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Effects of High-Intensity Interval Training (HIIT) on Metabolic Health among Individuals with Insulin Resistance

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ABSTRACT

Introduction

Insulin resistance (IR) is a key mechanism underlying metabolic syndrome, type 2 diabetes, non-alcoholic fatty liver disease, and cardiovascular disease. Its prevalence is rising worldwide, including in Poland, with many cases undiagnosed. Lifestyle strategies, especially physical activity, are essential. High-Intensity Interval Training (HIIT) has recently drawn attention as a time-efficient method with strong potential to improve metabolic health.

Aim

This study aims to evaluate the effects of HIIT on metabolic health in individuals with insulin resistance. The focus is on insulin sensitivity, glycemic control, lipid profile, blood pressure, and hepatic function, and compares HIIT with moderate-intensity continuous training (MICT).

State of Knowledge

HIIT involves repeated bouts of vigorous activity with recovery periods, usually at 80–100% of maximal heart rate. Evidence shows that it enhances glucose uptake, reduces HOMA-IR, lowers HbA1c, and improves lipid balance by decreasing LDL and raising HDL cholesterol. HIIT also reduces systolic blood pressure and improves liver parameters in non-alcoholic fatty liver disease. Benefits are often greater than or comparable to MICT, despite shorter training duration. The strongest effects occur when combined with weight reduction and dietary changes.

Summary (Conclusions)

HIIT is an effective non-pharmacological strategy for managing insulin resistance and related disorders. It improves glycemic control, lipid and blood pressure regulation, and markedly enhances insulin sensitivity. Thanks to its time efficiency and ease of integration into daily life, HIIT is a valuable therapeutic option that may significantly support prevention and treatment of insulin resistance in both clinical and public health contexts.

Keywords: high-intensity interval training, insulin resistance, metabolic health, glycemic control, lipid profile, blood pressure

INTRODUCTION

Definitions

HIIT (High-Intensity Interval Training)

High-Intensity Interval Training (HIIT) is a form of physical exercise characterized by repeated, short bursts of activity reaching high or very high levels of various intensity parameters. HIIT protocols have been widely implemented not only in professional and amateur sports but also in the prevention and treatment of cardiovascular diseases. Despite its longstanding application and broad range of uses, HIIT lacks a uniform definition [1]. Currently, four exercise intensity models are described in the literature. Works by Seiler, Casado, and Jamnick contributed to one of the most commonly used classification systems, which divides effort into six zones based on the Rating of Perceived Exertion (RPE) [2-4]. Zones 5 and 6 correspond to intensities achieved during HIIT sessions (RPE 17-19/20). Another widely used model is the three-zone division based on power output or physiological indicators. These include the moderate, high, and submaximal/very high-intensity zones, delineated by threshold values such as LT1 (first lactate threshold) and GET (gas exchange threshold), and in the case of HIIT, LT2 (second lactate threshold) and MLSS (maximal lactate steady state) [5-7].

The 2020 WHO guidelines, which offer a universal classification of aerobic exercise intensity, also describe three levels of effort: light, moderate, and vigorous, defined according to the metabolic equivalent of task (METs) and RPE values [8]. Activities exceeding 6 METs or an RPE of 7/10 are categorized as vigorous and consistent with HIIT. The American College of Sports Medicine, in its 11th edition of exercise guidelines, identifies five intensity zones: very light, light, moderate, vigorous, and near-maximal to maximal. Entering the last two zones corresponds to achieving 77-95% HRmax or >95% HRmax, 60-89% HRR or >90% HRR, 64%-90% VO2max or >91% VO2max, and an RPE of 14-17/20 or >18/20. Reaching these parameters also qualifies as HIIT. Weston, in turn, defined HIIT as exercise performed at 80% to 100% of peak heart rate [9].

Insulin Resistance

Insulin resistance (IR) is defined as a pathological condition in which peripheral tissues – primarily skeletal muscle, liver, and adipose tissue – show impaired responsiveness to insulin

despite normal or elevated circulating insulin levels. This dysfunction leads to reduced peripheral glucose uptake and its inefficient utilization, resulting in compensatory hyperinsulinemia [10, 11]. Mechanisms contributing to insulin resistance may involve defects in the insulin receptor itself or elements of its intracellular signaling cascade, ultimately disturbing glucose metabolism, lipid regulation, and inflammatory processes [11, 12].

Insulin resistance is a key component of multiple pathologies, including metabolic syndrome, type 2 diabetes mellitus (T2DM), non-alcoholic fatty liver disease (NAFLD), and cardiovascular diseases [11, 13]. Furthermore, it may develop asymptomatically over several years and precede clinically overt disturbances in glucose homeostasis, making it a critical target for early prevention and intervention strategies [10, 13].

Epidemiology

Insulin resistance represents a central pathophysiological mechanism in the development of metabolic syndrome and T2DM. Its global prevalence has risen alarmingly in recent decades. According to the World Health Organization (WHO), the number of individuals living with diabetes has quadrupled since 1990, reaching 830 million by 2022 [14] . Notably, 59% of those affected remain untreated, corresponding to more than 445 million adults over the age of 30. T2DM, which accounts for approximately 95% of all diabetes cases, arises due to chronic insulin resistance that is often undiagnosed for extended periods [14, 15].

Although not synonymous with insulin resistance, metabolic syndrome is frequently used as a practical marker of IR in epidemiological studies. Its prevalence varies by geographic region, ranging from approximately 10% to 30% [16]. In Poland, large-scale population-based studies such as WOBASZ and NATPOL have shown a significant increase in metabolic syndrome prevalence in recent years. In the WOBASZ II study (2013-2014), conducted among 19,751 Polish adults aged 20–74, metabolic syndrome prevalence reached 32.8% in women and 39.0% in men – a notable increase compared to the 2003–2005 WOBASZ survey (26.6% in women and 30.7% in men). The most marked rise was seen in carbohydrate metabolism disorders: in WOBASZ II, 47% of men and 35% of women met criteria for impaired fasting glucose or diabetes [17]. For comparison, in the NATPOL 2002 study using ATP III criteria, metabolic syndrome was found in 22.6% of women and 18.0% of men, confirming a growing trend in Poland [18].

Given the increasing prevalence of IR and its low recognition and treatment rates, there is an urgent need to implement integrated preventive and therapeutic strategies at both the individual and systemic levels.

Pathophysiology

Insulin resistance is characterized by the failure of normal insulin concentrations to elicit the expected biological response in target tissues. Both genetic and environmental factors contribute to this condition. Approximately half of IR cases are genetically determined, while the remainder are acquired, often resulting from obesity or physical inactivity [19].

From a pathophysiological standpoint, the core mechanism involves disruptions in intracellular signaling following insulin binding to the insulin receptor (IR). This includes impaired phosphorylation of the insulin receptor and its substrates (IRS), leading to insufficient activation of PI3K and AKT signaling pathways, which are essential for GLUT4 translocation to the cell membranę [20]. Consequently, glucose transport into muscle cells is compromised, resulting in hyperglycemia and compensatory insulin secretion [20, 21].

In skeletal muscle cells – the primary site of glucose uptake – IR manifests as decreased GLUT4 transcription, impaired glucose transport and phosphorylation, reduced glucose oxidation, and impaired glycogen synthesis. Mitochondrial dysfunction plays a central role, leading to reduced oxidative phosphorylation and intracellular lipid accumulation, particularly ceramides and diacylglycerols [21].

A key mechanism in the development of IR is chronic low-grade inflammation, particularly associated with visceral obesity. Enlarged adipose tissue (hypertrophy) produces pro-inflammatory cytokines such as TNF-α and IL-6. These cytokines exert both local and systemic effects, disrupting insulin signaling in multiple organs. At the cellular level, they activate stress kinases (e.g., JNK, IKKβ), which aberrantly phosphorylate IRS proteins on serine residues, thereby impairing signal transduction from the insulin receptor [22].

Emerging research emphasizes the role of insulin receptor endocytosis in IR pathogenesis. Upon activation, the receptor undergoes clathrin-mediated internalization, which modulates signal intensity and duration. Disruptions in this process, for example through AP2 complex dysfunction, may lead to excessive receptor degradation or impaired recycling, further exacerbating signal deficits [23].

The hormonal system also contributes to IR development. Activation of the mineralocorticoid receptor by aldosterone increases oxidative stress, reduces insulin receptor expression, and promotes IRS degradation, collectively impairing insulin signaling. Individuals with primary

hyperaldosteronism exhibit higher IR indices, which improve following mineralocorticoid receptor antagonist therapy [24].

In the liver, impaired glucose uptake is compounded by unrestrained gluconeogenesis, even in the presence of high insulin levels. Disturbances in lipid oxidation lead to triglyceride accumulation in hepatocytes, resulting in NAFLD, and in advanced stages, fibrosis and cirrhosis [22].

To compensate for insulin resistance, pancreatic β-cells increase insulin secretion, temporarily maintaining euglycemia. However, chronic hyperinsulinemia ultimately exhausts β-cell function, leading to T2DM onset [19, 20, 25].

The insulin resistance syndrome is not confined to glucose metabolism. It is frequently accompanied by dyslipidemia (elevated triglycerides, reduced HDL), hypertension, and increased cardiovascular disease risk [25, 26]. IR also plays a key role in the pathogenesis of polycystic ovary syndrome (PCOS) and NAFLD [25].

Thus, the pathophysiology of IR is multifactorial, encompassing interactions between hormonal signaling, energy metabolism, inflammation, and mitochondrial dysfunction. Understanding these mechanisms is crucial for developing effective therapeutic and preventive strategies.

CURRENT TREATMENT METHODS (LIFESTYLE VS. MEDICATION)

Diet and lifestyle interventions

The primary therapeutic approach to insulin resistance—especially in its early stages—should involve non-pharmacological methods based on lifestyle modifications. According to current literature, dietary adjustments play a crucial role, including caloric restriction (in cases of overweight or obesity), improving the quality of consumed products, and limiting carbohydrates with a high glycemic index and load. These measures should be complemented by regular physical activity, which enhances muscle sensitivity to insulin [27].

Both diet and physical activity play a pivotal role in the treatment of insulin resistance. Reducing intake of high-glycemic-index carbohydrates, increasing dietary fiber intake, and engaging in regular exercise - both aerobic and resistance training - improve tissue sensitivity to insulin. The greatest metabolic benefits are observed when weight loss is combined with physical activity and dietary habit changes [28].

According to recent data, high-intensity interval training (HIIT) significantly improves metabolic parameters, including increased insulin sensitivity and reduced HOMA-IR levels. Studies have shown that a 12-week HIIT intervention can reduce insulin resistance, especially when combined with spirulina supplementation, as evidenced by changes in levels of apolipoproteins A, B, and J [29].

The mechanisms underlying insulin resistance are complex and include chronic inflammation, oxidative stress, mitochondrial dysfunction, and gut dysbiosis. These elements play a crucial role in the development and progression of metabolic disorders [30]. Therefore, therapeutic strategies should target not only weight control but also aim to mitigate these pathological processes.

The qualitative composition of the diet directly influences insulin sensitivity. A diet rich in fiber, polyunsaturated fatty acids, and with a low glycemic load contributes to improved glucose metabolism. In particular, low-carbohydrate and Mediterranean dietary patterns have shown beneficial effects [31, 32].

Bioactive compounds found in vegetables and plant-based foods, such as sulforaphane, may enhance insulin signaling by regulating ceramide metabolism - lipid molecules involved in the pathogenesis of insulin resistance [33]. Incorporating such components into the diet may therefore provide additional therapeutic benefits, regardless of weight loss.

Pharmacological treatment

In more advanced cases, treatment of insulin resistance may require pharmacological intervention. Metformin, the first-line drug, improves glucose uptake in skeletal muscles by increasing the translocation of the GLUT4 transporter to the cell membrane. Moreover, it has been shown to positively affect gut microbiota composition and reduce hepatic glucose production, collectively enhancing tissue insulin sensitivity [34].

Hight-Intensity Interval Training

Table 1 summarizes selected studies evaluating the effects of high-intensity interval training (HIIT) on metabolic and clinical outcomes in individuals with insulin resistance. The table presents the type of intervention, assessed parameters, and reported results, providing a comparative overview of the current evidence.

Table 1. Impact of HIIT Protocols on Metabolic and Clinical Parameters in Insulin Resistance

PARAMETERS	TYPE OF INTERVENTION	OUTCOME	COMMENT
Lipid Profile [35]	• Phase 1: 30’ continuous at 50% VO_2peak (Weeks 1–2) • Phase 2: 4×(1’ at 80% + 4’ at 50%) (Weeks 3–6) • Phase 3: 6×(1’ at 85% + 4’ at 60%) (Weeks 7–12)	Reduction in LDL and total cholesterol Increase in HDL	Lipid profile improvement observed especially in patients with type 2 diabetes
NAS [36]	• 4×4 min intervals at 90–95% HR max	NAS decreased by 2–3 points	Participants had baseline NAS > 4/8

	• Active recovery: 3 min at 50% HRmax • Frequency: 3×/week		
Blood Pressure [37]	• HIIT: 4×4 min at 90–95% HR max, 3×/week, 12 weeks • Other protocols: 10×1' or 4–6×1', 3×/week for 12 weeks	SBP: significant reduction in most studies (~12 mmHg) DBP: variable response (0–8 mmHg decrease)	Effectiveness depends on interval duration; HIIT more effective than MICT, especially for SBP
HbA1c [35]	• HIIT: 4×4 min at 85–95% HR max + 3 min active recovery • 3×/week for 12 weeks	HbA1c reduction by 0.5% (from 7.7% to 7.2%; $p<0.01$)	HIIT more effective than MICT for glycemic control; significant HbA1c reduction in patients with type 2 diabetes
HOMA-IR [38]	• Low-volume HIIT with reduced intensity and extended interval duration	Moderate improvement in HOMA-IR, particularly with concurrent weight loss +1 interval = −2.2 HOMA-IR	HIIT has greater effect on peripheral than hepatic insulin resistance; better outcomes with longer duration & weight loss

Abbreviations: HIIT – High-Intensity Interval Training; MICT – Moderate-Intensity Continuous Training; LDL – Low-Density Lipoprotein cholesterol; SBP – Systolic Blood Pressure; DBP – Diastolic Blood Pressure; HR – Heart Rate; NAS – NAFLD Activity Score; NAFLD – Non-Alcoholic Fatty Liver Disease; HbA1c – Glycated Hemoglobin; HOMA-IR – Homeostatic Model Assessment of Insulin Resistance.

CONCLUSIONS

The analysis conducted demonstrates that high-intensity interval training (HIIT) represents an effective non-pharmacological intervention for the treatment and prevention of insulin

resistance and associated metabolic disorders. The studies included in this work confirmed the beneficial effects of HIIT on glycemic control, reduction of the HOMA-IR index, lowering of HbA1c levels, normalization of systolic blood pressure, and improvement in lipid profile. It was observed that the efficacy of HIIT protocols may be comparable to—or even exceed—that of moderate-intensity continuous training (MICT), while requiring significantly less time commitment. The most pronounced metabolic improvements were noted in interventions accompanied by weight loss, indicating a synergistic effect of physical activity and body weight management. HIIT demonstrated a particularly strong impact on peripheral insulin sensitivity, which is of key importance in the pathophysiology of insulin resistance and its complications. Considering the increasing prevalence of carbohydrate metabolism disorders and the limited effectiveness of current preventive strategies, the integration of HIIT into comprehensive therapeutic approaches may constitute a critical step toward more effective management of insulin resistance at both the individual and population levels.

Disclosure

Author's contribution

Conceptualization: Z. Kania-Bonicka; methodology: O. Dziechciarz; software: K.Wojtach; check: B. Głowacka; formal analysis: H. Mruzek; investigation: O. Kałwak; resources: K. Wojtach; data curation: Z. Kania-Bonicka; writing-rough preparation: O. Dziechciarz; writing – review and editing: B. Głowacka; visualization: H. Mruzek; supervision: O. Kałwak; project administration: Z. Kania-Bonicka

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The authors deny any conflict of interest.

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