Original Article

Hyperuricemia and metabolic syndrome in Taiwanese children

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Metabolic fitness in childhood is of increasing concern in transitional and advanced economies as the metabolic syndrome (MS) is recognized more often in this age group. As the MS appears, so also does hyperuricemia. Studies in Taiwan have identified both indigenous and Chinese with high prevalence of hyperuricemia. Data (1227 boys and 1057 girls, aged 6-12) from the Nutrition and Health Survey in Taiwan Elementary School Children (NAHSIT Children 2001-2002) were used to appraise the association between uric acid (UA) and MS in children. Mean serum urate increases by age, ranging from 5.69 mg/dL to 7.11 mg/dL for boys and 5.61 mg/dL to 6.13 md/dL for girls. Boys have higher UA concentrations (6.07 mg/dL vs. 5.74 mg/dL) and hyperuricemia (UA ≥ 7 mg/dL) rate (26.5% vs. 18.8%) than girls. Children of Mountain areas have higher rates of hyperuricemia (boys: 39.2%, girls: 30.1%). 5.56% of boys and 6.39% of girls were classified as having the MS by ATP III criteria. Serum urate was closely correlated with the MS parameters, and waist circumference (WC) in particular (r=0.387). WC alone accounted for 18% of variance of serum urate concentration. Both serum urate and hyperuricemia are significant risk factors for the MS (serum urate in mg/dL, OR: 1.54, 95% CI: 1.36-1.74; hyperuricemia, OR: 3.73, 95% CI: 2.47-5.62). Adjustment for age and region accentuate these relationships. Not only abdominal fatness, but also uric acid status, or both together may be of interest to public health workers and clinicians in regard to the transitional health problem of MS.

Key Words: serum urate, hyperuricemia, metabolic syndrome, abdominal obesity, elementary school children, Taiwan, Nutrition and Health Survey in Taiwan Elementary School Children (NAHSIT Children 2001-2002)

INTRODUCTION

There is increasing recognition of antecedents of chronic disease like diabetes and cardiovascular disease in early life.[1,2] Perhaps the most widespread risk cluster for such diseases is the so-called "metabolic syndrome" (MS).[3-6] This usually refers to the combination of abdominal obesity, insulin resistance, hypertension and dyslipidemia with hypertriglyceridemia and low HDL, and may refer to prothrombotic tendencies and the presence of inflammatory markers;[3-6] it is commonly associated with hyperuricemia as well.[7-10] There is some evidence from both cross-sectional and longitudinal observations that high uric acid in childhood may be prognostic of hypertension later in life in both Caucasians and African Americans.[11-13]

We and others have previously documented that there are relatively high prevalences of hyperuricemia in adult and elderly oriental populations, in particular indigenous people.[14-17] It is of interest to check whether this phenomenon applies across different age groups. Moreover, the opportunity is provided through a population representative sample from the Nutrition and Health Survey in Taiwan Elementary School Children (NAHSIT 2000-2001)[18] to consider possible links between uric acid status and MS in children from several Chinese and indigenous ethnic groups living in different areas of Taiwan.[19,20]

MATERIALS AND METHODS

Study participants

Data for this paper were obtained from the Nutrition and Health Survey in Taiwan Elementary School Children (NAHSIT Children 2001-2002). The NAHSIT Children 2001-2002 was a representative national survey aimed at studying the nutrition and health status of elementary school children aged 6-12 years in Taiwan. We classified 359 townships/districts in Taiwan into 13 strata according to the dietary patterns of the residents, degree of urbanization, and the geographic characteristics of the selected areas. A "probability proportional to population size" (PPS) method was

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used to select 104 schools (8 from each stratum) randomly. From each school, in turn, 24 students were drawn randomly, stratified by gender and age. A total of 1227 boys and 1057 girls underwent a physical examination and had available complete questionnaire information and serum urate data, making them eligible for inclusion in the present analysis. Informed consent has been signed by one of the parents of all participants. The study was approved by reviewers from the Department of Health in Taiwan. More details about the study design and sampling methodology are provided by Tu *et al*.[18]

Data collection

A face-to-face interview approach was used to collect information from both the parent/care giver and the child. Data included gender, age, residential location, physical activity, medical history and the use of medications. Detailed physical examination included anthropometric measurements and fasting blood samples were collected. The blood specimens were centrifuged immediately after collection. The serum specimens were aliquoted, frozen in a liquid nitrogen tank, and then delivered to the Academia Sinica where they were stored at -70°C. The frozen serum samples were analyzed in the clinical laboratory of the National Taiwan University Hospital within one month of collection (using the Hitachi 747, Japan).

Definition of variables

Hyperuricemia was defined by serum urate ≥ 7.0 mg/dL (416 μM) regardless of gender.[21, 22] The definition of metabolic syndrome was that used for adolescents in the third National Health and Nutrition Examination Survey, and modified from the new Adult Treatment Panel definition (NCEP-ATP III)[3, 4] with a revised fasting blood glucose criteria.[5] Participants needed to fulfill 3 or more of the following criteria: (1) waist circumference ≥ 90th percentile value for age (by year) and sex from this population representative sample; (2) serum triglyceride ≥ 110 mg/dL (1.24 mM); (3) HDL ≤ 40 mg/dL (1.03 mM); (4) SBP/DBP ≥ 90th percentile value for age (by year) and sex from this population representative sample ; (5) fasting blood glucose ≥ 100 mg/dL (5.55 mM). The impact of monthly variation in serum urate levels was found to be substantial in this population. This effect was adjusted for in regression analysis.

Statistical analysis

For descriptive purpose, all data were weighted to represent the elementary school children population in Taiwan. The sampling weight of each surveyed individual was calculated by dividing the sample size by the population size of his or her own sex/age group in the stratum. The SUDAAN version 8.0[23] was used to account for the effect of this multi-staged complex sampling design. We used Pearson correlation, partial correlation, regression and logistic regression without or with adjusting for other related factors to elucidate the relationship between serum urate and MS and its components regardless of the design effect of sampling. Statistical significance was defined as $p<0.05$.

RESULTS

Table 1 shows the gender-specific mean serum urate and MS parameters and the corresponding abnormal rates. Mean serum urate increases by age, ranging from 5.69 mg/dL (338 μM) and 5.61 mg/dL (334 μM) for 6 year old to 7.11 mg/dL (423 μM) and 6.13 md/dL (365 μM) for 12 year old boys and girls, respectively (data not shown). Boys have higher mean serum urate concentrations than girls (6.07 mg/dL vs. 5.74 mg/Dl; 361 μM vs. 341 μM). Using 7 mg/dL (416 μM) as the cut for hyperuricemia, the prevalence rates were 26.5% for boys and 18.8% for girls. Both boys and girls of Mountain areas have relatively higher prevalence rates of hyperuricemia, boys: 39.2%, ranked the first, girls: 30.1%, ranked the second (data not shown).

Regarding the MS components, 36.3% boys and 29.4% girls have serum glucose higher than 100 mg/dL with a mean of 97.3 mg/dL for boys and 95.6 mg/dL for girls. Prevalence of serum HDL less than 40 mg/dL is the lowest among the five metabolic syndrome components. Overall, 5.56% of boys and 6.39% of girls were classified as having the MS. The youngest had the lowest rate, however, no apparent age-sex pattern was found (data not shown).

Table 1. Sex-specific mean serum urate and the metabolic syndrome components; prevalence of hyperuricemia and of abnormal parameters of the metabolic syndrome; and of the metabolic syndrome itself in Taiwanese children aged 6-12 years [†]

Parameters	Boys			Girls		
	Mean	SE	Abnormal rate (%) [‡]	Mean	SE	Abnormal rate (%)
Serum urate (mg/dL)	6.07	0.06	26.5	5.73	0.07	18.8
Serum glucose (mg/dL)	97.3	0.35	36.3	95.6	0.44	29.4
Serum triglyceride (mg/dL)	74.0	1.45	11.7	77.1	1.20	15.1
Serum HDL (mg/dL)	59.5	0.46	4.38	58.5	0.55	5.99
Systolic blood pressure (mm-Hg)	98.4	0.36	10.7	96.3	0.31	11.6
Diastolic blood pressure (mm-Hg)	57.5	0.30	9.27	57.4	0.29	9.78
Waist circumference (cm)	60.7		10.4	56.9		10.6
Metabolic syndrome [§]			5.56			6.39

[†] All values are weighted to reflect their representation in the population; SE: standard error. [‡] Hyperuricemia is defined as serum urate ≥ 7.0 mg/dL (416 μM), the abnormal rate of individual components of metabolic syndrome were defined as: (1) waist circumference (WC) ≥ 90th percentile value for age (yr) and sex from this population representative sample; (2) serum triglyceride (TG) ≥ 110 mg/dL (1.24 mM); (3) HDL (high density lipoprotein cholesterol) < 40 mg/dL (1.03 mM); (4) SBP/DBP ≥ 90th percentile value for age (yr) and sex from this population representative sample; (5) fasting blood glucose (BG) ≥ 100 mg/dL (5.55 mM). [§] It requires the presence of 3 or more of the individual parameters to be abnormal.

Table 2. Pearson correlation coefficients† between concentration of serum urate and individual components of the metabolic syndrome in Taiwanese children aged 6-12 years

		Serum glucose (mg/dL)	Serum TG (mg/dL)	Serum HDL (mg/dL)	SBP (mm-Hg)	DBP (mm-Hg)	Waist circumference (cm)
Serum urate (mg/dL)	Crude	0.063*	0.122**	-0.193**	0.263**	0.141**	0.387**
	Age-adjusted	0.030	0.127**	-0.182**	0.207**	0.102**	0.346**

† Sample size for individual analysis was varied from 2232 to 2284. * $p < 0.005$, ** $p < 0.001$

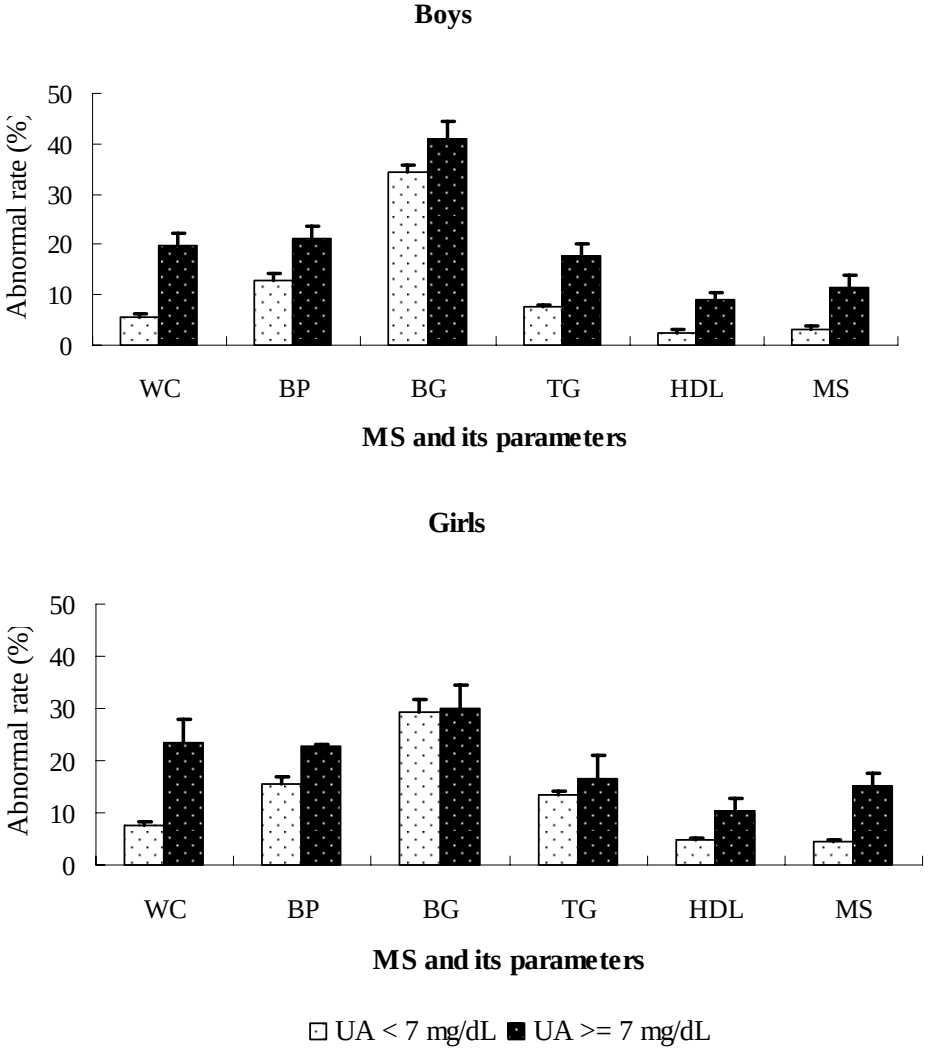

Figure 1. Prevalence (with standard error) of abnormal metabolic syndrome (MS) parameters and of the overall MS index, stratified for uric acid (UA) abnormality. Hyperuricemia is defined as serum urate ≥ 7.0 mg/dL (416 μM). Abnormalities of individual MS parameters and MS were defined as in Table 1.

Significant correlation coefficients were found between serum urate and parameters of MS (Table 2). The biggest Pearson correlation coefficient between serum urate and individual components of MS was 0.387 for waist circumference, while the smallest was serum glucose, which was 0.063. After adjusting for age, the coefficients, except for blood glucose, did not alter substantially. Stepwise regression showed that waist circumference (treated as continuous variable) alone accounted for more than 18% of variance of serum urate concentration (data not shown).

The prevalence of those whose individual MS parameters were abnormal and of the MS overall, stratified by serum urate and gender is illustrated in Fig 1. The largest differences in rate of abnormality between two uric acid groups in both genders were found in waist circumference, which were 14.2% (5.65% vs. 19.8%) for boys and 15.9% (7.69% vs. 23.6%) for girls, respectively. In terms of percent of difference, it was 250% for boys and 207% for girls. Both serum urate and hyperuricemia were significant predictors of MS. For every 1 mg/dL increase in serum urate, there was a 54% elevation in risk for MS (OR: 1.54, 95% CI: 1.36-1.74). For those whose serum urate was higher than 7 mg/dL, there was a 3.7 fold more MS (OR: 3.73, 95% CI: 2.47-5.62). After adjustment for gender, age, region (stratum) and monthly variation, the

Table 3. Risk of metabolic syndrome[†] predicted by uric acid in Taiwanese children aged 6-12 years (n = 1777)

	Uni-variable	Multi-variable [§]
	OR (95% CI)	OR (95% CI)
Serum urate (mg/dL)	1.54 (1.36-1.74)*	1.64 (1.44-1.88)*
Hyperuricemia [‡]	3.73 (2.47-5.62)*	4.22 (2.74-6.51)*

[†] Metabolic syndrome was defined as in Table 1. [‡] Hyperuricemia is defined as serum urate ≥ 7.0 mg/dL (416 μM). [§] Adjusted for gender, age (in year), monthly variation, regions; OR: odds ratio; CI: confidence interval. * $p < 0.001$

Table 4. Comparisons of family characteristics and parental demography between indigenous and non-indigenous children[†]

	Population	
	Indigenous (n=191)	Non-indigenous (n=1926)
Serum urate (Mean ± SD, mg/dL)	6.32±1.56	5.90±1.50
Paternal education level (%)		
Elementary school	20.3	5.83
Junior high school	36.1	24.1
Senior high school	36.0	41.9
College and above	7.57	28.2
Maternal education level (%)		
Elementary school	35.0	6.99
Junior high school	32.4	22.7
Senior high school	29.3	50.3
College and above	3.29	20.0
Paternal perceived health status (%)		
Very good	9.91	10.6
Good	22.5	36.1
Fair	52.7	48.4
Not good	13.1	4.53
Very bad	1.78	0.40
Maternal perceived health status (%)		
Very good	9.47	5.24
Good	14.6	28.8
Fair	63.8	57.4
Not good	11.1	8.06
Very bad	1.02	0.51
Family income (1000 NTD/month)		
< 40	43.8	22.6
40-80	44.2	50.2
>80	12.0	27.2
Poor living conditions[‡] (%)	6.78	2.04

[†] All values are weighted to reflect their representation in the population. [‡] These were characterized by a corrugated iron shelter, by being a squatter or living in a rooftop annex.

magnitudes were even stronger (Table 3).

DISCUSSION

In a comparison between three countries (Japan, USA and Taiwan), the highest mean serum uric acid was found in Taiwanese children. The differences in mean serum urate can be up to 2 mg/dL for boys and 1.5 mg/dL for girls.[9, 24] This high serum urate phenomenon is persistent from childhood to later life and applies to both genders in Taiwan. Children from the Mountain areas, mainly indigenous, have relatively higher levels of serum urate and rates of hyperuricemia. This is similar to findings for adults in general and for the elderly in particular.[14,15]

In the present study, significant correlations were found between serum urate and individual parameters of the MS. Waist circumference, as a surrogate for abdominal obesity accounts for the biggest variance in serum uric acid concentration, which is similar to findings in other countries.[7,8] This confirms that abdominal fatness is the likely basis of the metabolic abnormality which constitutes the insulin resistance syndrome.

Elevated fasting blood glucose may be a prerequisite for hyperuricemia in susceptible children who develop abdominal fatness. Since fasting glucose represents hepatic nocturnal gluconeogenesis, driven by free fatty acid flux from omental fat, this is plausible.[25] It is conceivable that uric acid, along with lactic acid and redox status, is a determinant of gluconeogenesis. Alternatively, with increased keto acid formation overnight, in those who are abdominal fat, uric acid will rise because of competition between acids for renal excretion;[22,26,27] of course, there may also be a genetic predisposition among some ethnic groups for under excretion of uric acid to further compound this linkage between abdominal fatness, hyperuricemia and higher blood glucose concentrations (i.e., the metabolic syndrome).[27]

The phenomenon of exaggerated nucleic acid turnover during growth in some children, perhaps with a genetic basis, has been taken into account in the present study by adjustment of uric acid status for age (uric acid production declines with age in children in accordance with changes in growth velocity).[28] Data have also been adjusted for region (stratum) since it is possible that such over-production of purines and uric acid may be peculiar to the indigenous mountain population of Taiwan.[19,20] Generally speaking indigenous children have higher serum uric acid concentrations than their Han counterparts (Table 4). This is of particular interest since, worldwide, indigenous people in transitional economies ultimately demonstrate more of the metabolic syndrome than others and hyperuricaemia may be a precursor of it.[29] The circumstances in which these Taiwanese indigenous children reveal such a metabolic disorder are ones where the parents, especially the mothers, are less educated, the economic and living conditions less good, and the sense of well-being and health inferior. These findings lead to a broader and socially important understanding of how prevention and correction of these phenomena may proceed.

The interrelationships between uric acid status and abdominal obesity, insulin resistance, hypertension and dyslipidemia are almost certainly complex and multidirectional. Based on the results of the present study, there are ways in which uric acid status may be determined by the MS or contribute to it.[9] We have calculated the gender-specific attributable risk measures of hyperuricemia in relation to MS in children, and an estimated 41.5% of boys and 30.5% of girls would not have MS if hyperuricemia was eliminated. The population attributable risk percent in Taiwanese children were more than twice higher than in elderly (men: 18.8%, women: 15.5%.) (data not shown).[15]

Nevertheless, the predictive power of hyperurceima in childhood for hypertension in later life in the Bogalusa

Study suggests that the measurement of uric acid status in childhood may be a useful public health measure for early recognition of the MS.[11,12,22] In turn, this may provide opportunities for early intervention and prevention. This may be particularly so in populations where small increments in abdominal fatness have great consequences for disordered energy and substrate metabolism.[29,30] Whether the specific reduction in serum urate could play a role in the reduction, and causality, of the MS is less clear, but possible.[31] The value of observations like the present ones of differential susceptibility to hyperuricemia among children is that detection can be convenient (with finger-prick blood and dry chemistry) and of considerable public health value.

However, although uric acid might be as discriminant as abdominal fatness for the MS at the population level, the cut off point for individual children may be arguable and require clinical judgment on factors not well incorporated into routine practice (like family history of hyperuricemia and cardiovascular risk in childhood).

It is known that fructose raises uric acid concentration, and that uric acid in turn increases the risk of hyperinsulinemia and the metabolic syndrome.[31] It is conceivable that differences in prevalence of hyperuricemia and the metabolic syndrome among children may relate to fructose intake, especially from soft drinks. We plan to analyze our food and beverage intake data on Taiwanese children[32] in regard to this question in a future publication.

In conclusion, consistent with the results of other age groups in Taiwan, we found a relatively high serum uric acid level and prevalence of hyperuricemia in children. In addition, the mean uric acid level and prevalence of hyperuricemia in people from the Mountain areas, mainly indigenes, were even higher. Our data show that the metabolic disorders of MS, abdominal fatness in particular, were significantly associated with hyperuricemia in Taiwanese children. It would be appear that, not only abdominal fatness, but also uric acid status, or both together may be of interest to public health works and clinician in regard to the transitional health problem of MS.

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AUTHOR DISCLOSURES

Meei-Shyuan Lee, Mark L. Wahlqvist, Hsiao-Li Yu, and Wen-Harn Pan, no conflicts of interest.

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Original Article

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臺灣兒童之高尿酸血症與代謝症候群

兒童代謝適能在過渡及高度經濟發展地區漸受關切，而代謝症候群(MS)在這個年齡層也愈常見。當 MS 出現，高尿酸血症也同時出現。臺灣的研究指出原住民及華人其高尿酸血症盛行率均極高。採用臺灣學童營養及健康調查(男：1227 人；女：1057 人)的資料，評估尿酸(UA)及 MS 在兒童的關係。平均血清 UA 隨年齡上升，男童範圍為 5.69 mg/dL 到 7.11 mg/dL，女童為 5.61 mg/dL 到 6.13 md/dL。男童 UA 濃度(6.07 mg/dL vs. 5.74 mg/dL)及高尿酸血症(UA ≥ 7 mg/dL)(26.5% vs. 18.8%)均較女童高。用 ATP III 標準，5.56%的男童及 6.39%的女童有代謝症候群。血清尿酸與 MS 指標緊密相關，尤其腰圍(r=0.387)。腰圍本身可解釋 18%血清尿酸的變異。血清尿酸及高尿酸血症均是代謝症候群的危險因數(UA 每 1 mg/dL, OR: 1.54, 95% CI: 1.36-1.74; hyperuricemia, OR: 3.73, 95% CI: 2.47-5.62)，控制年齡及區域後，關係更明顯。公共衛生及臨床工作者對代謝症候群這個變化中的健康問題，應多重視腹部肥胖或尿酸。

關鍵字：血清尿酸、高尿酸血症、代謝症候群、腹部肥胖、國小學童、臺灣、臺灣學童營養及健康調查 (NAHSIT Children 2001-2002)。