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Identifying Purine Intake Among People with Gout and Its Relationship with Uric Acid Level: a Cross-Sectional Study

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ABSTRACT Gout remains a prevalent chronic disease characterized by elevated uric acid levels, closely linked to unhealthy lifestyle behaviors, particularly high purine intake. Despite its significant health burden, limited studies have explored the direct relationship between purine consumption and uric acid levels in gout patients. This study aims to identify purine intake patterns among individuals with gout and examine their correlation with uric acid levels. A correlational design with a cross-sectional approach was employed, involving 66 purposively sampled participants from Village "X" in Banyuwangi, Indonesia. Purine intake was assessed via a validated questionnaire, while uric acid levels were measured using a GCU meter. Results revealed that 48.5% of participants consumed high-purine foods, and 66.7% exhibited elevated uric acid levels. A significant positive correlation was found between purine intake and uric acid levels (*p* = 0.000; *r* = 0.567), indicating that higher purine consumption was associated with increased uric acid concentrations. The findings underscore the need for targeted health interventions, such as education and dietary modifications, to reduce purine intake and mitigate hyperuricemia risks among gout patients. This study contributes to the growing evidence on dietary influences in gout management, emphasizing the importance of monitoring purine-rich foods to prevent disease progression.

INDEX TERMS purine intake, uric acid levels, hyperuricemia, gout, dietary patterns

I. INTRODUCTION

Gout is a chronic metabolic disorder characterized by hyperuricemia and the deposition of monosodium urate crystals in joints, leading to inflammation, severe pain, and long-term complications such as joint damage and kidney dysfunction [1]. Globally, gout prevalence has risen sharply, affecting 55.8 million individuals in 2020 a 22.5% increase since 1990 with projections estimating 95.8 million cases by 2050 [2], [3]. In Indonesia, gout prevalence reached 3.21% in 2023, with East Java reporting 12.16% of cases [4], [5]. This escalating burden underscores the urgent need to address modifiable risk factors, particularly dietary purine intake, which plays a critical role in uric acid metabolism [6], [7]. Problem Statement Despite extensive research on gout management, the direct relationship between dietary purine intake and uric acid levels remains inconsistently documented. While some studies confirm that high-purine foods (e.g., offal, seafood) elevate uric acid [8], [9], others suggest low-purine diets have negligible effects [10], [11]. Additionally, regional dietary habits and genetic predispositions further complicate these findings [12], [13]. For instance, in Indonesia, traditional diets rich in tempeh, tofu, and spinach often considered low-purine may paradoxically contribute to hyperuricemia when consumed excessively [14], [15]. This discrepancy highlights a critical gap in context-specific dietary guidelines for gout patients.

Recent studies employ diverse methodologies to investigate purine-uric acid dynamics, including cohort analyses [16], [17], cross-sectional surveys [18], and Mendelian randomization [19]. Advanced tools like GCU meters and food-frequency questionnaires (FFQs) have improved data accuracy [20], [21]. However, many studies focus on Western populations, neglecting regional dietary variations [22], [23]. For example, Zhou et al. [24] identified seafood as a primary purine source in coastal China, while Aihemaitijiang et al. [25] emphasized processed meats in urban diets. Such regional disparities necessitate localized research to tailor interventions effectively. Existing literature lacks consensus on:

1. Thresholds: The exact purine intake levels that trigger hyperuricemia in diverse populations [26], [27].
2. Temporal Effects: The duration of high-purine consumption required to elevate uric acid [28].
3. Cultural Diets: The impact of region-specific foods (e.g., Indonesian tempeh) on uric acid [29], [30].

This study addresses these gaps by examining purine-uric acid correlations in a homogeneous Indonesian cohort, controlling for comorbidities and dietary habits.

This study aims to:

1. Quantify purine intake among gout patients in rural Indonesia.
2. Analyze its correlation with uric acid levels.

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Vol. 5 no.3, pp. 120-124, June 2025

3. Propose context-specific dietary recommendations.

Contributions:

1. Empirical Evidence: Provides localized data on purine intake and uric acid levels in an understudied population [31].
2. Methodological Rigor: Combines validated FFQs with GCU meter measurements to enhance reliability [32].
3. Practical Implications: Informs culturally adapted dietary guidelines for Indonesian gout patients [33].

II. METHODS

A. STUDY DESIGN

This study employed a cross-sectional, correlational design to examine the relationship between purine intake and serum uric acid levels among individuals diagnosed with gout. The design was selected to analyze data at a single time point, ensuring efficiency in assessing dietary patterns and biochemical measurements without temporal confounders [21]. The study was non-randomized, with participants selected via purposive sampling to meet predefined inclusion criteria.

B. STUDY POPULATION AND SAMPLING

The target population comprised 70 adults (aged ≥20 years) diagnosed with gout in Village "X," Banyuwangi, Indonesia. A sample of 66 participants was selected based on the following inclusion criteria:

1. Confirmed gout diagnosis for ≥1 year.
2. No comorbid conditions (e.g., hypertension, kidney failure, cardiovascular disease).
3. Willingness to provide informed consent.

Exclusion criteria included:

1. Use of uric acid-lowering medications (e.g., allopurinol) in the past month.
2. Acute gout flare-ups during data collection.
3. Incomplete dietary or biochemical data.

Participants were recruited through community health records and local healthcare providers to ensure diagnostic accuracy [22].

C. DATA COLLECTION INSTRUMENTS

1. Purine Intake Assessment

The questionnaire was validated (Cronbach's α = 0.72) and pretested in a pilot study (n = 10). A structured questionnaire was administered, assessing:

a. Frequency of high-purine food consumption (e.g., offal, seafood, legumes).
b. Cooking methods (e.g., boiling, frying).
c. Daily intake quantities (categorized as low [<200 mg/day], moderate [200–400 mg/day], or high [>400 mg/day]) [23].

2. Uric Acid Measurement

a. Serum uric acid levels were measured using the EasyTouch GCU Meter, a portable device with 95% accuracy compared to laboratory tests [24].
b. Measurements were taken after an 8-hour fast to minimize dietary interference.
c. Classification:
1) Men: Normal (2.5–7.0 mg/dL), High (>7.0 mg/dL).
2) Women: Normal (1.5–6.0 mg/dL), High (>6.0 mg/dL) [25].

D. STUDY PROCEDURES

1. Ethical Approval

a. The study was approved by the Institutional Review Board of STIKES Rustida (No. 283/03/KEPK-STIKESBWI/VII/2024).
b. Participants provided written informed consent before enrollment.

2. Data Collection

a. Step 1: Baseline interviews to confirm eligibility and collect demographic data.
b. Step 2: Questionnaire administration (15–20 min) by trained researchers.
c. Step 3: Uric acid measurement via finger-prick blood test using the GCU meter.

3. Statistical Analysis

a. Descriptive statistics (frequencies, percentages) summarized participant characteristics.
b. Spearman's rank correlation (non-parametric) assessed the relationship between purine intake and uric acid levels due to skewed data distribution [26].
c. A p-value < 0.05 was considered statistically significant. Analyses were performed using SPSS v26.

E. QUALITY CONTROL AND LIMITATIONS

1. Training: Researchers were standardized in questionnaire administration and GCU meter use.
2. Calibration: The GCU meter was calibrated daily per manufacturer guidelines.
3. Data Verification: 10% of questionnaires were double-entered to ensure accuracy.

This study has several limitations that should be considered. First, the use of self-reported dietary data may introduce recall bias, as participants might not accurately remember or report their food intake. Second, the cross-sectional design with single-time measurements restricts the ability to draw causal inferences between variables. Lastly, the study's focus on a specific geographic region may limit the generalizability of the findings to broader populations with different cultural, dietary, or environmental contexts.

III. RESULTS

A. FREQUENCY DISTRIBUTION OF INDEPENDENT AND DEPENDENT VARIABLES

TABLE 1
Frequency Distribution of Independent and Dependent Variables

Variables	Category	f	%
Purine intake	Normal	23	34,8
	Poor	11	16,7
	High	32	48,5
Uric acid level	Normal	19	28,8
	Low	3	4,5
	High	44	66,7

TABLE 1 shows that the majority of participants consume high-purine foods (48,5%), whereas only a few consume low-purine foods (16,7%). This indicates that mostly purine consumption patterns among participants are unhealthy, and only a few participants understand the impact of purines on their body health. Meanwhile, based on uric acid levels, the majority of participants have high levels of

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Vol. 5 no.3, pp. 120-124, June 2025

uric acid (66.7%), while only a few participants have low levels of uric acid (4,5%). This indicates that high levels of uric acid are a sign of a problem with uric acid metabolism in the body, which can occur due to excess intake or impaired excretion.

B. THE RELATIONSHIP BETWEEN PURINE INTAKE WITH URIC ACID LEVELS

TABLE 2
The Relationship Between Purine Intake with Elevated Uric Acid Level

Purine Intake	Uric Acid Level						p-value; r
	Low		Normal		High		
	f	%	f	%	f	%	
Normal	3	4,5	9	13,6	**11**	**16,7**	0,000; 0,567
Low	0	0	10	15,2	**1**	**1,5**	
High	0	0	0	0	**32**	**48,5**	
n	3	4,5	19	28,8	**44**	**66,7**	

TABLE 2 shows that there is a relationship between purine intake with uric acid levels (p=0.000; r=0.567). The majority of participants who have high purine intake also have high uric acid levels (48.5%), whereas only a few participants with low purine intake also have low uric acid levels (1.5%). This indicates that there is a positive correlation between purine intake with uric acid levels. The enhancing consume of high purine intake could lead to elevated uric acid levels among patients with gout. The sources of high purine intake are especially found in offal, red meat, seafood, as well as a daily intake that is more than the normal baseline (less than 1000 mg per day).

IV. DISCUSSION

A. INTERPRETATION OF RESULTS

The study demonstrated a statistically significant positive correlation between purine intake and serum uric acid levels among individuals with gout (*p* = 0.000, *r* = 0.567). This finding aligns with the biochemical pathway in which purine metabolism generates uric acid as an end product [26]. Notably, 48.5% of participants consumed high-purine diets, predominantly from animal-based sources (e.g., offal, seafood), while 66.7% exhibited hyperuricemia (uric acid >7.0 mg/dL in men, >6.0 mg/dL in women). These results suggest that dietary habits play a critical role in exacerbating gout severity, particularly in populations with limited awareness of purine-rich foods [27].

The strength of the correlation (*r* = 0.567) indicates a moderate to strong relationship, reinforcing the need for dietary interventions in gout management. However, the absence of hyperuricemia in a subset of high-purine consumers (16.7%) implies individual variability in uric acid metabolism, possibly due to genetic factors or renal excretion efficiency [28]. This observation warrants further investigation into non-dietary determinants of hyperuricemia.

B. COMPARISON WITH PRIOR STUDIES

1. Similarities
2. Purine Intake and Hyperuricemia

a. Our findings corroborate multiple studies linking high-purine diets to elevated uric acid. For instance, Zhang et al. [29] reported a 1.5-fold increase in gout attacks with frequent seafood consumption (*p* < 0.01), while Zhou et al. [30] identified red meat as a key hyperuricemia risk factor (OR = 2.1, 95% CI: 1.3–3.4).

b. The dose-dependent relationship observed in our study mirrors results from a meta-analysis by Cheng et al. [31], where purine intake >400 mg/day increased hyperuricemia risk by 34%.

3. Regional Dietary Patterns

Similar to our cohort's preference for offal and seafood, studies in coastal Asian populations (e.g., China, Japan) highlight seafood as a primary purine source [32].

A. Low-Purine Diets

1) Contrary to our results, Chiu et al. [33] found no association between vegetarian diets and uric acid reduction (*p* = 0.12), suggesting that plant-based purines (e.g., spinach, legumes) may have a negligible impact.
2) Discrepancies may stem from differences in food preparation methods (e.g., boiling reduces purine content vs. frying) [34].

B. Genetic and Metabolic Factors

While our study focused on dietary intake, Yokose et al. [35] emphasized ABCG2 gene variants as a major hyperuricemia predictor (AUC = 0.81), indicating that diet alone may not explain all hyperuricemia cases.

C. LIMITATIONS AND IMPLICATIONS

The study's inability to establish causality or track long-term dietary effects limits conclusions about gout progression [36]. Recall bias may have led to underreporting of purine intake, particularly among participants with low health literacy [37]. Restricting the study to a single Indonesian village reduces generalizability to diverse populations with varying dietary habits [38].

Personalized dietary counseling should be integrated into gout management, emphasizing purine-restricted diets for high-risk patients [39]. Community-based nutrition education programs could mitigate gout prevalence in regions with high purine consumption [40]. Longitudinal studies are needed to explore:

a. The temporal relationship between purine intake and uric acid fluctuations.
b. Gene-diet interactions in hyperuricemia development [41].

V. CONCLUSION

This cross-sectional study examined the relationship between dietary purine intake and serum uric acid levels among individuals with gout in a rural Indonesian population. The findings revealed a statistically significant positive correlation ($r = 0.567$, $p = 0.000$), highlighting that participants with high-purine intake particularly from offal, seafood, and red meat tended to exhibit elevated uric acid levels. Specifically, 48.5% of the respondents consumed high-purine diets, while 66.7% demonstrated hyperuricemia, reinforcing the critical role of dietary patterns in the exacerbation of gout. These results contribute valuable empirical evidence to the existing body of knowledge, particularly in the context of regional dietary behaviors in Southeast Asia, which are often underrepresented in global gout research. Despite individual variations in uric acid metabolismlikely influenced by genetic or renal factors the

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findings strongly support the integration of purine-restricted dietary strategies into clinical management and public health programs. This study emphasizes the urgency of implementing community-level interventions, such as culturally adapted nutrition education and personalized dietary counseling, especially in rural settings with limited awareness of purine-related dietary risks. However, the study's cross-sectional nature limits causal inference, and the reliance on self-reported food intake introduces potential recall bias. Future research should adopt longitudinal designs to evaluate the temporal dynamics of purine metabolism, integrate genomic analyses to explore gene-diet interactions, and assess the long-term effectiveness of dietary interventions in reducing hyperuricemia and improving quality of life among gout patients.

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DATA AVAILABILITY

No datasets were generated or analyzed during the current study.

AUTHOR CONTRIBUTION

Y.A. conceptualized the study, designed the methodology, and supervised data collection. H.H. contributed to data analysis, interpretation, and manuscript drafting. S.N.T. assisted in literature review, statistical validation, and manuscript revision. All authors reviewed and approved the final version of the paper.

DECLARATIONS

ETHICAL APPROVAL

This study received ethical approval from STIKES Rustida (No. 283/03/KEPK-STIKESBWI/VII/2024). Informed consent was obtained from all participants. The authors declare no conflicts of interest. No external funding was received for this research. Data supporting the findings are available from the corresponding author upon reasonable request.

CONSENT FOR PUBLICATION PARTICIPANTS.

Consent for publication was given by all participants

COMPETING INTERESTS

The authors declare no competing interests.

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