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Comparison of Risk Scores for the Prediction of Stroke in African Americans: Findings from the Jackson Heart Study

Randi E. Foraker, PhD, MA, FAHA¹, Melissa Greiner, MS², Mario Sims, PhD³, Katherine L. Tucker, PhD⁴, Amytis Towfighi, MD⁵, Aurelian Bidulescu, MD, PhD, MPH⁶, Abigail B. Shoben, PhD⁷, Sakima Smith, MD, MPH⁸, Sameera Talegawkar, PhD, MHSc⁹, Chad Blackshear, MS¹⁰, Wei Wang, PhD¹⁰, N. Chantelle Hardy, MPH², and Emily O'Brien, PhD²

Randi E. Foraker: rforaker@cph.osu.edu; Melissa Greiner: Melissa.Greiner@duke.edu; Mario Sims: msims2@umc.edu; Katherine L. Tucker: Katherine_Tucker@uml.edu; Amytis Towfighi: atowfighi@dhs.lacounty.gov; Aurelian Bidulescu: abidules@indiana.edu; Abigail B. Shoben: ashoben@cph.osu.edu; Sakima Smith: Sakima.Smith@osumc.edu; Sameera Talegawkar: stalega1@jhu.edu; Chad Blackshear: cblackshear@umc.edu; Wei Wang: wwang@umc.edu; N. Chantelle Hardy: chantelle.hardy@duke.edu; Emily O'Brien: emily.obrien@duke.edu

¹The Ohio State University, Division of Epidemiology, Columbus, OH, USA

²Duke Clinical Research Institute, Durham, NC, USA

³University of Mississippi Medical Center, Department of Medicine, Birmingham, AL, USA

⁴University of Massachusetts at Lowell, Department of Clinical Laboratory and Nutritional Sciences, Lowell, MA, USA

⁵University of Southern California, Keck School of Medicine, Los Angeles, CA, USA

⁶Indiana University, School of Public Health, Bloomington, IN, USA

⁷The Ohio State University, Division of Biostatistics, Columbus, OH, USA

⁸The Ohio State University Medical Center, Columbus, OH, USA

⁹Johns Hopkins University, Bloomberg School of Public Health, Baltimore, MD, USA

¹⁰University of Mississippi Medical Center, Center of Biostatistics and Bioinformatics, Jackson, MS, USA

Abstract

Background—Evidence from existing cohort studies support the prediction of incident coronary heart disease and stroke using 10-year cardiovascular disease (CVD) risk scores and the American Heart Association/American Stroke Association's cardiovascular health (CVH) metric.

Reprints: Randi Foraker, The Ohio State University, 1841 Neil Avenue, 334 Cunz Hall, Columbus, OH 43210, Phone: 614-292-9916, Fax: 614-688-3533, rforaker@cph.osu.edu.

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Methods—We included all Jackson Heart Study participants with complete scoring information at the baseline study visit (2000-2004) who had no history of stroke (n=4,140). We used Kaplan-Meier methods to calculate the cumulative incidence of stroke, and used Cox models to estimate hazard ratios and 95% confidence intervals for stroke according to CVD risk and CVH score. We compared the discrimination of the two models according to the Harrell c-index, and plotted predicted versus observed stroke risk calibration plots for each of the two models.

Results—The median age of the African-American participants was 54.5 years, and 65% were female. The cumulative incidence of stroke increased across worsening categories of CVD risk and CVH. A one-unit increase in CVD risk increased the hazard of stroke (1.07, 1.06-1.08), while each one-unit increase in CVH corresponded to a decreased hazard of stroke (0.76, 0.69-0.83). As evidenced by the c-statistics, the CVH model was less discriminating than the CVD risk model (0.59, 0.55-0.64 versus 0.79, 0.76-0.83).

Conclusions—Both scores were associated with incident stroke in a dose-response fashion; however, the CVD risk model was more discriminating than the CVH model. The CVH score may still be preferable for its simplicity in application to broad patient populations and public health efforts.

Keywords

Risk scores; stroke; health disparities

Evidence from existing cohort studies support the prediction of incident coronary heart disease (CHD) and stroke using 10-year cardiovascular disease (CVD) risk scores (1) and the American Heart Association (AHA)/American Stroke Association's (ASA's) cardiovascular health (CVH) metric (2, 3). African Americans have higher rates of acute stroke compared with non-Hispanic whites, yet CVD risk scores have historically been validated in primarily white populations (1, 4). It remains unknown which risk scoring algorithm (10-year CVD risk versus CVH) is most accurate for predicting stroke in an African-American population.

A recent American College of Cardiology (ACC)/AHA collaboration resulted in guidelines for the clinical assessment of CVD risk (1). Applying these guidelines requires measurement of patient characteristics such as age, sex, race, cholesterol, blood pressure (BP), smoking status, diabetes, and antihypertensive treatment. Ten-year CVD risk scores stem from these new guidelines, and reflect risk estimates *for fatal* CHD and stroke and *non-fatal* myocardial infarction and stroke (1, 4). The ACC/AHA suggests that it is appropriate to use the updated 10-year CVD risk algorithm among non-Hispanic whites and African Americans (1).

Efforts to prevent CVD by promoting healthy behaviors and evidence-based treatments are underway via the Centers for Disease Control's *Million Hearts®* initiative (5), in partnership with AHA/ASA's *Life's Simple 7* (6). *Life's Simple 7* comprises seven modifiable CVH behaviors and factors: body mass index, cholesterol, BP, smoking status, fasting glucose, physical activity, and diet. The Reasons for Geographic And Racial Differences in Stroke (REGARDS) study recently reported a lower risk of stroke for every unit increase in CVH score among Caucasians and African Americans (2). While the 10-year CVD risk score may

have a broader appeal to clinicians, given the recently-released statin treatment guidelines (4), CVH may have greater public health relevance and ease of use outside of the clinical setting.

The objective of this study was to determine the predictive ability of two risk scores (10-year CVD risk versus CVH score) for stroke among participants of the Jackson Heart Study (JHS), a community-based cohort of African Americans in Jackson, Mississippi. The JHS presented an ideal setting in which to evaluate risk scores for the prediction of stroke in African Americans because baseline and follow-up data were carefully collected. We hypothesized that, at baseline, 10-year CVD risk would be high (and CVH low) in this population. We additionally anticipated that both the 10-year CVD risk and the CVH scores would predict incident stroke in this population, however, that the 10-year CVD risk score would more strongly predict incident stroke due to its inclusion of non-modifiable, yet highly predictive, risk factors.

Materials and Methods

We analyzed data from the JHS, a study designed to investigate risk factors for CVD among African Americans.(7) The JHS enrolled 5301 participants aged 21 to 95 years from four sources between 2000 and 2004: community volunteers from the Jackson metropolitan area (25%); residents of Jackson selected at random (17%); eligible residents in the Jackson site of the Atherosclerosis Risk in Communities (ARIC) cohort study (8) (31%); and family members of JHS participants (22%) or ARIC participants (5%). All participants underwent a baseline examination and data were collected on demographic characteristics, socioeconomic characteristics, medical history, physical examination results, laboratory values, cardiac testing, behavioral factors, and medications. Deaths and nonfatal events were subsequently ascertained via annual telephone calls, review of death certificates, and abstraction of medical records for relevant *International Classification of Diseases, Ninth Revision, Clinical Modification* codes through 2011. The JHS was approved by the institutional review boards of Jackson State University, Tougaloo College, and the University of Mississippi Medical Center. All study participants provided informed consent. The institutional review boards of the Duke University Health System and The Ohio State University approved use of JHS data for our study.

We included all JHS participants with complete risk score information at the baseline study visit. Participants who reported a prior history of stroke were excluded from all analyses (n=234/5301). Among the remaining n=5067 participants, the primary analysis excludes n=927 participants with any missing CVD risk or CVH score components (complete case) for a final total of n=4,140 (Supplemental Table 1).

The primary outcome of interest was incident stroke. Strokes were ascertained via directed patient queries during annual telephone follow-up and ongoing surveillance of hospitalizations, with subsequent transmission of hospital records and death certificates to a medical record abstraction unit for review. A computer-generated diagnosis with physician adjudication was used to classify hospitalized and fatal stroke events. We included all

ischemic and hemorrhagic strokes that occurred within 10 years of the baseline examination date based on a median of 9 years of follow-up time and a 75th percentile of 10 years.

10-year CVD risk scores were calculated using published coefficients (1). Specifically, we used published race-specific coefficients from pooled cohort equations, as recommended by the ACC/AHA and endorsed by the National Heart, Lung, and Blood Institute (1), to calculate a 10-year CVD risk score for each member of the JHS cohort at baseline (Supplemental Table 2). We then multiplied the CVD risk score times 100 to calculate a CVD risk percentage. For selected analyses, we assigned categories of CVD risk according to cutpoints in the extant literature: low (0–<5.0%), medium (5.0–<7.5%), and high (≥7.5%) (9, 10).

We estimated CVH using the Life's Simple 7 scoring system (3), introduced by the AHA/ASA and shown in detail in Supplemental Table 3, to investigate the association between CVH at baseline and incident stroke (11). We hypothesized that few JHS participants (i.e., <1%) would be classified as having ideal CVH for all behaviors and factors, based upon other local and national community samples (3, 12, 13). Thus, we incorporated the approach used by Kulshreshtha et al. to use an overall CVH score to represent the continuum of CVH that is based on the number of ideal (2), intermediate (1), and poor (0) CVH metrics present at baseline (2). The overall CVH score was summed to a total of 0 (worst) to 14 (best) points according published CVH categories (3). For selected analyses, we employed categories of CVH as follows: ideal CVH (10–14), intermediate CVH (5–9), poor CVH (0–4) (14).

We assessed the prevalence of poor, intermediate, and ideal CVH at baseline for each of the 7 health behaviors and factors. The JHS supported the calculation of a CVH score despite several variations from the precise 2010 AHA definition. In our data, CVH was defined for 5 of the 7 factors exactly as defined by the AHA (Table 1, smoking status, body mass index, cholesterol, BP, and fasting plasma glucose).

The healthy diet score comprises 5 components (not explicitly stated in Supplemental Table 3). We used the JHS food frequency questionnaire, a shorter version of the Delta food frequency questionnaire (15), to calculate the healthy diet score (16). We considered a healthy diet according to achievement of the 5 AHA ideal CVH items (17): 4.5 cups/day of fruits and vegetables; 3.5 oz servings/week of fish; 3 1-oz servings/day of whole grains; sodium (<1,500 mg/day); and 36 oz/week of sugar-sweetened beverages. We used an instrument derived from the Baecke questionnaire (18) to convert the frequency of participation in as many as 3 sports and/or exercise activities during the previous year to minutes per week of moderate or vigorous physical activity (3).

We first described the distribution of baseline characteristics in the overall JHS study population using frequencies and percentages for categorical variables, and median (Q1, Q3) for continuous variables. In addition, we presented the distribution of calculated CVD risk score (risk %) and CVH score (possible point range 0–14) using median (Q1, Q3) and (min, max). We additionally evaluated the distribution of CVH score components at baseline stratified by poor, intermediate, and ideal risk categories. Similarly, we described the

distribution of CVD risk components at baseline stratified by high, medium and low risk categories.

We used Kaplan-Meier methods to calculate the cumulative incidence of first stroke overall and by categories of low, medium and high CVD risk and ideal, intermediate, and poor CVH, respectively, at baseline. We tested for differences between the groups using log-rank tests. We estimated hazard ratios (HRs) and 95% confidence intervals (CIs) for incident stroke according to CVD risk and CVH score using Cox proportional hazards models. We chose Cox regression over logistic regression due to a stroke rate of 3%, and a competing risk of death (10%) over the follow-up period. Study participants were censored at the time of (a) death, (b) participant loss to follow-up, or (c) end of study follow-up (December 31, 2011). The CVD risk score percentage and the CVH score were included, respectively, as the single linear independent variable in Cox proportional hazards models. We did not conduct multivariable adjustment due to the primary interest in estimating the effect of CVD risk / CVH on incident stroke.

We evaluated each model (CVD risk and CVH) according to the increase in the hazard of incident stroke associated with a one-unit increase (percent) in CVD risk score or decrease (point) in CVH score. We compared the discrimination of the two models (CVD risk score versus CVH score) according to the Harrell c-index (19), and 95% CI using the method from Pencina and D'Agostino (20), and plotted predicted versus observed (Kaplan-Meier) stroke risk calibration plots by decile of predicted probability for each of the two models (21). To assess the clinical usefulness of the two models, we considered patients according to two thresholds for high risk of stroke: (a) based on published guidelines (CVD risk $\geq 7.5\%$; CVH score <5) and (b) CVD risk and CVH scores corresponding to the top 20% of the predicted probabilities from each model, respectively (22). At each threshold, we calculated the model's accuracy, sensitivity, false-negative rate, specificity, false-positive rate, and positive predictive value.

Most risk score components had very low missing rates ($<5\%$). However, 8.6% of participants were missing fasting glucose, and 7.2% had incomplete medication data. For the primary analysis, we excluded participants with any missing data for the risk score components as outlined above and in the supplementary tables. For the sensitivity analysis, missing data were handled as follows. For glucose, we imputed to either the non-fasting glucose measurement, when available, or otherwise to the overall median glucose value. We imputed all other continuous variables to the median value, dichotomous variables to 'no', and multichotomous variables to the most frequent categorical value. We selected this imputation strategy as it was most compatible with applying respective scores in clinical and public health settings. The analysis described above was then repeated on the imputed dataset ($n=5,067$). We used a 2-tailed $\alpha = .05$ to establish statistical significance and reported 95% CIs. We used SAS version 9.3 for all analyses (SAS Institute, Cary, North Carolina).

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Results

At baseline, the median age of the 4,140 eligible JHS participants was 54.5 years, and the majority were female (Table 2). Most JHS participants did not have a history of diabetes, and were not treated for BP, cholesterol, or diabetes at baseline. Most (87%) were non-smokers, yet nearly half (48%) reported zero hours of weekly physical activity and almost all (99%) consumed an unhealthy diet according to the CVH metric (Table 2). Meanwhile, the median CVD risk was 6.4%, and the median CVH score was 7.

For females, the median 10-year CVD risk was 5.1% and median CVH score was 7. For males, the median CVD risk was 8.3% and median CVH score was 7. For participants under 65 years, the median CVD risk was 4.3% and median CVH score was 7. For participants 65 years and older, the median CVD risk was 18.5% and median CVH score was 7. Overall, the 10-year CVD risk algorithm scored the largest proportion of participants (approximately 45%) in the “low” and “high” risk categories, while the CVH metric scored the most participants in the “intermediate” category (nearly 80%, data not shown). It should be noted that the proportion of participants classified in the same category of risk using both the CVD risk and the CVH metric were as follows: 10.6% for “low”, 9.8% for “medium”, and 5.9% for “high” (Supplementary Table 3).

Within 10 years of the baseline visit, 112 strokes occurred. The cumulative incidence of stroke was very similar whether calculated across categories of CVD risk or CVH (Figure 1), and the cumulative incidence of stroke across categories was statistically significant according to either scoring metric ($p<.001$). Specifically, the cumulative incidence of stroke and 95% CIs were 0.4 (95% CI, 0.2-0.9) for low CVD risk, 2.9 (1.6-5.1) for medium CVD risk, and 5.6 (4.6-6.9) for high CVD risk. Meanwhile, the cumulative incidence according to CVH categories was: 0.9 (0.3-2.9) for ideal CVH, 3.3 (2.7-4.1) for intermediate CVH, and 5.2 (3.2-8.6) for poor CVH (data not shown).

Meanwhile, the hazard of stroke increased significantly ($HR=1.07$, 95% CI=1.06, 1.08) with each percentage point increase in CVD risk, and decreased significantly ($HR=0.76$, 95% CI=0.69, 0.83) with each point increase in CVH. However, as evidenced by the c-statistics, the CVH model was less discriminating than the CVD risk model (0.59 95% CI (0.55, 0.64) versus 0.79 95% CI (0.76, 0.83)).

Table 3 shows classification statistics according to the guideline-defined elevated CVD risk threshold (7.5%) compared to the stroke risk threshold based on the upper 20% of model predicted probabilities for stroke (16%). Similarly, classification statistics are shown according to the guideline-defined poor CVH (score <5) compared to the CVH score threshold corresponding to the upper 20% of model predicted probabilities for stroke (<6). At these thresholds, the CVD risk model had higher sensitivity, but lower specificity, than the CVH model. We also observed that the positive predictive value was low for both models, which reflects the low prevalence of stroke in the JHS population (Table 3).

Figure 2 shows percent of strokes observed versus predicted in each decile of risk according to the CVD risk model and CVH model, respectively. The plots indicate that the CVD risk model under-predicts risk for those at higher risk (decile 9), while the CVH model over-predicts risk for those at the highest risk (deciles 9 & 10). There is a slight tendency for stroke risk to be over-predicted in the lower deciles of risk while using the CVD risk model (Figure 2).

Imputed data

We repeated all analyses with missing data imputed (n=5,067 participants). Substantive conclusions did not change.

Discussion

In a population-based cohort of African Americans, we observed the 10-year cumulative incidence of stroke increase across worsening categories of CVD risk and CVH. One-unit increases in CVD risk increased the hazard of stroke significantly, while one-unit increases in CVH corresponded to a significantly decreased hazard of stroke. As evidenced by the c-statistics, the CVH model was less discriminating than the CVD risk model, perhaps due to the inclusion of age and sex, important risk factors for CVD, in the CVD risk algorithm. However, stroke prediction was not well-calibrated in the upper and lower deciles of risk for either model. For the CVD risk model, we hypothesize that this poor calibration may be due to differences between our study population and those for which the CVD risk model was originally calibrated. For the CVH model, we acknowledge that there were few distinct scores with which to classify participants, and few were very high (CVH=0) or very low (CVH=14) risk.

We observed an average CVH score (7.0) in the JHS similar to that of the REGARDS national study (7.2) (2). With each one-point increase in CVH score, we reported a larger reduction in stroke risk (HR=0.76, 95% CI=0.69, 0.83) compared to that detected among African Americans in the REGARDS study (HR=0.93, 95% CI=0.87, 0.98) (2). Meanwhile, the c-statistic we observed for the CVD risk score (0.79) was similar to the area under the receiver operator curve reported in the ARIC study for the risk of stroke among black and white women (0.79) and men (0.75) in a model including systolic BP, hypertensive medication, current smoking, diabetes, previous CHD, and left ventricular hypertrophy.

Similar to our study, findings from the Northern Manhattan Study described a decreasing hazard of CVD according to the number of CVH metrics at ideal levels (range: 0-7) regardless of race/ethnicity (white, African American, or Hispanic) (23). An ARIC study sample which included African Americans also found a graded relationship between the number of ideal health metrics present and CVD incidence rates, which ranged from 37.1 per 1,000 person-years (lowest CVH) to 3.9 per 1,000 person-years (highest CVH) (3). Overall, our risk prediction results are consistent with those from other cohorts, which reported reductions in stroke with improved CVH (2) and lower CVD risk (24). Collectively, these findings indicate that maintenance of CVH and better management of modifiable risk factors reduces the risk of stroke.

Our study had several strengths. First, the collection of detailed CVD risk score component data in the JHS allowed for risk scores to be calculated and assessed for accurate stroke prediction. Systematic event ascertainment and adjudication maximized the capture of incident stroke, allowing for comprehensive characterization of risk prediction. Another distinguishing aspect of our study is that it arises from an exclusively African-American sample. The majority of existing risk scores have been developed in predominantly white populations (1), and ours is the first study to compare the performance of the CVH score with the 10-year CVD risk score in the same population. Our findings are relevant to the African-American population from a clinical and public health perspective, given its high burden of stroke and associated risk factors such as hypertension (25).

It should be acknowledged that the 10-year ACC/AHA risk assessment algorithms have created controversy regarding their tendency to over-estimate CVD risk, primarily among high-risk individuals (4, 26). Although the 10-year CVD risk score may be an over-estimate for certain high-risk cohort members (1, 27), we know that CVD risk increases in a graded, continuous fashion as levels of the risk factors become more adverse (28). The 10-year risk coefficients were developed from sex- and race-specific models which predicted the hazard of a hard atherosclerotic CVD event, which included incident non-fatal myocardial infarction or stroke as well as fatal coronary heart disease or stroke (1). Discrimination ability (c-statistics) of the models ranged from 0.71 in African-American men to 0.82 in African-American women (1). The CVD risk model may have provided more precise estimates compared to the CVH score for predicting stroke in this black population since race-specific coefficients were used in this analysis.

While the risk algorithms were subject to rigorous validation, individuals included in the original pooled cohort analyses were predominantly non-Hispanic whites (1), which underscored the need for a prospective evaluation of the CVD risk score in a cohort of African-Americans (4, 27). Since African-Americans have nearly twice the risk of stroke compared to non-Hispanic whites (25), we opted to evaluate the performance of the CVD risk score for the prediction of incident stroke – instead of total cardiovascular disease risk – in this population. Interestingly, our analysis found that the CVD risk algorithm under-predicted the risk of stroke in the 9th decile, and only slightly over-predicted the risk in the 10th decile.

The ARIC cohort, along with 4 other population-based studies, was used to develop the 10-year CVD risk score used for the current analysis (1). Thus, we acknowledge as a limitation the inherent overlap between these two populations. A limitation of these JHS data is the relatively small number of stroke events over follow-up. In addition, we acknowledge that our strategy for comparing the CVH and CVD scores to one another – computing observed versus predicted stroke by decile of risk – may have resulted in too much granularity for the CVH score, yet may not have provided a sufficient number of categories to adequately capture the CVD risk score. While this approach may have been the best way to compare the two scores to one another, it may not have accurately captured the performance of the scores across their respective spectra.

The current work evaluated and compared risk score models for the prediction of stroke without consideration of confounding factors. Future directions for this work include an investigation into the association between CVH and CVD in the presence of potential confounding factors. In addition, there have been recent public health campaigns (5, 6) and clinical interventions (29) to address these modifiable risk factors. Another opportunity for further investigation in community-based studies, such as ARIC, REGARDS, and JHS, would be to assess if there have been recent decreases in risk factor burden.

In our analyses, the CVD risk score resulted in better performance statistics (i.e., Harrell's c). While 10-year CVD risk calculators exist online for public use (30), we acknowledge the simplicity of the CVH scoring system may be easier to employ outside of the clinical setting. The CVH score, although not designed as a predictive score, may prove to be a better real-world method for patients and the public to understand how modifiable, health-promoting behaviors, such as diet and physical activity, can affect the risk of incident stroke.

Our results suggest that the CVD risk score provides better prediction for those at higher risk compared to those at lower risk, and thus those in poor CVH may be more accurately classified with the CVD risk score. In contrast, those at lower CVD risk could instead be advised to evaluate and manage their CVH score, as those individuals may find ways to improve their CVH even if they are already in generally good health. In contrast to the CVD risk score, all CVH behaviors and factors are modifiable. We acknowledge that both metrics have relatively poor calibration if the scores are very low or very high, possibly due to the low observed stroke rate in this population. Yet our findings suggest either metric can be used to identify patients at increased risk and to recognize improvements in certain modifiable behaviors and factors can reduce risk.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

AHA	American Heart Association
ARIC	Atherosclerosis Risk in Communities
ASA	American Stroke Association
BP	Blood pressure
CHD	Coronary heart disease
CI	Confidence interval
CVD	Cardiovascular disease
CVH	Cardiovascular health
HR	Hazard ratio
JHS	Jackson Heart Study
REGARDS	Reasons for Geographic And Racial Differences in Stroke

Highlights

- We compared the discrimination of two statistical models for the prediction of stroke among African Americans
- We observed increases in the hazard of stroke for every one-unit increase in 10-year cardiovascular risk and every one-unit decrease in the cardiovascular health metric
- The 10-year cardiovascular risk model was more discriminating than the cardiovascular health model, but the cardiovascular health metric may still be preferable for its simplicity

Figure 1. Kaplan-Meier Curves for Incident Stroke by Categories of Cardiovascular Risk (Left) and Cardiovascular Health (Right)

Figure 2.
Percent Observed (Kaplan-Meier) Versus Predicted Stroke Risk by Deciles of Risk for CVD Risk (Left) and CVH (Right) Models.*

*Decile group counts for CVH are not evenly distributed given there were only 14 unique stroke event predicted probabilities for the range of CVH scores from 0-13 and participants were disproportionately in the middle CVH range.

Table 1
Measures of CVH: 4 Health Behaviors and 3 Health Factors (Lloyd-Jones, 2011)(11)

	Poor Health	Intermediate Health	Ideal Health
Health Behaviors			
Smoking status	Yes	Former 12 months	Never or quit > 12 months
Body mass index	30 kg/m ²	25 - <30 kg/m ²	< 25 kg/m ²
Physical activity	None	0- <150 min/wk moderate or 0- <75 min/wk vigorous or 0- <150 min/wk moderate + vigorous	150 min/wk moderate or 75 min/wk vigorous or 150 min/wk moderate + vigorous
Healthy diet score	0 - 1 components	2 - 3 components	4 - 5 components
Health Factors			
Total cholesterol	240 mg/dL, treated or not	200- <240 mg/dL (treated or untreated) or treated to ideal on antihyperlipidemic	< 200 mg/dL and not on antihyperlipidemic
Blood pressure	Systolic 140 mm Hg or Diastolic 90 mm Hg, treated or not	Systolic 120- <140 mm Hg or Diastolic 80- <90 mm Hg (treated or untreated) or treated to ideal on antihypertensive	Systolic < 120 mm Hg and Diastolic < 80 mm Hg and not on antihypertensive
Fasting plasma glucose	126 mg/dL, treated or not	100- <126 mg/dL (treated or untreated) or treated to ideal on antidiabetic	< 100 mg/dL and not on antidiabetic

Table 2
Baseline Characteristics of JHS Participants with no Prior Stroke

N	4,140
Demographics	
Age, median (IQR), y	54.5 (44.8, 64.1)
Female sex, No. (%)	2,683 (64.8)
Medical History, No. (%)	
Diabetes	746 (18.0)
Physical examination/labs, median (IQR)	
BMI, kg/m ²	30.5 (26.8, 35.5)
Systolic blood pressure, mmHg	124.0 (114.0, 136.0)
Diastolic blood pressure, mmHg	79.0 (72.0, 86.0)
Total cholesterol, mg/dL	196.0 (172.0, 223.0)
HDL cholesterol, mg/dL	50.0 (41.0, 60.0)
Fasting plasma glucose, mg/dL	91.0 (85.0, 100.0)
Behavioral factors, No. (%)	
Smoking history, No. (%)	
Never smoked or quit >1 year	3,601 (87.0)
Former smoker quit ≤1 year	24 (0.6)
Current smoker	515 (12.4)
Physical activity	
None	1,979 (47.8)
0-<150 min/wk moderate or 0-<75 min/week vigorous or 0-<150 min/wk moderate/vigorous	1,338 (32.3)
≥150 min/wk moderate or ≥75 min/wk vigorous or ≥150 min/wk moderate/vigorous	823 (19.9)
Diet	
0-1 components	2,541 (61.4)
2-3 components	1,562 (37.7)
4-5 components	37 (0.9)
Medications, No. (%)	
Cholesterol treatment	471 (11.4)
Blood pressure treatment	1,999 (48.3)
Diabetes treatment	479 (11.6)
Stroke Risk Scores, Median (Q1,Q3) (Min, Max)	
CVD risk percentage	6.4 (2.2, 13.8) (0.0, 77.5)
Life simple 7 CVH score	7.0 (6.0, 9.0) (0.0, 13.0)

Table 3
Model Classification Statistics According to CVD Risk and CVH

	Stroke Prediction (CVD risk)		Stroke Prediction (CVH)	
	7.5%	16%	<5	<6
No. of subjects	1,886	839	306	750
Accuracy, %	56.2	80.3	90.6	80.9
Sensitivity, %	83.0	60.7	13.4	32.1
False-negative rate, %	17.0	39.3	86.6	67.9
Specificity, %	55.5	80.9	92.8	82.3
False-positive rate, %	44.5	19.1	7.2	17.7
Positive predictive value, %	4.9	8.1	4.9	4.8