Coronary Heart Disease Risk Factors in College Students¹,²

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ABSTRACT
More than one-half of young adults aged 18–24 y have at least 1 coronary heart disease (CHD) risk factor and nearly one-quarter have advanced atherosclerotic lesions. The extent of atherosclerosis is directly correlated with the number of risk factors. Unhealthy dietary choices made by this age group contribute to weight gain and dyslipidemia. Risk factor profiles in young adulthood strongly predict long-term CHD risk. Early detection is critical to identify individuals at risk and to promote lifestyle changes before disease progression occurs. Despite the presence of risk factors and pathological changes, risk assessment and disease prevention efforts are lacking in this age group. Most young adults are not screened and are unaware of their risk. This review provides pathological evidence along with current risk factor prevalence data to demonstrate the need for early detection. Eighty percent of heart disease is preventable through diet and lifestyle, and young adults are ideal targets for prevention efforts because they are in the process of establishing lifestyle habits, which track forward into adulthood. This review aims to establish the need for increased screening, risk assessment, education, and management in young adults. These essential screening efforts should include the assessment of all CHD risk factors and lifestyle habits (diet, exercise, and smoking), blood pressure, glucose, and body mass index in addition to the traditional lipid panel for effective long-term risk reduction.

Introduction
Coronary heart disease (CHD)⁵ risk in young adults aged 18–24 y is underestimated despite the high prevalence of CHD risk factors (1–4) and early signs of atherosclerosis in this age group (5,6). Obesity has more than doubled in children and more than tripled in adolescents over the past 30 y (7). This weight gain tracks forward and worsens in young adulthood (8). Heart disease risk increases by 2–4% for each year a young adult is obese (9). As many as 33% of young adults are overweight (1), and this excess weight leads to dyslipidemia (10) and increases in metabolic syndrome (11), diabetes (12), and CHD (3) risk. CHD accounts for 50% of cardiovascular disease (CVD) deaths and is 1 of the leading causes of death in young adults (13). CHD costs the United States $108.9 billion each year in health care services, medications, and lost productivity (14), which is more than for any other disease. A death occurs from CVD every 40 s in the United States, which would wipe out a college campus of 25,000 in <12 d (15).

More than half of young adults have at least 1 CHD risk factor, and this greatly increases lifetime heart disease risk (16). Because many CHD risk factors surface in adolescence (13,17–19) and track forward to adulthood (20), the AHA's 2020 Strategic Impact Goals along with the National Heart, Lung, and Blood Institute’s (NHLBI's) 2012 Expert Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents (21) emphasize primordial prevention beginning in childhood and adolescence (16). This concept of primordial prevention was introduced by Strasser (22) in 1978 and focuses on preventing the development of risk factors themselves (16). Dietary modifications are central to this approach (16).

Despite screening recommendations for all adults aged >20 y (23,24), <50% of women and <40% of men of this age are screened for CHD risk (25). In addition, the majority of young adults are unaware of their risk (26). Until primordial prevention strategies are implemented to avoid risk...
factor development in the first place, there is a need for improved screening, risk assessment, management, and education in this age group. Early detection and intervention are critical because 80% of CVD events are preventable through diet and lifestyle (27). Diets low in saturated fat and high in fruits and vegetables reduce the risk of new cardiac events by 73% (28). Despite this evidence, young adults have high intakes of solid fats, added sugars (29), and sodium (1,30), along with inadequate intakes of fruits and vegetables (31), whole grains (32,33), and fiber (30). The AHA recently issued a scientific statement recommending reductions in added sugar intake in response to research linking sugar to excess energy intake, obesity, dyslipidemia, and CHD risk (34). Sugar consumption has increased by nearly 20% from 1970 to 2005, supplying almost 500 kcal/d (35). Adolescents consume more sugar than any other age group (549 kcal) (34), and this continues into young adulthood (29). Collectively, these poor diet- ary choices contribute to the high prevalence of CHD risk factors in this age group (36–39).

In 2011, Magnussen et al. (40) reviewed findings from 2 population-based studies in Finland that support the ability to avoid or delay premature atherosclerosis by prevention efforts early in life. In 2012, Rubin and Borden (41) reviewed atherosclerotic versus nonatherosclerotic causes of CHD in young adults. Although these 2 recent reviews examined the causes of CHD in young adults (40,41), there is a need for a review of pathological evidence along with recent risk factor and screening data to highlight the need for increased screening, risk assessment, education, and management in this age group.

The purpose of this review is to demonstrate the need for improved screening and risk awareness of CHD in young adults by revealing pathological changes that start in childhood and manifest themselves in young adult CHD risk factors. In addition, successful population-based prevention/treatment strategies used in other populations will be discussed with a focus on how these strategies can be applied to this age group.

Current Status of Knowledge

Progression of atherosclerosis

Childhood risk factors correlated with extent of lesions. Research indicates that atherosclerosis has roots in childhood. In the 1950s and 1960s, Holman et al. (42) and Strong and McGill (43,44) were the first to show that fatty streaks were present in the aortas of children as young as 3 y of age without a congenital heart condition, and progressed to fibrous plaques by the second decade of life. This evidence of atherosclerosis early in life led to large observational studies in the 1970s and 1980s (45–48) that examined childhood CVD risk factors, lifestyle patterns, and the development of CVD later in life.

The Muscatine, Bogalusa Heart, Cardiovascular Risk in Young Finns, and Childhood Determinants of Adult Health (CDAH) studies are the largest cohorts that tracked childhood risk factors into adulthood, with an average follow-up time of 30 y (49) (Table 1). The Muscastine Study (1970) indicated that risk factors predictive of CHD in adulthood, such as total cholesterol (TC), TGs, blood pressure (BP) and obesity, are prevalent in school-aged children (48). The Bogalusa Heart Study (1973) linked these childhood risk

<table>
<thead>
<tr>
<th>Authors (ref) and study name (country)</th>
<th>Baseline year</th>
<th>No. and age of participants</th>
<th>Years of follow-up</th>
<th>Key outcomes</th>
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<tr>
<td>Lauer et al. (48)</td>
<td>1970</td>
<td>11,337; 5–18 y</td>
<td>1970–1981</td>
<td>TC: 37% &gt;200 mg/dL; TGs: 15% &gt;140 mg/dL; BP: 21% ≥140/90 mm Hg; weight: 33% ≥110% relative weight; elevated TC, TGs, BP, and relative weight in youth predict CHD in adults</td>
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<tr>
<td>Berenson et al. (47)</td>
<td>1973</td>
<td>12,164; 4–17 y</td>
<td>1977–1996</td>
<td>Pathological changes occur by 5–8 y of age; extent of lesions significantly related to concentrations of TC, LDL-C, TGs, BMI, and HDL-C and BP</td>
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<td>Raitakari et al. (45)</td>
<td>1980</td>
<td>3596; 3–18 y</td>
<td>1983–ongoing</td>
<td>CVD risk factors (elevated TC, LDL-C, BP, smoking) early in life lead to structural and functional vascular changes related to atherosclerosis; increased LDL-C, BP, obesity and cigarette smoke in adolescence predict increased cIMT and decreased elasticity in adulthood</td>
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<td>Gall et al. (46)</td>
<td>1985</td>
<td>8498; 7–15 y</td>
<td>2004–2006</td>
<td>Childhood physical activity, obesity, and TC are important determinants of adult CVD risk factors (obesity, IR, dyslipidemia, cIMT)</td>
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1 BP, blood pressure; CHD, coronary heart disease; cIMT, carotid intima media thickness; CVD, cardiovascular disease; HDL-C, HDL cholesterol; IR, insulin resistance; LDL-C, LDL cholesterol; ref, reference; TC, total cholesterol.
factors with atherosclerosis in young adults. This autopsy study showed that the extent of atherosclerotic lesions was directly correlated to antemortem concentrations of TC, TGs, LDL cholesterol, and HDL cholesterol; BP; BMI; and cigarette smoking in young adults (47,50). The Cardiovascular Risk in Young Finns Study (1980) provided longitudinal data to show that CHD risk factors such as TC, HDL cholesterol, LDL cholesterol, TGs, BMI, and systolic blood pressure (SBP) track forward to adulthood (8,45). Associations between childhood risk factors and those measured 27 y later were strongest for TC and LDL cholesterol. In addition, dietary intake and patterns showed significant tracking over time because individuals in the highest quintiles of either a traditional Finnish dietary pattern or a health-conscious dietary pattern remained in the same quintile 21 y later (51). The CDAH study (1985) supported the findings from the previous cohort studies and further demonstrated that healthy lifestyle behaviors such as consuming a diet low in saturated fat and sodium and being physically active were associated with a better cardiovascular risk profile even in young adults (52). Each of these studies contributed to the understanding that early life factors influence the development of adult CVD (40).

Further evidence was provided by the Pathobiological Determinants of Atherosclerosis (PDAY) Study (1987), which examined the onset and progression of atherosclerosis in >3000 individuals aged 15–34 y in the United States (53). Although earlier autopsy studies (1970s and 1980s) indicated that risk factors for CHD were associated with atherosclerosis in adults, the PDAY and Bogalus Heart studies provided evidence for this in children and young adults (47,53). PDAY found intimal lesions in all aortas and in more than half of the right coronary arteries of adolescents aged 15–19 y (5). These lesions progress to more advanced, clinically significant lesions by young adulthood (53).

As many as 10–20% of young adults have advanced atherosclerotic lesions (54). This progression correlates with the number of CHD risk factors; young adults with ≥3 childhood risk factors had a 9-fold increase in atherosclerotic plaque area compared with those with no risk factors (6). As shown in Table 1, risk factors in childhood were shown to be strong predictors of preclinical atherosclerosis, even after adjustment for adult risk factors (55,56). These findings are critical from a prevention standpoint because those at risk of developing atherosclerosis can be identified and treated decades before clinical manifestation of disease.

**Childhood risk factors associated with preclinical disease markers.** Hyperlipidemia early in life is directly related to pathological changes and functional abnormalities and strongly predicts CHD in adulthood (57). The development of noninvasive techniques in the 1990s to measure preclinical markers such as carotid artery intima media thickness (cIMT), arterial endothelial function, and coronary artery calcification allowed for the assessment of structural and functional changes indicative of preclinical atherosclerosis (58,59). The Muscatine, Bogalusa Heart, Cardiovascular Disease Risk in Young Finns, and CDAH studies provided evidence that these preclinical markers are associated with risk factors in childhood. Preclinical markers are strongly associated with the risk of CVD events (58), but longer follow-up times are needed to directly link childhood risk factors with clinical events (40). In the absence of these data, these surrogate disease markers serve as intermediate endpoints to assess the effects of risk factors and risk factor interventions before the clinical manifestation of disease and provide a better understanding of the evolution of CVD across the life span (40,49).

In an attempt to address the difficulties in obtaining sufficient follow-up CVD events data, the International Childhood Cardiovascular Risk Consortium (i3C) was developed in 2011 to pool data previously collected from childhood to adulthood in large multicohort studies for meta-analysis to increase the power to link longitudinal risk data with CVD events. Data from the 4 largest cohort studies (Muscatine, Bogalusa Heart, Cardiovascular Disease Risk in Young Finns, and CDAH) and from similar smaller studies (Minneapolis Childhood Cohort Studies, Princeton Lipid Research Clinics Study; Princeton; National Heart, Lung, and Blood Institute Growth and Health Study) were combined for a total number of 12,000 participants with major CVD risk factors measured at least once in childhood and adulthood. In an effort to determine the effects of child and adult elevated BP on cIMT, data were pooled from the Bogalusa Heart, Muscatine, Young Finns, and CDAH studies with a mean follow-up of 23 y. Participants were 6–18 y old at baseline and 27–45 y old at follow-up. Results indicated that elevated BP that persisted from childhood into adulthood increased cIMT (60). In a similar analysis using the same 4 cohort studies (n = 4380 participants aged 3–18 y at baseline; mean follow-up = 22 y), the influence of age on the associations between childhood risk factors and cIMT in adulthood was examined (61). Risk factors (TC, TGs, BMI, and SBP) measured in the oldest children (15–18 y olds) at baseline were the strongest predictors of increased cIMT >20 y later. These findings demonstrate that late adolescence is the optimal age for screening and these screenings can effectively identify those at risk of atherosclerosis in adulthood (61).

Another recent meta-analysis (2013) in young adults from the i3C (Bogalusa Heart, Young Finns, and CDAH studies) and from the Minneapolis Childhood Cohort Studies and the Princeton Follow-Up Study assessed the association of ideal cardiovascular health with cIMT in 5785 participants aged 20–38 y (62). Ideal cardiovascular health is emphasized in the AHA’s 2020 Strategic Impact Goals and is defined as BP <120/80 mm Hg, glucose <100 mg/dL, TC <200 mg/dL, BMI <25 kg/m², physical activity >150 min/wk of moderate/vigorous activity or >75 min/wk vigorous activity, non-smoking, and meeting 4–5 components of a healthy diet score (16). Ideal cardiovascular health was achieved by only 1% of young adults. The least commonly met goal was diet-related; only 7% met the criteria for ideal diet. Compliance was particularly poor for sodium and saturated fat intakes. The number of ideal cardiovascular health criteria was inversely
associated with cIMT, demonstrating that these 7 health metrics are related to vascular health in young adults. The goal of future analyses from i3C data is to determine the independent effects of childhood and early adult number of CVD risk factors on subsequent CVD occurrence (49). This will involve collecting CVD morbidity and mortality follow-up data, examining gene variants that increase disease risk, and harmonizing noninvasive vascular measures to obtain a better understanding of causal pathways to CVD events (49).

Although diet was not the main outcome in any of the studies in the i3C, it was measured in all studies. Future research should involve a pooled analysis to better understand the role that dietary intake in childhood and adolescence has on present and future CVD risk. Because diet is considered the first line of defense, this research would guide the development of both population-based and individual prevention efforts.

**Poor dietary choices negatively affect CHD risk factors**

**Adolescents.** Unhealthy diet choices are a major determinant of CHD risk (34, 63, 64). Recent NHANES data in 4673 adolescents ages 12–19 y show an alarmingly high prevalence of adolescents in poor and intermediate CHD risk factor categories (65). Adherence to the 5 components of the healthy diet score was assessed: >4.5 cups (0.001 m³) of fruits and vegetables/d, >2 servings [3.5 oz (99.2 g)] of fish/wk, >3 servings [1 oz (28.4 g)] of fiber-rich whole grains (>1.1 g of fiber per 10 g of carbohydrate)/d, <1500 mg of sodium/d, and <450 kcal (1884.1 kJ) from sugar-sweetened beverages/wk. Healthy diet score was the least prevalent component of ideal cardiovascular health (65). Less than 1% met the criteria for an ideal healthy diet score and 90% had diets classified as poor. Adolescents consume as much as 34% of energy from solid fats and added sugars (66), exceeding recommendations by >200%. The consumption of excess calories from solid fats and added sugars is a major contributor to weight gain, which increases CHD risk in a dose-response manner (67). Although not the focus of this article, these data highlight the most prevalent dietary quality issues in this age group.

Dietary patterns established early in life carry into adulthood and are strongly associated with CHD risk (51). The transition from adolescence to young adulthood is considered a high-risk period because of declines in diet quality and increases in body weight (68–70). This transition period is often marked by students entering college, living away from home for the first time, and experiencing increased independence and responsibility for food choices (68, 71). If adolescents enter this transition period with poor diet quality, their chances of making positive dietary changes without intervention/education are slim.

**College students.** College students consume excessive calories from high-fat snack foods (cookies, cake, chips, and ice cream), frequently skip meals, avoid certain nutrient-dense foods (fruits, vegetables, and low-fat dairy), and practice unhealthy weight-loss techniques (72–74). These unhealthy dietary choices and eating behaviors contribute to the declines in diet quality observed during this period. College students’ diets exceed recommendations of total fat (46% vs. 35% of energy) and saturated fat (13% vs. 10% of energy) (30). Intakes of total (24% of energy) and added (17% of energy) sugar also surpass guidelines (<10% of energy) (29, 75). College students also fail to meet whole-grain recommendations (32, 33), consuming just over 10% (10.5 g) of the recommended 3 ounces (85.1 g) (33). Similarly, fiber intake is inadequate, with only 43% of females and 51% of males meeting recommendations (30). More than 90% of college students exceed sodium recommendations (1). Dietary patterns high in solid fats, added sugars, and sodium and low in whole grains and fiber are known to exacerbate CHD risk factors (37, 63).

Changes in the college dining environment may play an important role in the worsening of eating behaviors and dietary intake during the transition from adolescence to young adulthood (76). Most dining halls are “all you can eat” styles and allow unlimited meal frequency. The campus food environment is no longer restricted to dining halls; students now have access to a variety of on-campus restaurants, cafes, snack bars, convenience stores, and vending machines (77, 78). Although there are a greater variety of options both on and off campus, there are few healthful options (77, 79).

In 2012, Horacek et al. (78) assessed the on-campus and off-campus dining environment at 15 universities. Unhealthy dining environments were widespread. Fast-food restaurants had significantly greater portion sizes and were more likely to have “combo meal” pricing compared with snack bars/cafes, dining halls and other sit down, fast casual and student union dining venues. Signs to encourage unhealthy or overeating were most common at fast-food restaurants and at snack bars/cafes. Dining halls had significantly more healthy entrees, nonfried vegetables, no-sugar-added fruits, vegetarian options, whole-wheat bread, and low-fat milk compared with all other dining settings. Dining halls, however, had 1 of the biggest barriers to healthy eating: “all you can eat” pricing. This all-you-can-eat environment and the wide variety of foods available in dining halls leads to larger portion sizes, increased energy intake, and weight gain (80). In the first semester, college students gain weight up to 11 times faster compared with young adults not in college (72) and maintain this weight throughout college (81) and into adulthood. This additional weight, most of which is excess body fat, can lead to dyslipidemia and increased heart disease risk (10).

**Prevalence of CHD risk factors in college students**

CHD risk factors in young adulthood can be the result of pathological changes from childhood. Only 20% of CHD in young adults is related to nonatherosclerotic factors (41). Results from the few cross-sectional studies that have assessed CHD risk in college students aged 18–24 y show an alarmingly high prevalence of young adults with abnormal risk factor profiles (Table 2). Huang et al. (82) reported...
that the most prevalent risk factors in a sample of 163 college students were elevated TC (12%) and low HDL cholesterol (14%). Impaired glucose metabolism was also a concern because just over 6% had prediabetes. Overweight students had worse risk factor profiles (waist circumference, BP, TC, LDL cholesterol, VLDL cholesterol, TGs, leptin, and insulin) compared with normal-weight students and were nearly 3 times as likely to have at least 1 metabolic syndrome component.

Fernandes et al. (2) assessed the prevalence of metabolic syndrome criteria in 189 first-year college students and found that 18% had elevated TGs and 20% had low HDL cholesterol for gender. Metabolic syndrome risk was also high: 28% met at least 1 of the criteria for metabolic syndrome and 4% had metabolic syndrome. Obese students were more likely to meet ≥3 metabolic syndrome criteria and had a higher prevalence of abnormal HDL cholesterol, WC, and BP compared with participants with a BMI <30 kg/m². Gender differences were also noted, with males having a higher prevalence of risk factors (Table 2).

In a similar study by Huang et al. (4) that examined the prevalence of metabolic risk and gender differences in a sample of 300 students, 24% had low HDL cholesterol, 9% had elevated fasting glucose, and 9% had elevated TGs. The overall prevalence of metabolic syndrome was low (1%), but one-third of the sample had at least 1 component. As shown in Table 2, males had a worse metabolic profile than did females.

In a larger study conducted in 1701 college students, Burke et al. (1) reported that more than half had at least 1 CHD risk factor. The sample had high rates of overweight/obesity (33%) and elevated LDL cholesterol (53%), TC (27%), and BP (47%). Males also had a worse risk factor profile (BMI, glucose, TC, HDL cholesterol, LDL cholesterol, SBP, and diastolic blood pressure) than did females in this study. In a subsequent analysis of the same data but with a larger sample size (n = 2103), nearly one-third had low HDL cholesterol, nearly two-thirds had high BP, and approximately one-fourth had elevated TC or LDL cholesterol (3). Metabolic syndrome was observed in up to 10% of the sample, and those with a higher BMI had a substantially greater number of individual metabolic syndrome risk factors. In addition, males had a higher risk prevalence (BMI, HDL cholesterol, LDL cholesterol, TGs, and BP).

The differences in prevalence rates between studies can be partially attributed to demographic differences between universities. Risk factor profiles can be expected to vary because of different ethnic breakdowns and lifestyle factors across geographically dispersed university samples (2). There were also gender differences: a higher prevalence of CHD risk factors was found in men. Risk factor profiles were worse in overweight and obese individuals, regardless of gender. Collectively, these studies demonstrate that dyslipidemia and metabolic dysfunction are a common and major concern in young adults. As previously discussed, poor dietary choices made by this age group contribute to the high prevalence of risk factors. These data underscore the need to identify those at risk, especially male and overweight/obese young adults, so that steps can be taken to prevent future CHD risk and manage existing risk factors. Data collected to date demonstrate that college students are at risk of heart disease, but additional research needs to be conducted in young adults who are not in college to get a more comprehensive profile of this age group.

### CHD risk factor screening in young adults

**Historically conflicting guidelines.** Data from the cross-sectional studies mentioned above demonstrate that CHD risk factor prevalence is high in this age bracket, yet universal risk assessment for primordial and primary prevention is lacking. Although the importance and need for screening for early detection and management of dyslipidemia are recognized from public health organizations, including the NHLBI, AHA, American Academy of Pediatrics (AAP), and U.S. Preventive Services Task Force (USPSTF), the majority of young adults are not screened (25). The absence of apparent disease in young adults contributes to the underestimation of risk in this age group by both young adults themselves and by health professionals (26,83,84). This underestimation of risk and historically differing risk assessment guidelines contribute to this problem (85).

A variety of approaches and attitudes toward screening in young adults has existed among health professionals over
the past 2 decades (85,86). This can be traced back to the 1990s, with the release of the National Cholesterol Education Program (NCEP) Adult Treatment Panel III guidelines in 1993, which recommended universal lipid screening, regardless of risk level, every 5 y for all adults older than age 20 y. The rationale for these recommendations was to detect individuals at risk early on so that early intervention could reduce long-term CHD risk. Although these guidelines have been endorsed by representatives from >40 different medical and health organizations, the American College of Physicians argued against the need for screening in young adults due to the low short-term risk of CHD is this age group (87). Despite the presence of detractors early on, however, the strength of these screening recommendations was evidenced by their inclusion in the 2004 NCEP Adult Treatment Panel III guidelines (17) and in more recent 2012 NHLBI Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents (21) and 2013 American College of Cardiology (ACC)/AHA Guidelines on Assessment of CVD Risk (23).

Different recommendations over the past 20 y from other organizations have also led to inconsistent screening practices (85). The 2008 guidelines from the USPSTF recommend screening in all men older than age 35 and in men 20–35 y of age and in women older than age 45 who are at increased risk (88). The USPSTF makes no recommendation, however, for or against routine screening in men and women >20 y of age who are not at increased risk of CHD and states that the optimal screening interval is uncertain. Young adults in the 18–24-y age bracket span both children/adolescent and adult recommendations, which further complicates the issue. Screening guidelines for children and adolescents have also been conflicting since 1992 due to different recommendations by the NCEP (89), AHA (90), USPSTF (91), AAP (92), and the National Lipid Association (93). This conflicting guidance over the past 20 y has made it difficult for a uniform screening protocol to be followed by doctors and other health professionals (85).

Much needed progress was made, however, with the release of the 2012 NHLBI Expert Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents (21) and the ACC/AHA Guidelines on Assessment of CVD Risk in 2013 (23). The NHLBI’s comprehensive, evidence-based guidelines represent a change in approach from targeted screening to universal screening, with an emphasis on primordial and primary prevention. This change was supported by the inability of previous high-risk, targeted screening approaches to detect up to 60% of children and adolescents with hypercholesterolemia (94). The 2012 evidence-based recommendations for lipid assessment recommend universal lipid screening by using a non-fasting non–HDL-cholesterol concentration between ages 9–11 and 17–21 y. Targeted screening is recommended between 2–8 and 12–16 y of age if risk factors are present. These new lipid screening guidelines are endorsed by the AAP, but the new expanded screening guidelines have not been without their detractors (85,95–97). There are concerns that the new guidelines may result in overdiagnosis, false-positives, and overuse of statins in children (95–97). Although some experts disagree with the conservative nature of the guidelines, they are a pivotal step in the shift toward primordial, population-based prevention strategies that are needed to reduce future risk (16,23,65,98,99).

More recent 2013 ACC/AHA CVD assessment guidelines also support the need for risk assessment early in life to motivate lifestyle changes in younger individuals who may be at low short-term risk but who could benefit from long-term risk assessment. Long-term risk assessment of traditional CVD risk factors is recommended every 4–6 y beginning at age 20 for those who are free from atherosclerotic CVD (23).

**Inadequate screening in young adults.** NHANES data from 1999–2006 on 2587 young adults aged 20–45 y indicated that two-thirds have at least 1 CVD risk factor. This is alarming because <50% of women and <40% of men reported being screened before the assessment visit. The screening rate for young adults in the 18–24-y age bracket can be expected to be even lower because screening rates increase with age (100). Younger males, in particular, are >50% less likely than their female counterparts to obtain preventive services (101). Data from NHANES show that women are more likely to have health insurance and see a health care provider (25). These low screening rates are especially concerning among young adults with multiple risk factors because the extent of atherosclerosis is directly correlated with the number of risk factors.

The AHA supports population-based strategies such as screenings at universities to identify at-risk individuals (16,98,102). Policy changes are needed to promote increased screening in primary care settings, clinics, schools, worksites, and community sites. These screenings are particularly important in the young-adult age group who may go otherwise undetected by the health care system (103), partly due to the underestimation of risk (26,83,84). As discussed in the AHA’s 2013 Science Advisory, screenings should include the assessment of all CHD risk factors including lifestyle habits (diet, exercise, and smoking), BP, glucose, and BMI in addition to the traditional lipid panel (98). Screening, however, must be accompanied by reliable interpretation of results, provision of appropriate educational material, and referral to a physician for those who need it for follow-up to be most effective. Young adults should be informed of the meaning of their results, the importance of dietary changes, and the appropriate follow-up steps that need to be taken depending on their other risk factors (103) (Fig. 1). As outlined in the 2013 AHA/ACC Guidelines on Lifestyle Management to Reduce Cardiovascular Risk and in the 2013 ACC/AHA Guidelines on Assessment of Cardiovascular Risk, heart healthy nutrition and physical activity behaviors are recommended for all adults older than age 18 for both prevention and treatment (23,104). These preventive efforts are essential for reducing CHD events later in life and for reducing the burden of CHD on a population level (98).
Future research is needed to better understand and eliminate barriers to screening. This needs to be done at the policy, provider, and patient level to improve suboptimal screening in young adults (105).

**Population-based nutrition interventions in college students**

Until primordial prevention strategies are successful in avoiding risk factor development altogether, risk factor screening needs to work in tandem with education and management for effective disease prevention. Strategies that focus on high-risk individuals are effective in reducing CHD events, but population-level strategies are needed to produce wide-scale risk reductions (16,98). Population-based interventions on college campuses are cost-effective strategies to manage existing risk factors by promoting lifestyle changes, which are the foundation for risk reduction efforts (104). The college setting is an ideal forum to reach large numbers of the young adult population because 12.5 million (nearly 50%) of those aged 18–24 y were enrolled in U.S. colleges and universities in 2010 (106). Interventions aimed at the college population represent an opportunity to promote healthy eating while lifestyle habits are still being formed and to target CHD risk factors before disease progression occurs.

Previous population-based strategies have proven to be successful in reducing CHD risk in other populations (16). In the late 1980s, a population-based approach was used to lower CHD risk in the island nation of Mauritius. The FA composition of imported cooking oil was changed to contain higher amounts of polyunsaturated fat instead of saturated fat. The mean TC concentration decreased from 225 mg/dL in 1987 to 182 mg/dL in 1992, decreasing the prevalence of hypercholesterolemia from 25% to 6% in men and from 22% to 5% in women (107,108). This intervention was a classic example of a population-based strategy that effectively shifted the entire distribution of risk. Estimates from the World Heart Federation show that a universal reduction in sodium intake by 1 g/d would lead to a 50% reduction in the number of individuals needing treatment for hypertension, a 22% decrease in deaths from stroke, and a 16% decrease in deaths from CHD (28).

Similar population-based strategies can be applied to the college setting. Although cafeterias can contribute to an obesogenic environment on college campuses, they also represent an opportunity to influence students’ diets for the better by providing nutrition information to guide healthy choices (109). To motivate students to choose healthier options, colleges need to identify healthy choices, provide nutrition information, and use point-of-selection signage (78). This nutrition information may provide the stimulus for students to reevaluate and change their eating habits (110). Pyramids that displayed energy and nutrient content of menu offerings at a university cafeteria led 71% of patrons to change their lunch selections by choosing meals lower in energy and fat (111).

Peterson et al. (112) reported increased awareness of healthy foods as the primary reason for selecting healthier food choices in a dining hall intervention consisting of signs, table tents, flyers, and benefit-based messages. Similar studies have also found that point-of-selection nutrition labels in dining halls resulted in better food choices and decreased energy intake at meals (113,114). In another study, students with the highest nutrition knowledge were 12 times more likely to meet dietary recommendations compared with those with the lowest knowledge (115). Drawing attention to nutrition and health in a campus dining hall setting has a positive affect on food choices (112). Relatively small changes in the physical environment can produce behavioral...
changes (116). For example, placing healthy foods in more prominent places and removing trays from dining halls are other inexpensive ways to prompt healthier dietary choices.

Recently, technology has been used to promote behavior change. Technology-based interventions are particularly appealing to the young-adult population and are quick, cost-effective, and convenient ways to transmit information to a large audience (117). For example, messages displayed on computer screens at “point of decision” spots in a college dining hall influenced students to increase their fruit intake (118). Poddar et al. (119) demonstrated that 8 wk of e-mail messages as part of a dairy intake intervention were effective in increasing dairy intake in college students relative to the comparison group. Greene et al. (31) found that a 10-lesson Web-based nutrition and physical activity intervention resulted in higher fruit and vegetable intake and greater physical activity in 1689 college students from 8 universities.

Other studies have also reported success with mobile technology-based interventions (120–124). Text messaging, in particular, has been used in a variety of behavioral intervention studies to provide reminders, cues, and positive reinforcement and enhance self-monitoring (125–128). All of these features are recognized as keys to successful maintenance of dietary changes (124). Text messaging is an especially appealing intervention mode for college students because 99.8% of college students own a cell phone and 97% of college students rely on text messaging as their main form of communication (129).

In conclusion, this review highlights the need for improved risk assessment and increased awareness in young adults. Cross-sectional studies provide evidence of the high prevalence of CHD risk factors in this age group. It is well established that these risk factors are associated with pathological changes and substantially increase lifetime CHD risk. Until successful primordial prevention strategies are part of the public health care infrastructure and prevent risk factors, the focus must be on improving education about and screening, assessment, and treatment of CHD risk factors. Targeting young adults at a time in their lives when lifelong habits are being developed is critical to prevent disease progression.

The low screening rates in this age group are concerning in light of the high prevalence of risk factors. Increased screening is the first step because young adults at risk must first be identified before treatment approaches can be initiated. College campuses provide an opportunity for population-based screening approaches. College students and health professionals on campus must first be made aware of the need for risk assessment and then risk reduction through lifestyle changes.

Future research needs to be done to identify the most effective and efficient ways of screening large numbers of young adults. Screenings embedded into course curricula in health courses, as part of university wellness programs or as a part of freshmen orientation, are potential avenues to increase screening rates in this age group. Increased screening needs to work in conjunction with education to effectively identify and manage CHD risk.

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Literature Cited
79. Levitsky DA, Youn T. The more food young adults are served, the more they overeat. J Nutr. 2004;134:2546–9.
80. Holm-Denoma JM, Joiner TE, Vohs KD, Heatherton TF. The “freshman fifteen” (the “freshman five” actually); predictors and possible explanations. Health Psychol. 2008;27(1 Suppl):S3–9.