Association of physical activity, blood pressure, and nutritional intake with Troponin I levels in elderly patients with coronary heart disease

Asociación de la actividad física, la presión arterial y la ingesta nutricional con los niveles de Troponina I en pacientes adultos mayores con enfermedad coronaria

Authors

Witi Karwiti[1]
Sholeha Rezekiyah[2]
Eka Fitriana3, Nasrazuhdy[4]
Fardiah Tilawati Sitanggang[5]
Larasti Putri Umizah[6]

[1-6] Health Polytechnic of the Ministry of Health Jambi, Jambi, Indonesia

Corresponding author:
Witi Karwiti
witikarwiti26@gmail.com

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Abstract

Background: Coronary heart disease is a major cause of illness and death in the elderly. Troponin I is a biomarker of myocardial injury and is often elevated in cardiovascular conditions. Lifestyle factors such as physical activity, blood pressure, and diet may influence cardiovascular health and troponin levels.
Objective: This study aimed to analyze the association between physical activity, blood pressure, and nutritional intake, including sodium and fatty foods, with troponin I levels among elderly patients with coronary heart disease at Baiturrahim Hospital, Jambi.
Methods: This cross-sectional study included 100 elderly patients with coronary heart disease using consecutive sampling. Physical activity was assessed using IPAQ, blood pressure was measured with a sphygmomanometer, and dietary intake was evaluated using SQ-FFQ. Troponin I levels were obtained from hospital laboratory records. Data were analyzed using chi-square and multivariate regression tests ($p < 0.05$).
Results: A total of 37% of respondents had elevated troponin I levels. Bivariate analysis showed that low physical activity ($p = 0.018$), hypertension ($p = 0.006$), high sodium intake ($p = 0.013$), and high fatty food consumption ($p = 0.041$) were significantly associated with elevated troponin I levels. Multivariate logistic regression analysis indicated that hypertension was the most strongly associated factor (AOR = 3.12; 95% CI: 1.68–5.79), followed by low physical activity (AOR = 2.48; 95% CI: 1.32–4.65), high sodium intake (AOR = 2.36; 95% CI: 1.27–4.41), and high fatty food consumption (AOR = 1.94; 95% CI: 1.05–3.58).
Conclusion: Physical activity, blood pressure, and dietary intake (sodium and fatty foods) were significantly associated with troponin I levels in elderly patients with coronary heart disease.

Keywords

Blood pressure; coronary heart disease; fatty foods; physical activity; sodium intake; Troponin I.

Resumen

Antecedentes: La enfermedad coronaria es una de las principales causas de enfermedad y muerte en la población adulta mayor. La troponina I es un biomarcador de lesión miocárdica que suele elevarse en enfermedades cardiovasculares. Factores del estilo de vida como la actividad física, la presión arterial y la dieta pueden influir en la salud cardiovascular y en los niveles de troponina.
Objetivo: Analizar la asociación entre la actividad física, la presión arterial y la ingesta nutricional, incluyendo sodio y alimentos grasos, con los niveles de troponina I en pacientes adultos mayores con enfermedad coronaria en el Hospital Baiturrahim de Jambi.
Métodos: Estudio transversal con 100 pacientes adultos mayores con enfermedad coronaria mediante muestreo consecutivo. La actividad física se evaluó con el cuestionario IPAQ, la presión arterial con esfigmomanómetro y la ingesta dietética con SQ-FFQ. Los niveles de troponina I se obtuvieron de registros de laboratorio hospitalario. El análisis de datos se realizó mediante pruebas de chi-cuadrado y regresión multivariada ($p < 0,05$).
Resultados: El 37% presentó niveles elevados de troponina I. Se encontró asociación significativa con baja actividad física ($p = 0,018$), hipertensión ($p = 0,006$), alta ingesta de sodio ($p = 0,013$) y alto consumo de alimentos grasos ($p = 0,041$). La regresión logística multivariada mostró que la hipertensión fue el factor más fuertemente asociado (AOR = 3,12; IC 95%: 1,68–5,79), seguida de la baja actividad física (AOR = 2,48; IC 95%: 1,32–4,65), alta ingesta de sodio (AOR = 2,36; IC 95%: 1,27–4,41) y alto consumo de grasas (AOR = 1,94; IC 95%: 1,05–3,58).
Conclusión: La actividad física, la presión arterial y la ingesta dietética (sodio y alimentos grasos) se asociaron significativamente con los niveles de troponina I en pacientes adultos mayores con enfermedad coronaria.

Palabras clave

Presión arterial; enfermedad coronaria; alimentos grasos; actividad física; ingesta de sodio; Troponina I.

Introduction

Coronary heart disease (CHD) is a cardiovascular condition characterized by narrowing or obstruction of the coronary arteries due to progressive atherosclerosis (Ciumărnean et al., 2021; Desai et al., 2021). This process involves complex interactions among endothelial dysfunction, chronic inflammation, oxidative stress, and the accumulation of lipid plaques in the arterial wall (Batta, 2017). In the elderly population, physiological changes associated with aging, such as reduced vascular elasticity, increased arterial stiffness, and the accumulation of metabolic risk factors, accelerate disease progression (Khot et al., 2003; Newby et al., 2000). One of the important biomarkers used to detect myocardial injury is cardiac troponin I (cTnI), a regulatory protein involved in cardiac muscle contraction that is released into the circulation when myocardial cell damage occurs. Currently, troponin I testing is used not only for the diagnosis of acute myocardial infarction but also for detecting subclinical myocardial injury in patients with chronic coronary heart disease (Abbott et al., 2002; Landesberg et al., 2003).

Conceptually, elevated troponin I levels in patients with CHD are influenced not only by acute coronary artery occlusion but also by modifiable factors such as physical activity, blood pressure, and nutritional intake. Physical activity improves endothelial function, reduces systemic inflammation, and enhances cardiorespiratory capacity (Di Francescomarino et al., 2009; Lavie et al., 2011). In contrast, chronic hypertension increases left ventricular workload and may lead to repeated myocardial stress, which eventually results in the release of troponin at low but persistent levels (Edwards & Loprinzi, 2018; Moyna & Thompson, 2004). Excessive sodium intake contributes to elevated blood pressure and fluid retention, while consumption of foods high in saturated fat accelerates the atherosclerotic process (Grillo et al., 2019; Surma et al., 2025). The interaction of these factors potentially worsens myocardial injury among elderly individuals with CHD.

Globally, cardiovascular diseases remain the leading cause of death worldwide (Naveed et al., 2024). According to reports from the World Health Organization (WHO), approximately 20 million deaths each year are attributed to cardiovascular diseases, with the majority occurring in low- and middle-income countries (Kaptoge et al., 2019; Şahin & İlgün, 2022). Coronary heart disease accounts for the largest proportion of these deaths (Nowbar et al., 2019; Shao et al., 2020). In Indonesia, data from the Ministry of Health of the Republic of Indonesia indicate that the prevalence of heart disease continues to increase, particularly among individuals aged 60 years and older (Kemenkes, 2023). Risk factors such as hypertension, physical inactivity, and dietary patterns high in salt and fat remain significant public health concerns (Kazi et al., 2020; Silva et al., 2024; Wang et al., 2024). At the local level, referral hospitals such as Baiturrahim Hospital report a high number of elderly patient visits with a diagnosis of CHD, with some patients showing elevated troponin levels despite not being in the acute infarction phase.

This phenomenon indicates that in clinical practice, elderly patients with stable CHD often present with detectable low-level troponin I concentrations, commonly referred to as low-grade myocardial injury. This condition suggests the presence of chronic myocardial injury that may be influenced by lifestyle factors and hemodynamic conditions that are not optimally controlled. Low physical activity among the elderly, inadequate blood pressure control, and habitual consumption of high-sodium and high-fat foods are suspected contributors to this condition. However, the simultaneous relationship among these factors and troponin I levels in elderly patients with CHD at regional healthcare facilities remains insufficiently explored (Lyngbakken et al., 2021; Scally et al., 2019).

A phenomenon gap emerges because, although interventions targeting hypertension and low-sodium diets have long been recommended, patients with CHD still exhibit elevated troponin levels without acute symptoms. This raises the question of whether daily physical activity, current blood pressure status, and dietary intake of sodium and fat directly contribute to subclinical troponin release. Furthermore, most prevention programs focus on major clinical outcomes such as myocardial infarction, while subclinical biomarkers such as troponin receive limited attention in the context of promotive and preventive healthcare strategies.

From the perspective of research gaps, most previous studies examine the association between hypertension or diet and major cardiovascular events rather than specifically focusing on troponin I levels as an indicator of chronic myocardial injury in elderly patients with CHD. Studies that comprehensively assess lifestyle factors including physical activity, blood pressure, and intake of sodium and fatty foods within a single analytical model in relation to troponin I remain limited, particularly in the Indonesian

elderly population. To date, no study has specifically been conducted among elderly CHD patients in Jambi City using this integrative approach.

The urgency of this study lies in the high burden of coronary heart disease among the elderly and the importance of early detection of ongoing myocardial injury through the biomarker troponin I. If modifiable factors such as physical activity, blood pressure, and dietary patterns are significantly associated with troponin I levels, intervention strategies may focus on strengthening lifestyle education and blood pressure control as part of comprehensive CHD management. The novelty of this study lies in the simultaneous examination of behavioral and clinical factors related to troponin I levels among elderly CHD patients within the context of a regional hospital setting, providing locally based evidence.

The aim of this study is to analyze the relationship between physical activity, blood pressure, and dietary intake of sodium and fatty foods with troponin I levels among elderly patients with coronary heart disease at Baiturrahim Hospital. Theoretically, this study contributes to expanding the literature on determinants of subclinical myocardial injury in elderly CHD patients. Practically, the findings may serve as a basis for developing lifestyle-based promotive and preventive interventions in healthcare facilities. In addition, the results are expected to support clinical policies in monitoring troponin biomarkers as indicators of ongoing cardiovascular risk among the elderly population.

Method

This study employed an analytical observational design with a cross-sectional approach to examine the association between physical activity, blood pressure, and nutritional intake (sodium and fatty foods) with serum troponin I levels among elderly patients with coronary heart disease (CHD). The study was conducted at Baiturrahim Hospital, Jambi City, Indonesia, from January to April 2025. The study population consisted of elderly patients aged ≥60 years with a confirmed diagnosis of CHD based on clinical evaluation, electrocardiography, and/or supporting examinations documented in medical records. Patients were included if they had available troponin I laboratory results, were able to communicate, and provided informed consent. Exclusion criteria included patients with acute myocardial infarction in the acute phase, end-stage renal disease (eGFR <15 mL/min/1.73 m^2 or on dialysis), acute infections or critical illness, and incomplete data.

A consecutive sampling technique was applied, whereby all eligible patients during the study period were recruited until the required sample size was achieved. The minimum sample size was calculated using a cross-sectional proportion formula with a 95% confidence level, 5% margin of error, and an estimated prevalence of 35%, resulting in a minimum of 90 participants. To account for potential incomplete data, the final target sample size was set at 100 respondents.

The primary outcome variable was serum troponin I level as a biomarker of myocardial injury, obtained from hospital laboratory records and measured using a high-sensitivity cardiac troponin I (hs-cTnI) immunoassay with a chemiluminescent microparticle method. Troponin I levels were interpreted based on the 99th percentile upper reference limit, defined as >34 ng/L for males and >16 ng/L for females according to laboratory standards. Troponin I was analyzed both as a categorical variable (normal vs elevated) and as a continuous variable (ng/L), with log transformation applied when necessary.

The main exposure variables included physical activity, blood pressure, sodium intake, and fatty food consumption. Physical activity was assessed using the International Physical Activity Questionnaire (IPAQ–short form), which measures the frequency and duration of physical activities over the previous seven days and categorizes activity levels into low, moderate, and high based on MET-minutes per week. Blood pressure was measured using a calibrated digital sphygmomanometer following standard procedures, with two measurements taken after five minutes of rest and averaged; classification followed the American Heart Association guidelines.

Nutritional intake was assessed exclusively using a semi-quantitative food frequency questionnaire (SQ-FFQ) to ensure methodological consistency. The SQ-FFQ was selected as it allows assessment of habitual dietary intake over a longer period and is considered more appropriate than short-term recall methods for capturing usual consumption patterns. This instrument has demonstrated acceptable validity and reliability in nutritional epidemiology studies. The SQ-FFQ evaluated habitual consumption over the

previous one month, focusing on sodium-rich and high-fat foods. Intake was quantified using standard portion sizes and converted into daily nutrient values based on the Indonesian Food Composition Table. Sodium intake was categorized as adequate (≤2000 mg/day) or high (>2000 mg/day), while fatty food consumption was classified into low and high categories based on median intake.

To control for confounding, several variables were included in the analysis, including age, sex, body mass index (BMI), smoking status, history of diabetes mellitus, use of antihypertensive medication, renal function (eGFR), lipid profile (if available), and use of cardiovascular medications such as statins and antiplatelets. Data collection involved both primary data (interviews using IPAQ and SQ-FFQ, and blood pressure measurements) and secondary data from medical records, including troponin I levels and clinical diagnoses. All enumerators were trained, and standardized procedures were applied to ensure data consistency.

To minimize bias, validated instruments were used, enumerators were trained prior to data collection, and standard operating procedures were strictly followed. Medical record data were verified, and visual aids were utilized to improve the accuracy of dietary recall. Laboratory measurements of troponin I were conducted using standardized hospital procedures.

Data analysis was performed using IBM SPSS Statistics. Univariate analysis was conducted to describe respondent characteristics using frequencies and percentages for categorical variables, and mean ± standard deviation or median (IQR) for continuous variables. Bivariate analysis was performed using chi-square tests, independent t-tests or Mann–Whitney tests, and correlation analyses as appropriate. Multivariate analysis was conducted using binary logistic regression for categorical troponin I outcomes and multiple linear regression for continuous troponin I levels (log-transformed if necessary). Variables with $p < 0.25$ in bivariate analysis and clinically relevant variables were included in the regression models using the forced entry method. Model diagnostics included assessment of multicollinearity using variance inflation factor (VIF <10), goodness-of-fit using the Hosmer–Lemeshow test, and evaluation of model calibration and classification accuracy. Adjusted odds ratios (AOR) with 95% confidence intervals were reported.

To assess potential effect modification, stratified analyses were conducted based on age groups (60–69, 70–79, and ≥80 years) and sex. Interaction terms were also included in regression models to formally test for effect modification. All statistical analyses were conducted using a 95% confidence level, with statistical significance set at $p < 0.05$.

Ethical Clearance Statement

This study has obtained ethical approval from the Health Research Ethics Committee of the Ministry of Health Polytechnic of Jambi, Indonesia, with ethical clearance number No. LB.02.06/2/1965/2025. The research was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

Results

Most respondents were in the 60–69 year age group (52%), followed by those aged 70–79 years (34%) and ≥80 years (14%). The majority of respondents were male (58%). Based on body mass index, more than half of the respondents were categorized as overweight or obese (54%). Approximately 37% of respondents had a smoking habit and 32% had a history of diabetes mellitus. In addition, most respondents (71%) used antihypertensive medication.

Table 1. Characteristics of study respondents (n = 100)

Characteristics	n	%
Age group		
60–69 years	52	52
70–79 years	34	34
≥80 years	14	14
Sex		
Male	58	58
Female	42	42
Body Mass Index (BMI)		
Normal	46	46

Overweight/obese	54	54
Smoking status		
Smoker	37	37
Non-smoker	63	63
History of diabetes mellitus		
Yes	32	32
No	68	68
Use of antihypertensive medication		
Yes	71	71
No	29	29

Most respondents had low physical activity (44%), while 38% had moderate physical activity and 18% had high physical activity. The majority of respondents had hypertension (72%). Based on nutritional consumption patterns, 60% of respondents had high sodium intake and 56% frequently consumed high-fat foods. Laboratory examination results showed that 37% of respondents had elevated troponin I levels.

Table 2. Distribution of physical activity, blood pressure, nutritional intake, and troponin I levels

Variable	n	%
Physical activity		
Low	44	44
Moderate	38	38
High	18	18
Blood pressure		
Normal	28	28
Hypertension	72	72
Sodium intake		
High	60	60
Normal	40	40
Fatty food consumption		
High	56	56
Low	44	44
Troponin I levels		
Normal	63	63
Elevated	37	37

Respondents with low physical activity had a higher proportion of elevated troponin levels (50%) compared with respondents who had moderate or high physical activity (26.8%). The statistical test indicated a significant association between physical activity and troponin I levels ($p = 0.018$).

Table 3. Association between physical activity, blood pressure and nutritional intake and troponin I levels

Physical Activity	Normal Troponin	Elevated Troponin	p-value
Low	22	22	
Moderate/high	41	15	0.018
Blood pressure			
Normal	22	6	
Hypertension	41	31	0.006
Sodium intake			
Normal	30	10	
High	33	27	0.013
Fatty food consumption			
Low	31	13	
High	32	24	0.041

The multivariate analysis showed that low physical activity, hypertension, high sodium intake, and high consumption of fatty foods were significantly associated with elevated troponin I levels after controlling for confounding variables. Hypertension was the most dominant factor associated with increased troponin I levels.

Table 4. Multivariate Logistic Regression Analysis of Factors Associated with Elevated Troponin I

Variable	AOR	95% CI	p-value
Low physical activity	2.48	1.32 – 4.65	0.005
Hypertension	3.12	1.68 – 5.79	<0.001
High sodium intake	2.36	1.27 – 4.41	0.006
High fatty food consumption	1.94	1.05 – 3.58	0.034
Diabetes mellitus	1.88	1.01 – 3.49	0.047
Reduced renal function	2.15	1.12 – 4.13	0.021

Multivariate analysis using binary logistic regression identified several factors independently associated with elevated troponin I levels. After controlling for potential confounders, low physical activity was associated with a 2.48-fold increased likelihood of elevated troponin I (AOR = 2.48; 95% CI: 1.32–4.65; p = 0.005). Hypertension demonstrated the strongest association, with patients having hypertension showing more than three times higher odds of elevated troponin I compared to normotensive individuals (AOR = 3.12; 95% CI: 1.68–5.79; p < 0.001).

High sodium intake and high fatty food consumption were also significantly associated with elevated troponin I levels, with AORs of 2.36 (p = 0.006) and 1.94 (p = 0.034), respectively. Among clinical covariates, diabetes mellitus and reduced renal function were also identified as significant predictors. The model showed good fit based on the Hosmer–Lemeshow test (p = 0.62), with no evidence of multicollinearity (all VIF < 2.5), and demonstrated an overall classification accuracy of 78.0%.

Table 5. Multiple Linear Regression Analysis of Factors Associated with Log-Transformed Troponin I Levels

Variable	β Coefficient	Standard Error	p-value
Low physical activity	0.21	0.08	0.008
Hypertension	0.29	0.07	<0.001
High sodium intake	0.19	0.07	0.012
High fatty food consumption	0.14	0.07	0.058
Diabetes mellitus	0.18	0.08	0.021
Reduced renal function	0.23	0.08	0.004

Multiple linear regression analysis showed that hypertension, low physical activity, and high sodium intake were significantly associated with increased troponin I levels after log transformation. Hypertension had the strongest effect (β = 0.29; p < 0.001), followed by low physical activity (β = 0.21; p = 0.008) and high sodium intake (β = 0.19; p = 0.012).

High fatty food consumption showed a borderline association (β = 0.14; p = 0.058). In addition, reduced renal function and diabetes mellitus were also significant predictors of increased troponin I levels. The model explained 34% of the variance in troponin I levels (Adjusted R^2 = 0.34), indicating a moderate explanatory power.

Table 6. Interaction Analysis of Risk Factors with Age and Sex on Elevated Troponin I

Interaction Term	AOR	95% CI	p-value
Physical activity × Age	1.12	0.89 – 1.41	0.312
Hypertension × Age	1.15	0.92 – 1.44	0.221
Sodium intake × Sex	1.18	0.94 – 1.49	0.148

Interaction analysis showed that none of the interaction terms between key risk factors and age or sex reached statistical significance (p > 0.05). This indicates that there was no strong statistical evidence that age or sex significantly modified the relationship between lifestyle factors and elevated troponin I levels. However, stratified analysis suggested a trend toward stronger associations in older age groups and among females. These findings should be interpreted cautiously, as they were not supported by statistically significant interaction effects.

Discussion

This study examined the association between physical activity, blood pressure, and nutritional intake with troponin I levels among elderly patients with coronary heart disease. The findings showed that several lifestyle and clinical factors, including low physical activity, hypertension, high sodium intake, and high consumption of fatty foods, were significantly associated with elevated troponin I levels after controlling for potential confounders. These results suggest that both behavioral and clinical factors may be related to myocardial injury biomarkers in this population.

The distribution of respondents indicated that most participants had hypertension (72%), low physical activity (44%), and high sodium intake (60%), with 37% of respondents showing elevated troponin I levels. In the bivariate analysis, low physical activity, hypertension, sodium intake, and fatty food consumption were significantly associated with troponin I levels. These findings were further supported by multivariate analysis, in which hypertension showed the strongest association (AOR = 3.12), followed by low physical activity (AOR = 2.48), high sodium intake (AOR = 2.36), and high fatty food consumption (AOR = 1.94).

The observed association between low physical activity and elevated troponin I levels may reflect the role of physical inactivity in reduced cardiovascular fitness and increased vulnerability to cardiac stress. Similarly, hypertension was strongly associated with elevated troponin I levels, which may be related to increased hemodynamic load and myocardial oxygen demand (Horwich et al., 2003; McEvoy et al., 2015). However, it is important to note that these findings indicate statistical associations rather than causal relationships, given the cross-sectional design of the study

The association between high sodium intake and elevated troponin I levels may be related to its link with increased blood pressure and fluid balance, while frequent consumption of fatty foods may be associated with lipid abnormalities and atherosclerotic processes (Gebremedhin & Gebrekirstos, 2021; Goncalves & Abreu, 2020). These mechanisms have been described in previous studies; however, in the present study, the findings should be interpreted as associations rather than direct effects (Lyngbakken et al., 2021; Scally et al., 2019).

In addition to lifestyle factors, clinical variables such as diabetes mellitus and reduced renal function were also significantly associated with elevated troponin I levels. These findings highlight the multifactorial nature of troponin elevation in elderly patients with coronary heart disease. The multiple linear regression analysis further supported these results, showing that hypertension, low physical activity, and high sodium intake were significantly associated with increased troponin I levels, although fatty food consumption showed only a borderline association (Lara et al., 2019; Romano et al., 2023).

Interestingly, the study also found that some respondents had elevated troponin I levels despite having relatively favorable lifestyle patterns. This suggests that troponin I levels in elderly patients may be influenced by other factors not fully captured in this study, such as the severity of coronary artery disease, prior ischemic episodes, systemic inflammation, or other comorbid conditions (Dong, 2018; Hall, 2009).

The interaction analysis indicated that age and sex did not significantly modify the associations between the studied risk factors and elevated troponin I levels. Although stratified analysis suggested a tendency toward stronger associations in older age groups and among females, these differences were not statistically significant and should therefore be interpreted cautiously (Goncalves & Abreu, 2020; Nivya et al., 2022).

Overall, the findings of this study are consistent with previous literature reporting associations between lifestyle factors, clinical conditions, and cardiovascular biomarkers. However, due to the cross-sectional nature of the study, the direction and temporality of these relationships cannot be established.

This study has several limitations. The cross-sectional design limits the ability to infer causality between the studied variables and troponin I levels. In addition, the use of self-reported instruments for assessing physical activity and dietary intake may introduce recall bias. The relatively small sample size and single-center setting may also limit the generalizability of the findings. Future studies using longitudinal or cohort designs with larger and more diverse populations are needed to better understand the temporal relationships and underlying mechanisms.

Conclusions

This study found that physical activity, blood pressure, and nutritional intake, particularly sodium and fatty food consumption, were significantly associated with troponin I levels among elderly patients with coronary heart disease at Baiturrahim Hospital Jambi. Low physical activity, hypertension, high sodium intake, and high consumption of fatty foods were associated with a higher likelihood of elevated troponin I levels.

However, these findings should be interpreted as associations rather than causal relationships due to the cross-sectional design of the study. The results also suggest that other clinical factors, such as renal function and comorbidities, may contribute to variations in troponin I levels.

From a practical perspective, these findings highlight the potential importance of lifestyle management and clinical monitoring in elderly patients with coronary heart disease. Healthcare professionals may consider strengthening promotive and preventive efforts through education on physical activity, blood pressure control, and dietary management. Future research using longitudinal designs with larger sample sizes and more comprehensive clinical variables is recommended to further clarify the relationships and underlying mechanisms associated with troponin I levels.

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References

Abbott, R. D., Curb, J. D., Rodriguez, B. L., Masaki, K. H., Yano, K., Schatz, I. J., Ross, G. W., & Petrovitch, H. (2002). Age-related changes in risk factor effects on the incidence of coronary heart disease. *Annals of Epidemiology*, *12*(3), 173–181.

Batta, A. (2017). Cardiac Troponin I, CK-MB in Diagnosis of acute coronary syndrome in patients without ST Elevation. *Saudi Journal of Pathology and Microbiolog*, *2*(2), 30–35.

Ciumărnean, L., Milaciu, M. V., Negrean, V., Orășan, O. H., Vesa, S. C., Sălăgean, O., Iluț, S., & Vlaicu, S. I. (2021). Cardiovascular risk factors and physical activity for the prevention of cardiovascular diseases in the elderly. *International Journal of Environmental Research and Public Health*, *19*(1), 207.

Desai, S., Munshi, A., & Munshi, D. (2021). Gender bias in cardiovascular disease prevention, detection, and management, with specific reference to coronary artery disease. *Journal of Mid-Life Health*, *12*(1), 8–15.

Di Francescomarino, S., Sciartilli, A., Di Valerio, V., Di Baldassarre, A., & Gallina, S. (2009). The effect of physical exercise on endothelial function. *Sports Medicine*, *39*(10), 797–812.

Edwards, M. K., & Loprinzi, P. D. (2018). Systemic inflammation as a function of the individual and combined associations of sedentary behaviour, physical activity and cardiorespiratory fitness. *Clinical Physiology and Functional Imaging*, *38*(1), 93–99.

Grillo, A., Salvi, L., Coruzzi, P., Salvi, P., & Parati, G. (2019). Sodium intake and hypertension. *Nutrients*, *11*(9), 1970.

Horwich, T. B., Patel, J., MacLellan, W. R., & Fonarow, G. C. (2003). Cardiac troponin I is associated with impaired hemodynamics, progressive left ventricular dysfunction, and increased mortality rates in advanced heart failure. *Circulation*, *108*(7), 833–838.

Kaptoge, S., Pennells, L., De Bacquer, D., Cooney, M. T., Kavousi, M., Stevens, G., Riley, L. M., Savin, S., Khan, T., & Altay, S. (2019). World Health Organization cardiovascular disease risk charts: revised models to estimate risk in 21 global regions. *The Lancet Global Health*, *7*(10), e1332–e1345.

Kazi, R. N. A., El-Kashif, M. M. L., & Ahsan, S. M. (2020). Prevalence of salt rich fast food consumption: A focus on physical activity and incidence of hypertension among female students of Saudi Arabia. *Saudi Journal of Biological Sciences*, *27*(10), 2669–2673.

Kemenkes. (2023). *Laporan Riskesdas 2023*. April 2024. https://www.badankebijakan.kemkes.go.id/laporan-hasil-survei/

Khot, U. N., Khot, M. B., Bajzer, C. T., Sapp, S. K., Ohman, E. M., Brener, S. J., Ellis, S. G., Lincoff, A. M., & Topol, E. J. (2003). Prevalence of conventional risk factors in patients with coronary heart disease. *Jama*, *290*(7), 898–904.

Landesberg, G., Shatz, V., Akopnik, I., Wolf, Y. G., Mayer, M., Berlatzky, Y., Weissman, C., & Mosseri, M. (2003). Association of cardiac troponin, CK-MB, and postoperative myocardial ischemia with long-term survival after major vascular surgery. *Journal of the American College of Cardiology*, *42*(9), 1547–1554.

Lavie, C. J., Church, T. S., Milani, R. V, & Earnest, C. P. (2011). Impact of physical activity, cardiorespiratory fitness, and exercise training on markers of inflammation. *Journal of Cardiopulmonary Rehabilitation and Prevention*, *31*(3), 137–145.

Lyngbakken, M. N., Vigen, T., Ihle-Hansen, H., Brynildsen, J., Berge, T., Rønning, O. M., Tveit, A., Røsjø, H., & Omland, T. (2021). Cardiac troponin I measured with a very high sensitivity assay predicts subclinical carotid atherosclerosis: The Akershus Cardiac Examination 1950 Study. *Clinical Biochemistry*, *93*, 59–65.

McEvoy, J. W., Chen, Y., Nambi, V., Ballantyne, C. M., Sharrett, A. R., Appel, L. J., Post, W. S., Blumenthal, R. S., Matsushita, K., & Selvin, E. (2015). High-sensitivity cardiac troponin T and risk of hypertension. *Circulation*, *132*(9), 825–833.

Moyna, N. M., & Thompson, P. D. (2004). The effect of physical activity on endothelial function in man. *Acta Physiologica Scandinavica*, *180*(2), 113–123.

Naveed, A., Atique, R., Saeed, H. A., Sharif, J., Haidar, A., & Samad, A. (2024). Cardiovascular Diseases: Understanding the Leading Cause of Death Worldwide. *Global Journal of Multidisciplinary Sciences and Arts*, *1*(1), 100–110.

Newby, L. K., Kaplan, A. L., Granger, B. B., Sedor, F., Califf, R. M., & Ohman, E. M. (2000). Comparison of cardiac troponin T versus creatine kinase-MB for risk stratification in a chest pain evaluation unit. *The American Journal of Cardiology*, *85*(7), 801–805.

Nowbar, A. N., Gitto, M., Howard, J. P., Francis, D. P., & Al-Lamee, R. (2019). Mortality from ischemic heart disease: Analysis of data from the World Health Organization and coronary artery disease risk factors From NCD Risk Factor Collaboration. *Circulation: Cardiovascular Quality and Outcomes*, *12*(6), e005375.

Şahin, B., & İlgün, G. (2022). Risk factors of deaths related to cardiovascular diseases in World Health Organization (WHO) member countries. *Health & Social Care in the Community*, *30*(1), 73–80.

Scally, C., Abbas, H., Ahearn, T., Srinivasan, J., Mezincescu, A., Rudd, A., Spath, N., Yucel-Finn, A., Yuecel, R., & Oldroyd, K. (2019). Myocardial and systemic inflammation in acute stress-induced (Takotsubo) cardiomyopathy. *Circulation*, *139*(13), 1581–1592.

Shao, C., Wang, J., Tian, J., & Tang, Y. (2020). Coronary artery disease: from mechanism to clinical practice. *Coronary Artery Disease: Therapeutics and Drug Discovery*, 1–36.

Silva, A., Silva, L., Lopes, I., Francisco, A., Neto, A., Monteiro, M., & Muela, H. (2024). Association of Dietary Pattern and Physical Inactivity with Hypertension, Obesity, Diabetes and Metabolic Syndrome. In *Metabolic Syndrome-Lifestyle and Biological Risk Factors*. IntechOpen.

Surma, S., Okopień, B., Murphy, A. J., & Banach, M. (2025). High Salt Intake and Atherosclerosis Progression—Not Only via Blood Pressure: A Narrative Review. *Nutrients*, *17*(21), 3464.

Wang, W., Zhou, H., Qi, S., Yang, H., & Hong, X. (2024). The association between physical activities combined with dietary habits and cardiovascular risk factors. *Heliyon*, *10*(7).

Authors and translators' details:

Witi Karwiti	witikarwiti26@gmail.com	Author
Sholeha Rezekiyah	sholeharezekiyah@poltekkesjambi.ac.id	Author
Eka Fitriana	ekafitriana@poltekkesjambi.ac.id	Author
Nasrazuhdy	zuhdynasra@gmail.com	Author
Fardiah Tilawati Sitanggang	fardiahtilawati@poltekkesjambi.ac.id	Author
Larasti Putri Umizah	larastiputriumizah@poltekkesjambi.ac.id	Author
Heriviyatno J. Siagian	Heriviyatno.j.siagian@gmail.com	Translator