# The Effect of Training Intensity on VO<sub>2</sub>max in Young Healthy Adults: A Meta-Regression and Meta-Analysis

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#### **ABSTRACT**

International Journal of Exercise Science 9(2): 230-247, 2016. Exercise training at a variety of intensities increases maximal oxygen uptake (VO2max), the strongest predictor of cardiovascular and all-cause mortality. The purpose of the present study was to perform a systematic review, meta-regression and meta-analysis of available literature to determine if a doseresponse relationship exists between exercise intensity and training-induced increases in VO<sub>2</sub>max in young healthy adults. Twenty-eight studies involving human participants (Mean age: 23±1 yr; Mean VO₂max: 3.4±0.8 1 min<sup>-1</sup>) were included in the meta-regression with exercise training intensity, session dose, baseline VO2max, and total training volume used as covariates. These studies were also divided into 3 tertiles based on intensity (tertile 1: ~60-70%; 2: ~80-92.5%; 3: ~100-250% VO<sub>2</sub>max), for comparison using separate meta-analyses. The fixed and random effects metaregression models examining training intensity, session dose, baseline VO<sub>2</sub>max and total training volume was non-significant (Q4=1.36; p=0.85; R<sup>2</sup>=0.05). There was no significant difference between tertiles in mean change in VO<sub>2</sub>max (tertile 1:+0.29±0.15 l/min, ES (effect size) =0.77; 2:+0.26±0.10 l/min, ES=0.68; 3:+0.35±0.17 l/min, ES=0.80), despite significant (p<0.05) reductions in session dose and total training volume as training intensity increased. These data suggest that exercise training intensity has no effect on the magnitude of training-induced increases in maximal oxygen uptake in young healthy human participants, but similar adaptations can be achieved in low training doses at higher exercise intensities than higher training doses of lower intensity (endurance training).

KEY WORDS: Maximal oxygen uptake, exercise training, exercise intensity, training volume, training dose, intensity dose-response, young adults.

## INTRODUCTION

Over the past 30 years maximal aerobic capacity (VO<sub>2</sub>max) has emerged as a strong predictor of adverse health outcomes such as cardiovascular disease and all-cause mortality (32, 42). Exercise training is an effective means of achieving improvements

in  $VO_2$ max, with a rise of one metabolic equivalent (3.5 ml  $O_2$  ·kg<sup>-1</sup> min<sup>-1</sup>) in  $VO_2$ max associated with a 10-25% improvement in survival (30). Thus, exercise training represents a potentially important preventative approach to reduce the risk of disease development in currently healthy adults. Similar to any form of preventative

medicine, there is a need for exercise prescription to be optimized with the goal of prescribing the most effective exercise intensity for improving VO<sub>2</sub>max.

Despite the obvious importance identifying the optimal intensity of exercise training for improving VO<sub>2</sub>max, there is surprisingly little evidence available describing what this intensity, or range of intensities might be. **Exercise-training** programs consisting of extended duration, continuous exercise at a moderate intensity (endurance training; END) have long been known to improve VO<sub>2</sub>max (16,24). More recently repeated intervals of short duration high-intensity exercise (interval training) have been demonstrated to be an effective alternative to END for improving VO<sub>2</sub>max (5, 8, 16, 26, 32, 38, 42). Interestingly, while several researchers report the potency of training high-intensity at improving VO<sub>2</sub>max, these studies frequently use small samples sizes (N:10-27) and employ one (1, 3, 14, 27, 30, 36, 37, 40, 43, 45, 48, 54, 59) or two training intensities (4-8, 15, 16, 21, 24, 26, 33, 35, 38, 49-53, 57), or do not extend to supramaximal exercise intensities (4, 6, 11, 15, 18, 21, 25, 27, 29, 31, 33, 37, 39, 43, 46, 49, 51, 52, 59). Unfortunately, as a result of these limitations, none of these reports adequately the intensity dose-response describe relationship with training-induced increases in VO<sub>2</sub>max. While many investigators have reported the ability of a wide range of both high and low intensity exercise-training intensities to improve VO<sub>2</sub>max, whether an optimal intensity exists for increasing VO<sub>2</sub>max remains unclear. This represents a critical gap in our understanding of the response to exercise training.

While individual studies have failed to characterize the optimal training intensity, the combination of accumulated results through meta-regression and meta-analysis may provide additional information on the impact of training intensity on VO<sub>2</sub>max. This approach will allow for both an examination of the magnitude of training-induced increases in VO<sub>2</sub>max across a large range of training intensities (i.e. submaximal continuous to supra-maximal "all-out" interval exercise) and the larger sample size will improve the external validity (i.e. generalizability) of results obtained from numerous smaller training studies. Thus, the purpose of the present study was to systematic review, conduct a metaregression and meta-analysis of existing literature in an attempt to determine the effect of exercise intensity on traininginduced increases in VO<sub>2</sub>max. In addition, we examined the impact of training session dose, baseline VO<sub>2</sub>max and total training volume on increases in VO<sub>2</sub>max. It is hoped that this work will help generate hypotheses for future studies aimed at determining the optimal exercise dose (both intensity and duration) for improving VO<sub>2</sub>max in healthy adults.

#### **METHODS**

Systematic Review

An extensive review of the available literature was performed to identify articles that evaluated the effect of exercise training on changes in aerobic capacity. For the purposes of this study, aerobic capacity was defined as the VO<sub>2</sub>max (or peak) value obtained from an incremental exercise test to volitional fatigue, while exercise training intensity was defined as a percentage of aerobic capacity. The review was conducted

between August and December, 2013 and consisted of searches of the PubMed database using the search terms: "VO<sub>2</sub>max" and "exercise training" or "high intensity interval training" or "endurance training" or "sprint interval training". Studies of interest were limited to those involving humans.

#### Inclusion and Exclusion Criteria

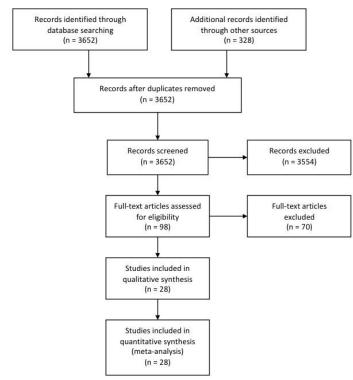
Articles following our inclusion criteria were considered for the systematic review: 1) the effects of training were examined in healthy human participants, 2) mean and standard deviation (SD)/standard error of the mean (SEM) were reported for VO<sub>2</sub>max both before and after training, 3) VO<sub>2</sub>max was either reported as absolute values (l/min) or were reported as relative values (ml/kg/min) and were accompanied by body mass such that absolute values could be calculated, 4) details regarding the sample size (n) intervention length (weeks), training frequency (number of sessions per week), exercise intensity (percentage of VO<sub>2</sub>max), and training protocol (duration, rest periods, interval length) were either expressly reported or could be calculated from the data presented, and 5) training protocol utilized was consistent throughout the intervention (i.e. either continuous steady state, or interval training was performed but not both). Studies were omitted for the following reasons: 1) any of the above criteria were not met, 2) participant population was not free of chronic illness. and 3) the training intervention examined was less than four weeks, or greater than eight weeks in duration in order to establish the impact of short to moderate term training (4-8 weeks) on VO2max. Shorter duration interventions (i.e. < 4 weeks) were excluded in order to eliminate the presence of little, or no change

in VO<sub>2</sub>max due to insufficient training duration impacting the analyses.

## Selection of Studies

Following a preliminary review of titles and abstracts of the initial articles identified using the search terms outlined above, 98 articles were further evaluated utilizing our full inclusion/exclusion criteria. Based on this criteria, a further 70 articles were excluded resulting in a total of 40 study groups from 28 articles being included in the meta-regression/analyses (Fig. 1). These 28 articles were evaluated for methodological quality (Table 1) using the modified Physiotherapy Evidence Base Database (PEDro) scale (56). Characteristics of each study are outlined in Table 2.

Figure 1



**Figure 1.** Study Selection Flow Diagram. Flow chart demonstrating the process of study selection. Studies included in quantitative analysis refers to the number of publications from which study groups (n=40) were extracted.

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**Table 1:** Methodological quality of training programs included in the present study as evaluated using a modified Physiotherapy Evidence Base Database (PEDro) scale.

Study	1	2	3	4	5	6	7	8	9	10	Total
Allemeier et al. (1)	0	0	0	0	0	1	1	1	1	0	4
Ben Abderrahman et al. (3)	0	1	0	1	0	1	1	1	1	1	7
Burgomaster et al. (5)	1	0	0	1	0	1	1	1	1	0	6
Burke et al. (6)	0	0	0	1	0	1	1	1	1	0	5
Clark et al. (7)	1	1	0	1	0	1	1	1	1	0	7
Connolly et al. (11)	0	1	0	0	0	1	1	1	1	0	5
Duffield et al. (14)	0	N/A	N/A	N/A	0	1	1	N/A	1	0	3
Dunham et al. (15)	1	1	0	1	0	1	1	1	1	0	7
Emonson et al. (18)	1	1	0	1	0	1	1	1	1	0	7
Hautala et al. (27)	1	N/A	N/A	N/A	0	1	1	N/A	1	1	5
Helgerud et al. (28)	0	1	0	1	0	1	1	1	1	1	7
Helgerud et al. (29)	0	1	0	1	0	1	1	1	1	0	6
Kargotich et al. (31)	0	1	0	1	0	1	1	1	1	0	6
Kim et al. (33)	0	1	0	1	0	1	1	1	1	0	6
Macpherson et al. (35)	0	0	0	1	0	1	1	1	1	0	5
Marles et al. (36)	0.5	N/A	N/A	N/A	0	1	1	N/A	1	0	3.5
McMillan et al. (37)	0	N/A	N/A	N/A	0	1		N/A	1	0	2
Mendes et al. (39)	0	1	0	1	0	1	1	1	1	0	6
Metcalfe et al. (40)	1	1	0	1	0	1	1	1	1	0	7
Perry et al. (43)	1	N/A	N/A	N/A	0	1	1	N/A	1	0	4
Poole et al. (45)	0	N/A	N/A	N/A	0	1	1	N/A	1	0	3
Prieur et al. (46)	0	1	0	N/A	0	1	1	1	1	0	5
Sugawara et al. (48)	0	N/A	N/A	N/A	0	1	1	N/A	1	0	3
Swart et al. (49)	0	1	0	1	0	1	1	1	1	1	7
Tabata et al. (50)	0	N/A	N/A	N/A	0	1	1	N/A	1	0	3
Trilk et al. (54)	1	1	0	1	0	1	1	1	1	1	8
Williams et al. (57)	1	0	0	0	0	1	1	1	1	0	5
Ziemann et al. (59)	0	1	0	1	0	1	1	1	1	1	7

Items not described in the methodology of the study being evaluated were assumed to have not been done and awarded a value of zero. Items not applicable to studies as a result of a lack of a control or comparison group were denoted by N/A and were awarded a value of zero. Evaluation was performed independently by 2 reviewers and reported as the average of the 2 scores. PEDro evaluation criteria, as previously described (56): 1. Eligibility criteria were specified. 2. Participants were randomly allocated to groups. 3. Allocation was concealed. 4. The groups were similar at baseline regarding the most important prognostic indicators. 5. There was blinding of all assessors who measured the primary outcome. 6. Measures of at least one key outcome were obtained from more than 70% of the participants initially allocated to groups. 7. All participants for whom outcome measures were available received the treatment or control condition as allocated or, where this was not the case, data for at least one key

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outcome were analyzed by 'intention to treat'. 8. The results of between-group statistical comparisons are reported for the primary outcome. 9. The study provides the point measures and measures of variability for at least one key outcome. 10. Sample size calculations were explained.

**Table 2:** Participant and training program characteristics of included studies organized by training intensity (lowest to highest).

		Participant Characteristics		Training Program Characteristics								
	(n) F)		лах		suc		r of in)	_	Interval Protocols			
Study	Group size (n) (Sex; M/F)	Age (yr)	Pre-training VO <sub>2</sub> max (ml ½g <sup>-1</sup> min <sup>-1</sup> )	Mode	Frequency (sessions per week)	Intensity (% of VO <sub>2</sub> max)	Average Duration of Training Bout (min)	Program Length (weeks)	Average # of Intervals	Work: rest ratio (s)		
Clark et al. (7)	15 (0/15)	19	49.5	R	3	60	50.0	8	N/A	N/A		
Burgomaster et al. (5)	10 (5/5)	23	41	С	5	65	50.0	6	N/A	N/A		
Macpherson et al. (35)	10 (6/4)	23	44	R	3	65	45.0	6	N/A	N/A		
Dunham et al. (15)	7 (4/3)	21	32.5	С	3	65	45.0	4	N/A	N/A		
Helgerud et al. (29)	10 (10/0)	25	55.8	R	3	70	45.0	8	N/A	N/A		
Sugawara et al. (48)	10 (10/0)	20	44.5	С	4	70	60.0	8	N/A	N/A		
Connolly et al. (11)	10 (2/8)	22	41.7	SS	3	70	30.0	6	N/A	N/A		
Connolly et al. (11)	7 (4/3)	22	47.5	R	3	70	30.0	6	N/A	N/A		
Tabata et al. (50)	7 (7/0)	23	52.9	С	5	70	60.0	6	N/A	N/A		
Mendes et al. (39)	13 (13/0)	25	44.9	С	3	70	31.5	6	N/A	N/A		
Kargotich et al. (31)	10 (10/0)	22	44.2	С	5	70	90.0	6	N/A	N/A		
Emonson et al. (18)	9 (9/0)	30	42.4	С	3	70	45.0	5.0	N/A	N/A		
Emonson et al. (18)	9 (9/0)	28	40.8	С	3	70	45.0	5.0	N/A	N/A		
Prieur et al. (46)	8 (6/2)	21	44.1	С	6	70	2.0	4.0	2	60:15		
Ziemann et al. (59)	10 (10/0)	21	50.2	С	3	80	9.0	6	6	90:180		

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Swart et al. (49)	6 (6/0)	30	60.3	С	2	80	32.0	4.0	8	240:90
Helgerud et al. (28)	9 (9/0)	18	58.9	R	2	82.5	16.0	8	4	240:180
Helgerud et al. (29)	10 (10/0)	25	59.6	R	3	85	24.5	8	N/A	N/A
Kim et al. (33)	11 (11/0)	20	49.8	R	4	85	6.0	8	12	30:240
Hautala et al. (27)	20 (20/0)	30	53.7	R	5	85	60.0	8	N/A	N/A
Burke et al. (6)	10 (0/10)	20	40	С	4	90	0.5	7.0	N.R.	30:30
Burke et al. (6)	11 (0/11)	21	39.9	С	4	90	2.0	7.0	N.R.	120:120
Perry et al. (43)	8 (5/3)	24	45.3	С	3	90	40.0	6	10	240:120
Dunham et al. (15)	8 (5/3)	20	33.3	С	3	90	5.0	4	5	60:180
McMillan et al. (37)	11 (11/0)	17	63.4	BDD	2	93	4.0	8	4	60:180
Helgerud et al. (29)	10 (10/0)	25	60.5	R	3	93	11.8	8	47	15:15
Helgerud et al. (29)	10 (10/0)	25	55.5	R	3	93	16.0	8	4	240:180
Duffield et al. (14)	10 (0/10)	20	37.4	С	3	100	16.0	8	8	120:60
Macpherson et al. (35)	10 (5/5)	24	46.8	R	3	100	2.5	6	5	30:180
Ben Abderrahman et al. (3)	9 (9/0)	20	58.7	R	3	105	2.5	8	5	30:30
Poole et al. (45)	8 (8/0)	22	50.8	С	3	105	20.0	7.0	10	120:120
Ben Abderrahman et al. (3)	9 (9/0)	21	59.4	R	3	105	2.5	7.0	5	30:30
Williams et al. (57)	8 (8/0)	27	43	С	3	110	12.0	4.0	12	60:60
Marles et al. (36)	9 (9/0)	19	43.5	С	3	120	9.0	6	3	180:360
Tabata et al. (50)	7 (7/0)	23	48.2	С	5	170	2.7	6	8	20:10
Allemeier et al. (1)	11 (11/0)	23	48.7	С	3	~25 0	1.5	6	3	30:1200

Burgomaster et al. (5)	10 (5/5)	24	41	С	3	~25 0	2.5	6	5	30:270
Metcalfe et al. (40)	7 (7/0)	26	36.3	С	3	~25 0	0.5	6	2	15:300
Metcalfe et al. (40)	8 (8/0)	24	32.5	С	3	~25 0	0.5	6	2	15:300
Trilk et al. (54)	14 (14/0)	30	21.6	С	3	~25 0	2.8	4.0	6	30:240

**Note**: BDD, ball-dribbling drills; C, Cycle; F, female; M, male; N/A, not applicable; N.R., not reported; R, running; s, seconds; SS, snow-shoeing; yr, years. Average # of intervals in training intervention characteristics reflects the average of intervals performed per training session.

## Data Extraction and Synthesis

Subject characteristics (age and weight), sample size, intervention length, training frequency, training intensity and training protocol were extracted. For studies where progression was built into the training intervention the mean value was calculated where appropriate. Before and after training VO<sub>2</sub>max data was extracted in the forms of means  $\pm$  SD/SEM. Where values were reported in relative terms (ml/kg/min), absolute VO<sub>2</sub>max was calculated using the mean body weights for the appropriate time points. When SEM was reported SD was calculated using the reported SEM and sample size. For each study, or each training intervention employed in a study if more than one, the absolute change in VO<sub>2</sub>max (l/min) was calculated, while the pooled standard deviation (SD<sub>P</sub>) was calculated using the sample size (n) and standard deviations (SD) from before (1) and after (2) training in each study or study group:

$$SD_p = \sqrt{\frac{(n_1 - 1)SD_1 + (n_2 - 1)SD_2}{n_1 + n_2 - 2}}$$

95% confidence intervals were calculated for the change in  $VO_2$ max using the resulting  $SD_P$  (20). Cohen's d effect sizes (ES) for the change in  $VO_2$ max for each study, or each training intervention employed in a study if more than one, were also calculated using the difference in means (M) from before (1) and after (2) training and the SD<sub>P</sub>:

$$ES = c(d) \frac{M_2 - M_1}{SD_p}$$

Where c(d) is the small sample size bias correction given by:

$$c(d)=1-\frac{3}{4(n_1+n_2)-9}$$

ES and weighted mean effect sizes (T) were assessed for normal distribution, both kurtosis and skewness, by converting to z by dividing the score by the standard error. Normalcy was considered present if all z values for both kurtosis and skewness were less than +/- 1.96 (19).

Training session dose was calculated by multiplying total exercise session time (seconds) by the relative exercise intensity (% of VO<sub>2</sub>max), and dividing by 10 000. Total training volume was determined by multiplying the training session dose by the total number of training sessions completed as part of each training intervention. Both session dose and total training volume are

presented as arbitrary units (AU), and intervention characteristics for all studies

evaluated and each tertile are presented in Table 3.

**Table 3:** Training intervention characteristics for all studies and each tertile, mean (SEM) unless otherwise indicated.

Tertile	n	Study Sample Size	Study Length (wks)	Training Frequency (days/wk)	Intensity (% of VO <sub>2</sub> max)	CONT	INT	Session Dose (AU)‡	Total Training Volume (AU)‡
All	40	9.8(0.4)	6.4(0.2)	3.3(0.1)	105 (60-250)	15	25	12.0(1.6)	256(41)
1	14	9.7(0.6)	6.0(0.3)	3.7(0.3)	68 (60-70)	13	1	18.4(2.2)	387(59)
2	13	10.3(0.9)	6.9(0.4)	3.2(0.2)	87 (80-92.5)	2	11	10.7(2.4)	230(85)§
3	13	9.2(0.5)	6.2(0.3)	3.1(0.2)	167 (100-250)	0	13	8.5(2.8)§	142(54)§

Note: AU, arbitrary units; CONT, continuous; INT, interval; n, number of studies;  $VO_2$ max; maximal oxygen uptake; wk(s), week(s). Intensity is presented as a mean (range of exercise intensities). Session dose represents total exercise time from an individual training session (seconds) multiplied by the exercise intensity (% of  $VO_2$ max) divided by 10 000. Total training volume represents the dose per session multiplied by the total number of training sessions for the intervention.

‡Significant difference between tertiles (p<0.05); one-way ANOVA's on both session dose and total training volume.

## Meta-Regression

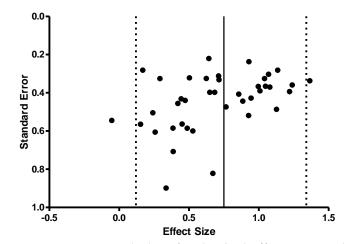
In order to determine whether exercise training intensity, session dose, total training volume, or baseline VO<sub>2</sub>max explain heterogeneity of training effects between the 28 studies examined (40 training groups), we performed a metaregression using these training characteristics as covariates as described by Pigott (44) (Supplementary File S1). A random-effects model was chosen as we believed that the variation among effect sizes could not be explained by sampling error alone; additionally, a random-effects model analysis is more appropriate (20), particularly for the analysis of continuous variables (e.g. exercise intensity, training volume, etc.) (34). Both fixed and random meta-regression analysis performed using weighted mean effect sizes

(T) and fixed-effects inverse variance weights (w) in SPSS 20.0 (SPSS, Chicago, IL, USA) using macros created by Wilson (58). Statistical significance for all regression analysis was accepted at p<0.05. A funnel plot was also constructed in GraphPad Prism v 5.01 (GraphPad Software Inc., La Jolla, CA) using the upper and lower 95% confidence intervals and effect size in order to evaluate the possibility of publication bias (Fig. 2).

## Meta-Analysis

In order to further examine the effect of exercise training intensity on the magnitude of changes in VO<sub>2</sub>max the 40 included study groups were divided into 3 tertiles by intensity resulting in moderate-, high-, and supramaximal-intensity groups. Studies using repeated Wingate bouts (i.e. sprint

Significantly (p<0.05) different from tertile 1 (post-hoc comparisons).



**Figure 2.** Funnel plot of Cohen's d effect sizes and standard error for the 40 study groups included. Mean effect size represented by the solid bar with upper and lower 95% confidence intervals represented by the dashed lines.

interval training; SIT) were assumed to have employed a training intensity of ~250% of VO2max. Each individual tertile was then subject to a fixed-effects model metaanalysis as described by Field and Gillett (20), which uses weighted means for comparison (Supplementary File Participant characteristics for all included studies and for each training intensity tertile are presented in Table 4. One-way ANOVA's were used to compare baseline VO<sub>2</sub>max, weighted change scores VO<sub>2</sub>max, session dose and total training volume between different intensity tertiles. A two-way, repeated measures ANOVA was used to compare the effect of training (tertile 1, 2 or 3) and time (Pre/Post-training) on maximal oxygen uptake. A Bonferonni correction was used for post hoc pairwise comparison of means for main effects and significant interactions. All statistical analysis was performed in SPSS 20.0. Statistical significance was accepted at p<0.05 and all meta-analysis population data are presented as means ± standard error of measurement (SEM).

#### **RESULTS**

Training intervention and participant characteristics for all studies included are presented in Tables 3 and 4, respectively. All studies involved either continuous training (n=15) or interval training (n=25) with modes of exercise including running (n=13), cycling (n=25), snowshoeing (n=1), and/or soccer ball dribbling drills (n=1).

Two reviewers independently determined the quality assessment of the studies included in the analysis using a modified PEDro scale. The average of the reviewers scores were used with a mean score of 5.4/10 for all included studies. There was no difference in mean PEDro scores between the 3 intensity tertiles (tertile 1: 5.6; 2:5.7; 3: 5.3). Specific eligibility criteria, blinded group allocation, blinding of assessors, and an explanation of sample size calculations were rarely performed in the studies included (mean percentage of studies: 32, 0, 0, and 21%, respectively), while random group allocation, utilization of comparable between-group groups baseline), statistical comparisons, and measures of variability for VO2max were reported for most studies (mean percentage of studies: 75, 84, 100, and 100%, respectively).

No outliers were found in the data; skewness (ES:-0.05;T:-0.05) and kurtosis (ES:-0.86;T:-0.26) were within normal limits. Visual inspection on the funnel plot (Fig. 2) suggests the potential for publication bias with smaller studies with larger effect sizes (lower right hand side of the plot), which suggests that results from the meta-regression and meta-analysis should be interpreted with caution. This analysis revealed two studies that fell outside the

95% confidence limits, studies by Helgerud et al. (2007; ES = -0.053) and Duffield et al.

(2006; ES = 1.365).

**Table 4:** Participant characteristics for all studies and each tertile, data presented as mean (SEM).

	Sex		ex	A 00 [rm]	Weight (Ira)	VO <sub>2</sub> ma	x [l/min]
	n	M	F	Age [yr]	Weight (kg)	Pre	Post
All	390	281	109	23 (1)	73 (2)	3.4 (0.1)	3.7 (0.1)*
1	136	95	41	23 (1)	72 (3)	3.2 (0.2)	3.5 (0.2)
2	134	107	27	23 (1)	74 (2)	3.8 (0.2)	4.1 (0.2)
3	120	79	41	22 (2)	74 (3)	3.2 (0.2)	3.5 (0.2)

Note: \*Main effect of training (p<0.05); two-way ANOVA on pre and post training VO<sub>2</sub>max of each tertile. F, female; kg, kilogram; l, litres; M, male; min, minute; n, number of participants; Pre, pre-training; Post, post-training; VO<sub>2</sub>max; maximal oxygen uptake; yr, years.

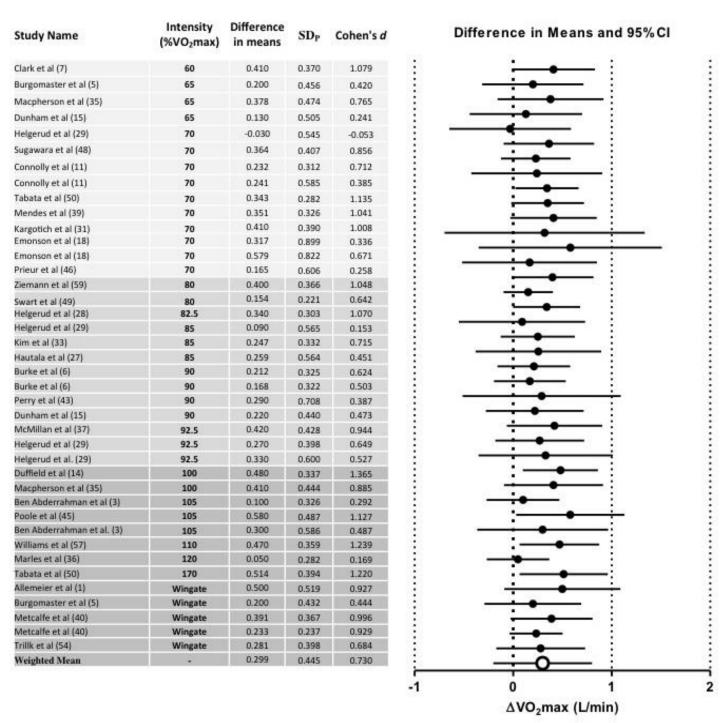
## Meta-Regression

Following training, VO<sub>2</sub>max increased (p<0.05) in all of studies except for one (29) with Cohen's d effects sizes ranging between -0.53 and 1.37. The weighted mean change in VO<sub>2</sub>max (1/min), 95% credibility intervals, small sample size corrected Cohen's d effect sizes, pooled SD, and training intensity (% of VO<sub>2max</sub>) for each study are presented in Figure 3. The weighted mean Cohen's d effect size was 0.73 (95% CI's 1.34-0.11) and homogeneous (Q39 = 11.47; p > 0.01). The homogeneity of ES ( $Q \le N - 1$ ) resulted in the random effects variance component  $(v_{\theta})$ being calculated as zero, resulting in equivalent inverse variance weights for both the fixed and random effects models (i.e. results from both regression models were equivalent). The random effects metaregression model (Table 5) examining training intensity, session dose, baseline VO<sub>2</sub>max, and total training volume was nonsignificant (Q3 = 1.36; p=0.85; R<sup>2</sup> = 0.05; ES=0.76).

#### Meta-analysis

Tertile one consisted of predominantly continuous training modes, while tertile two and three were predominately interval style training modes (Table 3). Ten tertile 1 (total n=14) studies employed cycle ergometers, while treadmill/track running was used in three, and snowshoeing in one. Six tertile 2 (total n=13) studies used cycle ergometers, while six ran on treadmills/track, and one dribbled soccer balls. Nine tertile 3 (total n=14) employed cycle ergometers (n=9), while four utilized treadmill/track running.

One-way ANOVA's on baseline (Table 4) and weighted change scores for absolute VO<sub>2</sub>max (Fig. 4A) demonstrated significant difference between tertiles. The weighted mean change of VO<sub>2</sub>max was 0.30 1/min (95% CI: -0.52 to 1.12 1/min) with population effects for each tertile (Fig. 4B) corresponding to a moderate-large effect of training (9).Α two-way **ANOVA** demonstrated a main effect of training (p<0.05) for absolute VO<sub>2</sub>max (Table 4). Session dose was significantly (p<0.05) lower in tertile 3 than tertile 1, while total training volume was significantly (p<0.05) lower in tertile 2 and 3 compared to tertile 1 (Table 3).



**Figure 3.** Forest plot of mean difference in absolute oxygen consumption (VO<sub>2</sub>max) with 95% credibility intervals (CI's) for each study (filled circles) and the total for all studies (open circle) included in the meta-regression and analysis. Training intensity (% of VO<sub>2</sub>max), pooled standard deviation (SD<sub>P</sub>), and Cohen's effect sizes (Cohen's d) are also shown for each individual, and all studies included. Interventions are organized by ascending order of training intensity with studies assigned to tertile one, two and three, represented by light, medium, and dark grey, respectively. Note: l, liters; min, minutes.

·					
	β	SE	-95% CI	+95% CI	р
Constant	.740	.374	.006	1.47	.048
Training Intensity	.001	.001	002	.003	.600
Training Volume	.000	.000	.000	.000	.532
Session Dose	.000	.000	.000	.000	.916

.083

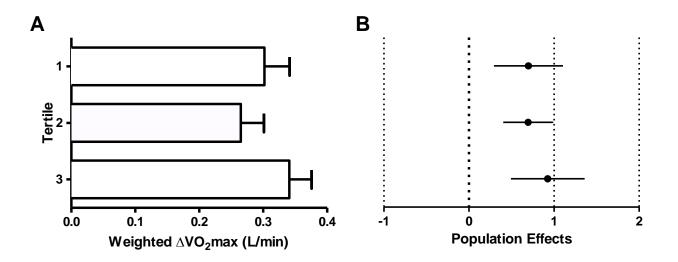
-.196

.130

.692

**Table 5:** Random effects meta-regression model summary and coefficients.

-.031



**Figure 4.** Weighted change in VO<sub>2</sub>max and population effects for each tertile. (A) Weighted mean change and pooled SEM in absolute oxygen consumption (VO<sub>2</sub>max) for each tertile (Tertile 1: 60-70% of VO<sub>2</sub>max; 2: 80-92.5% of VO<sub>2</sub>max; 3: 100-250% of VO<sub>2</sub>max). (B) Forest plot of population effects for each tertile with 95% Cl's. Note: CI, credibility interval; l, liters; min, minutes; SEM, standard error of measurement.

#### DISCUSSION

Baseline VO<sub>2</sub>max

Not surprisingly, exercise training across a wide range of intensities is an effective (T= 0.73; 95% CI 1.35, 0.11) means of increasing VO<sub>2</sub>max in young, healthy adults (39 of 40 study groups reported a positive effect of training; Fig. 3). Interestingly, meta-regression analysis revealed that Cohen's d effect sizes for the change in VO<sub>2</sub>max following training were homogeneous, indicating that the magnitude of the increase in VO<sub>2</sub>max was similar across the range of studies examined. Consistent with this

homogeneity, exercise training intensity, session dose, total training volume, and baseline VO₂max were unable to explain variances in effect sizes. Separate meta-analyses performed on the different training intensity tertiles confirmed that the magnitude of training-induced increases in VO₂max were unaffected by exercise training intensity. Session dose was lowest in the highest intensity tertile (tertile 3; ≥100% VO₂max), while total training volume was significantly (p<0.05) reduced in both tertiles 2 and 3 compared to the

lowest intensity tertile ( $\sim$ 60-70% of VO<sub>2</sub>max).

*Impact of exercise intensity on VO2max* 

Results from the meta-regression and metaanalyses performed in the current study suggest that increasing exercise training intensity above ~60% of VO2max does not provide additional increases in VO<sub>2</sub>max in healthy adults. These results are consistent with several recent reports of comparable increases in VO2max following highintensity interval training and moderateintensity endurance training (5,8,22,38). Importantly, the current analyses extend the results of previous studies that failed to provide a comprehensive examination of the effect of training intensity on traininginduced increases in VO2max due to the examination of only 2 different exercise intensities (4-6, 8, 15, 16, 21, 25, 26, 35, 38, 50-53), and/or a failure to examine the impact of supra-maximal training (4, 6, 15, 21, 25, 29, 51, 52). Our results provide a more complete description of the impact of training intensity on improvements in VO<sub>2</sub>max and suggest that there is not a positive relationship between exercise training intensity and the resulting change in VO<sub>2</sub>max. The findings of the present study should be interpreted with caution, as evidence for a potential publication bias was observed (Fig. 2). However, this evidence comes primarily from exercise-training studies with similarly sized participant populations (~8-10), thus the apparent aggregation of plotted values suggesting the presence of publication bias is likely, at least partly, explained by similar sample sizes. It should also be noted that the training interventions analyzed in the current study work-matched (kilocalories not expended per session and over the total

training period). The lack of work-matched comparisons remains a major shortcoming in the exercise intensity literature and represents an important area for future research.

Impact of session dose and training volume on VO<sub>2</sub>max

While meta-regression failed to identify a relationship between either session dose or total training volume and the effect of training on  $VO_2$ max (due homogeneity of the effect sizes for the studies evaluated), a significant reduction in session dose (tertile 3 vs. tertile 1) and total training volume (tertiles 2 and 3 vs. tertile 1) was observed for studies utilizing higher intensities of training (Table 3). These results demonstrate the potency of high-intensity exercise as a stimulus for increasing VO<sub>2</sub>max and are in agreement with numerous reports demonstrating elevations in VO<sub>2</sub>max following high-intensity, and sprint interval training (2,23,47). While the mechanisms by which low dose highintensity exercise elicits increases in VO<sub>2</sub>max remain controversial, a recent report suggests that improvements in aerobic exercise capacity following high-intensity are primarily the result of training peripheral adaptations (35). Though data supporting this assertion are equivocal (12,13), these results suggest that the mechanisms responsible for improvements in VO<sub>2</sub>max following long duration moderate intensity exercise (i.e. END) and high-intensity exercise may be different.

The findings that high-intensity submaximal and near-maximal training produces equivalent gains in VO<sub>2</sub>max following comparable session doses and training volumes as supra-maximal training

suggest that high-intensity, sub- and nearmaximal exercise (~80-92.5% VO<sub>2</sub>max) may represented the optimal exercise intensity range; providing comparable improvements in maximal oxygen uptake in a lower per session training dose than tertile 1 (p=0.075; see Table 3 for values) and at a lower training intensity than tertile 3 (Table 3). What remains unknown, is the effect of the increasing work associated protocols requiring maximal supramaximal intensities (i.e. would workmatched high intensity protocols yield superior results). While a recent report from our lab suggests that supramaximal exercise (~133% of VO<sub>2</sub>max) reduces the activation of mitochondrial biogenesis compared to work-matched maximal exercise (17), it is not currently known if these acute exercise responses will translate to differences in VO<sub>2</sub>max following training. While the results of the present study question the hypothesis that higher intensities of training would impair the adaptive response to training, the evaluation of training intensity worked-matched interventions across remain an important area of future study.

#### Limitations

While our results provide important new insight into the training response following short duration (4-8 weeks) exercise training in humans, this study posses several potential limitations that must be acknowledged. We sought to examine the impact of short-term (4-8 weeks) exercise training on improvements in VO<sub>2</sub>max; therefore, it remains unknown if the lack of exercise intensity effect observed in the short-term studies included in our analysis would persist following moderate and long duration training interventions. Additionally, the included studies utilized

participant populations consisting of young recreationally/highly active adults relatively similar age, weight, baseline VO<sub>2</sub>max, and disease status (i.e. nondiseased). Thus, it is not currently known if other populations (overweight/obese, children, elderly, diseased populations, etc.) would respond similarly to different intensities of short-term exercise training. It would also be of interest for future studies to examine the impact of other variable that might influence training adaptation (age, baseline VO<sub>2</sub>max, body composition, etc.) that we were not able to be examined in the current study. Finally, evaluation included studies using a modified PEDro scale revealed that several methodological criteria were consistently absent. None of the included studies concealed the allocation of participants to groups (i.e. the person assigning participants to groups was aware of what group they were being assigned) or blinded assessors. Further, very few outlined specific eligibility criteria (~32%) or explained sample size calculations (~21%). methodological This suggests that oversights may be routine in this area of research and highlight specific areas for improvement of future study design to increase the internal and external validity of study findings. Given these systematic limitations, the validity of the results from the analyses performed in the present study must also be interpreted with caution.

# Perspectives for exercise prescription

While training-induced increases in VO<sub>2</sub>max reduce the risk of cardiovascular disease development and all-cause mortality (30), a very small percentage of individuals accumulate the necessary amount of physical activity believed to be required to maintain and/or increase maximal oxygen

uptake (10,55). This is partly due to a lack of knowledge regarding what exercise intensity, or range of intensities, might be the most efficient at improving an individuals' VO2max. From this perspective, our results suggest that training at, or greater than ~60% of VO<sub>2</sub>max improves maximal oxygen uptake, with no additional benefit with increasing exercise intensity. Additionally, our results also highlight the ability of higher intensity training to elicit comparable increases in VO2max in significantly shorter training bouts and lower training volumes than moderate intensity training. These findings support previous investigations several demonstrating the time-efficiency of highintensity training at improving VO<sub>2</sub>max (5,38) and suggest that physical activity expanded guidelines should be incorporate recommendations for highintensity exercise (at or above ~100% of VO<sub>2</sub>max). Importantly, evaluation of the studies included in the present investigation questions the external validity of results from studies in this area of research and stress the need for future investigations of high methodological quality for accurate prescription exercise development generalizable to the population.

### Conclusions

Collectively, the results of the analyses carried out in the current work suggest that training at any intensity above ~60% of VO<sub>2</sub>max is likely to improve maximal oxygen uptake in healthy adults. While the lack of a positive effect of increasing training intensity on the increase in VO<sub>2</sub>max suggests minimal additional benefit to higher intensity training, it is important to highlight the fact that higher intensities of training induced adaptations following significantly

lower training session doses and total training volumes. Our observations also suggest that high-intensity, sub- and nearmaximal exercise (~80-92.5% VO<sub>2</sub>max) may be the ideal exercise intensity range for eliciting improvements in VO<sub>2</sub>max as both training volume, and exercise intensity are compared to moderate and supramaximal intensity training, respectively. While our data supports claims regarding the efficiency and potency of high-intensity training, they also highlight future directions for research examining the impact of exercise intensity on improvements in VO<sub>2</sub>max and the mechanisms by which high-intensity exercise achieves its potency.

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