Long-Term Effects of Childhood Risk Factors on Cardiovascular Health During Adulthood



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ABSTRACT: The primary purpose of this article is to provide a broad overview of the research on the long-term effects of childhood risk factors on cardiovascular diseases (CVDs) during adulthood and to outline recommendations for the prevention of CVDs based on evidence-based interventions. CVDs are the leading cause of death and a major cause of disability in the United States and globally. Risk factors for CVDs are already identifiable in children and youth, and include both modifiable factors (eg, unhealthy diet, physical inactivity, tobacco smoking) and factors that cannot be changed (eg, age, heredity, sex). A fundamental issue has been the severity of the long-term effects of childhood risk factors (ie, behavioral and intermediate risk factors) on subsequent cardiovascular health. It is clear from the empirical evidence that risk factors for CVDs can develop during childhood and adolescence. These risk factors in childhood have been linked to adverse health outcomes, including CVDs, during adulthood. The findings thus far suggest that, in order to be effective and reduce the risk of adulthood CVDs, intervention strategies should begin during childhood. The findings also underscore the importance of adopting a healthy lifestyle as early in life as possible.

KEYWORDS: cardiovascular diseases, heart diseases, health behavior, risk factors, biological markers

CITATION: Shrestha and Copenhaver. Long-Term Effects of Childhood Risk Factors on Cardiovascular Health During Adulthood. *Clinical Medicine Reviews in Vascular Health* 2015:7 1–5 doi: 10.4137/CMRVH.S29964.

TYPE: Short Review

RECEIVED: June 17, 2015. RESUBMITTED: July 19, 2015. ACCEPTED FOR

PUBLICATION: July 01, 2015.

ACADEMIC EDITOR: Garry Walsh, Editor in Chief

PEER REVIEW: Two peer reviewers contributed to the peer review report. Reviewers' reports totaled 655 words, excluding any confidential comments to the academic aditor.

FUNDING: Authors disclose no funding sources

COMPETING INTERESTS: Authors disclose no potential conflicts of interest

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Paper subject to independent expert blind peer review. All editorial decisions made by independent academic editor. Upon submission manuscript was subject to antiplagiarism scanning. Prior to publication all authors have given signed confirmation of agreement to article publication and compliance with all applicable ethical and legal requirements, including the accuracy of author and contributor information, disclosure of competing interests and funding sources, compliance with ethical requirements relating to human and animal study participants, and compliance with any copyright requirements of third parties. This journal is a member of the Committee on Publication Ethics (COPE).

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Cardiovascular Diseases

Globally, cardiovascular diseases (CVDs) are the leading cause of death and a major cause of disability in adults. An estimated 17.5 million people died from CVDs in 2012, representing 31% of all global deaths. More than 3 million of these deaths occurred before the age of 60 and could have been prevented. In the US alone, nearly 787,000 people died from CVDs in 2011, and it has remained a key factor of rising healthcare costs. The American Heart Association reports that one in every three US adults currently has one or more forms of CVDs. The direct and indirect costs of CVDs total more than \$320 billion, which includes health expenditures and opportunity costs. ²

Over the past four decades, major progress has been made in the prevention, diagnosis, and treatment of CVDs and their risk factors. Death rates from CVDs have declined, and people are living longer and healthier lives. Many adults in the US, nevertheless, are at increased risk for CVDs despite these encouraging developments. Morbidity and mortality associated with CVDs still continue to inflict a heavy burden on patients, their families, and the national healthcare system. In this article, we present an overview of the research studies linking childhood risk factors to cardiovascular health

later in life and also suggest recommendations for prevention of CVDs based on evidence-based interventions (EBIs).

Risk Factors for CVDs

CVDs are multifactorial; some risk factors are modifiable, and some (eg, age, heredity, and sex) cannot be modified.^{1,3} Among the important modifiable behavioral risk factors for CVDs are a diet that is high in saturated fat and sodium, physical inactivity, tobacco smoking, and excessive alcohol consumption. The effects of these behavioral risk factors may show up in individuals as "intermediate risk factors," which include high blood cholesterol, high blood pressure, raised blood lipids, obesity, and diabetes mellitus. Stress may also contribute to CVD risk.^{1,3} The risk for CVDs is higher among Mexican Americans, American Indians, Native Hawaiians, and some Asian Americans, in part due to higher prevalence of obesity and diabetes in these populations. Most CVDs can be prevented by addressing long-term exposure to the major behavioral risk factors - such as tobacco smoking, physical inactivity, unhealthy diets, and the harmful use of alcohol - using population-wide approaches and improved access to individual healthcare interventions. 1,3



Modifiable Childhood Risk Behaviors and Clinical Outcomes of CVDs in Adulthood

Empirical studies from the last three decades have greatly improved our understanding of CVDs and their modifiable risk factors. Although CVDs are usually diagnosed in adulthood, unfortunately, their origin often begins early in life. Compelling research results suggest that childhood influences may contribute to the risk of CVDs later in life. For example, risk factor (ie, behavioral and intermediate) levels tended to be high already in childhood and those with elevated levels tended to be more likely to have adverse risk factor levels as adults. The constellation of childhood risk factors has been associated with preclinical vascular markers in adulthood, as described in the following. The constellation of childhood risk factors has been associated with preclinical vascular markers in adulthood, as

Diet. Healthy eating habits are established early in life (ie, childhood) and plays a crucial role in primary prevention of a range of behavioral risk factors associated with CVDs later in life.²³ A growing body of evidence on the effectiveness of long-term dietary intervention for the reduction of risk factors for CVDs has suggested that changes in specific dietary macronutrients (eg, dietary fat and carbohydrates) and micronutrients (eg, sodium and calcium) have a significant influence on the risk of CVDs. 23-27 Children who consume large portion-sizes, more calories than they burn, and high-energydense foods gain excess weight and body fat and increase their CVD risks.²³ For example, the Cardiovascular Risk in Young Finns Study demonstrated that healthful dietary patterns developed early in life and the cardiovascular health benefits accumulated from such patterns track into adulthood. 18 Similarly, findings from the Amsterdam Growth and Health Longitudinal Study, an observational study with a long follow-up period of 23 years, suggested that adherence to a Mediterranean dietary pattern and higher fiber intake are associated with less stiff arteries in adulthood. 28,29

Physical activity. There have been an increasing number of reports that have examined the impact of youth physical activity and change in physical activity between youth and adulthood on adult preclinical vascular measures. From the Young Finns Study, Juonala et al showed that higher physical activity in youth is associated with reduced carotid intimalmedial thickness (CIMT) progression over 6 years in adulthood.¹⁹ In the European Youth Heart Study, higher youth levels of objectively measured moderate and vigorous physical activity at a mean age of 15 years were associated with reduced arterial stiffness 12 years later.³⁰ Interestingly, those who maintained a stable physical activity level or had a small increase in moderate and vigorous physical activity from youth to adulthood tended to have reduced arterial stiffness as adults independent of other lifestyle factors.³⁰ Though additional work is needed to elucidate all the complexities of intensity as well as the association of sedentary behavior independent of physical activity levels on adult vascular markers, these data do point toward the benefits of childhood physical activity on long-term cardiovascular health.

Active and passive smoking. Recent studies have highlighted the importance of preventing smoking and exposure to environmental tobacco smoke in children because of its direct association with CVD risks. Active smoking in adolescence is associated with increased CIMT and decreased carotid artery elasticity in young adulthood. For example, Raitakari et al showed that CIMT in adulthood is significantly associated with both smoking in childhood and adult smoking status.²¹ Similarly, a cohort study of 732 young adults by Geerts et al showed that exposure to tobacco smoke from parents induces vascular damage in offspring in early adulthood. The same study found that the exposure of the cardiovascular system to tobacco smoke in utero was associated with the vessels being more vulnerable later in life. 31 Similarly, environmental tobacco smoke in childhood has been linked with risk factors for and CVD processes (eg, dyslipidemia, impaired endothelial function) in adulthood.³² Therefore, the prevention or cessation of tobacco use could have a significant implication on cardiovascular health promotion and risk reduction among adults.

Obesity. Obesity is associated with a number of cardiovascular risk factors, such as hypertension, dyslipidemia, and diabetes mellitus.³³ Furthermore, childhood obesity is associated with an increased all-cause adult mortality. 34-37 Likewise, studies on the effect of obesity measured in childhood and adolescence on future cardiovascular mortality have also shown a positive relationship. 10,38-41 Ferreira et al found that the roots of the association of body fatness and body fat distribution with large artery structural and functional properties at age 36 were already present in adolescence. 11 Similarly, the Muscatine Study by Davis et al found that higher CIMT in young and middle-aged adults was associated with childhood cardiovascular risk factors.⁴² The Young Finns Study showed that higher body mass index (BMI) measured in childhood was significantly associated with CIMT, measured in adulthood.²¹ Another prospective study by Tirosh et al found that an elevated BMI early in life was associated with an increased risk of CVDs in adulthood.²⁰

Blood lipids. There has been a considerable amount of evidence that supports the association of adverse levels and patterns of lipids and lipoproteins to the development of CVDs later in life. For example, in the Muscatine Study - a longitudinal study that looked at risk factors for CVDs in school-age children and adults - carotid ultrasound in adults indicated that CIMT was positively associated with levels of total cholesterol measured in childhood.⁴² Similar results were witnessed in the Bogalusa Heart Study, in which childhood low-density lipoprotein cholesterol (LDL-C) was found to predict increased CIMT in adulthood.⁴³ In the Young Finns Study - a population-based prospective cohort study - associations between risk factor exposures in adolescence, including LDL-C, predicted CIMT in adulthood independently of adult risk factor levels.²¹ Therefore, evidence is mounting that the risk factors begin in childhood and are associated with the same CVDs risk factors that are well established in adults.



Blood pressure. Elevated blood pressure has been established as a very convincing risk factor for CVDs in children and adults.²³ In addition, the presence of hypertension in children and adolescents has been linked with a range of CVDs in adulthood. For example, Daniels et al showed that 8% of children and adolescents with hypertension already had left ventricular mass index elevated to a level associated with a fourfold increased risk of CVDs in adults with hypertension.²³ These results underscore the clinical importance of blood pressure elevation in children and adolescents. In the Muscatine Study, Davis et al demonstrated that childhood diastolic blood pressure was a significant predictor of CIMT in adult males. 42 Similarly, the International Childhood Cardiovascular Cohort Consortium showed that individuals with consistently raised blood pressure in childhood had significantly increased likelihood of developing of carotid atherosclerosis.²² The Young Finns Study found that systolic blood pressure measured in childhood and adolescence was inversely linked with adulthood brachial artery flow-mediated dilation, which is an indicator of endothelial function.⁴⁴

Much of these compelling historical data reinforcing the association between childhood risk behaviors and preclinical vascular markers came from epidemiological studies that followed participants for a significant period of time (eg, childhood through early adulthood) to observe which behavioral factors contributed to the development of CVDs. Collectively, such studies have played a key role in the understanding of multifactorial origins of CVDs in adult life and developing specifically tailored interventions for CVDs prevention.

Reducing the Burden of CVDs: Evidence and Future Recommendations

The evidence that CVD risks may be initiated in childhood requires risk factors to be widely explored during this period, with the aim of designing earlier and more effective preventive strategies and reducing morbidity and mortality in adult life. Therefore, based on existing evidence, we suggest holistic population-based approach for reduction of morbidity and mortality from CVDs through changes in modifiable risk-related behaviors, primarily high-fat and high-sodium diets, sedentariness, and cigarette smoking beginning in childhood and continuing through adulthood. ^{23,25,26,45–56}

Because dietary habits and food preferences are established in early childhood, it is important to intervene early to improve nutritional patterns of children and adolescents. Clearly, improvements in dietary patterns and physical activity and maintenance of a healthy weight throughout childhood and adolescence are likely to prevent the development of CVDs in children and subsequently in adults.²³ Therefore, interventions should emphasize individual adherence to prudent dietary patterns of low total and saturated fat (ie, diet containing omega-3 fatty acids) and cholesterol; provide children with more fruits, vegetables, fiber, and fat-free or low-fat dairy items; encourage the consumption of less dietary

salt and sodium and limited or no intake of sugar-sweetened beverages; and control of portion-sizes in early childhood and throughout adolescence.^{24–26,46–53,56}

At an individual level, healthcare providers should evaluate a child's level of physical activity and sedentary behaviors at each well-child visit at a minimum. If needed, they should provide age-appropriate recommendations (taking into account the child's and family's preferences and resources available) for increasing the child's involvement in physical activities and limiting sedentary behaviors. Time spent taking part in age-appropriate sports should also be highlighted, along with time spent in school or day-care physical education.

Beginning early in life and extending through adolescence, parents are instrumental in influencing their child's behavioral practices. ^{57,58} Parental role-modeling of physical activity and healthy eating behavior is important in promoting such behaviors among children. Parents should provide their child with the ability and opportunity to make healthy eating choices and should be involved in the child's dietary intervention to mitigate risk. ⁵⁹ Furthermore, parents should encourage their children to participate in physical activities (both at home and school), and they should engage in such activities with their children. Furthermore, interventions can focus on using age-appropriate behavioral change strategies, as part of individual and family counseling, specifically designed to increase levels of physical activity and to decrease sedentary behaviors. ²³

Evidence supports the need for both individual and population-based approaches for the prevention of smoking initiation and interventions for smoking cessation for children, youth, and adults. ⁵⁴ Parents and guardians should make sure to maintain a smoke-free home environment and to avoid exposure to secondhand smoke (SHS) in other environments. Schools need to focus on the assessment of smoking status with clear, firm, and consistent messages about the importance of remaining smoke-free. Interventions should also deliver nonsmoking messages in clinical encounters through educational materials in the clinical/office-based setting and advocate for efforts designed to reduce smoking initiation in community-based settings.

Preventing obesity, optimizing cholesterol levels, and managing dyslipidemia in childhood and adolescence should remain a priority for CVD risk reduction during adulthood. At an individual level, children should have their weight and height measured and BMI calculated at every visit with the healthcare provider. Parental obesity, family medical history, BMI trajectory, and CVDs risk factors need to be considered in the management of weight and in CVDs risk reduction. Population-based interventions should focus on activities that prevent excess weight gain and promote optimal lipid levels through lifestyle modifications, which include a healthy diet, adequate physical activity, and limited screen time. ²³ Dietary recommendations for children and youth should emphasize on a balanced caloric intake with sufficient physical activity to



achieve a normal weight and increased consumption of fruits, vegetables, whole grains, fish, and low-fat dairy products. ⁴² Individuals at risk due to family history of CVDs or presence of elevated levels of LDL-C should be offered or placed on therapeutic lifestyle change, with an emphasis on an adequate trial of dietary therapy and increased physical activity.

Using Policy, Systems, and Environmental (PSE) Change to Combat CVDs

The PSE changes in neighborhoods, schools, workplaces, transportation systems, and healthcare settings play a significant role in shaping health behaviors of individuals beginning from childhood through adulthood, which eventually determines health outcomes (ie, CVDs).55 Access to affordable fruits and vegetables, design of sidewalks and bike lanes within communities, and smoke-free policies in workplaces and businesses directly increase the likelihood that children and parents eat healthy and nutritious food, walk to school or work, and avoid exposure to second-hand smoke. PSEs in communities that make healthy choices easy, safe, and affordable can have a positive impact on the way children and youth live, learn, work, and play.60 Furthermore, partnerships with community leaders in education, government, transportation, and business are essential in creating sustainable change for children to practice health behaviors and thus reduce the burden of CVDs in adulthood.

Conclusions

A growing body of evidence shows that risk factors for CVDs can develop during childhood and adolescence. When risk factors develop at an early age, they are likely to continue over time, maintaining a high-risk status. This pattern has been linked to adverse health outcomes, including CVDs, during adulthood. Thus, because the development of CVDs has its origins in childhood, approaches to prevention must be directed at the developing child and adolescent. Although this is challenging, it can be achieved by promoting healthy behaviors, minimizing behavioral risk factors through individual behavior change, and instituting and maintaining optimum health behaviors early in life through PSE changes. Although substantial progress has been made in terms of understanding the risk factors of CVDs and reducing CVDs mortality rates, considerable opportunity exists for further reducing CVDs and its complications by improving and applying healthy behavioral interventions early in life. Future research needs to be done to examine the molecular pathway of how childhood behaviors impact cardiovascular health later in life. Furthermore, additional research needs to be carried out that is directed at developing, testing, and assessing cost-effective interventions, particularly focused at improving health behaviors among children and youth. Moving forward, the identified EBIs should be incorporated into larger health systems and policies that positively affect cardiovascular health at the population level.

Author Contributions

Conceived and designed the study: RS. Analyzed the data: RS. Wrote the first draft of the manuscript: RS. Contributed to the writing of the manuscript: RS, MC. Agree with manuscript results and conclusions: RS, MC. Jointly developed the structure and arguments for the paper: RS, MC. Made critical revisions and approved final version: RS, MC. Both authors reviewed and approved of the final manuscript.

REFERENCES

- WHO. Global Status Report on Noncommunicable Diseases 2014. World Health Organization. Geneva: 2014.
- Mozaffarian D, Benjamin EJ, Go AS, et al. Heart disease and stroke statistics 2015 update: a report from the American heart association. *Circulation*. 2015;131(4): e29–322.
- Barrett-Connor E, Ayanian JZ, Brown ER, et al. A Nationwide Framework for Surveillance of Cardiovascular and Chronic Lung Diseases. National Academies Press. Washington, DC, US: 2011.
- Juhola J, Magnussen CG, Viikari JS, 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.
- Chen X, Wang Y. Tracking of blood pressure from childhood to adulthood: a systematic review and meta-regression analysis. *Circulation*. 2008;117(25):3171–80.
- Freedman DS, Patel DA, Srinivasan SR, et al. The contribution of childhood obesity to adult carotid intima-media thickness: the Bogalusa heart study. Int J Obes (Lond). 2008;32(5):749–56.
- Baker JL, Olsen LW, Sørensen TIA. Childhood body-mass index and the risk of coronary heart disease in adulthood. N Engl J Med. 2007;357(23):2329–37.
- Morrison JA, Glueck CJ, Horn PS, Yeramaneni S, Wang P. Pediatric triglycerides predict cardiovascular disease events in the fourth to fifth decade of life. *Metabolism*. 2009;58(9):1277–84.
- 9. Morrison JA, Glueck CJ, Wang P. Childhood risk factors predict cardiovascular disease, impaired fasting glucose plus type 2 diabetes mellitus, and high blood pressure 26 years later at a mean age of 38 years: the Princeton-lipid research clinics follow-up study. *Metabolism*. 2012;61(4):531–41.
- Gunnell DJ, Frankel SJ, Nanchahal K, Peters TJ, Davey Smith G. Childhood obesity and adult cardiovascular mortality: a 57-y follow-up study based on the Boyd Orr cohort. Am J Clin Nutr. 1998;67(6):1111–8.
- Ferreira I, Twisk JW, van Mechelen W, Kemper HC, Seidell JC, Stehouwer CD.
 Current and adolescent body fatness and fat distribution: relationships with carotid intima-media thickness and large artery stiffness at the age of 36 years.
 J Hypertens. 2004;22(1):145–55.
- Mahmood SS, Levy D, Vasan RS, Wang TJ. The Framingham heart study and the epidemiology of cardiovascular disease: a historical perspective. *Lancet*. 2014;383(9921):999–1008.
- Hartiala O, Magnussen CG, Kajander S, et al. Adolescence risk factors are predictive of coronary artery calcification at middle age: the cardiovascular risk in young Finns study. J Am Coll Cardiol. 2012;60(15):1364–70.
- 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;112:1486–93.
- Raitakari OT, Juonala M, Ronnemaa T, et al. Cohort profile: the cardiovascular risk in young finns study. Int J Epidemiol. 2008;37:1220–6.
- Wijnstok NJ, Hoekstra T, van Mechelen W, Kemper HC, Twisk JW. Cohort profile: the Amsterdam growth and health longitudinal study. Int J Epidemiol. 2013;42:422–9.
- Magnussen C, Smith K, Juonala M. What the long term cohort studies that began in childhood have taught us about the origins of coronary heart disease. Curr Cardiovasc Risk Rep. 2014;8(2):1–10.
- Mikkilä V, Räsänen L, Laaksonen MM, et al. Long-term dietary patterns and carotid artery intima media thickness: the cardiovascular risk in young finns study. Br J Nutr. 2009;102:1507–12.
- Juonala M, Viikari JS, Kähönen M, et al. Life-time risk factors and progression of carotid atherosclerosis in young adults: the cardiovascular risk in young Finns study. Eur Heart J. 2010;31:1745–51.
- Tirosh A, Shai I, Afek A, et al. Adolescent BMI trajectory and risk of diabetes versus coronary disease. N Engl J Med. 2011;364(14):1315–25.
- Raitakari OT, Juonala M, Kahonen M, et al. Cardiovascular risk factors in childhood and carotid artery intima-media thickness in adulthood: the cardiovascular risk in young Finns study. *JAMA*. 2003;290:2277–83.
- Juhola J, Magnussen CG, Berenson GS, et al. Combined effects of child and adult elevated blood pressure on subclinical atherosclerosis: the international childhood cardiovascular cohort consortium. *Circulation*. 2013;128:217–24.



- Daniels SR, Pratt CA, Hayman LL. Reduction of risk for cardiovascular disease in children and adolescents. *Circulation*. 2011;124:1673–86.
- Aburto NJ, Ziolkovska A, Hooper L, Elliott P, Cappuccio FP, Meerpohl JJ. Effect of lower sodium intake on health: systematic review and meta-analyses. BMJ. 2013;346:f1326.
- He FJ, Li J, Macgregor GA. Effect of longer-term modest salt reduction on blood pressure. Cochrane Database Syst Rev. 2013;4:CD004937.
- Brehm BJ, Seeley RJ, Daniels SR, D'Alessio DAA. Randomized trial comparing a very low carbohydrate diet and a calorie-restricted low fat diet on body weight and cardiovascular risk factors in healthy women. *J Clin Endocrinol Metab*. 2003;88(4):1617–23.
- Cook NR, Cutler JA, Obarzanek E, et al. Long term effects of dietary sodium reduction on cardiovascular disease outcomes: observational follow-up of the trials of hypertension prevention (TOHP). BMJ. 2007;334(7599):885–8.
- van de Laar RJ, Stehouwer CD, van Bussel BC, Prins MH, Twisk JW, Ferreira I.
 Adherence to a Mediterranean dietary pattern in early life is associated with lower arterial stiffness in adulthood: the Amsterdam growth and health longitudinal study. *I Intern Med.* 2013;273(1):79–93.
- 29. van de Laar RJ, Stehouwer CD, van Bussel BC, et al. Lower lifetime dietary fiber intake is associated with carotid artery stiffness: the Amsterdam growth and health longitudinal study. *Am J Clin Nutr.* 2012;96:14–23.
- Ried-Larsen M, Grøntved A, Kristensen PL, Froberg K, Andersen LB. Moderate-and-vigorous physical activity from adolescence to adulthood and subclinical atherosclerosis in adulthood: prospective observations from the European youth heart study. Br J Sports Med. 2013;49(2):107–12.
- Geerts CC, Bots ML, Grobbee DE, Uiterwaal CS. Parental smoking and vascular damage in young adult offspring: is early life exposure critical? The atherosclerosis risk in young adults study. *Arterioscler Thromb Vasc Biol.* 2008;28:2296–302.
- DHHS. The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General. Atlanta GA: Centers for Disease Control and Prevention (US); 2006.
- Chopra I, Kamal KM, Candrilli SD, Kanyongo G. Association between obesity
 and therapeutic goal attainment in patients with concomitant hypertension and
 dyslipidemia. *Postgrad Med.* 2014;126(1):66–77.
- 34. Jee SH, Sull JW, Park J, et al. Body-mass index and mortality in korean men and women. N Engl J Med. 2006;355(8):779–87.
- Flegal KM, Graubard BI, Williamson DF, Gail MH. CAuse-specific excess deaths associated with underweight, overweight, and obesity. JAMA. 2007;298(17):2028–37.
- Berrington de Gonzalez A, Hartge P, Cerhan JR, et al. Body-mass index and mortality among 1.46 million white adults. N Engl J Med. 2010;363(23):2211–9.
- Zheng W, McLerran DF, Rolland B, et al. Association between body-mass index and risk of death in more than 1 million Asians. N Engl J Med. 2011;364(8):719–29.
- 38. Kushner RF. Body Weight and Mortality. Nutr Rev. 1993;51(5):127–36.
- Wannamethee S, Shaper A, Walker M. Weight change, body weight and mortality: the impact of smoking and ill health. Int J Epidemiol. 2001;30(4):777–86.
- Rimm EB, Stampfer MJ, Giovannucci E, et al. Body size and fat distribution as predictors of coronary heart disease among middle-aged and older US men. Am J Epidemiol. 1995;141(12):1117–27.
- Manson JE, Willett WC, Stampfer MJ, et al. Body weight and mortality among women. N Engl J Med. 1995;333(11):677–85.
- Davis PH, Dawson JD, Riley WA, Lauer RM. Carotid intimal-medial thickness is related to cardiovascular risk factors measured from childhood through middle age: the Muscatine study. *Circulation*. 2001;104(23):2815–9.

- 43. Li S, Chen W, Srinivasan SR, et al. Childhood cardiovascular risk factors and carotid vascular changes in adulthood: the Bogalusa heart study. *JAMA*. 2003;290: 2271_6
- 44. Juonala M, Viikari JS, Ronnemaa T, Helenius H, Taittonen L, Raitakari OT. Elevated blood pressure in adolescent boys predicts endothelial dysfunction: the cardiovascular risk in young Finns study. *Hypertension*. 2006:424–30.
- Health Epoigfe, Children RRI, Adolescents. Expert panel on integrated guidelines for cardiovascular health and risk reduction in children and adolescents: summary report. *Pediatrics*. 2011;128(suppl 5):S213–56.
- American Heart Association Nutrition Committee, Lichtenstein AH, Appel LJ, et al. Diet and lifestyle recommendations revision 2006: a scientific statement from the American heart association nutrition committee. *Circulation*. 2006:114(1):82–96.
- 47. Johnson RK, Appel LJ, Brands M, et al. @@ American Heart Association Nutrition Committee of the Council on Nutrition, Physical Activity, and Metabolism and the Council on Epidemiology and Prevention@@. Dietary sugars intake and cardiovascular health: a scientific statement from the American heart association. Circulation. 2009:120(11):1011–20.
- Howard BV, Van Horn L, Hsia J, et al. Low-fat dietary pattern and risk of cardiovascular disease: the women's health initiative randomized controlled dietary modification trial. *JAMA*. 2006;295(6):655–66.
- 49. Appel LJ, Moore TJ, Obarzanek E, et al. A clinical trial of the effects of dietary patterns on blood pressure. N Engl J Med. 1997;336(16):1117–24.
- Mozaffarian D, Micha R, Wallace S. Effects on coronary heart disease of increasing polyunsaturated fat in place of saturated fat: a systematic review and meta-analysis of randomized controlled trials. *PLoS Med.* 2010;7(3):e1000252.
- Turpeinen O, Karvonen MJ, Pekkarinen M, Miettinen M, Elosuo R, Paavilainen E. Dietary prevention of coronary heart disease: the Finnish mental hospital study. *Int J Epidemiol.* 1979;8(2):99–118.
- 52. Leren P. The Oslo diet-heart study: eleven-year report. Circulation. 1970;42(5):935–42.
- Dayton S, Pearce ML, Hashimoto S, Dixon WJ, Tomiyasu U. A controlled clinical trial of a diet high in unsaturated fat in preventing complications of atherosclerosis. *Circulation*. 1969;40(1S2):II-1-II-63.
- 54. DHHS. Preventing Tobacco Use Among Youth and Young Adults: A Report of the Surgeon General. Atlanta, GA: 2012.
- NACCHOO. Healthy Communities, Healthy Behaviors: Using Policy, Systems, and Environmental Change to Combat Chronic Disease. 2011. Available from: http://www.naccho.org/topics/HPDP/mcah/upload/issuebrief_pse_webfinal.pdf.
- Mozaffarian D, Wu JH. Omega-3 fatty acids and cardiovascular disease: effects on risk factors, molecular pathways, and clinical events. *J Am Coll Cardiol*. 2011;58(20):2047–67.
- Hoerr SL, Hughes SO, Fisher JO, Nicklas TA, Liu Y, Shewchuk RM. Associations among parental feeding styles and children's food intake in families with limited incomes. *Int J Behav Nutr Phys Act.* 2009;6:55.
- Hingle MD, O'Connor TM, Dave JM, Baranowski T. Parental involvement in interventions to improve child dietary intake: a systematic review. *Prev Med.* 2010;51(2):103–11.
- Rennie KL, Johnson L, Jebb SA. Behavioural determinants of obesity. Best Pract Res Clin Endocrinol Metab. 2005;19(3):343–58.
- RWJF. Where We Live Matters for Our Health: Neighborhoods and Health. 2008. Available from: http://www.commissiononhealth.org/PDF/888f4a18eb90-45be-a2f8-159e84a55a4c/Issue%20Brief%203%20Sept%2008%20 -%20 Neighborhoods%20and%20Health.pdf.