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Home blood pressure telemonitoring reveals race-specific patterns of target organ damage

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Background: Racial differences in cardiac and renal target organ damage (TOD) may persist at comparable blood pressure levels. This study compared TOD in high-risk, non-African-American Black and White patients in relation to the home blood pressure (HBP).

Methods: UPRIGHT-HTM (NCT04299529) is an ongoing international trial comparing risk stratification strategies in asymptomatic patients, aged 55–75 years, with ≥5 risk factors. Patients engage in HBP telemonitoring (OMRON HEM 9210-T). After 34.7 months (median), 287 Black and 154 White patients underwent echocardiography. At baseline, their chronic kidney disease (CKD) grade was assessed by cross-classification of the race-free estimated glomerular filtration rate and albuminuria (2024 KDIGO guideline). HBP was stratified by the 2024 ESC thresholds. Linear and logistic regression models, including a race-by-HBP interaction term, were applied to assess associations with the home systolic HBP.

Results: The number of HBP readings was 252 215. Median systolic/diastolic HBP was 127/77 mmHg with 142 patients (32.2%) having home hypertension. Fewer Black patients received statins or combination therapy for hypertension or diabetes. Among nonhypertensive White compared to Black patients, left atrial dimensions, mitral annular s', and stroke volume had a steeper slope in relation to systolic HBP. All patients had concentric left ventricular remodeling, but only 4 Black and 13 White patients had an ejection fraction < 50%. CKD grade was worse in Black than White patients without association with HBP.

Conclusions: TOD primarily affects the kidney in Black and the heart in White patients. Intensifying pharmacological treatment in sub-Saharan Africa, including antihypertensives, lipid-lowering agents, antidiabetic medications, and aspirin, should create an opportunity for improved overall cardiovascular and metabolic prevention.

Graphical abstract: http://links.lww.com/HJH/D54

Keywords: chronic kidney disease, echocardiography, home blood pressure, left ventricular structure and function, racial differences, target organ damage

Abbreviations: BP, blood pressure; CI, 95% confidence interval; CKD, chronic kidney disease; CVD, cardiovascular disease; E/A, ratio of the peak velocities of the transmitral blood during early diastole to late diastole; e'/a', ratio of the peak mitral annular velocities during early diastole to late diastole; E/e', ratio of the E-to-e' velocities reflecting left ventricular filling pressure; eCRF, Electronic case report form; HDL, high-density lipoprotein; HSBP, home systolic blood pressure measured by telemonitoring; HTM, home blood pressure telemonitoring; IQR, interquartile range;

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KDIGO, Kidney Disease Improving Global Outcomes; LAD, left atrial diameter; LAV, left atrial volume; LAVI, left atrial volume indexed to body surface area; LDL, low-density lipoprotein; LVDD, left ventricular diastolic dysfunction; LVEDD, left ventricular end-diastolic diameter; LV, left ventricle or left ventricular; LVM, left ventricular mass; LVMI, left ventricular mass indexed to body surface area; s', peak velocity of the mitral annulus during systole; SV, left ventricular stroke volume; TOD, target organ damage; UPRIGHT-HTM, Urinary Proteomics Combined with Home Blood Pressure Telemonitoring for Healthcare Reform Trial

INTRODUCTION

The racial disparities between Black and White individuals in the disease burden due to cardiovascular disease (CVD) and chronic kidney disease (CKD) are well documented in the United States [1–3]. Among all racial groups, African-Americans have the highest hypertension prevalence [1,4]. The American studies are confounded by genetic admixture, but the African ancestry undoubtedly plays a pivotal role in the high vulnerability of African-Americans to CVD and CKD [5–7]. Few studies compared the CVD and CKD predisposition of Black people born and living in sub-Saharan Africa with White Europeans [8–10] or White South- Americans [11–13].

Home blood pressure telemonitoring (HTM) provides a long-term estimate of the blood pressure (BP) burden, which is the main driver of CVD and CKD [14]. An important feature of HTM is that target organ damage (TOD) is more closely associated with home than office BP [15]. The Urinary Proteomics Combined with Home Blood Pressure Telemonitoring for Healthcare Reform Trial (UPRIGHT-HTM) is an ongoing clinical trial [16,17] with the objective to compare the management of high-risk patients based on HTM combined or not with urinary proteomic profiling. UPRIGHT-HTM patients are recruited, using the same inclusion and exclusion criteria at clinical sites located in Europe, sub-Saharan Africa, Asia, and South America. To be eligible, patients must have at least five established risk factors. The trial demonstrated that long-term HTM is feasible in widely diverse settings [18]. In UPRIGHT-HTM, echocardiography and measurement of the glomerular filtration rate are applied to assess target organ damage. In 2024, the European Society of Cardiology (ESC) proposed lower treatment targets in the management of elevated BP and hypertension [19] compared with the 2018 guidelines [20]. UPRIGHT-HTM data were analyzed with the objective to assess echocardiographic left ventricular (LV) structure and function and the stage of CKD according to the KDIGO criteria [21] in relation to HTM by the home BP strata proposed by the 2024 ESC guidelines [19].

METHODS

UPRIGHT-HTM (Clinicaltrials.gov identifier: NCT04299529) complies with the Helsinki declaration [22]. The Ethics Committee of University Hospitals Leuven, Belgium (Belgian registration number, B3222020000276), acting as the central Ethics Committee, and the Institutional Review Boards of all clinical sites approved the trial protocol [16]. Patients provided written informed consent using paper forms translated in the local language at each recruitment site. In line with the European General Data Protection Regulations [23], these forms, allowing the identification of patients, are filed in the local medical records [16] and are not accessible by the trial coordinating team.

Design of the UPRIGHT-HTM trial

UPRIGHT-HTM is an investigator-initiated randomized clinical trial, of which the rationale and protocol [16] and progress reports [17,18] have been published. The underlying hypothesis is that refinement of risk assessment in the intervention group by urinary proteomic profiling on top of HTM will motivate caregivers and patients to better risk management and therefore to less TOD and cardiovascular and renal complications. Eligible patients are asymptomatic adults of either sex, aged 55–75 years, with five or more guideline-defined [19,20] risk factors, preferably including hypertension, type–2 diabetes, or both. Furthermore, to qualify, patients must have an E-Mail address, internet access via an android smartphone, and be willing to engage in HTM during follow-up. On top of the exclusion criteria common to all clinical trials, study-specific exclusion criteria comprise an impracticable echocardiographic window, atrial fibrillation, or flutter or frequent extrasystoles, because these conditions interfere with echocardiographic imaging and the accuracy HTM using oscillometric devices [16]. After a screening visit and a run-in period of variable length (1–5 weeks or longer), patients are stratified by center and sex, randomized in a 1 : 1 proportion by means of an automated web-based computerized random function [16], and followed up at semi-annual and annual intervals.

Data collection

UPRIGHT-HTM is running as a patient-centered trial, mainly positioned at the patients' homes and primary care practices or out-patient clinics, using the CE-certified web-based and secured WiPaM platform (*Wi*reless *Pa*tient *M*onitoring), developed by RDSM NV, Hasselt, Belgium (https://www.rdsm.eu). HTM measurements are collected electronically by patients using an android-based app and by their caregivers via electronic case report forms (eCRFs). Data collection is described in detail in the online only Supplemental Material, http://links.lww.com/HJH/D53. For HTM, patients are using the validated [24] OMRON HEM 9210-T monitors (Omron Healthcare Co., Ltd, Kyoto, Japan) fitted with a cuff that accommodates the range of adults upper-arm circumferences. Patients are instructed to measure their blood pressure after 5 min of rest in the sitting position preferably within 1 h after awakening, before breakfast and before taking any medication, if possible, daily. The HTM measurements are averaged per patient and categorized according to the 2024 ESC guidelines [19] as nonelevated BP (<120/<70 mmHg), elevated BP (120/70 to <135/<85 mmHg) or hypertension (≥135/≥85 mmHg). If systolic and diastolic BP are in different categories, the highest category is used for classification. Caregivers complete the prerandomization eCRF showing that enrolled patients meet all eligibility criteria and providing information on anthropometrics, the patient's medical

history, and the use of medications by drug class. Patients are referred for echocardiography before or shortly after randomization and next at annual intervals. Venous blood samples are collected for hematological and biochemical measurements. Low-density lipoprotein (LDL) serum cholesterol is calculated from total serum cholesterol, high-density lipoprotein (HDL) serum cholesterol, and the serum triglyceride concentration by the Friedewald formula [25]. Dyslipidemia is present when serum total (≥4.91 mmol/l), LDL (≥2.97 mmol/l) or non-HDL (≥3.36 mmol/l) cholesterol or serum triglycerides (≥1.69 mmol/l) exceed recommended levels or when HDL serum cholesterol is low (<1.19 mmol/l in women and <1.03 mmol/l in men). The biological samples are processed by locally certified laboratories.

Echocardiography

The standardized protocol for echocardiography has been published [16]. All centers used General Electric echocardiographic devices (Horton, Norway) interfaced with appropriate probes (Table S1, Supplemental Digital Content, http://links.lww.com/HJH/D53). With patients in partial left decubitus and breathing normally, echocardiographic images are obtained together with an ECG signal along the parasternal long and short axes and from the apical 4- and 2-chamber long-axis views. All recordings from each echocardiographic window must include at least five cardiac cycles and are digitally stored for off-line analysis. M-mode echocardiograms of the left ventricle (LV) are recorded from the parasternal long-axis view under control of the 2D image with the ultrasound beam positioned just below the mitral valve at the level of the posterior chordae tendineae. Using tissue Doppler imaging, the observers then record low-velocity, high-intensity myocardial signals at a high frame rate, while adjusting the imaging angle to ensure a parallel alignment of the ultrasound beam with the myocardial segment of interest. From the apical window, the sonographer places a 5-mm Doppler sample at the septal, lateral, inferior, or posterior sites of the mitral annulus, or at a combination of these sites.

The end-diastolic LV dimensions are used to calculate left ventricular mass (LVM) by an anatomically validated formula [26]. Left atrial dimensions are measured in three orthogonal planes: the parasternal long, lateral, and superior-to-inferior axes and left atrial volume (LAV) is calculated using the prolate-ellipsoid method [27]. Both LVM (LVMI) and LAV (LAVI) are indexed to the body surface area. Left ventricular diastolic dysfunction (LVDD) is an abnormally low age-specific transmitral E/A ratio, indicative of impaired relaxation, or a mildly-to-moderately elevated LV filling pressure ($E/e' > 8.5$) with normal or decreased age-specific E/A ratio in patients with an ejection fraction of 50% or higher [28,29]. The intra- and inter-observer variability of the echocardiographic measurements have been published for Black Nigerians [30] and with similar results in White Polish patients [31].

Assessment of renal function

The glomerular filtration rate was derived from serum creatinine, using the race-free CKD-EPI Collaboration equation [32]. Creatinine was determined using Jaffe's method with modifications in certified laboratories that applied isotope-dilution mass spectrometry for calibration [33]. To determine CKD stage, the KDIGO recommendations were applied [21]. The presence of albuminuria was determined from mid-morning urine samples, taken on three consecutive days or on only two days if the first two samples were negative (<30 mg/l). The urinary albumin concentration was measured by automated laboratory devices, but to reduce costs, in particular in the low- and middle-income countries, the use of dipsticks was also allowed.

Statistical analysis

Data management and analysis were done using SAS version 9.4. (SAS Institute, Cary, NC). From the WiPaM platform, completely anonymized data were directly imported into SAS at the Study Coordination Centre. The central tendency (spread) of variables is represented by the arithmetic mean (SD) for normally distributed variables and by the median [interquartile range (IQR)] for nonnormally distributed continuous variables. Categorical variables are presented as proportions. For the current analysis, group means were compared by the unpaired *t*-test and proportions by Fisher exact test. Linear and logistic regression were applied to assess LV structure and function and renal function in relation to home systolic blood pressure (HSBP) per 10-mmHg increments in the level. We tested heterogeneity between Black and White patients by introducing the appropriate interaction terms in the model. Statistical significance was a two-sided α-level of 0.05.

RESULTS

Characteristics of participants

The selection of the 441 patients for the current analysis was based on data that accrued until 30 November 2025 (Fig. 1). The study included 287 Black and 154 White patients (Table 1). The Black patients were enrolled in Argentina ($N = 1$), Mozambique ($N = 14$), Nigeria ($N = 257$) and South Africa ($N = 15$), and the White patients in Argentina ($N = 34$), Belgium ($N = 1$), Poland ($N = 108$), and Slovenia ($N = 11$). Few Blacks reported smoking (Table 1). Black patients were 2.9 years [95% confidence interval (CI), 1.5–4.3 years] younger than White patients, but had a greater risk profile, as evidenced by the prevalence of albuminuria (20.9 vs. 1.3%) and dyslipidemia (83.6 vs. 59.7%). Black patients also had a higher systolic (130.0 vs. 126.5 mmHg) and diastolic (81.0 vs. 77.4 mmHg) home BP, resulting in a higher prevalence of hypertension (106/287 [36.9%] vs. 36/154 [23.4%]; $P < 0.001$). Black patients also had a faster office and home heart rate: 77.1 vs. 70.9 beats per minute and 74.6 vs. 70.5 beats per minute), respectively. After excluding patients on treatment with β-blockers, office heart rate was 77.8 (SD, 12.6) in 248 Black patients compared to 73.7 (11.5) beats per minute in 74 White patients (difference, 4.0 beats per minute; 95% CI, 0.8–7.2 beats per minute; $P = 0.014$) with a directionally similar difference in the long-term home heart rate 74.9 (9.7) vs. 72.1 (9.6) beats per minute (difference, 2.8 beats per minute; 95% CI, 0.3 to 5.3 beats per minute; $P = 0.031$). Black patients had significantly ($P < 0.001$) higher serum levels of total, LDL and non-HDL cholesterol, resulting in a higher total-to-HDL cholesterol ratio (Table 1).

Of 665 randomized patients, 215 patients were not included in the analysis, mainly because of delays in

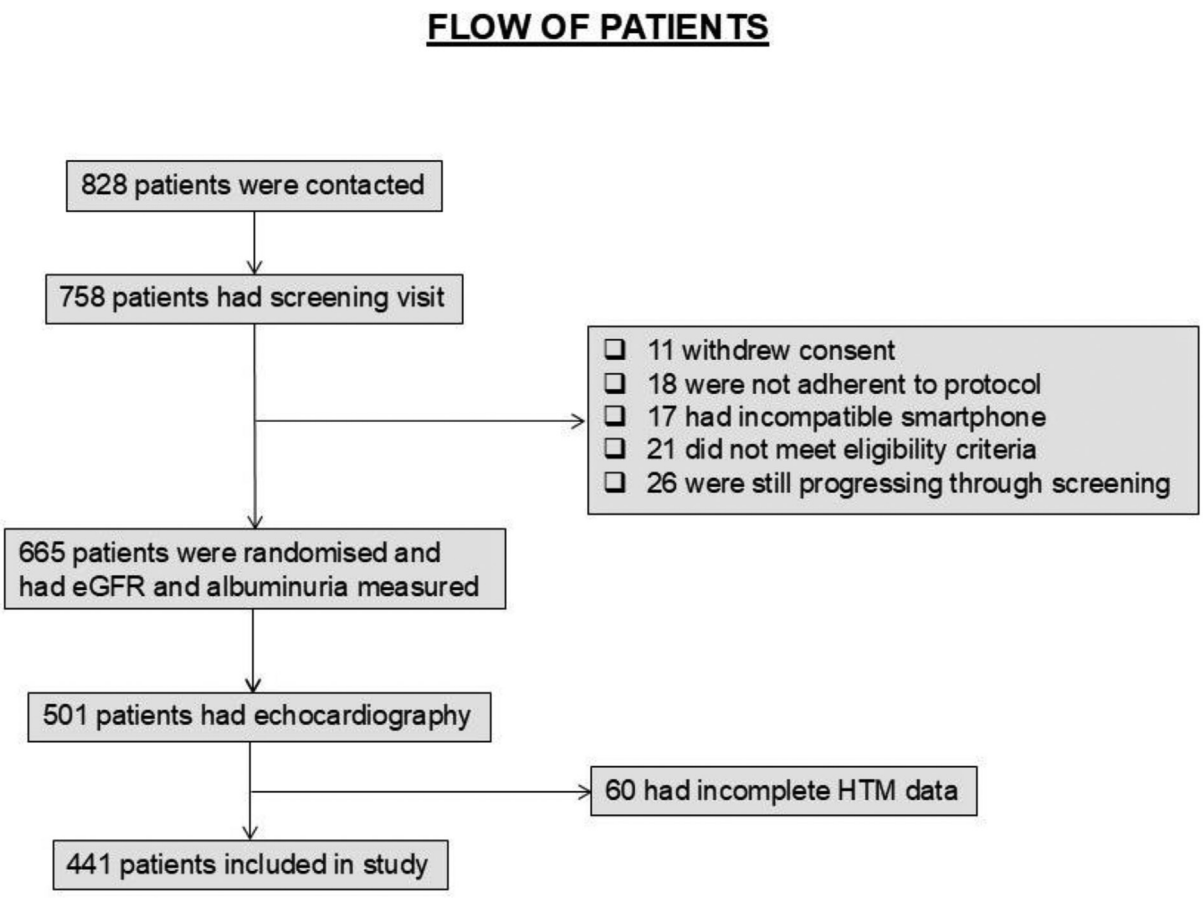

FIGURE 1 Consort diagram showing patient disposition, including screening, randomization, and selection of patients for the current analysis. eGFR is the glomerular filtration rate estimated from serum creatinine, using the race-free CDK-EPI Collaboration equation. Incomplete HTM data refers to home blood pressure telemonitoring from randomization until the echocardiographic examination.

completing the prerandomization eCRF, resulting in missing values (Table S2, Supplemental Digital Content, http://links.lww.com/HJH/D53). With few exceptions, analyzed and nonanalyzed patients had similar characteristics.

Pharmacological treatment

The analyses that follow were stratified by the home BP status according to the 2024 ESC guideline and race. The frequency of nonelevated home BP was low: 10 patients (3.5%) among Black and 11 (7.1%) among White participants. Patients with nonelevated or elevated home BP were therefore pooled. Results are summarized in Table S3, Supplemental Digital Content, http://links.lww.com/HJH/D53.

White compared to Black patients of any home BP category were more frequently treated with β-blockers and inhibitors of the renin-angiotensin system. Combining the home BP categories, the White vs. Black frequencies of the use of β-blockers or inhibitors of the renin-angiotensin system were 51.9 vs. 13.6% and 92.9 vs. 72.5%, respectively. Combination antihypertensive therapy was more common in White than Black patients (85.0 vs. 69.3%). Among the lipid-lowering drugs, irrespective of the home BP status, statins were more commonly prescribed to White than Black patients (75.3 vs. 46.0%). Among diabetic patients with nonelevated or elevated home BP, metformin (45.8 vs. 27.1%), incretin mimetics (4.2 vs. 0.6%), inhibitors of the sodium-glucose cotransporter 2 (8.5 vs. 2.8%), and combination antidiabetic therapy (50.0 vs. 28.2%) were more frequently prescribed to White than Black patients. This was also the case for aspirin in patients with hypertension (30.6 vs. 11.3%). Hypertensive White patients compared to the nonhypertensive group, including White patients with nonelevated and elevated BP, were more often treated with diuretics, dihydropyridine calcium-channel blockers, and aspirin.

Home blood pressure telemonitoring

Table S4, Supplemental Digital Content, http://links.lww.com/HJH/D53 summarizes the number of patients, the number of months of follow-up between randomization and the day of the echocardiographic examination, and the distribution of the home BP level over follow-up. Across the 11 recruitment sites, median follow-up was 34.7 months (IQR, 20.6–42.2 months) and the number of home BP readings collected amounted to 252 215, resulting in a median systolic/diastolic home BP level of 127/77 mmHg (IQR, 118/70 to 136/83 mmHg). Figure S1, Supplemental Digital Content, http://links.lww.com/HJH/D53 provides

TABLE 1. Characteristics of participants by race

Characteristic	Black patients	White patients	*P*
Number in group	287	154	
Number with characteristic, %			
Women	158 (55.1)	92 (59.7)	0.37
Current smoking	7 (2.4)	18 (11.7)	<0.001
Drinking alcohol	10 (2.3)	11 (7.4)	0.10
Body mass index ≥30 kg/m^2	124 (43.2)	79 (51.3)	0.11
Diabetes mellitus	173 (60.3)	93 (60.4)	>0.99
Albuminuria	60 (20.9)	2 (1.3)	<0.001
Dyslipidemia	240 (83.6)	92 (59.7)	<0.001
Antihypertensive medicines	276 (96.2)	147 (95.5)	0.80
Antidiabetic medicines	89 (31.0)	73 (47.4)	<0.001
Lipid-lowering medicines	133 (46.3)	118 (76.6)	<0.001
Anthropometrics			
Age, year	62.0 (7.5)	65.0 (6.2)	<0.001
Body height, cm	165 (8.9)	167 (8.9)	0.069
Body weight, kg	82.3 (19.0)	84.1 (14.1)	0.28
Body mass index, kg/m^2	30.1 (6.8)	30.1 (4.6)	0.99
Waist circumference, cm	96.8 (17.0)	100.7 (13.1)	0.016
Body surface area, kg/m^2	1.89 (0.22)	1.93 (0.20)	0.072
Hemodynamic measurements			
Office systolic pressure, mmHg	134.5 (17.2)	136.4 (16.0)	0.27
Office diastolic pressure, mmHg	82.8 (12.0)	83.9 (15.0)	0.42
Office heart rate, bpm	77.1 (12.8)	70.9 (12.0)	<0.001
Home systolic pressure, mmHg	130.0 (11.6)	126.5 (9.7)	0.002
Home diastolic pressure, mmHg	81.0 (8.5)	77.4 (6.4)	<0.001
Home heart rate, bpm	74.6 (9.7)	70.5 (9.2)	<0.001
Biochemical measurements			
Total serum cholesterol, mmol/l	4.94 (1.24)	4.46 (1.13)	<0.001
HDL serum cholesterol, mmol/l	1.31 (0.39)	1.37 (0.32)	0.12
Total-to-HDL serum cholesterol ratio	4.11 (1.59)	3.41 (1.08)	<0.001
LDL serum cholesterol, mmol/l	2.96 (1.67)	2.45 (1.05)	<0.001
Non-HDL serum cholesterol, mmol/l	3.63 (1.19)	3.11 (1.10)	<0.001
Triglycerides, mmol/l	1.47 (0.62)	1.43 (0.57)	<0.45

Current smoking is inhaling tobacco smoke on a daily basis. Drinking alcohol is the occasional or daily consumption of ethanol containing beverages. Body mass index is weight (kg) divided by height squared (m^2). Home blood pressure is the average of all available readings from randomisation until the echocardiographic examination.
HDL, high-density lipoprotein; LDL, low-density lipoprotein.

the distribution of the systolic and diastolic home BP by center.

LV structure and function

Patients with nonelevated and elevated BP

Comparison of Black and White patients with nonelevated and elevated home BP (Table 2), revealed differences in LV structure and systolic and diastolic LV function. With regard to LV structure, the left-atrium-to-aorta diameter ratio (1.25 vs. 1.34; $P=0.009$) and the LV end-diastolic diameter (45.6 vs. 47.2 mm; $P=0.025$), were smaller in Black patients, whereas Blacks had a greater relative wall thickness (47.3 vs. 44.8; $P=0.036$). Moving to systolic function (Table 2), Black compared to White patients had smaller mitral annular s′ (8.10 vs. 8.77 cm/s; $P=0.030$) and SV (64.5 vs. 69.2 mL; $P=0.042$), but standardization of SV to body surface area (stroke volume index) removed the racial difference ($P=0.22$). With regard LV diastolic function (Table 2), Black compared to White patients had smaller LAV (48.4 vs. 56.5 ml; $P<0.001$), smaller LAVI (26.2 vs. 29.7 ml/m^2; $P=0.004$), shorter isometric relaxation time (100.7 vs. 108.6 ms; $P=0.026$) and lower E/e′ (9.3 vs. 11.3; $P=0.004$) without racial difference in the LVDD prevalence (53.6 vs. 60.2%; $P=0.28$).

Patients with hypertension

In Black and White hypertensive patients, the echocardiographic measurements were not different with the exception of mitral annular s′, which was smaller in Black participants (7.62 vs. 8.71 cm/s; $P=0.025$).

Other measurements

The frequency of a LV ejection fraction below 50% was minor and amounted to 7 cases (2.4%) in Black and 15 (9.7%) in White participants. Heart rate measured in the supine position during the echocardiographic examination was 4.8 beats per minute (95% CI, 2.4 to 7.1 beats per minute; $P<0.001$) faster in Black than White patients.

Relation with home systolic BP

Table S5, Supplemental Digital Content, http://links.lww.com/HJH/D53 describes the associations between the echocardiographic traits and the home systolic BP in Black and White patients separately. The regression coefficients, given with 95% CI, express the change in the features of LV structure and systolic and diastolic LV function per 10 mmHg-increase in the home systolic BP (HSBP). Regression slopes were compared by introduction of a race-by-HSBP interaction term in the models. Left atrial diameter (LAD), LAV, LAVI, the LV end-diastolic diameter (LVEDD), and

TABLE 2. Echocardiographic structure and function by blood pressure status and ethnicity

Characteristic	Nonelevated and elevated blood pressure			Hypertension		
	Black patients	White patients	*P*	Black patients	White patients	*P*
Number in group	181	118		106	36	
M-mode LV structure						
Aortic root diameter, mm	28.8 (4.6)	28.4 (6.0)	0.58	30.1 (4.3)[a]	29.3 (6.1)	0.42
Left atrial diameter, mm	35.2 (5.5)	36.4 (6.3)	0.062	37.6 (6.9)[b]	38.3 (6.2)	0.57
Left atrium-to-aorta diameter ratio	1.25 (0.25)	1.34 (0.36)	0.009	1.27 (0.27)	1.37 (0.39)	0.090
Interventricular septum, mm	10.7 (2.4)	10.8 (1.6)	0.76	11.3 (2.4)[a]	11.2 (1.6)	0.69
Posterior wall thickness, mm	10.4 (1.9)	10.2 (1.5)	0.39	11.0 (2.2)[b]	10.6 (1.8)	0.29
End diastolic diameter, mm	45.6 (6.8)	47.2 (4.5)	0.025	47.3 (8.5)	48.7 (4.4)	0.36
End systolic diameter, mm	28.3 (6.1)	29.5 (5.3)	0.072	30.0 (8.3)[a]	29.7 (5.0)	0.83
Relative wall thickness	47.3 (11.0)	44.8 (7.5)	0.036	50.0 (12.8)	45.0 (7.1)	0.080
LV mass, g	139.6 (35.3)	143.3 (27.8)	0.34	155.2 (38.9)[c]	154.6 (30.8)[a]	0.93
LV mass index, g/m^2	75.4 (21.0)	75.4 (13.4)	0.99	81.2 (19.4)[a]	77.5 (15.1)	0.30
Echocardiographic LVH, *n* (%)	17 (9.4)	5 (4.2)	0.11	12 (11.3)	2 (5.6)	0.52
LV systolic function						
Heart rate during examination, bpm	74.6 (12.0)	69.7 (10.6)	<0.001	75.5 (14.0)	71.6 (10.9)	0.13
Fractional shortening, %	37.9 (8.8)	37.4 (9.2)	0.66	37.2 (8.9)	39.0 (8.7)	0.30
Ejection fraction, %	67.5 (10.1)	66.5 (11.7)	0.44	66.7 (10.1)	68.3 (10.6)	0.42
Mitral annular s', cm/s	8.10 (2.31)	8.77 (2.11)	0.030	7.62 (2.29)	8.71 (2.02)	0.025
Stroke volume, ml	64.5 (20.3)	69.2 (18.5)	0.042	67.2 (20.1)	76.4 (17.7)[a]	0.016
Stroke volume index, ml/m^2	34.9 (11.8)	36.5 (9.6)	0.22	35.3 (11.2)	38.4 (9.5)	0.13
Cardiac output (l/min)	4.81 (1.75)	4.83 (1.51)	0.94	5.07 (1.79)	5.43 (1.45)[a]	0.28
LV diastolic function						
Left trial volume, ml	48.4 (20.3)	56.5 (17.2)	<0.001	53.1 (21.0)	57.4 (18.5)	0.28
Left atrial volume index, ml/m^2	26.2 (11.2)	29.7 (8.8)	0.004	28.1 (11.9)	28.6 (8.6)	0.82
Mild enlargement, *n* (%)	23 (12.7)	26 (22.0)	<0.001	12 (11.3)	4 (11.1)	0.70
Moderate enlargement, *n* (%)	18 (9.9)	26 (22.0)		13 (12.3)	7 (19.4)	
Severe enlargement, *n* (%)	26 (14.4)	19 (16.1)		21 (19.8)	5 (13.9)	
Isometric relaxation time (ms)	100.7 (26.8)	108.6 (21.6)	0.026	108.6 (29.3)[a]	108.8 (22.9)	0.96
>100 ms, *n* (%)	72 (53.7)	48 (72.7)	0.014	55 (64.0)	16 (70.0)	0.81
Transmitral E/A ratio	7.19 (3.44)	7.60 (3.12)	0.30	7.37 (3.48)	7.75 (4.57)	0.61
Mitral annular e'/a' ratio	0.81 (0.32)	0.77 (0.31)	0.27	0.76 (0.36)	0.75 (0.35)	0.93
E/e' ratio	9.3 (3.9)	11.3 (7.7)	0.004	10.6 (4.1)[b]	11.5 (8. 6)	0.37
LV diastolic dysfunction, *n* (%)	97 (53.6)	71 (60.2)	0.28	68 (64.2)	20 (55.6)	0.43

Values are means (SD) or frequency (%). Stratification based on the home blood pressure (HBP) was done according to the thresholds proposed by the 2024 ESC guideline (EHJ 2024;45:3912–4018): nonelevated HBP, systolic <120 mmHg and diastolic <70 mmHg; elevated HBP, systolic 120-134 mmHg or diastolic 70–84 mmHg; and hypertension, systolic ≥130 mmHg or diastolic ≥85 mmHg. The frequency of nonelevated HBP was low: 10 patients (3.5%) among Blacks and 11 (7.1%) among Whites. Patients with nonelevated or elevated HBP are therefore pooled. The isovolumetric relaxation time is available in 175 Black and 74 White patients with nonelevated or elevated HBP and in 103 Black and 26 White patients with hypertension. *P*-values denote the significance of the unadjusted differences between Black and White patients. Continuously distributed variables are compared by Student's *t*-test and proportions by Fisher exact test. For abbreviations, see page 1. Significance of the difference between nonelevated and elevated HBP and hypertension by race.
[a]$P \leq 0.05$.
[b]$P \leq 0.01$.
[c]$P \leq 0\ .001$.

stroke volume (SV) increased less with HSBP in Black than White patients. The regression coefficients per 10 mmHg increase in HSBP were 1.265 vs. 1.272 mm ($P = 0.039$) for LAD, 1.272 vs. 4.642 ml ($P < 0.001$) for LAV, 0.489 vs. 1.833 mL/m^2 ($P = 0.005$) for LAVI, 0.299 vs. 1.047 mm ($P = 0.021$) for LVEDD, and 0.504 vs. 3.924 ml for SV. The relations of relative wall thickness and E/e' had a different direction in Black vs. White patients: 1.234 vs. −0.180 ($P = 0.007$) and 0.471 vs. −0.710 ($P = 0.008$), respectively. There were no racial differences in the associations with the HSBP for LV mass, LVMI, cardiac output, and the prevalence of LVDD.

Renal function

Table 3 lists the renal function measurements by home BP status and race. In Black patients with nonelevated and elevated BP and Black hypertensive patients, compared to the corresponding BP stratum of White patients, serum creatinine, eGFR, eGFR stage, and albuminuria were significantly higher. The racial profiles of the CKD grade, as derived by cross-classification of eGFR and albuminuria, according to the 2024 KDIGO Guideline, was significantly worse in Black than White patients (Table 3 and Fig. 2). In Black and White patients, serum creatinine ($P \geq 0.68$) or eGFR ($P \geq 0.72$) were not associated with HSBP.

DISCUSSION

This multinational analysis of high-risk individuals enrolled in the UPRIGHT-HTM trial [16,17], revealed distinct patterns of BP-mediated TOD among Black and White patients. Although both groups exhibited an elevated cardiometabolic risk profile, renal involvement was more prominent among Black patients, whereas cardiac structural and functional alterations prevailed among White patients. These findings suggest ethnicity-specific vulnerability of target organs in response to the cumulative influence of cardiovascular and metabolic risk factors. Black compared to White patients had a substantially higher cardiovascular

TABLE 3. Renal function by hypertension status and ethnicity

Characteristic	Nonelevated and elevated blood pressure			Hypertension		
	Black patients	White patients	*P*	Black patients	White patients	*P*
Number in group	181	118		106	36	
Glomerular filtration rate						
Serum creatinine, μmol/l	87.6 (27.2)	75.4 (14.7)	<0.001	86.6 (20.4)	77.2 (13.8)	0.011
eGFR, ml/min/1.73 m^2	87.7 (20.4)	95.0 (12.6)	<0.001	87.5 (17.8)	93.7 (11.0)	<0.050
≥90 ml/min/1.73 m^2, *n* (%)	95 (52.5)	93 (78.8)	<0.001	52 (49.1)	26 (72.2)	0.064
89–60 ml/min/1.73 m^2, *n* (%)	68 (37.6)	21 (17.8)		47 (44.3)	10 (27.8)	
59–45 ml/min/1.73 m^2, n (%)	12 (6.6)	4 (3.4)		6 (5.7)	0	
44–30 ml/min/1.73 m^2, *n* (%)	6 (3.3)	0		1 (0.9)	0	
Albuminuria, *n* (%)	38 (21.0)	1 (0.8)	<0.001	22 (20.8)	1 (2.8)	0.009
<30 mg/l, *n* (%)	143 (79.0)	117 (99.2)	<0.001	84 (79.2)	35 (97.2)	0.029
30–299 mg/l, *n* (%)	39 (19.9)	0		20 (18.9)	1 (2.8)	
≥300 mg/ml, *n* (%)	2 (1.1)	1 (0.8)		2 (1.9)	0	
KDIGO stage						
G1A1, *n* (%)	75 (41.4)	93 (78.8)	<0.001	41 (38.7)	25 (69.4)	0.070
G1A2, *n* (%)	21 (11.6)	0		9 (8.5)	1 (2.8)	
G1A3, *n* (%)	1 (0.6)	0		1 (0.9)	0	
G2A1, *n* (%)	53 (29.3)	20 (17.0)		37 (34.9)	10 (27.8)	
G2A2, *n* (%)	13 (7.2)	0		10 (9.4)	0	
G2A3, *n* (%)	0	1 (0.8)		1 (0.9)	0	
G3aA1, *n* (%)	11 (6.1)	4 (3.4)		5 (4.7)	0	
G3aA2, *n* (%)	1 (0.6)	0		1 (0.9)	0	
G3aA3, *n* (%)	0	0		0	0	
G3bA1, *n* (%)	4 (2.2)	0		0	0	
G3bA2, *n* (%)	1 (0.6)	0		0	0	
G3bA3, *n* (%)	1 (0.6)	0		0	0	

Values are means (SD) or frequency (%). Stratification of the home blood pressure (HBP) is based on the thresholds proposed by the 2024 ESC guideline (EHJ 2024; 45: 3912–4018): nonelevated HBP, systolic <120 mmHg and diastolic <70 mmHg; elevated HBP, systolic 120–134 mmHg or diastolic 70–84 mmHg; and hypertension, systolic ≥130 mmHg or diastolic ≥85 mmHg. The frequency of nonelevated or elevated HBP is low: 10 Black patients (3.5%) and 11 White patients (7.1%). Participants with nonelevated or elevated HBP are therefore pooled. Glomerular filtration rate is derived from serum creatinine by the race-free CKD-EPI Collaboration equation. The presence of albuminuria is determined from mid-morning urine samples, taken on three consecutive days or on only 2 days if the first two samples are negative (<30 mg/l). Renal function is classified according to the 2024 KDIGO guideline. G1–G3b refer to the classes of glomerular filtration rate and A1–A3 to the classes of albuminuria. Percentages do not always add up to 100% because of rounding. *P*-values denote the significance of the unadjusted differences between Black and White patients within each HBP category. Continuously distributed variables are compared by Student's *t*-test and proportions by Fisher exact test. None of the differences between the HBP categories are significant in Black ($0.12 \geq P \geq 0.90$) or White ($0.18 \leq P \leq 0.57$) patients.

risk profile (Table 1) and were less intensively treated for hypertension, dyslipidemia, and diabetes (Table S3, Supplemental Digital Content, http://links.lww.com/HJH/D53). Black patients often received monotherapy instead of combination therapy for hypertension and diabetes with fewer patients on β-blockers, inhibitors of the renin-angiotensin system, and metformin. The proportion of Black patients on statins was nearly two times smaller than in White individuals (132/287 [46.0%] vs. 116/154 [75.3%].

In this study, CKD stage was categorized according to the 2024 KDIGO guideline [21] and eGFR was derived from serum creatinine, using the race-free CKD-EPI Collaboration equation [32]. Race-free formulas were generated, because the equations with the race term led to an overestimation of eGFR in Blacks, potentially delaying treatment [34,35]. The prevalence of diabetes was similar in both races (60.3%), but as shown in Table 1, Blacks had more severe dyslipidemia and a higher systolic and diastolic home BP (130.0/81.0 vs. 126.5/77.4 mmHg), resulting in a higher prevalence of hypertension (36.9 vs. 23.4%). A recently published article [36] evaluated the previous eGFR equations with a race term [37,38] and the race-free equations [32,39,40]. eGFR was computed in multiethnic populations recruited in South Africa, Belgium, the Democratic Republic of Congo and the United States [36]. The new methods produced eGFR estimates, which predicted mortality and had near perfect reproducibility in the population as a whole. However, this article [36] also identified large intra-individual differences between the race-free eGFR estimates that may lead to misclassification of patients and showed that equations without race term tended to overestimate eGFR in Blacks [36]. Current recommendations favor equations based on serum cystatin C or a combination of serum creatine and cystatin C to improve accuracy [32,39,40], but many laboratories do not measure serum cystatin C, because this test is too expensive for routine application compared to serum creatinine [41].

The kidney emerged as the organ most susceptible to injury in Black patients, as reflected by a higher prevalence and severity of chronic kidney disease and albuminuria across the home BP categories. This observation aligns with previous reports indicating a heightened risk of hypertensive kidney disease in individuals of African ancestry [10,37,38], but extends prior knowledge by demonstrating this pattern in Blacks born and living in sub-Saharan Africa. Importantly, this setting minimizes the confounding influence of genetic admixture that characterizes many studies conducted in the United States [1–3]. In contrast, White patients showed a stronger association of indices of left ventricular remodeling and dysfunction with HSBP. These findings are consistent with the concept that myocardial structural adaptation may represent the dominant pathway of BP-mediated damage in populations of European ancestry. Differences in genetic and epigenetic variability [10,42],

(a) Blacks (n=287)

eGFR, mL/min/1.73 m²			Albuminuria, mg		
			A1	A2	A3
			<30	30-300	>300
G1	≥90		116 (40)	30 (10)	2 (0.7)
G2	60-89		90 (31)	23 (8.0)	1 (0.4)
G3a	45-59		16 (5.6)	2 (0.7)	0
G3b	30-44		5 (1.7)	1 (0.4)	1 (0.4)
G4	15-29		0	0	0
G5	<15		0	0	0

(b) Whites (n=154)

eGFR, ml/min/1.73 m²			Albuminuria, mg		
			A1	A2	A3
			<30	30-300	>300
G1	≥90		118 (77)	1 (0.7)	0
G2	60-89		30 (19)	0	1 (0.7)
G3a	45-59		4 (2.6)	0	0
G3b	30-44		0	0	0
G4	15-29		0	0	0
G5	<15		0	0	0

CKD grade: Low; Moderately increased; High; Very high

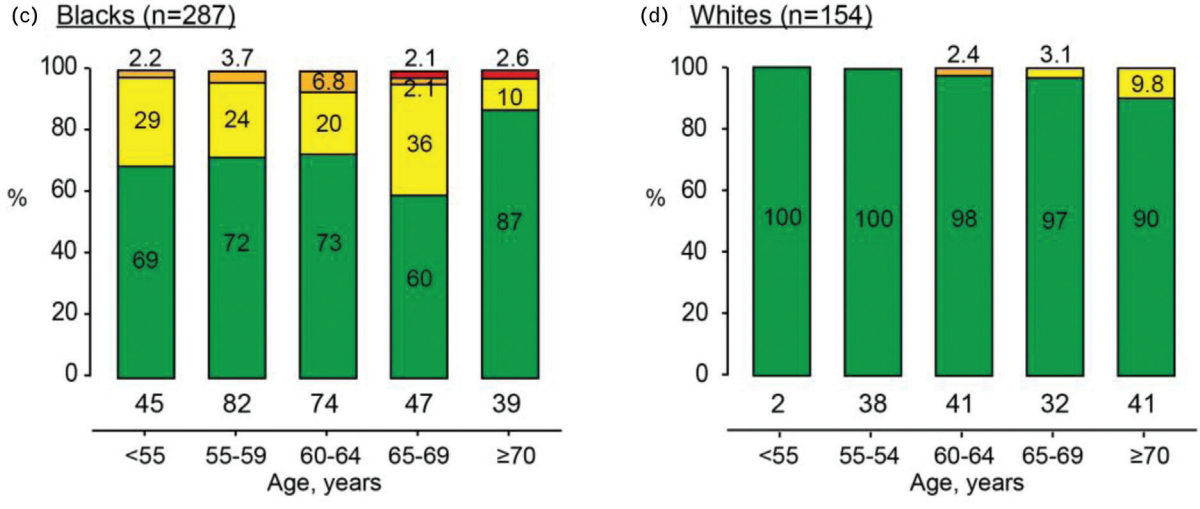

FIGURE 2 Grades of chronic kidney disease derived from the estimated glomerular filtration rate (eGFR) and albuminuria by race. The CKD grade is defined according to the 2024 KDIGO Guideline. (a, b) The number of patients (%) in cross-classified cells by race. (c, d) The percentage of patients at different CKD grades by age. CKD, chronic kidney disease.

dietary patterns, cardiovascular and metabolic risk factors, access to healthcare, and pharmacological treatment may further modulate these associations.

Among nonhypertensive patients, White compared to Black participants had greater LVEDD resulting in a slightly larger SV and faster mitral annular s′ (Table 2). On a continuous scale, relative wall thickness was slightly but significantly greater in Black than White patients (47.3 vs. 44.8), but on a categorical scale, using <0.44 vs. ≥0.44 as threshold, all Black and White patients, irrespective of their home BP status, showed concentric remodeling, a hallmark of an excessive LV load [43], most often attributable to hypertension. LVM and LVMI were similar in both races. Among nonhypertensive patients, LAV, LAVI, the isovolumetric relaxation time and E/e′ were greater in White than Black patients, suggesting a decline of diastolic LV function, but the prevalence of LVDD, as captured by a categorical variable derived from the age-specific E/A ratio as determined in a healthy reference population and E/e′ [28,29] was similar in Blacks and White patients (53.6 vs. 60.2%).

A remarkable finding was the racial heterogeneity in the associations of LV structure and systolic and diastolic LV with HSBP (Table S5, Supplemental Digital Content, http://links.lww.com/HJH/D53). The underlying reasons might be differences in the number of Black and White patients within each HSBP stratum (nonhypertensive vs. hypertensive patients), which affect the statistical power and significance levels, differences in the echocardiographic techniques, or in the number of home BP measurements characterizing each patient. However, some racially different slopes, expressed per 10-mmHg HSBP increment supported the findings reported in Table 2. There was a steeper slope in White than Black patients in relation to HSBP for LAD, the left atrium-to-aorta diameter ratio, mitral annular s′, SV, SVI, LAV and LAVI (Table S5, Supplemental Digital Content, http://links.lww.

com/HJH/D53). Remarkably, the relations of relative wall thickness and E/e′ with HSBP had a different direction in Black vs. White patients, being positive in the former and negative in the latter.

A recent publication addressed the intra- and inter-observer repeatability of the echocardiographic traits at the Nigerian recruitment sites [30]. The coefficients of variation for LVM were approximately 30%, while the intraclass correlation coefficients ranged from 0.74 to 0.84. The kappa statistics for LV hypertrophy varied from 0.49 to 0.64. For the LV ejection fraction, intra- and inter-rater coefficients of variation ranged from 13.0% to 14.5%, the intra-class correlations from 0.60 to 0.69. LVDD reproducibility was moderate, with kappa values around 0.50. These findings were consistent with most of the worldwide literature reviewed in the same article [30] and with a recently completed study conducted at First Department of Cardiology, Interventional Electrocardiology and Hypertension, Jagellonian University, Kraków, Poland [31].

CKD and LVDD often coexist [28,29,44,45] and share the same risk factors, in particular older age, diabetes mellitus, hypertension, and obesity. Hemodynamic and nonhemodynamic mechanisms underpin the two-way interaction between the kidney and the heart [46]. A decline in LV function and cardiac output activates the sympathetic nervous system and the renin-angiotensin system. The ensuing sodium and water retention and expansion of the circulating volume, although maintaining renal perfusion, increase LV afterload and aggravate LV dysfunction [46]. Furthermore, LVDD with preserved ejection fraction is accompanied with higher pressure in the central venous system [47], which in turn may increase renal intratubular pressure and reduce the ultrafiltration pressure, thereby reducing glomerular filtration. The drivers of the nonhemodynamic cardiorenal connection are the renin-angiotensin system, sympathetic nervous tone, inflammation, and the imbalance between nitric oxide and reactive oxygen production [48].

A remarkable finding, which was not abolished by removing all patients on β-blockers from the analysis, was the higher heart rate in Black than White patients. The ambient temperature is a possible explanation for this observation. The annual average temperature is within the 5–9°C range in Poland, 12°C in Mediterranean Slovenia, within the 16–18°C range in La Plata, Argentina, but as high as 26.9°C in Nigeria and around 23°C in the Potchefstroom area in South Africa (World Meteorological Organization). Exposure to high temperature leads to stimulation of the sympathetic system and parasympathetic tone withdrawal [49]. In 274 randomly recruited Flemish (50% women; mean age, 40.6 years), the odds of endothelial dysfunction, as assessed by the flow-mediated vasodilatation in the brachial artery, increased by 58% (95% CI, 4–141%; $P = 0.03$) for each 10°C increment in the ambient daily temperature averaged over 1 week [50].

Hypertension and diabetes drive the growing burden of noncommunicable disease in sub-Saharan Africa. In these countries and in other middle- or low-income countries, a framework shift is necessary to achieve better health outcomes through personalized care with focus on prevention, availability of generic low-cost medicines, easier accessibility of primary care facilities, and improved strategies for organizing healthcare [51]. Furthermore, an analysis of the Food and Agricultural Organization database and health indicators supplied by UNICEF and WHO revealed different dietary patterns in sub-Saharan Africa: a Westernized diet with high content of refined sugars, sweeteners, alcohol, meat, animal fats, eggs, and dairy in South Africa, and a high consumption of cereals, nuts, fish and vegetable oils in Nigeria, particularly in coastal areas [52]. The EAT Lancet Commission proposed that healthy diets should have an appropriate caloric intake and consist of a diversity of plant-based foods, low amounts of animal source foods, unsaturated rather than saturated fats, and small amounts of refined grains, highly processed foods, and added sugars [53].

Strengths and limitations

What sets this study apart is the involvement of Black patients, born and living in sub-Saharan Africa, and European and Argentinian Whites. While genetic admixture cannot be excluded in South African Black participants, presumably, it is less of a confounding factor compared with studies conducted in the United States [1,3]. Additionally, the large number of HTM readings over a long period combined with the refined ESC 2024 guidelines for classification of the BP status [19] are undoubtedly other strong points. However, the current results must be interpreted within the context of several limitations. First, although all recruitment sites applied the same eligibility criteria, the differences between Black and White patients should not only be attributed to race, because the contributions of environmental factors, lifestyle, diet, and background drug treatment undoubtedly all played a role. Second, the definition of LVDD was derived and validated in White Europeans [28,29], using a healthy study sample as reference. However, similar studies in sub-Saharan Blacks with a healthy reference group still have to be performed. Third, the current analysis did not include 88 Asian patients recruited in the Republic of China, because of delays in submitting eCRF data. However, Table S2, Supplemental Digital Content, http://links.lww.com/HJH/D53 shows that patients analyzed and not analyzed had largely comparable risk profiles. Finally, racial heterogeneity in the associations of LV structure and systolic and diastolic LV function with the HSBP made hampered a unified interpretation of these associations. However, the current study focused on racial differences in the regression slopes, evaluated by a race-by-HSBP interaction term. Significance of the associations was not corrected for multiple testing, because LV structural and functional features are highly inter-correlated, so that each test does not introduce an independent opportunity for a type-I error [54].

CONCLUSIONS

Given the eligibility criteria, UPRIGHT-HTM patients had a high-risk profile. The target organ with the highest vulnerability to the multiple risk factors was the kidney in Black patients and primarily the heart in White patients. Antihypertensive, antidiabetic and lipid-lowering medicines were less used in sub-Saharan Black patients compared to European and Argentinian White individuals. These findings

highlight the opportunity to intensify risk factor management in sub-Saharan Africa, using a personalized approach with focus on prevention, improved strategies for delivering healthcare, facilitating accessibility to primary care, mobilizing stakeholders, and educating people on the role of risk factors.

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Data availability: All relevant data are within the paper. Informed consent given by study participants did not include data sharing with third parties. However, anonymized data can be made available to investigators for focused noncommercial research based on a motivated request to be submitted to J.A.S., pending clearance by the Ethics Committee of the University Hospitals, Leuven, Belgium.

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The UPRIGHT-HTM investigators are listed in references 12 and 13.

Conflicts of interest

There are no conflicts of interest.

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