JIKM. 2025; 17(4): 192-200
DOI: 10.52022/jikm.v17i4.799
p-ISSN: 2085-4366, e-ISSN: 2684-950X

Jurnal Ilmiah Kesehatan Masyarakat
Media Komunikasi Komunitas Kesehatan Masyarakat

Relationship between Muscle Mass, Fasting Blood Glucose, and Vascular Elasticity in Medical Students

Raja Soaloon Purba[1], Nurfitri Bustamam[2*], Maria Selvester Thadeus[3], Marlina Dewiastuti[4]

[1]Undergraduate Medical Program, Faculty of Medicine, Universitas Pembangunan Nasional "Veteran" Jakarta, RS Fatmawati Street, Pondok Labu, South Jakarta 12450, Indonesia
[2]Department of Physiology, Faculty of Medicine, Universitas Pembangunan Nasional "Veteran" Jakarta, RS Fatmawati Street, Pondok Labu, South Jakarta 12450, Indonesia
[3]Department of Pathology Anatomy, Faculty of Medicine, Universitas Pembangunan Nasional "Veteran" Jakarta, RS Fatmawati Street, Pondok Labu, South Jakarta 12450, Indonesia
[4]Department of Internal Medicine, Faculty of Medicine, Universitas Pembangunan Nasional "Veteran" Jakarta, RS Fatmawati Street, Pondok Labu, South Jakarta 12450, Indonesia

Abstract

***Background**: Low vascular elasticity is one of the cardiovascular disease risk determinants. Sedentary lifestyles and high-glucose diets may reduce muscle mass and elevate blood glucose levels, potentially reducing vascular elasticity. This study aims to analyze the relationship between muscle mass and fasting blood glucose levels on vascular elasticity in medical students.*

***Methods:** A cross-sectional study was carried out involving 53 medical students aged 18–22 years using a stratified random sampling technique. Subject criteria were non-smokers, non-alcohol consumers, and had no history of diabetes, hypertension, or taking medications affecting blood pressure, blood glucose, or cholesterol levels. Muscle mass, fasting blood glucose, and vascular elasticity were measured using a bioelectrical impedance analyzer, capillary blood testing, and accelerated photoplethysmograph, respectively. Chi-square tests and multivariable logistic regression were used to analyze data.*

***Results:** No significant differences in age, blood pressure, BMI, or eating habits between groups with the suboptimal and those with normal+optimal vascular elasticity ($p > 0.05$). Multivariable logistic regression analysis showed that muscle mass significantly affected vascular elasticity ($p = 0.009$; OR = 9.656; CI = 1.757-53.075), after controlling with fasting blood glucose levels, which were not statistically significant ($p = 0.386$; OR = 2.329; CI = 0.344-15.776). Subjects with higher muscle mass were nearly 10 times more likely to have better vascular elasticity.*

***Conclusion:** Muscle mass is significantly associated with vascular elasticity in young adults, independent of fasting blood glucose levels. These findings emphasize the importance of maintaining adequate muscle mass. Promoting muscle mass through healthy lifestyle habits may contribute to preserving vascular elasticity and mitigating the risk of cardiovascular disease.*

***Keywords**: Fasting blood glucose, Muscle mass, Vascular elasticity, Young adult*

INTRODUCTION

Global data indicate that cardiovascular account for about 17.9 million deaths each year. In a 2016-2017 case-control study involving individuals over 40 years in Malang, East Java Province, 29.2% of the subjects were at high risk of cardiovascular disease.[1] Vascular elasticity is one of the crucial factors in the early detection of cardiovascular diseases. It refers to the capacity of arterial walls to

Correspondence*: Nurfitri Bustamam
E-mail: nurfitri.bustamam@upnvj.ac.id

Received: April, 24 2025
Accepted: July, 13 2025
Published: November, 20 2025

stretch and return to their original state in response to fluctuations in blood pressure. .[2] Vascular elasticity is affected by several factors, one of which is physical activity.[3] The Indonesian Ministry of Health reported that the prevalence of sedentary lifestyles increased from 26.1% in 2013 to 33.5% in 2018.[4] This trend of sedentary lifestyles is also observed among medical students. A study conducted at Yarsi University in 2018 reported that 52.5% of medical students led sedentary lifestyles.[5] Inadequate physical activity can contribute to a decrease in muscle mass.[6] Decreased muscle mass can lead to insulin resistance, lowered nitric oxide levels, increased endothelin production, elevated oxidative stress, and vascular smooth muscle proliferation, all of which can impair vascular elasticity.[7]

Vascular elasticity is also affected by blood glucose levels.[8] In Indonesia, the consumption of high-glucose diets increased from 53.1% in 2013 to 61.27% in 2018.[4] Chronic elevated blood glucose levels can cause endothelial dysfunction, which in turn contributes to a reduction in vascular elasticity.[9]

The growing global cardiovascular disease burden, coupled with the increasing prevalence of sedentary lifestyles and high-glucose diets among young adults, particularly in the medical student population, underscores the need for early identification of modifiable risk factors. While previous studies have explored the associations between muscle mass, blood glucose levels, and vascular elasticity in older adults, limited research has been conducted in younger, healthy populations. Moreover, existing research often examines these variables independently, without addressing their potential combined effects on vascular elasticity.

The concurrent trends of low physical activity and high sugar intake among Indonesian youth highlight the urgency of examining these interrelated factors. Gaining insight into how muscle mass and blood glucose contribute to vascular elasticity could inform early intervention strategies and support cardiovascular health from young adulthood. Therefore, this study aimed to investigate the relationship between muscle mass, fasting blood glucose levels, and vascular elasticity among medical students. This study hypothesizes that low muscle mass and high fasting blood glucose are associated with low vascular elasticity in this population.

METHOD

Participants and Study Design

A cross-sectional study design was used to investigate the relationship between muscle mass and fasting blood glucose levels on vascular elasticity in medical students. Data collection was conducted in December 2023 at the Medical Education and Research Center (MERCE) Physiology and Nutrition Laboratory, UPNVJ.

A self-administered questionnaire was used to collect subject characteristics, while eating habits were evaluated using the Adolescent Food Habit Checklist (AFHC) in Bahasa Indonesia. The AFHC demonstrated good validity and reliability with a Cronbach's alpha of 0.860.[10] Subsequently, all participants had their body composition, vascular elasticity, and fasting blood glucose measured.

The study population consisted of all active medical students registered at UPNVJ in the 2023/2024 academic year. The minimum required sample size was determined using the two-proportions formula, with parameters set at $\alpha = 5\%$, $\beta = 20\%$, $P_1 = 0.696$[11], and $P_2 = 0.341$.[4] Based on this calculation, a minimum of 48 subjects was required. To account for potential dropouts or missing data, a 10% adjustment was applied, increasing the target sample size to 53 subjects.

Stratified random sampling was employed to ensure representation across different subgroups of the population. In this study, the sample unit that was stratified was the year of study within the Faculty of Medicine UPNVJ. This technique was used to capture variability across academic levels, which may influence physical activity levels, dietary habits, and metabolic status. The inclusion criteria were medical students aged 18 years or older, non-smokers, and non-consumers of alcohol. Exclusion criteria included diabetes mellitus or hypertension histories, as well as the use of medications that could affect blood glucose levels, blood pressure, or cholesterol.

Measurements and Procedure

Participants were asked to remove any metal accessories and were provided with information about the study procedures before data collection. Subsequently, participants provided written informed consent. Muscle mass was assessed using Body Composition Analyzer (Tanita MC-980MA Plus), a device with a sensitivity of 75% and specificity of 92.7%.[12] During the measurement, participants stood barefoot on the device and grasped the hand electrodes for approximately 30 seconds. Muscle mass was then categorized as low, normal, or high, based on reference values provided by the Tanita Body Composition Analyzer.

Assessment of vascular elasticity was performed using the Accelerated Photoplethysmography (APG) SA-3000P, which demonstrates a sensitivity of 71.4% and specificity of 90%. Subjects were asked to sit comfortably after removing any remaining metal accessories. The APG sensor was then attached to the fingertip, and measurements were recorded for three minutes. Based on their scores, vascular elasticity was classified as suboptimal (<30), normal (30-70), or optimal (>70).[13]

Fasting blood glucose levels were measured following at least eight hours of overnight fasting. Capillary blood samples were obtained using a sterile lancet pen, and a drop of blood applied to a test strip inserted into a Sinocare glucometer. Glucose levels were recorded after 15 seconds. Fasting blood glucose levels were classified as normal (<100 mg/dL) or prediabetic (100–125 mg/dL). Participants with glucose levels above 125 mg/dL were excluded from the study.

The AFHC questionnaire was used to evaluate healthy eating habits. It comprises 23 items designed to identify both healthy and unhealthy dietary habits. Nine items include the additional option "Not applicable to me," in addition to the "Yes" and "No" response options. One point is given for each response that demonstrates a healthy eating pattern. The AFHC score was calculated by dividing the number of healthy eating responses multiplied by 23 by the total number of completed items. Scores below the mean were classified as indicating poor eating habits, whereas scores equal to or above the mean were classified as reflecting good eating habits.[14]

Statistical Analysis and Ethical Clearance

Data were analyzed using Chi-square or Fisher's exact tests, and subsequently subjected to multivariable logistic regression to evaluate the adjusted association between independent variables and vascular elasticity. The results are reported as odds ratios (OR) along with 95% confidence intervals (CI), reflecting the probability of having normal or optimal vascular elasticity in relation to muscle mass and fasting blood glucose levels. Potential confounding variables were identified by assessing changes in the odds ratio following the sequential exclusion of each covariate from the regression model. A variable was considered a confounder if its removal from the regression model led to a 10% change or greater in the OR of the primary exposure variable. Ethical approval was obtained from the Health Research Ethics Commission of Universitas Pembangunan Nasional Veteran Jakarta (Ethical Clearance No: 451/XI/2023/KEPK). All participants gave written informed consent.

RESULT

The study found that the median age of the subjects was 20 years. Most of the subjects were female, had normal blood pressure and body mass index (BMI), and exhibited poor eating habits. Statistical analyses indicated no significant differences in age, gender, BMI, or eating habits between the two vascular elasticity groups ($p > 0.05$) (Table 1). In this study, the majority of the subjects had normal muscle mass (56.6%) and normal fasting blood glucose levels (81.1%) (Table 2).

A significant crude association was found between muscle mass and vascular elasticity. Subjects with low muscle mass were 2.5 times more likely to have suboptimal vascular elasticity than those with normal or high muscle mass (OR = 2.533; 95% CI: 1.562–4.109) (Table 3). This association was based on the proportion of suboptimal vascular elasticity in the exposed group (low muscle mass: 86.7%)

versus the non-exposed group (normal and high muscle mass: 34.2%), indicating that suboptimal vascular elasticity was considerably more prevalent among individuals with low muscle mass.

Similarly, fasting blood glucose levels showed a significant crude association with vascular elasticity. Subjects with prediabetic fasting blood glucose levels were 1.9 times more likely to have suboptimal vascular elasticity than those with normal glucose levels (OR = 1.911; 95% CI: 1.195–3.055) (Table 4). Suboptimal vascular elasticity was observed in 80% of the exposed group (prediabetes) compared to 41.9% in the non-exposed group (normal glucose levels), suggesting a clear association between elevated fasting glucose and impaired vascular elasticity.

In the adjusted model derived from multivariable logistic regression analysis (conducted in two modeling stages), muscle mass continued to be a significant predictor of vascular elasticity (adjusted OR = 9.656; 95% CI: 1.757–53.075), whereas fasting blood glucose was not significant (adjusted OR = 2.329; 95% CI: 0.344–15.776) (Table 5). The findings show that muscle mass has a greater influence on vascular elasticity than fasting blood glucose. Individuals with low muscle mass are more likely to exhibit suboptimal vascular elasticity, even after adjustment for fasting blood glucose levels.

Table 1. Characteristics of Study Subjects Based on Vascular Elasticity Group

Characteristics	Vascular Elasticity		p-value
	Suboptimal n=26	Normal+Optimal n=27	
Age, median (min-max)	20 (18—22)	20 (18—22)	0.527*
Blood Pressure, mean ± SD			
Systolic (mmHg)	123.7 ± 3.7	122 ± 4.8	0.202*
Diastolic (mmHg)	80.4 ± 3.3	79.8 ± 3.3	0.513*
Gender, n (%)			
Male	8 (42.1)	11 (57.9)	0.638#
Female	18 (52.9)	16 (47.1)	
BMI, n (%)			
Underweight	5 (62.5)	3 (37.5)	0.736•
Normal	9 (56.3)	7 (43.8)	
Overweight	2 (40)	3 (60)	
Obese I	7 (46.7)	8 (53.3)	
Obese II	3 (33.3)	6 (66.7)	
Eating Habits, n (%)			
Poor	12 (44.4)	15 (55.6)	0.682#
Good	14 (53.8)	12 (46.2)	

Notes: *Mann-Whitney U test, #Chi-square test, •Chi-square-exact test

Table 2. Distribution of Muscle Mass and Fasting Blood Glucose Levels among Subject

Variable	Frequency (n)	Percentage (%)
Muscle Mass		
Low (Score -4 to -2)	15	28.3
Normal (Score -1 to +1)	30	56.6
High (Score +2 to +4)	8	15.1
Fasting Blood Glucose		
Normal (< 100 mg/dL)	43	81.1
Prediabetes (100 – 125 mg/dL)	10	19.9

Table 3. Relationship between Muscle Mass and Vascular Elasticity

Muscle Mass	Vascular Elasticity				Total		p-value	OR	95% CI
	Suboptimal		Normal + Optimal						
	n	%	n	%	N	%			
Low	13	86.7	2	13.3	15	100	0.002	2.533	1.562—4.109
Normal + High	13	34.2	25	65.8	38	100			

Table 4. Relationship between Fasting Blood Glucose Levels and Vascular Elasticity

Fasting Blood Glucose Levels	Vascular Elasticity				Total		p-value	OR	95% CI
	Suboptimal		Normal + Optimal						
	n	%	n	%	N	%			
Prediabetes	8	80	2	20	10	100	0.039	1.911	1.195 – 3.055
Normal	18	41.9	25	58.1	43	100			

Table 5. Estimated Model of the Causal Effect of Muscle Mass and Fasting Blood Glucose on Vascular Elasticity

Variable	p-value	OR	95% CI
Fasting Blood Glucose	0.386	2.329	0.344 – 15.776
Muscle Mass	0.009	9.656	1.757 – 53.075

DISCUSSION

Age is known to influence vascular elasticity. Aging can alter the balance between collagen and elastin in the vascular walls and promote vascular calcification, a key contributor to reduced elasticity. Aging also reduces nitric oxide (NO) bioavailability, resulting in vasoconstriction and reduced vascular compliance.[15] The median age of participants in both vascular elasticity groups was 20 years, with no statistically significant difference between the groups (p = 0.527). Therefore, age was not found to affect vascular elasticity in this study.

Elevated blood pressure is associated with fibrous tissue formation, resulting in thickening and reduced elasticity of vascular walls.[16] However, systolic (p = 0.202) and diastolic (p = 0.513) blood pressure did not differ significantly between the two groups. This suggests that blood pressure did not have a measurable impact on vascular elasticity in this population. These findings are in contrast to earlier research that found a significant association between hypertension and increased arterial stiffness in both young and older adults.[16,17] This discrepancy may be explained by the fact that most of the subjects in this study had blood pressure that was within the normal range, which reduced the variability required to identify meaningful differences.

Gender differences in vascular elasticity are commonly explained by differences in estrogen levels, with estrogen augmenting NO production, thereby preserving vascular function.[16] Gender distribution in this study was not significantly different between groups (p = 0.638), suggesting that gender does not influence differences in vascular elasticity.

A high BMI generally indicates greater adiposity. Excess fat tissue can cause increased blood pressure and produce pro-inflammatory agents that cause decreased vascular elasticity.[18] In the present study, BMI did not differ significantly between the vascular elasticity groups (p = 0.736). Therefore, within this study population, BMI does not affect vascular elasticity.

Dietary patterns also play a significant role in vascular health. Diets rich in antioxidants may reduce oxidative stress, whereas high salt intake can increase blood pressure and reactive oxygen species (ROS) production, while decreasing NO availability, ultimately causing vascular dysfunction.[19] Despite these associations, no significant differences in eating habits were found between groups ($p = 0.682$), suggesting that dietary habits in this study do not affect vascular elasticity. However, this result needs to be interpreted cautiously. The binary classification of eating habits into just "good" or "poor," according to the AFHC may lack the sensitivity to detect subtle but meaningful dietary influences on endothelial function or oxidative stress. A more detailed or quantitative dietary assessment, such as a food frequency questionnaire, may offer a more detailed understanding on the association between specific nutrient intakes and vascular function.

In this study, 71.7% of subjects had normal or high muscle mass, and 28.3% of subjects had low muscle mass. These findings align with a previous study, where 73.19% of subjects had normal muscle mass, 25.78% had low muscle mass, and only 0.51% had high or very high muscle mass. Variations in muscle mass can be influenced by multiple factors, including gender, age, physical activity, and dietary intake.[20]

In this study, 81.1% of participants had normal fasting blood glucose, while 18.1% were classified as having prediabetes. These results align with findings from a study at Tarumanegara University, which reported that 10.95% of participants had high blood sugar levels.[21] High fasting blood glucose levels have several risk factors, including genetics, lifestyle, cholesterol levels, and environmental factors.[22]

A significant crude association was identified between fasting blood glucose levels and vascular elasticity. Participants with low muscle mass had a 2.5 times higher risk of suboptimal vascular elasticity than those with normal or high muscle mass. A previous study employing bioelectrical impedance analysis to measure muscle mass and the cardio-ankle vascular index to assess vascular elasticity similarly identified muscle mass as an independent determinant of vascular elasticity.[23] Decreased muscle mass may contribute to insulin resistance, impaired glucose metabolism, increased oxidative stress, and chronic inflammation, each of which can reduce vascular elasticity.[24] Additionally, decreased muscle mass is associated with elevated pro-inflammatory markers, leading to endothelial dysfunction and further impaired vascular elasticity.[7] Chronic low-grade systemic inflammation linked to low muscle mass may also increase oxidative stress and endothelial dysfunction, ultimately resulting in decreased vascular elasticity.[25]

A significant crude association was also observed between fasting blood glucose and vascular elasticity. Participants with fasting blood glucose levels in the prediabetic range were at a 1.9 times higher risk of suboptimal vascular elasticity compared to those with normal range. Consistent with these findings, a cross-sectional study conducted among Caucasian populations suggested an association between elevated fasting blood glucose and reduced vascular elasticity, as assessed using baPWV and CAVI to assess vascular function.[26]

Hyperglycemia (high blood glucose level) is a cause of dyslipidemia, high triglycerides, and high free fatty acids, which amplify inflammation. It can also activate pro-inflammatory mediators such as NF-kB, IL-6, and TNF-α, leading to chronic inflammation. Hyperglycemia also increases the production of reactive oxygen species (ROS), leading to vascular dysfunction.[27] Elevated blood glucose levels also stimulate protein kinase C activation, which further worsens vascular function by enhancing vascular tone and vascular remodeling.[28]

Muscle mass was identified as the primary risk factor associated with decreased vascular elasticity, with an adjusted odds ratio of 9.656 (95% CI: 1.757—53.075). Previous studies reported similar associations, with ORs of 4.33 in males and 4.66 in females.[24] These findings reinforce the hypothesis that muscle mass has a key role in glucose metabolism and vascular health. Decreased muscle mass impairs glucose uptake, elevates inflammatory responses, and may lead to vascular remodeling and reduced elasticity.[25]

This study has some limitations that should be carefully considered. First, recruiting medical students from only one institution in a single center may have resulted in selection bias, which could have limited the diversity of subject characteristics like socioeconomic background, lifestyle, or regional health differences. This limitation was partially addressed through the use of stratified random sampling to ensure adequate representation across academic years. Second, self-reported measures, especially dietary habits, which are prone to social desirability bias and recall errors, may have introduced information bias. Standardized measurement procedures and validated questionnaires were used to reduce the risk. Third, the potential influence of unmeasured confounding factors cannot be entirely excluded, such as inflammatory biomarkers, genetic predisposition, or levels of physical activity. Future studies incorporating more comprehensive data and objective physiological parameters are warranted to strengthen causal inference.

The results are most generalizable to young, healthy, nonsmoking medical students with relatively homogeneous health profiles. Therefore, care should be taken when extrapolating to populations with pre-existing metabolic or cardiovascular conditions or to older adults. However, the findings offer important new information about the early vascular health risks in asymptomatic young adults.

CONCLUSION

Muscle mass has a greater influence on vascular elasticity than fasting blood glucose levels. After controlling for fasting blood glucose levels, low muscle mass is associated with a higher risk of suboptimal vascular elasticity than those with normal or high muscle mass. Therefore, maintaining or increasing muscle mass, in addition to controlling blood glucose levels within the normal range, is recommended as a preventive measure for preserving vascular elasticity.

In terms of policy perspective, the study emphasizes how crucial it is to support muscle mass maintenance through targeted lifestyle interventions, even in younger populations who may not yet exhibit clinical risk factors. Educational institutions, particularly medical schools, may consider integrating organized physical activity programs and nutrition education into student wellness strategies. Early preventive measures aimed at improving muscle health may serve as a cost-effective strategy for maintaining vascular function and lowering long-term cardiovascular risk.

REFERENCES

1. Maharani A, Sujarwoto, Praveen D, Oceandy D, Tampubolon G, Patel A. Cardiovascular disease risk factor prevalence and estimated 10-year cardiovascular risk scores in Indonesia: The SMARThealth Extend study. PLoS One. 2019;14(4):1–13.
2. Papaioannou TG, Protogerou AD, Stergiopulos N, Vardoulis O, Stefanadis C, Safar M, et al. Total arterial compliance estimated by a novel method and all-cause mortality in the elderly: The PROTEGER study. Age (Omaha). 2014;36(3):1555–63.
3. Szaló G, Hellgren M, Allison M, Råstam L, Lindblad U, Daka B. Longitudinal association between leisure-time physical activity and vascular elasticity indices. BMC Cardiovasc Disord [Internet]. 2021;21(1):1–8. Available from: https://doi.org/10.1186/s12872-021-01911-z
4. Kemenkes RI. Hasil Riset Kesehatan Dasar Tahun 2018. Vol. 53, Kemenkes. 2018. 1689–1699 p.
5. Rafi Faiq A, Zulhamidah Y, Widayanti E. Gambaran Sedentary Behaviour dan Indeks Massa Tubuh Mahasiswa Fakultas Kedokteran Universitas YARSI di Masa Pendidikan Tahun Pertama dan Kedua. Majalah Sainstekes [Internet]. 2019;5(2):66–73. Available from: https://academicjournal.yarsi.ac.id/index.php/sainstekes/article/view/925/546
6. Smith L, Tully M, Jacob L, Blackburn N, Adlakha D, Caserotti P, et al. The association between sedentary behavior and sarcopenia among adults aged ≥65 years in low-and middle-income countries. Int J Environ Res Public Health. 2020;17(5):1–10.

7. Aminuddin A, Noor Hashim MF, Mohd Zaberi NAS, Zheng Wei L, Ching Chu B, Jamaludin NA, et al. The Association Between Arterial Stiffness and Muscle Indices Among Healthy Subjects and Subjects With Cardiovascular Risk Factors: An Evidence-Based Review. Front Physiol. 2021;12(November).
8. Gomez-Sanchez L, Garcia-Ortiz L, Patino-Alonso MC, Recio-Rodriguez JI, Fernando R, Marti R, et al. Association of metabolic syndrome and its components with arterial stiffness in Caucasian subjects of the MARK study: A cross-sectional trial. Cardiovasc Diabetol. 2016;15(1):1–12.
9. Kobayashi R, Sato K, Sakazaki M, Nagai Y, Iwanuma S, Ohashi N, et al. Acute effects of difference in glucose intake on arterial stiffness in healthy subjects. Cardiol J. 2021;28(3):446–52.
10. Mahriani Y, Indriyanti R, Musnamirwan IA, Setiawan AS. A cross-sectional study on dietary assessment, oral hygiene behavior, and oral health status of adolescent girls. Front Nutr. 2022;9(October):1–9.
11. Guerrero-Pinedo F, Ochoa-Zárate L, Salazar CJ, Carrillo-Gómez DC, Paulo M, Flórez-Elvira LJ, et al. Association of traditional cardiovascular risk factors in adults younger than 55 years with coronary heart disease. Case-control study. SAGE Open Med. 2020;8.
12. Laksmi PW, Sukma FA, Setyohadi B, Nugroho P, Ariane A, Tirtarahardja G. The Need for a New Cut-off Value to Increase Diagnostic Performance of Bioelectrical Impedance Analysis Compared with Dual-Energy X-ray Absorptiometry to Measure Muscle Mass in Indonesian Elderly. Acta Med Indones. 2019;51(2):95–101.
13. Murakami T, Asai K, Kadono Y, Nishida T, Nakamura H, Kishima H. Assessment of arterial stiffness index calculated from accelerated photoplethysmography. Artery Res. 2019;25(1–2):37–40.
14. Ratih D, Ruhana A, Astuti N, Bahar A. Alasan pemilihan makanan dan kebiasaan mengkonsumsi makanan sehat pada mahasiswa UNESA Ketintang. Jurnal Tata Boga. 2022;11(1):22–32.
15. Vatner SF, Zhang J, Vyzas C, Mishra K, Graham RM, Vatner DE. Vascular Stiffness in Aging and Disease. Front Physiol. 2021;12(December):1–21.
16. DuPont JJ, Kenney RM, Patel AR, Jaffe IZ. Sex differences in mechanisms of arterial stiffness. Br J Pharmacol. 2019;176(21):4208–25.
17. Kim HL. Arterial stiffness and hypertension. Clin Hypertens [Internet]. 2023;29(1):1–9. Available from: https://doi.org/10.1186/s40885-023-00258-1
18. Abramowitz MK, Hall CB, Amodu A, Sharma D, Androga L, Hawkins M. Correction: Muscle mass, BMI, and mortality among adults in the United States: A population-based cohort study (PLoS ONE (2018) 13:4 (e0194697) DOI: 10.1371/journal.pone.0194697). PLoS One. 2018;13(5):1–16.
19. Leed A, Sheridan E, Baker B, Bamford S, Emmanouilidis E, Stewart F, et al. Dietary Intake and Arterial Stiffness in Children and Adolescents: A Systematic Review. Nutrients. 2023;15(9):1–29.
20. Nurfadhilah K, Surialaga S, Ibnusantosa RG. Gambaran Persentase Total Massa Otot dan Total Massa Lemak Tubuh pada Golongan Dewasa Muda. Prosiding Pendidikan Dokter. 2018;4(2):613–9.
21. Limanan D, Ciptono F. Gambaran Profil Gula Darah Sewaktu Pada Mahasiswa Kedokteran. Jurnal Serina Sains, Teknik dan Kedokteran. 2023;1(1):47–52.
22. Noventi I, Rusdianingseh R, Khafid M. Prevalensi, Karakteristik dan Faktor Resiko Prediabetes di Wilayah Pesisir, Pegunungan dan Perkotaan. Jurnal Ners dan Kebidanan (Journal of Ners and Midwifery). 2019;6(3):371–81.
23. Park HE, Chung GE, Lee H, Kim MJ, Choi SY, Lee W, et al. Significance of Low Muscle Mass on Arterial Stiffness as Measured by Cardio-Ankle Vascular Index. Front Cardiovasc Med. 2022;9(June):1–8.
24. Sampaio RAC, Sewo Sampaio PY, Yamada M, Yukutake T, Uchida MC, Tsuboyama T, et al. Arterial stiffness is associated with low skeletal muscle mass in Japanese community-dwelling older adults. Geriatr Gerontol Int. 2014;14(SUPPL.1):109–14.

25. Im IJ, Choi HJ, Jeong SM, Kim HJ, Son JS, Oh HJ. The association between muscle mass deficits and arterial stiffness in middle-aged men. Nutrition, Metabolism and Cardiovascular Diseases. 2017;27(12):1130–5.
26. Gomez-Sanchez L, Garcia-Ortiz L, Patino-Alonso MC, Recio-Rodriguez JI, Feuerbach N, Marti R, et al. Glycemic markers and relation with arterial stiffness in Caucasian subjects of the MARK study. PLoS One. 2017;12(4):1–17.
27. Kaur R, Kaur M, Singh J. Endothelial dysfunction and platelet hyperactivity in type 2 diabetes mellitus: Molecular insights and therapeutic strategies. Cardiovasc Diabetol [Internet]. 2018;17(1):1–17. Available from: https://doi.org/10.1186/s12933-018-0763-3
28. Li Y, Liu Y, Liu S, Gao M, Wang W, Chen K, et al. Diabetic vascular diseases: molecular mechanisms and therapeutic strategies. Signal Transduct Target Ther. 2023;8(1).