

Received	2025/09/10	تم استلام الورقة العلمية في
Accepted	2025/10/09	تم قبول الورقة العلمية في
Published	2025/10/12	تم نشر الورقة العلمية في

Correlation between serum uric acid and lipid profile in patients with type 2 diabetes mellitus

Omar B. Latiwesh¹; Hajir Othman²; Abdelfatah M. Elmahdawi¹;
Walid A. Augi¹; Hend A. Alomamy¹

1. College of Medical Technology-Benghazi, Libyan Ministry of
Technical and Vocational Education. Libya

2. Faculty of Dentistry, University of Benghazi. Libya

Email: omarbelhasan@gmail.com

Abstract

SUA is an independent risk factor for cardiovascular disease, and other type 2 diabetes related chronic complications. Patients with type 2 diabetes mellitus face a higher risk of cardiovascular disease. Diabetic dyslipidemia is a key risk factor for cardiovascular disease (CVD) and contributes to atherosclerosis. This study was conducted to examine the relationship between SUA and dyslipidemia in patients with type 2 diabetes mellitus to understand their roles in cardiovascular disease. **Subjects and methods:** The study included 103 patients with type 2 diabetes mellitus and 35 healthy individuals. Clinical information and medical history were obtained through a questionnaire that included variables such as age, sex, and others. Blood samples were collected from all participants and analyzed for FBG, SUA, TG, TC, LDL-C, and HDL-C. The data were analyzed statistically using SPSS. **Results:** SUA was higher in the diabetic group compared to the control group, but the difference was not statistically significant. SUA showed a significant positive relationship with TG and BMI. There was also a significant inverse relationship between SUA and both FBS and HDL-C. No significant correlation was found between SUA and TC or LDL-C. **Conclusion:** In T2DM patients with normal kidney function, serum uric acid has a strong association with the lipid profile, especially triglycerides. Because high serum uric acid can intensify the occurrence of cardiovascular diseases and other complications of

type 2 diabetes, it must be monitored and managed along with the lipid profile and blood glucose to reduce the risk of chronic diabetes complications.

Keywords: Serum uric acid, type 2 diabetes mellitus, cardiovascular disease. Triglyceride, Glucose, cholesterol.

الارتباط بين حمض اليوريك والدهون في الدم لدى مرضى السكري من النوع الثاني

عمر بالحسن الأطيوش¹، هاجر يوسف العوامي²، عبدالفتاح مفتاح المهدي¹، هند عوض
العمامي¹

1. كلية التقنية الطبية – بنغازي، وزارة التعليم التقني والفني. - ليبيا

2. كلية طب وجراحة الفم والأسنان – جامعة بنغازي – ليبيا

الإيميل: omarbelhasam@gmail.com

الملخص:

يعتبر حمض اليوريك عامل خطر لأمراض القلب والأوعية الدموية وللأمراض الأخرى المرتبطة بداء السكري من النوع الثاني. يعاني مرضى داء السكري من النوع 2 من خطر أعلى للإصابة بأمراض القلب والأوعية الدموية، ويُعد الخلل في دهون الدم عامل خطر رئيسي لأمراض القلب والأوعية الدموية كما يساهم في تصلب الشرايين. أجريت هذه الدراسة لفحص العلاقة بين حمض اليوريك والخلل في دهون الدم لدى مرضى داء السكري من النوع الثاني وكذلك لفهم مدى مساهمة كل منهما في أمراض القلب والأوعية الدموية. **العينات:** شملت الدراسة 103 مرضى مصابين بداء السكري من النوع الثاني و 35 فردًا سليمًا. تم الحصول على المعلومات السريرية والتاريخ الطبي من خلال استبيان تضمن متغيرات مثل العمر والجنس وغيرها. تم جمع عينات الدم من جميع المشاركين وتحليلها من حيث FBG و SU و TG و TC و LDL-C و HDL-C. تم تحليل البيانات إحصائيًا باستخدام SPSS. **النتائج:** كان SUA أعلى في مجموعة مرضى السكري مقارنة بالمجموعة الضابطة، ولكن الفرق لم يكن ذا دلالة إحصائية. أظهر SUA علاقة إيجابية كبيرة مع TG ومؤشر كتلة الجسم. كما وُجدت علاقة عكسية مهمة بين حمض اليوريك في الدم (SUA) وكلٍّ من FBS و HDL-C. ولم يُعثر على أي ارتباط مهم بين

حمض اليوريك في الدم (SUA) و TC أو LDL-C. **الخلاصة:** لدى مرضى السكري من النوع الثاني ذوي وظائف الكلى الطبيعية، يرتبط حمض اليوريك في الدم ارتباطاً وثيقاً بمستوى الدهون، وخاصةً الدهون الثلاثية. ولأن ارتفاع حمض اليوريك في الدم قد يُفاقم حدوث أمراض القلب والأوعية الدموية ومضاعفات داء السكري من الأخرى، يجب مراقبته وإدارته بالتزامن مع مستوى الدهون ومستوى سكر الدم للحد من خطر مضاعفات داء السكري المزمنة.

الكلمات الدالة: مرض السكري النوع الثاني، حمض اليوريك، أمراض القلب والأوعية الدموية، الجلوكوز، الدهون الثلاثية، الكوليسترول.

Introduction

Uric acid (UA) is the final product of purine nucleotide breakdown. The purine degradation pathway in humans is shorter than in other primates. As a result, humans have higher levels of UA in the blood and are more prone to hyperuricemia and gout. Numerous studies have identified serum uric acid (SUA) as an independent risk factor for cardiovascular disease, and it is closely linked to various components of metabolic syndrome, including obesity, hyperglycemia, dyslipidemia, and insulin resistance (Katsiki, Dimitriadis, and Mikhailidis 2021; Saito et al. 2021).

Over recent decades, type 2 diabetes mellitus has become an epidemic and one of the major public health challenges of the 21st century. According to the International Diabetes Federation (IDF 2019), the global prevalence of T2DM is expected to reach 642 million by 2040. Type 2 diabetes is associated with various complications and co-morbidities that can result in cardiovascular diseases, nephropathy, retinopathy, and neuropathy. These long-term complications are responsible for most deaths among people with type 2 diabetes. Therefore, it is essential to identify clinical biomarkers that can aid in the early detection and management of diabetic long-term complications. (Hu et al. 2021).

Patients with type 2 diabetes mellitus have a 2 to 4-fold higher risk of cardiovascular disease compared to those without diabetes. This increased risk cannot be explained solely by hyperglycemia, as other factors like hypertension and dyslipidemia also significantly contribute to the development of CVD in people with type 2 diabetes mellitus. (Stamler et al. 1993; Stern 1997).

Dyslipidemia is a major risk factor for cardiovascular disease (CVD) and contributes to atherosclerosis. It occurs twice as often in diabetic patients compared to the general population. Diabetic dyslipidemia is characterized by high triglycerides (TG), normal or slightly elevated low-density lipoprotein cholesterol (LDL-c), and reduced high-density lipoprotein cholesterol (HDL-c) levels. (Athysos 2011).

Elevated serum uric acid levels are significantly associated with dyslipidemia in non-diabetic, otherwise healthy individuals. Furthermore, conditions such as hypertriglyceridemia and mixed hyperlipidemia are directly linked to the development and progression of hyperuricemia (Ali et al. 2018; Fang et al. 2024).

Few studies have been conducted to evaluate the relationship between hyperuricemia and dyslipidemia in patients with type 2 diabetes mellitus, especially in Mediterranean and Arab countries. However, many studies have shown a significantly positive correlation between hyperuricemia and dyslipidemia in type 2 diabetic patients (Fayazi et al. 2023; Hu et al. 2021; Kumar et al. 2022).

To date, no study has been conducted in our country to examine the correlation between hyperuricemia and dyslipidemia in the diabetic population; therefore, our study was undertaken to evaluate serum uric acid in patients with type 2 diabetes mellitus and to explore the relationship between serum uric acid, and triglycerides, total cholesterol, HDL-cholesterol, and LDL-cholesterol, Fasting blood sugar, and body mass index.

Subjects and Methods:

This case-control study included 103 patients with type 2 diabetes mellitus (48 males and 55 females) and 35 healthy individuals (20 males and 15 females). The patients were recruited from the Center for Diagnosis and Treatment of Diabetes in Benghazi between November 2024 and February 2025. The healthy participants were recruited from the general population attending Alakeed Medical Laboratory.

Informed consent was obtained from all participants before the study. The diagnosis of diabetes was determined based on a previous history and in accordance with the American Diabetes Association criteria (2006), which include an A1c level of $\geq 6.5\%$, a fasting

plasma glucose level of ≥ 126 mg/dL, or a 2-hour plasma glucose level of ≥ 200 mg/dL during an oral glucose tolerance test.

Clinical information and medical history were obtained through the review of patient medical files and patient interviews. Face-to-face interviews were conducted using a questionnaire that included variables such as age, sex, date of diagnosis, physical activity level, adherence to a diet, and any existing health problems or prescriptions. The height and weight were measured, and obesity was defined as a body mass index (BMI) of 30 kg/m^2 or greater, where BMI was calculated by dividing the weight in kilograms by the height in meters squared.

In this study, we first measured the blood creatinine levels of the patients to exclude those with pre-existing kidney disease. All patients had stable metabolic conditions. Patients with any condition that could influence their metabolic status or the parameters studied, such as nephrotic syndrome, acute or chronic renal failure, anemia, other hematological disorders, cardiovascular diseases, malignancy, arthritis, inherited or genetic dyslipidemias, inflammatory conditions, gout, or cerebrovascular diseases, were excluded from the study. We also excluded patients with a history of smoking or alcohol consumption. Additionally, pregnant and lactating women were excluded from the study.

The medication history was documented, and patients using any drugs that could influence serum uric acid or lipid profile levels, such as insulin, statins, fibrates, or antihyperlipidemic medications, were also excluded. The control group consisted of healthy subjects who were not suffering from any acute infection, metabolic, or psychological disorder. They were non-smokers and not overweight. They had no history of inherited hyperuricemia or diabetes mellitus.

Venous blood samples were collected from all participants after at least 10 hours of fasting, and sera were separated from plain tubes and stored at -20°C until analysis. Serum samples were analyzed for fasting blood sugar, uric acid, total cholesterol (TC), triglycerides (TG), HDL-cholesterol, LDL-cholesterol, and

creatinine using a fully automated COBAS INTEGRA 400 plus (Roche, Germany).

The data were analyzed using the Statistical Package for the Social Sciences (SPSS) version 22. We divided the diabetic patients into two groups based on the cutoff SUA level of 5.5 mg/dL, and an independent sample t-test was performed to measure the differences between the two groups. Pearson's correlation coefficient was estimated to determine the degree of association between uric acid and other biochemical parameters. P value < 0.05 was considered significant.

Results:

This cross-sectional study included 103 patients with type 2 diabetes mellitus (48 males and 55 females) with a mean age and SD of 51 ± 11 , and 35 healthy individuals (20 males and 15 females) with a mean age and SD of 39 ± 15 .

As shown in Table 1, body mass index, fasting blood sugar, total cholesterol, LDL cholesterol, and triglyceride levels were significantly higher in the diabetic group compared to the control group. Meanwhile, HDL cholesterol concentration was higher in the control group than in the diabetic group, and this difference was statistically significant. SUA was higher in the diabetic group compared to the control group, but the difference was not statistically significant.

We divided the diabetic patients into two groups based on a SUA cutoff level of 5.5 mg/dL. We observed significant differences in triglycerides, HDL-C, and FBS between these groups. However, the differences in total cholesterol and LDL-C were not statistically significant, as shown in Table 2.

Table .1. Independent Samples T-Test Comparing the Diabetic Group and the Control Group

	Type 2 Diabetic Group	Healthy Control Group	P Value*
BMI (Kg/m ²)	31.2 ±6	24.9±4	0.00
FBS (mg/dl)	170.4±74	88 ±9	0.00
TG (mg/dl)	135±60	52±10	0.00
TC (mg/dl)	191±42	106±29	0.00
LDL-C (mg/dl)	177±37	107±28	0.00
HDL-C (mg/dl)	43.1±15	93.2±53	0.00
SUA (mg/dl)	4.6±1.4	4.2±0.9	0.22

BMI: Body Mass Index. FBS: Fasting Blood Sugar. TG: Triglycerides. TC: Total Cholesterol. LDL-C: Low Density Lipoprotein Cholesterol. HDL-C: High Density Lipoprotein Cholesterol. SUA: Serum Uric Acid. * P value < 0.05 is considered significant

Table .2. Independent Sample T-Test comparing diabetic patients grouped by SUA cutoff level of 5.5 mg/dL.

	Diabetic patients with SUA ≤ 5.5 (N= 56)	Diabetic patients with SUA > 5.5 (N= 47)	P Value*
BMI (Kg/m ²)	30.8±6	33.1±6	0.001
FBS (mg/dl)	178.6±82.5	149.3±41.5	0.015
TG (mg/dl)	126.6±56	159.1±63	0.000
TC (mg/dl)	191.8±36	187±53.8	0.924
LDL-C (mg/dl)	104.4±25.7	113±31.5	0.664
HDL-C (mg/dl)	45.8±16	36±7	0.010

BMI: Body Mass Index. FBS: Fasting Blood Sugar. TG: Triglycerides. TC: Total Cholesterol. LDL-C: Low Density Lipoprotein Cholesterol. HDL-C: High Density Lipoprotein Cholesterol. * P value < 0.05 is considered significant. N: number of cases.

Pearson correlation analysis in the diabetic group revealed a significant positive relationship between SUA and both TG ($r=0.350$, $P=0.000$) and BMI ($r=0.362$, $P=0.001$) as depicted in Figures 1 and 2. There was also a significant inverse relationship between SUA and both FBS ($r= -238$, $P= 0.015$) and HDL-C ($r= -252$, $P= 0.010$), as shown in Figures 3 and 4. No significant correlation was found between SUA and TC or LDL-C.

<http://www.doi.org/10.62341/ohaw0021>

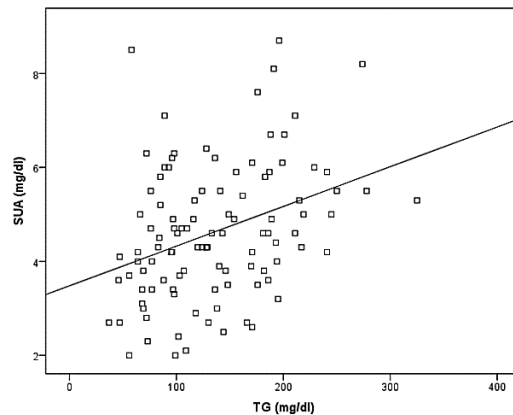


Fig.1. Correlation between SUA and TG.

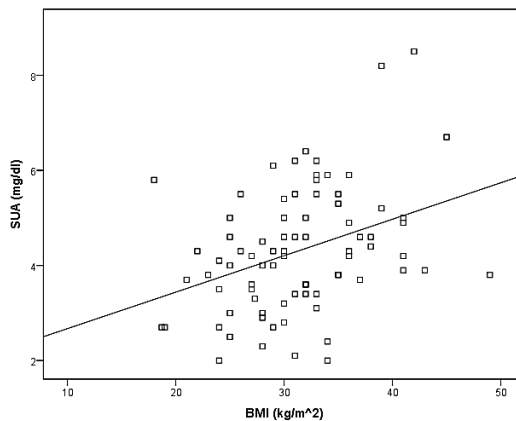


Fig.2. Correlation between SUA and BMI.

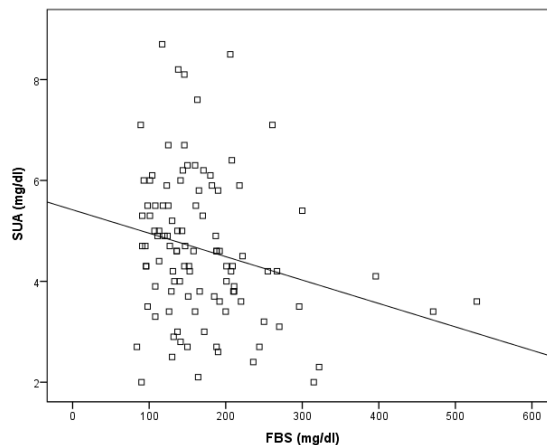


Fig.3. Correlation between SUA and FBS.

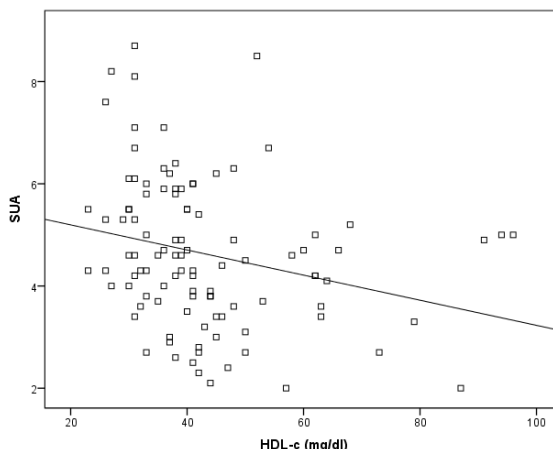


Fig.4. Correlation between SUA and HDL-C.

Discussion:

The present study observed higher serum uric acid (SUA) levels in the diabetic group compared to the control group, although this difference was not statistically significant. Moreover, in the diabetic group, SUA level showed a significant positive correlation with triglycerides (TG) and body mass index (BMI). In contrast, a significant negative correlation was found between SUA and both fasting blood sugar (FBS) and high-density lipoprotein cholesterol (HDL-C).

Many studies have shown high serum uric acid levels in patients with type 2 diabetes mellitus (Gill et al., 2013; Hussain et al., 2018; Khare et al., 2015), Whereas other studies have revealed lower serum uric acid levels in diabetic patients (Patel, Singh, and Kahlon 2024).

The mechanisms relating SUA to T2DM are not fully understood. Animal studies indicate that hyperuricemia may affect endothelial function by reducing nitric oxide (NO) bioavailability. Because insulin depends on NO to promote glucose uptake, this could impair glucose absorption. Therefore, hyperuricemia may significantly contribute to the development or progression of insulin resistance (Sluijs et al. 2013).

The role of insulin in uric acid metabolism is complex. Hyperinsulinemia can activate the hexose monophosphate pathway,

leading to increased purine synthesis and enhanced uric acid production. It can also promote the reabsorption of uric acid in the kidneys by stimulating the urate anion transporter in the proximal tubular brush border membrane, leading to higher SUA levels. On the contrary, high insulin levels typically result in lower blood glucose levels. This dual action enables insulin to regulate both uric acid and blood glucose levels simultaneously. Hence, this explains the inverse relationship between uric acid and blood glucose in the presence of hyperinsulinemia. (Fox 1981; Modan et al. 1987; Tsunoda et al. 2002)

In the diabetic group, Serum uric acid showed an inverse relationship with FBG, and this finding is aligned with the results of many studies (Nan et al. 2007; Patel et al. 2024; Xu et al. 2019). Other studies do not show any association between uric acid and FBG. (Satishkumar Modi and Sahi 2018). The inverse relationship between SUA and FBG observed in these diabetic subjects may be caused by increased renal excretion of uric acid in the presence of hyperglycemia.

Glucose and uric acid are both reabsorbed in the proximal renal tubules, so their reabsorption mechanisms may overlap. When glucose levels are high, as in type 2 diabetes mellitus, it can compete with uric acid for reabsorption, resulting in increased uric acid excretion in the urine. This is supported by the fact that both substances use similar transporters in the proximal tubules for reabsorption. Specifically, uric acid is reabsorbed through urate or anion exchanger and voltage-sensitive urate channel, and glucose is reabsorbed through similar mechanisms in the same region of the kidney. (Patel et al., 2024).

SUA was positively correlated with the body mass index of diabetic patients, and the correlation was statistically significant. This finding is consistent with that of Tanaka et al. (2015), who indicate that body mass index was significantly correlated with SUA levels in Japanese adult twins of both genders, after adjusting for genetic and environmental factors. Another study by Wang et al. (2014), revealed a positive association between BMI and SUA levels among healthy individuals in Jiangsu Province, China.

Obesity is implicated in raising SUA levels through two factors: overproduction and reduced renal excretion. A study conducted in patients with visceral fat obesity has revealed that elevated uric acid levels are strongly influenced by its overproduction, along with a decrease in urinary urate excretion. Moreover, the accumulation of visceral fat promotes free fatty acids influx into the liver, which stimulates the synthesis of triglycerides, followed by an associated increase in uric acid production through the activation of the uric acid synthesis pathway.. (Fabregat et al., 1987; Matsuura et al., 1998).

Our study found a strong positive correlation between SUA and TG, and a strong negative correlation between SUA and HDL-C. These findings exactly align with those of similar studies examining the relationship between SUA and dyslipidemia in patients with type 2 diabetes mellitus (Fayazi et al. 2023; Hu et al. 2021; Kumar et al. 2022). A study by Fayazi et al. (2023), revealed a significant negative relationship only between SUA and TG. In contrast, a study by Ali et al. (2019), indicated a significant positive correlation between SUA and TG, TC, and LDL, while an inverse correlation was observed between SUA and HDL.

Diabetic dyslipidemia contributes to the increased production of purine and its final catabolic product, uric acid (De Oliveira and Burini 2012). The relationship between higher SUA and dyslipidemia can be explained by the fact that higher central obesity is linked to insulin resistance and increased leptin production. These two factors reduce uric acid excretion. Along with this, increased production of TG accelerates the de novo synthesis of ribose-5-phosphate to phosphoribosyl pyrophosphate through the metabolic pathway nicotinamide adenine dinucleotide phosphate (NADPH), which in turn, increases uric acid production (Matsuura et al. 1998). Dyslipidemia can lead to the overactivity of xanthine oxidoreductase (XOR), a key enzyme in purine metabolism. This not only promotes the production of uric acid and reactive oxygen species (ROS), but also triggers oxidative stress. This oxidative stress could intensify the dyslipidemia, worsen insulin resistance,

and initiate a chain of metabolic changes within the body (Chen et al. 2013; Furuhashi 2020).

The limitations of the present study include the absence of information about urinary UA, so we cannot observe the effect of diabetes or dyslipidemia on the handling and excretion of UA, and the small sample size, which weakens the study's ability to represent the entire population. Moreover, the cross-sectional design cannot prove cause-and-effect relationships between SUA and dyslipidemia. Further studies with larger sample sizes, utilizing more sophisticated laboratory techniques, and advanced statistical software could provide more precise insights into the correlation between SUA and dyslipidemia in patients with type 2 diabetes mellitus.. gathering more information about the patients hypoglycemic medications, and conducting laboratory analyses of urinary uric acid could also provide a more clear understanding of the relationship between SUA and dyslipidemia in those patients.

Conclusion

In T2DM patients with normal kidney function, serum uric acid has a strong association with the lipid profile, especially triglycerides. Because high serum uric acid can intensify the occurrence of cardiovascular diseases and other complications of type 2 diabetes, it must be monitored and managed along with the lipid profile and blood glucose to reduce the risk of chronic diabetes complications.

References:

- Ali, Nurshad, Rasheda Perveen, Shahnaz Rahman, Shakil Mahmood, Sadaqur Rahman, Shiful Islam, Tangigul Haque, Abu Hasan Sumon, Rahanuma Raihanu Kathak, Noyan Hossain Molla, Farjana Islam, Nayan Chandra Mohanto, Shaikh Mirja Nurunnabi, Shamim Ahmed, and Mustafizur Rahman. 2018. "Prevalence of Hyperuricemia and the Relationship between Serum Uric Acid and Obesity: A Study on Bangladeshi Adults." *PLoS ONE* 13(11):e0206850. doi:10.1371/JOURNAL.PONE.0206850.

- Ali, Nurshad, Sadaqur Rahman, Shiful Islam, Tangigul Haque, Noyan Hossain Molla, Abu Hasan Sumon, Rahanuma Raihanu Kathak, Md Asaduzzaman, Farjana Islam, Nayan Chandra Mohanto, Mohammad Abul Hasnat, Shaikh Mirja Nurunnabi, and Shamim Ahmed. 2019. "The Relationship between Serum Uric Acid and Lipid Profile in Bangladeshi Adults." *BMC Cardiovascular Disorders* 19(1):42. doi:10.1186/S12872-019-1026-2.
- Athyros, Vasilios G. 2011. "Dyslipidaemia of Obesity, Metabolic Syndrome and Type 2 Diabetes Mellitus: The Case for Residual Risk Reduction After Statin Treatment." *The Open Cardiovascular Medicine Journal* 5(1):24–34. doi:10.2174/1874192401105010024,.
- Chen, Jiunn Horng, Wen Harn Pan, Chih Cheng Hsu, Wen Ting Yeh, Shao Yuan Chuang, Pin Yu Chen, Hui Chen Chen, Chwen Tzuei Chang, and Wei Lun Huang. 2013. "Impact of Obesity and Hypertriglyceridemia on Gout Development with or without Hyperuricemia: A Prospective Study." *Arthritis Care and Research* 65(1):133–40. doi:10.1002/ACR.21824,.
- Fabregat, Isabel, Elisa Revilla, and Alberto Machado. 1987. "Short-Term Control of the Pentose Phosphate Cycle by Insulin Could Be Modulated by the NADPH NADP Ratio in Rat Adipocytes and Hepatocytes." *Biochemical and Biophysical Research Communications* 146(2):920–25. doi:10.1016/0006-291X(87)90618-8,.
- Fang, Yicheng, Wendan Mei, Chenxu Wang, Xia Ren, Jian Hu, Fan Su, Lei Cao, Grace Tavengana, Mingfei Jiang, Huan Wu, and Yufeng Wen. 2024. "Dyslipidemia and Hyperuricemia: A Cross-Sectional Study of Residents in Wuhu, China." *BMC Endocrine Disorders* 24(1):2. doi:10.1186/S12902-023-01528-7.
- Fayazi, Haniyeh Sadat, Seyyedeh Sahereh Mortazavi Khatibani, Behrang Motamed, and Maryam Yaseri. 2023. "Evaluation of Levels of Uric Acid and Lipid Profile in Hospitalized Patients with Diabetes." *BMC Research Notes* 16(1). doi:10.1186/S13104-023-06429-5,.

- Fox, Irving H. 1981. "Metabolic Basis for Disorders of Purine Nucleotide Degradation." *Metabolism* 30(6):616–34. doi:10.1016/0026-0495(81)90142-6,.
- Furuhashi, Masato. 2020. "New Insights into Purine Metabolism in Metabolic Diseases: Role of Xanthine Oxidoreductase Activity." *American Journal of Physiology - Endocrinology and Metabolism* 319(5):E827–34. doi:10.1152/AJPENDO.00378.2020,.
- Gill, Anju, Sahiba Kukreja, Naresh Malhotra, and Namrata Chhabra. 2013. "Correlation of the Serum Insulin and the Serum Uric Acid Levels with the Glycated Haemoglobin Levels in the Patients of Type 2 Diabetes Mellitus." *Journal of Clinical and Diagnostic Research : JCDR* 7(7):1295. doi:10.7860/JCDR/2013/6017.3121.
- Hu, Yimeng, Qinge Li, Rui Min, Yingfeng Deng, Yancheng Xu, and Ling Gao. 2021. "The Association between Serum Uric Acid and Diabetic Complications in Patients with Type 2 Diabetes Mellitus by Gender: A Cross-Sectional Study." *PeerJ* 9. doi:10.7717/PEERJ.10691,.
- Hussain, Azhar, Omar B. Latiwesh, Farwa Ali, Mustafa Y. G. Younis, and Jamal A. Alammari. 2018. "Effects of Body Mass Index, Glycemic Control, and Hypoglycemic Drugs on Serum Uric Acid Levels in Type 2 Diabetic Patients." *Cureus* 10(8):e3158. doi:10.7759/CUREUS.3158.
- IDF. 2019. "IDF Diabetes Atlas 2019." *International Diabetes Federation* 1. <http://www.idf.org/about-diabetes/facts-figures>.
- Katsiki, Niki, George D. Dimitriadis, and Dimitri P. Mikhailidis. 2021. "Serum Uric Acid and Diabetes: From Pathophysiology to Cardiovascular Disease." *Current Pharmaceutical Design* 27(16):1941–51. doi:10.2174/1381612827666210104124320,.
- Khare, Shivam, Jogesh Kumar Vishandasani, and Archana Kansal. 2015. "To Study Serum Uric Acid In Type 2 Diabetes Mellitus Patient." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS) e-ISSN* 14(9):82–86. doi:10.9790/0853-14938286.
- Kumar, Sandeep, Himel Mondal, Manju Lata, Joshil Kumar Behera, and Bhagyajyoti Priyadarshini. 2022. "Correlation of Serum Uric

- Acid with Lipid Profile in Patients with Type 2 Diabetes Mellitus with Normal Creatinine Level: Report from a Tertiary Care Hospital in India.” *Journal of Family Medicine and Primary Care* 11(6):3066. doi:10.4103/JFMPC.JFMPC_2131_21.
- Matsuura, Fumihiko, Shizuya Yamashita, Tadashi Nakamura, Makoto Nishida, Shuichi Nozaki, Tohru Funahashi, and Yuji Matsuzawa. 1998. “Effect of Visceral Fat Accumulation on Uric Acid Metabolism in Male Obese Subjects: Visceral Fat Obesity Is Linked More Closely to Overproduction of Uric Acid than Subcutaneous Fat Obesity.” *Metabolism: Clinical and Experimental* 47(8):929–33. doi:10.1016/S0026-0495(98)90346-8,.
- Modan, M., H. Halkin, A. Karasik, and A. Lusky. 1987. “Elevated Serum Uric Acid - a Facet of Hyperinsulinaemia.” *Diabetologia* 30(9):713–18. doi:10.1007/BF00296994,.
- Nan, Hairong, Yanhu Dong, Weiguo Gao, Jaakko Tuomilehto, and Qing Qiao. 2007. “Diabetes Associated with a Low Serum Uric Acid Level in a General Chinese Population.” *Diabetes Research and Clinical Practice* 76(1):68–74. doi:10.1016/j.diabres.2006.07.022.
- De Oliveira, Erick Prado, and Roberto Carlos Burini. 2012. “High Plasma Uric Acid Concentration: Causes and Consequences.” *Diabetology and Metabolic Syndrome* 4(1). doi:10.1186/1758-5996-4-12,.
- Patel, Seema, Mitasha Singh, and Namrata Kahlon. 2024. “Association of Serum Uric Acid Levels with Glycated Haemoglobin in Diabetic Patients and Healthy Controls.” *Journal of Family Medicine and Primary Care* 13(11):5040–46. doi:10.4103/JFMPC.JFMPC_777_24.
- Saito, Yuichi, Atsushi Tanaka, Koichi Node, and Yoshio Kobayashi. 2021. “Uric Acid and Cardiovascular Disease: A Clinical Review.” *Journal of Cardiology* 78(1):51–57. doi:10.1016/j.jjcc.2020.12.013.
- Satishkumar Modi, Anuj, and Nita Sahi. 2018. “Serum Uric Acid Levels in Type 2 Diabetes Mellitus.” *Indian Journal of Basic and Applied Medical Research* 7:459. www.ijbamr.comP.

- Sluijs, Ivonne, Joline W. J. Beulens, Daphne L. Van Der A, Annemieke M. W. Spijkerman, Matthias B. Schulze, and Yvonne T. Van Der Schouw. 2013. "Plasma Uric Acid Is Associated with Increased Risk of Type 2 Diabetes Independent of Diet and Metabolic Risk Factors." *Journal of Nutrition* 143(1):80–85. doi:10.3945/jn.112.167221.
- Stamler, Jeremiah, Olga Vaccaro, James D. Neaton, and Deborah Wentworth. 1993. "Diabetes, Other Risk Factors, and 12-Yr Cardiovascular Mortality for Men Screened in the Multiple Risk Factor Intervention Trial." *Diabetes Care* 16(2):434–44. doi:10.2337/DIACARE.16.2.434,.
- Stern, M. P. 1997. "Glycemia and Cardiovascular Risk." *Diabetes Care* 20(10):1501–2. doi:10.2337/DIACARE.20.10.1501.
- Tanaka, Kentaro, Soshiro Ogata, Haruka Tanaka, Kayoko Omura, Chika Honda, and Kazuo Hayakawa. 2015. "The Relationship between Body Mass Index and Uric Acid: A Study on Japanese Adult Twins." *Environmental Health and Preventive Medicine* 20(5):347. doi:10.1007/S12199-015-0473-3.
- Tsunoda, Sei, Kei Kamide, Junichi Minami, and Yuhei Kawano. 2002. "Decreases in Serum Uric Acid by Amelioration of Insulin Resistance in Overweight Hypertensive Patients: Effect of a Low-Energy Diet and an Insulin-Sensitizing Agent." *American Journal of Hypertension* 15(8):697–701. doi:10.1016/S0895-7061(02)02953-9,.
- Wang, Honggang, Lizhen Wang, Rui Xie, Weijie Dai, Chengcheng Gao, Peng Shen, Xiaodan Huang, Faming Zhang, Xiaozhong Yang, and Guozhong Ji. 2014. "Association of Serum Uric Acid with Body Mass Index: A Cross-Sectional Study from Jiangsu Province, China." *Iranian Journal of Public Health* 43(11):1503. <https://pmc.ncbi.nlm.nih.gov/articles/PMC4449499/>.
- Xu, Linxin, Ting Li, Jianhong Yin, Gang Lin, Yali Xu, Yi Ren, Yan Wang, Jing Yang, and Liming Chen. 2019. "Association between Serum Uric Acid and Nonalcoholic Fatty Liver Disease in Community Patients with Type 2 Diabetes Mellitus." *PeerJ* 2019(8):e7563. doi:10.7717/PEERJ.7563/SUPP-1.

