## SYSTEMATIC REVIEW

# Effects of Low-Volume High-Intensity Interval Training (HIT) on Fitness in Adults: A Meta-Analysis of Controlled and Non-Controlled Trials

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Published online: 18 April 2014

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#### **Abstract**

Background Low-volume high-intensity interval training (HIT) appears to be an efficient and practical way to develop physical fitness.

Objective Our objective was to estimate meta-analysed mean effects of HIT on aerobic power (maximum oxygen consumption  $[VO_{2max}]$  in an incremental test) and sprint fitness (peak and mean power in a 30-s Wingate test).

Data Sources Five databases (PubMed, MEDLINE, Scopus, BIOSIS and Web of Science) were searched for original research articles published up to January 2014. Search terms included 'high intensity', 'HIT', 'sprint', 'fitness' and ' $VO_{2max}$ '.

Study Selection Inclusion criteria were fitness assessed pre- and post-training; training period  $\geq 2$  weeks; repetition duration 30–60 s; work/rest ratio <1.0; exercise intensity described as maximal or near maximal; adult subjects aged >18 years.

Data Extraction The final data set consisted of 55 estimates from 32 trials for  $VO_{2max}$ , 23 estimates from 16 trials for peak sprint power, and 19 estimates from 12 trials for mean sprint power. Effects on fitness were analysed as

percentages via log transformation. Standard errors calculated from exact *p* values (where reported) or imputed from errors of measurement provided appropriate weightings. Fixed effects in the meta-regression model included type of study (controlled, uncontrolled), subject characteristics (sex, training status, baseline fitness) and training parameters (number of training sessions, repetition duration, work/rest ratio). Probabilistic magnitude-based inferences for meta-analysed effects were based on standardized thresholds for small, moderate and large changes (0.2, 0.6 and 1.2, respectively) derived from between-subject standard deviations (SDs) for baseline fitness.

Results A mean low-volume HIT protocol (13 training sessions, 0.16 work/rest ratio) in a controlled trial produced a likely moderate improvement in the VO<sub>2max</sub> of active non-athletic males (6.2 %; 90 % confidence limits  $\pm 3.1$  %), when compared with control. There were possibly moderate improvements in the VO<sub>2max</sub> of sedentary males (10.0 %; ±5.1 %) and active non-athletic females  $(3.6\%; \pm 4.3\%)$  and a likely small increase for sedentary females (7.3 %;  $\pm 4.8$  %). The effect on the  $VO_{2max}$  of athletic males was unclear (2.7 %; ±4.6 %). A possibly moderate additional increase was likely for subjects with a 10 mL·kg<sup>-1</sup>·min<sup>-1</sup> lower baseline  $VO_{2max}$  (3.8 %;  $\pm 2.5$  %), whereas the modifying effects of sex and difference in exercise dose were unclear. The comparison of HIT with traditional endurance training was unclear  $(-1.6\%; \pm 4.3\%)$ . Unexplained variation between studies was 2.0 % (SD). Meta-analysed effects of HIT on Wingate peak and mean power were unclear.

Conclusions Low-volume HIT produces moderate improvements in the aerobic power of active non-athletic and sedentary subjects. More studies are needed to resolve the unclear modifying effects of sex and HIT dose on aerobic power and the unclear effects on sprint fitness.

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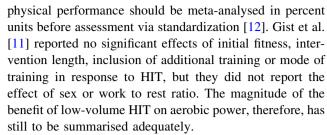
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## 1 Introduction

High-intensity interval training (HIT), which involves alternating bouts of intensive exercise with low-intensity recovery periods [1], is considered one of the most effective means of improving cardiorespiratory and metabolic function [2]. Athletes and coaches have historically used HIT to improve exercise performance, but the effectiveness of HIT to improve health-related outcomes has recently generated new interest [3]. In recent reviews, there appears to be a consensus for the benefit of high-intensity aerobic interval training in patient populations [3–6]. Weston et al. [6] meta-analysed ten studies and reported that highintensity aerobic interval training, typically performed at 85-95 % maximal heart rate (%HR<sub>max</sub>), increased cardiorespiratory fitness by almost double that of moderateintensity continuous training in patients with lifestyleinduced chronic disease. In contrast, HIT of similar intensity elicits improvements in maximal oxygen uptake (VO<sub>2max</sub>) slightly greater than is typically reported with continuous training in healthy, active adults [7].

HIT can encompass a considerable range of exercise intensities. For example, Buchheit and Laursen [8] recently defined HIT as "either repeated short (<45 s) to long (2-4 min) bouts of rather high- but not maximal-intensity exercise, or short (<10 s, repeated-sprint sequences) or long (>20-30 s, sprint interval session) all-out sprints, interspersed with recovery periods." As such, maximal, allout sprint training is classified as a form of high-intensity training at the highest end of the intensity spectrum [9, 10]. Here, the repeated bouts of relatively brief all-out (maximal) intermittent exercise necessitate shorter interval durations and longer recovery periods than those of traditional high-intensity aerobic interval programming, and the total weekly volume (duration) of exercise is therefore lower. There is accumulating evidence supporting improved aerobic exercise performance following this form of training. Kessler et al. [3] reviewed five studies with exercise intensity described as all-out and concluded that it was an effective means of improving  $VO_{2\text{max}}$ . Sloth et al. [10] meta-analysed standardized effects of low-volume all-out interval training on VO<sub>2max</sub> in 13 studies and reported an overall moderate effect (standardised change in the mean of 0.63). However, their meta-analysis did not account for the modifying effects of study and subject characteristics, or studies with reference groups representing traditional endurance training, rather than no training. Using similar inclusion criteria (e.g. 30-s all-out sprints) Gist and colleagues [11] meta-analysed 16 randomized controlled trials and reported a moderate effect (0.69) of HIT on  $VO_{2max}$  in comparison with no-exercise control groups and a trivial effect (0.04) when compared with endurance-training controls. However, effects on



Low-volume HIT may also have the potential to improve sprint power, as it increases enzymatic activities of anaerobic metabolism [13]. Most sporting activities depend upon the expression of power for short or sustained periods of time [14]. Furthermore, many basic daily activities are dependent on the ability to generate force at high velocity, and power training can improve mobility-related outcomes in the elderly [15] as well as increasing self-efficacy, satisfaction with physical function and overall life satisfaction [16]. A meta-analysis of the effect of HIT on sprint power is therefore timely. Our aim for this review was to use a mixed-model meta-analysis to provide estimates of the effect of low-volume HIT on fitness  $(VO_{2\text{max}}, 30\text{-s Wingate power})$  along with the modifying effects of study and subject characteristics.

## 2 Methods

## 2.1 Literature Search

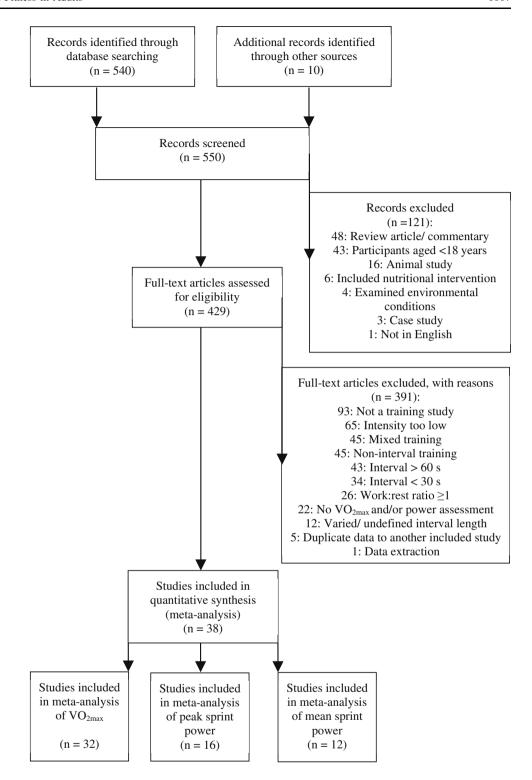
A search of five databases (PubMed, MEDLINE, Scopus, BIOSIS and Web of Science), along with the reference lists of original research and review articles published in English up to January 2014 was conducted by two of the authors (KT, MW). Our independent variable search terms were 'aerobic high intensity', 'high intensity', 'HIT', 'intervals', 'intensive', 'sprint', 'repeated sprint', and the dependent variable search terms were 'fitness', 'aerobic fitness', 'anaerobic fitness', 'VO<sub>2max</sub>' 'performance', 'endurance' and 'adaptations'. Independent variable search terms were combined with dependent variable search terms, giving a total of 49 combinations.

## 2.2 Study Selection

The most common model employed in low-volume HIT studies consisted of four to six 30-s 'all-out' efforts separated by  $\sim 4$  min of recovery, for a total of two to three minutes of intense exercise during a single training session. As such, our study selection criteria were  $VO_{2\text{max}}$  or 30-s Wingate power assessed pre- and post-training, training period  $\geq 2$  weeks, repetition duration 30–60 s, work/rest ratio <1.0, exercise intensity described as maximal or near maximal, and adult subjects aged >18 years. No inclusion



**Fig. 1** Flow diagram of study selection.  $VO_{2\text{max}}$  maximum oxygen consumption



criteria were used for participant fitness. Using the subject characteristic information provided by each study, participants were assigned to one of three groups: sedentary, active non-athletic or athletic.

The selection of studies for our meta-analysis was confined to studies predominantly utilizing the classic

Wingate protocol. In doing so, we acknowledge the exclusion of a large body of laboratory- and field-based HIT research utilizing longer interval durations (1–4 min) performed at high, but not maximal intensity, and with a work:rest ratio  $\geq 1.0$  [8]. Furthermore, by selecting  $VO_{2max}$  as our measure of aerobic fitness, we excluded field-



Table 1 Study and subject characteristics for maximum oxygen consumption estimates included in the meta-analysis

References	Study design	Subjects	Mean age, y	Sample size	Proportion of males	Duration (weeks)	Total sessions	Group	Exercise intensity <sup>a</sup>	No. of reps	f reps	Total reps	Rep duration (s)	Work/rest ratio	Effect on VO <sub>2max</sub> (%)	uo ×
										Start	End				Mean	SE
Rakobowchuk et al. [20]	NC	Non-ath	23.1	11	0.36	9	18	HIT	$100~\%P_{max}$	20	27	420	30	0.50	15.4	2.8
Siahkouhian et al. [21]	NC	Non-ath	19.1	12	1.00	8	24	HIT	All-out	9	7	191	30	0.13	13.9	3.0
Siahkouhian et al. [21]	NC	Ath	19.4	12	1.00	~	24	HIT	All-out	9	7	191	30	0.13	7.3	3.0
Trilk et al. [22]	C	Sed	30.1	14	0.00	4	12	HIT	All-out	4	7	99	30	0.13	13.4	2.5
Trilk et al. [22]	C	Sed	31.4	14	0.00	4	1	CON	1	ı	1	1	1	1	-0.5	2.5
Allemeier et al. [23]	C	Non-ath	22.7	11	1.00	9	15	HIT	All-out	3	3	45	30	0.03	12.5	2.8
Allemeier et al. [23]	C	Non-ath	24.0	9	1.00	9	ı	CON	ı	ı	1	1	ı	ı	-0.7	3.8
McKenna et al. [24]	NC	Non-ath	20.9	8	1.00	7	21	HIT	All-out	4	10	174	30	0.14	12.5	3.3
Macpherson et al. [25]	NC	Non-ath	24.3	10	09.0	9	18	HIT	All-out	4	9	06	30	0.13	11.5	2.9
Macpherson et al. [25]	NC	Non-ath	22.8	10	09.0	9	18	END	65 %VO <sub>2max</sub>	ı	ı	ı	2,700	I	12.5	2.9
Esfandiari et al. [26]	NC	Non-ath	24.5	~	1.00	2	9	HIT	95-100 %VO <sub>2max</sub>	∞	12	09	09	8.0	11.1	3.7
Esfandiari et al. [26]	NC	Non-ath	25.6	8	1.00	2	9	END	65 %VO <sub>2max</sub>	I	1	1	6,300	ı	4.5	3.7
Dunham and Harms [27]	NC	Non-ath	20.2	8	;	4	12	HIT	90 %P <sub>max</sub>	2	2	09	09	0.33	9.6	3.7
Dunham and Harms [27]	NC	Non-ath	21.3	7	;	4	12	END	$60-70 \% P_{max}$	I	1	1	2,700	ı	5.5	4.0
Bayati et al. [28]	C	Non-ath	25.0	~	1.00	4	12	HIT	All-out	3	4	48	30	0.13	9.6	4.0
Bayati et al. [28]	C	Non-ath	25.0	~	1.00	4	12	HIT	125 %P <sub>max</sub>	9	∞	96	30	0.25	6.7	4.1
Bayati et al. [28]	C	Non-ath	25.0	~	1.00	4	ı	CON	I	ı	ı	ı	ı	I	6.0—	5.5
Whyte et al. [29]	NC	Sed	32.1	10	1.00	2	9	HIT	All-out	4	9	30	30	0.11	9.5	3.2
Hazell et al. [30]	C	Non-ath	24.0	12	0.73	2	9	HIT	All-out	4	9	30	30	0.13	9.3	2.7
Hazell et al. [30]	C	Non-ath	24.0	12	;	2	ı	CON	I	I	ı	ı	ı	I	0.2	1.7
Jacobs et al. [31]	NC	Non-ath	27.0	16	1.00	2	9	HIT	$100~\%P_{max}$	∞	12	09	09	8.0	8.9	3.6
Sharp et al. [32]	NC	Non-ath	25.5	∞	1.00	~	32	HIT	All-out	∞	∞	256	30	0.13	8.3	3.3
Barnett et al. [33]	C	Non-ath	21.2	∞	1.00	∞	24	HIT	All-out	3	9	108	30	0.17	8.2	1.5
Barnett et al. [33]	C	Non-ath	21.2	∞	1.00	∞	ı	CON	I	ı	ı	ı	ı	ı	4.1	1.4
Harmer et al. [34]	C	Non-ath	24.0	7	0.57	7	21	HIT	All-out	4	10	174	30	0.14	8.2	3.5
Harmer et al. [34]	C	Non-ath	25.0	~	0.63	7	21	HIT	All-out	4	10	174	30	0.14	2.4	3.3
Shepherd et al. [35]	NC	Non-ath	22.0	~	1.00	9	18	HIT	All-out	4	9	06	30	0.11	7.6	3.7
Shepherd et al. [35]	NC	Non-ath	21.0	~	1.00	9	30	END	65 %VO <sub>2max</sub>	I	ı	ı	3,000	I	15.6	3.7
Burgomaster et al. [36]	NC	Non-ath	24.0	10	0.50	9	18	HIT	All-out	4	9	06	30	0.11	7.3	2.9
Burgomaster et al. [36]	NC	Non-ath	23.0	10	0.50	9	30	END	65 %VO <sub>2max</sub>	ı	ı	ı	3,000	ı	8.6	2.9
Bailey et al. [37]	C	Non-ath	21.0	∞	0.63	2	9	HIT	All-out	4	7	35	30	0.13	7.1	3.3
Bailey et al. [37]	C	Non-ath	21.0	~	0.63	2	9	END	90 %GET	I	1	1	1,200	I	0.0	3.3
Bailey et al. [37]	C	Non-ath	21.0	8	0.63	2	I	CON	I	I	ı	ı	I	I	-2.1	3.3
																1



Table 1 continued

References	Study design	Subjects Mean age, y		Sample size	Proportion of males	Duration (weeks)	Total sessions	Group	Exercise intensity <sup>a</sup>	No. of reps		Total reps	Rep duration (s)	Work/rest ratio	Effect on VO <sub>2max</sub> (%)	u
										Start	End				Mean	SE
MacDougall et al. [38]	NC	Non-ath	22.7	12	1.00	7	21	HIT	All-out	4	10	174	30	0.15	6.9	2.7
Tong et al. [39]	NC	Sed	22.9	8	0.25	9	18	HIT	120 %P <sub>max</sub>	20	20	360	30	0.50	9.9	3.3
Esfarjani and Laursen [40]	NC	Ath	19.0	9	1.00	10	20	HIT	130 %vVO <sub>2max</sub>	7	12	190	30	0.11	6.2	3.8
Esfarjani and Laursen [40]	NC	Ath	19.0	5	1.00	10	40	CON	$75 \% \text{vVO}_{2\text{max}}$	ı	1	1	3,600	ı	2.1	4.1
Burgomaster et al. [41]	NC	Non-ath	22.0	∞	1.00	2	9	HIT	All-out	4	7	30	30	0.13	5.5	3.2
Astorino et al. [42]	C	Non-ath	25.3	20	0.55	2	9	HIT	All-out	4	9	30	30	0.10	5.5	2.1
Astorino et al. [42]	C	Non-ath	22.8	6	0.55	2	ı	CON	ı	ı	1	1	ı	ı	1.6	3.1
Sandvei et al. [43]	NC	Non-ath	25.2	11	0.36	∞	24	HIT	All-out	5	10	189	30	0.17	5.1	2.8
Sandvei et al. [43]	NC	Non-ath	25.2	12	0.33	∞	24	END	70–80 %HR <sub>max</sub>	ı	1	1	2,700	ı	3.8	2.7
Rowan et al. [44]	NC	Ath	19.5	7	0.00	5	10	HIT	All-out	5	5	25	30	0.13	4.7	4.0
Rowan et al. [44]	NC	Ath	19.5	9	0.00	5	10	END	80 %VO <sub>2max</sub>	ı	1	1	2,400	ı	3.3	4.3
Stathis et al. [45]	NC	Non-ath	22.1	∞	0.75	7	21	HIT	All-out	3	10	153	30	0.13	4.2	3.3
Barnes et al. [46]	NC	Ath	24.9	5	1.00	9	12	HIT	$110~\% \text{vVO}_{2\text{max}}$	∞	16	74	40.5	0.33	3.6	4.7
Laursen et al. [47]	C	Ath	25.0	10	1.00	4	∞	HIT	175 %P <sub>max</sub>	12	12	96	30	0.11	3.1	2.9
Laursen et al. [47]	C	Ath	25.0	10	1.00	4	I	CON	ı	I	ı	ı	I	I	8.0	2.9
Harmer et al. [48]	NC	Non-ath	22.0	7	1.00	7	21	HIT	All-out	4	10	174	30	0.13	2.9	1.3
Laursen et al. [49]	C	Ath	23.5	7	1.00	2	4	HIT	100 %P <sub>max</sub>	20	20	80	09	0.25	2.3	3.5
Laursen et al. [49]	C	Ath	23.5	7	1.00	2	I	CON	ı	I	ı	ı	I	I	8.0-	3.5
Dalleck et al. [50]	NC	Non-ath	21.1	10	0.45	9	9	HIT	$110-120~\%P_{max}$	9	∞	42	30	0.14	-0.7	2.9
Dalleck et al. [50]	NC	Non-ath	21.1	10	0.45	9	12	HIT	$110-120~\%P_{max}$	9	∞	84	30	0.14	9.0-	2.9
Iaia et al. [51]	C	Ath	33.9	6	1.00	4	13.6	HIT	93 %V <sub>max</sub>	∞	12	124	30	0.17	-2.4	3.1
Iaia et al. [51]	C	Ath	33.9	8	1.00	4	16	CON	ı	I	ı	ı	3,138	ı	0.5	3.3

Studies are sorted from the largest to the smallest effects on VO<sub>2max</sub> in the intervention group

training incremental test), HIT low-volume high-intensity interval training group, NC non-controlled study, Non-ath non-athlete, Rep repetition, SE standard error, Sed sedentary, VO<sub>2max</sub> maximal oxygen uptake, %HR<sub>max</sub> intensity corresponding to a percentage of maximal heart rate (determined via a pre-training incremental test), %V<sub>max</sub> intensity corresponding to a percentage of maximum running velocity (determined via a pre-training 30-s all-out sprint run [51]), %VO<sub>2max</sub> intensity predicted to elicit a percentage of VO<sub>2max</sub> (on a treadmill [25] or on a cycle Ath athlete, C controlled study, CON control group, END endurance training group, GET gas exchange threshold (determined from a cluster of ventilatory measurements taken during a preergometer [26, 36], determined via a pre-training incremental test), %vVO<sub>2max</sub> percentage of the running speed predicted to elicit VO<sub>2max</sub> (determined during a pre-training incremental test [40, 46]), – indicates not applicable, ? indicates data not available

50]; a 'percentage of the highest 30 s power output completed' [47]; a 'percentage of peak work rate' [20, 39]; a 'percentage of final completed work rate maintained for 10 s' [28, 49]; a All-out: encompasses intensities described by the authors as "maximal" [24]; "near maximal" [43]; "sprints" [33]; "maximum efforts" [38, 44]; "supramaximal" [23]; "sprint training at the highest resistance maintained for 90 rpm" [32]; or "all-out" [21, 22, 25, 28–30, 34–37, 41, 42, 45, 48]. %P<sub>max</sub> encompasses intensities described as either a 'percentage of peak watt workload' percentage of peak power output [31]; a 'percentage of their final workload' [27] (all determined via a pre-training incremental test)



Table 2 Study and subject characteristics for peak power output included in the meta-analysis

References	Study	Subjects		Sample	Proportion	Duration	Total	Group	Exercise intensity a	No. c	No. of reps	Total	Rep	Work/rest	Effect on	on
	design		age, y	3776	OI IIIIaics	(wccks)	SCSSIONS		IIIICIISIII			rchs	(s)	Tano	power (%)	(%)
										Start	End				Mean	SE
Stathis et al. [45]	NC	Non-ath	22.1	∞	0.75	7	21	HIT	All-out	ж	10	153	30	0.13	17.1	2.9
Burgomaster et al. [52]	NC	Non-ath	22.0	∞	0.75	2	9	НІТ	All-out	4	7	30	30	0.25	15.7	2.9
Siahkouhian et al. [21]	NC	Non-ath	19.1	12	1.00	∞	24	нт	All-out	9	10	191	30	0.13	14.4	2.4
Forbes et al. [53]	NC	Non-ath	21.0	7	0.57	2	9	HIT	All-out	4	9	30	30	0.13	11.4	3.1
MacDougall et al. [38]	NC	Non-ath	22.7	12	1.00	7	21	HIT	All-out	4	10	174	30	0.13	10.6	2.4
Bayati et al. [28]	C	Non-ath	25.0	8	1.00	4	12	HIT	All-out	$\mathcal{E}$	4	48	30	0.13	10.3	2.7
Bayati et al. [28]	C	Non-ath	25.0	8	1.00	4	12	HIT	125 %P <sub>max</sub>	9	∞	96	30	0.25	7.5	3.1
Bayati et al. [28]	C	Non-ath	25.0	∞	1.00	4	I	CON	I	I	ı	ı	I	I	-0.4	3.0
Harmer et al. [48]	NC	Non-ath	22.0	7	1.00	7	21	HIT	All-out	4	10	174	30	0.14	9.4	3.1
Barnett et al. [33]	C	Non-ath	21.2	∞	1.00	∞	24	HIT	All-out	3	9	108	30	0.17	7.7	2.9
Barnett et al. [33]	C	Non-ath	21.2	∞	1.00	∞	I	CON	I	I	ı	ı	I	ı	8.9	2.0
Siahkouhian et al. [21]	NC	Ath	19.4	12	1.00	∞	24	НІТ	All-out	9	10	191	30	0.13	7.9	2.4
Richards et al. [54]	NC	Non-ath	29.0	12	0.42	2	9	HIT	All-out	4	7	30	30	0.13	6.9	2.4
McKenna et al. [24]	NC	Non-ath	20.9	∞	1.00	7	21	HIT	All-out	4	10	174	30	0.14	5.8	2.9
Esbjörnsson Liljedahl et al. [55]	NC	Non-ath	25.0	10	0.00	4	12	НІТ	All-out	ю	8	27	30	0.03	5.6	2.6
Richards et al. [54]	NC	Non-ath	25.0	11	0.27	2	9	HIT	All-out	4	7	30	30	0.13	5.5	2.5
Burgomaster et al. [41]	NC	Non-ath	21.0	∞	1.00	2	9	HIT	All-out	4	7	30	30	0.13	5.4	2.1
Whyte et al. [29]	NC	Sed	32.1	10	1.00	2	9	HIT	All-out	4	9	26	30	0.11	4.7	3.3
Allemeier et al. [23]	C	Non-ath	22.7	11	1.00	9	15	HIT	All-out	8	8	45	30	0.03	3.0	2.5
Allemeier et al. [23]	C	Non-ath	24.0	9	1.00	9	I	CON	ı	I	ı	1	ı	ı	3.0	3.3
Jansson et al. [56]	NC	Non-ath	27.0	7	1.00	4	10	HIT	All-out	3	3	30	30	0.03	2.4	3.1
McKenna et al. [57]	NC	Non-ath	18.8	9	1.00	7	21	HIT	All-out	4	10	174	30	0.13	2.3	3.3
Esbjörnsson	NC	Non-ath	26.0	9	1.00	4	12	HIT	All-out	3	3	27	30	0.03	1.4	3.3
Lujedani et ali. [33]																

Ath athlete, C controlled study, CON control group, HIT low-volume high-intensity interval training group, NC non-controlled study, Non-ath non-athlete, Rep repetition, SE standard error, Sed sedentary, %P<sub>max</sub> percentage of the final completed work rate maintained for 10 s during a pre-training incremental test [28], – indicates not applicable Studies are sorted from the largest to the smallest effects (intervention group) on peak power output

<sup>a</sup> All-out: encompasses intensities described as either "maximal" [24, 53, 55, 57]; "sprints" [33]; "maximum efforts" [38]; "supramaximal" [23]; and "all-out" [21, 28, 29, 41, 45, 48, 52, 54,



relevant performance measures, which may limit the application of our findings to athletic populations and sports performance. The recent meta-analysis by Bacon et al. [7] has, to an extent, addressed this gap in the literature. However, the number of studies excluded from the present study on interval duration, intensity and other measures of aerobic fitness (e.g. velocity at  $VO_{2max}$ , speed at lactate threshold, running economy and sports-specific tests) underscores the need for a dedicated review of studies using longer intervals at lower intensities.

To select relevant papers, all titles were initially screened by two authors (KT, MW) during the electronic searches to exclude studies that were beyond the scope of this meta-analysis. Following this initial selection process, there were 550 potentially eligible studies (Fig. 1). All study titles and abstracts were then screened independently by the same authors. Full-text versions of the remaining papers that met each of the eligibility criteria were then reviewed by these authors to determine final inclusion in the meta-analysis. Any disputed studies were taken to a third reviewer (AMB) for resolution. The final dataset for  $VO_{2\text{max}}$  consisted of 55 estimates from 32 trials, 11 of which were controlled trials. For peak sprint power, the final dataset consisted of 23 estimates from 16 trials, three of which were controlled trials. For mean sprint power, the dataset consisted of 19 estimates from 12 trials, three of which were controlled trials.

## 2.3 Data Extraction

Graph digitizer software (DigitizeIt, Germany) was used to obtain data values in studies where only plots were published. Accuracy was confirmed via intra- and interindividual reassessments of data extraction. Mean effects on VO<sub>2max</sub>, peak and mean sprint power in training and control groups were converted to a percentage change. For each converted effect, standard errors were calculated to indicate the level of imprecision. In studies where exact p values were given ( $VO_{2\text{max}}$  n = 7; peak power n = 4; mean power n = 5), standard errors were calculated directly via the corresponding t statistic and its degrees of freedom. Under the assumption that studies with similar test protocols and subject characteristics would have similar typical errors of measurement, the typical errors from these studies were then averaged (via the weighted mean variance) and assigned to the studies that did not report an exact p value. The standard error was then calculated via the relationship between typical error and standard error [17, 18]. Descriptive statistics for studies included in the metaanalysis for VO<sub>2max</sub>, peak and mean sprint power are shown in Tables 1, 2 and 3, respectively.

## 2.4 Publication Bias and Outliers

To investigate the extent of publication bias, we examined the standard error against the t value for each predicted effect for each outcome, and inspected the plot for signs of asymmetrical scatter [12]. Such a plot is an improved version of the funnel plot, as the scatter of the effects is adjusted for any uncertainty in the estimates and for the contribution of study covariates. Examination of these plots revealed no evidence of the asymmetrical scatter associated with publication bias.

# 2.5 Meta-Analytic Model

The general linear mixed-model procedure (Proc Mixed) in the Statistical Analysis System (Version 9.2, SAS Institute, Cary, NC, USA) was used to perform the meta-analysis. Fixed effects in the model included type of study (controlled, uncontrolled), study-level subject characteristics (sex, training status, baseline VO<sub>2max</sub>, peak and mean sprint power) and training parameters (number of training sessions, repetition duration, work/rest ratio). We determined the predicted effect of reference training conditions on VO<sub>2max</sub>, peak and mean sprint power using mean values of baseline fitness, number of sessions and work/rest ratio from all eligible studies. Performance effects were then calculated as the predicted effect under these reference training conditions. The modifying effects of predictors were also calculated, either as differences between levels of a nominal covariate (i.e. male/female, non-athletic/sedentary) or as the effect of approximately two standard deviations (SDs) of a numeric covariate (i.e. a typically high value minus a typically low value) [12]. Random effects in the model were the usual between-subject random effects and a novel within-study random effect to account for within-study repeated measurements (a control treatment and/or more than one training treatment). The residual was set to unity to properly weight the estimates by the inverse of the square or their standard errors. Unexplained true variation within and between studies was estimated by combining the variances for the random effects and was expressed as an SD. The SD was doubled before interpreting its magnitude with the scale used to interpret fixed effects [19], for the same reason that the magnitude of the effect of a linear covariate is evaluated with two SDs of the covariate [12].

# 2.5.1 Outcome Statistics

We expressed the uncertainty in the estimates of effects on fitness as 90 % confidence limits (CL) and as probabilities that the true value of the meta-analysed effect was trivial, beneficial or harmful in relation to threshold values for



Table 3 Study and subject characteristics for mean power output included in the meta-analysis

		•		•		•										
References	Study	Subjects Mean	Mean	Sample	Proportion of moles	Duration	Total	Treatment	Exercise	No. of reps		_	Rep	Work/	Effect on	uc
	uesigii		age, y	SIZE	oi maies	(weeks)	SCSSIOIIS	dnoig	mensity			reps	duration (s)	rest ratio	mean power (%)	(%)
										Start	End				Mean	SE
Siahkouhian et al. [21]	NC	Non-ath	19.1	12	1.00	8	24	HIT	All-out	9	10	191	30	0.13	17.5	3.1
Bayati et al. [28]	C	Non-ath	25.0	8	1.00	4	12	HIT	All-out	3	4	48	30	0.13	17.2	6.1
Bayati et al. [28]	C	Non-ath	25.0	8	1.00	4	12	HIT	125 %P <sub>max</sub>	9	∞	96	30	0.25	11.2	6.7
Bayati et al. [28]	C	Non-ath	25.0	∞	1.00	ı	ı	CON	ı	ı	1	1	ı	ı	0.3	4.4
Siahkouhian et al. [21]	NC	Ath	19.4	12	1.00	~	24	HIT	All-out	9	10	191	30	0.13	11.1	3.1
Stathis et al. [45]	NC	Non-ath	22.1	∞	0.75	7	21	HIT	All-out	3	10	153	30	0.13	10.7	3.8
Esbjörnsson Liljedahl et al. [55]	NC	Non-ath	25.0	10	0.00	4	12	HIT	All-out	3	3	27	30	0.03	10.1	2.9
Burgomaster et al. [41]	NC	Non-ath	21.0	∞	1.00	2	9	HIT	All-out	4	7	30	30	0.13	8.7	2.9
Barnett et al. [33]	C	Non-ath	21.2	~	1.00	~	24	HIT	All-out	3	9	108	30	0.17	7.1	1.6
Barnett et al. [33]	C	Non-ath	21.2	8	1.00	ı	ı	CON	ı	ı	1	1	ı	ı	2.8	1.0
McKenna et al. [24]	NC	Non-ath	20.9	~	1.00	7	21	HIT	All-out	4	10	174	30	0.14	6.1	3.8
Allemeier et al. [23]	C	Non-ath	22.7	11	1.00	9	15	HIT	All-out	3	8	45	30	0.03	5.0	3.2
Allemeier et al. [23]	C	Non-ath	24.0	9	1.00	9	ı	CON	I	ı	1	ı	ı	ı	5.0	4.4
Forbes et al. [53]	NC	Non-ath	21.0	7	0.57	2	9	HIT	All-out	4	9	30	30	0.13	4.5	1.3
Richards et al. [54]	NC	Non-ath	25.0	11	0.27	2	9	HIT	All-out	4	7	30	30	0.13	3.9	3.2
Whyte et al. [29]	NC	Sed	32.1	10	1.00	2	9	HIT	All-out	4	9	26	30	0.11	3.6	1.5
Jansson et al. [56]	NC	Non-ath	27.0	7	1.00	4	10	HIT	All-out	3	8	30	30	0.03	2.8	4.1
Esbjörnsson Liljedahl et al. [55]	NC	Non-ath	26.0	9	1.00	4	12	HIT	All-out	3	8	27	30	0.03	1.4	3.7
Richards et al. [54]	NC	Non-ath	29.0	12	0.42	2	9	HIT	All-out	4	7	30	30	0.13	1.1	3.1

Ath athlete, C controlled study, CON control group, HT low-volume high-intensity interval training group, NC non-controlled study, Non-ath non-athlete, Rep repetition, SE standard error, Sed sedentary, %P<sub>max</sub> percentage of the final completed work rate maintained for 10 s during a pre-training incremental test [28], – indicates not applicable Studies are sorted from the largest to the smallest effects (intervention group) on mean power output

<sup>a</sup> All-out: encompasses intensities described as either "maximal" [24, 53, 55]; "sprints" [33]; "supramaximal" [23]; and "all-out" [21, 28, 29, 41, 45, 54, 56]



benefit and harm. Probabilities were then used to make a qualitative probabilistic inference about the effect [12]. Given that improved aerobic functioning and power output have clinical application [3-7, 15, 16], main treatment effects were considered unclear if the chance of benefit (improved fitness) was high enough to warrant use of the intervention but with an unacceptable risk of harm (reduced fitness). An odds ratio of benefit to harm of <66 was used to identify such unclear effects. This ratio corresponds to a borderline possibly beneficial effect (25 % chance of benefit) and a borderline most unlikely harmful effect (0.5 % risk of harm). All other effects were deemed clinically clear and inference made via estimation of the probability that the true magnitude of the effect was at least as large as our pre-specified thresholds. In the absence of robust anchors for the smallest worthwhile clinical and practical effect on VO<sub>2max</sub> and sprint power, our inferences were based on standardized thresholds for small, moderate and large changes of 0.2, 0.6 and 1.2 SDs, respectively [12], and derived by averaging appropriate between-subject variances for baseline VO<sub>2max</sub>, peak and mean sprint power. For VO<sub>2max</sub>, magnitude thresholds were 3.2, 9.6 and 19.2 % for sedentary subjects, 1.4, 4.1 and 8.1 % for active non-athletic subjects, and 1.4, 4.2 and 8.4 % for athletic subjects. For peak and mean sprint power, thresholds were 1.7, 5.1 and 10.3 and 1.7, 5.2 and 10.5 %, respectively, for male subjects. The chance of the true effect being trivial, beneficial or harmful was interpreted using the following scale: <0.5 % most unlikely; 0.5–5 % very unlikely; 5–25 % unlikely; 25–75 % possibly; 75–95 % likely; 95-99.5 % very likely; >99.5 % most likely [12]. Modifying effects were evaluated non-clinically and deemed unclear if the 90 % CL overlapped the thresholds for the smallest worthwhile positive and negative effects [12].

## 3 Results

# 3.1 Maximum Oxygen Consumption

The meta-analysed effects on  $VO_{2max}$  of an average low-volume HIT protocol in a controlled trial are shown in Table 4. When compared with control, moderate improvements in  $VO_{2max}$  were likely for active non-athletic males and possible for sedentary males and active non-athletic females. A small improvement in  $VO_{2max}$  was likely for sedentary females. The effect on athletic males was unclear. With the exception of a possible moderate additional increase in  $VO_{2max}$  for subjects with a lower baseline value, the effects of all modifiers were unclear. The comparison of HIT with endurance training was unclear (-1.6~%;~90~% CL  $\pm 4.3~\%$ ). Unexplained variation expressed as a between-study SD was 2.0~%  $(\pm 2.7)$ .

## 3.2 Sprint Power

The meta-analysed effects of low-volume HIT on 30-s Wingate peak and mean sprint power in a controlled trial are shown in Tables 5 and 6, respectively. With the exception of a possibly moderate improvement in the peak sprint power of controls, all mean effects on sprint power were unclear. There were possibly moderate and likely small improvements in mean and peak sprint power, respectively, following a threefold increase in the number of training sessions. A moderately beneficial improvement in peak sprint power with a greater work/rest ratio was possible and a small additional increase in mean sprint power was possible for subjects with a lower baseline value. All other modifying effects were unclear. Unexplained variation between studies was  $2.4 \pm 2.5$  and  $1.0 \pm 2.9$  % for peak and mean sprint power, respectively.

# 4 Discussion

In the previous meta-analyses of Sloth et al. [10] and Gist et al. [11], low-volume HIT improved aerobic fitness and Wingate sprint power, but the effects on different subject groups and other modifying effects were either not analysed or not presented. Our meta-analysis broadens the scope of these previous reviews, as it is the first to include study and subject characteristics in the analysis. Our data revealed HIT to have an apparent adaptive effect on  $VO_{2max}$  that favours the less fit. Despite HIT effectively representing repeated Wingate tests, there was no clear effect on measures of performance in the test.

We found that a mean protocol of 13 HIT sessions with a work/rest ratio of 0.16 led to moderate improvements in the  $VO_{2\text{max}}$  of sedentary and non-athletic males and females. This main finding is consistent with the recent work of Sloth et al. [10], Gist et al. [11] and Bacon et al. [7], who reported standardized moderate effects on  $VO_{2\text{max}}$  for HIT and high-intensity aerobic interval training, respectively. A combination of central and peripheral adaptations promoting an enhanced availability, extraction and utilization of oxygen may explain such improvements following intensive interval-training protocols. However, mechanisms responsible for increased  $VO_{2\text{max}}$  following HIT were not the focus of our review. Comprehensive reviews of the possible underlying mechanisms are available elsewhere [9, 10].

Gibala et al. [58] reported low-volume HIT to be a timeefficient strategy for rapid physiological and performance improvements that are comparable to improvements following traditional endurance training. The random effects component of our mixed model enabled us to include studies where the reference group was traditional



**Table 4** Effects of low-volume high-intensity interval training on maximum oxygen consumption following reference training, with modifying effects for study characteristics, subject characteristics and training parameters

	Effect VO <sub>2ma</sub>		Inference
	Mean	±90 % CL	
Effect on treatment groups <sup>a</sup>			
Sedentary males	10.0	±5.1	Possibly moderate ↑
Sedentary females	7.3	$\pm 4.8$	Likely small ↑
Active non-athletic males	6.2	±3.1	Likely moderate ↑
Active non-athletic females	3.6	±4.3	Possibly moderate ↑
Athletic males	2.7	$\pm 4.6$	Unclear
Controls	1.2	$\pm 2.0$	Unclear
Modifying effects			
Baseline $VO_{2\text{max}}$ lower by $10 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$	3.8	±2.5	Possibly moderate ↑
Athlete vs. active non-athlete	2.4	$\pm 5.7$	Unclear
Threefold increase in work/rest ratio	-0.3	±2.0	Unclear
Threefold increase in no. of sessions	-0.3	±2.0	Unclear
Uncontrolled study	-0.7	$\pm 2.3$	Unclear
Sedentary vs. active non-athlete	-2.2	$\pm 5.7$	Unclear
Females	-2.5	$\pm 4.1$	Unclear
Replacement of training (male athletes only)	-2.9	±5.3	Unclear

Effects on treatment groups are presented as intervention minus control

Reference training: a controlled study of 13 low-volume HIT sessions with a work/rest ratio of 0.16 (0.14 for sedentary females)

CL confidence limit, HIT low-volume high-intensity interval training,  $VO_{2max}$  maximal oxygen uptake,  $\uparrow$  indicates increase

endurance training rather than no training. Here, the comparison between the two types of training was unclear. This finding is consistent with that of Gist et al. [11], who reported a trivial effect of HIT on  $VO_{2\text{max}}$  when compared with endurance training controls. More studies are therefore required to examine the effectiveness of HIT versus traditional endurance training for training-induced endurance gains. The effect of HIT on  $VO_{2\text{max}}$  was greater for the less fit, which is consistent with training in general having greater effects on the less fit [59]. For already highly trained athletes who replaced their usual training

**Table 5** Effects of low-volume high-intensity interval training on 30-s Wingate peak sprint power following reference training, with modifying effects for study characteristics, subject characteristics and training parameters

	Effect power	on peak (%)	Inference
	Mean	±90 % CL	
Effect on treatment groups <sup>a</sup>			
Males	1.8	$\pm 5.0$	Unclear
Controls	4.5	±3.8	Possibly moderate ↑
Modifying effects			
Fivefold increase in work/rest ratio	5.7	±3.5	Possibly moderate ↑
Threefold increase in sessions	2.9	$\pm 3.5$	Likely small ↑
Females	2.0	$\pm 6.3$	Unclear
Baseline peak power lower by 5 W/kg	1.6	±3.2	Unclear
Uncontrolled study	-1.0	±3.7	Unclear

Effects on treatment groups are presented as intervention minus control

Reference training: a controlled study of 12 low-volume HIT sessions with a work/rest ratio of 0.10

*CL* confidence limit, *HIT* low-volume high-intensity interval training, ↑ indicates increase

with HIT, as opposed to adding the HIT, the effect on  $VO_{2\text{max}}$  was unclear. This finding also indicates the need for more research, providing that elite athletes can be convinced to experiment with their normal training programmes [9]. Despite reporting no analytical data for the potentially modifying effect of training duration, Sloth et al. [10] and Gist et al. [11] reported no clear effects of the length of HIT intervention on the magnitude of  $VO_{2\text{max}}$  improvement. The data presented in our more extensive meta-analysis have still not resolved this issue.

On the basis of the CLs, low-volume HIT had an unclear effect on peak and mean sprint power that could at most be a moderate beneficial or a small harmful effect. These results contrast with those of Sloth et al. [10], who reported enhanced peak and mean power following HIT. Their assertion was based on nine studies [24, 28–30, 33, 36, 41, 42, 52], without a meta-analysis of the mean effect and its uncertainty. Three of these studies [30, 36, 42] were excluded from our analysis, owing to difficulties in obtaining precise baseline and post-intervention data during the data extraction process. An enhanced sprint power following HIT was expected, given that all-out training increases enzymatic activity related to anaerobic metabolism [13]. Furthermore, studies showing strong similarities between testing and training routines are more likely to



 $<sup>^{\</sup>rm a}$  Active non-athletic males: baseline  $VO_{\rm 2max}$  adjusted to 45 mL·kg $^{-1}$ ·min $^{-1}$ . Sedentary males: baseline  $VO_{\rm 2max}$  adjusted to 30 mL·kg $^{-1}$ ·min $^{-1}$ . Active non-athletic females: baseline  $VO_{\rm 2max}$  adjusted to 45 mL·kg $^{-1}$ ·min $^{-1}$ . Sedentary females: baseline  $VO_{\rm 2max}$  adjusted to 30 mL·kg $^{-1}$ ·min $^{-1}$ . Athletic males: baseline  $VO_{\rm 2max}$  adjusted to 60 mL·kg $^{-1}$ ·min $^{-1}$ 

<sup>&</sup>lt;sup>a</sup> Males, with baseline peak power output adjusted to 11.5 W/kg

**Table 6** Effects of low-volume high-intensity interval training on 30-s Wingate mean sprint power following reference training, with modifying effects for study characteristics, subject characteristics and training parameters

	Effect power	on mean (%)	Inference
	Mean	±90 % CL	
Effect on treatment groups <sup>a</sup>			
Males	2.2	$\pm 10.3$	Unclear
Controls	2.8	$\pm 8.2$	Unclear
Modifying effects			
Threefold increase in sessions	6.2	±3.9	Possibly moderate ↑
Baseline mean power lower by 4 W/kg	2.3	±3.7	Possibly small
Fivefold increase in work/rest ratio	1.5	±3.7	Unclear
Females	-0.1	$\pm 6.9$	Unclear
Uncontrolled study	-2.3	$\pm 4.3$	Unclear

Effects on treatment groups are presented as intervention minus control

Reference training: a controlled study of 14 low-volume HIT sessions with a work/rest ratio of 0.09

CL confidence limit, HIT low-volume high-intensity interval training, ↑ indicates increase

show training improvements [60]. However, when measured relative to controls, the meta-analysed effect of HIT on sprint power was unclear. Improvements of 4.5 % in peak sprint power and 2.8 % in mean sprint power of control subjects may have represented a learning effect on the Wingate test or provide some evidence of compensatory rivalry (e.g. greater effort by controls). There was some evidence of a dose–response relationship and a greater effect for the less fit. The finding of a possibly moderate enhancement in peak sprint power with a fivefold increase in repetition work/rest ratio could be explained by greater phosphocreatine resynthesis in the recovery phase [61, 62].

There was considerable uncertainty in the SDs representing the residual between-study variation in the mean effect of the treatment on the three measures of fitness, but in this sample of studies the observed magnitudes (after doubling the SDs) were small to moderate, depending on the measure of performance and the subject group. This SD needs to be added to and subtracted from the main effect to evaluate the magnitude of the HIT treatment in a specific setting. For example, the mean effect of HIT on  $VO_{2\text{max}}$  for active non-athletic males (6.2 %; moderate) in any given

setting could be anywhere from 4.2 % (very likely small) to 8.2 % (possibly large). Such differences between the effects of training in the different studies presumably reflect differences in subject characteristics and training protocols that are not properly accounted for by the published data. Some data may also have been analysed or reported erroneously.

We propose several areas for future research, along with suggestions for those publishing research in this area. Given that the age of participants included within our metaanalysis was mainly young adults, it is evident that research is required to clarify the effects of low-volume HIT in older populations. Moderating effects of changing the exercise dose on VO<sub>2max</sub> were unclear, as was the replacement of athletes' usual aerobic training with HIT, indicating that more research is necessary to investigate these predictors. However, we do recommend that modifying effects are interpreted with slight caution, as when a covariate is a subject characteristic averaged over study subjects, the observed meta-regression relationships might not hold at the individual study level [63]. The practicality of low-volume HIT warrants further investigation, given that repeated bouts of maximal exercise require high levels of motivation [9]. Adherence to unsupervised training also needs investigation [29]. We concur with the need to test the effectiveness of low-volume HIT via large-scale, multicentre, randomized clinical trials in various clinical populations and on long-term clinical outcome measures [64]. Of further benefit would be the reporting of full inferential statistics, such as SD of change scores or exact p values in training and control groups, to enable meta-analysis of the magnitude of individual responses. Finally, the findings of a training study are of very little or no value without precise information of the training itself [65]. We therefore encourage authors to report physiological responses during HIT sessions, as this practice will help to demonstrate that the fidelity of an intervention has been upheld for all subjects.

# 5 Conclusions

Low-volume HIT is increasingly being used for aerobic adaptations previously achieved with traditional endurance training. Our meta-analysis provides evidence of substantial improvements in the endurance fitness of sedentary and non-athletic subjects following repeated bouts of brief maximal intermittent exercise. The effect of HIT on sprint power should be determined with more studies.

**Acknowledgments** No sources of funding were used to assist in the preparation of this review. The authors have no conflicts of interest that are directly relevant to the content of this review.



<sup>&</sup>lt;sup>a</sup> Males, with baseline mean power output adjusted to 7.6 W/kg

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