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THE ROLE OF RESISTANCE TRAINING IN MODULATING INSULIN RESISTANCE: A NARRATIVE REVIEW

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ABSTRACT

Introduction: Insulin resistance (IR) is a key feature in the development of type 2 diabetes mellitus (T2DM) and a major contributor to metabolic dysfunction, with skeletal muscle being the primary site of insulin-stimulated glucose uptake. Resistance training (RT) has been shown to influence multiple mechanisms that improve insulin sensitivity, including increased muscle perfusion, enhanced muscle mass, activation of IRS-1/PI3K/Akt and AMPK/PGC-1α pathways, and promotion of GLUT4 translocation. RT may also alter muscle fiber composition and induce the release of myokines such as IL-6, irisin, and BDNF, which participate in the regulation of glucose and lipid metabolism and inflammation. Intervention studies report improvements in insulin sensitivity, glycemic control, and metabolic markers across different populations. This narrative review summarizes current evidence on how RT affects skeletal muscle insulin resistance and related metabolic adaptations.
Methods: A literature review was written using databases such as PubMed, Google Scholar and the official WHO website. The research was conducted using articles available as of March 2026.
Conclusions: Resistance training improves insulin sensitivity in skeletal muscle through enhanced perfusion, muscle hypertrophy, GLUT4 translocation, and activation of key signaling pathways. It may also modify muscle fiber composition and stimulate myokine release. Overall, RT consistently supports metabolic health and is a useful strategy for managing insulin resistance and type 2 diabetes.

KEYWORDS

Insulin Resistance, Insulin Sensitivity, Resistance Training, Resistance Exercise, Strength Exercise, Skeletal Muscle, Glucose Uptake, Type 2 Diabetes Mellitus, Myokines, AMPK, PGC-1α, GLUT4, BDNF, IL-6, Irisin, Metabolic Health

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Introduction

Despite extensive mechanistic knowledge of insulin resistance and the role of skeletal muscle, the effectiveness of resistance training in improving metabolic health across different populations remains an area of active research. Resistance training is a form of physical activity aimed at improving muscular fitness by working muscles against external resistance, using equipment such as free weights, machines, resistance bands, or body weight (Serafim et al., 2023). Beyond musculoskeletal benefits, RT has been shown to improve insulin sensitivity and overall metabolic health across populations, including adolescents and older adults (Shaibi et al., 2006; Castaneda et al., 2002; Roberts et al., 2013). According to the World Health Organization (WHO), diabetes is a chronic metabolic disease caused by insufficient insulin production or the body's inability to use insulin effectively, leading to high blood glucose and, over time, damage to nerves and blood vessels. Type 2 diabetes, the most common form, accounts for over 95% of cases. Insulin resistance plays a central role in the pathogenesis of type 2 diabetes mellitus and is a major risk factor for cardiovascular disease and hypertension (Garvey et al., 1998). In insulin resistance, tissues respond less effectively to insulin, whether it is produced naturally by the body (endogenous) or administered externally (exogenous), which results in reduced glucose uptake and utilization compared with healthy individuals (Lebovitz, 2001). Insulin resistance occurs when liver, muscle, and fat tissue respond poorly to insulin, reducing glucose uptake and triggering compensatory hyperinsulinemia (Freeman et al., 2023)

Impaired insulin action is considered a fundamental defect contributing to the development of T2DM and may precede the clinical manifestation of the disease by more than a decade (Shulman, 2000).The number of people worldwide with diabetes has increased dramatically, rising from 200 million in 1990 to 830 million in 2022, yet many cases of T2DM can be prevented through maintaining a healthy weight, regular physical activity, and management of risk factors (World Health Organization, 2024)

These effects make RT a promising strategy for the prevention and management of insulin resistance and T2DM. Given the central role of skeletal muscle in insulin-mediated glucose uptake, understanding how resistance training modulates these mechanisms is crucial for developing effective interventions to prevent and manage T2DM.

This narrative review aims to summarize current evidence on the mechanisms and effectiveness of resistance training in improving insulin sensitivity and metabolic health.

Insulin

Insulin is a key peptide hormone that regulates whole-body glucose balance and supports efficient glucose use by the body. It promotes glucose uptake in skeletal muscle and fat tissue, suppresses glucose production in the liver, and also acts on the brain, pancreatic β-cells, heart, and blood vessels; its maximum effect is referred to as "insulin responsiveness," while the concentration needed for a half-maximal effect defines "insulin sensitivity"(Muniyappa et al., 2008).

Individuals with liver insulin resistance show more unfavorable postprandial plasma metabolite responses, especially elevated triglycerides and lipid particles, compared to those with muscle insulin resistance, indicating tissue-specific differences in metabolic responses to insulin (Gijbels et al., 2024).

The hyperinsulinemic-euglycemic clamp is considered the most accurate method to assess insulin sensitivity in humans. Following an overnight fast, insulin is infused at a steady rate to increase plasma insulin above baseline, while glucose is infused at a variable rate to keep blood sugar within the normal range.The amount of glucose required to maintain euglycemia reflects the efficiency of insulin-stimulated glucose uptake in skeletal muscle and adipose tissue, while simultaneously suppressing hepatic glucose production (Muniyappa et al., 2008).

However, due to its complexity and cost, less invasive surrogate indices derived from fasting insulin and glucose, such as HOMA-IR and QUICKI, are commonly used as clinical and epidemiological research. Additional dynamic assessments, including the oral glucose tolerance test (OGTT) and frequently sampled intravenous glucose tolerance test (FSIVGTT), provide further insight into glucose metabolism and beta-cell function across physiological conditions (Freeman et al., 2023)

Results

1. Pathogenesis of insulin resistance

Skeletal muscle is responsible for roughly three-quarters of insulin-stimulated glucose disposal during hyperinsulinemic-euglycemic clamps. Following an oral glucose load, a portion of the glucose is oxidized immediately (30–40%), while another fraction (~15%) is stored in muscle as glycogen. After glycogen-depleting exercise, the amount stored can increase. (Jensen et al., 2011) Additionally, skeletal muscle can use both glucose and fatty acids for energy, primarily oxidizing fat during fasting when insulin levels are low, and switching to glucose after eating as insulin rises - an adaptive process referred to as metabolic flexibility. (Abdul-Ghani & DeFronzo, 2010) It underscores the key role of skeletal muscle in controlling postprandial glucose levels.

1.1 Mitochondria and Lipid Metabolism

One important mechanism of insulin resistance in skeletal muscle is mitochondrial dysfunction. This dysfunction may represent a key component in the pathogenesis of insulin resistance, as impaired oxidative capacity can reduce fatty acid oxidation, promote the accumulation of intramyocellular lipid intermediates, and consequently interfere with insulin signaling pathways (DeFronzo & Tripathy, 2009). In addition, fat and fatty acid metabolites in skeletal muscle, such as fatty acyl-CoA (FACoA) and diacylglycerol (DAG), interfere with insulin signaling by disrupting IRS-1 (insulin resistance substrate 1) phosphorylation, further promoting insulin resistance (Abdul-Ghani & DeFronzo, 2010). Finally, excessive accumulation of DAG, FACoA, and ceramides within skeletal muscle cells is also linked to impaired insulin action, as these lipid intermediates can disrupt insulin signaling through multiple direct and indirect mechanisms (Schmitz-Peiffer, 2002, as cited in Hulver & Dohm, 2004).

1.2 Disruptions in insulin signaling and GLUT4 dysfunction

Insulin signaling in skeletal muscle depends on the IRS-1/PI3K/Akt pathway (Xu et al., 2015) and disruptions in this signaling are a major cause of insulin resistance in skeletal muscle. Excessive serine phosphorylation of IRS-1 reduces the activation of PI3K and Akt and prevents GLUT4 transporters from moving from intracellular vesicles to the cell membrane, which decreases glucose uptake and contributes to hyperinsulinemia (Abdul-Ghani & DeFronzo, 2010). A key defect in insulin-resistant skeletal muscle and

adipose tissue is the reduced movement of GLUT4 transporters to the cell surface in response to insulin, leading to disrupted glucose regulation (van Gerwen et al. 2023). In type 2 diabetic patients Garvey et al. (1998) reported that skeletal muscle contained normal total GLUT4 levels, but insulin failed to effectively mobilize GLUT4 to the plasma membrane, suggesting the defect lies in trafficking and translocating GLUT4 rather than protein quantity.

1.3 AMPK and PGC-1α

Furthermore, in experimental models, chronic reductions in AMPK (5'AMP-activated protein kinase) activity are associated with the development of insulin resistance, whereas activation of AMPK is linked to improved insulin sensitivity (Ruderman et al., 2013).

AMPK is a key energy sensor in skeletal muscle that, when activated, promotes ATP-generating pathways, enhances glucose uptake via GLUT4 translocation, and supports metabolic and mitochondrial adaptations (Dziewulska et al. 2010; Lira et al. 2010; Hulett et al. 2022). Studies report mixed changes in AMPK activity in skeletal muscle of obese and type 2 diabetic individuals, but enhanced AMPK activity is consistently associated with improved insulin sensitivity (Kjøbsted et al. 2018). In skeletal muscle of T2DM patients and individuals with a family history of the disease, PGC-1α mRNA levels are reduced. Lower PGC-1α expression is also associated with higher fasting insulin and increased BMI, suggesting a link between PGC-1α dysregulation, lipid excess, and obesity (Lira et al. 2010).

1.4 Hormonal factors modulating insulin sensitivity

Sex hormone-binding globulin (SHBG) is a glycoprotein synthesized in the liver that binds circulating sex steroids, regulates the amount of free hormones in the bloodstream, and determines their availability to target tissues (Wallace et al., 2013). Reduced SHBG levels in the bloodstream are associated with an increased likelihood of developing T2DM (Ding et al., 2009) supporting its potential use as a biomarker for metabolic disturbances in men and women. This association appears to be mediated primarily through the regulation of fasting blood glucose, rather than by a direct effect on insulin secretion (Peter et al., 2010). Additionally, low SHBG concentrations are linked to insulin resistance and early disturbances in glucose metabolism, even before the onset of T2DM (Wallace et al., 2013).

1.5 Muscle fiber type

Skeletal muscle fibers are classified as slow-twitch (Type I) or fast-twitch (Type II) based on contraction speed and myosin isoforms (Bonato et al., 2024). Type IIa fibers exhibit higher capillary density, increased GLUT4 content, and greater glucose uptake, whereas individuals with T2DM and their first-degree relatives tend to have a higher proportion of Type IIx fibers, which are glycolytic and primarily glucose-dependent. Hyperinsulinemia, physical inactivity, and immobilization can shift muscle composition toward the glycolytic phenotype (Pesta et al., 2017).

1.6 Microcirculation and muscle perfusion

In patients with T2DM, the capillaries within skeletal muscle exhibit impaired permeability, which correlates with insulin resistance and elevated circulating insulin (Mooshage et al., 2023). Obesity, insulin resistance and diabetes mellitus can impair insulin-mediated capillary recruitment in skeletal muscle, reducing nutrient and oxygen delivery and contributing to metabolic dysfunction (as shown in Figure 4, Horton & Barrett, 2021).

1.7 Inflammation

Reactive oxygen species and proinflammatory cytokines can impair AMPK activity, which in turn worsens defects in insulin signaling (Ruderman et al., 2013).

Overall, insulin resistance in skeletal muscle results from the interplay of mitochondrial dysfunction, accumulation of harmful lipid metabolites, impaired insulin signaling and GLUT4 translocation, reduced AMPK and PGC-1α activity, unfavorable muscle fiber composition, impaired perfusion, chronic inflammation and hormonal imbalances, collectively leading to reduced glucose uptake and metabolic dysfunction.

2. Mechanisms of Resistance Training Effects on Skeletal Muscle Insulin Resistance

Skeletal muscle represents the principal tissue responsible for postprandial glucose disposal, accounting for the majority of insulin-stimulated glucose uptake following food intake (Merz & Thurmond, 2020). The efficiency of glucose uptake by skeletal muscle is determined by several factors, including muscle perfusion, GLUT4-dependent glucose transport, and intracellular metabolic processes (Hulett et al., 2022).

2.1 Improved Muscle Blood Flow and Microcirculation

Exercise training acutely increases muscle blood flow and, over time, promotes capillary growth, enhancing microvascular perfusion (Horton & Barrett, 2021). Six weeks of whole-body resistance training in individuals with T2DM improved muscle microvascular blood flow in response to an oral glucose load, along with better glycemic control and favorable changes in body composition (Russell et al., 2017).

2.2 Increased Muscle Mass and Neuromuscular Adaptations

Insulin resistance in skeletal muscle represents an early feature of metabolic dysfunction, including T2DM, as it limits both the magnitude and rate of glucose uptake. Consequently, interventions that improve muscle mass and insulin signaling capacity may contribute to improvements in insulin sensitivity and glycemic control (Petersen et al., 2007). Pesta et al. (2017) demonstrated that chronic resistance training induces increases in muscle mass and strength as a result of muscle hypertrophy and neuromuscular adaptations. These structural changes are commonly associated with improvements in glucose metabolism and glycemic regulation observed in resistance training interventions.

2.3 Regulation of Glucose Uptake via GLUT4

Glucose uptake in skeletal muscle is largely regulated by the GLUT4 transporter. Under insulin-stimulated conditions, GLUT4 translocation to the plasma membrane occurs via activation of the Akt/PKB signaling pathway, facilitating glucose entry into muscle cells. In addition, muscle contraction can stimulate GLUT4 translocation through insulin-independent pathways involving Ca^{2+}/CaMK and Rac1/actin signaling, thereby increasing glucose uptake during exercise (Hulett et al., 2022). Experimental evidence from resistance training studies supports the involvement of these mechanisms. In a six-week unilateral resistance training study, participants exercised one leg three times per week, while the other leg remained untrained. The trained leg showed higher levels of GLUT4, Akt/PKB, and glycogen synthase (Holten et al., 2004). Improvements in leg glucose clearance exceeded those expected from increases in muscle mass alone, suggesting the presence of metabolic adaptations in trained muscle. Importantly, resistance training targets defects in the IRS-1/PI3K/Akt pathway, central to skeletal muscle insulin resistance. In T2DM mice, the protein expression of IRS-1, p-PI3K, and p-Akt in skeletal muscle was significantly lower than in controls, but resistance exercise increased their levels, improving insulin signaling, GLUT4 translocation, insulin sensitivity, and glucose uptake (Zheng et al., 2024).

2.4 Activation of AMPK and PGC-1α

Resistance training has also been shown to influence energy-sensing and metabolic regulatory pathways. In young, previously untrained men, a six-week program of either traditional or functional resistance training resulted in significant increases in circulating levels of AMPK, PGC-1α, and irisin, accompanied by improvements in muscular fitness and body composition (Zuo et al., 2023). These findings indicate an association between resistance training and activation of the AMPK/PGC-1α/irisin signaling pathway. During physical exercise, muscle contractions increase calcium, nitric oxide, reactive oxygen species, and AMP/ADP, which activate kinases such as AMPK. This activation enhances glucose and fatty acid uptake and supports long-term metabolic and contractile adaptations in skeletal muscle (Spaulding & Yan, 2022). Upon activation, AMPK stimulates catabolic pathways that generate ATP and inhibits energy-consuming processes (Dziewulska et al. 2010). Simultaneously, it increases glucose uptake and improves muscle insulin sensitivity, in part by promoting GLUT4 transporter translocation to the plasma membrane (Spaulding & Yan, 2022).

Additionally, AMPK stimulates PGC-1α, a transcriptional coactivator induced in skeletal muscle by metabolic stimuli, including exercise. PGC-1α promotes mitochondrial biogenesis and GLUT4 expression, which enhances glucose uptake and insulin sensitivity (Lira et al., 2010). While excessive PGC-1α expression in inactive muscle may contribute to insulin resistance, regular physical activity enables PGC-1α to improve glucose homeostasis, increase mitochondrial metabolism, enhance glucose uptake, and optimize lipid utilization (Lira et al., 2010)

2.5 Muscle Fiber Type Remodeling

Research indicates that 4-6 weeks of moderate-intensity resistance training may promote a shift from type IIx to type IIa fibers, which exhibit enhanced glucose uptake, higher GLUT4 levels, and a stronger insulin response. Importantly, improvements in metabolic function can also occur without altering fiber type if the

muscle's intracellular metabolic mechanisms are functioning efficiently (Pesta et al., 2017). Active skeletal muscles act as an endocrine organ that releases a variety of signaling molecules known as myokines. These molecules exert autocrine, paracrine, and endocrine effects (Pedersen & Febbraio, 2012), modulating glucose metabolism and inflammatory responses (Pedersen et al., 2007).

2.6 Myokines

A systematic review with meta-analysis found that a single session of exercise, whether aerobic or resistance, led to elevated circulating levels of several myokines, such as IL-15, irisin, SPARC, oncostatin M (OSM), and decorin, in healthy adults. The extent of the increase differed depending on the specific myokine, and levels typically returned to baseline within 3 to 24 hours after exercise (Bettariga et al., 2024). Among the myokines that may be induced by resistance exercise are also interleukin-6 (IL-6) and brain-derived neurotrophic factor (BDNF) (Zunner et al., 2022)

- **IL-6**

IL-6 has both pro- and anti-inflammatory roles. In obesity, T2DM, and insulin resistance, the low-grade systemic inflammation is associated with increased levels of several cytokines, including IL-6 (likely reflecting dysregulation rather than causing it), whereas exercise-induced IL-6 exerts the anti-inflammatory effects by promoting the secretion of anti-inflammatory cytokines IL-1ra and IL-10 and suppressing proinflammatory TNF-α, which may contribute to protection against TNF-α-induced insulin resistance (Petersen & Pedersen, 2005). Experimental studies show that acute IL-6 exposure enhances insulin-stimulated glucose disposal in muscle, with associated AMPK activation, increased fatty acid oxidation, and GLUT4 translocation to the plasma membrane (Carey et al., 2006).

- **IRISIN**

Irisin is the PGC-1α-dependent myokine (Pedersen & Febbraio, 2012)

released by skeletal muscles in response to physical exercise, including resistance training, contributes to metabolic health by promoting the browning of white adipose tissue, enhancing glucose and lipid metabolism, and decreasing insulin resistance and adipose tissue inflammation (Zunner et al., 2022).

- **BDNF**

Resistance training can increase circulating levels of BDNF (brain-derived neurotrophic factor), particularly after several weeks of consistent exercise and regardless of the type of resistance training. In skeletal muscles, BDNF supports glucose metabolism through activation of the AMPK pathway (Zunner et al., 2022). Moosaie et al. (2023) reported that activation of the BDNF/TrkB/CREB signaling cascade improves hepatic insulin sensitivity and reduces glucose production in the liver, while also lowers blood glucose levels. Moreover, it decreases leptin and food intake, promotes the formation of glycolytic fibers in skeletal muscle, and protects pancreatic β cells in diabetes. In animal studies, chronic endurance exercise increased BDNF levels and improved insulin sensitivity via TrkB receptors (Jiménez-Maldonado et al., 2018) Although these findings come from animal studies using endurance training, they provide mechanistic insight into how BDNF might influence glucose metabolism, which could also be relevant for resistance training. In humans, lower circulating BDNF levels are associated with increased insulin resistance in patients with T2DM (Krabbe et al., 2007) emphasizing the potential metabolic importance of BDNF.

In summary, resistance training induces the release of multiple myokines from skeletal muscle, including IL-6, irisin, BDNF, and other factors such as IL-15, SPARC, OSM, and decorin. These myokines act locally and systemically to modulate glucose and lipid metabolism, reduce inflammation, and enhance insulin sensitivity. Collectively, these responses highlight the endocrine function of skeletal muscle and the mechanistic basis by which resistance exercise can improve metabolic health. All these effects complement the activation of AMPK, PGC-1α, and myokine pathways, illustrating that resistance exercise improves insulin sensitivity through both restoration of impaired insulin signaling and enhancement of metabolic and endocrine adaptations in skeletal muscle.

Evidence from Intervention Studies

Several intervention studies have investigated the effects of resistance training on insulin sensitivity across different populations, ranging from adolescents to overweight adults and individuals with T2DM. Shaibi et al. (2006) conducted a 16-week resistance training program in 22 overweight Latino adolescent males, comparing a twice-weekly training group with a non-exercising control group. Participants in the resistance training group significantly improved insulin sensitivity (by an average of 45%) independent of changes in fat or lean mass, whereas no improvement was observed in controls. Most individuals in the training group showed positive changes, suggesting that resistance training alone, regardless of body composition changes, can enhance metabolic function in this at-risk population. Shih & Kwok (2018) reported that overweight and obese

Taiwanese male adolescents participating in a 12-week exercise program showed significant reductions in body fat and waist circumference, along with improvements in insulin sensitivity (HOMA-IR) and pancreatic β-cell function. Lower glucose and insulin responses during OGTT were also observed after the intervention, and these metabolic improvements were present even in participants with impaired glucose tolerance. Croymans et al. (2013) reported that a 12-week resistance training program in overweight young men improved muscle insulin sensitivity (mISI) and β-cell function (disposition index) without affecting hepatic insulin resistance. Training also increased lean body mass, strength, and key glucose-transport proteins (HK2, GLUT4, AKT2), with substantial individual variability in response. In another study Van der Heijden et al. (2010) reported that a 12-week supervised resistance training program in obese adolescents increased strength and lean body mass without changing total body weight. The intervention improved hepatic insulin sensitivity by approximately 24% and reduced glucose production mainly through glycogenolysis, while peripheral insulin sensitivity, as well as total, visceral, hepatic, and intramyocellular fat, remained unchanged. A meta-analysis of 12 randomized controlled trials examining the effects of resistance training in adults aged ≥60 years reported significant improvements in insulin sensitivity, reflected by reductions in HOMA-IR compared with control groups. Subgroup analyses indicated that the greatest improvements were observed in higher-intensity programs lasting longer than 12 weeks. Additionally, some studies included in the analysis reported reductions in HbA1c, particularly in programs of moderate intensity and shorter duration (Jiahao et al., 2021). Castaneda et al. (2002) found that resistance training effectively supports metabolic health as an adjunct to standard care in older adults with T2DM. In a 16-week high-intensity progressive resistance training program involving older adults with T2DM, participants showed improved glycemic control, increased muscle glycogen and lean mass, decreased trunk fat and systolic blood pressure, and reduced diabetes medication use, whereas control participants exhibited no such improvements. Although the magnitude and site of improvements in insulin sensitivity vary between studies - sometimes affecting hepatic sensitivity, sometimes peripheral (muscle) sensitivity - overall, these intervention studies consistently demonstrate that resistance training enhances metabolic function across different ages and populations.

Recommendations

According to the American College of Sports Medicine (2009), the frequency of resistance training should be adjusted to experience, typically 2–5 sessions per week, using weights that challenge the muscles within a given repetition range. Programs should include both single- and multi-joint exercises, employ various types of muscle contractions, and set rest periods according to the goal, with longer rests for strength and power, and shorter rests for muscle growth or endurance. Liu et al. reported that high-intensity resistance training appears to produce greater reductions in HbA1c and insulin levels in individuals with T2DM compared with low-to-moderate intensity exercise.These findings suggest that exercise intensity is an important factor in maximizing the metabolic benefits of resistance training.

Discussion

Overall, studies indicate that resistance training consistently improves insulin sensitivity and other key metabolic functions across diverse populations, including adolescents and older adults, even in the absence of significant changes in body weight. While it is well established that resistance training enhances whole-body glucose tolerance and insulin sensitivity (Roberts et al., 2013; Shaibi et al., 2006), its effects on tissue-specific insulin sensitivity, such as in skeletal muscle and the liver, remain less well understood. The magnitude and location of these effects can vary among individuals and may depend on factors such as age, ethnicity, specific training program parameters, and the metabolic status of participants. Therefore, further research is needed to clarify the tissue-specific impacts of resistance training and to better understand the determinants of individual responses. The observed improvements in whole-body and tissue-specific insulin sensitivity may be mediated by increased skeletal muscle mass, enhanced microvascular perfusion, and upregulation of glucose transport proteins, such as GLUT4, in skeletal muscle. Overall, these findings suggest that resistance training can serve as a multi-level supportive intervention for improving insulin sensitivity and overall metabolic health, highlighting its potential as an adjunct to lifestyle or medical strategies aimed at preventing or managing insulin resistance and type 2 diabetes. Collectively, they support the inclusion of resistance training as a key component of lifestyle interventions, complementing dietary and pharmacological approaches.

Conclusions

Resistance training (RT) consistently improves insulin sensitivity and metabolic health across diverse populations, primarily through enhanced skeletal muscle perfusion, increased muscle mass, promotion of GLUT4 translocation, and activation of key signaling pathways such as IRS-1/PI3K/Akt and AMPK/PGC-1α. Additionally, RT influences muscle fiber composition and stimulates the release of myokines, including IL-6, irisin, and BDNF, which help regulate glucose and lipid metabolism and reduce systemic inflammation. Although individual responses may vary based on age, metabolic status, and training protocols, evidence supports RT as a safe, effective, and multi-level strategy for improving insulin resistance. Future research should clarify tissue-specific effects, optimal training parameters, and long-term impacts on metabolic health to strengthen the evidence base for personalized exercise interventions. Incorporating RT into lifestyle interventions, alongside dietary modifications and pharmacological treatments, represents a powerful, complementary approach to reducing insulin resistance and supporting the prevention and management of type 2 diabetes.

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