ATVB IN FOCUS:
Arterial Stiffness

Ethnicity and Arterial Stiffness

Aletta E. Schutte, Ruan Kruger, Lebo F. Gafane-Matemane, Yolandi Breet, Michél Strauss-Kruger, J. Kennedy Cruickshank

ABSTRACT: Early vascular aging reflects increased arterial stiffness of central blood vessels at young chronological ages and powerfully predicts cardiovascular events and mortality, independent of routine brachial blood pressure and other risk factors. Since ethnic disparities exist in routine blood pressure, in hypertension and cardiovascular outcomes, this review evaluates major studies comparing arterial stiffness through the life course between different ethnic groups or races (which have no biological definition)—in children, adolescents, young, and middle-aged adults and the very elderly. Most report that compared with white European-origin samples, populations of black African descent have increased central arterial stiffness throughout different life stages, as well as a more rapid increase in arterial stiffness at young ages. Exceptions may include African Caribbean origin people in Europe. Differences in vascular structure and function are clearest, where obesity, socioeconomic, and psychosocial factors are most marked. Few studies evaluate a wider spectrum of ethnic groups or factors contributing to these ethnic disparities. Genetic effects are not obvious; maternal risk and intergenerational studies are scarce. Nevertheless, across all ethnic groups, for given levels of blood pressure and age, some people have stiffer central arteries than others. These individuals are most at risk of vascular events and mortality and, therefore, may benefit from early, as yet untested, preventive action and treatment.

VISUAL OVERVIEW: An online visual overview is available for this article.

Key Words: age distribution ■ aortic stiffness ■ arteriosclerosis ■ blood pressure ■ ethnic groups ■ youth, African American

Arterial stiffness, measured as aortic or carotid-femoral pulse wave velocity (CFPWV), is now established as a significant independent predictor of future cardiovascular events and all-cause mortality.1,2 An 1 m/s increase in aortic PWV corresponds to an age-, sex-, and risk factor-adjusted increased risk of 14% and 15% for cardiovascular events and mortality, respectively.1 As aortic PWV predicts outcomes above and beyond traditional cardiovascular risk factors, including 24-hour blood pressure (BP),3 it can be considered an intermediary outcome rather than a risk factor. These meta-analyses did not report on ethnic-specific risk, but mounting evidence supports the notion that there are ethnic disparities in arterial stiffness, which may account for discrepant cardiovascular mortality. Although such ethnic differences are thought to be explained by BP, strong evidence suggests that factors beyond BP are involved. A prime US example could be the REGARDS study (Reasons for Geographic and Racial Differences in Stroke), which compared stroke risk of individuals from African (or black) ancestry with those from European (or white) descent.4 The impact of higher BP on stroke was 3 times greater for black than white adults—for a 10 mm Hg difference in systolic BP (SBP), increased risk was 24% for blacks but 8% for whites. Adjusting for the 2- to 3-fold excess diabetes mellitus nearly eliminated the excess.4 Many studies support the higher stroke risk in black populations, such as the Centers for Disease Control and Prevention’s Wide-Ranging Online Data for Epidemiologic Research indicating a crude rate of 52.3 stroke deaths/100 000 black versus 18.1/100 000 in white adults.5 BP alone may not be sensitive enough to...
detect cardiovascular risk appropriately. British data for people of direct African or African Caribbean descent show less dramatic but still clear differences in stroke rates compared with whites, in part, due to competing cause from less coronary heart disease allowing more people at risk of stroke, but also likely from earlier, better BP control with a national health system.6,7

To clarify the potential role of arterial stiffness in ethnic-specific risk, we review studies reporting ethnic comparisons in arterial stiffness across the life course, including all ethnic or racial groups. Despite that effort, most studies compared individuals from African ancestry (referred to as black) and those from European descent (white), whereas others, such as South Asians, were underrepresented. Clearly, these categories are arbitrary, and major differences occur within such large groupings, confounded by as yet ill-defined genetic, intergenerational, and lifestyle issues referred to below.

**ARTERIAL STIFFNESS THROUGHOUT THE LIFE COURSE**

Large conduit elastic arteries undergo structural changes in their vessel walls with aging. Changes include fractured elastic lamellae, increasing dominance of collagen and its cross-linkages and key vascular smooth muscle cell–extracellular matrix interactions which alter vascular smooth muscle cell tone and change over time resulting in the development of arteriosclerosis or increased stiffness.8 This biological process does not affect all individuals equally; animal and human evidence suggest that different segments of the aortic wall are affected differently,9 which may be highly relevant to ethnic issues particularly if investigated using magnetic resonance imaging.10 Factors such as genetic susceptibility, epigenetic imprinting during fetal life, socio-demographics, and health behaviors, including diet and physical activity, all contribute across the life course trajectory—with some individuals presenting with early vascular aging (Figure 1).11 The concept of early vascular aging, therefore, reflects increased arterial stiffness for an individual’s chronological age.8

The process of early vascular aging is complex. It may, therefore, be challenging to disentangle factors contributing to increased arterial stiffness in certain ethnic groups. As presented below, increased arterial stiffness is often found in ethnic groups who frequently experience lower socioeconomic status,12 thus limited health care, less desirable diets, and lifestyle behaviors throughout the life course, including during pregnancy and, therefore, across several generations.

Heritability of arterial stiffness in both the Framingham Offspring Study13 and a Brazilian population14 was found to be modest, and a 2007 study between black and white populations found no ethnic difference.15 Recently, using metabolomics, we found that higher PWV in black boys was uniquely associated with specific urinary amino acids regarded to have protective vascular functions. These amino acids play pivotal roles in collagen metabolism, glucose metabolism, and oxidative stress; and this ethnic-specific finding suggests that biosynthesis of nonessential amino acids may be upregulated to protect the vasculature against the onset of early vascular deterioration.16 In young normotensive black and white adults with similar brachial BP, higher central SBP and central pulse pressure (PP) of black youth were independently associated with urinary amino acids that play pivotal roles in collagen metabolism and oxidative stress.17 It remains

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**Nonstandard Abbreviations and Acronyms**

<table>
<thead>
<tr>
<th>Acronym</th>
<th>Description</th>
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<tr>
<td>AI</td>
<td>augmentation index</td>
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<tr>
<td>BMI</td>
<td>body mass index</td>
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<td>BP</td>
<td>blood pressure</td>
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<tr>
<td>CAVI</td>
<td>cardio-ankle vascular index</td>
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<td>CFPWV</td>
<td>carotid-femoral pulse wave velocity</td>
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<tr>
<td>CVD</td>
<td>cardiovascular disease</td>
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<td>DASH</td>
<td>Determinants of Adolescent, Now Young Adult, Social Wellbeing, and Health</td>
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<td>DBP</td>
<td>diastolic blood pressure</td>
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<tr>
<td>HELIUS</td>
<td>Healthy Life in an Urban Setting</td>
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<td>NOMAS</td>
<td>Northern Manhattan Study</td>
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<td>PP</td>
<td>pulse pressure</td>
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<tr>
<td>PWV</td>
<td>pulse wave velocity</td>
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<tr>
<td>REGARDS</td>
<td>Reasons for Geographic and Racial Differences in Stroke</td>
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<tr>
<td>SBP</td>
<td>systolic blood pressure</td>
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**Highlights**

- Early vascular aging, which reflects increased arterial stiffness at younger chronological ages, is a strong predictor of cardiovascular outcomes.
- Many studies that compare arterial stiffness between ethnic groups, spanning studies throughout the life course (including children as young as 6 years to the elderly), report ethnic disparities independent of blood pressure and other traditional cardiovascular risk factors.
- The majority of studies found that populations of African descent present with increased aortic stiffness from young ages onwards when compared to especially white populations.
- Ethnic disparities in arterial stiffness cannot yet be explained by genetic factors or by maternal risk factors, whereas obesity, socioeconomic status, and psychosocial factors were associated with increased arterial stiffness.
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plausible that by using newer polyomics technologies, certain genes may be identified to explain unique biological pathways that regulate arterial stiffness.

**ARTERIAL STIFFNESS THROUGHOUT THE LIFE COURSE—AMONG DIFFERENT ETHNICITIES**

Aging, the most obvious factor contributing to cardiovascular disease (CVD), is directly associated with increased arterial wall thickness, primarily attributed to medial thickening, independently of atherosclerosis. Replacement of elastic fibers by collagenous tissue reduces arterial elasticity, increasing central BP by the earlier return of reflected waves during systole. These structural changes partially explain the increasing incidence of hypertension and CVD with age. For detailed reviews of molecular and mechanical mechanisms, see publications by Lacolley et al and Shadwick.

In subsequent sections, we provide a brief overview of studies comparing arterial stiffness between ethnic groups over the life course. This is not a systematic nor exhaustive review, but we highlight key papers that included a range of arterial stiffness indices. CFPWV is regarded as a gold standard measure, but studies also include PWV spanning other sections of the arterial tree (carotid-radial, carotid-dorsalis-pedis, brachial-ankle), augmentation index (AI), augmentation pressure, PP, PP amplification, cardio-ankle vascular index (CAVI), and ultrasound derived carotid \(\beta\)-stiffness index.

**Children and Adolescents**

Although arterial stiffening is a manifestation of biological aging, the onset of arterial stiffening is proposed to start at young ages due to continuous adaptations in the molecular and biomechanical makeup of blood vessels. Increased aortic stiffness occurs in premature infants as measured by aortic wall thickness and vasomotor function. Together with excess systemic hypertension in preterm infants, low birth weight and other complications, such as bronchopulmonary dysplasia, all contribute to the early onset of arterial stiffening. Explanations for higher arterial stiffness in preterm infants include impaired elastin synthesis in the aortic wall, altering mechanical properties, for example, reduced arterial compliance and higher pulse wave reflections. Other studies investigated the impact of maternal risk factors in infants, surprisingly with an inverse possibly adaptive link between maternal BP and neonatal PWV in a multi-ethnic cohort. Later studies in mainly UK European and Pakistani-origin infants, found maternal anemia weakly linked with higher infant CFPWV but not with carotid-brachial PWV. Studies in children and adolescents reported higher arterial stiffness measures predominantly in black and Hispanic populations. As shown in Table 1 (Table I in the Data Supplement), most were performed in the United States comparing black and white children (with single studies from Brazil and South Africa). The youngest study cohort investigating ethnic differences in arterial stiffness parameters was performed in black and white boys, aged 6 to 8 years from South Africa. While PWV (in all regions) and intima-media thickness were highest in black compared to white boys, carotid ultrasound stiffness indices were similar between groups. In the late 1970s, findings from the Minneapolis Children's BP Study indicated that brachial PP was higher in white compared to black children (mean age 7.7 years at baseline). However, after a 9-year follow-up period, brachial PP was found to be almost 3 mm Hg higher in black adolescents when compared to their white counterparts (mean age 16.7 years).
A US study found higher CFPWV in black than white adolescents (mean age 10.7 years; n=107), with no differences in brachial or carotid PP, intima-media thickness, and central SBP after adjustment for confounders. CFPWV was also higher in Brazilian black compared with white adolescents (mean age, 11.9 years; n=771). A study in Memphis, Tennessee, of white, black, and Hispanic adolescents (mean age 13.7 years) measured CAVI,
higher values indicating stiffer arteries. While there were no differences in unadjusted CAVI, black boys had higher CAVI after adjusting for body mass index when compared to white and Hispanic boys with normal weight. Conversely, CAVI was lowest in obese black boys suggesting vascular adaptations to obesity in early life. Another study indicated
higher brachial-ankle PWV in black than white US adolescents (n=205, mean age, 15.9 years), with no difference in brachial PP. In other American studies, socioeconomic status disparities and carotid-femoral, as well as aorto-radial and aorto-dorsalis-pedis PWV, were higher in black than white adolescents, as was AI corrected for heart rate at 75 beats per minute among young patients with type 2 diabetes mellitus, indicating higher pulse wave reflections known to predict mortality and CVD events.

**Young Adults**

Important studies investigating arterial stiffness in young adults are indicated in Table 2 (Table II in the Data Supplement). While these studies mostly compared black and white populations, there are some comparisons of Hispanics and other ethnic groups. From these various cross-sectional and longitudinal studies, there is a clear trend that black populations, particularly men, had higher arterial stiffness compared with black women, and men or women of other ethnicities. The increased arterial stiffness in black populations has, in some cases, been related to socio-demographic and psychosocial factors. Healthy black men had higher central BP, CFPWV, carotid β-stiffness index, carotid intima-media thickness, and lower PP amplification despite younger mean age than white, 21.7 versus 23.6 years and after adjustments for heart rate, body fat, and cardiorespiratory fitness. Group differences were not significant comparing brachial structure and function, where routine BP is taken, confirming that early arterial alterations occur in central elastic conduit vessels. Another US study compared 3 populations; black adults (mean age 32 years) had higher AI corrected for heart rate at 75 beats per minute than Hispanic (mean 28 years) and white adults (mean 28 years), whereas PWV was also higher in the black versus white group after post hoc analysis.

In important longitudinal work, Liang et al reported higher carotid-dorsalis-pedis PWV over 7 years in apparently healthy, normotensive black than white Americans from late childhood through to early adulthood (Figure 2). Black men had a more prominent increase in PWV with aging than black women or white men and women. Ethnic differences persisted after adjustments for socioeconomic, anthropometric, hemodynamic, and lifestyle variables, where mean arterial pressure, waist circumference, parental marital status, and marijuana use were the major factors associated with differences in PWV.

Outside the United States, in the DASH study (Determinants of Adolescent, Now Young Adult, Social Wellbeing, and Health) in London, United Kingdom, unadjusted PWVs were similar in young (21–23 years) black Caribbean and white men, but higher than in other ethnicities (black African, Pakistani/Bangladeshi, others). In fully adjusted models, black African, black Caribbean, and Indian young women had lower stiffness compared to white women, with waist/height ratio, BP, and perceived racism effects showing a large impact on arterial stiffness. In the same study population, unadjusted AI was higher in Caribbean, West African, Indian, and Pakistani/Bangladeshi groups compared with white adults (where some were borderline significant). After multivariate adjustments, including SBP, differences remained robust. The African-PREDICT study (Prospective Study on the Early Detection and Identification of Cardiovascular Disease and Hypertension) in South Africa included only normotensive young healthy black and white adults (mean 24.4 years) and also reported no ethnic differences in CFPWV, AI corrected for heart rate at 75 beats per minute.
### Table 3. Major Studies Investigating Arterial Stiffness in Middle-Aged Adults and the Elderly

<table>
<thead>
<tr>
<th>Reference</th>
<th>Age, y</th>
<th>N</th>
<th>Blood Pressure (SBP/DBP mm Hg)</th>
<th>Arterial Stiffness (P&lt;0.05*)</th>
<th>Device</th>
<th>Comments and Additional Data in Supplement</th>
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</thead>
<tbody>
<tr>
<td>Brar et al;54; United Kingdom</td>
<td>Community controls: African Caribbean; 58±5; European: 62±4.8; Gujarati: 58±5</td>
<td>46; 54; 74</td>
<td>143±17/82±9; 136.3±20/77±3.10; 139.7±24/78.6±12</td>
<td>Desc. Aortic PWV (m/s)</td>
<td>Doppler</td>
<td>Unadjusted analyses</td>
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<tr>
<td>Cruickshank et al;55; United Kingdom</td>
<td>T2 Diabetes mellitus: African Caribbean: 58±5; European: 62±4.8; Gujarati: 58±5</td>
<td>55; 178; 127</td>
<td>150.1±26/84.4±13; 146.9±13/78.2±11; 141.4±22/78.1±10</td>
<td>Compliance coefficient (nm²/kPa)</td>
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<td>Carotid PP in supplement</td>
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<td>Goel et al;51; United States</td>
<td>Black: 45±10; White: 45±10; Hispanic: 41±9</td>
<td>Black: 130±18/80±10; White: 124±14/77±9; Hispanic: 120±15/75±9</td>
<td>Aortic arch PWV (m/s) (mean; 95% CI)</td>
<td>Stiffness derived from carotid intraluminal diameters and brachial blood pressure.</td>
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<td>PP in supplement</td>
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<td>Guo et al;63; China and Sweden</td>
<td>Swedish: 75±6.5; Chinese: 75±6.5</td>
<td>Black: 1264; White: 830; Hispanic: 450</td>
<td>130±18/80±10; White: 124±14/77±9; Hispanic: 120±15/75±9</td>
<td>Aortic arch PWV (m/s)</td>
<td>Cardiac magnetic resonance</td>
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<tr>
<td>Markert et al;56; United States</td>
<td>Black: 72±6; White: 74±9; Hispanic: 68±8</td>
<td>Black: 144±20/84±12; White: 138±19/79±10; Hispanic: 140±20/83±11</td>
<td>Carotid stiffness Black: 9.4±6.21*; White: 8.7±6.91; Hispanic: 8.4±5.60</td>
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<td>Unadjusted analyses</td>
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Age and BP are well-known contributors to arterial stiffness and need to be accounted for when investigating ethnic differences. Ferreira et al investigated the effects of age and BP on PWV in black and white Brazilian-born men (mean 26 years). In normotensives, white men had higher PWV than black men, whereas, in hypertensives, black men had higher PWV than white men. Furthermore, the slope of the age-adjusted PWV-SBP regression was steeper in black than white men. As above, although there were no differences in arterial stiffness indices between normotensive black and white South Africans, black adults showed a steeper decline in PP amplification between ages 20 and 30, which aligns with the findings in black American men. Together, these 2 studies suggest that early vascular aging manifests earlier in populations from African descent.

Table 3. Continued

<table>
<thead>
<tr>
<th>Reference</th>
<th>Age, y N</th>
<th>Blood Pressure (SBP/DBP mm Hg)</th>
<th>Arterial Stiffness (P&lt;0.05*)</th>
<th>Device</th>
<th>Comments and Additional Data in Supplement</th>
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<tr>
<td>Morris et al; United States</td>
<td>Black: 47±10; N: 386; White: 49±11</td>
<td>MAP (mm Hg) (Median [IQR]) Black: 92 (84–100); White: 88 (81–96)</td>
<td>CFPWV (m/s) (all peripheral) Black: 73±0.1*; White: 7.1±0.1</td>
<td>SphygmoCor</td>
<td>Adjusted for ethnicity, sex, age, smoking, history of HT/DM, BMI, MAP, glucose and lipids</td>
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<tr>
<td>South Africa</td>
<td>Black: 374; White: 378</td>
<td>Black: 126±20.9/84.4±13.2; White: 119±15.8/78.1±9.82</td>
<td>Carotid-radial PWV (m/s) Black: 8.7±1±5.4*; White: 7.6±3±1.37</td>
<td>Compilor SP</td>
<td>Unadjusted analyses</td>
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<tr>
<td>the Netherlands</td>
<td>Women: African Surinamese: 47.4±12.5; Ghanian: 48.3±10.8; Dutch: 45.1±14.3; South Asian Surinamese: 47.0±13.4</td>
<td>Women: African Surinamese: 1119; Ghanaian: 948; Dutch: 920; South Asian Surinamese: 604</td>
<td>Aortic PWV (m/s) Men: African Surinamese: 8.1±2±5.02; Ghanaian: 8.0±1±7.4; Dutch: 7.8±1±1.86; South Asian Surinamese: 8.2±2±2.20*</td>
<td>Arteriograph</td>
<td>Unadjusted analyses</td>
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<td>Men: African Surinamese: 721; Ghanaian: 725; Dutch: 877; South Asian Surinamese: 942;</td>
<td>Women: African Surinamese: 131.7±19.8/2.7±1.17; Ghanaian: 136.2±20.4/5.6±12.5; Dutch: 121±16.7/7.4±11; South Asian Surinamese: 126.6±20/78.9±12</td>
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Data are mean±SD unless otherwise specified. Black referring to people of African descent; Al75 indicates augmentation index corrected for heart rate at 75 beats per minute; BA-PWV: brachialankle PWV; BMI, body mass index; CFPWV, carotid-femoral pulse wave velocity; DM, diabetes mellitus; HT, hypertensive; MAP, mean arterial pressure; NT, normotensive; PP, pulse pressure; PPA, pulse pressure amplification; SBP, systolic blood pressure; and SES, socioeconomic status.

*Higher arterial stiffness.
†Where data have shown may be referred to as unadjusted, adjusted values may be available in the Reference’s text.

per minute, or PP amplification (adjusted for mean arterial pressure).45
Middle-Aged and Elderly Populations

Arterial stiffness at older ages has been extensively investigated. As in younger populations, the following large population-based studies in different world regions consistently found that black participants had increased arterial stiffness than the comparison group(s) (Table 3 and Table III in the Data Supplement). A cross-sectional study in urban Vitoria, Brazil, included 2015 participants (aged 36–47 years) from 4 different ethnic groups: Amerindians (29.2%), white (26.7%), Mulatto (38.3%), and black (5.8%). CFPWV was higher in the black group after adjustment for traditional covariates, including age, sex, and mean arterial pressure. In the US ARIC cohort (Atherosclerosis Risk in Communities), 268 black and 2459 white men and women (aged 45–64 years) with no history of CHD or stroke/transient ischemic attack were included. After adjustment for cardiovascular risk factors, mean β stiffness was 9% higher for black adults (mean±SE: 11.3±0.3) than for whites (10.3±0.1). In the multi-ethnic, population-based Dallas Heart Study of white, black, and Hispanic adults (43.7 years; n=2544) black and Hispanics, as compared to whites, displayed greater proximal aortic stiffness, assessed as aortic arch PWV or characteristic impedance, in fully adjusted models. Although blacks had a higher prevalence of numerous cardiovascular risk factors, none of these explained the ethnic differences in aortic arch PWV and characteristic impedance. In a bi-ethnic community-based sample from metropolitan Atlanta, United States (48 years; n=855), CFPWV was higher in the black group compared to their white counterparts, after adjustment for traditional risk factors. These differences persisted in a subgroup completely free of conventional risk factors. A South African study including black and white adults (40.7 years; n=750) also showed that carotid-radial and carotid-dorsalis-pedis PWV were elevated in black adults, in groups with normal or elevated BP.

However, in 2 UK studies, both in London and including people with testedly normal glucose tolerance from the community as well as with type 2 diabetes mellitus, ethnic differences in PWV between African Caribbeans and Europeans in similar locations were not found (Cruickshank et al52; Park et al57)—Table 3. PWV was a highly significant predictor of mortality, adjusted for age, BP, and ethnicity, as was glucose tolerance status. In both studies, type 2 diabetes mellitus and glucose intolerance worsened arterial stiffening as PWV, measured directly by doppler on the descending aorta. People of Indian (South Asian), origin, Gujerati in one and Sikh in Park’s, were included and had intermediate PWV values.

Among the very elderly, less data are available describing ethnic differences related to arterial stiffness. The NOMAS (Northern Manhattan Study)56 investigated carotid artery diameter and stiffness between 1536 black, white, and Hispanic participants (70 years) also showing greater carotid stiffness among the black and Hispanic groups compared with whites.

Although the majority of studies investigating ethnic differences included black populations, other ethnic differences are also evident, specifically among South Asians. Such findings are of importance as people of South Asian ethnicity have a high prevalence of heart disease, stroke, and small vessel disease. Ethnic minorities are often underrepresented in major works examining the role of arterial stiffness in CVD. Aiming to address this gap, a small Canadian study54 cross-sectionally recruited 22 South Asian participants (71 years) and compared various arterial stiffness measures to age- and sex-matched white participants. South Asians had higher carotid PP and lower compliance coefficients indicating stiffer vessels. Similarly, central PP and AI were higher in an older (69±6.1 years) South Asian sample compared to African Caribbean and white populations from the UK SABRE study (Southall and Brent Revisited).57 The HELIUS study (Healthy Life in an Urban Setting),58 a large prospective cohort study on health care utilization in Amsterdam, showed similar findings. There, Dutch (n=1797), South Asian Surinamese (n=1846), African Surinamese (n=1840), and Ghanaian (n=1673) participants were included. Unadjusted PWV was higher in African and South Asian groups compared with those of Dutch descent, in agreement with known increased cardiovascular risk. Increased PWV in some groups was mainly driven by conventional risk factors, specifically high BP.

PERSPECTIVES

Collectively, most studies performing ethnic-specific comparisons in arterial stiffness report that populations of African descent (and often also Hispanic populations) have higher arterial stiffness than white populations from as young as 624 to 70 years of age.56 Raised BP often accompanies arterial stiffness in black populations.56 Yet in analyses of normotensive or healthy subgroups, increased arterial stiffness persist5,59 supported by studies in children15,33,34 or young normotensive adults41,43 where black participants already have elevated arterial stiffness but normal or similar BP as their comparison groups. When tracking arterial stiffness with increasing age,19,41,46 it becomes clear that early vascular aging already occurring in adolescence or young adulthood predisposes black individuals to excess cardiovascular risk throughout the life course. Moreover, early vascular aging may be an important potentiating factor in the development of cardiovascular risk, adding to the already accelerated biological aging or weathering observed in black populations.60

Are specific ethnic groups genetically predisposed to develop arterial stiffness? Are socioeconomic, sociodemographic, psychological, maternal risk factors, and...
Health behaviors: the drivers? So far, studies provide evidence for modest heritability of arterial stiffness\textsuperscript{12,14} (with no ethnic difference\textsuperscript{15}), and slight evidence of maternal risk factors predicting increased stiffness\textsuperscript{61,62}. In contrast, psychosocial factors\textsuperscript{58} such as perceived racism,\textsuperscript{12} lower socioeconomic status,\textsuperscript{12} and health behaviors such as obesity\textsuperscript{12,25} do have significant impacts on arterial stiffness. Contributing factors to early vascular aging leading to excess risk remain poorly understood. There is a critical gap in studies evaluating these factors in detail, which would allow a more precise pinpointing of areas to address in the prevention and management of arterial stiffness and subsequent CVD development throughout the life course.

Identifying early vascular aging across all ethnic groups by assessment of aortic stiffness may add greater precision than BP alone to define those at risk for early onset of CVD and future events. It is certainly clear that at given ages and levels of peripheral or central BP, some individuals have stiffer vessels than others and are at higher cardiovascular risk.

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