Cardiovascular health in urban Suriname

The Healthy Life in Suriname (HELISUR) study

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CHAPTER 4

Hypertension and cardiovascular risk profile in urban Suriname: the HELISUR study


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ABSTRACT

Introduction:
Hypertension is the leading risk factor responsible for premature death worldwide, but its burden has shifted to low- and middle-income countries. Therefore, we studied hypertension and cardiovascular risk in the population of Suriname, a middle-income country with a predominantly urban population of African and Asian ancestry.

Methods:
A random sample of 1,800 non-institutionalized men and women aged 18–70 years was selected to be interviewed at home and examined at the local hospital for cardiovascular risk factors, asymptomatic organ damage, and cardiovascular disease.

Results:
The 1,157 participants examined (37% men) were mainly of self-defined Asian (43%) or African (39%) ancestry, mean age 43 years (SD 14). The majority of the population (71%) had hypertension or prehypertension, respectively, 40% and 31%. Furthermore, 72% was obese or overweight, while 63% had diabetes or prediabetes. Only 1% of the adult population had an optimal cardiovascular risk profile. Hypertension awareness, treatment, and control were respectively 68%, 56%, and 20%. In line with this, 22% of the adult population had asymptomatic organ damage, including increased arterial stiffness, left ventricular hypertrophy, microalbuminuria, or asymptomatic chronic kidney disease.

Conclusions:
In this first extensive cardiovascular assessment in the general population of this middle-income Caribbean country, high prevalence of hypertension with inadequate levels of treatment and control was predominant. The findings emphasize the need for collaborative effort from national and international bodies to prioritize the implementation of affordable and sustainable public health programs that combat the escalating hypertension and cardiovascular risk factor burden.
INTRODUCTION

Hypertension is the main preventable cause of disability and premature death worldwide, but the prevalence is increasing in low- and middle-income countries (LMIC), in contrast to a slight decrease in high-income regions. At present, more than 80% of the global burden of hypertension occurs in LMIC, with half of the mortality occurring at a relatively young age between 30 and 65 years. The economic burden of hypertension and cardiovascular disease (CVD) in terms of mortality- and disability-adjusted life years in these resource-limited settings is high. Although there are ample data from migrants of African, Asian, and Caribbean descent in North America and Europe, these data may not be applicable to populations in LMIC, where socioeconomic and environmental conditions as well as health systems and policies are different. Therefore, the United Nations emphasized the need for sufficient data on cardiovascular risk factors from these regions to combat the increasing cardiovascular mortality. Suriname is a multiethnic middle-income country in South America, where CVD is the main cause of death among its population of predominantly African and Asian ancestry. However, there are scant data on the prevalence of hypertension and other cardiovascular risk factors. Therefore, we assessed hypertension and cardiovascular health status in a random sample of the general population.

METHODS

Ethical clearance
The study was conducted according to the principles of the Declaration of Helsinki (59th WMA General Assembly, Seoul, October 2008) and in accordance with the Medical Research Involving Human Subjects Act. Ethical clearance was obtained from the Ethics Committee of the Ministry of Health in Suriname in 2012 (Approval nr. VG021-2012). Written informed consent was obtained from all participants.

The HELISUR study
The Healthy Life in Suriname (HELISUR) study is a cross-sectional population-based study conducted in the capital Paramaribo, the only main city in the country. The majority of the Surinamese population (73%) lives in an urban setting. Study procedures were published previously. A flowchart of the sampling procedure is depicted in Figure 1. In brief, a random sample of 1,800 non-institutionalized urban men and women aged 18–70 years was selected to be interviewed at home (response rate: 72%). The interview included demographic, socioeconomic, dietary, and health-related questions. Subsequently, participants were invited to be examined in our research facility at the
local Academic Hospital (response rate: 78%). The physical examination included anthropometric, medical, and physiological measurements including noninvasive hemodynamics, as well as laboratory tests administered by trained medical personnel. Main outcomes were the prevalence of hypertension, other cardiovascular risk factors, asymptomatic organ damage and CVD by sex, age, and ancestry.

Study procedures

Participants were seen by a trained investigator in the morning in the fasting state. Questionnaires filled out at home were checked and updated. Blood pressure was measured twice in the sitting position with an automated oscillometric device (WatchBP Office; Microlife AG, Widnau, Switzerland) and an appropriately adjusted cuff size on the left upper arm supported at heart level. Height and weight were measured twice to the nearest 0.1 cm and 0.1 kg, respectively, with participants wearing underwear. Body mass index was computed as mean weight (in kilograms) divided by the square of the mean height (in meters). We recorded 12-leads electrocardiograms, and estimated the aortic pulse wave velocity twice noninvasively in the supine position by analysis of the

Figure 1. Flowchart of the sampling procedure. The capital of Suriname is divided into 1,200 Census enumeration areas, consisting of 100–150 households. General Bureau of Statistics randomly selected 18 enumeration areas stratified by ancestry. Households were approached consecutively until 100 subjects per enumeration area were included (response rate: 72%). Preliminary assessment included the interviews administered at the subjects’ home. Eligible persons were subsequently invited to visit the hospital for the examination (response rate: 78%).
oscillometric pressure curves registered on the right upper arm, using the Arteriograph (TensioMed, Budapest, Hungary). Fasting venous blood samples and morning spot urine were collected for laboratory measurements.

**Definitions and outcome parameters**

The population of the capital Paramaribo consists of persons of African (43%), Asian (33%), and other ancestry. Persons of African ancestry are locally considered to be Creole or Maroon. Maroons recently migrated to the capital and therefore tend to have less admixture with other populations than Creoles. Persons of Asian ancestry migrated from India or the island of Java to Suriname in the late 19th century and early 20th century. For the current analysis, we used self-defined ancestry.

We used the following definitions: hypertension, systolic blood pressure ≥140 mm Hg, or diastolic blood pressure ≥90 mm Hg, or receiving antihypertensive drug therapy; prehypertension, systolic blood pressure 120–139 mm Hg or diastolic blood pressure 80–89 mm Hg, without antihypertensive drug therapy. Controlled hypertension included subjects with hypertension using antihypertensive medication with a systolic blood pressure <140 mm Hg and diastolic blood pressure <90 mm Hg. Furthermore, we used ethnic-specific cutoff values for body mass index: obesity ≥30 kg/m² in African-Surinamese participants (i.e., Creole and Maroons) and ≥27.5 kg/m² in Asian-Surinamese participants (i.e., Surinamese of South Asian and Indonesian ancestry). Overweight was defined as body mass index 25.0–29.9 kg/m² and 23.0–27.4 kg/m², respectively, in African and Asian ancestry participants. We considered participants to be diabetic if HbA1c ≥6.5% or when glucose-lowering medication was used, while prediabetes was defined as HbA1c 5.7–6.4% without the use of antihyperglycemic drugs. Diabetes control was defined as HbA1c <7.0%. Finally, dyslipidemia was defined as having at least one of the following: total cholesterol ≥6.2 mmol/l, low-density lipoprotein cholesterol ≥4.1 mmol/l, high-density lipoprotein cholesterol <1.0 mmol/l, triglycerides ≥2.3 mmol/l, or the use of lipid-lowering drugs. Borderline dyslipidemia was defined as total cholesterol 5.2–6.2 mmol/l, low-density lipoprotein cholesterol 3.4–4.1 mmol/l, high-density lipoprotein cholesterol 1.0–1.6 mmol/l, or triglycerides 1.7–2.3 mmol/l without the use of lipid-lowering drugs. Smoking was defined as using tobacco on daily basis.

An adverse cardiovascular risk profile was defined as the presence of any of the risk factors hypertension, obesity, diabetes, dyslipidemia, or smoking. If prehypertension, overweight, prediabetes, or borderline dyslipidemia was present, this was considered a suboptimal cardiovascular risk profile. An optimal cardiovascular risk profile was the absence of any of these risk factors.
Asymptomatic organ damage was defined as the presence of either increased arterial stiffness (pulse wave velocity $> 10$ m/s$^{19}$), left ventricular hypertrophy (LVH, Sokolow–Lyon criteria: $S$ in $V_1$ or $V_2 + R$ in $V_5$ or $V_6$ [whichever is larger] $\geq 35$ mm, or $R$ in aVL $\geq 11$ mm$^{20}$), microalbuminuria (30–300 mg/dl in spot urine$^{21}$), or asymptomatic chronic kidney disease (CKD), according to the European Society of Hypertension/European Society of Cardiology guidelines.$^{13}$ Estimated glomerular filtration rate was calculated using the CKD-EPI equation, and asymptomatic CKD was defined as estimated glomerular filtration rate 30–59 ml/min/1.73 m$^2$.21,22

We defined CVD as history of coronary heart disease (history of myocardial infarction, or coronary bypass surgery), history of stroke, or the presence of severe CKD. Severe CKD was defined as estimated glomerular filtration rate $<30$ ml/ min/1.73 m$^2$.22

Finally, educational level was assessed through the highest completed level of education, and categorized into 2 groups according to the International Standard Classification of Education (ISCED): low ($\leq$12 years of education, ISCED 1–2) or high ($>12$ years of education, ISCED 3).23

**Statistical analysis**

Descriptive characteristics were computed for the total group (primary) and for African (with subgroups of Creole and Maroon) vs. Asian (with subgroups of South Asian and Indonesian) ancestry participants. We calculated the prevalence and the odds ratios of hypertension and other cardiometabolic risk factors, asymptomatic organ damage, and CVD for Asian vs. African ancestry participants, using the unadjusted and adjusted model for sex, age, body mass index, and educational level. Model fit was assessed with the appropriate goodness-of-fit test for logistic regression models. Statistical analyses were performed with the SPSS statistical software package for Windows, version 20.0 (SPSS, Chicago, IL).

**RESULTS**

We analyzed data of 1,157 participants, 429 men and 728 women, with a mean age of 43 ± 14 years, as summarized in Table 1. Blood pressure was not optimal in 71% of the participants: 40% had hypertension and a further 31% was prehypertensive (Table 2, Figure 2). Hypertension awareness, treatment, and control were respectively 68, 56, and 20%. Hypertension prevalence did not differ by sex ($P = 0.33$) but men were more often prehypertensive than women (37 vs. 28%; $P < 0.01$). Figure 3 depicts hypertension prevalence by age. In logistic regression analysis, Asian ancestry participants were
Table 1. Characteristics of the participants by ancestry

<table>
<thead>
<tr>
<th></th>
<th>African</th>
<th>Creole (n=245)</th>
<th>Maroons (n=216)</th>
<th>South Asian (n=392)</th>
<th>Indonesian (n=105)</th>
<th>Total (n=1157)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Women †</td>
<td>59.2$^{ac}$</td>
<td>71.3$^b$</td>
<td>60.7$^c$</td>
<td>66.7$^d$</td>
<td>62.8</td>
<td>0.03</td>
<td></td>
</tr>
<tr>
<td>Age, years ‡</td>
<td>44.5 ± 13.5$^{ab}$</td>
<td>39.5 ± 13.8$^c$</td>
<td>43.5 ± 13.2$^a$</td>
<td>47.6 ± 13.0$^b$</td>
<td>43.3 ± 13.8</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td>High education †</td>
<td>19.2$^{ab}$</td>
<td>8.8$^b$</td>
<td>10.2$^a$</td>
<td>14.3$^c$</td>
<td>14.4</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>Systolic BP, mmHg ‡</td>
<td>132.7 ± 20.3</td>
<td>129.3 ± 22.6</td>
<td>128.5 ± 19.1</td>
<td>129.5 ± 19.2</td>
<td>129.8 ± 20.2</td>
<td>0.08</td>
<td></td>
</tr>
<tr>
<td>Diastolic BP, mmHg ‡</td>
<td>83.1 ± 11.6</td>
<td>80.6 ± 13.5</td>
<td>81.3 ± 11.0</td>
<td>80.2 ± 10.7</td>
<td>81.3 ± 11.6</td>
<td>0.06</td>
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</tr>
<tr>
<td>BMI, kg/m$^2$ ‡</td>
<td>28.1 ± 6.6$^{ab}$</td>
<td>28.7 ± 6.8$^a$</td>
<td>27.2 ± 5.6$^{b}$</td>
<td>26.7 ± 4.7$^{b}$</td>
<td>27.8 ± 6.2</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>Fasting glucose, mmol/L ‡</td>
<td>5.4 ± 1.8$^a$</td>
<td>4.8 ± 0.6$^b$</td>
<td>6.2 ± 2.8$^c$</td>
<td>5.9 ± 2.2$^{ac}$</td>
<td>5.6 ± 2.2</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td>Total cholesterol, mmol/L ‡</td>
<td>4.5 ± 1.1$^a$</td>
<td>4.4 ± 1.0$^b$</td>
<td>5.0 ± 1.0$^c$</td>
<td>5.1 ± 1.0$^{ab}$</td>
<td>4.7 ± 1.1</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td>LDL-cholesterol, mmol/L ‡</td>
<td>3.1 ± 1.0$^{a}$</td>
<td>2.9 ± 0.9$^{b}$</td>
<td>3.6 ± 0.9$^{a}$</td>
<td>3.6 ± 0.9$^{b}$</td>
<td>3.3 ± 0.9</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td>HDL-cholesterol, mmol/L ‡</td>
<td>1.2 ± 0.4$^a$</td>
<td>1.2 ± 0.3$^b$</td>
<td>1.1 ± 0.2$^{b}$</td>
<td>1.2 ± 0.3$^{b}$</td>
<td>1.2 ± 0.3</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td>Triglycerides, mmol/L ‡</td>
<td>1.1 ± 0.7$^a$</td>
<td>0.9 ± 0.5$^{b}$</td>
<td>1.5 ± 0.9$^{a}$</td>
<td>1.7 ± 2.1$^{b}$</td>
<td>1.3 ± 1.0</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td>PWV, m/s ‡</td>
<td>8.4 ± 2.3</td>
<td>8.1 ± 2.4</td>
<td>8.6 ± 2.5</td>
<td>8.4 ± 2.6</td>
<td>8.3 ± 2.6</td>
<td>0.21</td>
<td></td>
</tr>
<tr>
<td>eGFR, mL/min/1.73 m$^{2*}$</td>
<td>112.7 ± 23.5$^a$</td>
<td>117.8 ± 24.0$^{a}$</td>
<td>104.0 ± 20.2$^{b}$</td>
<td>100.5 ± 18.4$^{b}$</td>
<td>107.9 ± 22.2</td>
<td>&lt;0.01</td>
<td></td>
</tr>
</tbody>
</table>

Footnote Table 1. † Includes participants of other/mixed ancestry (n = 199). † Values are percentages. ‡ Values are means with SDs. Differences were tested across the four ancestry groups with $^a$ being significantly different from $^b$ and $^c$; $^b$ being significantly different from $^a$ and $^c$; $^c$ being significantly different from $^a$ and $^b$; all at a level of $P < 0.05$, depicted in bold. BMI, body mass index; eGFR, estimated glomerular filtration rate; HDL, high-density lipoprotein; LDL, low-density lipoprotein; PWV, pulse wave velocity.
Table 2. Cardiovascular risk factors, asymptomatic organ damage, and CVD

<table>
<thead>
<tr>
<th></th>
<th>African Creole (n=245)</th>
<th>Maroons (n=216)</th>
<th>South Asian (n=392)</th>
<th>Indonesian (n=105)</th>
<th>Total (n=1157)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
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<td>Hypertension</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>0.05</td>
</tr>
<tr>
<td>Aware†</td>
<td>63.8</td>
<td>72.8</td>
<td>71.0</td>
<td>67.4</td>
<td>68.0</td>
<td>0.28</td>
</tr>
<tr>
<td>Treated†</td>
<td>53.4</td>
<td>51.9</td>
<td>60.7</td>
<td>60.5</td>
<td>55.9</td>
<td>0.40</td>
</tr>
<tr>
<td>Controlled‡</td>
<td>45.2 (24.1)</td>
<td>33.3 (17.3)</td>
<td>29.5 (18.1)</td>
<td>38.5 (23.3)</td>
<td>35.7 (20.0)</td>
<td>0.28</td>
</tr>
<tr>
<td>Obesity</td>
<td>36.7</td>
<td>38.9</td>
<td>39.0</td>
<td>38.1</td>
<td>36.8</td>
<td>0.95</td>
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<tr>
<td>Diabetes mellitus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Aware†</td>
<td>40.7</td>
<td>33.3</td>
<td>68.6</td>
<td>61.5</td>
<td>58.8</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Treated†</td>
<td>40.7</td>
<td>16.7</td>
<td>64.6</td>
<td>61.5</td>
<td>54.4</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Controlled‡</td>
<td>22.7 (46.3)</td>
<td>50.0 (66.7)</td>
<td>15.8 (26.3)</td>
<td>31.3 (34.6)</td>
<td>20.6 (35.6)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td></td>
<td></td>
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<td>&lt;0.01</td>
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<tr>
<td>Smoking</td>
<td>22.9</td>
<td>7.9</td>
<td>20.9</td>
<td>14.3</td>
<td>18.0</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Early organ damage:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Increased arterial stiffness</td>
<td></td>
<td>15.9</td>
<td>17.9</td>
<td>19.0</td>
<td>16.6</td>
<td>0.41</td>
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<tr>
<td>LVH</td>
<td>3.3</td>
<td>9.3</td>
<td>1.3</td>
<td>1.0</td>
<td>3.4</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Microalbuminuria</td>
<td>2.9</td>
<td>3.7</td>
<td>1.8</td>
<td>2.9</td>
<td>2.8</td>
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<tr>
<td>Asymptomatic CKD</td>
<td>1.6</td>
<td>0.9</td>
<td>1.0</td>
<td>1.9</td>
<td>1.4</td>
<td>0.80</td>
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<tr>
<td>CVD:</td>
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<tr>
<td>Severe CKD</td>
<td>0.0</td>
<td>0.5</td>
<td>1.1</td>
<td>0.0</td>
<td>0.4</td>
<td>0.29</td>
</tr>
<tr>
<td>CHD</td>
<td>1.2</td>
<td>0.5</td>
<td>4.6</td>
<td>1.0 (2.0)</td>
<td>2.0</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Stroke</td>
<td>3.3</td>
<td>0.5</td>
<td>2.8</td>
<td>1.0</td>
<td>2.5</td>
<td>0.12</td>
</tr>
</tbody>
</table>

Footnote Table 2. Values are percentages. † Includes participants of other/mixed ancestry (n = 199). ‡ Percentage of subjects with hypertension/diabetes mellitus. † Percentage of treated subjects (percentage of all subjects with hypertension/diabetes mellitus). Differences were tested across the four ancestry groups with † being significantly different from ‡ and ‡ being significantly different from † and ‡; ‡ being significantly different from † and ‡; all at a level of P < 0.05, depicted in bold.

CHD, coronary heart disease; CKD, chronic kidney disease; CVD, cardiovascular disease; LVH, left ventricular hypertrophy.
In participants of 40 years and younger, hypertension was more often seen in African ancestry than in Asian ancestry participants (21% vs. 14%; $P = 0.06$).

**Figure 2.** Cardiovascular risk profile of the general population. Bars are % of the total population. The majority of the population had an adverse cardiovascular risk profile, mainly due to a high hypertension and dyslipidemia prevalence. DM, diabetes mellitus; HT, hypertension; PreDM, prediabetes; PreHT, prehypertension.

**Figure 3.** Cardiovascular risk factors, asymptomatic organ damage, and CVD by age. Bars are % of the total population. Cardiovascular risk factors, asymptomatic organ damage, and CVD were more prevalent in participants above 40 years than in participants of 40 years and below. *Significantly higher in participants above 40 years ($P < 0.01$). CVD, cardiovascular disease.

less likely to have high blood pressure than participants of African ancestry (**Figure 4**). In participants of 40 years and younger, hypertension was more often seen in African ancestry than in Asian ancestry participants (21% vs. 14%; $P = 0.06$).
The majority of the population (72%) was overweight (35%) or obese (37%). Four percent was underweight. A third of the population of ≤40 years was obese, and women were more often obese than men (46 vs. 21%; \( P < 0.01 \)). There were no differences between the 4 ancestry groups or between African and Asian ancestry participants.

A smaller proportion of the population had diabetes, 22%, but 41% of the participants had prediabetes. The majority of the participants with diabetes was aware of their condition (59%) and received treatment (54%), but only 21% of those treated were controlled (11% of all diabetics). There were no differences in diabetes prevalence between men and women (\( P = 0.19 \)), but Asian ancestry participants were more likely to have diabetes than participants of African ancestry (30 vs. 18%; \( P < 0.01 \)). No ethnic differences existed in the proportion of participants ≤40 years with diabetes (9% Asian vs. 7% African; \( P = 0.53 \)).
Ninety-five percent of the population had dyslipidemia (41%) or borderline lipid abnormalities (54%). Men (51%) and Asian ancestry participants (51%) had more dyslipidemia than women (37%; \( P < 0.01 \)) and African ancestry participants (33%; \( P < 0.01 \)). Moreover, dyslipidemia was more often prevalent in young Asian ancestry participants ≤40 years (39%) compared to their African counterparts (27%; \( P = 0.01 \)). Tobacco use was seen in 18% of the population. Smokers were more often men (35% vs. 8% in women; \( P < 0.01 \)), but no differences were apparent between ancestry or age groups.

The majority of the population (77%) had at least one cardiovascular risk factor, and this was 62% in the population subgroup of 40 years and younger. The inclusion of prehypertension, overweight, borderline dyslipidemia, and prediabetes resulted in 1% of the population with optimal cardiovascular parameters (2% in the population ≤40 years). Men (83%) and Asian ancestry participants (82%) had a higher risk factor burden than women (74%; \( P < 0.01 \)) or African ancestry participants (74%; \( P < 0.01 \)).

We found asymptomatic organ damage in 22% of the participants (9% in the population ≤40 years). In line with the high prevalence of hypertension, the majority had increased arterial stiffness (17%), followed by LVH and microalbuminuria (both 3%) and asymptomatic CKD (1%). Despite the higher risk factor burden, Asian ancestry participants were less likely to have asymptomatic organ damage than their African counterparts (21% vs. 24%; \( P = 0.21 \)). Moreover, asymptomatic organ damage was more often seen in African ancestry participants ≤40 years than in their Asian counterparts (13% vs. 5%; \( P < 0.01 \)).

Established CVD was found in 5% of the participants, including stroke (3%), coronary heart disease (2%), and severe CKD (0.4%). CVD occurred more often in men than in women (7 vs. 3%; \( P < 0.01 \)), mainly due to a higher prevalence of stroke (4 vs. 2% in respectively men and women; \( P < 0.01 \)), and in Asian ancestry participants than in African ancestry participants (6 vs. 3%; \( P = 0.01 \)), mainly due to a higher prevalence of coronary heart disease (4 vs. 1%; \( P < 0.01 \)). Multiple imputations did not alter our findings.

**DISCUSSION**

Our elaborate cardiovascular assessment indicates that the burden of hypertension and other cardiometabolic risk factors is very high in this middle-income country. The majority of the population had hypertension (40%) or prehypertension (31%). This is in line with the finding that stroke is the largest contributor to cardiovascular mortality in this country, accounting for 46% of all cardiovascular deaths. With only 1% of the adult
population now having an optimal cardiovascular risk factor profile, the coexistence of multiple cardiovascular risk factors, also in the young, and the low levels of control found in this study are ingredients for an increase in CVD of epidemic proportions in the coming decades, unless drastic primary preventive measures are taken.

At present, the main focus of health care for CVD in many LMIC is secondary prevention. Patients enter treatment programs after becoming symptomatic, when costly high-technology interventions are needed. However, the large proportion of individuals with suboptimal cardiovascular parameters underscores the importance and potential benefit of intensified screening and reduction of risk factor burden at an earlier stage. In line with this, clinicians could extend their case-finding strategy to virtually all adult patients as very few participants in our random sample (<5%) appeared to have an optimal cardiovascular risk profile. Importantly, while participants of Asian ancestry had a higher cardiovascular risk factor burden, the participants of African ancestry had more organ damage (arterial stiffness, LVH, microalbuminuria, and CKD). This suggests that clinicians should be aware of asymptomatic organ damage in the African ancestry patients despite an apparently lower risk factor burden.

This study has several strengths. First, we assessed the full spectrum of cardiovascular risk factors, and added noninvasive methods to assess early cardiovascular damage, including arterial stiffness. However, we need to establish whether these noninvasive assessments may help stratify cardiovascular risk profiles. Second, in Suriname, people of African and Asian ancestry live under relatively similar socioeconomic circumstances. This setting is a rather unique feature of our study as in previous studies these ancestry groups are predominantly compared with Caucasians with great differences in socioeconomic backgrounds. Lastly, we reported the complete cardiovascular risk factor profile, and thus provide high-quality cardiovascular data from a middle-income country in a region from which such data are scarce. A limitation is the assessment of hypertension and diabetes through the measurement of blood pressure and HbA1c within one visit. Although repeated tests are required for confirmation of the diagnosis, with epidemiological studies this is a common approach. Also, the diagnosis of LVH was based on ECG data rather than echocardiographic data. It should be noted that the use of Sokolow-Lyon criteria has a lower specificity and a greater sensitivity in people of African ancestry compared to Asians for detecting LVH. This might have overestimated the prevalence of LVH in the African ancestry group.

In conclusion, in order to provide data for preventive and intervention strategies, we conducted an extensive examination of cardiovascular risk factors, asymptomatic organ damage, and CVD in the population of Suriname. We found that only 1% of the popula-
tion had an optimal cardiovascular risk profile. Our findings underscore the urgent need for the implementation of cost-effective preventive strategies in LMIC to reduce blood pressure and other cardiovascular risk factors and prevent a surge in CVD of epidemic proportions.
REFERENCES


