Research Article
Research Progress on the Relationship between Dietary Patterns and Hyperuricemia

Tian Long and Liang Liu
Department of Clinical Nutrition, Huangshi Central Hospital, Affiliated Hospital of Hubei Polytechnic University, Edong Healthcare Group, Huangshi, Hubei 435000, China

Correspondence should be addressed to Tian Long; piscodragon@163.com

Received 20 July 2022; Revised 18 August 2022; Accepted 23 August 2022; Published 17 September 2022

Academic Editor: Ye Liu

Copyright © 2022 Tian Long and Liang Liu. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

As the final metabolite of purine metabolism, uric acid is critically associated with human health. The serum uric acid level is regulated by diet and the metabolic capacity of the human body. The impaired control of uric acid metabolism and excretion is associated with the increased level of serum uric acid, which ultimately results in hyperuricemia. Hyperuricemia is the “fourth-highest” after hypertension, hyperglycemia, and hyperlipidemia. With progress made in the relationship between diet and hyperuricemia, different dietary patterns and lifestyles have been discussed, such as exercise, the amount intake of meat, seafood, supplements with omega-3 fatty acids, sugar-sweetened soft drinks and energy drinks, and lower-fat-containing foods as well as drinking beer, wine, and spirits in the present article. This study demonstrated that a lower risk of hyperuricemia is substantially correlated with higher baseline adherence to MeDiet, and plant polyphenols can combat hyperuricemia by blocking xanthine oxidase.

1. Introduction

1.1. The Introduction of Uric Acid. Uric acid was discovered by Carl Scheele (1742–1786). At the same time, Tobern Bergman discovered that uric acid is a major constituent of bladder stones. In the same year, Murray Forbes suggested that both urine and blood may contain uric acid. Alfred Bar ing Garrod (1819–1909) discovered that the uric acid level is higher in the blood of gout patients. Nobel laureate Emil Fischer (1852–1919) identified uric acid as a purine compound. Studies of uric acid metabolism by isotope tracer techniques have shown that urate overproduction can be detected in about one-third of gout patients [1].

The studies of De Xie have shown that the final product of purine metabolism is uric acid. The uric acid is synthesized by the action of several enzymes, including xanthine oxidase (XO). The enzyme XO catalyzes the final two steps in the conversion of uric acid from purine. The catalytic reaction is hypoxanthine to xanthine and the second catalytic reaction is xanthine to uric acid [2]. As an endogenous antioxidant, uric acid substantially scavenges the free radicals, including monooxyperoxy radicals (ROS) and hydroxyl radicals (OH) [3]. In contrast, uric acid is unable to scavenge the superoxide radical, and uric acid exerts its antioxidant capacity in the presence of ascorbic acid in plasma. Although uric acid is a substantial antioxidant in the extracellular fluid, uric acid acts as a prooxidative agent in the cellular system [2]. In the cellular system, the prooxidative effect of uric acid is regulated by the NADPH oxidase-dependent regulatory pathway. Studies have confirmed that the uric acid level in plasma is considered a circulatory marker of oxidative damage, which is significantly observed in various clinical complications, including ischemia–reperfusion injury, ischemic liver injury, heart failure, chronic atherosclerosis, hyperlipidemia, and diabetes [4–7].

2. Background of Hyperuricemia

Uric acid, the final product of purine catabolism, is mainly catalyzed by the XO enzyme [8], and it is excreted from the body through urine and feces [9, 10]. Uric acid in serum is susceptible to purine- and fructose-rich diets and is
synthesized by the catabolism of macromolecules, including DNA, RNA, and ATP. The uric acid is mainly excreted by the kidney, and failure of renal function can also promote the re-elevation of serum uric acid. At the same time, hormones, including estrogen, can augment the excretion of uric acid and it is associated with lowering serum uric acid levels in premenopausal women. Renal vasoconstriction also reduces the excretion of urate. The serum urate level is impaired when the transport system is genetically altered, especially genetic polymorphisms of SLC2A9, which regulates Glut9, affect serum urate levels [11, 12]. When urate production and excretion are unbalanced, serum urate levels also increase significantly [13]. As the name suggests, hyperuricemia refers to an abnormally elevated serum uric acid concentration of >7 mg/dL (416 μmol/L) in men and >6 mg/dL in women, which is considered hyperuricemia [14]. The average levels of serum uric acid have been increasing in many populations over the last century [15]. Epidemiological studies show 170 million patients with hyperuricemia in China, compared with 32.5 million in the United States [16]. Findings from 2009 to 2010 showed that the prevalence of hyperuricemia in the US adult population was approximately 19.3% [17], and its prevalence increased over time [9, 18, 19]. According to NHANES data, the levels of uric acid in the United States significantly increased from 19% in 1988–1994 to 21.5% in 2007–2008 [20]. Results of an epidemiological survey study in the eastern coastal areas of China revealed the prevalence of hyperuricemia which was about 13% in 2008, while it was considered nonexistent in the 1980s. The results showed that the prevalence of hyperuricemia was higher in Oceania countries, including Thailand (Lohsoonthorn et al., 2006), China (Nan et al., 2006), the United States (Zhu et al., 2011), Australia (Nabipour et al., 2011), France (Zalokar et al., 1972), England (Gimeno et al., 2009), New Zealand and Scotland (Sturge et al., 1977), and to a lesser extent in African countries, Malaysia, Turkey, Iran, Saudi Arabia, Philippines, and regions of the Soviet Union (Kuo et al., 2015; Gosling et al., 2014) [21].

3. Complications of Hyperuricemia

Hyperuricemia is associated with gout and various cardiometabolic diseases, especially in type 2 diabetes, coronary artery disease, kidney disease, myocardial infarction, stroke, obesity, atherosclerotic heart disease, hypertriglyceridemia, and other metabolic syndromes [22]. Recent studies suggest that hyperuricemia is critically associated with the risk factor for these diseases [10]. In this article, we substantially discuss this relationship and evaluate the pros and cons of therapeutic interventions (Table 1).

3.1. Uric Acid is Associated with Metabolic Syndromes. A 10-year follow-up study showed that high SUA in children (10–15 years) is a crucial predictor of MS events in male subjects [23]. A prospective study evaluating 1,511 men and women aged 55 to 80 initially showed that they were not affected by any MS component. Later follow-up results showed that MS had a significantly higher incidence of hypertriglyceridemia, low high-density lipoprotein (HDL), and Htn [24]. A prospective meta-analysis study with more than 54,000 participants revealed that increased SUA was correlated with an augmented risk of MS and also a causal risk factor for nonalcoholic fatty liver disease (NAFLD) [25].

3.2. Chronic Kidney Disease. The kidneys are responsible for excreting 65–75% of uric acid daily, and the gastrointestinal tract is responsible for excreting the remaining 25–35% [26]. Impaired renal excretion is associated with about 90% of hyperuricemias [10]. Also, 60% of patients can develop hyperuricemia in advanced chronic kidney disease (CKD) [27]. Furthermore, CKD is a frequent independent risk factor for developing gout [28]. CKD can be defined as a structural or functional abnormality of the kidney that persists for more than 3 months [29]. These abnormalities included the lower glomerular filtration rate (GFR < 60 mL/min/1.73 m²) or the impairment of one or more than one markers of renal failure [29]. CKD is associated with the increasing prevalence of metabolic disease and cardiovascular diseases with an increasing problem of global public health, accounting for approximately 14% of the US population [30]. Over the past two decades, hyperuricemia is critically associated with the risk factor to develop or progress CKD, but its causality remains uncertain. Recent human epidemiological data and experimental evidence from animal models of mild hyperuricemia suggest that soluble serum urate may be directly involved with the pathogenesis of CKD [31, 32].

3.3. Systemic Hypertension. The risk of hypertension increases with age in patients with hyperuricemia [33, 34]. In children, studies discovered that an increased SUA level is substantially correlated to the development and pathogenesis of de novo primary hypertension [35]. A prospective meta-analysis study with 97,824 participants showed a higher level of SUA-predicted systemic hypertension [36]. Of the 118,000 healthy subjects from 40 to 70 years old were diagnosed with the level of SUA in 2002, and developed a quarter with systemic hypertension over the next 10 years. They found that subjects with SUA higher than 3 mg/dL showed a greater risk of developing hypertension. Within the normal range, a higher level of SUA is associated with a greater risk of developing hypertension [37, 62]. One of the important determinants of SUA is the glucose transporter "GLUT9" gene. GLUT9 transports UA. A family study of GLUT9 gene polymorphisms showed that hyperuricemia can lead to systemic Htn [38].

4. Dietary Pattern

The important organs, including the liver, muscles, and gut delivered two-thirds of the uric acid load, whereas the other food and drink constituents, including seafood, alcohol, fructose, and purine-rich meats delivered the rest of the uric acid. An important way to lower uric acid is to change patients’ diet and lifestyle.

5. Diet

Different types of foods contain different amounts of purines. It was previously mistakenly believed that avoiding
interest in the promotion of physical and environmental health. In chronic non-communicable diseases, a PBD can reduce the risk of morbidity and mortality in patients [46–50].

Several factors, including lower alcohol intake, weight loss, a diet with low purines, and total protein intake, are considered to treat and prevent gout attacks. However, reducing the intake of protein from animal sources will influence the utilization of refined carbohydrates and saturated and trans fats, which will have adverse effects on the cardiovascular system [51]. PBD is primarily centered on vegetables, legumes, grains, whole grains, and many other fruits. The results of a study revealed that the serum concentration of UA was lower in the vegetarian group than that in the nonvegetarian group [51].

### 7. Legumes

Beans are an important component of PBD plant-based dieters [52], are lower in natural fat, and showed a reduced level of the glycemic index. In PBD, beans supplemented a greater percentage of micro- and macronutrients, including dietary fiber, B vitamins, zinc, iron, and other essential minerals. Findings showed that soy foods were a crucial dietary agent which is associated with a longer survival time in older adults [53]. Soy foods are high in the diets of Asian countries, but soy products may increase the risk of gout. However, a prospective cohort study of 63,257 Chinese adults showed that legume-based products, including soy and nonsoy products, were correlated with a decreased risk of gout [70], and soy protein substantially increased the serum UA level. A plant food-rich diet has not been found to influence the risk of hyperuricemia and gout [54]. In their gout management recommendations, the British College of Rheumatology also encourages patients to include soy and plant proteins in their diets, while avoiding high-purine foods [55].

### 8. Plant Polyphenols

Vegetables and fruits are significantly correlated to reducing the incidence of various diseases such as hypertension, cardiovascular disease, cancer, diabetes, hyperuricemia, and gout [21]. It may be related to the presence of a large number of bioactive components (such as polyphenols) contained in vegetables and fruits [56]. Polyphenols are the most common functional bioactive components, and plant polyphenols can combat hyperuricemia by blocking XO to reduce UA synthesis, inhibit renal reabsorption of urate, and improve UA secretion [57].
9. Mediterranean Diet (MeDiet)

MeDiet is defined as a high intake of vegetables, fruits, legumes, nuts, olive oil, and whole grains and an intake of moderate level of poultry, dairy products, and wine. Since MeDiet showed the properties of antioxidant and anti-inflammatory activities [58], it may exhibit the role to reduce the SUA concentration, a crucial independent predictor of cardiovascular disease (CVD) in individuals with a high risk of cardiovascular disease [59]. In a study of 4,449 heart patients, a prospective analysis of older participants with high vascular risk showed that after a median follow-up of 5 years, 756 (24.9%) of 3,037 without hyperuricemia at baseline developed hyperuricemia compared with 964 at baseline. Four hundred twenty-two of those (43.8%) recovered from this condition. A cross-sectional study found an inverse association between the increased levels of adherence to the 14-item MeDiet score and decreased hyperuricemia. This demonstrated that a lower risk of hyperuricemia is substantially correlated with higher baseline adherence to MeDiet [60].

10. Recommended Lifestyle with Hyperuricemia Complications

The following health advice is for patients with gout, but also patients with hyperuricemia, cardiovascular disease, obesity, coronary artery disease, metabolic syndromes, diabetes, and hyperlipidemia. These recommendations are as follows [61]:

1. Exercise every day to lose weight. Weight loss was used to help reduce SUA in patients; however, the effect of this approach was modest (obese patients with gout lost an average of 7.7 kg over 16 weeks, a mean difference of 1.6 mg/dL SUA).
2. Limited intake of meat, seafood, and supplements with omega-3 fatty acids. Previous studies have revealed that consumption of meat and seafood is critically correlated with the development of hyperuricemia.
3. Intake of limited sugar-sweetened soft drinks and energy drinks is recommended. Intake of fructose is associated with increased SUA, and intracellular fructokinase produces an increased level of local ATP consumption and AMP production from the fructose [17]. It was reported that the supplemented high-calorie fructose (+35% excess energy) with extreme doses (213–219 g/day) substantially increased the level of SUA (mean difference = 0.5 mg/dL).
4. Eating lower-fat-containing foods or nonfat dairy-based products and reducing the intake of food-containing saturated fat, solid fat, and harmful cholesterol had little enforcement on reducing the level of SUA.
5. Cut down on drinking beer, wine, and spirits.

11. Conclusion

This study demonstrated that a lower risk of hyperuricemia is substantially correlated with higher baseline adherence to MeDiet, and plant polyphenols can combat hyperuricemia by blocking xanthine oxidase.

Data Availability

The data used to support this study are available from the corresponding author upon request.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Funding

No funding was received for this study.

References
update,” *Expert Opinion on Drug Discovery*, vol. 15, no. 8, pp. 943–954, 2020.

[14] T. Bardin and P. Richette, “Definition of hyperuricemia and gouty conditions,” *Current Opinion in Rheumatology*, vol. 26, no. 2, pp. 186–191, 2014.

[15] L. G. Sánchez-Lozada, E. Tapia, J. Santamaria et al., “Mild hyperuricemia induces vasoconstriction and maintains glomerular hypertension in normal and remnant kidney rats,” *Kidney International*, vol. 67, no. 1, pp. 237–247, 2005.

[16] G. Singh, B. Lingala, and A. Mithal, “Gout and hyperuricaemia in the USA: prevalence and trends,” *Rheumatology*, vol. 58, no. 12, pp. 2177–2180, 2019.

[17] E. Krishnan, “Reduced glomerular function and prevalence of gout: NHANES 2009-10,” *PLoS One*, vol. 7, no. 11, article ID e50046, 2012.

[18] G. Trifirò, P. Morabito, L. Cavagna et al., “Epidemiology of gout and hyperuricaemia in Italy during the years 20052009: a nationwide population-based study,” *Annals of the Rheumatic Diseases*, vol. 72, no. 5, pp. 694–700, 2013.

[19] Y. H. Rho, Y. Zhu, and H. K. Choi, “The epidemiology of uric acid and fructose,” *Seminars in Nephrology*, vol. 31, no. 5, pp. 410–419, 2011.

[20] Y. Zhu, B. J. Pandya, and H. K. Choi, “Prevalence of gout and hyperuricemia in the US general population: the national health and nutrition examination survey 2007–2008,” *Arthritis and Rheumatism*, vol. 63, no. 10, pp. 3136–3141, 2011.

[21] A. Mehmood, L. Zhao, C. Wang et al., “Management of hyperuricemia through dietary polyphenols as a natural medication: a comprehensive review,” *Critical Reviews in Food Science and Nutrition*, vol. 59, no. 9, pp. 1433–1455, 2019.

[22] C. Borghi, E. Agabiti-Rosei, R. J. Johnson et al., “Hyperuricemia and gout in cardiovascular, metabolic and kidney disease,” *European Journal of Internal Medicine*, vol. 80, pp. 1–11, 2020.

[23] H. L. Sun, D. Pei, K. H. Lue, and Y. L. Chen, “Uric acid levels can predict metabolic syndrome and hypertension in adolescents: a 10-year longitudinal study,” *PLoS One*, vol. 10, no. 11, article ID e0143786, 2015.

[24] N. Babio, M. A. Martínez-González, R. Estruch et al., “Associations between serum uric acid concentrations and metabolic syndrome and its components in the PREMED study,” *Nutrition, Metabolism, and Cardiovascular Diseases: NMCD*, vol. 25, no. 2, pp. 173–180, 2015.

[25] H. Yuan, C. Yu, X. Li et al., “Serum uric acid levels and risk of metabolic syndrome: a dose-response meta-analysis of prospective studies,” *The Journal of Clinical Endocrinology and Metabolism*, vol. 100, no. 11, pp. 4198–4207, 2015.

[26] A. C. Cannella and T. R. Mikuls, “Understanding treatments for gout,” *The American Journal of Managed Care*, vol. 11, 15 Suppl, pp. S451–S458, 2005, quiz S65–8.

[27] M. Madero, M. J. Sarnak, X. Wang et al., “Uric acid and long-term outcomes in CKD,” *American Journal of Kidney Diseases: The Official Journal of the National Kidney Foundation*, vol. 53, no. 5, pp. 796–803, 2009.

[28] E. Krishnan, “Chronic kidney disease and the risk of incident gout among middle-aged men: a seven-year prospective observational study,” *Arthritis and Rheumatism*, vol. 65, no. 12, pp. 3271–3278, 2013.

[29] Erratum: Kidney Disease: Improving Global Outcomes (KDIGO) CKD-MBD Update Work Group, “KDIGO 2017 clinical practice guideline update for the diagnosis, evaluation, prevention, and treatment of chronic kidney disease-mineral and bone disorder (CKD-MBD),” *Kidney International Supplement*, vol. 7, pp. 1–59, 2017.

[30] J. Coresh, E. Selvin, L. A. Stevens et al., “Prevalence of chronic kidney disease in the United States,” *Journal of the American Medical Association*, vol. 298, no. 17, pp. 2038–2047, 2007.

[31] T. O. Crisan, M. C. Cleophas, M. Oosting et al., “Soluble uric acid primes TLR-induced proinflammatory cytokine production by human primary cells via inhibition of IL-1β,” *Annals of the Rheumatic Diseases*, vol. 75, no. 4, pp. 755–762, 2016.

[32] T. Eleftheriadis, S. Golphinopoulos, G. Pissas, and I. Stefanidis, “Asymptomatic hyperuricemia and chronic kidney disease: narrative review of a treatment controversial,” *Journal of Advanced Research*, vol. 8, no. 5, pp. 555–560, 2017.

[33] F. Wei, N. Sun, C. Cai et al., “Associations between serum uric acid and the incidence of hypertension: a Chinese senior dynamic cohort study,” *Journal of Translational Medicine*, vol. 14, no. 1, p. 110, 2016.

[34] Y. Yokoi, T. Kondo, N. Okumura et al., “Serum uric acid as a predictor of future hypertension: stratified analysis based on body mass index and age,” *Preventive Medicine*, vol. 90, pp. 201–206, 2016.

[35] D. I. Feig and R. J. Johnson, “Hyperuricemia in childhood primary hypertension,” *Hypertension*, vol. 42, no. 3, pp. 247–252, 2003.

[36] J. Wang, T. Qin, J. Chen et al., “Hyperuricemia and risk of incident hypertension: a systematic review and meta-analysis of observational studies,” *PLoS One*, vol. 9, no. 12, article ID e114259, 2014.

[37] A. Leiba, S. Vinker, D. Dinour, E. J. Holtzman, and M. Shani, “Uric acid levels within the normal range predict increased risk of hypertension: a cohort study,” *Journal of the American Society of Hypertension: JASH*, vol. 9, no. 8, pp. 600–609, 2015.

[38] U. A. A. Sharaf El Din, M. M. Salem, and D. O. Abdulazim, “Uric acid in the pathogenesis of metabolic, renal, and cardiovascular diseases: a review,” *Journal of Advanced Research*, vol. 8, no. 5, pp. 537–548, 2017.

[39] X. Liu, S. Huang, W. Xu et al., “Association of dietary patterns and hyperuricemia: a cross-sectional study of the Yi ethnic group in China,” *Food & Nutrition Research*, vol. 62, 2018.

[40] R. Villegas, Y. B. Xiang, T. Elasy et al., “Purine-rich foods, protein intake, and the prevalence of hyperuricemia: the Shanghai Men’s health study,” *Nutrition, Metabolism, and Cardiovascular Diseases: NMCD*, vol. 22, no. 5, pp. 409–416, 2012.

[41] H. K. Choi, K. Atkinson, E. W. Karlson, W. Willett, and G. Curhan, “Purine-rich foods, dairy and protein intake, and the risk of gout in men,” *The New England Journal of Medicine*, vol. 350, no. 11, pp. 1093–1103, 2004.

[42] L. Zgaga, E. Theodoratou, J. Kyle et al., “The association of dietary intake of purine-rich vegetables, sugar-sweetened beverages and dairy with plasma urate, in a cross-sectional study,” *PLoS One*, vol. 7, no. 6, article ID e38123, 2012.

[43] T. J. Major, R. K. Topless, N. Dalbeth, and T. R. Merriman, “Evaluation of the diet wide contribution to serum urate levels: meta-analysis of population based cohorts,” *BMJ*, vol. 363, article ID k3951, 2018.

[44] L. A. MacFarlane and S. C. Kim, “Gout: a review of nonmodifiable and modifiable risk factors,” *Rheumatic Diseases Clinics of North America*, vol. 40, no. 4, pp. 581–604, 2014.
vegetarians and omnivores,” European Journal of Clinical Nutrition, vol. 61, no. 8, pp. 1011–1022, 2007.

[46] H. Lynch, C. Johnston, and C. Wharton, “Plant-based diets: considerations for environmental impact, protein quality, and exercise performance,” Nutrients Actions Search in PubMed Search in NLM Catalog Add to Search, vol. 10, no. 12, p. 1841, 2018.

[47] N. D. Barnard, S. M. Levin, and Y. Yokoyama, “A systematic review and meta-analysis of changes in body weight in clinical trials of vegetarian diets,” Journal of the Academy of Nutrition and Dietetics, vol. 115, no. 6, pp. 954–969, 2015.

[48] A. Satija, S. N. Bhupathiraju, E. B. Rimm et al., “Plant-based dietary patterns and incidence of type 2 diabetes in US men and women: results from three prospective cohort studies,” PLoS Medicine, vol. 13, no. 6, article ID e1002039, 2016.

[49] M. Dinu, R. Abbate, G. F. Gensini, A. Casini, and F. Sofi, “Vegetarian, vegan diets and multiple health outcomes: a systematic review with meta-analysis of observational studies,” Critical Reviews in Food Science and Nutrition, vol. 57, pp. 3640–3649, 2017.

[50] H. Kahleova, S. Levin, and N. Barnard, “Cardio-metabolic benefits of plant-based diets,” Nutrients, vol. 9, no. 8, p. 848, 2017.

[51] B. Jakše, B. Jakše, M. Pajek, and J. Pajek, “Uric acid and plant-based nutrition,” Nutrients, vol. 11, no. 8, p. 1736, 2019.

[52] K. Papier, T. Y. Tong, P. N. Appleby et al., “Comparison of major protein-source foods and other food groups in meat-eaters and non-meat-eaters in the EPIC-Oxford cohort,” Nutrients, vol. 11, no. 4, p. 824, 2019.

[53] I. Darmadi-Blackberry, M. L. Wahlqvist, A. Kouris-Blazos et al., “Legumes: the most important dietary predictor of survival in older people of different ethnicities,” Asia Pacific Journal of Clinical Nutrition, vol. 13, no. 2, pp. 217–220, 2004.

[54] J. A. Schmidt, F. L. Crowe, P. N. Appleby, T. J. Key, and R. C. Travis, “Serum uric acid concentrations in meat eaters, fish eaters, vegetarians and vegans: a cross-sectional analysis in the EPIC-Oxford cohort,” PLoS One, vol. 8, no. 2, article ID e56339, 2013.

[55] M. Hui, A. Carr, S. Cameron et al., “The British Society for Rheumatology guideline for the management of gout,” Rheumatology, vol. 56, no. 7, pp. 1056–1059, 2017.

[56] E. E. Mulvihill and M. W. Huff, “Les proprietes antiatherogenes des flavonoides: les repercussions pour la sante cardio-vasculaire,” The Canadian Journal of Cardiology, vol. 26, pp. 17A–21A, 2010, Suppl A:17a–21a.

[57] Y. Wang, Z. Lin, B. Zhang, A. Nie, and M. Bian, “Cichorium intybus L. promotes intestinal uric acid excretion by modulating ABCG2 in experimental hyperuricemia,” Nutrition & Metabolism, vol. 14, no. 1, p. 38, 2017.

[58] M. Bulló, R. Lamuela-Raventós, and J. Salas-Salavdó, “Mediterranean diet and oxidation: nuts and olive oil as important sources of fat and antioxidants,” Current Topics in Medicinal Chemistry, vol. 11, no. 14, pp. 1797–1810, 2011.

[59] G. Lippi, M. Montagnana, M. Franchini, E. J. Favaloro, and G. Targher, “The paradoxical relationship between serum uric acid and cardiovascular disease,” Clinica Chimica Acta; International Journal of Clinical Chemistry, vol. 392, no. 102, pp. 1–7, 2008.

[60] M. Guasch-Ferré, M. Bulló, N. Babio et al., “Mediterranean diet and risk of hyperuricemia in elderly participants at high cardiovascular risk,” The Journals of Gerontology Series A, Biological Sciences and Medical Sciences, vol. 68, no. 10, pp. 1263–1270, 2013.