

**عنوان مقاله:**  
GOUT Diet

**محل انتشار:**  
همایش علمی دانشجویان علوم تغذیه (سال: 1395)

تعداد صفحات اصل مقاله: 1

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**خلاصه مقاله:**

Introduction: Gout is one of the most common inflammatory arthritis's, caused by hyperuricemia, with an increasing prevalence. Hyperuricemia is often a consequence of renal under-excretion of uric acid as more than 70% of urate is excreted via the kidney primarily through the proximal tubule on-modifiable risk factors, including sex, age and race or ethnicity, have been under investigation for potential roles in gout development, In addition to these non-modifiable risk factors, modifiable or lifestyle factors play a significant role in reducing or increasing the risk of gout. Aim: This review focuses on the non-modifiable and modifiable risk factors of gout. With the increasing prevalence of gout, a strong knowledge of these risk factors for preclinical gout and hyperuricemia is important so that at-risk individuals can be identified and appropriately counseled. Result: More formal research from the 1960s demonstrated that alcohol administration caused decreased uric acid excretion and hyperuricemia. Ethanol ingestion increases serum lactate levels which inhibit uric acid excretion at the renal tubule. In terms of the production theory, ethanol prompts adenosine triphosphate (ATP) consumption leading to purine degradation, yielding an increase in plasma oxypurines and uric acid. Food rich in purines including meats, seafood, some vegetables, and animal protein have been theorized to lead to gout, as uric acid is the end product of purine degradation. Skepticism existed as protein can have a uricosuric effect which would actually lower urate levels. Despite moderate purine content in soy, soy has not been shown to be associated with gout and may be inversely associated with hyperuricemia. Overall as diets have come to include increasing quantities of fructose and sugar-sweetened beverages (main sweetener being fructose), these additives have come under investigation for their contribution to gout. Initial studies on these sweeteners found increased plasma uric acid and lactate levels probably driven either by purine nucleotide degradation or purine synthesis. For those at risk for gout. Vitamin C has been touted as protective against gout, ingestion of ascorbic acid was found to increase the fractional clearance of uric acid resulting in a reduction of serum uric acid. Supplementation with 500 mg/day of vitamin C significantly reduced serum uric acid levels in an RCT with a mean uric acid reduction of 0.5 mg/dl. Conclusion: Diet and lifestyle could be effective on recovering patients, so those at risk for hyperuricemia or ... gout should be educated on modifiab

**کلمات کلیدی:**

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