at start of menarche and menopause), medications (contraception, hormone therapy), and medical conditions (polycystic ovarian syndrome, autoimmune disorders, and history of certain chemotherapy or radiation therapy).

Mental health conditions are an additional nontraditional risk factor that have a significant effect on cardiovascular health. Women have anxiety and depression disproportionately to

men and are nearly twice as likely as men to be diagnosed with depression. Depressed mood can occur as a result of normal hormonal changes associated with puberty, menstruation, pregnancy, perimenopause, and menopause. In addition, environmental and cultural stressors are associated with the higher incidence of depression in women. Psychosocial stressors such as discrimination, poor social support, domestic violence, primary elder and child caregiving roles, and unequal power dynamics in the workplace play a significant role and may increase the risk of depression in women.[7–14] Incorporating screening for mental health conditions such as depression and posttraumatic stress disorder, which are more common in women and are associated with a higher risk of developing cardiometabolic risk factors and incident CVD, may better serve women and provide a more comprehensive assessment.[15]

In addition, SDOH, which include economic stability, neighborhood safety, education, social and community context, and access to quality health care, have a significant role in the development of CVD and vary across racial and ethnic groups.[16] This impact is recognized in the recent AHA scientific statement that has redefined overall cardiovascular health metrics from Life's Simple 7 to now Life's Essential 8.[17] This includes metrics pertaining to health behaviors (diet; physical activity; nicotine exposure, including vaping and exposure to secondhand smoke; and the new inclusion of sleep hygiene) and health factors (BMI, blood lipids, blood glucose, blood pressure), all of which may vary according to racial and ethnic cultural norms and beliefs.[16,17] Exposure to poor working conditions, poor living conditions, environmental hazards, reduced access to medical care, and other social determinants varies across racial and ethnic groups. Detailed information on the cardiovascular health of non-Hispanic Black, Hispanic/Latina, American Indian/Alaska Native, and Asian women and SDOH can be found in AHA scientific statements focused on these people of underrepresented races and ethnicities.[16,18–23] However, the influential SDOH have not yet been incorporated into traditional risk factor assessment tools.

CVD IN WOMEN ACROSS RACE AND ETHNICITY

This scientific statement uses the contemporary views of race and ethnicity not as biological variables but mainly as social, cultural, environmental, systemic, and demographic constructs that are invaluable in the assessment of access to health care, quality of care, and strategies to improve cardiovascular outcomes and to advance cardiovascular equity.[24–26]

Black Women

CVD is the leading cause of death for non-Hispanic Black women.[1] Black is used as an umbrella term for a heterogeneous population that often encompasses African American, African (East, West, South), and Afro Caribbean.[27] Nearly 60% of non-Hispanic Black women ≥20 years of age have CVD (coronary heart disease [CHD], heart failure, cerebrovascular accident [CVA], and hypertension)[1]; included within that are CVAs that disproportionately affect non-Hispanic Black women, making CVD prevalence rates the highest among this group.[18] Non-Hispanic Black women account for the greatest number of deaths, with 22.9% of all deaths resulting from heart disease (rheumatic heart disease, hypertensive heart disease, ischemic heart disease, endocarditis, heart failure, other myocardial/pericardial diseases) compared with 22.0% in non-Hispanic White women.[28]

As a result, they have a persistent life expectancy gap, with non-Hispanic Black women living 75 years on average compared with 80 years for non-Hispanic White women.[29] A key contributor to the disparities in care is that non-Hispanic Black women are disproportionately affected in the prevalence and magnitude of effect of traditional risk factors, driven most prominently by SDOH, which leads to this higher prevalence and earlier age at onset of CVD.[1]

Hispanic/Latina Women

CVD is the leading cause of death among Hispanic/Latina adults, which refers to individuals of any racial and ethnic background whose ancestry is from Mexico, Central America, South America, the Caribbean, or other Spanish-speaking countries.[19] In 2017, nearly 43% of Hispanic/Latina women had some form of CVD (CHD, heart failure, CVA, hypertension).[1] Paradoxically, despite a higher prevalence of diabetes, obesity, and metabolic syndrome, particularly in women, CVD death rates have remained 15% to 20% lower than in non-Hispanic White women.[1,30] This poorly characterized epidemiological phenomenon (the Hispanic paradox) may not apply equally to all subgroups, both sexes, and all types of CVD. It is possible that paradoxes in data may be secondary to the underappreciation of the diverse composition of the Hispanic/Latino population, which, in the United States, includes a mixture of races, ethnicities, and socioeconomic and demographic backgrounds. Unfortunately, research has been sparse, consisting mostly of cross-sectional studies focusing on Mexican American individuals or grouping diverse Hispanic/Latino subjects together. This approach to the study of the Hispanic/Latino population has contributed to epidemiological generalizations that do not take into account the possible heterogeneity in CVD risk profiles and may misrepresent their outcomes.[19]

American Indian and Alaska Native Women

American Indian and Alaska Native people are a heterogeneous population that includes 574 federally recognized tribes (and many tribes not federally recognized) and make up ≈2% of the US population.[20] CVD (heart disease and CVA) is the leading cause of death in American Indian/Alaska Native women,[31,32] and more than one-third of CVD-related deaths occur among American Indian/Alaska Native men and women <65 years of age.[20] Among American Indian/Alaska Native women, there is substantial variation in CVD-related death rates by geographic region, with the highest rates observed in the Southern and Northern Plains.[31,33] Most CVD-related deaths are a consequence of underlying CHD.[31,34]

In the SHS (Strong Heart Study), the largest longitudinal study of CVD among American Indian adults,[35] the prevalence of CHD among American Indian/Alaska Native women without and with diabetes was 17.8% and 26.5%, respectively.[36] The prevalence of modifiable risk factors in American Indian/Alaska Native women is high, with almost 50% of American Indian/Alaska Native adult women having at least 2 cardiovascular risk factors.[37] Unfortunately, understanding the cardiovascular health of American Indian/Alaska Native people remains challenging because many available national data sets underreport the burden of CVD and related deaths in American Indian/Alaska Native people as a result of small sample sizes, racial misclassification, or both.[38–40]

Asian Women

CVD is the leading cause of death for Asian women in the United States.[1] According to the Office of Management and Budget, federal Asian race and ethnicity classification is defined as having origins in the Far East, Southeast Asia, or the Indian subcontinent, including countries such as Cambodia, China, India, Japan, Korea, Malaysia, Philippines, Thailand, and Vietnam.[27] The prevalence of CVD is high in non-Hispanic Asian women (45%); however, there is considerable heterogeneity in both CVD risk factors and outcomes.[1] A report from US national mortality records of the 6 largest Asian subgroups (Asian Indian, Chinese, Filipino, Japanese, Korean, Vietnamese) from 2003 to 2011 revealed significant differences in causespecific death, with the highest CVD death rates among Asian Indian women. Although Asian Indian and Filapina women have the highest age-adjusted CVD death rates compared with other Asian subgroups, the rates are still significantly lower than for non-Hispanic White women.[41] Most studies that have examined traditional CVD risk factors in Asian American individuals have found associations similar to those reported for non-Hispanic White people; however, the prevalence varies among Asian subgroups.[21]

CVD RISK FACTORS ACROSS RACE AND ETHNICITY

Hypertension

Non-Hispanic Black women have among the highest prevalence of hypertension in the world at 55.3%.[1] Non-Hispanic Black women are less likely to have controlled blood pressure compared with non-Hispanic White women and are more likely to have controlled blood pressure compared with Mexican American women. As with other risk factors, there is a lack of consistent data on the prevalence of hypertension among Hispanic/Latina subgroups. According to AHA statistics, 40.8% of Hispanic/Latina women have hypertension.[1] Generally, Mexican American women have a significantly lower prevalence of hypertension compared with all other subgroups, whereas the prevalence is higher in Dominican, Puerto Rican, and Cuban individuals.[42] Hispanic/Latina Black women have a higher prevalence of hypertension than Hispanic/Latina White women.[42] The National Heart, Lung, and Blood Institute–sponsored Hispanic Community Health Study (HCHS)/SOL (Study of Latinos) showed that the prevalence of hypertension was higher among Puerto Rican women and lowest in South American women.[42,43]

Hypertension is a significant risk factor for CHD in American Indian/Alaska Native people, particularly among individuals with diabetes.[44,45] There are regional differences in the prevalence of hypertension in American Indian/Alaska Native women, with estimates ranging from 25% to 41%.[46–49] In a study of the 6 largest Asian American subgroups, a greater proportion of death from hypertensive disease was observed in all subgroups compared with non-Hispanic White women.[50] A New York City Health and Nutrition Examination Survey found substantially higher hypertension rates for Asian (38.0%) and Hispanic (33.0%) adults compared with non-Hispanic White adults (27.5%); however, subcategorization by sex was not reported. In addition, East/Southeast Asian adults had much higher odds of hypertension compared with non-Hispanic White adults (adjusted odds ratio, 2.8 [95% CI, 1.6–4.9]).[51] A Northern California study also found heterogeneity in age-adjusted hypertension rates from 30.0% in Chinese to 53.2% in Filipina women. Except

for Filipina, Mexican, and non-Hispanic Black women, most women of underrepresented race and ethnicity subgroups had lower or similar rates of hypertension compared with non-Hispanic White women.[52]

Dyslipidemia

The prevalence of elevated total cholesterol (>200 mg/dL) and low-density lipoprotein (LDL; >130 mg/dL) cholesterol levels is lower among non-Hispanic Black women compared with women of other races and ethnicities (33.4% and 24.3%, respectively).[1,53] Lipoprotein(a) (Lp[a]), which is an LDL-like particle with an additional apolipoprotein a attached to it, is highest among people of non-Hispanic Black ancestry, followed by those of South Asian, non-Hispanic White, Hispanic/Latino, and East Asian ancestry.[54] In the ARIC study (Atherosclerosis Risk in Communities), plasma Lp(a) was found to be positively associated with CVD events in Black and White adults; furthermore, high Lp(a) levels in non-Hispanic Black and non-Hispanic White women were found to be associated with a higher incidence of ischemic strokes.[55,56]

Dyslipidemia is highly prevalent among Hispanic/Latina women in the United States: 37.3% with total cholesterol levels ≥200 mg/dL, 26.3% with LDL ≥130 mg/dL, and 12.3% with high-density lipoprotein <40 mg/dL. Among adult American women, the mean triglycerides level is 86.8 mg/dL, whereas non-Hispanic Black and non-Hispanic Asian women have lower mean triglyceride levels, and levels are slightly higher (88.3 mg/dL) among non-Hispanic White women and highest (97.1 mg/dL) among Hispanic/Latina women.[1] Although NHANES (National Health and Nutrition Examination Survey) data demonstrated higher prevalence rates of hypercholesterolemia in Mexican American women,[1] HCHS/SOL showed that Puerto Rican women had the highest prevalence of hypercholesterolemia, followed by Cuban and Central American women. Female sex was one of the factors associated with dyslipidemia in HCHS/SOL.[57]

About 1 in 5 American Indian/Alaska Native women has been diagnosed with hyperlipidemia.[1] The SHS showed that the effect of LDL cholesterol on CHD risk was highest in those with diabetes[45]; thus, optimal management of lipids and glucose is critical to maximize cardiovascular health.[58] To date, the majority of lipid studies for Asian individuals have been conducted in their country of origin, with Asian Indian and Filipino people having a higher prevalence of low high-density lipoprotein cholesterol and high triglycerides.[59–61] One study from Northern California explored the variability of lipid profiles among Asian subgroups and found that Asian Indian and Chinese women had greater odds of low high-density lipoprotein cholesterol than non-Hispanic White women. In addition, compared with non-Hispanic White women, Chinese, Filipina, and Japanese women had a greater likelihood of high triglycerides.[61]

Furthermore, Lp(a) is an independent risk factor for CVD and is higher in Asian Indian individuals compared with non-Hispanic White individuals.[62] An analysis from INTERHEART found an association between high Lp(a) and increased CVD risk among Chinese, South Asian, and Southeast Asian adults; however, sex-specific data were not reported. The population-attributable risk of high Lp(a) and myocardial infarction was highest among South Asian adults (9.5%).[62]

Diabetes

Type 2 diabetes accounts for >95% of diabetes in the United States, and non-Hispanic Black women are more likely to develop it.[18] The prevalence of diagnosed diabetes among non-Hispanic Black US women ≥20 years of age is 13.2%.[1] Less is known about the prevalence of gestational diabetes. However, in a nationally representative sample of US women, the prevalence of gestational diabetes was higher in non-Hispanic Black women at 7.4% compared with 6.7% in non-Hispanic White women.[63]

Among Hispanic/Latina adults in NHANES, 14.1% of women had diagnosed diabetes compared with 7.3% of non-Hispanic White women.[64] The prevalence of diabetes differs among individuals with different Hispanic/Latina backgrounds, with a higher incidence seen in Mexican American and Puerto Rican women compared with other Hispanic/Latina women subgroups. In HCHS/SOL, 17% of women had diabetes. The prevalence was lowest in South American women (10.2%) and highest in Puerto Rican women (19.4%), followed by Mexican American women (18.5%), who were less likely to meet hemoglobin A1c and LDL cholesterol goals.[65]

American Indian women have a high prevalence (19%) of diagnosed diabetes, and diabetes is the primary risk factor for CHD in American Indian women.[34,38,66] However, the prevalence of diabetes varies by region; among SHS participants, the prevalence of diabetes was 72% among American Indian women in Arizona and 40% to 42% among American Indian women from Oklahoma, North Dakota, and South Dakota.[67] Although most studies report low levels of diabetes (≈5%–8%) in Alaska Native women,[48,68,69] data from the population-based Alaska Native Diabetes Registry demonstrate a recent increasing trend in the prevalence and incidence of diabetes for Alaska Native women.[46,70]

Type 2 diabetes is highly prevalent among Asian Indian and Filipina women, and several studies have found that Asian Indian people have higher rates of insulin resistance, whereas Filipino people have a higher prevalence of metabolic syndrome.[61,71,72] Although the associations between BMI and diabetes are strong across racial groups, the BMI cut point for increased risk of type 2 diabetes is lower for Asian people than for other racial groups, including non-Hispanic White and non-Hispanic Black people.[73]

From 2011 to 2016, the age- and sex-adjusted prevalence of undiagnosed diabetes and total diabetes was highest among non-Hispanic Asian groups compared with non-Hispanic White people. In addition, after adjustment for age, sex, and BMI, Southeast Asian individuals had the highest diabetes prevalence among all Asian subgroups.[74] The MASALA study (Mediators of Atherosclerosis of South Asians Living in America) found a higher prevalence of type 2 diabetes in South Asian women (22.4%) compared with women of other ethnicities from MESA (Multi-Ethnic Study of Atherosclerosis) even after adjustment for age, sex, and BMI (15.2% in non-Hispanic Black, 11.7% in Hispanic, 12.9% in Chinese American, and 5.6% in non-Hispanic White women).[75]

Obesity

The prevalence of obesity (BMI >30 kg/m^2) has continued to increase among adults in the United States, from 30.5% in 1999 to 39.5% in 2016. The highest age-adjusted obesity

(55.3%) and extreme obesity (15.3%) prevalences were in non-Hispanic Black women compared with non-Hispanic White, Hispanic/Latina, or non-Hispanic Asian women.[1] Obesity is highly prevalent in Mexican American[76] and Puerto Rican[43,77] women. There is a higher prevalence of obesity in Hispanic/Latina women than in Hispanic/Latino men and a significantly higher prevalence than in non-Hispanic White women (48.4% versus 37.8%, respectively) in prior and current studies. In HCHS/SOL, only 21.2% of women were at a healthy BMI.[43]

Obesity is 50% more likely in American Indian/Alaska Native adults compared with non-Hispanic White adults, with an age-adjusted percentage of 48.1% of American Indian/Alaska Native individuals ≥18 years of age with obesity. However, sex-specific data are not available.[78] Among Alaska Native women, obesity rates vary between 32.8% and 60%.[79,80] The mean BMI among American Indian/Alaska Native women is 30 to 34.9 kg/m^2, but data are limited, and obesity rates vary greatly.[49,79] In a separate subanalysis of the SHS, obesity was an inverse predictor for CVD in women after adjustment for smoking, which warrants further investigation.[34]

Although Asian American adults have a lower prevalence of overweight and obesity than adults in other racial groups, they have higher rates of hypertension, CVD, and type 2 diabetes at the same BMI levels.[81] As a result, the World Health Organization and American Diabetes Association have recommended lower BMI cut points for defining overweight (BMI ≥23 kg/m^2) and obesity (BMI ≥27.5 kg/m^2) in Asian individuals to increase the identification of cardiometabolic risk in this population.[73,82,83] In addition to environmental and genetic factors, increased cardiometabolic risk has been attributed to fat composition and distribution. Asian individuals generally have a higher percentage of body fat than non-Hispanic White individuals of the same age, sex, and BMI. Studies have shown a greater proportion of central adiposity and visceral body fat deposition in Chinese, Filipino, and Asian Indian people compared with non-Hispanic White and non-Hispanic Black people.[21,82] The International Diabetes Federation recommends a waist circumference cutoff of ≥88 cm clinically for non-Hispanic White women and ≥80 cm for South Asian women.[84,85] In addition, Asian women carry greater abdominal and visceral fat compared with non-Hispanic White women with similar adiposity and BMI, which may contribute to their elevated cardiometabolic risk.[86] A cross-sectional cohort study of MESA and MASALA found that South Asian individuals generally have greater intramuscular and visceral fat and less lean mass compared with Chinese American individuals.[87]

Tobacco Use

Data from the 2020 NHIS (National Health Interview Survey) indicate that overall tobacco use among non-Hispanic Black adults is comparable to that in non-Hispanic White adults, with 14.4% of non-Hispanic Black adults ≥18 years of age using cigarettes, followed by 1.6% using cigars, 1.6% reporting electronic cigarette use, and 1% using pipes.[88] Data show that non-Hispanic Black individuals initiate smoking at an older age and smoke fewer cigarettes per day; however, they die of smoking-related diseases at higher rates than their non-Hispanic White counterparts.[89] Sex-specific data based on race and ethnicity were not reported, but data from the National Center for Health Statistics showed decreasing rates of

current cigarette use in non-Hispanic Black women from 23.1% in 1990 to 1992 to 12.1% in 2016 to 2018.[90]

Data from the 2020 NHIS indicate that overall tobacco product use is lower for US Hispanic/Latino adults than for non-Hispanic White adults and non-Hispanic Black adults, with 8.0% of Hispanic/Latino adults ≥18 years of age using cigarettes, followed by 2.8% reporting electronic cigarette use, 2.2% using cigars, and 0.9% using pipes.[88] Data show that Hispanic/Latino adults born in the United States are more likely to smoke cigarettes than foreign-born Hispanic/Latino adults residing in the United States. In addition, Puerto Rican people were more likely to smoke cigarettes compared with Dominican and Mexican American people living in the United States.[89] Sex-specific data based on race and ethnicity were not reported, but data from the National Center for Health Statistics showed decreasing rates of current cigarette smoking in Hispanic/Latina women from 15.8% in 1990 to 1992 to 7.0% in 2016 to 2018.[90]

Traditional tobacco is an important part of the culture for many American Indian/Alaska Native people, and it is used for ceremonial and medicinal purposes.[91] However, the use of commercial tobacco (eg, cigarette smoking, vaping, chew) is higher for American Indian/Alaska Native individuals overall than for other racial and ethnic populations in the United States.[92] Approximately 1 in 3 American Indian/Alaska Native women report current smoking, and >50% of American Indian/Alaska Native women report being either current or former smokers, with the prevalence of smoking during pregnancy being 15% among American Indian/Alaska Native women.[88,93–95]

Data from the 2020 NHIS indicate that overall nicotine use among Asian adults is lower than among non-Hispanic White adults, with 8.0% of Asian adults ≥18 years of age using cigarettes, followed by 3.4% reporting electronic cigarette use, 0.9% using cigars, and 0.4% using pipes.[88] Sex-specific data based on race and ethnicity were not reported, but data from the National Center for Health Statistics show decreasing rates of current cigarette smoking in Asian women from 6.3% in 1990 to 1992 to 4.5% in 2016 to 2018.[90]

A summary of the CVD risk factors of hypertension, dyslipidemia, diabetes, obesity, and tobacco use specific to race and ethnicity is provided in Table 1.

NONTRADITIONAL RISK FACTORS AND SDOH

Sex-Specific Risk Factors

Much discussion has occurred over the utility of incorporating nontraditional risk factors into standard risk assessment tools. The US Preventive Services Task Force concluded in 2018 that there was insufficient evidence to say whether adding nontraditional risk factors such as high-sensitivity C-reactive protein or coronary artery calcium score to traditional risk assessment tools would benefit patients who do not have CVD symptoms.[96] However, there is much to consider when examining sex-specific factors and their impact on CVD risk. For example, pregnancy-related risk factors such as preeclampsia and eclampsia demonstrate notable racial and ethnic disparities, with the highest age-adjusted prevalence seen in non-Hispanic Black compared with Hispanic/Latina and non-

Hispanic White women. This places the mother at risk for severe morbidity and increases future cardiovascular risk.[97,98] Asian women may have the highest risk for developing cardiovascular complications from preeclampsia.[99] In the United States, 2 of 3 women who experience preeclampsia will die of heart disease.[100] Moreover, the impact of pregnancy-related risk factors extends to the offspring born to women with preeclampsia and increases their likelihood of having hypertension and obesity, placing them at higher risk for CVD, particularly heart disease and CVA.[100] Notably, nearly one-third of young adults with hypertension were born to mothers who experienced hypertension during pregnancy.[100]

The history of one's menstrual cycle, including the age at the start of menarche and menopause, is another nontraditional, sex-specific risk factor assessment that should be considered when evaluating women for CVD risk. Early menarche is associated with adiposity and metabolic syndrome, suggested to be attributable, in part, to increased lifetime exposure to estrogen.[101] In addition, early age at menarche has been associated with increased risk of CVD events and all-cause death.[102] Both early age and late age at menarche have been associated with increased risks of CHD,[103] with a suggestion that inflammatory biomarkers are at play in the development of angiographic coronary artery disease and could play a role in mediating atherosclerotic plaque destabilization.[104] With regard to menopause, it is imperative to state that menopause is a natural part of a woman's life cycle, and changes that occur during this phase of life can affect heart health. The prevalence of metabolic syndrome increases with menopause.[105] In addition, diminishing levels of estrogen lead to changes in the lipid profile by reducing protective high-density lipoprotein and elevating apolipoprotein B levels and triglycerides, thereby increasing the risk for CVD.[106] Decreasing estrogen levels have also been associated with increased intraarterial cholesterol deposition and increased visceral fat, which are associated with an increase in triglycerides and insulin resistance.[107]

Polycystic ovarian syndrome is a common sex-specific endocrine disorder that affects 5% to 10% of women of reproductive age that also has deleterious effects on the cardiovascular risk profile.[108] Because of the many clinical derangements associated with polycystic ovarian syndrome such as hypertension, altered lipid and glucose metabolism, vascular injury, and systemic inflammation, polycystic ovarian syndrome has previously been recommended to be considered a cardiovascular risk factor.[109] Similarly, those with an autoimmune disorders, of whom an estimated 80% are women, are at greater risk of developing CVD and premature CVD. Autoimmune diseases demonstrate a clear sex bias with a greater prevalence among women, occurring at a rate of 2:1, with some disorders being genetic and some sporadic.[110] Regardless of the mechanism of manifestation, patients with autoimmune disease are predisposed to accelerated atherosclerosis, increased CVD risk, and worse outcome from cardiovascular events as a result of inflammatory-mediated endothelial dysfunction and accelerated atherosclerosis.[111]

Social Determinants of Health

SDOH, including barriers to health care access and structural racism, are also primary determinants that complicate the prevention and management of CVD.[18] There is evidence that SDOH factors experienced in youth such as adverse neighborhood characteristics,

poverty, toxic exposures, inability to access health care, or inability to afford care may affect heart health into adulthood and contribute to CVD risk factors and outcomes in adulthood.[1,16,23]

Environmental factors such as air pollution, high long-term arsenic exposure, and cadmium and lead exposure have been linked to CVD, including CHD, CVA, and peripheral artery disease.[112,113] Asian communities in the United States are disproportionately exposed to air pollution compared with predominantly non-Hispanic White communities, although the number of studies is small.[114] In MESA, air pollution exposure was associated with carotid intima-media thickness, a predictor of CVD, in Chinese American women.[115] Disproportionate lead exposure among non-Hispanic Black women in the United States is of major concern because blood lead levels increase during periods of increased bone metabolism such as pregnancy and menopause and have been related to preeclampsia and increased hypertension risk.[116–119] Air pollution and arsenic exposures have been associated with increased CVD and diabetes risk in Hispanic/Latino communities.[50–55] American Indian communities in the Southwest and Midwest are also disproportionately affected by contaminant metal exposures, with arsenic and cadmium exposures related to increased risk of CVD among American Indian women.[113,120–122]

Evidence also suggests that perceived discrimination and racism are contributing factors. Such conditions lead to upregulation of the sympathetic nervous system and hypothalamic axis and inflammatory dysregulation, leading to an increased risk for CVD, contributing to cardiovascular health disparities, particularly among women of underrepresented races and ethnicities.[123] As a result, non-Hispanic Black women living in socially disenfranchised communities are 3 times more likely to have hypertension, with objective measures of structural racism strongly associated with hypertension and obesity in this population.[124] Researchers attribute carrying a higher allostatic load, a measure of physiological impact of stress on the body, to the combined weight of racism and sexism, with the terms weathering and superwoman schema used to explain the premature deterioration of health in non-Hispanic Black women exposed to stress, danger, and social disenfranchisement.[125–127] Additional information on the cardiovascular health of non-Hispanic Black, Hispanic/Latina, American Indian/Alaska Native, and Asian women and a detailed discussion on SDOH can be found in the AHA scientific statements focused on people of underrepresented races and ethnicities.[16,18–23]

CURRENT GUIDELINES ON CVD PRIMARY PREVENTIVE THERAPIES IN WOMEN

Notable Progress Made

Recent AHA/American College of Cardiology guidelines on the management of hypertension (2017), management of cholesterol (2018), and primary prevention of CVD (2019) have recognized the role of sex-specific risk factors in the evaluation and management of cardiovascular risk factors.[4,5,128] Current guideline-based recommendations on the management of cardiovascular risk factors and prevention of atherosclerotic CVD are summarized here and in Table 2.[4,5,128–131] AHA scientific statements on cardiovascular

considerations in pregnancy and menopause transition have further highlighted the need for nuanced management of cardiovascular risk in women.[105,132] In addition, statin treatment groups have been updated in the 2018 cholesterol guidelines, taking into account sex-specific risk factors.[5] Statin therapy is recommended for primary prevention in patients with a 10-year atherosclerotic CVD risk ≥20% or those with borderline or intermediate risk, depending on the presence of risk enhancers, including history of preeclampsia, premature menopause, and inflammatory conditions, that disproportionately affect women.[4] In addition, the American Diabetes Association's "Standards of Care in Diabetes—2023" highlights the management of women with prediabetes with a history of gestational diabetes, making recommendations for more frequent screening and more intensive lifestyle and treatment interventions.[133]

Recently, controversy arose after randomized clinical trials demonstrated no net benefit of low-dose aspirin for the primary prevention of atherosclerotic CVD in patients with diabetes, the elderly, or those with intermediate atherosclerotic CVD risk, regardless of sex.[134–136] However, for women at high risk of preeclampsia, special consideration is required. The American College of Obstetricians and Gynecologists recommends starting low-dose aspirin (81 mg/d) between 12 and 28 weeks of gestation and continuing it daily until delivery to prevent preeclampsia in women with high-risk features (chronic hypertension, history of preeclampsia, multifetal gestation, diabetes, renal disease, and autoimmune disease) or in women with >1 moderate-risk factors (BMI ≥30 kg/m^2, nulliparity, maternal age ≥35 years, family history of preeclampsia, Black race, low socioeconomic status, personal history of previous adverse pregnancy outcomes).[130,131] The US Preventive Services Task Force has similar recommendations to prescribe low-dose aspirin after 12 weeks of gestation to individuals at high risk of preeclampsia or with a combination of multiple moderate risk factors.[137]

CONSIDERATIONS AND CURRENT GAPS IN KNOWLEDGE

CVD prevention guidance statements have evolved over the decades, with recent emphasis on the consideration of the effects of nontraditional sex-specific risk factors on women's cardiovascular health. Despite this progress, more work needs to be done to refine the understanding we have of biological influencers of health. In addition, racial and ethnic refinements are missing, leaving us to extrapolate from racial and ethnic generalizations. Because of the lack of Hispanic/Latino, American Indian/Alaska Native, and Asian American representation in large registries used for cardiovascular risk assessment, including the American College of Cardiology/AHA Pooled Cohort Equation, specific risk calculators that more broadly incorporate race, ethnicity, and SDOH are currently not available, and the default White risk equation is applied, which may not accurately predict CVD risk.[4] Inadequate race and ethnicity data hamper optimal management of CVD risk factors and affect outcomes.

Future CVD prevention guidelines would be bolstered by culturally specific lifestyle recommendations tailored to the cultural norms and expectations that influence behaviors, beliefs, and attitudes about diet, physical activity, and healthy weight. Community-based approaches, faith-based community partnerships, and peer support in encouraging a better

lifestyle for chronic disease management among women could play an important role in improving the primary prevention of CVD. For example, the SAHELI study (South Asian Heart Lifestyle Intervention) demonstrated that an intervention with culturally targeted, community-based lifestyle interventions designed for medically underserved South Asian individuals was more effective at addressing CVD risk factors than print health education materials.[138] Key considerations in providing culturally competent care are the patient's preferred language and religion, dietary restrictions, sex identity, cultural norms and practices, health literacy, and cultural differences in communication style.[139]

Potential strategies to use include the following: (1) Provide barrier-free coverage for recommended evidence-based therapies; (2) incorporate screening and treatment for cardiovascular risk factors in all primary health care settings for all patients; (3) develop and increase access to culturally tailored consultations and services such as health education, preconception counseling, weight loss, and nutritional counseling; (4) share health messages through media campaigns in the preferred language of the target population with messaging sensitive to the specific cultural goup[89]; (5) increase enrollment of underrepresented populations in clinical trials; (6) mandate and enforce reporting of research participant data by sex and race and ethnicity; (7) create and promote programmatic pipelines for recruiting people who are underrepresented in medicine; and (8) recruit and retain women of underrepresented races and ethnicities in cardiovascular research.

CONCLUSIONS

CVD risk assessment in women is multifaceted in that it surpasses the traditional risk factors to include sex-specific biological risk factors and incorporates race and ethnicity and nonbiological factors: SDOH and behavioral and environmental factors (Figures 1 and 2). A greater focus on addressing adverse levels of all CVD risk factors among women of underrepresented races and ethnicities is warranted to avert future CVD morbidity and death. Adverse social factors such as health care access, migrant status, language barriers, discrimination, acculturation, and environmental racism (the disproportionate impact of environmental hazards on people of color) are common in communities of underrepresented races and ethnicities and pose a significant challenge in the diagnosis of CVD and the application of treatment modalities. Culturally sensitive, peer-led community and health care professional education is a necessary step in CVD prevention. Equitable access to guideline-approved, evidence-based cardiovascular preventive health care based on available data should be available for all women regardless of race and ethnicity. Despite this knowledge, these guidelines are not equally incorporated into practice, which highlights a call to action.

REFERENCES

1. 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. Circulation. 2022;145:e153–e639. doi: 10.1161/CIR.0000000000001052 [PubMed: 35078371]
2. Institute of Medicine (US) Committee on Women's Health Research. Women's Health Research: Progress, Pitfalls, and Promise. National Academies Press; 2010.

3. Lloyd-Jones DM, Elkind M, Albert MA. American Heart Association's 2024 Impact Goal: every person deserves the opportunity for a full, healthy life. Circulation. 2021;144:e277–e279. doi: 10.1161/CIRCULATIONAHA.121.057617 [PubMed: 34723639]
4. Grundy SM, Stone NJ, Bailey AL, Beam C, Birtcher KK, Blumenthal RS, Braun LT, de Ferranti S, Faiella-Tommasino J, Forman DE, et al. 2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA guideline on the management of blood cholesterol: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines [published correction appears in Circulation. 2019;139:e1182–e1186]. Circulation. 2019;139:e1082–e1143. doi: 10.1161/CIR.0000000000000698 [PubMed: 30586774]
5. Whelton PK, Carey RM, Aronow WS, Casey DE Jr, Collins KJ, Dennison Himmelfarb C, DePalma SM, Gidding S, Jamerson KA, Jones DW, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation. 2018;138:e484–e594. doi: 10.1161/CIR.0000000000000596 [PubMed: 30354654]
6. Rao G, Powell-Wiley TM, Ancheta I, Hairston K, Kirley K, Lear SA, North KE, Palaniappan L, Rosal MC; on behalf of the American Heart Association Obesity Committee of the Council on Lifestyle and Cardiometabolic Health. Identification of obesity and cardiovascular risk in ethnically and racially diverse populations. Circulation. 2015;132:457–472. doi:10.1161/CIR.0000000000000223 [PubMed: 26149446]
7. Kuehner C Why is depression more common among women than among men? Lancet Psychiatry. 2017;4:146–158. doi: 10.1016/S2215-0366(16)30263-2 [PubMed: 27856392]
8. Depressive disorders. In: Diagnostic and Statistical Manual of Mental Disorders DSM-5. 5th ed. American Psychiatric Association; 2013. Accessed August 22, 2022. https://dsm.psychiatryonline.org
9. National Institute of Mental Health. Depression in women: 5 things you should know. Accessed August 22, 2022. https://nimh.nih.gov/health/publications/depression-in-women/index.shtml
10. Office on Women's Health. Depression. Accessed August 22, 2022. https://womenshealth.gov/mental-health/mental-health-conditions/depression
11. Centers for Disease Control and Prevention. Depression among women. Accessed August 22, 2022. https://cdc.gov/reproductivehealth/depression/index.htm
12. American College of Obstetricians and Gynecologists. Frequently asked questions: labor, delivery, and postpartum care FAQ091: postpartum depression. Accessed August 22, 2022. https://acog.org/Patients/FAQs/Postpartum-Depression
13. American College of Obstetricians and Gynecologists. Frequently asked questions. Gynecological problems FAQ057: premenstrual syndrome (PMS). Accessed August 22, 2022. https://acog.org/Patients/FAQs/Premenstrual-Syndrome-PMS
14. World Health Organization. Gender and women's mental health. Accessed August 22, 2022. https://apps.who.int/iris/handle/10665/68884
15. Cho L, Davis M, Elgendy I, Epps K, Lindley KJ, Mehta PK, Michos ED, Minissian M, Pepine C, Vaccarino V, et al. ; ACC CVD Women's Committee Members. Summary of updated recommendations for primary prevention of cardiovascular disease in women: *JACC* state-of-the-art review. J Am Coll Cardiol. 2020;75:2602–2618. doi: 10.1016/j.jacc.2020.03.060 [PubMed: 32439010]
16. White-Williams C, Rossi LP, Bittner VA, Driscoll A, Durant RW, Granger BB, Graven LJ, Kitko L, Newlin K, Shirey M; on behalf of the American Heart Association Council on Cardiovascular and Stroke Nursing; Council on Clinical Cardiology; and Council on Epidemiology and Prevention. Addressing social determinants of health in the care of patients with heart failure: a scientific statement from the American Heart Association. Circulation. 2020;141:e841–e863. doi: 10.1161/CIR.0000000000000767 [PubMed: 32349541]
17. Lloyd-Jones DM, Allen NB, Anderson CAM, Black T, Brewer LC, Foraker RE, Grandner MA, Lavretsky H, Perak AM, Sharma G, et al. ; on behalf of the American Heart Association. 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 [PubMed: 35766027]

18. Carnethon MR, Pu J, Howard G, Albert MA, Anderson CA, Bertoni AG, Mujahid MS, Palaniappan L, Taylor HA Jr, Willis M; on behalf of the American Heart Association Council on Epidemiology and Prevention; Council on Cardiovascular Disease in the Young; Council on Cardiovascular and Stroke Nursing; Council on Clinical Cardiology; Council on Functional Genomics and Translational Biology; and Stroke Council. Cardiovascular health in African Americans: a scientific statement from the American Heart Association. Circulation. 2017;136:e393–e423. doi: 10.1161/CIR.0000000000000534 [PubMed: 29061565]

19. Rodriguez CJ, Allison M, Daviglus ML, Isasi CR, Keller C, Leira EC, Palaniappan L, Pina IL, Ramirez SM, Rodriguez B, et al. ; on behalf of the American Heart Association Council on Epidemiology and Prevention, Council on Clinical Cardiology, and Council on Cardiovascular and Stroke Nursing. Status of cardiovascular disease and stroke in Hispanics/Latinos in the United States: a science advisory from the American Heart Association. Circulation. 2014;130:593–625. doi: 10.1161/CIR.0000000000000071 [PubMed: 25098323]

20. Breathett K, Sims M, Gross M, Jackson EA, Jones EJ, Navas-Acien A, Taylor H, Thomas KL, Howard BV; on behalf of the American Heart Association Council on Epidemiology and Prevention; Council on Quality of Care and Outcomes Research; Council on Cardiovascular and Stroke Nursing; Council on Clinical Cardiology; and Council on Lifestyle and Cardiometabolic Health. Cardiovascular health in American Indians and Alaska Natives: a scientific statement from the American Heart Association. Circulation. 2020;141:e948–e959. doi: 10.1161/CIR.0000000000000773 [PubMed: 32460555]

21. Palaniappan LP, Araneta MR, Assimes TL, Barrett-Connor EL, Carnethon MR, Criqui MH, Fung GL, Narayan KM, Patel H, Taylor-Piliae RE, et al. ; on behalf of the American Heart Association Council on Epidemiology and Prevention, Council on Peripheral Vascular Disease, Council on Nutrition, Physical Activity, and Metabolism, Council on Clinical Cardiology, and Council on Cardiovascular Nursing. Call to action: cardiovascular disease in Asian Americans: a science advisory from the American Heart Association [published correction appears in Circulation. 2010;122:e516]. Circulation. 2010;122:1242–1252. doi: 10.1161/CIR.0b013e3181f22af4 [PubMed: 20733105]

22. Volgman AS, Palaniappan LS, Aggarwal NT, Gupta M, Khandelwal A, Krishnan AV, Lichtman JH, Mehta LS, Patel HN, Shah KS, et al. ; on behalf of the American Heart Association Council on Epidemiology and Prevention; Cardiovascular Disease and Stroke in Women and Special Populations Committee of the Council on Clinical Cardiology; Council on Cardiovascular and Stroke Nursing; Council on Quality of Care and Outcomes Research; and Stroke Council. Atherosclerotic cardiovascular disease in South Asians in the United States: epidemiology, risk factors, and treatments: a scientific statement from the American Heart Association [published correction appears in Circulation. 2018;138:e76]. Circulation. 2018;138:e1–e34. doi: 10.1161/CIR.0000000000000580 [PubMed: 29794080]

23. Havranek EP, Mujahid MS, Barr DA, Blair IV, Cohen MS, Cruz-Flores S, Davey-Smith G, Dennison-Himmelfarb CR, Lauer MS, Lockwood DW, et al. ; on behalf of the American Heart Association Council on Quality of Care and Outcomes Research, Council on Epidemiology and Prevention, Council on Cardiovascular and Stroke Nursing, Council on Lifestyle and Cardiometabolic Health, and Stroke Council. Social determinants of risk and outcomes for cardiovascular disease: a scientific statement from the American Heart Association. Circulation. 2015;132:873–898. doi: 10.1161/CIR.0000000000000228 [PubMed: 26240271]

24. Williams DR. Race and health: basic questions, emerging directions. Ann Epidemiol. 1997;7:322–333. doi: 10.1016/s1047-2797(97)00051-3 [PubMed: 9250627]

25. 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 [PubMed: 33170755]

26. Borrell LN, Elhawary JR, Fuentes-Afflick E, Witonsky J, Bhakta N, Wu AHB, Bibbins-Domingo K, Rodriguez-Santana JR, Lenoir MA, Gavin JR 3rd. Race and genetic ancestry in medicine: a time for reckoning with racism. N Engl J Med. 2021;384:474–480. doi: 10.1056/NEJMms2029562 [PubMed: 33406325]

27. Office of Management and Budget. Revisions to the standards for the classification of federal data on race and ethnicity. Fed Regist. 1997. Accessed September 14, 2022. https://www.govinfo.gov/content/pkg/FR-1997-10-30/pdf/97-28653.pdf
28. Heron M Deaths: leading causes for 2019. Natl Vital Stat Rep. 2021;70:1–114.
29. Arias E, Xu JQ. United States life tables, 2020. National Vital Statistics Reports; vol 71 no 1. Hyattsville, MD: National Center for Health Statistics; 2022. doi: 10.15620/cdc:118055
30. Sorlie PD, Backlund E, Johnson NJ, Rogot E. Mortality by Hispanic status in the United States. JAMA. 1993;270:2464–2468. [PubMed: 8031341]
31. Espey DK, Jim MA, Cobb N, Bartholomew M, Becker T, Haverkamp D, Plescia M. Leading causes of death and all-cause mortality in American Indians and Alaska Natives. Am J Public Health. 2014;104:S303–S311. doi: 10.2105/AJPH.2013.301798 [PubMed: 24754554]
32. Day GE PE, Lanier AP. Alaska native mortality rates and trends. Public Health Rep. 2009;124:54–64. doi: 10.1177/003335490912400109 [PubMed: 19413028]
33. Veazie M, Ayala C, Schieb L, Dai S, Henderson JA, Cho P. Trends and disparities in heart disease mortality among American Indians/Alaska Natives, 1990–2009. Am J Public Health. 2014;104:S359–S367. doi: 10.2105/AJPH.2013.301715 [PubMed: 24754556]
34. Howard BV, Lee ET, Cowan LD, Devereux RB, Galloway JM, Go OT, Howard WJ, Rhoades ER, Robbins DC, Sievers ML, et al. Rising tide of cardiovascular disease in American Indians. the Strong Heart Study. Circulation. 1999;99:2389–2395. doi: 10.1161/01.cir.99.18.2389 [PubMed: 10318659]
35. Lee ET, Welty TK, Fabsitz R, Cowan LD, Le NA, Oopik AJ, Cucchiara AJ, Savage PJ, Howard BV. The Strong Heart Study: a study of cardiovascular disease in American Indians: design and methods. Am J Epidemiol. 1990;132:1141–1155. doi: 10.1093/oxfordjournals.aje.a115757 [PubMed: 2260546]
36. Howard BV, Lee ET, Cowan LD, Fabsitz RR, Howard WJ, Oopik AJ, Robbins DC, Savage PJ, Yeh JL, Welty TK. Coronary heart disease prevalence and its relation to risk factors in American Indians: the Strong Heart Study. Am J Epidemiol. 1995;142:254–268. doi: 10.1093/oxfordjournals.aje.a117632 [PubMed: 7631630]
37. Hayes DK, Denny CH, Keenan NL, Croft JB, Sundaram AA, Greenlund KJ. Racial/ethnic and socioeconomic differences in multiple risk factors for heart disease and stroke in women: Behavioral Risk Factor Surveillance System, 2003. J Womens Health (Larchmt). 2006;15:1000–1008. doi: 10.1089/jwh.2006.15.1000 [PubMed: 17125418]
38. Anderson RN, Copeland G, Hayes JM. Linkages to improve mortality data for American Indians and Alaska Natives: a new model for death reporting? Am J Public Health. 2014;104:S258–S262. doi: 10.2105/AJPH.2013.301647 [PubMed: 24754614]
39. Rhoades DA. Racial misclassification and disparities in cardiovascular disease among American Indians and Alaska Natives. Circulation. 2005;111:1250–1256. doi: 10.1161/01.CIR.0000157735.25005.3F [PubMed: 15769765]
40. Jim MA, Arias E, Seneca DS, Hoopes MJ, Jim CC, Johnson NJ, Wiggins CL. Racial misclassification of American Indians and Alaska Natives by Indian Health Service Contract Health Service Delivery Area. Am J Public Health. 2014;104:S295–S302. doi: 10.2105/AJPH.2014.301933 [PubMed: 24754617]
41. Hastings KG, Jose PO, Kapphahn KI, Frank ATH, Goldstein BA, Thompson CA, Eggleston K, Cullen MR, Palaniappan LP. Leading causes of death among Asian American subgroups (2003–2011). PLoS One. 2015;10:e0124341. doi: 10.1371/journal.pone.0124341 [PubMed: 25915940]
42. Sorlie PD, Allison MA, Avilés-Santa ML, Cai J, Daviglus ML, Howard AG, Kaplan R, LaVange LM, Raij L, Schneiderman N, et al. Prevalence of hypertension, awareness, treatment, and control in the Hispanic Community Health Study/Study of Latinos. Am J Hypertens. 2014;27:793–800. doi: 10.1093/ajh/hpu003 [PubMed: 24627442]
43. Daviglus ML, Talavera GA, Avilés-Santa ML, Allison M, Cai J, Criqui MH, Gellman M, Giachello AL, Gouskova N, Kaplan RC, et al. Prevalence of major cardiovascular risk factors and cardiovascular diseases among Hispanic/Latino individuals of diverse backgrounds in the United States. JAMA. 2012;308:1775–1784. doi: 10.1001/jama.2012.14517 [PubMed: 23117778]

44. Zhang Y, Lee ET, Devereux RB, Yeh J, Best LG, Fabsitz RR, Howard BV. Prehypertension, diabetes, and cardiovascular disease risk in a population-based sample: the Strong Heart Study. Hypertension. 2006;47:410–414. doi: 10.1161/01.HYP.0000205119.19804.08 [PubMed: 16446387]
45. Howard BV, Robbins DC, Sievers ML, Lee ET, Rhoades D, Devereux RB, Cowan LD, Gray RS, Welty TK, Go OT, et al. LDL cholesterol as a strong predictor of coronary heart disease in diabetic individuals with insulin resistance and low LDL: the Strong Heart Study. Arterioscler Thromb Vasc Biol. 2000;20:830–835. doi: 10.1161/01.atv.20.3.830 [PubMed: 10712410]
46. Jolly SE, Howard BV, Umans JG. Cardiovascular disease among Alaska Native peoples. Curr Cardiovasc Risk Rep. 2013;7:43814445. doi: 10.1007/s12170-013-0362-5
47. Jolly SE, Koller KR, Metzger JS, Day GM, Silverman A, Hopkins SE, Austin MA, Boden-Albala B, Ebbesson SOE, Boyer BB, et al. Prevalence of hypertension and associated risk factors in Western Alaska native people: the Western Alaska Tribal Collaborative for Health (WATCH) Study. J Clin Hypertens (Greenwich). 2015;17:812–818. doi: 10.1111/jch.12483 [PubMed: 25644577]
48. Howard BV, Comuzzie A, Devereux RB, Ebbesson SOE, Fabsitz RR, Howard WJ, Laston S, MacCluer JW, Silverman A, Umans JG, et al. Cardiovascular disease prevalence and its relation to risk factors in Alaska Eskimos. Nutr Metab Cardiovasc Dis. 2010;20:350–358. doi: 10.1016/j.numecd.2009.04.010 [PubMed: 19800772]
49. Howard BV, Lee ET, Yeh JL, Go O, Fabsitz RR, Devereux RB, Welty TK. Hypertension in adult American Indians: the Strong Heart Study. Hypertension. 1996;28:256–264. doi: 10.1161/01.hyp.28.2.256 [PubMed: 8707391]
50. Jose PO, Frank AT, Kapphahn KI, Goldstein BA, Eggleston K, Hastings KG, Cullen MR, Palaniappan LP. Cardiovascular disease mortality in Asian Americans. J Am Coll Cardiol. 2014;64:2486–2494. doi: 10.1016/j.jacc.2014.08.048 [PubMed: 25500233]
51. Fei K, Rodriguez-Lopez JS, Ramos M, Islam N, Trinh-Shevrin C, Yi SS, Chernov C, Perlman SE, Thorpe LE. Racial and ethnic subgroup disparities in hypertension prevalence, New York City Health and Nutrition Examination Survey, 2013–2014. Prev Chronic Dis. 2017;14:E33. doi: 10.5888/pcd14.160478 [PubMed: 28427484]
52. Zhao B, Jose PO, Pu J, Chung S, Ancheta IB, Fortmann SP, Palaniappan LP. Racial/ethnic differences in hypertension prevalence, treatment, and control for outpatients in Northern California 2010–2012. Am J Hypertens. 2015;28:631–639. doi: 10.1093/ajh/hpu189 [PubMed: 25352230]
53. Braun LT, Wilbur J, Buchholz SW, Schoeny ME, Miller AM, Fogg L, Volgman AS, McDevitt J. Cardiovascular risk in midlife African American women participating in a lifestyle physical activity program. J Cardiovasc Nurs. 2016;31:304–312. doi: 10.1097/JCN.0000000000000266 [PubMed: 27074650]
54. Virani SS, Koschinsky ML, Maher L, Mehta A, Orringer CE, Santos RD, Shapiro MD, Saseen JJ. Global think tank on the clinical considerations and management of lipoprotein(a): the top questions and answers regarding what clinicians need to know. Prog Cardiovasc Dis. 2022;73:32–40. doi: 10.1016/j.pcad.2022.01.002 [PubMed: 35063437]
55. Virani SS, Brautbar A, Davis BC, Nambi V, Hoogeveen RC, Sharrett AR, Coresh J, Mosley TH, Morrisett JD, Catellier DJ, et al. Associations between lipoprotein (a) levels and cardiovascular outcomes in Black and White subjects: the Atherosclerosis Risk in Communities (ARIC) study. Circulation. 2012;125:241–249. doi: 10.1161/CIRCULATIONAHA.111.045120 [PubMed: 22128224]
56. Ohira T, Schreiner PJ, Morrisett JD, Chambless LE, Rosamond WD, Folsom AR. Lipoprotein (a) and incident ischemic stroke: the Atherosclerosis Risk in Communities (ARIC) study. Stroke. 2006;37:1407–1412. doi: 10.1161/01.STR.0000222666.21482.b6 [PubMed: 16675734]
57. Rodriguez CJ, Daviglus ML, Swett K, Gonzalez HM, Gallo LC, Wassertheil-Smoller S, Giachello AL, Teng Y, Schneiderman N, Talavera GA, et al. Dyslipidemia patterns among Hispanics/Latinos of diverse background in the United States. Am J Med. 2014;127:1186–1194.e1. doi: 10.1016/j.amjmed.2014.07.026 [PubMed: 25195188]
58. Lee JS, Chang PY, Zhang Y, Kizer JR, Best LG, Howard BV. Triglyceride and HDL-C dyslipidemia and risks of coronary heart disease and ischemic stroke by glycemic dysregulation

status: the Strong Heart Study. Diabetes Care. 2017;40:529–537. doi: 10.2337/dc16-1958 [PubMed: 28122840]

59. Karthikeyan G, Teo KK, Islam S, McQueen MJ, Pais P, Wang X, Sato H, Lang CC, Sitthi-Amorn C, Pandey MR, et al. Lipid profile, plasma apolipoproteins, and risk of a first myocardial infarction among Asians: an analysis from the INTERHEART Study. J Am Coll Cardiol. 2009;53:244–253. doi: 10.1016/j.jacc.2008.09.041 [PubMed: 19147041]
60. Misra A, Khurana L. Obesity-related non-communicable diseases: South Asians vs White Caucasians. Int J Obes (Lond). 2011;35:167–187. doi: 10.1038/ijo.2010.135 [PubMed: 20644557]
61. Frank ATH, Zhao B, Jose PO, Azar KMJ, Fortmann SP, Palaniappan LP. Racial/ethnic differences in dyslipidemia patterns. Circulation. 2014;129:570–579. doi: 10.1161/CIRCULATIONAHA.113.005757 [PubMed: 24192801]
62. Paré G, Çaku A, McQueen M, Anand SS, Enas E, Clarke R, Boffa MB, Koschinsky M, Wang X, Yusuf S; INTERHEART Investigators. Lipoprotein (a) levels and the risk of myocardial infarction among 7 ethnic groups. Circulation. 2019;139:1472–1482. doi: 10.1161/CIRCULATIONAHA.118.034311 [PubMed: 30667276]
63. Casagrande SS, Linder B, Cowie CC. Prevalence of gestational diabetes and subsequent type 2 diabetes among US women. Diabetes Res Clin Pract. 2018;141:200–208. doi: 10.1016/j.diabres.2018.05.010 [PubMed: 29772286]
64. National Center for Health Statistics, Centers for Disease Control and Prevention. National Health and Nutrition Examination Survey (NHANES) public use data files. Accessed April 1, 2019. https://www.cdc.gov/nchs/nhanes/
65. Stark Casagrande S, Fradkin JE, Saydah SH, Rust KF, Cowie CC. The prevalence of meeting A1C, blood pressure, and LDL goals among people with diabetes, 1988–2010. Diabetes Care. 2013;36:2271–2279. doi: 10.2337/dc12-2258 [PubMed: 23418368]
66. National Center for Health Statistics, Centers for Disease Control and Prevention. National Health Interview Survey, public-use data files. 2020. Accessed February 16, 2021. http://www.cdc.gov/nchs/nhis/nhis_2020
67. Lee ET, Howard BV, Savage PJ, Cowan LD, Fabsitz RR, Oopik AJ, Yeh J, Go O, Robbins DC, Welty TK. Diabetes and impaired glucose tolerance in three American Indian populations aged 45–74 years: the Strong Heart Study. Diabetes Care. 1995;18:599–610. doi: 10.2337/diacare.18.5.599 [PubMed: 8585996]
68. Redwood DG, Lanier AP, Johnston JM, Asay ED, Slattery ML. Chronic disease risk factors among Alaska Native and American Indian people, Alaska, 2004–2006. Prev Chronic Dis. 2010;7:A85–A85. [PubMed: 20550843]
69. Carter EA, MacCluer JW, Dyke B, Howard BV, Devereux RB, Ebbesson SO, Resnick HE. Diabetes mellitus and impaired fasting glucose in Alaska Eskimos: the Genetics of Coronary Artery Disease in Alaska Natives (GOCADAN) study. Diabetologia. 2006;49:29–35. doi: 10.1007/s00125-005-0071-9 [PubMed: 16369773]
70. Narayanan ML, Schraer CD, Bulkow LR, Koller KR, Asay E, Mayer AM, Raymer TW. Diabetes prevalence, incidence, complications and mortality among Alaska Native people 1985–2006. Int J Circumpolar Health. 2010;69:236–252. doi: 10.3402/ijch.v69i3.17618 [PubMed: 20501061]
71. Palaniappan LP, Kwan AC, Abbasi F, Lamendola C, McLaughlin TL, Reaven GM. Lipoprotein abnormalities are associated with insulin resistance in South Asian Indian women. Metabolism. 2007;56:899–904. doi: 10.1016/j.metabol.2007.01.020 [PubMed: 17570249]
72. Araneta MR, Wingard DL, Barrett-Connor E. Type 2 diabetes and metabolic syndrome in Filipina-American women: a high-risk nonobese population. Diabetes Care. 2002;25:494–499. doi: 10.2337/diacare.25.3.494 [PubMed: 11874936]
73. Hsu WC, Araneta MR, Kanaya AM, Chiang JL, Fujimoto W. BMI cut points to identify at-risk Asian Americans for type 2 diabetes screening. Diabetes Care. 2015;38:150–158. doi: 10.2337/dc14-2391 [PubMed: 25538311]
74. Cheng YJ, Kanaya AM, Araneta MRG, Saydah SH, Kahn HS, Gregg EW, Fujimoto WY, Imperatore G. Prevalence of diabetes by race and ethnicity in the United States, 2011–2016. JAMA. 2019;322:2389–2398. doi: 10.1001/jama.2019.19365 [PubMed: 31860047]

75. Kanaya AM, Herrington D, Vittinghoff E, Ewing SK, Liu K, Blaha MJ, Dave SS, Qureshi F, Kandula NR. Understanding the high prevalence of diabetes in U.S. south Asians compared with four racial/ethnic groups: the MASALA and MESA studies. Diabetes Care. 2014;37:1621–1628. doi: 10.2337/dc13-2656 [PubMed: 24705613]
76. Crespo CJ, Loria CM, Burt VL. Hypertension and other cardiovascular disease risk factors among Mexican Americans, Cuban Americans, and Puerto Ricans from the Hispanic Health and Nutrition Examination Survey. Public Health Rep. 1996;111(suppl 2):7–10.
77. Hales CM, Fryar CD, Carroll MD, Freedman DS, Aoki Y, Ogden CL. Differences in obesity prevalence by demographic characteristics and urbanization level among adults in the United States, 2013–2016. JAMA. 2018;319:2419–2429. doi: 10.1001/jama.2018.7270 [PubMed: 29922829]
78. US Department of Health and Human Services, Office of Minority Health. Obesity and American Indians/Alaska Natives. Accessed November 8, 2022. https://minorityhealth.hhs.gov/omh/browse.aspx?lvl=4&lvlid=40
79. Wilson C, Gilliland S, Moore K, Acton K. The epidemic of extreme obesity among American Indian and Alaska Native adults with diabetes. Prev Chronic Dis. 2007;4:A06. [PubMed: 17173714]
80. Risica PM, Schraer C, Ebbesson SO, Nobmann ED, Caballero B. Overweight and obesity among Alaskan Eskimos of the Bering Straits region: the Alaska Siberia project. Int J Obes Relat Metab Disord. 2000;24:939–944. doi: 10.1038/sj.ijo.0801259 [PubMed: 10951530]
81. Cho J, Juon HS. Assessing overweight and obesity risk among Korean Americans in California using World Health Organization body mass index criteria for Asians. Prev Chronic Dis. 2006;3:A79. [PubMed: 16776880]
82. WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. Lancet. 2004;363:157–163. doi: 10.1016/S0140-6736(03)15268-3 [PubMed: 14726171]
83. Gray LJ, Yates T, Davies MJ, Brady E, Webb DR, Sattar N, Khunti K. Defining obesity cut-off points for migrant South Asians. PLoS One. 2011;6:e26464. doi: 10.1371/journal.pone.0026464 [PubMed: 22039493]
84. International Diabetes Federation. The IDF consensus worldwide definition of the metabolic syndrome. 2005. Accessed July 2, 2022. https://idf.org/e-library/consensus-statements/60-idfconsensus-worldwide-definitionof-the-metabolic-syndrome.html
85. Cheng Y, Qin SK, Li J, Dai GH, Shen BY, Ying JE, Ba Y, Liang H, Wang XB, Xu Y, et al. A multicenter clinical study: personalized medication for advanced gastrointestinal carcinomas with the guidance of patient-derived tumor xenograft (PDTX). J Cancer Res Clin Oncol. 2022;148:673–684. doi: 10.1007/s00432-021-03639-x [PubMed: 33864522]
86. Lim U, Ernst T, Buchthal SD, Latch M, Albright CL, Wilkens LR, Kolonel LN, Murphy SP, Chang L, Novotny R, et al. Asian women have greater abdominal and visceral adiposity than Caucasian women with similar body mass index. Nutr Diabetes. 2011;1:e6. doi: 10.1038/nutd.2011.2 [PubMed: 23449381]
87. Shah AD, Kandula NR, Lin F, Allison MA, Carr J, Herrington D, Liu K, Kanaya AM. Less favorable body composition and adipokines in South Asians compared with other US ethnic groups: results from the MASALA and MESA studies. Int J Obes (Lond). 2016;40:639–645. doi: 10.1038/ijo.2015.219 [PubMed: 26499444]
88. Cornelius ME, Loretan CG, Wang TW, Jamal A, Homa DM. Tobacco product use among adults—United States, 2020. MMWR Morb Mortal Wkly Rep. 2022;71:397–405. doi: 10.15585/mmwr.mm7111a1 [PubMed: 35298455]
89. Centers for Disease Control and Prevention. Health disparities related to commercial tobacco and advancing health equity. Accessed July 5, 2022. https://cdc.gov/tobacco/health-equity/index.htm
90. Centers for Disease Control and Prevention, National Center for Health Statistics. Health, United States, 2020–2021, Table 019. Accessed July 5, 2022. https://cdc.gov/nchs/data/hus/2019/019-508.pdf

91. Kunitz SJ. Historical Influences on contemporary tobacco use by Northern Plains and Southwestern American Indians. Am J Public Health. 2016;106:246–255. doi: 10.2105/AJPH.2015.302909 [PubMed: 26691134]
92. Centers for Disease Control and Prevention American Indian and Alaska Native people experience a health burden from commercial tobacco. 2019. Accessed March 3, 2021 https://cdc.gov/tobacco/health-equity/aian/health-burden.html
93. Strong Heart Study Data Book: A Report to Indian Communities. US Department of Health and Human Services; 2001. NIH Publication No 01–3285.
94. Azagba S, Manzione L, Shan L, King J. Trends in smoking during pregnancy by socioeconomic characteristics in the United States, 2010–2017. BMC Pregnancy Childbirth. 2020;20:52. doi: 10.1186/s12884-020-2748-y [PubMed: 31973722]
95. Department of Health and Human Services, Centers for Disease Control and Prevention, Office on Smoking and Health. Tobacco use among U.S. racial/ethnic minority groups—African Americans, American Indians and Alaska Natives, Asian Americans and Pacific Islanders, and Hispanics: a report of the Surgeon General. 1998. Accessed February 14, 2021. https://cdc.gov/tobacco/sgr/1998/complete_report/pdfs/complete_report.pdf
96. Curry SJ, Krist AH, Owens DK, Barry MJ, Caughey AB, Davidson KW, Doubeni CA, Epling JW Jr, Kemper AR, Kubik M, et al. ; US Preventive Services Task Force. Risk assessment for cardiovascular disease with nontraditional risk factors: US Preventive Services Task Force recommendation statement. JAMA. 2018;320:272–280. doi: 10.1001/jama.2018.8359 [PubMed: 29998297]
97. Liese KL, Mogos M, Abboud S, Decocker K, Koch AR, Geller SE. Racial and ethnic disparities in severe maternal morbidity in the United States. J Racial Ethn Health Disparities. 2019;6:790–798. [PubMed: 30877505]
98. Howell EA, Egorova NN, Janevic T, Brodman M, Balbierz A, Zeitlin J, Hebert PL. Race and ethnicity, medical insurance, and within-hospital severe maternal morbidity disparities. Obstet Gynecol. 2020;135:285–293. doi: 10.1097/AOG.0000000000003667 [PubMed: 31923076]
99. Minhas AS, Ogunwole SM, Vaught AJ, Wu P, Mamas MA, Gulati M, Zhao D, Hays AG, Michos ED. Racial disparities in cardiovascular complications with pregnancy-induced hypertension in the United States. Hypertension. 2021;78:480–488. doi: 10.1161/HYPERTENSIONAHA.121.17104 [PubMed: 34098730]
100. Association of Black Cardiologists, Inc. We are the faces of black maternal health. Accessed August 22, 2022. https://Abcardio.org
101. Apter D, Vihko R. Early menarche, a risk factor for breast cancer, indicates early onset of ovulatory cycles. J Clin Endocrinol Metab. 1983;57:82–86. doi: 10.1210/jcem-57-1-82 [PubMed: 6222061]
102. Jacobsen BK, Oda K, Knutsen SF, Fraser GE. Age at menarche, total mortality and mortality from ischaemic heart disease and stroke: the Adventist Health Study, 1976–88. Int J Epidemiol. 2009;38:245–252. doi: 10.1093/ije/dyn251 [PubMed: 19188208]
103. Canoy D, Beral V, Balkwill A, Wright FL, Kroll ME, Reeves GK, Green J, Cairns BJ; Million Women Study Collaborators. Age at menarche and risks of coronary heart and other vascular diseases in a large UK cohort. Circulation. 2015;131:237–244. doi: 10.1161/CIRCULATIONAHA.114.010070 [PubMed: 25512444]
104. Lee JJ, Cook-Wiens G, Johnson BD, Braunstein GD, Berga SL, Stanczyk FZ, Pepine CJ, Bairey Merz CN, Shufelt CL. Age at menarche and risk of cardiovascular disease outcomes: findings from the National Heart Lung and Blood Institute-sponsored Women's Ischemia Syndrome Evaluation. J Am Heart Assoc. 2019;8:e012406. doi: 10.1161/JAHA.119.012406 [PubMed: 31165670]
105. El Khoudary SR, Aggarwal B, Beckie TM, Hodis HN, Johnson AE, Langer RD, Limacher MC, Manson JE, Stefanick ML, Allison MA; on behalf of the American Heart Association Prevention Science Committee of the Council on Epidemiology and Prevention; and Council on Cardiovascular and Stroke Nursing. Menopause transition and cardiovascular disease risk: implications for timing of early prevention: a scientific statement from the American Heart Association. Circulation. 2020;142:e506–e532. doi: 10.1161/CIR.0000000000000912 [PubMed: 33251828]

106. Mesalic L, Tupkovic E, Kendic S, Balic D. Correlation between hormonal and lipid status in women in menopause. Bosn J Basic Med Sci. 2008;8:188–192. doi: 10.17305/bjbms.2008.2980 [PubMed: 18498273]

107. Carr MC. The emergence of the metabolic syndrome with menopause. J Clin Endocrinol Metab. 2003;88:2404–2411. doi: 10.1210/jc.2003-030242 [PubMed: 12788835]

108. Cho LW, Randeva HS, Atkin SL. Cardiometabolic aspects of polycystic ovarian syndrome. Vasc Health Risk Manag. 2007;3:55–63. [PubMed: 17583175]

109. Scicchitano P, Dentamaro I, Carbonara R, Bulzis G, Dachille A, Caputo P, Riccardi R, Locorotondo M, Mandurino C, Matteo Ciccone M. Cardiovascular risk in women with PCOS. Int J Endocrinol Metab. 2012;10:611–618. doi: 10.5812/ijem.4020 [PubMed: 23843832]

110. Angum F, Khan T, Kaler J, Siddiqui L, Hussain A. The prevalence of autoimmune disorders in women: a narrative review. Cureus. 2020;12:e8094. doi: 10.7759/cureus.8094 [PubMed: 32542149]

111. Durante A, Bronzato S. The increased cardiovascular risk in patients affected by autoimmune diseases: review of the various manifestations. J Clin Med Res. 2015;7:379–384. doi: 10.14740/jocmr2122w [PubMed: 25883699]

112. Rajagopalan S, Landrigan PJ. Pollution and the heart. N Engl J Med. 2021;385:1881–1892. doi: 10.1056/NEJMra2030281 [PubMed: 34758254]

113. Moon K, Guallar E, Navas-Acien A. Arsenic exposure and cardiovascular disease: an updated systematic review. Curr Atheroscler Rep. 2012;14:542–555. doi: 10.1007/s11883-012-0280-x [PubMed: 22968315]

114. Jones MR, Diez-Roux AV, Hajat A, Kershaw KN, O'Neill MS, Guallar E, Post WS, Kaufman JD, Navas-Acien A. Race/ethnicity, residential segregation, and exposure to ambient air pollution: the Multi-Ethnic Study of Atherosclerosis (MESA). Am J Public Health. 2014;104:2130–2137. doi: 10.2105/AJPH.2014.302135 [PubMed: 25211756]

115. Jones MR, Diez-Roux AV, O'Neill MS, Guallar E, Sharrett AR, Post W, Kaufman JD, Navas-Acien A. Ambient air pollution and racial/ethnic differences in carotid intima-media thickness in the Multi-Ethnic Study of Atherosclerosis (MESA). J Epidemiol Community Health. 2015;69:1191–1198. doi: 10.1136/jech-2015-205588 [PubMed: 26142402]

116. Geronimus AT, Hillemeier MM. Patterns of blood lead levels in US black and white women of childbearing age. Ethn Dis. 1992;2:222–231. [PubMed: 1467759]

117. Nash D, Magder L, Lustberg M, Sherwin RW, Rubin RJ, Kaufmann RB, Silbergeld EK. Blood lead, blood pressure, and hypertension in perimenopausal and postmenopausal women. JAMA. 2003;289:1523–1532. doi: 10.1001/jama.289.12.1523 [PubMed: 12672769]

118. Poropat AE, Laidlaw MAS, Lanphear B, Ball A, Mielke HW. Blood lead and preeclampsia: a meta-analysis and review of implications. Environ Res. 2018;160:12–19. doi: 10.1016/j.envres.2017.09.014 [PubMed: 28938191]

119. Morello-Frosch R, Jesdale BM. Separate and unequal: residential segregation and estimated cancer risks associated with ambient air toxics in U.S. metropolitan areas. Environ Health Perspect. 2006;114:386–393. doi: 10.1289/ehp.8500 [PubMed: 16507462]

120. Tellez-Plaza M, Guallar E, Howard BV, Umans JG, Francesconi KA, Goessler W, Silbergeld EK, Devereux RB, Navas-Acien A. Cadmium exposure and incident cardiovascular disease. Epidemiology. 2013;24:421–429. doi: 10.1097/EDE.0b013e31828b0631 [PubMed: 23514838]

121. Nigra AE, Chen Q, Chillrud SN, Wang L, Harvey D, Mailloux B, Factor-Litvak P, Navas-Acien A. Inequalities in public water arsenic concentrations in counties and community water systems across the United States, 2006–2011. Environ Health Perspect. 2020;128:127001. doi: 10.1289/EHP7313 [PubMed: 33295795]

122. Pang Y, Peng RD, Jones MR, Francesconi KA, Goessler W, Howard BV, Umans JG, Best LG, Guallar E, Post WS, et al. Metal mixtures in urban and rural populations in the US: the Multi-Ethnic Study of Atherosclerosis and the Strong Heart Study. Environ Res. 2016;147:356–364. doi: 10.1016/j.envres.2016.02.032 [PubMed: 26945432]

123. Conway-Phillips R, Dagadu H, Motley D, Shawahin L, Janusek LW, Klonowski S, Saban KL. Qualitative evidence for Resilience, Stress, and Ethnicity (RiSE): a program to address

race-based stress among Black women at risk for cardiovascular disease. Complement Ther Med. 2020;48:102277. doi: 10.1016/j.ctim.2019.102277 [PubMed: 31987226]

124. Basile Ibrahim B, Barcelona V, Condon EM, Crusto CA, Taylor JY. The association between neighborhood social vulnerability and cardiovascular health risk among Black/African American women in the InterGEN Study. Nurs Res. 2021;70:S3–S12. doi: 10.1097/NNR.0000000000000523 [PubMed: 34074961]
125. Forrester S, Jacobs D, Zmora R, Schreiner P, Roger V, Kiefe CI. Racial differences in weathering and its associations with psychosocial stress: the CARDIA study. SSM Popul Health. 2019;7:003. doi: 10.1016/j.ssmph.2018.11.003 [PubMed: 31294072]
126. 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 [PubMed: 33563007]
127. 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 [PubMed: 35157523]
128. Arnett DK, Blumenthal RS, Albert MA, Buroker AB, Goldberger ZD, Hahn EJ, Himmelfarb CD, Khera A, Lloyd-Jones D, McEvoy JW, et al. 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines [published corrections appear in Circulation. 2019;140:e649–e650, Circulation. 2020;141:e60, and Circulation. 2020;141:e774]. Circulation. 2019;140:e596–e646. doi: 10.1161/CIR.0000000000000678 [PubMed: 30879355]
129. ACOG Practice Bulletin No. 202: gestational hypertension and preeclampsia. Obstet Gynecol. 2019;133:e1–e25. doi: 10.1097/AOG.0000000000003018
130. Gestational hypertension and preeclampsia: ACOG Practice Bulletin, Number 222. Obstet Gynecol. 2020;135:e237–e260. doi: 10.1097/AOG.0000000000003891 [PubMed: 32443079]
131. ACOG Committee Opinion No. 743 summary: low-dose aspirin use during pregnancy. Obstet Gynecol. 2018;132:254–256. doi: 10.1097/AOG.0000000000002709 [PubMed: 29939936]
132. Mehta LS, Warnes CA, Bradley E, Burton T, Economy K, Mehran R, Safdar B, Sharma G, Wood M, Valente AM, 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 Stroke Council. Cardiovascular considerations in caring for pregnant patients: a scientific statement from the American Heart Association [published corrections appear in Circulation. 2020;141:e904 and Circulation. 2021;143:e792–e793]. Circulation. 2020;141:e884–e903. doi: 10.1161/CIR.0000000000000772 [PubMed: 32362133]
133. ElSayed NA, Aleppo G, Aroda VR, Bannuru RR, Brown FM, Bruemmer D, Collins BS, Hilliard ME, Isaacs D, Johnson EL, et al. Classification and diagnosis of diabetes: standards of care in diabetes—2023. Diabetes Care. 2023;46(suppl 1):S19–S40. doi: 10.2337/dc23-S002 [PubMed: 36507649]
134. Bowman L, Mafham M, Wallendszus K, Stevens W, Buck G, Barton J, Murphy K, Aung T, Haynes R, Cox J, et al. ; ASCEND Study Collaborative Group. Effects of aspirin for primary prevention in persons with diabetes mellitus. N Engl J Med. 2018;379:1529–1539. doi: 10.1056/NEJMoa1804988 [PubMed: 30146931]
135. McNeil JJ, Wolfe R, Woods RL, Tonkin AM, Donnan GA, Nelson MR, Reid CM, Lockery JE, Kirpach B, Storey E, et al. ; ASPREE Investigator Group. Effect of aspirin on cardiovascular events and bleeding in the healthy elderly. N Engl J Med. 2018;379:1509–1518. doi: 10.1056/NEJMoa1805819 [PubMed: 30221597]
136. Gaziano JM, Brotons C, Coppolecchia R, Cricelli C, Darius H, Gorelick PB, Howard G, Pearson TA, Rothwell PM, Ruilope LM, et al. ; ARRIVE Executive Committee. Use of aspirin to reduce risk of initial vascular events in patients at moderate risk of cardiovascular disease (ARRIVE): a randomised, double-blind, placebo-controlled trial. Lancet. 2018;392:1036–1046. doi: 10.1016/S0140-6736(18)31924-X [PubMed: 30158069]

137. Aspirin use to prevent preeclampsia and related morbidity and mortality: US Preventive Services Task Force recommendation statement. JAMA. 2021;326:1186–1191. doi: 10.1001/jama.2021.14781 [PubMed: 34581729]

138. Kandula NR, Dave S, De Chavez PJ, Bharucha H, Patel Y, Seguil P, Kumar S, Baker DW, Spring B, Siddique J. Translating a heart disease lifestyle intervention into the community: the South Asian Heart Lifestyle Intervention (SAHELI) study; a randomized control trial. BMC Public Health. 2015;15:1064. doi: 10.1186/s12889-015-2401-2 [PubMed: 26475629]

139. Health Research & Educational Trust. Becoming a culturally competent health care organization. June 2013. Accessed September 24, 2022. www.hpoe.org

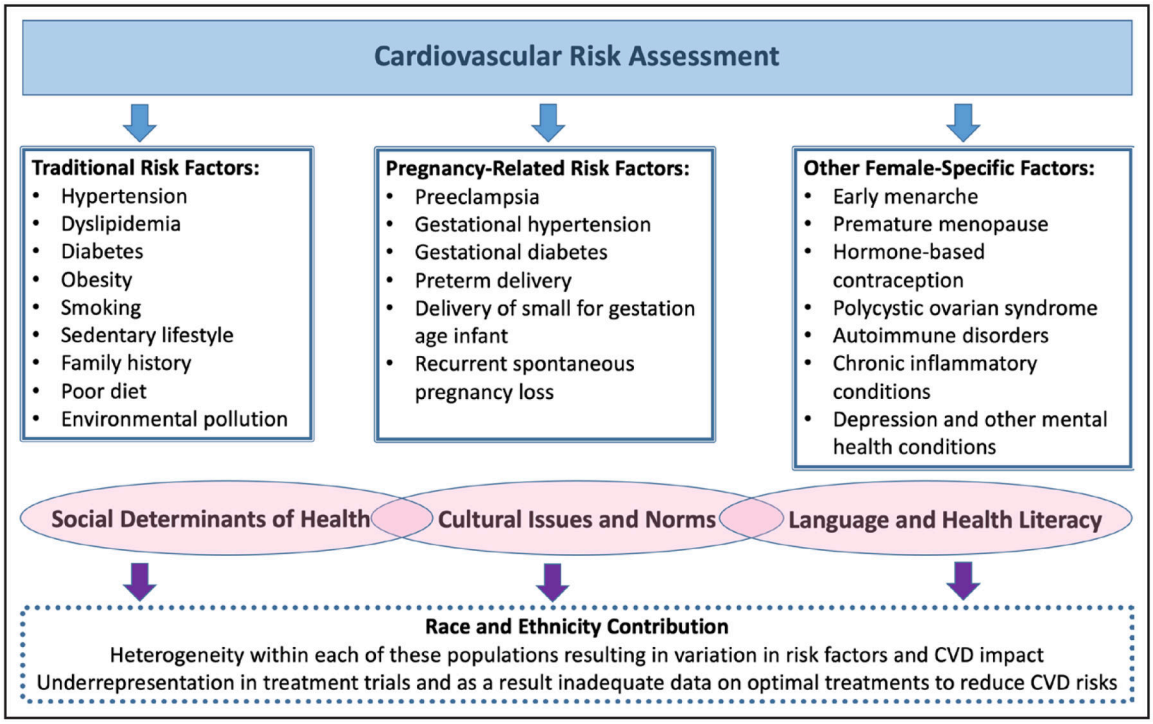

Figure 1. Cardiovascular risk factors in women and the impact of race and ethnicity contribution.

CVD indicates cardiovascular disease.

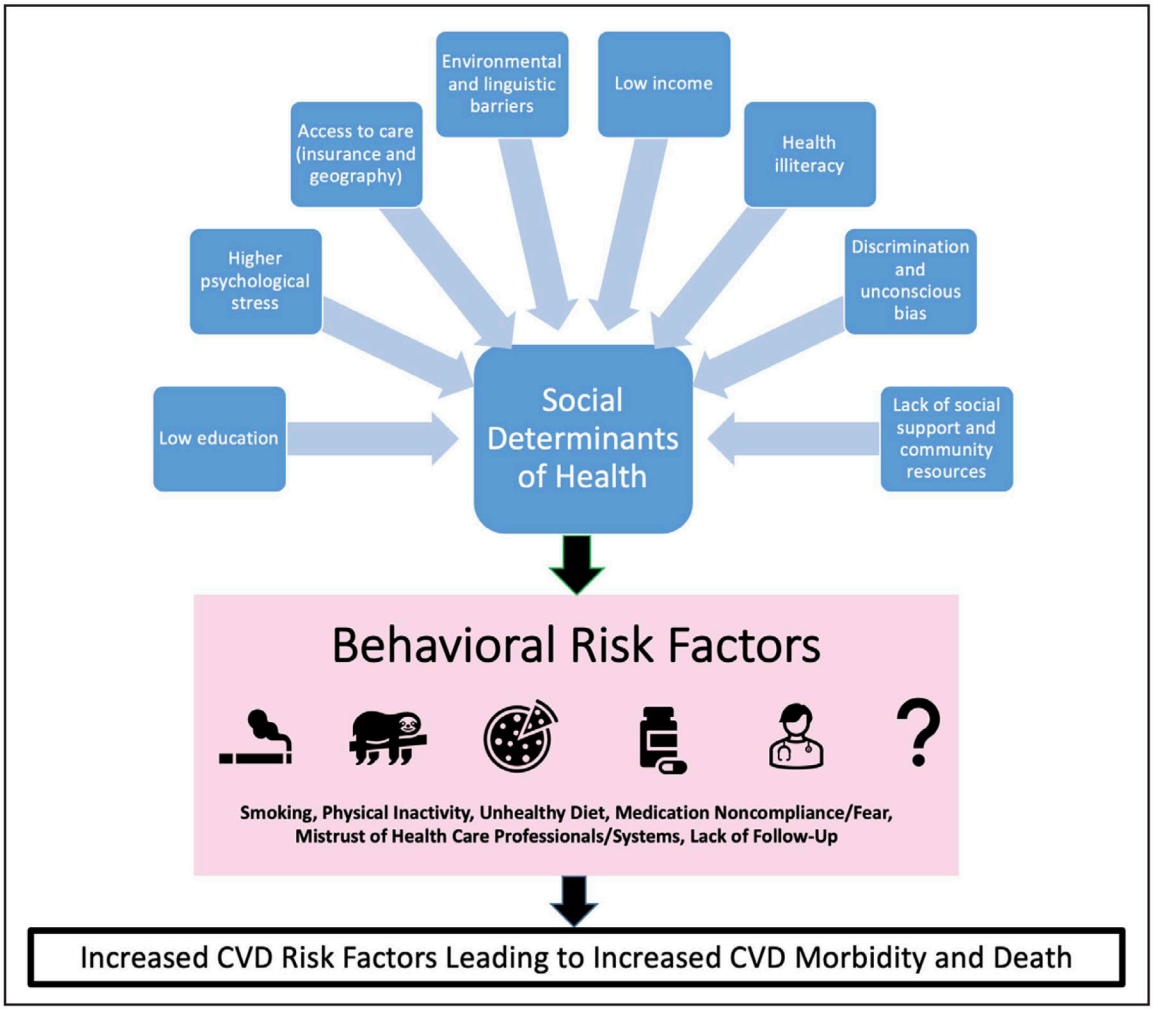

Figure 2. Nonbiological variables to be incorporated into the cardiovascular risk assessment of women.

CVD indicates cardiovascular disease.

Table 1.

Cardiovascular Risk Factor Profiles by Race and Ethnicity

	Hypertension	Dyslipidemia	Diabetes	Obesity	Tobacco use
Non-Hispanic Black women	Highest prevalence in world: 55.3%[1]	High Lp(a) levels associated with higher incidence of ischemic strokes[54]	Prevalence in women >20 y of age: 13.2%[1]	Highest age-adjusted prevalence of obesity and extreme obesity: 55.3% and 15.3%, respectively[1]	Decreasing rates of current smoking[90] More attempts to quit smoking, less successful at quitting[89] Die of smoking-related diseases at higher rates[89]
Hispanic/ Latina women	High prevalence: 40.8% Wide variation across subgroups: Black Hispanic/ Latina women have higher prevalence of hypertension than White Hispanic/Latina women[1]	High prevalence: 37.3% have elevated total cholesterol >200 mg/dL, 26.3% have elevated LDL >130 mg/dL, 12.3% have HDL <40 mg/dL[1]	Heterogeneity across subgroups: highest prevalence in Puerto Rican women (19.4%) and Mexican American women (18.5%)[64]	Higher prevalence in Hispanic/Latina women compared with Hispanic/ Latino men[1]	Decreasing rates of smoking[90] US-born Hispanic/Latino people more likely to smoke cigarettes than foreign-born Hispanic/Latino people[89]
American Indian/Alaska Native women	Prevalence ranges regionally from 25%−41%[46–49]	1 in 5 diagnosed with hyperlipidemia	High prevalence: 19%[38] Varies widely by region: 72% among American Indian women in Arizona[67]	High age-adjusted prevalence: 48.1% *[78] Mean BMI[49] 30–34.9 kg/m^2	Commercial tobacco use higher than in other racial and ethnic populations[92] 1 in 3 currently smoke[88] Prevalence of smoking during pregnancy: 15%[94]
Asian women	Heterogeneity across subgroups: from 30% in Chinese women to 53.2% in Filipina women[52]	Heterogeneity across subgroups: Asian Indian and Filipino people have higher prevalence of low HDL cholesterol and high triglycerides[59–61] Lp(a) higher in Asian Indian people than non-Hispanic White people[62]	Asian Indian people: higher rates of insulin resistance[71] Filipino people: higher prevalence of metabolic syndrome[72] Southeast Asian people: highest prevalence among all Asian subgroups[61]	Have greater proportion of body fat, central adiposity, and visceral fat[21,82]	Decreasing rates of smoking[90] Overall nicotine use lower than in non-Hispanic White people[88]

BMI indicates body mass index; HDL, high-density lipoprotein; LDL, low-density lipoprotein; and Lp(a), lipoprotein(a).

*Sex-specific data not available.

Table 2.

Current Guidelines and Considerations on CVD Preventive Therapies in Women[4,5,128–131]

General guideline recommendations	Sex-specific considerations
BP	
Encourage optimal BP of <120/80 mm Hg through lifestyle approaches BP categorization Normal: SBP <120 mm Hg and DBP <80 mm Hg Elevated: SBP 120–129 mm Hg and DBP <80 mm Hg Stage 1 hypertension: SBP 130–139 mm Hg or DBP 80–89 mm Hg Stage 2 hypertension: SBP ≥40 mm Hg or DBP ≥90 mm Hg BP treatment thresholds Stage 1 hypertension: clinical CVD, diabetes, or ASCVD risk ≥10% Stage 2 hypertension: no history of CVD and ASCVD risk <10%	Use thiazide diuretic as first-line agent in older women for osteoporosis prevention Avoid atenolol, ACE inhibitors, and ARBs during pregnancy Treat hypertension (≥140/90 mm Hg) during pregnancy Evaluate women with preeclampsia within 3–12 mo of delivery and treat cardiovascular risk factors
Lipids	
Encourage optimal lipid levels through lifestyle approaches: LDL cholesterol <100 mg/dL, HDL cholesterol >50 mg/dL, triglycerides <150 mg/dL, and non-HDL cholesterol <130 mg/dL Statin treatment groups: Clinical ASCVD Severe hypercholesterolemia (LDL ≥190 mg/dL) Diabetes Primary prevention: ASCVD risk ≥20% or 7.5%−<20%, depending on presence of risk enhancers	Stop statins 1–2 mo before attempting pregnancy in women at low to moderate risk. Consider continued use of water-soluble statins in very high-risk pregnant women. Statin use while breastfeeding is not recommended. Risk enhancers: history of preeclampsia, premature menopause (<40 y), inflammatory conditions (eg, rheumatoid arthritis or lupus) Consider other APOs to assess risk and to guide counseling: gestational hypertension, gestational diabetes, preterm delivery, delivering small-for-gestational-age infant
Aspirin	
Aspirin in individuals with CVD unless contraindicated Low-dose aspirin can be considered for primary prevention among select adults at high ASCVD risk and low bleeding risk.	Start aspirin at 12 wk of gestation to prevent preeclampsia in women at high risk of preeclampsia and consider in women at moderate risk of preeclampsia. Obtain and document pregnancy history throughout a woman's life course, and identify APOs as ASCVD risk factors.
Smoking cessation	
Recommend smoking cessation and avoidance of environmental tobacco	More directly address weight gain and anxiety management concerns with female smokers reluctant to quit.
Diet	
Diet emphasizing intake of vegetables, fruits, legumes, nuts, whole grains, and fish Diet limiting consumption of saturated fat, cholesterol, sodium, processed meats, refined carbohydrates, and sweetened beverages	
Exercise	
Engage in 150 min/wk of moderate-intensity or 75 min/wk of vigorous-intensity aerobic exercise Additional cardiovascular benefit with 300 min/wk of moderate-intensity or 150 min/wk of vigorous-intensity aerobic exercise	Engage in muscle-strengthening activities ≥2 d/wk
Weight loss/maintenance	

General guideline recommendations	Sex-specific considerations
Maintain or achieve an appropriate body weight through comprehensive lifestyle intervention Higher levels of physical activity recommended (≥300 min/wk) to maintain weight loss	
Environmental exposures	
Assessment of exposure to environmental hazards (eg, living near a highway, unregulated private well for drinking water, house built before 1978). Recommendations based on cardiovascular risk (eg, mask use, air filtration, green spaces) Environmental justice initiatives (eg, renovation of lead water pipes infrastructure, built environment, and green spaces).	Consider lead monitoring during pregnancy and menopause in high-risk populations (eg, child affected at home, old housing)

ACE indicates angiotensin-converting enzyme; APO, adverse pregnancy outcome; ARB, angiotensin receptor blocker; ASCVD, atherosclerotic cardiovascular disease; BMI, body mass index; BP, blood pressure; CVD, cardiovascular disease; DBP, diastolic blood pressure; HDL, high-density lipoprotein; LDL, low-density lipoprotein; and SBP, systolic blood pressure.

Writing Group Disclosures

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Expert witness	Ownership interest	Consultant/ advisory board	Other
Laxmi S. Mehta	The Ohio State University	None	None	None	None	None	None	None
Jennifer H. Mieres	Northwell Health	None	None	None	None	None	398 point 6 *; Atria Academy *	None
Roger S. Blumenthal	Johns Hopkins University	None	None	None	None	None	None	None
Rachel M. Bond	Chandler Regional Medical Center, Dignity Health	None	None	None	None	None	None	None
Rachel-Maria Brown	Northwell Health	None	None	None	None	None	None	None
Amanda M. Fretts	University of Washington	NIH (research supported by an NIH grant to study heart disease in American Indian individuals [Strong Heart Study]) *	None	None	None	None	None	None
Jennifer Lewey	University of Pennsylvania, Perelman School of Medicine Perelman Center for Advanced Medicine	NIH (receives grant funding from NHLBI) †	None	None	None	None	None	None
Gayenell S. Magwood	University of South Carolina	NIH †	None	None	None	None	None	Medical University of South Carolina (professor-unpaid) *; University of South Carolina (endowed professor) †
Ana Navas-Acien	Columbia University	NIH (PI of several grants to evaluate the role of environmental exposures in cardiovascular disease) †	None	None	None	None	None	Columbia University (professor) †
Garima Sharma	Ciccarone Center for the Prevention of Cardiovascular Disease, Johns Hopkins University School of Medicine	None	None	None	None	None	None	None
Gladys P. Velarde	University of Florida-Jacksonville	None	None	None	None	None	None	None
Eugene Yang	University of Washington Medical Center	None	None	None	None	None	None	None

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

*Modest.

†Significant.

Reviewer Disclosures

Reviewer	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Expert witness	Ownership interest	Consultant/advisory board	Other
Anandita Agarwala	Baylor Scott and White Heart Hospital Baylor Plano	None	None	None	None	None	None	None
Fathima A. Cader	Ibrahim Cardiac Hospital & Research Institute (Bangladesh)	None	None	None	None	None	None	None
Elizabeth A. Jackson	University of Alabama at Birmingham	NIH (grant paid to institution)[†]; Amgen (grant paid to institution)[†]; AHRQ (grant paid to institution)[†]	None	None	DeBlase Brown Everly[*]	None	Change Healthcare Technologies[*]; ACCF[*]; AHA (Editorial Board)[*]	UpToDate (royalties)[*]
JoAnn E. Manson	Brigham and Women's Hospital, Harvard Medical School	Mars Edge (Research grant to study cocoa flavanols and health)[†]	None	None	None	None	None	None
Deirdre Mattina	Cleveland Clinic	None	None	None	None	None	None	None
Jessica Pena	Dalio Institute of Cardiovascular Imaging, New York-Presbyterian Hospital and the Weill Cornell Medicine	None	None	None	None	None	None	None
Markella V. Zanni	MGH/Harvard Medical School	None	None	None	None	None	None	None

This table represents the relationships of reviewers that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all reviewers are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition

[*]Modest.

[†]Significant.