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High-intensity Interval Training and Obesity

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Abstract

High-intensity interval training (HIIT) has become a promising strategy to induce a number of metabolic adaptations and alter body composition. Recent evidence suggests that HIIT can be a time-efficient strategy to promote health in sedentary overweight/obese individuals. This may be contrary to the belief held by some health professionals that training programs at high intensity are not appropriate for optimizing fat oxidation and inducing weight loss in this population. This paper reviews the results of HIIT studies conducted with overweight/obese individuals. A special focus is on the effect of HIIT on fat oxidation and weight loss.

Keywords: Obesity; Weight loss; Over weight; Metabolic adaptations

Introduction

Obesity, a major risk factor in the development of type 2 diabetes and increased morbidity and mortality, has increased rapidly in recent decades [1,2]. Exercise represents an important intervention for weight loss through the potential to raise energy expenditure [3]. Exercise intensity plays an important role in governing substrate utilization (i.e., fat and carbohydrate) during exercise. Commonly, low/moderate intensity exercise is recommended for overweight and obese individuals to promote fat oxidization [4]. Low/moderate intensity exercise is also proposed to be an appropriate intensity for preventing injury and improving tolerance [5]. The reason to use this level of exercise intensity is to ensure that sedentary overweight/obese individuals could maintain the duration of exercise sessions [4,6]. Although some overweight/obese individuals have difficulty maintaining high-intensity exercise for sustained periods, High-Intensity Interval Training (HIIT) has been suggested as a promising strategy to induce a number of metabolic adaptations usually attributed to low-intensity exercise training including several skeletal muscle adaptations that result in enhanced skeletal muscle fat oxidation and improved glucose tolerance [7]. HIIT is superior to moderate-intensity continuous training in improving cardiorespiratory fitness, and is safe and well-tolerated [8]. HIIT has been reported to be more enjoyable than moderate-intensity continuous training [9]. A recent systematic review and meta-analysis concluded that HIIT is an equally effective alternative to moderate-intensity continuous training, with improvements in aerobic capacity in healthy, young people [10]. However, a growing body of evidence suggests that HIIT can be a time-efficient strategy to increase functional capacity and decrease risk for all-cause mortality in sedentary, overweight/obese individuals [11-13]. This review summarizes the results of HIIT studies conducted with overweight/obese individuals, with a special focus on the effect of HIIT on fat oxidation and weight loss.

HIIT Protocols

Commonly, HIIT is of short duration and performed above the lactate threshold, close to maximal oxygen consumption (VO_{2max}), and with intermittent periods of rest, allowing sedentary overweight/obese individuals sufficient time to recover and perform additional high-intensity bouts. HIIT is based on the Wingate test where an "all out" effort is performed; individuals start with 3 to 5 minutes of warm-up and then cycle for 30 seconds at maximum effort against a supra-maximal workload. A typical HIIT protocol consists of 4 to 6 Wingate tests separated by ~4 minutes of rest, for a total of 2 to 3 minutes of maximal exercise spread over 15 to 30 minutes. The Wingate protocol is extremely strenuous and uncomfortable, which is why HIIT is suited to active and motivated individuals [14-17]. However, a growing body of evidence suggests that HIIT, using the Wingate test, can be used by overweight/obese sedentary individuals [11,18-20]. Whyte et al. [18] found that 2 weeks of very high intensity sprint interval training (6 sessions of 4 to 6 repeats of 30s Wingate with 4-5min recovery) improved insulin sensitivity, increased resting fat oxidation, and reduced waist circumference and systolic blood pressure in overweight/obese sedentary men. Trilk et al. [11] also showed that 4 weeks of HIIT (4-7×30s "all out" sprints, 4min recovery) improved circulatory function (12% increase in VO_{2max}, 11.4% increase in stroke volume, and -8.1% decreases in resting heart rate) in overweight/obese sedentary women. Even a single bout of HIIT has been shown to improve obesity-related risk factors for diabetes in obese men [19,20]. Nie et al. [19] found that a single bout of HIIT (4×30s "all out" sprints, 4min recovery) improved glucose tolerance in obese men. More recently, improvement in insulin sensitivity and fat oxidation was found in overweight/obese sedentary men after a single bout of HIIT (4×30s "all out" sprints, 4.5min recovery) [20].

Although the findings from these studies suggest that "all out" HIIT could be an effective training modality for overweight/obese individuals, the Wingate test is limited by the need for a specialized cycle ergometer and high levels of motivation [21-23]. Therefore, a modified low-volume HIIT protocol has been used by a number of studies [24-26] and is likely to be more practical for sedentary and overweight/obese populations than repeated Wingate tests. Hood et al.

[24] examined the effect of a modified protocol (10×60s at 80-95% of heart rate reserve, 60s recovery) in sedentary overweight individuals who performed six training sessions over 2 weeks and found 35% increase in insulin sensitivity at ~72 h after the final training session. Another recent study found that 6 weeks of modified HIIT (10×60s at ~90% of HR_{max}, 60s recovery) improved body composition and skeletal muscle oxidative capacity in overweight and obese women [25]. A 12-week modified HIIT program (6-10×60s at ~75-95% HR_{max}, 75s recovery) showed that HIIT improved fat oxidation in sedentary, overweight women, but did not alter body weight or body composition [26]. Therefore, these results of these studies demonstrated that HIIT based on the Wingate test can be successfully prescribed for sedentary overweight/obese individuals to enhance health.

Other studies conducted with overweight/obese populations have used a modified HIIT protocol using treadmill running. This protocol includes 1×4 min at 90% of HR_{max} [12], 4×4 min at 90% of HR_{max}, separated by 3 min at 70% [12], 4×4 min at 90% of HR_{max}, separated by 3 min at 70% [27], 4×4 min at 90% of VO_{2peak}, separated by 4 min at 60% [28], and 4×4 min at 85–95% of HR_{max}, separated by 3 min at 50–60% of HR_{max} [29]. Collectively, the potential effect suggests that HIIT is a time-efficient strategy to enhance VO_{2max} and reduce very low-density lipoprotein, blood pressure, and fasting glucose in sedentary overweight/obese individuals (Table 1).

Wingate protocol	**Results**
Nie [19]: Single bout of HIIT: (4×30s "all out" sprints, 4 min recovery), obese men (BMI= 32.0 ± 4.0 kg/ m^2)	Glucose tolerance↑
Whyte et al. [20]: Single bout of HIIT: (4×30s "all out" sprints, 4.5 min recovery), overweight/obese sedentary men (BMI= 29.9 ± 9.1 kg/ m^2)	Insulin sensitivity ↑ Fat oxidation ↑
Whyte et al. [18]: 2 wk of HIIT: (4 -6 ×30s "all out" sprints, 4-5 min recovery), overweight/obese sedentary men (BMI= 31.0 ± 3.7 kg/ m^2)	Insulin sensitivity ↑ Resting fat oxidation↑ Waist circumference↓ Systolic blood pressure↓
Trilk et al. [11] 4 wk of HIIT: (4-7×30s "all out" sprints, 4 min recovery), overweight/obese sedentary women (BMI= > 25 kg/ m^2)	VO_{2max} ↑ Stroke volume↑ Resting heart rate↓
Modified HIIT protocol	Results
Hood et al. [24]: 2 wk (cycling) of HIIT: (10×60s at 80-95% of heart rate reserve, 60s recovery), sedentary overweight individuals (BMI= 27 ± 5 kg/ m^2)	Muscle oxidative capacity↑ Glucose transporter protein content↑ Insulin sensitivity↑
Gillen et al. [25]: 6 wk (cycling) of HIIT: (10×60s at ~90% of HR_{max}, 60s recovery), overweight and obese women (BMI= 29 ± 6 kg/ m^2)	Body composition↓ Skeletal muscle oxidative capacity↑
Astorino et al. [26]: 12 wk (cycling) HIIT: (6-10×60s at ~75-95% HR_{max}, 75s recovery), sedentary, overweight women (BMI= 25.2 ± 4.3)	Fat oxidation↑ Body weight ↔ Body composition ↔
Tjonna et al. [27] 3-12 months (running) of HIIT: (6-10×60s at ~75-95% of HR_{max}, 75s recovery), overweight adolescents (BMI= 33.2 ± 6.1 kg/m^2)	BMI↓ Total fat (%)↓ VO_{2max}↑
Tsekouras et al. [28]: 2 months (running) of HIIT (4×4 min at 90% of VO_{2peak}, separated by 4 min at 60% recovery), sedentary men (BMI= 20-30 kg/m^2)	VO_{2max}↑ Body weight↔ Body composition↔ Fasting plasmaVLDL-TG↓
Schjerve et al. [29]:12 wk (walking/running) of HIIT (4×4 min at 85–95% of HR_{max}, separated by 3 min at 50–60% of HR_{max}), obese adults (BMI= 36.6 ± 1.2 kg/m^2)	Aerobic work capacity↑ Endothelial function↑ Body weight↓ Body fat (%)↓
Alahmadi [30]: 4 wk (cycling) of HIIT: (30s at 90% VO_{2max}, 30s recovery), overweight and obese men (BMI= 28.2 ± 5 kg/m^2)	Fat oxidation↑

Leggate et al. [31]: 2 wk (cycling) of HIIT: (10×4 min at 85% VO_{2peak}, 2 min recovery), overweight/obese sedentary men (BMI= 29.1 ± 3.1 kg/m^2)	VO_{2peak}↑ Waist circumference↓ Subcutaneous adipose tissue↓
Heydari et al. [13]: 12 wk (cycling) of HIIT: (8s at 80–90% HR_{2peak}, 12s recovery), overweight men (BMI= 28.4 ± 0.5 kg/m^2)	$VO2_{peak}$↑ Abdominal fat↓ Trunk fat↓ Visceral fat↓
Tjonna et al. [32]: 16 wk (walking/running) of HIIT: (4×4 min at ~90% of HR_{max}, 3 min at 70% recovery), sedentary overweight individuals with metabolic syndrome (BMI= 29.8 ± 5.5 kg/m^2)	VO_{2max}↑ Body weight↓ Waist circumference↓ Arterial blood pressure↓
Skleryk et al. [33]: 2 wk of HIIT: (8–12×10s "all out" sprints, 80s recovery), obese men (BMI= 32.8 ± 4.7 kg/m^2)	VO_{2peak}↔ Insulin sensitivity↔ Body composition↔
Tjonna et al. [12]: 10 wk (walking/running) of HIIT: (1×4 min at 90% of HR_{max}) and (4×4 min at 90% of HR_{max}, separated by 3 min at 70% recovery), overweight men (BMI= 27.4 ± 1.9 kg/m^2)	VO_{2max}↑ Blood pressure↓ Fasting plasma glucose↓

Table 1: Findings from HIIT studies conducted with overweight/obese individuals

The Effect of HIIT on Fat Oxidation

An intensity that optimizes fat oxidation as the main energy source during exercise is important. Research has shown that obese individuals have an impaired utilization of free fatty acid in the skeletal muscle [34], which is thought to be a contributing factor to the aetiology of obesity and weight gain [35]. Endurance training is an effective strategy for obesity prevention and weight loss because it enhances lipolysis and fatty acid oxidation in the skeletal muscle [36]. This enhancement is known to be intensity-dependent, as the absolute rate of fat oxidation (g.min^{-1}) increases from low to moderate intensity and then decreases as exercise becomes more intense [37-40]. However, research has shown that six sessions of HIIT over 2 weeks can increase skeletal muscle oxidative capacity and endurance performance, and alter metabolic control [14]. The impact of 6 weeks of HIIT (10×4 min at ~90% of VO_{2peak}, 2 min recovery) on fat oxidation in untrained recreationally active individuals was investigated [41]. After 6 weeks of HIIT, fat oxidation during cycling at 60% VO_{2peak} increased significantly, suggesting that high-intensity exercise session is beneficial for increasing skeletal muscle capacities to oxidize fat. Talanian et al. [42] examined the effect of seven sessions of HIIT over 2 weeks on fat oxidation during exercise in eight moderately active women and found that fat oxidation increased by 36% compared with baseline [42]. Few studies have examined the effect of HIIT on fat oxidation in overweight/obese sedentary individuals. Whyte et al. [18] found that 2 weeks of HIIT (6 sessions of 4 to 6 repeats of 30s Wingate with 4-5 min recovery) increased resting fat oxidation in overweight/obese sedentary men. Recently, a 12-week intervention of HIIT (6-10×60s at ~75-95% of HR_{max}, 75s recovery) was shown to enhance fat oxidation in sedentary overweight women [26].

In our Lab, we conducted a 4 weeks study of supervised HIIT (30s at 90% VO_{2max}, 30s recovery) for 40 min duration, three times a week in overweight and obese men [30]. HIIT improved fat oxidation (~31%) during a 30-min constant load exercise session at 45% VO_{2max}, compared with baseline. It is apparent that HIIT may be a promising strategy to improve the ability of overweight/obese men to oxidize fat. In this study, compliance with the exercise training sessions was 100% and there were no reports of injury or negative feedback about the intensity.

The mechanism behind increased fat oxidation observed following HIIT is not fully understood. This is why muscle biopsy measurement is important to understand the potential biochemical mechanisms and adaptations responsible for increase in fat oxidation after HIIT. However, an extensive overview of biochemical mechanisms that may be responsible for an increased fat oxidation is beyond the scope of this review. In brief, it is notable that fatty acid transport proteins have been linked to enhanced fat oxidation. The increases in fatty acid translocase (FAT/CD36) and plasma membrane fatty acid-binding protein ($FABP_{pm}$) both found in the sarcolemma, the mitochondrial membrane, and in a cytoplasmic pool in skeletal muscle, could contribute to the enhanced fat oxidation by increasing the rate of free fatty acid transfer across the muscle and mitochondrial membrane [41]. Six weeks of HIIT (ten 4-min cycling bouts at 90% VO_{2peak} separated by 2-min of rest) increased fatty acid transport protein content in whole muscle (FAT/CD36 and $FABP_{pm}$), sarcolemmal ($FABP_{pm}$) and mitochondrial (FAT/CD36) membranes in the skeletal muscle of 10 untrained females, suggesting that increases in skeletal muscle fatty acid oxidation following exercise training at high intensity are related in part to changes in fatty acid transport protein content [43]. Enzymes increase the skeletal muscle's fat oxidation capacity following HIIT has also been reported [42]. Talanian et al. [42] examined the effect of seven sessions of high-intensity interval training over two weeks on fat oxidation during exercise in eight moderately active women. Training sessions consisted of ten 4-min bouts at 90% of VO_{2peak}, with 2-min rest periods between intervals. Two weeks of HIIT resulted in increased fat oxidation (36%) and maximal mitochondrial enzyme activities.

In summary, these findings suggest that HIIT induces skeletal muscle metabolic adaptation and improves the skeletal muscle capacity to oxidize lipids in overweight/obese people, which has implications for health and obesity prevention and management. However, information regarding the impact HIIT, compared with steady state exercise training, on fat oxidation in obese individuals is still relatively limited and more research is needed.

The Effect of HIIT on Weight Loss

Exercise represents an important intervention for weight loss as it has the potential to reduce body mass, increase fat-free mass, and maintain or elevate resting metabolic rate [3,44,45]. A number of studies have demonstrated that HIIT may induce weight loss in sedentary overweight/obese individuals. For example, a significant reduction in waist circumference and subcutaneous adipose tissue was found after 2 weeks of HIIT in overweight/obese sedentary men [18,31]. A 12-week HIIT program also resulted in significant reductions in total abdominal, trunk, and visceral fat in overweight young males [13]. Another study found that 16 weeks of HIIT (4×4 min at ~90% of HR_{max}, 3 min at 70% recovery) reduced body weight (3%) and waist circumference (5 cm) in sedentary overweight individuals with metabolic syndrome [32].

In a longer exercise intervention study, Tjonna et al. [27] found that HIIT decreased total fat by 0.9 and 2.4 kg at 3 and 12 months respectively. A recent study with overweight/obese women found that 6-week low-volume HIIT (10×60s at ~90% HR_{max}, 60s recovery) induced significant improvements in body composition, and DEXA revealed a reduction in abdominal and whole body level adiposity and an increase in leg lean mass [25].

In contrast, two recent studies using HIIT found no significant change in weight or body composition in sedentary, overweight/obese people [26,33]. Skleryk et al. [33] conducted a 2-week study and used a HIIT protocol that included 10s "all out" cycling efforts, which may not have been sufficient to alter body composition compared with a longer 30s protocol. Astorino et al. [26] used a longer HIIT protocol (60s at ~75-95% HR_{max}, 75s recovery) and body weight did not change after 12 weeks of training. Two possible explanations for the lack of weight loss in exercise interventions are increased energy intake due to the stimulatory effect of exercise on appetite and decreased non-exercise activity thermogenesis (NEAT) to compensate for the increase in exercise-induced energy expenditure [46,47].

There are few studies that have investigated the impact of HIIT on weight and body composition in sedentary overweight/obese individuals, resulting in negligible reduction in weight loss. Although the findings from studies reviewed here suggest that HIIT may promote weight loss in this population, more research lasting at least 12 weeks is needed.

Although the mechanism responsible for fat and weight loss after HIIT is unclear, one possible reason is an increase in post-exercise metabolism [48]. Excess post-exercise oxygen consumption (EPOC) response to HIIT may have a role in elevating post-exercise fat oxidation through the increased levels of catecholamine generated during acute HIIT [48]. Bracken et al. [49] reported increased catecholamine metabolism via elevated catechol-O-methyl transferase activity during HIIT. This increase in plasma epinephrine and norepinephrine at the end of HIIT could increase lipolysis and the availability of free fatty acids, resulting in increased overall fat oxidation during and after HIIT. Moreover, HIIT significantly increases muscle mitochondrial beta-hydroxyacyl-CoA dehydrogenase, which may enhance fat loss [42]. Body weight reduction observed after HIIT may be attributed to the high exercise intensity stimulating post-exercise metabolism (i.e. EPOC), which appears to be mainly supplied by fat during recovery time [27]. The need to remove lactate and H+ and to resynthesize glycogen during and after HIIT also increases fat oxidation [7]. Collectively, HIIT-induced weight loss could be explained by an increase in the lipolytic enzymes and negative energy balance through EPOC.

Decreased post-exercise appetite is another possible mechanism underlying HIIT-induced fat loss. Although the effect of HIIT on appetite suppression has not been investigated in overweight/obese individuals, a single bout of intense exercise has been found to suppress hunger immediately following cessation of the exercise [50]. A recent study with healthy men compared the effects of HIIT (6×30s Wingate tests) and endurance exercise (60 min exercise at 68.1 % of VO_{2max}) on appetite [51]. The men reported higher appetite perceptions in the hours after an acute bout of HIIT than after the endurance exercise.

There is no clear mechanism that explains why hunger level is suppressed after high-intensity exercise. However, there is evidence to suggest a marked effect of intense exercise on subjective hunger based on the reports of exercise-induced anorexia [52]. This may be partially explained by the considerable redistribution of blood flow away from the splanchnic circulation into the working muscles [52,53]. While acute exercise reduces liver and muscle glycogen stores, which may result in an immediate increase in hunger, chronic exercise training may induce more adaptations that may lead to more stable levels of metabolic fuels, resulting in a suppression of hunger [54].

Exercise represents an important intervention for weight loss, as it has the potential to reduce body mass, increase fat-free mass, and maintain or elevate resting metabolic rate [3,44,45]. Irrespective of weight loss, regular exercise has been shown to substantially reduce total fat, visceral fat, skeletal muscle lipid, and insulin resistance in obese individuals [55,56]. High postprandial blood triglyceride levels increase cardiovascular disease risk [57-60] and, by contrast, regular exercise can reduce the risk of cardiovascular disease [61-63]. Although moderate-intensity exercise may attenuate postprandial triacylglycerol [64-66], a growing body of evidence has suggested that HIIT has a greater effect on postprandial triacylglycerol. Research has shown that acute high-intensity endurance exercise is more effective to reduce postprandial triglyceride elevation, compared with moderate-intensity exercise [67,68]. HIIT can cause a significant reduction in postprandial lipemia by creating an energy deficit [69]. For an excellent review of the role of exercise on postprandial lipemia, see Freese et al. [70]. Finally, it is important to mention that the effect of HIIT on postprandial triacylglycerol is short-lived. For example, a single session HIIT may attenuate postprandial triacylglycerol 48 h after exercise, but this effect is abolished by day 3 [71,72].

Conclusion

Study findings suggest that HIIT is an effective and appropriate exercise strategy to enhance cardiorespiratory fitness, reduce metabolic risk factors, and optimize fat oxidation and weight loss in sedentary overweight/obese populations. This has important implications for public health strategies as increasing fat oxidation may help to induce a negative energy balance and decrease body fat,

both of which are integral components of any successful weight-management program.

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