

1-6-2014

Coronary Heart Disease Risk Factors in College Students

Jennifer Arts

Maria Luz Fernandez

Ingrid E. Lofgren

Follow this and additional works at: https://digitalcommons.uri.edu/nfs_facpubs

**The University of Rhode Island Faculty have made this article openly available.
Please let us know how Open Access to this research benefits you.**

This is a pre-publication author manuscript of the final, published article.

Terms of Use

This article is made available under the terms and conditions applicable towards Open Access Policy Articles, as set forth in our [Terms of Use](#).

1 **Title:** Coronary heart disease risk factors in college students

2 **Running title:** Heart disease risk in college students

3 **Corresponding author:** Ingrid Lofgren, University of Rhode Island, 10 Ranger Rd, Ranger Hall
4 Rm 112, Kingston, RI, 02881 email: ingridlofgren@uri.edu telephone: 401-874-5706 fax: 401-
5 874-5974

6 **All authors and institutional affiliations:** Jennifer Arts¹, Maria Luz Fernandez², Ingrid E.
7 Lofgren¹

8 ¹Department of Nutrition and Food Sciences, University of Rhode Island, Kingston, RI

9 ²Department of Nutritional Sciences, University of Connecticut, Storrs, CT

10 **Authors Names for Pub Med Indexing:** Arts J, Fernandez ML, Lofgren IE

11 **Word count:** 10,184

12 **Number of figures:** 1 **Number of tables:** 2

13 **Sources of financial support:** USDA

14 **Conflicts of interest:** Arts J, Fernandez ML, Lofgren IE – no conflicts of interest for any of the
15 authors

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

27

28

29

30

31

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
53 Determinants of Atherosclerosis; cIMT, carotid artery intima media thickness; i3C, International
54 Childhood Cardiovascular Risk Consortium; NGHS, National Heart, Lung, and Blood Institute

55 Growth and Health Study; NHANES, National Health and Nutrition Examination Survey; WC,
56 waist circumference; VLDL-C, VLDL cholesterol; DBP, diastolic blood pressure; American
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 ≥ 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/m², 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 m³) 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/m². 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.

474 **Acknowledgments:**

475 All authors have read and approved the final manuscript.

References:

1. Burke JD, Reilly RA, Morrell JS, Lofgren IE. The University of New Hampshire's Young Adult Health Risk Screening Initiative. *J Am Diet Assoc.* 2009; 109(10): 1751-8.
2. Fernandes J, Lofgren IE. Prevalence of metabolic syndrome and individual criteria in college students. *J Am Coll Health.* 2011; 59(4): 313-21.
3. Morrell JS, Lofgren IE, Burke JD, Reilly RA. Metabolic syndrome, obesity, and related risk factors among college men and women. *J Am Coll Health.* 2012; 60(1): 82-9.
4. Huang TT, Shimel A, Lee RE, Delancey W, Strother ML. Metabolic Risks among College Students: Prevalence and Gender Differences. *Metab Syndr Relat Disord.* 2007; 5(4): 365-72.
5. Strong JP, Malcom GT, McMahan CA, Tracy RE, Newman WP, 3rd, Herderick EE, Cornhill JF. Prevalence and extent of atherosclerosis in adolescents and young adults: implications for prevention from the Pathobiological Determinants of Atherosclerosis in Youth Study. *JAMA.* 1999; 281(8): 727-35.
6. Berenson GS, Bao W. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. *New England Journal of Medicine.* 1998; 338: 1650-1655.
7. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of obesity and trends in body mass index among US children and adolescents, 1999-2010. *JAMA.* 2012; 307(5): 483-90.
8. Juhola J, Magnussen CG, Viikari JS, Kahonen M, Hutri-Kahonen N, Jula A, Lehtimäki T, Akerblom HK, Pietikainen M, Laitinen T, et al. Tracking of serum lipid levels, blood pressure, and body mass index from childhood to adulthood: the Cardiovascular Risk in Young Finns Study. *J Pediatr.* 2011; 159(4): 584-90.
9. Reis JP, Loria CM, Lewis CE, Powell-Wiley TM, Wei GS, Carr JJ, Terry JG, Liu K. Association Between Duration of Overall and Abdominal Obesity Beginning in Young Adulthood and Coronary Artery Calcification in Middle Age. *JAMA.* 2013; 310(3): 280-288.
10. Boreham C, Twisk J, Murray L, Savage M, Strain JJ, Cran G. Fitness, fatness, and coronary heart disease risk in adolescents: the Northern Ireland Young Hearts Project. *Med Sci Sports Exerc.* 2001; 33(2): 270-4.
11. Sachedek JM, Kuder JF, Economos CD. Physical fitness, adiposity, and metabolic risk factors in young college students. *Med Sci Sports Exerc.* 2010; 42(6): 1039-44.
12. Narayan KM, Boyle JP, Thompson TJ, Gregg EW, Williamson DF. Effect of BMI on lifetime risk for diabetes in the U.S. *Diabetes Care.* 2007; 30(6): 1562-6.
13. Health, United States, 2010. 2010 [cited 2011 June 22]; Available from: <http://www.cdc.gov/nchs/data/hs/hs10.pdf>.
14. Heidenreich PA, Trogdon JG, Khavjou OA, Butler J, Dracup K, Ezekowitz MD, Finkelstein EA, Hong Y, Johnston SC, Khera A, et al. Forecasting the future of cardiovascular disease in the United States: a policy statement from the American Heart Association. *Circulation.* 2011; 123(8): 933-44.
15. Go AS, Mozaffarian D, Roger VL, Benjamin EJ, Berry JD, Borden WB, Bravata DM, Dai S, Ford ES, Fox CS, et al. Heart disease and stroke statistics--2013 update: a report from the American Heart Association. *Circulation.* 2013; 127(1): e6-e245.

16. Lloyd-Jones DM, Hong Y, Labarthe D, Mozaffarian D, Appel LJ, Van Horn L, Greenlund K, Daniels S, Nichol G, Tomaselli GF, et al. Defining and setting national goals for cardiovascular health promotion and disease reduction: the American Heart Association's strategic Impact Goal through 2020 and beyond. *Circulation*. 2010; 121(4): 586-613.
17. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation*. 2002; 106(25): 3143-421.
18. National Heart, Lung, Blood The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. 2004. National Institutes of Health. p. 1-104.
19. Health, United States, 2008 with Special Feature on the Health of Young Adults, 2008.
20. Camhi SM, Katzmarzyk PT. Tracking of cardiometabolic risk factor clustering from childhood to adulthood. *Int J Pediatr Obes*. 2010; 5(2): 122-9.
21. National Heart, Lung, and Blood Institute. Expert Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents, 2012.
22. Strasser T. Reflections on Cardiovascular-Diseases. *Interdisciplinary Science Reviews*. 1978; 3(3): 225-230.
23. Goff DC, Jr., Lloyd-Jones DM, Bennett G, Coady S, D'Agostino RB, Sr., Gibbons R, Greenland P, Lackland DT, Levy D, O'Donnell CJ, et al. 2013 ACC/AHA Guideline on the Assessment of Cardiovascular Risk: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2013.
24. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). *JAMA*. 2001; 285(19): 2486-97.
25. Kuklina EV, Yoon PW, Keenan NL. Prevalence of coronary heart disease risk factors and screening for high cholesterol levels among young adults, United States, 1999-2006. *Ann Fam Med*. 2010; 8(4): 327-33.
26. Munoz LR, Etnyre A, Adams M, Herbers S, Witte A, Horlen C, Baynton S, Estrada R, Jones ME. Awareness of Heart Disease Among Female College Students. *Journal of Womens Health*. 2010; 19(12): 2253-2259.
27. Stampfer MJ, Hu FB, Manson JE, Rimm EB, Willett WC. Primary prevention of coronary heart disease in women through diet and lifestyle. *N Engl J Med*. 2000; 343(1): 16-22.
28. World Heart Federation. Diet and cardiovascular disease. 2013.
29. Hirshberg S, Fernandes, J, Melanson, K, Dwiggin, J, Lofgren, I. Dietary Sugars Predict Chronic Disease Risk Factors in College Students. *Top Clin Nutr*. 2011; 26: 324-334.
30. Irazusta A, Hoyos I, Irazusta J, Ruiz F, Diaz E, Gil J. Increased cardiovascular risk associated with poor nutritional habits in first-year university students. *Nutrition Research*. 2007; 27(7): 387-394.
31. Greene GW, White AA, Hoerr SL, Lohse B, Schembre SM, Riebe D, Patterson J, Kattelman KK, Shoff S, Horacek T, et al. Impact of an Online Healthful Eating and Physical Activity Program for College Students. *Am J Health Promot*. 2012; 27(2): e47-58.

32. Rose N, Hosig K, Davy B, Serrano E, Davis L. Whole-grain intake is associated with body mass index in college students. *J Nutr Educ Behav.* 2007; 39(2): 90-4.
33. Ha EJ, Caine-Bish N. Interactive introductory nutrition course focusing on disease prevention increased whole-grain consumption by college students. *J Nutr Educ Behav.* 2011; 43(4): 263-7.
34. Johnson RK, Appel LJ, Brands M, Howard BV, Lefevre M, Lustig RH, Sacks F, Steffen LM, Wylie-Rosett J. Dietary sugars intake and cardiovascular health: a scientific statement from the American Heart Association. *Circulation.* 2009; 120(11): 1011-20.
35. Wells HF, B.J. Dietary Assessment of major trends in US food consumption, 1970-2005. Economic Research Service. US Department of Agriculture. 2008.
36. Anderson JW, Hanna TJ, Peng X, Kryscio RJ. Whole grain foods and heart disease risk. *J Am Coll Nutr.* 2000; 19(3 Suppl): 291S-299S.
37. Grundy SM, Denke MA. Dietary influences on serum lipids and lipoproteins. *J Lipid Res.* 1990; 31(7): 1149-72.
38. Anderson JW, Hanna TJ. Whole grains and protection against coronary heart disease: what are the active components and mechanisms? *Am J Clin Nutr.* 1999; 70(3): 307-8.
39. Fernandez ML, West KL. Mechanisms by which dietary fatty acids modulate plasma lipids. *J Nutr.* 2005; 135(9): 2075-8.
40. Magnussen CG, Niinikoski H, Juonala M, Kivimaki M, Ronnema T, Viikari JS, Simell O, Raitakari OT. When and how to start prevention of atherosclerosis? Lessons from the Cardiovascular Risk in the Young Finns Study and the Special Turku Coronary Risk Factor Intervention Project. *Pediatr Nephrol.* 2011.
41. Rubin JB, Borden WB. Coronary heart disease in young adults. *Curr Atheroscler Rep.* 2012; 14(2): 140-9.
42. Holman RL, Mc GH, Jr., Strong JP, Geer JC. The natural history of atherosclerosis: the early aortic lesions as seen in New Orleans in the middle of the of the 20th century. *Am J Pathol.* 1958; 34(2): 209-35.
43. Strong JP, Mc GH, Jr. The natural history of coronary atherosclerosis. *Am J Pathol.* 1962; 40: 37-49.
44. Strong JP, McGill HC, Jr. The pediatric aspects of atherosclerosis. *J Atheroscler Res.* 1969; 9(3): 251-65.
45. Raitakari OT, Juonala M, Ronnema T, Keltikangas-Jarvinen L, Rasanen L, Pietikainen M, Hutri-Kahonen N, Taittonen L, Jokinen E, Marniemi J, et al. Cohort profile: the cardiovascular risk in Young Finns Study. *Int J Epidemiol.* 2008; 37(6): 1220-6.
46. Gall SL, JK, Smith K, Dwyer T, Venn A. The childhood determinants of adult health study: a profile of a cohort study to examine the childhood influences on adult cardiovascular health. *Austral Epidemiol.* 2009; 16: 35-39.
47. Berenson GS, Srinivasan SR, Bao W, Newman WP, 3rd, Tracy RE, Wattigney WA. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa Heart Study. *N Engl J Med.* 1998; 338(23): 1650-6.
48. Lauer RM, Connor WE, Leaverton PE, Reiter MA, Clarke WR. Coronary heart disease risk factors in school children: the Muscatine study. *J Pediatr.* 1975; 86(5): 697-706.
49. Dwyer T, Sun C, Magnussen CG, Raitakari OT, Schork NJ, Venn A, Burns TL, Juonala M, Steinberger J, Sinaiko AR, et al. Cohort Profile: the international childhood cardiovascular cohort (i3C) consortium. *Int J Epidemiol.* 2013; 42(1): 86-96.

50. Frerichs RR, Srinivasan SR, Webber LS, Berenson GR. Serum cholesterol and triglyceride levels in 3,446 children from a biracial community: the Bogalusa Heart Study. *Circulation*. 1976; 54(2): 302-9.
51. Mikkila V, Rasanen L, Raitakari OT, Pietinen P, Viikari J. Consistent dietary patterns identified from childhood to adulthood: the cardiovascular risk in Young Finns Study. *Br J Nutr*. 2005; 93(6): 923-31.
52. Gall SL, Jamrozik K, Blizzard L, Dwyer T, Venn A. Healthy lifestyles and cardiovascular risk profiles in young Australian adults: the Childhood Determinants of Adult Health Study. *Eur J Cardiovasc Prev Rehabil*. 2009; 16(6): 684-9.
53. Zieske AW, Malcom GT, Strong JP. Natural history and risk factors of atherosclerosis in children and youth: the PDAY study. *Pediatr Pathol Mol Med*. 2002; 21(2): 213-37.
54. Strydom HC. Evolution and progression of atherosclerotic lesions in coronary arteries of children and young adults. *Arteriosclerosis*. 1989; 9(1 Suppl): I19-32.
55. Juonala M, Jarvisalo MJ, Maki-Torkko N, Kahonen M, Viikari JS, Raitakari OT. Risk factors identified in childhood and decreased carotid artery elasticity in adulthood: the Cardiovascular Risk in Young Finns Study. *Circulation*. 2005; 112(10): 1486-93.
56. Raitakari OT, Juonala M, Kahonen M, Taittonen L, Laitinen T, Maki-Torkko N, Jarvisalo MJ, Uhari M, Jokinen E, Ronnema T, et al. Cardiovascular risk factors in childhood and carotid artery intima-media thickness in adulthood: the Cardiovascular Risk in Young Finns Study. *JAMA*. 2003; 290(17): 2277-83.
57. McCrindle BW. Hyperlipidemia in children. *Thromb Res*. 2006; 118(1): 49-58.
58. Cohn JN, Quyyumi AA, Hollenberg NK, Jamerson KA. Surrogate markers for cardiovascular disease: functional markers. *Circulation*. 2004; 109(25 Suppl 1): IV31-46.
59. Mancini GB, Dahlof B, Diez J. Surrogate markers for cardiovascular disease: structural markers. *Circulation*. 2004; 109(25 Suppl 1): IV22-30.
60. Juhola J, Magnussen CG, Berenson GS, Venn A, Burns TL, Sabin MA, Srinivasan SR, Daniels SR, Davis PH, Chen W, et al. Combined Effects of Child and Adult Elevated Blood pressure on Subclinical Atherosclerosis: The International Childhood Cardiovascular Cohort Consortium. *Circulation*. 2013.
61. Juonala M, Magnussen CG, Venn A, Dwyer T, Burns TL, Davis PH, Chen W, Srinivasan SR, Daniels SR, Kahonen M, et al. Influence of age on associations between childhood risk factors and carotid intima-media thickness in adulthood: the Cardiovascular Risk in Young Finns Study, the Childhood Determinants of Adult Health Study, the Bogalusa Heart Study, and the Muscatine Study for the International Childhood Cardiovascular Cohort (i3C) Consortium. *Circulation*. 2010; 122(24): 2514-20.
62. Oikonen M, Laitinen TT, Magnussen CG, Steinberger J, Sinaiko AR, Dwyer T, Venn A, Smith KJ, Hutri-Kahonen N, Pahkala K, et al. Ideal Cardiovascular Health in Young Adult Populations From the United States, Finland, and Australia and Its Association With cIMT: The International Childhood Cardiovascular Cohort Consortium. *J Am Heart Assoc*. 2013; 2(3): e000244.
63. Welsh JA, Sharma A, Cunningham SA, Vos MB. Consumption of added sugars and indicators of cardiovascular disease risk among US adolescents. *Circulation*. 2011; 123(3): 249-57.
64. Hu FB, Manson JE, Willett WC. Types of dietary fat and risk of coronary heart disease: a critical review. *J Am Coll Nutr*. 2001; 20(1): 5-19.

65. Shay CM, Ning H, Daniels SR, Rooks CR, Gidding SS, Lloyd-Jones DM. Status of Cardiovascular Health in US Adolescents: Prevalence Estimates From the National Health and Nutrition Examination Surveys (NHANES) 2005-2010. *Circulation*. 2013; 127(13): 1369-76.
66. Slining MM, Popkin BM. Trends in intakes and sources of solid fats and added sugars among U.S. children and adolescents: 1994-2010. *Pediatr Obes*. 2013.
67. May AL, Kuklina EV, Yoon PW. Prevalence of cardiovascular disease risk factors among US adolescents, 1999-2008. *Pediatrics*. 2012; 129(6): 1035-41.
68. Nelson MC, Story M, Larson NI, Neumark-Sztainer D, Lytle LA. Emerging adulthood and college-aged youth: an overlooked age for weight-related behavior change. *Obesity* 2008; 16(10): 2205-11.
69. Gordon-Larsen P, Adair LS, Nelson MC, Popkin BM. Five-year obesity incidence in the transition period between adolescence and adulthood: the National Longitudinal Study of Adolescent Health. *Am J Clin Nutr*. 2004; 80(3): 569-75.
70. Gordon-Larsen P, The NS, Adair LS. Longitudinal trends in obesity in the United States from adolescence to the third decade of life. *Obesity (Silver Spring)*. 2010; 18(9): 1801-4.
71. McDade TW, Chyu L, Duncan GJ, Hoyt LT, Doane LD, Adam EK. Adolescents' expectations for the future predict health behaviors in early adulthood. *Soc Sci Med*. 2011; 73(3): 391-8.
72. Levitsky DA, Halbmaier CA, Mrdjenovic G. The freshman weight gain: a model for the study of the epidemic of obesity. *Int J Obes Relat Metab Disord*. 2004; 28(11): 1435-42.
73. Hendricks KM, Herbold NH. Diet, activity, and other health-related behaviors in college-age women. *Nutr Rev*. 1998; 56(3): 65-75.
74. Ha EJ, Caine-Bish N, Holloman C, Lowry-Gordon K. Evaluation of effectiveness of class-based nutrition intervention on changes in soft drink and milk consumption among young adults. *Nutr J*. 2009; 8: 50.
75. Nishida C, Uauy R, Kumanyika S, Shetty P. The joint WHO/FAO expert consultation on diet, nutrition and the prevention of chronic diseases: process, product and policy implications. *Public Health Nutr*. 2004; 7(1A): 245-50.
76. Kobayashi T, Umemura U, Iso H, Ishimori M, Tamura Y, Iida M, Shimamoto T. Differences in dietary habits, serum fatty acid compositions and other coronary risk characteristics between freshmen and fourth-year male university students. *Environ Health Prev Med*. 2001; 6(3): 143-8.
77. Horacek TM, Erdman MB, Reznar MM, Olfert M, Brown-Esters ON, Kattelman KK, Kidd T, Koenings M, Phillips B, Quick V, et al. Evaluation of the food store environment on and near the campus of 15 postsecondary institutions. *Am J Health Promot*. 2013; 27(4): e81-90.
78. Horacek TM, Erdman MB, Byrd-Bredbenner C, Carey G, Colby SM, Greene GW, Guo W, Kattelman KK, Olfert M, Walsh J, et al. Assessment of the dining environment on and near the campuses of fifteen post-secondary institutions. *Public Health Nutr*. 2012: 1-11.
79. Byrd-Bredbenner C, Johnson M, Quick VM, Walsh J, Greene GW, Hoerr S, Colby SM, Kattelman KK, Phillips BW, Kidd T, et al. Sweet and salty. An assessment of the snacks and beverages sold in vending machines on US post-secondary institution campuses. *Appetite*. 2012; 58(3): 1143-51.

80. Levitsky DA, Youn T. The more food young adults are served, the more they overeat. *J Nutr.* 2004; 134(10): 2546-9.
81. 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.
82. Huang TT, Kempf AM, Strother ML, Li C, Lee RE, Harris KJ, Kaur H. Overweight and components of the metabolic syndrome in college students. *Diabetes Care.* 2004; 27(12): 3000-1.
83. Collins KA, Dantico M, Shearer NBC, Mossman KL. Heart disease awareness among college students. *Journal of Community Health.* 2004; 29(5): 405-420.
84. Cleeman JI, Grundy SM. National Cholesterol Education Program recommendations for cholesterol testing in young adults. A science-based approach. *Circulation.* 1997; 95(6): 1646-50.
85. Saenger AK. Universal lipid screening in children and adolescents: a baby step toward primordial prevention? *Clin Chem.* 2012; 58(8): 1179-81.
86. Peterson AL, McBride PE. A review of guidelines for dyslipidemia in children and adolescents. *WMJ.* 2012; 111(6): 274-81; quiz 282.
87. Garber AM, Browner WS. Cholesterol screening guidelines. Consensus, evidence, and common sense. *Circulation.* 1997; 95(6): 1642-5.
88. U.S. Preventive Services Task Force. Screening for Lipid Disorders in Adults: Recommendation Statement. *Am Fam Physician.* 2009; 80(11): 1273-1274.
89. National Cholesterol Education Program (NCEP): highlights of the report of the Expert Panel on Blood Cholesterol Levels in Children and Adolescents. *Pediatrics.* 1992; 89(3): 495-501.
90. Kavey RE, Allada V, Daniels SR, Hayman LL, McCrindle BW, Newburger JW, Parekh RS, Steinberger J. Cardiovascular risk reduction in high-risk pediatric patients: a scientific statement from the American Heart Association Expert Panel on Population and Prevention Science; the Councils on Cardiovascular Disease in the Young, Epidemiology and Prevention, Nutrition, Physical Activity and Metabolism, High Blood Pressure Research, Cardiovascular Nursing, and the Kidney in Heart Disease; and the Interdisciplinary Working Group on Quality of Care and Outcomes Research: endorsed by the American Academy of Pediatrics. *Circulation.* 2006; 114(24): 2710-38.
91. U.S. Preventive Services Task Force. Screening for Lipid Disorders in Children: Recommendation Statement. 2007; Available from: <http://www.uspreventiveservicestaskforce.org/uspstf07/chlipid/chlipidrs.htm>.
92. Daniels SR, Greer FR. Lipid screening and cardiovascular health in childhood. *Pediatrics.* 2008; 122(1): 198-208.
93. Familial Hypercholesterolemia: Screening, Diagnosis and Management of Pediatric and Adult Patients Clinical Guidance from the National Lipid Association Expert Panel on Familial Hypercholesterolemia. *Journal of Clinical Lipidology.* 2011; 5(35): S 1-58.
94. Expert panel on integrated guidelines for cardiovascular health and risk reduction in children and adolescents: summary report. *Pediatrics.* 2011; 128 Suppl 5: S213-56.
95. Gillman MW, Daniels SR. Is Universal Pediatric Lipid Screening Justified? *Jama-Journal of the American Medical Association.* 2012; 307(3): 259-260.

96. Psaty BM, Rivara FP. Universal Screening and Drug Treatment of Dyslipidemia in Children and Adolescents. *Jama-Journal of the American Medical Association*. 2012; 307(3): 257-258.
97. Newman TB, Pletcher MJ, Hulley SB. Overly Aggressive New Guidelines for Lipid Screening in Children: Evidence of a Broken Process. *Pediatrics*. 2012; 130(2): 349-352.
98. Spring B, Ockene JK, Gidding SS, Mozaffarian D, Moore S, Rosal MC, Brown MD, Vafiadis DK, Cohen DL, Burke LE, et al. Better population health through behavior change in adults: a call to action. *Circulation*. 2013; 128(19): 2169-76.
99. Laitinen TT, Pahkala K, Magnussen CG, Viikari JS, Oikonen M, Taittonen L, Mikkilä V, Jokinen E, Hutri-Kahonen N, Laitinen T, et al. Ideal cardiovascular health in childhood and cardiometabolic outcomes in adulthood: the Cardiovascular Risk in Young Finns Study. *Circulation*. 2012; 125(16): 1971-8.
100. Disparities in screening for and awareness of high blood cholesterol--United States, 1999-2002. *MMWR Morb Mortal Wkly Rep*. 2005; 54(5): 117-9.
101. US Department of Health and Human Services. Utilization of Ambulatory Medical Care by Women: United States, 1997-98. 2001; Available from: http://www.cdc.gov/nchs/data/series/sr_13/sr13_149.pdf.
102. American Heart Association Scientific Position: Public Cholesterol Screening (Adults and Children). 2012 July 9, 2013]; Available from: http://www.heart.org/HEARTORG/Conditions/Cholesterol/SymptomsDiagnosisMonitoringofHighCholesterol/Public-Cholesterol-Screening-Adults-and-Children_UCM_305617_Article.jsp.
103. Carleton RA, Dwyer J, Finberg L, Flora J, Goodman DS, Grundy SM, Havas S, Hunter GT, Kritchevsky D, Lauer RM. Report of the Expert Panel on Population Strategies for Blood Cholesterol Reduction. A statement from the National Cholesterol Education Program, National Heart, Lung, and Blood Institute, National Institutes of Health. *Circulation*. 1991; 83(6): 2154-2232.
104. Eckel RH, Jakicic JM, Ard JD, Hubbard VS, de Jesus JM, Lee IM, Lichtenstein AH, Loria CM, Millen BE, Miller NH, et al. 2013 AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2013.
105. Robbins CL, Dietz PM, Bombard JM, Gibbs F, Ko JY, Valderrama AL. Blood pressure and cholesterol screening prevalence among U.S. women of reproductive age opportunities to improve screening. *Am J Prev Med*. 2011; 41(6): 588-95.
106. U.S. Department of Education NCfES. Digest of Education Statistics, 2011. 2012.
107. Dowse GK, Gareeboo H, Alberti KG, Zimmet P, Tuomilehto J, Purran A, Fareed D, Chitson P, Collins VR. Changes in population cholesterol concentrations and other cardiovascular risk factor levels after five years of the non-communicable disease intervention programme in Mauritius. Mauritius Non-communicable Disease Study Group. *BMJ*. 1995; 311(7015): 1255-9.
108. Uusitalo U, Feskens EJ, Tuomilehto J, Dowse G, Haw U, Fareed D, Hemraj F, Gareeboo H, Alberti KG, Zimmet P. Fall in total cholesterol concentration over five years in association with changes in fatty acid composition of cooking oil in Mauritius: cross sectional survey. *BMJ*. 1996; 313(7064): 1044-6.

109. Gorman N, Lackney JA, Rollings K, Huang TT. Designer schools: the role of school space and architecture in obesity prevention. *Obesity (Silver Spring)*. 2007; 15(11): 2521-30.
110. Conklin MT, Cranage, David A., Lambert, Carolyn U. . College students' use of point of selection nutrition information. *Topics in Clinical Nutrition*. 2005; 20(2): 97-108.
111. Turconi G, Bazzano R, Roggi C, Cena H. Helping consumers make a more conscious nutritional choice: acceptability of nutrition information at a cafeteria. *Public Health Nutr*. 2012; 15(5): 792-801.
112. Peterson S, Duncan DP, Null DB, Roth SL, Gill L. Positive changes in perceptions and selections of healthful foods by college students after a short-term point-of-selection intervention at a dining hall. *J Am Coll Health*. 2010; 58(5): 425-31.
113. Freedman MR, Connors R. Point-of-Purchase Nutrition Information Influences Food-Purchasing Behaviors of College Students: A Pilot Study (Reprinted from American Dietetic Association, vol 110, pg 1222-1226, 2010). *Journal of the American Dietetic Association*. 2011; 111(5): S42-S46.
114. Driskell JA, Schake MC, Detter HA. Using Nutrition Labeling as a Potential Tool for Changing Eating Habits of University Dining Hall Patrons. *Journal of the American Dietetic Association*. 2008; 108(12): 2071-2076.
115. Kresic G, Kendel Jovanovic G, Pavicic Zewel S, Cvijanovic O, Ivezic G. The effect of nutrition knowledge on dietary intake among Croatian university students. *Coll Antropol*. 2009; 33(4): 1047-56.
116. Kremers SPJ, Eves, F.F., Andersen, R.E. Environmental Changes to Promote Physical Activity and Healthy Dietary Behavior. *Journal of Environmental and Public Health*. 2012.
117. Sirriyeh R, Lawton R, Ward J. Physical activity and adolescents: an exploratory randomized controlled trial investigating the influence of affective and instrumental text messages. *Br J Health Psychol*. 2010; 15(Pt 4): 825-40.
118. Reed JA, Powers A, Greenwood M, Smith W, Underwood R. Using "point of decision" messages to intervene on college students' eating behaviors. *Am J Health Promot*. 2011; 25(5): 298-300.
119. Poddar KH, Hosig KW, Anderson-Bill ES, Nickols-Richardson SM, Duncan SE. Dairy intake and related self-regulation improved in college students using online nutrition education. *J Acad Nutr Diet*. 2012; 112(12): 1976-86.
120. Spring B, Duncan JM, Janke EA, Kozak AT, McFadden HG, Demott A, Pictor A, Epstein LH, Siddique J, Pellegrini CA, et al. Integrating Technology Into Standard Weight Loss Treatment: A Randomized Controlled Trial. *Arch Intern Med*. 2012: 1-7.
121. Spring B, Schneider K, McFadden HG, Vaughn J, Kozak AT, Smith M, Moller AC, Epstein LH, Demott A, Hedeker D, et al. Multiple behavior changes in diet and activity: a randomized controlled trial using mobile technology. *Arch Intern Med*. 2012; 172(10): 789-96.
122. Schembre SM, Yuen J. Project TwEATs. A feasibility study testing the use of automated text messaging to monitor appetite ratings in a free-living population. *Appetite*. 2011; 56(2): 465-8.
123. Kerr DA, Pollard CM, Howat P, Delp EJ, Pickering M, Kerr KR, Dhaliwal SS, Pratt IS, Wright J, Boushey CJ. Connecting Health and Technology (CHAT): protocol of a

- randomized controlled trial to improve nutrition behaviours using mobile devices and tailored text messaging in young adults. *BMC Public Health*. 2012; 12: 477.
124. Patrick K, Raab F, Adams MA, Dillon L, Zabinski M, Rock CL, Griswold WG, Norman GJ. A text message-based intervention for weight loss: randomized controlled trial. *J Med Internet Res*. 2009; 11(1): e1.
 125. Armstrong AW, Watson AJ, Makredes M, Frangos JE, Kimball AB, Kvedar JC. Text-message reminders to improve sunscreen use: a randomized, controlled trial using electronic monitoring. *Arch Dermatol*. 2009; 145(11): 1230-6.
 126. Hanauer DA, Wentzell K, Laffel N, Laffel LM. Computerized Automated Reminder Diabetes System (CARDS): e-mail and SMS cell phone text messaging reminders to support diabetes management. *Diabetes Technol Ther*. 2009; 11(2): 99-106.
 127. Hou MY, Hurwitz S, Kavanagh E, Fortin J, Goldberg AB. Using daily text-message reminders to improve adherence with oral contraceptives: a randomized controlled trial. *Obstet Gynecol*. 2010; 116(3): 633-40.
 128. Prabhakaran L, Chee WY, Chua KC, Abisheganaden J, Wong WM. The use of text messaging to improve asthma control: a pilot study using the mobile phone short messaging service (SMS). *J Telemed Telecare*. 2010; 16(5): 286-90.
 129. Hanley M. Student Smartphone Use Doubles; Instant Messaging Loses Favor. 2009 Feb 2013]; Available from: <http://chronicle.com/blogs/wiredcampus/student-smartphone-use-doubles-instant-messaging-loses-favor/24876>.