

STATE-OF-THE-ART REVIEW

Social Determinants of Cardiovascular Aging



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ABSTRACT

Social determinants of health are major drivers of adverse cardiovascular outcomes throughout the life course. As the population ages, understanding how social determinants influence vascular, myocardial, valvular, and electrophysiologic aging trajectories will be essential to improving cardiovascular outcomes. This review summarizes key frameworks for social determinants of health and cardiovascular aging, then examines social determinants' impacts on cardiovascular aging focusing on behavioral, biological, and health care-related mediators. Specifically, the review highlights race/ethnicity, gender, geographic context, and education as structural determinants with impact starting early in life; followed by food security, digital access, and financial security as intermediary determinants requiring targeted intervention in adulthood; then social connection, transportation access, and homebound status as intermediary determinants with outsized impact in elderhood. The review spotlights patient-, population-, and policy-level interventions for each determinant, as well as key considerations for improving atherosclerosis, heart failure, valvular, and atrial fibrillation outcomes, and priorities for study and intervention. (JACC Adv. 2025;4:102331) © 2025 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

BACKGROUND

The burden of age-associated cardiovascular disease (CVD) is rising as the global population ages.^{1,2} Morbidity and mortality from atherosclerosis, heart failure (HF), degenerative valve disease, and atrial fibrillation (AF) are unevenly distributed across the population, reflecting heterogeneous individual trajectories of cardiovascular aging.^{3,4} Social determinants of health influence cardiovascular risk factors and outcomes throughout the life course and

accelerate cardiovascular aging in socially vulnerable populations.^{5,6} Improving cardiovascular outcomes will therefore require a nuanced understanding of how social determinants of health impact cardiovascular aging and how to deploy preventive medical and lifestyle interventions accordingly.

In this state-of-the-art review, we first summarize contemporary perspectives and frameworks for social determinants of health and cardiovascular aging. We then build on these frameworks summarizing evidence on key social determinants of

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the [Author Center](#).

Manuscript received July 1, 2025; revised manuscript received September 30, 2025, accepted October 8, 2025.

**ABBREVIATIONS
AND ACRONYMS****ASCVD** = atherosclerotic
cardiovascular disease**AF** = atrial fibrillation**CVD** = cardiovascular disease**EP** = electrophysiologic**HF** = heart failure**MRI** = magnetic resonance
imaging**SNAP** = supplemental
nutrition assistance program**VHD** = valvular heart disease**WHO** = World Health
Organization

cardiovascular aging across the life course and corresponding evidence-based patient-, population-, and policy-level interventions. Finally, we share actionable insights on social determinants of cardiovascular aging in key age-associated cardiovascular conditions and consider future directions to promote healthy cardiovascular aging across the population.

**CONCEPTUAL FRAMEWORKS FOR
SOCIAL DETERMINANTS OF HEALTH**

Effectively addressing the role of social determinants of health in cardiovascular aging requires a nuanced, contemporary under-

standing of social determinants writ-large and existing conceptual frameworks for investigation and intervention. Social determinants of health are the nonmedical factors that influence health outcomes. Specifically, the Centers for Disease Control defines social determinants of health as “the conditions in the environments where people are born, live, learn, work, play, worship, and age that affect a wide range of health, functioning, and quality-of-life outcomes and risks.”⁷ Although the link between social context and health has been recognized for generations, the frameworks driving contemporary study and intervention crystalized in the late 1990s-2010s.

LIFE-COURSE APPROACH. First proposed in 1997 by Kuh and Ben-Shlomo,⁸ the life-course approach has been the central to understanding chronic disease epidemiology and heterogeneity in population aging. This approach recognizes the importance of life stage and timing to understand the association between social exposures and health outcomes. Two theoretical perspectives underlie this framework: the developmental perspective that focuses on the outsized impact exposures can have during critical periods for normal development and the structural perspective that focuses on the accumulated biopsychosocial consequences of health-impacting exposures with repeated and differential exposure across life stages.

The life-course approach has been applied to social determinants of health, with key implications for study and intervention.⁵ The cumulative and interactive nature of exposures over the life course necessitates study of multiple exposures and their interactions, as well as consideration of the life-stage timings of exposure and outcome: single-exposure studies with life stage-agnostic binary outcomes are insufficient to inform understanding and intervention. Improved understanding requires

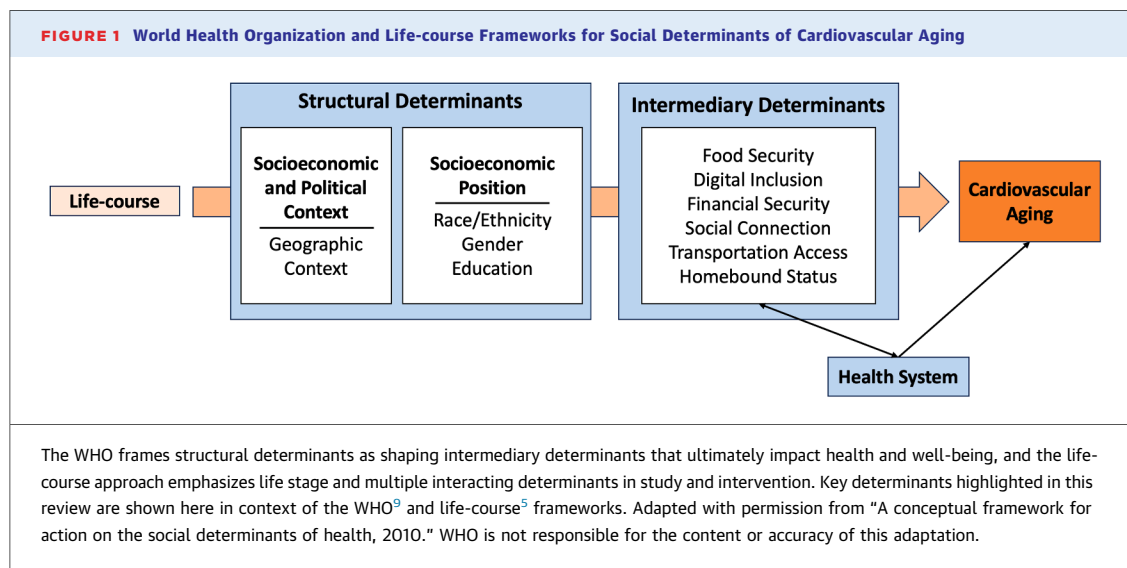
HIGHLIGHTS

- Addressing social determinants of cardiovascular aging is fundamental to improving cardiovascular outcomes in the aging global population.
- Structural determinants, particularly race/ethnicity, gender, geographic context, and education start impacting cardiovascular aging early and require multilevel interventions.
- Intermediary determinants, particularly food and financial security, transportation and digital access, social connection, and homebound status, affect cardiovascular aging in adulthood and elderhood and are modifiable with patient-, population-, and policy-level interventions.
- Further work should use life-course and implementation science approaches to develop tailored multimodal interventions for healthy cardiovascular aging.

epidemiologic studies of the ways social determinants shift health trajectories in childhood, adulthood, and elderhood, as well as mechanistic studies of biological adaptations to social determinants and their manifestations in later life stages. Finally, and most importantly, life-course approaches must be integrated into interventions to optimally address social determinants of health, given the major differences in individual characteristics and community interactions by life-stage.

WORLD HEALTH ORGANIZATION FRAMEWORK. The World Health Organization (WHO) Commission of Social Determinants of Health, led by Michael Marmot who popularized the term in his 1999 book *Social Determinants of Health*, combined the life-course approach with other key theories in the field into the pre-eminent contemporary framework.⁹ Informed by social causation theory, the commission categorizes determinants as “structural” or “intermediary.”

“Structural determinants” include both the socioeconomic and political context that stratifies populations into socioeconomic positions and the positions themselves, including race/ethnicity, gender, education, occupation, and income (Figure 1).⁹ This framing is informed by the work of Williams and Collins¹⁰ who elucidated the structural



mechanisms, for example, macroeconomic, social, and public policies, that stratify access to resources necessary to achieve health. For example, policies ranging from historical redlining to differential community investment have generated and perpetuated residential segregation that shapes housing, education, and employment opportunities that then influence socioeconomic status and health.^{10,11} Structural determinants such as race/ethnicity are, accordingly, social constructs associated with differential health trajectories driven by racism and discrimination in the socioeconomic and political context.^{11,12}

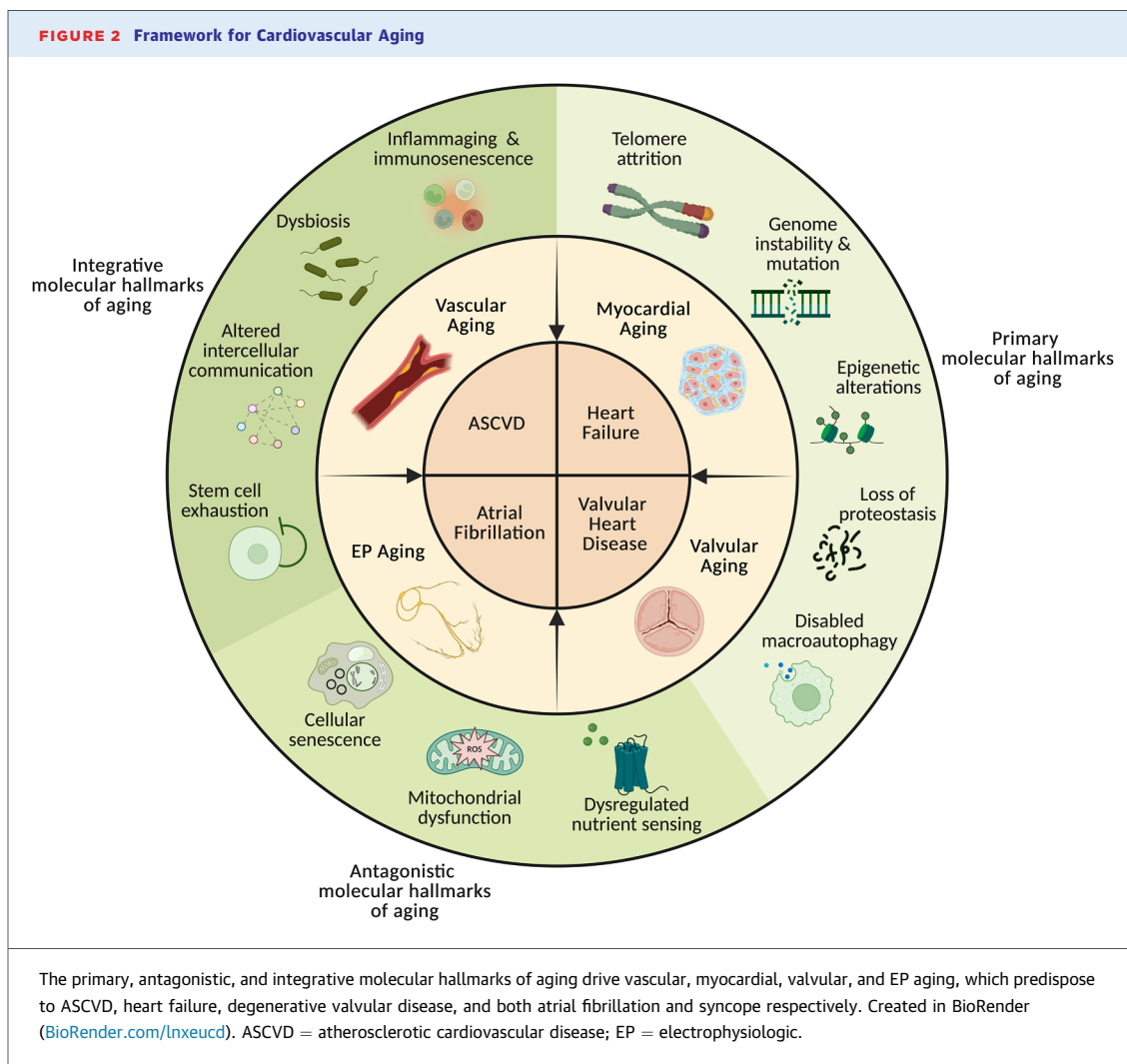
Socioeconomic positions then shape specific “intermediary determinants,” namely material circumstances as well as behavioral, biological, and psychosocial factors that more proximally drive exposure and vulnerability to health-compromising conditions throughout life.⁹ This framework calls attention to the need for interventions to tackle both structural and intermediary determinants on patient, population, and policy levels.

CARDIOVASCULAR AGING

Cardiovascular aging is the gradual process of molecular, cellular, and tissue-level change accrued to the cardiovascular system over the life course that increases the likelihood for, and adverse consequences of, CVD (Figure 2).⁴ It is a multifaceted physiologic process that occurs at varying rates across individuals and populations. These

heterogeneous trajectories are often modifiable; biopsychosocial stressors can accelerate cardiovascular aging, whereas preventive pharmacotherapies and lifestyle changes can slow its progression. In recent decades, Geroscience has broadened the recognized manifestations of cardiovascular aging, ranging from noninvasive imaging parameters to advanced cellular and molecular profiling (Table 1).¹³ These aging processes predispose to age-related CVDs and decrease resilience to stressors once these diseases are present. To inform high-level appraisal of the social determinants of cardiovascular aging, we will highlight key manifestations and consequences of cardiovascular aging across myocardial, valvular, vascular, and electrophysiologic (EP) domains.

MOLECULAR HALLMARKS OF CARDIOVASCULAR AGING. Molecular hallmarks of cardiovascular aging can be categorized as “primary” processes of decline with age and stressors, “antagonistic” maladaptive responses to these changes, and “integrative” hallmarks that precipitate CVD.^{14,15} The “primary” processes are telomere attrition; dysfunctional autophagy (decreased clearing of dysfunctional cytoplasmic components); loss of proteostasis (the balance between protein synthesis, folding, and degradation); genomic instability (mutation accumulation); and epigenetic modifications such as DNA methylation and histone modification.¹⁶ The “antagonistic” maladaptive responses are dysfunction of mitochondria, responsible for bioenergetics, and calcium homeostasis; neurohormonal dysregulation,



including renin-angiotensin-aldosterone system and β -adrenergic pathways; and cellular senescence, meaning, cell cycle arrest driven by oxidative stress and other stressors. The key “integrative” hallmark is the dyadic interplay between inflammaging—chronic immune activation with age, and immunosenescence—immune dysfunction and suppression with age, that can be adaptive when balanced and maladaptive when imbalanced.¹⁷ Stem cell exhaustion, dysbiosis, and altered intercellular communication are the final “integrative” molecular hallmarks that drive cardiovascular aging.^{14,18}

VASCULAR AGING. These primary, antagonistic, and integrative processes drive the 2 primary hallmarks of vascular aging: arterial stiffening and endothelial cell dysfunction. An aging cardiovascular continuum has been described, by which repetitive expansion with each systole over the life

course drives breakdown of elastic lamellae, increased collagen crosslinking, and thickening of the arterial media.¹⁹ These changes increase vascular stiffness and pulse-wave velocity, with reflected pressure waves returning during late systole instead of diastole, with resultant increased afterload and pulse wave extension into the peripheral microvasculature. Endothelial cell dysfunction decreases the effectiveness of the endothelial barrier to the movement of lipids and monocytes across the barrier, increases the likelihood for platelets to adhere to the endothelium, and decreases vasodilator response to endothelial-dependent stressors. These increase the likelihood for the development, progression, and clinical manifestations of atherosclerosis.²⁰

Physical exam changes associated with vascular stiffening include increased systolic pressure, pulse

TABLE 1 Cardiovascular Aging Physiology and Assessment

System	Cellular Processes	Tissue Processes	Noninvasive	Clinical
Vascular aging	Elastic lamellae breakdown Collagen crosslinking Arterial media thickening Endothelial dysfunction	Arterial stiffening Arteriosclerosis Atherosclerosis	↑ Systolic BP ↑ Pulse pressure ↑ ABI ↑ PWV Plaque geometry/inflammation Coronary calcification Carotid IMT Microvascular dysfunction	Hypertension CAD PAD Stroke Vascular dementia Sarcopenia
Myocardial aging	Myocyte attrition and senescence Myocyte hypertrophy Myocardial fibrosis	Ventricular stiffening Ventricular strain Atrial dilation	↑ LV mass ↓ LV volume ↓ GL strain ↑ Native T1 ↑ Extracellular volume ↑ E/A ratio ↑ E/e' ratio ↓ e' velocity ↑ LA volume	HFpEF HFrEF
Valvular aging	Valvular endothelial cell dysfunction Valvular interstitial cell fibroblastic and pro-calcific differentiation	Valvular thickening Valvular stiffening Valvular degeneration Valvular calcification	↑ Leaflet thickness ↑ Leaflet calcification ↑ Annular calcification ↓ Leaflet motion ↑ Pressure gradients ↑ Regurgitation	Aortic stenosis Aortic regurgitation Mitral regurgitation Mitral stenosis Tricuspid regurgitation
Electrophysiologic aging	Altered ion channel expression/function Conduction myocyte apoptosis Sinus node/conduction system fibrosis Decreased beta receptor sensitivity Decreased norepinephrine reuptake Decreased baroreceptor sensitivity Myocyte dysfunction and stretch	Sinus node dysfunction Chronotropic dysfunction Conduction delay Increased atrial ectopy	↓ Intrinsic HR ↓ Maximum HR AV block Bundle branch and fascicular blocks Orthostatic hypotension	Atrial fibrillation Atrial tachycardia Ventricular tachycardia Syncope Sick sinus syndrome Complete heart block

ABI = ankle-brachial index; AV = atrioventricular; BP = blood pressure; CAD = coronary artery disease; GL = global longitudinal; HFpEF = heart failure with preserved ejection fraction; HFrEF = heart failure with reduced ejection fraction; HR = heart rate; IMT = intima-media thickness, coronary artery disease, peripheral artery disease; LA = left atrium; LV = left ventricle; PAD = peripheral artery disease; PWV = pulse wave velocity.

pressure, and ankle-brachial index.¹⁹ Noninvasive markers of vascular aging include structural changes such as coronary artery calcification on computed tomography, geometric changes including increases in aortic dimensions and carotid intima-media thickness, and functional changes such as increased pulse-wave velocity and decreased aortic distensibility.⁴ Premature vascular aging contributes to hypertension and predisposes to atherosclerotic CVD (ASCVD), and microvascular dysfunction promotes vascular dementia and sarcopenia.²¹

MYOCARDIAL AGING. Dysfunctional autophagy and cellular senescence, combined with increased afterload from arterial stiffening, drive the primary processes of myocardial aging: myocyte attrition, cellular hypertrophy, and fibrosis. The aging

myocardium is thereby comprised of a smaller number of hypertrophied, senescent cardiomyocytes that release paracrine signals to neighboring fibroblasts, immune cells, and endothelial cells, promoting fibrosis, inflammation, and cellular dysfunction.²² These processes impair myocardial systolic coordination and diastolic relaxation, increasing left ventricular pressure and volume loads. The resultant constellation of atrial dilation, myocyte hypertrophy and fibrosis, and stimulation of stretch-activated ion channels facilitates arrhythmogenesis.²³

Echocardiography and cardiac magnetic resonance imaging (MRI) allow advanced noninvasive assessment of changes associated with myocardial aging. On echocardiography, declining diastolic function, global longitudinal strain, and ventricular volumes,

and increasing left atrial volume can reflect myocardial aging, as can increased myocardial native T1 and extracellular volume on MRI.²⁴⁻²⁹ Clinically, ventricular myocardial aging can drive HF, whereas atrial myocardial aging predisposes to AF.³⁰

VALVULAR AGING. Valvular endothelial and interstitial cells are also impacted by the molecular hallmarks of cardiovascular aging, mechanical stress imposed by myocardial aging, and increased pulse pressure associated with vascular aging.^{31,32} Aging valvular endothelial cells exhibit decreased proliferation and self-repair as well as disrupted endothelial to interstitial cell communication, which drives proliferation of valvular interstitial cells, increased elastic and collagen fiber production, and differentiation to procalcific cells.³¹⁻³³ The resultant thickening, stiffening, degeneration, and calcification are readily detectable by echocardiography, computed tomography, and MRI, and underlie a broad spectrum of valve diseases, the most common being aortic stenosis, mitral regurgitation, and aortic regurgitation.³⁴

ELECTROPHYSIOLOGIC AGING. The aforementioned primary, antagonistic, and integrative molecular processes affect the sinus node, conduction system, and autonomic nervous system and drive the EP consequences of aging.³⁵ With age and chronic hemodynamic stress, sinoatrial node and conduction system myocytes exhibit decreased expression and function of several ion channels, including voltage-gated calcium channels and channels responsible for funny current.³⁶ Simultaneously, these myocytes undergo age-related apoptosis, hypertrophy, and fibrotic extracellular matrix remodeling. These changes, as well as decreased density and function of beta-adrenergic receptors and decreased norepinephrine uptake, result in decreased resting and maximum heart rates, as well as atrioventricular and bundle branch blocks.³⁵⁻³⁷ This multifactorial chronotropic incompetence, paired with decreased alpha adrenergic receptor and baroreceptor density and sensitivity, increases the risk of orthostatic hypotension and syncope.³⁸ Finally, these EP changes along with the previously described processes of myocardial aging such as fibrosis, apoptosis, oxidative damage, inflammation, and stretch predispose to atrial and ventricular arrhythmias.^{35,39}

FRAILTY: INTEGRATED CARDIOVASCULAR AGING. Many of the molecular hallmarks of cardiovascular aging are shared with frailty, and vascular, myocardial, valvular, and EP aging all increases the risk of frailty.^{30,39-42} Frailty is a manifestation of biological aging, predominantly assessed as a physical

phenotype or deficit accumulation index.^{43,44} Among the 60-plus validated tools to measure frailty, the Fried frailty phenotype is considered the reference standard, and the essential frailty toolset, FRAIL scale, and clinical frailty scale are streamlined tools validated in the cardiovascular patient population that can be efficiently employed in busy clinical settings, guiding tailored implementation of frailty-directed and cardiovascular interventions.⁴⁵

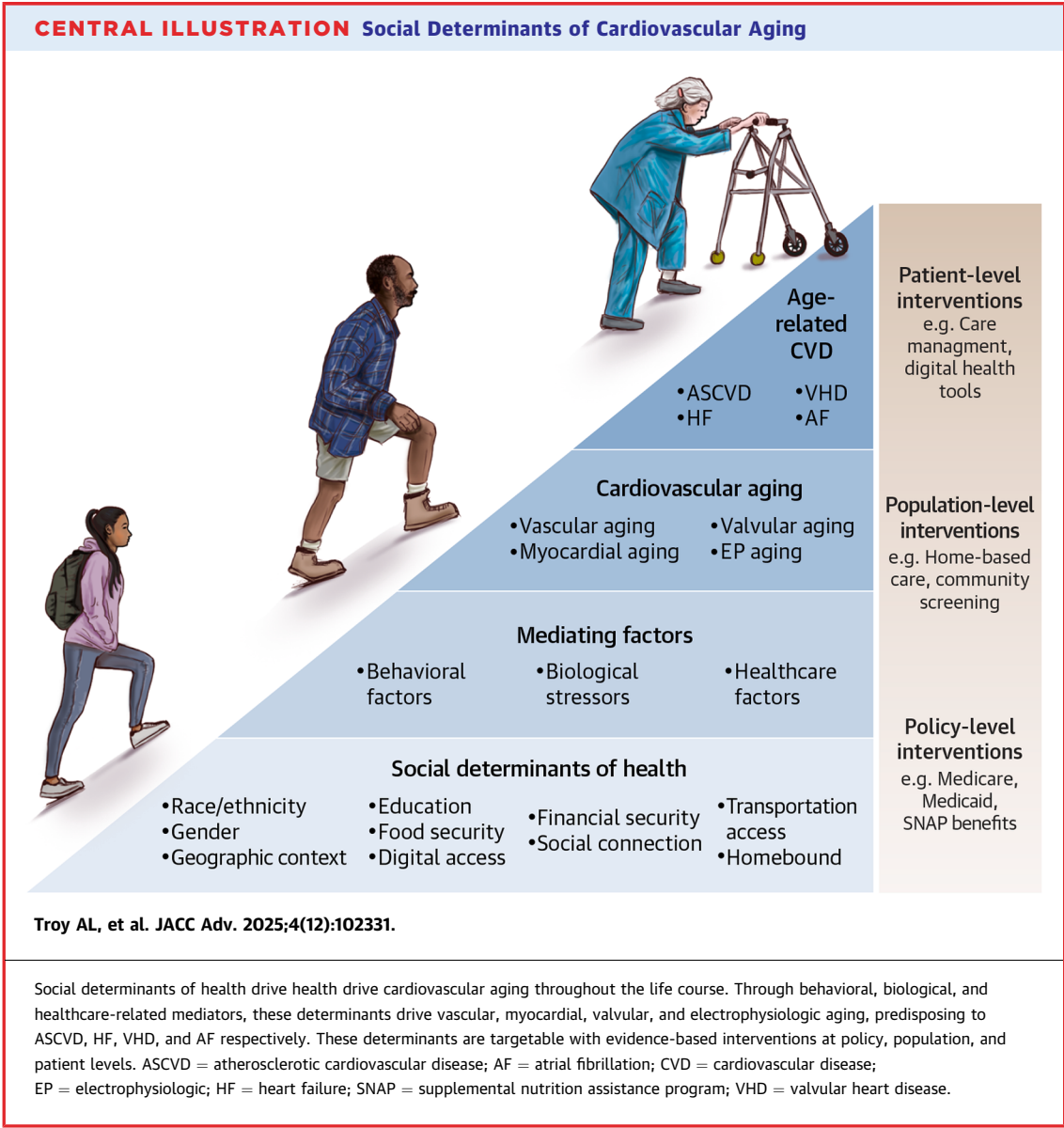
INTEGRATED FRAMEWORK FOR SOCIAL DETERMINANTS OF CARDIOVASCULAR AGING. In this state-of-the-art review, we advocate for adoption of the WHO framework with an emphasis on life-course perspectives to understand the social determinants of cardiovascular aging (**Central Illustration**). We will consider the impacts of multiple interacting determinants (**Table 2**), their behavioral-, biological-, and health care-related mechanisms of impact at critical periods across life stages, and highlight patient-, population-, and policy-level interventions to address their health effects (**Table 3**).

Specifically, we performed a scoping review of studies examining associations between key structural and intermediary social determinants of health and measures of cardiovascular aging, with priority given to molecular and imaging biomarkers followed by clinical manifestations of cardiovascular aging, and with particular emphasis on mechanisms and interventions. We will first discuss the key structural determinants, race/ethnicity, gender, geographic context, and education that start impacting social position and health trajectories in childhood. We will then address key intermediary determinants with impacts starting in early life and adulthood, namely food security, financial security, and digital access. Finally, we will explore key intermediary determinants with outsized impact during elderhood: social connection, transportation access, and homebound status.

STRUCTURAL DETERMINANTS: EARLY LIFE

Structural determinants, particularly race/ethnicity, gender, geographic context, and education have powerful effects on cardiovascular aging trajectories starting in early life and accumulating over the life course.

RACE/ETHNICITY. Cardiovascular aging trajectories begin to diverge by race and ethnicity in early development and childhood.^{12,46} Race and ethnicity are social constructs reflecting socioeconomic positions driven by macroeconomic policies and social context that impact access to health-promoting resources.^{10,11}



Multiple studies have demonstrated the association of race and ethnicity with divergent cardiovascular aging trajectories. Although social determinants differentially drive adverse outcomes across racial/ethnic groups, with recent evidence of accelerated myocardial aging across Asian, Hispanic, and Black American populations, cardiovascular aging trajectories remain underphenotyped in other minoritized populations relative to Black Americans.^{12,47} Vascular stiffness develops earlier in Black children compared to White children, driving

differences in hypertension and ankle brachial index in adulthood after adjusting for socioeconomic status.^{46,48} This accelerated vascular aging drives myocardial aging, with higher rates of systolic and diastolic cardiac dysfunction seen in Black adults.⁴⁹ This also leads to divergence in integrated cardiovascular aging, including race-based differences in “weathering”, or accelerated biologic aging, with Black individuals weathering 6 years faster than White individuals, and discrimination increasing the risk of frailty.^{50,51}

TABLE 2 Social Determinants and Cardiovascular Aging

	Vascular	Myocardial	Valvular	EP	Frailty
Race/ethnicity	Kruger et al, 2021	Kishi et al, 2015	-	Rodriguez et al, 2015	Mandelblatt et al, 2023
Gender	Böhm et al, 2009	Gori et al, 2014	Myasoedova et al, 2020	Westerman and Wenger et al, 2019	Muka et al, 2016
Education	Liu et al 2023	Yan et al 2006	-	Lunde et al 2020	Graf et al 2024
Geographic context	Liu et al, 2025	Turecamo et al, 2023	Tan et al, 2024	Essien et al, 2022	Marinacci et al, 2025
Food security	Liu et al, 2021	Gondi et al, 2022	-	Markson et al, 2023	Wennberg et al, 2024
Digital access	Troy et al, 2023	-	-	-	-
Financial security	Hamad et al, 2020	Mathews and Brewer 2022	-	Misialek et al, 2014	Hanlon et al, 2024
Social connection	Liang et al, 2023	Cene CW et al, 2022	-	Qiao et al, 2025	Davies et al, 2021
Transportation access	Remillard et al, 2022	Acquah et al, 2022	-	-	Fukuei et al, 2024
Homebound status	Qiu et al, 2010	Ornstein et al, 2015	-	-	Leff et al, 2024

These accumulated changes decrease cardiovascular longevity, with 7.9 million excess years of potential life lost between 2020 and 2022 in Black Americans relative to White Americans due to ischemic heart disease.^{52,53} Racism directly and indirectly impacts cardiovascular aging through toxic stress, which increases inflammatory markers and impacts DNA methylation in Black individuals, accelerating atherosclerosis and neurocognitive decline.⁵⁴⁻⁵⁶ Limited resources for preventive health behaviors in Black communities has driven low Life's Simple 7 scores in Black adults across the United States, with unequal access to cardiologists further impeding healthy cardiovascular aging.^{57,58}

A multipronged approach is needed to improve cardiovascular aging across racial/ethnic groups.⁵⁹ Evidence-based patient-level interventions include culturally sensitive lifestyle change programs like Black Impact, that improves Life's Simple 7 scores in Black men.^{58,60} On a population level, a blood pressure screening and pharmacist intervention in Black barbershops improves blood pressure compared with usual care.⁵⁷ Moreover, improving clinical trial enrollment of understudied racial and ethnic groups and increasing physician and researcher representation from these communities are vital to improve uptake of preventive pharmacotherapies in diverse populations.^{61,62} Ultimately, policy-level support of state- and federally-funded health centers and other resources in under-resourced communities, and continued Medicaid support are necessary for healthy cardiovascular aging across race and ethnicity.⁵⁹

GENDER. There are substantial gender-based differences in cardiovascular aging that start early in life.⁶³ Gender is a social construct with health impacts related to socioeconomic and political context separate from biological differences in sex.⁶³ Recognizing that historic underinvestigation of women's

cardiovascular health has contributed to gender-based outcome differences, we will first describe sex-based cardiovascular aging trajectories and then address gender-related mechanisms and opportunities.⁶⁴

Sex-based differences in vascular aging, specifically intima-media thickness, are seen starting in early childhood.⁶⁵ The vasculature of females also exhibits greater mineralocorticoid receptor expression, which has been associated with endothelial and microvascular dysfunction.⁶⁶ Heterogeneity in vascular aging drives disparate myocardial aging, with females more likely to have interstitial myocardial fibrosis, concentric remodeling, and HF with preserved ejection fraction.⁶⁷ In addition, valvular aging differs by sex, with females more likely to have mitral valve disease due to connective tissue disorders and men more likely to have aortic valve disease.⁴ These differences in cardiac remodeling and valvular disease also manifest in disparate EP aging, with men more likely to have AF and women having higher rates of stroke due to arrhythmia.⁶⁸ Estrogen has a protective effect before menopause, and premature cardiovascular aging is more common in males due to shorter telomeres and higher rates of CVD earlier in life.⁶⁹ Later, postmenopause, decreased estrogen increases oxidative stress and, thereby, risk of frailty in women.⁷⁰

Gender itself also impacts cardiovascular aging. Women are less likely to receive intensive risk factor modification and treatment when presenting with CVD.⁷¹ For example, women have been labeled to have "atypical" chest pain leading to underdiagnosis, undertreatment, and adverse outcomes because the clinical presentation of CVD has historically been defined through an androcentric lens.⁷² The current paradigm has broadened to emphasize myocardial infarction with nonobstructive coronary arteries, spontaneous coronary artery dissection, and

TABLE 3 Social Determinant-Directed Interventions to Improve Cardiovascular Aging			
	Patient	Population Health	Policy
Race/ethnicity	Community-based lifestyle change program Joseph et al, 2022	Hypertension screening and management in Black barber shops Victor et al, 2018	Support government-funded health centers in under-resourced communities Albert et al, 2024
Gender	Gender-specific cardiovascular risk factor questionnaires Nguyen et al, 2024	Increasing representation of women cardiologists Fatunde et al, 2025	Support inclusive and targeted clinical trials across sex Sullivan et al, 2021
Education	Poly pill use to improve adherence Muñoz D et al, 2019	Community-based risk-factor management classes, workshops Hassen et al, 2022	Vocational training to improve employment, access Flores 2022
Geographic context	Telehealth expansion Butzner and Cuffee 2021	Community gardens Litt et al, 2023	Regional planning and transfer networks Greenwood-Ericksen et al, 2020
Food security	Food prescriptions Hager et al, 2023	Meals on Wheels Mozaffarian et al, 2024	SNAP benefit expansions Troy et al, 2024
Digital access	Community partnerships for mobile health tools Johnson et al, 2023	Flexible cardiac rehabilitation models Mathews and Brewer 2021	Universal broadband and telehealth access Troy et al, 2023
Financial security	Multidimensional poverty index for risk assessment Butt et al, 2024	Capping out of pocket drug costs Narasimmaraj et al, 2023	Medicaid expansion Wadhwa et al, 2018
Social connection	Digital or in-person CBT Berkman et al, 2003	Caregiver support programs Rippon et al, 2024	Accessible, age-friendly community infrastructure Welch et al, 2024
Transportation access	Combined care coordination and transportation services Onyekere et al, 2016	Public transport vouchers, rural nonemergency medical transportation Lin and Cui, 2021	Integration of stakeholder input into transportation and infrastructure policy Summers et al, 2020
Homebound status	Home-based telemedicine Lindenfeld et al, 2023	Home-based primary care Zimbroff et al, 2021	Improved Medicare coverage for long-term services and supports DeCherrie et al, 2021
CBT = cognitive behavioral therapy.			

microvascular disease as major drivers of ischemic heart disease in women.⁷³ In men, on the other hand, higher male gender expressivity in adolescence is associated with increased hypertension and diabetes in adulthood.⁷⁴ There is also emerging evidence of unhealthy cardiovascular aging in sexual and gender minority populations, with mechanisms including impaired access to preventive care, minority stress, and hormonal therapies, although further investigation is needed.⁷⁵

There are numerous targets for intervention to reduce gender-based differences in cardiovascular aging trajectories across the life course.⁷⁶ On the patient level, increased education about CVD in women, development of gender-aware risk scores that include gynecologic, obstetric, and hormonal treatment histories, as well as cardiologists that focus specifically on women’s health can help optimize management of individual patient risk factors.⁷⁷ On a population level, efforts to optimize cardiovascular prevention in women should include increasing representation of women in cardiology, who currently comprise only 15% of the workforce, including systemic solutions such as mentorship programs, parental leave, and pumping breaks.⁷⁸ Innovative clinical research is needed, such as the

sensitivity analysis of the PARAGON-HF (Prospective Comparison of Angiotensin Receptor-Nephrilysin Inhibitor with Angiotensin-Receptor Blockers Global Outcomes in HF with Preserved Ejection Fraction) trial demonstrating improved sacubitril-valsartan-driven outcomes in women compared with men, as are guidelines that incorporate this evidence.^{79,80} This will require optimal gender representation in clinical trials across life stages, as delineated in the 2024 Food and Drug Administration draft guidance about diversity action plans for clinical trials that is at risk of incomplete implementation.⁸¹ On a policy level, future cardiac care models should include reducing gender-based outcome gaps as a metric for physicians and health systems to spur care delivery innovations that address unique needs of cardiovascular patients across gender.⁸²

GEOGRAPHIC CONTEXT. Geographic context, including neighborhood characteristics and rural vs urban status, interacts directly with many other determinants including race/ethnicity, gender, education, and food security, contributing to variations in cardiovascular aging trajectories through behavioral, biological, and health care-related mechanisms. Rurality and county-level differences in cardiovascular aging are largely driven by differences in access to care as well as

income, education, food availability, and housing stability that impact successful navigation of developmental milestones.^{83,84} Vascular aging has been accelerating in rural areas, manifesting in increased hypertension prevalence.⁸⁵ Geography also interacts with race and income to drive premature myocardial aging, with Black men in rural areas having compounded increased risk of developing HF.⁸⁶ Valvular and EP aging are also accelerated in rural residents due to cardiovascular risk factor exposure across the life course.^{87,88} These clinical risk factors drive premature cardiovascular mortality in rural areas.^{89,90} Lower access to primary and specialty cardiovascular care impacts preventive care and medical risk factor management needed for healthy cardiovascular aging.⁹¹⁻⁹³

Neighborhood-level factors are also associated with accelerated cardiovascular aging and weathering. In early life, neighborhood factors such as excessive noise, residing in a food desert, limited greenspace access, and low neighborhood walkability mediate premature vascular aging.⁹⁴ Environmental pollution is also associated with endothelial dysfunction, oxidative stress, and cell signaling dysfunction that can accelerate atherogenesis resulting in a higher burden of CVD.^{95,96} Racial residential segregation also promotes differences in cardiovascular aging between Black vs White residents.⁹⁷ Neighborhood violence and perceptions of violence increase psychological distress, predict development of metabolic syndrome, and increase inflammaging, with a particularly pronounced effect on women.⁹⁸ In elderhood, neighborhood socioeconomic disadvantage and individuals' senses of security and neighborhood belonging are associated with frailty as well as accelerated vascular and myocardial aging.⁹⁹⁻¹⁰¹

Achieving equity in cardiovascular aging by geographic context requires diverse interventions. On a patient level, increasing access to care for rural populations via telehealth has improved outcomes and the use of appropriate therapies, although such strategies risk widening the digital divide without concurrent broadband expansion.^{102,103} On a population level, infrastructure programs such as community gardens improve nutrition, access to food, and social cohesion.¹⁰⁴ Policy interventions such as securing the financial future of critical access hospitals, incentivizing physicians work in rural areas, and creating regional planning networks to link community facilities with tertiary care are needed to improve cardiovascular aging equity across geographic contexts.^{91,105,106}

EDUCATION. Educational attainment impacts cardiovascular aging trajectories through health care access, health literacy, and health behaviors throughout the life course. Individuals with lower educational attainment have increased risk of coronary artery disease, stroke, HF, and AF.¹⁰⁷⁻¹⁰⁹ These differences start early, with 1 study showing that lower educational attainment accelerates atherosclerosis progression in youth and, by adulthood, drives disparate rates of CVD.¹¹⁰ Among patients with acute myocardial infarction (MI), education is an independent risk factor for worse short- and long-term outcomes and lower medication adherence.¹¹¹ In adulthood and elderhood, differences in vascular risk factors by education can drive vascular dementia, with up to 44% increased dementia risk in those with lower educational attainment in a dose-response pattern.¹¹² Ultimately, lower educational attainment is associated with accelerated biologic aging, conferring decreased lifespan and healthspan.¹¹³

Divergent cardiovascular aging trajectories by educational attainment are driven by behavioral and health care-related differences connected to education itself and its socioeconomic consequences. Behaviorally, higher levels of education are associated with lower smoking rates, improved cholesterol management, and higher rates of physical activity.¹¹⁴ In addition, lower educational attainment is associated with lower medication adherence in older patients with polypharmacy and multimorbidity.¹¹⁵ Educational attainment is also associated with employment, health literacy, and health care access for patients with CVD.¹¹⁶

Educational interventions are needed throughout the life course to improve cardiovascular outcomes. On a patient level, assessing educational attainment and personalizing resources and decision aids accordingly during shared decision-making and counseling may improve preventive behaviors and care plan adherence. Single-pill combination use also improves medication adherence, a key mediator of divergent cardiovascular aging trajectories by education.¹¹⁷ Community-based interventions proven to improve CVD knowledge such as participatory workshops and group meetings, diabetes self-management classes, and mobile messaging interventions are needed to improve health behaviors and adherence.¹¹⁸ On a policy level, improving educational equity will require multiple approaches, from metrics that encourage interventions for populations with lower educational attainment, aligning school schedules with working days, incorporating

vocational training in high school to improve graduation rates, and improving teacher retainment given teachers' outsized early impact on health literacy.¹¹⁹

INTERMEDIARY DETERMINANTS: ADULTHOOD

In adulthood, the impacts of intermediary determinants like food security, digital access, and financial security increase along with autonomy in managing cardiovascular risk factors, necessitating targeted health-related social needs interventions to promote healthy cardiovascular aging.

FOOD SECURITY. Access to healthy food is one of the most important means of promoting healthy cardiovascular aging. Food insecurity, defined as having limited or uncertain access to adequate nutritious food needed for an active and healthy life, accelerates aging via multiple biologic pathways including allostatic load: wear and tear associated with repeated stress response activation.¹²⁰ Food insecurity increases risk of obesity, hypertension, and high predicted 10-year ASCVD risk, reflecting accelerated vascular aging.^{121,122} Food insecurity is associated both with increased mortality for patients with HF and with frailty in Medicare beneficiaries, reflecting myocardial and integrated cardiovascular aging, respectively.^{123,124} Malnutrition has also been associated with EP aging and cardiac cachexia decreases salutatory cytokine secretion, predisposing to AF.¹²⁵ Living in a food desert, an exposure at the intersection of food insecurity and geographic context, is associated with accelerated vascular aging: increased arterial stiffness, oxidative stress, inflammation, and 10-year ASCVD risk.¹²⁶

Food insecurity promotes consumption of foods that are less nutrient-dense, higher in fat and sodium, and more processed.¹²⁷ In addition, families may have to choose food over medicine due to financial scarcity, driving decreased medication adherence among the food insecure. Food insecurity is also associated with depression and stress, activating the sympathetic nervous symptom, impacting glucose and lipid levels, and accelerating vascular and EP aging, which may partially explain two-fold higher rates of food insecurity in patients with vs without CVD.¹²⁸ Ultraprocessed foods in particular decrease longevity, partially via inflammatory pathway modulation.¹²⁹

Targeted approaches are required to address food insecurity. Patient-level interventions, such as produce prescriptions and paired food insecurity screening and resource referrals can improve food security and cardiometabolic health.¹³⁰ On

population and policy levels, programs across the life course such as Supplemental Nutrition Assistance Program (SNAP), WIC, and Meals on Wheels can be helpful. For example, SNAP benefit expansions during the COVID-19 pandemic improved food insecurity, particularly among individuals with hypertension, hyperlipidemia, and diabetes, with 1 model projecting that subsidizing fruit and vegetables by 30% for SNAP participants could prevent >35,000 CVD deaths annually.¹³¹⁻¹³³ Numerous additional policies have been proposed to promote healthy cardiovascular aging, including improving nutrition standards for school meals, refining food labels, and strengthening regulatory standards around food content.¹³⁴

DIGITAL ACCESS. Bridging the digital divide is vital to improving access to cardiovascular care and equity in cardiovascular aging. Digital access has health implications across the life course and is especially helpful in maintaining social connectedness, access to resources, and societal engagement for older adults, all of which are associated with healthy cardiovascular aging. Broadband access in particular has been studied as a social determinant of health and is associated with cardioprotective resources such as employment and education.¹³⁵ On the county-level, low broadband access is associated with accelerated vascular aging, specifically premature coronary artery disease, stroke, and cardiovascular mortality, driven by higher age-adjusted prevalence of hypertension, hyperlipidemia, diabetes, smoking, and obesity, as well as fewer cardiologists and primary care physicians per capita.¹⁰² In addition, there has been increased focus on apps and other mobile technologies that help optimize cardiovascular health, but without expanded digital access, these resources remain underutilized by target populations.¹³⁶

Multiple strategies have been explored leveraging digital access to promote healthy cardiovascular aging. On a patient level, remote patient monitoring interventions have gained traction, with novel approaches for optimal efficacy and inclusion currently under investigation.^{137,138} On a population level, flexible cardiac rehabilitation models, including virtual, home-based, and community-based programs, can improve access and facilitate equitable adherence to guideline-directed therapies.^{139,140} Delivering targeted digital tools via community-based programs can help improve efficacy and uptake of these programs.¹⁴¹ On a policy level, universal access to both broadband and internet-capable devices is required for the aforementioned interventions to narrow

rather than widen the digital divide. However, despite recent federal funding to improve broadband access via the broadband equity, access, and deployment program, many patients remain with unmet digital needs, and this program's impact remains to be seen.¹⁴²

FINANCIAL SECURITY. Low income and low financial security are associated with cardiovascular risk factor development and premature cardiovascular aging. Although studies have shown that childhood low financial security leads to increased cardiovascular events in adulthood, the impact of financial security continues to accumulate throughout the life course, accelerating vascular, myocardial, and EP aging as well as frailty.¹⁴³⁻¹⁴⁷ Financial status impacts not only access to cardiovascular clinicians but also quality and quantity of care due to the high costs of cardiovascular services. Household family income has a dose-response relationship with cardiovascular aging and longevity, with a \$10,000 positive change in neighborhood median income increasing longevity by 10%.^{148,149} Financial toxicity associated with CVD management and medications is high.¹⁵⁰ In addition, financial status may impact care provided, with 1 study showing residents in a low-income area being less likely to receive coronary angiography after a ST-segment elevation myocardial infarction due to perceptions of medication adherence.¹⁵¹ Intergenerational impact of financial security has been observed, with low income in adulthood perpetuating cardiovascular risk across generations.¹⁵² Financial savings throughout adulthood also have important implications in elderhood, with evidence that economic benefits derived from the social security program improve health outcomes in older adults.¹⁵³

Low financial security may impact cardiovascular aging through multiple mechanisms. Biologically, it is associated with increased stress and sympathetic nervous system activation, which accelerates molecular hallmarks of cardiovascular aging.¹⁵⁴ Financial security also serves as an intermediary determinant driven by upstream structural determinants such as race/ethnicity, geography, education, and employment, and its impacts on cardiovascular aging are partially mediated downstream by food security, digital access, and social connection.

Multilevel policy approaches have been considered to ameliorate the impact of low financial security on cardiovascular outcomes. On a patient level, financial security screening during visits can support adherence by enabling cost-conscious prescribing and insurance navigation, with prior work

demonstrating utility of a multidimensional poverty index in cardiovascular risk assessment.¹⁵⁵⁻¹⁵⁷ Notably, cash transfers to low-income adults have been shown to improve stress and food security, but there is limited evidence that they improve physical health.¹⁵⁸ On population and policy levels, ensuring continued access to social programs with proven cardiovascular benefit such as Medicare, Medicaid, and Social Security is important to prevent exacerbation of income-based differences in cardiovascular aging trajectories.¹⁵⁹⁻¹⁶¹ In addition, legislation to reduce prescription drug spending, including capping out-of-pocket drug costs, has been projected to yield significant patient savings, with further work needed to differentiate the impact of prescription caps by household income.^{162,163}

INTERMEDIARY DETERMINANTS: ELDERHOOD

Although much of the cardiovascular aging trajectory has been set by the time adults reach elderhood, intermediary determinants, particularly social connection, transportation access, and homebound status, can make the difference between accelerated cardiovascular aging and curtailed longevity vs healthy aging in place.

SOCIAL CONNECTION. One in four Americans over age 65 experience social isolation.¹⁶⁴ There is abundant evidence that social isolation negatively impacts vascular aging and mortality in older adults.¹⁶⁵ Among 19,360 diabetic individuals in the U.K. Biobank, the most socially isolated had almost 2-fold increased risk of fatal MI or stroke, 36% higher risk of cardiovascular mortality, and 33% higher risk of all-cause mortality over 13 years median follow-up.¹⁶⁶ Also in the United Kingdom, social isolation is associated with higher incidence and worse prognosis of AF and increased risk of frailty, reflecting EP and integrated cardiovascular aging.^{167,168} Among 9,573 older adults in Copenhagen, participants with ≥ 3 intimate contacts with family, colleagues, or friends had a 17% lower risk of mortality and 18% lower risk of ischemic heart disease after adjustment for age, gender, and atherosclerotic risk factors.¹⁶⁹ And, in an Australian cohort of 19,114 healthy older adults, social isolation and low social support were associated with 66% increased incidence of CVD and 2-fold increased risk of mortality.¹⁷⁰

Moreover, social connection and gender interact, with social isolation particularly accelerating cardiovascular aging in women. In the Women's Initiative Extension Study II, older postmenopausal women with social isolation had 5% increased risk of incident CVD, with the most socially isolated having

13% to 27% higher risk of CVD than the least isolated.¹⁷¹ More specifically, social isolation has been associated with a 23% higher risk of HF hospitalization and 12% increased risk of coronary heart disease in women.^{172,173}

Social connection's impact on cardiovascular aging is mediated by behavioral factors such as physical activity and nutrition, psychological stressors, and adherence to medical recommendations.^{174,175} These mediators drive hyperactivation of the hypothalamic-pituitary-adrenocortical axis and sympathetic nervous system as well as glycemic dysregulation that accelerate molecular hallmarks of cardiovascular aging including inflammaging, oxidative stress, epigenetic modification, and neurohormonal dysregulation.¹⁷⁵⁻¹⁷⁸ Social isolation is associated with increased systolic and diastolic blood pressures, and loneliness with decreased heart rate variability, markers of vascular and EP aging, respectively.^{177,179,180}

Multifaceted interventions are required to target social isolation and its acceleration of cardiovascular aging. As part of its "Decade of Healthy Aging" initiative, the WHO supported development of evidence and gap maps for interventions to reduce social isolation.^{181,182} Although high-quality evidence is limited, commonly studied patient-level interventions include cognitive behavioral therapy, social skills trainings, group activities, and digital interventions to support interactions with family and friends. In the post-MI population, the ENRICH (Enhancing Recovery in Coronary Heart Disease Patients) randomized trial found that cognitive behavioral therapy reduced depression and social isolation scores at 6 months without a significant difference in death or recurrent MI at a mean of 29 months.¹⁸³ Nonrandomized studies supporting holistic approaches ranging from community-engaged music and arts to yoga and tai chi, from social networking sites and video games to exercise and pet therapy are promising, with several now undergoing randomized controlled trials.^{181,182,184,185} One example: pet therapy reduces blood pressure in nursing home residents, and pet ownership is associated with increased survival in patients with CVD, possibly assuaging cardiovascular impacts of social isolation via downregulation of sympathetic and upregulation of parasympathetic pathways, as well as oxytocin, dopamine, and endorphin release.^{186,187} On population and policy levels, increased state and federal support of caregiver support programs, area agencies on aging, and age-friendly infrastructure will be essential in coming decades.^{182,186-189}

TRANSPORTATION ACCESS. Over 5 million Americans delay medical care annually due to a lack of available transportation.¹⁹⁰ Among U.S. adults with ASCVD, approximately 5% delay care due to transportation barriers annually, with a higher risk in the low-income and Medicaid-insured populations. Transportation access has been associated with accelerated aging among Medicare beneficiaries with CVD, with those reporting a lack of transportation being 3 times as likely to have disability.¹⁹¹ Conversely, among individuals unable to engage with activities due to transportation disadvantage, hypertension and diabetes are highly prevalent: 77% and 39%, respectively.¹⁹² Specifically, the TRACE-CORE (Transitions, Risks, and Actions in Coronary Events Center for Outcomes Research and Education) study of acute coronary syndrome survivors assessed transportation barriers, financial barriers, and lack of a usual source of care, and found patients with at least 2 of those barriers had decreased health-related quality of life.¹⁹³ Transportation status, specifically nondriving status and public transportation nonuse, is also associated with premature frailty.¹⁹⁴

Transportation disadvantage is a powerful intermediary determinant of cardiovascular aging, as it can directly delay or defer indicated cardiovascular tests and treatments, allowing vascular and other aging processes to progress unchecked.¹⁹⁵ It is also a key mediator for structural determinants, given demonstrated differences in transportation access by race/ethnicity, gender, and rurality.^{196,197} Its impact is also mediated by other intermediary determinants, as transportation disadvantage limits food security and social connection and promotes homebound status.¹⁹² In elderhood, mobility and sensory impairment, financial limitations of fixed incomes, challenges with public transportation access, and insufficiently accessible infrastructure (eg, under-maintained sidewalks, ramps, and accessible bus stops) also drive transportation disadvantage, accelerating biological and cardiovascular aging.¹⁹⁸⁻²⁰⁰

Transportation access represents a vital frontier for intervention given its direct effect on cardiovascular care and clinical connection, with solutions needed to target transportation accessibility, affordability, availability, and safety for the aging population.²⁰¹ At the policy level, investment in age-friendly infrastructure, public transportation, and rural nonemergency medical transportation could support healthy cardiovascular aging, as could public transport vouchers for transportation disadvantaged individuals at elevated cardiovascular risk.²⁰¹ Population-level interventions including community-based shuttle services, adjustments in

public transportation routes, and health care-transit partnerships are most likely to succeed in partnership with local community organizations and stakeholders.²⁰² Finally, age-friendly patient-level resources have been deployed, including Cars2Care from the American College of Cardiology, as have combined care-coordination and transportation services using medical student volunteers.^{203,204}

HOMEBOUND STATUS. Homebound status, defined as never or rarely leaving home, and semi-homebound status, leaving home only with assistance or difficulty, are prevalent in the older population and accelerate cardiovascular aging.²⁰⁵ Between 2011 and 2019, approximately 5% of U.S. adults over age 70 were homebound, larger than the nursing home population, and more than twice that number were semi-homebound.^{205,206} During the COVID pandemic, however, prevalence of homebound status more than doubled to 13% of U.S. adults age 70 and above.²⁰⁶

The relationship between CVD and homebound status is understudied and likely bidirectional. Chronic diseases commonly contribute to homebound status, and CVD is the most common category of disorders among homebound older adults, with estimated prevalence between 20% to 44%, with homebound older adults having more advanced vascular, myocardial, and EP aging than the non-homebound, evidenced by higher prevalence of hypertension, stroke, MI, HF, and arrhythmia.^{205,207-209} Simultaneously, homebound status is associated with an increased risk of mortality both before and after adjusting for comorbidities and sociodemographic factors, an association compounded by coexisting social isolation.^{210,211}

Many social and geriatric determinants with strong cardiovascular impacts associate with homebound status. In a large national Medicare advantage care plan, frailty and dementia were independently associated with being homebound.²⁰⁷ Leading risk factors for homebound status include frailty, sarcopenia, reduced physical activity, limited access to health care, and coexisting medical conditions.^{21,205} Homebound individuals have a difficult time adhering to medical appointments, resulting in fragmented care and inappropriate medication prescribing; both polypharmacy and underprescription.^{212,213} Low financial security also interacts with homebound status, accelerating cardiovascular aging by generating barriers to health care access and affordable and efficient transportation.²¹⁴

Given the high risk and expense of hospital-based care in homebound older adults, there is a strong

imperative to develop and deploy digital and home-based solutions to improve cardiovascular aging in this population. Synchronous telemedicine has demonstrated promise; however, success has been partly dependent on caregiver assistance.^{215,216} Although outcomes have varied between programs, home-based care initiatives have been associated with decreased emergency department visits, hospital admissions, and health care costs, as well as increased primary and outpatient care utilization.²¹⁷ Finally, a study of 974 Medicare beneficiaries suggests that home-based long-term services and supports are underutilized, underscoring the need for improved coverage of home-based care by Medicare and other payers.^{218,219}

SOCIAL DETERMINANTS IN AGE-RELATED CARDIOVASCULAR CONDITIONS

Not only do social determinants of cardiovascular aging predispose to ASCVD, HF, valvular heart disease (VHD), and AF, they decrease resilience to stressors and represent targets to support healthy aging in place for patients with these conditions.

ATHEROSCLEROTIC CARDIOVASCULAR DISEASE.

Social determinants of cardiovascular aging have powerful impacts on ASCVD incidence and outcomes in adulthood and elderhood. It is the best studied of age-associated cardiovascular conditions, with documented associations with all structural and intermediary determinants discussed previously (Table 2). Some highlights: lower income, unemployment, and lower educational attainment increase the risk of ASCVD, particularly among Black Americans.^{220,221} In the National Health and Nutrition Examination Survey population, adults with income below \$25,000 a year and those with less than high school education have the highest risk of developing ASCVD.²²¹ Lower educational attainment in early life is associated with lower health literacy, fewer employment opportunities, and reduced lifetime earnings.²²⁰ Such disadvantages drive biologic stress and inflammaging, impact dietary behaviors, and decrease access to preventive health care, all predisposing to phenotypic coronary or polyvascular disease.²²⁰

Significant racial and ethnic differences persist in utilization of primary preventive medications and adherence to the national secondary prevention guidelines. Black and Hispanic Americans are less likely to receive guideline-directed antihypertensive medications or statins.^{222,223} These differences are multifactorial, with drivers including system-level underappreciation of cardiovascular risk factor

burden of among Black and Hispanic Americans and under-representation of these groups in landmark clinical trials. Furthermore, residents of rural areas face several challenges including limited access to health care services and lower-quality care compared to large urban centers.^{83,84,105} Thus, improving affordability of care for low-income communities, implementing preventive strategies for populations with lower health literacy such as single-pill combinations and community-based groups, and optimizing regional planning and transfer networks to ensure timely access to quality medical and procedural care are critical priorities to improve healthy vascular aging and ASCVD outcomes.

HEART FAILURE. Social determinants of cardiovascular aging are highly relevant to the development and management of HF, with mechanisms of impact including biological, behavioral, and health care-related factors. Data on the biology of adversity and myocardial aging are growing.^{6,224} Social determinants promote activation of the sympathoadrenomedullary and hypothalamic-pituitary-adrenal axes, conversion from classical to noncanonical β 2-adrenergic receptor signaling pathways, release of inflammatory cytokines, and multiple molecular hallmarks of cardiovascular aging, all associated with incident HF.^{6,225} Social determinants also impact lifestyle greatly, driving maladaptive health behaviors such as suboptimal diet and limited physical activity, well-known risk factors for incident HF, as well as increased HF decompensation, impaired quality of life, and worse prognosis among people with HF.^{99,101,226}

Social determinants can also impact physician behavior and thereby HF management. For example, recent data indicate that patients with HF who have low income are less likely to receive inpatient cardiology consultation during HF hospitalization.²²⁷ Explicit and implicit bias are important factors that can impact decision-making in both HF prevention and treatment. This may be particularly relevant when considering patient candidacy for advanced HF therapies like a left ventricular assist device or heart transplantation, where having social support and stable income are often important factors for patients to thrive with either advanced therapy. On the other hand, excluding patients negatively impacted by social determinants from these life-saving therapies can exacerbate well-described gaps in HF outcomes.

Social determinants directly impact all domains of access to HF care.²²⁸ For example: advanced HF specialists may not be physically available to people

in particular regions (accessibility) and residential settings (accommodation and availability), some people have underinsurance or financial insecurity limiting affordability of newer HF guideline-directed medical therapies (affordability), and people with lower educational attainment or health literacy may have challenges adhering to anticipatory guidance around weight checks, sodium intake, and diuretic adjustments (acceptability).

Promising strategies, including self-management programs, patient-caregiver dyadic interventions, and transitional care interventions have been tested to address HF across social determinant exposures.²²⁹⁻²³¹ The SMAC-HF (Self-Management and Care of Heart Failure) trial found that a multidisciplinary group clinic visit intervention for HF, a promising strategy to address HF in the setting of social isolation and low health literacy, improved medication adherence and increased hospitalization-free survival time.²³² The ENSPIRE (Education and Supportive Partners Improving Self-Care) study found that a patient-family partnership intervention designed to reduce dietary sodium and improve medication adherence compared with patient-family education and usual care in a majority Black population, and found both partnership and educational interventions improved dietary sodium intake and HF knowledge but not medication adherence.²³³ A randomized controlled trial of a primary care-based HF self-management program for patients of all educational and literacy levels reduced rates of hospitalization and mortality.²³⁴ Finally, a randomized controlled trial of a 3-month advance practice nurse-led transitional care intervention that included a home follow-up protocol for older adults hospitalized with HF increased the length of time to readmission or death, reduced readmissions, and decreased health care costs, representing a promising intervention to support healthy aging in homebound patients with HF.²³⁵ Harnessing implementation science and other rigorous and holistic approaches to the study of patient-, population-, and policy-level interventions to address social determinants will be necessary to improve cardiovascular aging in HF in coming decades.^{230,236}

VALVULAR HEART DISEASE. VHD is common in older adults and is independently associated with limiting symptoms and mortality.²³⁷ Although social determinants of cardiovascular aging have been less extensively studied in VHD compared with other age-associated CVD, race/ethnicity, gender, and geographic context are associated with VHD

diagnosis and access to procedural interventions.²³⁸⁻²⁴⁰ Black patients are diagnosed at younger ages and have more comorbidities such as hypertension, diabetes, and chronic kidney disease. Older Black patients are less likely to be referred to cardiology and receive diagnostic imaging, and more likely to decline intervention and be lost to follow-up than non-Black patients.²⁴¹ Older patients living in rural Florida counties faced significantly longer travel distance and times to transcatheter aortic valve replacement (TAVR), lower TAVR utilization rates, and higher adjusted TAVR mortality.²⁴⁰ County-level social vulnerability has also been associated with increased 5-year mortality in a national sample of older patients who underwent transcatheter or surgical mitral valve interventions with an adjusted HR of 1.1 (95% CI: 1.07-1.14).²⁴² Social determinants can interact when present together, with impacts on financial security that result in decreased odds of receiving transcatheter VHD therapies, similar to other expensive life-saving interventions.²⁴¹ Taken together, factors like race and ethnicity, geographic location, and socioeconomic status influence the age of onset of disease, comorbidity burden and complexity at presentation, and likelihood of receiving guideline-directed device and drug therapies, meriting further study as well as patient-, population-, and policy-level interventions.

ATRIAL FIBRILLATION. AF is a common consequence of myocardial and EP aging, and its prevalence, management, and associated outcomes are impacted by social determinants of health.^{243,244} Black race, lower income, neighborhood-level disadvantage, and social isolation are associated with earlier mortality among patients with AF.^{168,243} These and other social determinants accelerate cardiovascular aging, frailty, and dementia among the over 60 million older adults with AF globally, necessitating targeted screening, anticoagulation, and rhythm control strategies.^{39,245,246}

Although lower financial security is associated with increased incidence of AF, data regarding race/ethnicity, gender, and geographic context are mixed.²⁴⁷⁻²⁵⁰ These disparate results may be partially driven by differences in screening, as the STROKE-STOP (Systematic ECG Screening for Atrial Fibrillation Among 75 Year Old Subjects in the Region of Stockholm and Halland, Sweden) study reported decreased screening participation in individuals with lower income, lower educational attainment, and immigrant status.²⁵¹ In response, the STROKESTOP II investigators decentralized screening with 2 sites

close to lower-income neighborhoods, which increased participation across sociodemographic groups.²⁵² This result highlights the importance of social determinant-sensitive screening strategies. For example, mobile health approaches may work for many, but for homebound and digitally isolated older adults, home-based screening paired with other public health interventions such as vaccination or physical therapy could be a more optimal strategy.^{253,254}

Black race, lower income, and neighborhood-level disadvantage are associated with an increased risks of stroke and bleeding in the AF population.^{255,256} These complications increase frailty risk and are largely mediated by differences in anticoagulation. Minoritized race, lower educational attainment, neighborhood disadvantage, rurality, and lower socioeconomic status are associated with decreased likelihood of anticoagulation.^{256,257} Many of these determinants are also associated with prescription of warfarin as opposed to direct oral anticoagulant, lower anticoagulant adherence, higher likelihood of supratherapeutic warfarin dosing, and lower rates of left atrial appendage occlusion, explaining the higher rates of both stroke and bleeding.²⁵⁸⁻²⁶¹ Improving pharmacoequity for patients with AF will require improved patient education, clinician training, and decision aids for diverse populations, population health interventions such as automated electronic health record algorithms and mobile health interventions, as well as federal policies to improve direct oral anticoagulant affordability.^{162,262-265}

Finally, given the potential for rhythm control to slow AF-driven acceleration in cardiovascular aging, reducing well-established gaps and delays in guideline-based catheter ablation across race, gender, education, income, and geographic context is imperative.^{39,266-270} Progress will hinge on improving access to electrophysiologists through telemedicine and referral networks, increasing diversity in both clinical trials and the EP workforce, and ensuring comprehensive Medicare and Medicaid coverage for AF ablation.^{271,272}

CONCLUSIONS

Structural and intermediary social determinants of health drive vascular, myocardial, valvular, and EP aging throughout the life course. Some determinants and aging processes are more thoroughly studied than others (Table 2), with underexplored areas including digital access, transportation access, and homebound status, as well as determinants' impact

on valvular and EP aging. Although patient-, population-, and policy-level interventions have been proposed and studied across determinants (Table 3), the impacts of these interventions on cardiovascular aging and outcomes remain understudied across the risk and severity spectra for ASCVD, HF, VHD, and AF.

As the global population ages, improving cardiovascular outcomes will require focused study of, and interventions for, social determinants that incorporate the life-course approach, accounting for life stage and multiple intersecting determinants. Structural determinants will require policies reducing race- and gender-based discrimination, improving health literacy, and increasing access to preventive care in rural areas starting early in life. Intermediary determinants will require multilevel and multi-life stage interventions to improve food and financial security, digital and transportation access, as well as social connection and home support. Centering patients' holistic needs in research, interventions, advocacy, and medical care is paramount to advancing healthy cardiovascular aging for all.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

Dr Troy was supported by the National Heart, Lung, and Blood Institute Ruth L. Kirschstein Institutional National Research Service Award T32-HL007227. Dr Damluji receives research funding from the Pepper Scholars Program of the Johns Hopkins University Claude D. Pepper Older Americans Independence Center (OAIC) funded by the National Institute on Aging P30-AG021334 and he receives mentored patient-oriented research career development award from the National Heart, Lung, and Blood Institute K23-HL153771-01. Dr Cudjoe was supported by the National Institute on Aging (grant K23-AG075191) and the Johns Hopkins University Center for Innovative Medicine Human Aging Project as a Caryl & George Bernstein Scholar and he reported receiving personal fees from Edenbridge Healthcare and Papa, Inc, outside the submitted work. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

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KEY WORDS atherosclerotic cardiovascular disease, atrial fibrillation, cardiovascular aging, geriatric cardiology, health policy, heart failure, implementation science, population health, preventive cardiology, social determinants of health, valvular heart disease