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The Effects of Regular Aerobic Exercise on Blood-glucose Control in Sedentary Adults with Type 2 Diabetes

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Abstract. Sedentary behaviour has become a widespread health risk factor for modern adults, greatly increasing the risk of type 2 diabetes and worsening glycaemic regulation. Although pharmacological treatments are widely used for diabetes management, non-drug interventions such as aerobic exercise deserve further investigation. The study aims to explore how regular aerobic exercise would influence blood-glucose control among sedentary adults with type 2 diabetes. This narrative review synthesizes relevant published literature and physiological mechanisms including insulin sensitivity. Intervention outcomes such as changes in HbA1c and 24-hour glucose monitoring are compared between exercise and control groups. The results indicate that consistent aerobic training can effectively improve participants' blood glucose control. It is concluded that regular aerobic exercise is absolutely a meaningful approach for blood-glucose control, yet it should combine with personal condition changes to form practical exercise prescriptions.

Keywords: Type 2 diabetes, Sedentary lifestyle, Blood-glucose Control, Aerobic exercise, Sport intervention

1. Introduction

According to the latest International Diabetes Federation (IDF) Diabetes Atlas, nearly 10.5% of people around the world have been affected by diabetes mellitus (DM), with about half of adults (44.7%) remaining undiagnosed [1]. Type 2 diabetes mellitus (T2DM) is one of the most common types, which is a chronic metabolic disorder [2]. Globally, this subtype makes up approximately 90% of all diabetes cases [3]. The global disease burden of T2DM keeps mounting as a result of population ageing, urbanization and changing lifestyles [4]. According to research, one of the most significant causes of this chronic disease is sedentary lifestyle — a common living habit among both teenagers and elder adults in recent years [2]. It is predicted that the number of individuals aged 20-79 years with T2DM will grow to 640 million by 2030 [5]. In order to solve this considerable problem, many scientists have invented useful medicines and chemicals to remit the chronic disease. While medication is effective, it comes with side effects, compliance issues, and financial burdens; therefore, non-pharmacological interventions are valuable. Against this background, the present narrative review aims to examine the effects of regular aerobic exercise on glycaemic regulation among sedentary adults diagnosed with type 2 diabetes. By synthesizing existing literature and the underlying physiological pathways governing insulin sensitivity, this review compares glycaemic-

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related intervention outcomes such as changes in HbA1c and 24-hour glucose profiles across exercise and control groups. Regular aerobic exercise acts as a low-cost, accessible non-pharmacological intervention. Exploring its effects on glycemic control in sedentary T2DM adults helps establish evidence for lifestyle-based disease management.

2. Definition of key concepts

Sedentary lifestyle means a long term daily life pattern in which individuals accumulate substantial sedentary time across work, transportation or leisure. Individuals may or may not meet moderate-to-vigorous physical activity recommendations [6].

T2DM is a chronic metabolic disorder characterised by persistent hyperglycaemia, which arises from two main effects—insulin resistance and progressive insufficient compensatory insulin secretion from pancreatic β-cells, or both. Genetic susceptibility and modifiable environmental factors, such as obesity, sedentary lifestyle, unhealthy diet and ageing also contribute to it. It may remain asymptomatic for several years and can lead to microvascular and macrovascular complications if controlled poorly [7]. The primary diagnostic test for T2DM worldwide is HbA1c. An HbA1c value ≥ 6.5% is used to classify individuals as having T2DM [7]. Furthermore, there are also other tests for this chronic disease, such as OGTT and FPG. Elevated plasma glucose levels during an OGTT serve as a validated marker for identifying prediabetes and diabetes [8].. Aerobic exercise (also known as cardiorespiratory exercise) refers to rhythmic, sustained and long-term physical activity that involves large-muscle groups, in which oxygen is the primary source for energy production. It is performed at both moderate and vigorous intensity, elevates heart rate and breathing rate, and improves the function of the cardiovascular and respiratory systems [9].

3. Physiological mechanism of T2DM

A sedentary lifestyle is not only a common living habit but also a key contributor to stress on skeletal muscles. Roughly 80% of whole-body glucose uptake mediated by insulin is attributed to skeletal muscle. During prolonged sitting, muscles do not contract, which leads to impaired GLUT4 translocation. Muscle contraction activates the AMPK pathway (an insulin-independent pathway) to translocate GLUT4 glucose transporters to the cell membrane. Prolonged sitting shuts down this non-insulin-dependent route, while the insulin-stimulated PI3K/Akt pathway is also blunted by downstream signaling defects, so glucose entry into cells is substantially reduced [10]. This phenomenon is clinically observable: even in metabolically healthy young adults, 5 days of prolonged uninterrupted sitting can increase postprandial blood glucose peaks by more than 20%, before any measurable change in body weight or systemic inflammation. Furthermore, reduced muscle activity decreases fatty acid oxidation, raising intracellular lipid intermediates. These activate PKC- and JNK kinases, which phosphorylate insulin receptor substrate-1 (IRS-1) at serine residues instead of tyrosine, blocking downstream Akt signaling. The result would be insulin "knocks" but the cell fails to respond. Thirdly, sitting downregulates mitochondrial biogenesis (reduced PGC-1α expression), impairs electron transport chain efficiency, and increases ROS (reactive oxygen species) production. Oxidative stress further activates the NF-κB inflammatory pathway, exacerbating serine phosphorylation of IRS-1 [11-13].

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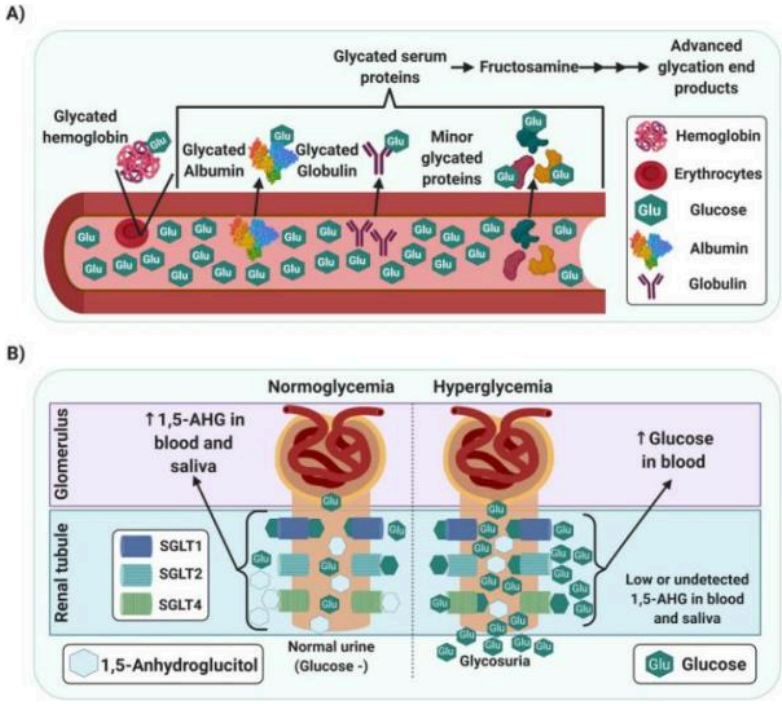


Figure 1. Physiological mechanism of T2DM [7]

4. Aerobic exercise intervention of T2DM

As described in the previous section, prolonged sedentary behaviour impairs glucose uptake via disrupted GLUT4 translocation, mitochondrial dysfunction and chronic low-grade inflammation in skeletal muscle. Regular aerobic exercise counteracts these sedentary-induced molecular defects through multiple interconnected physiological adaptions. Aerobic exercise exerts several beneficial effects on T2DM through a series of integrated physiological adaptations that target the root causes of hyperglycaemia and insulin resistance. First and foremost, regular endurance training enhances skeletal muscle insulin sensitivity significantly. This is achieved via upregulated insulin receptor substrate-1 (IRS-1) tyrosine phosphorylation and improved downstream Akt signalling, which restore the ability of cells to circulate insulin [14]. At the same time, physical exercise triggers the AMP-activated protein kinase (AMPK) signalling pathway. This contraction-dependent, insulin-independent signalling cascade directly boosts glucose uptake and metabolism. AMPK drives the translocation of GLUT4 glucose transporters to the sarcolemma, accelerating rapid glucose entry into muscle cells, and enhances glycogen synthase activity to raise intracellular glycogen stores [15]. The overall process leading to T2DM is shown in Figure 1. In addition to these direct metabolic impacts, aerobic exercise produces strong anti-inflammatory effects. It suppresses the secretion of pro-inflammatory mediators including tumour necrosis factor-α (TNF-α) and interleukin-6 (IL-6) from adipose tissue and immune cells, which in turn mitigates the chronic low-grade inflammation sustaining insulin resistance [16]. Finally, consistent aerobic exercise drives mitochondrial biogenesis (via upregulation of PGC-1α) and enhances oxidative capacity, which improves fatty acid oxidation, reduces intramyocellular lipid accumulation, and elevates cardiorespiratory fitness (maximal oxygen uptake, VO_2max) [17]. Two major GLUT4 translocation pathways are illustrated in Figure 2: insulin-dependent signaling and the insulin-independent contraction-triggered AMPK pathway activated by exercise. Collectively, these mechanisms lower both fasting and postprandial blood glucose, decrease HbA1c levels, and improve systemic

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metabolic health, forming the physiological basis for exercise as a cornerstone of T2DM management.

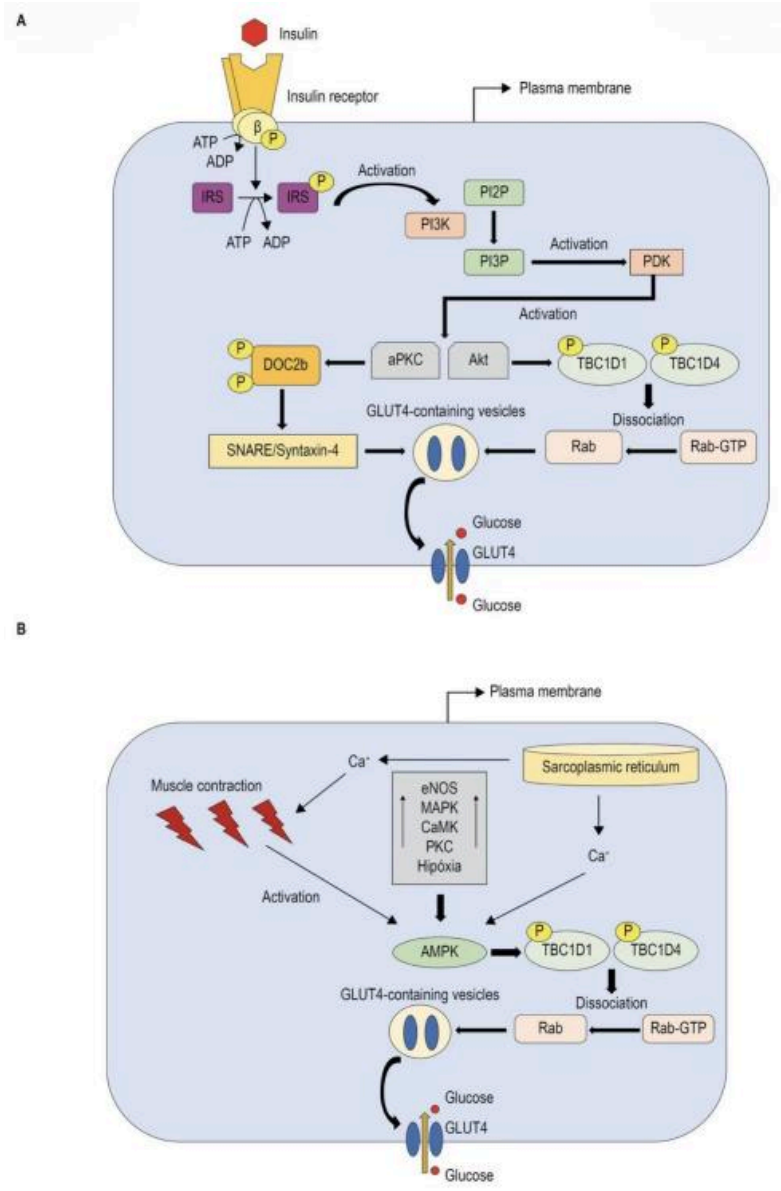


Figure 2. Representation of the main pathways that promote the translocation of GLUT4 containing vesicles to the membrane in the skeletal muscle induced by insulin (A) and insulin-independent pathway during physical exercise (B)

5. Intervention outcomes

5.1. Change in glycaemic and metabolic parameters induced by aerobic exercise

Existing evidence from numerous randomised controlled trials (RCTs) and meta-analyses demonstrates that regular aerobic exercise yields meaningful improvements in glycaemic control and a range of metabolic markers among individuals with T2DM. Glycated haemoglobin (HbA1c), the gold-standard marker for average blood glucose over 2-3 months, is commonly reported as the primary outcome. A network meta-analysis including 158 RCTs (17 059 participants) reported that

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aerobic training reduced HbA1c by -0.58% (95% Crl: -0.70 to -0.45). While other exercise modalities including HIIT, combined training and resistance training also yielded significant HbA1c reductions, detailed cross-modal comparisons are presented [17].

For fasting glucose and related metabolic markers, a meta-analysis focusing on East Asian T2DM participants showed aerobic training lowered fasting blood glucose with a mean difference of -1.49mg/L (95% Cl: -2.53 to -0.44). Another systematic review found exercise interventions improved multiple metabolic indices beyond glycaemia, including total cholesterol, triglycerides, HDL and LDL cholesterol. Intermittent aerobic exercise produced a fasting blood glucose reduction of 0.63 (95% CI: -1.08 to -0.17). Collectively, these metabolic changes represent clinically meaningful benefits for people living with T2DM.

5.2. Differences in efficiency by exercise intensity

Evidence shows that both HIIT and MICT reduce HbA1c effectively, with no significant between-group difference. A meta-analysis of 21 RCTs (792 participants) demonstrated similar outcomes between HIIT and MICT for HbA1c, fasting glucose, HOMA-IR, BMI, total cholesterol, triglycerides, LDL and blood pressure. HIIT, however, was more beneficial for increasing HDL cholesterol (SMD 0.44, $p = 0.023$). Compared with MICT, HIIT led to greater fasting glucose reduction and larger VO_2max improvements. Subgroup analyses suggested that moderate training duration (20–30 minutes), weekly training frequency ≤3 sessions and intervention periods ≤8 weeks optimised HIIT's HDL benefits [18, 19].

In older adults, the ranking of exercise modalities for HbA1c reduction was: HIIT > combined exercise > continuous aerobic exercise > mind–body exercise > resistance training [20]. Aerobic training was more effective than resistance training in reducing HbA1c (MD -0.20%, 95% CI -0.32 to −0.08; $p = 0.0007$), while combined training produced a significantly greater reduction in HbA1c than aerobic training alone (MD −0.17%, 95% CI −0.31 to −0.03; $p = 0.02$) [21].

5.3. Limitations of exercise intervention

Despite robust evidence supporting the efficacy of exercise interventions for T2DM, their practical application faces considerable challenges and potential risks.

Patients differ substantially in age, comorbidities (e.g., cardiovascular disease, peripheral neuropathy, and retinopathy), baseline fitness levels, and glycaemic control status. Exercise prescriptions must therefore follow individualised principles. The resulting increase in blood pressure can cause hemorrhaging of retinal vessels, which is why vigorous exercise is contraindicated for patients with proliferative retinopathy and for three months after laser retinal treatment. In patients with peripheral neuropathy, a history of recurrent falls, or uncontrolled hypertension, exercise may be relatively contraindicated. Exercise may induce cardiac arrhythmic events in patients with long QT syndrome. Inappropriate exercise modality, intensity, or duration may provoke adverse reactions including musculoskeletal injuries, fractures, joint damage, proteinuria, retinal haemorrhage, and even myocardial ischaemia.

Secondly, the greatest challenge of exercise intervention is long-term adherence. A systematic review found that the proportion of participants adhering to physical activity varied considerably across studies. The prevalence of physical exercise non-adherence among T2DM patients ranges widely, with reported rates from 11.7% to 73.6% depending on the population and setting. Studies show that although exercise programmes may initially motivate participants, attendance rates often

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decline significantly after 12 weeks, with some programmes experiencing near-complete dropout [22, 23].

6. Conclusion

In conclusion, a sedentary lifestyle drives T2DM through muscle insulin resistance, lipid dysregulation, and chronic inflammation, while aerobic exercise counteracts these processes by enhancing insulin sensitivity and glucose uptake. Intervention data confirm that regular aerobic training, particularly HIIT and combined exercise, significantly reduces HbA1c (by ~0.5%-0.8%), lowers fasting glucose, and improves VO_2max. However, limitations include short intervention durations, small sample sizes and poor real-world adherence. Future research should prioritise large-scale long-term trials, objective adherence monitoring, phenotype-based stratification, and implementation strategies to translate evidence into sustainable community programmes.

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