Childhood C-Reactive Protein in Predicting CRP and Carotid Intima–Media Thickness in Adulthood
The Cardiovascular Risk in Young Finns Study

Markus Juonala, Jorma S.A. Viikari, Tapani Rönnemaa, Leena Taittonen, Jukka Marniemi, Olli T. Raitakari

Background—Atherosclerosis begins in childhood, and inflammation may contribute to its pathophysiology. The value of measuring inflammatory markers in the pediatric risk assessment, however, is uncertain. We examined whether childhood C-reactive protein (CRP) levels predict CRP and carotid intima–media thickness (IMT) in adulthood.

Methods and Results—Study cohort included 1617 subjects, aged 3 to 18 years at baseline in 1980. These subjects were reexamined in 2001 at ages 24 to 39 years. In 2001, CRP was measured from fresh samples, and the subjects underwent carotid IMT study to evaluate subclinical atherosclerosis. Baseline (1980) CRP concentrations were measured from frozen samples in 2005. A significant tracking was observed between childhood and adult CRP levels. The age- and sex-specific correlations were the highest in the age group of 18 years at baseline ($r = 0.47$ in females, $r = 0.32$ in males, $P < 0.0001$). The association between childhood and adult CRP levels was independent of serum lipids, blood pressure, smoking, obesity indices, and insulin. In multivariate analysis, childhood risk factors that independently associated with increased adult IMT included elevated systolic blood pressure ($P < 0.0001$), high low-density lipoprotein–cholesterol ($P = 0.01$) and smoking ($P = 0.049$), but not CRP ($P = 0.95$).

Conclusions—Childhood CRP values predict weakly but significantly adult CRP, and this association is independent of other metabolic risk factors. Unlike conventional risk factors, however, childhood CRP does not predict adult IMT. (Arterioscler Thromb Vasc Biol. 2006;26:1883-1888.)

Key Words: CRP ■ tracking ■ childhood

The existing evidence indicates that prevention of atherosclerosis should begin in childhood.1 The risk factor–specific guidelines for primary prevention in children and adolescents include the assessment of conventional risk factors, such as serum lipids, blood pressure, smoking, and obesity, for identifying children at high risk of future cardiovascular disease.2 The rationale of focusing on these factors is based on observational evidence showing that they track from childhood to adulthood,3,4 are related with atherosclerotic changes in young people,5,6 and predict preclinical atherosclerosis in adulthood.7–9

The pathophysiology of atherosclerosis has recently been shown to include local inflammation as a promoter to plaque formation, as well as a trigger to plaque instability.10–12 In favor of this, several prospective epidemiological studies have shown that increased high-sensitive C-reactive protein (CRP) is an independent predictor of cardiovascular events.13 In support of the role of CRP in the early phases of atherosclerosis, we have shown in Finnish children that elevated CRP levels are associated with increased carotid intima–media thickness (IMT) and decreased brachial artery flow-mediated dilatation.14 Results from the Pathobiological Determinants of Atherosclerosis in Youth Study (PDAY) indicate that serum CRP level is independently associated with advanced atherosclerosis in young persons.15 These observations thus suggest that the assessment of inflammation by measuring serum CRP in childhood might add value to the pediatric cardiovascular risk estimations. Therefore, we have examined the tracking of CRP from childhood to adulthood and the value of childhood CRP measurement of predicting carotid IMT in adulthood. Study subjects were participants in the large population-based Cardiovascular Risk in Young Finns Study. CRP values were measured in 1617 subjects from their baseline samples collected in 1980, at ages 3 to 18 years, and during the 21-year follow-up in 2001, at ages 24 to 39 years.

Methods
The Cardiovascular Risk in Young Finns Study is an on-going 5-center follow-up study of atherosclerosis risk factors and precur-

1883

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From The Centre of Applied and Preventive Cardiovascular Medicine (M.J.) and Departments of Medicine (J.S.A.V., T.R.) and Clinical Physiology (O.T.R.), University of Turku; Department of Pediatrics (L.T.), Vaasa Central Hospital; and National Public Health Institute (J.M.), Department of Health and Functional Capacity, Turku, Finland.
Correspondence to Olli T. Raitakari, Department of Clinical Physiology, PO Box 52, 20521 Turku, Finland. E-mail olli.raitakari@utu.fi
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Arterioscler Thromb Vasc Biol is available at http://www.atvbaha.org DOI: 10.1161/01.ATV.0000228818.11968.7a
sors in Finnish children and adolescents. The first cross-sectional survey was conducted in 1980, when 3596 participants aged 3 to 18 years were randomly chosen from the national population register.\textsuperscript{16} In 2001, we reexamined 2283 of these individuals, aged 24 to 39 years.\textsuperscript{17} Subjects gave written informed consent, and the study was approved by the local ethics committees.

Clinical Characteristics and Risk Factors

Metabolic and cardiovascular risk factors were studied in childhood in 1980. Height and weight were measured, and body mass index (BMI) was calculated. Blood pressure was measured from brachial artery using a standard mercury sphygmomanometer. From 3-year olds, blood pressure was measured with an ultrasound device. Skinfold thicknesses (in childhood) were measured by Harpenden calipers in 1980. Serum insulin was measured using an immunoassay method. Smoking habits were assessed with a questionnaire in subjects aged 12 to 18 years. Those who had smoked during last 2 months were considered smokers. For the determination of serum lipid levels, venous blood samples were drawn after an overnight fast. All lipid determinations were done using standard methods. Low-density lipoprotein (LDL)-cholesterol concentration was calculated by the Friedewald formula.

CRP Measurements

In 2001, serum high-sensitive CRP was analyzed in 2252 subjects by an automated analyzer (Olympus AU400) using a turbidimetric immunoassay kit ("CRP-UL"-assay, Wako Chemicals, Neuss, Germany). The detection limit suggested by the manufacturer of the assay was 0.06 mg/L. In our laboratory, we have been able to measure reproducibly CRP concentrations between 0.02 and 0.06 mg/L. Therefore, we used a detection limit of 0.02 mg/L. The interassay coefficient of variation was 3.33\% at the mean level of 1.52 mg/L (n = 116) and 2.65\% at the mean level of 2.51 mg/L (n = 168). Childhood serum samples were taken in 1980 and stored at −20°C. These samples were analyzed in 2005 using the same method as in 2001. During the storage, the samples were not thawed or refrozen. We excluded subjects with CRP levels more than 10 mg/L in 1980 or 2001 (N = 65), diabetes (N = 13, CRP 4.29±5.46 mg/L in 2001), chronic rheumatic disease (N = 2, CRP 0.15±0.09 mg/L in 1980; N = 34, CRP 5.14±7.31 mg/L in 2001), history of recent infection (N = 113, CRP 3.34±6.62 mg/L in 2001) and pregnant women (N = 61, CRP 4.63±4.87 mg/L in 2001), lactating women (N = 53, CRP 2.48±5.27 mg/L in 2001), and those using oral contraceptives (N = 38, CRP 2.17±4.03 mg/L in 1980; N = 288, CRP 3.72±6.08 mg/L in 2001) from all analyses. Thus, the study cohort included 1617 subjects (693 females and 924 males). All analyses were repeated after inclusion of subjects using oral contraceptives, with essentially similar results. We also repeated all analyses after setting all CRP values to 0 that were below the recommended detection limit of the manufacturers (0.06 mg/L) (5\% of values in 1980 and <1\% values in 2001). This did not influence the main findings.

Stability of CRP in Storage

To study the stability of CRP stored at −20°C, we reanalyzed CRP in 2006 from spare serum samples of 39 subjects (age, 55.7±5.2 years [mean±SD]; males, 56\%; diabetics, 56\%; BMI, 29.1±5.2 kg/m²; LDL-cholesterol, 4.42±1.14 mmol/L) who had participated in another study in 2001 and had high-sensitive CRP measurements performed at that time in the same laboratory and with the methodology.\textsuperscript{18} Thus, the spare samples had been stored at −20°C for 5 years. During the storage, the samples had not been thawed or refrozen. The mean±SD (median) CRP values in serial measurements were 2.10±2.26 mg/L (1.7 mg/L) in 2001 and 1.90±1.81 mg/L (1.6 mg/L) in 2006. There was a good agreement between the measurements, the Spearman’s rank order correlation coefficient being $r=0.997$ (Figure). Coefficient of variation between the 2 measurements with 5-year interval was 6.5\%.

Carotid IMT

Carotid ultrasound studies were performed using Sequoia 512 ultrasound mainframes (Acuson, Mountain View, Calif) with 13.0-MHz linear array transducer, as previously described.\textsuperscript{8} In brief, the image was focused on the posterior (far) wall of the left carotid artery. Magnified image was recorded from the angle showing the greatest distance between the lumen–intima interface and the media–adventitia interface. At least 4 measurements of the common carotid far wall were taken 10 mm proximal to the bifurcation to derive mean carotid IMT. The between-visit (2 visits 3 months apart) coefficient of variation of IMT measurements was 6.4\%.\textsuperscript{8}

Statistical Analyses

The tracking of CRP from childhood to adulthood was estimated by calculating Spearman’s rank-order correlation coefficients. Univariate correlations between childhood risk factors and childhood and adult CRP were examined with Spearman’s correlation analysis. Variables correlating significantly with CRP in univariate analysis were included in multivariate analysis to establish significant determinants of childhood and adult CRP. To study whether childhood CRP is an independent determinant of adult IMT, we used multivariate modeling. The effect of CRP was adjusted for childhood risk factors that have been shown to predict IMT in this cohort.\textsuperscript{8} In multivariate regression models, values of CRP, insulin, and triglycerides were log-transformed because of skewed distributions. All analyses were performed using the Statistical Analysis System, SAS (version 8.1), and statistical significance was inferred at a 2-tailed probability value of $P<0.05$.

Results

CRP levels in childhood and adulthood are shown stratified by age and sex in Table 1. Mean values in childhood were 0.66 mg/L in girls and 0.64 mg/L in boys. Mean values in adults were 1.13 mg/L in women and 1.08 mg/L in men. No significant age trend was detected between ages 3 and 18 years. In all subjects, a significant tracking was observed between childhood and adulthood CRP levels with a Spearman’s correlation coefficient of $r=0.29$ ($P<0.0001$). A significant correlation between childhood and adult CRP values was observed in all age groups, except in 3-year olds. Tracking seemed better in older age groups and in females. The highest tracking correlations were seen in 18-year-old subjects (males $r=0.32$, females $r=0.47$, $P<0.0001$). By comparison, the 21-year tracking correlations of LDL-cho-
TABLE 1. CRP Levels in Childhood and Adulthood

<table>
<thead>
<tr>
<th></th>
<th>Childhood CRP, mg/L</th>
<th></th>
<th>Adult CRP, mg/L</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>Age, y</td>
<td>Median</td>
<td>Mean±SD</td>
</tr>
<tr>
<td>Females</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>73</td>
<td>3</td>
<td>0.43</td>
<td>1.37±2.24</td>
<td>0.47</td>
</tr>
<tr>
<td>104</td>
<td>6</td>
<td>0.31</td>
<td>0.76±1.21</td>
<td>0.36</td>
</tr>
<tr>
<td>106</td>
<td>9</td>
<td>0.32</td>
<td>0.68±1.00</td>
<td>0.35</td>
</tr>
<tr>
<td>142</td>
<td>12</td>
<td>0.16</td>
<td>0.43±0.85</td>
<td>0.20</td>
</tr>
<tr>
<td>144</td>
<td>15</td>
<td>0.19</td>
<td>0.58±1.33</td>
<td>0.24</td>
</tr>
<tr>
<td>124</td>
<td>18</td>
<td>0.23</td>
<td>0.48±0.66</td>
<td>0.28</td>
</tr>
<tr>
<td>All</td>
<td>693</td>
<td>0.29</td>
<td>0.66±1.25</td>
<td>0.29</td>
</tr>
<tr>
<td>Males</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>144</td>
<td>3</td>
<td>0.19</td>
<td>0.74±1.52</td>
<td>0.27</td>
</tr>
<tr>
<td>128</td>
<td>6</td>
<td>0.21</td>
<td>0.54±0.96</td>
<td>0.24</td>
</tr>
<tr>
<td>170</td>
<td>9</td>
<td>0.15</td>
<td>0.57±1.33</td>
<td>0.20</td>
</tr>
<tr>
<td>156</td>
<td>12</td>
<td>0.13</td>
<td>0.58±1.31</td>
<td>0.21</td>
</tr>
<tr>
<td>172</td>
<td>15</td>
<td>0.19</td>
<td>0.62±1.33</td>
<td>0.25</td>
</tr>
<tr>
<td>154</td>
<td>18</td>
<td>0.29</td>
<td>0.78±1.43</td>
<td>0.35</td>
</tr>
<tr>
<td>All</td>
<td>924</td>
<td>0.19</td>
<td>0.64±1.33</td>
<td>0.25</td>
</tr>
</tbody>
</table>

Total no. of subjects was 1617. CRP levels during childhood and adulthood levels were assessed in 1980 and 2001, respectively. Subjects with CRP levels >10 mg/L, diabetes, chronic rheumatic disease, and history of recent infection and pregnant women, lactating women, and those using oral contraceptives have been excluded.

TABLE 2. Univariate Spearman’s Correlation Coefficients Between Risk Factors Measured and CRP Measured

<table>
<thead>
<tr>
<th>Childhood Variable</th>
<th>r (Childhood CRP)</th>
<th>P (Childhood CRP)</th>
<th>r (Adult CRP)</th>
<th>P (Adult CRP)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LDL-cholesterol</td>
<td>-0.04</td>
<td>0.10</td>
<td>-0.01</td>
<td>0.85</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>-0.19</td>
<td>&lt;0.0001</td>
<td>-0.01</td>
<td>0.57</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>0.04</td>
<td>0.09</td>
<td>0.05</td>
<td>0.04</td>
</tr>
<tr>
<td>BMI</td>
<td>0.17</td>
<td>&lt;0.0001</td>
<td>0.15</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Skinfold thickness</td>
<td>0.27</td>
<td>&lt;0.0001</td>
<td>0.19</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>0.02</td>
<td>0.32</td>
<td>0.05</td>
<td>0.04</td>
</tr>
<tr>
<td>Insulin</td>
<td>0.01</td>
<td>0.79</td>
<td>0.12</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Age</td>
<td>0.01</td>
<td>0.72</td>
<td>0.06</td>
<td>0.02</td>
</tr>
<tr>
<td>Sex</td>
<td>0.08</td>
<td>0.0001</td>
<td>-0.01</td>
<td>0.76</td>
</tr>
<tr>
<td>Smoking*</td>
<td>0.11</td>
<td>0.0006</td>
<td>0.02</td>
<td>0.49</td>
</tr>
</tbody>
</table>

Total no. of subjects was 1617. Risk factors were measured at ages 3 to 18 years in 1980, and CRP was measured in 1980 and 2001. *Smoking data were available for subjects aged 12 to 18 years at baseline.

TABLE 3. Childhood Multivariate Determinants of Childhood CRP and Adult CRP

<table>
<thead>
<tr>
<th>Determinants of CRP</th>
<th>β±SE</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1: Determinants of childhood log-CRP*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female sex</td>
<td>0.176±0.058</td>
<td>0.003</td>
</tr>
<tr>
<td>Childhood HDL-cholesterol, mmol/L</td>
<td>-0.629±0.103</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Childhood BMI, kg/m²†</td>
<td>0.079±0.010</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Childhood smoking</td>
<td>0.390±0.063</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Model 2: Determinants of adulthood log-CRP‡</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-0.014±0.008</td>
<td>0.09</td>
</tr>
<tr>
<td>(log) Childhood CRP, mg/L</td>
<td>0.204±0.022</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Childhood BMI, kg/m²†</td>
<td>0.043±0.012</td>
<td>0.0006</td>
</tr>
<tr>
<td>(log) Childhood insulin, mU/L</td>
<td>0.143±0.046</td>
<td>0.002</td>
</tr>
<tr>
<td>Childhood systolic blood pressure, mm Hg</td>
<td>-0.001±0.003</td>
<td>0.63</td>
</tr>
<tr>
<td>(log) Childhood triglycerides, mmol/L</td>
<td>-0.012±0.080</td>
<td>0.88</td>
</tr>
</tbody>
</table>

Risk factors and CRP were measured at ages 3 to 18 years during childhood in 1980 and adult CRP at ages 24 to 39 years in 2001. Those factors with significant correlation in univariate analysis were included in multivariate models. *Model 1, childhood CRP (N=1598). †Skinfold thickness was an independent determinant of both childhood and adulthood CRP (both P<0.0001), when included in the models instead of BMI. ‡Model 2, adult CRP (N=1594).

lesterol (r=0.47), HDL-cholesterol (r=0.49) and BMI (r=0.39) were higher than that of CRP’s, whereas the tracking of systolic blood pressure (r=0.30) was similar to CRP (P always <0.0001).

Childhood Determinants of Childhood and Adult CRP and Adult Carotid IMT

Childhood BMI and smoking correlated directly, and high-density lipoprotein (HDL)-cholesterol inversely with childhood CRP (Tables 2 and 3). In univariate analyses, childhood BMI, blood pressure, triglycerides, and insulin correlated with adult CRP (Table 2). The effect of confounding factors on association between childhood and adulthood CRP was studied with multivariate analysis. In this model, the association between childhood and adult CRP levels was independent of other cardiovascular risk factors, and childhood BMI...
and insulin were also independently associated with adult CRP (Table 3).

Multivariate model of childhood determinants of adult IMT is shown in Table 4. CRP did not correlate with IMT, whereas childhood LDL-cholesterol, blood pressure, and smoking were independently associated with adult IMT.

**Discussion**

CRP is a sensitive biomarker of inflammation that has gained acceptance as a risk factor for atherosclerosis. As exposure to risk factors in childhood may contribute to the development of atherosclerosis later in life,5–8 we studied whether CRP measured in childhood predicts CRP and carotid IMT measured in adulthood. We observed a weak, but significant, 21-year tracking of CRP from childhood and found that this association was independent of confounding effects of metabolic and conventional cardiovascular risk factors. Contrary to traditional risk factors identified in childhood, however, childhood CRP levels did not predict increased carotid IMT in adulthood.

The 21-year tracking correlation for CRP was \( r = 0.29 \). Somewhat higher interindividual correlations have been reported earlier. Danesh et al10 observed a 12-year tracking correlation coefficient of \( r = 0.59 \) in 379 Icelandic men aged 56 years at baseline. Kayaba et al10 reported a 5-year tracking correlation coefficient of \( r = 0.43 \) in 368 Japanese subjects aged 30 to 69 years. In the Cholesterol and Recurrent Events (CARE) study,25 the 5-year tracking correlation was \( r = 0.60 \) in 214 subjects with a mean age of 59 years. Previous reports have suggested that the tracking of CRP is similar to that of blood pressure and serum cholesterol.22,24 In the present study, the 21-year tracking correlations of LDL-cholesterol, HDL-cholesterol, and BMI were higher than that of CRP, whereas the tracking of systolic blood pressure was similar to CRP. The discrepancies with previous reports have probably many reasons. Prior studies have been conducted in smaller study populations (N < 400 always), the follow-up intervals have been only up to 12 years (mainly 1 to 5 years), and all previously studied populations have been elderly. It is possible that because of sexual maturation, there is more physiological intrindividual variability in CRP levels during childhood and adolescence than later in life. In addition, changes in lifestyle may occur more frequently in adolescence than in adulthood, and it has been shown that CRP levels are affected by many lifestyle mediated environmental factors, such as obesity, smoking, and use of oral contraceptives.22–24 Therefore, it may not be surprising that there is a weaker tracking of CRP from childhood to adulthood than during adulthood. In line with this, the 21-year tracking correlations in the present study were the highest in the oldest age group (18-year olds at baseline).

A recent statement from the Centers for Disease Control and Prevention and the American Heart Association25 concluded that it is reasonable to measure CRP in adults as an adjunct to the measurement of established risk factors to assess cardiovascular risk. However, the value of measuring CRP in the pediatric risk assessment is unknown. In favor of this, there is much experimental evidence indicating that CRP may be causally related to the development of atherosclerosis, thus suggesting that CRP may represent a biochemical marker of the early atherosclerotic process. CRP can be found in arterial walls affected by atherosclerosis but not in a healthy vessel wall.26 CRP directly increases endothelial expression of adhesion molecules.11 CRP may influence monocyte chemotaxis during atherogenesis, and the deposition of CRP seems to precede the appearance of monocytes in early lesions.27 CRP may also mediate the uptake of LDL into macrophages.28 Thus, together, these experimental data suggest that CRP may have a direct proatherogenic role by disturbing endothelial function and promoting the formation of early atherosclerotic lesions. However, in a recent in vitro study, Taylor et al29 reported that azide and lipopolysaccharide contaminations in the commercial CRP preparations, not CRP per se, cause endothelial cell activation events. Their data thus suggested that a wide range of effects on endothelial cells ascribed to CRP in in vitro experiments may in fact be attributable to azide and lipopolysaccharide and not to CRP itself. In clinical studies conducted in adults, CRP levels have been consistently shown to predict the development of atherosclerotic disease.13 However, the findings concerning the associations between CRP and early markers of atherosclerosis have been controversial. Increased CRP concentration in healthy children has been related with increased carotid IMT.14 Furthermore, CRP levels in young adults have been related with atherosclerotic lesions in postmortem samples.15 However, in a recent report from the Dallas Heart Study involving more than 2500 subjects, CRP concentration was not an independent determinant of coronary artery calcification or aortic plaque.30 In the present study, we found that childhood CRP was not associated with carotid IMT. Therefore, these results do not encourage the measurement of CRP as part of the pediatric cardiovascular risk assessment.

Suggesting the validity of our analyses, childhood CRP levels were very similar to those reported previously from Finnish, Canadian, Taiwanese, UK, and US children and adolescents.31–35 The comparison to previous studies is shown in Table 5. In summary, median values between 0.2 to 0.4 mg/L have been reported in most previous studies. Our data are also in agreement with those published by Ford et al from US children.33 They demonstrated that CRP levels are lower in children compared with young adults and that during ages 3 and 18, the levels are not much influenced by age. The
transition to higher adult values starts to occur gradually between ages 16 to 19 years, with large intraindividual variation.33 Furthermore, as a sign of internal validity of our measurements, we found that metabolic risk factors, such as obesity indices and HDL–cholesterol (inverse correlation) and smoking, correlated significantly with childhood CRP levels. Importantly, childhood obesity indices correlated with similar magnitude with both childhood and adulthood CRP. In addition, girls using oral contraceptives had increased CRP values in adolescence, a similar finding that we have previously reported in adult women.24

Childhood BMI and insulin correlated with CRP values in adulthood in multivariate analysis. This finding is in line with several previous cross-sectional studies that have shown that the components of metabolic syndrome, especially obesity indices, are independent correlates of CRP levels.24,36 Possible mechanism for this association may include increased interleukin (IL)-6 production by adipocytes, stimulating hepatic CRP production.37 In a recent study by Calabro et al.,38 it was also demonstrated that human adipocytes can produce CRP under the stimulation of several proinflammatory cytokines.

### Study Limitations

Because baseline high-sensitive CRP is the key variable in this analysis, the potential of a measurement error is not trivial. We analyzed childhood CRP from serum samples that had been collected in 1980 and stored for 25 years at −20°C. It is generally thought that serum and plasma samples of CRP are stable for long periods when frozen.39–41 However, the long-term stability of high-sensitive CRP is unknown, as the high sensitive methods have been commercially available for less than a decade.42 During long-term storage, protein levels may be reduced as a result of proteolysis and aggregation. Thus, a possibility exists that the levels of childhood CRP values analyzed from stored samples may have been inaccurately and erroneously low. However, we observed that CRP levels had remained remarkably stable during 5-year storage. Most importantly, the ranking between the serial CRP values had remained nearly identical, as indicated by high rank-order correlation between the measurements. Thus, although the effects of long-term storage remains unknown, the results regarding medium-term storage suggest that CRP remains stable when stored at −20°C.

### Conclusions

In summary, we found that childhood CRP values are weakly, but significantly, predictive of adult CRP. This association is independent of conventional cardiovascular risk factors, ie, serum lipids, blood pressure, smoking, obesity indices, and insulin. However, the tracking of CRP from childhood to adulthood was lower than the tracking of serum lipids and BMI. In addition, CRP measured in childhood was not associated with adult carotid IMT, whereas childhood LDL–cholesterol, systolic blood pressure, BMI, and smoking were predictive of adult IMT.

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### Disclosure(s)

None.

### References

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