Effects of Dietary Factors on Gout: A Systematic Review

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ABSTRACT

Recent studies reveal that in spite of the increase in the prevalence of gout, the role of dietary risk factors in the development and management of this condition remains unclear.

Therefore, this review work aimed at clarifying the role of dietary factors in the risk and management of gout.

An extensive search of literatures published between 1960, to 2018 was performed on the databases of PubMed, CINAHL, Science direct, Cochrane, BMJ, Ann Rheum Dis, and BioMed to identify relevant Cohort, Prospective, and Population based, or Cross-sectional studies that examined the effect of diet on gout.

A total of 19 studies (2 Cohort studies, 5 Prospective studies, 1 Population based studies, and 11 Cross sectional studies) were included in this review work. And the methodological quality of these studies was evaluated using the quality assessment tool for observational and cross-sectional studies developed by the National Heart, Lungs and Blood Institute.

This work revealed that a positive association exist between intake of sugar sweetened beverages and the risk of gout. It also revealed an inverse relationship between the increase in coffee consumption and the risk of gout. The multivariate relative risk (RR) for incident gout based on coffee intake grouping of 0, <1, 1 - 3, 4 - 5, and > 6 cups per day, were 1.00, 0.97, 0.92, 0.60 (95% confidence interval 0.41 - 0.87), and 0.41 (95% confidence interval 0.19 - 0.88), respectively (*P* for trend is equal to 0.009).

In addition, this work also recorded a positive association between risk of gout and the consumption of either meat or sea food. The multivariate relative risk of gout among participants who consumed meat at a mean daily intake of 2.5 servings per day was recorded as 1.41 (95% CI, 1.07 to 1.86; P for trend = 0.02).

In summary, this research successfully clarified the role of dietary factors in both the risk and the management of gout. It also showed that while the consumption of sugar-sweetened beverages, purine - rich foods (like meat and seafood), and fructose rich fruits increased the risk for gout, the consumption of coffee and dairy products reduced the risk for gout. Therefore, it is safe to suggest that dietary risk factors should be considered when gout patients are being managed.

Keywords: Gout, Arthritis, Dietary factors, sugar-sweetened beverages, purine - rich foods fructose rich fruits.

INTRODUCTION

Gout is a disease of ancient origin, which in recent times is on the rise in most western regions of the world (Nuki & Simkin, 2009). It was described by the Hippocrates as the “unwalkable disease” and was associated with an imbalance of one out of the four humours of which an individual’s health is maintained. Overtime that exact excess “humour” has been clinically identified to be uric acid (Nuki & Simkin, 2009). Uric acid is insoluble and the end product in the metabolism of purines (Edward, Christian & Michael, 2013). About 70% of uric acid in the body is derived from the metabolism of endogenous purines, whilst the other 30% is from dietary purines (Edwards, 2008).

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The western diet has however developed into one that is rich in purine, notwithstanding income levels, thus possibly promoting the prevalence of gout in the United Kingdom (UK) and United States (US) (Edward, Christian & Michael, 2013; Zhu, Pandya & Choi, 2011 and Nuki & Simkin, 2009). Gout affects 1 - 2% of the UK population and is the commonest type of inflammatory arthritis (Edward, Christian & Michael, 2013). Also, Choi et al, 2004; and Roubenoof et al, 1991 in their studies further described gout as an inflammatory arthritis that most commonly affects men that is characterized by the elevation of serum uric acid (SUA) levels; and the general burden caused by the disease as being substantial and remains on the rise. Of all the many risk factors of gout, hyperuricemia (raised urate concentration in the serum), happens to be the major risk factor of gout. It leads to monosodium urate crystals deposition in, as well as around the joints thus resulting in severe pain and inflammation of the affected joints (Chandratre, Roddy, Clarson, et al, 2013). When left untreated the continued deposition of urate crystals can lead to an irreversible damage of the affected joint (Edward, Christian & Michael, 2013; and Vitart et al, 2008).

Most commonly, a decreased renal elimination or increased production of uric acid, results in hyperuricemia (Zhu, Pandya & Choi, 2012; and Milkus, Farrar, Bilker et al, 2005). The epidemiological studies of Zhu, Pandya & Choi, 2012; Roddy & Doherty, 2011; and Mikuls, Farrar, Bilker et al., 2005, reports that metabolic syndrome alongside its components (obesity, hypertension, insulin resistance and hyperlipidaemia), are adversely linked to gout.

Gout is a form of arthritis caused by a chronic increase of body uric acid level to a point that results in the formation of monosodium urate crystal in joints and other tissues (Zhu, Pandya, & Choi, 2011). Epidemiological studies have in recent decades indicated an increase in both the prevalence and incidence rate of gout. For instance, Lawrence, Hochberg, Kelsey, et al (1989) reported that, the prevalence of self-reported gout cases in United States increased from 4.8 per 1000 Americans in 1969 to 7.8 per 1000 Americans in 1976, and increased further to 8.3 per 1000 Americans in 1980. Also, Zhu, Pandya, & Choi (2011), reported that the National Health and Nutrition Examination Survey (NHANES) in USA found an increase in gout prevalence from 26.4 per 1000 Americans in (1988–1994) to 37.6 per 1000 Americans in 2007–2010 (Zhu, Pandya, & Choi 2011).

Similarly, an increase in the prevalence of gout is also evident in the United Kingdom. Currie (1976); Steven (1992) and Harris, Lloyd, Lewis (1995) in their separate studies revealed that the estimated lifetime prevalence of gout increased from 2.6/1000 in 1975 to 3.4/1000 in 1987 and increased further to 9.5/1000 in 1993 (Currie, 1976; Steven, 1992; and Harris, Lloyd, & Lewis, 1995). Also, Mikuls, Farrar, Bilker et al. (2005) highlighted an annual consultation prevalence of gout to be 13.9/1000 in 1999 (Mikuls, Farrar, Bilker et al., 2005).

Data from China and New Zealand also shows an increasing prevalence in gout in these countries. In New Zealand, the studies of Edward & Hyon, (2014) showed that a prevalence life time estimate amongst subjects of European descent increased from 3/1000 in 1958 to 9/1000 in 1966 and further increased to 29/1000 in 1992 (Edward & Hyon, 2014). Similarly, Nan, Qiao, Dong, et al. (2006) and Miao, Li, Chen, et al. (2008) indicated that Survey data from China shows an increase in the prevalence of gout from 3.6/1000 to 5.3/1000 between 2002 and 2004 (Nan, Qiao, Dong, et al., 2006; and Mio, Li, Chen, et al., 2008).

The prevalence of gout in New Zealand, China, United Kingdom, and USA as highlighted in the abovementioned studies had some variation. These variations could be the result of demographics, differences in genetics, the methodology adopted by each study, and diet/ lifestyle. But in spite of these variations, when the gout prevalence of each country in the study is considered in isolation, it gives credence to the conclusion that gout has become more prevalent in recent decades.

On the incidence of gout, result from a study in the USA where 1216 male medical students were recruited and observed for 29 years revealed a gout incidence of 1.73 per 1,000 person- years. This implied that about 5% of the observed students developed gout within the study period (Roubenoff et al., 1991). A similar incidence rate of 1.50 per 1000 persons' years was also observed in the Health Professionals Follow- Up Study (HPFS) that

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followed 47,150 male health workers for a period of time 12 years and found 720 incident gout cases (Choi et al., 2004)

The Framingham Heart Study that followed 1,951 men and 2,476 women between the age of 29 and 62 years reported a greater incidence of gout in men (4.0 per 1,000 persons - years) than women (1.4 per 1,000 persons - years) (Bhole et al., 2010).

In a large study of 1,775,505 individuals (aged 20 to 89 years) between the periods of January 2000 to December 2007, Cea et al. (2011) reported 24,768 incident gout cases, which translates to approximately 2.68 per 1,000 per - years.

Pathophysiology of Gout

Hyperuricemia (high uric acid level in the blood) is a very strong risk factor for gout, although not everyone with hyperuricemia develops gout (Champion, 1987 and Wortmann, 2002). Gout is associated with the formation of monosodium urate (MSU) crystals in the joints and soft tissues. The formation of these crystals begins when the level of serum uric acid (a product of sodium and uric acid) rises beyond the body's fluid saturated threshold. (Chandratre et al., 2013). A pictorial representation of the formation of MSU crystals in gout is shown in figure 1.0.

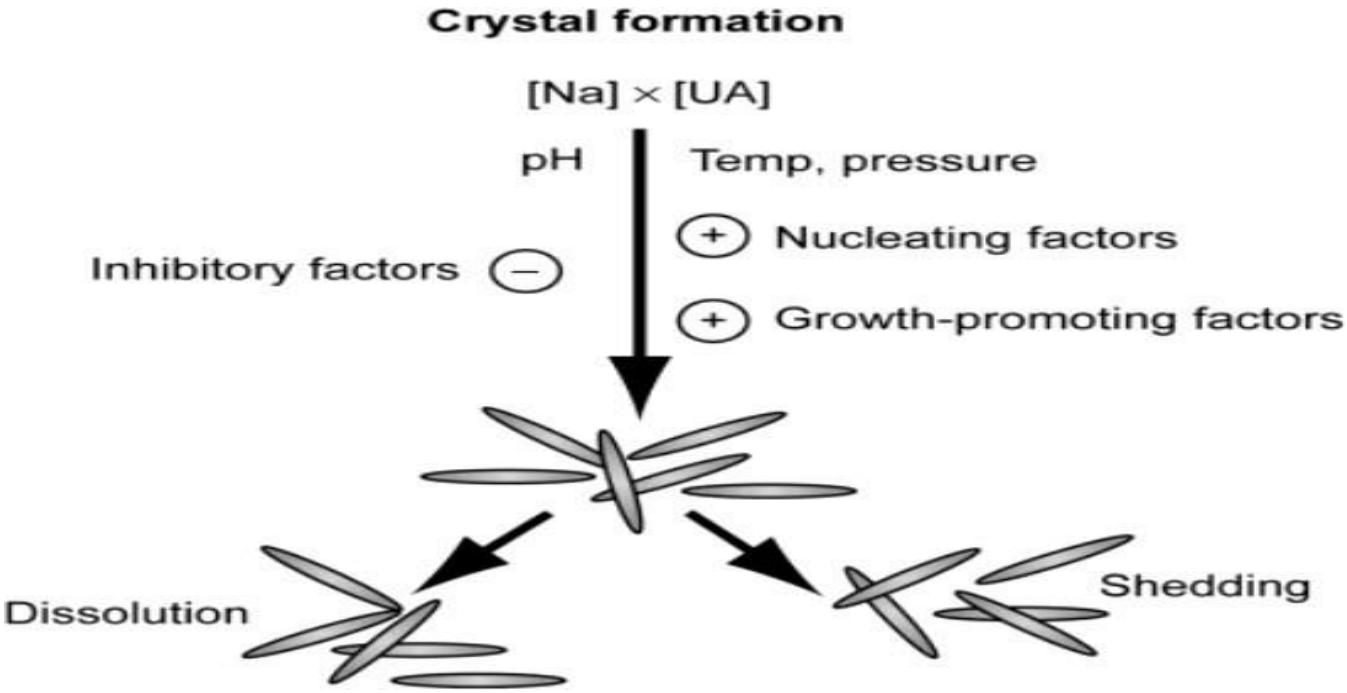

Figure 1: Crystal formation in gout (Dorherty, 2009).

Uric acid is a weak acid which exists in its ionized form as urate at a basic physiological pH of 7.40. Under these physiological conditions, monosodium urate can become insoluble in plasma. The insolubility of monosodium urate in plasma occurs because it has a saturation level in plasma of 6.8 mg/dl at a physiological pH of 7.40. Therefore, the formation of monosodium urate (MSU) crystals in the joints and soft tissues abounds when the plasma concentration exceeds the monosodium urate saturated level of 6.8 mg/dl. (Pittman, 1999 and Choi, 2005). Therefore, because the serum urate level in men and women is very close to the urate saturated level of 6.8 mg/dl, there is a high chance of monosodium urate (MSU) crystals formation in joints.

Additionally, Roddy (2007) reported that the formation of crystals in joints or soft tissues depends largely on the balance of tissue inhibitors. Although very little has been reported about such tissue factors but studies associated with osteoarthritis (OA) highlights that the balance of tissue inhibitors promotes the formation of crystals such as; the MSU crystals, calcium pyrophosphate crystals and the basic calcium phosphate crystals.

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The formation of monosodium urate crystals often occurs within the cartilage and fibrous tissues because the cartilage and fibrous tissues offer the crystals protection from contact with inflammatory mediators (Nathalie & Alexander, 2010).

Most often, these crystals remain within this initial protection for years without causing problems but once they are released into the joint space, they turn into highly phlogistic crystals that gets phagocytosed very quickly by monocytes and macrophages. This action further results in the activation of the NALP3 inflammation which triggers the release of IL-1 and other cytokines (Martinon et al., 2006). This severe inflammatory response results in the symptoms experienced by gout patients. Fig. 2 below describes multiple steps for triggering gout inflammation.

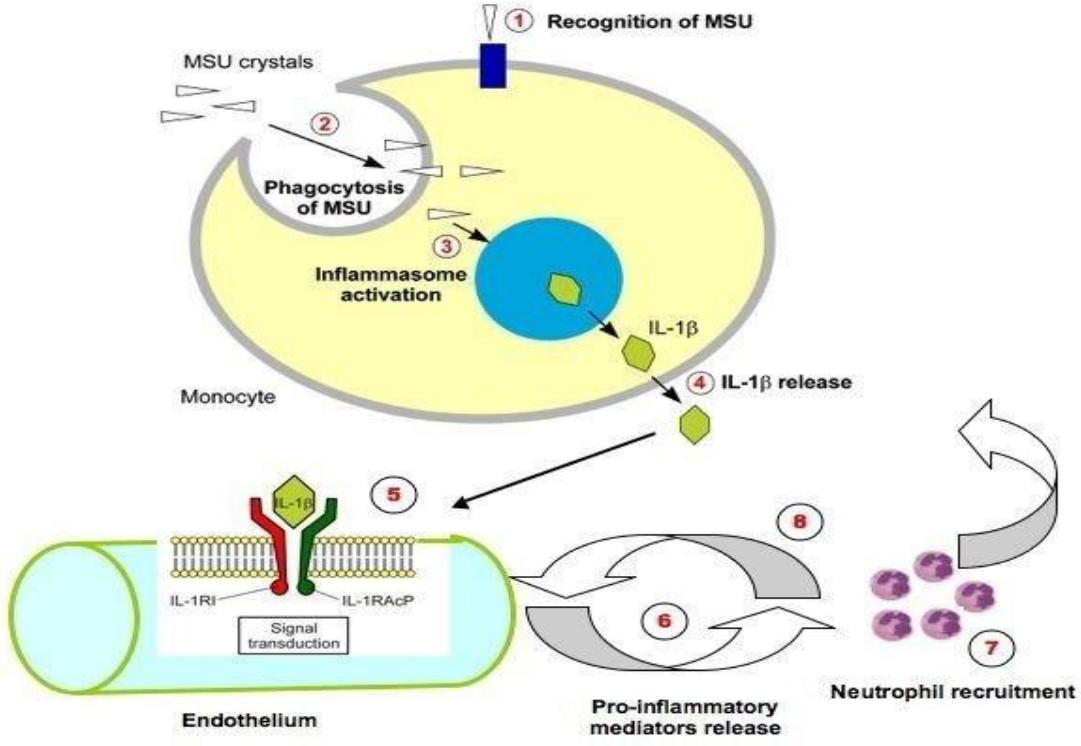

Figure 2: Pictorial description of multiple steps for triggering gout inflammation (Nathalie & Alexander, 2010; and Martinon et al., 2006).

(1) Components of innate immune system recognizes crystals of monosodium urate. (2) Monosodium urate is taken up by phagocytotic cells. (3) NALP3 (also known as cryopyrin comprising of the NLR protein) inflammasome is activated by monosodium urate (4) IL-1β is released from the cell (5) Activation of type 1 (IL1R) endothelial IL-1β (6) release of pro-inflammatory mediators (inclusive of IL-8) which in turn releases a potent chemokine for recruiting neutrophil (7) Neutrophil recruited into the inflamed site (8) Neutrophils releases pro-inflammatory compounds which includes more IL-1β.

The Genetic basis of gout

Often times, gout is prevalent in families. Possibly, this is related to factors such as family lifestyle and genetic/ hereditary. A genetic predisposition to gout could/ might be inherited by an individual/ sufferer, with various unusual defects of enzymes being a known cause (Emmerson, Nagel, Duffy & Martin, 1992). Gout has always been considered a monogenetic trait but results from recent studies suggest that it has a polygenic mode of inheritance. For instance, Genome-Wide Association Studies (GWAS) confirmed various variants that increases the risk of developing gout (Reginato et al., 2012; Yang et al., 2010; and Mount, 2013).

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The GWAS have uncovered 8 DNA sequence variations that are associated with different degrees of increased serum uric acid. These studies identified the SLC2A9, SLC22A11, and SLC22A12 genes for urate transporters as being highly associated with hyperuricemia (Yang et al., 2010).

Similarly, Glut-9 which is a transporter that correspond to the SLC2A9 genes was identified as a contributor toward the re-absorption of urate at the proximal tubules of the kidney; an activity that is responsible for about 3.7% of the serum uric acid variance in a patient (Reginato et al., 2012).

Also, recent reports suggest that DNA sequence variations in the genes responsible for the transportation of urate that is associated with hyperuricemia are found in people of European ancestry and in some Asian and black populations. It has been suggested that approximately 6% of the serum uric acid variation that is associated with hyperuricemia is a result of genetic variances (Obi & James, 2024; Eric & Denise, 2014; Reginato et al., 2012; Merrimam & Dalbeth, 2011 & Yang et al., 2010).

Development of Gout

The development/inflammation of gout has been in existence many years before now. It involves the deposition of monosodium crystals in joint tissues, tendons etc. The relationship between joint inflammation and the deposition of crystals was made by Garrod, when he developed a test known as “thread test assay of serum urate; which he explained saying that the deposition of serum urate soda might be seen as the cause of gout flares and not its effects (Nathalie & Alexander, 2010 and Garrod, 1876).

This development brought about the manifestation of acute gout. McCarty & Hollander, (1961) observed and reported that crystals seen in synovial fluid were composite of monosodium urate. However, there have been no doubts that urate crystal deposition results to intense inflammation (gout). Gout is made up of three phases: (1) Hyperuricemia phase, (2) deposition phase, and (3) Acute attack phase.

Over a period of 5 years, 22% of men develop gout and recorded a greater than 9 mg/ dL serum uric acid level (Choi, Atkinson, Karison, Willett & Curhan, 2004). Acute gout flares have been linked with fluctuations in the levels of serum uric acid and monosodium urate crystal which begins to form, as well as deposits in joints, tendons and bursas when serum uric acid levels are greater than 6.8 mg/ dL (Choi, Willett & Curhan, 2007; Choi, Atkinson, Karison, Willett & Curhan, 2004; Choi, Atkinson, Karison, Willett & Curhan, 2004; and Emmerson, Nagel, Duffy & Martin, 1992).

Association between diet and the development of gout

Although the association between dietary factors (which includes food rich in purines and alcoholic drinks) and gout has been recognised for decades, only in recent time have the associations been reported in quality prospective studies (Choi, Atkinson, Karison, Willett & Curhan, 2004; and Choi, Atkinson, Karison, Willett & Curhan, 2004).

The intake of alcohol is linked to gout. The studies of Choi, Willett and Curhan, (2007); Choi, Atkinson, Karison, Willett & Curhan, (2004); and Emmerson, Nagel, Duffy & Martin, 1992) reported that the consumption of beer is highly associated with the risk of gout development (where each additional 10g of alcohol intake is associated with a relative risk of 2.51, 1.77 to 3.55 with a 95% CI); consumption of wine and liquor is linked with low and moderate risks of gout respectively. Studies of Choi, Atkinson, Karison, Willett & Curhan, (2004) further reported that the risks of gout development is directly linked to the consumption of alcohol. The study further reported that a multivariate relative risk of 1.17 is associated with each additional 10g of alcohol intake per day, and a confidence interval of 95% (1.11 to 1.22). Again, the study showed that the risk for the development of gout is high in the consumption of beer; 2.51, 1.77 to 3.55 (multivariate relative risk) (same as one of their studies earlier mentioned together with the study of Emmerson, (1992) and consumption of spirit; 1.60, 1.19 to 2.16 (multivariate relative risk). However, wine consumption was least and reported a multivariate relative risk of 1.05.

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Furthermore, Choi, Atkinson, Karison, Willett & Curhan, (2007); and Emmerson, Nagel & Martin (1992), reported that a high intake/ consumption of meat and sea foods rich in purine are linked with increased levels of serum uric acid; however, increased intake of vegetables rich in purine is not linked with an increased risk of gout.

Additionally, in relation to the association of diet and the development of gout, the studies of Roddy, Mallen & Doherty, (2013); and Choi & Curhan, (2008) described dairy products as being protective against the development of gout (0.54, 0.42 to 0.74 multivariate relative risk at 95% confidence interval). They further reported that sugar sweetened soft drinks (particularly those containing fructose) increases the risk for the development of gout, with a recorded relative risk of 1.85, 1.08 to 3.16 relative risk. On the other hand, caffeinated and decaffeinated coffee intake is thought to play a vital role in decreasing the risks of gout development (0.41, 0.19 to 0.88 relative risk).

Dietary Risk Factors

Purine - rich food and Gout incidence

Over the years, they've been notable relationship between gout incidence and the habitual consumption of food rich in purines. Emmerson (1996) and Fam (2002) had earlier suggested in their respective studies that gout patients should be encouraged to avoid consistent consumption of red meat, sea food, and some vegetables rich in purine because these could contribute to the increase of purine level in the body (Emmerson, 1996; and Fam, 2002). Similarly, Choi, Atkinson, Karlson, Willett, & Curhan (2004) had highlighted information from Health Professionals Follow-up Study (HPFS) which associated higher risk of gout attack on consumption of high red meat diet. Their study also went ahead to suggest that high seafood intake had lesser but significant increase in the risk of gout attack compared to the risk level associated with high intake of red meat (Obi & Ishiekwen 2024; Choi et al, 2004). Furthermore, the study suggested that purine rich vegetable diets increases the risk of gout attack while diets rich in low-fat dairy products have relatively low risk of gout attack (Choi et al, 2004).

Fructose-Rich Beverages and Risk of Gout in Women

Choi, & Curhan (2008) revealed that the increase in the annual incidence of gout in women in the United States coincided with the increase in the consumption of soft drinks and fructose.

Although, soft drinks that are sweetened with sugar contain low purine levels, they contain high levels of fructose (a carbohydrate known to increase the level of uric acid in the body). The intake of fructose in humans leads to rapid increase in serum uric acid which increases the synthesis of purine (Choi, Ford, Gao, & Choi, 2008). According to Young, Yanyan & Hyon (2012) the fructose consumed by humans' result in the production of uric acid by increasing the degradation of Adenosine triphosphate to Adenosine monophosphate. Therefore, few minutes after the infusion of fructose, an increase in the level of serum uric acid id experienced.

Choi, Willett, Curhan, (2010) made an effort to study the link between fructose intake and the risk of incident gout in women. The study documented 778 newly diagnosed gout cases in women within a period of 22 years. And went ahead to characterize its cohort based on their consumption levels of sugar-sweetened soda and free fructose. Finally, Choi, Willett, Curhan, (2010) concluded by confirming that fructose and fructose-rich beverages are important risk factors to be considered in the primary prevention of gout among women.

Again, Choi & Curhan (2008) used data from HPFS to highlight relative association between sugar sweetened soft drinks and new gout cases. Their study also revealed that the highest quintile of fructose intake had an adjusted risk of gout that was twice the risk in the lowest quintile (Choi & Curhan, 2008).

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Soft drinks, fructose consumption, and the risk of gout in men

Choi, Atkinson, Karlson, Willett, Curhan (2004) and Choi, and Curhan, (2007) revealed that its prevalence and incidence have doubled over the last decades in the United States. And Choi, and Curhan, (2008) highlighted that the doubling in the prevalence and incidence rate of gout has a relationship with the substantial increase in the consumption of soft drinks and fructose.

In a prospective cohort study, Choi, and Curhan, (2008) "examined the relation between intake of sugar sweetened soft drinks and fructose and the risk of incident gout in men". The study used validated food frequency questionnaire to collect information on the soft drink and fructose intake of about 46,393 men over a period of 12 years.

Coffee Consumption and Risk of Incident Gout in Men

Coffee is widely consumed around the world with over 50% of Americans consuming coffee at an average of 2 cups per day (Salazar-Martinez, 2004 & Lundsberg, 1998). According to Kiyohara et al., (1999), Petrie (2004) & Greer et al., (2001) coffee may lower the risk of gout through reducing serum uric acid levels. However, the high level of coffee consumption and the current growing level in the overall disease burden of gout (Choi, Mount, & Reginato, 2005) makes the review on the impact of coffee consumption on incident gout important. Recently, Hyon, Walter, & Gary (2007) conducted a perspective study on the consumption of coffee and the risk of incident gout in men. In the study, 45,869 men who had no history of gout at baseline were observed for a period of 12 years. Both validated and supplementary questionnaires were then used to respectively collate participant's coffee intake and to ascertain if participant's met the American College of Rheumatology survey criteria for gout. At the end, the study highlighted that 1.65% of the participants reported incident cases of gout. And the study further reported that the relative risk for incident gout based on the following coffee consumption categories of 0, <1, 1–3, 4–5, and >6 cups per day were 1.00, 0.97, 0.92, 0.60, 0.41 respectively. With these results in mind, it could be inferred that prolonged consumption of coffee could result in a lower risk of incident gout.

METHODOLOGY

For this review, a systematic search for information relating to the effect of diet on gout was carried out using the following databases; PubMed, CINAHL, Science direct and Cochrane. Basic terms like Gout, dietary factors, gouty arthritis, food intake and gout, beverage intake and gout, diet and gout development, triggers of gout inflammation, gout and genetics, gout development, dietary risk factors of gout, purine - rich foods and gout occurrence, fructose - rich beverages and the risk of gout development in women, consumption of fructose, soft drinks and the risk of gout development in men, consumption of coffee and the risk of gout occurrence in men, and consumption of sugar sweetened beverages and the occurrence of gout, were used to search the aforementioned online databases. The European League Against Rheumatism (EULAR) as well as the American College of Rheumatology was also searched and most abstracts with titles or body capturing gout was reviewed. In addition to the databases searched, journal articles (i.e. manuscripts that were published in peer reviewed journals) was considered most appropriate to gather the needed information for this work and this included BMJ (01/01/1960 - 01/09/2018), Ann Rheum Dis (01/01/1990 - 01/09/2018), BioMed (01/01/1980 - 01/09/2018). The titles and abstracts of each article found in the search results (that was published in English language) were reviewed, and full text of articles that had the possibility of containing relevant information were obtained and reviewed thoroughly.

On completion of the initial literature search criteria, titles and abstracts was reviewed and citations which their titles or abstracts having no relevance to the aims and objectives of this review was excluded. Requests of manuscripts was requested from authors whose articles was not open to the public for details of further data.

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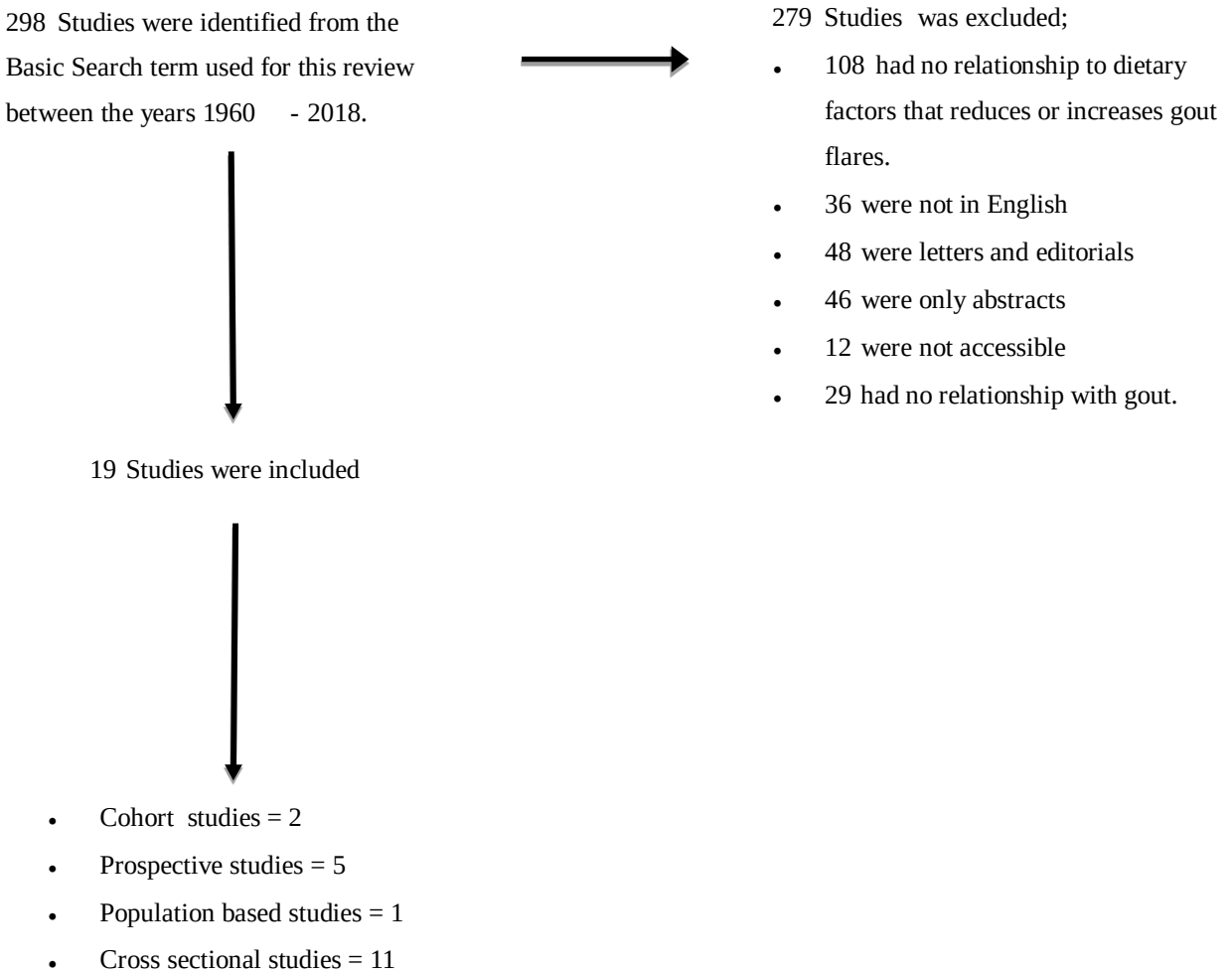
Flow Chart of Studies Included for Review



Results Sugar Sweetened Beverage and The Risk of Gout

As highlighted in Table 1.0, four (4) articles assessed the association between the consumption of sugar sweetened beverage and the risk of gout. Also four (4) additional articles (as shown in table 2.0) studied the relationship between intake of sugar sweetened beverages and either serum urate concentration or serum uric acid level that are both closely associated with the risk of gout. Six out of the eight studies are cross sectional analysis while two are prospective cohort studies. The average scientific - quality score for the reviewed studies is 7.5 out of 16.

Choi and Curhan (2007), and Choi, Willett and Curhan (2011) (The first and second articles presented in table 1.0) analysed the association between sugar sweetened beverages and the risk of gout. Both articles performed a follow-up study that revealed a positive relationship between increasing intake of sugar sweetened beverages and the risk of gout.

Jee, Earl, Xiang, and Hyon (2008) (article highlighted in table 1.0) studied the association between the consumption of both sugar sweetened soft drinks and diet soft drinks on serum uric acid level in men and women. It analysed data from the United States Third National Health and Nutrition Examination Survey (NHANES-III) and used a linear regression model to evaluate the relationship between beverage consumption and serum uric acid level. The study then presented results which reflected a difference of 0.4 mg/dl in the increase in the levels of serum uric acid between the least and highest intake of sugar-sweetened soft drink.

Similarly, Batt et al. (2013) (another article presented in table 1.0), explored the relationship between intake of sugar sweetened beverages and gout prevalence. It also investigated whether the interaction between the

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consumption of sugar sweetened beverages and SLC2A9 genotype could determine the risk of gout. Their work gathered both survey and clinical information on 1,634 New Zealand, European Caucasian, Maori and Pacific Island individuals within a period of 5 years and used logistic regression analysis to study the relationship between the intake of sugar sweetened beverages, and SLC2A9 genotype, and gout. The study went on to report a positive association between the intake of sugar - sweetened beverages and serum urate levels as (Table 1.0). And then concluded that they is a non-additive genotype - specific relationship between the consumption of sugar sweetened beverages and the SLC2A9 genotype.

Siqueira et al. (2018), performed a cross-sectional study on a baseline data from the Brazilian Longitudinal Study of Adult Health. 7,173 participants with a mean age of 50 years were included in the study which employed a multivariate linear regression model to investigate the relationship between the intake of sweetened beverages and serum uric acid levels. Results from the study by Siqueira et al. (2018) revealed an average increase of 0.11mg/dl and 0.30mg/dl of uric acid in men who consumed > 0 to <0.1 servings of sugar - sweetened soft drinks/day and those who consumed >1.0 servings per day respectively, similarly, women who consumed > 0 to <0.1 and >1.0 servings of sugar - sweetened soft drinks/day had an average increase of 0.12 mg/dl and 0.13 mg/dl of uric acid respectively.

Coffee/ tea Intake and the risk of Gout

Five cross sectional studies examined the association between tea intake and serum uric acid concentration (see Table 3.0 for details). Similarly, two prospective studies (detailed in Table 4.0) investigated the relationship between coffee intake and the risk of gout. The average scientific - quality score for the seven reviewed studies is 7.5 out of 16.

Choi, Willett, and Curhan (2007) conducted a prospective study that investigated the association between the intake of coffee and the risk of incident gout over a 12 y period in a cohort of 45,869 men with no history of gout at baseline. A validated food - frequency questionnaire was used to gather information on the consumption of coffee, decaffeinated coffee, tea, and total caffeine among the cohort. Results from the study confirmed 757 incident cases of gout that met the American College of Rheumatology survey criteria for gout. The results also revealed an inverse relationship between the increase in coffee consumption and the risk of gout. Furthermore, the result showed that the multivariate relative risk (RR) for incident gout based on coffee intake grouping of 0, <1, 1 - 3, 4 - 5, and > 6 cups per day, were 1.00, 0.97, 0.92, 0.60 (95% confidence interval 0.41 - 0.87), and 0.41 (95% confidence interval 0.19 - 0.88), respectively (*P* for trend is equal to 0.009). In addition, the results also highlighted the multivariate relative risk for incident gout based on decaffeinated coffee intake grouping as 1.00, 0.83, 0.67 (95% confidence interval 0.54 - 0.82), and 0.73 (95% confidence interval 0.46 - 1.17), respectively (*P* for trend = 0.002).

Choi and Curhan, (2010), conducted a prospective study that evaluated the association between the consumption of coffee, tea, and total caffeine and the risk of incident gout in 89,433 females who participated in the Nurses' Health Study. Participants were followed over a 26 years period and diet was assessed every 2 - 4 years using validated Food Frequency Questionnaire (FFQ). Incident cases of gout were identified using a gout survey which used criteria from the American College of Rheumatology. Using Cox proportional hazards modelling, the study found an inverse relationship between coffee consumption and the risk of gout. Based on coffee intake groupings of 0, 1 - 237, 238 - 947, and ≥ 948 mL /day (where 237 mL = one 8-ounce cup) the RR were 1.00, 0.97, 0.78 (95% confidence interval 0.64, 0.95), and 0.43 (95% confidence interval 0.30, 0.61); respectively (P for trend < 0.0001). Similarly, results from the study highlighted that the multivariate RR for incident gout based on decaffeinated coffee intake grouping of 0, 1 - 237, and ≥ 237 mL /day were 1.00, 1.02, and 0.77 (95% confidence interval 0.63, 0.95) respectively (P for trend is equal to 0.02),

Choi and Curhan, (2007), carried out a study that used linear regression modelling technique to investigate the association between serum uric acid concentration and the consumption of coffee, tea, and caffeine in 14, 758 men and women who participated in the Third National Health and Nutrition Examination Survey (NHANES - III) that was conducted between 1988 and 1994. The study used FFQ to gather information on participant's

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coffee and tea consumption. It classified participants' coffee intake into groups of; 0 cups of coffee per day, < 1 cup of coffee per day, 1 - 3 cups of coffee per day, 4 - 5 cups of coffee per day, and ≥ 6 cups of coffee per day. Serum uric acid concentration was inversely related to coffee consumption. In participants who consumed 4 to 5 cups of coffee per day, serum uric acid was 0.26 mg/ dL (95% confidence interval [95% CI] 0.11, 0.41) lower than in participants who consumed 0 cups of coffee per day. Similarly, participants who consumed ≥ 6 cups of coffee daily had a serum uric acid level that was lower than that of participants who consumed 0 cups of coffee daily by 0.43 mg/ dL (95% CI 0.23, 0.65; P for trend < 0.001).

Effect of Dairy, Protein and Purine-Rich Diet on Gout

As highlighted in Table 5.0 a prospective cohort study investigated the association between the intake of purinerich foods and the incidence of gout in a cohort of 47,150 men who had no gout at baseline. The scientific - quality score for the reviewed study is 7.0 out of 16

Choi et al., (2004), used a semi-quantitative FFQ to gather the cohort's dietary information and then used Cox proportional-hazards modelling technique to calculate the multivariate relative risk of gout in the cohort. There was a positive association between risk of gout and the consumption of either meat or sea food. The multivariate relative risk of gout among participants who consumed meat at a mean daily intake of 2.5 servings per day was 1.41 (95% CI, 1.07 to 1.86; P for trend = 0.02). Similarly, the participants who consumed seafood at a mean daily intake of 2.5 servings per day had a multivariate relative risk of 1.51 (95% CI, 1.17 to 1.95; P for trend = 0.02).

The study also revealed a negative association between dairy intake and the risk of gout. It also shows a reduction of 0.18 in the multivariate relative risk of gout between participants who consumed dairy product at a mean daily intake of 2.5 servings per day and those who consumed dairy product at a mean daily intake of 0.5 servings per day.

Effect of vitamin C Intake on Serum Uric Acid concentration and risk of gout.

One population-based study investigated the association between the intake of vitamin C and serum uric acid concentration. And one prospective study assessed the relationship between vitamin C intake and the risk of gout. (See Table 6.0 for details). The average scientific - quality score for the four reviewed studies is 7.5 out of 16.

Gao et al., 2008, used a population-based study to investigate the relationship between the intake of vitamin C and the concentration of Serum Uric Acid in men.

The study worked with the data of 1,387 participants of the Health Professional Follow - up Study (HPFS) who had no hypertension, had Body Mass Index of less than 30 kg/m^2 and who had participated in a prospective nested case - control study of serum uric acid and hypertension among men.

The Dietary intakes of the 1,387 participants were gathered using a validated semi - quantitative food frequency questionnaire (FFQ). The Harvard University Food Composition Database that was derived from the US Department of Agriculture sources was used to confirm the nutrient composition of the food consumed by participants. And the amount of supplemental vitamin C intake was obtained from participants' response. A negative relationship between greater intake of total vitamin C and serum uric acid concentrations was observed by Gao et al., 2008. The relationship was quite significant when the results for serum uric acid concentrations were adjusted for various parameters like blood pressure, body mass index, smoking, dairy protein, aspirin usages, presence of gout, alcohol, fructose, coffee, sea food and meat. The study went on to report adjusted mean uric acid concentration of 6.4, 6.1, 6.0, 5.7, and 5.7 mg/ dL, (P for trend < 0.001) for respective greater vitamin C intake of <90, 90 - 249, 250 - 499, 500 - 999, and ≥ 1000 mg/ dL.

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Also, the study recorded an inverse association between the intake of vitamin C and the prevalence of hyperuricemia (serum uric acid > 6 mg/ dL). The multivariate ORs for hyperuricemia based on the categories of total vitamin C intake was recorded as follows; 1 (reference), 0.58, 0.57, 0.38, and 0.34 (95% CI: 0.20 - 0.58; P for trend < 0.001).

Finally, the study concluded that vitamin C intake could play a role in the prevention of hyperuricemia and gout.

Choi et al., 2009, used a prospective study to investigate the association between the intake of vitamin C and the risk of gout in 46,994 male participants who had no history of gout at baseline. Every four years within the 20 years period that the study lasted, validated semi - quantitative food - frequency questionnaire was used to gather participants' information on the use of total and supplemental vitamins. The daily rate at which supplemental vitamin C was consumed was categorized into 0, 1 to 399, 400 to 700, 750 to 1250, and 1300 mg or more daily. And the participants' nutrient intake was determined from the frequency at which specified units of food was consumed and the corresponding nutrient content of specified portions from published data. The study went on to confirm 1,317 incident cases of gout. It also revealed that an increase in total vitamin C intake was associated with a decrease in the incidence of gout. For instance, men who took vitamin C at a rate of 500 - 999 mg/day, 1,000 - 1,499 mg/day, and ≥ 1,500 mg/day had a multivariate relative risk (MRR) of gout as 0.83 (95% CI, 0.71 to 0.97), 0.66 (0.52 to 0.86) , and 0.55 (0.38 to 0.80) respectively (P for trend < 0.001) as compared to the RR of gout of 1.0 recorded against men who had less than 250mg of vitamin C per day.

Also, the absolute risk reduction associated with total vitamin C intake in terms of cases per year were presented as 27 cases, 51 cases, and 69 cases per 100,000 person years for total vitamin C intake categories of 500 - 999, 1,000 - 1,499, and ≥ 1500 mg/day respectively.

Similarly, the study also revealed a negative relationship between increase in the intake of supplemental vitamin C and the incidence of gout. A quick comparison between the participants who did not use supplemental vitamin C and those who used it revealed that those who used 1,000 - 1,499 mg/day, and those who used ≥ 1500 mg/day recorded a multivariate RR of gout at 0.66 (0.49 to 0.88) and 0.55 (0.36 to 0.86) respectively (P for trend < 0.001). The study also noted that the negative relationship increase in the intake of supplemental vitamin C and the incidence of gout remained significant when the incidence of gout was adjusted for body mass index, alcohol use, and diary intake.

Finally, Choi et al., 2009, concluded that an inverse association exists between the risk of gout and the intake of either supplemental vitamin C or total vitamin C.

DISCUSSION

This review was carried out to boost the limited findings of the dietary effects on gout. The study exclusively dealt with data/ information from data bases aforementioned in chapter three; which mostly covered cohort studies, prospective studies and cross - sectional studies, of participants with or without gout and how their dietary changes influenced or impacted the effects of gout.

An important finding of this review is that increased intake of fructose or sugar sweetened beverages is associated with the risk of gout (see table 1.0 for details). This risk was found to increase significantly with higher intake levels of sugar sweetened beverages per week. For instance, men who consumed two or more servings of sugar sweetened beverages per day had 85% higher risk of incident gout compared to those who consumed under a serving of sugar sweetened beverages in a month. This relationship was solely independent of other risk factors for gout which includes age, alcohol consumption, body mass index, and hypertension, etc.

The review also identified a substantial positive relationship between the risk of incident gout and the consumption of sugar sweetened beverages. For instance, a 35% increase in the relative risk of gout per serving of sugar sweetened beverages was noticed compared to the 49% increase in the relative risk of gout per

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serving for beer and 15% increase in the relative risk of gout per serving for spirit. This review therefore, puts forward ample evidence that shows that sugar sweetened beverages are a very important risk factor to be considered in the prevention of gout.

In addition, this review work noticed a positive association between the risk of gout and increased consumption of meat. They was a substantial inverse relationship between dairy intake and the incidence of gout. These notable associations were also solely independent of other risk factors for gout which includes age, alcohol consumption, body mass index, and hypertension, etc.

However, some metabolic experiments that investigated the effect of artificial short-term loading of purine on serum uric acid level in both animals and humans presented by (Cliffort et al., 1976) and (Clifford & Story, 1976) could provide theoretical backing to the finding in this work but in spite of their findings, the following constraints hindered the use of their data in public health efforts; Firstly, the lack of information on the specific type and amount of purines in most processed or cooked food (Gibson et. al., 1983). And secondly, the remarkable difference in the bioavailability of various purines found in different foods (Zollner & Griebsch, 1974).

This work reviewed a study that addressed these constraints by investigating the association between common purine rich foods (like meat, seafood, etc.) and the incidence of gout. For instance, this work noticed that a 21% and 7% increase in the risk of gout is associated with each additional daily servings of meat and each additional weekly servings of seafood respectively. Also, no significant relationship was identified between the risk of gout and the intake of purine rich vegetables. These findings therefore provides sufficient data that could be used for public health efforts. For instance, reports by Emmerson, (1996) and Fam, (2000) indicated that gout patience are usually advised to reduce their intake of purine rich meals of both vegetable and animal source. But findings from this review suggests that patients' dietary intake restrictions should be limited to only purine rich foods of animal origin.

Furthermore, this work revealed an inverse association between serum uric acid concentration and vitamin C intake. For instance, approximately 0.6 – 0.7 mg/dL reduction in serum uric acid was noticed in participants who increased their vitamin C intake from less than 90 mg/d to 500 mg/d. This association was solely independent of other risk factors for gout which includes age, alcohol consumption, body mass index, and hypertension, etc. Also this revealed association could be seen as a clinically relevant association in the risk for incident gout as highlighted by Choi, (2014). These findings also correlates with the results from a metabolic experiment that studied the association between serum uric acid level and the short-term loading of high dose of vitamin C presented by Stein, Hassan, and Fox, (1976). The experiment revealed that, the intake of a single dose of 4 g of vitamin C results in the doubling of the fractional excretion of uric acid. Similarly, the daily intake of 8g of Vitamin C for a period of 3 to 7 days results in about 2.0 to 3.1 mg/ dl reduction in serum uric acid as a result of uricosuria.

Again, the consumption of sugar sweetened beverages is associated with the prevalence of gout, thus in managing gout, a reduction in the intake of sugar sweetened beverages would reduce gout prevalence as reported by the studies of Batt et al., in 2013.

CONCLUSION

This thesis has successfully clarified the role of dietary factors in both the risk and the management of gout. The thesis revealed that while the consumption of sugar-sweetened beverages, purine - rich foods (like meat and seafood), and fructose rich fruits increased the risk for gout, the consumption of coffee and dairy products reduced the risk for gout. In view of this findings, it is safe to suggest that dietary risk factors should be considered when gout patients are being managed. Also, health policies and strategies that discourages the excessive consumption of these diets should be implemented. Finally, this thesis is suggesting that future studies could focus on both primary and secondary prevention of gout, as results from such studies could give insight into the possibility of preventing gout.

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