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*Review Article*

# High-Intensity Intermittent Exercise and Fat Loss

**Stephen H. Boutcher**

*School of Medical Sciences, Faculty of Medicine, University of New South Wales, Sydney, NSW 2052, Australia*

Correspondence should be addressed to Stephen H. Boutcher, s.boutcher@unsw.edu.au

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The effect of regular aerobic exercise on body fat is negligible; however, other forms of exercise may have a greater impact on body composition. For example, emerging research examining high-intensity intermittent exercise (HIIE) indicates that it may be more effective at reducing subcutaneous and abdominal body fat than other types of exercise. The mechanisms underlying the fat reduction induced by HIIE, however, are undetermined. Regular HIIE has been shown to significantly increase both aerobic and anaerobic fitness. HIIE also significantly lowers insulin resistance and results in a number of skeletal muscle adaptations that result in enhanced skeletal muscle fat oxidation and improved glucose tolerance. This review summarizes the results of HIIE studies on fat loss, fitness, insulin resistance, and skeletal muscle. Possible mechanisms underlying HIIE-induced fat loss and implications for the use of HIIE in the treatment and prevention of obesity are also discussed.

## 1. Introduction

Most exercise protocols designed to induce fat loss have focused on regular steady state exercise such as walking and jogging at a moderate intensity. Disappointingly, these kinds of protocols have led to negligible weight loss [1, 2]. Thus, exercise protocols that can be carried out by overweight, inactive individuals that more effectively reduce body fat are required. Accumulating evidence suggests that high-intensity intermittent exercise (HIIE) has the potential to be an economical and effective exercise protocol for reducing fat of overweight individuals.

HIIE protocols have varied considerably but typically involve repeated brief sprinting at an all-out intensity immediately followed by low intensity exercise or rest. The length of both the sprint and recovery periods has varied from 6 s to 4 min. Most commonly the sprints are performed on a stationary cycle ergometer at an intensity in excess of 90% of maximal oxygen uptake ($\dot{V}O_{2\,max}$). Subjects studied have included adolescents, young men and women, older individuals, and a number of patient groups [3–12]. The most utilized protocol in past research has been the Wingate test which consists of 30 s of all-out sprint with a hard resistance [13]. Subjects typically perform the Wingate test 4 to 6 times separated by 4 min of recovery. This protocol amounts to 3 to 4 min of exercise per session with each session being typically performed 3 times a week for 2 to 6 weeks. Insight into the skeletal muscle adaptation to HIIE has mainly been achieved using this type of exercise [13]; however, as this protocol is extremely hard, subjects have to be highly motivated to tolerate the accompanying discomfiture. Thus, the Wingate protocol is likely to be unsuitable for most overweight, sedentary individuals interested in losing fat. Other less demanding HIIE protocols have also been utilised. For example, we have used an 8-second cycle sprint followed by 12 s of low intensity cycling for a period of 20 min [5]. Thus, instead of 4 to 6 sprints per session, as used in Wingate protocol studies, subjects using the 8 s/12 s protocol sprint 60 times at a lower exercise intensity. Total sprint time is 8 min with 12 min of low intensity cycling. For the HIIE Wingate protocols, total exercise time is typically between 3 to 4 min of total exercise per session. Thus, one of the characteristics of HIIE is that it involves markedly lower training volume making it a time-efficient strategy to accrue adaptations and possible health benefits compared to traditional aerobic exercise programs. This review summarises results of research examining the effect of different forms of HIIE on fitness, insulin resistance, skeletal muscle, subcutaneous, and abdominal fat loss.

## 2. Acute Response and Chronic Adaptations to High-Intensity Intermittent Exercise

Acute responses to HIIE that have been identified include heart rate, hormones, venous blood glucose, and lactate levels, autonomic, and metabolic reactivity. Heart rate response is dependent on the nature of the HIIE protocol but typically is significantly elevated during exercise and declines during the period between sprint and recovery. For example, Weinstein et al. [14], using the Wingate protocol, recorded peak heart rates of 170 bpm immediately after a 30-second maximal all-out cycle sprint. Heart rate response to the 8 s/12 s protocol typically averages around 150 bpm after 5 min of HIIE which increases to 170 bpm after 15 min of HIIE [15]. In this protocol, there is typically a small heart rate decrease of between 5–8 bpm during each 12-second recovery period. A similar pattern of heart rate response was found for an HIIE protocol consisting of ten 6-second sprints interspersed with 30 s recovery. Heart rate increased to 142 bpm after the first sprint and then increased to 173 bpm following sprint ten [16].

Hormones that have been shown to increase during HIIE include catecholamines, cortisol, and growth hormones. Catecholamine response has been shown to be significantly elevated after Wingate sprints for both men and women [17, 18]. Catecholamine response to HIIE protocols that are less intensive than the Wingate protocol have also been shown to be elevated. For example, Christmass et al. [19] measured catecholamine response to long (24 s/36 s recovery) and short (6 s/9 s recovery) bout intermittent treadmill exercise and found that norepinephrine was significantly elevated postexercise. Also Trapp et al. [15] found significantly elevated epinephrine and norepinephrine levels after 20 min of HIIE cycle exercise (8 s/12 s and 12 s/24 s protocols) in trained and untrained young women. Bracken et al. [16] examined the catecholamine response of 12 males who completed ten 6-second cycle ergometer sprints with a 30-second recovery between each sprint. From baseline, plasma epinephrine increased 6.3-fold, whereas norepinephrine increased 14.5-fold at the end of sprinting (Figure 1). The significant catecholamine response to HIIE is in contrast to moderate, steady state aerobic exercise that results in small increases in epinephrine and norepinephrine [20]. The HIIE catecholamine response is an important feature of this type of exercise as catecholamines, especially epinephrine, have been shown to drive lipolysis and are largely responsible for fat release from both subcutaneous and intramuscular fat stores [21]. Significantly, more $\beta$-adrenergic receptors have been found in abdominal compared to subcutaneous fat [22] suggesting that HIIE may have the potential to lower abdominal fat stores. Aerobic endurance training increases $\beta$-adrenergic receptor sensitivity in adipose tissue [23]. Interestingly, in endurance trained women, $\beta$-adrenergic sensitivity was enhanced, whereas the sensitivity of the anti-lipolytic $\alpha_2$ receptors was diminished [24]. However, no data are available concerning HIIE training effects on $\beta$ or $\alpha_2$ adrenergic receptor sensitivity of human adipocytes.

Nevill et al. [25] examined the growth hormone (GH) response to treadmill sprinting in female and male athletes and showed that there was a marked GH response to only 30 s of maximal exercise and the response was similar for men and women but greater for sprint compared to endurance trained athletes. GH concentration was still ten times higher than baseline levels after 1 hour of recovery. Venous blood cortisol levels have also been shown to significantly increase after repeated 100 m run sprints in trained males [26], after five 15-second Wingate tests [27], and during and after brief, all-out sprint exercise in type 1 diabetic individuals [28].

Venous blood lactate response to the Wingate test protocols has typically ranged from 6 to 13 mmol·L$^{-1}$. Lactate levels after the Wingate test are typically higher in trained anaerobic athletes and have been shown to be similar [18] and lower for trained women compared to trained men [17]. Lactate levels gradually increase during longer, lower intensity HIIE protocols. Trapp et al. [15] showed that 8 s/12 s HIIE for 20 min increased plasma lactate levels between 2 and 4 mmol·L$^{-1}$ after 5 min of HIIE for both trained cyclists and untrained females. Lactate rose to between 4 and 5 mmol·L$^{-1}$after 15 min of HIIE. During a 12 s/24 s HIIE condition lactate levels of the untrained were similar but were significantly higher for the trained female cyclists (between 7 and 8 mmol·L$^{-1}$after 15 min). Despite increasing lactate levels during HIIE exercise, it appears that free fatty acid transport is also increased. For example, a 20-minute bout of 8 s/12 s HIIE produced increased levels of glycerol indicating increased release of fatty acids [15] which peaked for untrained women after 20 min and after 10 min of HIIE for trained women.

HIIE appears to result in significant increases in blood glucose that are still elevated 5 min [29] and 30 min postexercise [18]. HIIE appears to have a more dramatic effect on blood glucose levels of exercising type 1 diabetic individuals. Bussau et al. [28] examined the ability of one 10-second maximal sprint to prevent the risk of hypoglycemia typically experienced after moderate aerobic exercise in type I diabetics. Twenty minutes of moderate-intensity aerobic exercise resulted in a significant fall in glycemia. However, one 10-second sprint at the end of the 20-minute aerobic exercise bout opposed a further fall in glycemia for 120 minutes, whereas in the absence of a sprint, glycemia decreased further after exercise. The stabilization of glycemia in the sprint trials was associated with elevated levels of catecholamines, growth hormone, and cortisol. In contrast, these hormones remained at near baseline levels after the 20 min of aerobic exercise. Thus, one 10-second all out sprint significantly increased glucose, catecholamines, growth hormone, and cortisol of type 1 diabetic individuals for 5 min after HIIE. Authors suggest that the addition of one 10-second sprint after moderate intensity aerobic exercise can reduce hypoglycemia risk in physically active individuals who possess type 1 diabetes.

Autonomic function has been analyzed after HIIE by assessing heart rate variability. Parasympathetic activation was found to be significantly impaired in a 10-minute recovery period after repeated sprint exercise [30] and a 1-hour recovery period [31] in trained subjects. Buchhiet et al. [30] have suggested that parasympathetic or vagal impairment is caused by the heightened sympathetic activity

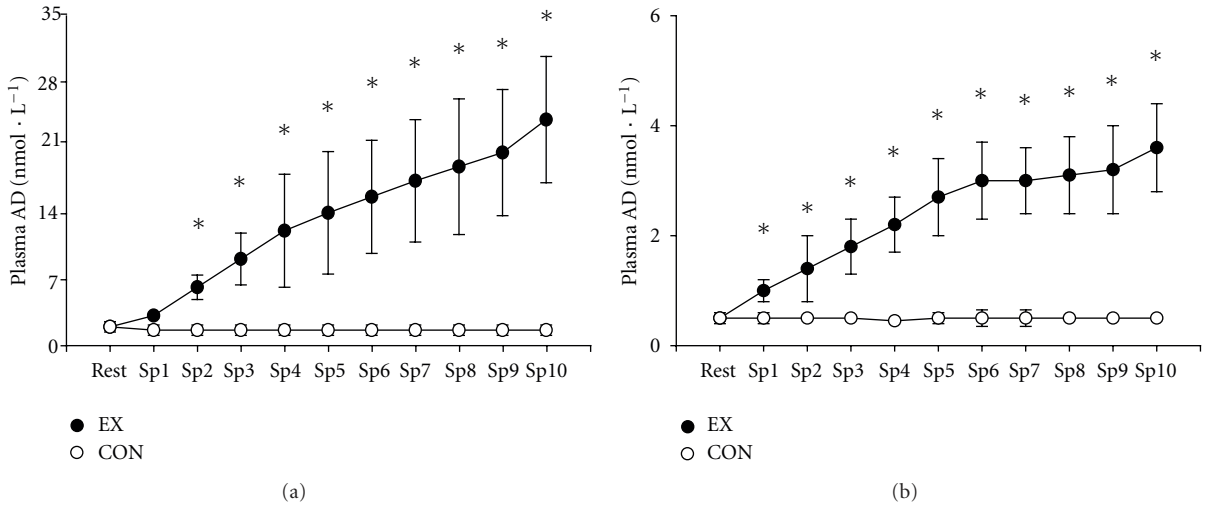


Figure 1: Plasma noradrenaline (NA) and adrenaline (AD) concentration of subjects at rest (CON) and following each 6-second sprint (EX) (mean ± SD, $n = 12$). *Indicates a significant difference from equivalent CON value ($P < .05$). (Adapted from Bracken et al. [16]).

that occurs during HIIE exercise and the persistent elevation of adrenergic factors and local metabolites during recovery (e.g., epinephrine, norepinephrine, and venous blood lactate).

With regard to metabolic response, HIIE initially results in decreased adenosine triphosphate (ATP) and phosphocreatine (PCr) stores followed by decreased glycogen stores [32] through anaerobic glycolysis [33]. Gaitanos et al. [29] have suggested that towards the end of an HIIE session, which consists of numerous repeat sprints (e.g., ten 6-second bouts of maximal sprinting), an inhibition of anaerobic glycogenolysis may occur. These authors have further suggested that at the end of the HIIE bout, ATP resynthesis may be mainly derived from PCr degradation and intramuscular triacylglycerol stores. However, this pattern of fuel utilization during HIIE has not been demonstrated in humans. After hard, all-out HIIE exercise, complete phosphagen recovery may take 3-4 min but complete restoration of pH and lactate to pre-exercise levels may take hours [33]. The recovery of the exercising muscle after HIIE to its pre-exercise state is undetermined. After a hard bout of aerobic exercise, recovery has typically been found to be biphasic with an initial rapid phase of recovery lasting 10 s to a few minutes followed by a slower recovery phase lasting from a few minutes to hours [33]. During recovery, oxygen consumption is elevated to help restore metabolic processes to baseline conditions. The postexercise oxygen uptake in excess of that required at rest has been termed excess postexercise oxygen consumption (EPOC). EPOC during the slow recovery period has been associated with the removal of lactate and $H^+$, increased pulmonary and cardiac function, elevated body temperature, catecholamine effects, and glycogen resynthesis [33]. Although EPOC does not appear to have been assessed after HIIE, it is enhanced after split aerobic exercise sessions. For example, magnitude of EPOC was significantly greater when 30-minute [34] and 50-minute [35] aerobic exercise sessions were divided into two parts. Also an exponential relationship between aerobic exercise intensity and EPOC magnitude has been demonstrated [36]. With regard to HIIE, it is feasible that the significant increase in catecholamines (Figure 1) and the accompanying glycogen depletion described earlier could induce significant EPOC. However, aerobic exercise protocols resulting in prolonged EPOC have shown that the EPOC comprises only 6–15% of the net total exercise oxygen cost [36]. Laforgia et al. [36] have concluded that the major impact of exercise on body mass occurs via the energy expenditure accrued during actual exercise. Whether HIIE-induced EPOC is one of the mechanisms whereby this unique form of exercise results in fat loss needs to be determined by future research. In summary, acute responses to a bout of HIIE include significant increases in heart rate, catecholamines, cortisol, growth hormone, plasma lactate and glucose levels, glycerol, and a significant decrease in parasympathetic reactivation after HIIE, and depletion of ATP, PCr, and glycogen stores.

Chronic responses to HIIE training include increased aerobic and anaerobic fitness, skeletal muscle adaptations, and decreased fasting insulin and insulin resistance (Table 1). Surprisingly, aerobic fitness has been shown to significantly increase following minimal bouts of HIIE training. For example, Whyte et al. [45] carried out a 2-week HIIE intervention with three HIIE sessions per week consisting of 4 to 6 Wingate tests with 4 min of recovery. Previously, untrained males increased their $\dot{V}O_{2\,max}$ by 7%. Increases in $\dot{V}O_{2\,max}$ of 13% for an HIIE program also lasting 2 weeks have been documented [42]. HIIE protocols lasting 6 to 8 weeks have produced increases in $\dot{V}O_{2\,max}$ of 4% [37] and 6–8% [39]. Longer Wingate-type HIIE programs lasting 12 to 24 weeks have recorded large increases in $\dot{V}O_{2\,max}$ of 41% [40] and 46% [6] in type 2 diabetic and older cardiac rehabilitation patients. The less intense protocols (8 s/12 s) coupled with longer duration conducted over 15 and 12 weeks resulted in a 24% [5] and 18% increase [46] in $\dot{V}O_{2\,max}$. Collectively, these results indicate that participation

Table 1: Effect of high-intensity intermittent exercise on subcutaneous and abdominal fat, body mass, waist circumference, $\dot{V}O_{2\,max}$, and insulin sensitivity.

| Study | Subcutaneous fat (kg) | Abdominal/ trunk fat (kg) | Body mass (kg) | Waist circumference (cm) | Type of HIIE | Length of intervention | $\dot{V}O_{2\,max}$ ml·kg·min$^{-1}$ | Insulin sensitivity |
|---|---|---|---|---|---|---|---|---|
| Boudou et al. [8] | ⇓ 18% | ⇓ 44% | ⇓ 1.9 kg (2%) | — | SSE + 5 × 2/3 min R | 8 weeks | — | ⇑ 58% |
| Burgomaster et al. [37] | — | — | ⇔ | — | 4–6 Wingate/4.5 min R | 6 weeks | ⇑ 7% | — |
| Dunn [46] | ⇓ 2.6 kg (8%) | ⇓ .12 kg (6%) | ⇓ 1.9 kg (3%) | ⇓ 3.5 cm (4%) | 60 × 8 s/12 s R | 12 weeks | ⇑ 18% | ⇑ 36% |
| Helgerud et al. [39] | — | — | ⇓ .8 kg (1%) | — | 15 s/15 s R | 8 weeks | ⇑ 6% | — |
| Helgerud et al. [39] | — | — | ⇓ 1.5 kg (2%) | — | 4 × 4 min/4 min R | 8 weeks | ⇑ 7% | — |
| Mourier et al. [40] | ⇓ 18% | ⇓ 48% | ⇓ 1.5 kg (2%) | ⇓ 1.00 cm (1%) | SSE + 5 × 2/3 min R | 8 weeks | ⇑ 41% | ⇑ 46% |
| Perry et al. [41] | — | — | ⇓ .2 kg (.03%) | — | 10 × 4 min/2 min R | 2 weeks | ⇑ 9% | — |
| Talanian et al. [42] | — | — | — | — | 10 × 4 min/2 min R | 2 weeks | ⇑ 13% | — |
| Tjønna et al. [43] | — | — | ⇓ 2.3 kg (2.5%) | ⇓ 5.0 cm (5%) | 4 × 4 min/3 min R | 16 weeks | ⇑ 26% | ⇑ 19% |
| Tjønna et al. [3] | ⇓ 2.4 kg (7%) | ⇓ 1.5 kg (8%)# | ⇑ .1 kg (.3%) | ⇓ 7.2 cm (7%) | 4 × 4 min/3 min R | 12 weeks | ⇑ 10% | ⇑ 29% |
| Trapp et al. [5] | ⇓ 2.5 kg (10%) | ⇓ .15 kg (10%)# | ⇓ 1.51 kg (2%) | — | 60 × 8 s/12 s R | 15 weeks | ⇑ 24% | ⇑ 33% |
| Tremblay et al. [38] | ⇓ 15%* | ⇓ 12%* | ⇓ .1 kg (.1%) | — | 15 × 30 s | 24 weeks | ⇑ 20% | — |
| Warburton et al. [44] | — | — | ⇓ 3.0 kg (4%) | — | 7 × 2 min/2 min R | 16 weeks | ⇑ 10% | — |
| Whyte et al. [45] | — | — | ⇓ 1.0 kg (1%) | ⇓ 2.4 cm (2%) | 4–6 Wingate/4.5 min R | 2 weeks | ⇑ 9% | ⇑ 25% |

Note: ⇑ indicates increased; ⇓ decreased; ⇔no change; —not recorded; *body fat was assessed by skin folds; # trunk fat; SSE = steady state exercise; Wingate = 30 s flat out sprint; R = recovery.

in differing forms of HIIE by healthy young adults and older patients, lasting from 2 to 15 weeks, results in significant increases in $\dot{V}O_{2\,max}$ from between 4% to 46% (Table 1). Mechanisms underlying the aerobic fitness response to HIIE are unclear although a major contributor is phosphocreatine degradation during repeated HIIE. Using thigh cuff occlusion to prevent PCr resynthesis during recovery, Trump et al. [47] showed that PCr contributed approximately 15% of the total ATP provision during a third 30-second bout of maximal isokinetic cycling. Muscle glycogenolysis made

a minor contribution to ATP provision during the third 30-second bout indicating that aerobic metabolism was the major source of ATP during repeated sprinting. Similarly, Putman et al. [48] showed that repeated bouts of HIIE resulted in a progressive increase in ATP generation so that by the third out of five 30-second Wingate bouts, the majority of ATP was generated oxidatively.

Other mechanisms underlying the HIIE increase in aerobic power are undetermined but may involve increased stroke volume induced by enhanced cardiac contractility [39], enhanced mitochondrial oxidative capacity, and increased skeletal muscle diffusive capacity [10]. There is also evidence indicating that muscle aerobic capacity is increased following HIIE due to increases in PGC-1a mediated transcription [49] occurring via AMPK activation ¡?bhlt?¿[50] . Harmer et al. [7] have suggested that these marked oxidative adaptations in the exercising muscle are likely to underlie the significant increases in peak and maximal oxygen uptake documented after regular HIIE.

Anaerobic capacity response to HIIE has typically been assessed by measuring blood lactate levels to a standardized exercise load or anaerobic performance on a Wingate test. A number of studies have demonstrated that HIIE lasting from 2 to 15 weeks results in significant increases in anaerobic capacity from between 5% to 28%. For example, Tabata et al. [51] used a 20 s/10 s protocol and found that in previously untrained males, anaerobic capacity, measured by maximal accumulated $O_2$ deficit, was increased by 28%. Whyte et al. [45] carried out a 2-week HIIE intervention and found that previously untrained males increased their anaerobic capacity by 8%, whereas Burgomaster et al. [32] found that Wingate test performance was increased by 5.4% after two weeks of HIIE.

A number of studies have taken muscle biopsies after Wingate test performance in order to examine skeletal muscle adaptations. In a series of studies, Gibala et al. [13, 52] have consistently found increased maximal activity and protein content of mitochondrial enzymes such as citrate synthase and cytochrome oxidase after HIIE training. For example, Talanian et al. [42] carried out an HIIE intervention that consisted of 2 weeks of HIIE exercise performed seven times with each session consisting of ten 4-minute bouts at 90% $\dot{V}O_{2\,max}$ separated by 2-minute resting intervals. $\dot{V}O_{2\,max}$ was increased by 13% and plasma epinephrine and heart rate were lower during the final 30 min of a 60-minute cycling steady state exercise trial at 60% of pretraining $\dot{V}O_{2\,max}$. Exercise whole body fat oxidation also increased by 36%, and net glycogen use was reduced during the steady state cycling trial. HIIE significantly increased muscle $\beta$ hydroxyacyl coenzyme A dehydrogenase and citrate synthase. Total muscle plasma membrane fatty acid binding protein content also increased significantly after HIIE. Thus, seven sessions of HIIE, over two weeks, induced marked increases in whole body and skeletal muscle capacity for fatty acid oxidation during exercise in moderately active women. Other studies have found similar results with studies reporting large increases in citrate synthase maximal activity after 2 weeks [32] and 6 weeks of HIIE [37]. Similarly, $\beta$ hydroxyacyl coenzyme A dehydrogenase activity, which catalyzes a key rate-limiting enzyme step in fat oxidation, also significantly increased after HIIE training [38]. Increases in oxidative muscle metabolism (e.g., hexokinase and citrate synthase activity) after 7 weeks of HIIE training with type 1 diabetic individuals have also been documented [7]. Collectively, markers of muscle oxidative capacity have been shown to significantly increase after six sessions of HIIE lasting as little as 2 weeks. Glycolytic enzyme content and activity has also been shown to increase after exposure to HIIE. Tremblay et al. [38] have shown that 16 weeks of HIIE significantly increased phosphofructokinase levels which is a key rate limiting enzyme in glycolysis, whereas Macdougall et al. [53] also showed increases in phosphofructokinase with a Wingate-type protocol carried out for 7 weeks. In summary, Wingate test HIIE protocols of between one and seven weeks have demonstrated marked increases in skeletal muscle capacity for fatty acid oxidation and glycolytic enzyme content and activity.

The effect of HIIE training on fasting insulin and insulin resistance is shown in Table 1. As can be seen all studies that have assessed insulin response to HIIE have recorded significant improvements of between 23% and 58% increase in insulin sensitivity. Insulin sensitivity has typically been assessed by measuring fasting insulin, HOMA-IR, and by glucose tolerance tests. In healthy, nondiabetic individuals, the improvement in fasting insulin and insulin resistance has ranged from 23% to 33% [37, 39, 42, 45], whereas in individuals possessing type 2 diabetes, two studies have reported greater insulin sensitivity improvements of 46% [40] and 58% [8]. Babraj et al. [4] used a glucose tolerance test to assess insulin sensitivity after an intervention that consisted of 2 weeks of HIIE performed three times per week with each session consisting of four to six 30-second all out sprints separated by resting interval of between 2 to 4 min. Glucose (12%) and insulin areas under the curve (37%) were significantly attenuated with a sustained improved insulin action until at least three days after the last exercise session. This was achieved without a change in body weight and with a total exercise energy increase of only 500 kcal for the two weeks. Authors suggest that the small increase in energy expenditure contrasts to the 2000–3000 kcal per week experienced during a typical aerobic training program. The mechanism(s) underlying these large increases in insulin sensitivity reported in these studies is likely due to the skeletal muscle adaptations previously discussed involving marked increases in skeletal muscle capacity for fatty acid oxidation and glycolytic enzyme content [25]. In summary, chronic exposure to HIIE results in significant increases in aerobic and anaerobic fitness, increased skeletal muscle capacity for fatty acid oxidation and glycolytic enzyme content, and increased insulin sensitivity.

## 3. High-Intensity Intermittent Exercise and Fat Loss

The majority of research examining HIIE has focused on short-term (2 to 6 weeks) programs on skeletal muscle adaptation [13]. However, some studies have utilized longer

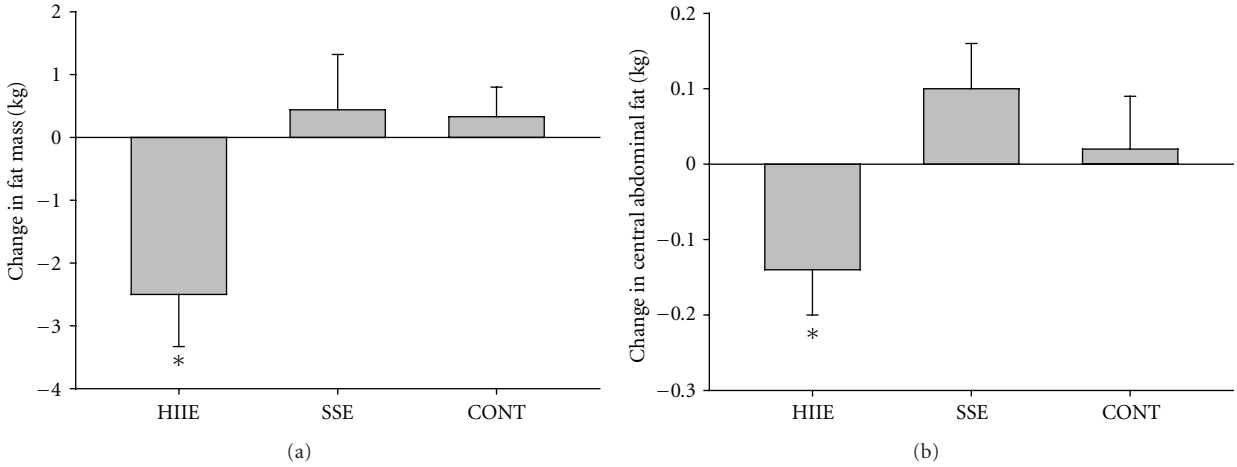


Figure 2: Subcutaneous (a) and abdominal fat loss (b) after 15 weeks of high-intensity intermittent exercise. HIIE: high-intensity intermittent exercise, SSE: steady state exercise, Cont: control. *Significantly different from control and SSE groups ($P < .05$). (Adapted from Trapp et al. [5]).

programs to determine the effect of HIIE on subcutaneous and abdominal fat loss. For example, Tremblay et al. [38] compared HIIE and steady state aerobic exercise and found that after 24 weeks subjects in the HIIE group lost more subcutaneous fat, as measured by skin folds, compared to a steady state exercise group when exercise volume was taken into account (Table 1). More recently, Trapp et al. [5] conducted an HIIE program for 15 weeks with three weekly 20-minute HIIE sessions in young women. HIIE consisted of an 8-second sprint followed by 12 s of low intensity cycling. Another group of women carried out an aerobic cycling protocol that consisted of steady state cycling at 60% $\dot{V}O_{2\,max}$ for 40 min. Results showed that women in the HIIE group lost significantly more subcutaneous fat (2.5 kg) than those in the steady state aerobic exercise program (Figure 2(a)). Dunn [46] used a similar HIIE protocol together with a fish oil supplementation and a Mediterranean diet for 12 weeks. In 15 overweight young women, the combination of HIIE, diet, and fish oil resulted in a 2.6 kg reduction in subcutaneous fat (8%) and a 36% increase in insulin sensitivity (Table 1). The amount of subcutaneous fat lost was similar to that observed in the Trapp et al. [5] study suggesting that shorter HIIE interventions (12 versus 15 weeks) are also effective for reducing subcutaneous fat.

With regard to abdominal fat, Trapp et al. [5] found that 15 weeks of HIIE led to significantly reduced abdominal fat (.15 kg) in untrained young women (Figure 2(b)), whereas Dunn [46] found that 12 weeks of HIIE led to a.12 kg decrease in abdominal fat. As women in these studies possessed relatively low abdominal fat levels, it is possible that the greater abdominal fat of men may demonstrate greater reductions after HIIE. For example, Boudou et al. [8], in a study involving older type 2 diabetic males, found that after 8 weeks of HIIE no change in body mass occurred; however, abdominal adiposity was decreased by 44% (Table 1). Mourier et al. [40] found a 48% reduction in visceral fat, measured by MRI, compared to an 18% decrease in subcutaneous fat following an exercise regimen consisting of steady state exercise two days per week and HIIE one day a week for 8 weeks in type 2 diabetic men and women. Tjønna et al. [3] examined 32 middle-aged metabolic syndrome men and women who performed 16 weeks of HIIE three times per week. $\dot{V}O_{2\,max}$ increased by 26% and body weight was reduced by 2.3 kg. Whyte et al. [45] examined ten overweight males aged 32 years after two weeks of HIIE consisting of 6 sessions of a 4–6 repeats of a Wingate test. $\dot{V}O_{2\,max}$ increased (8%) and significant change in waist circumference was also found (Table 1). Although the effects of HIIE on fat free mass has not been extensively examined, one study using DEXA found that trunk muscle mass was significantly increased after 15 weeks [5], whereas another study using MRI showed a significant 24% increase in thigh muscle cross sectional area after HIIE [8].

A summary of the results of studies examining the effects of HIIE on subcutaneous and abdominal fat, body mass, and waist circumference is illustrated in Table 1. As can be seen studies that carried out relatively brief HIIE interventions (2 to 6 weeks) only resulted in negligible weight loss. However, the majority of subjects in these short-term Wingate test studies have been young adults with normal BMI and body mass. Studies that used longer duration HIIE protocols with individuals possessing moderate elevations in fat mass [5] have resulted in greater weight/fat reduction. Interestingly, the greatest HIIE-induced fat loss was found in two studies that used overweight type 2 diabetic adults (BMI > 29 kg/m$^2$) as subjects [8, 40]. Given that greater fat loss to exercise interventions has been found for those individuals possessing larger initial fat mass [54], it is feasible that HIIE will have a greater fat reduction effect on the overweight or obese. Thus, more studies examining the effects of HIIE on obese or overweight individuals are needed.

Possible mechanisms underlying the HIIE-induced fat loss effect include increased exercise and postexercise fat oxidation and decreased postexercise appetite. As mentioned, Gaitanos et al. [29] have suggested that towards the end of an HIIE session that consists of numerous repeat

sprints (e.g., ten 6-second bouts of maximal sprinting) an inhibition of anaerobic glycogenolysis occurs and ATP resynthesis is mainly derived from PCr degradation and intramuscular triacylglycerol stores. That increased venous glycerol accompanied HIIE in both trained female cyclists and untrained women [15] supports the notion that acute HIIE progressively results in greater fatty acid transport. Also Burgomaster et al. [55] and Talanian et al. [42] have shown that 6 to 7 sessions of HIIE had marked increases in whole body and skeletal muscle capacity for fatty acid oxidation.

As mentioned previously, the EPOC or postexercise response to HIIE does not appear to have been examined. It is feasible that the catecholamines generated by HIIE (Figure 1) could influence postexercise fat metabolism. Increased fat oxidation after HIIE may also occur as a result of the need to remove lactate and $H^+$ and to resynthesize glycogen. The elevated GH levels documented after a bout of HIIE [25] may also contribute to increased energy expenditure and fat oxidation.

It is also feasible that HIIE may result in suppressed appetite. In rats, hard exercise has been repeatedly reported to reduce food intake [56]. The mechanisms underlying the anorectic effects of exercise are not known but exercise may reduce food intake by facilitating the release of corticotropin releasing factor (CRF) a potent anorectic peptid [56]. It has been shown that hard running and swimming exercise results in elevated levels of CRF in rats [57, 58] and increases in indirect markers of CRF in humans [59]. Rivest and Richard [57] and Kawaguchi et al. [58] showed that injecting a corticotropin-releasing factor (CRF) antagonist into the hypothalamus of rats prevented the effects of exercise on food intake and body weight reduction suggesting that CRF plays a major role in the anorexia caused by exercise in rats. Bi et al. [59] also provided evidence to support the importance of CRF in mediating the long-term effects of exercise on food intake and body weight in rats. Human studies also show a considerable decrease in subjective hunger after intensive aerobic exercise [56]. However, this exercise-induced anorexia has been observed only for a short time after hard exercise (>60% $\dot{V}O_{2\,max}$). Mechanisms underlying this effect in humans are undetermined but could include the CRF peptide effect previously discussed and an exercise-induced redistribution of splanchnic blood flow. For example, a 60%–70% decrease in splanchnic blood flow in humans exercising at 70% $\dot{V}O_{2\,max}$ has been documented [60] and at maximal exercise splanchnic blood flow is reduced by approximately 80% [61]. In summary, there is evidence to suggest that regular HIIE results in increased fat oxidation during exercise; however, the effects of HIIE on postexercise fat oxidation and appetite suppression have not been examined.

## 4. Conclusions and Clinical Implications

Research examining the effects of HIIE has produced preliminary evidence to suggest that HIIE can result in modest reductions in subcutaneous and abdominal body fat in young normal weight and slightly overweight males and females. Studies using overweight male and female type 2 diabetic individuals have shown greater reductions in subcutaneous and abdominal fat. The mechanisms underlying the fat reduction induced by HIIE, however, are undetermined but may include HIIE-induced fat oxidation during and after exercise and suppressed appetite. Regular HIIE has been shown to significantly increase both aerobic and anaerobic fitness and HIIE also significantly lowers insulin resistance and results in increases in skeletal muscle capacity for fatty acid oxidation and glycolytic enzyme content.

Some important issues for future HIIE research include optimization of type and nature of HIIE protocols, individual fat loss response to HIIE, and suitability of HIIE for special populations. The most utilized protocol has been the Wingate test (30 s of all-out sprint). This protocol amounts to 3 to 4 min of cycle exercise per session with each session being typically performed 3 times a week. This protocol, although remarkably short in duration, is extremely hard and subjects have to tolerate significant discomfiture. Thus, the Wingate protocol is likely to be unsuitable for most overweight, sedentary individuals interested in losing fat. Other less demanding HIIE protocols have included an 8-second cycle sprint followed by 12 s of low intensity cycling for a period of 20 min [5], a 15-second cycle sprint followed by 15 s of low intensity cycling for a period of 20 min [45], and a 2-minute cycle sprint followed by 3 min of low intensity cycling for a period of 20 min [8]. A challenge for future research is to identify the minimal dose of HIIE for the maximum health benefit. As discussed earlier, reducing the length of HIIE training from 15 to 12 weeks still resulted in significant subcutaneous and abdominal fat loss [46]. Thus, more research is needed to identify the optimal length and intensity of the HIIE protocol for achieving varying health outcomes.

With regard to modality, studies have primarily utilized a stationary cycle ergometer, thus, little is known about the effects of other potential HIIE modalities such as rowing, walking, running, stair climbing, and swimming. That insulin resistance has recently been shown to primarily be located in leg muscle [62] suggests that HIIE exercise that focuses on the legs is likely to show the greatest insulin sensitivity increases. How leg muscle adaptations to HIIE impact on subcutaneous and abdominal fat loss and other health markers compared to other regional adaptations is undetermined.

It is unclear if the increase in insulin sensitivity following HIIE training is simply a response to the last exercise session or a result of more permanent skeletal muscle adaptations. Whyte et al. [45] have provided evidence to suggest that for short-term HIIE training of two weeks, the increase in insulin sensitivity was largely a result of the last HIIE session. They assessed insulin resistance 24 hours and 72 hours after the sixth HIIE session in a two-week training program. Insulin sensitivity had increased by 25% at 24 hours after HIIE but had returned to preintervention levels after 72 hours. In contrast to these results, Babraj et al. [4] used a glucose tolerance test to assess insulin sensitivity after a similar intervention and found that insulin sensitivity was improved until at least three days after the last exercise session. Why these similar HIIIE protocols produced differing results

differ is not clear and also whether HIIE programs lasting longer than two weeks display a similar effect has not been established.

Individual variability in fat loss to HIIE and other forms of exercise is an important issue for future research. For example, in the intervention previously described [5] there were significant individual differences in the fat loss response to HIIE. Fat response ranged from a loss of 8 kg to a gain of .10 kg. If fat loss responders alone were examined in this study (women who lost rather than gained fat), then average fat loss was 3.94 kg. As there are likely to be responders and nonresponders in every exercise, fat loss trial calculating mean fat loss alone hides the significant fat loss achieved by some individuals. Thus, it is feasible that HIIE fat loss programs are effective for producing a clinical decrease in fat (greater than 6% of fat mass) for some but not all participants. Boutcher and Dunn [63] have highlighted a range of program design factors and individual factors that are behavioral, inherited, and physiological in origin that may affect individual fat loss response to exercise. Therefore, research is needed to identify the major individual factors that both enhance and impede fat loss response to HIIE-based interventions.

A small number of studies have examined the effects of HIIE fat loss and health of special populations and patients. These have included overweight adolescents [3], older adults [6], type 1 [7] and type 2 diabetic individuals [8], paraplegics [9], intermittent claudication [10], chronic obstructive pulmonary disease [11], and cardiac rehabilitation patients [12]. Encouragingly, these studies have shown that HIIE appears to be both safe and beneficial for these patient groups. Future research needs to establish the most beneficial HIIE protocol that is both optimal and sustainable for different types of patients.

In conclusion, regular HIIE produces significant increases in aerobic and anaerobic fitness and brings about significant skeletal muscle adaptations that are oxidative and glycolytic in nature. HIIE appears to have a dramatic acute and chronic effect on insulin sensitivity. The effects of HIIE on subcutaneous and abdominal fat loss are promising but more studies using overweight individuals need to be carried out. Given that the major reason given for not exercising is time [64], it is likely that the brevity of HIIE protocols should be appealing to most individuals interested in fat reduction. The optimal intensity and length of the sprint and rest periods together with examination of the benefits of other HIIE modalities need to be established.

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