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Exercise-Based Interventions in Type 2 Diabetes Mellitus: Effects on Glycemic Control and Metabolic Adaptations. A Narrative Review

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Abstract

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by persistent hyperglycemia, insulin resistance, and progressive β-cell dysfunction, contributing to increased cardiovascular and metabolic complications. Alongside pharmacological treatment, structured physical activity is a fundamental component of contemporary diabetes management, owing to its broad metabolic and functional benefits.

This narrative review aimed to summarize current evidence regarding exercise-based interventions in T2DM, with particular emphasis on their effects on glycemic control, physiological mechanisms, and implications for individualized exercise prescription. A literature review was conducted using peer-reviewed publications published between 2016 and 2026 identified through PubMed/MEDLINE, Scopus, and Google Scholar. The analyzed literature included randomized controlled trials, systematic reviews, meta-analyses, umbrella reviews, mechanistic studies, consensus statements, clinical guidelines, and selected intervention studies related to exercise physiology and diabetes management.

Current evidence indicates that aerobic training, resistance training, high-intensity interval training (HIIT), and combined exercise modalities improve glycemic control, insulin sensitivity, and cardiometabolic outcomes through distinct physiological pathways. Exercise-induced adaptations include enhanced skeletal muscle glucose uptake, improved insulin signaling, increased GLUT4 translocation, and mitochondrial adaptations.

Combined exercise programs appear to provide the most consistent metabolic benefits, whereas HIIT may represent a time-efficient alternative for selected individuals. These findings support individualized exercise prescription as an important strategy for optimizing long-term management of T2DM.

Keywords: type 2 diabetes mellitus, exercise, physical activity, aerobic training, resistance training, high-intensity interval training, HIIT, glycemic control, HbA1c, insulin sensitivity, GLUT4, AMPK, exercise prescription, cardiorespiratory fitness

Introduction

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disease characterized by persistent hyperglycemia and progressive impairment of glucose homeostasis. Its prevalence continues to increase worldwide, including in Poland, where it affects approximately 5–8% of adults [1]. This trend is driven by population aging, sedentary behavior, increasing obesity rates, and progressive metabolic dysfunction, creating a substantial and growing challenge for healthcare systems. Although advances in pharmacological management have improved clinical

outcomes and reduced mortality, prolonged survival contributes to an increasing long-term healthcare and functional burden.

The clinical course of T2DM is primarily determined by insulin resistance affecting skeletal muscle, liver, and adipose tissue, resulting in impaired glucose utilization and progressive metabolic dysregulation [2]. Over time, compensatory hyperinsulinemia contributes to the gradual deterioration of pancreatic β-cell function and to a decline in insulin secretory capacity. These alterations are accompanied by disturbances in lipid metabolism and increased cardiovascular risk, further complicating long-term disease management.

Persistent hyperglycemia contributes to the development of both microvascular and macrovascular complications, including diabetic retinopathy, nephropathy, peripheral neuropathy, and cardiovascular disease, significantly reducing functional capacity and quality of life [1]. Consequently, contemporary management of T2DM extends beyond pharmacological glycemic control and increasingly emphasizes comprehensive long-term risk reduction through lifestyle-based interventions.

Dietary modification, particularly low-carbohydrate and low-glycemic dietary approaches, is an important component of metabolic management and may improve insulin sensitivity and glycemic regulation; however, long-term effectiveness depends strongly on patient adherence [3]. Therefore, behavioral support, continuous patient education, and multidisciplinary care remain essential for achieving sustainable clinical outcomes.

Physical activity is currently recognized as one of the core therapeutic pillars in the non-pharmacological management and functional rehabilitation of T2DM. Regular exercise improves skeletal muscle glucose utilization, enhances insulin sensitivity, reduces visceral adiposity, and helps preserve lean body mass [4]. These adaptations contribute to improved metabolic regulation, better glycemic outcomes, and enhancement of overall functional status. Beyond glycemic control, regular physical activity is associated with improved cardiovascular fitness and broader clinical benefits.

In clinical practice, three principal exercise modalities are commonly implemented: aerobic training, resistance training, and high-intensity interval training (HIIT). Aerobic exercise primarily improves cardiorespiratory capacity and long-term glycemic stability; resistance training supports the preservation of skeletal muscle mass and peripheral glucose disposal. In contrast, HIIT provides a time-efficient strategy associated with improvements in selected cardiometabolic outcomes [6,7]. Due to their complementary physiological and clinical effects, combined exercise interventions are increasingly recognized as a promising and clinically applicable strategy for improving metabolic outcomes in individuals with T2DM [5].

Despite substantial evidence supporting exercise-based interventions, considerable variability remains in clinical outcomes. Uncertainty persists regarding the optimal exercise dose, intensity, frequency, and modality selection, as well as implementation strategies, across different patient populations. Exercise responsiveness also appears to be influenced by factors such as age, sex, disease duration, baseline functional status, and diabetes-related comorbidities [6,8]. Furthermore, current evidence remains inconclusive regarding the comparative effectiveness of HIIT versus moderate continuous exercise and resistance-based interventions for long-term HbA1c reduction and sustained metabolic improvement [7,8].

Therefore, this narrative review aims to synthesize current clinical evidence on exercise interventions for T2DM, with particular emphasis on their therapeutic potential and practical implementation in individualized diabetes management. The objective is to support evidence-based exercise prescription and facilitate integration of structured physical activity into contemporary clinical care pathways for individuals with T2DM [5–7].

Materials and Methods

This narrative review aimed to summarize the current evidence on exercise-based interventions for the management of type 2 diabetes mellitus (T2DM). The review focused on the metabolic and clinical effects of aerobic training, resistance training, high-intensity interval training (HIIT), and combined exercise modalities.

A literature search was conducted using peer-reviewed publications published between 2016 and 2026. The included literature comprised randomized controlled trials, systematic reviews, meta-analyses, umbrella reviews, clinical guidelines, consensus statements, mechanistic studies, and selected clinical intervention studies related to exercise physiology, metabolic adaptation, and diabetes management.

Publications were identified through searches of PubMed/MEDLINE, Scopus, and Google Scholar. The search strategy included combinations of the following keywords: “type 2 diabetes mellitus”, “exercise”, “physical

activity", "aerobic training", "resistance training", "high-intensity interval training", "HIIT", "glycemic control", "HbA1c", "insulin sensitivity", "GLUT4", "AMPK", "exercise prescription", and "cardiorespiratory fitness".

Priority was given to studies evaluating clinically relevant outcomes, including glycated hemoglobin (HbA1c), fasting plasma glucose, insulin resistance, body composition, cardiorespiratory fitness, and functional outcomes. Additionally, mechanistic studies investigating exercise-induced metabolic adaptations—including GLUT4 translocation, AMPK activation, mitochondrial adaptation, inflammatory regulation, and insulin signaling pathways—were included to support physiological interpretation of clinical findings.

Recommendations and consensus documents from the World Health Organization (WHO), the American Diabetes Association (ADA), the ADA/EASD consensus report, and the American College of Sports Medicine (ACSM) were incorporated to contextualize exercise prescription and current standards of diabetes care.

The collected evidence was synthesized narratively to identify clinically relevant exercise strategies and practical approaches supporting individualized metabolic management in individuals with T2DM.

Overview of Current Evidence

Physical activity consistently improves glycemic control in type 2 diabetes mellitus, leading to reductions in HbA1c, fasting glucose, and insulin resistance. All major exercise modalities (aerobic, resistance, HIIT, and combined training) demonstrate beneficial effects, with combined training and HIIT generally showing the greatest improvements in metabolic outcomes. However, evidence indicates substantial inter-individual variability in response to exercise interventions. The most consistent determinant of long-term effectiveness is adherence to regular physical activity rather than specific training modality or intensity.

Biological and Cellular Mechanisms of Physical Activity in T2DM

Metabolic Mechanisms: GLUT4, AMPK, and Mitochondria

The primary physiological pathway through which physical exercise modulates carbohydrate kinetics relies on the accelerated, contraction-induced translocation of glucose transporter type 4 (GLUT4) proteins to the sarcolemma and T-tubules of skeletal muscle cells. This adaptive mechanism is directly activated by mechanical muscle contraction. It operates independently of the proximal insulin signaling cascade, effectively bypassing innate receptor mutations and post-receptor signaling defects characteristic of insulin-resistant diabetic tissues [33].

Exercise-stimulated transmembrane glucose clearance is regulated by parallel downstream signaling cascades activated by structural and energetic cellular stress [14]. Myocyte contraction triggers a dual, interconnected pathway involving both metabolic and calcium-dependent signaling networks. A rapid decrease in the intracellular ATP-to-AMP ratio serves as a direct stimulus for 5'-AMP-activated protein kinase (AMPK)—a master metabolic switch controlling cellular energy homeostasis and intracellular glucose flux. Concurrently, repeated depolarization of the cell membrane induces a transient release of calcium from the sarcoplasmic reticulum, thereby activating calcium/calmodulin-dependent protein kinase II (CaMKII). Both cascades converge at the level of Rab GTPase-activating protein regulation (TBC1D1 and AS160), facilitating the docking of GLUT4 vesicles on the cell surface [14].

Chronic adaptation to structured physical training induces a marked upregulation of the total GLUT4 protein pool within target myocytes, driving long-term improvements in systemic insulin sensitivity in patients with T2DM. Concurrently, systematic physical exercise supports mitochondrial adaptation in skeletal muscle, thereby expanding cellular oxidative capacity. These ultrastructural adaptations enhance intracellular fatty acid beta-oxidation, minimizing the accumulation of lipotoxic lipid intermediates (such as diacylglycerols and ceramides) that physically disrupt the insulin signaling cascade, thus contributing to improved metabolic regulation and peripheral physical capacity [14].

The Role of Physical Activity in the Prevention of Complications

Beyond immediate post-exercise glycemic control, regular physical activity may help prevent long-term diabetic complications, counteracting the progression of diabetic microvascular and macrovascular angiopathies. Clinical data confirm that systematic training significantly reduces the incidence of cardiovascular disease (CVD) and

all-cause mortality by optimizing central hemodynamics, mitigating peripheral insulin resistance, and favorably restructuring the systemic metabolic profile. At the microcirculatory level, targeted exercise may help reduce the progression of selected diabetic complications and improve vascular function by mitigating chronic low-grade vascular inflammation and reducing localized oxidative stress, which damages capillary basement integrity.

These vascular benefits are closely linked to enhanced endothelial function and improved vascular wall tone. Shear stress induced by accelerated pulsatile blood flow during skeletal muscle contraction stimulates endothelial nitric oxide synthase (eNOS), thereby increasing nitric oxide bioavailability and optimizing organ perfusion. Concurrently, systematic training lowers circulating triglyceride concentrations, balances atherogenic plasma lipoprotein fractions, and restores elasticity of large blood vessels [33].

Improvements in macro- and microcirculation exert potent neuroprotective and angioprotective effects within the distal lower extremities, thereby eliminating hypoxic and ischemic conditions in peripheral nerves and cutaneous tissues. Consequently, regular physical activity may help preserve lower-limb function and reduce selected risk factors associated with diabetic foot progression [33]. In clinical rehabilitation, maintaining mobility and supporting tissue perfusion through appropriately prescribed physical activity may help preserve functional independence and reduce selected lower-limb complications [33].

Improvement of Insulin Response and Cellular Resilience

Structured exercise interventions significantly improve insulin response kinetics through multi-level systemic metabolic adaptations.

Increased expression and localized translocation of GLUT4 proteins accelerate transmembrane glucose clearance, contributing directly to reductions in peripheral insulin resistance. Chronic exercise improves metabolic flexibility, increasing the skeletal muscle's functional capacity to utilize glucose under dynamic physical loads.

The therapeutic impact of physical exercise extends beyond peripheral insulin-responsive tissues, modulating pancreatic architecture and secretory function via systemic and paracrine pathways. Systematic training increases circulating concentrations of growth factors—primarily insulin-like growth factors (IGFs)—and key signaling molecules that regulate multi-organ homeostasis. By creating a physiological environment conducive to islet cell survival and stimulating potential beta-cell proliferation, physical exercise stabilizes insulin secretion dynamics. It is associated with improved beta-cell function over time, halting the progression of glucose tolerance impairment [15].

Simultaneously, physical activity acts as a non-pharmacological stimulator of the systemic anti-inflammatory and antioxidant response networks. Training upregulates the activity of endogenous antioxidant enzymes and suppresses the transcription of pro-inflammatory cytokines. This protects the insulin receptor substrate (IRS) from pathological serine phosphorylation, which, under conditions of oxidative stress, blocks intracellular metabolic signaling. These molecular adaptations are structurally anchored in improved mitochondrial quality control, enhanced thermodynamic efficiency of oxidative phosphorylation, and support cellular ATP turnover [15].

Sexual Dimorphism in Physiological and Glycemic Responses

Biological sex significantly influences the clinical efficacy of kinesiotherapy in patients with T2DM. Variations in response to the training stimulus are determined by distinct phenotypic characteristics: baseline body composition, skeletal muscle mass volume, adipose tissue distribution (visceral versus peripheral), and a unique endocrine profile. Although a structured 10-week exercise program leads to a significant reduction in glycated hemoglobin (HbA1c) across all patient cohorts, male participants exhibit a greater and more rapid reduction in HbA1c than female participants do. From a sports physiology perspective, this dynamic is closely linked to greater baseline lean body mass and higher hypertrophic and mechanical potential of male muscle fibers subjected to external resistance loads.

These physiological relationships have been verified in interventional trials, including a cohort study involving 67 adults with T2DM randomized to groups performing combined training (walking and structured exercise) or an isolated protocol. Empirical results confirmed clear sex differences in metabolic clearance and adaptive dynamics. This suggests that contemporary lifestyle medicine must move away from universal, template exercise

recommendations. Programming physical activity in T2DM requires strict individualization and precise adjustment of loads to the patient's sex, body composition, baseline activity level, and hormonal profile [11].

Clinical Programming and Prescription of Physical Activity Volumes

Physical activity must be treated as a structured, dose-dependent clinical intervention in the management of T2DM, rather than a general lifestyle recommendation. According to World Health Organization guidelines, adults should accumulate 150–300 minutes of moderate-intensity or 75–150 minutes of vigorous-intensity aerobic exercise weekly, with established benefits for reductions in cardiovascular and all-cause mortality [29].

In patients with T2DM, the American Diabetes Association recommends at least 150 minutes of moderate-to-vigorous aerobic activity per week, distributed over at least 3 days, with no more than 2 consecutive days without activity [30]. This distribution reflects the transient nature of exercise-induced improvements in insulin sensitivity, which typically persist for 24–48 hours following a single bout of activity, largely mediated by sustained GLUT4 translocation and enhanced non–insulin-dependent glucose uptake [30].

Aerobic training should be complemented by resistance exercise performed at least twice weekly to optimize metabolic outcomes [31]. Resistance training increases skeletal muscle mass, thereby expanding the primary peripheral site of glucose disposal and improving overall insulin sensitivity. In addition, it increases resting energy expenditure and improves body composition [31].

Current clinical recommendations also emphasize frequent interruptions of prolonged sedentary time, with brief bouts of light physical activity. Such interruptions attenuate postprandial glycemic excursions and support endothelial function, reinforcing the metabolic benefits of structured exercise [32]. Collectively, these components require individualized prescription based on patient age, disease duration, comorbidities, and baseline functional capacity [30,32].

Integration of Physical Activity with Daily Lifestyle

Effective exercise-based interventions require integration into daily behavioral patterns rather than isolation from other therapeutic activities. Optimizing insulin sensitivity and preserving beta-cell function depends on appropriate modulation of exercise intensity, training modality, and weekly volume. Embedding physical activity into daily routines, combined with structured dietary management, improves glycemic stability and reduces postprandial variability [8].

Barriers, Psychosocial Determinants, and Support Strategies

Despite well-documented clinical benefits, adherence to physical activity recommendations among people with T2DM remains suboptimal. Common barriers include multimorbidity, reduced functional capacity, aging-related physical decline, limited health literacy, and insufficient self-regulation skills [9,21].

Motivational determinants differ substantially by exercise modality. Aerobic training is more strongly associated with social interaction and a perceived sense of belonging, whereas resistance training is primarily linked to self-efficacy and perceived competence. Across both modalities, long-term adherence is primarily determined by behavioral self-regulation, defined as the clinical ability to plan, monitor, and maintain consistent exercise behavior [16]. Therefore, structured behavioral and educational interventions are essential components of effective long-term disease management strategies.

Clinical Safety Frameworks and Risk Stratification

Before exercise prescription, a comprehensive clinical assessment is recommended to evaluate cardiovascular risk and identify microvascular or macrovascular complications. This is particularly important in sedentary individuals and patients with hypertension, obesity, or long-standing T2DM, where the risk of acute cardiovascular events is elevated.

In patients presenting with diabetic retinopathy, high-intensity isometric exercise and Valsalva-like maneuvers should generally be avoided due to transient increases in intraocular pressure and the associated risk of retinal damage or hemorrhage [33].

In individuals with peripheral neuropathy, high-impact or repetitive weight-bearing activities should generally be limited to reduce the risk of unrecognized foot trauma and related complications [33].

Exercise-induced hypoglycemia is an important clinical consideration, particularly in patients using insulin or insulin secretagogues. Standard care includes rigorous glycemic monitoring before, during, and after exercise to enable appropriate nutritional or pharmacological adjustments. Exercise progression should follow the principle of gradual overload, with continuous adjustments based on functional capacity and comorbidities. Patient education regarding glycemic control, hydration, and appropriate protective footwear is a critical component of risk reduction and long-term adherence [33].

Aerobic Training Paradigms and Metabolic Adaptation

Aerobic exercise consists of sustained, rhythmic activities that engage large muscle groups, such as walking, cycling, and swimming. It is characterized by improvements in cardiovascular and metabolic efficiency, including increased maximal oxygen uptake.

For patients with T2DM, a minimum of 150 minutes of moderate-to-vigorous aerobic activity per week is recommended. Individual sessions should last at least 10 minutes, with optimal adaptations achieved through bouts of approximately 30 minutes or longer distributed throughout the week. This structure is associated with clinically meaningful reductions in HbA1c and improvements in insulin sensitivity.

Aerobic exercise prescription should follow the principle of progressive overload, with gradual increases in duration, frequency, or intensity, adjusted according to individual health status and comorbidities to ensure safety and sustainability [30].

Aerobic Intervention Paradigms and Glycemic Kinetics

Efficacy Thresholds and Dose–Response Relationships

Aerobic exercise has a well-established role in improving glycemic control across the continuum from prediabetes to type 2 diabetes mellitus (T2DM). Evidence from systematic reviews and meta-analyses indicates that structured endurance training reduces fasting blood glucose (FBG), postprandial glucose (2hPG), and glycated hemoglobin (HbA1c), thereby lowering the risk of both microvascular and macrovascular complications [10].

In individuals with prediabetes, aerobic training performed 3–4 times per week induces clinically relevant improvements in HbA1c, body mass index (BMI), and fasting glycemia. While longer session duration may modestly enhance outcomes, clinically meaningful benefits can be achieved even with moderate training volumes, which supports the feasibility of implementation in initially untrained populations [11,22].

In patients with established T2DM, a minimum intervention period of approximately 12 weeks and at least 150 minutes of moderate-to-vigorous aerobic exercise per week is generally required to produce sustained improvements in HbA1c. However, meta-analytic evidence suggests a non-linear dose–response relationship, in which further increases in weekly exercise volume or intensity beyond recommended thresholds do not necessarily yield proportional reductions in HbA1c [17].

This finding has important implications for clinical programming. It suggests that exercise prescriptions should prioritize adherence, feasibility, and long-term sustainability rather than maximal training volume. Achieving the minimum effective dose that stimulates skeletal muscle glucose uptake appears sufficient for clinically relevant metabolic improvement while reducing the risk of injury or acute metabolic complications [11,22].

Temporal Dynamics and Training Adaptations

Long-term efficacy of aerobic interventions is supported by evidence from controlled trials, including the INTENSITY study, which implemented a 28-week structured training program of approximately 150 minutes

per week at moderate-to-high intensity. The intervention resulted in a significant reduction in HbA1c, confirming the effectiveness of sustained aerobic training for long-term glycemic control.

Importantly, improvements in glycemic outcomes were observed regardless of disease duration. Patients with both short-term (<5 years) and long-standing (>5 years) T2DM demonstrated comparable reductions in HbA1c, suggesting that aerobic exercise remains effective across stages of disease progression and levels of metabolic impairment [18].

Exercise Intensity and Metabolic Outcomes

Meta-analytic evidence from randomized controlled trials indicates that structured aerobic exercise produces significant improvements in metabolic parameters, including a mean reduction in HbA1c of -0.71% (95% CI: -1.11 to -0.31; $p = 0.0005$), alongside improvements in insulin resistance measured by HOMA-IR, fasting plasma glucose, and fasting insulin levels [9].

The duration of intervention is a key determinant of effect magnitude, with longer programs associated with progressively greater reductions in HbA1c ($p = 0.002$). This supports the importance of sustained participation in exercise programs as a determinant of long-term metabolic control.

Regarding intensity, moderate training primarily improves glycemic stability and daily glucose regulation, whereas higher-intensity aerobic exercise yields greater improvements in cardiorespiratory fitness. These adaptations are associated with improved cardiovascular risk profiles, suggesting that intensity modulation may be particularly relevant for macrovascular protection, while moderate exercise is sufficient for glycemic regulation [9].

Overall, aerobic exercise demonstrates a non-linear but clinically significant effect on glycemic control in T2DM. The most consistent determinant of efficacy is not maximal intensity or volume, but sustained adherence to a structured program meeting minimal recommended thresholds. This supports a clinical shift toward individualized, adherence-centered exercise prescription rather than escalation toward high-intensity or high-volume regimens [9,10,17,18].

Physiological Biomarkers and Remote Biometric Control in Personalized Aerobic Exercise

Methodological Biomarkers for Intensity Monitoring and Prescription Tailoring

To optimize metabolic outcomes while ensuring hemodynamic safety, prescribing aerobic exercise should rely on individualized physiological metrics rather than standardized templates. Heart rate–based indices, including percentages of maximal heart rate and heart rate reserve, are widely used noninvasive surrogates of exercise intensity and approximate myocardial oxygen consumption, enabling workload adjustment based on real-time cardiovascular tolerance.

However, these indices have important limitations. During conditions involving altered peripheral perfusion or localized muscular occlusion, heart rate responses may not accurately reflect systemic oxygen demand, potentially leading to misclassification of exercise intensity and overestimation of metabolic load [31].

To address this limitation, heart rate monitoring should be complemented by subjective assessment tools such as the Borg Rating of Perceived Exertion (RPE). In patients with autonomic neuropathy or those receiving beta-blocker therapy, where heart rate responses are attenuated, RPE (typically 10–12 on the 6–20 scale) represents a more reliable indicator of exercise intensity regulation [31,33].

Technological Interventions and Remote Biometric Control

Recent advances in digital health have enabled the development of closed-loop exercise systems in which workload is dynamically adjusted based on continuous physiological feedback, particularly heart rate monitoring [19]. Originally applied in highly vulnerable clinical populations, these systems demonstrate the feasibility of remote supervision and automated intensity control.

In the context of T2DM, such technologies may enhance adherence to home-based exercise programs while improving safety through continuous monitoring and real-time adjustment of training intensity. This approach enables multidisciplinary teams to maintain individualized therapeutic control outside traditional clinical environments and reduce the risk of exercise-related adverse events [19,31,33].

Resistance Training Protocols in Metabolic Rehabilitation

Molecular and Myological Mechanisms of Mechanical Loading

Resistance training involves structured exercise performed against external loads, including free weights, machines, bodyweight exercises, and elastic resistance, targeting major skeletal muscle groups to induce neuromuscular adaptation and hypertrophy.

From a metabolic perspective, resistance training improves glycemic control by increasing skeletal muscle mass, which is the primary site of insulin-mediated glucose disposal. These adaptations enhance insulin signaling, improve substrate oxidation, and contribute to reductions in both microvascular and macrovascular complications associated with T2DM. Additional benefits include improved bone mineral density, neuromuscular function, and overall cardiometabolic risk profile [30].

Training Prescription and Functional Adaptation

Current clinical guidelines recommend resistance training 2–3 times per week on non-consecutive days to allow adequate recovery and optimize adaptive responses.

Evidence suggests that higher-load resistance training may produce greater improvements in HbA1c and GLUT4-mediated glucose transport. However, clinically meaningful benefits are also observed across low-to-moderate intensity protocols, which remain effective in improving muscular strength, functional capacity, and daily physical performance.

Importantly, resistance training demonstrates effectiveness across a broad range of intensities and patient phenotypes. This flexibility supports its use as a scalable, adaptable intervention for individuals with T2DM, including older adults and those with reduced functional capacity or sarcopenia, thereby promoting long-term adherence and metabolic stability [30].

Multidirectional Paradigms in Resistance Training: Traditional Loading, Blood Flow Restriction, and Tonic Kinetics

Diversity of Strength Training Modalities and Metabolic Adaptations

Contemporary evidence in sports medicine and metabolic endocrinology indicates that resistance training in type 2 diabetes mellitus (T2DM) should extend beyond traditional high-load protocols to include multiple complementary modalities. These include conventional resistance training, blood flow restriction training (BFRT), and low-load protocols performed with slow movement tempo and sustained muscular tension [20–22].

These modalities improve skeletal muscle strength, facilitate translocation of glucose transporter type 4 (GLUT4), reduce glycated hemoglobin (HbA1c), and improve cardiometabolic risk profiles [20–23]. This heterogeneity supports the use of resistance training as a flexible, scalable intervention for the management of T2DM. Current guidelines highlight its role not only in glycemic regulation but also in attenuating sarcopenia, improving body composition, and supporting long-term metabolic homeostasis [29,30].

Traditional High-Load Training Versus Blood Flow Restriction (BFRT)

Both traditional high-load resistance training and BFRT demonstrate comparable efficacy in improving muscular strength and metabolic outcomes in individuals with T2DM, although they differ in mechanical load and clinical application.

BFRT may induce comparable muscular and metabolic adaptations using low external loads, typically 20–30% of one-repetition maximum (1RM) [29,31]. The method combines partial arterial inflow restriction with venous

occlusion, resulting in localized hypoxia and metabolite accumulation. These conditions promote recruitment of high-threshold motor units and support muscular adaptation. and stimulate downstream anabolic signaling pathways associated with muscular adaptation [28,29,31].

Due to its low mechanical stress, BFRT is particularly suitable for older adults and patients with comorbidities such as osteoarthritis or advanced diabetic microvascular complications. Evidence indicates that BFRT improves lower-limb strength, particularly in the quadriceps, thereby enhancing gait stability and functional independence [29,31]. Additionally, it improves glucose homeostasis and lipid metabolism, making it a viable alternative or adjunct to traditional resistance training for the rehabilitation of T2DM [20–23].

Metabolic Orchestration and Glycemic Kinetics of Resistance Modalities

Cellular Mechanisms, Sarcopenia, and Tissue Specificity

Resistance training significantly affects glucose homeostasis in skeletal muscle, primarily by increasing insulin sensitivity and modulating intracellular metabolic signaling pathways. This intervention is particularly relevant in aging populations, in whom insulin resistance, low-grade systemic inflammation, and unfavorable changes in body composition are commonly observed. Age-related sarcopenia, defined as the progressive loss of skeletal muscle mass and contractile function, is associated with impaired insulin receptor substrate (IRS) signaling and reduced metabolic efficiency. These changes contribute to impaired lipid oxidation and accumulation of lipotoxic intermediates, which further aggravate metabolic dysregulation in skeletal muscle [12].

Although both endurance and resistance training improve insulin sensitivity and glucose regulation in older adults, their underlying adaptations differ. Endurance training primarily enhances mitochondrial biogenesis, oxidative enzyme activity, and capillary density. In contrast, resistance training primarily increases skeletal muscle cross-sectional area, particularly in type I and type II fibers, and enhances GLUT4 expression and insulin receptor availability.

Baseline phenotypic characteristics, including visceral adiposity and the degree of pancreatic β-cell dysfunction, influence the metabolic response to resistance training. In individuals with established T2DM, resistance training increases skeletal muscle mass, thereby expanding peripheral glucose storage capacity. This increases postprandial glucose disposal even in the presence of reduced insulin secretion, supporting short-term glycemic stability [12,13].

Systemic Glycemic Control and Chronic Metabolic Adaptations

Long-term resistance training is associated with improvements in fasting plasma glucose (FPG) and glycated hemoglobin (HbA1c) in individuals with T2DM, as demonstrated in clinical trials and systematic reviews. Evidence suggests that the magnitude of HbA1c reduction may be influenced by intervention duration and biological sex. These differences are likely related to variations in baseline muscle mass, hormonal profiles, and fat distribution between male and female participants. In contrast, age and weekly training frequency do not appear to be strong independent modifiers of the overall glycemic response [11,12].

At the molecular level, chronic resistance training induces sustained alterations in skeletal muscle metabolism. Metabolomic analyses indicate changes in acylcarnitines, branched-chain amino acid metabolites, and tricarboxylic acid (TCA) cycle intermediates, reflecting improved mitochondrial function and substrate utilization [13]. These adaptations are associated with improved fatigue resistance and metabolic efficiency.

Importantly, chronic adaptations differ from acute post-exercise responses, indicating that resistance training induces long-term remodeling of skeletal muscle metabolism rather than transient metabolic shifts. This supports the role of resistance exercise as a chronic regulator of metabolic flexibility in T2DM, rather than a short-term glycemic modulator [12,13].

Synergistic Potency of Combined Training Paradigms

Cellular Integration of Concurrent Modalities on Biphasic Insulin Secretion

The integration of aerobic and resistance training within a concurrent exercise framework represents an effective non-pharmacological strategy for improving metabolic control in individuals with type 2 diabetes mellitus. Evidence from randomized controlled trials indicates that combined training improves biphasic insulin secretion and increases glucose effectiveness to a greater extent than either modality applied in isolation.

Following 12-week structured interventions, improvements are observed in both first-phase (acute insulin response to glucose) and second-phase (sustained insulin release) secretion dynamics. These adaptations improve postprandial glucose regulation and overall carbohydrate homeostasis. Aerobic training primarily enhances microvascular function and mitochondrial capacity, supporting early-phase insulin responsiveness. In contrast, resistance training increases skeletal muscle mass, thereby improving peripheral glucose disposal and contributing more strongly to later-phase insulin dynamics through non-insulin-dependent mechanisms.

Current evidence suggests that reductions in visceral adiposity, lipotoxic burden, and circulating atherogenic lipid fractions are key upstream factors associated with improved β-cell function and preservation of insulin secretory capacity [24].

Comparative Efficacy and Clinical Prescription

Moderate-intensity aerobic training (approximately 50%–85% of maximal heart rate is associated with consistent improvements in insulin sensitivity, β-cell function, and glycated hemoglobin (HbA1c) levels. In contrast, resistance training shows more heterogeneous effects on systemic insulin resistance, with outcomes varying across populations and training designs.

Concurrent training, combining aerobic and resistance modalities, appears to mitigate the limitations observed in single-modality interventions. This approach enhances both pancreatic insulin secretion dynamics and peripheral glucose uptake through coordinated increases in glucose transporter type 4 (GLUT4) translocation and skeletal muscle metabolic capacity.

Overall, exercise prescription should be individualized, taking into account baseline functional status, comorbidities, and metabolic targets to support safety, adherence, and long-term effectiveness [24].

High-Intensity Interval Training (HIIT): Biomechanical Architecture and Chrono-Efficiency

Chrono-efficiency and training dose characteristics

High-Intensity Interval Training (HIIT) consists of repeated bouts of high-intensity exercise alternated with periods of passive rest or low-intensity recovery [25].

In clinical exercise physiology, HIIT is primarily characterized by high time-efficiency, enabling meaningful physiological adaptations with low total weekly training volume, typically 1–3 sessions per week [25,26].

Evidence from studies involving sedentary, overweight, and obese populations indicates that HIIT improves cardiorespiratory fitness and cardiometabolic health, even at low frequencies. A single weekly HIIT session over 8 weeks has been shown to improve peak oxygen uptake (VO_2peak), reduce visceral adiposity, and lower resting blood pressure [26].

These adaptations support the use of low-frequency HIIT as a feasible intervention for populations with limited exercise tolerance or low adherence.

Time-efficient protocols also improve behavioral adherence by reducing the perceived time burden, a major barrier to structured physical activity in inactive individuals [25,26].

Integration within long-term training models

HIIT should not be applied as a standalone intervention in metabolic rehabilitation. Optimal programming integrates HIIT with lower-intensity continuous or resistance-based exercise within a structured weekly distribution. Long-term adaptations, including mitochondrial biogenesis, improved capillary density, and shifts in substrate oxidation, depend on cumulative training exposure rather than on isolated high-intensity sessions. Therefore, HIIT functions as a high-intensity stimulus within a broader training continuum rather than a complete intervention model. Training effectiveness depends on the interaction of three variables: intensity, interval structure, and weekly frequency [27].

Evidence supports a polarized distribution model in which most training volume is performed below the first lactate threshold (LT1), with HIIT providing a limited but potent high-intensity stimulus. This approach minimizes autonomic strain while maintaining sufficient stimulus for cardiometabolic adaptation [26,27].

Metabolic efficacy in type 2 diabetes mellitus

Meta-analytic evidence indicates that HIIT significantly improves glycemic control and cardiorespiratory fitness in patients with type 2 diabetes mellitus (T2DM). Data from 10 systematic reviews, including 2,954 participants, demonstrate greater reductions in glycated hemoglobin (HbA1c) and greater increases in VO_2peak than in sedentary controls and moderate-intensity continuous training (MICT).

The primary mechanistic basis includes the recruitment of high-threshold motor units and the depletion of intramuscular glycogen stores, which activate the AMP-activated protein kinase (AMPK) and PGC-1α signaling pathways. This enhances insulin-independent glucose uptake via GLUT4 translocation to the sarcolemma.

Clinically, HIIT is associated with reductions in fasting plasma glucose, fasting insulin levels, and insulin resistance as measured by HOMA-IR [7].

Cardiometabolic and vascular adaptations

Beyond glycemic regulation, HIIT improves body composition by reducing visceral fat mass and preserving lean tissue. Repeated exposure to high shear stress increases endothelial nitric oxide synthase (eNOS) activity, improving endothelial function, arterial compliance, and resting blood pressure.

Additional benefits include improvements in lipid profiles, particularly reductions in triglycerides and increases in HDL cholesterol, supporting a reduction in overall cardiovascular risk burden in T2DM populations [7].

Clinical implementation and safety considerations

HIIT is associated with high adherence due to reduced time requirements and perceived efficiency. However, its clinical implementation requires careful patient selection.

The acute hemodynamic and sympathetic responses induced by supramaximal workloads may pose risks in individuals with advanced diabetic complications, including proliferative retinopathy, autonomic neuropathy, or occult cardiovascular disease.

Therefore, individualized risk stratification and gradual progression of intensity are required. Further long-term trials are needed to refine safety thresholds and significantly improve prescription models in high-risk diabetic populations [7].

Interval Duration Metrology and Downstream Metabolic Adaptations

Dose structure and training adaptation sensitivity

The duration of work and rest intervals in high-intensity interval training (HIIT) is a key programming variable that influences metabolic and anthropometric adaptations in individuals with type 2 diabetes mellitus (T2DM). Evidence indicates that physiological responses to interval training are dose-dependent and vary according to both interval duration and total weekly training volume [7].

Longer work intervals (approximately 60–120 seconds per bout), particularly when combined with higher weekly training volume, are associated with greater improvements in visceral adiposity, glycemic control, and cardiorespiratory fitness than shorter interval formats and sedentary conditions [7,28]. In contrast, very short sprint protocols may produce smaller or less consistent effects on metabolic outcomes in T2DM populations.

Compared with moderate-intensity continuous training (MICT), extended-duration HIIT protocols appear to produce more pronounced reductions in body mass, waist circumference, fasting plasma glucose (FPG), and glycated hemoglobin (HbA1c), particularly in individuals with established insulin resistance [7].

Metabolic mechanisms associated with interval duration

The physiological responses to interval duration are linked to differential engagement of energy systems. Short-duration intervals predominantly rely on phosphagen (ATP-PCr) and fast glycolytic pathways, producing high acute metabolic stress with relatively limited sustained systemic adaptation.

Longer intervals increase reliance on glycolytic and oxidative metabolism, leading to greater metabolic perturbations, including intramuscular glycogen depletion, lactate accumulation, and transient reductions in local oxygen availability. These conditions are associated with activation of AMP-activated protein kinase (AMPK), a central regulator of cellular energy homeostasis [7].

Activation of AMPK is linked to downstream signaling involving peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PGC-1α) and increased translocation of glucose transporter type 4 (GLUT4), supporting enhanced insulin-independent glucose uptake in skeletal muscle.

Extended interval protocols are also associated with increased post-exercise oxygen consumption (EPOC), which contributes to elevated lipid oxidation and improved systemic substrate utilization.

Inflammatory and adipose tissue–related adaptations

Longer HIIT interventions have been associated with reductions in markers of chronic low-grade inflammation, including tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and C-reactive protein (CRP) [28]. These changes may improve adipose tissue function and reduce visceral fat accumulation.

Collectively, improvements in skeletal muscle glucose uptake capacity and reductions in systemic inflammatory burden are associated with reduced insulin resistance indices (e.g., HOMA-IR) and improved long-term glycemic control in T2DM populations [7,28].

Clinical interpretation

Overall, evidence suggests that longer work-interval HIIT protocols, combined with sufficient weekly training volume, may elicit more consistent improvements in metabolic and anthropometric outcomes than shorter-interval formats in individuals with type 2 diabetes mellitus. However, the magnitude of adaptation is also influenced by baseline fitness, disease severity, and total weekly exercise adherence.

Further studies are required to refine optimal interval duration thresholds across different T2DM phenotypes and to establish individualized prescription models balancing efficacy and safety [7,28].

Discussion

This narrative review supports exercise training as a central component of contemporary management of type 2 diabetes mellitus (T2DM), with consistent evidence demonstrating clinically meaningful improvements in glycemic control, insulin sensitivity, functional capacity, and overall cardiometabolic health. Across aerobic, resistance, high-intensity interval training (HIIT), and combined exercise modalities, structured physical activity improves metabolic outcomes through partially overlapping yet clinically distinct mechanisms.

One of the most important observations emerging from the current evidence is the absence of a universally superior exercise modality across all clinical endpoints. Aerobic exercise demonstrates consistent benefits for cardiorespiratory fitness and long-term glycemic stability; resistance training supports preservation of skeletal muscle mass and peripheral glucose disposal, whereas HIIT offers a time-efficient strategy associated with favorable cardiometabolic adaptations. Combined exercise interventions appear to integrate complementary physiological responses and may provide broader clinical benefits than isolated modalities [6–8,32].

Importantly, current evidence suggests that the relationship between exercise dose and metabolic benefit is non-linear. Achievement of guideline-based activity thresholds appears sufficient to induce clinically meaningful improvements in glycemic outcomes, whereas further increases in training volume or intensity do not consistently translate into proportional reductions in HbA1c [17]. This observation shifts attention from maximizing exercise exposure toward improving feasibility, long-term implementation, and sustained participation.

Clinical response to exercise remains highly heterogeneous, posing an important challenge for exercise prescription in T2DM populations. Factors including disease duration, body composition, baseline physical capacity, age, and diabetes-related complications appear to influence training responsiveness. Although some studies suggest sex-related differences in adaptation, current evidence indicates that baseline physiological characteristics may account for a substantial proportion of the observed variability [11,12]. These findings support individualized and phenotype-oriented exercise prescription rather than standardized exercise algorithms.

From a physiological perspective, exercise improves glucose regulation by enhancing skeletal muscle glucose utilization, improving insulin responsiveness, and promoting favorable changes in metabolic flexibility. However, available evidence suggests that these adaptations are multifactorial and cannot be attributed to a single dominant pathway. Therefore, clinical decision-making should prioritize patient characteristics and implementation feasibility rather than the mechanistic superiority of individual exercise modalities.

Despite strong evidence supporting exercise interventions, long-term adherence remains a significant barrier to their translation into routine clinical practice. Behavioral factors, psychosocial determinants, functional limitations, and healthcare system constraints frequently reduce sustained participation in structured exercise programs. Accordingly, future interventions should focus not only on optimizing exercise prescription but also on improving implementation strategies, behavioral support, and integration of digital health tools to support long-term engagement.

Collectively, current evidence supports a transition from modality-centered exercise recommendations to individualized, patient-centered therapeutic models. Long-term adherence, sustainability, and adaptation of exercise programs to patient-specific clinical characteristics appear to represent the primary determinants of successful metabolic management in individuals with T2DM.

Conclusion

Exercise training is an effective and clinically applicable therapeutic strategy that supports long-term metabolic control in individuals with type 2 diabetes mellitus.

Aerobic exercise, resistance training, HIIT, and combined exercise interventions contribute to clinically meaningful improvements in glycemic regulation, insulin sensitivity, and cardiometabolic health through partially distinct but complementary physiological adaptations.

Current evidence suggests that the long-term effectiveness of exercise interventions depends less on identifying a single superior modality and more on the sustainable implementation of structured physical activity tailored to individual patient characteristics.

Therefore, contemporary exercise prescription should move beyond generalized recommendations and increasingly adopt individualized, multimodal approaches that account for functional capacity, comorbidities, metabolic goals, and long-term adherence.

Future research should prioritize optimizing exercise dose–response relationships, refining clinically applicable prescription models, and developing strategies to support sustainable implementation and behavioral adherence.

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AI

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