Jurnal Ilmiah Mandala Education (JIME)
Vol. 8, No. 2, April 2022
p-ISSN : 2442-9511, e-2656-5862
DOI:10.36312/jime.v8i2.3035/http://ejournal.mandalanursa.org/index.php/JIME

Association of Body Mass Index with The Risk Of Gout Arthritis in Male and Female with Underweight, Normal Weight, Overweight, Obese

Slamet Raharjo[1], Olivia Andiana[2*]
[1,2]Department of Sport Science, Faculty of Sport Science, State University of Malang, Malang, Indonesia

Article Info
Article history:
Accepted: 21 Febuari 2022
Publish: 03 April 2022

Keywords:
Body mass index
gout arthritis
uric acid
hyperuricemia
adolescents

ABSTRAK
Penelitian ini bertujuan untuk menganalisis hubungan antara indeks massa tubuh (IMT) underweight, normal weight, overweight, obese dengan risiko kejadian gout arthritis pada remaja laki-laki dan perempuan Kota Malang. Penelitian ini menggunakan metode korelasional dengan pendekatan cross-sectional study. Sebanyak 101 remaja yang terdiri dari 59 laki-laki dan 42 perempuan, usia 16 – 23 tahun, IMT underweight, normal weight, overweight, dan obese ikut berpartsisipasi dalam penelitian ini. Kadar asam urat diukur menggunakan Easy Touch (Easy Touch, Hsinchu, Taiwan) dengan satuan konsentrasi mg/dL, sedangkan IMT dihitung dengan cara berat badan (BB) (kg) dibagi tinggi badan (TB) (m^2). Pearson's correlation coefficient digunakan untuk menganalisis hubungan antara IMT dengan kadar asam urat. Hasil penelitian menunjukkan rata-rata IMT underweight (17.64 ± 0.26 kg/m^2), normal weight (21.94 ± 1.33 kg/m^2), overweight (25.81 ± 0.51 kg/m^2), obese (29.92 ± 2.80 kg/m^2), dan kadar asam urat (6.09 ± 1.61 mg/dL) dengan (r = 0.838; $p \leq 0.001$). Berdasarkan hasil penelitian disimpulkan terdapat hubungan positif antara IMT dengan kadar asam urat. IMT menunjukkan korelasi positif yang kuat dengan kadar asam urat.

Article Info
Article history:
Diterima: 21 Febuari 2022
Terbit: 03 April 2022

Abstract
The current study aims to analyze the correlation between body mass index (BMI) in underweight, normal weight, overweight, obese with the risk of gout arthritis in male and female adolescents in Malang. We conduct a correlational with a cross-sectional study approach. A total of 101 adolescents consisting of 59 men and 42 women, aged 16 – 23 years, with body mass index in categories underweight, normal weight, overweight, and obese are participated in this study. Uric acid levels were measured using Easy Touch (Easy Touch, Hsinchu, Taiwan) with a concentration unit of mg/dL, while BMI was calculated by dividing body weight (BW) (kg) with body height (BH) (m^2). Pearson's correlation coefficient was used to analyze the correlation between BMI and uric acid levels. The results showed that the mean BMI was underweight (17.64 ± 0.26 kg/m^2), normal weight (21.94 ± 1.33 kg/m^2), overweight (25.81 ± 0.51 kg/m^2), obese (29.92 ± 2.80 kg/m^2), and levels of uric acid (6.09 ± 1.61 mg/dL) with (r = 0.838; $p \leq 0.001$). Based on the results of the study, it was concluded that there was a positive correlation between BMI and uric acid levels. BMI showed a strong positive correlation with uric acid levels.

Corresponding Author:
Olivia Andiana
Department of Sport Science, Faculty of Sport Science, State University of Malang
Email: olivia.andiana.fik@um.ac.id

1. INTRODUCTION

Hyperuricemia is a condition of uric acid levels in the blood that are more than 7.0 mg/dL (Nasrul, 2012) with an estimated incidence of 8.9% to 24.4% of the world population (Skoczyńska et al., 2020; Benn et al., 2018; Perez-Ruiz et al., 2015). Gout arthritis is an inflammatory joint condition caused by monosodium urate crystals that deposit in distal joints and peripheral tissues (Butler et al., 2021). Susceptibility in monosodium urate crystals formation due to excessive levels of uric acid in the blood, one of the purine nucleotide breakdown end products (Martillo et al., 2014; Choi et al., 2005). Gout forms due to an increase in uric acid levels in the blood (hyperuricemia) (Roddy & Doherty, 2010). The accumulation of monosodium urate crystals in joints causes local inflammation (Almatsier, 2006).

In the current era, the rapidly increased prevalence of patients with hyperuricemia. Emerging evidence suggests that the prevalence of hyperuricemia is not only in developed countries, but is also increasing in low-income and middle-income (Kanwar & Kabra, 2016; Zhu, 2011). The results by Wurangian (2014) reported that from 54 students with obesity, the prevalence of obese students who experienced hyperuricemia in Minahasa Regency was 8 people (14.82%) consisting of 3 male students (5.56%) and 5 female students (9.26%). Despite most of the obese students having normal blood uric acid levels, some of them showed hyperuricemia. Hyperuricemia has the potential for gout arthritis following the results of the study by Budiono (2016) which reported that there was a relationship between hyperuricemia and the incidence of gout arthritis in both men and women.

Non-modifiable risk factors include age and gender, while modifiable risk factors include lifestyle and other diseases such as diabetes mellitus (DM), hypertension, making these individuals have a greater risk of developing gout arthritis (Singh et al. al., 2017). In addition, an increase in uric acid levels is triggered by an increase in body mass index (BMI), waist circumference, and dyslipidemia (Tanaka et al., 2015). Results by Anggraini et al. (2018) show that respondents who experience gouty arthritis have a history of hypertension and obesity on average. According to Rosdiana et al. (2018), uric acid levels decrease along with weight loss. On the contrary, the increase in uric acid levels is triggered by an increase in BMI, waist circumference, and dyslipidemia (Wang et al., 2014). However, the relationship between BMI categories underweight, normal weight, overweight, and obese with the risk of developing gouty arthritis in adolescent boys and girls are still not clearly understood.

2. RESEARCH METHODS

We conduct a correlational with a cross-sectional study approach. A total of 101 adolescents consisting of 59 males and 42 females, aged 16 – 23 years participated in this study, with body mass index (BMI) categories underweight, normal weight, overweight, and obese. All respondents obtain information about the research both orally and written. After obtaining information, all respondents filled out and sign an informed consent as a precondition before participating in the study.

Data collection technique. Measuring height using Stadiometer (Portable Seca® Stadiometer, North America). Weight is measured using a digital scale (OMRON Model HN-289, Omron Co., Osaka, Japan). BMI was calculated by dividing body weight (BW) (kg) with body height (BH) (m^2). Measuring the risk of gout arthritis by using a uric acid level indicator using Easy Touch (Easy Touch, Hsinchu, Taiwan) with a concentration unit of mg/dL. Blood sampling for measurement of uric acid levels was carried out on the capillary blood vessels on the fingerstick. Measurement of uric acid levels and BMI were performed at the same time.

Data analysis used a statistical software packet for social science (SPSS) version 17.0. Shapiro-Wilk test for data normality test. The correlation analysis between body mass index and uric acid levels was performed using Pearson's correlation coefficient test. All data are displayed with Mean ± Standard Deviation (SD). All statistical analyzes used a significant level ($p \leq 0.01$).

3. RESULTS AND DISCUSSION

Based on the analysis results show that the mean age (18.61 ± 2.09 years of age), body weight (BW) (63.94 ± 9.17 kg), body height (BH) (1.62 ± 0.07 meters), body mass index (BMI) underweight (17.64 ± 0.26 kg/m^2), normal weight (21.94 ± 1.33 kg/m^2), overweight (25.81 ± 0.51 kg/m^2), obese (29.92 ± 2.80 kg/m^2), and uric acid levels (6.09 ± 1.61 mg/dL). The results of the analysis of the percentages of age, gender, BMI, and uric acid levels of respondents are presented in Tables 1 – 4, while the results of the analysis of the relationship between BMI and uric acid levels can be seen in Figure 1. Figure 1 shows that there is a positive correlation between BMI and uric acid levels (r = 0.838; $p \leq 0.001$). BMI showed a strong positive correlation with uric acid levels.

Table 1. Results of the analysis of the percentage of respondents' age

No	Age	Amount	Percentage (%)
1	16	19	18.81
2	17	17	16.83
3	18	25	24.76
4	19	3	2.97
5	20	11	10.89
6	21	16	15.84
7	22	7	6.93
8	23	3	2.97
Total		**101**	**100**

Table 2. Results of the analysis of the percentage of respondents' gender

No	Gender	Amount	Percentage (%)
1	Male	59	58.41
2	Female	42	41.59
Total		**101**	**100**

Table 3. Results of the analysis of the percentage of respondents' body mass index

No	Category	Body Mass Index (kg/m^2)	
		Amount	Percentage (%)
1	Underweight	11	10.89
2	Normal Weight	41	40.59
3	Overweight	19	18.81
4	Obese	30	29.71
Total		**101**	**100**

Table 4. Results of the percentage analysis of respondents' uric acid levels

No	Category	Uric Acid Level (mg/dL)	
		Amount	Percentage (%)
1	Normal	58	57.43
2	High	43	42.57
Total		**101**	**100**

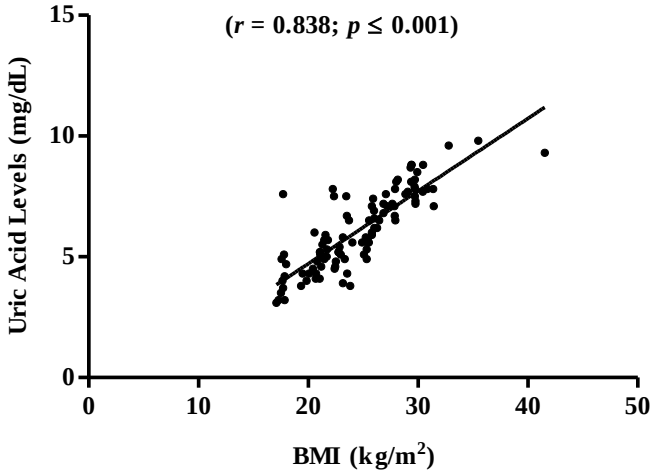

Figure 1. Results of correlation analysis between body mass index and uric acid levels**.** Pearson correlation coefficients (*r*) and *p*-value are shown in each graph.

This study aims to analyze the correlation between body mass index categories including underweight, normal, overweight, and obesity with the risk of gout arthritis in male and female adolescents. The main finding in this study is that there is a positive correlation between body mass index and uric acid levels. Body mass index showed a strong positive correlation with uric acid levels. Reinforced by Juraschek et al. (2013), shows that in both survey periods, for the average American adult standing 1.76 meters (5 feet 9 inches), a BMI of 1 unit higher, corresponding to a greater 3.1 kg (~6.8 pounds) weight, was associated with a 5% greater prevalence of uric acid, even after adjusting for serum uric acid. Dalbeth et al. (2020), also revealed that in a fasting state, people with high BMI experienced an increase in serum uric acid levels.

The instant lifestyle tends to make people lazy to do physical activity and pay less attention to the nutritional content of the food they eat. High protein intake can trigger an increase in uric acid levels in the blood (Soeroso, 2011). Lack of physical activity also triggers an increase in uric acid levels (Jaliana & Suhadi, 2018). Results by Syarifuddin et al. (2019) showed that respondents who had high uric acid levels were respondents who had low levels of physical activity, as many as 84.2% of 38 respondents. In addition to lack of physical activity, there are also types of foods with high purine content, such as offal, beef, sardines, duck meat, sea fish, shellfish, chicken, shrimp, nuts, tempeh, mushrooms, tapai, tofu, also in some vegetables such as melinjo, beans, cassava leaves, kale, cauliflower, and spinach (Dewi & Asnita, 2016). The factors that can affect uric acid levels in adolescents are 1). Heredity is one of the many risk factors for gout. People with families suffering from gout have a greater risk of developing gout (Sari & Syamsiyah, 2017). When there is a history of gouty arthritis in the family tree, it may trigger the onset of gouty arthritis (Sustrani et al., 2007). If there are family members who suffer from gouty arthritis, then their offspring are likely to suffer from gouty arthritis (Suiraoka, 2012). 2). Diet or consumption of high-purine foods from plant sources can increase the risk of gout arthritis (Az-zahra et al., 2014). One of the factors that cause an increase in uric acid levels is the consumption of foods containing high purines or about > 1000 mg/day (Kusumayanti et al., 2015). Purine is one of the organic base compounds that compose nucleic acids and is included in the amino acid group which is an element of protein formation (Kanwar & Kabra, 2016: 4). 3). History of comorbidities is a history of diseases that can accompany a certain disease (Bilotta, 2012). The incidence of gout arthritis can be exacerbated by hypertension and diabetes mellitus (Lingga, 2012). According to Anggraini et al. (2018) said that the incidence of

gouty arthritis was experienced by respondents who were obese. Obesity or being overweight is a form of malnutrition and metabolic disorders. Obesity is a feature of the population with gouty arthritis (Bilotta, 2012). Several epidemiological studies have shown that elevated uric acid levels are associated with several diseases including diabetes mellitus, dyslipidemia, obesity, hypertension, cardiovascular and metabolic syndromes (Huda et al., 2014; Desai et al., 2018).

Emerging evidence suggests that hyperuricemia is not only occurring in developed countries (Kanwar & Kabra, 2016; Zhu & Choi, 2010) but is also increasing in low- and middle-income countries with high frequency. In particular, a higher body mass index (BMI) has a doubled risk of developing gout compared to individuals with a normal BMI (McAdams et al., 2012). High BMI can increase blood pressure and serum triglyceride concentrations, leading to hypertriglyceridemia, which leads to an increase in uric acid in the body (de Oliviera & Burini 2012), so obesity is not only said to be a risk factor for the incidence of gout arthritis but has the potential to develop gouty arthritis (Lee et al., 2015). According to epidemiological studies on metabolic syndrome, increased uric acid levels are triggered by increased BMI, waist circumference, and dyslipidemia (Tanaka et al., 2015; Wang et al., 2014). Therefore, the incidence of increased levels of uric acid that triggers the occurrence of gouty arthritis is caused by an unfavorable lifestyle disorder and is associated with obesity (Tanaka et al., 2015). Obesity has greater potential as a trigger for gout arthritis compared to individuals with normal weight (Bilotta, 2012). Masheb et al. (2015), suggests that someone who is overweight sometimes has a pattern of eating more than what is needed. In addition, excess body weight causes stress on the joints so uric acid is difficult to excrete in the body and is at risk of triggering insulin resistance (McAdams et al., 2011).

Risk factors that cause people to develop gout are age, excessive intake of purine compounds, excessive alcohol consumption, obesity, excessive physical activity, hypertension and heart disease, certain drugs (especially diuretics), and impaired kidney function (Jaliana & Suhadi). , 2018). Research conducted by Nishida et al. (2011) stated that moderate physical activity had a significant relationship with low uric acid levels in obese respondents. Nishida et al. (2011) stated that moderate physical activity in subjects with obesity can increase insulin sensitivity and increase urine volume so that it has an impact on decreasing serum uric acid levels. Therefore, each individual is recommended to do 150 minutes of physical activity a week or 30 minutes a day for 5 days at moderate intensity (Parfitt et al., 2011). Research conducted by Chen et al. (2015) revealed that individuals who exercise at least 15-30 minutes a day such as walking, or brisk walking can reduce the increase in uric acid levels in the blood. Regular moderate-intensity physical activity can reduce uric acid levels in the blood (Garcia & Rodriguez, 2018).

4. CONCLUSION

Based on the results of the study, it was concluded that there was a positive correlation between body mass index and uric acid levels. BMI showed a strong positive correlation with uric acid levels. The higher the body mass index, the higher the risk of developing gout arthritis in both males and females.

5. REFERENCES

Almatsier, S. (2006). Penuntun Diet. Jakarta: Penerbit PT Gramedia Pustaka Utama.

Anggraini E., Dewi Ari P., Novayelinda R. (2018). Faktor-Faktor Yang Berhubungan Dengan Kejadian Gout Arthritis Masyarakat Melayu. JOM FKp, 5(2), 683-692.

Az-zahra, F., Nurwahid, D., & Pangastuti, R. (2014). Pola makan, obesitas, dan frekuensi serangan pada pasien artritis gout. Jurnal Gizi Klinik Indonesia, 11(1), 12-19.

Benn, C. L., Dua, P., Gurrell, R., Loudon, P., Pike, A., Storer, R. I., & Vangjeli, C. (2018). Physiology of Hyperuricemia and Urate-Lowering Treatments. Frontiers in medicine, 5, 160. https://doi.org/10.3389/fmed.2018.00160.

Bilotta, K.A.J. (2012). Kapita selekta penyakit: dengan implikasi keperawatan,ed 2. Jakarta: EGC

Budiono, A., Manampiring, A. E., & Bodhi, W. (2016). Hubungan kadar asam urat dengan status gizi pada remaja di Kecamatan Bolangitang Barat Kabupaten Bolaang Mongondow Utara. Jurnal e-Biomedik, 4(2).

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. https://doi.org/10.3390/jpm11030231.

Chen, J. H., Wen, C. P., Wu, S. B., Lan, J. L., Tsai, M. K., Tai, Y. P., Lee, J. H., Hsu, C. C., Tsao, C. K., Wai, J. P., Chiang, P. H., Pan, W. H., & Hsiung, C. A. (2015). Attenuating the mortality risk of high serum uric acid: the role of physical activity underused. Annals of the rheumatic diseases, 74(11), 2034–2042. https://doi.org/10.1136/annrheumdis-2014-205312.

Choi, H. K., Mount, D. B., Reginato, A. M., American College of Physicians, & American Physiological Society (2005). Pathogenesis of gout. Annals of internal medicine, 143(7), 499–516. https://doi.org/10.7326/0003-4819-143-7-200510040-00009.

Dalbeth, Nicola & Allan, Jordyn & Gamble, Gregory & Horne, Anne & Woodward, Owen & Stamp, Lisa & Merriman, Tony. (2020). Effect of body mass index on serum urate and renal uric acid handling responses to an oral inosine load: experimental intervention study in healthy volunteers. Arthritis Research & Therapy. 22. https://doi.org/10.1186/s13075-020-02357-y.

de Oliveira, E. P., & Burini, R. C. (2012). High plasma uric acid concentration: causes and consequences. Diabetology & metabolic syndrome, 4, 12. https://doi.org/10.1186/1758-5996-4-12.

Desai, R. J., Franklin, J. M., Spoendlin-Allen, J., Solomon, D. H., Danaei, G., & Kim, S. C. (2018). An evaluation of longitudinal changes in serum uric acid levels and associated risk of cardio-metabolic events and renal function decline in gout. PloS one, 13(2), e0193622. https://doi.org/10.1371/journal.pone.0193622.

Dewi, A. P & Asnita, L. (2016). Buku Ajar Perawatan Lansia Penderita Nyeri Sendi Dalam Keluarga dan Masyarakat. Pekanbaru: UR Press.

García-Esquinas, E., & Rodríguez-Artalejo, F. (2018). Association between serum uric acid concentrations and grip strength: Is there effect modification by age?. Clinical nutrition (Edinburgh, Scotland), 37(2), 566–572. https://doi.org/10.1016/j.clnu.2017.01.008.

Huda, N., Hossain, S., Rahman, M., Karim, M. R., Islam, K., Al Mamun, A., & Arefin, A. (2014). Elevated levels of plasma uric acid and its relation to hypertension in arsenic-endemic human individuals in Bangladesh. Toxicology and applied pharmacology, 281(1), 11-18.

Jaliana, J., & Suhadi, S. (2018). Faktor-Faktor yang Berhubungan dengan Kejadian Asam Urat pada Usia 20-44 Tahun di RSUD Bahteramas Provinsi Sulawesi Tenggara Tahun 2017. Jurnal Ilmiah Mahasiswa Kesehatan Masyarakat, 3(2).

Juraschek, S. P., Miller, E. R., 3rd, & Gelber, A. C. (2013). Body mass index, obesity, and prevalent gout in the United States in 1988-1994 and 2007-2010. Arthritis care & research, 65(1), 127–132. https://doi.org/10.1002/acr.21791.

Kanwar, G., & Kabra, R. 2016. Serum uric acid level and obesity: An association. International Journal of healthcare Sciences, 4(1), 52-55

Kusumayanti, D.G.A., Wiardani NK, Antarini NAA. (2015). Pola konsumsi purin dan kegemukan sebagai faktor risiko hiperurisemia pada msyarakat Kota Denpasar. Jurnal Skala Husada, 12(1), 27 – 31.

Lee, J., Lee, J. Y., Lee, J. H., Jung, S. M., Suh, Y. S., Koh, J. H., & Park, S. H. (2015). Visceral fat obesity is highly associated with primary gout in a metabolically obese but normal weighted population: a case control study. Arthritis research & therapy, 17(1), 79.

Lingga, L. 2012. Bebas penyakit asam urat tanpa obat. Jakarta: PT. Agro Media Pusaka.

Martillo, M. A., Nazzal, L., & Crittenden, D. B. (2014). The crystallization of monosodium urate. Current rheumatology reports, 16(2), 400. https://doi.org/10.1007/s11926-013-0400-9.

Masheb, R. M., Lutes, L. D., Kim, H. M., Holleman, R. G., Goodrich, D. E., Janney, C. A., Kirsh, S., Richardson, C. R., & Damschroder, L. J. (2015). High-frequency binge eating predicts weight gain among veterans receiving behavioral weight loss treatments. Obesity (Silver Spring, Md.), 23(1), 54–61. https://doi.org/10.1002/oby.20931.

McAdams DeMarco, M. A., Maynard, J. W., Huizinga, M. M., Baer, A. N., Köttgen, A., Gelber, A. C., & Coresh, J. 2011. Obesity and younger age at gout onset in a community-based cohort. Arthritis care & research, 63(8), 1108-1114.

Nasrul, S.E. (2012). Hiperurisemia pada Pra Diabetes. Jurnal Kesehatan Andalas, 1(2): 86-90. https://doi.org/10.25077/jka.v1i2.49.

Nishida, Y., Iyadomi, M., Higaki, Y., Tanaka, H., Hara, M., & Tanaka, K. 2011. Influence of physical activity intensity and aerobic fitness on the anthropometric index and serum uric acid concentration in people with obesity. Internal medicine, 50(19), 2121-2128.

Parfitt, Ekkekakis, P& Petruzzello, S. J. (2011). The pleasure and displeasure people feel when they exercise at different intensities. Sports medicine, 41(8), 641-671.

Perez-Ruiz, F., Dalbeth, N., & Bardin, T. (2015). A review of uric acid, crystal deposition disease, and gout. Advances in therapy, 32(1), 31–41. https://doi.org/10.1007/s12325-014-0175-z.

Roddy, E., & Doherty, M. 2010. Gout. Epidemiology of gout. Arthritis research & therapy, 12(6), 223.

Rosdiana, D. S., Khomsan, A., & Dwiriani, C. M. (2018). Pengetahuan Asam Urat, Asupan Purin Dan Status Gizi Terhadap Kejadian Hiperurisemia Pada Masyarakat Perdesaan. Media Pendidikan, Gizi, dan Kuliner, 7(2).

Sari, Y.N.I., & Syamsiyah, N.. (2017). Berdamai dengan asam urat. Jakarta: Bumi Medika.

Singh, J. A., Ramachandaran, R., Yu, S., Yang, S., Xie, F., Yun, H., Zhang, J., & Curtis, J. R. (2017). Is gout a risk equivalent to diabetes for stroke and myocardial infarction? A retrospective claims database study. Arthritis research & therapy, 19(1), 228. https://doi.org/10.1186/s13075-017-1427-5.

Skoczyńska, M., Chowaniec, M., Szymczak, A., Langner-Hetmańczuk, A., Maciążek-Chyra, B., & Wiland, P. (2020). Pathophysiology of hyperuricemia and its clinical significance - a narrative review. Reumatologia, 58(5), 312–323. https://doi.org/10.5114/reum.2020.100140.

Soeroso, J & Algristian, H. (2011). Asam urat. Jakarta: Niaga Swadaya. Diperoleh tanggal 20 Maret 2018 dari https://books.google.co.id/books.

Suiraoka. (2012). Penyakit Degeneratif. Yogyakarta: Nuha Medika.

Sustrani L., Alam S., & Hadibroto. (2007). Asam Urat. Jakarta: PT Gramedia Pustaka Utama.

Syarifuddin, L. A., Taiyeb, A. M., & Caronge, M. W. (2019). Hubungan Pola Makan dan Aktivitas Fisik dengan Kadar Asam Urat Dalam Darah Pada Penderita Asam Urat (Gout) di Wilayah Kerja Puskesmas Sabbangparu Kabupaten Wajo. In Seminar Nasional Biologi.

Tanaka, K., Ogata, S., Tanaka, H., Omura, K., Honda, C., Osaka Twin Research Group, & Hayakawa, K. (2015). The relationship between body mass index and uric acid: a study on Japanese adult twins. Environmental health and preventive medicine, 20(5), 347–353. https://doi.org/10.1007/s12199-015-0473-3.

Wang, H., Wang, L., Xie, R., Dai, W., Gao, C., Shen, P., Huang, X., Zhang, F., Yang, X., & Ji, G. (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), 1503–1509.

Wurangian, V. G. (2014). Gambaran Asam Urat Pada Remaja Obes Di Kabupaten Minahasa. Jurnal e-Biomedik, 2(1). https://doi.org/10.35790/ebm.v2i1.4019.

Zhu, Y., Pandya, B. J., & Choi, H. K. (2011). Prevalence of gout and hyperuricemia in the US general population: the National Health and Nutrition Examination Survey 2007-2008. Arthritis and rheumatism, 63(10), 3136–3141. https://doi.org/10.1002/art.30520.

Zhu, Y., Zhang, Y., & Choi, H. K. (2010). The serum urate-lowering impact of weight loss among men with a high cardiovascular risk profile: the Multiple Risk Factor Intervention Trial. Rheumatology (Oxford, England), 49(12), 2391–2399. https://doi.org/10.1093/rheumatology/keq256.