Role of Culture, Diet, Genetics in Gout Management

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Contributor: Youssef M. Roman

Gout is a metabolic disorder, and one of the most common inflammatory arthritic conditions, caused by elevated serum urate (SU). Gout prevalence is globally rising, partly due to global dietary changes and the growing older adult population. Gout was known to affect people of high socioeconomic status. Currently, gout disproportionately affects specific population subgroups that share distinct racial and ethnic backgrounds. While genetics may predict SU levels, nongenetic factors, including diet, cultural traditions, and social determinants of health (SDOH), need to be evaluated to optimize patient treatment outcomes. A cultural assessment may inform the development of culturally tailored dietary recommendations for patients with gout. Causal and association studies investigating the interaction between diet, genetics, and gout, should be cautiously interpreted due to the lack of reproducibility in different racial groups. Optimal gout management could benefit from a multidisciplinary approach, involving pharmacists and nurses.

acculturation gout management hyperuricemia culture diet genetics

1. Introduction

Gout is a metabolic disorder, and one of the most common inflammatory arthritic conditions worldwide, caused by persistent hyperuricemia. Developing gout is multifactorial, ushering in different methodological approaches to ascertain the risk factors associated with developing hyperuricemia and gout. Despite substantial advancement in understanding the biological basis of gout, it remains one of the most poorly managed chronic conditions in healthcare. Uncontrolled gout is associated with a poor quality of life, joint damage, an increase in missed days of work, and a higher utilization of the healthcare system resources [1][2][3].

Gout is a chronic inflammatory condition caused by persistent hyperuricemia, leading to the formation and deposition of monosodium urate crystals into and around the distal joints. The development of hyperuricemia and gout is heterogenous, and, therefore, different research approaches are needed to identify and quantify the distinct risk factors in the pathogenesis of both conditions. For example, the Mendelian Randomization (MR) approach provides a pathway to ascertain causality, exploiting the natural randomization of allele causal disease. However, this approach is not without limitations, possibly due to the pleiotropic effect of the selected instrumental variables [4]. As gout continues to disproportionately affect non-EUR populations, there is a growing need to increase the representation of minorities in genetic research and cross-validation of genetic findings in multiple populations. To that end, it recognized that developing hyperuricemia and gout is a multifactorial process founded in genetics and modulated by epigenetic factors, including medications, lifestyle factors, diet, and the potential interactions

between all of them. While genetic polymorphisms in *ABCG2* and *SLC2A9* remain two of the most significant signals in developing hyperuricemia and gout across different populations, evaluating nongenetic factors across selected populations through a cultural lens is an adjunct approach to further stratify hyperuricemia and gout risk and optimize gout management. This encompassing approach could be a valuable tool for gout patients with strong cultural identities and distinct racial or ethnic backgrounds. A summary of the major genes associated with regulating uric acid in humans is listed in **Table 1**.

Table 1. Summary of major urate regulation genes.

Gene	Protein	Possible Functions
ABCG2	ATP binding cassette subfamily G member 2: ABCG2	Regulating renal and gut excretion of urate. Gene polymorphisms are strongly linked to urate underexcretion and the risk of early-onset gout in men. Genetic polymorphisms may also influence the therapeutic response to allopurinol and other statin medications.
GCKR	Glucokinase regulator	Regulatory protein that inhibits glucokinase in the liver and pancreatic islet cells by forming an inactive complex with the enzyme. Gene polymorphisms are associated with fasting glucose, maturity-onset type-2 diabetes, hyperuricemia, and gout.
LRRC16A	Capping protein regulator and myosin 1 linker 1: CARMIL1	Cytoskeleton-associated protein. Gene polymorphisms are associated with urate concentrations and gout subtypes.
PDZK1	PDZK domain-containing scaffolding protein	Mediates the localization of cell surface proteins and plays a critical role in cholesterol metabolism. Gene polymorphisms are linked to dyslipidemia, hyperuricemia, and gout.
SLC2A9	Solute carrier family 2 member 9: GLUT9	Regulating renal uric acid reabsorption. Gene polymorphisms are linked to the risk of gout in women.
SLC16A9	Solute carrier family 16 member 9: MCT9	Regulating monocarboxylic acid transporter. Gene polymorphisms are linked to uric acid concentrations.
SLC17A1	Solute carrier family 17 member 1: NPT1	Sodium phosphate cotransporter. Gene polymorphisms are linked with hyperuricemia and gout.
SLC22A11	Solute carrier family 22 member 11: OAT4	Urate reabsorption transporter. A target for some uricosuric drugs. Gene polymorphisms are associated with hyperuricemia.
SLC22A12	Solute carrier family 22 member 12: URAT1	Uric acid reabsorption transporter. A major target for uricosuric drugs. Gene polymorphisms are associated with hyperuricemia and gout. Loss of function in the gene can also lead to hypouricemia.

2. Heritability of Urate Levels and Urate-Modifying Factors

Twin studies have demonstrated that serum urate (SU) levels and hyperuricemia are genetically linked with heritable estimates of 40 and 60%, respectively [5][6]. While high SU levels are strongly predictive for developing gout, not all hyperuricemia cases will result in gout, suggesting that gout is a trait influenced more by the environmental factors than the inherited factors [5]. This knowledge supports that many cases of gout could be preventable. Furthermore, specific dietary and other social and behavioral factors could significantly influence SU levels [7]. For example, social lifestyle factors such as smoking and alcohol intake could decrease and increase SU levels, respectively [8]. Health and nutritional supplements (e.g., niacin, vitamin C, cherries, and fish oil) and physical activity levels can further modulate SU concentrations and the prognostications of chronic hyperuricemia [9][10][11]. Certain medications may also affect SU levels, which warrants using or avoiding certain prescription drugs in patients with gout when compelling indications persist [12]. A summary of the effect of major dietary patterns and lifestyle factors on uric acid levels and gout risk is listed in **Table 2**.

Table 2. Effect of dietary patterns and lifestyle factors on serum urate and gout risk management.

Diet/Food/Lifestyle Factor	Serum Urate Level	Incident Gout	Gout Flare Risk	ACR 2020 Recommendations [13]	References
DASH diet	↓	↓	↓	No recommendation	[14][15][16]
Mediterranean diet	↓	↓	↓	No recommendation	[17]
Ketogenic diet	↓	No data	No data	No recommendation	[18]
Low-fat dairy products	↓	↓	↓	No recommendation	[19][20]
Cherries	↓	↓	↓	No recommendation	[21][22]
Coffee	↓↔	↓	↓	No recommendation	[23][24][25][26]
Tea	↑↔	No data	No data	No recommendation	[25][26][27]
High-fructose corn syrup (HFCS)	↑	↑	↑	Conditionally recommends limiting the intake of HFCS	[28][29]
Weight loss	↓	↓	↓	Conditionally recommends a weight loss program	[30][31]

Diet/Food/Lifestyle Factor	Serum Urate Level	Incident Gout	Gout Flare Risk	ACR 2020 Recommendations [13]	References
Physical exercise	↓	No data	No data	No recommendation	[9][30]
Smoking	↑↓	↓	No data	No recommendation	[32][33][34]
Alcohol	↑	↑	↑	Conditionally recommends limiting alcohol intake	[35][36][37]
Vitamin B complex (B6-B12-Folic acid)	↓	No data	No data	No recommendation	[38]
Vitamin C	↓↔	No data	↓	Conditionally recommends against use	[10][39][40]
Fish Oil/Omega-3-fatty acids	↓	No data	↓	No recommendation	[11][41][42]

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3. Gout Risk and Acculturation

Dietary habits are mirrors of cultural customs and traditions. Certain population subgroups tend to follow a specific diet for personal, social, and religious norms [43][44]. Nonetheless, immigration or acculturation could significantly impact the lifestyle and dietary habits of the same population groups [44][45][46]. These changes could have significant effects on the individuals' overall health, ranging from energy expenditure-related activities to their gut microbiome. Collectively, these changes could have a consequential impact on developing cardiometabolic risk factors, including hyperuricemia and gout. Similarly, Filipinos living in the US were reported to have higher gout rates and elevated mean serum urate levels compared with Filipinos living in the Philippines, suggesting significant gene–environment interactions [47][48]. Comparatively, developing hyperuricemia or gout among migrant groups could be owing to a more permissive environment, increasing the penetrance of risk alleles by equivalent effects [44][49]. For example, the joint effect of alcohol consumption and carrying the risk allele of *ABCG2* rs2231142 G > T was associated with a greater risk for developing hyperuricemia than the risk allele alone, especially among women [50]. Therefore, ascertaining the dietary and social lifestyle habits among distinct racial and ethnic groups could shed additional light on the hypothesis of gene–diet/gene–social habits interactions and population-specific risk for developing hyperuricemia or gout [35][51]. Additionally, this ascertainment may provide more evidence on the role of preserving a cultural identity in disease prevention and the generational effect on disease onset among immigrant groups.

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4. Gout Risk and Health Beliefs

5. Gout and Social Determinants of Health

Many factors could lead to ethnic and racial disparities in the prevalence and management of chronic diseases, including gout [43][44][58][59][60]. While the biomedical framework for inequality remains the operational framework to provide insights into addressing gout health disparities, little is known about the role of SDOH and gout. This role is compounded by the association between gout and multiple cardiometabolic diseases [61]. It is presumed that optimal management of gout-related comorbidities may confer added benefits for gout management itself. Therefore, optimal management of gout is believed to be within the context of the optimal chronic disease management framework. However, the management of gout remains poor despite the availability of effective and affordable treatments. Many factors, ranging from individual to organizational causes, can lead to suboptimal management of gout. On an individual level, financial barriers, health insurance, health literacy, and access to

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healthy food choices are significant contributors for optimal gout management. On an organizational level, the knowledge base of gout management among healthcare professionals, adequate training of gout in health profession programs, patient–physician relationships, and effective guidelines for gout management are crucial elements for delivering optimal care for patients with gout. Assessing the role of SDOH in patients with gout may provide a window of opportunity to address treatment goals and eliminate barriers to receiving care. Furthermore, future gout studies are also needed to quantify the impact of SDOH on disease onset and treatment outcomes to inform the value of a comprehensive assessment for optimal gout management.

6. Multidisciplinary Approach to Gout Management

A multidisciplinary approach to chronic disease management improves treatment outcomes across many disease states. Similarly, gout management could be optimized by engaging multiple healthcare professionals in addition to physicians, including nurses, pharmacists, and dietitians. According to the 2020 American College of Rheumatology guidelines, an augmented treat-to-target protocol by nonphysician providers is conditionally recommended over the usual care [13]. Recognizing that patients with gout are more than likely to have other comorbidities, which also require close monitoring and complex treatment regimens, the pharmacist is well-positioned to optimize gout management outcomes. Indeed, pharmacist-led interventions to improve adherence to allopurinol and gout treatment, and better treatment outcomes than the usual care. Specifically, in a one-year pharmacist-led intervention, incorporating automated telephone technology, led to a 30% improvement in adherence rates to allopurinol and more than a two-fold increase in the number of patients achieving SU levels less than 6 mg/dL, compared with the usual care. Moreover, participants in the intervention arm were twice as likely to have their allopurinol dose escalated compared with the standard of care [62]. These results support the role of a pharmacist-staffed gout management clinic to improve gout treatment outcomes compared with the standard of care. Regardless of the clinical setting, including virtual gout clinics, pharmacist involvement in gout management significantly improves the number of individuals achieving SU levels of less than 6 mg/dL, with these patients having fewer gout flares than those receiving the standard of care [63][64]. As new patient care models continue to emerge, community-based pharmacy practice may also play a role in health equity and increasing access to healthcare among patients with chronic diseases [65].

7. Pharmacogenomics and Gout Management

Pharmacogenetics (PGx) is a growing field within precision medicine, focusing on how gene variations affect the patient's response to treatment. Pharmacogenetics is a promising tool to optimize the selection and dosing of medications, including ULTs among patients with gout [66]. Genetic and experimental findings have demonstrated that genetic polymorphisms associated with SU pathology are also of pharmacogenetic interest [66]. Patients with gout often present with several comorbidities, warranting the use of several acute and long-term medications that increase their pill burden and the risk of adverse drug events. Implementing PGx testing can identify individuals who are more or less likely to benefit from a given treatment, thereby potentially improving medication adherence and reducing pill burden. The strongest evidence today suggests that individuals carrying

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the HLA-B*5801 allele are at a higher risk of serious and life-threatening skin reactions when taking allopurinol [13][67].

Emerging evidence of clinically significant drug–gene pairs among various gout therapies is growing. Allopurinol remains the most widely prescribed ULT; therefore, identifying sources of variability in response to allopurinol is an active area of research. Genes that were found to modulate the response to allopurinol include *AOX*, *ABCG2*, and *SLC22A12* [68][69][70][71]. Meanwhile, *UGT1A1* appears to modulate the response to febuxostat. While CYP2C9 may modulate the toxicity of benzbromarone, the *SLC22A12* and ABCB1 were found to modulate the response to both benzbromarone and probenecid. The genes *CYP2D6*, *ABCB1*, gene cluster (rs6916345 G > A), and SEPHS1 were recently reported to modulate the safety and efficacy of colchicine [66][72]. Finally, *HCG22* and *IL1RN* could be linked with the response to corticosteroids and anakinra, respectively [66].

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