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The Importance of Resistance Training in Addressing Insulin Resistance and Type 2 Diabetes: A Narrative Review

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Abstract

Introduction: Insulin resistance and type 2 diabetes mellitus are serious global health challenges, which are caused by aging population, increasing levels of obesity and insufficient amount of physical activity. Although pharmacological treatment is still a central part of managing diabetes, a substantial number of patients are not able to achieve optimal glycemic control, showing the need for effective lifestyle interventions. Resistance training has become a promising strategy which targets main pathophysiological mechanisms underlying insulin resistance.

Purpose: This narrative review aims to summarize and critically evaluate current evidence on the effects of resistance training on insulin sensitivity, metabolism of glucose and cardiometabolic health across diverse populations, including patients with type 2 diabetes, insulin resistance, obesity, metabolic syndrome, and older adults.

Material and Method: A comprehensive literature search was conducted using the PubMed/MEDLINE database and included studies published up to 2025. Only peer-reviewed articles written in English were included in the review. Particular emphasis was placed on randomized controlled trials, systematic reviews, and meta-analyses.

Results: Evidence from randomized controlled trials and meta-analyses demonstrates that resistance training improves glycemic control, reduces insulin resistance as well as enhances body composition and functionality. Comparative analyses indicate that resistance training provides

benefits that are complementary to aerobic exercise, dietary interventions, and pharmacotherapy, particularly when weight loss strategies are added.

Conclusions: The findings support resistance training as a safe and effective component of comprehensive management strategies for insulin resistance and type 2 diabetes.

Key words: Resistance training; Insulin resistance; Type 2 diabetes mellitus; Glycemic control; Skeletal muscle; Metabolic health

1. Introduction

1.1 Epidemiology of Insulin Resistance and Type II Diabetes

Insulin resistance and type 2 diabetes mellitus (T2DM) constitute major and rapidly expanding global public health problems. A recent systematic review and meta-analysis estimated the global prevalence of insulin resistance at approximately 26.53% among adults, showing that more than one in four individuals worldwide exhibit a defect in insulin sensitivity, a crucial precursor of T2DM [1]. Epidemiological analyses indicate that T2DM affects hundreds of millions of people worldwide, with approximately 462 million patients living with this disease in 2017, which represents over 6% global prevalence and considerable mortality burden [2]. More recent estimations further document that both the absolute and relative burden of T2DM continued to increase between 1990 and 2021, driven by aging population, demographic changes, and risk factors such as obesity and lack of physical activity, with projections suggesting continued rises in prevalence and disease impact in upcoming decades [3]. These trends highlight the urgent need for effective preventive strategies which target insulin resistance and its progression to diabetes at the population level.

1.2 Limitations of Conventional Treatments

Despite the availability of multiple pharmacological options for type 2 diabetes mellitus (T2DM), a substantial proportion of patients fail to achieve and maintain target glycemic control with conventional therapies. Reviews have consistently shown that recommended glycemic goals are achieved by less than half of people with T2DM, with <50–56% of patients reaching personalized targets in real-world settings, even with intensive glucose-lowering regimens and routine monitoring in high-income countries [4,5]. Conventional treatment regimens, including metformin, sulfonylureas, and insulin, while effective in reducing glycosylated hemoglobin, are often limited by adverse effects, declining efficacy over time, and difficulties with adherence, particularly as the disease progresses and β -cell function deteriorates [6,7]. Clinically, barriers such as therapeutic inertia — the delay in initiating or intensifying treatment when glycemic targets are not met — contribute significantly to suboptimal outcomes, with many patients spending years above target before treatment adjustments occur [8]. Moreover, patient-related factors, including fear of hypoglycemia, weight gain, injection burden, and psychosocial perceptions of treatment as a “failure,” further compromise adherence and engagement with therapy [8]. Collectively, these limitations highlight that pharmacological approaches alone do not comprehensively address the multifaceted pathophysiology of T2DM — including insulin resistance, obesity, lifestyle factors, and psychosocial determinants — underscoring the need for integrative strategies that combine optimized medical therapy with lifestyle interventions and individualized patient support.

1.3 Mechanisms of Insulin Resistance

Insulin resistance, defined as a diminished responsiveness of insulin-target tissues to physiological insulin concentrations, underlies the pathogenesis of type 2 diabetes mellitus and is driven by multiple interrelated molecular and cellular mechanisms [9]. A central feature of insulin resistance is impaired insulin signaling, where defects at the level of the insulin receptor and downstream signaling pathways, including altered insulin receptor substrate (IRS) phosphorylation and disrupted PI3K activation, reduce glucose uptake and glycogen synthesis in skeletal muscle and adipose tissue [10]. Mitochondrial dysfunction further contributes to insulin

resistance by impairing oxidative phosphorylation and lipid handling, leading to an imbalance between fatty acid supply and oxidation that promotes accumulation of lipid intermediates that interfere with normal insulin action [10]. Chronic low-grade inflammation, particularly in adipose tissue and liver, plays a key role by releasing pro-inflammatory cytokines that activate serine kinases, exacerbating insulin signaling defects and promoting systemic metabolic dysfunction [11]. Another major mechanism is ectopic fat deposition — the accumulation of triglycerides and lipid metabolites in non-adipose tissues such as liver, skeletal muscle, and pancreas — which is strongly associated with cellular lipotoxicity and impaired insulin action; this phenomenon is observed both in obesity and in conditions with limited adipose storage capacity, such as lipodystrophy [12]. Collectively, these pathogenic processes impair glucose homeostasis at multiple levels and represent key targets for therapeutic interventions aimed at restoring insulin sensitivity.

1.4 Role of Skeletal Muscle in Glucose Homeostasis

Skeletal muscle plays a central role in whole-body glucose homeostasis and is the primary site of insulin-mediated glucose disposal in humans. Under postprandial conditions, approximately 80% of glucose uptake occurs in skeletal muscle, highlighting its dominant contribution to systemic glycemic regulation [13]. Insulin stimulates glucose uptake in muscle fibers primarily through translocation of glucose transporter type 4 (GLUT-4) to the cell membrane, a process tightly linked to intracellular insulin signaling and muscle metabolic capacity [13]. Beyond insulin-dependent mechanisms, skeletal muscle also regulates glucose homeostasis through contraction-mediated glucose uptake, which occurs independently of insulin and provides an alternative pathway for glucose disposal during and after physical activity [14]. Collectively, these findings underscore skeletal muscle as a key metabolic organ in glucose regulation and a critical therapeutic target for interventions aimed at improving insulin sensitivity and metabolic health.

1.5 Mechanisms by Which Resistance Training Influences Glucose Metabolism

Resistance training (RT) improves glucose metabolism through several distinct but complementary physiological mechanisms. One of the most well-documented adaptations is an increase in skeletal muscle GLUT-4 expression and translocation, which enhances insulin-stimulated glucose uptake and contributes to improved glycemic control, including in individuals with type 2 diabetes [15]. RT also activates adenosine monophosphate-activated protein kinase (AMPK) during muscle contraction, promoting glucose uptake through insulin-independent pathways and providing an alternative mechanism for glucose disposal in insulin-resistant states [16].

In addition, RT induces favorable changes in mitochondrial content and function, improving oxidative capacity and metabolic efficiency of skeletal muscle, which supports enhanced substrate utilization and insulin sensitivity [17]. RT further contributes to glucose homeostasis by promoting a reduction in intramuscular and visceral fat, depots closely associated with insulin resistance and metabolic dysfunction [18]. Another important mechanism is the increase in muscle mass and insulin-responsive tissue volume, which expands the body's capacity for glucose uptake and storage, thereby improving whole-body insulin sensitivity [19]. Finally, RT exerts beneficial effects on chronic inflammation and oxidative stress, reducing pro-inflammatory markers and enhancing antioxidant defenses, which together support improved insulin signaling and metabolic health [20]. Collectively, these mechanisms explain the robust and clinically meaningful effects of RT on glucose regulation.

2. Materials and Methods

This narrative clinical review was conducted to summarize and critically evaluate current evidence on the effects of resistance training on insulin resistance and type 2 diabetes mellitus. A comprehensive literature search was performed using the PubMed/MEDLINE database, covering studies published up to 2025. Search terms included combinations of “resistance training,” “strength training,” “insulin resistance,” “type 2 diabetes,” “glycemic control,” “HbA1c,” “met-

abolic syndrome,” “obesity,” and “older adults.” Only peer-reviewed articles published in English were considered. Priority was given to randomized controlled trials, systematic reviews, and meta-analyses involving adult populations, including individuals with type 2 diabetes, insulin resistance, obesity, metabolic syndrome, or age-related sarcopenia. Studies focusing exclusively on aerobic exercise without a resistance training component were excluded unless used for comparative purposes. Relevant articles were screened based on title and abstract, followed by full-text assessment for eligibility. Data extraction focused on study design, population characteristics, intervention protocols, and key metabolic and clinical outcomes. The findings were synthesized qualitatively to provide an integrated overview of the efficacy, mechanisms, and clinical applicability of resistance training in the management of insulin resistance and type 2 diabetes.

3. Results

3.1 RT vs No Exercise

Evidence from randomized controlled trials and meta-analyses consistently demonstrates that resistance training (RT), compared with non-exercise or usual-care control conditions, leads to clinically meaningful improvements in glycemic control, insulin sensitivity, and cardiometabolic health in individuals with type 2 diabetes (T2DM). A recent meta-analysis showed that RT significantly reduces fasting insulin, HOMA-IR, fasting plasma glucose, and HbA1c, while also inducing modest reductions in body mass index and increases in skeletal muscle mass and strength, indicating simultaneous metabolic and functional benefits [21]. This analysis also reported reductions in systemic inflammation, reflected by lower C-reactive protein concentrations, although effects on pro-inflammatory cytokines were inconsistent [21].

Additional meta-analytic evidence confirms that RT favorably modifies multiple cardiovascular risk factors in T2DM, including triglycerides, waist circumference, body fat percentage, blood pressure, and high-density lipoprotein cholesterol, highlighting benefits beyond glycemic indices alone [22]. Similar improvements in clustered metabolic syndrome components—such

as fasting glucose, central adiposity, and blood pressure—have been reported in systematic reviews focusing on populations with abnormal glucose metabolism [23,24]. In middle-aged and older women with T2DM and overweight or obesity, RT significantly reduced fasting glucose and HbA1c, with greater glycemic improvements observed when training intensity exceeded 70% of one-repetition maximum, suggesting an intensity-dependent metabolic response [25].

Mechanistic evidence from controlled trials indicates that RT enhances glucose disposal primarily through increases in fat-free mass, thereby expanding the volume of insulin-responsive tissue, rather than by altering the intrinsic insulin sensitivity of muscle fibers—a mechanism distinct from endurance training adaptations [26]. Importantly, a meta-regression analysis demonstrated that greater gains in muscular strength were associated with larger reductions in HbA1c, underscoring the relevance of neuromuscular adaptation for glycemic improvement [27]. Evidence from structured exercise RCTs further shows that RT, delivered alone or within multimodal programs, significantly improves fasting and postprandial glucose, insulin resistance indices, functional capacity, and quality of life compared with non-exercise controls [28].

RCTs in specific clinical subgroups reinforce these findings. In individuals with T2DM and possible sarcopenia, progressive resistance-based interventions produced greater reductions in HbA1c and larger improvements in muscle strength than control conditions [29]. Finally, pooled evidence from multiple meta-analyses confirms that RT consistently lowers HbA1c and fasting glucose across diverse study designs and populations, with training duration and participant characteristics influencing the magnitude of response [30]. Collectively, the convergence of evidence from RCTs and meta-analyses establishes RT as an effective standalone intervention for improving glycemic control, cardiometabolic risk, and functional health in T2DM when compared with no structured exercise.

3.2 Resistance vs Aerobic Training

Clinical trials comparing resistance training (RT), aerobic training (AT), and their combination demonstrate that these exercise modalities exert both overlapping and distinct effects on glyce-mic control and cardiometabolic health in individuals with impaired glucose regulation and type 2 diabetes mellitus (T2DM). In the STRRIDE-AT/RT trial, eight months of RT alone did not significantly improve the metabolic syndrome (MetS) score, whereas AT led to modest improvements and combined AT+RT produced a significant reduction, suggesting that aerobic components are particularly important for improving clustered MetS risk, while RT alone may be insufficient for this composite outcome [31]. Similarly, in obese patients with T2DM under-going dietary treatment, combined aerobic and resistance training did not outperform aerobic training alone for insulin sensitivity, endothelial function, adipokine profiles, or inflammatory markers, indicating that AT may exert stronger effects on certain cardiometabolic pathways in this population [32].

However, more recent evidence highlights important contexts in which RT provides distinct or superior benefits. In individuals with normal-weight T2DM, strength training produced greater reductions in HbA1c than aerobic training, alongside selective increases in appendicular lean mass relative to fat mass; changes in muscle composition independently predicted improve-ments in glyce-mic control, underscoring a muscle-mediated mechanism specific to RT [33]. Comparable findings have been reported in prediabetic populations, where RT led to greater short-term improvements in glycosylated serum protein than AT following a 12-week interven-tion, suggesting enhanced control of recent glyce-mic exposure [34]. Network meta-analytic evidence further supports the efficacy of combined AT+RT for improving BMI, HbA1c, insulin resistance, and inflammatory markers, while also indicating that no single exercise modality is universally superior across all outcomes [35].

Large randomized trials reinforce the complementary nature of these modalities. Both AT and RT independently improve glyce-mic control in T2DM, but the greatest reductions in HbA1c are consistently observed with combined training protocols performed three times per week over extended periods [36,37]. Meta-analyses focusing specifically on combined aerobic and

resistance training demonstrate significant benefits across glycemic, blood pressure, inflammatory, fitness, and quality-of-life outcomes compared with single-modality training, particularly in individuals with overweight or obesity [38]. Additional evidence suggests that combined training may also improve inflammatory profiles and autonomic function in patients with diabetic cardiovascular autonomic neuropathy [39]. Collectively, these findings indicate that while RT and AT each confer meaningful metabolic benefits, their relative effectiveness depends on the targeted outcome and population, with combined training often providing the most comprehensive improvements.

3.3 RT in Overweight and Obese Patients

Clinical evidence from randomized controlled trials and meta-analyses indicates that resistance training (RT) is an effective intervention for improving insulin resistance and cardiometabolic health in overweight and obese adults. A recent meta-analysis focusing on adults with overweight or obesity without diabetes demonstrated that RT significantly reduced fasting insulin and HOMA-IR, confirming an independent effect of RT on insulin resistance even in the absence of overt hyperglycemia [40]. Subgroup analyses revealed greater improvements among men compared with women and in individuals younger than 40 years, suggesting that sex and age may moderate responsiveness to RT [40]. In obese men with type 2 diabetes, short-term RT interventions have been shown to lower fasting glucose and insulin concentrations and elicit near-significant reductions in HbA1c, alongside substantial gains in muscular strength and endurance compared with non-training controls [41].

Mechanistic insights from controlled trials in overweight and obese young men further demonstrate that RT improves glucose tolerance and skeletal muscle insulin sensitivity, as reflected by favorable changes in oral glucose tolerance test–derived indices and increased expression of key proteins involved in glucose transport and metabolism, including GLUT-4 and AKT2 [42]. Evidence from clinical populations with complex phenotypes also supports the metabolic relevance of RT. In overweight or obese breast cancer survivors, combined resistance and aerobic

exercise significantly improved markers of sarcopenic obesity, insulin signaling, adipokine profiles, and metabolic syndrome components compared with usual care, with sustained benefits observed at follow-up [43].

Large meta-analyses in individuals with type 2 diabetes and concurrent overweight or obesity confirm that RT performed as a standalone intervention improves waist circumference, lipid profiles, fasting glucose, insulin, and HbA1c compared with standard care without exercise [44]. Broader exercise-based evidence in overweight and obese adults further indicates consistent reductions in blood pressure, insulin resistance, and intrahepatic fat following structured training programs, with particularly pronounced effects observed in those with type 2 diabetes [45]. Collectively, these findings establish RT as a clinically effective strategy for improving insulin resistance, body composition, and cardiometabolic risk in overweight and obese populations.

3.4 RT in Elderly Patients

Evidence from randomized controlled trials and meta-analyses consistently indicates that resistance training (RT) improves insulin sensitivity, glycemic control, and musculoskeletal health in older adults. A meta-analysis of RCTs in elderly populations demonstrated that RT significantly reduced HOMA-IR and HbA1c, with subgroup analyses revealing that training intensity, intervention duration, and population characteristics substantially influenced outcomes [46]. High-intensity and longer-term RT protocols produced the largest improvements in insulin resistance, whereas moderate-intensity and shorter-duration programs appeared particularly effective for reducing HbA1c in older adults with type 2 diabetes (T2D) [46]. These findings align with earlier meta-analytic evidence showing that RT consistently lowers HbA1c and markedly increases muscular strength in elderly individuals with T2D, even when changes in lean body mass are modest [47].

Controlled mechanistic studies further support these clinical findings. RT has been shown to enhance insulin-stimulated glucose uptake and improve muscle function in both healthy elderly individuals and older adults with diabetes, with concurrent benefits for functional capacity and

reduced risk of falls and fractures [48]. In older hypertensive adults, RT significantly increased insulin-mediated glucose disposal measured by hyperinsulinemic–euglycemic clamp techniques, alongside gains in lean body mass and strength, despite minimal changes in circulating inflammatory markers [49]. Landmark RCTs in older adults with T2D demonstrated that high-intensity progressive RT performed three times weekly over 16 weeks substantially reduced HbA1c, increased muscle glycogen storage, and allowed for reductions in diabetes medication dosage in the majority of participants compared with controls [50]. Similarly, twice-weekly progressive RT significantly improved insulin sensitivity and reduced fasting glucose while decreasing abdominal adiposity in older men with T2D, independent of weight loss [51].

Beyond glycemic outcomes, RT plays a critical role in counteracting age-related sarcopenia. Meta-analytic evidence confirms that resistance exercise effectively increases lean body mass in aging adults, particularly when higher training volumes are employed [52]. In older individuals with established sarcopenia, network meta-analyses indicate that resistance-based exercise programs—alone or combined with aerobic and balance training—are among the most effective interventions for improving physical function and quality of life, with additional benefits observed when nutritional support is included [53]. Collectively, these findings support RT as a safe and effective intervention for improving metabolic health, preserving muscle mass, and enhancing functional outcomes in elderly populations.

3.5 RT Combined With Weight Loss

Clinical evidence indicates that resistance training (RT) combined with weight loss produces superior metabolic outcomes compared with either intervention alone, particularly in older and overweight populations. In postmenopausal women, RT significantly improved insulin action and reduced hyperinsulinemia regardless of weight change; however, reductions in body weight and fat mass occurred only when RT was combined with caloric restriction, highlighting the complementary effects of exercise and energy deficit [54]. Importantly, gains in muscular strength were observed independent of weight loss, underscoring the neuromuscular benefits of RT [54].

Randomized controlled trials in older adults with overweight or obesity further demonstrate that RT combined with caloric restriction leads to meaningful improvements in metabolic syndrome components, including reductions in abdominal obesity, blood pressure, triglycerides, and very-low-density lipoprotein cholesterol, whereas RT alone did not significantly alter these cardiometabolic risk factors despite improving strength [55]. In older patients with type 2 diabetes, high-intensity progressive RT combined with moderate weight loss resulted in significantly greater reductions in HbA1c compared with weight loss alone, despite similar losses in body weight and fat mass [56]. Notably, lean body mass increased with RT plus weight loss but declined with weight loss alone, indicating that RT preserves metabolically active tissue during caloric restriction [56]. Together, these findings support RT combined with weight loss as an effective strategy for improving glycemic control while maintaining lean mass in older, metabolically compromised individuals.

4. Conclusions

Resistance training represents a safe, effective, and clinically relevant intervention for improving insulin sensitivity, glycemic control, and cardiometabolic health across diverse populations, including individuals with type 2 diabetes, insulin resistance, obesity, and older adults. Evidence from randomized controlled trials and meta-analyses consistently demonstrates that resistance training reduces HbA1c, fasting glucose, insulin resistance indices, and central adiposity while preserving or increasing lean body mass and muscular strength. Importantly, resistance training provides metabolic benefits that are complementary to dietary interventions, pharmacotherapy, and aerobic exercise, and it plays a critical role in mitigating age-related sarcopenia and functional decline. When combined with weight loss or other lifestyle modifications, resistance training enhances glycemic outcomes while protecting metabolically active tissue, supporting long-term metabolic health. Despite heterogeneity in training protocols and study designs, the overall body of evidence supports the inclusion of resistance training as a core component of lifestyle management strategies for insulin resistance and type 2 diabetes. Future research should prioritize standardized training prescriptions, long-term clinical outcomes, and personalized approaches to maximize the therapeutic potential of resistance training in clinical practice.

5. Disclosures:

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