Recent Advances in Preventive Cardiology and Lifestyle Medicine

Reduction of Risk for Cardiovascular Disease in Children and Adolescents

Stephen R. Daniels, MD, PhD; Charlotte A. Pratt, PhD, RD; Laura L. Hayman, RN, PhD

Atherosclerotic cardiovascular disease (CVD) is the number 1 cause of death in the United States and other developed nations. After decades of study, risk factors for the development of atherosclerotic CVD have been identified. These risk factors include older age, male sex, a positive family history, hypertension, dyslipidemia, diabetes mellitus, cigarette smoking, and obesity. As these risk factors have been studied, it has become clear that both genetics and lifestyle are important contributors to increased risk. The primary lifestyle components are diet, physical activity, and smoking.

A fundamental issue has been the timing of the development of atherosclerosis. Timing is critical because it determines, at least in part, when interventions should occur. Both the prevention of risk factor development (primordial prevention) and modification of risk factors once they are established (primary prevention) are important. An impediment to understanding the development of atherosclerosis has been the lack of simple, noninvasive methods to follow the atherosclerotic process longitudinally. This problem led to the Bogalusa Heart Study and the Pathobiological Determinants of Atherosclerosis in Youth (PDAY). These pathology studies investigated the aorta and coronary arteries in autopsies of young individuals who died of accidental causes. The investigators were able to evaluate the extent of atherosclerosis and the presence of risk factors. They found that even relatively advanced levels of atherosclerosis, including fibrous plaques, can be present in adolescents and young adults. They also found that increased body mass index (BMI), systolic and diastolic blood pressures, and low-density lipoprotein cholesterol (LDL-C); low levels of high-density lipoprotein cholesterol (HDL-C); diabetes mellitus; and the presence of cigarette smoking were all associated with greater atherosclerotic plaque coverage and more advanced atherosclerotic lesions.

Subsequent studies using noninvasive measures of atherosclerosis, including carotid intima-medial thickness (CIMT) and arterial distensibility, have resulted in similar findings. Therefore, evidence is mounting that atherosclerosis begins in childhood and is directly associated with the same CVD risk factors that are well established in adults.

When these results are taken together, they emphasize the need for appropriate CVD prevention strategies in children and adolescents. Through these preventive efforts, it should be possible to maintain low-risk status into adulthood. Low-CVD-risk status maintained to 50 years of age is associated with a very low future risk of CVD. This is a fundamental principle behind the American Heart Association’s 2020 goals, which are focused on achieving and monitoring cardiovascular health.

In this report, we summarize the evidence and the current published recommendations regarding the epidemiology of risk factors for atherosclerotic CVD in childhood. We outline the recommended clinical approaches to prevent risk factor development and review cut points for identifying risk factors and approaches for ameliorating high-risk status once it has developed. The National Heart, Lung and Blood Institute (NHLBI) has formed an advisory group to review evidence and to make recommendations regarding the prevention of CVD in children and adolescents. This work is in progress and has not been published. This report is an overview and a synthesis of previously published recommendations.

Diet

Healthy dietary patterns develop in childhood and are important for primordial and primary prevention of risk factors related to CVD from childhood and adolescence through adulthood. Evidence of the effectiveness of long-term dietary intervention for the reduction of risk factors for CVD in children is limited, but ample data suggest that changes in specific dietary macronutrients (eg, dietary fat and carbohydrates) and micronutrients (eg, sodium and calcium) have an impact on the risk of CVD.

How much energy a child or adolescent should consume depends on age, sex, stage of growth, body weight and size, and level of physical activity. Table 1 presents an estimate of caloric needs by age, sex, and activity level. Children who are sedentary need less energy compared with those who are active.

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Table 1. Suggested Caloric Intake by Sex, Age, and Activity Level

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age, y</th>
<th>Sedentary*</th>
<th>Moderately Active†</th>
<th>Active‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>2–3</td>
<td>1000–1200</td>
<td>1000–1200</td>
<td>1000–1400</td>
</tr>
<tr>
<td></td>
<td>4–8</td>
<td>1200–1400</td>
<td>1400–1600</td>
<td>1400–1800</td>
</tr>
<tr>
<td></td>
<td>9–13</td>
<td>1400–1600</td>
<td>1600–2000</td>
<td>1800–2200</td>
</tr>
<tr>
<td></td>
<td>14–18</td>
<td>1800</td>
<td>2000–2200</td>
<td>2400</td>
</tr>
<tr>
<td>Male</td>
<td>2–3</td>
<td>1000–1200</td>
<td>1000–1200</td>
<td>1000–1400</td>
</tr>
<tr>
<td></td>
<td>4–8</td>
<td>1200–1400</td>
<td>1400–1600</td>
<td>1600–2000</td>
</tr>
<tr>
<td></td>
<td>14–18</td>
<td>1800–2400</td>
<td>2400–2800</td>
<td>2800–3200</td>
</tr>
</tbody>
</table>


*Sedentary means a lifestyle that includes only the physical activity of independent living.
†Moderately active means a lifestyle that includes physical activity equivalent to walking about 1.5 to 3 miles/d at 3 to 4 mph, in addition to the activities of independent living.
‡Active means a lifestyle that includes physical activity equivalent to walking >3 miles/d at 3 to 4 mph, in addition to the activities of independent living.

Thus, the types and amount of food groups needed to meet caloric needs also vary15 (http://www.mypyramid.gov).

There is evidence linking diet to cardiovascular health.13,14 Table I in the online-only Data Supplement presents strong and moderately strong evidence relating macronutrients, micronutrients, foods, and food environment to health. There is strong and/or moderately strong evidence of a positive association between adiposity and macronutrients: dietary fat, total energy intake, and energy density of foods, as well as sugar-sweetened beverages16 and portion sizes (Table I in the online-only Data Supplement). Children and adolescents who consume large portion sizes, more calories than they expend, and high-energy-dense foods gain excess weight and body fat and increase their CVD risks. Those who eat breakfast have been reported to be at lower risk of being overweight or obese and more likely to consume adequate intakes of essential nutrients such as calcium and iron.13,17–19 Based on 21 years of follow-up data from youth 3 to 18 years of age, the Cardiovascular Risk in Young Finns Study20 demonstrated that healthful dietary patterns developed in childhood and the cardiovascular health benefits accrued from such patterns track into adulthood. Clinical trials have demonstrated that diets low in total fat (<30% of energy), saturated fat (8%–10% of energy), and cholesterol (200–300 mg/d) significantly reduced total cholesterol (TC), LDL-C, and C-reactive protein.21

Because dietary habits and preferences are established in early childhood, it is important to intervene early to improve dietary 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 CVD in children and adolescents and subsequently in adults. Primary care providers should counsel their pediatric patients and their families to adhere to prudent dietary patterns of low total and saturated fat and cholesterol; to provide youth with more fruits, vegetables, fiber, and fat-free or low-fat dairy; to encourage the consumption of less dietary salt and sodium and limited or no intake of sugar-sweetened beverages; and to control portion sizes in early childhood and throughout adolescence.

Physical Activity

Evidence accumulated over the past several decades supports a multitude of benefits associated with a physically active lifestyle in children and youth.22 Health-related benefits of regular physical activity documented in clinical and epidemiological studies and summarized in recent comprehensive reviews include improved cardiovascular fitness, increased bone mass, improved psychological well-being, and lower risk of obesity and elevated blood pressure.22,23 In contrast, results of several observational studies of children and adolescents (4–18 years of age) and young adults (19–21 years of age) demonstrate associations between increased time spent in sedentary activities and decreased levels of physical activity, adverse lipid profiles, increased levels of obesity, and related cardiovascular risk factors, including hypertension and insulin resistance.24,25 Longitudinal data from the Cardiovascular Risk in Young Finns Study and the Muscatine Study, similar to observations of adults, indicate that optimal cardiovascular risk profiles are seen in individuals who are consistently physically active.24,25

Tracking of levels of physical activity from childhood to young adulthood has also been documented, with the most consistent tracking observed for high levels of physical activity at 9 to 18 years of age predicting higher levels of physical activity in adulthood.26 Physically active children and youth are more likely to engage in other health-promoting behaviors and less likely to engage in health-compromising behaviors than their less active counterparts.27 Finally, results of studies that include interventions designed to increase physical activity and to decrease sedentary time have demonstrated reductions in systolic and diastolic blood pressures,28 decreased measures of body fat,29 decreased BMI,30 improved cardiorespiratory fitness,31 and improved cardiometabolic risk profiles.32 However, the results are not consistent across studies, and the dose (duration and intensity) of physical activity required to modify cardiovascular risk factors in children and youth remains to be clarified. Current recommendations for healthy children and youth (≥6 years of age) include at least 1 hour of moderate to vigorous physical activity (MVPA) daily with vigorous-intensity physical activity and muscle- and bone-strengthening activities on at least 3 days of the week.22,33 Moderate intensity is defined as 3.0 to 6.0 metabolic equivalents (METs), and >6.0 METs is considered vigorous, expending 3.5 to 7.0 and >7.0 kcal/min, respectively. The reduction of sedentary time (leisure screen time) to <2 h/d is also recommended.33
management of parent engagement in physical activities, optimiz- 
ing physical activity behaviors in offspring. Encour- 
gement of sedentary behaviors, including advocacy and support for 
daily physical education in schools, are needed to improve levels of 
physical activity in US children and youth.36

Healthcare providers should assess physical activity and 
sedentary behaviors at every well-child visit.37–39 Although 
no valid and reliable instruments are currently available for 
assessment of physical activity in pediatric primary care 
settings, general topics for questions include the amount of 
time regularly spent walking, bicycling, and in outdoor play; 
use of playgrounds, parks, and gymnasiums; and interactive 
play/games with other children and adolescents. Time spent 
participating in age-appropriate organized sports should also 
be addressed, along with time spent in school or day-care 
physical education that includes a minimum of 30 minutes of 
coordinated large-muscle exercise (for children ≥2 years of age). 
Sedentary behaviors, including the number of hours per 
day spent in leisure screen time such as television viewing 
and computer gaming, should also be assessed.

Primary healthcare providers should provide age-
appropriate suggestions (that consider the child and family’s 
resources and preferences) for increasing physical activity 
and limiting sedentary behaviors in children and youth. For 
example, for families with children <5 years of age, recom-
mendations would include unlimited active play time in a safe 
supportive environment, family activity at least once a week, 
and limitation of screen time to <2 h/d. Screen time should 
be zero from birth to 2 years of age. In addition, television 
should not be permitted in the child’s bedroom. Beginning 
early in life and extending through adolescence, parental role 
modeling of physical activity behaviors is important in 
promoting physical activity behaviors in offspring. Encour-
gagement of parent engagement in physical activities, opti-
mal with the children, is advised. During childhood and 
adolescence, 1 h/d of MVPA and vigorous-intensity activity on 
least 3 d/wk and <2 h/d of sedentary activity are recom-
mended, along with matching physical activity recommenda-
tions with the child’s energy intake. Theory-based age-
appropriate behavioral change strategies should be included as 
part of individual and family counseling designed to increase 
levels of physical activity and to decrease sedentary behaviors.

Although additional research is needed to determine the most 
efficient and effective methods for implementation in pediatric 
primary care settings, available evidence points to the promise 
and potential of multicomponent, tailored interventions that 
incorporate principles of behavior change and are delivered by 
multidisciplinary teams.36,40

Obesity

Childhood obesity has reached epidemic proportions in the 
United States, with an estimated 17% of children and adoles-
cents being obese.41 Obesity prevalence is estimated at 10.4% 
among preschool children, 19.6% among 6 to 11 year olds, and 
18.1% among adolescents 12 to 19 years of age.41 Among 
infants and toddlers, ≈10% were above the 95th percentile of the 
weight-for-recumbent-length growth charts.41 Mexican American 
males and non-Hispanic black girls have a higher prev-
lence of obesity compared with non-Hispanic whites (29.2%, 
26.8%, and 14.5%, respectively). Higher obesity prevalence was 
reported in the National Health and Nutrition Examination 
Survey (NHANES) in children of low socioeconomic status 
(<130% of poverty level; boys, 21.1%; girls, 19.3%) compared 
with those of high socioeconomic status (>350% of poverty 
level; boys, 11.9%; girls, 12%).32

Obesity in children and adolescents is determined from 
BMI (weight in kilograms divided by height in meters 
squared) and the corresponding BMI-for-age percentile on a 
Centers for Disease Control BMI-for-age growth chart (http:// 
apps.nccd.cdc.gov/dnpabmi/Calculator.aspx); obesity is de-
efined as BMI ≥95th percentile and overweight as BMI ≥85th 
to <95th percentile.43 Obesity in childhood and adolescence is 
associated with numerous adverse health outcomes. Car-
diovascular risk factors such as hypertension, type 2 diabetes 
mejllitus, metabolic syndrome, sleep apnea, left ventricular 
hypertrophy,44,45 and abnormal lipid profiles (eg, high triglyc-
erides, low HDL-C) are higher in obese than in normal-weight 
youth.36,46 For example, obese girls had a 6-fold higher prev-
ance and a 2- to 3-fold higher incidence of hyperten-
sion compared with their normal-weight counterparts.47 Obes-
ity in childhood and adolescence substantially increases the 
risk of adult obesity.58,49 Obese 2- to 5-year-olds had >4 times 
the likelihood of becoming obese adults compared with 
normal-weight children.49 Eighty percent of children who 
were overweight at 10 to 15 years of age were obese adults at 
25 years of age.48 Twenty five percent of obese adults were 
overweight as children.48 Obesity in childhood has been 
associated with increased arterial stiffness and CIMT and 
increased risk of coronary heart disease in adult life.7,9,50,51

Prevention of obesity in childhood and adolescence is the 
mainstay for CVD risk reduction. An improvement in weight 
status and a decrease in adiposity are associated with a 
decline in blood pressure and an improvement of blood lipids 
(ie, TC, HDL-C, and triglycerides), insulin resistance, and 
inflammatory markers (see Table II in the online-only Data 
Supplement).36

A suggested approach to overweight and obesity manage-
ment in children and adolescents has been published.40 All 
children, regardless of their weight status, should have their 
weight and height measured and BMI calculated at every visit 
with the healthcare provider. The BMI percentile and, for 
infants and children, percentile for weight for recumbent 
height can then be determined. Parental obesity, family 
medical history, BMI trajectory, and CVD risk factors (eg, 
diabetes mellitus) are considered in the management of 
weight and in CVD risk reduction. For healthy-weight chil-
dren and adolescents (BMI, 5th–84th percentile), the goal is 
to prevent excess weight gain through lifestyle modifications
Table 2. Components of a Staged Approach to Weight Management for Children and Adolescents

<table>
<thead>
<tr>
<th>Stage</th>
<th>Components</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Prevention plus (&quot;plus&quot; means providers must spend more time and increase intensity of the recommendation and follow-up 3–6 mo beyond usual care)</td>
<td>≥5 servings of fruits and vegetables daily; ≥2 h of screen time daily; no television in child’s bedroom; no sugar-sweetened beverages; ≥60 min of physical activity daily; (graded) family-based approach to lifestyle changes; acknowledge cultural differences</td>
</tr>
<tr>
<td>2. Structured weight management (SWM)</td>
<td>Prevention plus; family-based and balanced macronutrient diet emphasizing small amounts of energy-dense foods; high-quality, nutrient-rich diet to prevent muscle loss; controlled portion sizes; reduced television and screen time; self-monitoring of diet and physical activity; medical screening</td>
</tr>
<tr>
<td>3. Comprehensive multidisciplinary intervention</td>
<td>Prevention and structured weight management but more frequent patient-provider contact, more active use of behavioral strategies, more formal monitoring, and feedback to improve adherence; multidisciplinary teams; strong parental involvement for children &lt;12 y of age; assessment of diet, physical activity, and body fat at specified intervals; food monitoring; structured dietary and physical activity intervention to improve diet quality and result in weight loss</td>
</tr>
<tr>
<td>4. Tertiary care center</td>
<td>Continue diet and activity counseling in stages 2 and 3 plus meal replacement, very low-energy diet, medication, and/or surgery</td>
</tr>
</tbody>
</table>


that include eliminating the intake of sugar-sweetened beverages,13 increasing the intake of fruits and vegetables to ≥5 servings daily, limiting television and electronic media use to ≤2 h/d, and participating in at least 60 minutes of MPVA daily (prevention stage).

Table 2 describes the components of the staged approach to weight management, and Table 3 presents a suggested staged approach to weight management and healthy weight goals for children and adolescents by age and BMI percentiles. For children and adolescents who are overweight (85th–94th percentile) or obese (≥95th percentile), a staged lifestyle behavioral approach of increasing intensity plus a structured weight management protocol and/or a comprehensive and multidisciplinary weight management protocol consisting of supervised counseling by a trained physician, nurse practitioner, or a registered trained dietitian are recommended.40 The management plan includes a low-energy-dense and balanced diet, supervised physical activity of at least 60 min/d, ≤1 h/d of screen time, and increased self-monitoring of dietary and physical activity behaviors.40

For overweight and obese youth, the goal is to improve BMI to less than the 85th percentile (Table 3). Lower energy (caloric) intake is strongly associated with reduced adiposity in children,13 and caloric expenditure must exceed intake to achieve weight loss. Referral of severely obese children and those with obesity-related comorbidities to a pediatric weight management program is highly recommended. The 4-stage intervention and treatment modality for overweight and obese youth has been endorsed by many organizations, including the American Academy of Family Physicians and the American Academy of Pediatrics (AAP).

Table II in the online-only Data Supplement presents examples of obesity prevention and nutrition interventions that were reported in the literature from 2000 to 2010. Interventions that improved BMI were multicomponent (included dietary and physical activity improvements)52 and multidisciplinary (included pediatricians or primary care physicians and registered dietitians or nurses).53 Interventions that resulted in weight loss or reduction in excess weight gain were often family based and included behavioral therapy. Intervention duration varied from 4 weeks to 4 years. The NHLBI Working Group on Childhood Obesity Prevention and Treatment recently provided research recommendations to prevent and treat obesity in children to reduce future CVD risk.54

Blood Pressure

Elevated blood pressure is determined for children when measured blood pressure exceeds certain percentile values based on studies in normal populations. To determine the blood pressure percentile, it is important to take into account age, sex, and height, variables that are normally associated with blood pressure level. The Fourth Report on Blood Pressure in Children from the NHLBI has established levels of blood pressure that should trigger clinical action.55 Prehypertension is defined as blood pressure between the 90th and 95th percentiles for age, sex, and height or 120/80 mm Hg (the level for prehypertension in adults), whichever is lower. When prehypertension is identified, the recommended clinical approach is lifestyle intervention to improve BMI when obesity is present, to lower dietary sodium, and to increase the level of physical activity. Blood pressure elevation or hypertension is defined by systolic or diastolic blood pressure above the 95th percentile on a persistent basis. Persistence is defined by elevation on 3 consecutive occasions. Stage 1 hypertension is present when blood pressure is above the 95th percentile but below the 99th percentile plus 5 mm Hg (~12 mm Hg above the 95th percentile). Stage 2 hypertension is present when blood pressure is above the 99th percentile plus 5 mm Hg.55

The management approach to hypertension is presented in the Figure. For stage 1 hypertension, it is important to evaluate left ventricular mass to determine whether left ventricular hypertrophy is present. Left ventricular hypertrophy is the most useful marker of hypertensive target organ abnormality and, when present, is an indication that more aggressive treatment is indicated. The initial treatment for stage 1 hypertension is management of lifestyle and improvement of BMI as for prehypertension. However, if blood pressure elevation persists despite lifestyle change or if target organ damage is present, then pharmacological intervention may be indicated. When stage 2 hypertension is present, this
may signal the presence of a secondary form of hypertension. A workup for underlying causes of hypertension should be instituted on the basis of the presence of suspicious history or abnormal physical examination. Stage 2 hypertension may also require earlier intervention with pharmacological therapy.55

The recommended approach to blood pressure elevation in children and adolescents comes from decades of epidemiological and clinical research on children and adults. First, hypertension has been established as a very potent risk factor for cerebrovascular disease and coronary artery disease in adults.2,56 Treatment of high blood pressure has also been shown to directly lower the risk of CVD in adults.56,57 This has led to major initiatives to ensure that hypertension is recognized and treated and that appropriate blood pressure is achieved in adults.58 Although levels of awareness and

Table 3. Suggested Staged Approach to Weight Management and Healthy Weight Goals for Children and Adolescents

<table>
<thead>
<tr>
<th>Age Range, y</th>
<th>BMI Range (Percentile or Absolute Value)</th>
<th>Staged Approach</th>
<th>Healthy Weight Goals</th>
</tr>
</thead>
<tbody>
<tr>
<td>2–5</td>
<td>5th–84th</td>
<td>Prevention stage; foster healthy lifestyle in all children; follow national recommendations for food consumption and physical activity</td>
<td>Normal weight; continue to maintain weight</td>
</tr>
<tr>
<td></td>
<td>85th–94th</td>
<td>PP; SWM after 3–6 mo of no weight change or parental obesity</td>
<td>Weight maintenance until BMI &lt;85th percentile or slowing of weight gain as indicated by downward deflection in BMI curve</td>
</tr>
<tr>
<td></td>
<td>≥95th</td>
<td>PP; SWM after 3–6 mo of no improvement on PP</td>
<td>Weight maintenance until BMI &lt;85th percentile or slowing of rate of weight gain</td>
</tr>
<tr>
<td></td>
<td>≥99th or &gt;21 kg/m²</td>
<td>PP; SWM after 3–6 mo of no improvement on SWM, comorbidity, or family history of obesity</td>
<td>Gradual, carefully monitored weight loss of no more than 1 lb/mo until BMI is &lt;85th percentile</td>
</tr>
<tr>
<td>6–11</td>
<td>5th–84th</td>
<td>Prevention stage; prevention; foster healthy lifestyle in all children; follow national recommendations for food consumption and physical activity</td>
<td>Normal weight; continue to maintain normal weight</td>
</tr>
<tr>
<td></td>
<td>85th–94th</td>
<td>PP; SWM after 3–6 mo if increasing BMI percentile or medical condition persists</td>
<td>Weight maintenance until BMI is &lt;85th percentile or slowing of weight gain as indicated by downward deflection in BMI curve</td>
</tr>
<tr>
<td></td>
<td>95th–98th</td>
<td>PP; SWM depending on responses, age, health risks, and motivation; comprehensive multidisciplinary intervention if no improvement on SWM</td>
<td>Weight maintenance until BMI is &lt;85th percentile or carefully monitored weight loss of no more than 1 lb/mo until &lt;85th percentile</td>
</tr>
<tr>
<td></td>
<td>≥99th</td>
<td>PP; SWM depending on age, response to treatment, health risks, and motivation; advance to comprehensive multidisciplinary intervention after 3–6 mo on SWM if comorbidity persists; refer to tertiary care center for evaluation</td>
<td>Weight loss not to exceed an average of 2 lb/wk (0.9 kg/wk) until &lt;85th percentile</td>
</tr>
<tr>
<td>12–18</td>
<td>5th–84th</td>
<td>Prevention stage; foster healthy lifestyle in all children; follow national recommendations for food consumption and physical activity</td>
<td>Normal weight; continue to maintain normal weight</td>
</tr>
<tr>
<td></td>
<td>85th–94th</td>
<td>PP; SWM after 3–6 mo of PP if BMI percentile or medical condition persists</td>
<td>Weight maintenance until BMI is &lt;85th percentile or slowing of weight gain as indicated by downward deflection in BMI curve</td>
</tr>
<tr>
<td></td>
<td>95th–98th</td>
<td>PP or SWM depending on age, degree of obesity, health risks, and motivation; advance to intensive intervention depending on response to treatment, age, health risks, and motivation</td>
<td>Weight loss until BMI is &lt;85th percentile; no more than 2 lb/wk; if greater weight loss is noted, monitor patient for causes of weight loss &gt;=2 lb/wk</td>
</tr>
<tr>
<td></td>
<td>≥99th</td>
<td>PP; SWM depending on age, response for treatment, degree of obesity, health risks, and motivation; advance from SWM to comprehensive multidisciplinary intervention stage after 3–6 mo with comorbidity and no improvement; refer to tertiary care center for evaluation</td>
<td>Weight loss not to exceed an average of 2 lb/wk until &lt;85th percentile</td>
</tr>
</tbody>
</table>


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effective treatment for adults are not optimum, they have improved steadily over time.58

Elevated blood pressure levels are known to track over time. Thus, an adolescent with elevated blood pressure is much more likely to become an adult with high blood pressure than an adolescent with normal blood pressure.59,60 Factors that increase the likelihood of blood pressure remaining high are excess weight gain over time and initiation of cigarette smoking.60 Other factors that may increase the persistence of high blood pressure but have been less well studied are low levels of physical activity and dietary patterns that result in elevated sodium. The use of oral contraceptives in adolescent girls and young women may also contribute.60

Unfortunately, the prevalence of high blood pressure has been increasing in children and adolescents.61,62 This is due in part to the epidemic of obesity in this population, but it appears that there are likely to be other, yet-to-be-discovered factors responsible for this increase.63 Din-Dzietham et al64 used national survey data from 1963 to 2002 to evaluate trends in hypertension for 8- to 17-year-old children and adolescents. They found that both prehypertension and hypertension are increasing in children and adolescents. They reported that an increase in abdominal obesity over time may be more important than overall obesity in relation to blood pressure elevation. There were also differences by ethnic group. Blacks and Mexican Americans had a greater prevalence of prehypertension and hypertension than whites. Male subjects had a higher prevalence of hypertension than female subjects.

McNiece et al62 also evaluated the prevalence of hypertension in adolescents. After 3 screenings, they found that 15.7% of adolescents had prehypertension and 3.2% had hypertension, including 2.6% with stage 1 hypertension and 0.6% with stage 2 hypertension. The only correlate of hypertension was obesity, whereas greater BMI, male sex, and black race were associated with prehypertension.

These results are quite important from a clinical perspective in that they demonstrate that primary care providers can expect to see prehypertension and even hypertension relatively commonly in children and adolescents. Providers should be ready to identify and treat hypertension in this age group. Unfortunately, studies have shown that appropriate identification and treatment of hypertension is not occurring at an optimum level for pediatric patients. Hansen et al64 examined data on >14 000 children via medical records with at least 3 blood pressure measurements. Of those, 3.6% met the criteria for hypertension. However, only 26% of those were actually diagnosed with hypertension. Of all of the children with at least 1 blood pressure measurement documenting elevated blood pressure, only 9% had a subsequent blood pressure recorded to determine whether the elevation was persistent. This means that measurement of blood pressure, appropriate interpretation, and clinical intervention must be applied more systematically in pediatric primary care.

Blood pressure elevation in children and adolescents has been demonstrated to be associated with early atherosclerosis in autopsy studies5,5 and has been shown to be related to increased left ventricular mass. For example, Daniels et al65 showed that 8% of children and adolescents with hypertension already had left ventricular mass index elevated to a level associated with a 4-fold increased risk of CVD in adults with hypertension. Lande et al66 have also described problems with neurocognitive function in children with hypertension. These results underscore the clinical importance of blood pressure elevation in children and adolescents. It is clear that hypertension is already having an impact on the heart and vascu-
lature even at an early age. Initial treatment should focus on weight loss if obesity is present and other nonpharmacological approaches. Couch et al have shown that a Dietary Approaches to Stop Hypertension–type diet can be successful in reducing blood pressure even without a reduction in BMI in adolescents. For those patients in whom lifestyle change is not effective, pharmacological intervention may be needed. There is now substantial clinical trial evidence that pharmacological agents from a variety of classes can be safe and effective in lowering elevated blood pressure in children and adolescents. Unfortunately, no studies have documented the comparative effectiveness of 1 agent or 1 class of agents over another. This means that several classes, including diuretics, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, calcium channel blockers, and β-adrenergic blockers, could be chosen as first-line agents. Preliminary data demonstrate that pharmacological treatment can be effective in lowering left ventricular mass and blood pressure in children.

It is important to include blood pressure measurement as part of a cardiovascular risk assessment for children. Blood pressure should be a routine part of health-maintenance visits starting at 3 years of age. This should allow better lifetime control of blood pressure and lower risk for the development of CVD.

Lipids/Lipoproteins

Over the past 40 years, data have accumulated linking adverse levels and patterns of lipids and lipoproteins to the initiation and progression of the atherosclerotic process in children and adolescents. However, no multidisciplinary, longitudinal population-based studies have been conducted linking absolute levels of lipids and lipoproteins in childhood to incident CVD in adult life, and no randomized, controlled trials have demonstrated that reducing atherogenic lipids and lipoproteins in early life prevents CVD in adulthood. Several lines of evidence, however, clearly support the need for primary prevention, including reduction of adverse levels of lipids and lipoproteins beginning early in life. This evidence includes the natural history observed for individuals with genetic dyslipidemias, such as homozygous familial hypercholesterolemia, in whom LDL-C levels are quite high and there is substantial increased risk of evolving atherosclerotic CVD.

Postmortem studies conducted as part of the Bogalusa Heart Study and the PDAY Study have demonstrated that early atherosclerotic lesions (fatty streaks) and advanced lesions (fibrous plaques) are significantly related to elevations in TC, LDL-C, and non–HDL-C and low levels of HDL-C, as well as to the presence and intensity of other potentially modifiable risk factors, including obesity, hypertension, and cigarette smoking. In subsequent analyses using a risk score derived from these results, non–HDL-C was found to be the major correlate of coronary atherosclerosis, with a 30 mg/dL increase in non-HDL-C equivalent to 2 years of vascular aging. Noninvasive imaging studies have also been used to examine the association of risk factors for CVD and vascular structure and function in childhood and adolescence and atherosclerosis in young adult life. In the Musscatine Study, a longitudinal, observational study of CVD risk factors in children and youth, carotid ultrasound in adults (33–42 years of age) indicated that CIMT was positively associated with levels of TC and BMI measured in childhood. Similar results were observed in the Bogalusa Heart Study, in which childhood LDL-C and BMI were found to predict increased CIMT in adulthood. In the Young Finns Study, a population-based prospective cohort study, associations between risk factor exposures in adolescence, including LDL-C, BMI, cigarette smoking, and systolic blood pressure, predicted CIMT in adulthood independently of adult risk factor levels. Results of other imaging studies designed to assess subclinical atherosclerosis reaffirm these observations, indicating that abnormal levels of atherogenic lipids and lipoproteins in childhood and adolescence are associated with endothelial dysfunction, coronary artery calcium, and increased CIMT.

Taken together, the results of pathology and imaging studies indicate that adverse levels of lipids and lipoproteins (and other potentially modifiable established risk factors) are associated with accelerated atherosclerotic processes in childhood and adulthood and point to the importance of primordial and primary prevention beginning in early life.

Data from the NHANES for 1999 to 2006 indicate that the prevalence of abnormal lipid levels among all youths 12 to 19 years of age was 20.3%. Abnormal serum lipid levels were classified on the basis of cutoff points suggested in AAP and AHA guidelines: high LDL-C, ≥130 mg/dL; low HDL-C, ≤35 mg/dL; and high triglycerides, ≥150 mg/dL. Of note, the prevalence varied by BMI: 14.2% of normal-weight, 22.3% of overweight, and 42.9% of obese youths had at least 1 abnormal lipid level.

Tracking of lipids and lipoproteins, particularly TC and LDL-C, has also been documented in children from diverse racial/ethnic groups and is particularly evident in the upper and lower extremes of the distribution. In the Musscatine Study, for example, 75% of children who were 5 to 18 years of age at baseline and had TC levels greater than the 90th percentile had elevated TC (≥200 mg/dL) at 20 to 25 years of age. The Bogalusa Heart Study reported that 70% of children with elevated TC in childhood persisted with elevated levels in adulthood. Tracking is relevant to primary prevention because of the potential for identifying children at risk for CVD early in life.

Data from the Lipid Research Clinics indicate that serum lipids and lipoproteins increase throughout the first 2 years of life and approximate levels in young adults by 2 years of age. These observations of primarily non-Hispanic whites and blacks contributed to the first guidelines issued by the NHLBI National Cholesterol Education Panel (NCEP) recommending selective screening for high-risk children after 2 years of age when lipids and lipoproteins begin to track. Data from the Lipid Research Clinics and NHANES indicate that puberty/maturation has an important impact on levels of lipids and lipoproteins. In addition, patterns of change during this developmental period vary by sex and race/ethnicity. For example, in the 1988 to 1994 NHANES, at 4 to 19 years of age, the mean cholesterol was 165 mg/dL; at 9 to 11 years of age, however, age-specific values for mean
cholesterol peaked at 171 mg/dL.83 Female subjects had higher TC and LDL-C than male subjects and, after pubertal development, had higher HDL-C than male subjects. Black children had higher HDL-C and lower triglyceride concentrations than their non-Hispanic white and Hispanic counterparts.83

Current recommendations for management of dyslipidemia in children and youth issued by the AAP86 and AHA84 reflect accumulating evidence. There is currently a lack of consensus within the pediatric healthcare and research communities on targeted versus universal screening. The AAP recommends a targeted approach to screening. Specifically, this approach recommends screening children (>2 years of age) who have a family history of premature CVD or who have parents with dyslipidemia. Screening is also recommended for children for whom family history is unknown or children who present with other CVD risk factors, including hypertension, obesity, and diabetes mellitus. Since the NCEP recommended targeted screening, research has shown that the approach has limitations for capturing at-risk children.85–90 Studies of the effectiveness of the targeted approach found that 35% to 46% of children and youth had cholesterol levels measured on the basis of positive family history or elevated cholesterol levels.85,89,91 Other research has shown that 30% to 60% of children and adolescents with elevated cholesterol levels will be missed by this approach.84,90,92 Clearly, with the increase in prevalence of childhood obesity and its comorbidities, the number of children who qualify for screening has increased.76

Cut points recommended by NCEP82 and AAP76 used to identify children and adolescents with abnormal lipid and lipoprotein levels are presented in Table 4. These cut points are recommended for children 2 to 18 years of age. The AHA has recommended that triglyceride levels of ≥150 mg/dL and HDL-C levels of ≤35 mg/dL be considered abnormal for children and adolescents.39 A single cut point for all children and youth may be limited by differences in age, sexual maturation, and race/ethnicity.93 Table III in the online-only Data Supplement presents lipid and lipoprotein distributions in children and youth 4 to 19 years of age.83 These data were collected in 1981 before the increase in prevalence of obesity with standardized protocols and the use of plasma specimens. As suggested by the AAP, these tables and percentiles, stratified by age, are applicable in clinical practice.

Optimizing cholesterol levels and managing dyslipidemia in children and youth includes both a population approach and an individual/high-risk approach.76,84 The population approach focuses on promoting optimal lipid levels in all children and youth and emphasizes health behaviors, a cornerstone of cardiovascular health promotion and risk reduction in childhood.36,94 Although dietary restrictions are generally not recommended for children during the first 2 years of life, research has demonstrated the safety and efficacy of a diet consisting of total fat of <30% of the caloric intake, saturated fat of <10% of the intake, and cholesterol intake of <200 mg/dL per day, with 1.5% cow’s milk after 12 months of age.95 More recent data from this study (Special Turku Risk Intervention Program) indicate beneficial effects in boys (lowering of LDL-C) and decreased prevalence of obesity in girls compared with age-matched control subjects.95 The updated AHA dietary recommendations for children and youth (≥2 years of age) emphasize a balanced caloric intake with sufficient physical activity (60 min/d) to achieve a normal weight and increased consumption of fruits, vegetables, whole grains, fish, and low-fat dairy products.96 In addition, a reduction of daily intake of trans fatty acids, which are known to increase LDL-C levels, to <1% of total intake is recommended.96 Recently, the AHA published strategies designed to assist healthcare providers in implementing these guidelines with children and families.97

The individual approach focuses on children and adolescents identified as being at risk because of family history of CVD or because they present with elevated levels of LDL-C or other major risk factors. Management for these patients initially involves therapeutic lifestyle change, with an emphasis on an adequate trial of dietary therapy (and increased physical activity). Research has shown that with good adherence to a saturated fat- and cholesterol-restricted diet (<7% and <200 mg/d, respectively), LDL-C levels can be decreased 10% to 15% from baseline.81 Children with genetic dyslipidemias may present with baseline levels that cannot be reduced to suggested goals. Interindividual differences in LDL-C levels in response to reduced intakes of saturated fat and cholesterol are well documented and attributable to a number of factors. Thus, after an adequate trial of therapeutic dietary lifestyle change, some children and adolescents will be candidates for pharmacological intervention. Other nonpharmacological approaches to reducing adverse LDL-C have also been recommended for children and adolescents, including additional fiber (calculated as the child’s age plus 5 g/d up to a dose of 20 g/d at 15 years of age).84 Results have not been consistent across studies, but some show modest (~7%) reductions of LDL-C.84 Plant stanols and sterols, currently added to several food products such as margarines, orange juice, and cereal bars, have been shown to reduce cholesterol concentrations by ~5% to 10% with minimal adverse effects.84

The AAP currently recommends that pharmacological interventions be considered in children ≥8 years of age if LDL-C levels persist at ≥190 mg/dL (with no other risk factors present) and for children with levels that persist at ≥160 mg/dL and have other risk factors such as obesity, hypertension, positive family history of CVD, and/or cigarette smoking. For children with diabetes mellitus, the AAP recommends pharmacological intervention for children whose LDL-C levels are ≥130 mg/dL (approximately the 95th percentile).76 Medications such as statins, bile acid-binding resins,98 and cholesterol absorption inhibitors are
currently available for treatment of dyslipidemia in children and adolescents.84 Therapeutic lifestyle change remains a cornerstone of CVD risk reduction in childhood and should continue along with pharmacological intervention in children and adolescents.

Smoking/Tobacco Exposure

Tobacco use continues to be the single leading preventable cause of death in the United States and is responsible for ≈4 million annual deaths worldwide.99,100 Because >80% of established adult smokers begin smoking before 18 years of age101 and in view of the unequivocal evidence linking tobacco use, particularly cigarette use, and adverse health and developmental outcomes,99,102 prevention of smoking initiation and cessation interventions are essential components of cardiovascular health promotion and risk reduction for children and adolescents.37,39,103

Recent population-based data on tobacco use among middle and high school students in the United States prompted a call to action for pediatric healthcare providers and public health advocates.104 Specifically, data from the Centers for Disease Control and Prevention National Youth Tobacco Survey indicated that from 2000 to 2009 the prevalence of tobacco use among middle school students declined (15.1%–8.2%), as did cigarette use (11.0%–5.2%) and cigarette smoking experimentation (29.8%–15.0%). Similar trends were observed for high school students (tobacco use, 34.5%–23.9%; cigarette use, 28.0%–17.2%; smoking experimentation, 39.4%–30.1%). No change was observed, however, in susceptibility to initiate cigarette smoking as measured by self-report of openness to trying cigarette smoking.104,105 In view of 2006 to 2009 prevalence and susceptibility data, both individual/clinical and public health strategies must continue unabated to reduce tobacco use. Restrictions on advertising, promotion, and availability of tobacco products to children and adolescents should be combined with implementation of evidence-based, community-wide, comprehensive tobacco control policies.106,107

The AAP Committee on Environmental Health called attention to the health hazards of environmental tobacco smoke in 1997 with evidence-based recommendations for pediatricians and child health care providers.108 Environmental tobacco smoke in childhood has been linked with acute and chronic respiratory conditions, middle ear effusions, risk factors for and CVD processes (ie, dyslipidemia, impaired endothelial function), and increased risk for selected cancers in adulthood.102 The adverse effects of intrauterine exposure to tobacco smoke on fetal and childhood development and overall health have also been well documented and include low birth weight for gestational age and associations with selected behavioral and cognitive-information processing problems.102

Smoking prevention and cessation interventions are effective in adults.103,109,110 Theory-based behavioral interventions are central to the prevention of initiation and smoking cessation and are generally applicable to children and youth, although the evidence base is not as substantial. Office-based counseling directed at children and youth for the prevention or cessation of tobacco use has been a cornerstone of cardiovascular health promotion, risk reduction, and general pediatric preventive care.37,39

Christakis and colleagues110 conducted a systematic review of smoking prevention interventions delivered by healthcare providers; 1 study showed a significant positive effect on the prevention of smoking initiation. A more recent systematic review of family-based smoking prevention interventions that included 14 randomized, controlled trials111 showed that 4 of the 9 randomized controlled trials that tested a multicomponent family intervention and included a control group demonstrated significant positive effects, whereas 1 of the 5 that tested family intervention versus a school-based intervention had a significant positive outcome. Overall, a significantly lower rate of smoking initiation was achieved in ≈40% of interventions. The quality of the implementation of the intervention and the amount of training of the implementer were related to positive outcomes; however, the number of intervention sessions was not more recently, Pbert and colleagues112 completed a randomized, controlled trial designed to examine the effect of a pediatric practice-based smoking prevention and cessation intervention for adolescents. The provider- and peer-delivered intervention was based on the 5A model (ask, advise, assess, assist, arrange) and consisted of brief counseling by the pediatric provider, followed by 1 visit and 4 telephone calls by older peer counselors. Results indicated that compared with usual care, nonsmokers who received the provider- and peer-delivered intervention were significantly more likely to self-report having remained abstinent at 6- and 12-month follow-ups. Smokers who received the intervention, however, were more likely to self-report having quit at the 6-month but not the 12-month follow-up. Methodological limitations notwithstanding, including self-report of abstinence, the results support the feasibility and short-term efficacy of theory-based multicomponent interventions for the prevention of smoking initiation and smoking cessation in primary care pediatric office settings. The results also underscore the need for prevention of initiation of smoking.112 Adding to the need for emphasis on the prevention of smoking initiation in pediatric primary care settings are the conflicting results reported in recent systematic reviews of the benefits of office-based counseling on smoking cessation.113 This Cochrane review found that interventions that used pharmacological aids were not effective; however, those that incorporated behavioral change strategies, including motivational interviewing and stages of change, achieved significant positive results at the 6-month follow-up. Additional research on the efficacy and effectiveness of combining behavioral change strategies and pharmacotherapy for long-term abstinence in adolescent smokers is warranted.

Evidence supports the need for both individual/clinical- and population-based approaches to the prevention of smoking initiation and interventions for smoking cessation for children and youth. Current recommendations for pediatric healthcare providers emphasize a developmental approach with assessment of smoking status at every well-child visit.37,39 Parents and guardians are advised to maintain a smoke-free home environment and to avoid exposure to secondhand smoke in other environments. For infants and
Table 5. Schedule for Integrated Cardiovascular Promotion in Children

<table>
<thead>
<tr>
<th>Family History</th>
<th>Cholesterol</th>
<th>Obesity</th>
<th>Blood Pressure</th>
<th>Diet</th>
<th>Physical Activity</th>
<th>Smoking</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, 0–2 y</td>
<td>Early heart disease before 55 y of age</td>
<td>Parent cholesterol screening</td>
<td>Diet history</td>
<td>Parent physical activity</td>
<td>Parental/household smoking</td>
<td></td>
</tr>
<tr>
<td>Parent total cholesterol ≥240 mg/dL</td>
<td>Parent obesity</td>
<td>Early foods influence future food preferences</td>
<td>No television viewing for infant</td>
<td>If yes, counsel to quit; referral to smoking cessation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age, 2–5 y</td>
<td>Update family history</td>
<td>Fasting lipids screening</td>
<td>Plot height, weight, and BMI (kg/m²) on growth charts</td>
<td>Start routine BP measures at 3 y of age (determine if &gt;90th or 95th percentile for sex, age, and height)</td>
<td>Diet history</td>
<td>Encourage active child-parent play</td>
</tr>
<tr>
<td>Early heart disease before 55 y of age</td>
<td>Based on family history</td>
<td>BMI percentiles</td>
<td>Low saturated-fat diet, including 1% or nonfat milk</td>
<td>Limit sedentary behaviors, including television viewing, to &lt;2 h/d</td>
<td>If yes, counsel to quit; referral to smoking cessation</td>
<td></td>
</tr>
<tr>
<td>Parent total cholesterol ≥240 mg/dL</td>
<td></td>
<td>Moderate salt intake</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age, 6–10 y</td>
<td>Update family history</td>
<td>Fasting lipids screening</td>
<td>Plot height, weight, and BMI (kg/m²) on growth charts</td>
<td>BP measures</td>
<td>Diet history</td>
<td>Physical activity history</td>
</tr>
<tr>
<td>Early heart disease before 55 y of age</td>
<td>Based on family history</td>
<td>BMI percentiles</td>
<td>BP percentiles</td>
<td>Low saturated-fat diet, including 1% or nonfat milk</td>
<td>Lifestyle and family activities</td>
<td>If yes, counsel to quit; referral to smoking cessation</td>
</tr>
<tr>
<td>Parent total cholesterol ≥240 mg/dL</td>
<td></td>
<td>Moderate salt intake</td>
<td>Reduce sedentary behaviors such as television viewing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age, &gt;10 y</td>
<td>Update family history</td>
<td>Fasting lipids screening</td>
<td>Plot height, weight, and BMI (kg/m²) on growth charts</td>
<td>BP measures</td>
<td>Diet history</td>
<td>Physical activity history</td>
</tr>
<tr>
<td>Early heart disease before 55 y of age</td>
<td>Based on family history</td>
<td>BMI percentiles</td>
<td>BP percentiles</td>
<td>Low saturated-fat diet, including 1% or nonfat milk</td>
<td>Lifestyle and family activities</td>
<td>If yes, counsel to quit; referral to smoking cessation</td>
</tr>
<tr>
<td>Parent total cholesterol ≥240 mg/dL</td>
<td></td>
<td>Moderate salt intake</td>
<td>Daily moderate to vigorous activity (60 min/d), reduce sedentary behaviors</td>
<td>Antismoking counseling for the child</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


young children, intervention is directed at parents/guardians and others in the child’s household who smoke. Parents/guardians who smoke should be advised of the immediate health risks to their children; motivated to quit through the use of behavioral prescriptions such as motivational interviewing, stages of change, and/or the 5A model; and referred to smoking cessation programs if appropriate. During the school-age years, assessment of smoking status with clear, firm and consistent messages about the importance of remaining smoke-free is advised. Five strategies for discussing smoking with children are suggested for pediatric healthcare providers, who are powerful role models for children and youth. First, encourage young children to actively avoid environmental smoke whenever possible. Second, emphasize the importance of not experimenting with smoking, “not even a puff.” Third, point out the harmful health consequences of smoking and the addictive habit-forming qualities of nicotine. Fourth, present information to counter the myths of media influences (ie, smoking is enjoyable). Fifth, 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.37

In counseling adolescents, a group most at risk for smoking initiation, pediatric healthcare providers are advised to assess personal smoking history at every nonurgent encounter and to provide clear, consistent messages about nonsmoking. For adolescent smokers, ongoing support and counseling either personally or through community-based smoking cessation programs will most likely yield positive outcomes. Resources such as quit-line numbers and community-based cessation programs and information on pharmacotherapy for cessation should be made readily available.37

Conclusion

It is clear that risk factors for atherosclerotic CVD can develop during childhood and adolescence. This result from both genetic and environmental factors. When risk factors develop at an early age, they are likely to track over time, maintaining a high-risk status. This tracking is reinforced by ongoing and new adverse health behaviors.

This means that the development of CVD has its origins in families and that approaches to prevention must be directed at the developing child and adolescent and the family environment. Pediatricians and family physicians should view the prevention of risk factor development (primordial prevention) and the development of atherosclerotic lesions (primary prevention) as an important responsibility. This article has outlined the main risk factors of concern. Healthcare providers should view this from an integrated perspective, meaning that each risk factor and the behaviors underlying it should be addressed through the use of a developmental approach at every health-maintenance visit.

Kavey et al19 have presented a blueprint for an integrated approach to pediatric primordial and primary prevention of atherosclerotic CVD. This approach is presented in Table 5. Using this approach should maximize opportunities for prevention. This improves the likelihood that children and adolescents will maintain low-risk status into young adulthood.

Prevention of atherosclerotic CVD is best achieved by maintaining cardiovascular health. Although this is challenging, it can be achieved by instituting and maintaining optimum health behaviors early in life and stressing improvement of the family environment as the most important strategy to achieve these goals.

Disclosures

Dr Daniels has served as a consultant/advisory board member for Merck, Shering-Plough. Dr Hayman has received research grants for Health Behaviors of African-American and European-American Lung Cancer Patients and Family Members (UMB-DF/HCC Comprehensive Cancer Partnership Program, NIH/NCl, 1U56CA118635-03, 11/01/2008-12/31/2010) and Improving Fitness in Children at Increased Cardiometabolic Risk: Piloting an Interactive Fitness Program (Harvard Catalyst/The Harvard Clinical and Translational Science Center, NIH #1 UL1RR 025758-02, 11/01/2009-6/30/2011). Dr Pratt reports no conflicts. The views expressed and positions taken in this document are not necessarily those of the National Heart, Lung, and Blood Institute.

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Reduction of Risk for CVD in Childhood

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Keywords: atherosclerosis ■ diet ■ hypertension ■ obesity
Reduction of Risk for Cardiovascular Disease in Children and Adolescents
Stephen R. Daniels, Charlotte A. Pratt and Laura L. Hayman

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Supplemental Table 1. Strong- and moderately-rated evidence linking diet and CVD risk:
Evidence from the 2010 Dietary Guidelines Advisory Committee

<table>
<thead>
<tr>
<th>Topic</th>
<th>Conclusion</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MACRONUTRIENTS AND ENERGY</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Is intake of dietary fat associated with adiposity in children?</td>
<td>Moderate evidence from prospective cohort studies suggests that increased intake of dietary fat is associated with greater adiposity in children. However, there were no studies conducted under isocaloric conditions.</td>
<td>Moderate</td>
</tr>
<tr>
<td>Is total energy intake associated with adiposity in children?</td>
<td>Moderately strong evidence from recent prospective cohort studies that identified plausible reports of energy intake, support a positive association between total energy (caloric) intake and adiposity in children.</td>
<td>Moderate-Strong</td>
</tr>
<tr>
<td>Is dietary energy density associated with adiposity in children?</td>
<td>Moderately strong evidence from methodologically rigorous longitudinal cohort studies of children and adolescents suggests that there is a positive association between dietary energy density and increased adiposity in children.</td>
<td>Moderate-Strong</td>
</tr>
<tr>
<td>Is intake of sugar-sweetened beverages associated with adiposity in children?</td>
<td>Strong evidence supports the conclusion that greater intake of sugar-sweetened beverages is associated with increased adiposity in children.</td>
<td>Strong</td>
</tr>
<tr>
<td><strong>MICRO NUTRIENTS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>What is the effect of a reduced sodium intake on blood pressure in children from birth to age 18 years?</td>
<td>A moderate body of evidence has documented that as sodium intake decreases, so does blood pressure in children, birth to 18 years of age.</td>
<td>Moderate</td>
</tr>
<tr>
<td>Is intake of calcium and/or dairy (milk and milk products) related to adiposity in children?</td>
<td>Moderate evidence suggests that there is no relationship between intake of calcium and/or dairy (milk and milk products) and adiposity in children and adolescents.</td>
<td>Moderate</td>
</tr>
<tr>
<td><strong>FOODS AND FOOD ENVIRONMENT</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>What is the relationship between the environment, body weight and fruit/vegetable consumption?</td>
<td>An emerging body of evidence has documented the impact of the food environment and select behaviors on body weight in both children and adults. Moderately strong evidence now indicates that the food environment is associated with dietary intake, especially less consumption of</td>
<td>Moderate</td>
</tr>
<tr>
<td>Question</td>
<td>Response</td>
<td>Strength</td>
</tr>
<tr>
<td>-------------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>----------</td>
</tr>
<tr>
<td>What is the relationship between eating out and body weight?</td>
<td>Strong and consistent evidence indicates that children and adults who eat fast food are at increased risk of weight gain, overweight and obesity. The strongest documented relationship between fast food and obesity is when one or more fast food meals are consumed per week. There is not enough evidence at this time to similarly evaluate eating out at other types of restaurants and risk of weight gain, overweight and obesity.</td>
<td>Strong</td>
</tr>
<tr>
<td>What is the relationship between breakfast and body weight?</td>
<td>Moderate evidence suggests that children who do not eat breakfast are at increased risk of overweight and obesity. The evidence is stronger for adolescents. There is inconsistent evidence that adults who skip breakfast are at increased risk for overweight and obesity.</td>
<td>Moderate</td>
</tr>
<tr>
<td>Is breakfast intake associated with achieving recommended nutrient intakes?</td>
<td>Moderate evidence supports a positive relationship between the behavior of breakfast consumption and intakes of certain nutrients in children, adolescents and adults.</td>
<td>Moderate</td>
</tr>
<tr>
<td>What is the relationship between portion size and body weight?</td>
<td>Strong evidence documents a positive relationship between portion size and body weight.</td>
<td>Strong</td>
</tr>
<tr>
<td>What is the relationship between screen time and body weight?</td>
<td>Strong and consistent evidence in both children and adults shows that screen time is directly associated with increased overweight and obesity. The strongest association is with television screen time.</td>
<td>Strong</td>
</tr>
</tbody>
</table>

### Supplemental Table 2. Examples of obesity prevention and treatment interventions in children and adolescents

<table>
<thead>
<tr>
<th>Reference</th>
<th>Population</th>
<th>Intervention</th>
<th>Outcomes (e.g., Diet, food pattern, nutrients/weight/BMI/blood pressure/blood lipids/glucose/sedentary behavior-TV viewing, physical activity)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diaz RG et al. Lifestyle intervention in primary care settings improves obesity parameters in Mexican youth. <em>J Am Diet Assoc.</em> 2010;110:285-90. (53)</td>
<td>Mexican youth, 9-17 years; RCT; 12 months, BMI or weight circumference &gt;90th percentile</td>
<td>Shape Down program adapted for Mexican youth with culturally appropriate topics. 12 weekly 2-hour sessions on improving self-esteem, emotions, communication, and lifestyle behaviors—nutrition, weight regulation, energy balance, physical activity. Dietitian advice and physician consult. Control condition: 10-15 consult with physician, encouraged 30 min PA, food guide pyramid suggestions.</td>
<td>Mean changes in body weight for the lifestyle group and the control group were -0.8 kg (-3.2, 1.5) vs. +5.6 kg (3, 8.2; P&lt;0.001). Mean changes in BMI were -1.8 (-2.6, -0.9) vs. +0.4 (-0.5, 1.3; P&lt;0.001), intervention vs. control, respectively. Significant differences in primary outcomes (weight -3.5 kg, P=0.02; BMI -1.2, P=0.03) in favor of the lifestyle group at 12 months.</td>
</tr>
<tr>
<td>Kalarchian MA et al. Family-based treatment of severe pediatric obesity: randomized controlled trial. <em>Pediatrics.</em> 2009;124:1060-8.</td>
<td>192 children 8-12 years old (mean +/- SD: 10.2 +/- 1.2 years). The average BMI percentile for age and gender was 99.18 (SD: 0.72)</td>
<td>Family-based intervention; 20 group meetings, 1 hr each, for 6 mo. Lifestyle coach, 6 group sessions and telephone calls. Intervention focused on Stop Light eating plan ranging from 1200 to 1800 kcal; strategies to increase PA, decreased sedentary hours, decreased emotional eating, goal setting. Control condition-2 nutrition consults. Offered intervention after 18 mo.</td>
<td>Significant decreases in child percent overweight, relative to usual care, at 6 months. Intervention vs. Usual Care - 7.58% vs. 0.66% decrease in child percent overweight at 6 months.</td>
</tr>
<tr>
<td>Vitola BE et al. Weight loss reduces liver fat 8 obese adolescents, BMI</td>
<td>Four-week behavioral therapy sessions; Healthy Habits weight loss program used to</td>
<td>5% stable weight loss for 4 weeks improved hepatic triglycerides by 61.6%, insulin</td>
<td></td>
</tr>
</tbody>
</table>

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<tbody>
<tr>
<td>&quot;Obeldicks” is 12-month outpatient physical activity, nutrition and behavior therapy intervention Psychological care for 3 mo intensive—nutrition course 1.5 hrs for 6 months, 30min/mo of psychological therapy with family, 3 mo of individual care. Physical activity 1/wk for 12 mo, ball games, jogging, trampoline jumping, and TV reduction. Food based dietary guidance- 15% pro, 55% CHO, 5% sugar, 30% fat.</td>
<td>Lifestyle intervention significantly reduced BMI-z score (mean -0.22; 95%CI -0.18 to -0.26), compared to control (mean +0.15; 95%CI +0.13 to +0.18), and metabolic syndrome prevalence (from 19% to 9%).</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>30 min physical activity daily, elimination of sweets and sweetened beverages, increase in whole grains. Low-fat dairy products and whole-grain bread were promoted and all sweets and sweetened drinks were eliminated in intervention schools.. Duration: 4 years, 8/2001 to 6/2005</td>
<td>The prevalence of overweight and obesity decreased by 3.2% (from 20.3 to 17.1) in intervention schools compared with an increase of 2.8% (from 16.1 to 18.9) in control schools (P&lt;0.05). A larger proportion of the children who were initially overweight reached normal weight in the intervention group (14%) compared with the control group (7.5%), P=0.017.</td>
</tr>
<tr>
<td>Study</td>
<td>Participants</td>
</tr>
<tr>
<td>--------------------------------------</td>
<td>-------------------------------------------------------------------------------</td>
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<tr>
<td>Nowicka P et al. Family weight school treatment: 1-year results in obese adolescents. <em>Int J Pediatr Obes</em>. 2008;3:141-7.</td>
<td>72 obese adolescents 12-19 years old. Referred by pediatrician and school nurses. Duration: 1 year.</td>
</tr>
<tr>
<td>Saelens BE et al. Efficacy of maintenance treatment approaches for childhood overweight: a randomized controlled trial. <em>JAMA</em>. 2007;298:1661-73.</td>
<td>204 healthy 7- to 12-year-olds, 20% to 100% above median body mass index (BMI) for age and sex, with at least 1 overweight parent.</td>
</tr>
<tr>
<td>Study</td>
<td>Participants</td>
</tr>
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<tr>
<td>Savoye M et al. Effects of a weight management program on body composition and metabolic parameters in overweight children: a controlled trial. <em>JAMA</em>. 2007;297:2697-704.</td>
<td>209 overweight children (body mass index [BMI] &gt;95th percentile for age and sex), ages 8 to 16 years of mixed ethnic groups.</td>
</tr>
<tr>
<td>Kalavainen MP et al. Clinical efficacy of group-based treatment of childhood obesity compared with routinely given individual counseling. <em>Int J Obes (Lond)</em>. 2007;31:1500-8.</td>
<td>70 obese children (weight for height 115-182%) aged 7-9 years.</td>
</tr>
</tbody>
</table>
**Supplemental Table 3. Lipid and Lipoprotein Distributions in Children and Adolescents, Aged 5-19 Years**

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5-9y</td>
<td>10-14y</td>
</tr>
<tr>
<td><strong>Total Cholesterol, mg/dL</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>50th percentile</td>
<td>153</td>
<td>161</td>
</tr>
<tr>
<td>75th percentile</td>
<td>168</td>
<td>173</td>
</tr>
<tr>
<td>90th percentile</td>
<td>183</td>
<td>191</td>
</tr>
<tr>
<td>95th percentile</td>
<td>186</td>
<td>201</td>
</tr>
<tr>
<td><strong>Triglyceride, mg/dL</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>50th percentile</td>
<td>48</td>
<td>58</td>
</tr>
<tr>
<td>75th percentile</td>
<td>58</td>
<td>74</td>
</tr>
<tr>
<td>90th percentile</td>
<td>70</td>
<td>94</td>
</tr>
<tr>
<td>95th percentile</td>
<td>85</td>
<td>111</td>
</tr>
<tr>
<td><strong>LDL, mg/dL</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>50th percentile</td>
<td>90</td>
<td>94</td>
</tr>
<tr>
<td>75th percentile</td>
<td>103</td>
<td>109</td>
</tr>
<tr>
<td>90th percentile</td>
<td>117</td>
<td>123</td>
</tr>
<tr>
<td>95th percentile</td>
<td>129</td>
<td>133</td>
</tr>
<tr>
<td><strong>HDL, mg/dL</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5th percentile</td>
<td>38</td>
<td>37</td>
</tr>
<tr>
<td>10th percentile</td>
<td>43</td>
<td>40</td>
</tr>
<tr>
<td>25th percentile</td>
<td>49</td>
<td>46</td>
</tr>
<tr>
<td>50th percentile</td>
<td>55</td>
<td>55</td>
</tr>
</tbody>
</table>

Adapted from the Lipid Research Clinics Pediatric Prevalence Study (83).