USDA and ODS-NIH Database for the Purine Content of Foods

Release 3.0

Prepared by:

Katherine C. Heydorn[1A], Xianli Wu[1], Timothy J. Garrett[2], Manasi Kamat[2], Deepesh Pandey[1A], Johanna T. Dwyer[3A], Edwina Wambogo[3], Abby G. Ershow[3A*], Stephen P. Juraschek[4], Pamela R. Pehrsson[1*]

[1] Methods and Application of Food Composition Laboratory
Agricultural Research Service
U.S. Department of Agriculture (USDA)

[2] Clinical and Translational Science Institute Southeast Center for Integrated Metabolomics (CTSI-SECIM)
University of Florida (UF)

[3] Office of Dietary Supplements
National Institutes of Health (NIH)

[4] Beth Israel Deaconess Medical Center and Harvard Medical School

* Retired
[A] Contractor

September 2026

U.S. Department of Agriculture
Agricultural Research Service
Beltsville Human Nutrition Research Center
Methods and Application of Food Composition Laboratory (MAFCL)
10300 Baltimore Avenue
Building 307C, BARC – EAST
Beltsville, Maryland 20705 Tel. 301-504-0630
MAFCL web site: https://www.ars.usda.gov/northeast-area/beltsville-md-bhnrc/beltsville-human-nutrition-research-center/methods-and-application-of-food-composition-laboratory/
FoodData Central database web site: https://fdc.nal.usda.gov/

Supported by: National Institutes of Health-Office of Dietary Supplements (NIH-ODS) and the U.S. Department of Agriculture (USDA)

The authors gratefully acknowledge Dr. Pavel Gusev and Ms. Karen Andrews for their contributions to and review of this document. The authors also acknowledge Dr. Katherine Phillips, Virginia Tech, Dr. Leslie Thompson, Texas Tech University, and Dr. Xiaojuan Wang, University of Maryland, for preparation of the USDA samples.

Disclaimer:

The U.S. Department of Agriculture (USDA) prohibits discrimination in all its programs and activities on the basis of race, color, national origin, age, disability, and where applicable, sex, marital status, familial status, parental status, religion, sexual orientation, genetic information, political beliefs, reprisal, or because all or a part of an individual's income is derived from any public assistance program (Not all prohibited bases apply to all programs.). People with disabilities who require alternative means for communication of program information (Braille, large print, audiotape, etc.) should contact USDA's TARGET Center at (202) 720-2600 (voice and TDD). To file a complaint of discrimination, write to USDA, Director, Office of Civil Rights, 1400 Independence Avenue, S.W., Washington, DC 20250-9410 or call (800) 795-3272 (voice), or (202) 720-6382 (TDD). USDA is an equal opportunity provider and employer.

List of Abbreviations:

Abbreviation	Definition
ATP	Adenosine triphosphate
CTSI-SECIM	Clinical and Translational Science Institute Southeast Center for Integrated Metabolomics
DASH	Dietary Approaches to Stop Hypertension
DNA	Deoxyribonucleic acid
DQES	Data Quality Evaluation System
DS	Dietary supplement
DSLD	Dietary Supplement Label Database
FDC	FoodData Central
FF	Foundation Foods
GTP	Guanosine-5'-triphosphate
HILIC-HRMS/MS	Hydrophilic Interaction Liquid Chromatography coupled with Tandem High-Resolution Mass Spectrometry
LOD	Limit of detection
MAFCL	Methods and Application of Food Composition Laboratory
NFNAP	National Food and Nutrient Analysis Program
NHANES	National Health and Nutrition Examination Survey
NIH-ODS	National Institutes of Health Office of Dietary Supplements
QC	Quality control
RNA	Ribonucleic acid
SEM	Standard error of the mean
SSB	Sugar-sweetened beverage
SU	Serum urate
TTU	Texas Tech University
UF	University of Florida
USDA	United States Department of Agriculture
WWEIA	What We Eat in America

Purine Content of Foods (Release 3.0) Table of Contents

A. **Introduction: Purines in Health and Disease 5**

Metabolism and Physiological Function of Purines in Humans 5

Hyperuricemia and Gout 7

B. **Impact of Diet on Urate Levels 8**

Dietary Factors and Urate Levels 8

Eating Patterns and Urate Levels 9

C. **The Need for Purine Data 10**

Estimating Total Intake of Various Purines is Challenging 10

Data are Lacking for Purines in Foods and Dietary Supplements 10

D. **Creation of the USDA Purine Database 11**

Purpose 11

Database Format and Reporting Protocols 12

Literature Purine Data 13

1. Data Sources 13
2. Analytical Laboratory Methodology 14
3. Data Evaluation Methods 14
4. Literature Data Discussion 14

USDA Analytical Purine Data 15

1. USDA Samples: Sources and Sampling 15
2. Analytical Laboratory Methodology and Quality Control 21
3. Data Discussion 22

E. **Database Strengths and Limitations 25**

F. **Summary: Plans for Foods and Dietary Supplements in Future Data Releases 26**

G. **References 27**

A. Introduction: Purines in Health and Disease

Metabolism and Physiological Function of Purines in Humans

Purines are a class of nitrogenous compounds essential to all living organisms. Chemically, purines are heterocyclic aromatic organic compounds featuring a pyrimidine ring fused to an imidazole ring. Adenine, guanine, hypoxanthine, and xanthine are the primary purine bases in plants and animals (**Figure 1**). In foods, purines exist as free bases, nucleosides, nucleotides, and nucleic acids in RNA and DNA, with nucleic acids being the predominant form (Inazawa et al., 2014).

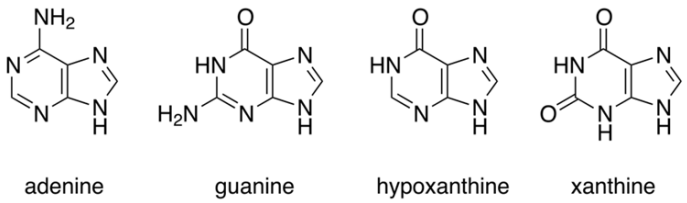


Figure 1. Chemical structures of four major purines

In the human body, purines are synthesized through the *de novo* synthetic pathway from the degradation of nucleic acids in live and dying cells. They are also obtained exogenously by consuming foods, beverages, and dietary supplements (DS) (Nelson & Voruganti, 2021; Ridi & Tallima, 2017). After ingestion, free purines are efficiently absorbed, mainly in the duodenum and jejunum of the small intestine. The metabolic pathways of purines in humans are summarized in **Figure 2.** The various purines are related chemically to each other. They are absorbed from the diet in a similar manner but are metabolized differently (**Figure 2**) (Clifford et al., 1976; Brulé et al., 1992). They then can be utilized through salvage pathways or can be catabolized into uric acid. In humans, uric acid is the final product of both endogenous and exogenous purine metabolism, with approximately 70% excreted by the kidneys and 30% through the intestines.

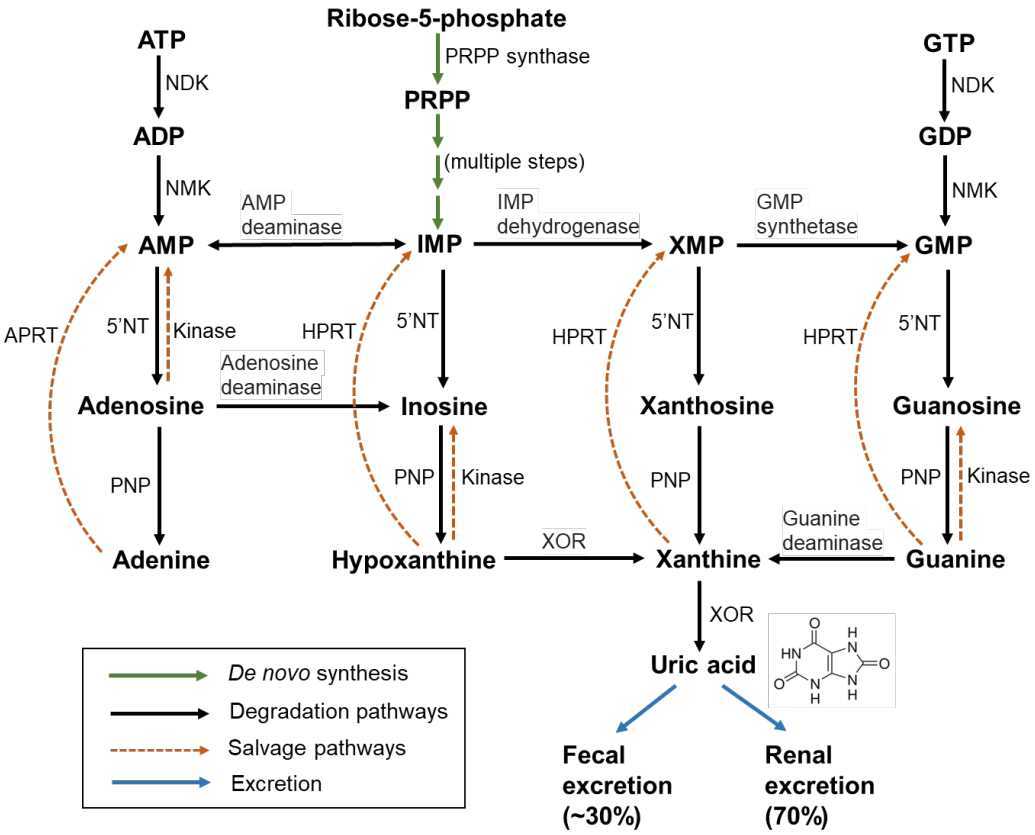


Figure 2. Metabolic pathways of purines and urate production in the human body (Abbreviations: ADP, adenosine diphosphate; AMP, adenosine monophosphate; ATP, adenosine triphosphate; GDP, guanosine diphosphate; GMP, guanosine monophosphate; GTP, guanosine triphosphate; PRPP, phosphoribosyl pyrophosphate; IMP, inosine monophosphate; XMP, xanthosine monophosphate; NDK, nucleoside diphosphate kinase; NMK, nucleoside monophosphate kinase; 5'NT, 5'nucleotidase; APRT, adenine phosphoribosyl transferase; HPRT, hypoxanthine-guanine phosphoribosyl transferase; PNP, purine nucleotide phosphorylase; XOR, xanthine oxidoreductase)

Purines have many important biochemical functions including acting as metabolic signals within and between cells, providing energy, and regulating enzymatic activity (Nelson & Voruganti, 2021). Two purines, adenine and guanine, form nucleotides (such as ATP and GTP) which are components of the nucleic acids DNA and RNA (Kaneko et al., 2020; Nelson & Voruganti, 2021). Purine nucleosides such as adenosine and inosine in the body are key molecules controlling intracellular energy homoeostasis and nucleotide synthesis (Kaneko et al., 2020). Besides acting as chemical messengers, these purines act as endogenous ligands that bind to and activate plasmalemmal purinoceptors, which mediate the extracellular communication that is sometimes referred to as "purinergic signaling" (Huang et al., 2021). Some purines serve specialized roles in the nervous system like guanosine and adenosine, which exert trophic effects following trauma (Rathbone et al., 1999; Jinnah et al., 2013). In addition, uric acid, the end metabolite of purines, is not simply a waste metabolic product, but also serves as an antioxidant for tissue repair, mediator of immune response, and in mitigating against neurological and autoimmune diseases (Nelson & Voruganti, 2021; Ridi & Tallima, 2017).

Hyperuricemia and Gout

Elevated serum urate (SU) levels (> 7 mg/dL in men and > 6 mg/dL in women) indicate that the condition known as hyperuricemia is present. Hyperuricemia is a common disorder that affects people of all ages and genders. Hyperuricemia prevalence rates in the United States are 20.2% in men and 20.0% in women, according to the National Health and Nutrition Examination Survey (NHANES) 2015-2016 (Chen-Xu et al., 2019). Hyperuricemia is caused by many factors, including lowered urate excretion. A causal relationship between hyperuricemia and the pathogenesis of a variety of other health problems has been proposed (Chittoor & Voruganti, 2020). It is associated with a higher risk of gout, hypertension, cardiovascular disease, and kidney disease (Chittoor & Voruganti, 2020).

Gout, a painful form of inflammatory arthritis caused by deposits of monosodium urate crystals in joints, occurs in about 25% of individuals with hyperuricemia. The presence of hyperuricemia and gout are worldwide health issues (Juraschek et al., 2021; Luk & Simkin, 2005). The gout disease burden grew globally from 1990 to 2017 due in part to simultaneous dietary and lifestyle changes, aging populations, and a rise in elevated body mass index. Gout is generally highest in developed regions and countries such as New Zealand, Australia, and the United States (Safiri et al., 2020; Xia et al., 2020). In recent years, Japan has experienced an annual increase in hyperuricemia and gout, possibly due to shifting dietary patterns; hyperuricemia prevalence affected 20-25% of Japanese adult males in 2010 while gout prevalence exceeded 1% in males aged 30 years and older (Hisatome et al., 2020). Recent modeling suggests the global burden of prevalent gout is expected to increase by more than 70% from 2020 to 2050 largely due to population growth and ageing (GBD 2021 Gout Collaborators, 2024). According to NHANES 2015-2016, nearly 4% of U.S. adults (9.2 million people) have gout, including 5.2% of men and 2.7% of women (Chen-Xu et al., 2019). The lower incidence of gout in women may be related to endocrine differences, because estrogen may inhibit the onset of arthritis and promote urate excretion (Hak et al., 2010; Luk & Simkin, 2005). Epidemiologic studies have linked dietary factors with gout risk (Chittoor & Voruganti, 2020; Nuki & Simkin 2006; Zhang et al., 2012). Traditionally, the causes of gout were attributed to an affluent or “western” lifestyle, including red meats, seafoods, and alcohol. Additional causes include obesity, ethnicity, biological sex, and genetics (Kuo et al., 2015; MacFarlane & Kim, 2014; Todd & Wright, 2020).

Accumulating evidence suggests that elevated SU levels may lead to a higher risk of cardiovascular disease, hypertension, dyslipidemia, obesity, metabolic syndrome, type 2 diabetes, and chronic renal disease (Vareldzis, Perez, & Reisin, 2024.). For example, individuals with gout who also experienced a cardiovascular event were more likely to have had a recent gout flare than gout patients who did not have a cardiovascular event, suggesting that hyperuricemia and cardiovascular issues are closely related and that episodes of gout are linked to increased risk for cardiovascular events (Cipolletta et al., 2022). In addition, other potential underlying mechanisms may include regulating molecular signals, such as inflammatory response, oxidative stress, insulin resistance, endoplasmic reticulum stress, and endothelial dysfunction (Yanai et al., 2021).

Conventional treatment options for hyperuricemia as well as gout with varying amounts of evidence include exercise, weight loss if warranted, restriction of alcoholic and sugary beverages containing fructose, anti-inflammatory agents, and urate-lowering medications, as well as avoiding excessive meat and seafood intake (Mallen et al., 2017; Richette & Bardin, 2017).

B. Impact of Diet on Urate Levels

Dietary Factors and Urate Levels

Diet plays an integral role in hyperuricemia and gout via the contribution of dietary purines, leading to increased urate production as shown in observational and intervention studies. Traditional dietary management of hyperuricemia emphasizes reducing the intake of high purine foods such as meats and seafood, as well as limiting the consumption of alcohol and sugar-sweetened beverages (MacFarlane & Kim, 2014; Nuki & Simkin, 2006; Rai et al., 2017). Frequent and high consumption of these foods and beverages are associated with elevated SU levels (Kaneko et al., 2014; Li et al., 2018; Villegas et al., 2012; Zgaga et al., 2012). A higher risk of hyperuricemia was observed with higher intake of red meat, poultry, seafood, and legumes in a large cross-sectional study of Chinese adults independent of sex, age, geographic region, body mass index, hypertension, consumption of refined grains, and alcohol intake (Aihemaitijiang et al., 2020). However, different cuts of meat and different types of seafood vary substantially in their total purine content and purine base composition, highlighting the importance of having analytically determined purine data for specific foods to inform appropriate dietary choices instead of avoiding all meats and all seafood.

High purine containing vegetables have not been associated with the risk of hyperuricemia or gout (Choi et al., 2004; Li et al., 2018; Villegas et al., 2012; Zgaga et al., 2012, Jakse et al., 2019). However, data on the purine content of various types of vegetables, such as different Brassicas (e.g., broccoli and cauliflower), are inconsistent and may lead to unnecessary dietary restrictions. Since various mechanisms affect hyperuricemia, restricting all purine-rich foods would unnecessarily eliminate certain foods that confer other health benefits. Therefore, these studies highlight the need for an accurate understanding of which food products, including DS, are high in purine content and increase the risk of hyperuricemia and gout. In addition, published purine values for food items can vary considerably across studies, potentially reflecting differences in food varieties, preparation method(s), analytical methodology, and other factors. Analytically determined values for foods and supplements representative of U.S. food supply are therefore needed to improve estimates of dietary purine intake.

Dietary factors besides total purine intake can impact urate levels, such as levels of specific uricogenic purine bases in foods. Uricogenic bases are purine bases that increase serum and urinary urate concentrations (Sarwar & Brulé, 1991). Hypoxanthine has shown the greatest dietary impact on increasing gout risk due to its effect on urate levels in the body (Clifford et al., 1976). Adenine also seems to increase urate levels (Clifford et al., 1976; Sarwar & Brulé, 1991; Brulé et al., 1992). The ratio of the four major purine bases (adenine, guanine, hypoxanthine, and xanthine) relative to total purines in a food seems influential in affecting SU, especially the impact of hypoxanthine (Kaneko et al., 2014). Therefore, reporting individual purine bases in addition to total purine contents may provide a more complete characterization of the purine composition of foods and their potential contribution to urate production.

Consuming dairy foods containing milk may decrease the risk of gout and hyperuricemia due to inverse associations with serum urate (Choi et al., 2004; Li et al., 2018; Zgaga et al., 2012). In one large observational study, low-fat dairy products were inversely associated with the incidence of gout (Choi et al., 2004). The protective effects of dairy have been attributed to the milk proteins casein and lactalbumin (Choi et al., 2005) and to the dairy fractions glycomacropeptide and G600 milk fat extract (Dalbeth et al., 2012). Soy food intake is negatively associated with hyperuricemia and gout risks (Villegas et al., 2012;

Li et al., 2018). High levels of meat and seafood consumption in the diet, but not total protein intake, have been linked to higher serum urate levels; in contrast, dairy intake has been inversely associated with serum urate levels (Choi et al., 2005).

Sugar-sweetened beverages (SSBs), especially those high in fructose, have a well-documented biochemical and epidemiologic link to hyperuricemia. The relationship is driven by fructose metabolism, which rapidly consumes cellular ATP and generates uric acid (Luk & Simkin, 2005; Nelson & Voruganti, 2021). SSBs also appear to compete with urate excretion, which further elevates SU (Maiuolo et al., 2016; Chittoor & Voruganti, 2020). A recent meta-analysis of 22 studies spanning from 2008 – 2024 concluded that SSB consumption was significantly associated with increased risks of hyperuricemia and gout, particularly among adult men (Lu et al., 2025). However, most patients' exposure to SSBs were self-reported or relied on food frequency questionnaires. Clinical research is necessary to quantify potential associations between SSB consumption and increases in serum urate.

Alcohol consumption contributes meaningfully to elevated SU and hyperuricemia through multiple biochemical pathways (Pillinger & Keenan, 2008). The purine content of alcoholic beverages is only one of several factors involved in inducing changes to SU. Alcohol metabolism itself also plays an important role. Ethanol metabolism accelerates adenine nucleotide degradation, increasing urate production. Concurrently, ethanol promotes lactic acidosis and, in heavier use, ketoacid formation, both of which competitively inhibit renal urate excretion (Yamamoto et al., 2005; Todd & Wright, 2020). Epidemiologic studies consistently demonstrate a dose-dependent increase in hyperuricemia and gout risk, with men showing greater susceptibility (Nieradko-Iwanicka, 2022). Gout patients are typically advised to limit alcohol to one drink per day for women or two drinks per day for men (Khanna et al., 2012).

High doses of vitamin C (i.e., 400-500 mg/day) have beneficial effects on uric acid levels such as increased urinary urate excretion (Azzeh et al., 2017; Berger et al., 1977; Stein et al., 1976) and reduced SU concentration (Huang et al., 2005; Juraschek et al., 2011). In addition, vitamin C is highly associated with a reduced risk of gout (Choi et al., 2009). Vitamin C supplementation at 500 mg/day reduced SU and the incidence of new or recurrent gout diagnoses in middle-aged male physicians (Juraschek et al., 2022).

Weight loss is often recommended to gout patients since a lower body mass index has been associated with a lower risk of gout (Choi et al., 2005). However, reducing subjects' body mass indices led to either higher urate levels or no effects in a randomized clinical trial; more studies are needed (Hu et al., 2021).

The effects of diet on urate levels differ by race, sex, body weight, and by the presence of certain genetic polymorphisms. For example, legume intake increased serum urate among African Americans compared to White adults, while alcohol intake increased serum urate to the same extent among African Americans and White adults in the United States (Beydoun et al., 2018). Dairy intake had an inverse effect on serum urate among African Americans but not White women (Beydoun et al., 2018). a cross-sectional analysis using NHANES 2011-2018 found that gout prevalence was twice as common among Asian adults in the United States than in any other race/ethnic groups (Yokose et al., 2023). The higher prevalence of gout in Asian populations may also be influenced by genetic polymorphisms (Butler et al., 2021).

Eating Patterns and Urate Levels

The Dietary Approaches to Stop Hypertension (DASH) diet, which focuses on fruit, vegetables, and low-fat dairy products, is being studied as a dietary intervention pattern for gout. Since nutrients and foods are

consumed as part of a total diet, research on the effect of eating patterns has presented an opportunity to examine the combinations of foods and micronutrients on hyperuricemia and gout risk (Rai et al., 2017).

The DASH diet is associated with lower gout risk among men (Rai et al., 2017) and lowered SU (Juraschek et al., 2016; Rai et al., 2017; Belanger et al., 2021). In a recent study, gout patients were instructed to follow the DASH diet along with other guidelines such as avoiding red meat, organ meat, shellfish, and excess alcohol consumption (Juraschek et al., 2021), presumably because those foods and beverages are traditionally considered high dietary sources of purines. In these and other scenarios, data for the purine content of specific foods can be beneficial for making food choices and optimizing established healthy dietary patterns for adults at risk for gout.

C. The Need for Purine Data

Estimating Total Intake of Various Purines is Challenging

There have been few, if any, well-controlled trials documenting the relationship between the consumption of dietary purines and serum urate in healthy individuals or in those with hyperuricemia or diagnosed gout. One of the major hurdles in conducting such intervention studies and other types of clinical trials is that the exact amounts of individual purines in most foods, and especially in cooked foods, are unknown (Gibson et al., 1983; Choi et al., 2004). Additionally, the bioavailability of different purines varies considerably in specific foods, so total purine content alone is insufficient - it is also important to know what specific purines are present. For example, studies which supplemented subjects' diets with RNA or DNA showed that RNA influenced urate concentration more than an equivalent amount of DNA (Zollner & Griebsch, 1974). Other dietary studies have found that oral doses of adenine and hypoxanthine increased subjects' SU levels while guanine and xanthine had no effect (Clifford et al., 1976). Overall, very few intervention trials have been conducted in recent years to examine how individuals with hyperuricemia respond to dietary changes.

We are not aware of research conducted to establish dietary guidance on intakes of various purines. While recommended intake levels for purines have been established internationally, such as Japanese recommendations limiting purine intake to 400 mg/day to prevent hyperuricemia and gout (Kaneko et al., 2020), current U.S. dietary guidance does not provide recommended intake levels of individual or total purines for healthy adults or patients with hyperuricemia. The American College of Rheumatology rated the certainty of evidence as "low" between limiting purine intake and gout management in its recent review (FitzGerald et al., 2020), but it has been noted that there was a dose-response relationship between higher consumption of purines and risk for gout flares (acute episodes of joint pain and swelling) (Zhang et al., 2012). These gaps highlight the need for more research to determine potential diet-disease relationships, as well as the need for research and clinical resources such as databases that can improve estimation of total and individual purine intake.

Data are Lacking for Purines in Foods and Dietary Supplements

There is very little high-quality information available on the purine content of commonly consumed U.S. foods. Systematic searches for sources of purine food composition data for dietary guidance to assist patients or clinicians reveal that data are rarely or poorly documented, may rely on older analytical data, and often lack information on individual purine bases. Websites focusing on gout treatment often provide only limited total purine values and do not give data on which purine bases are present in foods, nor do

they cite their data sources. A table of purine values is provided in the seminal food composition reference (Pennington & Spungen, 2012), but it is not in the public domain, there is a fee for accessing data, and the data are outdated and limited to that available over a decade ago.

Some dietary guidance sources group foods into categories (low, medium, high, very high) using a nominal scale or an ordinal scale (numbering 1 through 4) to guide patients on which foods are high in purine content and should be restricted in frequency and amounts consumed. Such categorizations should be based on reliable analytically determined values generated using consistent sampling and assay methodology and should be relevant to the food supply of the patient's country of residence if they are to provide actionable and effective guidance. Many other patient-facing materials on health websites (e.g., Cleveland Clinic or WebMD) and patient advocacy groups (e.g., the Arthritis Foundation) provide similar recommendations for restricting purines, but they often focus on rarely or uncommonly consumed foods (e.g., game and organ meats) and provide limited quantitative information on purine concentrations in commonly consumed U.S. foods.

Also, no North American-based studies have published analytical data on the purine content of commonly sold DS available for U.S. consumers. However, concentrated amounts of purines have been found in some Japanese DS (Kaneko et al., 2014), suggesting that DS potentially could represent a substantial source of purines intake. Some DS on the U.S. market, such as products promoted for increasing energy or losing weight, are labeled as containing purine nucleotides or related compounds (e.g., adenosine, ATP, GTP) or purine-rich constituents (e.g., yeast extracts). Many other DS may have purines as an undisclosed significant component but are not required by law to specifically list purines in the Supplements Facts panels. These products may therefore represent an unrecognized source of dietary purines, particularly for individuals advised to limit their purine intake.

The absence of a centralized and consistently structured database poses challenges for estimating dietary purine exposure and for developing evidence-based dietary guidance, particularly for at-risk groups such as patients already suffering from hyperuricemia and gout who may rely on poorly documented guidance and the spread of anecdotal home remedies. Data for the purine content of foods and DS are limited and needed to strengthen the evidence base between purine intakes in health and disease outcome, as well as to address other gaps in understanding their physiological relevance (Choi et al., 2004).

D. Creation of the USDA Purine Database

Purpose

The "USDA and ODS-NIH Database for the Purine Content of Foods" is designed to function as an essential source of publicly available dietary purine data for researchers, health professionals, and others striving to monitor purine consumption (USDA & NIH ODS, 2021). The purine database was developed as a collaborative effort among scientists at USDA's Methods and Application of Food Composition Laboratory (MAFCL) in Beltsville, MD, the Office of Dietary Supplements National Institutes of Health (ODS-NIH) in Bethesda, MD, the University of Florida Clinical and Translational Science Institute Southeast Center for Integrated Metabolomics (UF CTSI-SECIM) in Gainesville, FL, the Beth Israel Deaconess Medical Center in Boston, MA, and the Department of Animal and Food Sciences at Texas Tech University in Lubbock, TX.

The first USDA and ODS-NIH purine database release (Release 1.0) was published on the USDA MAFCL website in February 2023. Release 1.0 of the database provided existing data for purine bases in foods (396 foods and 15 alcoholic beverages) sourced from mostly non-U.S. validated literature with a usable format.

Release 2.0 of the database, which was published in March 2025, contained analytical data for 61 foods and 14 purine-containing DS samples collected from the U.S. market. All validated literature data published in Release 1.0 was also included to allow for comparisons among foods. Foods and DS selected for analysis were based on comprehensive and representative sampling plans detailed below which were designed to address data gaps observed after the publication of Release 1.0.

Release 3.0 of the includes new analytical values for 43 commonly consumed foods and 14 purine-containing DS products collected from the U.S. market. In addition, a lot-to-lot comparison study was conducted on 5 DS previously analyzed in Release 2.0 to evaluate differences in the purine content of select supplements between lots. The aims of determining purine contents in these samples were as follows: a) to generate new analytical data for the purine database for commonly consumed U.S. foods using validated methodology based on nationally representative intake patterns, b) to elucidate the purine content of insufficiently characterized food categories (e.g., cereal grains, fruits, nuts and seeds), c) to ascertain the purine content of the top consumed seafood and organ meat products sold in the U.S., d) to observe possible changes in the content or base form of purines and uric acid after cooking, and e) to determine the analytical content of purine-containing DS from a subset of commonly consumed supplement products.

Database Format and Reporting Protocols

The Purine Database Release 3.0 is presented as an Excel data file, which contains a total of 7 tabs. Tabs are separated into North American food data (United States and Canada), international food data, alcohol data, DS per serving unit (per individual unit such as one tablet, one capsule or a specific weight), DS per serving (e.g., a recommended intake such as two tablets or three capsules), DS per daily serving size (based on recommended daily intake serving values as provided on labels), and literature-derived purine content. Data were grouped into typical U.S. food categories for presentation in the database (e.g., beef organ meats, legumes, vegetables). USDA's analytical data are presented in bold text along with the values from literature that were reviewed to enable interpretation and for planning future work. Sample size (n), the four major purine bases (adenine, guanine, hypoxanthine, xanthine), and total purines (sum of four individual purines) are all reported. Uric acid was also disclosed due to measurable amounts found in some foods. Footnotes are given where descriptions or explanations of specific foods are needed.

For foods, means, standard error of the mean (SEM), minimum (Min), and maximum (Max) values were reported as milligrams per 100 grams for solid foods and milligrams per 100 milliliters for liquids. The data are presented based on a raw fresh weight basis or on a cooked weight basis for cooked foods. When applicable, other reporting units from literature were converted into these units for consistency in observing data across studies. Data cells with a dash ("- ") indicate data that was not calculable or measured, whereas values listed as "ND" (not detected) in literature were reported when purine measurements were below the studies limit of detection.

More samples were procured for four seafood items previously analyzed in Release 2.0, including raw catfish, raw Atlantic salmon, and raw, mixed species shrimp. New data were averaged with the corresponding values in Release 2.0 to provide updated estimates of purine content. For "Cod, Atlantic, wild, raw" in Release 2.0, the food name was revised to "Cod, mixed species, wild, raw" after combining datasets due to minimal purine differences among Atlantic and Pacific cod species.

Dietary supplements are presented as 6 categories (brewer's yeast, chlorella, chlorella and spirulina, RNA/DNA (nucleotide complex), royal jelly, and spirulina) and are grouped under their respective dosage forms (capsule, liquid, powder, tablet) to best represent the purine content associated with ingestion of each form. Purine contents of DS were reported as milligrams per serving unit, as milligrams per serving, and as milligrams per recommended daily serving as listed on their respective Supplements Facts panels.

Literature Purine Data

1. Data Sources

For Release 1.0 published in February 2023, literature data for four purine bases (adenine, guanine, hypoxanthine, and xanthine) in foods and alcoholic beverages were compiled. Food listings from literature were reported in fourteen studies conducted in various countries. Data came primarily from Japan; less than 20% of the food items in the database were from North American sources (United States and Canada).

Data from the scientific literature were re-examined in 2024 to obtain an accurate number of foods and to determine which studies, if any, measured uric acid in food; only one of the fourteen studies contained this information. Six foods from Kaneko et al. (2014) were separated into two different food descriptions based on an incorrect grouping of data. The food descriptions modified in Release 2.0 were:

- Beef brisket (split into beef brisket and beef shin)
- Red king crab (split into red king crab and snow crab)
- Bean sprouts (split into "with" and "without bean" descriptors)
- Mustard spinach (komatsuna) (split into "baby" and "mature" varieties)
- Spinach (split into "baby" and "mature" varieties)
- Removal of the food longcod (split into arabesque greenling and fat greenling)

Footnotes were also added to three foods from Kaneko et al. (2014) (white rice, asparagus, and bamboo shoots) to indicate when USDA combined data for the same food due to similar purine measurements and processing conditions.

Tab 1 displays 75 foods from North American sources to facilitate inspection of data for foods analyzed domestically. *Tab 2* reports 327 foods from other countries which provide a broadened perspective of available published data. *Tab 3* includes 13 alcoholic beverages from Japan, Austria, Hungary, Romania, and Poland. Food data from countries beyond North America can be applicable in the U.S. due to availability in Asian or other specialty markets. However, differences among plant species, growing conditions, storage, laboratory methods, and other factors can impact reported purine values and should be considered when referencing data from various sources.

The studies were published between 1976 and 2020, with nearly half of them occurring prior to 1989. Other studies were found but were not included because they reported total purine values but not values for individual bases or disseminated their data only in the German or Japanese languages.

2. Analytical Laboratory Methodology

Analytical laboratory methods for measuring purine content in the foods and beverages in the database are listed with their respective data sources in Tab 7. High-performance liquid chromatography (HPLC), which has been proven to be effective for many years in measuring purine content of foods, was used in all the published studies except for one, which used capillary zone electrophoresis for analysis of beers.

3. Data Evaluation Methods

The purine literature data were evaluated for quality using procedures developed by scientists at USDA's MAFCL (formerly the Nutrient Data Laboratory) (Bhagwat et al., 2009). Scientists used a modified version of the USDA Data Quality Evaluation System (DQES) to assign a numeric score to each data source (Holden et al., 2002, 2005). Five categories of documentation were initially evaluated: sampling plan, sample handling, number of samples, analytical method, and analytical quality control. However, scoring of the analytical method could not be accomplished due to lack of standardized evaluation criteria. Notable characteristics of the studies reporting data used in this purine database (except for additional data published in 2020) were previously reported (Wu et al., 2019).

4. Literature Data Discussion

As observed in Tabs 1 and 2, purine content varied among foods, and it also varied for the same food in different studies. For example, total purine in raw beef cuts in the database ranged from 77 to 123 mg/100 g (chuck ribs and round, respectively), while liver had up to 220 mg/100 g. Among canned seafood items in the database, clams had 62 mg/100 g, while anchovies had 321 mg/100 g. Furthermore, the overall category of finfish and shellfish ranged from 7.7 to 1,400 mg total purines/100 g.

Dark and traditional-type beers had higher adenine and hypoxanthine values than other types of beers and other alcoholic beverages reported, although the highest total purine value among alcoholic beverages was only 13.5 mg/100 g (or about 46 mg/12 ounce serving) (*Tab 3*).

Uric acid was evaluated in vegetarian dishes, soy protein concentrate, and poultry (*Tab 2*). Negligible uric acid was observed in most vegetarian dishes and the soy protein concentrates. Raw chicken liver contained 51 mg uric acid/100 g; this value corroborates prior scientific consensus that purine metabolism occurs in the liver prior to excretion via the intestines and renal system.

Variations in purine content may be due to natural differences in foods (such as vegetable cultivars or animal breeds), different sampling plans, sample handling, cooking, laboratory methods, and other factors. Overall, purine values are generally highest in animal-based products (especially organ meats) and lowest in dairy, eggs, grains, fruits, and most vegetables. Moreover, in most cases products from animal sources (other than dairy and eggs) contain higher amounts of uricogenic bases hypoxanthine and adenine than other food groups. The food category with the highest mean hypoxanthine values was the "soups and sauces" group which was mostly influenced by the dehydration process and reporting of certain sauces as mixes, not as consumed. Thus, the user of the data would need to account for the dilution factor of water to correct to "as consumed." Mean hypoxanthine levels were lowest in plant-

based foods, dairy, eggs, and sweets. Mean values for adenine were highest in organ meat products (chicken and beef liver, pork kidney), dried yeast, and cod milt (i.e., the cod's sperm filled gland) and were lowest in dairy and eggs, fruits, and sweets.

USDA Analytical Purine Data

1. USDA Samples: Sources and Sampling

Food Samples

USDA food samples came from several sources and included both archived and recently acquired material. Recently procured samples were preferred whenever possible to provide data that best represented the current U.S. food supply. Therefore, most USDA samples for purine analysis were acquired in conjunction with the FoodData Central (FDC) Foundation Foods (FF) program, a new food composition database designed to provide expanded metadata on a diverse number of raw foods and ingredients (USDA ARS, 2024a). Foods from this program are primarily convenience-sampled with the goal of collecting a diverse, representative number of samples from manufacturers and distributors. Foods were obtained at large grocery stores throughout Maryland and Virginia from January 2023 – April 2025. All samples were processed by the food composition laboratory at Virginia Tech in Blacksburg, VA.

A few of the foods selected to address data gaps were from the National Food and Nutrient Analysis Program (NFNAP) (Haytowitz and Pehrsson, 2018) archived samples. They were steamed lobster, steamed oysters, and prepared blue crab (collected and processed in May 2009), cooked chickpeas (July 2010), canned tuna (January 2017), and 2% milk (April 2018).

To determine the purine content of animal-based products and the effects of cooking, 13 types of meat were selected based on 2017-2018 consumption data published in 2020 from the What We Eat in America (WWEIA) database, the dietary intake interview portion of the national food survey NHANES (USDA ARS, 2024b). Meat samples were purchased with unique lot numbers from different manufacturers in Lubbock, TX supermarkets in June through October 2023 through collaboration with Texas Tech University (TTU). Meats were stored in a food-grade refrigerator (4 °C) after purchase. To prepare analytical samples, meats were chopped into pieces, placed in a strainer, and submerged into liquid nitrogen until solidly frozen. The frozen samples were subsequently homogenized with a blender, transferred into double-sealed plastic bags (Ziploc brand), and were frozen at -20 °C or colder until ready to aliquot. All samples were processed prior to expiration dates listed on the packages. When applicable, selected meat samples were cooked at TTU based on a raw-to-cooked paired meat sampling plan (**Table 1**). Subsamples of raw or unprepared (for processed foods) material were collected from each meat cut before cooking to allow for a direct comparison of purine content. Processed meats were prepared for analysis according to label directions (e.g., boiling). Raw meats were cooked without added ingredients such as oils, spices, and/or butter. Conventional recipes that most closely emulated American consumers' cooking methods for each meat were used.

In 2025, USDA identified database gaps in the organ meat and lamb and game food categories necessitating new analytical data. Therefore, purine contents in selected game and organ meats were analyzed before and after cooking. A total of 3 organ meats (beef liver, chicken gizzards, pork liver), and lamb chops were procured by TTU in August 2025. Samples were purchased from supermarkets in Lubbock, TX, as described above, except for pork liver. Commercial-grade pork liver was obtained from

"Raider Red Meats," an on-campus store managed by TTU that contains meat products from animals raised and slaughtered by the Gordon W. Davis Meat Science Laboratory. Due to sample limitations, only one sample was procured, and the sample was not cooked. All meat samples were homogenized, processed and cooked at TTU based on a raw-to-cooked paired meat sampling plan (**Table 1**).

USDA also completed a study to quantify the purine content of commonly consumed seafood products and to evaluate the effects of cooking. Six types of seafood were chosen based on WWEIA data as described above. Seafoods were purchased with unique lot numbers from different manufacturers in Lubbock, TX supermarkets from April – August 2025 through collaboration with TTU. Samples were stored, homogenized, and processed as described previously (**Table 2**). When applicable, skin was removed from fish fillets prior to homogenization and disposed of, except for Atlantic salmon. For this food, slight modification of the protocol was adopted to assess whether purine content differed between skin and flesh. Therefore, four sets of samples were prepared for Atlantic salmon per biological sample: raw salmon flesh, raw salmon skin, cooked salmon flesh, and cooked salmon skin. The finfish samples and salmon skin were cooked using the cooking protocol described in **Table 2**. After cooking, samples were immediately submerged into liquid nitrogen until completely frozen and prepared for homogenization. Raw shrimp were de-shelled, and tails and heads were removed prior to homogenization. Shrimp were cooked based on the method described in **Table 2**. After cooking, shrimp were de-shelled, heads and tails were removed, and the samples were immediately submerged into liquid nitrogen until frozen for homogenization.

Typically, at least 6 to 8 individual samples per food to were obtained to provide a representative sample size. However, for some foods, a smaller sample size was deemed adequate due to their low observed variability, material limitations, or the exclusion of outlier data. Blind duplicates and replicates were included for most foods.

In some cases, literature data was used to identify foods critical in U.S. diets warranting additional analysis due to low (n) or lower than expected purine content. For example, only one data point was observed in literature for chicken eggs, which reportedly did not contain purines (Kaneko et al., 2014). Therefore, a total of 6 raw chicken eggs were convenience sampled from supermarkets (n = 3) and a local farm (n = 3) in Alachua, FL, in September 2024. Each set of eggs were boiled in liquid chromatography (LC) grade water for approximately 8-10 minutes until fully cooked. Samples were subsequently peeled, and the egg whites and yolks were separated. The egg whites were chopped finely with a knife, while the yolks were crushed with a fork. Similarly, two foods (plain, whole grain, uncooked oats and raw asparagus) were evaluated in Release 3.0 to address data discrepancies in published literature. Samples were purchased in March 2026 from Maryland supermarkets.

Table 1. Meat and offal (organ meat) cooking methodology at Texas Tech University and moisture losses after cooking.

Meat	Cooking method[1]	Cooking condition	Weight loss (%) (Mean ± SD)
Release 2.0			
Bacon	Pan-fried	Cooked in griddle at 400°F (204.4°C), flipped every 3-4 minutes until internal temperature reached 145°F (62.8°C)	70.06 ± 2.42
Beef Chuck Roast	Braised	Braised in Dutch oven at 250°F (121.1°C) with deionized water until internal temperature reached 185°F (85.0°C)	35.04 ± 4.50
Chicken Breast	Roasted	Cooked in oven to 375°F (190.6°C) until internal temperature reached 165°F (73.8°C)	25.29 ± 3.22
Frankfurter (beef)	Boiled	Boiled in deionized water for 4-5 minutes	7.52 ± 1.35
Frankfurter (mixed meat)	Boiled	Boiled in deionized water for 4-5 minutes	4.36 ± 1.95
Ground Beef (73% lean)	Pan-fried	Cooked in skillet at 400°F (204.4°C) until internal temperature reached 165°F (73.8°C)	49.59 ± 0.93
Ground Beef (93% lean)	Pan-fried	Cooked in skillet at 400°F (204.4°C) until internal temperature reached 165°F (73.8°C)	35.08 ± 4.90
Pork Loin	Pan-fried	Cooked in griddle at 400°F (204.4°C), flipped every 5-6 minutes until internal temperature reached 165°F (73.8°C)	36.80 ± 4.24
Pork Sausage	Pan-fried	Cooked in skillet at 375°F (190.6°C) for 2 minutes and 15 seconds on each side until internal temperature reached 155°F (68.3°C)	29.34 ± 5.76
Release 3.0			
Beef liver	Pan-fried	Cooked in skillet at 400°F (204.4°C) until internal temperature reached 165°F (73.8°C)	20.73 ± 4.85
Chicken Gizzards	Instant Pot	Pressure cooked in an Instant Pot at 400°F (204.4°C) for 45 – 60 minutes until tender and internal temperature reached 165°F (73.8°C)	26.30 ± 1.78
Lamb Chops	Pan-fried	Cooked in skillet at 400°F (204.4°C) until internal temperature reached 165°F (73.8°C)	30.48 ± 5.31

[1] Meats were prepared without added oils, spices, butter, or other added ingredients

Table 2. Seafood cooking methodology at Texas Tech University and moisture losses after cooking.

Seafood	Cooking method[1]	Cooking condition	Weight loss (%) (Mean ± SD)
Release 3.0			
Atlantic Salmon	Roasted	Cooked in oven at 425°F (218.3°C) for 10 minutes on each side until internal temperature reached 145°F (63°C)	20.60 ± 3.39
Atlantic Salmon, skin only	Roasted	Cooked in oven at 425°F (218.3°C) for 10 minutes on each side until internal temperature reached 145°F (63°C)	40.68 ± 10.28
Catfish	Roasted	Cooked in oven at 425°F (218.3°C) for 10 minutes on each side until internal temperature reached 145°F (63°C)	30.78 ± 3.48
Cod	Roasted	Cooked in oven at 425°F (218.3°C) for 10 minutes on each side until internal temperature reached 145°F (63°C)	31.81 ± 0.49
Shrimp	Boiled	Boiled in deionized water for 3-4 minutes	29.50 ± 4.95
Tilapia	Roasted	Cooked in oven at 425°F (218.3°C) for 10 minutes on each side until internal temperature reached 145°F (63°C)	23.31 ± 12.03
Yellowfin tuna	Roasted	Cooked in oven at 425°F (218.3°C) for 10 minutes on each side until internal temperature reached 145°F (63°C)	20.72 ± 4.61

[1] Seafoods were prepared without added oils, spices, butter, or other added ingredients

Dietary Supplement (DS) Samples

A sampling plan was developed by USDA in 2024 to identify and procure DS with the goal of obtaining analytical purine content from a subset of commonly consumed supplements. The sampling plan was based on a thorough evaluation of published literature, U.S. market data from online web searches using the ODS Dietary Supplement Label Database (DSLD) and retail vendors, analytical data from Kaneko et al. (2014), and an intake analysis on a representative sample in the U.S. population using the US National Health and Nutrition Examination Survey (NHANES) data spanning 2017–2020. Five DS categories containing "very high" levels of purines (>300 mg/100 g) were chosen: brewer's yeast, chlorella, royal jelly, RNA/DNA (nucleotide complex), and spirulina. Searches of NHANES DS data files with these key terms led to the identification of 38 potentially high-purine products available in the U.S. market.

A USDA in-house, ranked scoring system was used to prioritize the most popular DS available for purchase by U.S. consumers (**Table 3**). A score of "2" was given to products found in NHANES due to their use by American consumers, and "1" to products found in DSLD and online retail websites including Amazon, Walmart, and others. A variety of online retail platforms were researched /surveyed to ensure that a diverse range of purine-rich DS were represented. Ultimately, 14 representative DS samples characterized by 3 dosage forms (tablet, capsule, and powder) were selected for inclusion in the Purine Database Release 2.0.

In 2025, the DS sampling plan was expanded to include 14 new products representing the top purine-containing DS on the U.S. market. Web-scraped data was collected from the online retailers Amazon and Walmart by a third-party, Abt Associates, over a period of three months (January – March 2025). Eight keywords were used to search for products: "beer yeast", "brewer's yeast", "spirulina", "chlorella", "royal jelly", "DNA", "RNA," and "nucleotide". Supplements were selected based on the 1) total number of units sold (sales volume), (2) number of consumer ratings over the products' lifetime, (3) search result ranking, (4) Amazon "best seller" status, and (5) Amazon sponsorship status (i.e., the presence of paid advertisements when browsing products). Duplicate search results were excluded from each category, and DS were excluded based on the following criteria:

1. Zero sales in past 3 months
2. Items unrelated to the DS search category, such as products not intended for ingestion by humans
3. Products without Supplement or Nutrition Facts labels
4. Product blends that did not report the exact amount of the search ingredient on the label
5. DS already analyzed for purine content in Release 2.0

After a preliminary examination of data, results were grouped into three categories based on similar search terms: "Beer yeast and brewer's yeast," "DNA, RNA and nucleotide," and "Chlorella and Spirulina." Products were sorted based on monthly average units sold (largest to smallest), and the top products in each category were chosen for purine analysis. A new DS category, "Chlorella and Spirulina," was identified based on several top-consumed product containing equal formulations of these ingredients. DS identified from the web scraping were subsequently evaluated under USDA's comprehensive DS sampling plan as detailed above (**Table 3**). In sum, 14 representative DS samples characterized by 4 dosage forms (capsule, liquid, powder, and tablet) were selected for inclusion in the Purine Database Release 3.0, bringing the total number of DS in the purine database to N = 28.

Evaluation of lot-to-lot variability was also performed on 5 previously analyzed DS products in Release 2.0 to assess differences in purine content between different lots or caused by reformulation. These 5 DS were selected based on product category, USDA score, and availability on online U.S. market retail websites (**Table 3**). All supplements were reviewed by USDA to ensure lot numbers were unique and that the main ingredients and dosages were consistent across batches prior to purine analysis.

Supplements were procured by USDA and UF CTSI-SECIM from online retailers including Amazon, iHerb, and manufacturer websites. For each sample, at least 200 pills of the same lot with an expiration date of at least one year were purchased.

Table 3. List of dietary supplements (DS) identified for purine analysis using USDA's US market-based sampling plan and web scraping data.

Product Name (dose)	Dosage Form	Brand Code	NHANES	DSLD	Online Retail Vendor							Avg. Monthly Amazon Sales[α]	USDA score
					Amazon	iHerb	Walmart	Pureformulas	Vitamin Shoppe	GNC	Other		
Release 2.0													
Brewer's Yeast, 650 mg	Tablet	A	0	1	1	1	1	1	0	-	0	-	5
Brewer's Yeast, 500 mg	Tablet	B	0	1	1	1	1	0	0	-	0	-	4
Brewer's Yeast, 500 mg	Tablet	C	0	1	1	1	1	0	0	-	0	-	4
Brewer's Yeast, 1000 m	Capsule	J	0	0	1	0	1	0	0	-	1	-	3
Chlorella, 200 mg	Tablet	D	2	1	1	1	1	1	1	-	0	-	8
Chlorella, 1000 mg	Tablet	A	0	1	1	1	1	1	0	-	0	-	5
RNA/DNA (nucleotide complex), 500mg	Capsule	F	0	1	1	1	1	1	0	-	0	-	5
Royal Jelly, 1500 mg	Capsule	A	0	1	1	1	1	1	0	-	0	-	5
Ginseng & Royal Jelly, 100 mg	Capsule	A	0	1	0	1	1	1	0	-	0	-	4
Spirulina, Blend	Powder	E	2	1	0	0	0	0	0	-	1	-	4
Spirulina, 1000 mg	Tablet	G	2	1	1	1	1	1	1	-	1	-	9
Spirulina, 500 mg	Tablet	H	2	1	1	1	1	0	0	-	0	-	6
Spirulina, 500 mg	Tablet	I	0	1	1	1	1	0	0	-	0	-	4
Spirulina, 500 mg	Tablet	A	2	1	1	1	1	1	0	-	0	-	7
Release 3.0													
Brewer's Yeast, 15000 mg	Powder	K	0	0	1	0	0	-	-	0	1	5667	2
Brewer's Yeast, 16000 mg	Powder	A	0	0	1	1	1	-	-	1	1	1333	5
Chlorella and Spirulina, 3000 mg	Tablet	L	0	0	1	1	1	-	-	0	0	4000	3
Chlorella and Spirulina, 1000 mg	Tablet	M	0	0	1	0	0	-	-	0	0	3000	1
Chlorella and Spirulina, 600 mg	Capsule	N	0	0	1	0	1	-	-	0	0	6000	2
Chlorella, 3000 mg	Tablet	O	0	0	1	0	1	-	-	0	0	4000	2
Chlorella, 970 mg	Tablet	P	0	0	1	1	0	-	-	0	1	1000	3
RNA/DNA (nucleotide complex), 270mg	Tablet	Q	0	1	1	0	0	-	-	0	1	650	3
RNA/DNA (nucleotide complex), 110mg	Capsule	R	0	0	1	0	1	-	-	0	1	2000	3
RNA/DNA (nucleotide complex), 150mg	Capsule	S	0	0	1	0	1	-	-	0	1	100	3
Royal Jelly, 1500 mg	Capsule	U	0	0	1	0	1	-	-	0	0	267	2
Royal Jelly, 370 mg	Liquid	T	0	0	1	0	1	-	-	0	0	67	2
Spirulina, 3000 mg	Powder	K	0	0	1	0	0	-	-	0	1	1333	2
Spirulina, 3000 mg	Powder	L	0	0	1	1	1	-	-	0	0	600	3

A score of "1" was given to products found in DSLD and online retail websites including Amazon, Walmart, and others, and a score of "2" was given to products found in NHANES due to their use by American consumers. Data entries with a dash "-" were not collected.

[α] Sales were averaged over a period of three months from web scraping data collected in January to March 2025.

2. Analytical Laboratory Methodology and Quality Control

Sample Preparation and Extraction

All food samples except for chicken eggs, raw asparagus, and plain, whole grain, uncooked oats (as described above) were homogenized prior to shipment at UF CTSI-SECIM. Upon arrival, samples were transferred into a freezer and stored in -80 °C until analysis. Unprocessed dietary supplements were stored at room temperature.

The United States Pharmacopoeia (USP) has developed analytical reference standards in the form of monographs and general chapters for dietary and herbal supplements. However, monographs for the 5 categories of purine-containing DS do not exist. Therefore, for the preparation of supplements, a total of 20 pills (30 if pills were ≤ 20 mm) per DS were homogenized following USP general guidance (U.S. Pharmacopoeia (USP), <2040>). Tablets and capsules (along with the shell) were homogenized using a coffee grinder. The jars were cleaned out between grinding with deionized water and methanol. Fine, powdered samples were processed as-is without further homogenization. Resulting material was prepared in duplicate, and remaining material was stored at -80 °C for retesting as needed. Additionally, brewer's yeast tablets from the same batch of material were prepared and analyzed individually a total of 6 times for reproducibility.

Purines were extracted using acid hydrolysis according to previously published methodology with minor modification (Kaneko et al., 2014). 1.0 g of material was transferred to a 16-mL glass tube. Ten mL of liquid chromatography–mass spectrometry (LC-MS) grade water was added to each sample along with 0.5 mL of 2,6-diaminopurine as an internal standard. Samples were sonicated using a high-powered sonicator (Model #FS-220, Fisher Scientific, Waltham, MA) for 15 minutes.

Acid hydrolysis was performed by adding 3 mL of 70% perchloric acid ($HClO_4$) solution with heating in a water bath for 60 minutes at 95 °C. The samples were then neutralized by adding 3.5 mL of ammonium hydroxide (NH_4OH) to pH 8.0. Samples were filtered through a Grade 1 Whatman qualitative filter paper (70 mm diameter) (Cytiva, Wilmington, DE). An aliquot of the filtrate was transferred to a clean tube along with 20 µL of purine internal standards and 200 µL of precipitation solution (45:5 acetonitrile:acetone with 0.01% ammonium hydroxide). Samples were centrifuged at 20,000 x g for 10 minutes at 4 °C before transferal to HPLC vials.

Qualitative and Quantitative Analysis

Purine content was determined using Hydrophilic Interaction Liquid Chromatography coupled with Tandem high-resolution mass spectrometry (HILIC-HRMS/MS). Samples were analyzed using a Thermo Scientific Vanquish UHPLC system coupled to a Thermo-Scientific Q-Exactive orbitrap mass spectrometer operated in negative heated electrospray ionization mode. Chromatographic separation was performed using a HILIC-Z column (1.9 µm, 2.1 mm X 150 mm; Agilent, Santa Clara, CA). Mobile phase A was 15 mM ammonium formate (NH_4HCO_2) in 1:9 water: acetonitrile (pH 9.9) and mobile phase B was 15 mM ammonium formate in 9:1 water: acetonitrile (pH 9.9). The pH was adjusted with ammonium hydroxide. Both mobile phases were spiked with 0.5% medronic acid ($CH_6O_6P_2$). The injection volume was 3 µL with a flow rate of 0.4 mL/min.

A mixture of purine standards (xanthine, hypoxanthine, guanosine, guanine, adenine, inosine, AMP, IMP, GMP and uric acid) was prepared at a concentration of 500 µM and a mixture of internal standards

(inosine-$^{15}N_4$, adenine-$^{15}N_5$, xanthine-^{13}C,$^{15}N_2$, hypoxanthine-^{13}C,$^{15}N_2$, guanosine-$^{13}C_2$,^{15}N, and AMP-$^{13}C_5$; ≥ 95% purity) was prepared at a concentration of 10 µg/mL. This mixture of internal standards was spiked into each calibration level and samples. A ratio of area under the curve for each analyte to the area under the curve for the respective internal standard was plotted to calculate the amount of each analyte in the sample. Using this stock mixture of standards, an external calibration curve ranging from 50 ng/mL to 25,000 ng/mL was prepared. For hypoxanthine, a separate external curve was prepared with concentration range 250 ng/mL to 400 µg/mL.

Quality Control

Quality control (QC) samples were prepared from separate stock solutions of purine standards at three levels: 750 ng/mL, 2500 ng/mL and 7500 ng/mL. Hypoxanthine QC levels were prepared at 750 ng/mL, 7500 ng/mL, 25000 ng/mL and 75000 ng/mL. QCs were acquired every 10 samples to check the performance of the instrument and stability of the analytes throughout the batch. Blind duplicates and/or replicate material were included in each batch of analyses to evaluate the repeatability and reproducibility of the data. The limit of detection (LOD) for purines in food and DS samples was 50 ng/mL. Sample errors for all the analyses were maintained under 15% to account for the inherent variability of foodstuffs.

Moisture Content Analysis for Seafood and Organ Meats

After cooking, the raw and cooked seafood and organ meat samples and blind duplicates were shipped overnight on dry ice to USDA's MAFCL in Beltsville, MD, for moisture content determination. Samples were frozen at -80° C upon arrival until analysis. The drying method was slightly modified based on the Association of Official Agricultural Chemists (AOAC) Method 950.46 (AOAC, 2023).

Samples were grouped into 6 different batches; the first batch of material was thawed overnight at 4° C, while remaining batches were thawed at room temperature for 1.5 hours prior to analysis. For all 6 groups of samples, the contents of each sample bag were manually mixed thoroughly to reincorporate any released liquid and ensure sample homogeneity. All samples were kept sealed during thawing and manual mixing, and no drip loss occurred. The weight of a labeled, dry aluminum weighing dish was recorded with a high-precision analytical balance (Sartorius Quintix224-1S, 220 g × 0.1 mg). Approximately 1.0 grams of material were spread thinly and uniformly across the bottom of the aluminum weighing dish. The weight of the dish and wet sample were recorded. Samples were placed into a Lab-Line L-C Oven (Lab-Line Instruments, Melrose Park, IL) at 120°C for 4.5 hours. Samples were removed, placed into a desiccator, and weighed again to determine the difference in moisture content before and after drying.

3. Data Discussion

Food Samples

USDA's analytical laboratory data (*Tab 1*) shows the purine contents of foods varies depending on category, food type, cultivar, cooking status, and more. Meats and organ meats contained more purines than foods in the dairy, legumes, and vegetables categories. Variation in purine content was also observed within food categories. For example, the total amount of purines in pork products ranged from 141 to 448 mg/100 g depending on the meat cut, processing state, and cooking status. Notably, the purine content measured in most of USDA's meat samples were higher compared to literature values apart from the turkey and organ meat values (Clifford et al., 1976; Young, 1980). Certain foods were higher or lower in purine content than the rest of the foods within the same category. These included green peas, which were

higher in total purines (72 mg/100 g), and cooked chickpeas, which were lower in total purines (11 mg/100 g) compared to other legumes which ranged from 21 to 35 mg/100 g. Less than 1.0 mg/100 g total purines were measured in chicken egg whites and yolks, which is comparable to data in Kaneko et al. (2014). These analytical data are consistent with dietary guidance classifying meat and offal as purine-rich foods (Albayda, M, 2026; National Kidney Foundation, 2025).

Care should be taken in interpreting differences between the raw and cooked meats. For all raw to cooked paired meat products, total purine content on a milligram per 100-gram basis increased due to moisture and fat loss from cooking (Table 2). For example, raw bacon appeared to contain over three times fewer total purines than cooked bacon (141 vs. 448 mg/100 g). When moisture loss from cooking was considered, negligible differences between the purine content of the two products were observed. Processed meats (e.g., frankfurters), however, retained their total purine content before and after boiling because they were already pre-cooked and ready-to-eat (RTE). These findings suggest that cooked meats should be considered when developing sampling plans for the analysis of purine content in foods. Furthermore, the inclusion of these products in databases could facilitate more accurate dietary estimations of total purine intake.

The total purines in raw and cooked seafood ranged from 34 to 404 mg/100 g for the finfish and shellfish species. Generally, shellfish and mollusks contained lower amounts of total purines compared to finfish. Like the paired meats, purine contents in cod, tilapia, and yellowfin tuna increased on a milligram per 100-gram basis after moisture and fat losses from cooking. However, for catfish, Atlantic salmon, and shrimp, purine contents declined after cooking. Interestingly, raw and cooked Atlantic salmon skin contained the highest quantity of purines (404 and 335 mg/100 g, respectively) of all seafood samples, suggesting that purines are highly concentrated in the skin. A high variation of the total purines was also observed for the salmon skin samples. The purine content of fish skin is variable and may depend upon factors such as fish species, age of the fish when caught, and quality of aquaculture feed.

A selection of highly consumed fruits and nuts in the U.S. were analyzed for Release 3.0 to fulfill critical database gaps for these food groups. The total purine contents for fruits (1.8 – 3.7 mg/100 g) and nuts (16 – 51 mg/100 g) were comparable to foods in the dairy category and other plant-based foods. Our findings for whole almonds (23 mg/100 g) and walnuts (16 mg/100 g) were aligned with values published in Kaneko et al. (2024). These results corroborate existing dietary recommendations and provide new analytical information for underrepresented food categories.

There was stratification of uricogenic bases across categories and food types. Hypoxanthine was the predominant purine base found in meats and most seafood, whereas dairy, fruits, nuts, legumes, and vegetables were primarily composed of adenine and guanine. Many of the shellfish analyzed, such as cooked lobster and oyster, crab legs, and frozen squid, also contained primarily adenine and guanine. Organ meats were primarily composed of adenine and guanine, while hypoxanthine was a small contributor to total purine content except in the profile of chicken gizzards. The foods' purine composition remained stable in meat and seafood samples after cooking except for ground beef ("73% lean meat / 27% fat" and "93% lean meat / 7% fat") and in Atlantic salmon skin. In these samples, an increase in the percentage of hypoxanthine and decreases in the percentages of adenine, guanine, and xanthine after cooking were observed.

Concentrations of uric acid in foods were measured and reported in the database when they were present as a foundation for future research. Uric acid was not detected in most samples because it does not exist in plants, and it is not the final metabolic product for almost all animal foods and seafoods studied. The presence of uric acid in animal foods has only been reported in one publication (Havlik et al., 2010), in which a significant amount of uric acid (51 mg/100 g) was detected in chicken liver. Chicken legs and beef were also found to contain low levels of uric acid. A significant amount of uric acid was detected in USDA's raw and cooked beef liver samples – 123 and 49 mg/100 g, respectively.

Blue crab contained 32 – 34 mg/100 g uric acid. Concentrations of less than 1.0 mg/100 g were observed in other foods including beef, dairy, poultry, sausage, and seafood categories. Interestingly, low concentrations of uric acid were detected in lobster and snow crab legs, and uric acid was undetected in shrimp. Unlike purines, the bioavailability of dietary uric acid is largely unknown, although it is known that it can be potentially absorbed through uric acid transporters in the intestine. The impact of uric acids in foods on SU levels has not been studied, but the topic certainly warrants further research.

When differences in USDA's data exist compared to published values from the literature, they may be attributable to vegetable cultivars, animal species, geographic differences, agricultural and animal husbandry methodology changes over time, and manufacturer processing disparities. The studies' sampling plan methodologies and analytical instrumentation may have also impacted measurements. Statistical comparisons between USDA and literature data were not possible due to low (n) and data access limitations. Overall, the findings in this study highlight a need for current data on the purine content of foods, and a reevaluation of dietary guidance based on this for populations interested in lowering purine consumption.

Dietary Supplement Samples

To facilitate analysis, products from the 6 DS categories were grouped based on their dosage form (capsule, liquid, powder, and tablet). For instance, all brewer's yeast tablet product results were averaged and included in the database as one entry. This approach consolidated 28 total products into 12 entries in the database with each representing a distinct dosage form.

Dietary supplement data is presented in three formats: as "per serving unit" (*Tab 4*), as "per serving size" (*Tab 5*), and as "per daily serving" (*Tab 6*). The analytical results indicate that purine content in DS varied significantly across categories, dosage form, and brand. Brewer's yeast powder exhibited the highest total amount of purines (155 mg/serving size), while liquid royal jelly contained the lowest (2.7 mg/serving size). In descending total order of total purine content per serving, the ranking was as follows: (1) brewer's yeast [powder] > (2) chlorella [tablet] > (3) spirulina [tablet] > (4) nucleotide complex [capsule] > (5) spirulina [powder] > (6) chlorella and spirulina [tablet] > (7) brewer's yeast [tablet] > (8) nucleotide complex [tablet] > (9) chlorella and spirulina [capsule] > (10) brewer's yeast [capsule] > (11) royal jelly [capsule] > (12) royal jelly [liquid]. Royal jelly was consistently lowest in total purine content irrespective of dosage form.

Adenine and guanine constituted over 90% of the total purines in all tested supplements except for spirulina tablet (89.1%). Notably, uric acid was not detected in any of the DS samples. An internal comparison revealed that USDA's analytical DS data (in mg/100 g) were consistently lower in average total purines compared to published literature values from Kaneko et al. (2014) apart from the spirulina

tablet. This discrepancy is likely attributable to geographical differences and variations in manufacturing processes. USDA also observed considerable differences in total purine content across product lots and ingredient categories. Among the 5 new DS lots examined in 2025, 3 contained 40 – 60% fewer total purines compared to same products analyzed in 2024. For the other 2 DS, minimal differences in purine content (1 – 3 %) between lots were observed. All supplement lots were purchased within a 12-month period and were tested well before their expiration dates.

Finally, reproducibility was assessed in 2024 to ensure analytical consistency among the DS. The relative standard deviation percentage for the total number of purines in brewer's yeast was 0.7, indicating good reproducibility of the sample preparation and extraction protocols.

E. Database Strengths and Limitations

This database has several strengths. To the authors' knowledge, this is the first study to examine the purine content in a representative selection of widely consumed purine-containing DS in the United States. Furthermore, the database provides information for the contents of purine bases in foods covering many different food categories, which were obtained from an extensive literature search and directly assayed analytical data. Literature data were based on research findings from studies whose published reports met the minimal criteria for inclusion using established data evaluation protocols. Compared to Release 1.0, USDA's new analytical data provides new and updated values for many foods collected from the U.S. market. The foods selected for analysis were expected to contain substantial amounts of purines or were analyzed to fill knowledge gaps. The selected foods are of relevance and importance to the U.S. population based on dietary intake patterns extrapolated from WWEIA.

In addition, the purine contents in raw and cooked pairs of selected commonly consumed meat and seafood products were included in the database. This data will assist with more accurate estimations of purine intake as most animal products are cooked before consuming. Another strength is that multiple samples were purchased and analyzed to understand variability. Finally, this analytical purine data allows for more accurate intake estimates. Thus, the database can be used by clinicians, public health researchers, and consumers to manage the dietary intake of purines and to facilitate treatment of subgroups diagnosed with hyperuricemia and gout.

Limitations of the database include the possible significant discrepancies between the literature data and USDA's analytical data. It should be noted that nearly half of all literature data in the database originated from studies performed 3 decades ago. Most of the literature data only presented a sample (n) of 1. Because of this and the small sample (n) observed in the studies, statistical comparisons between USDA's analytical data and literature data were not possible. The USDA foods selected for analysis were partially chosen based on their anticipated purine content presumed from literature data. Therefore, analytical data for some food categories (e.g., baked goods, sweets) are not included in this release. The analytical values provide representative estimates of purine content in select foods, but do not necessarily represent values for the entire U.S. market due to the nature of convenience sampling and product formulation differences over time. Similarly, the new DS published in Release 3.0 were not present in NHANES, nor were most products available in DSLD. Although web-scraped data is valuable for interpreting consumer patterns, it is retrospective over a small duration of time and may be ineffective in future evaluation efforts. Furthermore, despite the dozen new DS added in Release 3.0, categories and dosage forms are still limited in sample size (n = 1 – 4); additional analyses may improve representativeness.

F. Summary: Plans for Foods and Dietary Supplements in Future Data Releases

USDA's Purine Database Release 3.0 provides new analytical data for 43 food listings from 9 food groups and 14 new dietary supplements from a total of 6 categories, expanding the database to cover a wider range of products. These listings are built on USDA's Release 2.0 dataset, which included analytical data for 61 foods and 14 dietary supplements. The database includes previously published literature data from 14 published international studies for 401 food listings from 20 food groups and 15 alcoholic beverages. These literature data are useful in further strengthening the scientific evidence base for identifying additional U.S. foods for purine analysis and in determining foods of substantial purine content for which expanded sample sizes would be advantageous. These new, validated analytical purine data provide valuable insights into the purine content of commonly available foods and purine-rich DS in the U.S. and may be used for improving estimates of dietary purine intake. This database is dynamic and will be expanded in 2027 when additional analyses of U.S. foods become available.

USDA's MAFCL will continue to procure and analyze foods from the U.S. food supply relevant for estimating purine intake for the next data release. Of particular interest are single-ingredient staples of the American diet not yet analyzed by USDA (e.g., bananas, corn, potatoes, and rice). Data generated for these foods will be used to create U.S. estimates of purine intake that will aid clinicians, researchers, and public health professionals in understanding and managing hyperuricemia and gout. In addition, the long-term stability of purines may be assessed in foods currently frozen in storage. Furthermore, literature-derived purine data on foodstuffs and DS published after February 2023 will be identified and evaluated for potential inclusion using established data evaluation criteria, and acceptable data will be added to the database. Importantly, the expanded dataset will also enable more rigorous human studies by providing standardized, analytically verified purine values that can be used to validate dietary assessment methods, examine individual differences in purine metabolism, and clarify relationships between purine intake, serum urate, and gout risk. Improved understanding of the links between dietary purine exposure and hyperuricemia in human populations is essential to develop more precise and evidence-based dietary recommendations for individuals at risk.

Information on the food and DS contents of various methylxanthines may prove a useful adjunct to this purine dataset due to their chemical similarity, as well. Methylxanthines are chemical derivatives of the purine base xanthine which are naturally present in some plants (e.g., cocoa and coffee beans, tea leaves, cola nuts, guarana, and yerba mate). Methylxanthines such as caffeine, theobromine, and theophylline act as central nervous system stimulants causing diuresis, cardiovascular and metabolic effects, bronchial relaxation, and gastric acid secretion (Franco, Oñatibia-Astibia, & Martínez-Pinilla, 2013). Caffeine decreases the concentration of urate by inhibiting xanthine oxidase activity (Chittoor & Voruganti, 2020). Relatedly, coffee consumption has shown an inverse association with gout incidence (Choi et al., 2007). Coffee consumption also has an inverse association with hyperuricemia in men but is associated with increased hyperuricemia in women (Li et al., 2018). On the other hand, tea consumption does not appear to be associated with hyperuricemia, urate levels, or gout (Zhang et al., 2017). Relevant foods and supplements could be assayed and included as data fields to allow better estimates of total dietary intake and as effect modifiers in studies of purine metabolism. A methylxanthine database containing analytical data from the USDA-NIH NFNAP program is planned for release.

G. References

Aihemaitijiang, S., Zhang, Y., Zhang, L., Yang, J., Ye, C., Halimulati, M., Zhang, W., Zhang, Z. (2020). The association between purine-rich food intake and hyperuricemia: A cross-sectional study in Chinese adult residents. *Nutrients, 12, 3835.*

Albayda, M. (2026, August 5). *Gout Diet: What to Eat and What to Avoid*. Health. https://www.hopkinsmedicine.org/health/expert-qa/gout-diet-what-to-eat-and-what-to-avoid (accessed on 10 August 2026).

AOAC. AOAC Official Method 950.46. Loss on Drying (Moisture) in Meat. In Official Methods of Analysis of AOAC International (22nd Edition); Association of Official Analytical Chemists International: Gaithersburg, MD, USA, 2023.

Azzeh, F.S., Al-Hebshia, A.H., Al-Essimiia, H.D., Alarjahb, M.A. (2017). Vitamin C supplementation and serum uric acid: A reaction to hyperuricemia and gout disease. *PharmaNutrition, 5, 47-51.*

Battison, A.L. (2013). Subcuticular urate accumulation in an American lobster (Homarus americanus). *Veterinary Pathology, 50(3), 451-6.*

Belanger, M.L., Wee, C.C., Mukamal, K.J., Miller, E.R., Sacks, F.M., Appel, L.J., Shmerling, R.H., Choi, H.K., Juraschek, S.P. (2021). Effects on dietary macronutrients on serum urate: results from the OmniHeart trial. *American Journal of Clinical Nutrition, 113, 1593-1599.*

Berger L., Gerson, C.D., Yu,T.F. (1977). The effect of ascorbic acid on uric acid excretion with a commentary on the renal handling of ascorbic acid. *American Journal of Medicine*, *62, 71–76.*

Beydoun, M.A., Fanelli-Kaczmarski, M.T., Canas, J.-A., Beydoun, H.A., Evans, M.K., Zonderman, A.B. (2018). Dietary factors are associated with serum uric acid trajectory differentially by race among urban adults. *British Journal of Nutrition, 120, 935-935.*

Bhagwat, S.A., Patterson, K.Y., Holden, J.M. (2009). Validation study of the USDA's data quality evaluation system. *Journal of Food Composition and Analysis*, *22*, *366-372*.

Brodkorb, A., Egger, L., Alminger, M., Alvito, P., Assuncão, R., Balance, S., Bohn, T., Bourlieu-Lacanal, C., Boutrou, R., Carrière, F., Clemente, A., Corredig, M., Dupont, D., Dufour, C., Edwards, C., Golding, M., Karakaya, S., Kirkhus, B., Feunteun, S.L., Lesmes, U., Macierzanka, A., Mackie, A.R., Martins, C., Marze, S., McClements, D.J., Ménard, O., Minekus, M., Portmann, R., Santos, C.N., Souchon, I., Singh, R.P., Vegarud, G.E., Wickham, M.S.J., Weitschies, W., Recio, I. (2019). INFOGEST Static in Vitro Simulation of Gastrointestinal Food Digestion. *Nature Protocols, 14.4, 991–1014.*

Brulé, D., Sarwar, G., Savoie, L. (1992). Changes in serum and urinary uric acid levels in normal human subjects fed purine-rich foods containing different amounts of adenine and hypoxanthine. *Journal of the American College of Nutrition, 11, 353-358.*

Butler, F, Alghubayshi, A, Roman, Y. (2021) The Epidemiology and Genetics of Hyperuricemia and Gout across Major Racial Groups: A Literature Review and Population Genetics Secondary Database Analysis. *Journal of Personalized Medicine, 11(3), 231.*

Chen-Xu, M., Yokose, C., Rai, S.K., Pillinger, M.H., Choi, H.K. (2019). Contemporary prevalence of gout and hyperuricemia in the United States and decadal trends: the National Health and Nutrition Examination Survey, 2007-2016. *Arthritis & Rheumatology,71, 991-999.*

Choi, H.K., Atkinson, K., Karlson, E.W., Willett, W., Curhan, G. (2004). Purine-rich foods, dairy and protein intake, and the risk of gout in men. *New England Journal of Medicine*, *350, 1093-1103.*

Choi, H.K., Liu, S., Curhan, G. (2005). Intake of purine-rich foods, protein, and dairy products and relationship to serum levels of uric acid. *Arthritis & Rheumatism, 52(1), 283-289.*

Choi, H.K., Willett, W., Curhan, G. (2007). Coffee consumption and risk of incident gout in men: a prospective study. *Arthritis & Rheumatism, 56(6), 2049-2055.*

Choi, H.K., Gao, X., Curhan, G. (2009). Vitamin C intake and the risk of gout in men, A prospective study. *Archives of Internal Medicine 169(5), 502-507.*Chittoor, G., Voruganti, V. (2020). Hyperuricemia and Gout. In R. Caterina, J. Martinez, & M. Kohlmeier (Eds.), *Principles of Nutrigenetics and Nutrigenomics: Fundamentals of Individualized Nutrition (pp. 389-394).* Academic Press: Cambridge MA.

Cipolletta, E., Tata, L.J., Nakafero, G., Avery, A.J., Mamas, M.A., Abhishek, A. (2022). Association between gout flare and subsequent cardiovascular events among patients with gout. *Journal of American Medical Association, 328(5), 440-450.*

Clifford, A.J., Riumallo, J.A., Young, V.R., Scrimshaw, N.S. (1976). Effects of oral purines on serum and urinary uric acid of normal, hyperuricaemic and gouty humans. *Journal of Nutrition, 106, 428–5.*

Dalbeth, N., Ames, R., Gamble, G.D., Horne, A., Wong, S., Kuhn-Sherlock, B., MacGibbon, A., McQueen, F.M., Reid, I.R., Palmano, K. (2012). Effects of skim milk powder enriched with glycomacropeptide and G600 milk fat extract on frequency of gout flares: a proof-of-concept randomised controlled trial. *Annals of the Rheumatic Diseases, 71. 929-934.*

FitzGerald, J.D., Dalbeth, N., Mikuls, T., Brignardello-Petersen, R., Guyatt, G., Abeles, A.M., Gelber, A.C., Harrold, L.R., Khanna, D., King, C., Levy,G., Libbey, C., Mount, D., Pillinger, M.H., Rosenthal, A., Singh, J.A., Sims, J.E., Smith, B.J., Wenger. N.S., Bae, S.S., Danve, A., Khanna, P.P., Kim, S.C., Lenert, A., Poon, S., Qasim, A., Sehra, S.T., Sharma, T.S.K., Toprover, M., Turgunbaev, M., Zeng, L., Zhang, M.A., Turner, A.S., Neogi, T. (2020). American College of Rheumatology Guideline for the Management of Gout. *Arthritis Care & Research***,** *72 (6), 744–760*.

Franco, R., Oñatibia-Astibia, A., Martínez-Pinilla, E. (2013). Health benefits of methylxanthines in cacao and chocolate. *Nutrients, 18;5 (10), 4159–4173.*

GBD 2021 Gout Collaborators. (2024). Global, regional, and national burden of gout, 1990-2020, and projections to 2050: a systematic analysis of the Global Burden of Disease Study 2021. *The Lancet Rheumatology, 6(8), e507-e517.*

Gibson, T., Rodgers, A.V., Simmonds, H.A., Court-Brown, F., Todd, E., Meilton. V. (1983). A controlled study of diet in patients with gout. *Annals of Rheumatic Diseases, 42, 123–127.*

Hak, A.E., Curhan, G.C., Grodstein, F., Choi, H.K. (2010). Menopause, postmenopausal hormone use and risk of incident gout. *Annals of Rheumatic Diseases, 69(7), 1305-1309.*

Havlik, J., Plachy, V., Fernandez, J., Rada, V. (2010). Dietary purines in vegetarian meat analogues. *Journal of the Science of Food and Agriculture, 90(14), 2352-2357.*

Haytowitz, D.B., Pehrsson, P.R. (2018). USDA's National Food and Nutrient Analysis Program (NFNAP) produces high-quality data for USDA food composition databases: Two decades of collaboration, *Food Chemistry, 238(1), 134-138*.

Hisatome, I., Ichida, K., Mineo, I., Ohtahara, A., Ogino, K., Kuwabara, M., Ishizaka, N., et al. (2020). Japanese society of gout and uric & nucleic acids 2019: Guidelines for management of hyperuricemia and gout 3rd edition. *Gout and Uric & Nucleic Acids,.44 (Supplement), 1-40.*

Holden, J.M., Bhagwat, S.A., Patterson, K.Y. (2002). Development of a multi-nutrient data quality evaluation system. *Journal of Food Composition and Analysis, 15*, *339–348*.

Holden, J.M., Bhagwat, S.A., Haytowitz, D.B., Gebhardt, S.E., Dwyer, J.T., Peterson, J., Beecher, G.R., Eldridge, A.L., Balentine, D. (2005). Development of a database of critically evaluated flavonoids data: application of USDA's data quality evaluation system. *Journal of Food Composition and Analysis, 18, 829-844.*

Horne, F.R. (1968). Nitrogen excretion in crustacea. I. The herbivorous land crab Cardiosoma guanhumi Latreille. *Comparative Biochemistry and Physiology, 26(2), 687-95.*

Hu, J-R., Yeh, H-C., Mueller, N.T., Appel, L.J., Miller, E.R., Maruthur, N.M., Jerome, G.J., Chang, A.R., Gelber, A.C., Juraschek, S.P. (2021). Effects of a behavioral weight loss intervention and metformin treatment on serum urate: results from a randomized clinical trial. *Nutrients, 13, 2673*.

Huang, H-Y., Appel, L.J., Choi, M.J., Gelber, A.C., Charleston, J., Norkus, E.P., Miller, E.R. (2005). The effects of vitamin C supplementation on serum concentrations of uric acid. *Arthritis & Rheumatism, 52(6), 1843-1847.*

Huang, Z., Xie, N., Illes, P., Di Virgilio, F., Ulrich, H., Semyanov, A., Verkhratsky, A., Sperlagh, B., Yu, S.G., Huang, C., Tang, Y. (2021). From purines to purinergic signalling: molecular functions and human diseases. *Signal Transduction and Targeted Therapy, 6(1), 162.*

Inazawa, K., Sato, A., Kato, Y., Yamaoka, N., Fukuuchi, T., Yasuda, M., Mawatari, K., Nakagomi, K., Kaneko, K (2014). Determination and profiling of purines in foods by using HPLC and LC-MS. *Nucleosides, Nucleotides & Nucleic Acids*, *33(4-6), 439-444*.

Jakse, B., Jakse, B., Pajek, M., Pajek, J. (2019). Uric Acid and Plant-Based Nutrition. *Nutrients*,*11:1736*.Juraschek, S.P., Miller, E.R., Gelber, A.C. (2011). Effect of oral vitamin C supplementation on serum uric acid: a meta-analysis of randomized controlled trials. *Arthritis Care & Research, 63(9), 1295-1306.*

Juraschek, S.P., Gelber, A.C., Choi, H.K., Appel, L.J., Miller, E.R. (2016). Effects of the dietary approaches to stop hypertension (DASH) diet and sodium intake on serum uric acid. *Arthritis & Rheumatology*, *68, 3002-3009*.

Juraschek, S.P., Miller, E.R., Wu, B., White, K., Charleston, J., Gelber, A.C., Rai, S.K., Carson, K.A., Appel, L.J., Choi, H.K. (2021). A randomized pilot study of DASH patterned groceries on serum urate individuals with gout. *Nutrients, 13, 538*.

Juraschek, S.P., Gaziano, J.M., Glynn, R.J., Gomelskaya, N., Bubes, V.Y., Buring, J.E., Shmerling, R.H., Sesso, H.D. (2022). Effects of vitamin C supplementation on gout risk: results from the Physicians' Health Study II trial. *American Journal of Clinical Nutrition, 0: 1-8.*

Kaneko, K., Aoyagi, Y., Fukuuchi, T., Inazawa, K., Yamaoka, N. (2014). Total purine and purine base content of common foodstuffs for facilitating nutritional therapy for gout and hyperuricemia. *Biological and Pharmaceutical Bulletin, 37, 709-721.*

Kaneko, K., Takayanagi, F., Fukuuchi, T., Yamaoka, N., Yasuda, M., Ken-ichi Mawatari, K., Fujimori, S. (2020). Determination of total purine and purine base content of 80 food products to aid nutritional therapy for gout and hyperuricemia. *Nucleosides, Nucleotides & Nucleic Acids, 39 (10-12), 1449-1457.*

Khanna, D., Fitzgerald, J.D., Khanna, P.P., Bae, S., Singh, M.K., Neogi, T., Pillinger, M.H., Merill, J., Lee, S., Prakash, S., Kaldas, M., Gogia, M., Perez-Ruiz, F., Taylor, W., Lioté, F., Choi, H., Singh, J.A., Dalbeth,,N., Kaplan, S., Niyyar, V., Jones, D., Yarows, S.A., Roessler, B., Kerr, G., King, C., Levy, G., Furst, D.E., Edwards, N.L., Mandell, B., Schumacher, H.R., Robbins, M., Wenger, N., Terkeltaub, R. (2012). American College of Rheumatology guidelines for management of gout. Part 1: systematic nonpharmacologic and pharmacologic therapeutic approaches to hyperuricemia. *Arthritis Care and Research*, *64*, *1431-1446.*

Kuo, C-F., Grainge, M.J., Zhang, W., Doherty, M. (2015). Global epidemiology of gout: prevalence, incidence and risk factors. *Nature Reviews Rheumatology, 11(11),649-662.*

Li, R., Yu, K., Li, C. (2018). Dietary factors and risk of gout and hyperuricemia: a meta-analysis and systematic review. *Asia Pacific Journal of Clinical Nutrition, 27(6), 1344-1356.*

Luk, A.J., Simkin, P.A. (2005). Epidemiology of hyperuricemia and gout. *American Journal of Managed Care, 11, S435-442.*

MacFarlane, L.A., Kim, S.C. (2014). Gout: a review of non-modifiable and modifiable risk factors. *Rheumatic Disease Clinics of North America, 40(4), 581-604.*

Mallen, C.D., Davenport, G., Hui, M., Nuki, G. Roddy, E. (2017). Improving management of gout in primary care: a new UK management guideline. *British Journal of General Practice, 67, 284-285.*

National Kidney Foundation. (2025). *What to eat (and avoid) if you have gout*. National Kidney Foundation. https://www.kidney.org/news-stories/what-to-eat-and-avoid-if-you-have-gout (accessed on 10 August 2026).

Nelson, K.L., Voruganti, V.S. (2021). Purine metabolites and complex diseases: role of genes and nutrients. *Current Opinion in Clinical Nutrition and Metabolic Care*, *24(4), 296-302.*

Nieradko-Iwanicka, B. (2022). The role of alcohol consumption in pathogenesis of gout. *Critical Reviews in Food Science and Nutrition, 62(25), 7129-7137*.

Nuki, G., Simkin, P.A. (2006). A concise history of gout and hyperuricemia and their treatment. *Arthritis Research and Therapy, 8, S1.*

Pennington, J.A.T., Spungen, J. (2012). Bowes & Church's food values of portions commonly used, 19th edition. eBook. ISBN: 9781451157475 1451157479

Pillinger, M.H., Keenan, R.T. (2008). Update on the management of hyperuricemia and gout. *Bulletin of the NYU Hospital for Joint Diseases, 66(3), 231-239.*

Rai, S.K., Fung, T.T., Lu, N., Keller, S.F., Curhan, G.C., Choi, H.K. (2017). The dietary approaches to stop hypertension (DASH) diet, Western diet, and risk of gout in men: prospective cohort study. *The BMJ*, *357, 1794.*

Rathbone, M.P., Middlemiss, P.J., Gysbers, J.W., Andrew, C., Herman, M.A.R., Reed, J.K., Ciccarelli, R., Di Iorio, P., Caciagli, F. (1999). Trophic effects of purines in neurons and glial cells. *Progress in Neurobiology, 59(6), 663-690.*

Richette, P., Bardin, T. (2017). Purine-rich foods: an innocent bystander of gout attacks? *Annals of Rheumatic Diseases, 71(9), 1435-1436.*

Ridi, R.E., Tallima, H. (2017). Physiological functions and pathogenic potential of uric acid: a review. *Journal of Advanced Research, 8, 287-493*.

Sarwar, G., Brulé, D. (1991). Assessment of the uricogenic potential of processed foods based on the nature and quantity of dietary purines. *Progress in Food and Nutrition Science, 15(3), 159-181.*

Safiri, S., Kolahi, A.-A., Cross, M., Carson-Chahhoud, K., Hoy, D.; Almasi-Hashiani, A., Sepidarkish, M., Ashrafi-Asgarabad, A., Moradi-Lakeh, M., Mansournia, M.A., et al. (2020). Prevalence, incidence, and years lived with disability due to gout and its attributable risk factors for 195 countries and territories 1990–2017: A systematic analysis of the global burden of disease study 2017. *Arthritis and Rheumatology, 72, 1916–1927.*

Stein, H.B., Hasan, A., Fox, I.H. (1976). Ascorbic acid-induced uricosuria: a consequence of megavitamin therapy. *Annals of Internal Medicine, 84, 385–388.*

Todd, E., Wright, A. (2020). Gout: origin, treatment, and prevention. *BIOS, 91(1), 66-73.*

U.S. Department of Agriculture (USDA) and National Institutes of Health, Office of Dietary Supplements (NIH ODS). (2021.) Statement of work: Development of a database on purines in foods and dietary supplements. Unpublished. June 23, 2021.

U.S. Department of Agriculture (USDA), Agricultural Research Service (ARS). (2024a). FoodData Central. https://fdc.nal.usda.gov/ (accessed on 21 October 2024).

U.S. Department of Agriculture (USDA), Agricultural Research Service (ARS). (2024b). WWEIA/NHANES Related Links. https://www.ars.usda.gov/northeast-area/beltsville-md-bhnrc/beltsville-human-nutrition-research-center/food-surveys-research-group/docs/wweianhanes-related-links/ (accessed on 22 October 2024).

U.S. Department of Health and Human Services (USDHHS), National Institutes of Health Office of Dietary Supplements (NIH ODS). Dietary Supplement Label Database (DSLD). Accessed June 22, 2021. Available from: https://dsld.nlm.nih.gov/dsld/.

United States Pharmacopeia and National Formulary (USP-NF). (2018). <2040> Disintegration and Dissolution of Dietary Supplements.

Vareldzis, R., Perez, A., Reisin, E. (2024). Hyperuricemia: An Intriguing Connection to Metabolic Syndrome, Diabetes, Kidney Disease, and Hypertension. *Current Hypertension Reports, 26, 237-245.*

Villegas, R., Xiang, Y.B., Elasy, T., Xu, W.H., Cai, H., Cai, Q., Linton, M.F., Fazio, S., Zheng, W., Shu, X.O. (2012). Purine-rich foods, protein intake, and the prevalence of hyperuricemia: The Shanghai Men's Health Study. *Nutrition, Metabolism and Cardiovascular Diseases, 22, 409-416.*

Wu, B., Roseland, J.M., Haytowitz, D.B., Pehrsson, P.R., Ershow, A.G. (2019). Availability and quality of published data on the purine content of foods, alcoholic beverages, and dietary supplements. Journal of *Food and Composition Analysis, 84, 103281.*

Xia, Y., Wu, Q.,Wang, H., Zhang, S., Jiang, Y., Gong, T., Xu, X., Chang, Q., Niu, K., Zhao, Y. (2020). Global, regional and national burden of gout, 1990–2017: A systematic analysis of the global burden of disease study. *Rheumatology, 59, 1529–1538.*

Yamamoto, T., Moriwaki, Y., Takahashi, S. (2005). Effect of ethanol on metabolism of purine bases (hypoxanthine, xanthine, and uric acid). *Clinica Chimica Acta, 356(1-2), 35-57*.

Yanai, H., Adachi, H., Hakoshima, M., Katsuyama, H. (2021). Molecular Biological and Clinical Understanding of the Pathophysiology and Treatments of Hyperuricemia and Its Association with Metabolic Syndrome, Cardiovascular Diseases and Chronic Kidney Disease. *International Journal of Molecular Sciences, 22(17), 9221*.

Yokose, C, McCormick, N, Lu, N, Tanikella, S, Lin, K, Joshi, AD, Raffield, LM, Warner, E, Merriman, T, Hsu, J, Saag, K, Zhang, Y, Choi, HK. (2023). Trends in Prevalence of Gout Among US Asian Adults, 2011-2018. *JAMA Network Open, 6(4), e239501.*

Young, L.L. Evaluation of four purine compounds in poultry products. (1980). *Journal of Food Science, 45(4)*, *1064-5*.

Yu, L., Wang, Y., Huang, R., Pan, H., Xie, Z., Wen, C., Huang, L., Li, X. (2025). Sugar-sweetened beverages and the risk of hyperuricemia and gout: a meta-analysis. *Frontiers in Nutrition, 12:1669129.*

Zgaga, L., Theodoratou, E., Kyle, J., Farrington, S.M., Agakov, F., Tenesa, A., Walker, M., McNeill, G., Wright, A.F., Rudan, I., Dunlop, M.G., Campbell, H. (2012). The association of dietary intake of purine-

rich vegetables, sugar-sweetened beverages and dairy with plasma urate, in a cross-sectional study. *Plos One, 7, e38123*.

Zhang, Y., Chen, C., Choi, H., Chaisson, C., Hunter, D., Niu, J., Neogi, T. (2012). Purine-rich foods intake and recurrent gout attacks. *Annals of the Rheumatic Diseases, 71(9), 1448-1453.*

Zhang, Y., Cui, Y., Li, X., Li, L., Xie, X., Huang, Y, Deng, Y., Zeng, C., Lei, G. (2017). Is tea consumption associated with the serum uric acid level, hyperuricemia or the risk of gout? A systematic review and meta-analysis. *BMC Musculoskeletal Disorders, 18, 95.*

Zollner, N., Griebsch, A. (1974). Diet and gout. *Advances in Experimental Medicine and Biology, 41, 435–442.*