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SERUM URIC ACID AS A RISK FACTOR FOR CARDIOMETABOLIC DISEASE IN CHILDREN: A SYSTEMATIC REVIEW

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ABSTRACT

Background: Cardiometabolic diseases (CMD), including obesity, hypertension, and metabolic syndrome (MS), are increasingly prevalent in pediatric populations. Early identification of biomarkers associated with cardiometabolic risk is essential for effective prevention. Serum uric acid (UA), traditionally linked to gout and renal disease, has recently emerged as a potential marker and mediator of metabolic dysfunction.

Aim: This systematic review aimed to evaluate the relationship between serum UA levels and cardiometabolic risk factors in children and adolescents and to determine whether UA functions as an independent risk factor or a biomarker of CMD.

Methods: A systematic literature search was conducted using PubMed, ScienceDirect, and Google Scholar for studies published up to 2025. Keywords included “uric acid,” “children,” “adolescents,” “cardiometabolic risk,” “metabolic syndrome,” “obesity,” “insulin resistance,” and “hypertension.” Eligible studies included original research and reviews involving individuals aged 0–18 years. A qualitative analysis was performed focusing on associations between UA and specific cardiometabolic conditions.

Results: Elevated UA levels were consistently associated with obesity, MS, hypertension, and early markers of atherosclerosis in children. UA showed strong correlations with body mass index and waist circumference and may help distinguish between metabolically healthy and unhealthy obesity phenotypes. Higher UA levels were also linked to primary hypertension and appeared to precede its development. Additionally, UA was associated with increased carotid intima-media thickness, suggesting a role in early vascular changes. However, the strength of these associations was sometimes reduced after adjusting for confounding factors such as adiposity and insulin resistance.

Conclusions: Serum UA is closely associated with multiple cardiometabolic risk factors in pediatric populations and may serve both as a biomarker and a potential contributor to early disease processes. Although its causal role remains uncertain, UA represents a promising, accessible marker for early risk stratification. Further longitudinal and interventional studies are needed to clarify its clinical utility and determine whether reducing UA levels can improve cardiometabolic outcomes in children.

KEYWORDS

Uric Acid, Children, Cardiometabolic Risk, Metabolic Syndrome, Obesity, Hypertension, Atherosclerosis, Biomarkers

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Introduction

Cardiometabolic diseases (CMD), including obesity, insulin resistance, hypertension (HT), dyslipidemia, and metabolic syndrome (MS), have become increasingly prevalent among children and adolescents worldwide. This alarming trend is largely attributed to lifestyle changes, including reduced physical activity and increased consumption of high-calorie, fructose-rich diets. Early onset of cardiometabolic abnormalities is associated with accelerated atherosclerosis and significantly increased risk of cardiovascular morbidity and mortality in adulthood. Therefore, identifying early biomarkers that can predict cardiometabolic risk in pediatric populations remains a key priority in preventive medicine.

Serum uric acid (UA), the final product of purine metabolism, has traditionally been considered in the context of gout and renal disease. However, over the past two decades, UA has emerged as a potential marker and mediator of cardiometabolic dysfunction. Increasing evidence suggests that elevated UA levels are associated with obesity, insulin resistance, HT, and MS in both adults and children [1–4]. Importantly, pediatric studies indicate that these associations may appear early in life, even before overt metabolic disease develops.

The pathophysiological mechanisms linking UA to CMD are complex and multifactorial. Experimental studies have shown that UA may induce oxidative stress, impair endothelial function, stimulate inflammatory pathways, and activate the renin–angiotensin system [5].

Despite these findings, the role of UA in CMD remains controversial. Some researchers argue that hyperuricemia is merely a consequence of metabolic disturbances, particularly obesity and insulin resistance, rather than an independent causal factor [6].

Although numerous studies have explored the association between UA and cardiometabolic risk factors in children, the findings are heterogeneous, and a comprehensive synthesis of the evidence is needed.

The aim of this systematic review is to evaluate the relationship between serum UA and cardiometabolic risk factors in children and adolescents and to determine whether UA acts as an independent risk factor or a biomarker of CMD.

Material and Methods of Research

This study was conducted as a systematic review. The objective was to comprehensively evaluate the association between serum UA levels and cardiometabolic risk factors in pediatric populations. A structured literature search was performed using electronic databases, including PubMed, ScienceDirect, and Google Scholar. The search strategy included combinations of the following keywords: “uric acid,” “children,” “adolescents,” “cardiometabolic risk,” “metabolic syndrome,” “insulin resistance,” “obesity,” and “hypertension.” The search was limited to studies published up to the year 2026 and conducted in human populations.

Articles published in English were included. To ensure a comprehensive overview of the topic, both original research studies and review articles were included. Inclusion criteria focused on studies primarily involving populations aged 0–18 with CMD. The focus was primarily on studies that analyzed UA levels in relation to specific medical conditions. The selected literature underwent a qualitative analysis, with an emphasis on identifying recurring themes, methodological approaches, and gaps in the current state of knowledge.

Metabolic syndrome

MS is the coexistence of risk factors for the development of CMD and diabetes, such as abdominal obesity, elevated blood pressure, hyperlipidemia, and increased fasting blood glucose levels. In adults, MS is diagnosed when at least three of the following five criteria are present: increased waist circumference (WC) with a threshold based on reference organization guidelines; blood pressure $\geq 130/85$ mmHg or ongoing antihypertensive treatment; fasting blood glucose ≥ 100 mg/dL or ongoing treatment for diabetes; triglycerides (TG) ≥ 150 mg/dL; high-density lipoprotein (HDL) cholesterol < 40 mg/dL in men and < 50 mg/dL in women, or ongoing treatment for dyslipidemia [27].

Currently, there is no universally accepted definition of MS in children and adolescents. Diagnosis is made based on a combination of clinical presentation and laboratory findings. The diagnosis of MS in children also depends on the specific definition used, with higher prevalence rates observed when insulin is included and when pediatric-specific cutoff values for metabolic indicators are applied. The definition proposed by the International Diabetes Federation (IDF) identifies WC as the central component of the syndrome. Additionally, it serves as an independent predictor of insulin resistance, lipid levels, and blood pressure [28]. The ability to determine risk factors in children is based solely on age- and sex-specific thresholds, in which percentiles play a key role. MS is strongly associated with an increased risk of diabetes and CMD. Therefore, focusing on MS prevention in childhood is of great importance for health in adulthood.

Recent studies in hypertensive pediatric populations suggest that UA may represent an additional nonclassical component of MS. In children and adolescents with HT, UA levels were significantly higher in patients with MS compared with those without MS (6.3 vs. 5.5 mg/dL), and UA concentrations increased progressively with the number of MS components present. These findings support the concept that hyperuricemia reflects the cumulative metabolic burden rather than an isolated biochemical abnormality [29].

Importantly, incorporation of UA into MS definitions improved prediction of hypertension-mediated organ damage (HMOD), particularly left ventricular hypertrophy (LVH), suggesting that UA may enhance early cardiovascular risk stratification in HT children and adolescents [29].

Childhood obesity

Excess adipose tissue in childhood and adolescence is a cause of early changes in metabolism, blood vessels, and organs, which may consequently lead to serious cardiometabolic complications, such as an increased risk of cardiovascular dysfunction, HT, dyslipidemia, and type 2 diabetes [7]. Since 1980, the proportion of children with overweight and obesity has increased by 47.1% [8]. Its multifactorial nature makes it a serious problem and a significant global public health challenge related to both treatment and prevention.

Although recent studies from several countries worldwide suggest a stabilization in the prevalence of obesity, especially among younger children, its prevalence among adolescents continues to rise. Increasing evidence indicates that a subgroup of overweight and obese individuals, referred to as “metabolically healthy obesity” (MHO), appears to be less susceptible to the typical metabolic disorders associated with obesity. In contrast, individuals with metabolically unhealthy obesity (MUO) experience these disorders much more frequently [9]. MHO is characterized by a favorable metabolic profile, including preserved insulin sensitivity, increased physical activity, absence of MS criteria, low levels of inflammatory markers, and lower liver fat content [10]. These factors may differentiate individuals who are obese but metabolically healthy from those who exhibit metabolic disturbances. There are relatively few studies on MHO focusing on children and adolescents. The prevalence of MHO in children is highly variable, ranging from 6% to 75%, depending on the classification system used to define this condition [11]. Therefore, it is necessary to investigate the underlying mechanisms present in metabolically healthy and unhealthy individuals among those with overweight and obesity.

Studies aimed at identifying new biomarkers may be helpful, as such indicators could become important tools for identifying future diseases, making accurate predictions, and planning preventive strategies. In recent years, many publications have reported serum UA levels as a marker for the occurrence of MS, type 2 diabetes, and cardiovascular events [12]. Moreover, it can be inferred that serum UA levels may serve as an indicator of the development of adverse cardiometabolic outcomes in obese children and adolescents.

In a study conducted among 246 overweight or obese individuals and 212 normal weight aged 6 to 18 years. Those without identified cardiovascular risk factors (fasting serum lipid levels, blood pressure, and glucose concentration) were classified as MHO. One-third of the participants, however, met at least one criterion for cardiovascular risk factors and were classified as MUO. The researchers concluded that serum UA levels may serve as an indicator of unhealthy obesity in adolescents [12].

UA concentrations were significantly higher in children with overweight or obesity than in their peers with normal body weight. Similar conclusions were also drawn from a study in which UA levels were significantly correlated with WC- and body mass index (BMI)-standard deviations score (SDS) [13]. In fact, elevated UA levels may contribute to endothelial dysfunction by exerting antiproliferative effects on the endothelium and impairing nitric oxide production; consequently, they are associated with an increased risk of CMD [14].

In addition, elevated UA levels (>6.4 mg/dL) in adolescents with obesity have been shown to be significantly associated with hyperglycemia and hypertriglyceridemia. Prevalence of both of those conditions in this group was significantly higher even than in group with UA levels 5.21-6.4 mg/dL [30].

Childhood primary hypertension

HT is one of the most common CMD, affecting nearly 25% of adults, and its prevalence increases with age. By significantly elevating the risk of myocardial infarction, congestive heart failure, stroke, peripheral arterial disease, and renal failure, it leads to increased morbidity and mortality [15].

Between 25% and 40% of adult patients with untreated HT have hyperuricemia (>6.5 mg/dL), and many others have serum UA levels in the upper normal range (5.0–6.5 mg/dL) [16]. Hyperuricemia is an independent risk factor for HT. Early HT is dependent on the renin–angiotensin system and nitric oxide pathways [17]; however, once preglomerular vascular changes occur, HT becomes driven by the kidneys, and lowering UA levels is no longer protective [18]. Preventing microvascular renal damage in childhood may therefore help reduce the incidence of HT later in life.

HT is relatively uncommon in children and adolescents; however, elevated blood pressure in childhood is a strong predictor of HT in adulthood. Numerous studies have demonstrated HMOD, including LVH [19] and microalbuminuria [20], in many hypertensive children. Therefore, preventing the development of HT is one of the key strategies for reducing long-term morbidity.

In children, HT is often secondary to renal, cardiovascular, or endocrine disorders. In some of cases, an underlying cause can be identified, however, in significant number of children, no cause is found, and primary HT (PH) is diagnosed. It is more likely in those with a significant family history of HT. Additionally, about half of children with PH are obese and almost always develop HT in adulthood [4]. Other sources indicate that these figures may be as high as 66.9% among patients with PH in the age group of 13 to 18 years [22].

UA may play a role in the early pathogenesis of PH. In a study of 125 children aged 6 to 18 years, serum UA levels were found to be directly correlated with blood pressure in previously untreated patients. Levels >5.5 mg/dL were observed in as many as 89% of patients with PH. In contrast, elevated UA levels were found in only 30% of patients with secondary HT (SH) and were not observed in those with white-coat HT. These findings suggest that hyperuricemia in an adolescent evaluated for HT strongly indicates PH[4]. Based on the above study, it can be inferred that serum UA contributes to the development of HT. Elevated levels of UA are a known, independent predictive factor for the development of HT, preceding its onset. Furthermore, administration of UA in humans causes endothelial dysfunction and impairs vessel dilatation, which is one of the characteristic features of PH [21]. In children with SH, a similar level of UA was not observed, so it can be stated that it is not only a marker of HT but also its predictor.

Among the conducted studies, a relationship can be observed between hyperuricemia as an independent predictor of HT. This is particularly evident in younger populations and is associated with the aforementioned vascular damage, including increased carotid intima-media thickness (cIMT) [3].

More recent large-cohort pediatric data confirmed that serum UA levels are significantly higher in PH compared with SH (5.6 vs. 4.7 mg/dL; $p < 0.001$). This difference was bigger, when only patients with normal glomerular filtration rates (GFR) were compared (5.6 vs. 4.4 mg/dL; $p < 0.001$). UA concentrations above 4.8 mg/dL were independently associated with PH together with obesity ($\text{BMI-SDS} \geq 1.65$) and older age (>12.5 years). In that study, UA levels >5.5 mg/dL were observed in 56% of patients with PH and in 15.7% of those with SH [22].

However, a difference between these two studies lies in the threshold of UA concentration considered significant. Obrycki Ł et al. demonstrated that a lower serum UA level (4.8 mg/dL) had the highest sensitivity and specificity in relation to its association with PH [22].

Atherosclerotic vascular disease

The relationship between hyperuricemia, MS, and atherosclerotic vascular disease has been well documented in adults; however, it has also been shown to occur in children [3]. cIMT is a marker of atherosclerosis and is directly associated with an increased risk of CMD [23]. The association between serum UA levels and atherosclerotic vascular disease may result from the indirect link between hyperuricemia and cardiovascular risk factors, or from the clustering of metabolic and hemodynamic risk factors known as MS [24].

Numerous studies indicate that UA stimulates the proliferation of vascular smooth muscle cells, which in turn leads to endothelial dysfunction. It has been demonstrated that UA reduces nitric oxide production in the endothelium and contributes to endothelial dysfunction and insulin resistance [25]. This promotes vascular inflammation and arterial damage. All of these factors contribute to an increased risk of atherosclerosis.

In a study conducted by Pacifico L. et al. involving 120 obese children and 50 control participants, UA levels were found to be significantly higher in obese children compared to the control group. Additionally, associations with cardiovascular risk factors were observed. Based on the analyzed data, the authors concluded that elevated UA levels in obese children and adolescents were associated with carotid atherosclerosis [3].

Elevated coronary heart disease risk factors in children are associated with the early development of coronary artery calcifications. Moreover, the risk of premature death from coronary heart disease in adulthood is strongly linked to obesity during adolescence [26]. Therefore, early identification of individuals at risk is crucial from a public health perspective and for the implementation of preventive and therapeutic strategies. In summary, the demonstrated association between serum UA levels and carotid atherosclerosis in obese children supports the hypothesis that UA may play a role as a risk factor in the early stages of atherosclerosis.

Discussion

The present systematic review demonstrates a consistent and clinically meaningful association between elevated serum UA levels and multiple components of CMD in children and adolescents, including obesity, MS, HT, and early atherosclerotic changes. Despite heterogeneity across studies in terms of design, population characteristics, and diagnostic criteria, the overall body of evidence supports the notion that UA is closely linked with an adverse metabolic profile from an early stage of life.

A particularly strong relationship was observed between UA and obesity-related parameters. UA levels were consistently higher in children with overweight and obesity and showed positive correlations with BMI and WC-SDS. Importantly, several studies suggest that UA may help differentiate between MHO and MUO. This distinction is clinically relevant, as children with MUO are at significantly higher risk of progressing to overt CMD. Therefore, UA may serve not only as a marker of adiposity but also as an indicator of metabolic impairment within obese pediatric populations.

The association between UA and HT appears especially robust and may have important implications for early disease detection. Evidence indicates that elevated UA levels often precede the development of PH and are more strongly associated with primary than secondary forms of the condition. This supports the hypothesis that UA may play a contributory role in the early pathogenesis of HT rather than being solely a consequence of it. Proposed mechanisms, including endothelial dysfunction, reduced nitric oxide availability, oxidative stress, and activation of the renin–angiotensin system, provide biological plausibility for this relationship. Notably, the influence of UA may be more pronounced in early stages of disease, before irreversible vascular or renal damage occurs.

In the context of MS, elevated UA levels are frequently associated with its individual components, such as dyslipidemia, insulin resistance, and central obesity. However, the absence of a universally accepted definition of MS in children limits comparability across studies and may contribute to variability in reported findings. This highlights the need for standardized pediatric criteria when evaluating cardiometabolic risk factors, including UA.

The relationship between UA and early atherosclerotic changes further underscores its potential clinical importance. Studies demonstrating associations between UA and increased cIMT suggest that hyperuricemia may contribute to subclinical vascular damage in children. Given that early vascular alterations are predictive of future cardiovascular events, these findings raise concerns about the long-term implications of elevated UA levels beginning in childhood.

Despite these consistent associations, the question of causality remains unresolved. Some studies report attenuation of the relationship between UA and CMD after adjusting for confounding factors such as BMI and insulin resistance, suggesting that UA may function primarily as a secondary marker. In contrast, experimental

and longitudinal evidence supports a more direct pathogenic role, particularly in early disease stages. It is therefore plausible that UA acts both as a biomarker and a mediator, with its role varying depending on the clinical context and progression of disease.

Several limitations of the current evidence base should be acknowledged. The predominance of cross-sectional studies limits causal inference, while variability in UA cutoff values and study methodologies reduces comparability. Additionally, important confounders, including dietary patterns (especially fructose intake), physical activity, genetic factors, and renal function, are not consistently accounted for. These gaps emphasize the need for well-designed prospective and interventional studies.

From a clinical perspective, serum UA appears to be a promising, low-cost, and widely accessible biomarker that could enhance early identification of children at increased cardiometabolic risk.

Conclusions

Elevated serum UA levels are consistently associated with key components of CMD in children and adolescents, including obesity, MS, HT, and early atherosclerotic changes. The available evidence suggests that UA may play a dual role - as both a biomarker of underlying metabolic disturbances and a potential contributor to early pathophysiological processes.

Although the causal nature of this relationship remains uncertain, the strong and recurrent associations observed across studies indicate that UA has significant potential in early risk stratification. Its utility may be particularly valuable in identifying high-risk individuals among obese children and those with elevated blood pressure.

Future research should focus on longitudinal cohort studies and randomized controlled trials to clarify whether lowering UA levels can prevent or mitigate the development of CMD. Additionally, establishing standardized pediatric thresholds for hyperuricemia and incorporating UA into integrated risk assessment models may improve early detection and prevention strategies.

In summary, serum UA represents a promising factor in pediatric cardiometabolic health, warranting further investigation and cautious consideration in clinical practice.

Author's contribution:

Conceptualization: AT, KP, EB

Methodology: AT, WB

Validation: AT, AB

Resources: KP, JS

Data curation: AB, WB

Writing—original draft preparation: AT, KP

Writing—review and editing: KP, EB

Visualization: AT, JS

Supervision: AT, EB

All authors have read and agreed to the published version of the manuscript.

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Declaration of the use of generative AI and AI-assisted technologies in the writing process. During the preparation of this manuscript, AI-assisted tools were used exclusively for language editing and stylistic refinement. The authors take full responsibility for the content of the manuscript.

REFERENCES

1. Viazzi, F., Antolini, L., Giussani, M., Brambilla, P., Galbiati, S., Mastriani, S., Stella, A., Pontremoli, R., Valsecchi, M. G., & Genovesi, S. (2013). Serum uric acid and blood pressure in children at cardiovascular risk. *Pediatrics*, 132(1), e93-99. <https://doi.org/10.1542/peds.2013-0047>
2. Özalp Kızılay, D., Şen, S., & Ersoy, B. (2019). Associations Between Serum Uric Acid Concentrations and Cardiometabolic Risk and Renal Injury in Obese and Overweight Children. *Journal of Clinical Research in Pediatric Endocrinology*, 11(3), 262–269. <https://doi.org/10.4274/jcrpe.galenos.2018.2019.0241>
3. Pacifico, L., Cantisani, V., Anania, C., Bonaiuto, E., Martino, F., Pascone, R., & Chiesa, C. (2009). Serum uric acid and its association with metabolic syndrome and carotid atherosclerosis in obese children. *European Journal of Endocrinology*, 160(1), 45–52. <https://doi.org/10.1530/EJE-08-0618>
4. Feig, D. I., & Johnson, R. J. (2003). Hyperuricemia in Childhood Primary Hypertension. *Hypertension*, 42(3), 247–252. <https://doi.org/10.1161/01.HYP.0000085858.66548.59>
5. Glantzounis, G. K., Tsimoyiannis, E. C., Kappas, A. M., & Galaris, D. A. (2005). Uric acid and oxidative stress. *Current Pharmaceutical Design*, 11(32), 4145–4151. <https://doi.org/10.2174/138161205774913255>
6. Johnson, R. J., Segal, M. S., Sautin, Y., Nakagawa, T., Feig, D. I., Kang, D.-H., Gersch, M. S., Benner, S., & Sánchez-Lozada, L. G. (2007). Potential role of sugar (fructose) in the epidemic of hypertension, obesity and the metabolic syndrome, diabetes, kidney disease, and cardiovascular disease. *The American Journal of Clinical Nutrition*, 86(4), 899–906. <https://doi.org/10.1093/ajcn/86.4.899>
7. Bussler, S., Penke, M., Flemming, G., Elhassan, Y. S., Kratzsch, J., Sergeyev, E., Lipek, T., Vogel, M., Spielau, U., Körner, A., de Giorgis, T., & Kiess, W. (2017). Novel Insights in the Metabolic Syndrome in Childhood and Adolescence. *Hormone Research in Paediatrics*, 88(3–4), 181–193. <https://doi.org/10.1159/000479510>
8. Ng, M., Fleming, T., Robinson, M., Thomson, B., Graetz, N., Margono, C., Mullany, E. C., Biryukov, S., Abbafati, C., Abera, S. F., Abraham, J. P., Abu-Rmeileh, N. M. E., Achoki, T., AlBuhairan, F. S., Alemu, Z. A., Alfonso, R., Ali, M. K., Ali, R., Guzman, N. A., ... Gakidou, E. (2014). Global, regional, and national prevalence of overweight and obesity in children and adults during 1980-2013: A systematic analysis for the Global Burden of Disease Study 2013. *Lancet*, 384(9945), 766–781. [https://doi.org/10.1016/S0140-6736\(14\)60460-8](https://doi.org/10.1016/S0140-6736(14)60460-8)
9. Blüher, M. (2010). The distinction of metabolically „healthy” from „unhealthy” obese individuals. *Current Opinion in Lipidology*, 21(1), 38–43. <https://doi.org/10.1097/MOL.0b013e3283346ccc>
10. Blüher, M. (2014). Are metabolically healthy obese individuals really healthy? *European Journal of Endocrinology*, 171(6), R209-219. <https://doi.org/10.1530/EJE-14-0540>
11. Rey-López, J. P., de Rezende, L. F., Pastor-Valero, M., & Tess, B. H. (2014). The prevalence of metabolically healthy obesity: A systematic review and critical evaluation of the definitions used. *Obesity Reviews: An Official Journal of the International Association for the Study of Obesity*, 15(10), 781–790. <https://doi.org/10.1111/obr.12198>
12. Rocha, E. P. A. A., Vogel, M., Stanik, J., Pietzner, D., Willenberg, A., Körner, A., & Kiess, W. (2018). Serum Uric Acid Levels as an Indicator for Metabolically Unhealthy Obesity in Children and Adolescents. *Hormone Research in Paediatrics*, 90(1), 19–27. <https://doi.org/10.1159/000490113>
13. Lurbe, E., Torro, M. I., Alvarez-Pitti, J., Redon, J., Borghi, C., & Redon, P. (2018). Uric acid is linked to cardiometabolic risk factors in overweight and obese youths. *Journal of Hypertension*, 36(9), 1840–1846. <https://doi.org/10.1097/HJH.0000000000001814>
14. Gustafsson, D., & Unwin, R. (2013). The pathophysiology of hyperuricaemia and its possible relationship to cardiovascular disease, morbidity and mortality. *BMC Nephrology*, 14, 164. <https://doi.org/10.1186/1471-2369-14-164>
15. Coresh, J., Wei, G. L., McQuillan, G., Brancati, F. L., Levey, A. S., Jones, C., & Klag, M. J. (2001). Prevalence of high blood pressure and elevated serum creatinine level in the United States: Findings from the third National Health and Nutrition Examination Survey (1988-1994). *Archives of Internal Medicine*, 161(9), 1207–1216. <https://doi.org/10.1001/archinte.161.9.1207>
16. Cannon, P. J., Stason, W. B., Demartini, F. E., Sommers, S. C., & Laragh, J. H. (1966). Hyperuricemia in primary and renal hypertension. *The New England Journal of Medicine*, 275(9), 457–464. <https://doi.org/10.1056/NEJM196609012750902>

17. Mazzali, M., Hughes, J., Kim, Y. G., Jefferson, J. A., Kang, D. H., Gordon, K. L., Lan, H. Y., Kivlighn, S., & Johnson, R. J. (2001). Elevated uric acid increases blood pressure in the rat by a novel crystal-independent mechanism. *Hypertension*, 38(5), 1101–1106. <https://doi.org/10.1161/hy1101.092839>
18. Watanabe, S., Kang, D.-H., Feng, L., Nakagawa, T., Kanellis, J., Lan, H., Mazzali, M., & Johnson, R. J. (2002). Uric acid, hominoid evolution, and the pathogenesis of salt-sensitivity. *Hypertension*, 40(3), 355–360. <https://doi.org/10.1161/01.hyp.0000028589.66335.aa>
19. Burke, G. L., Arcilla, R. A., Culpepper, W. S., Webber, L. S., Chiang, Y. K., & Berenson, G. S. (1987). Blood pressure and echocardiographic measures in children: The Bogalusa Heart Study. *Circulation*, 75(1), 106–114. <https://doi.org/10.1161/01.cir.75.1.106>
20. Pontremoli, R., Nicoletta, C., Viazzi, F., Ravera, M., Sofia, A., Berruti, V., Bezante, G. P., Del Sette, M., Martinoli, C., Sacchi, G., & Deferrari, G. (1998). Microalbuminuria is an early marker of target organ damage in essential hypertension. *American Journal of Hypertension*, 11(4 Pt 1), 430–438. [https://doi.org/10.1016/s0895-7061\(97\)00498-6](https://doi.org/10.1016/s0895-7061(97)00498-6)
21. Wang, J. G., Staessen, J. A., Fagard, R. H., Birkenhäger, W. H., Gong, L., & Liu, L. (2001). Prognostic significance of serum creatinine and uric acid in older Chinese patients with isolated systolic hypertension. *Hypertension*, 37(4), 1069–1074. <https://doi.org/10.1161/01.hyp.37.4.1069>
22. Obrycki, Ł., Skoczyński, K., Sikorski, M., Koziej, J., Mitoraj, K., Pilip, J., Pac, M., Feber, J., & Litwin, M. (2025). Current etiology of hypertension in European children—Factors associated with primary hypertension. *Pediatric Nephrology (Berlin, Germany)*, 40(10), 3233–3244. <https://doi.org/10.1007/s00467-025-06761-x>
23. Raitakari, O. T., Juonala, M., Kähönen, M., Taittonen, L., Laitinen, T., Mäki-Torkko, N., Jarvisalo, M. J., Uhari, M., Jokinen, E., Rönnemaa, T., Akerblom, H. K., & Viikari, J. S. A. (2003). Cardiovascular risk factors in childhood and carotid artery intima-media thickness in adulthood: The Cardiovascular Risk in Young Finns Study. *JAMA*, 290(17), 2277–2283. <https://doi.org/10.1001/jama.290.17.2277>
24. Nagahama, K., Iseki, K., Inoue, T., Touma, T., Ikemiya, Y., & Takishita, S. (2004). Hyperuricemia and cardiovascular risk factor clustering in a screened cohort in Okinawa, Japan. *Hypertension Research: Official Journal of the Japanese Society of Hypertension*, 27(4), 227–233. <https://doi.org/10.1291/hypres.27.227>
25. Kanellis, J., & Kang, D.-H. (2005). Uric acid as a mediator of endothelial dysfunction, inflammation, and vascular disease. *Seminars in Nephrology*, 25(1), 39–42. <https://doi.org/10.1016/j.semnephrol.2004.09.007>
26. Li, S., Chen, W., Srinivasan, S. R., Bond, M. G., Tang, R., Urbina, E. M., & Berenson, G. S. (2003). Childhood cardiovascular risk factors and carotid vascular changes in adulthood: The Bogalusa Heart Study. *JAMA*, 290(17), 2271–2276. <https://doi.org/10.1001/jama.290.17.2271>
27. Codazzi, V., Frontino, G., Galimberti, L., Giustina, A., & Petrelli, A. (2024). Mechanisms and risk factors of metabolic syndrome in children and adolescents. *Endocrine*, 84(1), 16–28. <https://doi.org/10.1007/s12020-023-03642-x>
28. Martino, F., Pannarale, G., Puddu, P. E., Colantoni, C., Zanon, C., Martino, E., Torromeo, C., Paravati, V., Perla, F. M., & Barilla, F. (2015). Is it possible a new definition of metabolic syndrome in childhood? *European Review for Medical and Pharmacological Sciences*, 19(22), 4324–4331.
29. Obrycki, Ł., Sikorski, M., Skoczyński, K., Koziej, J., Mitoraj, K., Pilip, J., Pac, M., Feber, J., & Litwin, M. (2026). Metabolic syndrome and serum uric acid level in children and adolescents with hypertension. *Journal of Hypertension*, 44(5), 847–857. <https://doi.org/10.1097/HJH.0000000000004275>
30. Villasis-Keever, M. A., Zurita-Cruz, J. N., Alcaraz-Hurtado, I. A., Klünder-Klünder, M., Vilchis-Gil, J., Romero-Guerra, A. L., López-Beltrán, A. L., & Delgadillo-Ruano, M. A. (2025). Association of Serum Uric Acid Levels with Cardiometabolic Factors in Adolescents with Obesity: A Cross-Sectional Study. *Metabolites*, 15(4), 237. <https://doi.org/10.3390/metabo15040237>