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## Coronary Heart Disease Risk Factors in College Students

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16 **Abbreviations:** CHD, coronary heart disease; CVD, cardiovascular disease; AHA, American  
17 Heart Association; NHLBI, National Heart, Lung, and Blood Institute; CDAH, Childhood  
18 Determinants of Adult Health; TC, total cholesterol; BP, blood pressure; LDL-C, LDL  
19 cholesterol; HDL-C, HDL cholesterol; SBP, systolic blood pressure; PDAY, Pathobiological  
20 Determinants of Atherosclerosis; cIMT, carotid artery intima media thickness; i3C, International  
21 Childhood Cardiovascular Risk Consortium; NGHS, National Heart, Lung, and Blood Institute  
22 Growth and Health Study; NHANES, National Health and Nutrition Examination Survey; WC,  
23 waist circumference; VLDL-C, VLDL cholesterol; DBP, diastolic blood pressure; American  
24 Academy of Pediatrics, AAP; US Preventive Services Task Force, USPSTF; National  
25 Cholesterol Education Program Adult Treatment Program III, NCEP ATP III; American College  
26 of Cardiology, ACC

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32 **Abstract:**

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

49 Abbreviations: CHD, coronary heart disease; CVD, cardiovascular disease; AHA, American  
50 Heart Association; NHLBI, National Heart, Lung, and Blood Institute; CDAH, Childhood  
51 Determinants of Adult Health; TC, total cholesterol; BP, blood pressure; LDL-C, LDL  
52 cholesterol; HDL-C, HDL cholesterol; SBP, systolic blood pressure; PDAY, Pathobiological  
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57 Academy of Pediatrics, AAP; US Preventive Services Task Force, USPSTF; National  
58 Cholesterol Education Program Adult Treatment Program III, NCEP ATP III; American College  
59 of Cardiology, ACC

60 **Introduction:**

61 Coronary heart disease (CHD) risk in young adults, ages 18-24, is underestimated despite  
62 the high prevalence of CHD risk factors (1-4) and early signs of atherosclerosis in this age group  
63 (5, 6). Obesity has more than doubled in children and more than tripled in adolescents over the  
64 past 30 years (7). This weight gain tracks forward and worsens in young adulthood (8). Heart  
65 disease risk increases by 2-4% for each year a young adult is obese (9). As many as 33% of  
66 young adults are overweight (1) and this excess weight leads to dyslipidemia (10) and increases  
67 in metabolic syndrome (11), diabetes (12) and CHD (3) risk. Coronary heart disease accounts for  
68 50% of cardiovascular disease (CVD) deaths and is one of the leading causes of death in young  
69 adults (13). Coronary heart disease costs the US \$108.9 billion each year in health care services,  
70 medications and lost productivity (14), which is more than any other disease. A death occurs  
71 from CVD every 40 seconds in the US, which would wipe out a college campus of 25,000 in less  
72 than 12 days (15).

73 More than half of young adults have at least one CHD risk factor and this causes a spike  
74 in lifetime heart disease risk (16). Since many CHD risk factors surface in adolescence (13, 17-  
75 19) and track forward to adulthood (20), the American Heart Association's (AHA) 2020  
76 Strategic Impact Goals along with the National Heart, Lung and Blood Institute's (NHLBI) 2012  
77 Expert Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children

78 and Adolescents (21) emphasize primordial prevention beginning in childhood and adolescence  
79 (16). This concept of primordial prevention was introduced by Strasser in 1978 (22) and focuses  
80 on preventing the development of risk factors themselves (16). Dietary modifications are central  
81 to this approach (16).

82         Despite screening recommendations for all adults over age 20 (23, 24), less than 50% of  
83 women and 40% of men of this age are screened for CHD risk (25). In addition, the majority of  
84 young adults are unaware of their risk (26). Until primordial prevention strategies are  
85 implemented to avoid risk factor development in the first place, there is a need for improved  
86 screening, risk assessment, management and education in this age group. Early detection and  
87 intervention are critical since 80% of CVD events are preventable through diet and lifestyle (27).  
88 Diets low in saturated fat and high in fruits and vegetables reduce the risk of new cardiac events  
89 by 73% (28). Despite this evidence, young adults have high intakes of solid fats, added sugars  
90 (29) and sodium (1, 30), along with inadequate intakes of fruits and vegetables (31), whole  
91 grains (32, 33) and fiber (30). The AHA recently issued a scientific statement recommending  
92 reductions in added sugar intake in response to research linking sugar to excess energy intake,  
93 obesity, dyslipidemia and CHD risk (34). Sugar consumption has increased by nearly 20% from  
94 1970 to 2005, supplying almost 500 kcal/day (35). Adolescents consume more sugar than any  
95 other age group (549 kcals) (34) and this continues into young adulthood (29). Collectively,  
96 these poor dietary choices contribute to the high prevalence of CHD risk factors in this age group  
97 (36-39).

98         In 2011, Magnussen et al. (40) reviewed findings from two population based studies in  
99 Finland that support the ability to avoid or delay premature atherosclerosis by prevention efforts  
100 early in life. In 2012, Rubin et al. (41) reviewed atherosclerotic versus non-atherosclerotic

101 causes of CHD in young adults. Although these two recent reviews have examined the causes of  
102 CHD in young adults (40, 41), there is a need for a review of pathological evidence along with  
103 recent risk factor and screening data to highlight the need for increased screening, risk  
104 assessment, education and management in this age group.

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

## 110 **Current Status of Knowledge:**

### 111 **Progression of Atherosclerosis**

#### 112 *Childhood Risk Factors Correlated with Extent of Lesions*

113         Research indicates that atherosclerosis has childhood roots. In the 1950's and 60's  
114 Holman et al., McGill et al. and Strong et al. (42-44) were the first to show that fatty streaks  
115 were present in the aortas of children as young as 3 years of age, without a congenital heart  
116 condition, and progressed to fibrous plaques by the second decade of life. This evidence of  
117 atherosclerosis early in life led to large, observational studies in the 1970s and 1980s (45-48) to  
118 examine childhood CVD risk factors, lifestyle patterns and the development of CVD later in life.

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

124 (48). The Bogalusa Heart Study (1973) linked these childhood risk factors with atherosclerosis in  
125 young adults. This autopsy study showed that the extent of atherosclerotic lesions was directly  
126 correlated to antemortem levels of TC, TG, LDL cholesterol (LDL-C), HDL cholesterol (HDL-  
127 C), BP, BMI and cigarette smoking in young adults (47, 50). The Cardiovascular Risk in Young  
128 Finns Study (1980) provided longitudinal data to show that CHD risk factors such as TC, HDL-  
129 C, LDL-C, TG, BMI, and systolic blood pressure (SBP) track forward to adulthood (8, 45).  
130 Associations between childhood risk factors and those measured 27 years later were strongest for  
131 TC and LDL-C. In addition, dietary intake and patterns showed significant tracking over time as  
132 individuals in the highest quintiles of either a traditional Finnish dietary pattern or a health-  
133 conscious dietary pattern remained in the same quintile twenty-one years later (51). The CDAH  
134 study (1985) supported the findings from the previous cohort studies and further demonstrated  
135 that healthy lifestyle behaviors such as consuming a diet low in saturated fat and sodium and  
136 being physically active were associated with a better cardiovascular risk profile even in young  
137 adults (52). Each of these studies contributed to the understanding that early life factors influence  
138 the development of adult CVD (40).

139 Further evidence was provided by the Pathobiological Determinants of Atherosclerosis  
140 (PDAY) study (1987), which examined the onset and progression of atherosclerosis in over  
141 3,000 subjects in the US ages 15-34 years (53). Although earlier autopsy studies (1970's and  
142 80's) indicated that risk factors for CHD were associated with atherosclerosis in adults, PDAY  
143 and Bogalusa provided evidence for this in children and young adults (47, 53). PDAY found  
144 intimal lesions in all aortas and more than half of the right coronary arteries of adolescents ages  
145 15-19 years (5). These lesions progress to more advanced, clinically significant lesions by young  
146 adulthood (53).

147 As many as 10-20% of young adults have advanced atherosclerotic lesions (54). This  
148 progression is correlated with the number of CHD risk factors; young adults with  $\geq 3$  childhood  
149 risk factors had a 9-fold increase in atherosclerotic plaque area compared to those with none (6).  
150 As shown in **Table 1**, risk factors in childhood were shown to be strong predictors of preclinical  
151 atherosclerosis even after adjustment for adult risk factors (55, 56). These findings are critical  
152 from a prevention standpoint as those at risk of developing atherosclerosis can be identified and  
153 treated decades before clinical manifestation of disease.

#### 154 *Childhood Risk Factors Associated with Preclinical Disease Markers*

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

167 In an attempt to address the difficulties in obtaining sufficient follow-up CVD events  
168 data, the International Childhood Cardiovascular Risk Consortium (i3C) was developed in 2011



169 to pool data previously collected from childhood to adulthood in large, multi-country cohort  
170 studies for a meta-analysis to increase the power to link longitudinal risk data with CVD events.  
171 Data from the four largest cohort studies (Muscatine, Bogalusa, Cardiovascular Disease Risk in  
172 Young Finns, and CDAH) and from similar smaller studies (Minneapolis, Princeton, National  
173 Heart, Lung, and Blood Institute Growth and Health Study (NGHS) were combined for a total  
174 number of 12,000 participants with major CVD risk factors measured at least once in childhood  
175 and adulthood. In an effort to determine the effects of child and adult elevated BP on cIMT, data  
176 was pooled from the Bogalusa, Muscatine, Young Finns and CDAH with a mean follow-up of 23  
177 years. Participants were 6-18 years old at baseline and 27-45 years old at follow-up. Results  
178 indicated that elevated blood pressure that persisted from childhood into adulthood increased  
179 cIMT (60). In a similar analysis using the same four cohort studies (n=4,380 ages 3-18 years at  
180 baseline, mean follow-up=22 years), the influence of age on the associations between childhood  
181 risk factors and cIMT in adulthood was examined (61). Risk factors (TC, TG, BMI, SBP)  
182 measured in the oldest children (15-18 year olds) at baseline were the strongest predictors of  
183 increased cIMT more than 20 years later. These findings demonstrate that late adolescence is the  
184 optimal age for screening and these screenings can effectively identify those at risk of  
185 atherosclerosis in adulthood (61).

186 Another recent meta-analysis (2013) on young adults from the i3C consortium (Bogalusa,  
187 Young Finns, CDAH studies) and from the Minneapolis Childhood Cohort Studies and the  
188 Princeton Follow-Up Study assessed the association of ideal cardiovascular health with cIMT  
189 (62) in 5,785 participants ages 20-38 years (62). Ideal cardiovascular health is emphasized in the  
190 AHA's 2020 Strategic Impact Goals and is defined as blood pressure <120/80 mmHg, glucose  
191 <100 mg/dL, TC <200 mg/dL, BMI <25 kg/m<sup>2</sup>, physical activity >150 min/wk

192 moderate/vigorous or >75 min/wk vigorous, nonsmoking and 4-5 components of a healthy diet  
193 score (16). Ideal cardiovascular health was achieved by only 1% of young adults. The least  
194 commonly met goal was diet-related; only 7% met the criteria for ideal diet. Compliance was  
195 particularly poor for sodium intake and saturated fat intake. The number of ideal cardiovascular  
196 health criteria was inversely associated with cIMT, demonstrating that these 7 health metrics are  
197 related to vascular health in young adults. The goal of future analyses from i3C data is to  
198 determine the independent effects of childhood and early adult levels of CVD risk factors on  
199 subsequent CVD occurrence (49). This will involve collecting CVD morbidity and mortality  
200 follow-up data, examining gene variants that increase disease risk and harmonizing non-invasive  
201 vascular measures to obtain a better understanding of causal pathways to CVD events (49).

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

## 207 **Poor Dietary Choices Negatively Impact CHD Risk Factors**

### 208 *Adolescents*

209         Unhealthy diet choices are a major determinant of CHD risk (34, 63, 64). Recent data  
210 from the National Health and Nutrition Examination Survey (NHANES) show an alarmingly  
211 high prevalence of poor and intermediate CHD risk factors in a nationally representative sample  
212 of 4673 adolescents ages 12-19 years (65). Adherence to the five components of the healthy diet  
213 score was assessed: >4.5 cups (0.001 m<sup>3</sup>) of fruits and vegetables per day, > two 3.5 oz (99.2 g)

214 servings of fish per week, > three 1 oz (28.4 g) servings of fiber-rich whole grains (>1.1 g of  
215 fiber per 10 g of carbohydrate) per day, <1500 mg of sodium per day and <450 kcals (1884.1 kJ)  
216 from sugar-sweetened beverages per week. Healthy diet score was the least prevalent component  
217 of ideal cardiovascular health (65). Less than 1% met the criteria for an ideal healthy diet score  
218 and 90% had diets classified as poor. Adolescents consume as much as 34% of energy intake  
219 from solid fats and added sugars (66), exceeding recommendations by over 200%. Consumption  
220 of excess calories from solid fats and added sugars is a major contributor to weight gain, which  
221 increases CHD risk in a dose-response manner (67). Although not the focus of this paper, this  
222 data highlights the most prevalent dietary quality issues in this age group.

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

### 230 *College Students*

231         College students consume excessive calories from high-fat snack foods (cookies, cake,  
232 chips, ice cream), frequently skip meals, avoid certain nutrient-dense foods (fruits, vegetables,  
233 low-fat dairy) and practice unhealthy weight-loss techniques (72-74). These unhealthy dietary  
234 choices and eating behaviors contribute to the declines in diet quality observed during this  
235 period. College students' diets exceed recommendations of total fat (46% versus 35% of energy)  
236 and saturated fat (13% versus 10% of energy) (30). Total sugar (24% of energy) and added sugar

237 (17% of energy) intake also surpass guidelines (<10% of energy) (29, 75). College students also  
238 fail to meet whole grain recommendations (32, 33), consuming just over 10% (10.5 g) of the  
239 recommended 3 oz (85.1 g) (33). Similarly, fiber intake is inadequate with only 43% of females  
240 and 51% of males meeting recommendations (30). Over 90% of college students exceed sodium  
241 recommendations (1). Dietary patterns high in solid fats, added sugars and sodium and low in  
242 whole grains and fiber are known to exacerbate CHD risk factors (37, 63).

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

250         In 2012, Horacek et al. (78) assessed the on-campus and off-campus dining environment  
251 at fifteen universities. Unhealthy dining environments were widespread. Fast-food restaurants  
252 had significantly greater portion sizes and were more likely to have “combo meal” pricing  
253 compared to snack bars/cafes, student unions, and sit down, fast casual and dining halls. Signs to  
254 encourage unhealthy or overeating were most common at fast-food restaurants and at snack  
255 bars/cafes. Dining halls had significantly more healthy entrees, non-fried vegetables, no-sugar  
256 added fruit, vegetarian options, whole wheat bread and low-fat milk compared to all other dining  
257 settings. Dining halls, however, had one of biggest barriers to healthy eating: “all-you-can eat”  
258 pricing. This “all-you-can-eat” environment and the wide variety of foods available in dining  
259 halls leads to larger portion sizes, increased energy intake and weight gain (80). In the first

260 semester, college students gain weight up to 11 times faster compared to young adults not in  
261 college (72) and maintain this weight throughout college (81) and into adulthood. This  
262 additional weight, most of which is excess body fat, can lead to dyslipidemia and increased heart  
263 disease risk (10).

#### 264 **Prevalence of CHD Risk Factors in College Students**

265         Coronary heart disease risk factors in young adulthood can be the result of pathological  
266 changes from childhood. Only 20% of CHD in young adults is related to non-atherosclerotic  
267 factors (41). Results from the few cross-sectional studies that have assessed CHD risk in college  
268 students, ages 18-24 years show an alarmingly high prevalence of young adults with abnormal  
269 risk factor profiles (**Table 2**). Huang et al. (82) reported that the most prevalent risk factors in a  
270 sample of 163 college students were elevated TC (12%) and low HDL-C (14%). Impaired  
271 glucose metabolism was also a concern as just over 6% had pre-diabetes. Overweight students  
272 had worse risk factor profiles (waist circumference (WC), BP, TC, LDL-C, VLDL cholesterol  
273 (VLDL-C), TG, leptin, insulin) compared to normal weight students and were nearly 3 times  
274 more likely to have at least one metabolic syndrome component.

275         Fernandes et al. (2) assessed the prevalence of metabolic syndrome criteria in 189 first  
276 year college students and found that 18% had elevated TG and 20% had low HDL-C for gender.  
277 Metabolic syndrome risk was also high; 28% met at least one of the criteria for metabolic  
278 syndrome and 4% had metabolic syndrome. Obese students were more likely to meet 3 or more  
279 metabolic syndrome criteria and had a higher prevalence of abnormal HDL-C, WC and BP  
280 compared to subjects with a BMI<30 kg/m<sup>2</sup>. Gender differences were also noted, with males  
281 having a higher prevalence of risk factors (**Table 2**).

282 In a similar study by Huang et al. (4) that examined prevalence of metabolic risk and  
283 gender differences in a sample of 300 students, 24% had low HDL-C, 9% had elevated fasting  
284 glucose and 9% had elevated TG. Overall prevalence of metabolic syndrome was low (1%) but  
285 1/3 of the sample had at least one component. As shown in **Table 2**, males had a worse  
286 metabolic profile than females.

287 In a larger study performed on 1,701 college students, Burke et al. (1) reported that more  
288 than half had at least one CHD risk factor. The sample had high rates of overweight/obesity  
289 (33%) and elevated LDL-C (53%), TC (27%) and BP (47%). Males also had a worse risk factor  
290 profile (BMI, glucose, TC, HDL-C, LDL-C, SBP and diastolic blood pressure (DBP)) than  
291 females in this study. In a subsequent analysis of the same data but with a larger sample size,  
292 (n=2,103) nearly 1/3 had low HDL-C, nearly 2/3 had high BP and approximately 1/4 had  
293 elevated TC or LDL-C (3). Metabolic syndrome was observed in up to 10% of the sample and  
294 those with a higher BMI had a significantly greater number of individual metabolic syndrome  
295 risk factors. In addition, males had higher risk prevalence (BMI, HDL-C, LDL-C, TG, BP).

296 The differences in prevalence rates across studies can be partially attributed to  
297 demographic differences between universities. Risk factor profiles can be expected to vary due to  
298 different ethnic breakdowns and lifestyle factors across geographically dispersed university  
299 samples (2). There were also gender differences; a higher prevalence of CHD risk factors was  
300 found in men. Risk factor profiles were worse in overweight and obese individuals, regardless of  
301 gender. Collectively, these studies demonstrate that dyslipidemia and metabolic dysfunction are  
302 a common and major concern in young adults. As previously discussed, poor dietary choices  
303 made by this age group contribute to the high prevalence of risk factors. These data underscore  
304 the need to identify those at risk, especially male and overweight/obese young adults, so that

305 steps can be taken to prevent future CHD risk and manage existing risk factors. Data collected  
306 to-date demonstrates that college students are at risk for heart disease but additional research  
307 needs to be done on young adults not in college to get a more comprehensive profile of this age  
308 group.

## 309 **CHD Risk Factor Screening in Young Adults**

### 310 *Historically Conflicting Guidelines*

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

320 A variety of approaches and attitudes toward screening in young adults has existed  
321 among health professionals over the past two decades (85, 86). This can be traced back to the  
322 1990's, with the release of the National Cholesterol Education Program Adult Treatment Panel  
323 III (NCEP ATP II) guidelines in 1993 that recommended universal lipid screening, regardless of  
324 risk level, every 5 years for all adults over age 20 years. The rationale for these  
325 recommendations was to detect individuals at risk early on so that early intervention could  
326 reduce long-term CHD risk. Although these guidelines have been endorsed by representatives

327 from over 40 different medical and health organizations, the American College of Physicians  
328 argued against the need for screening in young adults due to the low short-term risk for CHD in  
329 this age group (87). Despite the presence of detractors early on, however, the strength of these  
330 screening recommendations was evidenced by their inclusion in 2004 NCEP ATP III Guidelines  
331 (17) and in more recent 2012 NHLBI Guidelines for Cardiovascular Health and Risk Reduction  
332 in Children and Adolescents (21) and 2013 American College of Cardiology (ACC)/AHA  
333 Guidelines on Assessment of CVD Risk (23).

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

345 Much needed progress was made, however, with the release of the 2012 NHLBI Expert  
346 Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and  
347 Adolescents (21) and the ACC/AHA Guidelines on Assessment of CVD Risk in 2013 (23). The  
348 NHLBI's comprehensive, evidence-based guidelines represent a change in approach from  
349 targeted screening to universal screening with an emphasis on primordial and primary



350 prevention. This change was supported by the inability of previous high-risk, targeted screening  
351 approaches to detect up to 60% of children and adolescents with hypercholesterolemia (94). The  
352 2012 evidence-based recommendations for lipid assessment recommend universal lipid  
353 screening by a non-fasting non-HDL-C level between ages 9-11 and 17-21 years of age.  
354 Targeted screening is recommended between 2-8 and 12-16 years of age if risk factors are  
355 present. These new lipid screening guidelines are endorsed by the AAP but the new expanded  
356 screening guidelines have not been without their detractors (85, 95-97). There are concerns that  
357 the new guidelines may result in over diagnosis, false-positives, and overuse of statins in  
358 children (95-97). Although some experts disagree with the conservative nature of the guidelines,  
359 they are a pivotal step in the shift toward primordial, population-based prevention strategies that  
360 are needed to reduce future risk (16, 23, 65, 98, 99).

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

#### 366 *Inadequate Screening in Young Adults*

367 National Health and Nutrition Examination Survey data from 1999-2006 on 2587 young  
368 adults ages 20-45 years, indicated that 2/3 have at least one CVD risk factor. This is alarming  
369 since less than 50% of females and less than 40% of males reported being screened prior to the  
370 assessment visit. The screening rate for young adults in the 18-24 year age bracket can be  
371 expected to be even lower as screening rates increase with age (100). Younger males, in

372 particular, are more than 50% less likely than their female counterparts to obtain preventive  
373 services (101). Data from NHANES show that women are more likely to have health insurance  
374 and see a healthcare provider (25). These low screening rates are especially concerning among  
375 young adults with multiple risk factors as the extent of atherosclerosis is directly correlated with  
376 the number of risk factors.

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

394 is needed to better understand and eliminate barriers to screening. This needs to be done at the  
395 policy, provider and patient level to improve suboptimal screening in young adults (105).

### 396 **Population-Based Nutrition Interventions in College Students**

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

408       Previous population-based strategies have proven to be successful in reducing CHD risk  
409 in other populations (16). In the late 1980's, a population-based approach was used to lower  
410 CHD risk in the island nation of Mauritius. The fatty acid composition of imported cooking oil  
411 was changed to contain higher levels of polyunsaturated fat instead of saturated fat. The mean  
412 TC concentration fell from 225 mg/dL in 1987 to 182 mg/dL in 1992, decreasing the prevalence  
413 of hypercholesterolemia from 25% to 6% in men and from 22% to 5% in women (107, 108).  
414 This intervention was a classic example of a population-based strategy that effectively shifted the  
415 entire distribution of risk. Estimates from the World Heart Federation show that a universal  
416 reduction in sodium intake by 1 gram/day would lead to a 50% reduction in the number of

417 individuals needing treatment for hypertension, a 22% decrease in deaths from stroke and a 16%  
418 drop in deaths from CHD (28).

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

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

438           Recently, technology has been used to promote behavior change. Technology-based  
439 interventions are particularly appealing to the young adult population and are quick, cost-

440 effective and convenient ways to transmit information to a large audience (117). For example,  
441 messages displayed on computer screens at “point of decision” spots in a college dining hall  
442 influenced students to increase their fruit intake (118). Poddar et al. (119) demonstrated that 8  
443 weeks of email messages as part of a dairy intake intervention were effective in increasing dairy  
444 intake in college students relative to the comparison group. Greene et al. (31) found that a 10-  
445 lesson, web-based nutrition and physical activity intervention resulted in higher fruit and  
446 vegetable intake and greater physical activity in 1689 college students from eight universities.

447         Other studies have also reported success with mobile technology-based interventions.  
448 (120-124). Text messaging, in particular, has been used in a variety of behavioral intervention  
449 studies to provide reminders, cues, positive reinforcement and enhance self-monitoring (125-  
450 128). All of these features are recognized as keys to successful maintenance of dietary changes  
451 (124). Text messaging is an especially appealing intervention mode for college students as  
452 99.8% of college students own a cell phone and 97% of college students rely on text messaging  
453 as their main form of communication (129).

#### 454 **Conclusions:**

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

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

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

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