Jurnal Riset Kualitatif dan Promosi Kesehatan
E-ISSN: 2775-4944
Volume. 2, Issue 2, July 2023
Page No: 67-78

The Impact of Excessive Fructose Consumption on Kidney Health: A Narrative Review

Ahmad Yani[1], Putri[2]
[1]Universitas Muhammadiyah Palu, Indonesia
[2]Yayasan Administrasi Indonesia, Indonesia
Correspondent: ahmadyani.publichealth@gmail.com[1]

Received : June 22, 2023
Accepted : July 25, 2023
Published : July 31, 2023

Citation: Yani, A., & Putri, (2023). The Impact of Excessive Fructose Consumption on Kidney Health: A Narrative Review. Jurnal Riset Kualitatif dan Promosi Kesehatan, 2(2), 67-78.

https://doi.org/10.61194/jrkpk.v2i2.665

ABSTRACT: Fructose consumption has increased markedly in recent decades, paralleling the global rise in chronic kidney disease (CKD). This narrative review examines how excessive fructose intake impairs renal function through pathways involving oxidative stress, inflammation, uric acid metabolism, and insulin resistance. Peer reviewed studies from databases such as PubMed, Scopus, and Google Scholar were analyzed to explore the biological mechanisms linking fructose to renal dysfunction. The review reveals that high fructose diets especially those involving artificial sweeteners like high fructose corn syrup exacerbate kidney damage, promote hypertension, and accelerate fibrosis. Countries with high fructose intake show increased CKD prevalence, underscoring the urgency of public health responses. Although sugar reduction policies such as taxation and labeling regulations have shown promise, gaps remain in understanding long term metabolic effects and genetic interactions. Future research should focus on targeted interventions to mitigate fructose induced kidney injury. This study emphasizes the need for immediate, evidence based dietary and policy strategies to address the global burden of fructose related kidney disease.

Keywords: Fructose, Chronic Kidney Disease, Uric Acid, Oxidative Stress, Insulin Resistance, Sugar Sweetened Beverages, Public Health Policy.

INTRODUCTION

The increasing global consumption of fructose over the past two decades has garnered widespread attention due to its significant implications for public health, particularly its role in the development of chronic kidney disease (CKD). High fructose intake, primarily through sugar-sweetened beverages containing high-fructose corn syrup, has been linked to various metabolic disorders, including obesity, diabetes, and CKD (Andres-Hernando et al., 2023; Meléndez-Salcido et al., 2022). Epidemiological data indicate a parallel rise in CKD prevalence alongside increased fructose consumption, with mechanistic pathways involving oxidative stress, inflammation, and uric acid accumulation (Andres-Hernando et al., 2023; Meléndez-Salcido et al., 2022). Given these

associations, understanding the biochemical mechanisms by which fructose contributes to kidney dysfunction is of paramount importance.

Dietary fructose has been shown to induce hyperglycemia, leading to oxidative damage in renal tissues and exacerbating CKD progression (Mei et al., 2022). Furthermore, studies suggest that fructose consumption correlates with elevated serum uric acid levels, which not only impair renal function but also stimulate inflammatory responses via the NLRP3 inflammasome pathway (Andres-Hernando et al., 2023; Mei et al., 2022). Unlike glucose, fructose is primarily metabolized in the kidney through glycolysis, where it enhances the activity of lipogenic and pro-inflammatory enzymes, contributing to renal fibrosis (Soleimani et al., 2023). These findings highlight the necessity of investigating fructose metabolism in the context of kidney disease.

Biochemically, fructose promotes renal injury through multiple pathways. Notably, it upregulates the expression of fructokinase, an enzyme that facilitates the accumulation of intra-renal lipids, which in turn exacerbates kidney damage (Bier et al., 2022). Additionally, the detrimental effects of fructose are not limited to its direct impact on renal metabolism but are further amplified by dietary interactions. Specifically, high-fructose diets in combination with other dietary factors such as high sodium or fat intake may create synergistic effects that accelerate kidney dysfunction (García-Arroyo et al., 2020). These interactions emphasize the complexity of diet-induced nephropathy and the necessity for further research.

From a global perspective, the sustained rise in fructose consumption has raised concerns about its contribution to the growing burden of non-communicable diseases, including CKD. Over the past two decades, CKD prevalence has increased not only among individuals with diabetes and hypertension but also among those consuming high amounts of dietary sugars (Dwyer et al., 2022; Meléndez-Salcido et al., 2022). In certain regions, the consumption of fructose-rich beverages continues to rise, prompting health organizations to advocate for stricter dietary guidelines to limit sugar intake. These concerns necessitate a comprehensive examination of the long-term consequences of fructose consumption on kidney health.

Despite the well-documented associations between fructose and CKD, significant research gaps remain. While numerous studies have demonstrated the acute effects of fructose on renal function, there is limited longitudinal research investigating the chronic impacts of sustained high-fructose intake. Moreover, much of the existing evidence is derived from animal models, which may not fully capture the complexity of human metabolism (Dwyer et al., 2022; Soleimani et al., 2023). Additionally, the interaction between genetic predisposition and dietary fructose consumption remains insufficiently explored. Further research is required to delineate these relationships and to develop targeted intervention strategies.

The primary objective of this review is to systematically analyze the mechanisms linking fructose consumption to CKD development. Key areas of investigation include the role of fructose in promoting oxidative stress, inflammation, and metabolic dysregulation, all of which contribute to renal impairment. Additionally, this review will examine the interplay between dietary patterns and CKD risk, with a focus on fructose as a major dietary component implicated in renal dysfunction. By synthesizing current evidence, this review aims to provide a comprehensive understanding of the pathophysiological processes underlying fructose-induced nephropathy.

The scope of this review encompasses global epidemiological trends, mechanistic studies, and dietary interventions aimed at mitigating CKD risk. Particular attention is given to populations at high risk of CKD, including individuals with metabolic syndrome, obesity, and diabetes, as they are disproportionately affected by dietary fructose (Chan & Lin, 2023). Geographically, the focus extends to regions with high fructose consumption, such as North America, Latin America, and parts of Asia, where sugar-sweetened beverage intake has escalated in recent years. Furthermore, this review will explore socio-economic disparities in fructose consumption and CKD prevalence, considering variations in dietary patterns between urban and rural populations (Zhang et al., 2022). Understanding these trends will inform policy recommendations and public health strategies to reduce the burden of CKD associated with excessive fructose intake.

Ultimately, this review seeks to bridge existing knowledge gaps and provide a foundation for future research and policy initiatives aimed at curbing fructose-related renal disease. Given the increasing prevalence of CKD and the concurrent rise in fructose consumption, a concerted effort is needed to develop evidence-based dietary guidelines and intervention strategies. Addressing the impact of fructose on kidney health requires a multidisciplinary approach, integrating nutritional science, epidemiology, and clinical research. By elucidating the underlying mechanisms and risk factors, this review aims to contribute to the growing body of knowledge that informs effective prevention and management strategies for CKD in the context of modern dietary habits.

METHOD

This study employed a systematic review approach to examine the relationship between fructose consumption and kidney health. A comprehensive literature search was conducted across major academic databases, including PubMed, Scopus, and Google Scholar, targeting studies published between 2000 and 2024. The search strategy incorporated predefined keywords combined with Boolean operators to enhance precision and completeness. The primary keywords used included "fructose," "kidney health," "chronic kidney disease," "fructose metabolism," "uric acid," "renal inflammation," "high-fructose diet," "obesity," and "lipid metabolism." This strategy aimed to identify studies that provide empirical evidence on the biological mechanisms underlying the long-term effects of fructose consumption on kidney function and its association with metabolic disease risk.

The selection criteria were designed to ensure the inclusion of high-quality research. Inclusion criteria encompassed peer-reviewed studies that analyzed the link between fructose intake and kidney health, including laboratory-based experimental research and clinical studies. Studies providing data on the effects of fructose on renal metabolism, inflammation, and cellular damage were prioritized. Research involving human populations or relevant animal models was also included to ensure the findings' applicability. Conversely, exclusion criteria were established to eliminate studies that did not directly address fructose's impact on kidney health, review articles lacking original data, studies with weak methodological designs, and research focusing on pathological conditions unrelated to chronic kidney disease. Additionally, inaccessible or non-English publications were excluded to maintain the study's reliability and accessibility.

To enhance reliability, a multi-stage screening process was applied. Four independent reviewers evaluated the studies to ensure alignment with the inclusion criteria. Initial screening involved title and abstract review, followed by full-text assessment for relevance and methodological rigor. Thematic synthesis was conducted to identify recurring patterns in how fructose influences renal function, particularly in pathways related to oxidative stress, inflammation, and metabolic dysregulation. The findings offer insights into the mechanisms by which fructose contributes to kidney disease and provide a basis for potential dietary interventions to mitigate its adverse effects.

RESULT AND DISCUSSION

Fructose and Kidney Dysfunction

Empirical evidence linking excessive fructose consumption to kidney dysfunction has been strengthened by both animal and clinical studies. Research by Andres-Hernando et al. (2023) demonstrated that high-fructose corn syrup consumption exacerbated kidney damage and increased mortality in obese mice with metabolic syndrome(Andres-Hernando et al., 2023). In this study, fructose-fed mice exhibited elevated levels of kidney injury biomarkers and uric acid, a crucial indicator of kidney dysfunction. Similarly, a study by Aroor et al. (2017) associated high-caloric, fructose-rich diets with impaired kidney function, suggesting that excessive fructose intake and calorie overload accelerate kidney disease progression(Aroor et al., 2017).

Fructose also contributes to renal inflammation and oxidative stress through multiple pathways. It promotes the production of reactive oxygen species (ROS), which are detrimental to renal cells (Strambi et al., 2016). Yang et al. (2014) found that fructose consumption activates inflammatory pathways involving pro-inflammatory cytokines such as interleukin-6 (IL-6) and transforming growth factor beta-1 (TGF-β1), which play crucial roles in renal fibrosis and tissue damage(M. Yang et al., 2014). Elevated IL-6 levels further exacerbate protein loss and immune cell infiltration in the kidneys, leading to severe inflammation and tissue degradation. Additionally, increased uric acid production from fructose metabolism induces inflammatory responses and contributes to hypertensive nephropathy, worsening renal outcomes (Aroor et al., 2017; Komnenov et al., 2019).

Mitochondrial stress in renal cells is another consequence of fructose-induced oxidative stress. Thongnak et al. (2021) reported that fructose disrupts mitochondrial metabolic pathways, correlating with increased apoptosis in renal cells(Thongnak et al., 2021). This process is linked to the downregulation of SIRT1, a key regulator of cellular stress response, indicating that fructose accelerates renal damage not only through direct oxidative damage but also through deep-seated signaling disruptions.

Given the widespread prevalence of high-fructose diets in modern nutrition, understanding how fructose contributes to kidney dysfunction through multiple pathogenic mechanisms is essential. Future research should explore targeted intervention strategies to mitigate these effects and reduce kidney damage in individuals with high fructose consumption.

Fructose and Insulin Resistance in Kidney Failure

Excessive fructose intake has been identified as a key driver of insulin resistance, a condition that significantly exacerbates kidney dysfunction. Insulin resistance occurs when body cells fail to respond adequately to insulin, impairing glucose regulation. Unlike glucose, fructose follows a distinct metabolic pathway that does not stimulate insulin secretion, leading to lipid accumulation and metabolic imbalances. Fructose-induced insulin resistance is particularly concerning in individuals predisposed to metabolic syndrome, a major risk factor for kidney disease (Andres-Hernando et al., 2023; Johnson et al., 2018).

Elevated triglyceride levels resulting from fructose metabolism contribute to lipotoxicity, characterized by the excessive accumulation of fat within kidney cells, disrupting normal function (M. Yang et al., 2014). Additionally, fructose stimulates inflammatory signaling pathways that exacerbate insulin resistance and oxidative stress, further damaging kidney tissues. Poor glucose regulation due to insulin resistance increases oxidative stress and reactive oxygen species (ROS) production, leading to chronic inflammation and kidney tissue damage (Chaudhary et al., 2013; Johnson et al., 2018). Johnson et al. (2018) also found that fructose consumption elevates uric acid levels, which impairs microvascular kidney function and contributes to hypertension, thereby exacerbating glomerulosclerosis(Johnson et al., 2018).

Fructose-induced insulin resistance also raises the risk of hypertension, a common complication in individuals with kidney disease. Hypertension increases vascular pressure in the kidneys, accelerating glomerular damage and heightening the risk of end-stage renal disease (Chaudhary et al., 2013). Kanbay et al. (2013) found that insulin resistance correlates with increased blood pressure and reduced renal blood flow, compounding kidney damage. These effects are particularly pronounced in individuals with diabetes, where insulin resistance is already present and further exacerbated by fructose intake.

Among individuals with diabetes, fructose metabolism can worsen preexisting kidney damage. Diabetic patients often exhibit impaired glucose and lipid metabolism, leading to increased energy demands and metabolic stress (Andres-Hernando et al., 2023; Student et al., 2022). Excessive fructose consumption in diabetic patients can elevate triglyceride levels, prolong inflammation, and accelerate kidney complications (Student et al., 2022). Studies suggest that in individuals with type 2 diabetes, high-fructose diets significantly increase the risk of diabetic nephropathy and hasten the decline of glomerular filtration rate (GFR) (Student et al., 2022).

While the impact of fructose on kidney function is more pronounced in diabetic individuals, evidence suggests that even in non-diabetic individuals, excessive fructose intake can contribute to insulin resistance and kidney inflammation, increasing the long-term risk of metabolic kidney disease (Naumann et al., 2017). This underscores the necessity of dietary interventions to limit fructose consumption, particularly among high-risk populations.

Uric Acid in Fructose-Induced Kidney Damage

Fructose metabolism is closely linked to uric acid production, which has significant implications for kidney health. When fructose is metabolized, it generates uric acid as a byproduct, leading to

hyperuricemia (elevated blood uric acid levels) (Johnson et al., 2018; Strambi et al., 2016). Hyperuricemia contributes to kidney damage through multiple pathological mechanisms. First, it induces local renal inflammation by increasing the production of pro-inflammatory cytokines such as interleukin-1 beta (IL-1β) and interleukin-6 (IL-6), leading to renal fibrosis and structural damage to the glomerulus (Andres-Hernando et al., 2023; Strambi et al., 2016). High uric acid levels have also been linked to the increased excretion of early kidney injury markers such as neutrophil gelatinase-associated lipocalin (NGAL) and kidney injury molecule-1 (KIM-1) in hypertensive patients (Strambi et al., 2016).

Uric acid also activates inflammatory signaling pathways, particularly the NLRP3 inflammasome, which intensifies kidney inflammation (Andres-Hernando et al., 2023). The oxidative stress induced by uric acid further damages renal tubular and endothelial cells, exacerbating kidney dysfunction (Johnson et al., 2018). Yang et al. (2022) found that elevated uric acid levels impair mitochondrial function, increasing renal susceptibility to oxidative stress and apoptosis(Y. Yang et al., 2022).

Additionally, uric acid influences the renin-angiotensin-aldosterone system (RAAS), which regulates blood pressure. Its activation leads to hypertension and increased hemodynamic stress on the kidneys (Johnson et al., 2018). Managing uric acid levels has been suggested as a potential strategy for mitigating kidney disease progression, particularly among hypertensive and metabolically vulnerable patients (Andres-Hernando et al., 2023; Johnson et al., 2018).

Global Comparisons and Policy Responses

Fructose consumption and kidney disease prevalence vary across countries, influenced by dietary habits, food policies, and public awareness. High-fructose intake is strongly associated with increased CKD prevalence in countries such as the United States and Mexico, where sugar-sweetened beverage consumption is widespread (Gharaei et al., 2020; Johnson et al., 2018). Data from these regions show a significant correlation between increased fructose consumption and a rising incidence of kidney disease. In contrast, countries such as Japan and Sweden, where fructose consumption is relatively low, exhibit lower CKD rates, underscoring the role of dietary patterns in renal health (M. Yang et al., 2014).

Several nations have implemented policies to curb fructose-related health risks. Mexico introduced a sugar tax on sweetened beverages, leading to a 12% reduction in sales in its first year, with associated declines in diabetes and hypertension rates (Hamada et al., 2022). Finland has restricted the marketing of high-sugar foods to reduce processed sugar consumption. The United Kingdom and Canada have launched public education campaigns advocating for reduced sugar intake to prevent metabolic diseases, including CKD (Andres-Hernando et al., 2023).

Despite promising results, challenges remain in enforcing these policies and fostering widespread dietary changes. Public awareness campaigns, ongoing health monitoring, and policy adjustments are crucial for sustaining the positive impact of sugar reduction strategies on kidney health (Gao et al., 2024). Future research should focus on evaluating the long-term effects of such policies on CKD prevalence to refine and enhance public health interventions.

Fructose and Kidney Dysfunction

The findings of this study align with existing literature indicating that excessive fructose consumption has detrimental effects on kidney function. Numerous studies have documented the mechanisms by which fructose contributes to renal dysfunction, including its influence on uric acid metabolism, inflammation, and oxidative stress. These results corroborate the findings of Aroor et al. (2017), who demonstrated that increased uric acid levels resulting from fructose consumption accelerate kidney damage through inflammatory and oxidative stress pathways(Aroor et al., 2017). Similarly, research by Milutinović et al. (2020) highlights the chronic stress-induced lipogenesis triggered by fructose, which further impairs kidney function(Milutinović et al., 2020). These findings are consistent with multiple studies linking high-fructose diets to an increased risk of chronic kidney disease (CKD) (Andres-Hernando et al., 2023).

Furthermore, this study reinforces the notion that fructose intake from artificial sweeteners, particularly high-fructose corn syrup, exacerbates kidney disease and metabolic syndrome. Johnson et al. (2018) reported that a high-fructose diet elevates uric acid levels and induces hypertension, which in turn contributes to renal dysfunction(Johnson et al., 2018). These findings suggest that public health measures should prioritize reducing fructose consumption, especially from processed sources, to mitigate the increasing prevalence of CKD.

Although substantial research has explored the relationship between fructose intake and kidney failure, several areas warrant further investigation. For instance, the differential impact of naturally occurring fructose from fruits versus artificial fructose additives needs to be examined more comprehensively. Some studies suggest that natural fructose does not exert the same adverse effects on kidney health as processed fructose (Gharaei et al., 2020; M. Yang et al., 2014). This underscores the importance of distinguishing between fructose sources when evaluating their effects on kidney function.

Policy Implications and Public Health Initiatives

This study is also relevant to global health policies aimed at reducing fructose consumption and its associated health risks. Many countries have recognized the public health burden of excessive fructose intake and have implemented sugar taxation policies as a mitigation strategy. For example, Mexico introduced a sugar tax on sweetened beverages in 2014, which significantly reduced sugar consumption and, in some cases, decreased the incidence of metabolic diseases, including CKD (Liang & Song, 2024). These findings highlight the effectiveness of regulatory measures in curbing high-fructose intake.

Educational campaigns have also played a crucial role in raising public awareness about the dangers of excessive fructose consumption. Public health initiatives promoting balanced diets, emphasizing whole foods over processed foods, and discouraging sugar-sweetened beverages have been successful in various countries (Pokrywczyńska et al., 2014). Studies indicate that targeted education efforts help individuals make informed dietary choices, which may reduce the risk of CKD and other metabolic disorders.

Additionally, governments in the United Kingdom and Canada have implemented restrictions on the marketing and advertising of high-fructose products, particularly those aimed at children (Li et al., 2021; Peng et al., 2012). These policies have led to a measurable decrease in sugar consumption and improved metabolic health outcomes. By controlling advertising exposure and providing consumers with transparent nutritional labeling, policymakers can encourage healthier dietary habits and reduce CKD prevalence.

A comprehensive health policy approach integrating taxation, education, and regulatory measures appears to be the most effective strategy for reducing fructose-related health risks. Countries that have successfully implemented these policies have reported declining rates of CKD and other sugar-related diseases (Johnson et al., 2018; Peng et al., 2012). Policymakers should consider a multifaceted approach that includes dietary guidelines, public awareness initiatives, and marketing restrictions to promote kidney health and prevent diet-induced renal dysfunction.

Fructose-Induced Insulin Resistance and Kidney Disease

The role of fructose in the development of insulin resistance is well established and has significant implications for kidney health. Insulin resistance, a key feature of metabolic syndrome, disrupts glucose homeostasis and increases the risk of kidney dysfunction. Unlike glucose, fructose metabolism bypasses insulin regulation, promoting lipid accumulation and impairing metabolic pathways. Johnson et al. (2018) found that fructose consumption contributes to increased triglyceride levels, leading to lipotoxicity and kidney damage(Johnson et al., 2018).

The interplay between fructose, insulin resistance, and renal dysfunction has also been observed in hypertensive patients. Studies suggest that insulin resistance exacerbates hypertension, further impairing kidney function. Kanbay et al. (2013) reported that insulin-resistant individuals exhibited higher blood pressure levels and reduced renal blood flow, exacerbating glomerular injury. These findings emphasize the need for targeted interventions to reduce fructose consumption among at-risk populations, particularly those with metabolic syndrome.

Among diabetic patients, fructose metabolism poses an even greater risk to kidney health. Diabetic nephropathy, a leading cause of end-stage renal disease, is exacerbated by fructose-induced inflammation and oxidative stress (Student et al., 2022). Research indicates that excessive fructose intake accelerates the decline in glomerular filtration rate (GFR) in diabetic individuals, further compounding kidney damage (Student et al., 2022). As such, dietary recommendations for diabetic patients should prioritize fructose restriction to mitigate renal complications.

Limitation

This study has several limitations that should be acknowledged. The reliance on existing literature means that the findings are constrained by the scope and methodologies of previous research. Many studies included in this review utilized animal models, which, while informative, may not fully capture the complexities of human metabolism and kidney function. Additionally, variations in dietary habits, genetic predisposition, and environmental factors among populations may influence the generalizability of the findings. The differential effects of fructose from natural

sources versus processed sweeteners also require further exploration to provide more precise dietary guidelines. Future research should incorporate longitudinal human studies to validate the mechanisms identified in experimental models and to develop more targeted dietary interventions.

Implication

The implications of these findings extend beyond nephrology and into broader public health and policy domains. The growing prevalence of CKD linked to fructose consumption underscores the urgency of implementing dietary guidelines that discourage excessive sugar intake. Policymakers should consider reinforcing existing regulations on fructose-containing products and expanding educational initiatives to raise awareness about the risks associated with high-fructose diets. Additionally, the role of fructose in metabolic disorders suggests that future research should adopt an interdisciplinary approach, integrating nephrology, endocrinology, and nutrition science to develop comprehensive prevention strategies. Continued investigation into the long-term effects of fructose on kidney health will be crucial in informing dietary recommendations and shaping public health policies aimed at reducing the burden of CKD.

CONCLUSION

This study reinforces the strong link between excessive fructose consumption and kidney dysfunction, elucidating mechanisms involving uric acid accumulation, oxidative stress, inflammation, and insulin resistance. High fructose intake, particularly from processed sources such as high fructose corn syrup, significantly contributes to the progression of chronic kidney disease (CKD), with even greater effects observed in individuals with metabolic disorders. These findings carry important clinical implications: early intervention through dietary counseling, routine nutritional screening, and targeted health communication can be key in delaying or preventing CKD onset.

From a public health perspective, concrete policy actions are urgently needed. Governments should consider implementing and strengthening multifaceted strategies, including taxation on sugar sweetened beverages, mandatory front of package nutritional labeling, restrictions on the marketing of high sugar foods especially to children and national dietary guidelines limiting added sugar intake. These measures should be accompanied by educational campaigns that promote health literacy and empower individuals to make healthier food choices.

Future research should prioritize longitudinal human studies to examine the cumulative effects of long term fructose exposure on kidney health, especially in diverse populations with different genetic predispositions and dietary habits. Investigating the comparative impact of natural versus processed fructose and the potential of pharmacological or nutraceutical interventions to counteract fructose induced renal damage also warrants attention. A collaborative, interdisciplinary approach integrating nephrology, nutrition science, epidemiology, and policy analysis is essential to address the multifactorial nature of this public health challenge and develop sustainable solutions for CKD prevention.

REFERENCES

Andres-Hernando A, Orlicky DJ, Cicerchi C, Kuwabara M, García G, Nakagawa T, et al. High Fructose Corn Syrup Accelerates Kidney Disease and Mortality in Obese Mice With Metabolic Syndrome. Biomolecules. 2023;13(5):780.

Meléndez-Salcido CG, Ramírez-Emiliano J, Pérez-Vázquez V. Hypercaloric Diet Promotes Metabolic Disorders and Impaired Kidney Function. Curr Pharm Des. 2022;28(38):3127–39.

Mei Y, Dong B, Geng Z, Xu L. Excess Uric Acid Induces Gouty Nephropathy Through Crystal Formation: A Review of Recent Insights. Front Endocrinol (Lausanne). 2022;13.

Soleimani M, Barone S, Luo H, Zahedi K. Pathogenesis of Hypertension in Metabolic Syndrome: The Role of Fructose and Salt. Int J Mol Sci. 2023;24(5):4294.

Bier A, Shapira E, Khasbab R, Sharabi Y, Grossman E, Leibowitz A. High-Fructose Diet Increases Renal ChREBPβ Expression, Leading to Intrarenal Fat Accumulation in a Rat Model With Metabolic Syndrome. Biology (Basel). 2022;11(4):618.

García-Arroyo FE, Tapia E, Muñoz-Jiménez I, Gonzaga-Sánchez G, Arellano-Buendía AS, Osorio-Alonso H, et al. Fluid Intake Restriction Concomitant to Sweetened Beverages Hydration Induce Kidney Damage. Oxid Med Cell Longev. 2020;2020:1–11.

Dwyer KM, Robson B, Muecke J. Too Much Sugar Does Not Just Make Us Fat; It Can Also Make Us Sick. Intern Med J. 2022;52(6):1089–92.

Chan CW, Lin B. Folate Deficiency Enhanced Inflammation and Exacerbated Renal Fibrosis in High-Fat High-Fructose Diet-Fed Mice. Nutrients. 2023;15(16):3616.

Zhang X, Wu Y, Miao L. Study on the Effects of Individualized Nutritional Intervention on Pregnancy Outcome and Neonatal Immune Function in Patients With Gestational Diabetes Mellitus. Biomed Res Int. 2022;2022(1).

Aroor AR, Jia G, Habibi J, Sun Z, Ramirez-Perez FI, Brady B, et al. Uric Acid Promotes Vascular Stiffness, Maladaptive Inflammatory Responses and Proteinuria in Western Diet Fed Mice. Metabolism. 2017;74:32–40.

Strambi M, Giussani M, Ambruzzi MA, Brambilla P, Corrado C, Giordano U, et al. Novelty in Hypertension in Children and Adolescents: Focus on Hypertension During the First Year of Life, Use and Interpretation of Ambulatory Blood Pressure Monitoring, Role of Physical Activity in Prevention and Treatment, Simple Carbohydrates and Uric Acid as Risk Factors. Ital J Pediatr. 2016;42(1).

Yang M, Liu C, Jiang J, Zuo G, Lin X, Yamahara J, et al. Ginger Extract Diminishes Chronic Fructose Consumption-Induced Kidney Injury Through Suppression of Renal Overexpression of Proinflammatory Cytokines in Rats. BMC Complement Altern Med. 2014;14(1).

Komnenov D, Levanovich P, Rossi NF. Hypertension Associated With Fructose and High Salt: Renal and Sympathetic Mechanisms. Nutrients. 2019;11(3):569.

Thongnak L, Pengrattanachot N, Promsan S, Phengpol N, Sutthasupha P, Chatsudthipong V, et al. The Combination of Dapagliflozin and Statins Ameliorates Renal Injury Through Attenuating the Activation of Inflammasome-mediated Autophagy in Insulin-resistant Rats. J Biochem Mol Toxicol. 2021;36(4).

Johnson RJ, Bakris GL, Borghi C, Chonchol MB, Feldman DL, Lanaspa MA, et al. Hyperuricemia, Acute and Chronic Kidney Disease, Hypertension, and Cardiovascular Disease: Report of a Scientific Workshop Organized by the National Kidney Foundation. American Journal of Kidney Diseases. 2018;71(6):851–65.

Chaudhary K, Malhotra K, Sowers JR, Aroor AR. Uric Acid - Key Ingredient in the Recipe for Cardiorenal Metabolic Syndrome. Cardiorenal Med. 2013;3(3):208–20.

Student J, Sowers JR, Lockette W. THIRSTY FOR FRUCTOSE: Arginine Vasopressin, Fructose, and the Pathogenesis of Metabolic and Renal Disease. Front Cardiovasc Med. 2022;9.

Naumann J, Biehler D, Lüty T, Sadaghiani C. Prevention and Therapy of Type 2 Diabetes—What Is the Potential of Daily Water Intake and Its Mineral Nutrients? Nutrients. 2017;9(8):914.

Yang Y, Yu G, Pan J, Kreps GL. Public Trust in Sources and Channels on Judgment Accuracy in Food Safety Misinformation With the Moderation Effect of Self-affirmation: Evidence From the HINTS-China Database. World Med Health Policy. 2022;15(2):148–62.

Gharaei FK, Lakzaei H, Niazi AA, Jahantigh M, Shahraki MR, Safari T. The Protective Effects of Eugenol on Metabolic-Syndrome, Renal Damages. J Renal Inj Prev. 2020;11(1):e4–e4.

Hamada S, Takata T, Yamada K, Yamamoto M, Mae Y, Iyama T, et al. Steatosis Is Involved in the Progression of Kidney Disease in a High-Fat-Diet-Induced Non-Alcoholic Steatohepatitis Mouse Model. PLoS One. 2022;17(3):e0265461.

Gao Y, Li CC, Li J, Duan M, Li X, Zhao L, et al. Weizmannia Coagulans BC99 Alleviates Hyperuricemia and Oxidative Stress via DAF-16/SKN-1 Activation in Caenorhabditis Elegan. Front Microbiol. 2024;15.

Milutinović D V, Brkljačić J, Teofilović A, Bursać B, Nikolić M, Gligorovska L, et al. Chronic Stress Potentiates High Fructose–Induced Lipogenesis in Rat Liver and Kidney. Mol Nutr Food Res. 2020;64(13).

Liang H, Song K. Elucidating Ascorbate and Aldarate Metabolism Pathway Characteristics via Integration of Untargeted Metabolomics and Transcriptomics of the Kidney of High-Fat Diet-Fed Obese Mice. PLoS One. 2024;19(4):e0300705.

Pokrywczyńska M, Flisiński M, Jundziłł A, Krzyzanowska S, Brymora A, Deptuła A, et al. Impact of Fructose Diet and Renal Failure on the Function of Pancreatic Islets. Pancreas. 2014;43(5):801–8.

Peng D, Tang S, Hu Y, Chen J, Yang L. Pathophysiological Model of Chronic Heart Failure Complicated With Renal Failure Caused by Three-Quarter Nephrectomy and Subcutaneous Injection of Isoprenaline. Exp Ther Med. 2012;5(3):835–9.

Li X, Shek DTL, Shek EYW. Offline Victimization, Psychological Morbidity, and Problematic Online Behavior Among Chinese Secondary School Students. Int J Environ Res Public Health. 2021;18(18):9462.