Predicted Long-Term Cardiovascular Risk Among Young Adults in the National Longitudinal Study of Adolescent Health

Cari Jo Clark, ScD, MPH, Alvaro Alonso, MD, PhD, Rachael A. Spencer, MPA, Michael Pencina, PhD, Ken Williams, MS, PStat, and Susan A. Everson-Rose, PhD, MPH

Objectives. We estimated the distribution of predicted long-term cardiovascular disease (CVD) risk among young adults in the United States.

Methods. Our data were derived from National Longitudinal Study of Adolescent Health participants (n = 14 333; average age: 28.9 years). We used a Framingham-derived risk prediction function to calculate 30-year risks of "hard" and "general" CVD by gender and race/ethnicity.

Results. Average 30-year risks for hard and general CVD were 10.4% (95% confidence interval [CI] = 10.1%, 10.7%) and 17.3% (95% CI = 17.0%, 17.7%) among men and 4.4% (95% CI = 4.3%, 4.6%) and 9.2% (95% CI = 8.9%, 9.5%) among women. Average age-adjusted risks of hard and general CVD were higher among Blacks and American Indians than among Whites and lower among Asian/Pacific Islander women than White women. American Indian men continued to have a higher risk of general CVD after adjustment for socioeconomic status. Four percent of women (95% CI = 3.6%, 5.0%) and 26.2% of men (95% CI = 24.7%, 27.8%) had a 20% or higher risk of general CVD. Racial differences were detected but were not significant after adjustment for socioeconomic status.

Conclusions. Average CVD risk among young adults is high. Population-based prevention strategies and improved detection and treatment of high-risk individuals are needed to reduce the future burden of CVD. (*Am J Public Health.* 2014;104:e108–e115. doi:10.2105/AJPH.2014.302148)

Estimated costs of cardiovascular disease (CVD) in the United States are expected to be more than $1 trillion by 2030.[1] This fact, along with growing evidence of the cost effectiveness of primordial and primary prevention,[2] has prompted increasing attention to the extent of CVD risk early in life. Elevated CVD risk is detectable in childhood[3] and pronounced by young adulthood; among young adults 18 to 25 years of age, 34.2% report current smoking,[4] and 23.8% and 16.8% are overweight and obese, respectively.[5] Hypertension rates are estimated to be as high as 7.2% among those 18 to 24 years of age[5] and may be as high as 20.9% among those 25 to 32 years of age.[6] These individual statistics, although alarming, do not account for the co-occurrence of risk factors and the differing strength of their relationship to CVD as is done with risk prediction.

The application of risk prediction functions to large epidemiological data sets could serve as a useful indicator of the burden of CVD among young adults.[7] However, most functions were designed to be used with middle-aged or older adults, and they predict risk over a 5- to 10-year time period. This time frame is too short for most young adults given that incident disease will occur over decades, rather than years, after assessment. Existing research involving data from the National Health and Nutrition Examination Survey has shown that the overwhelming majority (82%) of US adults have a low 10-year risk of coronary heart disease (CHD), but nearly two thirds of these individuals have a high long-term risk of CVD.[8]

Longer-term risk assessments are a better predictor of subclinical[9,10] and clinical CVD[11] than shorter-term risk prediction functions and take account of competing causes of death, thereby providing a more realistic assessment of the overall burden of CVD.[7,11] Existing longer-term estimates suggest a high lifetime risk of CVD among both men (60%) and women (56%).[12] However, these estimates have been made in reference to individuals 45 years of age. Although considerable data are available on levels of individual risk factors among young adults, concurrent consideration of multiple risk factors better discriminates longer-term risk than any single risk factor.[13] To our knowledge, no estimates of this type currently exist for the US young adult population. Thus, estimates of CVD risk among young adults are needed to more accurately estimate the extent of risk in this population and to more accurately predict future disease burden.

The 30-year Framingham risk score (30-year FRS) is the only longer-term risk prediction function designed to be used with young adults. The function was developed with the Framingham offspring cohort, and analyses of these data represent the only estimates of the extent of long-term risk among young adults. However, the participants in that study were recruited in the 1970s, and CVD risk factors have since changed significantly; there has been an increase in average body mass index (BMI)[14] and in the prevalence of diabetes,[15] whereas the prevalence of smoking has decreased.[16] A more accurate estimate of the extent of long-term CVD risk among young adults requires an application of the 30-year risk prediction function to a contemporary sample of young adults. In this study, we began to fill this gap by using the 30-year FRS to provide the first, to our knowledge, nationally representative estimates of long-term CVD risk by gender and race/ethnicity. We also examined racial/ethnic differences in the prevalence of "high" CVD risk (a risk score of 20% or higher).

METHODS

We used data from the National Longitudinal Study of Adolescent Health (Add Health),[17] a school-based longitudinal study of a nationally representative sample of US adolescents in grades 7 to 12 during the 1994–1995 school year. A sample of 80 high schools and 52 middle schools was selected with unequal probability of selection. Incorporating systematic sampling methods and implicit stratification into the Add Health study design ensured that the sample was representative of US schools with respect to region of the country, degree to which the location was urbanized, school size, school type, and race/ethnicity. Student rosters were used to select a sample of 20 745 adolescents at baseline (wave 1) that included a number of oversampled groups such as adolescents with disabilities and minority adolescents (Chinese, Cuban, Puerto Rican, and Black adolescents with at least 1 parent with a college degree). A parent was also surveyed at baseline to gather information on the adolescent's health status and behaviors, home environment, and interpersonal relationships.

Three waves followed, the most recent of which (wave 4, 2008–2009) included data from which the 30-year FRS could be calculated. Response rates in the 4 waves were 79%, 89%, 77%, and 80%, respectively. Our analysis was restricted to participants with valid sampling weights at wave 4 (n = 14 800) who had not been diagnosed with heart disease or cancer and were Hispanic, non-Hispanic White, non-Hispanic Black, American Indian/Native American (hereafter American Indian), or Asian/Pacific Islander (n = 14 333).

Measures

We used the 30-year FRS, a prediction model developed by Pencina et al.,[11] to estimate participants' risk of developing CVD over a 30-year time frame. In the prediction function, information on age, gender, and other risk factors is used to predict the risk of occurrence of 2 composite CVD endpoints: "hard" CVD (coronary death, myocardial infarction, and fatal and nonfatal stroke) and "general" CVD (coronary death, myocardial infarction, coronary insufficiency, angina pectoris, stroke, transient ischemic attack, intermittent claudication, and congestive heart failure).[11] We also generated a dichotomous measure of "high" general CVD risk with a cut point of 20%, the highest risk level defined in the National Cholesterol Education Program Expert Panel's guidelines on the detection, evaluation, and treatment of high blood cholesterol in adults.[18] These guidelines refer to a 10-year risk prediction function, but there are currently no validated cut points for the 30-year risk score. However, a 20% risk in the Add Health population would indicate that 20 of 100 individuals will develop CVD over 30 years (which would be between the ages of 54 and 64 years for this population).

Risk factors used to calculate the 30-year FRS include gender, age, systolic blood pressure (SBP), use of antihypertensive medication, smoking, diabetes, and BMI (weight in kilograms divided by the square of height in meters), all of which were ascertained from interview data, anthropometric measurements, and biological specimens taken at wave 4. Standardized approaches to height and weight measurements were used.[19] Participants' SBP was measured after a 5-minute seated rest[20]; 3 measurements were obtained at 30-second intervals, and the latter 2 readings were averaged to calculate resting SBP. Interviewers used a medication inventory to assess use of antihypertensive medication in the preceding 4 weeks.[21]

Cigarette smoking in the 30 days preceding the interview was ascertained via self-report. The validity of self-reported smoking has been shown to be consistently high in population-based studies.[22] Respondents were considered to have diabetes if they had a fasting glucose level of 126 milligrams per deciliter or above, a nonfasting glucose level of 200 milligrams per deciliter or above, or an HbA1c of 6.5% or above; if they reported having been diagnosed with diabetes by a health provider (except during pregnancy); or if they had used antidiabetic medication in the preceding 4 weeks.[23]

Race/ethnicity was assessed at wave 1; participants were asked if they were Hispanic and were asked to select a racial category (White, Black/African American, American Indian/Native American, Asian/Pacific Islander, or other). Participants could choose more than 1 racial category and were asked to select the one that best described their racial background.

Socioeconomic status (SES) was measured via multiple indicators of current, early life, and neighborhood SES, given that the accumulation of low-SES experiences across the life span is a consistent contributor to poor adult health[24] and that SES at the neighborhood level may have a unique impact on health.[25] Wave 4 SES indicators included educational attainment and household income, categorized into theoretically meaningful levels, and financial stress, defined as a positive response to any of 6 items indicating an inability to pay one's full rent or mortgage or one's utility bill, loss of telephone or utility service, food scarcity, and eviction.

Family SES at baseline (1994–1995), when the participants were in grades 7 through 12, was measured with 3 variables: parental education (a 10-category variable ranging from no education to education or training beyond college), parents' response (yes or no) to a question asking whether they had enough money to pay their bills, and neighborhood poverty (the proportion of families in respondents' census block group with an income below the poverty line in 1989).

Statistical Analysis

Descriptive statistics were calculated by gender and race/ethnicity. We used bivariate linear and logistic models to examine differences in study variables according to race/ethnicity. Restricted cubic spline functions[26] were used to examine the linear associations of continuous variables (educational attainment, household income, parental income, and block group poverty) with CVD risk. The associations involving household and parental income were determined to be linear, but the associations involving educational attainment and poverty were not. Quadratic terms for the latter 2 variables were included in subsequent regression models to optimally control for these potential confounders.

We used linear models adjusted for age and race/ethnicity to calculate mean 30-year FRS values for hard and general CVD separately for men and women. Gender-disaggregated models were used because the risk of CVD is known to be much higher among young men than young women,[15] and therefore a combined mean score is less informative than gender-specific means. We included age and

race/ethnicity to adjust for differences in risk that might simply be a result of differences in the sociodemographic composition of the subgroups (i.e., gender and race/ethnicity) being compared. We computed additional models incorporating current and baseline measures of SES to determine whether any potential racial/ethnic differences could be attributed to racial/ethnic differences in SES. Similar logistic models were computed to examine racial/ethnic differences in the prevalence of high CVD risk.

In our regression analyses, we used PROC MI in SAS version 9.3 (SAS Institute, Cary, NC) to perform multiple imputation by gender and racial/ethnic group. Twenty-five data sets were generated. In addition to the study variables, we included several auxiliary variables in the imputation process to improve power and reduce nonresponse bias.[27] Wave 4 auxiliary variables included the individual CVD score components, waist circumference, frequency of moderate exercise and alcohol consumption, and whether the participant had been born in the United States. Wave 1 auxiliary variables included parental employment, welfare receipt, and block group measures of unemployment rate and the proportion of households with children that were headed by women. Per Add Health guidelines,[28] we used SUDAAN version 11 (RTI International, Research Triangle Park, NC) survey procedures in computing descriptive statistics and regression models to correct for design effects and unequal probability of selection so that our estimates would be unbiased and nationally representative.

RESULTS

The mean age of the participants was 28.9 years (95% confidence interval [CI] = 28.6, 29.1); 68% were non-Hispanic White (n = 7931), 16% were non-Hispanic Black (n = 3101), 0.7% were American Indian (n = 109), 3.2% were Asian/Pacific Islander (n = 883), and 12.1% were Hispanic (n = 2309). On average, men and women in the sample were overweight (average BMIs of 29.2 for women and 29.0 for men) and had normal to elevated SBP (120.0 mm Hg among women and 129.9 mm Hg among men). Approximately one third of women and 42.7% of men were current smokers, and nearly 6% of both men and women had diabetes (Tables 1 and 2).

Overall, Blacks, American Indians, and Hispanics were of lower SES than Whites, and Asian/Pacific Islanders were of higher SES than Whites. Relative to White women, American Indian women had consistently worse risk profiles, Black women had worse risk profiles with the exception of smoking, and Asian/Pacific Islanders had generally more favorable risk profiles. Differences in risk profiles between Hispanic women and White women were mixed; Hispanics had higher BMIs and a higher percentage had diabetes, but fewer of these women smoked. Men exhibited similar but less pronounced differences in risk patterns.

Average 30-year risks for hard and general CVD were 10.4% (95% CI = 10.1%, 10.7%) and 17.3% (95% CI = 17.0%, 17.7%) among men and 4.4% (95% CI = 4.3%, 4.6%) and 9.2% (95% CI = 8.9%, 9.5%) among women, respectively. Table 3 presents results by gender and race/ethnicity. Relative to White women, mean age-adjusted 30-year risk scores were higher among Black and American Indian women, lower among Asian/Pacific Islander women, and similar among Hispanic women. In the case of men, mean age-adjusted 30-year risks of hard and general CVD were higher among Blacks and American Indians than among Whites, and risks were similar among Hispanics, Asian/Pacific Islanders, and Whites. Adjustment for SES attenuated all Black–White differences to nonsignificance and amplified the reduced risk among Hispanic women relative to White women. American Indian men had a higher risk of general CVD than White men after adjustment for SES.

Four percent of women (95% CI = 3.6%, 5.0%) and 26.2% of men (95% CI = 24.8%, 27.8%) had a 20% or greater risk of CVD. Significantly more Blacks than Whites and significantly fewer Asian/Pacific Islander men than White men had a 20% or higher risk in age-adjusted models; adjustment for SES attenuated all differences to nonsignificance (Figure 1).

DISCUSSION

We found that, among a nationally representative sample of young adults, predicted long-term CVD risk is sizable, risks are disproportionately high among Blacks and American Indians, and a substantial number of men have "high" CVD risk (a risk score of 20% or higher). Our findings extend previous research on longer-term risks among middle-aged and older adults to younger adults[12,13,29] and provide the first estimates of the extent and distribution of long-term CVD risk in the US young adult population. Longer-term risk prediction is increasingly being promoted to more accurately estimate the likelihood of a CVD event associated with an adverse risk profile in young adulthood and to provide a more realistic assessment of the future CVD burden. However, until this study, no estimate of the extent of long-term risk among young adults has been available.

Our findings support the need for intensified prevention, identification, and treatment of CVD risk factors. The extent of predicted CVD risk (9% among women and 17% among men) and the high prevalence of individual risk factors we documented highlight the need for a population-based approach to CVD prevention, which has the potential to save more lives than a focus solely on high-risk individuals.[30] However, the 2 approaches are complementary,[30] and the sizable subpopulation of high-risk young adults in this study indicates the need for both strategies. Twenty-six percent of men and 4% of women had a 1 in 5 chance or greater of developing CVD over a 30-year window, which for the members of this cohort would be well in advance of their 65th birthday. These percentages represent more than 3.5 million young adults and suggest that better identification and more intensive risk factor treatment are needed.

Guidelines recently released by the American College of Cardiology and the American Heart Association recommend longer-term risk assessment of all individuals 20 to 39 years of age, as well as individuals 40 to 59 years of age who have a low (< 7.5%) 10-year risk of CVD, to guide decisions on the intensity and type of lifestyle modification.[31] Adverse health behaviors are highly prevalent and are the major contributors to CVD risk, and behavior change is both feasible and effective in reducing CVD risk.[32] However, the impact of communicating long-term predicted risk scores on behavior change has not been studied. Sharing 10-year CHD risk scores with adult patients at moderate to high risk has been

TABLE 1—Characteristics of Female Participants, by Race/Ethnicity: National Longitudinal Study of Adolescent Health, United States, 2008–2009

Characteristic	Total (n = 7572), No., No. (%), or Mean (95% CI)	White (n = 4132), No., No. (%), or Mean (95% CI)	Black (n = 1750), No., No. (%), or Mean (95% CI)	American Indian (n = 57), No., No. (%), or Mean (95% CI)	Asian/Pacific Islander (n = 425), No., No. (%), or Mean (95% CI)	Hispanic (n = 1208), No., No. (%), or Mean (95% CI)
Age, y	28.8 (28.5, 29.0)	28.7 (28.4, 28.9)	29.0 (28.6, 29.4)	28.7 (28.1, 29.3)	29.1 (28.5, 29.7)	28.9 (28.4, 29.4)
Education						
< high school	459 (7.5)	227 (6.5)	124 (9.4)	6 (8.3)	9 (3.2)	93 (11.7)
High school	1029 (14.2)	553 (12.9)	224 (16.9)	13 (21.3)	35 (7.7)	204 (19.0)
Some college	3351 (44.3)	1745 (43.0)	828 (47.8)	30 (51.6)	171 (41.3)	577 (47.2)
≥ college	2731 (33.9)	1607 (37.6)	573 (25.5)	8 (18.8)	210 (47.8)	333 (22.0)
Missing	2 (0.1)	0	1 (0.4)	0	0	1 (< 0.1)
Financial stress						
No	5548 (72.7)	3157 (76.2)	1061 (55.9)	31 (50.4)	376 (86.6)	923 (73.1)
Yes	2019 (27.1)	973 (23.6)	688 (44.1)	26 (49.6)	49 (13.4)	283 (26.6)
Missing	5 (0.2)	2 (0.2)	1 (< 0.1)	0	0	2 (0.3)
Household income, $						
< 25 000	1291 (17.8)	592 (14.6)	503 (35.7)	16 (26.8)	37 (10.4)	143 (12.7)
25 000–49 999	1985 (25.9)	1041 (25.1)	521 (28.2)	12 (27.7)	83 (21.8)	328 (28.3)
50 000–74 999	1744 (22.9)	1005 (24.1)	332 (17.0)	16 (24.4)	84 (24.0)	307 (23.8)
≥ 75 000	2087 (26.3)	1273 (30.0)	268 (11.6)	7 (11.4)	197 (40.7)	342 (22.5)
Missing	465 (7.1)	221 (6.2)	126 (7.5)	6 (9.7)	24 (3.1)	88 (12.8)
Parental education						
< high school	1162 (14.8)	392 (9.6)	256 (18.7)	16 (30.9)	38 (19.1)	460 (36.7)
High school	1919 (28.8)	1215 (30.9)	419 (29.0)	10 (21.6)	45 (10.8)	230 (21.6)
Some college	1838 (24.4)	1110 (26.3)	432 (22.5)	18 (29.9)	64 (11.8)	214 (18.7)
≥ college	1544 (18.7)	944 (21.6)	341 (12.1)	11 (14.1)	137 (28.9)	111 (8.7)
Missing	1109 (13.4)	471 (11.6)	302 (17.7)	2 (3.6)	141 (29.4)	193 (14.3)
Parental ability to pay bills						
No	1193 (15.0)	474 (11.5)	381 (23.2)	13 (29.3)	42 (13.0)	283 (23.8)
Yes	5134 (70.2)	3134 (75.9)	1026 (57.1)	40 (65.8)	234 (55.0)	700 (60.0)
Missing	1245 (14.8)	524 (12.7)	343 (19.8)	4 (4.9)	149 (32.1)	225 (16.2)
Neighborhood poverty score[a]	0.12 (0.10, 0.14)	0.09 (0.07, 0.10)	0.24 (0.21, 0.28)	0.21 (0.10, 0.32)	0.10 (0.06, 0.13)	0.15 (0.12, 0.18)
Neighborhood poverty score missing	113	59	41	0	3	10
BMI, kg/m^2	29.2 (28.8, 29.6)	28.4 (28.0, 28.8)	32.6 (31.9, 33.4)	32.0 (26.8, 37.2)	25.6 (24.1, 27.2)	29.8 (29.1, 30.5)
BMI missing	111	56	26	3	8	18
SBP, mm Hg	112.0 (119.5, 120.5)	119.6 (119.0, 120.2)	122.6 (121.9, 123.3)	123.9 (119.5, 128.3)	117.9 (115.6, 120.2)	118.9 (117.8, 120.1)
SBP missing	270	114	67	2	8	79
Hypertensive medication use						
No	7364 (97.3)	4027 (97.5)	1677 (95.7)	54 (96.4)	413 (98.8)	1193 (98.3)
Yes	208 (2.7)	105 (2.5)	73 (4.3)	3 (3.6)	12 (1.2)	15 (1.7)
Current smoker						
No	5226 (65.8)	2587 (61.0)	1355 (75.6)	28 (42.7)	325 (78.1)	931 (77.2)
Yes	2301 (33.6)	1533 (38.6)	371 (22.7)	29 (57.3)	99 (21.6)	269 (21.9)
Missing	45 (0.7)	12 (0.4)	24 (1.8)	0	1 (0.4)	8 (0.9)
Diabetes						
No	7031 (93.9)	3943 (95.8)	1528 (86.4)	46 (87.5)	396 (95.2)	1118 (93.3)
Yes	541 (6.1)	189 (4.2)	222 (13.6)	11 (12.5)	29 (4.9)	90 (6.7)

Note. BMI = body mass index; CI = confidence interval; SBP = systolic blood pressure. Data on age, hypertensive medication use, and diabetes were not missing for any participants. The sample size was n = 7572.

[a]The proportion of families in respondents' census block group with an income below the poverty line in 1989.

TABLE 2—Characteristics of Male Participants, by Race/Ethnicity: National Longitudinal Study of Adolescent Health, United States, 2008–2009

Characteristic	Total (n = 6761), No., No. (%), or Mean (95% CI)	White (n = 3799), No., No. (%), or Mean (95% CI)	Black (n = 1351), No., No. (%), or Mean (95% CI)	American Indian (n = 52), No., No. (%), or Mean (95% CI)	Asian/Pacific Islander (n = 458), No., No. (%), or Mean (95% CI)	Hispanic (n = 1101), No., No. (%), or Mean (95% CI)
Age, y	29.0 (28.7, 29.2)	28.9 (28.6, 29.2)	29.2 (28.8, 29.7)	28.7 (27.9, 29.5)	29.1 (28.5, 29.6)	29.0 (28.6, 29.5)
Education						
< high school	641 (10.7)	312 (8.8)	158 (16.6)	9 (22.9)	17 (1.7)	145 (15.5)
High school	1294 (21.3)	704 (20.2)	280 (25.6)	15 (31.4)	53 (14.8)	242 (23.4)
Some college	2943 (41.4)	1621 (41.5)	616 (40.2)	22 (38.5)	159 (31.5)	525 (45.5)
≥ college	1881 (26.5)	1162 (29.6)	296 (17.2)	6 (7.2)	229 (52.0)	188 (15.6)
Missing	2 (0.1)	0	1 (0.5)	0	0	1 (< 0.1)
Financial stress						
No	5290 (76.9)	3012 (78.6)	956 (68.1)	28 (53.3)	415 (90.2)	879 (76.9)
Yes	1460 (22.9)	783 (21.3)	393 (31.4)	23 (46.4)	43 (9.8)	218 (22.5)
Missing	11 (0.2)	4 (0.1)	2 (0.5)	1 (0.3)	0	4 (0.6)
Household income, $						
< 25 000	873 (14.5)	432 (12.6)	300 (26.9)	8 (20.7)	29 (7.4)	104 (10.7)
25 000–49 999	1735 (26.1)	988 (26.2)	355 (24.8)	17 (34.2)	81 (13.3)	294 (29.9)
50 000–74 999	1553 (22.2)	933 (23.6)	266 (17.2)	11 (12.9)	82 (20.5)	261 (21.7)
≥ 75 000	2131 (29.5)	1246 (31.8)	287 (18.1)	10 (16.4)	230 (47.6)	358 (27.5)
Missing	469 (7.7)	200 (5.8)	143 (13.1)	6 (15.8)	36 (11.3)	84 (10.2)
Parental education						
< high school	910 (14.0)	313 (8.9)	144 (16.9)	4 (16.8)	30 (11.3)	419 (39.7)
High school	1708 (27.7)	1118 (30.6)	310 (25.6)	18 (37.0)	65 (9.7)	197 (18.8)
Some college	1804 (26.4)	1142 (29.2)	363 (23.6)	16 (28.7)	84 (15.0)	199 (17.4)
≥ college	1424 (19.6)	864 (21.7)	301 (15.7)	10 (8.1)	144 (38.4)	105 (8.4)
Missing	915 (12.2)	362 (9.6)	233 (18.3)	4 (9.3)	135 (25.5)	181 (15.7)
Parental ability to pay bills						
No	968 (14.4)	413 (11.2)	271 (23.4)	8 (20.2)	37 (12.6)	239 (21.2)
Yes	4762 (71.3)	2960 (77.5)	823 (55.7)	40 (70.5)	281 (61.7)	658 (59.5)
Missing	1031 (14.2)	426 (11.3)	257 (21.0)	4 (9.3)	140 (25.8)	204 (19.4)
Neighborhood poverty score[a]	0.12 (0.10, 0.14)	0.09 (0.07, 0.10)	0.23 (0.20, 0.26)	0.21 (0.09, 0.34)	0.07 (0.05, 0.08)	0.16 (0.14, 0.18)
Neighborhood poverty score missing	105	57	28	2	6	12
BMI, kg/m^2	29.0 (28.7, 29.3)	28.8 (28.5, 29.1)	29.1 (28.5, 29.7)	32.9 (28.5, 37.3)	28.0 (26.6, 29.3)	30.1 (29.3, 30.9)
BMI missing	96	49	19	2	8	18
SBP, mm Hg	129.9 (129.4, 130.4)	129.9 (129.4, 130.5)	130.0 (128.7, 131.3)	134.2 (127.1, 141.3)	129.1 (126.4, 131.8)	129.6 (128.3, 131.0)
SBP missing	212	83	57	3	14	55
Hypertensive medication use						
No	6555 (96.6)	3664 (96.1)	1314 (97.4)	50 (95.0)	448 (97.5)	1079 (98.1)
Yes	206 (3.4)	135 (3.9)	37 (2.6)	2 (5.0)	10 (2.5)	22 (2.0)
Current smoker						
No	3972 (56.0)	2133 (55.3)	793 (54.4)	23 (43.5)	300 (66.7)	723 (60.3)
Yes	2719 (42.7)	1649 (44.3)	520 (41.1)	29 (56.5)	156 (33.3)	365 (37.5)
Missing	70 (1.3)	17 (0.5)	38 (4.5)	0	2 (< 0.1)	13 (2.2)
Diabetes						
No	6318 (93.7)	3646 (95.7)	1182 (86.6)	45 (85.1)	429 (95.8)	1016 (91.6)
Yes	443 (6.3)	153 (4.4)	169 (13.5)	7 (15.0)	29 (4.2)	85 (8.4)

Note. BMI = body mass index; CI = confidence interval; SBP = systolic blood pressure. Data on age, hypertensive medication use, and diabetes were not missing for any participants. The sample size was n = 6761.

[a]The proportion of families in respondents' census block group with an income below the poverty line in 1989.

TABLE 3—Mean 30-Year Cardiovascular Disease Risk, by Gender and Race/Ethnicity: National Longitudinal Study of Adolescent Health, United States, 2008–2009

	Hard CVD[a]				General CVD[b]			
Race/Ethnicity	Mean (95% CI)[c]	*P*	Mean (95% CI)[d]	*P*	Mean (95% CI)[c]	*P*	Mean (95% CI)[d]	*P*
Women (n = 7572)								
White (Ref)	4.3 (4.1, 4.5)		4.4 (4.3, 4.6)		8.9 (8.5, 9.2)		9.2 (8.9, 9.4)	
Black	5.2 (4.9, 5.5)	< .01	4.7 (4.4, 5.0)	.1	10.5 (10.0, 10.9)	< .01	9.6 (9.1, 10.0)	.15
American Indian	7.2 (4.2, 10.3)	.05	6.7 (4.0, 9.4)	.1	13.4 (9.3, 17.6)	.03	12.5 (8.9, 16.2)	.08
Asian/Pacific Islander	3.3 (2.7, 3.8)	< .01	3.7 (3.1, 4.2)	< .01	7.1 (6.1, 8.0)	< .01	7.7 (6.8, 8.6)	< .01
Hispanic	4.0 (3.6, 4.3)	.17	3.7 (3.3, 4.1)	< .01	8.3 (7.8, 8.9)	.09	7.9 (7.3, 8.5)	< .01
Men (n = 6761)								
White (Ref)	10.1 (9.8, 10.5)		10.3 (10.1, 10.6)		17.1 (16.7, 17.6)		17.4 (17.0, 17.8)	
Black	11.6 (10.9, 12.3)	< .01	11.0 (10.3, 11.7)	.08	18.6 (17.8, 19.4)	< .01	17.7 (16.9, 18.5)	.53
American Indian	15.4 (10.8, 19.9)	.03	14.4 (10.1, 18.6)	.08	23.5 (18.4, 28.9)	.02	22.3 (17.6, 26.9)	.04
Asian/Pacific Islander	9.2 (7.8, 10.7)	.23	10.0 (8.6, 11.3)	.57	15.7 (13.7, 17.7)	.15	16.7 (14.9, 18.5)	.41
Hispanic	10.7 (9.8, 11.6)	.27	10.3 (9.4, 11.2)	.88	17.6 (16.5, 18.7)	.43	17.1 (15.9, 18.2)	.56

Note. CI = confidence interval; CVD = cardiovascular disease. The sample size was n = 14 333.
[a]Defined as coronary death, myocardial infarction, and fatal and nonfatal stroke.
[b]Defined as coronary death, myocardial infarction, coronary insufficiency, angina pectoris, stroke, transient ischemic attack, intermittent claudication, and congestive heart failure.
[c]Adjusted for age.
[d]Adjusted for age and socioeconomic status (socioeconomic status indicators include education, financial stress, income, and early life measures of parental education, parental ability to pay bills, and block group poverty).

shown to have a modest positive impact on the accuracy of risk perception and motivation to initiate risk reduction; however, evidence of the impact on absolute CVD risk is less conclusive.[33] Further research is needed to understand whether the use of longer-term CVD risk prediction influences risk communication, behavior change, and, ultimately, risk factor levels and other hard clinical endpoints.[31]

Racial/ethnic disparities in long-term CVD risk have not been explored since the 30-year FRS was developed with a predominantly White, middle-class population. We found that mean age-adjusted 30-year risk scores were higher among Blacks and American Indians than among Whites. However, racial/ethnic differences in predicted CVD risk were almost entirely eliminated after adjustment for SES with the exception of a higher risk of general CVD among American Indian men and lower risks of hard and general CVD among Asian/Pacific Islander and Hispanic women. Of the groups assessed, American Indian men and women had the highest CVD scores, were most likely to be at "high" general CVD risk (20% or higher), and often had the highest prevalence rates of the individual risk factors considered. These findings confirm the disparities in CVD risk found among older adult American Indian samples.[15,34] Although the small number of American Indians in our sample limited our power to detect statistically significant differences, we present the findings for this group because they highlight a health disparity that warrants further research.

Our findings underscore the importance of SES in Black–White disparities in CVD risk. Previous research has shown that SES adjustment eliminates or markedly reduces Black–White differences in CVD risk factors,[35] estimated 10-year CHD risk,[36] incident CHD,[37] and CVD mortality.[38] Adjustment for SES also highlighted the reduced risk among Hispanic women relative to White women. In light of the high proportion of immigrants among Asians and Hispanics, further work should examine 30-year CVD risk by nativity and acculturation given that these factors also affect CVD risk.[39–43]

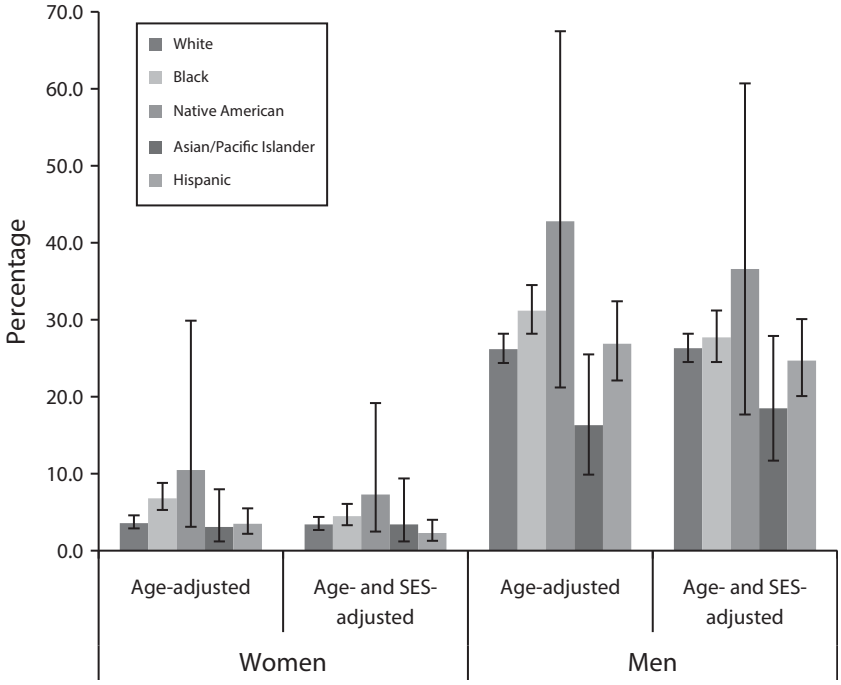
These racial/ethnic differences need to be validated in further research. Shorter-term Framingham risk functions perform well with White and Black adults,[44] but this may not be the case with the 30-year prediction function. One strength of the 30-year score is that it can account for competing causes of death, but this also may be a limitation because rates of competing causes of death refer to the White, middle-class population with which the prediction function was developed. Death rates are not similar across racial/ethnic[45–47] or socioeconomic groups.[45,46]

Furthermore, in one study a 10-year Framingham function underestimated risk among individuals of low SES by nearly 30%,[48] probably as a result of a higher underlying risk of death unaccounted for by the function. Therefore, the longer-term risk prediction function may perform more poorly in individuals with considerably different risks of death from non-CVD causes. Because Blacks and low-SES individuals are at higher risk for CVD,[15,49] it is essential that assessment tools perform well in these racial and socioeconomic groups. The most recent CVD assessment guidelines recommend the use of separate 10-year risk prediction functions for Whites and Blacks. However, the role of SES in the accuracy of these predictions was not mentioned. Given our finding that many disparities in predicted risk were eliminated when SES was controlled, further research is needed to ascertain whether SES-specific risk prediction functions might be more accurate in detecting risk and risk differences than racially specific tools.

Limitations

Our study must be interpreted within the confines of its limitations. BMI was used in the 30-year FRS values because lipid data were not available. In previous research in which the 10-year CVD risk equation was used, models relying on BMI performed well relative to the model requiring laboratory data.[50] Furthermore, the Asian/Pacific Islander and Hispanic categories included a wide range of groups with differing CVD risks.[43,51] Research involving more nuanced racial/ethnic categories is needed.

Also, residual confounding may have been present in our study owing to measurement error and the possibility that the SES indicators were not commensurate across racial/ethnic



Note. Socioeconomic status (SES) indicators include education, financial stress, income, and early life measures of parental education, parental ability to pay bills, and block group poverty. The sample size was n = 14 333.

FIGURE 1—Percentages of participants with a 20% or higher risk of general cardiovascular disease, by gender and race/ethnicity: National Longitudinal Study of Adolescent Health, United States, 2008–2009.

groups.[52] As noted, the Framingham risk scores were developed with a predominantly White population, and in previous research these scores have overestimated CHD risks in non-Black racial/ethnic minority groups.[44] Additional research in other cohorts is needed to validate our findings. Comparable estimates should be made with data from the National Health and Nutrition Examination Survey to ascertain whether methodological differences in study design affect estimated distributions of risk.

Conclusions

This study provides the first estimates, to our knowledge, of long-term CVD risk among young adults, and our results highlight disparities that will perpetuate an unacceptable status quo if left unaddressed. Our findings underscore the need for more population-based prevention efforts, which have been shown to be effective[30,53,54] and cost-effective[2] ways to prevent CVD. Our study identified a substantial number of men at high long-term risk for CVD. Although more research is needed to determine cut points for high long-term risk, it is clear that many young adults need intensive lifestyle or pharmacological treatments to prevent CVD. Better identification might start with more extensive use of the 30-year FRS in clinical practice with younger adults, as recommended by the most recent American Heart Association and American College of Cardiology risk assessment guidelines.[31] ■

About the Authors

Cari Jo Clark is with the Department of Medicine and the Division of Epidemiology and Community Health, School of Public Health, Program in Health Disparities Research, University of Minnesota, Minneapolis. Alvaro Alonso is with the Division of Epidemiology and Community Health, School of Public Health, University of Minnesota. Rachael A. Spencer is an independent researcher in Atlanta, GA. Michael Pencina is with the Clinical Research Unit and the Department of Biostatistics and Bioinformatics, Duke University School of Medicine, Durham, NC. Ken Williams is with KenAnCo Biostatistics, San Antonio, TX. Susan A. Everson-Rose is with the Department of Medicine and the Program in Health Disparities Research, University of Minnesota.

Correspondence should be sent to Cari Jo Clark, ScD, MPH, School of Public Health, University of Minnesota, 717 Delaware St SE, Suite 166, Minneapolis, MN 55414 (e-mail: cjclark@umn.edu). *Reprints can be ordered at http://www.ajph.org by clicking on the "Reprints" link.*

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Contributors

C. J. Clark, A. Alonso, M. Pencina, K. Williams, and S. A. Everson-Rose designed the study. C.J. Clark analyzed the data. A. Alonso, M. Pencina, and K. Williams supervised the data analysis. C.J. Clark and R. A. Spencer drafted the article. A. Alonso, R. A. Spencer, M. Pencina, K. Williams, and R. A. Everson-Rose critically reviewed the article. All of the authors contributed to interpretation of the findings.

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Human Participant Protection

The parent study was approved by the institutional review board of the University of North Carolina at Chapel Hill, and participants provided written informed consent. No protocol approval was needed for our study because deidentified data were used.

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