Cardiovascular Disease Risk in Children and Adolescents

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Atherosclerosis is a global health issue beginning in childhood. Children’s early development of cardiovascular disease (CVD) risk factors may include exposure to key biological components responsible for vascular inflammation in young adults. A retrospective medical record review based on the 2008 American Academy of Pediatrics childhood lipid screening recommendations examined current and initial CVD risk factors for 227 at-risk school-aged children during wellness exams to better determine the age when children are most likely to convert from being risk-free to at risk for CVD. An original risk trend algorithm analyzed risk clustering and risk trend progression. Risk onset was young (M=2.88 years), with family risk most prevalent and risk clustering and accumulation noted, perhaps precluding primordial prevention.

CARDIOVASCULAR DISEASE IS a complex and global health problem that begins in childhood. Atherogenic dyslipidemia, hypertension, left ventricular hypertrophy, and insulin resistance are evidence of cardiovascular disease in children (Hayman et al., 2007). Data from the Bogalusa Heart Study (Berenson, Srinivasan, Bao, Newman, Tracy, & Wattigney, 1998; Newman, Freedman, & Voors, 1986), and Pathobiological Determinants of Atherosclerosis in Youth (PDAY) (Gidding et al., 2006), both prospective pediatric cardiovascular epidemiological studies, showed that atherosclerosis has its origins in childhood, is associated with risk factor development at early ages, and progresses to measureable vascular changes in young adulthood.

Ripatti et al. (2010) define coronary heart disease (CHD) as myocardial infarction (MI), unstable angina pectoris, coronary revascularization, or sudden cardiac death, and define cardiovascular disease (CVD) as the previous factors plus ischemic stroke events. Libby and Theroux (2005) detail how years of research has modified our understanding of the biological processes involved in atherosclerosis from the previously held perception that coronary artery disease (CAD) was a localized disease composed of arterial stenosis and plaques that obstructed blood flow. Atherosclerosis is now recognized as an arterial remodeling disorder (hardening and thickening of arterial walls) based on exposure to systemic inflammatory factors involved with diseases like dyslipidemia, hypertension, diabetes, obesity, and even bacterial infections that raise CVD risk (Libby & Theroux, 2005).

Christakis (2011) surmised that problems with clinical decision making tools and prediction rules for disease related back to “the underlying philosophical approach that many clinicians have to the practice of medicine”; further stating “they are either unable or unwilling to articulate a treatment threshold as aficionados of evidence-based medicine would have them do and then act on it” (p. 91). He advocated that patients either have a particular disease or not, and that tools that do not “perfectly predict outcomes” make it difficult for clinicians to tolerate the possibility of the risk of a missed diagnosis and subsequent “perceived potential [negative] consequences of inaction” (both p. 91) regardless of the predicted chance of having the disease. This merits
consideration when nurse clinicians assess cardiovascular health and identify CVD risk in children, as the nursing intervention approach would differ for promoting primordial prevention, or defining potential disease risk (threshold), and treating known disease.

Strasser (1978) originally developed the concept of primordial prevention as a way to address world health problems, being concerned with preserving risk-factor free populations in developing societies from penetration of risk factor epidemics [like CVD]. More recently Berenson and Srinivasan (2010) suggest that primordial prevention in pediatrics is aimed to improve lifestyle and health behaviors in children and in family cultures, before abnormal risk factors develop. Gupta and Deedwania (2011) developed a continuum for CVD prevention that has primordial prevention as its base. They direct health care professionals to strive for risk-free populations at baseline and to recognize areas such as socioeconomic status as an individual risk factor for CVD, thus incorporating public health financing and access to care issues into their model, which continues on with promotion of smoking cessation, healthy diets and physical activity and focusing medical education on preventive care. As wellness and preventative care are at the heart of nursing education, this model is an adequate framework for childhood CVD health promotion.

The purpose of the current study is three-fold. First, to determine the developmental age when children first tend to accrue CVD risk factors, converting from no risk to at risk for CVD and identifying the likely threshold age between primordial and primary prevention; second, to evaluate risk factor profiles and risk clustering; and third, to understand likely risk trend progression for the study population.

Background

A literature review was completed through PubMed and CINHAL for English language research conducted worldwide, predominantly from 2000 to 2011, using search terms focused on atherosclerosis, cardiovascular health and disease, cardiovascular risk recognition, children, adolescents, family history, primordial prevention, and advanced practice nursing. Secondary sources were included if they were meta-analyses of relevant studies, or recommendations from research panel groups and governmental agencies.

Historically relevant, the Framingham heart study was the first to identify age, hypertension, hyperlipidemia, smoking, and diabetes as major determinants for CVD, calling them coronary risk factors (Dawber & Kannel, 1966; Kannel, Dawber, Kagan, Revotskiw, & Stokes, 1961). Recent longitudinal studies of CHD in adults using Framingham risk factor data, developed and examined the validity and reliability of CHD risk score prediction tools for estimating 10-year risk for likelihood of actual cardiovascular events in adults (Framingham Risk Score-FRS). The FRS using traditional CHD risk factors effectively predicted CHD risk in a white middle-age population (Wilson, D’Agostino, Levy, Belanger, Silberhaz, & Kannel, 1998), and later was determined reliable for predicting 5 to 10-year CHD risk in white and black men and women, and could be used in other ethnic groups when recalibrated for risk factor prevalence (D’Agostino, Grundy, Sullivan, & Wilson, 2001). Lloyd-Jones et al. (2004) investigated using the FRS to stratify lifetime CHD risk, finding it predicted short-term risk for men and women, stratified lifetime risk well for women at any age, less well in younger men, and improved lifetime risk prediction in older men.

There has been global interest in pediatric studies of the reliability of CVD risk factor assessment approaches for predicting future CHD in young persons. Several wide-ranging studies provide supportive background for the current study aims to determine age when children first develop CVD risk, and the importance of trending future CVD risk in at risk children. Juonala et al. (2010) conducted an epidemiologic study that pooled data from four international prospective cohort studies (Australia, Finland, two in the United States) to define the age when childhood CVD risk scores began to relate to carotid artery intima-media thickness (IMT), a marker of subclinical atherosclerosis in adults. IMT has consistently associated with future CVD morbidity. Risk factor scores in all four cohorts demonstrated that higher numbers, or clustering of specific childhood CVD risk factors (high total cholesterol, triglycerides, blood pressure, and body mass index) showed stronger predictive associations for higher young adult carotid IMT for children 9 years or older, and were not significant for children at 3 and 6 years of age.

Corvalan, Uauy, Kain, and Martorell (2010) studied 4-year old Chilean preschoolers to clarify the association of obesity with CVD risk in preschoolers, measuring the contribution of obesity indicators (BMI), and measures of central obesity (waist circumference) to cardiometabolic (CM) risk factors [elevated C-reactive protein (C-RP), diabetes, dyslipidemia, hypertension, and interleukin-6-pro-inflammatory risk factors]. Both obesity factors were positively, but weakly, correlated with CM risk status, but were not specifically associated with elevated lipid concentrations. Corvalan et al. (2010) cautioned that BMI and waist circumference were poor indicators for CM risk factors and CVD risk screening in preschoolers as a population because children grow and mature at differing rates. Other researchers agreed that a single threshold to identify CVD risk across all childhood is inappropriate (Eissa, Wen, Mihalopoulos, Grunbaum, & Labarthe, 2009; Lloyd-Jones et al., 2010; Reinehr, Stoffel-Wagner, Roth, & Andler, 2005). This prompts nurse clinicians to evaluate other CVD risk factors in addition to obesity when assessing risk in young children.

McMahan et al. (2005) used PDAY data that combined measures of traditional CHD risk factors in young people 15–34 years old who died of non-cardiac causes with post-mortem measurements of atherosclerotic lesions in their
coronary arteries and abdominal aortas to develop the PDA\-Y Risk Score (RS) to predict the likelihood of young people having advanced preclinical atherosclerosis lesions. The PDA\-Y RS weighted risk factors differently than the FRS due to differences in how a risk factor may affect CHD, but sufficiently determined risk for future CHD in young persons.

Childhood CVD risk factor clustering (simply a higher number of distinct risk factors per individual) is noted to be more predictive of CVD when an adult. Pouroubrahim et al. (2006) found that CVD risk factors are more prevalent, and more clustered in high-risk families. Shah, Dolan, Gao, Kimball, and Urbina (2011) evaluated whether clustering CVD risks could detect early atherosclerotic lesions in youth, and compared risk clustering to the PDA\-Y RS for predicting early CVD. Results found that the presence of two or more risk factors per individual was reliably associated with vascular changes in adolescents but, a key point for the current study, could not detect when (or age) these changes occurred in relation to when children began accruing CVD risk factors.

There is growing evidence that having a family history (FMHx) of premature CHD is an independent risk factor for CVD (Lloyd-Jones et al., 2004; Pouroubrahim et al., 2006; Ripatti et al., 2010) and is a significant predictor of coronary heart disease (Assimes, 2011; Makedou, Kourt, Makedou, Lazaridou, & Varlamis, 2005). FMHx was not included when the Framingham risk profile was adopted as cholesterol treatment threshold recommendations by the Adult Treatment Panel III (ATP III) of the National Cholesterol Education program (NCEP) (Assimes, 2011). ATP III did agree, though, that one’s risk for CHD increases with younger CHD onset in affected first-degree family members, and with having more first-degree relatives with CVD (NCEP, 2002).

The role that genetics research has in CVD risk assessment is gathering momentum. Nurse clinicians may see future clinical practice implications for family history assessment as genetic assessment technology tools are refined. Genomewide-association studies (GWAS) examine the genetic pathways involved in CAD susceptibility, and aim to identify genetic CAD risk markers, called single nucleotide polymorphisms (SNPs). Past studies of genetic variants have previously explained only small variations in CAD disease risk (Ripatti et al., 2010; Sandhu, Wood, & Young, 2010). Recently Harismendy et al. (2011) conducted research using emerging genetic technology to successfully identify SNPs and genetic enhancers that link carrier CAD susceptibility with inflammatory signaling responses in human endothelial cells.

CVD risk assessment in children and adolescents is a clinical challenge because it must evaluate several areas. The 2008 American Academy of Pediatrics (AAP) guidelines for lipid screening and management of hypercholesterolemia in children and adolescents (Daniels, Greer, & the Committee on Nutrition, 2008) were used in the current study. AAP recommends evaluating all 2 to 10 year olds at each well visit for presence of CVD risk in six areas, four of which address arterial and systemic inflammation. Risk areas include: (a) family history for CVD demographics of dyslipidemia, or for premature CVD in biological parents, grandparents and sibling male relatives ≤55 years of age, or female relatives ≤65 years of age, (b) unknown family history; (c) overweight or obesity; (d) hypertension; (e) cigarette smoking; and (f) diabetes. Lipid studies are recommended for children with one or more risk factors. Normal lipid results would prompt retesting in 3–5 years. AAP proposed that markedly abnormal lipid results may warrant pharmacotherapy in addition to diet and activity changes.

Lipid screening in children recognizes that total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C) levels increase rapidly in the neonate, and then only gradually until 2 years old, becoming fairly constant from age 2 years to early adolescence. A 10–20% decrease for TC and LDL-C is noted in adolescents 12–16 years old (Kwiterovich, 2008). Friedman, Morrison, Daniels, McCarthy, and Sprecher (2006) found significant variation in sensitivities by age for TC and LDL-C levels in children, with the lowest sensitivities at 14–16 years (LDL-C, 22%; TC 18%) and highest sensitivities at 5–10 years (LDL-C, 69%; TC 63%). High-sensitive C-reactive protein (h-s CRP), a lab blood biomarker for inflammation in adults, is not routinely measured in at risk children, due to lack of general reliability and validity as a predictor for inflammation with childhood CVD risk factors besides obesity, shown in one study (Reinehr et al., 2005). All studies, when taken together, corroborate AAP recommendations to evaluate several CVD risk factors, and to screen lipids in at risk children who are 2–10 years old.

Method

Participants and Sampling

The current study was conducted at a private, pediatric primary care practice, one of three solely-pediatric practices serving a population (under 18 years old) of 7307 in two counties in rural Midwest United States. Two pediatricians and one certified pediatric nurse practitioner (PNP), the researcher, are clinicians at the study site.

A convenience sample of all school-aged child/adolescent patients, 4 through 17 years (N=316), scheduling a wellness physical exam with the researcher from June to September 2010 was evaluated for the presence of CVD risk factors. Children were accompanied by a caregiver, defined as an adult biological parent or legal guardian who could provide “self-reported” information about the child’s family history for dyslipidemia and premature CVD. All children with CVD risk factors (n=227, 72% of the convenience sample) were included as the target sample (M=10.65 years, 57% were female, 43% male). Age stratification was used in the
PDAY risk score and Framingham risk score development study samples. In the current study the target sample was also aged-stratified, but into developmental groups representing preschool/kindergarten (4 through 6 years, $n=47$), elementary (7 through 9 years, $n=35$), middle school, grades 5–8 (10 through 13 years, $n=101$), and high school (14 through 17 years, $n=44$). Demographics for the target sample are summarized in Table 1.

Children from the target sample who met three further inclusion criteria were included in a second section of the study for risk trend analysis. Criteria were identifiable age of initial CVD risk onset, age of risk onset different from age at current exam, and previous wellness care at the study practice with written exam findings documented in their medical records ($n=159$, 70% of target sample). Children older than 10 years were included here to evaluate their initial CVD risk data and to extend the age of the population in the sample to allow for examining risk trends.

Exclusion criteria for both sections of the study included lack of CVD risk factors at the current exam ($n=89$, 28% convenience sample), children or adolescents arriving without a caregiver ($n=8$, 2.5% convenience sample), emancipated minors, and pregnant teens.

**Measures**

Cardiovascular disease was conceptually defined in this study as the presence of atherosclerosis, and the other myocardial and vascular changes listed previously that predispose individuals to increased morbidity and mortality from clinical cardiovascular events (Lloyd-Jones et al., 2010). Congenital and infectious CVD were not included in this definition. Risk is a concept that elucidates the probability of specific undesirable outcomes or situations, and effects of that occurrence (Hansson, 2004). Trend is defined to mean a tendency or movement toward something, or in a particular direction.

Caregivers updated written family health histories for each child at their current well exam, providing information about dyslipidemia and premature CVD (hypertension, myocardial infarction, heart surgery for CHD, sudden death, or stroke) according to AAP guidelines. Information was verbally clarified with the researcher. Research centered on individual children’s available written medical records for current and all prior well exams, which were examined for presence and type of CVD risk, going back to as young as 2 years of age when available (AAP lower end age for lipid screening) or to the earliest age that data existed. Children were considered at risk at ages 2 and 3 years if they had positive family history for CVD or BMI $\geq 85$th percentile.

BMIs were calculated from weight and height data (kg/m$^2$) gathered individually at current and all prior well exams, then compared to Center for Disease Control and Prevention (CDC)/National Center for Health Statistics (2000) standardized growth charts, and to CDC BMI-for-age tables (Center for Disease Control and Prevention, 2009) for normal weight curve, overweight ($\geq 85$th percentile) or obesity ($\geq 95$th percentile). Blood pressures (BPs) were obtained by experienced staff nurses using size-appropriate, manual calibrated McKesson BP cuffs. If elevated, BP was re-measured later in the visit, recording the lowest result. The National Heart Lung and Blood Institute (NHLBI, 2004) defines hypertension in children and adolescents as BP $\geq 95$th percentile by age, gender, and height percentile. NHLBI (2004) pediatric blood pressure tables were used to evaluate BPs from the current well exam, and retrospectively from previous exams back to

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Demographics for CVD At Risk Children.</th>
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<tbody>
<tr>
<td>Variables</td>
<td>Stratified Age of Patients</td>
</tr>
<tr>
<td></td>
<td>4–6 years</td>
</tr>
<tr>
<td>Total number of children</td>
<td>47</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>23 (49%)</td>
</tr>
<tr>
<td>Male</td>
<td>24 (51%)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>43</td>
</tr>
<tr>
<td>Black</td>
<td>0</td>
</tr>
<tr>
<td>Native American</td>
<td>1</td>
</tr>
<tr>
<td>Hispanic</td>
<td>1</td>
</tr>
<tr>
<td>Asian</td>
<td>0</td>
</tr>
<tr>
<td>Mixed</td>
<td>2</td>
</tr>
<tr>
<td>Current patient insurance status</td>
<td></td>
</tr>
<tr>
<td>Private</td>
<td>27 (57%)</td>
</tr>
<tr>
<td>Medicaid</td>
<td>19 (40%)</td>
</tr>
<tr>
<td>None</td>
<td>1 (2.1%)</td>
</tr>
<tr>
<td>Current patient age (mean for age group)</td>
<td>4.64 years</td>
</tr>
</tbody>
</table>
the child’s age of initial identifiable risk, for hypertension. Urine samples were screened for glycosuria using Siemens Multistix 10 SG urinalysis reagent strips. Adolescents were privately asked about substance use including smoking habits. Demographic data were gathered from patient administration forms. Caregivers of CVD at risk children were educated about AAP recommendations for lipid screening, and lipid panels were ordered when indicated. The nurse researcher counseled all children/adolescents and their caregivers about healthy lifestyle habits including diet and physical activity at the well exam.

Research Design

Study design was approved by the university institutional review board and the study practice’s pediatrician executive. Ethical standards for research were followed. All data collection was completed or verified by the researcher. This study presents descriptive, retrospective data from written medical records of well exams for children having at least one CVD risk factor at their current exam, back through prior well exams to as young as 2 years of age, as available. The study was conducted in two sections. The first section was an analysis of individual current and prior well exams to identify which CVD risk factors were present in at risk children, and whether it was possible to determine an age when children initially tend to accrue any CVD risk factors. The second section developed CVD risk profiles and analyzed these for risk trending over time.

CVD risk profiles were developed by clustering risk factors into three profile categories: FMHx-only, FMHx plus modifiable risk factors (BMI/HTN), and modifiable risk factors (BMI/HTN) only. All FMHx risk factors were combined in one FMHx classification. The child’s initial and current risk profiles were compared for patterns of risk change using an algorithm model designed for this analysis (Figure 1).

The concept of risk treatment threshold was introduced in the second study section, and examined with two separate models that both incorporate differences in initial and current risk profiles but utilize different clinical approaches. The first model, called the straight-classification model (SCM), was based solely on the presence or absence of modifiable CVD risk (BMI or HTN) or FMHx risk. The second model, termed clinical observation model (COM), reviewed records for the presence of CVD risk factors plus noted clinician documentation of incremental clinical improvement (decreases) in the same modifiable CVD risk, even though risk may have persisted, or worsening modifiable risk (increases) to represent risk degree or severity. This distinction is relevant as children have differing individual growth and maturation rates, and small changes may noticeably affect their BMI and HTN classifications. The COM approach begs Christakis (2011) comments about clinical usefulness of prediction

![Figure 1](image-url)  
**Figure 1** CVD risk profiles and risk trend analysis algorithm. $^1$RISKi: initial risk profile; $^2$RISKc: current risk profile.
tools, but is related to categorizing strategies for primordial, primary, and secondary prevention of disease. It may also be useful when evaluating children with a history of modifiable CVD risk factors at previous well visits, but having since lowered BMI and BPs, are now a subset within the current risk-free group and merit close follow-up. Children’s risk profiles were combined by developmental age groups, and as a whole for the group analysis. **PASW Statistics 18.0.0 (2009)** was used for statistical data analysis.

### Results

In the first section of the study the age of CVD risk onset was identified for 73% of the target sample ($n=166$, $M=2.88$ years, SD 2.95 years), found to be younger than defined preschool age of 4 years and the mean age was similar across all developmental age groups. Ten percent ($n=22$) of children presented with CVD risk factors for the first time at their current exam. **Table 2** summarizes the distribution of the participant’s CVD risk factors at current well exams. Risk factors for cigarette use and diabetes mellitus were evaluated, but not included in the table, having identified only one person with each of these risk factors in the sample. Of note, a recent survey of Michigan high school students documented that 18% of students admitted smoking cigarettes in the prior month (Anderson, Lyon-Callo, Heiler, Miller, & Theisen, 2008).

Crosstab analysis was run for each risk factor, stratified by age, gender, and race. Gender was not significant for particular CVD risk factors, or for risk factor trends. Ninety-one percent of participants were white: analysis for ethnic differences was not significant. **Table 3** details distributions for initial and current CVD risk profiles. A chi-square test of independence was calculated comparing initial and current CVD risk profiles. There was an interaction showing significant association between initial and later risk profiles [$\chi^2(4)=131.59$, $p<.001$]. Overall, 89% ($n=190$) of the target sample had some component of FMHx as a current risk factor. In the risk trend subset study group, 87% ($n=139$) had some component of FMHx risk at age of initial risk onset, and 92% ($n=146$) had FMHx risk at current exams. Risk changes were most notable in the FMHx + BMI/HTN profile subgroup at current exam, almost tripling risk within this sample. Children in the study were accumulating modifiable risk factors over time, thus clustering and increasing CVD risk. Risk trend analysis using the SCM showed smaller variations between the initial and current risk profiles than with the COM as less than 3% ($n=4$) of children had improvement in risk status from initial to current risk, 73% ($n=116$) had no change in risk, and 24.5% ($n=39$) had increased risk. The COM incorporates incremental changes in modifiable risk factors and found 4.4% ($n=5$) had improvement in risk status, 55% ($n=94$) had no change in risk, and 39% ($n=59$) had increased risk (1%

### Table 2  Distribution of CVD Risk Factors at Current Wellness Exams.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Number of children by stratified age groups</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>4–6 years</td>
</tr>
<tr>
<td><strong>BMI</strong></td>
<td></td>
</tr>
<tr>
<td>Normal (&lt;85%)</td>
<td>Count (%)</td>
</tr>
<tr>
<td><strong>HTN</strong></td>
<td></td>
</tr>
<tr>
<td>Normotensive</td>
<td>Count (%)</td>
</tr>
<tr>
<td><strong>FMHx dyslipidemia</strong></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>Count (%)</td>
</tr>
<tr>
<td><strong>FMHx premature CVD</strong></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>Count (%)</td>
</tr>
<tr>
<td><strong>FMHx unknown/known</strong></td>
<td></td>
</tr>
<tr>
<td>Unknown</td>
<td>Count (%)</td>
</tr>
</tbody>
</table>

### Table 3  Distributions of Initial and Current CVD Risk Factor Profiles.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Number (%) of children by stratified age groups</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>4–6 years</td>
</tr>
<tr>
<td><strong>RISKi</strong>:</td>
<td></td>
</tr>
<tr>
<td>FMHX-only</td>
<td>27 (64%)</td>
</tr>
<tr>
<td>FMHX + BMI/HTN</td>
<td>6 (14%)</td>
</tr>
<tr>
<td>BMI/HTN</td>
<td>9 (21%)</td>
</tr>
<tr>
<td>Totals</td>
<td>42</td>
</tr>
<tr>
<td><strong>RISKc</strong>:</td>
<td></td>
</tr>
<tr>
<td>FMHX-only</td>
<td>19 (45%)</td>
</tr>
<tr>
<td>FMHX + BMI/HTN</td>
<td>16 (38%)</td>
</tr>
<tr>
<td>BMI/HTN</td>
<td>7 (17%)</td>
</tr>
</tbody>
</table>

* RISKi: initial risk profile.
† RISKc: current risk profile.
Discussion

CVD risk is a growing reality for very young children, and this is supported by the current study determination that children often change from no risk to being at risk for CVD before preschool. Nurse clinicians can use the AAP recommendations to identify CVD risk in children perhaps previously considered too young for lipid screening, and to target interventions as early in life as possible to reduce risk.

A large number of children in the current study were considered at risk solely due to family history. Group trend for CVD risk in this study found that the majority of children tended to stay in similar risk profile categories over time, although 29% in the FMHx-only risk profile initially, accumulated unhealthy weight/HTN risk, clustering risk, and switching profiles. Twenty-four percent in the BMI/HTN-only profile initially, eventually had relatives with CVD. Clearly, the growing prevalence of CVD in adult family members limits the potential goal for primordial prevention in children in the current study if family history and genetics are considered part of a child’s risk profile when the child has no other risk factors. Children may accumulate CVD risk factors over time, including family history risk as first-degree adults grow older and develop dyslipidemia and CVD, with adult disease reflecting onto the child’s own risk profile. This finding emphasizes the importance of updating FMHx risk at each well child exam. It is important to note that 28% of the children in the original convenience sample did not have any CVD risk factors, so a “healthy behaviors” nursing approach for primordial prevention similar to Gupta and Deedwania’s model (2011) is appropriate for them.

The COM risk trend analysis approach actually identified more children with changes in risk clustering and trends over time, identifying smaller increments of change in BMI or BP (typical for children) which could easily classify children into different risk profiles. There may be disadvantages to using standardized BMI and HTN tables in children to represent obesity and hypertension. Small incremental changes in weight can effect classifications for BMI percentile for age. HTN risk classification may be difficult in anxious children having falsely elevated BPs.

Risk prevention and intervention efforts are best tailored for children as members of a family unit. Children share family genomics (family history), environmental factors, lifestyle and health behaviors with their caregivers, and may mimic adult’s poor food choices, physical inactivity, or initiating smoking, contributing to future prevalence of atherosclerosis (Berenson, 2009; Lloyd-Jones et al., 2010). While children may be genetically predisposed to developing inflammatory CVD changes with risk factor exposure, healthy lifestyle practices could potentially lower the “inevitability” of gene expression. Genome research may provide practical means for genetic testing in the future that would help clinicians evaluate family risk for developing atherosclerosis, prompting intensive nursing and medical interventions for primary prevention in health management.

Nurse clinicians must repeatedly and realistically address evolving CVD risk with children and their caregivers in developmentally appropriate ways. In pediatrics prevention efforts must also evaluate how accurately caregivers perceive and acknowledge their child’s CVD risk, as successful intervention requires motivation from both child and caregiver to activate necessary behavioral changes to reduce risk. These are concepts for future research.

Advanced practice nurses (APNs) are expanding their roles in primary care practice to meet present patient needs for support managing health and risk for disease. Nurses can use clinically-observed trends in a child’s CVD risk profile, and the knowledge that as risk clustering develops in children this increases likelihood to develop advanced atherosclerotic lesions, to individualize patient treatment thresholds for primordial and primary prevention, and to plan patient-specific education and access to resources. Conrad & Schneider (2011) see this as a unique opportunity to enhance visibility of nursing interventions and outcomes in electronic health records that are currently used primarily to document medical care.

In fact, the present study findings, completed in spring 2011, are reflective of NHLBI (2011) expert panel guidelines about cardiovascular health and risk reduction in children and adolescents released later the same year. Three areas of the panel’s literature review of evidence-based and observational studies were especially relevant to the current study. The review found that a child’s CVD risk was strongly inversely related to the parent’s age at the time of the parents’ initial CVD event, and that the association of increased family history CVD risk was sustained for gender, racial and ethnic groups, prompting recommendations for FMHx screening beginning at 2 years old. Second, studies found a strong association for increases in CVD risk for children with risk factor clustering. And third, the panel advocated for the importance of placing clinical recommendations in a developmental context when considering expression of
disease, and approaches to patient and caregiver teaching and recommendations which address both primordial prevention (beginning in infancy), and primary prevention risk screening and reduction. The NHLBI panel incorporated the AAP lipid screening guidelines in their lengthy recommendations, but did go further than AAP by recommending universal screening for dyslipidemia with a non-fasting non-HDL–C level at 10 years old.

Limitations

There were limitations to this study. Children joined the clinical practice at differing ages so ages for initial data points varied. Some children had well visits at irregular intervals rather than annually, having differing numbers of wellness exams to evaluate. State vaccination requirements for sixth graders were changed just before the study began so more middle school students than usual needed wellness exams during the study period. Fully-vaccinated older students perhaps were under-represented as some student–athletes opt to have sports pre-participation exams at volunteer screening clinics held at area schools during June 2010. Genetic risk seemed quite high for the group, perhaps in part due to the self-reported nature of FMHx risk, with caregivers sometimes estimating ages when grandparents developed dyslipidemia or CVD. The AAP recommendation to screen when family histories are unknown may incorrectly place children in the FMHx risk group who are actually risk-free. The target population was not ethnically diverse and taken from only one pediatric practice site so it is difficult to generalize these findings to other ethnic groups, or settings other than a private health care facility. While there is a sizeable local Native American population, representation may have been lower as some Native children receive well care at a tribal health center.

Conclusions

A child’s exposure to CVD risk factors such as obesity, HTN, dyslipidemia, diabetes, and smoking have the potential to prompt systemic inflammatory responses that may lead to arterial changes. Nurse clinicians must be willing to define and address CVD risk treatment thresholds when present, even for very young children. Although genetic screening for gene variants related to inflammatory-responses is a complex and fledgling approach to CVD risk assessment, evidence linking atherosclerosis pathophysiology to familial predisposition for inflammation when exposed to CVD risk factors is essential to understanding the concept of CVD risk. It prompts us to screen other family members when early CVD risk is identified, and encourages developmentally age-appropriate prevention efforts to decrease modifiable childhood CVD risk.

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