# Effects of high-intensity interval training on cardiometabolic health: a systematic review and meta-analysis of intervention studies

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## ABSTRACT

The current review clarifies the cardiometabolic health effects of high-intensity interval training (HIIT) in adults. A systematic search (PubMed) examining HIIT and cardiometabolic health markers was completed on 15 October 2015. Sixty-five intervention studies were included for review and the methodological quality of included studies was assessed using the Downs and Black score. Studies were classified by intervention duration and body mass index classification. Outcomes with at least 5 effect sizes were synthesised using a random-effects meta-analysis of the standardised mean difference (SMD) in cardiometabolic health markers (baseline to postintervention) using Review Manager 5.3. Short-term (ST) HIIT (<12 weeks) significantly improved maximal oxygen uptake ($VO_2$ max; SMD 0.74, 95% CI 0.36 to 1.12; p<0.001), diastolic blood pressure (DBP; SMD −0.52, 95% CI −0.89 to −0.16; p<0.01) and fasting glucose (SMD −0.35, 95% CI −0.62 to −0.09; p<0.01) in overweight/obese populations. Long-term (LT) HIIT (≥12 weeks) significantly improved waist circumference (SMD −0.20, 95% CI −0.38 to −0.01; p<0.05), % body fat (SMD −0.40, 95% CI −0.74 to −0.06; p<0.05), $VO_2$ max (SMD 1.20, 95% CI 0.57 to 1.83; p<0.001), resting heart rate (SMD −0.33, 95% CI −0.56 to −0.09; p<0.01), systolic blood pressure (SMD −0.35, 95% CI −0.60 to −0.09; p<0.01) and DBP (SMD −0.38, 95% CI −0.65 to −0.10; p<0.01) in overweight/obese populations. HIIT demonstrated no effect on insulin, lipid profile, C reactive protein or interleukin 6 in overweight/obese populations. In normal weight populations, ST-HIIT and LT-HIIT significantly improved $VO_2$ max, but no other significant effects were observed. Current evidence suggests that ST-HIIT and LT-HIIT can increase $VO_2$ max and improve some cardiometabolic risk factors in overweight/obese populations.

## INTRODUCTION

The WHO[1] and the American College of Sports Medicine (ACSM)[2] recommend at least 150 min of moderate-intensity physical activity (40–60% maximum oxygen uptake ($VO_2$ max)[3]) or 75 min of vigorous-intensity physical activity (60–85% $VO_2$ max) per week for healthy adults to maintain or improve health. Despite the established therapeutic potential of moderate-intensity to vigorous-intensity physical activity, 31.1% of the adult worldwide (43% US population) fails to meet the minimum physical activity guidelines.[4] Frequently cited barriers to engagement in physical activity are lack of time, low motivation and poor adherence.[5 6] To this end, several investigators[7–9] have examined the efficacy of high-intensity interval training (HIIT; ≥85% $VO_2$ max[3]) to maintain or improve health as an alternative to longer duration, continuous, moderate-intensity to vigorous-intensity physical activity approaches recommended by the WHO and ACSM. One of the primary advantages of HIIT, compared to lesser-intensity exercise, is that HIIT requires less time be spent exercising, while providing similar or greater health-related benefits, compared to established physical activity recommendations.[10 11] As a result, it has been theorised that HIIT can mitigate the most commonly cited barrier to physical activity which is 'lack of time'.[5 6]

Interval training refers to intermittent exercise that involves alternating short bursts of higher-intensity activity with lower-intensity activity for recovery or rest.[3 12] HIIT is an enhanced form of interval training involving brief, high-intensity, anaerobic exercise (ranging from 85% to 250% $VO_2$ max for 6 s to 4 min) separated by brief, but slightly longer bouts of low-intensity aerobic rest (ranging from 20% to 40% $VO_2$ max for 10 s to 5 min).[13] Numerous studies have demonstrated greater health-related benefits from HIIT compared to traditional moderate-intensity continuous training (MICT).[14–17] Compared with MICT, HIIT has been reported to more effectively increase aerobic capacity ($VO_2$ max)[14–17] and reduce risk factors associated with metabolic syndrome, including blood pressure (BP),[17] insulin action[16] and lipogenesis,[16] in a variety of patient populations. Recent systematic reviews and meta-analyses have reported HIIT, compared to MICT, can significantly increase peak oxygen uptake in individuals with lifestyle-induced cardiometabolic diseases[18] and to stimulate modest improvements in $VO_2$ max compared to pretraining values in active non-athletic and sedentary individuals.[19]

As such, HIIT is a promising method by which to reduce cardiometabolic risk factors.[20] However, reviews of HIIT, compared to MICT, to date have focused on cardiorespiratory fitness[18 19 21] and vascular function.[22] A single meta-analysis[23] of six studies examined the effect of HIIT on traditional cardiovascular disease (CVD) risk factors in individuals with cardiometabolic disorders and found HIIT and MICT to have similar effects on metabolic risk factors (body composition, BP, lipid profile and glucose). However, no review, to date, has sought to provide an extensive review of the effect of HIIT on traditional and novel markers of

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CVD risk factors such as inflammation. The need for a systematic review/meta-analysis examining the efficacy of HIIT on markers of cardiometabolic health is particularly relevant as a growing number of individuals who develop CVD, despite the absence of traditional CVD risk factors (ie, hypertension, elevated blood glucose and high cholesterol).[24] Additionally, the higher exertion and unique pattern of HIIT may induce changes in novel markers of CVD risk. For example, high-intensity exercise has been shown to reduce disease-related inflammation (interleukin 6 (IL-6) and tumour necrosis factor (TNF)-α) in animals.[25] Therefore, to further examine the potential benefit of HIIT, this study will include novel markers of CVD risk which previous reviews have not examined.

Therefore, we conducted a systematic review and meta-analysis to synthesise the effects of HIIT on cardiometabolic health markers, including body mass, waist circumference (WC), hip circumference (HC), body mass index (BMI), waist-to-hip ratio (WHR), % body fat, resting heart rate (HR), BP, $VO_2$ max, fasting glucose, glycosylated haemoglobin, insulin, total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides, C reactive protein (CRP), IL-6, TNF-α, TNF receptor-1 (TNFR1) and TNF receptor-2 (TNFR2) in adults.

## METHODS

A systematic search of clinical trials, randomised controlled trials, controlled clinical trials and comparative studies was initially performed on 25 March 2015 and completed on 15 October 2015 based on the PRISMA guidelines[26] following the protocol of a systematic review on physical activity.[27] Articles were retrieved from PubMed using the following search criteria: (high intensity interval training OR high-intensity interval training OR high intensity interval exercise OR high-intensity interval exercise OR high intensity intermittent exercise OR high-intensity intermittent exercise OR sprint interval training OR HIIT OR HIIE) AND (fasting plasma glucose OR glycosylated hemoglobin OR HbA1c OR triglycerides OR insulin OR total cholesterol OR LDL cholesterol OR HDL cholesterol OR CRP OR C-reactive protein OR IL-6 OR interleukin-6 OR TNF-alpha OR TNF receptor-1 OR TNFR1 OR TNF receptor-2 OR TNFR2 OR 'body mass index' OR BMI OR waist circumference OR hip circumference OR waist-to-hip ratio OR resting heart rate OR per cent body fat OR lean body mass OR resting blood pressure OR maximum heart rate OR $VO_2$ max) AND Humans[MeSH] AND Adult[MeSH] AND English[lang].

Initially, titles and abstracts of identified articles were checked for relevance by two reviewers (RBB and PST). Subsequently, the reviewers independently reviewed the full text of potentially eligible papers. Any disagreement between the reviewers for inclusion was resolved through discussion. Additional articles were identified via hand-searching and reviewing the reference lists of relevant papers. Figure 1 presents the flow of papers through the study selection process.

Studies were considered to be eligible for inclusion according to the following criteria: (1) participants were ≥18 years of age.

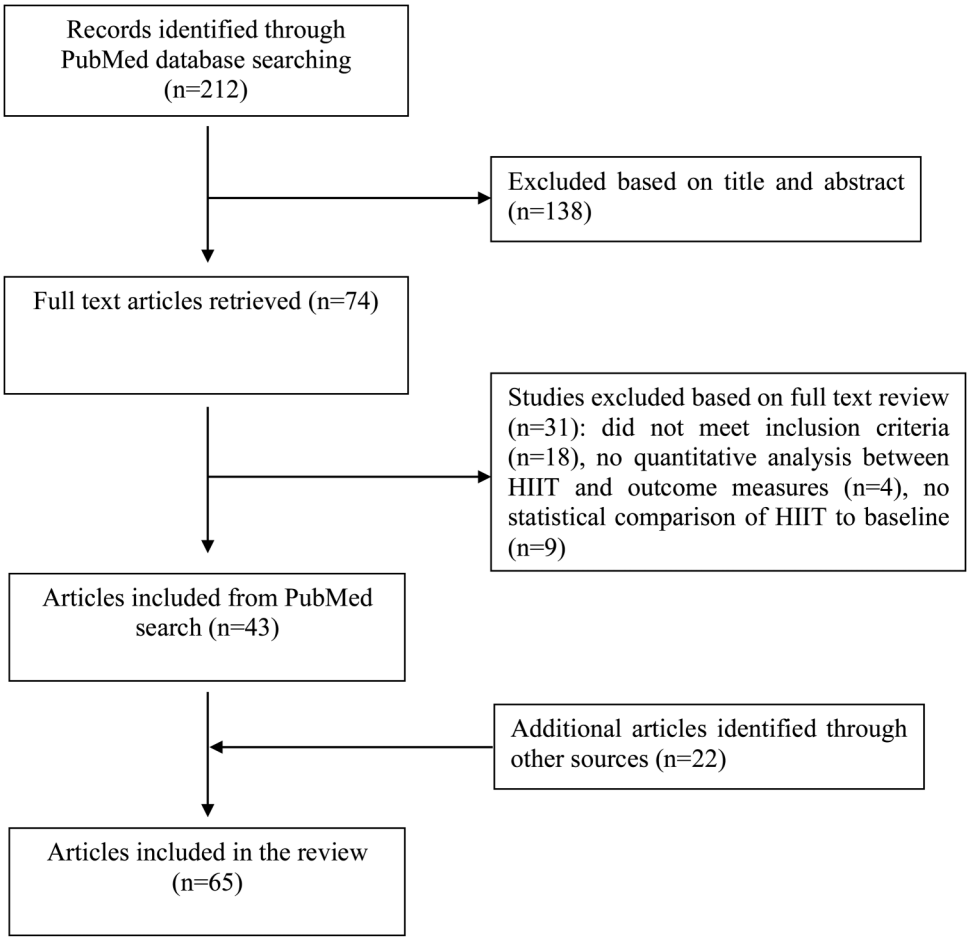


**Figure 1** Flow diagram of study selection. HIIT, high-intensity interval training.

(2) The study examined at least one of the following cardiometabolic health markers in humans: body mass, WC, HC, BMI, WHR, % body fat, HR, BP, $VO_2$ max, fasting glucose, glycosylated haemoglobin, insulin, total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides, CRP, IL-6, TNF-α, TNFR1 or TNFR2. (3) The study employed an intervention (randomised or non-randomised) of either short-term (ST-HIIT; <12 weeks) or long-term (LT-HIIT; ≥12 weeks) HIIT defined as activities with intermittent bouts of activity that were performed at maximal effort, ≥85% $VO_2$ max, ≥85% HR reserve or the relative intensity of at least 90% HR max.[3 28] (4) The study included a HIIT session lasting ≤4 min/set interspersed with an interval of rest or active recovery. (5) The study included quantitative analyses (statistical comparisons of the intervention to baseline/pretraining values) of the effect of HIIT on at least one of the outcome measures mentioned above (criteria ii). (6) The study reported baseline BMI of HIIT participants or baseline BMI can be calculated from the provided data. (7) The article was published or accepted for publication in a refereed journal from 1970 up to the search date. (8) The study was published in English. To differentiate the effect of HIIT duration on cardiometabolic markers, a classification of ST-HIIT (<12 weeks) and LT-HIIT (≥12 weeks) based on previous studies[20 29 30] was used in this review.

Two authors (RBB and PST) independently assessed the quality of the studies that met the inclusion criteria (table 1). The risk of bias and strength of evidence from individual studies were assessed using the Downs and Black Checklist,[31] which uses a scoring system to assess the strength of reporting, external validity, internal validity and statistical power. The maximum score that can be received is 32. Adapted from another systematic review,[32] the score obtained by each study was divided by 32 and multiplied by 100 to provide a 'Study Quality Percentage'. Study quality percentages were then classified as high (66.7% or higher), fair (between 50.0% and 66.6%) and low (<50.0%).[32]

Studies were stratified based on the duration of the training intervention and BMI classification. Mean BMI values were classified as follows: normal weight (18.5–24.99 kg/m$^2$), overweight (≥25–29.99 kg/m$^2$) and obese (≥30 kg/m$^2$).[33] For outcome markers examined in five or more studies, a meta-analysis was conducted in Review Manager 5.3 (The Cochrane Collaboration, Copenhagen, Denmark) using the inverse-variance statistical method. Previous meta-analyses have applied a minimum of five effect sizes to be included in the meta-analysis.[34 35] Too few studies of LT-HIIT with normal weight participants were available for meta-analysis. In instances where insufficient statistical information was reported to calculate mean change in health markers (ie, relevant measurements had been taken but the pretraining and post-training values were not reported or were presented in graphs), the authors were contacted and asked to provide the missing data. When no reply was received, the study/outcome was excluded from the meta-analysis. The effect size of the standardised mean difference (SMD) in cardiometabolic health markers from preintervention to postintervention in each study was calculated and pooled using the random-effects model (appropriate for data gathered from the published literature). Effect sizes were quantified as large (>0.8 SMD), medium (0.5 SMD–0.8 SMD) small (0.2 SMD–0.5 SMD) or non-significant (<0.2 SMD).[36] Heterogeneity of included studies was assessed using the $I^2$ statistic with heterogeneity estimates of 25%, 50% and 75% representing low, moderate and high heterogeneity, respectively. The significance level was set at $p<0.05$.

Where significant heterogeneity was found, meta-regression was performed using Comprehensive Meta-analysis V.3 (Biostat, New Jersey, USA) in an attempt to determine whether intervention duration, total HIIT time used (min), BMI and baseline level of the outcome variable mediated the observed changes. Total HIIT time was calculated by multiplying session duration (HIIT in minutes×number of repetitions, maximum repetition was used for studies where range was provided) by the frequency and duration of intervention. Intervention duration, total HIIT time used (min), BMI and baseline levels were used since these factors are likely to impact the outcome variables.

Where meta-analysis is not possible due to insufficient studies, a modified form of coding system described by Sallis *et al*[37] was used to summarise the studies reporting the effect of HIIT on cardiometabolic markers. If 0–33% of the studies reported a statistically significant difference between HIIT and cardiometabolic markers, the result was categorised as no effect (0). If 34–59% of the studies reported a statistically significant difference, the result was categorised as inconsistent (?). If 60–100% of the studies reported a statistically significant difference, the result was rated as positive (+) or negative (−), respective of the direction of the effect. When four or more studies supported a difference or no difference, it was coded as ++, −− or 00 to indicate consistent observations. The ?? code indicated a marker that has been examined in four or more studies with inconsistent findings.

## RESULTS

The search identified 212 articles published between the years 1981 and October 2015. Of these, 138 articles were excluded as they were not relevant to the scope of this review. Of the remaining 74 articles, 43 met the inclusion criteria and were included in the review. An additional 22 articles were identified through the reference lists of the included studies. As a result, 65 studies were included in the final analysis.

General study characteristics are summarised in table 1; more detailed study characteristics are presented in online supplementary table S1, and the effects of ST-HIIT and LT-HIIT on cardiometabolic health markers reported in each study are presented in online supplementary tables S2 and S3, respectively. All studies included in this analysis employed a HIIT intervention. The number of study participants in HIIT groups ranged from 5 to 85. Generally, participants were young (18–35 years old) men and women. The HIIT protocols ranged from an acute single session (a single 30 min bout of HIIT) to longer term multiple sessions (four HIIT sessions, lasting 4 min per session, three times per week for 52 weeks). Exercise modalities included treadmill running, swimming and cycling. Nineteen of 65 (29%) studies examined HIIT in participants with a current medical condition (hypertension, diabetes, metabolic syndrome, post myocardial infarction, coronary artery disease and patients who had a transplanted heart). Thirty-seven of 65 (57%) studies examined HIIT in overweight/obese participants, based on BMI. The summary estimates of the effect of ST-HIIT and LT-HIIT on cardiometabolic health variables in normal weight and overweight/obese populations are provided in figures 2 and 3. Forest plots on the effects of ST-HIIT and LT-HIIT on cardiometabolic markers are provided in online supplementary figures S1–S4. Table 2 provides a summary coding of the studies (not included in the meta-analysis) reporting the effect of ST-HIIT and LT-HIIT on cardiometabolic health markers.



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**Table 1** Summary of included studies, sorted by duration of intervention and BMI

| Study reference | Disease status* | Duration of intervention | Weight status (based on mean BMI) | Study design | Sample size N=HIIT group (total) | Age range (mean±SD) | Baseline activity level* | Downs and Black score | Study quality |
|---|---|---|---|---|---|---|---|---|---|
| [38] | No | 16 weeks | Normal | RCT | 16 (44) | 20–30 (24.4±3.8) | Physically inactive | 18 | Fair |
| [17] | No | 16 weeks | Normal | RCT | 16 (44) | 20–30 (24.4±3.8) | Physically inactive | 18 | Fair |
| [39] | No | 15 weeks | Normal | NRCT | 10 (27) | 18–32 | Not engaged in a regular exercise | 13 | Low |
| [15] | MI | 12 weeks | Normal | RCT | 9 (27) | (76.5±9) | No data | 19 | Fair |
| [40] | No | 12 weeks | Normal | CT | 10 (48) | (21.4±1.3) | Sports students (15 hour activities/ week) | 14 | Low |
| [41] | HTx | 12 months | Overweight | RCT | 20 (43) | (51±17) | No data | 20 | Fair |
| [42] | HTx | 24 weeks | Overweight | RCT | 24 (48) | 20–72 (56) | No data | 21 | Fair |
| [16] | MS | 16 weeks | Overweight | RCT | 9 (28) | (55.3±13.2) | No data | 16 | Fair |
| [43] | HPN | 15 weeks | Overweight | RCT | 21 (62) | 36–49 (44±2) | Sedentary | 19 | Fair |
| [44] | CAD | 12 weeks | Overweight | RCT | 85 (174) | 40–75 (57±8.8) | No data | 23 | High |
| [45] | No | 12 weeks | Overweight | RCT | 13 (38) | 18–55 (41.8±2.7) | Physically inactive | 22 | High |
| [46] | CAD | 12 weeks | Overweight | RCT | 24 (49) | 47–78 (64.4) | No data | 22 | High |
| [47] | DM2 | 12 weeks | Overweight | RCT | 14 (43) | 50–70 (61.2±2.8) | No previous exercise training | 17 | Fair |
| [48] | No | 12 weeks | Overweight | RCT | 10 (27) | (22.7±5.4) | Sedentary (<1 hour/week of structured exercise) | 19 | Fair |
| [49] | No | 12 weeks | Overweight | RCT | 17 (34) | 18–35(24.4±4.7) | Physically inactive | 19 | Fair |
| [50] | No | 12 weeks | Overweight | RCT | 20 (38) | 18–35 (24.9±4.3) | Physically inactive | 21 | Fair |
| [51] | No | 12 weeks | Overweight | RCT | 25 (46) | (24.7±4.8) | Physically inactive | 18 | Fair |
| [52] | DM2 | 12 weeks | Overweight | RCT | 7 (15) | 55–75 (62±3) | <150 min of structured exercise per week | 21 | Fair |
| [53] | MI | 12 weeks | Overweight | RCT | 35 (107) | (56.7±10.4) | No data | 20 | Fair |
| [54] | HPN | 12 weeks | Overweight | RCT | 25 (73) | (52.5±7.4) | No data | 19 | Fair |
| [55] | MS | 12 weeks | Obese | RCT | 11 (31) | (49.8±9.1) | Physically inactive | 20 | Fair |
| [56] | No | 12 weeks | Overweight | RCT | 17 (60) | 19–20 | No data | 18 | Fair |
| [57] | MS | 12 weeks | Obese | RCT | 11 (43) | (49.9±10.1) | No data | 20 | Fair |
| [58] | No | 12 weeks | Obese | RCT | 14 (40) | (46.9±2.2) | No data | 17 | Fair |
| [59] | No | 10 weeks | Normal | CT | 6 (17) | (19±2) | Moderately trained | 14 | Low |
| [60] | No | 10 weeks | Normal | CT | 22 (22) | (20.2±0.7) | No data | 15 | Low |
| [61] | No | 8 weeks | Normal | RCT | 14 (42) | 20–30 (26.4±6.5) | Sedentary lifestyle | 21 | Fair |
| [62] | No | 8 weeks | Normal | RCT | 11 (23) | 18–35 (25.2±0.7) | Sedentary to moderately trained | 16 | Fair |
| [63] | No | 8 weeks | Normal | RCT | 20 (36) | 21–36 (29.8±4.5) | Untrained | 19 | Fair |
| [64] | No | 8 weeks | Normal | RCT | 10 (55) | (24.6±3.8) | No data | 18 | Fair |
| [65] | No | 7 weeks | Normal | RCT | 9 (24) | (20.9±0.8) | Well trained | 15 | Low |
| [66] | No | 7 weeks | Normal | CT | 12 (12) | (22.7±2) | Physically active | 14 | Low |
| [67] | No | 6 weeks | Normal | RCT | 8 (16) | (22±1) | Sedentary (<1 hour of exercise/week) | 16 | Fair |
| [68] | No | 6 weeks | Normal | RCT | 8 (16) | (22±1) | Sedentary | 15 | Low |
| [69] | No | 6 weeks | Normal | RCT | 19 (44) | (21.7±4.4) | Recreationally active | 18 | Fair |
| [70] | No | 6 weeks | Normal | RCT | 5 (15) | (20±1) | Recreationally active | 16 | Fair |
| [9] | No | 6 weeks | Normal | CT | 10 (20) | (24±1) | Active but untrained | 14 | Low |
| [71] | No | 6 weeks | Normal | RCT | 13 (55) | 18–31 (21±1) | No data | 19 | Fair |

Continued

**Table 1** Continued

| Study reference | Disease status* | Duration of intervention | Weight status (based on mean BMI) | Study design | Sample size N=HIIT group (total) | Age range (mean±SD) | Baseline activity level* | Downs and Black score | Study quality |
|---|---|---|---|---|---|---|---|---|---|
| [72] | No | 6 weeks | Normal | RCT | 10 (20) | (23.6±3.2) | Not engaged in regular exercise | 16 | Fair |
| [73] | No | 6 weeks | Normal | CT | 7 (14) | (20±1) | Physically active | 15 | Low |
| [74] | No | 5 weeks | Normal | RCT | 8 (16) | (20±1) | Recreationally active | 15 | Low |
| [75] | No | 4 weeks | Normal | RCT | 8 (15) | (20.2±2.1) | Physically active | 16 | Fair |
| [76] | No | 4 weeks | Normal | RCT | 8 (24) | (25±0.8) | Habitually active but untrained | 16 | Fair |
| [77] | No | 2 weeks | Normal | CT | 20 (29) | (25.3±5.5) | Recreationally active | 15 | Low |
| [78] | No | 60 min | Normal | RCD | 11 (11) | (22.3±4) | Physically active | 17 | Fair |
| [79] | No | 30 min | Normal | RCD | 8 (8) | (34±7) | Physically active | 17 | Fair |
| [80] | DM1 | 30 min | Normal | RCD | 7 (7) | (21.6±4) | Physically active | 14 | Low |
| [81] | CAD | 10 weeks | Obese | RCT | 15 (28) | (60±7) | No data | 20 | Fair |
| [82] | No | 10 weeks | Overweight | RCT | 13 (26) | (42.2±2.4) | Physically inactive | 20 | Fair |
| [83] | CAD | 10 weeks | Overweight | RCT | 8 (21) | (62.9±11.2) | No data | 20 | Fair |
| [84] | No | 8 weeks | Obese | RCT | 7 (21) | 18–64 (40.9±11.7) | Sedentary | 17 | Fair |
| [85] | No | 8 weeks | Overweight | RCT | 7 (15) | 20–40 | Recreationally active | 16 | Fair |
| [86] | MS | 6 weeks | Obese | RCT | 22 (45) | 30–65 (51.9±9.2) | Physically inactive | 19 | Fair |
| [87] | No | 6 weeks | Overweight | RCT | 8 (16) | (27±8) | ≤2 sessions/week of structured exercise | 16 | Fair |
| [88] | No | 6 weeks | Overweight | CT | 10 (20) | (24.3±3.3) | Recreationally active | 15 | Low |
| [89] | No | 4 weeks | Obese | RCT | 14 (28) | (30.1±6.8) | Sedentary | 16 | Fair |
| [14] | CAD | 4 weeks | Overweight | RCT | 28 (59) | (60.2±6.9) | No data | 20 | Fair |
| [90] | No | 4 weeks | Overweight | RCT | 10 (42) | (23.62±4.78) | Recreationally active | 21 | Fair |
| [91] | No | 2 weeks | Obese | RCT | 8 (16) | (38.7±5.5) | Sedentary | 20 | Fair |
| [92] | No | 14 days | Overweight | CT | 12 (12) | (23.7±5.2) | Physically inactive | 15 | Low |
| [93] | No | 2 weeks | Overweight | CT | 11 (29) | (25.3±5.5) | Recreationally active | 14 | Low |
| [94] | No | 2 weeks | Overweight | CT | 7 (7) | (45±5) | Sedentary | 16 | Fair |
| [95] | DM2 | 2 weeks | Obese | CT | 8 (8) | (63±8) | Sedentary | 18 | Fair |
| [96] | No | 2 weeks | Overweight | RCT | 21 (31) | (29±3) | Recreationally active | 17 | Fair |
| [97] | No | 2 weeks | Obese | CT | 10 (10) | 18–40 (32.1±8.7) | Sedentary (<1 hour/week of structured exercise) | 15 | Low |

Physical inactivity is defined as not meeting at least 150 min of moderate-intensity physical activity or 75 min of vigorous-intensity physical activity.
*Disease status and baseline activity level (using the terminology of the primary study) presented were as per inclusion criteria.
BMI, body mass index; CAD, coronary artery disease; CT, clinical trial; DM2, diabetes mellitus type 2; HIIT, high-intensity interval training; HPN, hypertension; HTx, heart transplant recipient; min, minute; MI, myocardial infarction; MS, metabolic syndrome; NRCT, non-randomised controlled trial; RCD, randomised cross-over design; RCT, randomised clinical trial.



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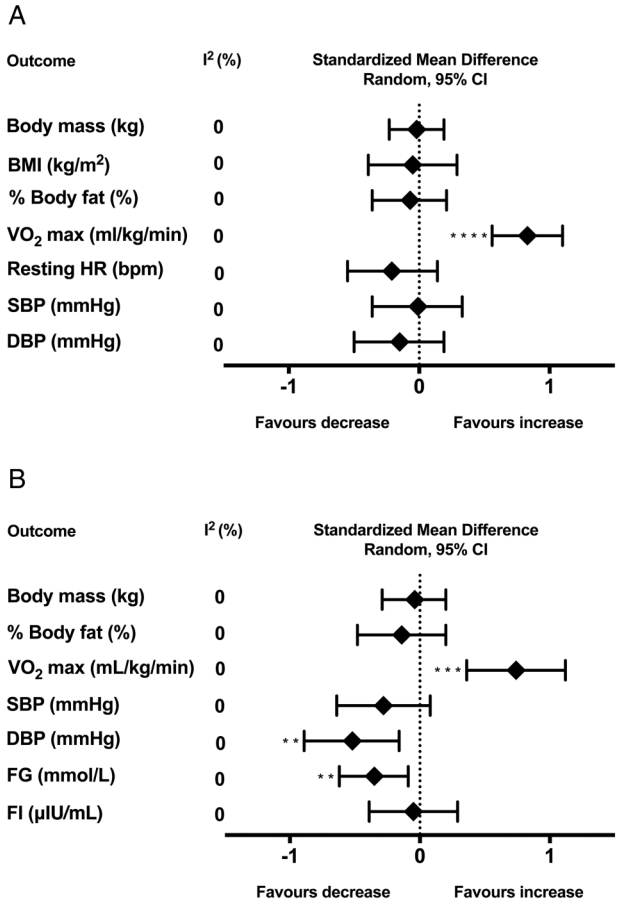


**Figure 2** Summary estimates of the effect of ST-HIIT on cardiometabolic health variables in (A) normal weight and (B) overweight/obese populations. BMI, body mass index; DBP, diastolic blood pressure; FG, fasting glucose; FI, fasting insulin; HR, heart rate; $I^2$, I-squared statistic for heterogeneity; SBP, systolic blood pressure; ST-HIIT, short-term high-intensity interval training; $VO_2$ max, maximal oxygen uptake. **p<0.01, ***p<0.001, ****p<0.00001.

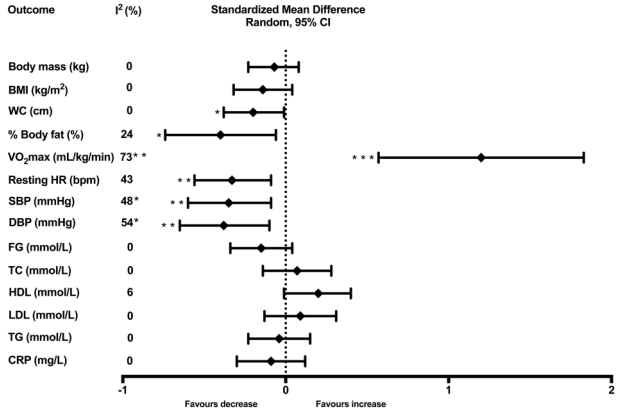


**Figure 3** Summary estimates of the effect of LT-HIIT on cardiometabolic health variables in overweight/obese populations. BMI, body mass index; CRP, C reactive protein; DBP, diastolic blood pressure; FG, fasting glucose; HDL, high-density lipoprotein cholesterol; HR, heart rate; $I^2$, I-squared statistic for heterogeneity; LDL, low-density lipoprotein cholesterol; LT-HIIT, long-term high-intensity interval training; SBP, systolic blood pressure; TC, total cholesterol; TG, triglycerides; $VO_2$ max, maximal oxygen uptake; WC, waist circumference. *p<0.05, **p<0.01, ***p<0.001.

### HIIT and body composition measures

ST-HIIT showed no effect on body mass (SMD −0.02, 95% CI −0.23 to 0.19; p=0.87), BMI (SMD −0.05 95% CI −0.39 to 0.29; p=0.77) and % body fat (SMD −0.07, 95% CI −0.36 to 0.21; p=0.62) in normal weight populations, with no significant heterogeneity across studies for body mass, BMI and % body fat ($I^2$=0%; p=1.0 for all outcomes). Similarly, ST-HIIT showed no effect on body mass (SMD −0.04, 95% CI −0.29 to 0.20; p=0.72) and % body fat (SMD −0.14, 95% CI −0.48 to 0.20; p=0.42) in overweight/obese populations, with no significant heterogeneity across studies ($I^2$=0%; p=1.0). The summary coding revealed no evidence for the effect of ST-HIIT on WC in normal weight populations and no evidence for the effect of ST-HIIT on BMI in overweight/obese populations. Conversely, summary coding showed that ST-HIIT reduced WC in overweight/obese populations.

There were insufficient studies to examine the effect of LT-HIIT on body composition in normal weight populations. LT-HIIT reduced WC (SMD −0.20, 95% CI −0.38 to −0.01; p<0.05) and % body fat (SMD −0.40, 95% CI −0.74 to −0.06; p<0.05) by a small effect in overweight/obese populations, with no significant heterogeneity across studies ($I^2$=0%; p=0.74 for WC, $I^2$=24%; p=0.25 for % body fat). However, LT-HIIT showed no effect on body mass (SMD −0.07, 95% CI −0.23 to 0.08; p=0.37) and BMI (SMD −0.14, 95% CI −0.32 to 0.04; p=0.12) in overweight/obese populations, with no significant heterogeneity across studies ($I^2$=0%; p=0.95 for body mass, $I^2$=0%; p=0.62 for BMI). Furthermore, summary coding revealed that the effect of LT-HIIT on WHR was inconsistent in overweight/obese populations.

### HIIT and cardiorespiratory measures

ST-HIIT increased $VO_2$ max (SMD 0.83, 95% CI 0.56 to 1.10; p<0.00001) by a large effect in normal weight populations and increased $VO_2$ max (SMD 0.74, 95% CI 0.36 to 1.12; p<0.001) by a medium effect in overweight/obese populations, with no significant heterogeneity across studies ($I^2$=0%; p=0.52 for normal weight; $I^2$=0%; p=0.62 for overweight/obese). While the summary coding revealed that LT-HIIT increased $VO_2$ max in normal weight populations. LT-HIIT improved $VO_2$ max (SMD 1.20, 95% CI 0.57 to 1.83; p<0.001) by a large effect in overweight/obese populations, with significant heterogeneity across studies ($I^2$=73%; p<0.01).

ST-HIIT showed no effect on resting HR (SMD −0.21, 95% CI −0.55 to 0.14; p=0.24) in normal weight populations, with no significant heterogeneity across studies ($I^2$=0%; p=0.81). There was no evidence in the summary coding for the effect of ST-HIIT on resting HR in overweight/obese populations and no evidence for the effect of LT-HIIT on resting HR in normal weight populations. In contrast, LT-HIIT decreased resting HR (SMD −0.33, 95% CI −0.56 to −0.09; p<0.01) by a small effect in overweight/obese populations, with no significant heterogeneity across studies ($I^2$=43%; p=0.07).

ST-HIIT showed no effect on systolic BP (SBP; SMD −0.01, 95% CI −0.36 to 0.33; p=0.95) and diastolic BP (DBP; SMD −0.15, 95% CI −0.50 to 0.19; p=0.39) in normal weight populations, with no significant heterogeneity across studies ($I^2$=0%; p=0.91 for SBP; $I^2$=0%; p=0.98 for DBP). While ST-HIIT showed no effect on SBP (SMD −0.28, 95% CI −0.64 to 0.08; p=0.12) and decreased DBP (SMD −0.52, 95% CI −0.89 to −0.16; p<0.01) by a medium effect in overweight/obese populations, with no significant heterogeneity across studies ($I^2$=0%; p=0.98 for SBP; $I^2$=0%; p=0.79 for DBP). The summary

**Table 2** Summary coding of studies examining the effect of HIIT on cardiometabolic health variables

| | | Normal weight population | | Overweight/obese population | |
|---|---|---|---|---|---|
| HIIT duration ion | Marker | n/N (%)*†‡ | Effect (0/−/+/?)§ | n/N (%)*†‡ | Effect (0/−/+/?)§ |
| ST-HIIT | BM | NR | NA | NR | NA |
| | BMI | NR | NA | 1/4 (25%) | No (0) |
| | WC | 0/2 (0%) | No (0) | 2/3 (67%) | Positive (+) |
| | WHR | 0/1 (0%) | NA | 0/0 (0%) | NA |
| | % BF | NR | NA | NR | NA |
| | RHR | NR | NA | 1/3 (33%) | No (0) |
| | $VO_2$ max | NR | NA | NR | NA |
| | SBP | NR | NA | NR | NA |
| | DBP | NR | NA | NR | NA |
| | FG | 2/4 (50%) | Inconsistent (?) | NR | NA |
| | FI | 0/2 (0%) | No (0) | NR | NA |
| | HbA1c | 0/0 (0%) | NA | 1/1 (100%) | NA |
| | TC | 1/2 (50%) | Inconsistent (?) | 1/3 (33%) | No (0) |
| | HDL | 1/3 (33%) | No (0) | 0/4 (0%) | No (00) |
| | LDL | 1/2 (50%) | Inconsistent (?) | 1/3 (33%) | No (0) |
| | TG | 0/2 (0%) | No (0) | 0/5 (0%) | No (00) |
| | CRP | 0/0 (0%) | NA | 0/1 (0%) | NA |
| | IL-6 | 1/2 (50%) | Inconsistent (?) | 0/1 (0%) | NA |
| | TNF-α | 0/1 (0%) | NA | 0/1 (0%) | NA |
| LT-HIIT | BM | 0/1 (0%) | NA | NR | NA |
| | BMI | 0/1 (0%) | NA | NR | NA |
| | WC | 0/0 (0%) | NA | NR | NA |
| | WHR | 0/0 (0%) | NA | 2/4 (50%) | Inconsistent (?) |
| | % BF | 0/0 (0%) | NA | NR | NA |
| | RHR | 0/2 (0%) | No (0) | NR | NA |
| | $VO_2$ max | 4/4 (100%) | Positive (++) | NR | NA |
| | SBP | 1/2 (50%) | Inconsistent (?) | NR | NA |
| | DBP | 1/2 (50%) | Inconsistent (?) | NR | NA |
| | FG | 0/2 (0%) | No (0) | NR | NA |
| | FI | 1/1 (100%) | NA | 0/3 (0%) | No (0) |
| | HbA1c | 0/0 (0%) | NA | 1/5 (20%) | No (00) |
| | TC | 0/2 (0%) | No (0) | NR | NA |
| | HDL | 0/2 (0%) | No (0) | NR | NA |
| | LDL | 0/1 (0%) | NA | NR | NA |
| | TG | 0/2 (0%) | No (0) | NR | NA |
| | CRP | 0/0 (0%) | NA | NR | NA |
| | IL-6 | 0/0 (0%) | NA | 0/2 (0%) | No (0) |
| | TNF-α | 0/0 (0%) | NA | 1/1 (100%) | NA |

*n=number of studies reporting difference in the expected direction.
†N=number of identified studies of interest.
‡(%)=percentage of studies reporting differences in the expected direction.
§Summary effect. No effect (0): 0–33% of studies reported significant differences; inconsistent (?): 34–59% of studies reported significant differences; positive (+) or negative (−) effect: 60–100% of studies demonstrated significant differences; ≥4 studies: positive (++), negative (−−), no effect (00), inconsistent findings (??).
% BF, body fat percentage; BM, body mass; BMI, body mass index; CRP, C reactive protein; DBP, diastolic blood pressure; FG, fasting glucose; FI, fasting insulin; HbA1c, glycosylated haemoglobin; HDL, high-density lipoprotein cholesterol; HIIT, high-intensity interval training; IL-6, interleukin 6; LDL, low-density lipoprotein cholesterol; NA, not applicable; NR, no reported summary coding (a meta-analysis was performed); RHR, resting heart rate; SBP, systolic blood pressure; TC, total cholesterol; TG, triglycerides; TNF-α, tumour necrosis factor-α; $VO_2$ max, maximal oxygen uptake; WC, waist circumference; WHR, waist-to-hip ratio.

coding revealed that the effect of LT-HIIT on SBP and DBP was inconsistent in normal weight populations. LT-HIIT decreased SBP (SMD −0.35, 95% CI −0.60 to −0.09; p<0.01) and DBP (SMD −0.38, 95% CI −0.65 to −0.10; p<0.01) by a small effect in overweight/obese populations, with significant heterogeneity across studies ($I^2$=48%, p<0.05 for SBP; $I^2$=54%, p<0.05 for DBP).

### HIIT and glucose metabolism measures

The summary coding revealed that the evidence for the effect of ST-HIIT on fasting glucose was inconsistent and no evidence for the effect of ST-HIIT on fasting insulin in normal weight populations. Alternatively, ST-HIIT reduced fasting glucose (SMD −0.35, 95% CI −0.62 to −0.09; p<0.01) by a small effect and showed no effect on fasting insulin (SMD −0.05, 95% CI −0.39 to 0.29; p=0.76) in overweight/obese populations, with no significant heterogeneity across studies ($I^2$=0%, p=0.51 for fasting glucose; $I^2$=0%, p=0.95 for fasting insulin). The summary coding revealed no evidence for the effect of LT-HIIT on fasting glucose in normal weight populations. Similarly, LT-HIIT demonstrated no effect on fasting glucose (SMD −0.15, 95% CI −0.34 to 0.04; p=0.11) in overweight/obese populations, with no significant heterogeneity across studies ($I^2$=0%; p=1.0). There was no evidence in the summary

coding for the effect of LT-HIIT on fasting insulin and HbA1c in overweight/obese populations.

### HIIT and blood lipid measures

The summary coding revealed that the evidence for the effect of ST-HIIT on total cholesterol and LDL cholesterol was inconsistent and no evidence for the effect of ST-HIIT on HDL cholesterol and triglycerides in normal weight populations. There was no evidence in the summary coding for the effect of ST-HIIT on total cholesterol, HDL cholesterol, LDL cholesterol and triglycerides in overweight/obese populations. The summary coding revealed no evidence for the effect of LT-HIIT on total cholesterol, HDL cholesterol and triglycerides in normal weight populations. LT-HIIT demonstrated no effect on total cholesterol (SMD 0.07, 95% CI −0.14 to 0.28; p=0.51), HDL cholesterol (SMD 0.20, 95% CI −0.01 to 0.40; p=0.06), LDL cholesterol (SMD 0.09, 95% CI −0.13 to 0.31; p=0.42) and triglycerides (SMD −0.04, 95% CI −0.23 to 0.15; p=0.67) in overweight/obese populations, with no significant heterogeneity across studies ($I^2$=0%, p=0.73 for total cholesterol; $I^2$=6%, p=0.38 for HDL; $I^2$=0%, p=0.93 for LDL; $I^2$=0%, p=0.91 for triglycerides).

### HIIT and inflammation measures

The summary coding revealed that the evidence for the effect of ST-HIIT on IL-6 was inconsistent in normal weight populations. There was no evidence in the summary coding for the effect of LT-HIIT on IL-6 in overweight/obese populations. LT-HIIT showed no effect on CRP (SMD −0.09, 95% CI −0.30 to 0.12; p=0.39) in overweight/obese populations, with no significant heterogeneity across studies ($I^2$=0%; p=0.94).

### Meta-regression

Table 3 shows the β-coefficients and CIs for the meta-regression analyses. Intervention duration, total HIIT time used (min), BMI and baseline level of the outcome variable did not predict the improvements observed in SBP and DBP. Intervention duration (β (95% CI)=0.77 (0.35, 1.18); $R^2$=0.94) and BMI (β (95% CI)=0.84 (0.29, 1.38); $R^2$=0.73) predicted changes in $VO_2$ max. Greater increases in $VO_2$ max were associated with longer intervention duration and higher BMI.

## DISCUSSION

Results suggest that HIIT is an effective intervention to improve cardiometabolic health in overweight/obese populations. Specifically, ST-HIIT beneficially influenced WC, $VO_2$ max, fasting glucose and DBP, whereas LT-HIIT was found to beneficially influence WC, % body fat, $VO_2$ max, resting HR, SBP and DBP in overweight/obese populations.

Meta-analysis of ST-HIIT revealed no significant effect on body composition in normal weight populations, whereas too few studies are currently available examining the effect of LT-HIIT in normal weight populations. ST-HIIT reduced WC in overweight/obese populations and LT-HIIT significantly improved WC and % body fat in overweight/obese populations. The average change in WC was 2.13 cm for ST-HIIT and 2.23 cm for LT-HIIT, both above the cut-off value of >2 cm WC decrease which is suggested to confer improvements in metabolic syndrome risk factors.[98] These findings suggest that HIIT is an effective stimulus for reducing body fat levels (even in the absence of weight loss) for those individuals with large fat mass. Possible mechanisms underlying HIIT-induced fat loss include generation of catecholamines that increased fat oxidation and fat release from visceral fat stores, decreased postexercise appetite and increased excess postexercise oxygen consumption resulting in an elevated fat loss state.[99 100] A catecholamine response has been shown to be significantly elevated after HIIT.[101 102] Since $\beta_3$-adrenergic receptors are located mainly in the adipose tissue[103] and β-adrenergic receptor sensitivity in adipose tissue is increased following exercise,[104] these factors might explain why HIIT is effective in reducing body fat in overweight/obese individuals. One intriguing finding is the absence of weight loss despite observed decrease in body fat, this is likely to be a consequence of gain in muscle mass. HIIT is known to recruit more fast type II muscle fibres leading to greater muscle hypertrophy and muscle mass.[105 106] This adaptation is likely to induce health benefits as increase in muscle mass improves insulin sensitivity.[107] As an example, elevated muscle mass was found positively associated with reduced incidence of insulin resistance and metabolic syndrome.[108 109] Thus, if HIIT can be successfully implemented in settings outside of clinical trials, it may offer an additional strategy to assist with adipose reduction in overweight/obese populations. However, more studies are required to determine whether HIIT could be a successful population-based strategy for producing health adaptations.

Results from meta-analysis consistently revealed that ST-HIIT significantly improved $VO_2$ max by medium effects to large effects in normal weight (SMD 0.83, 95% CI 0.56 to 1.10; p<0.00001) and overweight/obese (SMD 0.74, 95% CI 0.36 to 1.12; p<0.001) populations, with an aggregate improvement of 3.80 and 4.43 mL/kg/min, respectively. The summary coding revealed that LT-HIIT increased $VO_2$ max in normal weight populations, whereas the meta-analysis showed that LT-HIIT significantly improved $VO_2$ max by large effects in overweight obese (SMD 1.20, 95% CI 0.57 to 1.83; p<0.001) populations with an aggregate improvement of 6.04 mL/kg/min. These findings are similar to previous meta-analyses which have demonstrated that HIIT improves aerobic fitness by moderate effects to large effects (Hedges' g=0.63, 95% CI 0.39 to 0.87; SMD 0.86, 95% CI 0.72 to 0.99) in healthy sedentary and recreationally active young adults[12 19 21 110] and in adults with cardiometabolic disorders.[18 23] Notably, ST-HIIT and LT-HIIT improve $VO_2$ max in normal weight and overweight/obese populations

**Table 3** Meta-regression coefficients of the effect of significant moderators on selected outcomes

| | β (95% CI) | | | |
|---|---|---|---|---|
| **Marker** | **Intervention duration** | **Total HIIT time used (min)** | **BMI** | **Baseline level** |
| $VO_2$ max | 0.7677 (0.3510 to 1.1844)* | 0.0002 (−0.0017 to 0.0021) | 0.8366 (0.2948 to 1.3785)* | −0.3928 (−1.0735 to 0.2879) |
| SBP | 0.0192 (−0.0575 to 0.0960) | 0.0003 (−0.0004 to 0.0010) | −0.1169 (−0.3611 to 0.1274) | −0.0210 (−0.0432 to 0.0011) |
| DBP | 0.0191 (−0.0620 to 0.1001) | 0.0003 (−0.0004 to 0.0010) | −0.0809 (−0.3434 to 0.1816) | 0.0053 (−0.0177 to 0.0283) |

*p<0.05.
BMI, body mass index; DBP, diastolic blood pressure; HIIT, high-intensity interval training; SBP, systolic blood pressure; $VO_2$ max, maximal oxygen uptake.

with larger gains observed for longer training periods. This has implications for the use of HIIT as part of lifestyle modification strategies and is consistent with training responses to stimuli. The ability of ST-HIIT and LT-HIIT has clinical applications in individuals that need to improve their aerobic fitness as HIIT is able to increase $VO_2$ max rapidly via increasing mitochondrial density, resulting in the generation of more ATP for working muscles, thereby producing greater force generation for a longer duration.[111] HIIT is also able to increase stroke volume induced by increased cardiac contractility[64] and increase skeletal muscle diffusive capacity,[112] thus improving aerobic capacity.

This review also found LT-HIIT to significantly decrease resting HR in overweight/obese populations, but not in normal weight populations subjected to LT-HIIT and in normal weight/overweight/obese populations subjected to ST-HIIT. The decrease in resting HR following LT-HIIT may be explained by increased stroke volume[64] and improved cardiac autonomic function via increased baroreflex-mediated modulation of the sinoatrial node.[113] Taken together, these improvements in cardiorespiratory fitness ($VO_2$ max) and HR response provided by HIIT are important since both are independent predictors of all-cause and CVD mortality.[114–116]

BP is another commonly assessed measure related to cardiovascular health. ST-HIIT showed no significant effect on SBP and DBP in normal weight populations. While ST-HIIT showed no significant effect on SBP in normal weight and overweight/obese populations, ST-HIIT significantly improved DBP in overweight/obese populations with an average reduction of 4.74 mm Hg. This lack of change observed in SBP following ST-HIIT is perhaps due to the fact that most of the participants in this group were middle aged to older aged (40.9–62.9 years old), and it is well known that SBP increases progressively with age.[117] It is possible that longer HIIT intervention periods are required to produce a significant effect in SBP in this population. This is supported by the observation that LT-HIIT significantly decreased SBP and DBP in overweight/obese populations. The average reduction is 4.57 mm Hg for SBP and 2.94 mm Hg for DBP, above 4 mm Hg SBP reduction which is expected to decrease CVD mortality by 5–20%.[118] The findings of the current study demonstrate the beneficial impact of HIIT in overweight/obese populations. The mechanisms responsible for the BP lowering effect of HIIT may result from intensity-dependent increases to blood flow velocity, resulting in increased levels of endothelial nitric oxide (NO).[119 120] Increases in endothelial NO availability and bioactivity improve NO-dependent vasodilation in the vasculature, resulting in improved peripheral compliance and decreased BP.[121]

ST-HIIT and LT-HIIT showed no significant effect on glucose/insulin response in normal weight populations. No changes in fasting glucose/insulin were observed in overweight/obese populations subjected to LT-HIIT, but a decrease in fasting glucose was observed in overweight/obese populations subjected to ST-HIIT. The reason for improvement in glucose response in ST-HIIT is not fully known, but activation of AMP-activated kinase (AMPK) has been shown to increase glucose uptake in skeletal muscle via increased translocation of GLUT4.[122 123] Interestingly, no significant effect was observed in glucose/insulin response following LT-HIIT in overweight/obese populations. One explanation is that all the LT-HIIT studies that examined glucose metabolism were conducted in participants with a pre-existing medical condition (coronary artery disease, metabolic syndrome and hypertension) that is known to independently influence glucose metabolism, thus possibly obscuring the effect of HIIT on glucose metabolism.

There was no evidence to suggest that ST-HIIT and LT-HIIT influence blood lipids in normal weight and overweight/obese populations. Although the exact cause is unknown, a possible explanation is that HIIT decreases fatty acid release in the circulation due to decreased blood flow in adipose tissue mediated by α2-adrenergic receptors during high plasma catecholamine concentrations.[124] This is an area that requires further examination.

The role of inflammation in diabetes and CVD risk is increasingly acknowledged.[125] Inflammation contributes to the development of CVD by narrowing arteries[126] and diabetes by promoting insulin resistance.[127] ST-HIIT demonstrated no effect on IL-6 in normal weight populations, whereas LT-HIIT demonstrated no effect on CRP and IL-6 in overweight/obese populations. One LT-HIIT study reported improvements in TNF-α in obese, middle-aged to older-aged adults. At present, the effect of HIIT on inflammation is not clear. The small number of studies combined with the varied populations made synthesis of studies difficult. Furthermore, evidence suggests that the duration of individual bouts of exercise is the single most important factor that determines the magnitude of the systemic IL-6 response.[128] The short bouts of exercise used in some of the studies included in this review may not be long enough to elicit a pronounced IL-6 response despite the high intensity of HIIT. Thus, studies addressing these issues are encouraged to better understand the impact of HIIT on inflammatory markers.

This review demonstrates that HIIT performed <12 weeks and ≥12 weeks can significantly improve $VO_2$ max in normal weight and overweight/obese populations, <12 weeks of HIIT can significantly improve WC, fasting glucose and DBP in overweight/obese populations and at least 12 weeks of HIIT appears to promote significant reductions in WC, % body fat, resting HR, SBP and DBP in overweight/obese populations. However, despite the results observed for ST-HIIT and LT-HIIT in overweight/obese populations, there were too few studies of LT-HIIT in normal weight populations. The number of health-related benefits elicited by HIIT in overweight/obese populations is possibly related to the changes in fat mass following HIIT. Another explanation is that increase in adipose tissue induces metabolic dysregulation increasing responsiveness to HIIT. However, although improvements in cardiorespiratory fitness and body composition were observed, there were no evidence to suggest that lipid metabolism and inflammatory markers are influenced by ST-HIIT or LT-HIIT, possibly due to the different metabolic consequences of the different HIIT protocols causing different metabolic adaptations. The challenge therefore for future research is to identify the optimal length, work-to-rest ratio of HIIT that would provide maximum health benefit.

There are some limitations to this meta-analysis. The effect of sprint interval training (HIIT above 100% $VO_2$ max) separate to HIIT was not examined and is a potential area for future reviews. Most studies included are of low (16/65) to fair (46/65) quality and used relatively small sample sizes. The substantial heterogeneity found in several meta-analysed health markers (WC, $VO_2$ max, HR, SBP and DBP) suggests differences in population cohorts and study design as possible sources. This issue was addressed by stratifying the results by study duration and BMI, and by performing a meta-regression. Additional studies conducted in larger and more diverse samples are required to address these limitations of primary studies.

## CONCLUSION

Findings of this review indicate that HIIT may constitute an effective training protocol for improving $VO_2$ max and several



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cardiometabolic risk factors such as WC, % body fat, resting HR, SBP, DBP and fasting glucose in overweight/obese populations. Taken as a whole, in overweight/obese populations, performing HIIT results in significant, positive, physiological adaptations that improve cardiometabolic health and may reduce the development and progression of disease-related risk factors that are associated with overweight/obesity and low aerobic fitness. However, whether these metabolic adaptations following LT-HIIT extend to normal weight populations still needs further examination.

As HIIT activity regime requires minimal time commitment, HIIT may serve as a time-efficient substitute or as a compliment to commonly recommended MICT in improving cardiometabolic health. Clinicians are encouraged (after an appropriate pre-exercise screening and under supervised conditions) to use HIIT performed at least three times a week for 12 weeks as part of their exercise programme to enhance cardiorespiratory fitness and to reduce body fat in overweight/obese populations.

## What are the findings?

- At least 12 weeks of high-intensity interval training (HIIT) improves cardiometabolic risk factors such as waist circumference, % body fat, resting heart rate, systolic blood pressure and diastolic blood pressure in overweight/obese populations.
- Improvements in aerobic capacity are larger with longer training periods.
- The effect of HIIT on inflammation is not clear due to the limited number of studies available.

## How might it impact on clinical practice in the future?

- High-intensity interval training (HIIT) performed at least 3 times a week for 12 weeks results in significant, positive, physiological adaptations that improve cardiometabolic health in overweight/obese populations.
- HIIT may reduce the development and progression of disease-related risk factors that are associated with overweight/obesity and low aerobic fitness.
- HIIT may be especially attractive to overweight/obese populations interested in improving cardiometabolic health but with limited time available.

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