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Mechanisms of Zone 2 Aerobic Exercise on Glycemic Control in Prediabetic Populations: A Narrative Review

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Abstract: Prediabetes represents a critical window for the development of type 2 diabetes, with exercise intervention serving as a core management strategy. Zone 2 aerobic exercise, characterized by low-to-moderate intensity and sustained fat oxidation, has recently attracted attention for its potential to improve glucose metabolism. This narrative review synthesizes domestic and international research to explore the underlying mechanisms by which Zone 2 aerobic exercise improves glycemic control in prediabetic populations, including enhanced mitochondrial biogenesis, improved fatty acid oxidation capacity, ameliorated insulin signaling pathways, modulation of chronic low-grade inflammation, and gut microbiota remodeling. Current evidence suggests that Zone 2 aerobic exercise can improve insulin sensitivity through multi-target, multi-pathway synergistic effects, thereby reducing the risk of diabetes progression. Future high-quality randomized controlled trials are needed to validate its long-term efficacy and optimal exercise prescription parameters.

Keywords: Prediabetes; Zone 2 aerobic exercise; Glycemic control

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1. Introduction

Prediabetes refers to an intermediate metabolic state in which blood glucose levels are elevated above normal but have not yet reached the diagnostic threshold for type 2 diabetes, encompassing impaired fasting glucose (IFG), impaired glucose tolerance (IGT), or both [1]. The global prevalence of prediabetes is approximately 37.4%, and without effective intervention, approximately 5–10% of prediabetic individuals progress to type 2 diabetes each year [2].

Exercise intervention is the cornerstone of prediabetes management. The American Diabetes Association recommends at least 150 minutes of moderate-intensity aerobic exercise per week [3]. However, traditional exercise prescriptions have predominantly focused on moderate-intensity continuous training (MICT) or high-intensity interval training (HIIT), with relatively limited research on Zone 2 aerobic exercise.

Zone 2 aerobic exercise is defined as low-to-moderate intensity sustained exercise performed below the first lactate threshold (LT1), with fat serving as the primary fuel substrate [4]. Its characteristics include relatively low exercise intensity (approximately 60–70% of maximum heart rate), prolonged sustainability, and low perceived exertion, making it suitable for individuals with limited exercise foundations. Increasing evidence has demonstrated that Zone 2 exercise offers unique advantages in improving metabolic health [5,6].

This review aims to synthesize the mechanisms by which Zone 2 aerobic exercise improves glycemic control in prediabetic populations, providing a theoretical basis for clinical exercise prescription.

2. Physiological characteristics of zone 2 aerobic exercise

2.1. Definition and intensity demarcation

The intensity of Zone 2 aerobic exercise is typically defined as being below the first lactate threshold (LT1) or the first ventilatory threshold (VT1) [4]. At this intensity, blood lactate concentration remains below 2 mmol/L, the body relies predominantly on aerobic metabolism, and fat oxidation rate reaches its peak [7].

In terms of heart rate, Zone 2 exercise corresponds to 60–70% of maximum heart rate (HRmax) or 40–59% of heart rate reserve (HRR) [8]. From the perspective of rating of perceived exertion (RPE), it corresponds to a score of 11–13 on the Borg scale (light to somewhat hard) [9].

2.2. Metabolic characteristics

The core metabolic feature of Zone 2 exercise is maximized fat oxidation. San-Millán and Brooks (2018) demonstrated that well-trained athletes can achieve fat oxidation rates of 1.0–1.5 g/min at Zone 2 intensity, whereas sedentary individuals achieve only 0.3–0.5 g/min. This difference reflects variations in mitochondrial function and metabolic flexibility.

Additionally, Zone 2 exercise can significantly activate the AMPK (AMP-activated protein kinase) pathway, promoting the translocation of glucose transporter protein GLUT4 to the cell membrane and increasing muscular glucose uptake [10].

3. Pathological mechanisms of prediabetes

The development of prediabetes involves multiple interrelated pathophysiological processes. The primary alteration is insulin resistance, wherein skeletal muscle, liver, and adipose tissue exhibit decreased sensitivity to insulin, resulting in reduced efficiency of glucose uptake and utilization [11]. Concurrently, pancreatic β-cell function progressively deteriorates, and insulin secretory capacity becomes insufficient to compensate for peripheral insulin resistance, leading to disruption of glucose homeostasis [12].

At the cellular level, elevated free fatty acid levels lead to increased intramyocellular lipid deposition, including accumulation of intramyocellular triglycerides and diacylglycerol, which interfere with insulin signal transduction [13]. Chronic low-grade inflammation also constitutes an important pathological foundation, with elevated levels of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) activating inflammatory pathways including JNK and IKKβ, thereby suppressing insulin receptor substrate function [14]. Furthermore, decreased skeletal muscle mitochondrial quantity and function, accompanied by reduced fatty acid oxidation capacity, further exacerbate lipid metabolic dysfunction and insulin resistance [15]. These mechanisms are intricately interconnected and mutually

reinforcing, forming a vicious cycle that ultimately drives the progression from prediabetes to type 2 diabetes.

4. Mechanisms of zone 2 aerobic exercise in improving glycemic control

4.1. Enhancement of mitochondrial biogenesis and function

Mitochondria serve as the primary site of fatty acid oxidation. Prediabetic individuals commonly exhibit skeletal muscle mitochondrial dysfunction, manifested as reduced mitochondrial density and decreased oxidative phosphorylation capacity [15]. Zone 2 aerobic exercise can improve mitochondrial function through multiple molecular pathways.

Energy stress induced by exercise activates AMPK, which phosphorylates and activates PGC-1α (peroxisome proliferator-activated receptor gamma coactivator 1-alpha), initiating the mitochondrial biogenesis program [16]. Activated PGC-1α further cooperates with nuclear respiratory factors NRF-1 and NRF-2, as well as mitochondrial transcription factor A (TFAM), to promote mitochondrial DNA replication and mitochondrial protein synthesis [17]. At the functional level, regular Zone 2 exercise can increase the activity of mitochondrial electron transport chain complexes I through IV, enhancing ATP production efficiency and significantly improving the oxidative phosphorylation capacity of skeletal muscle [18].

Hood et al. (2016) demonstrated that six weeks of moderate-intensity aerobic exercise increased skeletal muscle mitochondrial content by 40–50%, with a significant improvement in fatty acid oxidation capacity.

4.2. Enhancement of fatty acid oxidation capacity and reduction of lipotoxicity

Intramyocellular lipid deposition, particularly diacylglycerol and ceramides, is an important contributor to insulin resistance [13]. These lipid metabolites can activate protein kinase C, which phosphorylates inhibitory sites on insulin receptor substrate-1 (IRS-1), thereby blocking insulin signal transduction [19].

Zone 2 aerobic exercise reduces lipotoxicity through synergistic effects at multiple levels. At the level of fatty acid uptake, exercise upregulates the expression of fatty acid transport proteins such as CD36 and FATP, promoting efficient fatty acid entry into mitochondria [20]. At the level of oxidative metabolism, exercise increases the activity of carnitine palmitoyl transferase-1, the rate-limiting enzyme for long-chain fatty acid entry into the mitochondrial matrix for β-oxidation [21]. Through sustained fat oxidation, intramyocellular DAG and ceramide levels are significantly reduced, restoring the sensitivity of the insulin signaling pathway [22].

Van Aggel-Leijssen et al. (2002) found that six weeks of indoor cycling exercise (intensity approximately 60% VO_2 max) significantly reduced intramyocellular triglyceride content in prediabetic male subjects and improved insulin sensitivity.

4.3. Improvement of insulin signaling pathways

The core cascade of the insulin signaling pathway proceeds as follows: insulin binds to the insulin receptor (IR) on the cell membrane, activating insulin receptor substrate (IRS), which in turn activates phosphoinositide 3-kinase and protein kinase B, ultimately promoting the translocation of glucose transporter protein GLUT4 to the cell membrane to facilitate glucose uptake [23].

Zone 2 aerobic exercise can enhance this pathway at multiple levels. At the receptor level, exercise upregulates the expression density of insulin receptors in skeletal muscle, increasing insulin binding capacity [24]. At the signal transduction level, exercise restores the normal tyrosine phosphorylation state of IRS-1 by

reducing inhibitory serine phosphorylation caused by inflammatory cytokines and lipid metabolites, ensuring effective downstream signal transmission [25]. At the effector level, mechanical stress and metabolic signals induced by exercise activate Akt, promoting GLUT4 translocation to the cell membrane [26]. Additionally, regular exercise can increase skeletal muscle GLUT4 protein expression by 30–50% through transcriptional regulation [27].

Richter and Hargreaves (2013) noted that a single bout of exercise can increase GLUT4 translocation through contraction-dependent pathways, while regular exercise increases total GLUT4 content through transcriptional regulation. The synergistic effects of both mechanisms significantly enhance skeletal muscle glucose uptake capacity.

4.4. Modulation of chronic low-grade inflammation

Chronic low-grade inflammation is an important driver of insulin resistance. In prediabetic individuals, increased macrophage infiltration in adipose tissue and elevated levels of pro-inflammatory cytokines such as TNF-α, IL-6, and C-reactive protein can activate JNK and IKKβ pathways, suppressing insulin signaling [14].

Zone 2 aerobic exercise exerts significant anti-inflammatory effects. At the cellular level, exercise reduces the proportion of M1-type (pro-inflammatory) macrophages in adipose tissue while increasing M2-type (anti-inflammatory) macrophage infiltration, thereby improving the inflammatory microenvironment of adipose tissue [28]. At the circulating cytokine level, regular exercise significantly reduces peripheral blood levels of TNF-α, IL-6, and CRP [29]. Notably, IL-6 released from skeletal muscle during exercise has anti-inflammatory properties, promoting the secretion of anti-inflammatory cytokines IL-10 and IL-1 receptor antagonist [30]. At the molecular mechanism level, exercise inhibits NF-κB nuclear translocation, reducing the transcriptional activation of pro-inflammatory genes [31].

The systematic review by Gleeson et al. (2011) demonstrated that regular moderate-intensity exercise can reduce CRP levels by 20–30% and TNF-α by 15–20%, an anti-inflammatory effect that is of considerable significance for improving insulin sensitivity.

4.5. Remodeling of gut microbiota

In recent years, the relationship between gut microbiota and metabolic health has attracted considerable attention. Prediabetic individuals commonly exhibit gut dysbiosis, characterized by an elevated Firmicutes-to-Bacteroidetes ratio and reduced abundance of short-chain fatty acid -producing bacteria [32].

Zone 2 aerobic exercise can improve gut microbiota composition and function across multiple dimensions. First, exercise increases the α-diversity of gut microorganisms, promoting a healthier microbial community structure [33]. Second, exercise promotes the proliferation of beneficial bacteria such as *Akkermansia muciniphila* and *Faecalibacterium prausnitzii*, which are the primary producers of SCFAs [34]. SCFAs, particularly butyrate and propionate, can activate G protein-coupled receptors GPR41 and GPR43, improving intestinal barrier function, reducing gut permeability, and attenuating endotoxemia [35]. Additionally, exercise can alter the bile acid metabolic profile, activating the farnesoid X receptor and TGR5 receptor, thereby improving glucose and lipid metabolism [36].

Allen et al. (2018) found that six weeks of aerobic exercise significantly increased SCFA-producing bacteria in subjects, and the elevation of SCFA levels was positively correlated with improved insulin sensitivity.

4.6. Other potential mechanisms

Beyond the core mechanisms described above, Zone 2 aerobic exercise may indirectly improve glycemic control through additional pathways. At the vascular level, exercise increases the bioavailability of nitric oxide, improving endothelial function and microcirculation, thereby facilitating glucose delivery to skeletal muscle [37]. At the level of cellular quality control, exercise activates skeletal muscle autophagy, clearing damaged mitochondria and protein aggregates to maintain cellular metabolic homeostasis [38]. At the level of gene regulation, exercise can modulate the expression of metabolism-related genes through epigenetic mechanisms including DNA methylation and histone modification [39]. Furthermore, exercise can optimize sleep quality and reduce cortisol levels, indirectly promoting glycemic control by improving sleep and attenuating stress responses [40].

5. Summary of current research evidence

5.1. Animal studies

Multiple animal studies have confirmed the metabolic benefits of Zone 2 exercise. Zhang et al. (2017) found that 12 weeks of moderate-intensity treadmill exercise (15 m/min, 60 min/day) significantly improved insulin sensitivity in high-fat diet-induced prediabetic mice, with mechanisms closely related to AMPK activation and enhanced mitochondrial biogenesis [41].

5.2. Human studies

In human randomized controlled trials, the landmark Diabetes Prevention Program study demonstrated that lifestyle intervention, including exercise, reduced diabetes incidence by 58% [42]. Uusitupa et al. (2003) found that 12 weeks of moderate-intensity aerobic exercise reduced HbA1c by 0.2–0.4% in prediabetic individuals [43]. In the Chinese population, long-term follow-up from the Da Qing Diabetes Prevention Study showed that 16 weeks of cycle ergometer exercise (5 sessions/week, 45 min/session) significantly improved fasting plasma glucose and oral glucose tolerance test results [44].

Regarding dose-response relationships, current evidence suggests that 150–300 minutes of moderate-intensity exercise per week yields the greatest metabolic benefits, with an optimal frequency of 4–5 sessions per week and a recommended duration of 30–60 minutes per session [45,46].

5.3. Comparison with other exercise modalities

Compared to high-intensity interval training (HIIT), Zone 2 aerobic exercise offers unique advantages across multiple dimensions. In terms of exercise intensity, Zone 2 exercise is classified as low-to-moderate intensity, whereas HIIT requires 85–95% of maximum heart rate, placing higher demands on cardiopulmonary function. Regarding fat oxidation efficiency, Zone 2 exercise, with intensity maintained within the fat oxidation peak zone, achieves significantly higher fat oxidation per unit time compared to HIIT. In terms of adherence, Zone 2 exercise, due to its low perceived exertion and minimal subjective discomfort, is more conducive to long-term participation. Regarding exercise injury risk, Zone 2 exercise carries substantially lower joint loading and muscle damage risk than HIIT. Therefore, for prediabetic individuals with limited exercise foundations, older age, or multiple comorbidities, Zone 2 aerobic exercise represents a safer and more feasible choice [47].

6. Conclusions and future directions

6.1. Main conclusions

In summary, Zone 2 aerobic exercise can effectively improve glycemic control in prediabetic populations through multi-target, multi-pathway synergistic mechanisms, including enhancing mitochondrial biogenesis and function, increasing fatty acid oxidation capacity to reduce lipotoxicity, improving insulin signaling pathways, modulating chronic low-grade inflammation, and remodeling gut microbiota. This exercise modality is characterized by low intensity, high sustainability, strong adherence, and favorable safety profiles, making it suitable as a first-line exercise intervention strategy for prediabetic populations.

6.2. Practical recommendations

Based on current evidence, it is recommended that prediabetic individuals select exercise modalities such as cycle ergometry, brisk walking, jogging, or swimming, maintaining exercise intensity at 60–70% of maximum heart rate or an RPE of 11–13, exercising for 30–60 minutes per session, 4–5 times per week, with a minimum intervention duration of 12 weeks to achieve significant metabolic improvements.

6.3. Future research directions

Future research should focus on several key areas: conducting multi-center, large-scale randomized controlled trials to validate the long-term efficacy of Zone 2 exercise and its diabetes prevention effects; exploring dose-response relationships among different combinations of exercise frequency, duration, and intensity to establish individualized exercise prescription models; utilizing metabolomics, transcriptomics, and metagenomics to elucidate molecular mechanisms in greater depth; developing intelligent exercise prescription systems based on wearable devices for real-time monitoring and dynamic adjustment of exercise intensity; and investigating the combined effects of Zone 2 exercise with dietary interventions and pharmacotherapy to provide more comprehensive evidence-based support for the integrated management of prediabetes.

Disclosure statement

The author declares no conflict of interest.

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