Oshada; Jurnal Kesehatan
https://nawalaeducation.com/index.php/O/index
Volume 2 Nomor 5, October 2025
e-ISSN : 3047-017X
DOI : https://doi.org/10.62872/4h5kgg80

Sedentary Lifestyle and Blood Glucose Regulation: A Physiological Perspective on Metabolic Imbalance

Albert Manggading Hutapea[1], Dwight Mahaputera Marulitua Hutapea[2],
Universitas Advent Indonesia[1], Universitas Prima Indonesia[2]
e-mail: amhutapea@unai.edu[1]

Input	: 20 September 2025	Revised	: 14 October 2025
Accepted	: 26 October 2025	Published	: 30 October 2025

ABSTRACT

The increasing prevalence of sedentary behavior represents a global phenomenon that significantly contributes to the rising burden of metabolic disorders, particularly impaired blood glucose regulation. Glucose regulation is a central physiological mechanism in maintaining metabolic homeostasis, and its disruption has profound implications for cardiometabolic health. This study aims to synthesize scientific evidence on the relationship between sedentary lifestyle and blood glucose regulation from a physiological perspective, with a focus on the metabolic mechanisms underlying metabolic imbalance. A Systematic Literature Review with a qualitative synthesis approach was conducted using peer-reviewed journal articles published over the past ten years. A total of 22 eligible studies were analyzed through physiological thematic analysis and narrative synthesis. The findings indicate that sedentary behavior disrupts glucose regulation through reduced skeletal muscle activity, impaired GLUT-4 translocation, decreased insulin sensitivity, mitochondrial dysfunction, and increased adipose tissue inflammation. Over time, these alterations contribute to the progression of prediabetes, type 2 diabetes mellitus, and elevated cardiometabolic risk. This review highlights the critical role of physical activity in preserving metabolic balance and preventing glucose dysregulation.

Kata kunci: *blood glucose regulation; insulin resistance; metabolic imbalance; sedentary lifestyle; systematic literature review.*

INTRODUCTION

Global lifestyle changes over recent decades have demonstrated a significant shift toward sedentary patterns of living, characterized by prolonged periods of low energy expenditure activities such as extended sitting, increased use of digital devices, and reduced daily physical activity. This phenomenon is not limited to developed countries but has also increased rapidly in developing nations as a consequence of urbanization, the transformation of work toward service-based sectors, and the widespread penetration of digital technology into everyday life. Global health organizations report that more than one quarter of the world's adult population does not meet minimum physical activity recommendations, and the proportion of daily sedentary time continues to rise

across age groups. This condition positions sedentary lifestyle as a global public health issue with broad implications for the increasing prevalence of metabolic disorders, including obesity, metabolic syndrome, and type 2 diabetes mellitus (Musa et al., 2022).

One of the most critical health implications of increased sedentary behavior is impaired blood glucose regulation, which represents a central physiological mechanism in maintaining metabolic homeostasis and overall energy balance. Glucose regulation involves dynamic interactions among the musculoskeletal, endocrine, and energy metabolism systems, with insulin serving as the key hormone mediating glucose uptake into peripheral tissues. When this regulatory mechanism is disrupted, the body experiences an imbalance between glucose production, distribution, and utilization, ultimately leading to chronic hyperglycemia. Impaired glucose regulation is not only a defining feature of diabetes mellitus but also constitutes an early stage in the broader spectrum of metabolic dysfunction (Richter-Stretton et al., 2020).

From a metabolic physiology perspective, skeletal muscle plays a dominant role in glucose regulation because this tissue accounts for the majority of postprandial glucose uptake. Muscle contraction facilitates the translocation of GLUT-4 glucose transporters to the cell membrane through insulin independent pathways, thereby enabling glucose uptake even when insulin sensitivity is reduced. However, under sedentary conditions, reduced muscle contraction leads to diminished activation of these glucose uptake pathways, causing the body to rely increasingly on insulin action to maintain normoglycemia. Excessive dependence on insulin over time contributes to declining insulin sensitivity and the development of insulin resistance as a maladaptive physiological response (Yaribeygi et al., 2021).

In addition to skeletal muscle dysfunction, sedentary lifestyle also contributes to structural and functional changes in adipose tissue that exacerbate metabolic imbalance. An imbalance between energy intake and expenditure leads to the accumulation of adipose tissue, particularly metabolically active visceral fat. This tissue functions not only as an energy storage site but also as an endocrine organ that secretes various adipokines and proinflammatory cytokines. The resulting low grade inflammatory environment disrupts insulin signaling pathways in target tissues, including muscle and liver, thereby accelerating the development of impaired glucose regulation. Abranches et al. (2015) demonstrated that adipose tissue dysfunction serves as a key link between sedentary behavior, chronic inflammation, and impaired glucose homeostasis.

This phenomenon is further supported by epidemiological data indicating increased prevalence of impaired blood glucose regulation with longer durations of sedentary behavior, even among individuals with normal body mass index. This finding suggests that sedentary lifestyle exerts physiological effects that extend beyond obesity alone. Recent studies also indicate that brief interruptions of prolonged sitting with light physical activity can improve postprandial glycemic responses, highlighting the sensitivity of

glucose regulatory systems to acute changes in physical activity. These findings reinforce the view that sedentary behavior constitutes an active physiological determinant rather than a passive consequence of insufficient exercise (Cavallo et al., 2022).

Although the relationship between sedentary lifestyle and impaired glucose regulation has been widely investigated, existing research approaches remain conceptually and methodologically fragmented. Cavallo et al. (2022), in their study titled The Association Between Sedentary Behaviour, Physical Activity and Type 2 Diabetes Markers: A Systematic Review of Mixed Analytic Approaches, emphasized the strong association between sedentary behavior and type 2 diabetes markers, but focused primarily on analytic heterogeneity and statistical associations rather than synthesizing underlying physiological pathways. Meanwhile, Yaribeygi et al. (2021), through Pathophysiology of Physical Inactivity Dependent Insulin Resistance: A Theoretical Mechanistic Review Emphasizing Clinical Evidence, provided an in depth mechanistic explanation of insulin resistance resulting from physical inactivity, but did not specifically distinguish sedentary lifestyle as a behavioral pattern characterized by prolonged sitting. On the other hand, Damayanti (2025), in The Impact of Sedentary Behavior on Blood Glucose Levels in Type 2 Diabetes Mellitus Patients: A Literature Review, focused on clinical populations with type 2 diabetes mellitus and emphasized glycemic outcomes without integrating cross tissue physiological mechanisms from preclinical stages.

Based on these three studies, the research gap addressed in this study lies in the absence of a systematic and comprehensive synthesis that integrates the physiological mechanisms linking sedentary lifestyle to blood glucose dysregulation as a continuous process of metabolic imbalance. Cavallo et al. (2022) emphasized associative relationships without mapping biological pathways, Yaribeygi et al. (2021) discussed physical inactivity in general without specific focus on sedentary behavior, while Damayanti (2025) oriented toward clinical outcomes without integrating cross tissue physiological mechanisms. As a result, understanding of the interaction among skeletal muscle dysfunction, insulin resistance, adipose tissue inflammation, and hormonal imbalance within the context of sedentary lifestyle remains fragmented and insufficiently synthesized at a conceptual level.

Based on this gap, the novelty of the present study lies in its integrative approach grounded in metabolic physiology, synthesizing cross study scientific evidence to map the principal mechanistic pathways linking sedentary lifestyle to impaired blood glucose regulation. This study aims to synthesize scientific findings on the relationship between sedentary behavior and glucose regulation from a physiological perspective, identify key metabolic mechanisms underlying metabolic imbalance, and formulate an evidence based conceptual framework that can strengthen both theoretical and practical foundations for the prevention of metabolic disorders.

METODOLOGI

This study employs a Systematic Literature Review (SLR) design to comprehensively synthesize scientific evidence regarding the relationship between sedentary lifestyle and blood glucose regulation from a physiological perspective. The SLR approach was selected because it enables the systematic, transparent, and replicable integration of findings across studies, making it particularly suitable for examining complex biological mechanisms distributed across multiple disciplines, including physiology, endocrinology, and metabolic health. The review procedure followed methodological principles recommended for systematic literature reviews in the fields of health and biomedical sciences (Xiao & Watson, 2017).

The literature search was conducted using reputable scientific databases, namely PubMed, Scopus, Web of Science, ScienceDirect, and Google Scholar, covering publications from the last ten years. The search strategy employed combinations of the keywords "sedentary lifestyle" or "sedentary behavior" paired with "blood glucose regulation", "insulin sensitivity", and "metabolic imbalance". Inclusion criteria comprised peer reviewed journal articles with experimental, clinical, or systematic review designs that examined physiological mechanisms of glucose regulation in adult or adolescent human subjects. Non peer reviewed studies, articles focusing exclusively on social aspects without biological implications, and publications without accessible full texts were excluded to ensure the quality of the synthesized evidence (Cavallo et al., 2022).

The study selection process followed the PRISMA flow systematically. During the initial identification stage, 684 articles were retrieved from all databases. After duplicate removal, 512 articles were screened based on titles and abstracts, resulting in 176 articles deemed relevant for full text assessment. At the eligibility stage, 41 articles met the inclusion criteria and methodological quality standards, and ultimately 22 studies were included in the final synthesis. Extracted data included study characteristics, physiological parameters such as blood glucose levels and insulin sensitivity, and identified metabolic mechanisms. These data were then analyzed using physiological thematic analysis and synthesized narratively to map the principal biological pathways underlying metabolic imbalance associated with sedentary behavior (Xiao & Watson, 2017).

As summarized in Table 1, the reviewed studies consistently indicate that sedentary behavior disrupts glucose regulation through skeletal muscle inactivity, insulin resistance, mitochondrial dysfunction, and adipose tissue inflammation

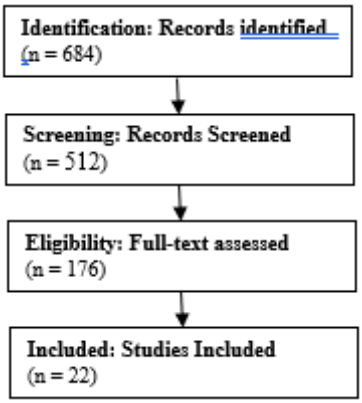

Figure 1. PRISMA Flow Diagram of the Systematic Literature Review

RESULT AND DISCUSSION

Table 1. Summary of Reviewed Studies on Sedentary Lifestyle and Blood Glucose Regulation

Author(s) & Year	Study Title / Reference	Research Objective	Study Design / Methodology	Sample / Data Source	Key Variables / Concepts	Key Findings / Results
Abranches et al., 2015	Obesity and diabetes: the link between adipose tissue dysfunction and glucose homeostasis	Examine the role of adipose tissue dysfunction in glucose regulation	Narrative review	Human metabolic and clinical studies	Adipose tissue, adipokines, insulin signaling	Adipose tissue dysfunction promotes insulin resistance and impaired glucose homeostasis
Cavallo et al., 2022	Sedentary behaviour, physical activity and type 2 diabetes markers	Analyze associations between sedentary behavior and T2DM markers	Systematic review	Population-based studies	Sedentary time, physical activity, glycemic markers	Sedentary behavior independently worsens glycemic control
Damayanti, 2025	Sedentary behavior and blood glucose in T2DM patients	Review effects of sedentary behavior on blood glucose	Literature review	Type 2 diabetes patients	Sedentary duration, blood glucose	Prolonged sedentary behavior increases blood glucose levels
Devi et al., 2023	TNF-α and blood glucose after HIIT	Examine inflammatory and glucose responses to	Experimental study	Overweight women	TNF-α, blood glucose, exercise	Acute exercise reduces inflammation and

		acute exercise				glucose levels
Fazzi et al., 2017	Sedentary behaviours during pregnancy	Assess sedentary patterns and metabolic risk	Systematic review	Pregnant women	Sedentary behavior, metabolic risk	High sedentary time associated with metabolic risk
Gupta et al., 2025	AR/VR in gastronomy and consumer engagement	Explore digital engagement patterns	Narrative review	Consumer behavior studies	Digital engagement, sedentary behavior	Digital immersion may reinforce sedentary lifestyles
Ibeojo et al., 2025	Exercise and glucose regulation in T2DM	Identify mechanisms linking exercise and glucose control	Systematic review	Type 2 diabetes patients	Exercise, insulin sensitivity	Exercise improves glucose regulation pathways
Ispas et al., 2025	Physical activity and diabetes management	Review effects of physical activity on diabetes outcomes	Systematic review	Diabetic populations	Physical activity, glycemic control	Increased activity improves metabolic outcomes
Iyer, 2025	Lifestyle modification and yoga in T2DM	Examine non-pharmacological diabetes management	Comprehensive review	Adult T2DM patients	Lifestyle change, yoga	Lifestyle modification improves glycemic control
Jędrysik et al., 2024	Biomarkers in metabolic disorders	Review biomarkers predicting metabolic disorders	Review and predictive analysis	Biomarker studies	GLP-1, GIP, IGFBP-7	Biomarkers reflect metabolic dysregulation
Kajita et al., 2024	Sphingosine-1-phosphate and glucose homeostasis	Investigate molecular regulation of glucose metabolism	Experimental and review	Molecular and animal studies	S1P signaling, glucose homeostasis	S1P regulates obesity and glucose balance
Khan et al., 2023	Anthropometric indices and insulin resistance	Analyze obesity indicators and insulin resistance	Literature review	Obese adult populations	BMI, insulin resistance	Anthropometric indices correlate with insulin resistance
Mthembu et al., 2022	Exercise, insulin resistance	Examine exercise effects on	Review	T2DM patients	Mitochondrial function,	Exercise improves mitochondr

	and mitochondria	skeletal muscle metabolism			insulin resistance	ial and insulin function
Muguerza-Rodríguez et al., 2025	Gut microbiota, physical activity and diabetes	Review gut microbiota role in glucose regulation	Systematic review	Human studies	Gut microbiota, exercise	Physical activity improves microbiota-mediated glucose regulation
Musa et al., 2022	Screen-based sedentary behavior and metabolic syndrome	Assess digital sedentary behavior effects	Systematic review	Adolescents	Screen time, metabolic syndrome	Screen-based sedentary behavior increases metabolic risk
Obeidat et al., 2025	Trends of metabolic syndrome	Examine long-term metabolic syndrome trends	Narrative review	Global epidemiological data	Metabolic syndrome trends	Sedentary lifestyle accelerates metabolic disorders
Richter-Stretton et al., 2020	Skeletal muscle and metabolic syndrome	Examine muscle role in glucose metabolism	Narrative review	Human physiology studies	Skeletal muscle, glucose uptake	Muscle inactivity impairs glucose regulation
Sabillah et al., 2025	Resistance training and glucose reduction	Test acute resistance training effects	Randomized controlled trial	Sedentary women	Resistance training, blood glucose	Resistance exercise acutely lowers blood glucose
Xing et al., 2024	Exercise-responsive metabolites and insulin signaling	Investigate metabolite-mediated insulin signaling	Experimental study	Human metabolic studies	Glutamate, insulin signaling	Exercise enhances insulin signaling pathways
Yaribeygi et al., 2021	Physical inactivity-dependent insulin resistance	Explain mechanisms of inactivity-induced insulin resistance	Theoretical mechanistic review	Clinical and experimental evidence	Insulin signaling pathways	Physical inactivity induces insulin resistance
Zhang et al., 2022	Exercise intervention in diabetes research	Analyze global research trends	Bibliometric analysis	International publications	Exercise, diabetes research	Exercise is central to diabetes intervention

Effect of Sedentary Lifestyle on Blood Glucose Regulation Mechanisms

A sedentary lifestyle affects blood glucose regulation primarily through a

reduction in skeletal muscle activity, which represents the main peripheral tissue responsible for postprandial glucose uptake. Under normal physiological conditions, skeletal muscle contraction stimulates the translocation of the glucose transporter GLUT-4 to the cell membrane through insulin independent pathways, thereby enabling efficient glucose uptake even when insulin levels are low. However, in individuals with prolonged sedentary behavior, reduced muscle contraction leads to diminished activation of this pathway, which directly results in elevated postprandial blood glucose levels. Richter-Stretton et al. (2020) emphasize that skeletal muscle functions as an active regulator of glucose metabolism and is highly sensitive to changes in physical activity levels.

Reduced glucose uptake by skeletal muscle due to sedentary behavior increases the body's reliance on insulin dependent pathways to maintain normoglycemia. This condition compels the pancreas to increase insulin secretion as a compensatory response to elevated circulating glucose levels. In the short term, this compensatory mechanism may maintain glucose levels within the normal range; however, repeated and prolonged insulin stimulation contributes to desensitization of insulin receptors in target tissues. Yaribeygi et al. (2021) explain that physical inactivity induces molecular alterations in insulin signaling pathways, including reduced phosphorylation of insulin receptor substrate 1 and impaired activation of the PI3K-Akt pathway, which is the primary pathway for cellular glucose uptake.

Beyond alterations in insulin signaling, a sedentary lifestyle also affects skeletal muscle mitochondrial function, which plays a critical role in energy metabolism. Low levels of physical activity reduce mitochondrial oxidative capacity, thereby decreasing the ability of muscle cells to efficiently oxidize fatty acids and glucose. The accumulation of unoxidized metabolic substrates contributes to lipotoxicity and increased oxidative stress, which further impairs insulin sensitivity. Mthembu et al. (2022) demonstrate that skeletal muscle mitochondrial dysfunction represents a key therapeutic target in addressing insulin resistance induced by sedentary lifestyles.

The physiological effects of a sedentary lifestyle on glucose regulation are also mediated by changes in adipose tissue, particularly increased metabolically active visceral adiposity. The accumulation of adipose tissue resulting from energy imbalance enhances the secretion of proinflammatory adipokines such as tumor necrosis factor alpha and interleukin 6, which directly disrupt insulin signaling pathways in skeletal muscle and the liver. This low grade inflammatory environment exacerbates glucose dysregulation by increasing hepatic glucose production and reducing peripheral glucose uptake. Abranches et al. (2015) affirm that adipose tissue dysfunction is a key component in the pathophysiology of glucose homeostasis disorders associated with sedentary lifestyles.

Overall, the findings discussed in this section indicate that sedentary lifestyle induces blood glucose dysregulation through complex interactions among reduced skeletal muscle activity, impaired insulin signaling, mitochondrial metabolic dysfunction, and adipose tissue inflammation. These physiological processes do not operate independently but rather reinforce one

another in generating a progressive state of metabolic imbalance. Cavallo et al. (2022) emphasize that without adequate physical activity intervention, physiological changes resulting from sedentary behavior may progress from transient glucose disturbances to chronic metabolic dysfunction, thereby increasing the risk of type 2 diabetes mellitus.

Insulin Sensitivity Dysfunction and Metabolic Imbalance

A sedentary lifestyle contributes significantly to reduced insulin sensitivity through disruption of the balance between glucose uptake and utilization by peripheral tissues. Under normal physiological conditions, insulin facilitates glucose entry into cells through activation of insulin signaling pathways involving the insulin receptor, insulin receptor substrate, and the PI3K-Akt pathway. However, low levels of physical activity result in reduced stimulation of these pathways, leading to a progressive decline in insulin effectiveness in lowering blood glucose levels. Zhao et al. (2023) explain that insulin resistance represents a central mechanism linking sedentary behavior with systemic metabolic disorders.

Insulin sensitivity dysfunction resulting from sedentary lifestyle is also closely associated with metabolic alterations in adipose tissue, particularly increased visceral adiposity. Visceral adipose tissue produces inflammatory mediators that interfere with insulin signaling in target tissues, including skeletal muscle and the liver. This low grade inflammatory environment exacerbates insulin resistance by inhibiting insulin receptor substrate phosphorylation and increasing hepatic glucose production. Abranches et al. (2015) demonstrate that adipose tissue dysfunction is a key factor in the development of insulin resistance and glucose regulation disorders in individuals with low physical activity levels.

In addition to the role of adipose tissue, sedentary lifestyle also influences insulin sensitivity through alterations in mitochondrial function and energy substrate metabolism. Reduced mitochondrial oxidative capacity due to physical inactivity leads to the accumulation of lipotoxic intracellular lipids, thereby disrupting insulin signaling pathways. This condition reinforces insulin resistance and creates a metabolic vicious cycle between impaired energy oxidation and hyperglycemia. Mthembu et al. (2022) emphasize that mitochondrial dysfunction represents one of the primary mechanisms underlying insulin resistance induced by sedentary lifestyles.

To summarize the principal mechanisms of insulin sensitivity dysfunction associated with sedentary lifestyle, Table 2 presents a synthesis of physiological findings across studies linking sedentary behavior with the involved metabolic pathways.

Table 2. Physiological Mechanisms Linking Sedentary Lifestyle to Insulin Resistance

Physiological Component	Sedentary-Induced Alteration	Metabolic Consequence
Skeletal muscle activity	Reduced muscle contraction and GLUT-4 translocation	Decreased peripheral glucose uptake

Insulin signaling pathway	Impaired IRS-1 and PI3K-Akt activation	Reduced insulin sensitivity
Adipose tissue function	Increased visceral fat and pro-inflammatory adipokines	Chronic low-grade inflammation
Mitochondrial metabolism	Decreased oxidative capacity	Lipid accumulation and lipotoxicity
Hepatic glucose regulation	Increased gluconeogenesis	Elevated fasting blood glucose

Analysis of Table 2 indicates that insulin resistance resulting from a sedentary lifestyle is the outcome of multiple interacting physiological systems rather than a single isolated pathway. Reduced skeletal muscle activity, adipose tissue dysfunction, impaired mitochondrial metabolism, and increased hepatic glucose production mutually reinforce one another in creating a progressive metabolic imbalance. The integration of these mechanisms explains why interventions that focus solely on glycemic control without addressing sedentary behavior often yield limited outcomes. Yaribeygi et al. (2021) emphasize that physiologically based mechanistic approaches are required to disrupt the cycle of insulin resistance induced by chronic physical inactivity.

Long-Term Physiological Consequences for Metabolic Health

Chronic exposure to sedentary behavior leads to the accumulation of blood glucose regulation disturbances that ultimately drive progression toward prediabetes and type 2 diabetes mellitus. As insulin resistance develops persistently, compensatory pancreatic mechanisms through increased insulin secretion become ineffective, resulting in relative pancreatic beta cell failure. This condition is characterized by sustained elevations in fasting and postprandial blood glucose levels, as well as a loss of metabolic flexibility in responding to fluctuations in energy intake. Obeidat et al. (2025) demonstrate that the transition from normoglycemia to impaired glucose regulation occurs gradually and is strongly influenced by the duration and intensity of sedentary behavior.

In addition to its effects on glucose metabolism, sedentary lifestyle disrupts systemic energy homeostasis through alterations in lipid metabolism and energy substrate distribution. Prolonged insulin resistance reduces the capacity of adipose tissue to safely store lipids, thereby increasing the release of free fatty acids into the circulation. Elevated free fatty acid levels further exacerbate insulin resistance in skeletal muscle and the liver and enhance hepatic glucose production. Consequently, a pathological interaction emerges between impaired glucose and lipid metabolism, accelerating the development of metabolic syndrome. Khan et al. (2023) emphasize that insulin resistance represents the central link between lipid metabolic dysfunction and increased risk of chronic metabolic diseases.

Over the long term, metabolic imbalance resulting from sedentary lifestyle also exerts widespread effects on cardiometabolic health through increased oxidative stress, endothelial dysfunction, and systemic inflammation. Chronic

hyperglycemia and insulin resistance enhance the formation of advanced glycation end products, which impair vascular function and accelerate atherosclerosis. Furthermore, persistent low grade inflammation aggravates tissue damage and reduces the adaptive capacity of the cardiovascular system. Zhang et al. (2022) demonstrate that metabolic disturbances induced by physical inactivity constitute a major determinant of increased cardiometabolic morbidity and mortality globally.

Overall, this discussion underscores that the physiological consequences of sedentary lifestyle extend beyond elevated blood glucose levels alone and encompass a spectrum of progressive and systemic metabolic disorders. The interaction among insulin resistance, lipid metabolism dysfunction, chronic inflammation, and vascular damage forms a pathological pathway that reinforces long-term metabolic imbalance. These findings highlight the urgency of early intervention targeting sedentary behavior as a key strategy for preventing metabolic disorders and related chronic diseases, particularly through integrated and sustained physical activity based approaches (Ispas et al., 2025).

CONCLUSION

Based on the results of the systematic literature review, this study concludes that sedentary lifestyle exerts significant physiological effects on blood glucose regulation through complex interactions among skeletal muscle dysfunction, reduced insulin sensitivity, adipose tissue inflammation, and energy metabolism imbalance. Scientific evidence indicates that low levels of physical activity impair glucose uptake by skeletal muscle, increase reliance on insulin, and accelerate the development of insulin resistance, which constitutes the primary foundation of metabolic imbalance and progression toward chronic glucose disorders (Yaribeygi et al., 2021).

Furthermore, metabolic imbalance induced by sedentary behavior affects not only glucose metabolism but also lipid metabolic systems and broader cardiometabolic health. Persistent insulin resistance promotes the accumulation of dysfunctional lipids, reinforces low grade systemic inflammation, and accelerates vascular damage, thereby contributing to increased risk of type 2 diabetes mellitus and cardiovascular disease. The synthesis of these findings affirms that sedentary lifestyle represents an active physiological determinant within the spectrum of metabolic disorders rather than a passive lifestyle factor (Zhang et al., 2022).

Based on these findings, this study recommends that health interventions prioritize the reduction of sedentary behavior through increased regular physical activity as a primary strategy for preventing glucose regulation disorders and metabolic imbalance. For future research, longitudinal and mechanistic studies integrating physiological biomarkers are required to strengthen causal understanding of the relationship between sedentary lifestyle and metabolic dysfunction, as well as to develop more

effective and sustainable evidence based interventions in public health and clinical contexts (Ispas et al., 2025).

REFERENCE

Abranches, M., Oliveira, F., Conceição, L., & Pelúzio, M. (2015). Obesity and diabetes: the link between adipose tissue dysfunction and glucose homeostasis. Nutrition Research Reviews, 28, 121 - 132. https://doi.org/10.1017/s0954422415000098.

Cavallo, F., Golden, C., Pearson-Stuttard, J., Falconer, C., & Toumazou, C. (2022). The association between sedentary behaviour, physical activity and type 2 diabetes markers: A systematic review of mixed analytic approaches. PLoS ONE, 17. https://doi.org/10.1371/journal.pone.0268289.

Damayanti, L. (2025). The impact of sedentary behavior on blood glucose levels in type 2 diabetes mellitus patients: A literature review. Journal of Evidence-based Nursing and Public Health. https://doi.org/10.61511/jevnah.v2i1.2025.1735.

Devi, A., Rejeki, P., Argarini, R., Shakila, N., Yosnengsih, Y., Ilmi, S., Karimullah, A., Ayubi, N., & Herawati, L. (2023). Response of TNF-α Levels and Blood Glucose Levels after Acute High-Intensity Intermittent Exercise in Overweight Women. Retos. https://doi.org/10.47197/retos.v48.94305.

Fazzi, C., Saunders, D., Linton, K., Norman, J., & Reynolds, R. (2017). Sedentary behaviours during pregnancy: a systematic review. The International Journal of Behavioral Nutrition and Physical Activity, 14. https://doi.org/10.1186/s12966-017-0485-z.

Gupta, P., Das, E., Islam, D., Gogoi, D., & Daolagupu, N. (2025). Augmented Reality (AR) And Virtual Reality (VR) In Gastronomy: Transforming Consumer Engagement and Enhancing Immersive Dining Experiences In India. Journal of Information Systems Engineering and Management. https://doi.org/10.52783/jisem.v10i34s.5858.

Ibeojo, D., Oden, J., Ojukwu, E., & Avramenko, I (2025). Exercise and Glucose Regulation in Type 2 Diabetes: A Systematic Review of Mechanisms and Pathways. Impact Surgery. https://doi.org/10.62463/surgery.167.

Ispas, S., Twakor, A., Mîndrescu, N., Ispas, V., Tofolean, D., Hutanu, E., Petcu, A., Deacu, S., Iordache, I., Bica, C., Petcu, L., Gherghiceanu, F., Popoviciu, M., & Stoian, A. (2025). From Sedentary to Success: How Physical Activity Transforms Diabetes Management: A Systematic Review. Journal of Mind and Medical Sciences. https://doi.org/10.3390/jmms12010010.

Iyer, S. (2025). Investigating the Role of Lifestyle Modifications and Yoga Intervention in the Management of Type 2 Diabetes: A Comprehensive Review. Journal of Information Systems Engineering and Management. https://doi.org/10.52783/jisem.v10i34s.5794.

Jędrysik, M., Wyszomirski, K., Różańska-Walędziak, A., Grosicka-Maciąg, E., Walędziak, M., & Chełstowska, B. (2024). The Role of GLP-1, GIP, MCP-1 and IGFBP-7 Biomarkers in the Development of Metabolic Disorders: A Review

and Predictive Analysis in the Context of Diabetes and Obesity. Biomedicines, 12. https://doi.org/10.3390/biomedicines12010159.

Kajita, K., Ishii, I., Mori, I., Asano, M., Fuwa, M., & Morita, H. (2024). Sphingosine 1-Phosphate Regulates Obesity and Glucose Homeostasis. International Journal of Molecular Sciences, 25. https://doi.org/10.3390/ijms25020932.

Khan, K., Wanjari, A., Acharya, S., & Quazi, S. (2023). Anthropometric Indices With Insulin Resistance in Obese Patients: A Literature Review. Cureus, 15. https://doi.org/10.7759/cureus.41881.

Mthembu, S., Mazibuko-Mbeje, S., Ziqubu, K., Nyawo, T., Obonye, N., Nyambuya, T., Nkambule, B., Silvestri, S., Tiano, L., Muller, C., & Dludla, P. (2022). Impact of physical exercise and caloric restriction in patients with type 2 diabetes: Skeletal muscle insulin resistance and mitochondrial dysfunction as ideal therapeutic targets.. Life sciences, 120467 . https://doi.org/10.1016/j.lfs.2022.120467.

Muguerza-Rodríguez, L., Mier, A., Ponce-González, J., Casals, C., & Corral-Pérez, J. (2025). Systematic Review on the Importance of Gut Microbiota in the Regulation of Type 2 Diabetes Through Physical Activity and Exercise. Current Issues in Molecular Biology, 47. https://doi.org/10.3390/cimb47070505.

Musa, S., Elyamani, R., & Dergaa, I. (2022). COVID-19 and screen-based sedentary behaviour: Systematic review of digital screen time and metabolic syndrome in adolescents. PLoS ONE, 17. https://doi.org/10.1371/journal.pone.0265560.

Obeidat, A., Ahmad, M., Ghabashi, M., Alazzeh, A., Habib, S., Al-Haijaa, D., & Azzeh, F. (2025). Developmental Trends of Metabolic Syndrome in the Past Two Decades: A Narrative Review. Journal of Clinical Medicine, 14. https://doi.org/10.3390/jcm14072402.

Richter-Stretton, G., Fenning, A., & Vella, R. (2020). Skeletal muscle - A bystander or influencer of metabolic syndrome?. Diabetes & metabolic syndrome, 14 5, 867-875 . https://doi.org/10.1016/j.dsx.2020.06.006.

Sabillah, M., Wibawa, J., Ayubi, N., Sceisarriya, V., Ndayisenga, J., & Kurnaz, M. (2025). Physiological Mechanisms of Acute Resistance Training in Reducing Blood Glucose Levels in Women with a Sedentary Lifestyle: A Randomized Controlled Trial. Al-Rafidain Journal of Medical Sciences (ISSN 2789-3219). https://doi.org/10.54133/ajms.v9i2.2482.

Xiao, Y., & Watson, M. (2017). Guidance on Conducting a Systematic Literature Review. Journal of Planning Education and Research, 39, 112 - 93. https://doi.org/10.1177/0739456x17723971.

Xing, X., Sun, Q., Wang, R., Wang, Y., & Wang, R. (2024). Impacts of glutamate, an exercise-responsive metabolite on insulin signaling.. Life sciences, 122471 . https://doi.org/10.1016/j.lfs.2024.122471.

Yaribeygi, H., Maleki, M., Sathyapalan, T., Jamialahmadi, T., & Sahebkar, A. (2021). Pathophysiology of Physical Inactivity-Dependent Insulin Resistance: A Theoretical Mechanistic Review Emphasizing Clinical Evidence. Journal of Diabetes Research, 2021. https://doi.org/10.1155/2021/7796727.

Zhang, Z., Zhu, Y., Wang, Q., Chang, T., Liu, C., Zhu, Y., Wang, X., & Cao, X. (2022). Global Trends and Research Hotspots of Exercise for Intervening Diabetes: A Bibliometric Analysis. Frontiers in Public Health, 10. https://doi.org/10.3389/fpubh.2022.902825.

Zhao, X., An, X., Yang, C., Sun, W., Ji, H., & Lian, F. (2023). The crucial role and mechanism of insulin resistance in metabolic disease. Frontiers in Endocrinology, 14. https://doi.org/10.3389/fendo.2023.1149239.