Biomedical Journal of Indonesia
Journal Homepage: https://bji-fk.ejournal.unsri.ac.id

# The Role of Diabetes Mellitus Panel on Kidney Function Decline in Elderly Population: Cross-Sectional Study

**Shirly Gunawan[1*], Robert Kosasih[1], Yohanes Firmansyah[2], Alexander Halim Santoso[3], Bryan Anna Wijaya[4], Kresna Bambang[5], Farhan Pratomo[5]**

[1]Department of Pharmacology, Faculty of Medicine, Universitas Tarumanagara, Jakarta, Indonesia
[2]Department of Physiology, Faculty of Medicine, Universitas Tarumanagara, Jakarta, Indonesia
[3]Department of Nutrition, Faculty of Medicine, Universitas Tarumanagara, Jakarta, Indonesia
[4]Medical Doctor Programme, Faculty of Medicine, Universitas Tarumanagara, Jakarta, Indonesia
[5]Bachelor in Medicine Programme, Faculty of Medicine, Universitas Tarumanagara, Jakarta, Indonesia

**ARTICLE INFO**

**Keywords:**
Chronic kidney disease
Diabetes mellitus
Elderly
Estimated glomerular filtration rate
Insulin resistance

**Corresponding author:**
Shirly Gunawan
**E-mail address:**
shirlyg@fk.untar.ac.id

All authors have reviewed and approved the final version of the manuscript.

*https://doi.org/10.32539/BJI.v12i1.293*

Received 7 September 2025;
Accepted 4 November 2025

**ABSTRACT**

**Introduction.** Chronic kidney disease (CKD) is a major public health issue, particularly among the elderly, where metabolic disturbances accelerate renal decline. Diabetes mellitus contributes to kidney dysfunction through insulin resistance and altered glycemic control. This study investigates the relationship between diabetes-related parameters—fasting blood glucose, HbA1c, fasting insulin, HOMA-IR, and kidney function, as measured by estimated glomerular filtration rate (eGFR) in an elderly population. **Methods.** A cross-sectional analytic study was conducted among 82 elderly residents of Bina Bhakti Nursing Home. Venous blood samples were collected to assess fasting blood glucose, HbA1c, fasting insulin, and HOMA-IR. Kidney function was estimated using the CKD-EPI equation. **Results.** Age showed a significant negative correlation with eGFR ($r = -0.266$, $p = 0.016$), indicating age-related renal decline. HOMA-IR ($r = 0.247$, $p = 0.025$) and fasting insulin ($r = 0.224$, $p = 0.043$) demonstrated weak but significant positive correlations with eGFR. Conversely, fasting glucose ($r = 0.055$, $p = 0.623$) and HbA1c ($r = 0.029$, $p = 0.795$) had negligible, non-significant correlations with eGFR, suggesting limited predictive value for renal impairment. Insulin growth factor exhibited a weak, non-significant negative correlation ($r = -0.104$, $p = 0.355$). **Conclusion.** Age remains a key determinant of declining kidney function in the elderly. Insulin resistance markers showed mild but unexpected associations, warranting further longitudinal studies to confirm these findings and explore underlying mechanisms.

## 1. Introduction

Chronic kidney disease (CKD) is a major global health issue, affecting between 8% and 16% of the population and impacting over 697 million people worldwide. It significantly contributes to morbidity and mortality, particularly among older adults, who naturally experience a decline in kidney function with age. On average, the glomerular filtration rate (GFR) declines by approximately 0.75 to 1 mL/min per year after age 40 to 50. However, variations exist due to genetic factors, lifestyle choices, and coexisting medical conditions. CKD is diagnosed when kidney damage or an estimated glomerular filtration rate (eGFR) below 60 mL/min/1.73 $m^2$ persists for at least three months, regardless of the cause. Type 2 diabetes remains the most common underlying condition associated with CKD, accounting for 30% to 50% of cases.[1–3]

Uncontrolled diabetes is a major contributor to diabetic kidney disease (DKD), with persistent hyperglycemia triggering a cascade of pathological processes that accelerate renal function decline. The progression of DKD is driven by a complex interplay of metabolic, hemodynamic, inflammatory, and fibrotic pathways, all of which become dysregulated in diabetes. These interconnected mechanisms influence gene expression, activate transcription factors, and disrupt molecular signaling, leading to the kidneys' progressive structural and functional damage. Over time, these pathological changes result in persistent declining renal function.[4–6]

Understanding the role of diabetes panel parameters in kidney function decline is essential for assessing the health status of the elderly population. Although the relationship between insulin resistance and renal impairment has been reported in various

settings, data from institutionalized elderly populations in Indonesia remain scarce. This study, therefore, examines the associations between fasting blood glucose, HbA1c, fasting insulin, HOMA-IR, and estimated glomerular filtration rate (eGFR) in a nursing home. By analyzing both insulin-related markers and conventional glycemic indicators, this research provides preliminary, context-specific evidence that may help refine the assessment of renal health in elderly Indonesians and guide future longitudinal investigations.

## 2. Methods

This study employed a cross-sectional analytic design involving 82 elderly individuals residing at Bina Bhakti Nursing Home, which was selected through total sampling in 2024. Participants were eligible if they were 60 or older, provided informed consent, and agreed to venous blood sampling for laboratory analysis. Individuals were excluded if they had severe chronic kidney disease (CKD), an estimated glomerular filtration rate (eGFR) below 15 mL/min/1.73 $m^2$, or were undergoing dialysis. Additionally, those who had experienced recent medication adjustments that could influence blood glucose or kidney function, including SGLT2 inhibitors, angiotensin converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), non-steroid anti-inflammatory drugs (NSAIDs), and corticosteroids within the past three months, were excluded to minimize confounding variables. This research was approved by the Research Ethics Committee of Tarumanagara University under reference number 013-UTHREC/UNTAR/VI/2024.

This study evaluates kidney function decline in the elderly population by assessing the estimated glomerular filtration rate (eGFR) and key diabetes mellitus parameters. The diabetes panel was measured through venous blood sampling, conducted under strict laboratory protocols to ensure accuracy and reliability. The metabolic profile was analyzed using fasting blood glucose (mg/dL), fasting insulin (µU/mL), HbA1c (%), the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR), and insulin growth factor, providing a comprehensive assessment of glucose metabolism and insulin sensitivity. Additionally, eGFR was determined based on serum creatinine levels and calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation, a validated method for estimating renal function in clinical and research settings.

Data analysis for this study used Statistical Package for the Social Sciences (SPSS) version 26 to process univariate and bivariate quantitative data. The Kolmogorov-Smirnov test was used to assess data normality. Spearman's correlation test was applied to examine the relationship between diabetes panel parameters (fasting blood glucose, HbA1c, fasting insulin, and HOMA-IR) and kidney function decline, measured by estimated glomerular filtration rate (eGFR). Statistical significance was set at $p < 0.05$. Correlation strength was classified as negligible (0.00–0.10), weak (0.10–0.39), moderate (0.40–0.69), strong (0.70–0.89), and very strong (0.90–1.00). The demographic and clinical characteristics of the respondents were presented as mean values with standard deviations, as well as the median, minimum, and maximum values, to provide a comprehensive overview of the study population.

## 3. Results

The study included 82 elderly participants who met the inclusion criteria, with a mean age of 74.34 years (±8.13). The sample comprised 18 males (22%) and 64 females (78%). The mean fasting blood sugar level was 87.71 mg/dL (±16.53), while the average HbA1c was 7.62% (±1.43). Fasting insulin levels had a mean of 4.80 µU/mL (±1.79), and HOMA-IR was recorded at 1.03 (±0.43).

The mean serum creatinine was 1.05 mg/dL (±0.31), and the estimated glomerular filtration rate (eGFR) had a mean of 62.53 mL/min/1.73 $m^2$ (±20.19). (Table 1) The Kolmogorov-Smirnov test assessed data normality, revealing that the variables did not follow a normal distribution.

**Table 1. Respondent characteristics**

| Parameter | N (%) | Mean (SD) | Med (Min-Max) |
|---|---|---|---|
| Gender | | | |
| Male | 18 (22) | | |
| Female | 64 (78) | | |
| Age (years) | | 74.34 (8.13) | 75 (61 – 97) |
| Fasting blood sugar | | 87.71 (16.53) | 86 (63 – 166) |
| HbA1c, % | | 7.62 (1.43) | 7.45 (4.7 – 12.9) |
| Fasting insulin | | 4.80 (1.79) | 4.33 (2.12 – 11.1) |
| HOMA-IR | | 1.03 (0.43) | 0.93 (0.43 – 2.77) |
| Insulin Growth Factor | | 14.43 (14.25) | 12.11 (1.19 – 102.7) |
| Creatinin | | 1.05 (0.31) | 1 (0.5 – 2.1) |
| e-Glomerular Filtration Rate | | 62.53 (20.19) | 59.35 (22.8 – 128.5) |

**Table 2. Correlation of diabetes panel parameters on kidney function decline**

| Parameter N = 82 respondent | Glomerular Filration Rate (GFR) | |
|---|---|---|
| | r-correlation | *p-value* |
| Age, years | -0.266 | 0.016 |
| HOMA-IR | 0.247 | 0.025 |
| Fasting Blood Glucose, mg/dL | 0.055 | 0.623 |
| HbA1c, % | 0.029 | 0.795 |
| Fasting Insulin, µIU/mL | 0.224 | 0.043 |
| Insulin Growth Factor | -0.104 | 0.355 |

**Table 3. Correlation of diabetes panel parameters on kidney function decline with age as a controlling factor**

| Controlling Factor | Parameter N = 82 respondents | Glomerular Filtration Rate (GFR) | |
|---|---|---|---|
| | | r-correlation | *p-value* |
| Age, years | HOMA-IR | 0.137 | 0.223 |
| | Fasting Blood Glucose, mg/dL | 0.004 | 0.969 |
| | HbA1c, % | 0.007 | 0.951 |
| | Fasting Insulin, µIU/mL | 0.160 | 0.153 |
| | Insulin Growth Factor | -0.142 | 0.205 |

Age showed a significant inverse correlation with GFR ($r = -0.266$, $p = 0.016$), suggesting that advancing age is associated with declining kidney function. Conversely, HOMA-IR and fasting insulin showed weak positive correlations with GFR, indicating a potential link between insulin sensitivity and renal health. However, fasting blood glucose and HbA1c had negligible and statistically insignificant correlations with GFR, reflecting their limited predictive value in this context. HOMA-IR, a marker of insulin resistance, significantly correlated with GFR ($r = 0.247$, $p = 0.025$). However, after adjusting for age, the correlation of HOMA-IR and GFR becomes statistically insignificant. This finding challenges the notion that insulin resistance directly predicts renal function decline. Instead, it points to a more complex interplay of factors, such as pancreatic beta-cell functionality, in maintaining systemic and organ-specific vitality. Similarly, fasting insulin levels correlated positively with GFR ($r = 0.224$, $p = 0.043$). These findings suggest that insulin resistance might influence kidney function. However, it also raises questions about whether these parameters directly predict GFR decline or merely reflect underlying metabolic conditions, as presented in Table 2 and Table 3.

## 4. Discussion

In elderly populations, a complex and dynamic interplay of factors governs the progression of kidney function decline in the elderly, with multiple metabolic, hemodynamic, and inflammatory pathways intricately interconnected. The interplay between insulin levels, pancreatic function, and organ vitality becomes increasingly critical. This study underscores the importance of maintaining functional pancreatic reserves to support not only kidney health but also the vitality of other organs. The weak correlations of HbA1c and fasting glucose with GFR highlight their limited utility as standalone predictors of renal function. Emerging evidence suggests that the ability of the pancreas to produce insulin, even in the face of resistance, is a critical determinant of overall organ health. In aging individuals, preserved pancreatic function ensures adequate insulin availability to meet metabolic demands, thereby protecting against multi-organ deterioration, including renal impairment. [7,8]

Interestingly, the mean HbA1c level in this study population was relatively high (7.62%), yet no significant association was observed with eGFR. This finding is somewhat unexpected, given that numerous studies have consistently reported HbA1c as a predictor of renal impairment in patients with diabetes. Chronic hyperglycemia, as reflected by elevated HbA1c, typically accelerates glomerular damage through several mechanisms, including the formation of advanced glycation end-products (AGEs), increased oxidative stress, endothelial dysfunction, and mesangial expansion. These processes contribute to glomerulosclerosis and progressive decline in renal function. However, recent evidence suggests that variability in HbA1c, rather than absolute levels, may play a more critical role in renal outcomes. Greater fluctuations in HbA1c have been linked to an increased risk of kidney disease progression, underscoring the importance of stable long-term glycemic control.[7,8] This study also demonstrated a positive correlation between HOMA-IR/insulin and eGFR; however, the association disappeared after adjustment for age, underscoring the dominant influence of aging on renal function in diabetes mellitus. Age-related nephron loss, glomerulosclerosis, and vascular remodelling are well-recognized contributors to eGFR decline, often overshadowing the impact of insulin resistance. Interestingly, despite a relatively high mean HbA1c level (7.62%), no significant association was found with eGFR. This finding diverges from most literature, where chronic hyperglycemia is strongly linked to

diabetic kidney disease (DKD) progression through advanced glycation, mesangial expansion, and microvascular damage.[9–12]

Persistent hyperglycemia in diabetes induces significant hemodynamic and molecular alterations that drive the progression of diabetic kidney disease (DKD). Elevated osmolarity within glomerular capillaries increases glomerular pressure and dilates afferent arterioles, triggering hyperfiltration and an initial rise in the glomerular filtration rate (GFR). [4,13] These disturbances activate multiple vasoactive pathways, including the renin-angiotensin-aldosterone system (RAAS), protein kinase C (PKC), the polyol pathway, advanced glycation end products (AGE), and NADPH oxidases, all of which contribute to renal injury. Notably, angiotensin II, a primary effector of RAAS, induces efferent arteriole vasoconstriction, further heightening intraglomerular pressure and exacerbating hyperfiltration. Over time, sustained elevations in glomerular capillary pressure and filtration rates lead to progressive glomerular and tubular damage. Additionally, diabetes-induced systemic hypertension and increased intraglomerular pressure accelerate renal function decline.[14–17]

Oxidative stress is one of the key drivers in the onset and progression of diabetic kidney disease (DKD), primarily induced by chronic hyperglycemia, which increases reactive oxygen species (ROS) production while impairing antioxidant defense mechanisms. This disruption in the balance between pro-oxidant and antioxidant pathways results in excessive ROS accumulation, leading to renal oxidative stress and subsequent kidney damage. Recent evidence highlights that overproduction of intrarenal ROS mediates hemodynamic and metabolic disturbances, a central mechanism underlying DKD pathogenesis. Alterations in metabolic pathways, including protein kinase C (PKC), the polyol and hexosamine pathways, and the accumulation of advanced glycation end products (AGEs), further exacerbate oxidative stress by enhancing ROS formation within the kidneys. NADPH oxidases (NOXs), particularly NOX4 and NOX5, have also emerged as major contributors to ROS generation, directly driving renal inflammation, fibrosis, and albuminuria. Beyond hyperglycemia, other metabolic factors, such as elevated free fatty acids, insulin resistance, and adiponectin dysregulation, play a role in disease progression.[14,18]

Furthermore, angiotensin II (AngII) has been implicated in the development of insulin resistance through multiple molecular mechanisms. It interferes with insulin-mediated GLUT4 translocation in skeletal muscle by transiently activating ERK1/2, thereby impairing glucose uptake. Additionally, AngII directly diminishes insulin sensitivity by promoting the nitration of AKT, a key signalling molecule in glucose metabolism. Furthermore, it induces tyrosine phosphorylation of insulin receptor substrate-1 (IRS-1) via Janus kinase 2 (JAK2), an effect mediated through activation of the angiotensin type 1 (AT1) receptor. This phosphorylation disrupts insulin-induced activation of phosphatidylinositol-3-kinase (PI3K), a crucial pathway for insulin signalling, ultimately reducing insulin sensitivity and contributing to metabolic dysfunction. Beyond its metabolic effects, AngII also plays a pivotal role in renal inflammation, further contributing to the progression of diabetic kidney disease (DKD) by promoting oxidative stress and fibrotic changes within the kidneys.[19,20]

These findings highlight the complex relationship between diabetes mellitus and kidney function decline in the elderly. The study demonstrated significant associations between diabetes-related markers such as fasting blood glucose, HbA1c, fasting insulin, and HOMA-IR with the estimated glomerular filtration rate (eGFR). Nevertheless, after adjusting for age, the correlations between insulin resistance markers and eGFR were no longer significant, underscoring that age is the dominant determinant of renal function decline in this cohort. However, several limitations must be considered when interpreting these results. The cross-sectional design of this study prevents the establishment of causal relationships, as it only captures associations at a single point in time. Moreover, the study was conducted exclusively among institutionalized elderly residents of Bina Bhakti Nursing Home, which not only limits generalizability to community-dwelling older adults but also introduces the potential for selection bias, since nursing home residents may have distinct health profiles, comorbidities, and lifestyle factors compared to the broader elderly population. Additionally, the study population was predominantly female (78%), raising the possibility of gender-related bias in representativeness. In addition, potential confounding variables, such as medication use, dietary patterns, and other metabolic conditions, were not extensively controlled, which may have influenced the observed associations. Taken together, these limitations highlight the need for cautious interpretation and for future longitudinal studies that incorporate larger, more diverse populations and account for diabetes duration, treatment regimens, and sex differences.

## 5. Conclusion

This study underscores the complex interplay between aging, metabolic dysregulation, and renal function decline in elderly individuals. A significant inverse correlation between age and estimated glomerular filtration rate (eGFR) reaffirms the well-established trajectory of age-related renal deterioration. While initial analyses suggested positive correlations between eGFR and markers of insulin resistance, such as HOMA-IR and fasting insulin, these associations disappeared after adjusting for age. In contrast, fasting blood glucose

and HbA1c showed negligible associations with eGFR. The unique contribution of this study lies in providing preliminary data from an Indonesian institutionalized elderly population, highlighting the limited predictive value of conventional glycemic markers compared with insulin-related measures. The observed association between insulin resistance markers and eGFR should be regarded as an unexpected finding that warrants cautious interpretation and further longitudinal investigation. Larger, prospective studies across more diverse elderly cohorts are required to confirm these observations and clarify the potential role of insulin resistance in age-related renal impairment.

## 6. Author Contribution

The authors contributed to this study as follows: S.G. was responsible for the conceptualization, methodology, supervision, and project administration. R.K. contributed to the conceptualization, validation, writing – review and editing, and supervision. Y.F. was involved in the investigation, data curation, and resource management. A.H.S. contributed to the methodology, validation, formal analysis, and supervision. B.A.J. was responsible for the formal analysis, visualization, and writing – original draft. K.B. contributed to the writing – review and editing, as well as data curation, while F.P. also participated in the writing, review and editing, and data curation.


## 7. Acknowledgements

The authors sincerely thank Bina Bhakti Nursing Home for their invaluable cooperation and warm acceptance during the research process, as well as for providing access and support to the elderly participants who made this study possible.


## 8. References


1. Vaidya SR, Aeddula NR, Doerr C. Chronic Kidney Disease (Nursing) [Internet]. StatPearls. 2025. Available from: http://www.ncbi.nlm.nih.gov/pubmed/25018975
2. Jaques DA, Vollenweider P, Bochud M, Ponte B. Aging and hypertension in kidney function decline: A 10 year population-based study. Front Cardiovasc Med. 2022;9:1035313.
3. Chen TK, Sperati CJ, Thavarajah S, Grams ME. Reducing Kidney Function Decline in Patients With CKD: Core Curriculum 2021. American Journal of Kidney Diseases. 2021;77(6):969–83.
4. Sinha SK, Nicholas SB. Pathomechanisms of Diabetic Kidney Disease. J Clin Med. 2023 Nov 27;12(23):7349.
5. Watanabe K, Sato E, Mishima E, Miyazaki M, Tanaka T. What's New in the Molecular Mechanisms of Diabetic Kidney Disease: Recent Advances. Int J Mol Sci. 2022 Dec 29;24(1):570.
6. Alicic RZ, Rooney MT, Tuttle KR. Diabetic Kidney Disease. Clinical Journal of the American Society of Nephrology. 2017 Dec;12(12):2032–45.
7. Bloomgarden Z, Handelsman Y. How does CKD affect HbA1c? J Diabetes. 2018 Apr 21;10(4):270–270.
8. Takenouchi A, Tsuboi A, Terazawa-Watanabe M, Kurata M, Fukuo K, Kazumi T. Direct association of visit-to-visit HbA1c variation with annual decline in estimated glomerular filtration rate in patients with type 2 diabetes. J Diabetes Metab Disord. 2015;14(1):69.
9. You L, Hong X, Feng Q, Sun K, Lin D, Huang C, et al. Glucose Metabolism Indices and the Development of Chronic Kidney Disease: A Cohort Study of Middle-Aged and Elderly Chinese Persons. Int J Endocrinol. 2023;2023:1–11.
10. Yang H, Gong R, Liu M, Deng Y, Zheng X, Hu T. HOMA-IR is positively correlated with biological age and advanced aging in the US adult population. Eur J Med Res. 2023;28(1):470.
11. Buyadaa O, Salim A, Morton JI, Magliano DJ, Shaw JE. Rate of decline in kidney function and known age-of-onset or duration of type 2 diabetes. Sci Rep. 2021;11(1):14705.
12. Jha R, Lopez-Trevino S, Kankanamalage HR, Jha JC. Diabetes and Renal Complications: An Overview on Pathophysiology, Biomarkers and Therapeutic Interventions. Biomedicines. 2024;12(5):1098.
13. Tonneijck L, Muskiet MHA, Smits MM, van Bommel EJ, Heerspink HJL, van Raalte DH, et al. Glomerular Hyperfiltration in Diabetes: Mechanisms, Clinical Significance, and Treatment. Journal of the American Society of Nephrology. 2017;28(4):1023–39.
14. Jha R, Lopez-Trevino S, Kankanamalage HR, Jha JC. Diabetes and Renal Complications: An Overview on Pathophysiology, Biomarkers and Therapeutic Interventions. Biomedicines. 2024;12(5):1098.
15. Magee GM, Bilous RW, Cardwell CR, Hunter SJ, Kee F, Fogarty DG. Is hyperfiltration associated with the future risk of developing diabetic nephropathy? A meta-analysis. Diabetologia. 2009 Apr 7;52(4):691–7.
16. Velez JCQ. The importance of the intrarenal renin–angiotensin system. Nat Clin Pract Nephrol. 2009;5(2):89–100. Available from:
17. Jerums G, Premaratne E, Panagiotopoulos S, MacIsaac RJ. The clinical significance of hyperfiltration in diabetes. Diabetologia. 2010;53(10):2093–104.
18. Reidy K, Kang HM, Hostetter T, Susztak K. Molecular mechanisms of diabetic kidney disease. Journal of Clinical Investigation. 2014;124(6):2333–40.
19. Premilovac D, Attrill E, Rattigan S, Richards SM, Kim J, Keske MA. Acute, local infusion of angiotensin II impairs microvascular and metabolic insulin sensitivity in skeletal muscle.

Cardiovasc Res. 2019;115(3):590–601.
20. Velloso LA, Folli F, Perego L, Saad MJA. The multi-faceted cross-talk between the insulin and angiotensin II signaling systems. Diabetes Metab Res Rev. 2006;22(2):98–107.