ORIGINAL RESEARCH

# Prevalence and Cardiovascular Health Impact of Family History of Premature Heart Disease in the United States: Analysis of the National Health and Nutrition Examination Survey, 2007–2014

Ramal Moonesinghe, PhD; Quanhe Yang, PhD; Zefeng Zhang, MD, PhD; Muin J. Khoury, MD, PhD

***Background—***Because family history is a known risk factor for heart disease, it is important to characterize its public health impact in terms of population prevalence of family history of heart disease, the burden of heart disease attributable to family history, and whether family history interacts with modifiable risk factors for heart disease.

***Methods and Results—***We used population data from NHANES (the National Health and Nutrition Examination Survey [2007–2014]) to measure the association of self-reported family history of premature heart disease (FHPHD) with cardiovascular disease (n=19 253) and to examine the association between cardiovascular health metrics and FHPHD (n=16 248). Using logistic regression and multivariable adjustment, family history odds ratios were 5.91 (95% CI, 3.34–10.44) for ages 20 to 39, 3.02 (95% CI, 2.41–3.79) for ages 40 to 59, and 1.87 (95% CI, 1.54–2.28) for age ≥60 for cardiovascular disease. The prevalence of cardiovascular disease for the population with a FHPHD (15.72%; 95% CI, 13.81–17.64) was more than double the prevalence of cardiovascular disease for those without a family history (6.25%; 95% CI, 5.82–6.69). Compared with participants with optimum cardiovascular health, the prevalence ratio for FHPHD was 1.98 (95% CI, 1.40–2.79) for those with inadequate cardiovascular health.

***Conclusions—***Millions of people who are at high risk of having cardiovascular disease could be identified using FHPHD. FHPHD can become an important component of public health campaigns that address modifiable risk factors that plan to reduce the overall risk of heart disease. **(*J Am Heart Assoc.* 2019;8:e012364. DOI: 10.1161/JAHA.119.012364.)**

**Key Words:** cardiovascular disease • cardiovascular disease prevention • cardiovascular disease risk factors • family history

In spite of declining death rates from cardiovascular disease (CVD), heart disease is still the leading cause of death in the United States. An estimated 92.1 million US adults have at least 1 type of CVD and by 2030, around 44% of the US adult population is expected to have some form of CVD.[1] An important risk factor that has long been known to be associated with heart disease is family history of heart disease.[2–5] On the basis of a large international case-control study (the INTERHEART [Effect of Potentially Modifiable Risk Factors Associated With Myocardial Infarction] study) to estimate the association of myocardial infarction (MI) and parental history of MI, the odds ratios (ORs) of MI ranged from 1.67 to 6.56 depending on the number of parents who had an MI and whether the parent had an MI before age 50.[6] The increased risk of heart disease attributable to family history can be caused by shared genetic, environmental, and behavioral factors. The role of genetic factors in excess familial risk of heart disease increases with early onset of heart disease in the family and number of affected people.[7] Genetic conditions, most commonly familial hypercholesterolemia, account for a small proportion of excess familial risk, but causes of most familial cases of heart disease remain unknown.[7]

With the goal of improving cardiovascular health, the American Heart Association designed a health campaign, "My Life Check—Life's Simple 7," that was based on 7 cardiovascular metrics: body mass index, smoking, physical activity, dietary intake, total cholesterol, blood pressure, and fasting glucose.[8] To monitor the overall cardiovascular health in the US population, Lloyd-Jones et al[9] defined levels for each metric from ideal to intermediate to poor. Several independent studies have substantiated the associations of these metrics with cardiovascular health and mortality.[10–14] Social

*From the Office of Minority Health and Health Equity (R.M.) and Office of Public Health Genomics (M.J.K.), Centers for Disease Control and Prevention, Atlanta, GA; Division for Heart Disease and Stroke Prevention, National Center for Chronic Disease Prevention and Health Promotion, Atlanta, GA (Q.Y., Z.Z.).*

Accompanying Tables S1 and S2 are available at https://www.ahajournals.org/doi/suppl/10.1161/JAHA.119.012364

**Correspondence to:** Ramal Moonesinghe, Office of Minority Health and Health Equity, Centers for Disease Control and Prevention, 4770 Buford Highway, Mailstop TCU-2 Atlanta, GA 30341. E-mail: rmoonesinghe@cdc.gov

Received February 14, 2019; accepted June 11, 2019.

## Clinical Perspective

### What Is New?

- Using a representative sample of the US population, this study showed that family history of premature heart disease is common in the United States (estimated 27.8 million people over 20 years of age), and millions of people who are at increased risk for cardiovascular disease can be identified using family history.
- Among young people (20–39 years of age) with cardiovascular disease, about 1 in 3 could be attributed to the family history of premature heart disease (burden of 0.3 million young people).
- Among people without prevalent cardiovascular disease, people with family history of heart disease have a much less favorable heart health rating compared with people without a family history, including several modifiable risk factors (such as physical activity and cholesterol).

### What Are the Clinical Implications?

- Awareness of family history of premature heart disease and associated modifiable risk factors is important for clinicians to make appropriate diagnosis, start early treatment, and promote healthy lifestyles in their patients, and for the public to collect, understand, and act on their family history to reduce their risk of premature heart disease.

risk factors such as low income, low education, minority race/ethnicity, and single living were associated with lower levels of Life's Simple 7 scores after adjusting for age and sex.[15]

Because a positive family history of premature heart disease is a known risk factor for heart disease, it is important to characterize its public health impact in terms of population prevalence of family history of heart disease, the burden of heart disease attributable to family history, and whether family history interacts with modifiable risk factors for heart disease. To the best of our knowledge, such analyses have not been done in a representative sample of the US population. NHANES (the National Health and Nutrition Examination Survey) provides a unique opportunity to conduct such an analysis, as the survey is population based, representative, and weighted and collects information on heart disease, heart disease risk factors, and family history of premature heart disease (FHPHD, under age 50).

## Materials and Methods

All data and materials used in this study are publicly available at the National Center for Health Statistics website.[16]

### NHANES

NHANES is a series of cross-sectional surveys using stratified, multistage probability samples designed to provide assessments on the health and nutrition status of the civilian, noninstitutionalized US population. NHANES is conducted by the Centers for Disease Control and Prevention's National Center for Health Statistics and has continuously collected data based on personal interviews and physical examination of survey participants in 2-year cycles since 1999. The present study included samples of adults aged ≥20 years in the cycles 2007 to 2014. Detailed methods of the NHANES survey construction and sampling strategy are available elsewhere (https://www.cdc.gov/nchs/data/series/sr_02/sr02_162.pdf). Participants complete an in-home interview for basic demographic and health information along with a scheduled visit to a mobile examination center for physical examination and laboratory testing. Informed consent was obtained from all participants for both parts of the survey, and the National Center for Health Statistics ethics review board approved all the protocols. Pregnant women were excluded because of the effect of pregnancy on glucose measurement.

### Family History of Premature Heart Disease

If a participant reported that they had ever been diagnosed with coronary heart disease, angina, heart attack, or stroke by a doctor or other health professional, we defined that person as having CVD. Participants were asked whether any of their close biological (blood) relatives, including father, mother, sisters, or brothers, were ever told by a health professional that they had a heart attack or angina before the age of 50. We defined participants as having a reported family history of premature heart disease (FHPHD) if they responded "yes" to this question. Further information on family history of CVD is not available in NHANES 2009–2014 to do a more comprehensive analysis.

### Statistical Analysis

We used a logistic regression model to measure the association between CVD and self-reported FHPHD. We included other risk factors in the model: age group (20–39, 40–59, ≥60 years), sex, race/ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, others [non-Hispanic]), body mass index (BMI) (<25, 25–29.9, ≥30 kg/m$^2$), current smoker (yes/no), and leisure time physical activity (yes/no). We did not consider other racial/ethnic groups because sample sizes were too small for meaningful analysis. We defined participants as physically active if the participant had ≥150 min/week moderate intensity activity or ≥75 min/week vigorous

intensity activity or 2×min/week vigorous intensity activity+min/week moderate intensity activity ≥150. We also included income-to-poverty ratio (<1/≥1) and education (less than high school completion/high school completion or greater) in the model as indicators of socioeconomic status. Income-to-poverty ratio is the ratio of family income to poverty guidelines. We also tested for significant interactions between family history and other risk factors by including 1 interaction term at a time in the model.

We estimated the population attributable fraction (PAF) for FHPHD using the formula, $PAF = \frac{G(R-1)}{G(R-1)+1}$, where $G$ is the prevalence of FHPHD in the population and $R$ is the prevalence ratio. When the prevalence of CVD was <10% for a given population, we used OR as an approximate estimate for prevalence ratio.[17] When the prevalence of CVD was relatively large (>10%), the approximate estimate of prevalence ratio was obtained from ORs, using the formula, prevalence ratio $= \frac{OR}{(1-p_0)+OR\times p_0}$, where $p_0$ is the prevalence of CVD in the population who do not have a FHPHD.[17] Next, we calculated the number of cases impacted by FHPHD in the population by multiplying PAF and the number of cases with CVD. To calculate the total number of cases with CVD by age group, we used the distribution of the civilian noninstitutionalized US population obtained from the Census Bureau's Current Population Survey as recommended by the National Center for Health Statistics.[18] We multiplied the average population size for the 4 survey cycles by the prevalence of CVD. After excluding participants with missing data for the variables used in the logistic regression, the study sample consisted of 19 253 nonpregnant adult respondents aged ≥20 that included 1787 CVD cases and 2304 participants with FHPHD.

### *Cardiovascular health metrics*

Next, we compared the cardiovascular health metrics between participants with and without FHPHD for those who had not yet developed CVD. Cardiovascular health metrics included BMI, smoking, physical activity, healthy dietary scores, total cholesterol, blood pressure, and fasting plasma glucose. The definitions of ideal, intermediate, and poor cardiovascular health metrics for adults were given in Table 3 of Lloyd-Jones et al[9]. Instead of using their dietary criteria, we used Healthy Eating Index–2010 (HEI-2010) scores, which were calculated from dietary information collected by a single 24-hour dietary recall.[19] The Healthy Eating Index–2010 is a measure of diet quality that tracks the federal dietary guidelines for Americans. It has 12 components: total fruit, whole fruit, total vegetables, greens and beans, whole grains, dairy, total protein foods, seafood and plant protein, fatty acid, refined grains, sodium, and empty calories.[19] The total scores range from 0 to 100, with higher scores indicating a healthier diet. We categorized participants with Healthy Eating Index–2010 scores ≤50, 51 to 80, and ≥81 as having poor diet, intermediate diet, and ideal diet, respectively.[12]

To maximize the sample size, we used both fasting plasma glucose and hemoglobin $A_{1c}$ values to determine whether participants have diabetes mellitus or prediabetes. When both $A_{1c}$ and fasting plasma glucose were available for a participant, if either one met the criteria for diabetes mellitus, then the participant was classified as having diabetes mellitus (poor health). If neither met the criteria for having diabetes mellitus, but at least one met criteria for having prediabetes, then the participant was classified as having prediabetes (intermediate health).[20] Participants who reported having diabetes mellitus or being treated with insulin or oral medication to lower blood glucose and had a hemoglobin $A_{1c}$ concentration of 5.7% to 6.4% were considered to have intermediate health. Mean blood pressure was estimated from up to 3 readings, obtained under standard conditions during a single physical examination. Use of antihypertensive, cholesterol-lowering, and glucose-lowering medications were self-reported.

A score of 0, 1, or 2 was assigned to each cardiovascular health metric to represent poor, intermediate, or ideal health, respectively. Based on the sum of scores for all 7 cardiovascular metrics, an overall score, ranging from 0 to 14, was categorized as inadequate (0–4), average (5–9), or optimum (10–14) cardiovascular health.[21,22] We compared characteristics of participants with an FHPHD with those without a family history using t tests. We used polytomous logistic regression to estimate the adjusted prevalence ratios (PRs) of FHPHD comparing average or inadequate cardiovascular health with optimum cardiovascular health, adjusted for age, sex, race/ethnicity, education, and income-to-poverty ratio. For this analysis, we excluded participants with missing cardiovascular health metric scores, missing values of covariates, and FHPHD. We also excluded participants with a history of CVD and participants with BMI <18.5. The sample for this analysis consisted of 16 431 nonpregnant adult respondents aged ≥20 years that included 1863 participants with a family history of CVD.

The family history question in NHANES asked only about heart attack or angina, whereas the CVD definition also included stroke or coronary heart disease. We conducted a sensitivity analysis to examine the consistency of results obtained for the association of FHPHD with CVD and cardiovascular health metrics by changing the outcome variable to heart attack or angina instead of CVD.

## Heart Age Calculation

Finally, we compared the predicted mean heart age for participants aged 30 to 74 years with an FHPHD with those without a family history. Based on the Framingham study participants, D'Agostino et al[23] presented simple sex-specific

risk functions to evaluate the 10-year risk of developing overall CVD. The nonlaboratory predictors for the multivariable risk factor algorithm included age, BMI, treated and untreated systolic blood pressure, smoking, and diabetes mellitus. They also introduced heart age, the estimated age of a person's vascular system based on these predictors. The difference between heart age and chronological age provides an effective way to communicate risk for developing CVD.[24]

We age-adjusted heart age and excess heart age (defined as the difference between heart age and chronological age) using the age groups 30 to 39, 40 to 49, 50 to 59, and 60 to 74 years and the 2000 US standard population. The survey data were analyzed using SURVEYFREQ and SURVEYLOGISTIC procedures in SAS version 9.3 (SAS Institute, Cary, NC) that takes into account the complex survey design of the NHANES, and the sample weights were adjusted for pooling 4 cycles of NHANES data.

## Results

Table 1 gives the prevalence of reported FHPHD and the estimated population with FHPHD for the US population by age group, sex, race/ethnicity, education, income-to-poverty ratio, and BMI. The prevalence of FHPHD in the US population aged ≥20 years was 12.55% (95% CI, 11.81–13.29). Based on the average US population during 2007 to 2014, around 27.8 million people aged ≥20 had an FHPHD. The prevalence of reported FHPHD was significantly higher among age groups 40 to 59 (14.21%; 95% CI, 12.80–15.62) and ≥60 (15.09; 95% CI, 13.43–16.75) compared with the age group 20 to 39 (9.02%; 95% CI, 8.23–9.81). By race/ethnicity groups, the non-Hispanic white population had the highest prevalence of FHPHD, an estimate of 13.82% (95% CI, 12.76–14.87). The prevalences of FHPHD for non-Hispanic black and Hispanic populations were 11.17% (95% CI, 9.97–12.37) and 8.91% (95% CI, 8.11–9.71), respectively. Females had a higher prevalence of FHPHD (14.03%; 95% CI, 13.05–15.0) compared with males (10.98 95% CI, 10.07–11.90). Populations with BMI ≥30, less than high school education, and income-to-poverty ratio <1 had significantly higher prevalence of FHPHD than those with BMI <30, more education, and income-to-poverty ratio ≥1, respectively.

During 2007 to 2014, the crude prevalence of CVD in the US population ≥20 years was 7.44% (95% CI, 6.95, 7.94). The prevalence of CVD for the population with a FHPHD (15.72%; 95% CI, 13.81–17.64) was more than double the prevalence of CVD for those without a family history (6.25%; 95% CI, 5.82–6.69). Table 2 presents the prevalence of CVD among those with and without an FHPHD for the populations with risk factors related to CVD, and Table 3 describes the adjusted odds ratios (aORs) of these risk factors associated with CVD in the logistic regression model. The prevalence of CVD for those with an FHPHD was more than double the prevalence of CVD for those without an FHPHD in all categories of these risk factors except people aged ≥60 years and people with BMI 25 to 29.9. All the interaction terms of risk factors with family history were nonsignificant except for age in the logistic regression model. Because the interaction of FHPHD and age was significant ($P$<0.001), we present the aOR of family history for each age group in Table 3. The aOR of FHPHD for age 20 to 39 was 5.91 (95% CI, 3.34–10.44); for age 40 to 59 was 3.02 (95% CI, 2.41–3.79); and for age ≥60 was 1.87 (95% CI, 1.54–2.28). During 2007 to 2014, the average US population for the age groups 20 to 39, 40 to 59, and ≥60 were 81.5, 84.2, and 56.1 million, respectively. Using the prevalence estimates of CVD for these age groups (1.05%, 5.32%, and 19.66%, respectively), we

**Table 1.** Unadjusted Prevalence of Self-Reported FHPHD and Estimated Population With FHPHD by Selected Characteristics, US Population Aged 20 Years and Older, NHANES 2007–2014

| | FHPHD Prevalence (95% CI) | Estimated Population With FPHPD (millions) |
|---|---|---|
| Total | 12.55 (11.81–13.29) | 27.84 |
| Age, y | | |
| 20–39 | 9.02 (8.23–9.81) | 7.35 |
| 40–59 | 14.21 (12.80–15.62) | 11.96 |
| ≥60 | 15.09 (13.43–16.75) | 8.45 |
| Sex | | |
| Male | 10.98 (10.07–11.90) | 11.71 |
| Female | 14.03 (13.05–15.00) | 16.15 |
| Race/Ethnicity | | |
| Non-Hispanic white | 13.82 (12.76–14.87) | 20.77 |
| Hispanic | 8.91 (8.11–9.71) | 2.76 |
| Non-Hispanic black | 11.17 (9.97–12.37) | 2.81 |
| Other* | 10.02 (8.09–11.94) | 1.54 |
| BMI (kg/m$^2$) | | |
| <25 | 9.86 (8.76–10.95) | 6.55 |
| 25–29.9 | 12.13 (11.11–13.15) | 8.79 |
| ≥30 | 15.08 (14.00–16.16) | 12.51 |
| Education | | |
| Less than high school completion | 15.38 (13.71–17.05) | 5.25 |
| High school completion or greater | 12.04 (11.33–12.74) | 22.59 |
| Income-to-poverty ratio† | | |
| <1 | 15.02 (12.94–17.10) | 5.18 |
| ≥1 | 12.10 (11.39–12.81) | 22.67 |

BMI indicates body mass index; FHPHD, family history of premature heart disease; NHANES, National Health and Nutrition Examination Survey.
*Non-Hispanic Asians, non-Hispanic multiracial, and non-Hispanic other race.
†A ratio of family income to poverty guidelines.

**Table 2.** Estimates of Prevalence of CVD Among Those With and Without an FHPHD for the Populations Aged 20 Years and Older With Risk Factors, NHANES, 2007–2014

| | Prevalence of CVD | | |
|---|---|---|---|
| | Overall (95% CI) | With FHPHD (95% CI) | Without FHPHD (95% CI) |
| Self-reported family history | | | |
| Age 20–39 (y) | 1.05 (0.77–1.32) | 4.63 (2.58–6.68) | 0.69 (0.46–0.93) |
| Age 40–59 (y) | 5.32 (4.60–6.03) | 12.32 (10.17–14.47) | 4.16 (3.53–4.78) |
| Age ≥60 (y) | 19.66 (18.38–20.94) | 29.81 (25.99–33.63) | 17.84 (16.59–19.09) |
| Sex | | | |
| Females | 6.38 (5.75–7.01) | 13.30 (11.11–15.49) | 5.26 (4.66–5.85) |
| Males | 8.57 (7.78–9.36) | 19.00 (15.71–22.30) | 7.28 (6.53–8.02) |
| Race/Ethnicity | | | |
| Non-Hispanic white | 8.05 (7.35–8.75) | 16.35 (13.94–18.76) | 6.72 (6.07–7.36) |
| Hispanic | 4.68 (4.03–5.33) | 10.11 (7.56–12.66) | 4.15 (3.53–4.77) |
| Non-Hispanic black | 7.62 (6.96–8.28) | 14.34 (11.24–17.43) | 6.78 (6.15–7.40) |
| Other* | 6.85 (5.05–8.64) | 19.32 (11.90–26.73) | 5.46 (3.97–6.94) |
| BMI (kg/m$^2$) | | | |
| <25 | 5.39 (4.65–6.13) | 13.76 (9.85–17.67) | 4.48 (3.85–5.11) |
| 25–29.9 | 6.70 (5.93–7.46) | 11.75 (9.08–14.42) | 6.00 (5.19–6.81) |
| ≥30 | 9.74 (9.03–10.44) | 19.53 (16.17–22.89) | 7.99 (7.33–8.65) |
| Income-to-poverty ratio† | | | |
| ≥1 | 7.21 (6.84–8.00) | 15.27 (12.94–17.61) | 6.10 (5.60–6.59) |
| <1 | 8.71 (7.49–9.93) | 17.70 (14.32–21.08) | 7.12 (6.01–8.23) |
| Education | | | |
| High school or greatercompletion† | 6.69 (6.17–7.22) | 14.69 (12.7216.65) | 5.60 (5.12–6.07) |
| Less than high school completion | 11.55 (10.34, 12.77) | 20.18 (15.6124.75) | 9.98 (8.78–11.18) |
| Physical activity | | | |
| Active‡ | 4.46 (3.85–5.08) | 11.23 (8.57–13.88) | 3.62 (3.13–4.11) |
| Not active | 10.05 (9.39–10.70) | 18.88 (15.84–21.92) | 8.63 (7.96–9.29) |
| Current smoker | | | |
| No | 7.03 (6.50–7.55) | 14.34 (11.97–16.71) | 6.09 (5.61–6.57) |
| Yes | 9.15 (7.83–10.47) | 19.37 (15.46–23.29) | 6.96 (5.80–8.12) |

BMI indicates body mass index; CVD, cardiovascular disease; FHPHD, family history of premature heart disease; NHANES, National Health and Nutrition Examination Survey.
*Non-Hispanic Asians, non-Hispanic multiracial, and non-Hispanic other race.
†A ratio of family income to poverty guidelines.
‡≥150 min/week moderate activity or ≥75 min/week vigorous activity or ≥150 min/week moderate+vigorous activity.

estimated that 0.9, 4.5, and 11.0 million people had CVD in the age groups 20 to 39, 40 to 59, and ≥60, respectively. Since the PAFs for reported FHPHD for these 3 age groups were 30.68%, 22.30%, and 8.58%, respectively, ≈0.3, 1.0, and 1.0 million cases with CVD could be attributed to having an FHPHD.

The aOR for males was 75% higher (95% CI, 1.49–2.06) than that of females. The odds of having CVD was 24% lower for Hispanics (95% CI, 0.63–0.91), compared with that of non-Hispanic whites. The aOR for the association between BMI ≥30 and prevalence of CVD was 1.55 (95% CI, 1.34–1.80) compared with that of BMI <25. The aORs for current smokers, those who were not physically active, had less than high school education, and income-to-poverty ratio <1 ranged from 1.36 to 1.66.

Table 4 presents a comparison of the distributions of the populations free of CVD distributed in categories: ideal, intermediate, and poor health, between those with and without an FHPHD for each "Life's Simple 7" cardiovascular metric. The percentages of the population with an FHPHD in ideal health were significantly less than that for the population without a family history for BMI risk (26.7 versus 30.7), smoking risk (71.0

**Table 3.** Estimates of aORs From the Logistic Regression Analysis of Factors Related to Diagnosed CVD for the Population Aged 20 Years and Older With Risk Factors, NHANES, 2007–2014

| | aOR (95% CI) |
|---|---|
| Self-reported family history[‡] | |
| Age 20–39 (FHPHD: Yes vs No[†]) (y) | 5.91 (3.34–10.44) |
| Age 40–59 (FHPHD: Yes vs No[†]) (y) | 3.02 (2.41–3.79) |
| Age ≥60 (FHPHD: Yes vs No[†]) (y) | 1.87 (1.54–2.28) |
| Sex | |
| Females[†] | |
| Males | 1.75 (1.49–2.06) |
| Race/Ethnicity | |
| Non-Hispanic white[†] | |
| Hispanic | 0.76 (0.63–0.91) |
| Non-Hispanic black | 1.07 (0.93–1.24) |
| Other* | 1.20 (0.89–1.63) |
| BMI (kg/m$^2$) | |
| <25[†] | |
| 25–29.9 | 1.01 (0.86–1.18) |
| ≥30 | 1.55 (1.34–1.80) |
| Income-to-poverty ratio[§] | |
| ≥1[†] | |
| <1 | 1.49 (1.23–1.80) |
| Education | |
| High school completion or greater[†] | |
| Less than high school completion | 1.36 (1.16–1.59) |
| Physical activity | |
| Active[†,‖] | |
| Not active | 1.63 (1.38–1.93) |
| Current smoker | |
| No[†] | |
| Yes | 1.66 (1.35–2.06) |

aOR indicates adjusted odds ratio; BMI, body mass index; CVD, cardiovascular disease; FHPHD, family history of premature heart disease; NHANES, National Health and Nutrition Examination Survey.
*Non-Hispanic Asians, non-Hispanic multiracial, and non-Hispanic other race.
[†]Reference group.
[‡]Because the interaction of FHPHD and age was significant, aOR of family history for each age group was presented.
[§]A ratio of family income to poverty guidelines.
[‖]≥150 min/week moderate activity or ≥75 min/week vigorous activity or ≥150 min/week moderate+vigorous activity.

versus 78.6), physical activity risk (42.6 versus 48.4), cholesterol risk (40.7 versus 47.0), blood pressure risk (35.6 versus 44.7), and diabetes mellitus risk (68.3 versus 74.5). The percentages of the population with an FHPHD in poor health were significantly higher than that for the population without a family history for BMI (39.9 versus 35.0), smoking (27.0 versus 19.3), physical activity (37.6 versus 33.7), diet (53.4 versus 48.6), blood pressure (16.6 versus 13.8), and diabetes mellitus (8.2 versus 6.6). The overall effects of these risk factors were reflected in the mean scores of the 7 health metrics for the population with and without an FHPHD. The overall mean score of the 7 health metrics for the population with FHPHD was significantly lower compared with the population without family history (7.9 versus 8.6). Similar results can be seen when we categorize the overall score to inadequate, average, and optimum health.

The results of the sensitivity analysis corresponding to Tables 3 and 4 when the outcome is heart attack or angina instead of CVD are presented in Table S1 and S2, respectively. The pattern of associations and the difference in distributions of the cardiovascular health metrics were largely consistent for both outcomes.

Table 5 shows the associations between inadequate and average cardiovascular health, and reported FHPHD. After controlling for other variables in the model, the adjusted PRs for inadequate and average cardiovascular health were 1.98 (95% CI, 1.40–2.78) and 1.59 (95% CI, 1.31–1.91), respectively, for those with a FHPHD compared with those without, relative to those with optimum cardiovascular health. Out of all the variables considered in the model, age group ≥60 years compared with age group 20 to 39 years had by far the highest adjusted PRs (inadequate—8.58 [95% CI, 6.56–11.17] and average—4.25 [95% CI, 3.61, 5.01]). The adjusted PRs for non-Hispanic blacks were 2.38 (95% CI, 1.82–3.12) and 1.78 (95% CI, 1.49–2.13), respectively, compared to non-Hispanic whites, and the adjusted PR for those with less than a high school education were 3.19 (95% CI, 2.42–4.20) and 2.00 (95% CI, 1.77–2.27), respectively.

Table 6 presents the predicted age-adjusted mean heart age and the predicted age adjusted mean excess heart age for the overall population aged between 30 and 74 as well as for those with and without an FHPHD. The estimated heart age for those with an FHPHD was significantly higher (57.6 versus 55.0) compared with those without a family history. Similarly, the excess heart age (heart age—chronological age), for those with an FHPHD was significantly higher (9.6 versus 7.0) compared with those without a family history, indicating that, on average, US adults aged 30 to 74 with an FHPHD have a heart age that is 9.6 years older than their actual age.

## Discussion

Our findings confirm the public health importance of family history as a risk factor associated with CVD. Using a population-based representative survey, we show that reported FHPHD is common in the United States (12.5%, or 27.8 million people over age 20). These data suggest that millions of people who are

**Table 4.** Distribution of Ideal, Intermediate, and Poor[9] Cardiovascular Health for Each Metric for Adults 20 Years and Older Free of CVD, NHANES 2007–2014

| Cardiovascular Health Metric | Overall | No Family History of CVD | Family History of CVD |
|---|---|---|---|
| Body mass index risk (%, SE) | | | |
| Ideal (<25 kg/m$^2$) | 30.3 (0.68) | 30.7 (0.67) | 26.7* (1.38) |
| Intermediate (25–29 kg/m$^2$) | 34.2 (0.66) | 34.3 (0.70) | 33.5 (1.36) |
| Poor (≥30 kg/m$^2$) | 35.5 (0.59) | 35.0 (0.65) | 39.9* (1.16) |
| Smoking risk (%, SE) | | | |
| Ideal (never smoked or quit smoking ≥12 months ago) | 77.7 (0.64) | 78.6 (0.61) | 71.0* (1.85) |
| Intermediate (quit smoking <12 months ago) | 2.1 (0.18) | 2.1 (0.20) | 2.0 (0.43) |
| Poor (current smoker) | 20.2 (0.60) | 19.3 (0.56) | 27.0* (1.85) |
| Physical activity risk (%, SE) | | | |
| Ideal (≥150 min/week moderate or ≥75 min/week vigorous or ≥150 min/week moderate+vigorous) | 47.7 (0.94) | 48.4 (0.93) | 42.6* (1.90) |
| Intermediate (1–149 min/week moderate or 1–74 min/week vigorous or 1–149 min/week moderate+vigorous) | 18.1 (0.52) | 17.9 (0.56) | 19.8 (1.23) |
| Poor (none) | 34.2 (0.95) | 33.7 (0.93) | 37.6* (1.84) |
| Diet risk (%, SE) | | | |
| Ideal (Healthy Eating Index score ≥81) | 2.5 (0.18) | 2.4 (0.20) | 2.6 (0.54) |
| Intermediate (Healthy Eating Index score 51–80) | 48.3 (0.94) | 48.9 (0.97) | 44.0* (1.83) |
| Poor (Healthy Eating Index score ≤50) | 49.2 (1.00) | 48.6 (1.00) | 53.4* (2.0) |
| Cholesterol risk (%, SE) | | | |
| Ideal (<200 mg/dL) | 46.3 (0.80) | 47.0 (0.81) | 40.7* (1.74) |
| Intermediate (200–239 mg/dL or treated to goal) | 39.9 (0.72) | 39.2 (0.72) | 45.2* (1.61) |
| Poor (≥240 mg/dL) | 13.8 (0.47) | 13.8 (0.48) | 14.1 (1.13) |
| Blood pressure risk (%, SE) | | | |
| Ideal (SBP <120/DBP <80 mm Hg) | 43.6 (0.81) | 44.7 (0.83) | 35.6* (1.53) |
| Intermediate (SBP 120–139 or DBP 80–89 mm Hg or treated to goal) | 42.2 (0.71) | 41.5 (0.70) | 47.8* (1.61) |
| Poor (SBP ≥140 or DBP ≥90 mm Hg) | 14.2 (0.47) | 13.8 (0.43) | 16.6* (1.49) |
| Diabetes mellitus risk (%, SE) | | | |
| Ideal (glucose <100 mg/dL and $A_{1c}$ <5.7%) | 73.7 (0.44) | 74.5 (0.48) | 68.3* (1.30) |
| Intermediate (glucose 100–125 mg/dL or 5.7% ≤$A_{1c}$ <6.5% or treated to goal) | 19.5 (0.38) | 18.9 (0.42) | 23.5* (1.11) |
| Poor (diagnosed diabetes mellitus or glucose ≥126 mg/dL or $A_{1c}$ ≥6.5%) | 6.8 (0.21) | 6.6 (0.23) | 8.2* (0.71) |
| Mean score (SE) of 7 health metrics† | 8.5 (0.05) | 8.6 (0.05) | 7.9* (0.1) |
| Categories of 7 health metrics†(%, SE) | | | |
| Inadequate (0–4) | 5.3 (0.24) | 5.0 (0.26) | 7.7* (0.78) |
| Average (5–9) | 58.8 (1.04) | 57.7 (1.05) | 66.6* (1.74) |
| Optimum (10–14) | 35.9 (1.03) | 37.3 (1.03) | 25.7* (1.81) |

CVD indicates cardiovascular disease; DBP, diastolic blood pressure; NHANES, National Health and Nutrition Examination Survey; SBP, systolic blood pressure; SE, standard error.
*Difference in percentage between populations with and without family history is significant at 0.05 level.
†A score of 0, 1, or 2 was assigned to each cardiovascular health metric to represent poor, intermediate, or ideal health. The overall score for the 7 health metrics ranged from 0 to 14.

at risk for CVD in the United States can be identified using family history. Among people 20 years and older in the United States, 7.4% had CVD, almost 13.4% of whom have their CVD attributable to family history (burden of 2.3 million people). In our study, younger people with an FHPHD tended to have higher odds of prevalent CVD, compared with their peers without an FHPHD. Among people in the age group 20 to 39 with CVD, around 29% could be attributed to FHPHD (burden of 0.3 million

**Table 5.** Estimates of PRs From Polytomous Logistic Regression for the Population Without Cardiovascular Disease When Inadequate CVH and Average CVH Were Compared With the Population With Optimum CVH, Adults Aged ≥20, NHANES 2007–2014

| | Inadequate CVH‡ PR (95% CI) | Average CVH‡ PR (95% CI) |
|---|---|---|
| Self-reported family history | | |
| No† | | |
| Yes | 1.98 (1.40–2.79) | 1.59 (1.31–1.92) |
| Age, y | | |
| 20–39† | | |
| 40–59 | 6.13 (4.58–8.21) | 2.67 (2.31–3.08) |
| ≥60 | 8.58 (6.56–11.22) | 4.25 (3.61–5.01) |
| Sex | | |
| Female† | | |
| Male | 1.26 (0.99–1.62) | 1.29 (1.15–1.44) |
| Race/Ethnicity | | |
| Non-Hispanic white† | | |
| Hispanic | 1.04 (0.74–1.46) | 1.10 (0.91–1.33) |
| Non-Hispanic black | 2.38 (1.82–3.12) | 1.78 (1.49–2.13) |
| Other* | 0.81 (0.48–1.36) | 0.70 (0.59–0.85) |
| Income-to-poverty ratio§ | | |
| ≥1† | | |
| <1 | 1.98 (1.42–2.76) | 1.27 (1.02–1.59) |
| Education | | |
| High school completion or greater† | | |
| Less than high school completion | 3.19 (2.42–4.20) | 2.00 (1.77–2.27) |

CVH indicates cardiovascular health; NHANES, National Health and Nutrition Examination Survey; PR, prevalence ratio.
*Non-Hispanic Asians, non-Hispanic multiracial, and non-Hispanic other race.
†Reference group.
‡A score of 0, 1, or 2 was assigned to each cardiovascular health metric to represent poor, intermediate, or ideal health. On the basis of the sum of scores for all 7 cardiovascular metrics, an overall score, ranging from 0 to 14, was categorized as inadequate (0–4), average (5–9), or optimum (10–14) cardiovascular health.
§A ratio of family income to poverty guidelines.

**Table 6.** Estimates of Age-Standardized Mean Heart Age* and Mean Excess Heart Age for the US Population Aged 30 to 74 Without CVD, NHANES 2007–2014

| | Overall Years (SE) | Without Family History of CVD† Years (SE) | With Family History of CVD† Years (SE) |
|---|---|---|---|
| Chronological age | 48.0 (0.04) | 48.0 (0.04) | 48.0 (0.10) |
| Heart age | 55.3 (0.18) | 55.0 (0.18) | 57.6‡ (0.39) |
| Excess heart age† | 7.3 (0.17) | 7.0 (0.17) | 9.6‡ (0.38) |

CVD indicates cardiovascular disease; NHANES, National Health and Nutrition Examination Survey; SE, standard error.
*Age-standardized by the direct method to the US 2000 census population using the age groups 30 to 39, 40 to 49, 50 to 59, 60 to 69, and 70 to 74 years.
†Excess heart age is the difference between heart age and chronological age.
‡Difference in age for the population with and without family history of heart disease is significant ($P<0.05$).

people). The PAF for parental history of MI (14.8%) for younger individuals (men ≤55 years and women ≤60 years) was also significantly higher in the INTERHEART study.[25]

Family history was not included in any version of the Framingham risk score to estimate CVD risk, and only a few risk calculators include family history of CVD to assess a patient's risk.[25,26] However, both parental and sibling history were found to improve prediction of CVD.[27,28] The joint 2013 American College of Cardiology/American Heart Association Taskforce guideline for the assessment of cardiovascular risk recommends for FHPHD to be considered if, after quantitative risk assessment, a risk-based treatment is uncertain. The Work Group supported revising risk assessment upward for males <55 and females <65 years of age with FHPHD.[29] As risk from family history depends on number of first-degree relatives affected, type of relatives, and the age of onset of CVD, it has been shown that using more sophisticated definitions of family history variables compared with a simple binary approach significantly improved the predictive ability of coronary heart disease risk models.[30]

The prevalence of CVD in people with an FHPHD is more than double the prevalence of CVD among people without a family history. Among people without prevalent CVD, our findings show that people with an FHPHD have a much less favorable heart health rating compared with people without a family history. Either the percentages of the population with an FHPHD in ideal health were significantly less than that for the population without a family history, or the percentages of the population with a FHPHD in poor health were significantly higher than that for the population without a family history for the 7 cardiovascular metrics considered. After controlling for other variables, the PR for FHPHD was almost double for inadequate health relative to optimal health. Our findings also show that both heart age and excess heart age were significantly higher among those who have an FHPHD compared with those without a family history. Another study of association between family history of diabetes mellitus and FHPHD and lifestyle risk factors in the US population based on NHANES 2009–2012 found that participants with an FHPHD were more likely to be current smokers and participants with a family history of both diabetes mellitus and CVD were more likely to have obesity compared with participants with no family history.[31] There was no association between family history and dietary factors or physical activity. However, their study sample did not exclude those who had already developed CVD.

The findings that people with FHPHD are in worse heart health than those without FHPHD may be unexpected to some

since the 7 cardiovascular metrics considered in our study are based on modifiable risk factors, and lifestyle modifications can reduce the risk of developing CVD. It is reasonable to expect that knowing their family history, people with FHPHD could be more motivated to make positive lifestyle and behavior changes than those without a family history or may have a more fatalistic approach to their own heart health. These data have important implications for targeting public heath, clinical, and public health education program interventions to this high-risk group. Better strategies to collect comprehensive family history of CVD may improve the effectiveness of family history as a tool for preventing CVD.[32]

## Study Limitations

There are several limitations in our study. NHANES is a cross-sectional survey, and cannot be used to show causal effect of the FHPHD on CVD. The risk of FHPHD on CVD is well known in the literature, and thus in calculating numbers attributed to FHPHD, we assumed the causality of FHPHD on CVD. Moreover, the collection of family health history information is limited in the NHANES. There have been no population-based surveys that examined the accuracy of self-reported family histories of CVD. However, other studies that investigated the accuracy of family history of CVD found that offspring report of parental history may be unreliable and may lead to overestimates of risk associated with parental CVD.[27,33,34] The differences in prevalence of reported family history between men and women also suggest that there may be a recall or knowledge bias.[35] The accuracy of self-reports of the medical history varies on the participants' knowledge of the pertinent information, ability to recall it, and inclination to report it.[36] A study conducted on patients with hypercholesterolemia enrolled in primary care centers in Germany to obtain more information on the accuracy of patient-provided data, on cardiovascular conditions compared with medical records showed excellent and substantial agreement for patient self-report and medical record regarding diabetes mellitus and hypertension but showed only moderate agreement for both MI and stroke.[37] Misreporting of medical history including FHPHD and history of CVD could introduce bias in the association study.

As the participants were asked whether any of their close biological (blood) relatives, including sisters or brothers, were ever told by a health professional that they had a heart attack or angina before the age of 50, the family history question did not distinguish between full and half siblings. Therefore, our results may also include second-degree relatives in addition to the first degree relatives. The inclusion of half siblings may dilute the impact of family history on CVD. Furthermore, the age cutoff point for the definition of premature heart disease is higher than 50 years in some studies of CVD. For example, Lloyd-Jones et al[27] defined premature parental CVD as the occurrence of a validated parental event before age 55 years in a father or age 65 years in a mother. These were also the cut points recommended by the National Cholesterol Education Program Third Adult Treatment Panel[38] and Seventh Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure.[39] A lower cut point for age for the definition of premature heart disease may increase the impact of FHPHD on CVD.

Because the definition of FHPHD is based only on heart attack or stroke, we examined the possible effect of this definition on CVD outcomes, and the patterns of associations were largely consistent for the consistent definition of FHPHD and CVD outcomes. As Healthy Eating Index–2010 was from the first-day 24-hour dietary recall, the energy intake may be underestimated by as much as 11%.[40] Another limitation in our study is the absence of specific data on genetic factors, including polygenic risk scores[41] and major genetic conditions such as familial hypercholesterolemia. However, with an estimated prevalence of 1 in 250 people, familial hypercholesterolemia accounts for a small proportion of people with an FHPHD.[42]

In conclusion, millions of people who are at high risk of having or developing CVD could be identified using FHPHD. In addition, FHPHD is associated with increased prevalence of modifiable risk factors for CVD. FHPHD can become be an important component of public health campaigns that plan to reduce the overall risk of heart disease by working on modifiable risk factors. Each year since 2004, the Surgeon General has declared Thanksgiving to be National Family History Day and encourages Americans to use his "My Family Health Portrait," which is an Internet-based tool that makes it easy to record family health history.[43] Further work is needed to assess how this knowledge can be used in public health programs such as the "Million Hearts" initiative that are targeting the reduction of heart disease in the US population.[44]

## Acknowledgments

The findings and conclusions in this article are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention. Data Access and Responsibility: Dr Moonesinghe had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

## Disclosures

None.

## References

1. Benjamin EJ, Blaha MJ, Chiuve SE, Cushman M, Das SR, Deo R, de Ferranti SD, Floyd J, Fornage M, Gillespie C, Isasi CR, Jiménez MC, Jordan LC, Judd SE, Lackland D, Lichtman JH, Lisabeth L, Liu S, Longenecker CT, Mackey RH,

Matsushita K, Mozaffarian D, Mussolino ME, Nasir K, Neumar RW, Palaniappan L, Pandey DK, Thiagarajan RR, Reeves MJ, Ritchey M, Rodriguez CJ, Roth GA, Rosamond WD, Sasson C, Towfighi A, Tsao CW, Turner MB, Virani SS, Voeks JH, Willey JZ, Wilkins JT, Wu JH, Alger HM, Wong SS, Muntner P. Heart disease and stroke statistics-2017 update: a report from the American Heart Association. *Circulation*. 2017;135:e146–e603.

2. Khaw KT, Barrett-Connor E. Family history of heart attack: a modifiable risk factor? *Circulation*. 1986;74:239–244.
3. Barrett-Connor E, Khaw KT. Family history of heart attack as an independent predictor of death due to cardiovascular disease. *Circulation*. 1984;69:1065–1069.
4. Veronesi G, Gianfagna F, Giampaoli S, Chambless LE, Mancia G, Cesana G, Ferrario MM. Improving long-term prediction of first cardiovascular event: the contribution of family history of coronary heart disease and social status. *Prev Med*. 2014;64:75–80.
5. Miller EM, Hinton RB. A pediatric approach to family history of cardiovascular disease: diagnosis, risk assessment, and management. *Pediatr Clin North Am*. 2014;61:187–205.
6. Chow CK, Islam S, Bautista L, Rumboldt Z, Yusufali A, Xie C, Anand SS, Engert JC, Rangarajan S, Yusuf S. Parental history and myocardial infarction risk across the world: the INTERHEART Study. *J Am Coll Cardiol*. 2011;57:619–627.
7. Austin MA, Hutter CM, Zimmern RL, Humphries SE. Familial hypercholesterolemia and coronary heart disease: a HuGE association review. *Am J Epidemiol*. 2004;160:421–429.
8. My Life Check—Life's Simple 7. American Heart Association. Available at: http://www.heart.org/HEARTORG/Conditions/My-Life-Check—Lifes-Simple-7_UCM_471453_Article.jsp#.W1TTOfZFy71. Accessed October 15, 2018.
9. Lloyd-Jones DM, Hong Y, Labarthe D, Mozaffarian D, Appel LJ, Van Horn L, Greenlund K, Daniels S, Nichol G, Tomaselli GF, Arnett DK, Fonarow GC, Ho PM, Lauer MS, Masoudi FA, Robertson RM, Roger V, Schwamm LH, Sorlie P, Yancy CW, Rosamond WD. Defining and setting national goals for cardiovascular health promotion and disease reduction: the American Heart Association's strategic Impact Goal through 2020 and beyond. *Circulation*. 2010;121:586–613.
10. González HM, Tarraf W, Rodríguez CJ, Gallo LC, Sacco RL, Talavera GA, Heiss G, Kizer JR, Hernandez R, Davis S, Schneiderman N, Daviglus ML, Kaplan RC. Cardiovascular health among diverse Hispanics/Latinos: Hispanic Community Health Study/Study of Latinos (HCHS/SOL) results. *Am Heart J*. 2016;176:134–144.
11. Folsom AR, Yatsuya H, Nettleton JA, Lutsey PL, Cushman M, Rosamond WD. Community prevalence of ideal cardiovascular health, by the American Heart Association definition, and relationship with cardiovascular disease incidence. *J Am Coll Cardiol*. 2011;57:1690–1696.
12. Ford ES, Greenlund KJ, Hong Y. Ideal cardiovascular health and mortality from all causes and diseases of the circulatory system among adults in the United States. *Circulation*. 2012;125:987–995.
13. Murray CJ, Atkinson C, Bhalla K, Birbeck G, Burstein R, Chou D, Dellavalle R, Danaei G, Ezzati M, Fahimi A, Flaxman D, Foreman , Gabriel S, Gakidou E, Kassebaum N, Khatibzadeh S, Lim S, Lipshultz SE, London S, Lopez , MacIntyre MF, Mokdad AH, Moran A, Moran AE, Mozaffarian D, Murphy T, Naghavi M, Pope C, Roberts T, Salomon J, Schwebel DC, Shahraz S, Sleet DA, Murray , Abraham J, Ali MK, Bartels DH, Birbeck G, Burstein R, Chen H, Criqui MH, Dahodwala , Jarlais , Ding EL, Dorsey ER, Ebel BE, Ezzati M, Fahami , Flaxman S, Flaxman AD, Gonzalez-Medina D, Grant B, Hagan H, Hoffman H, Kassebaum N, Khatibzadeh S, Leasher JL, Lin J, Lipshultz SE, Lozano R, Lu Y, Mallinger L, McDermott MM, Micha R, Miller TR, Mokdad AA, Mokdad AH, Mozaffarian D, Naghavi M, Narayan KM, Omer SB, Pelizzari PM, Phillips D, Ranganathan D, Rivara FP, Roberts T, Sampson U, Sanman E, Sapkota A, Schwebel DC, Sharaz S, Shivakoti R, Singh GM, Singh D, Tavakkoli M, Towbin JA, Wilkinson JD, Zabetian A, Murray , Abraham J, Ali MK, Alvardo M, Baddour LM, Benjamin EJ, Birbeck G, Bolliger I, Burstein R, Carnahan E, Chou D, Chugh SS, Cohen A, Colson KE, Cooper LT, Couser W, Criqui MH, Dabhadkar KC, Dellavalle RP, Jarlais , Dicker D, Dorsey ER, Duber H, Ebel BE, Engell RE, Ezzati M, Felson DT, Finucane MM, Flaxman S, Flaxman AD, Fleming T, Foreman , Forouzanfar MH, Freedman G, Freeman MK, Gakidou E, Gillum RF, Gonzalez-Medina D, Gosselin R, Gutierrez HR, Hagan H, Havmoeller R, Hoffman H, Jacobsen KH, James SL, Jasrasaria R, Jayarman S, Johns N, Kassebaum N, Khatibzadeh S, Lan Q, Leasher JL, Lim S, Lipshultz SE, London S, Lopez , Lozano R, Lu Y, Mallinger L, Meltzer M, Mensah GA, Michaud C, Miller TR, Mock C, Moffitt TE, Mokdad AA, Mokdad AH, Moran A, Naghavi M, Narayan KM, Nelson RG, Olives C, Omer SB, Ortblad K, Ostro B, Pelizzari PM, Phillips D, Raju M, Razavi H, Ritz B, Roberts T, Sacco RL, Salomon J, Sampson U, Schwebel DC, Shahraz S, Shibuya K, Silberberg D, Singh JA, Steenland K, Taylor JA, Thurston GD, Vavilala MS, Vos T, Wagner GR, Weinstock MA, Weisskopf MG, Wulf S, Murray ; U.S. Burden of Disease Collaborators. The state of US health, 1990–2010: burden of diseases, injuries, and risk factors. *JAMA*. 2013;310:591–608.
14. Yang Q, Cogswell ME, Flanders WD, Hong Y, Zhang Z, Loustalot F, Gillespie C, Merritt R, Hu FB. Trends in cardiovascular health metrics and associations with all-cause and CVD mortality among US adults. *JAMA*. 2012;307:1273–1283.
15. Caleyachetty R, Echouffo-Tcheugui JB, Muennig P, Zhu W, Muntner P, Shimbo D. Association between cumulative social risk and ideal cardiovascular health in US adults: NHANES 1999–2006. *Int J Cardiol*. 2015;191:296–300.
16. National Health and Nutrition Examination Survey. Available at: https://wwwn.cdc.gov/nchs/nhanes/default.aspx. Accessed June 17, 2019.
17. Osborn J, Cattaruzza MS. Odds ratio and relative risk for cross sectional data. *Int J Epidemiol*. 1995;24:463–464.
18. Centers for Disease Control and Prevention, National Health and Nutrition Examination Survey, Response rates and population totals. Available at: https://wwwn.cdc.gov/nchs/nhanes/ResponseRates.aspx. Accessed October 16, 2018.
19. Guenther PM, Casavale KO, Reedy J, Kirkpatrick SI, Hiza HA, Kuczynski KJ, Kahle LL, Krebs-Smith SM. Update of the Healthy Eating Index: HEI-2010. *J Acad Nutr Diet*. 2013;113:569–580.
20. American Diabetes Association. Standards of medical care in diabetes–2010. *Diabetes Care*. 2010;33(suppl 1):S11–S61.
21. Kulshreshtha A, Goyal A, Veledar E, McClellan W, Judd S, Eufinger SC, Bremner JD, Goldberg J, Vaccarino V. Association between ideal cardiovascular health and carotid intima-media thickness: a twin study. *J Am Heart Assoc*. 2013;2:e000282. DOI: 10.1161/JAHA.113.000282.
22. Zefeng Z, Jackson SL, Merritt R, Gillespie C, Yang Q. Association between cardiovascular health metrics and depression among U.S. adults: National Health and Nutrition Examination Survey, 2007–2014. *Ann Epidemiol*. 2019;31:49–56.e2.
23. D'Agostino RB Sr, Vasan RS, Pencina MJ, Wolf PA, Cobain M, Massaro JM, Kannel WB. General cardiovascular risk profile for use in primary care: the Framingham Heart Study. *Circulation*. 2008;117:743–753.
24. Yang Q, Zhong Y, Ritchey M, Cobain M, Gillespie C, Merritt R, Hong Y, George MG, Bowman BA. Vital Signs: Predicted heart age and racial disparities in heart age among U.S. adults at the State level. Available at: https://www.cdc.gov/mmwr/preview/mmwrhtml/mm6434a6.htm. Accessed October 16, 2018.
25. Prabhakaran D, Jeemon P. Should your family history of coronary heart disease scare you? *Mt Sinai J Med*. 2012;79:721–732.
26. Garg N, Muduli SK, Kapoor A, Tewari S, Kumar S, Khanna R, Goel PK. Comparison of different cardiovascular risk score calculators for cardiovascular risk prediction and guideline recommended statin uses. *Indian Heart J*. 2017;69:458–463.
27. Lloyd-Jones DM, Nam BH, D'Agostino RB Sr, Levy D, Murabito JM, Wang TJ, Wilson PW, O'Donnell CJ. Parental cardiovascular disease as a risk factor for cardiovascular disease in middle-aged adults: a prospective study of parents and offspring. *JAMA*. 2004;291:2204–2211.
28. Murabito JM, Pencina MJ, Nam BH, D'Agostino RB Sr, Wang TJ, Lloyd-Jones D, Wilson PW, O'Donnell CJ. Sibling cardiovascular disease as a risk factor for cardiovascular disease in middle-aged adults. *JAMA*. 2005;294:3117–3123.
29. Goff DC Jr, Lloyd-Jones DM, Bennett G, Coady S, D'Agostino RB Sr, Gibbons R, Greenland P, Lackland DT, Levy D, O'Donnell CJ, Robinson JG, Schwartz JS, Shero ST, Smith SC Jr, Sorlie P, Stone NJ, Wilson PWF. 2013 ACC/AHA guideline on the assessment of cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2014;129:S49–S73.
30. Ciampi A, Courteau J, Niyonsenga T, Xhignesse M, Lussier-Cacan S, Roy M. Family history and the risk of coronary heart disease: comparing predictive models. *Eur J Epidemiol*. 2001;17:609–620.
31. Akhuemonkhan E, Lazo M. Association between family history of diabetes and cardiovascular disease and lifestyle risk factors in the United States population: the 2009–2012 National Health and Nutrition Examination Survey. *Prev Med*. 2017;96:129–134.
32. Hunt SC, Gwinn M, Adams TD. Family history assessment: strategies for prevention of cardiovascular disease. *Am J Prev Med*. 2003;24:136–142.
33. Murabito JM, Nam BH, D'Agostino RB Sr, Lloyd-Jones DM, O'Donnell CJ, Wilson PW. Accuracy of offspring reports of parental cardiovascular disease history: the Framingham Offspring Study. *Ann Intern Med*. 2004;140:434–440.
34. Bensen JT, Liese AD, Rushing JT, Province M, Folsom AR, Rich SS, Higgins M. Accuracy of proband reported family history: the NHLBI Family Heart Study (FHS). *Genet Epidemiol*. 1999;17:141–150.
35. Hariri S, Yoon PW, Moonesinghe R, Valdez R, Khoury MJ. Evaluation of family history as a risk factor and screening tool for detecting undiagnosed diabetes in a nationally representative survey population. *Genet Med*. 2006;8:752–759.
36. Goldman N, Lin IF, Weinstein M, Lin YH. Evaluating the quality of self-reports of hypertension and diabetes. *J Clin Epidemiol*. 2003;56:148–154.
37. Englert H, Müller-Nordhorn J, Seewald S, Sonntag F, Völler H, Meyer-Sabellek W, Wegscheider K, Windler E, Katus H, Willich SN. Is patient self-report an

adequate tool for monitoring cardiovascular conditions in patients with hypercholesterolemia? *J Public Health*. 2010;32:387–394.

38. *Circulation*. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. 2002;106:3143–3421.
39. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, Jones DW, Materson BJ, Oparil S, Wright JT Jr, Roccella EJ. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension*. 2003;42:1206–1252.
40. Espeland MA, Kumanyika S, Wilson AC, Reboussin DM, Easter L, Self M, Robertson J, Brown WM, McFarlane M; TONE Cooperative Research Group. Statistical issues in analyzing 24-hour dietary recall and 24-hour urine collection data for sodium and potassium intakes. *Am J Epidemiol*. 2001;153:996–1006.
41. Knowles JW, Ashley EA. Cardiovascular disease: the rise of the genetic risk score. *PLoS Med*. 2018;15:e1002546.
42. Bucholz EM, Rodday AM, Kolor K, Khoury MJ, de Ferranti SD. Prevalence and predictors of cholesterol screening, awareness, and statin treatment among US adults with familial hypercholesterolemia or other forms of severe dyslipidemia (1999–2014). *Circulation*. 2018;137:2218–2230.
43. My family health portrait- a tool from the Surgeon General. Available at: https://phgkb.cdc.gov/FHH/html/index.html. Accessed May 6, 2019.
44. Frieden TR, Berwick DM. The "Million Hearts" initiative–preventing heart attacks and strokes. *N Engl J Med*. 2011;365:e27.