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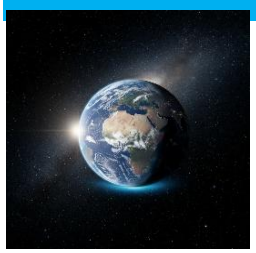

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The Role of Physical Activity in Improving Insulin Sensitivity: A Narrative Review

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Abstract

Background: Insulin resistance is a key metabolic disorder underlying the development of type

2 diabetes and other cardiometabolic diseases. Lifestyle factors, particularly physical activity, play an important role in improving insulin sensitivity and overall metabolic health. Numerous studies indicate that regular exercise influences glucose metabolism through multiple physiological and molecular mechanisms.

Aim: The aim of this review was to summarize current evidence on the relationship between physical activity and insulin sensitivity, with particular emphasis on the biological mechanisms involved and the effects of different types of exercise.

Materials and Methods: A PubMed search of English-language studies published in the last 10 years was conducted to evaluate the effects of physical activity on insulin sensitivity and metabolic health.

Results: The reviewed studies indicate that both acute and long-term physical activity improve insulin sensitivity through multiple mechanisms, including increased glucose uptake in skeletal muscle, enhanced mitochondrial function, and reduction of visceral adipose tissue. Aerobic exercise, resistance training, and high-intensity interval training are all associated with beneficial metabolic effects. In addition, reducing sedentary behavior and increasing daily activity may further improve glucose metabolism.

Conclusions: Regular physical activity represents an effective non-pharmacological strategy for improving insulin sensitivity and preventing metabolic diseases. The greatest benefits appear to result from long-term lifestyle interventions combining exercise, healthy diet, and weight management.

Keywords: physical activity, exercise, insulin resistance, insulin sensitivity, skeletal muscle, type 2 diabetes, metabolic health.

1. Introduction

Insulin resistance (IR) is a key pathophysiological mechanism underlying many metabolic disorders, particularly type 2 diabetes. It is defined as a reduced responsiveness of target tissues-primarily skeletal muscle, liver, and adipose tissue-to physiological concentrations of insulin. As a result, increasing amounts of insulin must be secreted to maintain glucose homeostasis, initially leading to compensatory hyperinsulinemia; over time, together with progressive pancreatic β-cell dysfunction, this may result in hyperglycemia [31]. At the cellular level, insulin action largely depends on activation of the insulin receptor signaling pathway involving

IRS proteins, PI3K, and Akt, which promotes translocation of the GLUT4 transporter to the cell membrane and enables glucose uptake by cells. Disruptions in this pathway reduce insulin-stimulated glucose transport and contribute to the development of metabolic disorders[1,31].

Skeletal muscle plays a central role in glucose homeostasis, accounting for the majority of insulin-stimulated glucose uptake in the body [7,31]. In insulin resistance, metabolic flexibility becomes impaired, resulting in decreased glucose utilization, disturbances in fatty-acid oxidation, and deterioration of metabolic homeostasis [27]. In addition, ectopic lipid accumulation in skeletal muscle and the liver promotes the formation of metabolites such as diacylglycerols and ceramides that interfere with insulin signaling. These processes are frequently accompanied by chronic low-grade inflammation, oxidative stress, mitochondrial dysfunction, and endothelial dysfunction [1,32].

The clinical significance of insulin resistance extends beyond glucose metabolism and is strongly associated with metabolic syndrome, hypertension, dyslipidemia, non-alcoholic fatty liver disease, and increased cardiovascular risk [1,36]. Improving insulin sensitivity is therefore a major target in the prevention and management of metabolic disorders.

Among lifestyle factors, physical activity is one of the best-documented non-pharmacological strategies for improving insulin sensitivity. Regular exercise increases glucose uptake in skeletal muscle, improves mitochondrial function, enhances GLUT4 expression, and positively influences lipid and glucose metabolism [1,7]. These benefits have been observed in both aerobic and resistance training, although the mechanisms of adaptation may differ depending on the type of exercise performed [1,14,40]. Physical activity may also improve insulin sensitivity through mechanisms such as improved endothelial function, reduced inflammation, enhanced oxidative capacity of skeletal muscles, and decreased accumulation of harmful lipid metabolites [7].

Evidence suggests that not only structured exercise but also overall daily physical activity plays an important role in metabolic regulation. Prolonged sedentary behavior is associated with impaired insulin sensitivity, whereas even light activity such as walking or regularly interrupting sitting time may improve postprandial glucose and insulin metabolism [3,20,21,23,38].

However, the metabolic response to physical activity may vary depending on factors such as age, obesity, cardiorespiratory fitness, sex, hormonal status, and the presence of metabolic diseases [2,5,24,29]. Although a single bout of exercise can transiently increase insulin sensitivity, regular training leads to long-term metabolic adaptations that improve muscle metabolism and glucose regulation [4,6,25].

Overall, physical activity represents one of the most important components in the prevention and management of insulin resistance. However, further research is needed to determine which types and intensities of physical activity are most effective in improving insulin sensitivity and metabolic health. Therefore, the aim of this review is to summarize current evidence on the relationship between physical activity and insulin sensitivity, focusing on biological mechanisms, different types of exercise, and the role of reducing sedentary behavior in the prevention and management of metabolic disorders**.**

2. Pathophysiology of Insulin Resistance

2.1 Mechanisms of Insulin Resistance

Insulin resistance is a complex metabolic disorder resulting from an impaired response of tissues to the action of insulin. Under physiological conditions, insulin regulates glucose metabolism by increasing glucose uptake in skeletal muscle and adipose tissue and by suppressing hepatic glucose production. This process occurs primarily through the activation of the insulin receptor signaling pathway involving IRS, PI3K, and Akt, which leads to the translocation of the GLUT4 transporter to the cell membrane and enables glucose transport into the cell [31]. In insulin resistance, disturbances occur within this signaling pathway, resulting in reduced glucose uptake and impaired glycemic control.

One of the major mechanisms contributing to the development of insulin resistance is the ectopic accumulation of lipids in metabolic tissues such as skeletal muscle and the liver. Excess lipids lead to the formation of metabolites such as diacylglycerols and ceramides, which activate protein kinases that interfere with insulin signaling by inhibiting the activity of IRS and Akt [31]. This process may be further exacerbated by chronic low-grade inflammation, oxidative stress, endoplasmic reticulum stress, and mitochondrial dysfunction [32]. The interaction of

these factors contributes to the progressive impairment of glucose metabolism and an increased risk of developing type 2 diabetes.

Insulin resistance is also associated with disrupted metabolic communication between various organs, including adipose tissue, skeletal muscle, and the liver. Numerous signaling molecules are involved in this process, including free fatty acids, adipokines, myokines, and pro-inflammatory cytokines, which influence the regulation of glucose and lipid metabolism throughout the body [34].

Due to the complexity of the mechanisms underlying insulin resistance, the accurate assessment of pancreatic β-cell function and tissue insulin sensitivity is also of great importance. Both dynamic and static methods are used in scientific research. Among the most precise techniques are the hyperglycemic clamp used to evaluate β-cell function and the hyperinsulinemic-euglycemic clamp, which is considered the gold standard for assessing insulin sensitivity. However, these methods are expensive, time-consuming, and difficult to apply in routine clinical practice. Therefore, in epidemiological studies and clinical settings, simpler indices calculated from fasting glucose and insulin concentrations or from oral glucose tolerance test results are more commonly used. These include HOMA-IR, HOMA-β, QUICKI, and the insulinogenic index [33]. Nevertheless, each of these methods has certain limitations because glucose and insulin metabolism is a complex process involving interactions between multiple organs and regulatory mechanisms. Consequently, the choice of an appropriate assessment method should depend on the aim of the study, the characteristics of the studied population, and the organizational capabilities of a given research center [33].

2.2 Role of Skeletal Muscle in Glucose Uptake

Skeletal muscle plays a crucial role in maintaining glucose homeostasis, as it accounts for the majority of insulin-stimulated glucose uptake in the body [31]. Under physiological conditions, insulin increases glucose transport into muscle cells through activation of the IRS-PI3K-Akt signaling pathway and the translocation of the GLUT4 transporter to the cell membrane [7]. As a result, muscles can store glucose in the form of glycogen or utilize it as a source of energy.

In insulin resistance, the ability of skeletal muscle to uptake glucose is significantly reduced. This may result from impaired insulin signaling, decreased GLUT4 translocation, and the

accumulation of lipids and lipid metabolites within muscle cells [7,31]. Additionally, disturbances in skeletal muscle microcirculation may limit the delivery of glucose and insulin to muscle fibers, further impairing glucose metabolism [7].

Mitochondrial function also plays an important role in the regulation of muscle metabolism. A decline in mitochondrial oxidative capacity may lead to impaired fatty acid oxidation and increased lipid accumulation in skeletal muscle, which promotes the development of insulin resistance [5]. Therefore, proper functioning of skeletal muscle represents one of the key elements in maintaining normal insulin sensitivity.

2.3 Adipose Tissue Dysfunction

Adipose tissue plays an important role in the regulation of energy metabolism; however, under conditions of obesity it may undergo significant dysfunction. Enlarged adipocytes begin to secrete increased amounts of free fatty acids and pro-inflammatory cytokines, leading to chronic low-grade inflammation [32]. Excess free fatty acids may be transported to other tissues, such as skeletal muscle and the liver, where they promote the formation of lipotoxic metabolites that interfere with insulin signaling.

Adipose tissue also functions as an active endocrine organ that secretes adipokines involved in metabolic regulation. Disturbances in the secretion of these molecules may affect glucose metabolism, inflammatory processes, and cardiovascular function [34]. As a result, adipose tissue dysfunction plays a significant role in the development of insulin resistance and other metabolic disorders.

2.4 Visceral Fat and Metabolic Risk

Particular importance in the development of metabolic disorders is attributed to visceral fat, which refers to adipose tissue located within the abdominal cavity. Excess accumulation of visceral fat is strongly associated with insulin resistance, lipid abnormalities, and an increased risk of cardiovascular diseases [36]. Compared with subcutaneous adipose tissue, visceral fat

exhibits greater metabolic activity and secretes larger amounts of free fatty acids and inflammatory mediators.

An increase in visceral fat leads to an elevated influx of free fatty acids into the liver, which may enhance hepatic glucose production and disrupt lipid metabolism [31]. Moreover, excess abdominal fat contributes to the development of metabolic syndrome, characterized by the coexistence of abdominal obesity, hypertension, dyslipidemia, and insulin resistance [36]. For this reason, the reduction of visceral fat represents one of the most important targets of lifestyle interventions aimed at preventing metabolic diseases.

3. Effects of Physical Activity on Insulin Sensitivity

Physical activity is one of the most important lifestyle factors influencing the improvement of tissue insulin sensitivity and the regulation of glucose metabolism. Numerous studies indicate that both a single bout of physical exercise and long-term training can improve insulin sensitivity through various metabolic, molecular, and physiological mechanisms [4,6]. The beneficial effects of physical activity include increased glucose uptake in skeletal muscle, improved mitochondrial function, reduction of inflammation, and a decrease in adipose tissue mass, particularly visceral fat [1,7].

Research findings also suggest that higher levels of daily physical activity may be associated with improved insulin sensitivity and a more favorable metabolic profile, including in individuals with type 1 diabetes. In a study involving adult patients with T1D, a greater number of daily steps and higher levels of moderate-to-vigorous physical activity were associated with higher values of the estimated glucose disposal rate (eGDR), lower insulin requirements, and improved glycemic control [9].

One of the most important mechanisms underlying the improvement of insulin sensitivity during physical activity is the increased transport of glucose into muscle cells. During muscle contraction, insulin-independent mechanisms are activated, leading to the translocation of the GLUT4 transporter to the cell membrane and increased glucose uptake by skeletal muscle [7]. This mechanism is partly associated with the activation of AMP-activated protein kinase (AMPK), which acts as a cellular energy sensor and plays a key role in the regulation of glucose

and fatty acid metabolism. As a result, physical exercise can increase glucose utilization by muscles even in conditions of reduced insulin sensitivity.

Studies also indicate that a single session of physical exercise can lead to a transient improvement in insulin sensitivity. After exercise, the ability of skeletal muscle to uptake glucose increases, and this effect may persist for several hours up to even a few days following physical activity [4]. This phenomenon is partly related to the depletion of muscle glycogen stores, which increases cellular demand for glucose during the recovery period. In addition, exercise activates numerous cellular signaling pathways responsible for metabolic adaptation to increased energy demand [6]. Multi-omic analyses have shown that even a single bout of exercise can trigger thousands of molecular changes involving energy metabolism, inflammatory responses, oxidative stress, and tissue repair processes [22].

Regular training leads to long-term metabolic adaptations that may result in sustained improvements in insulin sensitivity. These adaptations include an increased number of mitochondria in skeletal muscle, improved oxidative capacity, and enhanced expression of proteins involved in energy metabolism [5]. As a result, muscles become more efficient in utilizing both glucose and fatty acids as energy sources. Physical training also increases capillary density in skeletal muscle, which improves the delivery of oxygen, glucose, and insulin to muscle fibers [7].

Physical activity also positively influences lipid metabolism and the reduction of adipose tissue. Regular exercise can reduce the amount of visceral fat and limit lipid accumulation in skeletal muscle and the liver, which are among the key factors contributing to the development of insulin resistance [15,16]. Additionally, exercise may reduce the concentration of lipids associated with metabolic disturbances, such as triacylglycerols and sphingolipids, thereby improving the function of insulin signaling pathways [17].

An important aspect of the metabolic effects of physical activity is the release of myokines, signaling molecules produced by contracting muscles. Myokines can regulate metabolic processes in various organs, influence glucose and lipid metabolism, and reduce chronic inflammation associated with obesity [13]. Consequently, physical activity exerts not only local effects in skeletal muscle but also systemic effects that contribute to improved overall metabolic function.

It is also important to emphasize that the metabolic benefits of physical activity are not limited to high-intensity exercise. Studies indicate that even moderate or light physical activity may improve insulin sensitivity compared with a sedentary lifestyle [8]. In contrast, prolonged sitting is associated with impaired insulin sensitivity, particularly in skeletal muscle, which represents the primary site of glucose utilization in the body [3]. It has also been demonstrated that regularly interrupting sedentary time with short periods of movement, such as walking or standing, can improve postprandial glucose and insulin metabolism [21,23].

At the same time, the metabolic response to physical activity may vary depending on multiple factors, including age, degree of obesity, body composition, and level of physical fitness [5,24]. In some cases, physical activity alone, without concurrent weight loss, may not lead to sustained improvements in all metabolic indicators, particularly in individuals with high levels of adipose tissue [29]. Therefore, many studies emphasize the importance of comprehensive lifestyle interventions that combine physical activity with dietary modifications and the reduction of excess body weight [16].

The effectiveness of such adaptations has also been confirmed in interventional studies. In a randomized study involving individuals with type 2 diabetes, a 12-week program of regular physical exercise led to significant improvements in glycemic control and reductions in insulin resistance, assessed among others using the HOMA-IR index [12].

4. Types of Exercise and Their Effects on Insulin Sensitivity

Different forms of physical activity may influence insulin sensitivity through distinct physiological mechanisms. However, research indicates that most types of exercise training contribute to improved glucose metabolism and reduced insulin resistance, particularly when performed regularly [14].

Aerobic exercise, such as running, cycling, or brisk walking, increases glucose utilization by skeletal muscles and improves cardiorespiratory fitness. Regular aerobic activity may enhance the expression of the GLUT4 transporter, improve mitochondrial function, and reduce adipose tissue mass, thereby contributing to improved insulin sensitivity [14,28].

Resistance training influences glucose metabolism primarily by increasing muscle mass and enhancing the capacity of muscles to store glycogen. Greater muscle mass corresponds to a higher capacity of the body to uptake glucose, which may lead to improved insulin sensitivity [14,40]. Cellular-level studies also indicate that both long-term endurance and resistance training can induce similar metabolic adaptations in muscle cells. In muscle stem cells derived from physically active individuals, greater insulin-stimulated glycogen synthesis has been observed compared with individuals leading a sedentary lifestyle, suggesting a beneficial effect of regular physical activity on glucose metabolism in skeletal muscle [39].

High-intensity interval training (HIIT) involves performing short periods of very intense exercise interspersed with phases of rest or low-intensity activity. Studies suggest that HIIT may improve insulin sensitivity to a degree comparable to traditional aerobic training while requiring shorter training sessions [4]. Interventional studies have also demonstrated that a 16-week HIIT program in individuals with metabolic syndrome resulted in improvements in body composition, increased aerobic capacity, and reductions in fasting insulin levels and the HOMA-IR index. These effects were observed regardless of the time of day at which the training was performed, although some metabolic parameters improved slightly more after morning training sessions [41].

Combined training programs that include both aerobic and resistance exercise are also increasingly emphasized as effective strategies for improving metabolic health. Such programs may produce particularly beneficial metabolic effects because they simultaneously improve cardiorespiratory fitness, increase muscle mass, and promote reductions in adipose tissue [14].

Overall, different forms of physical activity can effectively improve insulin sensitivity; however, the greatest metabolic benefits appear to be associated with regular exercise and the combination of various types of physical training. Meta-analyses indicate that both aerobic and resistance training can improve parameters of glucose metabolism in individuals with type 2 diabetes, and their combination may lead to additional metabolic benefits [11].

5. Factors Modifying the Effects of Physical Activity on Insulin Sensitivity

The effects of physical activity on insulin sensitivity may be modified by various biological and environmental factors, including body composition, age, lifestyle, and diet. Understanding these factors is essential for the proper design of interventions aimed at improving glucose metabolism and preventing metabolic diseases.

5.1 Obesity and Body Composition

One of the most important factors contributing to the development of insulin resistance is excess body weight, particularly increased visceral adipose tissue. Excess adiposity, especially in the abdominal region, is associated with elevated levels of free fatty acids and increased inflammatory activity, which can impair insulin signaling in skeletal muscle and the liver [20,32]. Studies indicate that weight reduction, particularly through a combination of physical activity and dietary modifications, can significantly improve insulin sensitivity and reduce the risk of developing type 2 diabetes [18]. Additionally, higher aerobic fitness is associated with lower levels of visceral adipose tissue and improved regulation of lipid metabolism [23].

5.2 Age and Mitochondrial Function

The aging process is associated with a gradual decline in insulin sensitivity and metabolic changes occurring across multiple tissues. With increasing age, reductions in skeletal muscle mass (sarcopenia), impairments in insulin signaling, and decreased mitochondrial oxidative capacity are commonly observed [5]. Furthermore, aging is associated with deteriorating skeletal muscle function, reduced muscle strength, and an increased risk of frailty, which is linked to metabolic disturbances, including insulin resistance [13,37].

Since skeletal muscle represents the primary site of insulin-stimulated glucose uptake in the body, structural and metabolic changes occurring within this tissue may significantly contribute to the development of insulin resistance [5]. Aging is also associated with mitochondrial dysfunction, which may lead to reduced substrate oxidation and increased lipid accumulation within skeletal muscle, further impairing glucose metabolism.

However, regular physical activity can substantially counteract many of these processes. Both endurance and resistance training may improve mitochondrial function, increase the expression of glucose transporters, and enhance the ability of skeletal muscles to utilize glucose as an

energy source [5,13,40]. As a result, physical activity may mitigate some of the adverse effects of aging, improve skeletal muscle function, and support the maintenance of normal insulin sensitivity in older adults [37].

5.3 Sedentary Behaviour and Daily Activity

Prolonged sedentary behavior, such as extended periods of sitting, represents an independent risk factor for the development of metabolic disorders. Studies have shown that greater amounts of time spent in a seated position are associated with higher levels of adipose tissue insulin resistance and increased visceral fat accumulation [32]. This relationship is particularly pronounced in older individuals, those with higher body mass index, and individuals with lower cardiorespiratory fitness [32]. Importantly, even short breaks in sedentary time involving light physical activity may lead to improved glycemic control and reduced postprandial glucose and insulin concentrations [21].

5.4 Diet and Lifestyle Interventions

Physical activity often produces the greatest metabolic benefits when combined with appropriate lifestyle modifications, particularly those related to dietary habits. Diet plays a key role in regulating glucose metabolism and in the prevention of insulin resistance [18]. Research indicates that even a moderate weight reduction of approximately 7–10%, combined with at least 150 minutes of moderate physical activity per week, can significantly reduce the risk of developing type 2 diabetes and improve glycemic control [18].

Additional factors such as meal timing, carbohydrate quality, glycemic index of foods, and adequate dietary fiber intake may also influence glycemic responses and insulin secretion [18]. Increasing evidence also highlights the importance of so-called chrononutrition, which refers to the timing and composition of meals. Consuming a greater proportion of daily energy intake earlier in the day, maintaining regular meal timing, and following an appropriate order of food consumption may positively affect postprandial glycemia and insulin sensitivity [35].

The importance of combining physical activity with dietary interventions has also been confirmed in experimental studies. In animal models, switching to a low-fat diet was shown to be the primary factor driving reductions in body weight and adipose tissue mass, while physical

activity further improved insulin sensitivity and enhanced the beneficial metabolic effects of dietary changes [19]. These findings suggest that the greatest metabolic benefits may be achieved through the simultaneous implementation of dietary modifications and regular physical activity [19].

The effectiveness of combining physical activity with dietary interventions has also been demonstrated in interventional studies conducted in various populations. In a study involving postmenopausal women, a 12-week aerobic training program performed three times per week in combination with a calorie-restricted diet resulted in greater weight loss, improved insulin sensitivity, and beneficial changes in hormonal parameters compared with diet alone [26]. These findings suggest that physical activity may enhance the effects of dietary interventions by increasing glucose uptake in skeletal muscle and reducing adipose tissue, particularly in the abdominal region [26].

6. Clinical and Practical Implications of Physical Activity for Insulin Resistance

Physical activity represents one of the most important components in the prevention and management of insulin resistance and metabolic diseases. Numerous studies indicate that regular physical exercise can significantly improve insulin sensitivity by increasing glucose uptake in skeletal muscles, enhancing mitochondrial function, and reducing excess adipose tissue [14,18]. For this reason, physical activity-based interventions are widely recommended as a primary strategy for the prevention of type 2 diabetes.

Randomized studies suggest that even relatively short training programs may produce meaningful metabolic benefits. In one study, a 12-week program of moderate-intensity exercise resulted in improved glycemic control, increased insulin sensitivity, and improved functional capacity in individuals with type 2 diabetes [12].

6.1 Physical Activity Guidelines for Improving Insulin Sensitivity

Lifestyle guidelines recommend performing at least 150 minutes of moderate-intensity physical activity per week, such as brisk walking, cycling, or swimming. Regular aerobic exercise increases glucose utilization in skeletal muscle and improves cardiorespiratory fitness, thereby supporting improved glucose metabolism [14].

Increasingly, the importance of combining aerobic training with resistance training is also emphasized. Resistance exercise increases muscle mass and enhances the capacity of skeletal muscle to store glycogen, which further supports glucose metabolism and improves insulin sensitivity [5].

6.2 Role of Exercise in the Prevention of Type 2 Diabetes

Regular physical activity plays a crucial role in the prevention of type 2 diabetes. Studies indicate that intervention programs involving increased physical activity, dietary modification, and moderate weight reduction can significantly reduce the risk of developing this disease [18].

Even modest weight loss of approximately 7-10%, when combined with regular physical activity, may lead to significant improvements in glycemic control and increased tissue insulin sensitivity [18].

6.3 Breaking Sedentary Behaviour

In addition to planned exercise, increasing attention is being paid to reducing sedentary behavior. Prolonged sitting is associated with impaired glucose metabolism and an increased risk of insulin resistance [32].

Introducing short breaks in sedentary time, such as several minutes of light physical activity throughout the day, may reduce postprandial glucose and insulin concentrations and improve overall metabolic control [21].

6.4 Combined Lifestyle Interventions

The greatest therapeutic benefits are typically observed with comprehensive lifestyle interventions that include both physical activity and dietary modifications. A well-balanced diet,

reduction of excess caloric intake, and regular physical activity may together contribute to improved insulin sensitivity and a reduction in cardiovascular risk factors [18].

Such an approach is particularly important for individuals with overweight, obesity, or metabolic syndrome.

Physical activity therefore represents an effective strategy in the prevention and treatment of insulin resistance. Regular exercise, reduction of sedentary time, and comprehensive lifestyle modifications can significantly improve metabolic health and reduce the risk of developing type 2 diabetes.

7. Discussion

Insulin resistance is a central metabolic disturbance underlying type 2 diabetes and other cardiometabolic diseases. The evidence reviewed in this study indicates that physical activity improves insulin sensitivity through mechanisms involving skeletal muscle, adipose tissue, and systemic metabolic regulation, supporting its role as a fundamental component of metabolic disease prevention and management.

One of the main mechanisms responsible for improved insulin sensitivity is the effect of exercise on skeletal muscle metabolism. Skeletal muscle is the primary site of insulin-stimulated glucose uptake, and physical activity enhances glucose transport through both insulin-dependent and insulin-independent pathways [5,14]. Acute exercise increases glucose uptake mainly through greater GLUT4 translocation, whereas long-term training induces adaptations such as increased mitochondrial density, improved oxidative capacity, and enhanced insulin signaling [5,7].

Adipose tissue also plays an important role in insulin resistance. Excess adiposity, particularly visceral fat, is associated with chronic low-grade inflammation and elevated free fatty acid release, which impair insulin signaling in the liver and skeletal muscle [32,36]. Physical activity may therefore improve insulin sensitivity not only by increasing glucose uptake in muscle but also by reducing visceral fat and improving adipose tissue function. Higher cardiorespiratory fitness has likewise been associated with better regulation of lipolysis and lower adipose tissue insulin resistance [38,42].

The available evidence suggests that metabolic health depends not only on structured exercise but also on the overall pattern of daily movement. Prolonged sedentary behavior is associated with higher insulin resistance and poorer glycemic control, whereas even short interruptions of sitting time with light activity may improve postprandial glucose and insulin responses [3,20,21,23,38].

Different forms of exercise may improve insulin sensitivity through partly distinct mechanisms. Aerobic training improves cardiorespiratory fitness and mitochondrial function, resistance training increases muscle mass and glycogen storage capacity, and high-intensity interval training provides significant metabolic benefits despite shorter duration [4,14,28,40]. These findings suggest that combining different forms of physical activity may provide the greatest metabolic benefit.

At the same time, the metabolic response to exercise varies between individuals and may depend on age, obesity, body composition, physical fitness, and the presence of metabolic diseases [2,5,24,29,40]. In some groups, particularly older individuals and those with obesity, physical activity alone may not lead to equally pronounced improvements in insulin sensitivity without accompanying weight reduction [16,24,29]. The greatest benefits are therefore typically observed with comprehensive lifestyle interventions combining physical activity, dietary modification, and weight loss [15,16,18,24,30].

Some studies also suggest population-specific differences in response to physical activity. For example, in individuals with type 1 diabetes, total physical activity measured in MET-hours per week was not significantly associated with insulin resistance or non-alcoholic fatty liver disease, whereas participation in sports and higher exercise intensity were linked to more favorable metabolic profiles and higher eGDR values [10]. Interventional studies further confirm the benefits of structured exercise programs. A systematic review and meta-analysis including 11 studies with 846 individuals with type 2 diabetes showed that regular exercise training improved several parameters of glucose metabolism, including fasting glucose levels [11]. In postmenopausal women, combining aerobic exercise with caloric restriction also improved insulin sensitivity and hormonal profiles [26].

However, several limitations should be acknowledged. The available studies differ in methodology, intervention duration, type of exercise, and methods used to assess insulin

sensitivity, which limits direct comparison of results. In addition, many studies rely on indirect markers of insulin resistance rather than gold-standard methods such as the hyperinsulinemic-euglycemic clamp.

Overall, current evidence indicates that physical activity is one of the most effective non-pharmacological strategies for improving insulin sensitivity. Long-term, regular, and individualized exercise interventions-especially when combined with other lifestyle modifications-appear to provide the greatest metabolic benefits.

8. Conclusion

Insulin resistance is a major metabolic disturbance contributing to the development of type 2 diabetes and other cardiometabolic diseases. Current evidence indicates that physical activity plays a key role in improving insulin sensitivity through multiple physiological and metabolic mechanisms, including increased glucose uptake in skeletal muscle, improved mitochondrial function, reduction of visceral fat, and favorable effects on lipid and glucose metabolism.

Aerobic exercise, resistance training, and high-intensity interval training have all been shown to improve insulin sensitivity, while reducing sedentary behavior may further support glycemic control. The greatest metabolic benefits appear to result from regular, long-term physical activity combined with other lifestyle modifications, particularly healthy diet and weight management.

Physical activity should therefore be considered an effective, accessible, and non-pharmacological strategy for the prevention and management of metabolic diseases. Future research should focus on identifying the most effective exercise models and developing personalized interventions that maximize metabolic benefits.

Disclossure

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