Quantifying the effects of four weeks of low volume high intensity sprint interval training on O2max through assessment of haemodynamics

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ABSTRACT

Background: Sprint interval training is a popular workout modality. Studies have eluded to a positive effect on maximal oxygen uptake, however little is known about the mechanistic basis of this adaptation. Therefore, the purpose of this study was to determine the effects of a short-term high-intensity sprint interval training (SIT) intervention on O2max through quantification of both the respiratory and haemodynamic responses.

Participants: Thirty-seven physically active participants undertook 4-weeks of either cycling-based SIT (8 x 20 s at 170% P-O2max with 10 s recovery) or continuous exercise training (CET) (30 min at 70% P-O2max) 3 times per week. O2max, blood-based markers and haemodynamic responses were assessed pre and post the intervention period. O2max was assessed using breath-by-breath open circuit spirometry, while haemodynamic responses were monitored using thoracic impedance cardiography.

Results: O2max exhibited a non-significant 4.1% increase (ES = 0.24) for SIT with 7.0% p = 0.007 (ES = 0.40) increase for CET. Haemodynamic responses (maximal cardiac output, maximal stroke volume) displayed non-significant responses for CET and SIT while a-vO2dif-max increased from 15.8 ± 4.8 to 18.3 ± 2.9 ml.100 ml-1) (p = 0.02) (ES = 0.63) in SIT.

Conclusions: O2max is a function of maximal cardiac output and a-vO2dif-max, so for a meaningful change to occur in cardiorespiratory fitness, there must be a concomitant increase in O2 delivery. This study demonstrates that a low volume SIT intervention evokes peripherally mediated responses (a-vO2dif) and anaerobic substrate utilisation rather than O2 delivery components. Future works should address the time course of the responses and when assessing O2max-based responses that due attention be given to the haemodynamic responses as means of quantification of the response.

Keywords: maximal oxygen uptake, cardiac output, high intensity training, anaerobic capacity

INTRODUCTION

Maximal oxygen uptake (O2max) provides a representation of the integrated response of the respiratory, cardiovascular and muscular systems to take-up, distribute and utilise oxygen during an exercise challenge to volitional exhaustion. Furthermore, O2max is one of the most widely accepted diagnostic tests in both athletic and clinical populations in the assessment of cardio-respiratory fitness and health (Ward 2018). A manifestation of the O2max response is attributable to the delivery and utilisation of oxygen at the muscle, as reflected through the Fick principle, which dictates that O2max is a function of maximal cardiac output (max) and the maximal amount of O2 extracted at the muscle, determined by the arterio-venous oxygen difference (a-vO2dif-max). Thus, during such an exercise challenge, an imbalance results between the supply of O2 to the muscle as expressed by and the extraction of O2 as reflected by the a-vO2dif. At the point of O2max, rises to a maximal value while a-vO2dif exhibits a plateau-like response, with the response of being attributed to, in a healthy population, a continuing rise in heart rate (HR) with stroke volume (SV) either levelling-out at ~50-60% O2max in untrained, or rising in conjunction with the HR response, in trained populations (Calbet et al 2007). Although recent advances in understanding and modelling have led to more sophisticated and complex interpretations of the factors which limit O2max (Ferretti 2014), the prevailing consensus is for an O2 delivery () limitation. Furthermore, the roles of haemoglobin (Hb) and muscle blood flow also appear to be critical to the magnitude of the O2max, with both maximal O2 extraction (Wagner 2000) and maximal cardiac output (Krip et al 1997; Calbet et al 2004) being dependent upon these. Hence, given the association between max and a-vO2dif-max and their derivatives and O2max, it is prudent, when ascertaining the magnitude of change following a training intervention, to establish the underlying changes in O2 delivery and extraction.

Sprint interval training is a popular exercise regimen which has received considerable attention and has been reported to stimulate biological responses similar to those seen with more conventional higher volume low intensity continuous-based exercise (Burgomaster et al 2005; Burgomaster et al 2006; Astorino et al 2012; Astorino et al 2017). The additional obvious attractiveness of this form of exercise is the significant reduction in exercise time, compared to the more conventional model of endurance training. A typical SIT session has a duration of ~2-10 min compared to a recommended minimum duration of ~30 min for continuous-based exercise, with the application of SIT-based interventions exhibiting significant increases in O2max. Yet despite the apparent significant changes in O2max following either high intensity interval training (HIIT) or a SIT intervention, it is noteworthy that very few studies have reported any associated changes in , SV, HR and a-vO2dif. Of the studies that have reported haemodynamic responses (Warburton et al 2004; Daussin et al 2007; Astorino et al 2017), meaningful improvements in O2max (7-8%) across 20 sessions were accompanied by an 8-11% increase in max, with little or no change in a-vO2dif-max or HRmax. However, the interventions in these studies ranged from HIIT using longer intervals (4 min at lactate turn-point, 1 min at 90% Pmax) through to HIIT using shorter intervals of 4 x 30s maximal efforts. In contrast, no change in max was observed following a six-week interval training intervention despite a 12% increase in O2max, with the authors suggesting that there would have been a peripherally mediated response to account for this discrepancy (Macpherson et al 2011).

Changes in O2max with training attributed to an increased stroke volume and red blood cell volume, with longer duration interventions promoting concentric cardiac hypertrophy and an increased ventricular compliance (Ekblom & Hermansen 1968, Calbet et al 2015), with the locus of the training stimulus attributed to the imposed training load. Indeed, the magnitude of gain in O2max was shown to be greater where the intervention was longer, more intense and accompanied by a higher training frequency (Bacon et al 2013).

However, despite the apparent association between training load (intensity and duration) and the adaptive nature of O2max, a number of studies have emerged highlighting significant increases in O2max in as little as 2-4 weeks using between 6-12 sessions of SIT (Burgomaster et al 2005; Burgomaster et al 2006; Bailey et al 2009; Babraj et al 2009; Little et al 2010; Astorino et al 2012). Changes in markers of biogenesis have been observed following such intervention including significant increases in mitochondrial density, carbohydrate metabolism, aerobic and anaerobic enzyme activity, lactate production and insulin sensitivity culminating in an increase in O2max.

However, to date, the actual mechanisms which contribute to the increase in O2max following a short duration SIT intervention through either increased O2 delivery or extraction have not been addressed. Accordingly, there is still uncertainty as to the nature of the factors which mediate these reported responses for O2max following a period of SIT. Therefore, the purpose of this study was to evaluate the response of O2max to a four-week low volume SIT intervention addressing the cardiorespiratory and haemodynamic responses in a physically active population.

METHOD

Participants: Following local institutional ethical approval (Anglia Ruskin University, Faculty Research Ethics Panel) 36 physically active participants volunteered and agreed to participate (age, 17.2 ± 1.2 yrs; height, 173.7 ± 8.9 cm; mass, 67.7 ± 14.0 kg). Of which n = 10 (age, 17.0 ± 0 yrs; height, 173.6 ± 8.7 cm; mass, 69.3 ± 17.0 kg) formed the CET group, n = 8 formed the control group (CON) (age, 17.0 ± 0 yrs; height, 172.6 ± 9.6 cm; mass, 70.1 ± 17.1 kg) and n = 18 formed the SIT cohort (age, 17.0 ± 0.5 yrs; height, 173.1 ± 9.2 cm; mass, 62.4 ± 6.9 kg). Group sizes were determined *a-priori* using previously established criteria for O2max (44.8 ± 5.7 ml.kg-1.min-1) with an anticipated/reported increase, following HIIT of 5.7 ml.kg-1.min-1 from a meta-analysis of 14 studies (Gist et al 2014). Assuming a power (ß) of 80% and an alpha level of 0.05, minimum sample sizes were estimated to be eight (ClinCalc.com). Inclusion criteria for participation in this study was to be undertaking >60 min of physical activity per week and to be free from any contra-indication to exercise, such as asthma, hypertension, musculoskeletal injury, or a recent viral infection.

Study design: Using repeated measures matched pairs design, participants from all groups reported to the laboratories for testing on 3 occasions. The first visit served as a familiarisation trial with the remaining two for data collection. Assessments were undertaken both pre and post the 4-week training intervention of maximal oxygen uptake (O2max), and associated cardiorespiratory and haemodynamic responses, with groups being matched for the intervention according to their pre-test O2max scores as highlighted in figure 1. All testing was completed at the same relative time of day between the hours of 13:00-17:00 to minimise diurnal variation. Throughout all trials participants cycled on a pre-calibrated ergometer (Lode Excalibur Sport, Groningen, Netherlands) with personal geometries for the ergometer established during the familiarisation trials and standardised thereafter. Throughout all trials cadence was held at a constant 60 rpm, with the test being terminated either due to ensuing volitional exhaustion, or cadence decreasing >5 rpm than that prescribed for >10 s. Gas exchange responses were recorded on a breath-by-breath basis using a pre-calibrated metabolic cart (Metalyzer 3B, Cortex, Germany).

Determination of O2max: Using an incremental stress test with a ramp rate of 0.42 W.s-1 following an initial 60 s period at 100 W, O2max was determined. Each trial was preceded by a self-selected warm-up, established during the familiarisation trial, which was subsequently prescribed for all experimental trials. O2max was initially confirmed using a conventional approach of assessing a ∆O2 across the final 2 consecutive 30 breath periods, where a response of ≤1.5 ml.kg-1.min-1 classified a maximal effort and O2-plateau (Gordon et al 2017). Additional confirmation was also sought using the heart rate plateau method (Keiller and Gordon 2018), where a ∆HR ≤2 b.min-1 is used to confirm a maximal effort. From this test, the power output at O2max (P-O2max) was derived for establishment of the SIT and CET intensities.

Pulmonary gas exchange responses: Throughout all trials, respiratory volumes and flow along with expired gas concentrations (O2, CO2 and N2) were drawn directly from a mouthpiece and low resistance turbine assembly with gas analysis occurring at a rate of 60 ml.min-1. Using custom metabolic cart software, the respiratory responses and gas concentrations were aligned, allowing presentation of respiratory gas exchange variables (O2, CO2, *V*E and RER). Prior to all trials, the metabolic cart was calibrated for both volume and flow according to manufacturer’s specifications. Gas exchange responses were visually inspected, and a rolling 15-breath average applied to the data.

Haemodynamic responses: Using thoracic impedance cardiography (Physioflow, Mantec, France), the haemodynamic responses to exercise during the O2max trials were assessed. This approach has been validated previously in exercising populations (Richard et al 2001) with acceptable test-re-test reliability (Siebenmann et al 2014). With participants sat in a rested state, electrode lead placement sites were prepared for the following anatomical positions: the skin along the supra-clavicular fossa aligned to the base of the neck, along the xiphoid and across the chest, according to the V1/V6 position for ECG lead placement, with all sites being shaved, cleaned with an alcohol swab and then agitated to increase signal conductivity using an abrasive gauze. At each location, a pair of electrodes (Ag/AgCL) (Skintact, Leonhard Lang, GmbH, Innsbruck, Austria) were applied, with each pair containing a transmitting and receiving electrode. All leads were taped to the skin to minimise movement, thereby reducing any noise artefacts in the signal. Additionally, a tubigrip sheath was worn around the chest further reducing the movement of wires on the electrodes. With leads in place, the participant adopted a seating position on the ergometer while blood pressure (Omron HEM – 7200, Omron Health Care, Hoofddorp, Netherlands) was recorded across the antecubital space twice. The participants then remained motionless and quiet for a further two minutes, allowing for calibration using the averaged baseline blood pressure value. HR was determined from the R-R interval from the ECG signal while was established according to = HR x SV x BSA, where BSA = body surface area (Charloux et al 2000). Both SV and HR were recorded on a beat-to-beat basis during exercise, and subsequently reduced to 10 s averages for analysis. The a-vO2dif response during exercise was calculated using a reverse Fick equation where, a-vO2dif (ml.100 ml-1) = O2 (ml.min-1) / (l.min-1).

Blood chemistry: Prior to all exercise tests, with the participants being in a rested state, a capillary blood sample (120 µl) was collected for determination of both haematological and biochemical makers (Opti CCA-TS, Una Health, Stoke on Trent, UK). An additional capillary sample (20 µl) was collected for determination of both lactate and glucose concentrations (Biosen C-Line Clinic (EKF-diagnostic GmbH, Germany)). Finally, a further follow-up sample was collected immediately upon completion of the VO2max trial.

Training interventions: SIT training consisted of 3-sessions.wk-1 for 4-weeks of exercise at 170% P-O2max, comprising of 8 x 20 s bouts of work interspersed with 10 s recovery. Prior to each session the participants completed a prescribed warm-up for 5 min at 100 W, while during the intervening recovery periods they were instructed to gently rotate the pedals while the resistance was removed from the flywheel. Throughout the SIT trials, strong verbal encouragement was provided and upon completion of the session, they undertook a prescribed recovery again at 100 W for a minimum of 5 min. As with the SIT, CET was completed over a 4-week period with 3-sessions.wk-1 with all trials completed at 70% P-O2max for 30 min.session-1. All training commenced at least 48-h after completion of the baseline laboratory tests, with all sessions undertaken on weight-loaded cycle ergometers (Monark Ergomedic 893 E, Sweden). Sessions were grouped with five participants training at the same time, but from within the same training group (SIT or CET), with all trials being performed in a climate-controlled laboratory and at the same time of day 12-2pm. Training sessions took place on Monday, Wednesday and Friday thus allowing for 48-h recovery between each session. The criteria for a successful completion of the intervention was 80% (10/12 sessions) in accordance with previous work (Gaskill et al 2001). Those in the CON group were advised to continue their normal physical activity regime throughout the course of the study.

Data analysis: Using SPSS version 21 (SPSS, Chicago, Illinois) data are expressed as mean ± SD. Normality was assessed using a Shapiro-Wilk test, while homogeneity of variance was confirmed within each group using Levene’s test. Identification of differences between groups in physiological and haematological variables was assessed using a one-way ANOVA, while a two-way ANOVA was used to identify differences in O2max, max, SVmax, and a-vO2dif-max, while *post-hoc* analysis was effected using LSD. Effect sizes (ES) were identified through both partial eta-squared (ŋ2p) and Cohen’s d, interpreted as trivial, <0.2; small 0.2-0.6; medium, 0.6-1.2 and large >1.2 (Batterham et al 2006). Magnitude-based inferences for all variables are reported according to the methods of Hopkins (2002). Under the conditions the effect of both SIT and CET on all measures were examined using 95% CI and as a likelihood that the true value represents substantial change. For an effect to be deemed unclear, its confidence intervals would overlap the thresholds for substantives, in other words the effect could be substantially positive or negative, beneficial or detrimental, with the smallest substantial change (either positive or negative) set at 0.8% (Hopkins et al 2009).

Transfer of training effects were estimated according to the approach of Zatsiorsky (1995) whereby O2max was referred to as the trained exercise and the untrained exercises are the composite variables.

RESULTS

Adherence to training: Of the total number of training sessions (448) which could have been undertaken, there was a completion rate of 430 (96%), of which the response rate for SIT was 271 (94%) and CET was 157 (98%). The total training durations for the groups were, ~50 min for SIT and 270 min for CET across the four-week training period. Prior to commencement of the 4-week training interventions there were non-significant differences between groups (p> 0.05) for any of the key physiological, biochemical or anthropometric variables.

Response of O2max: Post-training the SIT group displayed a non-significant (p > 0.05) increase of 2.0 ± 5.3 ml.kg-1.min-1 (4.1%) with CET exhibiting an increase of 3.6 ± 3.7 ml.kg-1.min-1 (7.0%) (p = 0.007). However, as displayed in table one using magnitude based inferences the outcome for SIT is considered very likely trivial while for CET, likely trivial. The absolute responses (l.min-1) were 0.26 ± 0.23 (p = 0.002), 0.12 ± 0.34 (p > 0.05) and -0.10 ± 0.23 (p > 0.05) for CET, SIT and CON respectively, while their magnitude based inferences are also reflected in table one and individual responses are reflected in figure two.

Haemodynamic responses: SIT-based training resulted in a non-significant increase in max and SVmax (p > 0.05) while a-vO2dif-max displayed a significant (p = 0.02) increase of 2.5 ± 3.3 ml.100ml-1. The CET group also presented non-significant differences for max, SVmax as well as a-vO2dif-max (p > 0.05) but with a significant increase in HRmax of 4 ± 5 b.min-1 (p = 0.02). The magnitude-based inferences for all groups are in table one. The CON group displayed non-significant responses (p > 0.05) across all haemodynamic markers.

Haematological and biochemical responses: Table one reflects the changes in both Hb and HcT while table two highlights the blood-based biochemical markers, BLa, GLu, pH and HCO3-. CET exhibited a significant increase in Hb of 0.4 ± 0.6 g.dl-1 (p= 0.04) with non-significant responses observed for all other markers (p > 0.05). SIT resulted in non-significant changes in Hb and HcT (p > 0.05), however, maximal BLa (mM) recorded at termination of the O2max trial exhibited an increase of 0.96 ± 1.36 mM (p = 0.02) (ES = 0.50) (possibly beneficial). Of note were the significant decreases in resting BLa (mM) of 0.2 ± 0.4 (p = 0.03), resting GLu, 0.3 ± 0.2 mM (p = 0.02) and HCO3- of 0.8 ± 0.8 mM (p = 0.01) for CON.

Change in body mass: The CET and SIT groups presented a non-significant increase in body mass of 0.5 ± 1.0 and 0.2 ± 1.0 kg (p > 0.05) while there was a 0.4 ± 1.3 kg decrease in mass for CON (p > 0.05).

Transfer of training: The estimated transfer effects are presented in figure three, whereby the larger the value the greater the potential effect.

DISCUSSION

The purpose of this study was to compare the response of O2max to a 4-week period of SIT with that of CET in a group of young physically active participants. The primary outcome was that, despite exposure to an on-training duration of ~50 min across the SIT intervention, there was a significant but non-meaningful change in O2max, with non-significant responses for max, HRmax or SVmax. However there was a significant and possibly beneficial increase in the a-vO2dif-max. Furthermore, despite an on-training duration of ~360 min with the CET displaying a significant increase in O2max, the benefit of this was deemed to be likely trivial. Indeed, similar responses were evident in this group for all other parameters besides HRmax and Hb which also showed significant increases but those were considered as being possibly, or likely trivial, respectively.

This study has attempted to validate the response of O2max to both CET and SIT through assessing changes in its primary constituents, namely SV, , HR and a-vO2dif.. The fundamental principle governing O2max is Fick’s which states that O2max is a function of max and CaO2-CvO2. Indeed, studies across both sedentary and trained populations show that the changes in O2max are ostensibly due to variations in maximal stroke volume (Montero et al 2015), supported by considerably less variability in HRmax and a-vO2dif (Calbet et al 2004). The role of O2 delivery and by extension max have been well established with training following bed rest (16 weeks) (Ekblolm et al 1968) showing an increase in O2max of 14% coupled with an 8% increase in max while a period of detraining results in ~4-14% decrease in O2max coupled with ~8% reduction in max (Coyle et al 1984). These changes would appear to be the consequence of a reduced SVmax attributed to both a lowering of blood volume (Coyle et al 1986) and reduced SVmax, a consequence of a decreased left ventricular diastolic dimension (Martin et al 1986). It is thus contended, that a change in O2max cannot occur without fundamental changes in O2 delivery.

A limited number of HIIT/SIT based studies have assessed haemodynamic responses and showed varying results, particularly when considered in conjunction with the O2max response. A 7-8% increase in O2max corresponded with 8-11% increase in max (Astorino et al 2017) while in a similar study, a 12% increase in O2max following a 6-week intervention resulted in no change in max (Daussin et al 2007). Such data suggest that HIIT/SIT-based exercise can elicit a change in VO2max due to an increase in max, but that this may well be a function of the intervention type. Certainly, 10 repetitions of 60 s at an intensity of 110% of peak aerobic power interspersed with 75 s recovery (Astorino et al 2017), as opposed to 5 min periods of work, with 4 min at lactate turn-point and the final 60 s at 90% peak aerobic power (Daussin et al 2007) yielded significantly different outcomes. Secondly, there is a time course interaction for the adaptation to cardiac output. An increase in max is characterised by both an eccentric and concentric hypertrophy of the ventricle as a consequence of increased contractility of the heart and ventricular blood volume (Blomqvist & Saltin 1983), with a minimum duration for adaptation of 4 weeks adopting continuous exercise of 20-60 min per session (Ribeiro et al 2017).

In contrast, studies using intervention periods of 2-4 weeks have shown only small increases in O2max in the order of 2.2 ml.kg-1.min-1 (ES= 0.28). (Burgomaster et al 2005; Burgomaster et al 2006; Bailey et al 2009; Babraj et al 2009; Little et al 2010; Astorino et al 2012). These findings are in very close agreement with those recorded in the current study of △O2max of 2.0 ml.kg-1.min-1 (ES = 0.24). The mechanisms responsible for the modest increase in O2max following SIT are likely linked to an increased glycolytic flux, due in part to increased activity of glycolytic enzymes PFK and LDH (Parra et al 2000; Weber et al 2002). Under exercise conditions experienced during the SIT sessions, there is insufficient O2 to meet the raised metabolic demand at the muscle, consistent with the notion of a hypoxic state at a cellular level. Such conditions are shown to be associated with peripherally orientated adaptations, including an increased (muscle) capillarisation and mitochondrial density (Blomqbist et al 1983; Daussin et al 2008), supported through the 13.7% increase in the a-vO2dif-max observed in this study. An increased O2 delivery at a cellular level has previously been demonstrated in sprint interval studies (Burgomaster et al 2008; MacPerson et al 2011) with the mechanism for this adaptation being attributed to a reduced O2 partial pressure at the muscle, as a consequence of a reduced blood flow to the exercising muscle (Essen 1978). These findings are supported by the significant increase in post-exercise blood lactate concentration following SIT in agreement with work from Ready et al (1981) citing a similar response following 2-weeks of sprint-interval training.

Coupled with these considerations is the recognition that O2max, besides being a composite of these broad O2 delivery and utilisation parameters, it is affected by a myriad of additional factors, relating to barometric pressure, water vapour pressure of gas, gas temperature, tidal volumes and breathing frequencies. Indeed, these respiratory-based components were demonstrated through applied, multiple regression under both steady state and incremental exercise conditions to account for 99.4% of the variability in the O2 response (Robergs et al 2010). Additionally, given the reported daily variations in O2max and associated composite factors, the control of such tests and their environments is critical to establishing a meaningful result. A recent review (Ward 2018) reinforced this, suggesting that small individual errors through data collection or calibration can manifest in significant errors in the final gas-exchange computation. It is therefore feasible that previously reported changes in O2max in the absence of concurrent haemodynamic responses are a consequence of errors in O2 collection and/or interpretation. Indeed, this study demonstrates that SIT evoked a significant increase in O2max but when assessed through ES and magnitude-based inferences, which reflect the degree of substantive change, this response was likely to be trivial. These findings support the notion of appropriate ratification of O2max using standardised approaches (Robergs et al 2010, Ward 2018) and quantification of the response to training through assessment of the haemodynamic characteristics.

This study is though not without some confounding issues. The period of the training intervention was relatively short and as such it was not possible to fully address the time-course for the haemodynamic adaptations. Coupled with this, the relatively short time period of the intervention precluded the application of a progressive overload to be applied, which again could have contributed to the blunted O2max response in both the SIT and CET groups.

CONCLUSIONS

This study has demonstrated that the application of a 4-week SIT intervention had no meaningful effect on O2max, despite a significant and possibly beneficial increase in a-vO2dif-max and that CET across the same time period resulted in an increase in O2max, but this was deemed to be likely trivial. It is suggested that future studies that aim to address changes in O2max should support the findings with reference to the requisite components (, SV and a-vO2dif) and that recognition should be given to the sensitivity of O2max as a measure when evaluating training-based responses. Furthermore, future works need to establish the role of progressive training overload when applying SIT on haemodynamic responses and increase the timeframe of application in order to address the time course of the biological adaptations.

REFERENCES

Astorino TA (2009) Alterations in O2max and the O2 plateau with manipulation of sampling interval. Clinical Physiol Func Imaging, 29: 60-67

Astorino TA, Allen RP, Roberson DW, Jurancich M (2012) Effect of high intensity interval training on cardiovascular function, O2max and muscular force. J Strength Cond Res, 26 (1): 138-145

Astorino TA, Edmunds RM, Clark A, King L, Gallant RM, Namm S, Fischer A, Wood KA (2017) High-intensity interval training increases cardiac output and O2max. Med Sci Sport Exerc 49:

Bacon AP, Carter RE, Ogle EA, Joyner MJ (2013) O2max trainability and high intensity interval training in humans: A meta-analysis. PLoS One doi:10.1371/journal.pone.0073182

Babraj JA, Vollaard NB, Keast C, Guppy FM, Cottrell G, Timmons JA (2009) Extremely short duration high intensity interval training substantially improves insulin action in young healthy males. BMC Endocr Disord. 9: 3 doi:10.1186/1472-6823-9-3

Bailey SJ, Wilkerson D, Dimenna F, Jones AM (2009) Influence of repeated sprint training on pulmonary O2 uptake and muscle deoxygenation kinetics in humans. J Appl Physiol. 106: 1875-1887

Batterham AM, Hopkins WG (2006) Making meaningful inferences about magnitudes. Int J Sports Physiol Perform 1: 50-57

Beltz NM, Gibson AL, Janot JM, Kravitz L, Mermier CM, Dalleck LC (2016) Graded exercise testing protocols for the determination of O2max: historical perspectives, progress and future considerations. J Sports Med <http://dx.doi.org/10.1155/2016/3968393>

Blomqvist CG, Saltin B (1983). Cardiovascular adaptations to physical training. Ann Rev Physiol 45: 169-189

Bouchard C (1986) Genetics of aerobic power and capacity. In: Malina RM, Bouchard C (eds) Sport and Human Genetics, Human Kinetics, Champaign IL, 59-88

Burgomaster KA, Hughes SC, Heigenhauser GJF, Bradwell SN, Gibala MJ (2005) Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans. J Appl Physiol, 98 (6) 1985-1990

Burgomaster KA, Heigenhauser GIF, Gibala MJ (2006) Effect of short term sprint interval training on human skeletal muscle carbohydrate metabolism during exercise and time-trial performance. J Appl Physiol, 100 (6) 2041-2047

Burgomaster KA, Howarth KR, Phillips SM, Rakobowchuk M, MacDonald MJ, McGee SL, Gibala, MJ (2008) Similar metabolic adaptations during exercise after low volume sprint interval and traditional endurance training in humans. J physiol, 586: 151-160

Calbet JAL, Jensen‐Urstad M, Van Hall, G, Holmberg HC, Rosdahl H, Saltin B (2004) Maximal muscular vascular conductances during whole body upright exercise in humans. J Physiol, 558: 319-331

Calbert JAL, Gonzalez-Alonso J, Helge JW, Sondergaard H, Munch-Anderson T, Boushel R, Saltin B (2007) Cardiac output and arm blood flow during incremental exercise on the cycle ergometer. J Appl Physiol 103: 969-978

Calbert JAL, Jensen-Urstad M, Van Hall G, Holmberg HC, Rosdahl, H, Saltin B (2004) Maximal muscular vascular conductances during whole body upright exercise in humans. J Physiol 558: 319-331

Coyle EF, Hemmert MK, Coggan AR (1986) Effects of detraining on cardiovascular responses to exercise: role of blood volume. J Appl Physiol 60: 95-99.

Coyle EF, Martin WH, Sinacore DR, Joyner MJ, Hagberg JM, and Holloszy JO (1984) Time course of loss of adaptations after stopping prolonged intense endurance training. J Appl Physiol 57: 1857-1864

Daussin FN, Ponsot F, Dufour SP (2007) Improvement of O2max by cardiac output and oxygen extraction adaptation during intermittent versus continuous training. Eur J Appl Physiol 101: 377-383

Daussin FN, Zoll J, Dufour SP, Ponsot E, Lonsdorfer-Wolf E, Doutreleau S, Mettauer, B, Piquard, F, Geny B, Richard R (2008) Effect of interval versus continuous training on cardiorespiratory and mitochondrial functions: relationship to aerobic performance improvements in sedentary subjects. Am J Physiol-Reg I 29: 264-272

Ekblom B, Hermansen L (1968) Cardiac output in athletes. J Appl Physiol 25: 619-625

Essén B (1978) Studies on the regulation of metabolism in human skeletal muscle using intermittent exercise as an experimental model. Acta physiol Scand 454: 1-31

Ferretti G (2014) Maximal oxygen consumption in healthy humans: theories and facts. Eur J Appl Physiol 114: 2007-2036

Gaskill SE, Walker AJ, Serfass RA, Bouchard C, Gagnon J, Rao DC, Skinner JS, Wilmore JH, Leon AS (2001) Changes in ventilatory threshold with exercise training in a sedentary population: The Heritage family study. Int J Sport Med 22: 586-592

Gist NH, Fedewa MV, Dishman RK, Cureton KJ (2014) Sprint interval training effects on aerobic capacity: A systematic review and meta-analysis. Sport Med 44: 269-279

Gordon D, Scruton A, Barnes R, Baker J, Prado L, Merzbach V (2017) The effects of menstrual cycle phase on the incidence of plateau at O2max and associated cardiorespiratory dynamics. Clin Physiol Func Imaging 38: 689-698

Hopkins WG, Marshall SW, Batterham AM, Hanin J (2009) Progressive statistics for studies in sports medicine and exercise science. Med Sci Sport Exerc 41: 3-12

Hopkins WG (2002) Probabilities of clinical or practical significance. Sport Sci 6: 16

Howley ET, Bassett DR, Welch HG (1995) Criteria for maximal oxygen uptake. Med Sci Sport Exerc 27: 1292-1301

Keiller D, Gordon D (2018) Confirming maximal oxygen uptake: Is heart rate the answer? Int J Sport Med 39: 198-203

Krip B, Gledhill N, Jamnik V, Warbuton D (1997) Effect of alterations in blood volume on cardiac function during maximal exercise. Med Sci Sport Exerc 29: 1469-1476

Macpherson RE, Hazell TJ, Olver TD, Paterson DH, Lemon PW (2011) Run sprint interval training improves aerobic performance but not maximal cardiac output. Med Sci Sport Exerc 43: 115-122

Martin WH, Coyle EF, Bloomfield SA, Ehsani AA (1986) Effects of physical deconditioning after intense endurance training on left ventricular dimensions and stroke volume. J Am Coll Cardiol 7: 982-989

Medbø JI, Mohn AC, Tabata MI, Bahr R, Vaage O, Sejersted OM (1988) Anaerobic capacity determined by maximal accumulated O2 deficit. J Appl Physiol 64: 50-60

Montero D, Diaz-Canestro C, Lundby C (2015) Endurance training and VO2max: Role of maximal cardiac output and oxygen extraction. Med Sci Sport Exerc 47: 2024-2033

Parra J, Cadefau JA, Rodas G, Amigo N, Cusso R (2000) The distribution of rest periods affects performance and adaptations of energy metabolism induced by high‐intensity training in human muscle. Acta Physiol Scand 169: 157-165

Poole, DC, Jones AM (2017) Measurement of the maximum oxygen uptake O2max: O2peak is no longer acceptable. J Appl Physiol 122: 997-1002

Poole DC, Wilkerson DP, Jones AM (2008) Validity of criteria for establishing maximal O2 uptake during ramp exercise tests. Eur J Appl Physiol 102: 403-410

Richard R, Lonsdorfer-Wolf E, Charloux A, Doutreleau S, Buchheit M, Oswald-Mammosser M, Lampert E, Mettauer B, Geny B, Lonsdorfer J (2001) Non-invasive cardiac output evaluation during a maximal progressive exercise test, using a new impedance cardiograph device. Eur J Appl Physiol. 85: 202-207

Ready AE, Eynon RB, Cunningham DA (1981) Effect of interval training and detraining on anaerobic fitness in women. Can J Appl Sport Sci. 6: 114-118

Ribeiro PA, Boidin M, Juneau M, Nigam A, Gayda M (2017) High-intensity interval training in patients with coronary heart disease: Prescription models and perspectives. Ann Phys Rehabil Med 60: 50-57

Robergs RA, Dwyer D, Astorino T (2010) Recommendations for improved data processing from expired gas analysis indirect calorimetry. Sport Med 40: 95-111

Siebenmann C, Rasmussen P, Sørensen H, Zaar M, Hvidtfeldt M, Pichon A, Secher H, Lundby C (2014) Cardiac output during exercise: A comparison of four methods. Scand J Med Sci Sports. 25: 20-27

Tabata I, Nishimura K, Kouzaki M, Hirai Y, Ogita F, Miyachi M, Yamamoto K (1996) Effects of moderate-intermittent training on anaerobic capacity and O2max. Med Sci Sport Exerc 28: 1327-1330

Taylor HL, Buskirk E, Henschel A (1955) Maximal oxygen intake as an objective measure of cardio-respiratory performance. J Appl Physiol 8: 73-80

Wagner PD (2000) New ideas on limitations to O2max. Exerc Sport Sci Rev 28: 10-14

Warburton DER, Haykowsky MJ, Quinney HA Blackmore D, Teo KK, Taylor DA, McGavock J, Humen DP (2004) Blood volume expansion and cardiorespiratory function: effects of training modality. Med Sci Sport Exerc 36: 991-1000

Ward S (2018) Open-circuit respirometry: Real time, laboratory-based systems. Eur J Appl Physiol 118: 875-898

Weber CL, Schneider DA (2002) Increases in maximally accumulated oxygen deficit after high-intensity interval training are not gender dependent. J Appl Physiol 92: 1795-1801

Zatsiorsky V (1995) Science and practice of strength training, Human Kinetics, Champaign Illinois , 7-11