WHAT’S KNOWN ON THIS SUBJECT: Uric acid (UA) is associated with hypertension in children, after body weight adjustment. Whether the whole spectrum of variables, such as visceral adiposity, insulin resistance, puberty, and renal function, influence the relationship between UA and blood pressure is unknown.

WHAT THIS STUDY ADDS: In a cohort of children at relatively high cardiovascular risk, the association between UA and blood pressure levels is independent of several well-known factors implicated in the development of hypertension, such as insulin resistance, pubertal status, and renal function.
Serum uric acid (UA) was shown to be a strong correlate of established hypertension in children. Preliminary studies have also reported an association between hyperuricemia and non-sustained hypertension, and, recently, a relationship between changes in serum UA over time and blood pressure (BP) from childhood to adulthood has been reported.

These findings have sparked growing interest in the relationship between UA and BP in children that may lead to pathophysiological insights and greater knowledge on the cause-effect relationship between these factors.

The complex interaction between serum UA, BP changes, and other well-established risk factors has been elucidated only in part, and controversy remains as to whether UA is an independent causal factor or merely a marker of the development of hypertension. It has been suggested that several factors, such as age, puberty, obesity, insulin resistance, and impaired renal function, all of which are related to UA levels, may play a role in the development of hypertension. In 2 recent trials, serum UA reduction by allopurinol or probenecid resulted in BP normalization in children and adolescents with increased BP levels, thus supporting a causative role for UA in hypertension.

The purpose of the current study was to investigate the relationship between serum UA and BP, as well as several possibly confounding factors, such as puberty, anthropometric parameters, insulin resistance, and renal function in a large group of children at relatively high cardiovascular (CV) risk.

METHODS

Subjects

We studied a cohort of children aged between 6 and 18 years (mean = 10.8 years), consecutively referred by their primary care pediatricians to the San Gerardo Hospital, Unit for Cardiovascular Risk Assessment in Children, because of evidence of elevated BP values and/or because of positive family history of CV disease. The latter was defined as the presence in 1 or both of the parents of at least 1 among the following: hypertension, type 2 diabetes, dyslipidemia, early ischemic heart disease, and cerebrovascular disease.

None of these children were affected by impaired glucose tolerance, diabetes, or renal insufficiency. Specific diagnostic tests that are needed to rule out secondary hypertension were carried out in all children. Children with secondary forms of hypertension were excluded from the study.

Informed consent was obtained from the children’s parents, and the local ethical committee approved the study protocol.

Definition of Terms and Groups

Height, weight (mobile digital scale [SECA, Vogel & Halke GmbH, Hamburg, Germany], precision 100 g), and waist circumference were measured with the subjects in their underwear. BMI was calculated as weight (kg)/height (m²). BMI z-scores were calculated by using the Centers for Disease Control and Prevention charts (available at http://www.cdc.gov/nchs/). Weight was approximated to hectograms, whereas height precision was 5 mm. Weight class was defined according to the tables of the International Obesity Task Force, distinguishing among normal weight (NW), overweight (OW), and obese (OB).

Waist circumference was measured to the nearest 0.1 cm by a nonelastic flexible tape with the children in a standing position. The tape was applied horizontally midway between the lowest rib margin and the iliac crest. Waist-to-height ratio (WHR) was calculated by dividing waist circumference by height. A cutoff of 0.5 was chosen to distinguish between low and high WHR category, as suggested by several authors.

Pubertal stage was assessed by a medical examination and children were classified into 2 categories: prepubertal and pubertal according to Tanner staging. BP was measured by using an aneroid sphygmomanometer with the appropriate cuff for the child’s upper arm size. The sphygmomanometer was calibrated before starting the study and once a month thereafter with a mercury sphygmomanometer. Systolic BP (SBP) was defined by the first Korotkoff sound (appearance of sounds) and diastolic BP (DBP) was identified by the fifth Korotkoff sound (disappearance of sounds). BP values were approximated to the nearest 2 mm Hg. Measurements were performed while children were sitting with their back supported and the cubital fossa supported at the heart level, after a rest of at least 5 minutes. BP was taken 3 times (3–5-minute intervals) and SBP and DBP percentiles were calculated according to the normograms recommended by the National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents. Each child was classified according to the percentile of the mean of the 3 measurements as being normotensive (NT) if both SBP and DBP percentiles were <90th, prehypertensive (PH) if the SBP and/or DBP percentile was ≥90th but both were <95th, or hypertensive (HT) if the SBP and/or DBP percentile was ≥95th. Children who were referred by family pediatricians because of evidence of elevated BP values, but whose SBP and DBP percentiles were both <90th percentile when BP was measured at the Cardiovascular Risk in Children Unit, were classified as having transiently elevated BP (TH). Family history of hypertension was defined as the presence of at least 1 parent with hypertension.

Biochemical Parameters

Blood samples were taken from all subjects after a 12-hour fasting period.
to measure plasma glucose and insulin, and serum UA, total cholesterol, triglycerides, and high-density lipoprotein concentrations. Plasma glucose was measured by a glucose oxidase method and insulin was evaluated by chemiluminescent immunometric assay. Homeostasis Model Assessment (HOMA) index was calculated by dividing the product of plasma insulin (µU/mL) and plasma glucose (mmol/L) by 22.5.24 Glomerular filtration rate was estimated (eGFR) by means of the updated Schwartz formula using serum creatinine and height measurements and a k constant of 0.413.25 Serum creatinine level was measured by using the Jaffe method referable to the standardized reference measurement procedure (isotope dilution mass spectrometry), as recommended.

**Statistical Methods**

The cohort was categorized into groups according to hypertension category, weight class, and UA gender-specific quartiles. The continuous variables were described by mean and SD and compared across groups by Fisher-Snedecor and Student’s t tests. The categorical variables were described by the proportion of subjects falling into each category. Proportions were compared across groups by the χ2 test. Box-plots were used to describe the distribution of UA according to hypertension category and weight class based groups. Correlation between continuous variables was assessed by the Pearson correlation coefficient. Multiple linear regression was used to assess the influence of age, pubertal status, gender, BMI z-score (or WtHr) and eGFR on UA. The impact of UA was considered on SBP z-score and on DBP z-score by a multiple linear regression model adjusting for age, pubertal status, gender, BMI z-score (or WtHr), and HOMA index. Logistic regression models were used to assess the impact of the aforementioned factors on the odds ratio (OR) of being TH, PH, and HT with respect to NT in 3 separate models on the TH versus NT, PH versus NT, and HT versus NT, respectively.

No data were missing for any of the mentioned variables. All analyses and graphics were conducted with the package R (available at http://cran.r-project.org/).

**RESULTS**

Among 648 children consecutively referred to our outpatient clinic for CV risk assessment between November 2004 and March 2012, 147 were excluded (12 because they were <6 years of age, and 135 because data for 1 or more of the variables included in the present analyses were missing). In the resulting final cohort of 501 subjects (55.9% boys and 44.1% girls), mean age was 10.8 years (SD = 2.4 years), 156 (31.1%) were NT, 122 (24.4%) were TH, 87 (17.4%) were PH, and 136 (27.1%) were HT. Among them, 33.3% and 40.5% were OW or OB, respectively.

Elevated BP (PH or HT) was found in 32.1% among 131 children with NW, in 42.5% among 167 OW and 54.2% among 203 OB. Table 1 reports the clinical characteristics of study subjects on the basis of the various BP categories. As expected, there was a trend toward higher BMI z-score and higher percentages of OW or OB, increasing WtHr, and HOMA index as BP categories rose. Moreover, the prevalence of pubertal, OB, children whose WtHr was above 0.50 progressively increased from the lower to the upper BP categories. Despite similarities in age, family history of hypertension, renal function, fasting serum glucose, and lipid profile, the children showed a significant rise in

### TABLE 1 Clinical Characteristics of Study Subjects According to BP Category (501 Children)

<table>
<thead>
<tr>
<th></th>
<th>NT, n = 156</th>
<th>TH, n = 122</th>
<th>PH, n = 87</th>
<th>HT, n = 136</th>
<th>P Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age, y</strong></td>
<td>10.5</td>
<td>10.2</td>
<td>10.7</td>
<td>10.8</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><strong>Gender, %</strong></td>
<td>63 (50.0)</td>
<td>62 (50.8)</td>
<td>60 (50.0)</td>
<td>61 (50.7)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><strong>Puberty, %</strong></td>
<td>56 (55.9)</td>
<td>61 (50.0)</td>
<td>50 (57.5)</td>
<td>69 (50.7)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><strong>Weight, kg</strong></td>
<td>45.8</td>
<td>14.8</td>
<td>14.9</td>
<td>16.5</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><strong>Height, m</strong></td>
<td>1.4</td>
<td>1.0</td>
<td>1.5</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td><strong>BMI, kg/m²</strong></td>
<td>21.6</td>
<td>4.5</td>
<td>22.8</td>
<td>4.1</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><strong>BMI z-score</strong></td>
<td>1.04</td>
<td>1.2</td>
<td>1.37</td>
<td>0.8</td>
<td>0.001</td>
</tr>
<tr>
<td><strong>Weight class, %</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&lt;.001</td>
</tr>
<tr>
<td><strong>NW</strong></td>
<td>61 (38.1)</td>
<td>31 (24.8)</td>
<td>66 (54.1)</td>
<td>68 (50.0)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><strong>OW</strong></td>
<td>83 (53.2)</td>
<td>54 (44.3)</td>
<td>50 (57.5)</td>
<td>42 (30.9)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><strong>OB</strong></td>
<td>56 (55.9)</td>
<td>40 (32.8)</td>
<td>42 (49.4)</td>
<td>36 (41.4)</td>
<td>&gt;.001</td>
</tr>
<tr>
<td><strong>WC, cm</strong></td>
<td>71.8</td>
<td>12.0</td>
<td>74.7</td>
<td>11.6</td>
<td>1.38</td>
</tr>
<tr>
<td><strong>WtHr</strong></td>
<td>49.8</td>
<td>7.4</td>
<td>51.1</td>
<td>6.2</td>
<td>1.58</td>
</tr>
<tr>
<td><strong>WtHr &gt;50%, %</strong></td>
<td>78 (50.0)</td>
<td>66 (54.1)</td>
<td>60 (69.0)</td>
<td>86 (63.2)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><strong>SBP, mm Hg</strong></td>
<td>108.7</td>
<td>7.1</td>
<td>113.2</td>
<td>7.1</td>
<td>13.5</td>
</tr>
<tr>
<td><strong>DBP, mm Hg</strong></td>
<td>64.6</td>
<td>8.6</td>
<td>66.4</td>
<td>8.0</td>
<td>9.5</td>
</tr>
<tr>
<td><strong>BMI, %</strong></td>
<td>0.40</td>
<td>0.56</td>
<td>0.73</td>
<td>0.46</td>
<td>0.37</td>
</tr>
<tr>
<td><strong>BMI z-score</strong></td>
<td>0.20</td>
<td>0.53</td>
<td>0.38</td>
<td>0.50</td>
<td>0.76</td>
</tr>
<tr>
<td><strong>Family history of HT, %</strong></td>
<td>50 (50.0)</td>
<td>30 (50.0)</td>
<td>35 (50.0)</td>
<td>42 (50.0)</td>
<td>.82</td>
</tr>
<tr>
<td><strong>Creatinine, mg/dL</strong></td>
<td>0.02</td>
<td>0.10</td>
<td>0.03</td>
<td>0.12</td>
<td>0.11</td>
</tr>
<tr>
<td><strong>eGFR, mL/min</strong></td>
<td>116</td>
<td>198</td>
<td>118</td>
<td>182</td>
<td>116</td>
</tr>
<tr>
<td><strong>UA, mg/dL</strong></td>
<td>3.9</td>
<td>0.9</td>
<td>4.2</td>
<td>1.1</td>
<td>0.4</td>
</tr>
<tr>
<td><strong>Glucose, mg/dL</strong></td>
<td>85</td>
<td>7.3</td>
<td>82</td>
<td>6.9</td>
<td>82</td>
</tr>
<tr>
<td><strong>Insulin, mM/L</strong></td>
<td>10.8</td>
<td>6.3</td>
<td>10.9</td>
<td>6.8</td>
<td>12.5</td>
</tr>
<tr>
<td><strong>Total cholesterol, mg/dL</strong></td>
<td>168</td>
<td>37.4</td>
<td>157</td>
<td>25.8</td>
<td>163</td>
</tr>
<tr>
<td><strong>HDL cholesterol, mg/dL</strong></td>
<td>58</td>
<td>14.1</td>
<td>55</td>
<td>13.5</td>
<td>54</td>
</tr>
<tr>
<td><strong>Triglycerides, mg/dL</strong></td>
<td>68</td>
<td>35.6</td>
<td>68</td>
<td>35.4</td>
<td>78</td>
</tr>
</tbody>
</table>

Values are mean and SD unless otherwise indicated. HDL, high-density lipoprotein; WC, waist circumference.

* P < .05 was considered statistically significant.

a HOMA index = plasma insulin (µU/mL) × plasma glucose (mmol/L)/22.5.
serum UA levels along with increasing BP levels.

The distribution of UA levels based on weight classes is shown in Fig 1. There was a significant trend in increasing UA levels from NW to OB.

Univariate analysis showed that among the whole study group, UA was significantly related to both systolic BP (SBP) (z-score, \( r = 0.21, P < .001 \)) and DBP (z-score, \( r = 0.11, P = .02 \)).

A multiple regression model showed that the only variables significantly related to SBP (z-score) were BMI (z-score) (P < .001), HOMA index (P = .02), and serum UA (P = .03), whereas UA and pubertal status were related to DBP (z-score) (P = .03 and P = .02, respectively).

When WtHr was included in the multiple linear regression model as a measure of weight excess instead of BMI (z-score), the only variables significantly related to SBP (z-score) were WtHr (P < .001) and UA (P = .03), whereas WtHr was related to DBP (z-score) (P = .01). Including eGFR in the statistical model yielded superimposable results.

Mean UA level was 4.38 (SD = 1.14) in boys and 4.15 (SD = 0.95) in girls (\( t \)-test statistic = -2.5, \( P = .01 \)). Table 2 shows the clinical characteristics of study subjects on the basis of gender-specific serum UA quartiles. It can be noticed that, as UA quartiles increased, children were more likely to be older, pubertal, and OB. Moreover, there was a trend toward higher BMI z-score, WtHr, BP parameters, serum creatinine, and more unfavorable HOMA index and lipid profile.

After adjusting for pubertal status, gender, BMI z-score, and HOMA index, the OR for the presence of HT among the whole population was 1.40 (95% confidence interval [CI] 1.12–1.75, \( P < .01 \)) for each 1 mg/dL increase in UA and 1.67 (95% CI 1.00–2.82, \( P = .05 \)) for subjects in the IV UA quartile (>4.7 mg/dL for girls; >5.0 mg/dL for boys). The impact of UA was not found to be significant when we compared children with TH versus those with NT. Using NT as comparison, the risk of being PH and of being HT increased by at least 50% (\( OR = 1.60 \) and \( P < .01, OR = 1.54 \) and \( P < .01 \), respectively, Table 3) for each 1 mg/dL increase in UA, whereas it doubled (\( OR = 2.25, 95\% CI 1.15–4.38, P = .01 \) and OR = 2.04, 95% CI 1.12–3.73, \( P = .02 \), respectively) for children in the IV UA quartile. The relationship between the distribution of BP categories and UA quartiles is emphasized in Fig 2. When WtHr was used instead of BMI (z-score) as a measure of weight excess, the OR for the presence of HT among children with TH versus those with NT was 2.17, 95% CI = 1.10–4.28 (\( P = .03 \)) and 1.95, 95% CI = 1.06–3.60 (\( P = .03 \)) respectively. Including eGFR in the statistical model yielded almost superimposable results.

**DISCUSSION**

In this large group of children at relatively high CV risk, serum UA was independently associated with BP levels across different BP categories, from normotension to transient and then prehypertension, up to established hypertension.

Although the association between mild hyperuricemia and hypertension has previously been described in several studies both in adults and in children,\(^ 1,3,4,6,26 \) a relationship between UA and prehypertension has mainly been reported in adults,\(^ 27 \) with only a few, relatively small studies carried out in children with white coat and borderline hypertension.\(^ 4,5,16 \)
Although the clinical setting of our study is cross sectional, it provides us with an opportunity to investigate the complex association between UA and other variables (such as insulin resistance, obesity, puberty, and BP) in a group of subjects in whom pathophysiological mechanisms and changes leading to the development of hypertension are still at play. This is even more interesting in light of the recently reported finding that UA levels may be a predictor of the future development of hypertension in adults.5 The relationship between UA and BP that we observed becomes clinically relevant for higher UA values, since children in the top UA quartile (ie, ≥4.7 mg/dL for boys and ≥4.7 mg/dL for girls) showed a twofold higher risk of being PH and HT (Table 3, Fig 2). On the other hand, although a trend toward higher UA values was present across all BP categories, UA levels could not help identify children with nonsustained hypertension. With the aim of better elucidating the role of UA in the development of essential hypertension, in the current study we excluded patients with renal insufficiency and/or diabetes. However, we analyzed the relationship between UA and those parameters, such as eGFR and HOMA index, which, even in the range of normality, could be implicated in the development of hypertension in children.13 Even slightly elevated serum UA levels have shown to be strong, independent predictors of the development of type 2 diabetes in adult cohorts of unselected general populations,28 as well as of HT patients.29,30 Moreover, recent data strongly support the hypothesis that insulin resistance in children might play a role in hypertension development regardless of obesity and fat distribution.13 Other variables, such as puberty and renal function,10,14 have also been shown to bear an influence on the link between UA and BP in children. To date, only a few studies have investigated the relationship of sexual maturity1,2 and estimated renal function1,3,4,31 with UA and BP, and none have taken insulin resistance into consideration. In the current study, HOMA index was found to be linearly related to UA levels and increased along with UA quartiles (Table 2, Fig 1). A trend toward higher UA values was present across all BP categories, and UA levels could not help identify children with nonsustained hypertension. On the other hand, although a trend toward higher UA values was present across all BP categories, UA levels could not help identify children with nonsustained hypertension. The prevalence of OW and OB was relatively high in our study sample, as expected on the basis of recruitment criteria. Nonetheless, the association between UA and BP persisted after adjusting for BMI z-score and for WHtR, a finding that clearly supports a pathogenetic role for serum UA in the early phases of hypertension development.

Table 2 shows that UA gender-specific quartiles (501 children)

<table>
<thead>
<tr>
<th>Variable</th>
<th>TH versus NT, n = 278</th>
<th>PH versus NT, n = 243</th>
<th>HT versus NT, n = 292</th>
</tr>
</thead>
<tbody>
<tr>
<td>OR</td>
<td>(95% CI)</td>
<td>p</td>
<td>OR</td>
</tr>
<tr>
<td>Puberty, boys</td>
<td>1.97 (1.13–3.43)</td>
<td>.01</td>
<td>1.09 (0.58–2.06)</td>
</tr>
<tr>
<td>Gender, boys</td>
<td>1.11 (0.67–1.84)</td>
<td>.69</td>
<td>1.15 (0.65–2.03)</td>
</tr>
<tr>
<td>BMI, z-score</td>
<td>1.53 (1.15–2.05)</td>
<td>.01</td>
<td>1.43 (1.03–1.98)</td>
</tr>
<tr>
<td>UA, mg/dL</td>
<td>1.17 (0.89–1.54)</td>
<td>.26</td>
<td>1.60 (1.15–2.22)</td>
</tr>
<tr>
<td>HOMA index</td>
<td>0.82 (0.67-1.01)</td>
<td>.06</td>
<td>0.98 (0.77–1.18)</td>
</tr>
</tbody>
</table>

* P < .05 was considered statistically significant.

HOMA index = plasma insulin (μU/mL) × plasma glucose (mmol/L)/225.
and logistic regression analysis, the relationship between this marker of insulin resistance and BP levels and categories almost completely loses its strength, whereas serum UA maintains its independent link to BP. Although these results cannot help to define whether serum UA is influenced by BP elevation and insulin resistance or vice versa, they certainly point toward a complex relationship among these factors.

As for renal function, we found that eGFR was similar in all BP categories, and the addition of this variable to linear and logistic regression analyses left the relationship between UA and BP unchanged. Even accounting for body mass and puberty, the independent role of UA variations on BP values did not decrease.

Our data confirm and expand those previously reported by Loeffler et al. in a large study conducted on a representative sample of adolescents in the United States. In fact, we were fortunate enough to obtain a larger study sample from our records and therefore were able to demonstrate an association between SBP as well as DBP and serum UA levels even in a fully adjusted multivariate linear regression analysis.

Our study has both strengths and weaknesses that deserve to be commented on. Among the former, the fact that, unlike other previous large studies, the definition of elevated BP and the allocation of children into different BP categories was not based on a single physical examination. BP was measured in at least 2 different occasions: previously by the family pediatrician and then again in our clinic, and in both cases the mean of 3 measurements was used for analysis. To fully account for age, gender, and height, according to the tables published in the fourth report on the diagnosis, evaluation, and treatment in children and adolescents, we used BP z-scores rather than raw BP values, which makes our observations more reliable as compared with those of a variety of previous reports.

Furthermore, to the best of our knowledge, for the first time several clinical and biohumoral variables have been concurrently collected and analyzed in a group of referred children.

Last, there are some limitations to our study. First, given the cross-sectional nature of its design, it would be inappropriate to derive definitive conclusions regarding the cause-effect relationship between UA and BP levels, even though the association we report persisted even when several well-known confounding factors were taken into account. Second, the lack of a control group may limit the implications of our observations. Nevertheless, within the context of children with relatively high CV risk, our data show a clear trend in the BP-UA association along with progressively worse BP categories, from transient to established hypertension. Higher UA levels show a significant association with the occurrence of higher BP levels even among a cohort of children at relatively high CV risk. Third, ambulatory blood pressure monitoring was not performed in our study and therefore we could not classify any subject into the “white-coat hypertension” or “masked hypertension” category. However, there is a general consensus in current pediatric literature that diagnosis of hypertension in children and adolescents be based on office BP measures and not on ambulatory blood pressure monitoring.

**CONCLUSIONS**

In children at relatively high CV risk, UA shows a strong relationship with BP values across different BP categories, from normal BP to transient, up to pre- and finally to established hypertension. The association between UA and BP levels is independent of several well-known factors potentially implicated in the development of hypertension, such as insulin resistance, pubertal stage, and renal function. These data support the need for further large, prospective, and interventional studies to clarify the pathophysiological mechanisms underlying the relationship between serum UA and BP.
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Serum Uric Acid and Blood Pressure in Children at Cardiovascular Risk

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