

Association of Sleep Duration and Sleep Quality With Metabolic Markers Among Construction Workers: A Cross-Sectional Study

Edward Timothy Setio¹, James Dendy¹, Muhammad Iqbal Taufani¹, Azizah Bonitha Zahrah Santoso¹, Jennifer Angeline¹, Louisa Angie¹, Kayleen Eleora Honggoseputro¹, Nasya Malfa Aqilah¹, Galuh Retno Nur Pratiwi¹, Joyceline Chika Tanely⁵, Citrawati Dyah Kencono Wungu^{2*,3}, Shafira Meidyana⁴

¹ Faculty of Medicine, Medical Program, Universitas Airlangga, Surabaya 60132, Indonesia

² Department of Physiology and Medical Biochemistry, Faculty of Medicine, Universitas Airlangga, Surabaya 60132, Indonesia

³ Institute of Tropical Disease, Universitas Airlangga, Surabaya 60115, Indonesia

⁴ Department of Public Health – Preventive Medicine, Universitas Airlangga, Surabaya 60132, Indonesia

⁵ School of Medicine and Health Sciences, Atma Jaya Catholic University of Indonesia, Indonesia

ABSTRACT

This study evaluates the association between sleep duration, sleep quality, and metabolic markers among construction workers in Sidoarjo Regency, Indonesia. This cross-sectional study enrolled 47 construction workers via consecutive sampling. Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI); blood glucose and uric acid were measured using EasyTouch GCU, while blood pressure was measured separately. Because most continuous variables were non-normally distributed, we assessed associations using Spearman's rank correlation. Sleep duration was negatively correlated with blood glucose ($p=0.028$) and positively correlated with uric acid ($p=0.015$). Supplementary quadratic regression showed no evidence of curvature in the sleep duration–uric acid relationship, and categorical sensitivity analyses (hyperuricemia-based) found consistent but non-significant directions in the Mann-Whitney U test ($p=0.072$) and logistic regression ($p=0.174$). Sleep quality was not significantly associated with any metabolic marker (all $p>0.05$). Sleep duration was consistently associated with metabolic markers in this sample. Sleep duration showed a significant positive correlation with uric acid that was not corroborated by categorical and regression-based sensitivity analyses, and should therefore warrant larger, multi-site samples in future studies.

Keywords: Sleep duration, Metabolic syndrome, Construction workers, Uric acid, Blood glucose

*Corresponding Author:

Citrawati Dyah Kencono Wungu

Email: citrawati.dyah@fk.unair.ac.id

INTRODUCTION

Metabolic syndrome is an increasingly important global public health concern. A recent systematic review and modeling analysis estimated that approximately 1.54 billion adults worldwide had metabolic syndrome in 2023, with prevalence reaching 31.0% among women and 25.7% among men.¹ Metabolic syndrome is a cluster of interrelated conditions, including abdominal obesity, insulin resistance, dyslipidemia, and hypertension, which substantially increase the risk of cardiometabolic complications.² Individuals with metabolic syndrome have a 2.35-fold higher risk of cardiovascular disease compared with those without the syndrome (95% CI: 2.02–2.73).³ The

global prevalence has increased substantially over the past two decades, rising from 11.9% in 2000 to 28.4% in 2023.¹ This growing burden is attributed to a combination of genetic and environmental factors, particularly unhealthy dietary patterns and sedentary lifestyles.¹

Poor sleep, whether from insufficient sleep or low-quality sleep, is increasingly recognized as a major trigger for metabolic issues. An updated meta-analysis including 36 cross-sectional and 9 longitudinal studies found that short sleep duration was associated with a higher risk of incident metabolic syndrome (RR 1.28, 95% CI 1.07–1.53). However, the magnitude of this association may vary across populations, with previous subgroup analyses reporting different effect estimates among Asian and Caucasian populations.⁴ Irregular sleep patterns disrupt our circadian rhythms and alter key hormones that regulate appetite and energy metabolism, such as

leptin, ghrelin, and insulin.⁵

Construction work involves intense physical labor and unpredictable shifts.⁶ These factors may take a toll on sleep and metabolic health, yet few studies have examined sleep duration and sleep quality together against multiple metabolic markers within a single occupational cohort.⁷ This study addresses this gap by assessing blood glucose and uric acid as the primary metabolic health markers.^{8,9} These markers are easy to test on-site and offer a clear picture of cardiovascular risk. By connecting them directly to two modifiable habits (sleep duration and subjective sleep quality assessed via questionnaires), we can create highly practical health screening tools for workplaces with limited resources. A study among blue-collar workers in Poland showed that the cardiometabolic profile, including glucose and uric acid, which differs by occupational status, supports an occupation-based screening approach; this was demonstrated in a cross-sectional study of three populations with different employment statuses in Poland.¹⁰

While the association between sleep and metabolism is well-documented globally,⁴ it remains unclear whether these same patterns apply to the rapidly industrializing region in developing countries. This group faces unique working conditions and dietary habits that differ greatly from previously studied populations; in developing countries, risks for work-related ill-health and injuries are estimated to be 10 to 20 times higher than in developed countries.¹¹ Therefore, this study aims to fill this gap by evaluating the relationship between sleep duration, sleep quality, and key metabolic markers among construction workers at a project site in Sidoarjo Regency, Indonesia. Sidoarjo was selected as the study setting because previous local studies have documented poor sleep quality and substantial occupational demands among workers, highlighting the relevance of sleep-related factors in the region's working population.

METHODS

Study Design and Setting

This cross-sectional study was conducted among workers at a construction project (Natura Residence) in Sidoarjo Regency, East Java, Indonesia, in April 2026.

Study Population

A total of 55 workers met the inclusion criteria and were enrolled through consecutive

sampling into the study (Figure 1). During data collection, eight participants were subsequently excluded from the final analysis because the point-of-care device (EasyTouch® GCU) failed to register uric acid levels due to values below the device's reading threshold (3mg/dL), which may be due to technical or organic factors in the participants. As uric acid was one of the three primary metabolic outcomes, each affected participant's dataset (including sleep, smoking, and other metabolic measurements) was excluded, yielding a final analytic sample of 47 participants.

Participants whose uric acid values fell below the lower detection limit of the point-of-care analyzer (EasyTouch GCU), as the device does not register a numeric reading below this threshold. Because exclusion was determined directly by proximity to the lower boundary of the measurement range, this process was not random with respect to the outcome: the excluded participants were, by definition, those with the lowest true uric acid concentrations in the sample. This introduces directional selection bias in the analytic subset by systematically removing the low end of the uric acid distribution and truncating the range of values available for correlation analysis. As a result, associations involving uric acid in this study should be interpreted as applying to the range of values the device could detect, and may not generalize to individuals with clinically low uric acid levels.

Inclusion criteria: (1) aged 18–59 years (working age); (2) employed on the Sidoarjo Regency (Indonesia) construction project; (3) willing to provide signed informed consent.

Exclusion criteria: (1) pregnant or breastfeeding; (2) taking medication known to affect metabolism or blood pressure; (3) history of metabolic disease; (4) incomplete metabolic measurement resulting from a point-of-care device reading.

Variables and Measurements

The independent variables were sleep duration and sleep quality. Blood pressure was also recorded as a baseline characteristic but was not analyzed inferentially. Dependent variables included random blood glucose and random uric acid. Smoking status was assessed as a baseline characteristic but was not analyzed inferentially. The authors have considered several confounding variables including age, sex, physical activity, and medical history. However, because of limited variability and sample size, only age was included

in the exploratory multivariate models. Blood glucose and uric acid were measured using the EasyTouch® GCU Blood Glucose/Cholesterol/Uric Acid Multi-Function Monitoring System (Biopetik Technology Corp., Hsinchu, Taiwan). Volunteers measured blood pressure using a sphygmomanometer. Remaining variables were collected using structured questionnaires.

Outcomes

To analyze the effects of sleep on metabolic markers, the primary outcomes of interest were random blood glucose and uric acid. Blood pressure was categorized as either normal (below the threshold of 120/80 mmHg, marked as 0) or abnormal (above 120/80 mmHg, marked as 1). The primary exposures for respondents include sleep duration and sleep quality using the Pittsburgh Sleep Quality Index (PSQI). Sleep quality was categorized as normal ($PSQI \leq 5$) and poor ($PSQI > 5$). Smoking status was classified as current smoker, former smoker, or non-smoker (nominal data). Data on blood pressure and smoking status were also collected as baseline characteristics, but were not included in the inferential analysis.

Statistical Analysis

Jamovi (version 2.6) was utilized to analyze the data. Initially, normality of continuous variables was assessed using the Shapiro-Wilk test. Because non-normally distributed variables predominated in this study, all continuous variables were reported as median (interquartile range, IQR) for consistency, and non-parametric statistical methods were used throughout. Spearman's rank correlation coefficient was used with the corresponding 95% confidence intervals. A two-sided $p < 0.05$ was considered statistically

significant.

We also ran multivariable linear regression models for both glucose and uric acid, adjusting for age, PSQI, and sleep duration. Model assumptions were assessed using Q-Q plots, showing departures from normality. As such, and given the small sample size, we report regression estimates as hypothesis-generating rather than confirmatory. Three sensitivity analyses were conducted additionally. First, because of outlying uric acid values, we repeated correlations after excluding them from the analysis. Second, because Spearman's correlation tests only for a monotonic association and cannot distinguish linear from curvilinear relationships, a quadratic sleep-duration term was added to test for curvature. Lastly, we dichotomized uric acid and re-examined its association with sleep duration using the non-parametric Mann-Whitney U test and logistic regression.

Ethical Considerations

This study was conducted in accordance with the Declaration of Helsinki, approved by the ethics committee of the Faculty of Medicine, Universitas Airlangga (approval number: 60/EC/KEPK/FKUA/2026, approved on March 8th, 2026), as part of the parent study "Effects of Sleeping Habits, Eating Habits, and Lung Health Education Towards the Health Condition of Construction Workers in Sidoarjo Regency." Written informed consent was obtained from all participants prior to enrollment. During the data analysis, subjects' names and identities were not included and were coded to maintain confidentiality. Access to the questionnaire, general check-up, and sphygmomanometer data was restricted to certain authors, as previously disclosed to participants.

RESULTS

Participant Characteristics

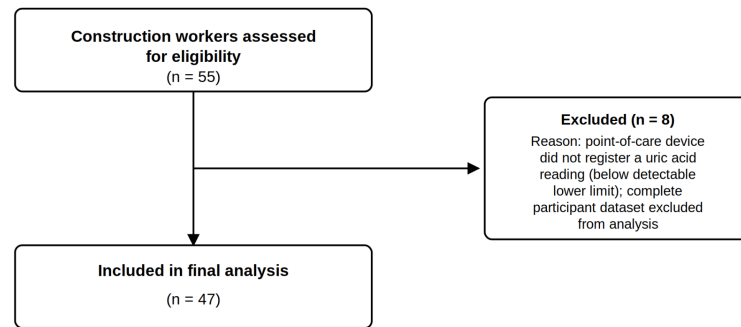


Figure 1. Flow diagram of participant recruitment, exclusion, and final analytic sample.

Figure 1. Flow Diagram of Participant Recruitment and Selection

Figure 1 shows a flow diagram illustrating participant recruitment and selection. Of the 55 construction workers initially assessed for eligibility, eight participants were excluded because of technical problems during general health check-up (GCU) measurements, leaving 47 participants for the final analysis. Table 1 presents the baseline demographic and clinical characteristics of the 47 participants. As most continuous variables were found to deviate from

the normal distribution on Shapiro–Wilk testing, all variables are reported uniformly as median (IQR). In addition, Table 2 presents the distribution of sleep-quality categories and their corresponding metabolic marker values. Smoking status was distributed as 27 current smokers (57.4%), 4 former smokers (8.5%), and 16 who had never smoked (34.0%). These descriptive comparisons were not subjected to inferential testing.

Table 1. Baseline Characteristics of Study Participants (N = 47)

Characteristic	n (%)	
Sex	Male: 42 (89.4)	Female: 5 (10.6)
Smoking Status	Never Smoked: 16 (34.0)	Smoked: 31 (66.0)
Blood Pressure	Normotensive: 15 (31.9)	Abnormal: 32 (68.1)
Variable	Median	IQR
Age (yrs)	42.00	19.50
Sleep duration (hrs)	7.00	1.25
Sleep quality (PSQI score)	4.00	3.00
Uric acid (mg/dL)	6.10	1.60
Glucose (mg/dL)	103.00	19.00

Of the 47 participants, most were male (n = 42, 89.4%), while 5 participants (10.6%) were female. Based on the 2019 Indonesian Hypertension Management Consensus issued by the Perhimpunan Dokter Hipertensi Indonesia (PERHI),¹² blood pressure values up to 120/80mmHg were considered normotensive in

this study, whereas values above this threshold were classified as abnormal. Accordingly, 32 participants (68.1%) were classified as abnormal and 15 (31.9%) as normotensive. Sleep duration ranged from 2 to 10 hours, with a median duration of 7.0 hours (IQR 1.25).

Table 2. Metabolic Markers and Sleep Duration According to Sleep Quality

Sleep quality	n (%)	Glucose, median (IQR)	Uric acid, median (IQR)	Sleep duration, median (IQR)
Normal (PSQI ≤5)	32 (68.1%)	102.50 (17.00)	6.15 (1.97)	7.00 (1.12)
Poor (PSQI >5)	15 (31.9%)	105.00 (19.00)	6.10 (0.80)	6.00 (2.25)

Correlation Between Sleep Parameters and Metabolic Markers

Associations between sleep variables (sleep duration as continuous and sleep quality as ordinal data) and metabolic markers were assessed using Spearman's rank correlation as

provided in Table 3. Sleep duration was significantly and inversely correlated with blood glucose and significantly and positively correlated with uric acid. Sleep quality (PSQI) was not significantly correlated with either metabolic marker.

Table 3. Spearman Correlation Between Sleep Variables and Metabolic Markers

Outcome	Sleep duration ρ (95% CI), p	Sleep quality ρ (95% CI), p
Glucose	-0.322 (-0.56–0.03), 0.028*	-0.007, 0.963
Uric acid	0.352 (0.06–0.59), 0.015*	-0.288, 0.050

*Statistically significant ($p < 0.05$).

Sensitivity and Exploratory Analysis

Excluding statistical outliers in uric acid did not change the direction, magnitude, or significance of the sleep duration–uric acid or sleep duration–glucose correlations. To test for a curvilinear relationship, we added a quadratic sleep–duration term to an exploratory regression model predicting uric acid. The quadratic term was not statistically significant ($\beta = -0.05$, $p = 0.374$), and the linear term also lost significance once the quadratic term was included ($p = 0.230$). As such, this provided no statistical evidence of a curvature within the range of sleep duration observed in this sample, indicating that the association more closely reflected an approximately linear, increasing pattern.

When uric acid was categorized, participants with hyperuricemia ($n=14$) had a higher median sleep duration than those without ($n=33$) (7.00 vs 6.00 hours), consistent with the previous finding. However, this difference did not manage to reach statistical significance (Mann–Whitney $U = 307.5$, $p = 0.072$). Logistic regression estimated an odds ratio of 1.41 (95% CI 0.86–2.32, $p = 0.174$) for hyperuricemia per additional hour of sleep duration.

Exploratory Regression Analysis

Multivariable linear regression models for glucose and uric acid, each including PSQI (either normal or abnormal sleep quality), sleep duration, and age, did not identify any statistically significant predictor (all $p > 0.05$). The overall model fit was also modest (glucose: $R^2 = 0.141$, $aR^2: 0.0363$; uric acid: $R^2 = 0.128$, $aR^2: 0.0217$). Residual Q–Q plots for both models deviated from normality, so these exploratory models should be cautiously interpreted.

DISCUSSION

Principal Findings

Among the construction workers in this study, sleep duration, rather than sleep quality, was the primary behavioral factor associated with the metabolic markers examined. Specifically, shorter sleep was associated with higher blood glucose levels, whereas longer sleep was surprisingly linked to higher uric acid. Otherwise, sleep quality showed no association with metabolic markers. To double-check these results, we ran sensitivity analyses using a curved relationship test and by grouping uric acid into categories. These checks generally supported our main correlation findings, although the categorical analysis did not reach statistical significance, which we discuss in detail below.

Sleep Duration and Blood Glucose

This study identified an inverse relationship between sleep duration and glucose concentration in blood, indicating that shorter sleep may be linked with higher glucose levels. This direction is consistent with existing evidence, placing this finding as confirmatory to this phenomenon. At the cellular level, acute sleep restriction reduces insulin-stimulated PKB (Akt) signals in skeletal muscle, and lowers insulin sensitivity as shown by an 18.6% drop in the Matsuda index after two nights of restricted sleep in healthy men.¹³ At the hormonal level, shortened sleep has been associated with a decrease in leptin and increase in ghrelin, forming a feeling of hunger and food intake. Meanwhile, elevated cortisol further lowers insulin action and increases hepatic glucose output.^{14,15} These molecular and hormonal changes combine into measurable glycemic differences at the population level. This was evidenced in previous research, where among Korean non-regular workers, shorter sleep duration was associated with impaired fasting glucose.¹⁴ Additionally, a South Asian community sample found that those sleeping

under 6 hours had significantly higher fasting glucose than longer sleepers.¹⁶

Sleep Duration and Uric Acid

This study found a positive association between sleep duration and uric acid, indicating that longer sleep was linked with higher levels of uric acid. This direction contrasts with most existing literature, which typically reports the opposite pattern as observed among Chinese government employees,¹⁷ and in elderly Mediterranean population.¹⁸ Several explanations, such as a non-linear, U-shaped relationship between sleep duration and uric acid (both unusually short and unusually long sleep increase risk relative to a mid-range optimum), have been reported in a large network meta-analysis.¹⁹ However, this explanation could not be supported by the exploratory analysis conducted by the author, providing no statistical evidence of curvature within the observed range of sleep duration. A methodological explanation should also be considered. As noted in the limitations, participants were excluded specifically for having undetectably low uric acid, which trimmed the bottom end of the distribution before the correlation was ever calculated. That kind of exclusion can push a correlation upward on its own, independent of any real biology. Given how directly this exclusion connects to the outcome itself, it is a more likely driver of the divergence from prior literature than an occupation-specific confounder we simply did not measure. Another possible explanation is reduced physical activity, which has been associated with elevated inflammatory and metabolic markers, including uric acid.^{17,19} Because this finding diverges in direction from most prior literature, it should be treated more as hypothesis-generating for this specific population rather than confirmatory, generalizing theory.

Sleep Quality (PSQI) and Metabolic Markers

Unlike sleep duration, sleep quality, as defined by PSQI results, was not significantly correlated with glucose or uric acid. This divergence between duration and quality is not unusual, as PSQI by nature is retrospective and subjective. Mendelian randomization work shows that objectively measured sleep traits and self-reported sleep measures may reflect distinct exposures,²⁰ which may explain why duration was more sensitive than PSQI.

Although the absence of significant associations with sleep quality may appear

counterintuitive, this trend has been reported in a study of Taiwanese adults, reporting that poor sleepers had lower levels of uric acid and blood glucose compared to good sleepers. The original authors attributed this pattern to confounding by age and body composition.⁹ In the present study, another possible explanation is that the relatively small sample size and the distribution of PSQI scores limited the ability to detect modest associations with metabolic outcomes. Construction-worker-specific studies have also shown that poor sleep quality is highly prevalent and closely linked with fatigue, depressive symptoms, and chronic disease,⁶ suggesting that subjective PSQI scores in this occupational setting may reflect broader occupational and psychosocial influences in addition to metabolic health.

Strengths and Limitations

This study represents a population novelty, focusing on construction workers, an occupation notably underrepresented in the sleep-metabolic literature despite plausible occupational exposures including irregular shift patterns, heat stress, and physical exertion that may plausibly affect sleep and metabolic parameters. Despite this novelty, several limitations apply. The sample was modest ($n = 47$) after exclusions, limiting statistical power. Several exposure variables relied on self-report, including sleep quality and smoking status, with smoking captured only as a categorical variable rather than pack-years. Glucose and uric acid were measured via non-fasting point-of-care capillary testing rather than laboratory assay; non-fasting glucose values in particular are more variable and less clinically interpretable than fasting measurements.

Additionally, the point-of-care device could not register values below its detection threshold, and participants with uric acid levels below this threshold were excluded from analysis. This exclusion was not random with respect to uric acid level, potentially truncating the lower end of the distribution and introducing a bias that may have influenced the observed positive association between sleep duration and uric acid; the unexpected direction of this association should therefore be interpreted cautiously and requires confirmation in larger studies using laboratory-based measurements.

Regression models were further limited by non-normal residuals driven by a small number of influential observations, low explained variance

(adjusted $R^2 = 0.036$ for glucose, 0.022 for uric acid), and no statistically significant predictors; accordingly, regression estimates are reported as hypothesis-generating rather than confirmatory.

Implications and Future Research

The finding that shorter sleep is linked to higher blood glucose aligns perfectly with well-established global research. When sleep is cut short, it alters the body's neuroendocrine system, triggering a stress response that reduces insulin sensitivity and disrupts glucose handling. What makes this study unique, however, is that this relationship still holds true even within a workforce facing intense physical labor and irregular shifts, factors that usually affect metabolic health. This suggests that getting enough sleep is a powerful, independent shield for blood sugar control, even in highly demanding work environments.

There's also a more specific issue worth flagging: the eight participants excluded from this study weren't dropped at random. They were excluded because the point-of-care device couldn't register a uric acid reading below its detection floor, meaning the participants we lost were, by definition, the ones with the lowest uric acid values. This effectively cut off the low end of the uric acid distribution before any analysis even began. If sleep duration also happened to differ between the excluded and included groups, this alone could push the sleep–uric acid correlation in a positive direction, regardless of any real biological relationship. Because this exclusion was tied directly to the outcome we measured, it's arguably a simpler and more likely explanation for our counterintuitive finding than an unmeasured confounder, and we should keep it in mind when interpreting the result.

CONCLUSION

Among 47 construction workers in Sidoarjo Regency, Indonesia, sleep duration showed more consistent associations with metabolic health markers than subjective sleep quality (PSQI). While shorter sleep was linked to higher blood glucose (matching well-known research on how sleep loss lowers insulin sensitivity), longer sleep was unexpectedly related to higher uric acid. This contrast to the typical U-shaped patterns found in larger studies should be treated as a preliminary finding, given our modest sample size and snapshot design. Overall, these results suggest that adjusting sleep duration, perhaps through better shift scheduling, is a practical way to

support worker health, though larger, long-term studies using objective tracking tools are needed to confirm this.

ACKNOWLEDGEMENTS

The authors would like to thank the members of the Research and Academic Division of the Asian Medical Students' Association (AMSA) Universitas Airlangga Bryan Nathaniel, Samuel Alexius, Beatrice Carina, Pramudya Dhafa, Kaleb Stanfier Hewis, Elizabeth Blessya, Jacqueline Angela, Jens Kefas, Achmad Nurul Falah, Muhammad Wildan Barizal, Yudistira Putra, Najya Alysia Paprinha, Jonathan Samuel, Jilan Nafisi, Adel Demisa, and Raras Nur Anggraeny for their contributions to data collection, along with the Community Outreach Division of AMSA Universitas Airlangga for their support in participant recruitment during this study.

AUTHOR CONTRIBUTIONS

Conceptualization: Setio ET. Methodology: Wungu CDK. Investigation: Dendy J, Taufani MI, Santoso ABZ, Angeline J, Honggoseputro KE, Angie L, Aqilah NM, Pratiwi GRN. Data curation: Taufani MI. Formal analysis: Dendy J, Tanely JC. Validation: Meidyana S. Writing – original draft: Setio ET, Dendy J, Taufani MI, Santoso ABZ, Angeline J. Writing – review & editing: Wungu CDK, Meidyana S, Tanely JC, Honggoseputro KE, Angie L, Aqilah NM, Pratiwi GRN. Supervision: Wungu CDK. Project administration: Santoso ABZ.

REFERENCE

1. Noubiap JJ, Nansseu JR, Nyaga UF, Ndoadoumgue AL, Ngouo AT, Tounouga DN, et al. Worldwide trends in metabolic syndrome from 2000 to 2023: a systematic review and modelling analysis. *Nat Commun.* 2025 Dec 6;17(1):573. doi:10.1038/s41467-025-67268-5
2. Swarup S, Ahmed I, Grigoroza Y, Zeltser R. Metabolic Syndrome. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 [cited 2026 Jul 6]. Available from: <http://www.ncbi.nlm.nih.gov/books/NBK459248/>
3. Mottillo S, Filion KB, Genest J, Joseph L, Pilote L, Poirier P, et al. The metabolic syndrome and cardiovascular risk a systematic review and meta-analysis. *J Am Coll Cardiol.* 2010 Sep 28;56(14):1113–32. doi:10.1016/j.jacc.2010.05.034

4. Xie J, Li Y, Zhang Y, Vgontzas AN, Basta M, Chen B, et al. Sleep duration and metabolic syndrome: An updated systematic review and meta-analysis. *Sleep Med Rev.* 2021 Oct;59:101451. doi:10.1016/j.smrv.2021.101451
5. Beccuti G, Pannain S. Sleep and obesity. *Curr Opin Clin Nutr Metab Care.* 2011 Jul;14(4):402–12. doi:10.1097/MCO.0b013e3283479109
6. Kim Y, Lee S, Lim J, Park S, Seong S, Cho Y, et al. Factors Associated with Poor Quality of Sleep in Construction Workers: A Secondary Data Analysis. *Int J Environ Res Public Health.* 2021 Feb 25;18(5):2279. doi:10.3390/ijerph18052279
7. Hashemipour S, Yazdi Z, Ghorbani A. Occupational Difference in Association of Poor Sleep Quality and Metabolic Syndrome: Differences between Workers and Employees. *Sleep Disord.* 2021 Sep 17;2021:1–8. doi:10.1155/2021/9947027
8. Lu Q, Wu S, Wang S, Xiao J. Influence of occupation type on the association between sleep duration and impaired fasting glucose: results from a Chinese population-based study. *BMJ Open.* 2021 Jun;11(6):e042066. doi:10.1136/bmjopen-2020-042066
9. Chou YT, Li CH, Shen WC, Yang YC, Lu FH, Wu JS, et al. Association of sleep quality and sleep duration with serum uric acid levels in adults. *PLOS ONE.* 2020 Sep 17;15(9):e0239185. doi:10.1371/journal.pone.0239185
10. Sołtysik BK, Kostka J, Karolczak K, Watała C, Kostka T. What is the most important determinant of cardiometabolic risk in 60–65-year-old subjects: physical activity-related behaviours, overall energy expenditure or occupational status? A cross-sectional study in three populations with different employment status in Poland. *BMJ Open.* 2019 Jul;9(7):e025905. doi:10.1136/bmjopen-2018-025905
11. Ahasan MR, Partanen T. Occupational Health and Safety in the Least Developed Countries-A Simple Case of Neglect. *J Epidemiol.* 2001;11(2):74–80. doi:10.2188/jea.11.74
12. Perhimpunan Dokter Hipertensi Indonesia. Konsensus Penatalaksanaan Hipertensi 2019 di Indonesia. Jakarta: Perhimpunan Dokter Hipertensi Indonesia.
13. Chaput JP, Dutil C, Sampasa-Kanyinga H. Sleeping hours: what is the ideal number and how does age impact this? *Nat Sci Sleep.* 2018;10:421–30. doi:10.2147/NSS.S163071
14. Reynolds AC, Dorrian J, Liu PY, Van Dongen HPA, Wittert GA, Harmer LJ, et al. Impact of five nights of sleep restriction on glucose metabolism, leptin and testosterone in young adult men. *PLOS ONE.* 2012;7(7):e41218. doi:10.1371/journal.pone.0041218
15. Mosavat M, Mirsanjari M, Arabiat D, Smyth A, Whitehead L. The Role of Sleep Curtailment on Leptin Levels in Obesity and Diabetes Mellitus. *Obes Facts.* 2021;14(2):214–21. doi:10.1159/000514095
16. Joo J, Lee JG, Kim S, Lee J, Lee JH, Lee KJ. Association between sleep duration and impaired fasting glucose according to work type in non-regular workers: data from the first and second year (2016, 2017) of the 7th Korean National Health and Nutrition Examination (KNHANE) (a cross-sectional study). *Ann Occup Environ Med.* 2020;32:e29. doi:10.35371/aoem.2020.32.e29
17. An Y, Li X, Ouyang F, Xiao S. Association between nocturnal sleep duration and the risk of hyperuricemia among Chinese government employees: A cross-sectional study. *Front Public Health.* 2022;10:1055778. doi:10.3389/fpubh.2022.1055778
18. Papandreou C, Babio N, Díaz-López A, Martínez-González MÁ, Becerra-Tomas N, Corella D, et al. Sleep Duration is Inversely Associated with Serum Uric Acid Concentrations and Uric Acid to Creatinine Ratio in an Elderly Mediterranean Population at High Cardiovascular Risk. *Nutrients.* 2019 Apr 1;11(4):761. doi:10.3390/nu11040761
19. Luo C, Zhang F, Shen D, Sun J, Zhang Y, Xie Z, et al. Nighttime sleep duration and the prevalence of hyperuricemia: a systematic review and network meta-analysis. *Front Neurosci.* 2025 May 9;19:1436116. doi:10.3389/fnins.2025.1436116
20. Liu J, Richmond RC, Anderson EL, Bowden J, Barry CJS, Dashti HS, et al. The role of accelerometer-derived sleep traits on glycated haemoglobin and glucose levels: a Mendelian randomization study. *Sci Rep.* 2024 Jun 28;14(1):14962. doi:10.1038/s41598-024-58007-9