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Effect of high-intensity interval training on omentin-1 serum levels, gene expression, and insulin resistance in type 2 diabetic rats

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Abstract

Background: Omentin-1 plays an important role in insulin function. Despite numerous studies, the effect of interval training on this adipokine is still vague. This study aimed to elucidate the effect of high-intensity interval training (HIIT) on serum glucose, insulin, insulin resistance (IR), omentin-1 serum levels, and gene expression in the visceral adipose tissue in type 2 diabetic (T2D) rats.

Methods: In an experimental study, 20 male rats (8-10 weeks, weight: 250-270 g) were randomly divided into 2 groups: diabetic control (N=10) and diabetic training (N=10). The training protocol was 30 minutes of HIIT (1-min run, 2-min rest) performed 5 days a week for 4 weeks. Fasting blood glucose, insulin resistance, omentin-1 serum level, and gene expression were measured in the visceral fat 48 hours after the last exercise for both groups.

Results: The HIIT resulted in lower serum glucose and insulin resistance (P = 0.001), higher serum omentin-1 levels (P = 0.001), and higher visceral fat gene expression (P = 0.004) in the training group compared to the control group.

Conclusion: Lower serum glucose and insulin resistance and higher omentin-1 serum levels and gene expression in the training group can prove the effectiveness of HIIT training in T2D, although further research is required.

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Introduction

The pervasiveness of type 2 diabetes (T2D) is expanding all over the world (1). Researchers evaluated 171 million diabetic patients in 2000 and anticipate this number to rise to 366 million in 2030 (2). Common reasons for T2D are insulin disorder in the main glucose-consuming tissues (for example, muscles, liver, and fat tissues), fat amassing, metabolic perturbation, and adipokine secretion problems (3). A standout amongst the most critical adipokines, identified with metabolic clutters, is omentin. Omentin (additionally called intelectin-1 or intestinal lactoferrin receptor) is a 38-40 kDa adipokine, a 313-amino peptide, which was distinguished from a copy deoxyribonucleic acid (cDNA) library in visceral omental fat tissue by Yang et al. (2003) (4). There are 2 omentin genes situated in the 1q22–q23 chromosomal locale, which deliver omentin-1 and omentin-2 (5). Omentin-1 is the prevalent isoform in the human plasma and fat tissue (6).

It has been demonstrated that omentin-1 levels are diminished in patients with impeded glucose control (7), type 1 (8), and type 2 diabetes (7, 9). Diminished omentin-1 levels in diabetic patients may be due to the disturbance in omentin biosynthesis or hyperglycemia and hyperinsulinemia (10). Tan et al. showed that insulin and glucose diminish omentin generation and its messenger ribonucleic acid (mRNA) expression in the omental fat tissue (8). Yang et al. expressed that treatment with recombinant omentin-1 raised insulin-invigorated glucose transport in vitro (4). These findings proposed the conceivable role of omentin decrease in insulin resistance (IR). Afterward, Yang et al. (2006) demonstrated that omentin expanded Akt phosphorylation in the absence and presence of insulin (11). Increased Akt phosphorylation can enhance insulin-animated glucose uptake and phosphorylation in subcutaneous and visceral adipocytes (11). Different studies demonstrated the anti-inflammatory, antiatherogenic, and antidiabetic properties of omentin (12). Likewise, omentin-1 levels had a negative correlation with the body mass index (BMI), fasting insulin, homeostasis model assessment of insulin resistance (HOMAIR), and plasma glucose (13).

Some factors can modify omentin patterns. The most important one is physical activity (14, 15). It has been shown that 1 training session can increase omentin-1 gene expression and control hyperglycemia in diabetic rats (16). Other studies proved that 12 weeks of aerobic training could increase omentin-1 levels in obese men (17), but 4 weeks of resistance training failed to improve omentin levels (18).

Another type of physical activity that is considered a powerful method to enhance cardiorespiratory fitness and decrease cardiometabolic dangers is high-intensity interval training (HIIT) (19). Phillips et al. demonstrated that HIIT is a practical and time-efficient training program to change cardiometabolic risk factors in adults with the risk factors for T2D (19), although they did not study the omentin-1 cascade. Due to the significance of omentin-1 in metabolic control (particularly in IR), it can be very helpful to investigate the effect of physical activity on omentin-1 levels and metabolic indexes of T2D. The main reason for not exercising is the "perceived lack of time," which is one of the most frequently cited barriers (20). Therefore, because of the time-efficient nature of HIIT, this study was designed to investigate the effect of 4 weeks of HIIT on glucose serum levels, insulin, IR, omentin-1 serum levels, and gene expression in the visceral adipose tissue of T2D rats.

Methods

All the experimental procedures were approved by the Ethics Committee of Kerman University of Medical Sciences (ethics code: 99000443). All the animals received humane care according to the criteria outlined in the Guide for the Care and Use of Laboratory Animals prepared by the National Academy of Sciences and published by the National Institutes of Health (NIH publication 86-23, revised in 1985).

Male Wistar rats were purchased from Baqiyatallah Animal Center (Tehran, Iran). The animals were housed with a 12:12-h light-dark cycle at ambient temperature (22 °C) with 30-40% relative humidity. The rats were assigned to their respective diets, which consisted of chow and tap water ad libitum. After acclimatization for 1 week, they were assigned randomly to one of the following groups (n=10): control or training. These groups were matched based on body weight. The training group performed the HIIT protocol 5 times per week for 4 weeks, but the control group did not perform any kind of training. Each training section consisted of 10 repetitions of 1 minute of running, followed by 2 minutes of rest. The total time for each session was 30 minutes. Running speed commenced from 30 m/min and increased by 10 m/min weekly. Before the HIIT protocol started, the rats ran for 5 minutes at a speed of 10 m/min for warm-up. The same protocol was used for cool-down (22). The rats were sacrificed 2 days after the last exercise session to preclude acute effects on the measured variables. They were fasted for 4 hours, anesthetized via isoflurane inhalation, and killed by decapitation.

In this examination, we utilized nicotinamide and streptozotocin for diabetes induction (21). Nicotinamide (95 mg/kg body weight) (Sigma, Saint-Louis, MO, USA), dissolved in saline, was injected intraperitoneally 15 min before the administration of STZ (60 mg/kg body weight) (Sigma, Saint-Louis, MO, USA), which was dissolved in citrate buffer (pH 4.5) promptly before use. Seven days later, a drop of blood was taken from the tail and set on a glucometer. Rats with moderate diabetes and hyperglycemia (blood glucose ≥ 126 mg/dL) were included in the investigation (22).

After 2 months of diabetes induction, the training protocol was started when the rats were 17 weeks old. The training group performed an HIIT protocol for 4 weeks, 5 days a week (Saturdays, Sundays, Tuesdays, Wednesdays, and Thursdays).

Both groups were sacrificed 48 hours after the last training session. The rats were anesthetized and then euthanized (23). The rats were euthanized by cervical

dislocation. Blood was drawn from the heart, and soleus muscle biopsy was performed. Body weight, soleus muscle weight, and the expending sustenance were weighed by Sartorius computerized scales (Germany). Omentin-1 serum levels were estimated by an ELISA pack (Zelbio, Germany). Fasting blood glucose was estimated by enzymatic strategy (Pars Pack, Iran) utilizing an autoanalyzer (Technicon RA-1000, USA). Insulin resistance was estimated by HOMA-IR via the accompanying equation (24): HOMA-IR index = (fasting insulin (μU/mL) × fasting glucose (mmol/L))/ 22.5.

The adipose tissue (20 mg) was powdered and then transferred to a small tube for the expansion of the QIAzol Lysis (1 mL) and treated with gDNA Eliminator. The RNA was reagent cleaned utilizing RNeasy, in addition to the Universal Mini Kit (Qiagen, Hilden, Germany) (feline. No. 73404) as per the producer's instruction. Real-time polymerase chain reaction (PCR) quantification of the T cell factor (TCF) mRNA was performed with the Rotor-Gene 6000 system using the One Step SYBR PrimeScript RT PCR kit (Takara Co). Melting curve analysis was performed at the end of the PCR cycles to validate the specificity of the expected PCR product. The relative ΔΔCT technique was employed to measure the TCF mRNA expression. We utilized RNA Polymrasell as the normalizer. Table 1 presents the primer sequences.

Table 1. Primer sequences

Primer sequences	
Omentin	F: CCAGTCAGCAAGGCAACAGAG R: CCAGATGCCCAGGTTCTCAG

The results are expressed as means ± standard error (SE). The Shapiro-Wilk Test was used to assess the normality of the data distribution, and the independent t-test was used to assess differences between the means. The statistical significance was set to $P \leqslant 0.05$. Statistical analysis was performed in SPSS v. 21 (IBM Corp., Armonk, NY, USA).

Results

As shown in Table 2 diabetes induction with nicotinicamide and streptozotocin injection increased mouse body weight (BW) in both groups significantly ($p = 0.001$). In addition, after 4 weeks HIIT, the training group had less body weight than control. There was no significant difference between groups after 2 months diabetes induction. On the other hand, daily food consumption (DFC) was not significantly different between groups. To test the hypothesis if diabetes induced obesity, we measured mouse BW before and after 2 months (Table 2).

The blood glucose was less in training group than control. In addition, this was true for IR as well. In addition, blood insulin levels were higher in training group (Table 2). All three differences can prove training effects.

To test if HIIT can compensate these reduction, Omentin-1 serum levels and gene expression in adipose tissue were assessed in both groups (Table 2). Both variables were higher in training group which can reflect the net effect of HIIT. As expected SMW was higher in training group after 4 weeks HIIT (Table 2).

Table 2. Effect of HIIT on BW, biochemical variables, and omentin-1 gene expression in the two groups

Variable	Control group	Training group
BW (before diabetes induction) (g)	248.3 ± 18.8	250 ± 15.1
BW (after diabetes induction) (g)	327 ± 28	311 ± 42.1
BW (after 4 weeks of training) (g)	343 ± 24*	323 ± 16
Glucose (mg/dL)	272 ± 16*	228 ± 27
Insulin (mg/dL)	0.172 ± 0.10	0.186 ± 0.14*
IR	6 ± 16.52*	5 ± 4.67
SMW (after 4 weeks of training) (g)	0.125 ± 0.18	0.133 ± 0.1*
DFC (g)	32.4 ± 2.1	33.2 ± 2.6
OSL (mg/mL)	97.2 ± 0.42	103.2 ± 1.8*
OGE in the adipose tissue (after 4 weeks of training)	1.02 ± 0.8	1.52 ± 0.50*

BW: Body weight, IR: Insulin resistance, SMW: Soleus muscle weight, DFC: Daily food consumption, OSL: Omentin-1 serum levels, OGE: Omentin-1 gene expression, HIIT: High-intensity interval training. * Shows a significant difference between the groups.

Discussion

Diabetes induction resulted in increasing rats' weight in the first 2 months and, continued to the end of study but, it was lower in training group. After 4 weeks training period, BW in the training group was lower remarkably ($p \leq 0.05$). In addition, SMW was higher in the training group after 4 weeks training period which means HIIT not only restricted weight gain but also, increased muscle mass and improved body composition subsequently.

Blood glucose control is a standout amongst the most critical variable in diabetes. Blood glucose and IR were lower in the training group after the training period, which is in line with Babraj et al. and Little et al. (25, 26). Altogether, these findings demonstrate that HIIT can be exceptionally compelling for glycemic control in T2D, and its time is equivalent to 33% of physical activity proposed for diabetic patients (27). It has been reported that almost no diabetic patient has enough physical activity due to lack of time (26). Based on our data, HIIT with less time and an acceptable level of impact can be an important step in solving this problem.

After 4 weeks of HIIT, omentin-1 serum levels and gene expression in the visceral adipose tissue were considerably higher in the training group. These findings are consistent with those of Ouerghi et al. and Alizadeh et al. despite shorter periods (4 weeks versus 8 weeks) (16, 28). Different investigations have detailed that visceral fat tissue is an active endocrine organ. Fat tissue synthesizes and secretes numerous hormones and cytokines (called adipokine), which contribute to fat metabolism (cholesterol transporting protein), irritation (TNFα), and insulin affectability (adiponectin and omentin) (16). Omentin-1 is an adipokine that is principally secreted by visceral fat tissue, and its gene is associated with T2D and cardiac problems (16, 29). It has been shown that omentin-1 plays an important role in carbohydrate metabolism and glucose consumption in the muscle, and increasing the consumption of glucose in the muscles decreases BW and increases the lipolysis of adipose tissue (30). Besides, omentin-1 enhances the phosphorylation of protein kinase B (Akt) and the absorption of insulin-mediated glucose, which plays an important role in maintaining glucose homeostasis (31). Akt is a serine/tyrosine-protein kinase that plays a key role as a secondary messenger in cellular functions such as glucose metabolism, proliferation, and apoptosis (8). An increment of the omentin-1 gene expression increased Akt phosphorylation, which raised glucose uptake mediated by insulin (16). Besides, physical activity seems to increase glucose uptake and consumption (16). The responsible mechanisms include greater muscle blood flow, increased insulin binding to its receptor, and higher insulin receptor and glucose transporter 4 (GLUT4) transfer to the membrane surface of the cell (16). Our results confirmed the lower amount of blood glucose and insulin resistance and higher insulin levels in the training group compared to the controls. Therefore, it seems that 4 weeks of HIIT can raise the AMP kinase activity and trigger the Akt signaling pathway, followed by an increased protein synthase and insulin secretion. Studies demonstrated that omentin-1 improves insulin sensitivity and glucose metabolism in the muscles, liver, and adipose tissues (11, 32) because omentin-1 plays a determining role in stimulating skeletal muscle insulin and glucose receptors' translation and translocation to the membrane (33). Omentin-1 serum levels are regulated by inflammation and decrease in T2D (28). Since T2D is chronic inflammation, it can be stated that increased serum levels and gene expression of omentin-1 in the visceral fat tissue were due to the dominance of HIIT.

Conclusion

In the last decades, researchers have tried to find a solution for IR in T2D. Studies showed that physical activity can improve IR. We have demonstrated that HIIT is a time-efficient exercise that can improve IR through increasing omentin-1 serum levels and gene expression in the adipose tissue. Since the main reason people do not exercise is the lack of time, HIIT can be a very useful method.

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Ethical statement

All the experimental procedures were approved by the Ethics Committee of Kerman University of Medical Sciences (Ethics code: 99000443). All the animals received humane care according to the criteria outlined in the Guide for the Care and Use of Laboratory Animals prepared by the National Academy of Sciences and published by the National Institutes of Health (NIH publication 86-23, revised in 1985).

Conflicts of interest

All the contributing authors declare that there is no conflict of interest.

Author contributions

NR, MD, SSK, and NS designed the study and performed the animal work. NR, MD, SSK, NS, and KK analyzed the data and wrote the manuscript. All the authors confirmed the final version of the manuscript.

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