The Acute Effects of Continuous and Intermittent Blood Flow Restriction on Sprint Interval Performance and Muscle Oxygen Responses

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THE ACUTE EFFECTS OF CONTINUOUS AND INTERMITTENT BLOOD FLOW
RESTRICTION ON SPRINT INTERVAL PERFORMANCE AND MUSCLE OXYGEN
RESPONSES

by

AARON WIZENBERG
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ABSTRACT

The purpose of this investigation was to examine the effects of intermittent and continuous blood flow restriction (BFR) during sprint interval training (SIT) on muscle mitochondrial function and perceived effort. Fifteen men volunteered to participate in this investigation and were randomly assigned to complete SIT with CBFR, IBFR, and NoBFR. Each SIT session consisted of 2, 30s maximal sprints on a cycle ergometer with a resistance of 7.5% of body mass. Peak power (PP), total work (TW), ratings of perceived exertion (RPE), sprint decrement score (Sdec), and muscle oxygen responses were measured during each sprint. Before (pretest) and after (posttest) the sprints muscle mitochondrial functioning was assessed. There were similar reductions (17,835.6 ± 966.2 to 12,687.2 ± 675.2 J) in TW from Sprint 1 to Sprint 2 for all 3 conditions, and TW was lower (collapsed across Time) for CBFR (14,320.7 ± 769.1 J) than IBFR (15,548.0 ± 840.5 J) and NoBFR (15,915.4 ± 771.5 J). PP decreased to a greater extent from Sprint 1 to Sprint 2 during CBFR (25.5 ± 11.9%) and IBFR (23.4 ± 9.3%) than NoBFR (13.4 ± 8.6%). There were no differences in Sdec (84.3 ± 1.7 %, 86.1 ± 1.5 %, 87.2 ± 1.1%, for CBFR, IBFR and NoBFR, respectively) or RPE that increased from Sprint 1 (8.5 ± 0.3) to Sprint 2 (9.7 ± 0.1) among conditions. Muscle oxygen responses increased across time and were similar for IBFR and NoBFR, while changes in deoxyhemoglobin and total hemoglobin were greater for CBFR. Collectively, the findings of the present study indicated that applying BFR to maximal aerobic exercise is capable of eliciting acute physiological adaptations that may be superior with CBFR relative to IBFR and NoBFR.
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**I: INTRODUCTION**

The training variables that exhibit the most potent effect on muscle adaptation have received considerable debate. Previous investigations (16,18,31,48,57) have manipulated exercise load, frequency of exercise, exercise volume, rest, and mode of exercise to examine how these variables contribute to muscle adaptation. The most potent exercise variable to stimulate muscle adaptation has yet to be elucidated, although exercising at or near volitional exhaustion, regardless of load, appears to be a primary mechanism facilitating muscle growth (19,31,41,58).

The mechanisms mediating muscle adaptation may be a function of metabolic stress and/or mechanical tension (46). Mechanical tension induces mechanotransduction (29,80) that facilitates increases in muscle protein synthesis (3). Metabolic stress, however, results from the accumulation of metabolites (i.e., lactate, inorganic phosphate, and hydrogen) associated with intense exercise bouts (e.g., above the anaerobic threshold) that may facilitate local hypoxia within the muscle (66,69). Unlike mechanical tension, metabolic stress can be achieved without utilizing heavy training loads (46) and is not limited to resistance exercise-based approaches. Thus, alternate exercise interventions including low-load training or high-intensity aerobic exercise may facilitate muscular adaptations through the accumulation of metabolic byproducts.

An effective and efficient exercise modality to induce metabolite accumulation and muscle adaptation is high-intensity interval training (HIIT). HIIT consists of alternating bouts of high-intensity exercise (i.e., above the respiratory compensation point) with intermittent rest periods. HIIT has been associated with improving performance- and health-related outcomes
An alternate and potentially more efficient application of HIIT is sprint interval training (SIT). SIT is a unique form of HIIT which implements discrete supramaximal exercise bouts with similar or longer rest periods than HIIT (27,51,65). For example, ten sessions of SIT that consisted of 8-12, 30-second maximal cycling sprints (130-150% of peak power output [PP] achieved at the maximum value of oxygen consumption [VO₂max]) separated by 2-minutes of rest resulted in similar increases in VO₂max (10.9% ± 7.6%) as HIIT that consisted of 5-7, 2.5-minute submaximal cycling sprints (70-80% PP of VO₂max) separated by 60-seconds of rest (8.9% ± 6.1%) (2). Additionally, following six sessions of progressive SIT that consisted of 4-7 repeated, maximal cycling sprints (all-out effort against a resistance of 7.5% of body mass) separated by 4-minutes of rest, there was a training-induced increase (26 ± 5 to 51 ± 11 minutes) in time to exhaustion (TTE) at 80% of VO₂peak, but no change in TTE for the control group (14). The increase in TTE was associated with increases in citrate synthase (38%) and muscle glycogen (26%) content (14). Therefore, SIT may be a viable alternative to HIIT.

The application of blood flow restriction (BFR) as an exercise adjunct accelerates the accumulation of metabolites compared to non-BFR conditions (66). It is possible, therefore, that the application of BFR during SIT (SIT+BFR) would elicit a more robust training response. For example, four weeks of SIT+BFR that consisted of 4-7 repeated, maximal cycling sprints (all-out effort against a resistance of 7.5% of body mass) separated by 4.5-minutes of rest increased VO₂max by 5.9% compared to SIT without BFR (41). Furthermore, VO₂max increased by approximately 4.5% following 4 weeks of progressive SIT+BFR that consisted of 4-7 repeated, maximal cycling sprints (all-out effort against a resistance of 7.5% of body mass) separated by 4.5-minutes of rest compared to a 0.7% increase in VO₂max for SIT without BFR (68). The
increases in VO\textsubscript{2max} were associated with increased expression of HIF-1\(\alpha\) mRNA that increased 2 fold and 1.5 fold with SIT+BFR and SIT, respectively (68). Thus, these findings (68), in conjunction with those of Burgomaster et al. 2005, suggested that the positive adaptations associated with SIT may be due, in part, to improvements in mitochondrial function as assessed by citrate synthase and HIF-1\(\alpha\). Mitochondrial function and/or capacity can be assessed non-invasively using near-infrared spectroscopy (NIRS). For example, the recovery of muscle oxygen consumption (mVO\textsubscript{2}) as assessed by NIRS was correlated with VO\textsubscript{2max} (35) and was sensitive to training-induced improvements in aerobic capacity (54).

No previous investigations, however, have examined the acute responses of muscle mitochondrial function utilizing NIRS following intermittent and continuous SIT+BFR. For example, intermittent and continuous BFR applied during low-load resistance exercise was associated with similar muscular adaptations, but continuous BFR induced greater discomfort (14-20 RPE) than intermittent BFR (14-17 RPE) (79). While intermittent SIT+BFR was tolerated during repeated maximal sprints (33,41,68), less is known regarding the effects of continuous SIT+BFR (75). Thus, there is a lack of available information regarding the effects of intermittent versus continuous SIT+BFR on indices of mitochondrial function and perceived effort. Therefore, the purpose of this investigation was to examine the acute effects of intermittent versus continuous SIT+BFR on muscle mitochondrial function and perceived effort. Based on previous investigations (14,22,23,67,68), it was hypothesized that intermittent and continuous SIT+BFR will induce greater changes in muscle mitochondrial function compared to SIT without BFR. Furthermore, continuous BFR was hypothesized to be associated with greater perceived effort than intermittent SIT+BFR and SIT.
II: REVIEW OF LITERATURE

SIT Protocols

Bishop et al. (7)

The purpose of this investigation was to determine if repeated sprint ability (RSA) is related to muscle buffer capacity and pH changes in blood and muscle. Thirty-four untrained women (mean (SD) age: 19 (1) yr) performed a graded exercise test (GXT) to determine lactate threshold (LT) via the modified Dmax method and VO2peak. A minimum of 48h later the participants performed a 5x6s repeated sprint ability (RSA) test on a cycle ergometer. The total work recorded during the first 6 s of the 10 s sprint was used as the criterion score during the 5x6s RSA test. To determine if RSA is associated with muscle buffer capacity and pH changes in blood and muscle, capillary blood samples, Mean VO2peak, lactate threshold (LT) were collected, and work decrement score was calculated. There were significant correlations between total work and VO2peak (r = 0.60), LT (r = 0.55), and LT (W) (r = 0.53). The strongest correlation was between total work and work completed in the first sprint (r = 0.87). There were no significant correlations between total work and any muscle or blood variables. Work decrement and VO2peak were correlated (r = -0.62) as was LT (r = -0.56). Work decrement and change in H+ were also correlated, (r=0.41), and blood H+ and muscle buffer capacity in vivo were correlated (r = 0.72). Power decrement and VO2peak were correlated (r = -0.44), LT (r = -0.47), and power decrement and blood H+, (r = 0.36) and muscle buffer capacity (r = -0.53). The results support an association between aerobic fitness and RSA in untrained to moderately trained subjects. However, less than 40% of the variance in RSA was explained by aerobic measures. Oxygen delivery, not oxidative capacity of the muscles is the
primary limiting factor of VO$_{2\text{peak}}$, so VO$_{2\text{peak}}$ may not be a strong predictor of oxidative capacity of the muscles. Altogether, these findings show that VO$_{2\text{peak}}$ and LT are significant predictors of RSA, but also that muscle buffer capacity is linked to RSA. The ability to buffer H+ is important to maintain performance with short recovery periods. The best predictor of performance decrement was a combination of aerobic and muscle buffer capacity.

**Burgomaster et al. (14)**

The purpose of this investigation was to examine the effect of six sessions of SIT on mVO2, VO$_{2\text{peak}}$, and time to fatigue at a high intensity. Sixteen participants completed performance tests consisting of a VO$_{2\text{peak}}$ test and an endurance capacity test at the initial visit and at a 2-week follow-up. 8 of the participants were assigned to the control group (CON) and 8 to the SIT group wherein they performed 6 sessions over 14 days of RSE consisting of 30s all out efforts on a cycle ergometer with a resistance of .075kg/kg body mass. Number of intervals increased from 4-7 over the course of the study. After training the average increase in time to exhaustion was 100%, doubling their time to exhaustion. The control group showed no change in performance. VO$_{2\text{peak}}$ was unchanged in both groups, however expired ventilation and RER were lower in the SIT group. Peak power was higher at the end of the training, as was the fatigue index (FI) when compared to the first training session scores. Maximal citrate synthase activity increased by 38% after the SIT intervention, and resting muscle glycogen increased by 26%. The results of this study show a significant increase in endurance capacity and point to changes in the muscle signaling the increases, as VO$_{2\text{peak}}$ was not increased. The increases also came from a very low training volume, only 15 minutes total over the course of 2 weeks, highlighting the effectiveness of supramaximal SIT training on untrained subjects under long duration sprints.
Thompson et al. (70)

This investigation aimed to determine if 4 weeks of nitrate supplementation enhanced performance and metabolic adaptations in SIT. Thirty-six participants (Eighteen male (age 27 ± 8 yr, height 1.79 ± 0.08 m, body mass 80 ± 13 kg, $\dot{V}O_{2peak}$ 50.4 ± 11.4 ml·kg$^{-1}$·min$^{-1}$; means ± SD) and 18 female (age 23 ± 4 yr) were assigned to three groups: 1) SIT+PL; 2) SIT + BR; 3) NT + BR. All groups completed the same exercise tests at the same absolute work rates. Participants performed a GXT to determine VO$_{2peak}$ and gas exchange threshold (GET). SIT groups performed 4x30s (Increasing to 5x30s) all out sprints at 7.5% of their bodyweight resistance on a cycle ergometer with 4-minute rest periods of active recovery. Blood pressure, blood lactate, muscle biopsies for metabolites and muscle pH, and VO$_2$ were taken. SIT+BR increased resting plasma by [NO$^{-3}$] by ~590% at 2 wk and ~960% at 4 wk, and NT+BR increased resting plasma by [NO$^{-3}$] by ~505% at 2 wk and ~1,050% at 4 wk, but there was no change in resting plasma with SIT+PL. Resting plasma was greater at 4 weeks than at 2 weeks. SBP was not different between groups pre-intervention. SBP was reduced at 2 and 4 wk by 5 ± 6 and 6 ± 4 mmHg, respectively, in SIT+BR and by 4 ± 5 and 10 ± 6 mmHg, respectively, in NT+BR. SBP was unchanged in SIT+PL. DBP was not different and remained unchanged throughout the interventions. MAP was reduced by 3 ± 5 mmHg at 4 wk in both SIT+BR and NT+BR but was unchanged with SIT+PL. Peak work rate increased in all groups after 4 weeks, and after 2 weeks in SIT+BR and SIT+PL. VO$_{2peak}$ was not different between groups pre-intervention. SIT+BR increased VO$_{2peak}$, but VO$_{2peak}$ was unchanged in SIT+PLC and NT+BR. Work rate (WR) at the GET was changed following the intervention in SIT+BR. End-exercise $\dot{V}O_2$ was significantly reduced in SIT+BR and NT+BR, but not in SIT+PL. Time to task failure was improved in SIT+PL and SIT+BR by 163 ± 144 s & 170 ± 90 s respectively. There was no difference in the change in the time to task failure from
preintervention to postintervention between the SIT+BR and SIT+PL. Blood lactate was lower in SIT+BR but not SIT+PL or NT+BR. ATP and PCR were unchanged in all groups. Muscle lactate and pH was improved (lower lactate, higher pH) in SIT+BR. Type IIx fibers were lower in SIT+BR post intervention, and higher in SIT+PL. There were no differences in type 1, or type IIa fiber proportions alone, but when combined there was a higher proportion of type 1 and IIa fibers in SIT+BR, and a lower proportion in SIT+PL. The results of this investigation show that the combination of SIT and nitric oxide supplementation provided greater improvements in incremental exercise performance compared with either alone and led to greater improvements in muscle pH and lactate. Resting plasma levels were elevated, and SBP was lowered with NO supplementation, but not with SIT alone. 4 weeks of SIT has no effect on the O2 cost of submaximal exercise.

Skelly and Gillen (59)

The purpose of this review was to determine the best SIT protocol to induce skeletal muscle remodeling. The review looked at the study by Fiorenza et al. (2018) that involved 12 trained male cyclists completing 3 experimental trials of intermittent exercise (RS), speed endurance (SE), or continuous moderate intensity (CM) cycling. RS consisted of 18x5s all out sprints with 30s of passive recovery, SE involved 6x20s all out sprints with 2min of passive recovery. The SIT protocols were matched for volume and work to rest ratio. The CM protocol was 50m of cycling at 70% VO_{2peak}. Muscle biopsies were collected. The authors concluded that initial events associated with mitochondrial biogenesis are dependent on metabolic stress (RS vs SE) and that high intensity exercise can compensate for reduced exercise volume only when marked metabolic perturbation happens (SE vs CM). The study compared the two work-matched low volume SIT protocols and found that SE elicited greater metabolic stress than RS, evidenced by a higher
exercise induced increase in muscle lactate and lower muscle pH. The greater disturbance associated with SE was associated with a higher PGC-1a mRNA expression in recovery. SE and CM evoked similar increases in mitochondrial gene expression despite the difference in exercise volume, showing that not all low-volume SIT protocols are equal. The greater glycolytic contribution to energy provision associated with 20s as compared to 5s may be an important signal for the enhanced skeletal muscle remodeling. 3x20s all out sprints induces mitochondrial biogenesis and improves markers of cardiometabolic health (Gillen et al. 2014). The protocol by Gillen et al. Evoked large increases in mitochondrial content despite having half the number of 20s sprints. This was in untrained males; trained males may need greater intervals to initiate mitochondrial responses. Increases in plasma adrenaline and muscle H+ concentrations, muscle glycogen and PCr usage during exercise were predictors of post exercise transcription of mitochondrial genes. Enhanced muscle glycogen utilization and lactate accumulation associated with NaHCO3 supplementation contribute to an elevated mitochondrial response. A study has not been conducted yet to determine if NaHCO3 supplementation and SE could augment rates of glycogenolysis and PGC-1a mRNA expression further to optimize the stimulus. Women may not see the same results though, as research has shown that women have a lower catecholamine response, reduced rate of glycogen usage, and lower blood lactate accumulation, which may result in lower metabolic stress. The results of this review show that there is importance in inducing metabolic stress to mediate skeletal muscle mitochondrial responses to SIT training. Future investigations seeking to maximize skeletal muscle remodeling should understand that higher duration sprints evoke higher metabolic stresses and greater changes in mitochondrial genesis.

Paquette et al. (44)

The purpose of this investigation was to compare the physiological and performance
enhancing effects of HIIT to SIT in short race durations with upper body muscles as the primary muscles used. Twelve participants (21±3 yr) performed a max incremental test on a kayak ergometer and time trials (TT) over three racing distances, 200, 500, and 1000m. Then the participants were paired for sex and VO$_{2\text{max}}$ and randomized into training groups: A HIIT group and a SIT group. Both groups performed 9 training sessions over 4 weeks followed by a 4-day recovery with reduced loads before the final incremental test and time trials. NIRS, blood lactate, VO$_{2\text{max}}$ and time performance were all recorded. There were no differences in age, weight, and VO$_{2\text{max}}$ between groups at the start of the study. There were no differences between groups in performance for the 1000 m, 500, and 200m TT. Performance in the 1000m TT improved from 4:42.2 ± 0:33.7 min to 4:30.3 ± 0:18.8 min in HIIT and from 4:27.1 ± 22.3 min to 4:25.1 ± 24.8 min in SIT. Performance in the 500-m improved from 2:10.0 ± 0:11.3 min to 2:07.3 ± 0:11.9 min in HIIT and from 2:05.0 ± 0:08.4 min to 2:04.6 ± 0:12.7 min in SIT. Performance in the 200-m improved from 46.1 ± 4.5 s to 44.7 ± 5.3 s in HIIT and from 44.8 ± 3.8 s to 44.3 ± 5.4 s in SIT. There was a difference between groups for change in performance in favor of HIIT over SIT in the 1000m TT, and small differences for the 500m and 200m TT. In 1000m trials, the slowest performance improved the most in HIIT but not SIT. In the 200m TT a faster performance was associated with a larger improvement and was associated with SIT more than HIIT. Quality of life scores were lower in HIIT than in SIT. VO$_{2\text{max}}$, HR$_{\text{max}}$ and lactate thresholds didn’t change in either group. Performance improved from 2.1% to 3.8% in HIIT and by .5% to 1.3% in SIT, greater than the typical variability of performance for the sport. The results of this study show that HIIT led to greater improvements in performance in all the TT compared to SIT. HIIT induced lower muscle deoxygenation at submaximal intensities, and training led to greater deoxygenation in the biceps and vastus lateralis during the 100m in HIIT and a greater deoxygenation in the latissimus
dorsi during the 100m in SIT (but did not change the muscles max deoxygenation in the 500m TT or the 200m TT. The best predictors of the increase in performance for the 1000m TT were the training related increases in max deoxygenation in the lats and VL, which relates to the contribution of peripheral adaptations to performance in sports with shorter duration races.

Vernillo et al. (71)

This investigation aimed to examine the effects of repeated sprint training on parasympathetic reactivation in adults. Eighteen participants (24.3 ± 3.7 yr) were randomly assigned to either of two groups: SIT or CON. Participants were tested at three points, pre-intervention, mid-intervention, and post-intervention. HR was collected, Sprint decrement score, and PNSr were collected. Testing was performed on an indoor track. The SIT consisted of 15 m sprints with 17s of passive recovery. Total test time was 6 minutes, performed three times per week with at least 48 hours of recovery between sets. The CON group just performed normal daily activities with no intervention. The CON group had lower Sdec scores at the pre-test. In the SIT group, Sdec was decreased between the pretest and mid test, and largely decreased at the post test. Sdec decreased in the CON group at the mid test and at the post test, although the SIT group had greater improvement in their Sdec. Total sprint time was greater in the SIT group than in the CON group at the mid test and posttest. HRR60s gradually increased in the SIT group as the training intervention progressed, reaching higher values compared to the pretest at both the mid test and posttest. These results show higher performance and parasympathetic levels in the SIT group compared to the CON group, showing that parasympathetic reactivation is higher following repeated sprint training. The change in postexercise HR recovery at 60 seconds was largely correlated with the improvement in RS performance. HRR and HRV are both used to assess adaptations of cardiac functions to exercise. Lower levels are associated with heart disease,
hypertension and myocardial infarction. The authors suggest that there is a strong rationale for using RS training, and that their results suggest long term RS exercise may have a significant and positive effect on postexercise parasympathetic reactivation and may help decrease cardiovascular disease risk.

Buchheit et al. (11)

The purpose of this investigation was to compare the effects of active recovery compared to passive recovery on muscle deoxygenation during RSE. Ten participants (26.9 ± 3.7 yr) performed 6x4s sprint intervals on a non-motorized treadmill with 21s of active or passive recovery. Mean running speed, speed decrement, VO₂, HHb, and blood lactate were collected for both conditions. On three different visits, separated by at least 48 hours, in a random order, participants completed two sets of 6x4s maximal sprints with 21 seconds of active or passive recovery. Mean average speed was significantly lower and sprint decrement score was significantly higher in the active recovery condition. All other kinetic parameters were also significantly higher in passive recovery. Active recovery has significantly lower max speed and stride frequency during the six 4s all out sprints. HR, VO₂, VCO₂, RER, RR and Ve values were all significantly higher for active recovery compared to passive recovery. There was no significant difference between HRₘₐₓ reached during the trials between active recovery and passive recovery. Mean values of HR, VO₂, blood lactate, and RPE were significantly higher in active recovery compared to passive. Mean post exercise blood lactate values were also significantly higher in active recovery. However, there were no differences between the 3- and 5-minute post exercise lactate levels between conditions. Mean VO₂ change was significantly lower in active recovery compared to passive recovery (13.1 ± 0.5 ml.min⁻¹.kg⁻¹ versus 19.7 ± 4.6 ml.min⁻¹.kg⁻¹). Mean recovery of deoxyhemoglobin was significantly lower in active recovery. The results of this study
show that cardiorespiratory stress was significantly higher in active recovery compared to passive recovery. The increase in VO$_2$ required to run at 2 meters per second resulted in an increased VO$_2$ during recovery periods even after the first sprint. The higher oxygen cost, with inadequate recovery/delivery of oxygen likely resulted in the increased muscle deoxygenation seen in active recovery, the authors mention.

**Racinais S et al. (49)**

The purpose of this investigation was to investigate muscle deoxygenation and changes in neural drive during repeated cycling sprints. Nine participants (25 ± 2 yr) performed 10x6s maximal sprints against a load of .9 N*Kg body mass with 30 seconds of active recovery. Peak power was significantly decreased across sprint repetitions, with an increase in oxygen uptake across sprints as well. The increase in HHb was constant throughout sprint repetitions but failed to recover to previous levels after each sprint. RMS activity did not significantly change across sprint repetitions. MVIC from rest to postexercise showed a significant decrease in force. Peak twitch force produced decreased, along with a significant decrease in percent voluntary activation estimated by twitch. RMS/M-wave ratio was significantly decreased post sprint as well. The results of this study showed that there was a significant power decrease across repeated sprints with an increase in muscle deoxygenation during sprints of 6 seconds with 30 seconds of active recovery. Neural drive was also decreased post sprints. Even with progressive muscle deoxygenation, oxygen usage remained the same in the muscle throughout the sprints. RMS activity during acceleration of sprints was significantly decreased, showing a decrease in neural drive to the muscles, confirmed by a decrease in voluntary activation and RMS/M-wave ratio.

**Nalçakan et al. (43)**

The aim of this investigation was to determine if reducing sprint duration from 20s to 10s
influences acute responses and aerobic capacity adaptations. Thirty-six participants (22 ± 3 yr) were randomly assigned to either a standard REHIT protocol (REHIT20) or a reduced REHIT protocol (REHIT10). In the REHIT20 group participants would perform 20s sprints, while REHIT10 would perform 10s sprints. Both groups completed 2 sprint repetitions during their exercise sessions and had three sessions per week for six weeks. Body mass, VO$_{2\text{max}}$, mood states, and RPE were measured in all participants. Body mass increased from pre- to post-intervention, with no difference between groups. Increases in VO$_{2\text{max}}$ were greater in the REHIT20 group. Significant exercise induced improvements in mood states were observed for tension, depression and vigor as well as total mood disturbance with no difference between groups. Although several other studies found no differences between 2-4 weeks of 4-6x30s sprints vs 10s sprints, this investigation observed a significant increase in VO$_{2\text{max}}$ in the longer sprint duration group. The results of this study show that reducing sprint duration from 20s to 10s minimizes or removes the VO$_{2\text{max}}$ improvements following SIT training. The shorter duration does not impact RPE or acute responses on mood state.

Minahan et al. (40)

The purpose of this investigation was to determine the effect of deceleration on repeated sprint running compared to no deceleration. Fourteen participants completed two randomly assigned trials of RSE, with or without deceleration. The RSE consisted of 4 sets of 4x6s sprints with 30 seconds of recovery between sprints and 144 seconds between sets. Countermovement jump performance, velocity, sit and reach distance, soreness, and blood analysis were measured in all participants. Peak and mean velocities were not different between groups, and there was no difference in speed decrement score between groups. The average oxygen uptake for the entire protocol was not significantly different between groups. Blood lactate was not different between
groups. CMJ height was not different between groups. There was no difference between groups in perceived soreness. The results of this study show that the removal of deceleration from repeated sprint running has no impact on metabolism or performance during or after the repeated sprint exercise. There are also no changes in markers of muscle damage.

Wang et al. (73)

The aim of this investigation was to determine the influence of fatigue of lower limb muscle groups on exercise performance during SIT. Ten participants (21 ± 4.67 yr, 7 men, 3 women) volunteered for the study. All were highly trained competitive elite cyclists, with 6 days per week and 8 hours per day of training for at least 7 years. The SIT protocol consisted of 5x6s sprints with 24 seconds of recovery between each sprint. The resistance was set at 8-10 for men and 6-8 for women. EMG signals were collected during each trial. Power and cadence progressively decreased with increasing sprints. The MNF of the rectus femoris, vastus lateralis, hamstrings, and gastrocnemius decreased significantly with increasing sprints. MNF of the hamstrings and TA showed significant increase after recovery time, while the MNF of the SOL showed significant decrease after recovery time. Based on previous research, which showed that peak muscle activity of the VAS, RF, and GAS during sprint cycling is nearly 100% of MVIC values, the authors suggest that the fatigue of these muscles play a large role in fatigue development during SIT. The results of this study showed that the HAM and TA recovered significantly during rests, while the RF, VAS, HAM, and GAS fatigued progressively. The degree of fatigue in the HAM and VAS were closely related to the decrease in exercise performance.

Wang et al. (74)

The aim of this investigation was to determine the effects of SIT and β-alanine (BA) supplementation on performance. Thirty-eight participants were randomly assigned to either a
normoxic β-alanine group (NB, n = 11, 22.6 ± 2.9 yr), a normoxic placebo group (NP, n = 8, 22.6 ± 2.9 yr), a hypoxic placebo group (HP, n = 9, 22.7 ± 2.8), and a hypoxic β-alanine group (HB, n = 10, 22.5 ± 2.7). The SIT protocol consisted of 2 sessions per week for 4 weeks of 3 sets of 5x10s sprints with 7.5% body mass resistance with 20 seconds of active recovery between reps and 5 minutes between sets. Repeated sprint testing consisted of 6x10s cycling at 7.5% body mass resistance with 60s of active recovery between sets. Participants who were in the supplementation groups received 6.4g of sustained release BA per day. Blood samples, biochemical analysis, GXT, and 3 minute all out tests were measured in all participants. There were significant differences between SpO2 measures at rest, during training and the decline from rest to training between hypoxic and normoxic conditions. Resting SpO2 were higher in both normoxic conditions compared to hypoxic conditions (96.4 ± 0.9% for NB and 97.2 ± 0.7% for NP compared to 88.8 ± 2.1% for HB and 89.6 ± 2.1% for HP). SpO2 during training was also significantly higher in normoxic conditions compared to hypoxic conditions, with values of 94.1 ± 3.5% for NB and 95.7 ± 0.8% for NP compared to 78.6 ± 2.7% in HB and 81.5 ± 4.6% HP. Anthropometric and hematological measurements were unchanged in all conditions after the training intervention. Greater VO_{2max} and PP values were observed after the training intervention, with no difference between the BA groups and placebo groups. Hypoxic vs normoxic did not affect 3-minute all out performance. A main effect for supplement was seen for AWC with HB and NB being greater than HP and NP. A main effect for condition was seen for total work, with HB and HP being greater than NB and NP. No effect for condition or supplement was seen for lactate concentration or heart rate 60 seconds posttest. Repeated sprint training in hypoxia and BA supplementation improves different aspects of performance. Hypoxia resulted in greater values for fatigue threshold, exercise tolerance, recovery and total work capacity compared to normoxia. BA resulted in greater AWC
compared to placebo. There were no changes in hemoglobin and hematocrit values in any group. Hypoxia improved exercise tolerance via better maintenance of steady-state VO\textsubscript{2} and lactate at high intensities. The results of this study show that SIT in hypoxia improved fatigue threshold, exercise tolerance, cardiovascular recovery, and total work capacity to a greater extent compared to SIT in normoxia. BA supplementation maintained the AWC following SIT training.

Macdougal et al. (37)

The purpose of this investigation was to determine the effects of SIT on glycolytic and oxidative enzyme activity in skeletal muscle. Twelve participants (22 ± 2 yr) were assigned to complete a 3 sessions per week for 7 weeks program consisting of 4-10x30s sprints with 4 minutes of recovery (decreasing to 2 minutes 30 seconds). All measurements were performed before the 7-week intervention and after. Measurements tested anaerobic power, aerobic power, and enzyme activity. Anaerobic power was tested via 4 repeated Wingates with 4 minutes of rest between. Aerobic power was measured via a VO\textsubscript{2max} test. Enzyme activity was measured from muscle biopsies. PP and total work for sprints 2-4 were significantly higher after training. VO\textsubscript{2max} increased from 3.73 ± 0.13 to 4.01 ± 0.08 l/min (P ≤ 0.05). Body mass did not significantly change after training, therefore relative VO\textsubscript{2max} was also increased. Muscle enzyme HEX was 56% higher after training, a significant increase. PFK was 49% higher after training, a significant increase. Although total phosphorylase activity and LDH changed by 9% and 7% respectively, they did not change significantly. Citrate synthase activity increased significantly, by 36%, MDH by 29%, and SDH by 65%. The results of this study show that 7 weeks of SIT training significantly improved VO\textsubscript{2max}, PP, and maximum activity in glycolytic and oxidative enzymes in skeletal muscle. The authors speculate that the increase in PP may have resulted from the increased enzyme activity and Na-K pump capacity while increased mitochondrial activity may have resulted from increased
pyruvate flux rate.

Yamagishi and Babraj (78)

The purpose of this investigation was to determine the time course of adaptations to SIT with different durations. Twenty-five participants were randomly assigned to the 15 second (15TG, 30 second (30TG) group, or the control group (CON). Participants performed baseline measurements three times to determine VO$_{2\text{peak}}$, critical power (CP), and a 10km time trial, and completed these again after the training protocol. The SIT protocol for 15TG consisted of 4-6x15s sprints with 2 minutes of active recovery at 40% VO$_{2\text{peak}}$. The 30TG protocol consisted of 4-6x30s sprints with 4 minutes active recovery at 40% VO$_{2\text{peak}}$. Both training groups performed their respective protocol twice a week for 9 weeks. The CON group did not perform a training intervention. Following the 9 weeks blood pressure and body composition did not change in either of the SIT groups or in the CON group. Both training groups significantly improved VO$_{2\text{peak}}$, 15TG by 12.1% and 30TG by 12.8%. O2 pulse was also significantly increased in both groups. Time trial performance significantly increased by 16.2% in 15TG and 12.8% in 30TG. Critical power was significantly increased in 15TG, with a similar increase in 30TG, but not significantly. No performance measures were changed in CON. Blood lactate significantly increased following the sprints in both groups. VO$_{2\text{peak}}$ rapidly improved with training, with the highest values observed after 3 weeks in both groups. VO$_{2\text{peak}}$ gain dropped after the third week. O2 pulse followed the same pattern in both groups, while TTE (time to exhaustion) was not significantly increased until the 6$^{\text{th}}$ week of training in both groups, with the highest values obtained after 9 weeks. RSA was not significantly changed after 9 weeks in 30TG. 15TG significantly improved PP and total work in RSA. The results of this study demonstrate 15 second sprints can cause similar or better adaptations compared with 30 second sprints. In addition to this, the adaptations to VO$_{2\text{peak}}$ are
rapid and plateau after 3 weeks, while TTE performance does not improve until the 6\textsuperscript{th} week of training and continued to improve through the 9\textsuperscript{th} week of training.

**Blood Flow Restriction**

Kojima et al. (33)

The aim of this investigation was to determine if BFR during rest would accentuate deoxygenation levels without compromising fatigue resistance. Seven active males (age: 21.7 ± 0.8 yr) completed RSE (5x10s, 40s rest between sprints) on an electromagnetically braked cycle ergometer at 7.5\% body weight resistance either with or without BFR (140 mmHg for 30s) (BFR vs CON trial) during rest periods between sprints on different days. Muscle oxygenation, power output, and heart rate were recorded during exercise, lactate concentrations were evaluated before and after exercise. Oxy-Hb levels were lower in the BFR trial than in the CON trial during sprint and rest periods. There were no significant differences in mean power output between trials. Total power output was not different between trials either. Peak power output was significantly decreased in trial 4 and 5 with BFR when compared to CON. Both oxy-Hb and StO2 levels were significantly lower with BFR during rest, but deoxy-Hb and total Hb levels are not. Power production was not comprised either, showing that BFR during rest of RSE could be effective for producing sustained local hypoxia in working muscles. BFR during rest interferes with reoxygenation, however, total-Hb, a marker of blood volume, remained unchanged between trials. However, blood lactate concentrations were not significantly different between trials, which means that metabolic processes may not be changed in BFR trials of RSE. Training stimulus was
increased without a decrease in training quality with BFR. The results of the study show that BFR during rest can accentuate deoxygenation levels without decreasing mean power output or changing blood lactate concentration when compared to a non-BFR rest.

Taylor et al. (68)

The aim of this investigation was to determine if a combination of sprint interval training (SIT) and blood flow restriction (BFR) enhances max aerobic physiology and performance and the mechanisms of adaptation. 28 trained males (age: 27 ± 7 vs 26 ± 5 yr, cycling 120 ± 66 km per week) participated in the studies. Participants were assigned to either a SIT and BFR group or SIT only group. Study 2 was acute, and subjects performed a bout of SIT with postexercise BFR or SIT alone. Eight subjects (age: 32 ± 7 yr; height: 180±10; body mass 75.3±9.1; VO2max 4.3±.41) participated in study 2. In study 1, the subjects were randomly assigned to the CON or BFR intervention, all measures were conducted on an SRM cycle ergometer. VO2max and maximal aerobic power (MAP) were established. Subjects completed a 15 km time trial and completed 2 sessions of the SIT program (4/5/6/7 x 30s 4.5 minutes recovery on a mechanically braked cycle ergometer at 0.075 kg) per week for 8 weeks. In the BFR group, participants immediately dismounted and would lay supine on a couch for lower limb BFR to 130mmHG for 2 minutes. In study 1, average power output for CON was greater than BFR, and therefore total work done was greater in CON than in BFR. Relative (and absolute) VO2max increased in BFR but were unchanged in CON. The findings of this study demonstrate that combing BFR with SIT can increase VO2max in trained individuals. The exact mechanisms behind the improvement are unclear, however the results suggest skeletal muscle remodeling in capillary density could be one factor behind the increase. VO2max was not improved through SIT alone, but it's important to note that VO2max is not a direct measure of performance. Despite an improvement in VO2max
with BFR, 15km time to exhaustion (TTe) performance remained the same.

Willis et al. (75)

This study aimed to measure the changes in oxygenation, physiological responses, and neuromuscular fatigue as a result of various levels of BFR during RST. Eleven (six men and five women; 26.7 ± 4.2 yr, 68.0 ± 14.0 kg) healthy and recreationally active people (4 hours per week minimum training) participated in the study. Participants would perform 3 sets of: 10s sprints with 20 seconds of recovery until exhaustion. VO$_{2peak}$, NIRS of the VL and prefrontal cortex, and neuromuscular fatigue were measured. RST were conducted with either 0%, 45%, or 60% occlusion (conducted on different testing visits), with 0.8 Nm*kg torque. Blood pressure cuffs were bilateral and inflated to the pressure condition of that test day 1 min prior to pre-RST measurements. For the RST, BFR cuffs were inflated 5 sec before the start of the test and remained inflated for the entire duration of the test and through the post-RST measurements. Task failure was defined as a cadence < 70 rpm. Recovery was at a resistance of 20 W. BFR at 45% and 60% decreased the number of sprints performed to exhaustion by 47.4 ± 25.4% and 65.8 ± 36.9%, respectively, when compared to 0% BFR. Total work was similarly decreased by 52.5 ± 22.9% at 45% and 68.6 ± 32.6% at 60%. Maximal heart rate was lower at 60% by 7.6 ± 8.6% compared to 0%. Peak oxygen uptake was reduced by 12.6 ± 9.3% at 45% (P < 0.05) and 18.2 ± 7.2% at 60% (P < 0.001) during RST when compared with 0%. Peak oxygen uptake was reduced by 6.1 ± 6.2% from 45% to 60%. Other physiological variables did not result in any significant differences (RER, RR, blood lactate). Set duration and occlusion percentage both decreased VL oxygenation (delta HHb) in BFR. MVC, VAL, and RMS/M-wave all decreased at the 60% BFR compared to 0%. There was a decrease in MVC and VAL from 45% to 60%. The results of this study show that performance (number of sprints and total work) decreased with increased BFR, which also
decreased VO\textsubscript{2}peak. Muscle volume blood changes increased with both levels of BFR, and large decrements of MVC, VAL, and RMS/M-wave were induced with BFR. Change in HHb may not be a valid measure of Oxygen extraction. Total hemoglobin was not constant.

Willis et al. (76)

The purpose of this investigation was to determine the vascular and oxygenation responses to RSE with BFR, systemic hypoxia, or both. Sixteen participants (eleven men and five women; mean ± SD; 26.4 ± 4.0 yr old; 73.8 ± 9.8 kg; 1.79 ± 0.07 m) completed 4 conditions (400m, 3800m, 0% and 45% BFR) of RSE of 10s sprinting 20s recovery to exhaustion on an arm cycle. The Wingate mode of the arm cycler ergometer was fixed with 0.4Nm * kg for each participant. VO\textsubscript{2}peak, HR, and NIRS data were obtained throughout the RST. With BFR, cuffs were placed bilaterally and inflated 5s before the RST began and remained inflated until the end of the post-RST measures. Task failure was defined as <70rpm. The total number of sprints was lower in both hypoxic conditions (3800 0% BFR, and 3800 45% BFR) when compared with the control. Mean power was unchanged through all conditions. Total work decreased 23% from control (400, 0%) and hypoxia alone, as well as 53% between the control and hypoxia with BFR, which was a decrease of 39% from normoxia with BFR. VO\textsubscript{2} was decreased in both hypoxic conditions compared to normoxia, and max HR was lower in hypoxia with BFR than the control condition. Blood flow was 52% lower in hypoxia with BFR than just hypoxia, and 48% lower in normoxia with BFR compared to hypoxia without BFR. Systemic hypoxia demonstrates a higher shear rate compared to BFR only. The results of this study indicate that arm cycling is impaired the most by systemic hypoxia mainly due to decreased convective oxygen delivery. BFR alone and with systemic hypoxia caused greater changes in total hemoglobin causing vascular responses unique to BFR. There were no additional effects of the combination of BFR and systemic hypoxia when
compared to BFR alone. Hypoxia induced vasodilation may increase blood volume to increase blood flow and allow greater oxygen utilization, which may limit oxygen delivery (VO2peak). BFR blunts the effect of hypoxia induced vasodilation, even when combined with high intensity. The investigation shows that arms and legs show different vascular properties and responses when BFR and hypoxia are introduced in high intensity exercise. In systemic hypoxia variations in perfusion are likely from vasodilation, whereas changes in perfusion under BFR may result from stimulation of specific mechanisms.

**Paradis-Deschênes et al. (45)**

This investigation aimed to determine the benefits of SIT with ischemic preconditioning (IPC) on performance and physiological adaptations. Twenty participants completed the study. There were 16 lab visits with 8 training sessions over a 4-week period. Eight of the visits were for testing evaluations. Participants were pair-matched based on age, VO2peak, MAP, and time trial (TT) performance in addition to relative peak power (PP) and mean power output (MPO) obtained during a Wingate. Participants were then randomly assigned to either IPC or placebo (PLA) group. Both groups had the same training intervention, consisting of 4-7x30s springs with 4.5 min recovery periods. For the IPC condition, blood pressure cuffs were inflated to 220mmHg while PLA received 20mmHg for 5 minutes. SpO2, HR, NIRS, power output, RPE, and blood measurements were collected. Both groups increased power output over the course of the training. From pre- to post- training, IPC maintained the index of muscle blood volume, while it declined in PLA. The time to completion for the 5km time trial was faster in IPC compared to PLA. PLA did not alter change in deoxyhemoglobin but increased the change in tissue saturation index from pre to post. IPC increased change in total hemoglobin from mid- to post training, as change in deoxyhemoglobin. Compared to PLA, IPC increased change in deoxyhemoglobin from pre to post.
training the first and second half of the TT. IPC also increased SpO2 and HR after training compared to PLA. Ischemic preconditioning before SIT led to greater improvements in MPO (5%), 5-km completion time (~2%), and increased fatigue resistance during a Wingate (~8.5%). PP during the Wingate was similarly increased in both groups. The authors suggest that muscle perfusion and peripheral O2 extraction were increased after IPC based on their findings. The results of this study show that 4 weeks of IPC applied before SIT causes greater increases in time trial performance than the same protocol without IPC. This was associated with increased local perfusion and muscle oxygen extraction without changing hematocrit, VO2max, or VO2peak.

Mitchell et al. (41)

The aim of this investigation was to determine the efficacy of SIT with BFR on enhancing CP. Twenty-one participants (23 ± 5 yr) volunteered in the study. All participants had a VO2max of at least 60ml*kg. All participants were randomized into two groups, SIT on its own (CON) and SIT with blood flow restriction (BFR). All participants completed baseline testing measures followed by a 4 week SIT protocol and finally the post-training testing measures. VO2max and MAP were measured during the testing protocols and some participants consented to provide muscle biopsies. The SIT protocol consisted of 2 sessions per week for 4 weeks each session consisted of 4-7x30s sprints with recovery periods of 4.5 minutes in a semi-supine position. For the BFR condition blood flow was occluded at 120mmHg within 25 seconds of sprint completion and maintained for 2 minutes. Absolute and relative VO2max increased in the BFR group but not in the CON group. Absolute MAP was unchanged in both groups while relative MAP increased with training in both groups. Both absolute and relative peak power increased after the SIT protocol in both groups. All measures of capillarization were unchanged with training in both groups. Mitochondrial enzyme protein content was unchanged in both groups after training. The study
demonstrates that 4 weeks of SIT increased CP in trained individuals. BFR did not enhance the effect of the SIT training, however. Muscle capillary content and mitochondrial protein content did not change with SIT in either group. VO\textsubscript{2max} did increase more substantially with BFR than SIT. The results of the study show that the addition of BFR during rest periods of SIT did not cause greater increases in CP than SIT without BFR.

**Systemic Hypoxia**

Bejder et al. (5)

This investigation aimed to determine if hypoxic living conditions can increase power output during exhaustive cycling, a time trial, 3-minute performance, and RSA. Seven highly trained athletes (5 men, 2 women 31 ± 2 yr, 185 ± 4 cm, and 80 ± 10 kg for men and 29 ± 4 yr, 168 ± 6 cm, and 63 ± 4 kg for women) spent 8h per day in a tent with either hypoxic air (LHTL) or normoxic air (PL). All participants performed a VO\textsubscript{2peak} test, a time trial, a 3 minute all out test, and a RSA test under both conditions. The RSA test consisted of 8x30s all out sprinting at the same resistance as the 3-minute test, 115% of VO\textsubscript{2peak}, with 1 minute of rest between sprints. Peak workload during the VO\textsubscript{2peak} test was not significantly different, time trial mean power output (MPO) and 3 minute all out MPO were also not significantly different. Mean and peak power during RSE was not significantly different between conditions. These results show that normobaric LHTL does not alter power performance in VO\textsubscript{2peak}, time trial, 3-minute all out, or RSA tests.

Gatterer et al. (25)

The aim of this investigation was to examine running distance and acceleration patterns during hypoxic versus normoxic repeated sprinting, as well as determine if RPE, oxidative stress,
and breathing frequency are involved in the performance outcomes. Eight participants were randomly assigned to perform two sprint sessions, one in normobaric hypoxia and one in normoxia. The sprinting sessions consisted of 3 sets of 5x10s repeated maximal shuttle running, with the number of runs completed recorded. Participants had 30 seconds of rest between the five reps of 10 seconds, and 5 minutes between the three sets. After the sprints RPE and capillary blood were taken. HR, Ventilation rate (VR) were taken continuously throughout the sessions. Total distance covered during the sprints was not different between conditions (Δ −8.3 ± 14.3 m, 95% CI −20.2 to 3.6, p = 0.144, ICC: 0.656). Sprinting distance decreased during the third (last) set only under hypoxic conditions. Acceleration patterns did not differ between conditions. Breathing frequency during both sprinting and recovery in both conditions was increased in hypoxia (p = 0.003, ES: 1.5, ICC: 0.978 and p = 0.020, ES: 1.03, ICC: 0.963, respectively). Increases in breathing frequency from normoxia to hypoxia were associated with individual reductions in sprint differences (r = −0.792, p = 0.019). Heart rate response, blood lactate, RPE, and redox status were all similar between conditions. The results of this study indicate that in lower volume hypoxic conditions sprint performance is not decreased compared to normoxic conditions. Preserving the total work in sprinting maintains the mechanical stress, while the hypoxic conditions increase the physiological stress placed on the body. The authors mention that the main factor in performance loss is believed to be oxygen availability. However, other factors may influence performance in hypoxia. Breathing patterns, RPE, and oxidative stress may also play a role in performance loss. In this study only breathing frequency differed between conditions suggesting that in low volume sprinting done in hypoxic conditions, breathing frequency may play a larger role in performance loss than RPE and oxidative stress.
The purpose of this investigation was to determine if 12 sessions of repeated sprint training in hypoxic conditions improved performance to a greater extent than equivalent training in normoxia. Thirty participants (males age 18.4 ± 1.5 yr) completed 12 sessions of SIT on a non-motorized treadmill of 10x6s sprinting with 30s of recovery over 4 weeks in either hypoxia or normoxia. Participants completed endurance and sprint tests pre and post training while measuring speed, HR, blood lactate, muscle deoxygenation, RER, VO2, VCO2, VE, and SaO2. Distance covered in the Yo-Yo IR1 test was significantly increased with training, with greater distance covered in the hypoxic group compared to the normoxic group. Five-meter sprint performance and time taken in the 20 m RS test improved after training, with no significant differences between groups. There were no significant changes to 10 or 20 m sprint performance or speed. There was a tendency for greater oxygen consumed during the testing after hypoxic training compared to normoxic training, with a significant increase in total VCo2 for both groups. There were no significant differences in VCo2 between groups. There was no significant difference in total VE, SaO2, HR, or lactate concentrations after training or between groups. Cerebral deoxygenation was lower across sprints after hypoxic training compared to no response after normoxic training. Quadriceps deoxygenation did not change. Total distance covered in the RSA tests improved after training in both groups but was not significantly different between groups. Speed decrement during the RSA test improved in both groups. There was a greater degree of improvement in the hypoxic group, although it was not significant due to the high variability. The authors suggest the performance improvement may have been a result of the lower cerebral deoxygenation in the hypoxic group as cerebral deoxygenation has been linked to central fatigue. The results of this study demonstrate that 4 weeks of hypoxic training is able to elicit greater performance in the Yo-
Faiss et al. (20)

The aim of this investigation was to compare repeated sprint training hypoxia versus normoxia and to assess blood perfusion and molecular response at the muscular level. Fifty participants age (35 ± 7 yr) completed 8 training sessions over 4 weeks. Participants were randomly assigned to either the normoxic group (RSN, n = 20), hypoxic group (RSH, n = 20), or the control group (CON, n = 10). The CON group completed only the PRE and POST sessions, without specific training in the 4-week interim. Blood samples and muscle biopsies were collected (only five subjects in CON had muscle biopsies taken). Training sessions consisted of 3 sets of 5x10s sprints with 3 minutes of active recovery at 120W. Lactate dehydrogenase (LDH) activity, RPE, NIRS, EMG, RSA tests, 3-minute critical power, and pain scores were taken. Wingate's were performed at 0.8 Nm*kg. Total work and training intensity were similar for RSN and RSH, with mean HR being higher in RSH. From pre-test to post-test the average power of all sprints during the RSA test increased (6±7% vs. 7±8%, NS) in RSH and RSN respectively, but not in CON. The number of sprints before exhaustion was increased in RSH but not RSN (9.4±4.8 vs. 13.0±6.2, p<0.01; 9.3±4.2 vs. 8.9±3.5, NS). 10s average power in successive sprints was significantly improved until the 9th sprint in RSH and the 7th sprint in RSN. In RSH the 10s power output was significantly better in the 10th and 11th sprints in Post compared to the 9th sprint in pre. Significant group by time interactions (RSH vs RSN, Pre- vs Post-) were found in the number of sprints and total work performed in the RSA test. Average power in the 3 minute all out test were unchanged between pre and post in all groups. RSH and RSN improved single 10s sprint and Wingate performances similarly. After training Δ[tHb]av increased to a greater extent in RSH than in RSN (F=15.8, p <0.01). Δ[HHb]av increased similarly in RSH and RSN. The average Δ[tHb]/Power ratio increased to a greater extent in RSH than in RSN. Average RMS activity of the VL was not
different in RSH, RSN or CON. Average RMS activity of the BF was not different in RSH, RSN, or CON. mRNA gene concentrations of hypoxia inducible factor, carbonic anhydrase III, monocarboxylate transporter-4, and lactate dehydrogenase were increased in RSH only. Mitochondrial transcription factor A (TFAM), peroxisome proliferator-activated receptor gamma coactivator 1α, and monocarboxylate transporter-1 were decreased in RSH only. LDH activity was increased in RSH but not in RSN, citrate synthase activity was not different and did not vary significantly in RSH and in RSN. The results of this study show that SIT in hypoxia results in greater RSA to exhaustion when compared to the same training in normoxia. Increased variations of blood perfusion occur from hypoxic SIT, molecular adaptations large enough to improve RSA performance occur with hypoxic SIT. The mechanisms involved in these improvements are likely different than ones associated with the Live High Train Low method the author's mention. The authors mention that in RSH the amplitude of blood flow variations were increased during sprints, and the increased mRNA expressions suggest that a potential increase of the glycolytic activity in muscle may occur.

Billaut et al. (6)

The purpose of this investigation was to examine the interaction between the development of peripheral fatigue, muscle recruitment, and performance during RSE. Ten participants (22.8 ± 4.4 yr) were randomly assigned to complete either a hypoxic or normoxic trial first. All participants performed three sets of 5x5 s cycle sprints against various resistances with 5 minutes of recovery in between sprints. Pedaling rate was matched between sprints. Arterial oxygen saturation (SpO2), blood lactate concentration, NIRS, EMG, twitch, and RPE were collected. Performance for the first sprint was not significantly affected by hypoxia, however total mechanical work was lower in hypoxia. The reduction in mechanical work in hypoxia was significantly larger than in normoxia.
There was no significant interaction for any of the physiological variables measured. There were large differences in average arterial oxygen saturation between the two conditions, however. Changes in muscle tissue saturation index (TSI) were not significantly different between the two conditions, which the authors state supports the vastus lateralis being in a similar state of oxygenation regardless of the environmental conditions. Cerebral TSI was affected by the conditions, being significantly lower in hypoxia compared to normoxia. Changes in blood lactate concentrations were similar in both conditions throughout all sprint sets. Total quadriceps EMG activity was significantly lower over the course of the RSE in both conditions, but the main effect of the condition indicated that RMSsum was 13.7% lower in hypoxia than in normoxia. With the twitch, mean potentiated quadriceps twitch force (Qtw,pot) was reduced in both conditions, but was not significantly different between conditions. M-wave amplitude and duration were unchanged after RSE in both conditions, with no significant effect of hypoxia compared to normoxia. Average maximal voluntary force of the quadriceps were reduced from pre to post exercise in both groups and was significantly lower in hypoxia compared to normoxia. The RMSMVC/Mamp ratio decreased from pre to post exercise in both groups and was significantly lower in hypoxia compared to normoxia. RPE also increased during the RSE in both groups, however, it was similar between groups. The results show that with RSE in hypoxia, central oxygenation, quadriceps activation, and cycling performance were all lower when compared to normoxia. The magnitude of the quadriceps fatigue induced by the sprints was similar between conditions. The authors interpreted the results of the study to suggest that the CNS regulated the development of peripheral muscle fatigue via reductions in muscle recruitment and exercise intensity. Lower performance in hypoxia, despite similar muscle conditions as evidenced by twitch and M-wave responses may indicate anticipatory regulation of exercise performance. The results,
however, do not rule out the effect of oxygen desaturation on reducing motor output to the working muscle.

Bowtell et al. (9)

This investigation aimed to determine the effects of various levels of hypoxia on RSA, cardiorespiratory, and neuromuscular responses to construct a hypoxic dose response. Nine participants (23.6 ± 3.7 yr) completed 10x6s sprints with 30 seconds of recovery under 5 different conditions: Normoxia and four levels of hypoxia (12%, 13%, 14%, 15%). Muscle oxygenation was collected with NIRS, blood oxygen saturation, HR, and pulmonary gas exchange were collected during the RSE. Treadmill speed was recorded, capillary blood samples were taken pre- and post- the warm-up, before and immediately after and 5 minutes after the RSE protocol. EMG activity was also measured. Peak speed during the 10 sprints was lower in hypoxic conditions. Fatigue index was significantly greater during the 12% trial versus the 21% trial and Sdec was greater in the 12% trial than all others except 14%. Blood oxygen saturation was significantly lower in hypoxic conditions during both sprints and recovery intervals between sprints. Blood oxygen saturation decreased to a greater extent in hypoxic conditions than normoxic conditions. HR was increased during sprints across repetitions and to various levels between conditions. Minute ventilation increased across sprints and was higher and increased more quickly in hypoxic conditions. VO2mean during sprints and recovery between sprints increased across sprints and was significantly different between conditions. Total oxygen consumed for the entire protocol was lower in the hypoxic conditions. Oxygen consumed during recovery periods (30s) was a significantly higher proportion of total oxygen consumed in normoxic conditions than in hypoxic conditions, however this was reversed in the inter-set recovery periods (5 minutes). Blood lactate was higher in hypoxic conditions post sprints. Max and PostMax Tissue deoxyhemoglobin
increased across sprints. MaxHHb was significantly lower in the normoxic conditions compared to the hypoxic conditions. The total area under the normalized HHb curve was significantly different between conditions, with a lower total area under the curve in the normoxic conditions compared to hypoxic conditions. Normalized iEMG decreased across sprints with a strong tendency for a main effect of condition for the iEMG data, with lower iEMG in hypoxic conditions than in normoxic conditions. The sum of the iEMG across the 10 sprints was also lower under hypoxic conditions. The duration of muscle activation decreased across sprints and was significantly shorter in hypoxic conditions. All EMG data were normalized to corresponding MVC peak values in each trial. The results of the investigation show that physiological responses related to RSA were larger as inspired oxygen decreased to 13%, with higher HR, minute ventilation, oxygen debt, muscle deoxygenation, and lower EMG. Physiological demands were attenuated at 12%. Sprint performance was not significantly impacted by hypoxic conditions and fatigue development was only significantly increased relative to normoxia at the lowest hypoxic condition. Central and peripheral responses were attenuated in the 12% condition whereas in the 13% condition responses were amplified by lower oxygen availability. Sprint speed was preserved in the 14% and 15% conditions. Total oxygen consumed during hypoxia was significantly lower, with significantly higher HR and minute ventilation. The authors suggest that lower blood lactate concentration and deoxyhemoglobin at 12% means that local metabolic factors are not the cause of the greater fatigue development in 12% compared to other conditions. The lower iEMG in hypoxia may be due to reduced central drive or impaired neuromuscular transmission because of decreased sarcolemmal excitability. Overall, physiological demands increase as inspired oxygen fraction decreased to 13% with similar peak speeds. Fatigue development only significantly worsened at 12% FIO2.
The purpose of this investigation was to determine the difference in oxygen usage in men and women during RSE. Ten men and ten women performed 10x10s cycling sprints with 30 seconds of rest under normoxia or hypoxia. Work, SpO2, O2Hb, HHb were monitored in all participants. All participants performed an initial 10s sprint in order to match them based on the mechanical work completed. After a familiarization visit, all participants were randomly assigned to a normoxic or hypoxic condition first. The second condition was completed 7 days later. All sprints had a resistive load of .9 N*Kg of body mass. In both conditions total work and percent decrement in work were similar for men and women. SpO2 was similar at rest in men and women and was affected by condition. Hypoxia caused lower values (men: –12.5 ± 7.3%; women: –14.7 ± 8.1%). Significant reductions in SpO2 were recorded across sprints 2-10 in normoxia, and significant reductions in sprints 1-10 in hypoxia. Men and women were equally affected by the hypoxic conditions. In normoxia, there was a 7x increase in deoxyhemoglobin following the first sprint in men, and a 4x increase in women. From sprints 1-10 change in HHb remained the same. Change in THb (Total blood volume) rose significantly. The authors suggest that this shows a reduction in the rate of muscle deoxygenation in subsequent sprints. In hypoxia, the pattern and magnitude of deoxygenation were similar to normoxia in both sexes. Change in muscle HHb differed between the sexes, however. The magnitude of sprint induced HHb was significantly less for women compared with men. Cerebral oxygenation was reduced in hypoxia in both men and women across every sprint. The results of this study show that both men and women reach maximal muscle deoxygenation during RSE. When work is matched men and women experience similar arterial desaturation. The results indicated that work matched men and women experienced similar systemic, cerebral, and muscular adjustments during sprinting. Cerebral deoxygenation imposes a
limit on RSA.

Smith & Billaut (60)

The aim of this investigation was to use NIRS to monitor central and peripheral oxygenation during RSA tests to better understand the factors associated with fatigue and improve performance in RSA. Thirteen participants (23.6 ± 3.7 yr) were randomized to either a normoxic or hypoxic RSA trial first and completed the other trial 7 days later. All participants completed the sprints against a resistance of 0.9 N*kg of body mass. SpO2, NIRS, and EMG were collected during the RSA tests. Condition had no effect on initial sprint scores. There was a significant decline in work in sprints 2-10 in both conditions, with hypoxia having a greater decrement than normoxia. Normoxia also had a higher total amount of work performed when compared to normoxia. SpO2 decreased significantly in sprints 1-10 and was affected by condition. Hypoxia had lower values than normoxia, with significant decrements in all sprints (1-10) than normoxia (sprints 2-10). Overall, changes in SpO2 were larger and occurred earlier in hypoxia. In normoxia, muscle oxygenation decreased rapidly during the first sprint (Shown by a decrease in oxyhemoglobin (O2Hb) and an increase in deoxyhemoglobin (HHb)), with a decrease in total blood volume (THb). From sprints 2-10 change in deoxyhemoglobin remained unchanged with a rise in oxyhemoglobin and total blood volume. In hypoxia muscle oxygenation followed a similar pattern (a reduction in the rate of muscle deoxygenation). The magnitude of effect on deoxygenation was the same in both trials. For cerebral oxygenation, oxygenation was increased rapidly in sprints 1 and 2 in normoxia. From sprints 3-10 NIRS signals fluctuated. Cerebral oxygenation in hypoxia was attenuated during sprint 1 without a significant change in blood volume. Changes in oxyhemoglobin and deoxyhemoglobin were larger in hypoxia compared to normoxia throughout the RSA test. There was no change in total hemoglobin across the sprints.
between normoxia and hypoxia. There was a significant decline in the sum iEMG signals for sprints 6-10 when compared to the initial sprint in both conditions. There was a significant interaction of sprint x condition, with lower values in sprints 6-10 in normoxia and 4-10 in hypoxia. Mechanical work reductions were correlated negatively with increased deoxyhemoglobin in the prefrontal lobe in both conditions, with a greater rate of change in hypoxia than normoxia. Greater cerebral deoxyhemoglobin changes also correlated negatively with changes in sum iEMG in both trials, with a greater rate of change in hypoxia. The results of the study show that changes in cerebral oxygenation are unlikely to limit RSA in normoxia, however they may affect performance in RSA in hypoxia. Tissue oxygenation did not vary significantly between normoxia and hypoxia.

Calbet JA et al. (15)

This investigation aimed to determine which factor limits muscle VO2 in hypoxia, arterial partial pressure, or oxygen content. Seven subjects (24.3 ± 0.5 yr) participated in the study. Catheters were inserted into the femoral vein, antecubital vein, and femoral artery in all participants. After 9-10 weeks at 5260 m elevation chronic hypoxia tests were conducted. Acute hypoxia tests at sea level were carried out at least 6 months after the subjects returned to Denmark. All subjects performed leg extensions of 30 W leg extensions increased by 10-15 W every 2 minutes until exhaustion. After 60 minutes of rest subjects performed cycle ergometer exercise starting at 30 W and increasing by 20-40 W every minute until exhaustion. The results of this study show that active muscle mass has a significant impact on tolerance to exercise in hypoxia. Under acute hypoxia, reducing the size of the active muscle reduced the effect of hypoxia on VO2peak by 62%. In chronic hypoxia the effect was removed. The authors concluded that mechanism responsible for the reduction in VO2peak in acute and chronic hypoxia is the reduction of oxygen delivery to active muscles. Even with low arterial saturation (55 mmHg) skeletal muscle is able to
achieve normoxic peak VO2 values assuming that oxygen delivery is sufficient.

**Vogt et al. (72)**

The purpose of this investigation was to determine molecular adaptations to exercise training of various intensities under normoxic versus hypoxic conditions. Thirty participants were randomly assigned to one of four groups. They trained for 30 minutes five times per week for 6 weeks. Two groups trained at a high intensity, with one of the two under normoxic conditions and the other under hypoxic conditions (Nor-High; Hyp-High). The other two groups trained at a low intensity under normoxic or hypoxic conditions (Nor-Low; Hyp-Low). VO2max, muscle biopsies, and molecular markers were taken for all participants. VO2max and Wmax were increased under all conditions. Mitochondrial density increased significantly under hypoxic conditions, and with high intensity under normoxia. Subsarcolemmal mitochondrial fraction increased significantly under hypoxia. HIF-1a mRNA increased under hypoxic conditions. MRNA coding for myoglobin and vascular endothelial growth factor increased under high intensity hypoxic conditions only. The results of this study show that high intensity hypoxic training induces the largest changes in molecular adaptations. These molecular adaptations suggest better oxygen transport and utilization in skeletal muscle.

**Morales-Alamo et al. (42)**

The purpose of this investigation was to determine if hypoxia increases AMPK phosphorylation in response to sprint exercise. Ten participants (25 ± 4 yr) were randomly assigned to perform 30 second sprints in normoxia or hypoxia first. All participants completed VO2 tests to determine VO2peak, max heart rate, and max power output in normoxia. Wingates were isokinetic at 100rpm. Pre and post exercise muscle biopsies were taken. Serum insulin levels were increased up to 62% following the Wingate test and decreased back to pre-exercise levels after 2
hours. AMP to ATP ratio was similarly increased after the exercise in both conditions. Glycolytic rate was 50% higher in hypoxia compared to normoxia. Muscle lactate concentration was 48% higher in hypoxia compared to normoxia. The reduction in NAD to NADH ratio was significantly higher in hypoxia. Hypoxia prevented the exercised induced AMPKa phosphorylation. The results of this study show that hypoxia lowers the AMPKa phosphorylation after exercise. Muscle lactate concentrations are increased under hypoxia, as shown by the greater reduction in NAD to NADH ratio. Sprinting exercise requires a glycolytic rate above the mitochondrial capacity of pyruvate oxidation. Power decreased by 6% in hypoxia, with a 37% reduction in VO2. This demonstrates a reduced ability to deliver oxygen. Glycolytic rate was greater in hypoxia compared to normoxia. Overall, this shows that skeletal muscle signaling response to sprint exercise is modified in hypoxia. AMPKa phosphorylation was blunted in hypoxia and the authors propose two mechanisms that may be responsible. The lower NAD to NADH ratio after sprints combined with SIRT1 protein levels may blunt the SIRT1/LKB1 mediated phosphorylation of AMPKa, and AMPKa phosphorylation may have been blunted due to increased Ser-AMPKa/SER-AMPKa2 phosphorylation.

Brocherie et al. (10)

The aim of this investigation was to compare the effects of repeated sprints in hypoxia compared with normoxia. Sixteen participants were randomly assigned to either repeated sprints in hypoxia (RSH) or repeated sprints in normoxia (RSN). All participants completed their normal football training at sea level excluding sprinting and explosive exercises, while exercise interventions and tests were done based on group condition (RSH or RSN). Training interventions consisted of 2-3 sets of 5-6x15 seconds running with 15 seconds of passive recovery. 5-10 minutes recovery between sets. RSE consisting of 4-6 sets of 3-4x5 seconds sprinting with 45 seconds of
rest between reps and 3 between sets. Countermovement jump height, 40-meter sprint distance, and RSA were measured in all participants. Average arterial oxygen saturation values decreased in RSH compared to RSN over the course of the study (5 weeks). Countermovement jump height increased similarly in both groups. Sprinting performance in both groups improved significantly after the intervention when compared to the baseline measures, with no significant difference between groups. RSH was likely to most likely beneficial while RSN was possibly beneficial (RSH had greater magnitude for sprint distance). RSApeak performance was improved in both groups, and RSA average scores were also improved in both groups. RSH seemed possibly beneficial compared to RSN. Sprint decrement score remained unchanged between pre and post scores in both groups. Agility performance parameters were improved in both groups with a very likely advantage for RSH compared to RSN. The results of the study show that repeated sprints in hypoxia and normoxia both improve performance in young football players. However, sprints in hypoxia are more efficient at improving performance than sprints in normoxia.

Woorons et al. (77)

The purpose of this investigation was to examine the effects of repeated sprint exercise in hypoventilation induced hypoxia on physiological adaptations, performance, and RSA. Eighteen (34.6 ± 11 yr) cyclists completed six RSE sessions over three weeks. Participants were matched for performance and then randomly assigned to either the normoxic group (RSN) or the hypoxic induced group (RSH-VHL). RSA was tested with 10x6s sprints with 30s of passive recovery between sprint repetitions. Repetitions of sprints were progressively increased over the course of the training period to reach 3x8 sprints at the last session. Sets were separated by 3 minutes of recovery. In the VHL group subjects were told to start each repetition exhaling normally and then holding their breath until the end of the six seconds. RSA performance, gas exchange, HR, SpO2,
NIRS, and RPE were measured during the trials. The total training load was not different between groups, with no difference in the mean number of 6s sprints completed per subject between groups. Mean power output was not different between groups before the RSA tests but was higher after the tests in the VHL group (unchanged in RSN). Average MPO and percent decrement score were not different between groups. MPO and percent decrement score were higher at post than at pre in VHL, while they were unchanged in RSN. VO2 was not different between groups at the start of the intervention, but at the post-test was higher in VHL. Average tidal volume was not different between groups. VO2 was significantly higher at post- than at pre- in VHL from the 4th sprint on, while it was unchanged in RSN. Average SpO2 and HR were not different in RSN and VHL. Total deoxyhemoglobin was higher at Post than at pre in both groups, with no change for the average values of total hemoglobin. There were no differences between groups in RPE or blood lactate concentration. The results of this study show that six sessions of RSH-VHL improved RSA with an increase in VO2 without any change in tissue oxygenation. Under normoxia these conditions were not improved. Anaerobic performance over a Wingate was also improved under RSH-VHL compared to RSN.

Soo et al. (63)

The purpose of this investigation was to determine the effects of various levels of hypoxia on RSA. Nine participants performed 3 trials in a randomized order. Participants performed 10 × 4s sprints with 30 seconds of recovery between them (15 seconds passive, 15 seconds active). The trials were conducted in normoxia (SL), FiO2 17% (MH), and FiO2 13% (SH). Following the sprints participants rested passively for 8 minutes before performed 5 × 4s sprints with 30 seconds of recovery in normoxia. Neuromuscular measurements were taken by having participants perform a 4s MVIC with a superimposed 80Hz doublet followed after 3 seconds by another 80 Hz doublet,
a 20 Hz doublet, and three single twitches on the relaxed state separated by 3 seconds. MPO decreased to a larger extent in SH compared to SL and MH. Average sprint performance for the first five sprints at set two (after the 8-minute passive rest) was not different from average sprint performance of the first five in set one. RMS activity was significantly reduced at sprint five and again at 10 in all conditions. After rest, RMS activity significantly recovered for sprint 11 when compared to 10. HR was significantly higher at sprint 5 and 15 compared to 1. HR was significantly lower at sprint 11 compared to 5, 10, and 15. SpO2 was significantly reduced with increasing severity of hypoxia. Blood lactate values were similar between conditions. RPE was significantly higher in SH compared to SL after 5 sprints. SH caused a larger performance decrement during the first set of sprints, with increased sensations of discomfort, difficulty breathing, and limb discomfort. Muscle contractility at the end of the first set was similar between conditions. Voluntary activation reductions were not meaningful. Following the 8 minutes of rest, single sprint performance and sensations of discomfort, difficulty breathing, and limb discomfort were recovered in all conditions. RSA was decreased in set two after the SH condition. The results of this study show that recovery of sensations of fatigue and discomfort may play a larger role than neuromuscular function integrity in subsequent RSA.

**Traditional Training (MICT)**

Kriel et al. (34)

This purpose of this investigation was to determine the acute effects of SIT when compared to CMIE on local oxygen utilization, post exercise blood pressure, and enjoyment in inactive young men. 11 inactive men, (mean ± SD; age 23 ± 4 yr) completed a VO$_{2\text{max}}$ test followed by two conditions, SIT (.075 kg/kgbw 4x30s with 2 minutes recovery) and a work matched continuous
moderate intensity exercise (CMIE, 50% of peak power output during $\text{VO}_{2\text{max}}$ and then work matched to the SIT) on a cycle ergometer. Changes in O2Hb and HHb were measured using NIRS. As O2Hb data are affected by muscular compression and perfusion changes, only HHb data is shown. VO$_2$ and HR were collected continuously, with SBP and DBP responses measured at rest immediately post exercise and every 2 minutes during the 6-minute recovery period. VO$_2$ and HR were higher during SIT compared to CMIE. Differences in SBP over time were found in SIT and CMIE, but not between conditions. For DBP there was a main effect for condition and time, and a significant condition x time interaction. Physical activity enjoyment was greater for CMIE compared to SIT. The results of this study show that there was a higher oxygen utilization for SIT, a lower PACES score, and a lower post exercise DBP at recording 2, 4, and 6, indicating a higher level of physiological stress. A higher VO$_2$ and HR do not necessarily show an increase in local oxygen utilization at the site of measurement. In large locomotor muscles, oxygen utilization is similar to that achieved during a work matched session of CMIE.

Fiorenza et al. (21)

The aim of this investigation was to examine the impact of metabolic stress on regulation of molecular responses promoting skeletal muscle mitochondrial biogenesis. 12 trained male cyclists completing 3 experimental trials of intermittent exercise (RS), speed endurance (SE), or continuous moderate intensity (CM) cycling. RS consisted of 18x5s all out sprints with 30s of passive recovery, SE involved 6x20s all out sprints with 2min of passive recovery. The SIT protocols were matched for volume and work to rest ratio. The CM protocol was 50m of cycling at 70% VO2peak. Muscle lactate accumulation was larger in SE than in RS and CM. Muscle pH decreased in RS and SE and decreased more in SE than RS. Muscle glycogen was lowered in all trials, with post exercise glycogen lower in CM than in RS and SE. Plasma adrenaline was
increased by exercise in RS and SE, being ~2.5x and ~3.5x higher in RS and SE than in CM, respectively. The change in plasma adrenaline was greater in RS and SE than in CM. Phosphorylated AMPKα increased immediately after exercise in all trials (RS, P = 0.006; SE, P = 0.001; CM, P < 0.001), with no difference between trials. Muscle PGC-1α mRNA content was elevated 3 h after exercise relative to Pre in all trials, with SE and CM inducing a greater response than RS. Mean power output of 902 ± 33 and 669 ± 26 W (mean ± SEM) during RS and SE, respectively. Multiple linear regression analysis showed that change in muscle PCr, [H+] and glycogen, and plasma adrenaline, predicted the PGC-1α mRNA response to RS and SE. The results of this study show that the PGC-1α mRNA response to low volume intense intermittent exercise is greater with higher muscle lactate accumulation, greater muscle pH drop, and greater plasma adrenaline levels.

Litleskare et al. (36)

This investigation aimed to compare the specific adaptations of moderate intensity continuous training (MICT) and SIT performed as running. 25 participants (25 ± 1 yr) were randomly assigned to perform either MICT or SIT three times per week for eight weeks. The MICT group exercised starting at 30-minutes and increasing to 60 at 70-70% HRpeak and the SIT group for 5-30s sprints, building up to 10, with 3 minutes of recovery between sets. VO2max, HR, RER, RSA, lactate and running economy were assessed before and after the intervention. There were no significant differences between groups prior to the intervention. VO2max was measured three times prior to the intervention and increased from test to test. VO2max was improved in both CT (47.9 ± 1.5 --> 49.7 ± 1.5) and SIT (50.5 ± 1.6 --> 53.5 ± 1.5) after training, by 3.8% and 5.5% respectively. 20m shuttle run performance increased in both groups, as did maximal O2pulse. Both groups had improved running economy after the intervention, with CT decreasing VO2 at all
stages, and SIT decreasing at stage 4, and RE at 2 and 4. HR was lower after CT but was unchanged after SIT. HR remained unchanged at a velocity close to 70% VO2max in CT, while O2 pulse increased. In SIT, however, HR increased and O2 pulse remained unchanged. RER was also reduced in the CT group for that intensity, but not changed after SIT. Lactate was reduced in both groups. The results show that VO2max was increased in both groups, sprint performance is improved in both groups, but SIT begins to perform better with more sprints. Only CT improved HR and o2pulse at submaximal intensities. The authors suggest that the improvements in VO2max are due to peripheral adaptations. Even though HR was unchanged in submaximal exercise for SIT, running economy improved. Further testing at a specific exercise intensity (70%) showed that cardiac output after SIT is unchanged. Increased cardiac output leads to higher O2pulse and decreased HR at submaximal intensities. Overall, although CT improved cardiac output and SIT did not, both groups improved VO2max, endurance capacity and sprint performance. SIT improved RSA significantly more compared to CT. The training specific adaptations of SIT appear to improve skeletal muscle buffer capacity, and oxidative potential of the muscle, with similar VO2max improvements with less total work than CT.

Saanijoki et al. (55)

The purpose of this investigation was to determine responses to SIT in comparison to MICT in insulin-resistant subjects. Twenty-six participants (49 ± 4 yr) were randomized into SIT or MICT groups. All participants were sedentary with impaired glucose tolerance (HbA1c < 7.5 mmol*L). Participants completed six training sessions in a two-week period. The SIT sessions were comprised of 4-6x30s sprints with 4 minutes of recovery. Resistive load was set at 10% of fat free mass in kilograms. MICT sessions were comprised of 40-60min at 60% peak workload. RPE, perceived stress questionnaire (PSQ), positive and negative affect schedule (PANAS), visual
analogue scale (VAS) and VO2 were measured. Body mass, BMI and fat free mass (FFM) were unchanged after the intervention. Fat percent reduced and peak load was improved in both groups. VO2peak response was different between SIT and MICT, with only SIT improving VO2peak. Lactate was higher after SIT compared to MICT. RPE decreased more over time in SIT compared to MICT. SIT sessions increased perceived stress (PSQ), although these scores decreased post exercise. This increased PSQ score was mitigated after the third SIT session. PANAS positive score was initially decreased by SIT, but by the end of the intervention scores were increased compared to SIT, the opposite effect of MICT. Pain and pain alleviation were both higher in the SIT group compared to MICT. MICT increased motivation to exercise more than SIT. Pain ratings in SIT decreased significantly. The results of this study show that although SIT has significantly increased stress, pain, and RPE ratings compared to MICT, after several sessions these ratings are not significantly different than for MICT. The authors conclude that very strenuous SIT appears to be tolerable for insulin-resistant adults.

MacInnis et al. (38)

The purpose of this investigation was to determine the role of exercise intensity in promoting training-induced increases in skeletal muscle mitochondrial content. Ten participants (23 ± 1 yr) performed unilateral GXT to measure single leg VO2peak and peak power. Each leg was randomly assigned to complete six sessions of HIIT or MICT on one leg, with the other leg exercising 10 minutes later. HIIT and MICT were work matched and duration matched. The HIIT exercise consisted of 4x5min cycling at 65% average peak watts, with 2.5 min active recovery between bouts. MICT consisted of 30 minutes of cycling at 50% peak watts to match the HIIT group work. All sessions were conducted at a cadence of ~80RPM. Heart rate, muscle biopsies, RPE and blood lactate were all measured. Citrate synthase and mitochondrial respiration were
measured from the muscle biopsies. HR and RPE were higher during HIIT sessions compared to MICT, as was blood lactate levels (8.0 ± 0.8 vs. 5.5 ± 0.7 mm; P < 0.001). After the intervention single leg peak watts increased from 150 ± 9.3 to 163 ± 10.0 W in the HIIT group, and 155 ± 8.4 to 163 ± 8.3 W in the MICT group. There were no differences between groups. Training did not increase single leg VO2peak in either group. Citrate synthase activity increased for HIIT more than MICT (39% vs 11% P=0.02). Interval work compared to the continuous work caused greater increases in mitochondrial content of skeletal muscle. The results of this study show that higher intensity training, even if work matched, causes greater increases in skeletal muscle mitochondrial capacity. Mitochondrial function was unchanged, however.

Gibala et al. (26)

The purpose of this investigation was to compare the changes in exercise capacity as well as molecular and cellular adaptations in skeletal muscle after low volume SIT and high-volume ET. Sixteen males were randomly assigned to either SIT (22 ± 1 yr) or ET (21 ± 1 yr). Both protocols consisted of six sessions across 14 days. For the SIT protocol participants completed 4-6x30s sprints with 4 minutes of recovery between them. The ET protocol consisted of 90-120 minutes of continuous cycling at 65% of VO2peak. Both groups completed 50kj and 750kj timed trials before and after the 2-week intervention. Performance on the 750kj trial decreased by 10.1% in SIT and 7.5% in ET, with no significant difference between groups. MPO increased in both groups, from 212 ± 17 to 234 ± 16 W in the SIT, and from 199 ± 13 to 212 ± 12 W in the ET group (main effect for time, P < 0.001). The time to complete the 50kj test also decreased by 4.1% in the SIT group and 3.5% in ET with a main effect for time (P = 0.02). MPO also increased in the 50kJ time trial from 435 ± 23 to 453 ± 25 W in the SIT group and 416 ± 39 to 433 ± 40 W in the ET group (main effect for time, P = 0.02). COX max activity increased after training, however there
was no difference between groups. There were training induced increases in COX II and COX IV protein contents with no difference between groups. Muscle buffering capacity increased after training by 7.6% for SIT and 4.2% for ET, with no significant difference between groups. Muscle glycogen content increased after training by 28% and 17% for SIT and ET respectively, with no significant difference between groups. Although the difference in total training volume was ~90% lower in SIT compared to ET, both groups experienced similar increases in muscle buffer capacity, glycogen content, time to completion in 50kJ and 750kJ trials, and muscle oxidative capacity as shown by COX and COX subunits. The results of the study show that intense interval training is a time efficient way to cause rapid muscle and performance adaptations comparable to traditional endurance training.

Hoier et al. (30)

The purpose of this investigation was to determine if intense intermittent exercise is a sufficient stimulus to cause capillary growth in skeletal muscle conditioned by MICT. Nine participants (31.7 yr) participated in the study. All participants completed a 4-week conditioning period of 60 minutes cycling at ~65% VO2max 3x per week. Following this conditioning phase participants had two data collection and measurement days and a high intensity intermittent exercise protocol. Order of experimental days were randomized for all participants. The HIIE protocol consisted of 24x1 minute bouts of intense cycling with 1.5 minutes of recovery in between bouts. This training was performed 3x week for 4 weeks as well. The training load was ~117% of the pre-conditioning VO2peak, increasing to ~124% after two weeks. Muscle biopsies were taken microdialysis probes were inserted before subjects performed 10 minutes of exercise at 10 W. Subjects also would complete another test with 60 minutes of continuous cycling at ~62% VO2peak and one of the 24 bouts of cycling with 1.5 minutes of rest. The initial conditioning
phase increased the capillary to fiber (C: F) ratio from 2.38 ± 0.16 to 3.09 ± 0.24 and capillary density (CD) from 497 ± 23 to 579 ± 35 caps mm−2. Citrate synthase levels also increased by 45% and VO2max increased from 3.30 ± 0.21 to 3.59 ± 0.21 l min−1. Following the HIIE protocol C:F ratio was not different, and CD was also unchanged. mRNA expression of several angiogenic factors increased after the MICT protocol, however the stimulus during 4 weeks of HIIE was not sufficient to induce capillary growth. The HIIE exercise induced only ~60% of the interstitial VEGF compared with MICT. The results of this study show that in skeletal muscle conditioned by MICT, HIIE is not enough of a stimulus to cause capillary growth due to the angiogenic response. It did lead to increases in mRNA expressions of proteins involved in angiogenesis, and lowered response in interstitial VEGF levels.

Burgomaster et al. (13)

The aim of this investigation was to compare changes in muscle oxidative capacity after 6 weeks of SIT vs ET. Twenty participants (5 men and 5 women per group, 24 ± 1 SIT and 23 ± 1 yrET) volunteered for the study. Participants were randomly assigned to either the SIT group of the ET group. All participants completed pre-tests before the 6-week intervention and post-tests after. Muscle biopsies, HR, RER and VO2peak measurements were collected on those testing days. The ET protocol consisted of continuous cycling on an ergometer 5x per week for 6 weeks at 65% VO2peak. Exercise time was increased from 40-60 minutes over the course of the 6 weeks. VO2peak tests were re-taken after 3 weeks, and training loads were adjusted accordingly. The SIT protocol consisted of 3 sessions per week of repeated sprints (performed in the manner of a Wingate). The number of Wingates completed increased over the duration of the study from 4-6 with a constant active recovery period of 4.5 minutes between sprints. VO2peak increased after training with no difference between groups. PP during the Wingate test increased by 17% and 7%
in the SIT and ET groups respectively, with no significant differences between groups. Average RER was reduced after training and rates of fat oxidation were increased while carbohydrate oxidation was decreased after training in both groups. Muscle glycogen was increased, muscle glycogenolysis was decreased, and muscle PCr content was increased in both groups after training. There were no significant differences between groups. Muscle ATP was reduced after 6 weeks of SIT compared to ET. Adaptations in select markers of muscle CHO and fat metabolism and metabolic control were similar between SIT and ET after 6 weeks of training. Training volume was ~90% lower in SIT compared to ET. The results of this study show that low-volume SIT induces similar changes in markers of whole-body and skeletal muscle metabolism during exercise comparable to changes caused by high-volume ET.

Kavaliauskas et al. (32)

This investigation aimed to compare physiological and performance adaptations following either 2 weeks of SIT or 2 weeks of uphill running (UST). Seventeen participants (28 ± 5 yr) were randomly assigned to either a control (CON), SIT, or UST group. The control group completed both running and cycling VO2peak tests in a randomized fashion. The SIT group completed only cycling VO2peak tests while the UST group completed only running VO2peak tests. The SIT protocol consisted of 3 sessions per week for 2 weeks. Each session consisted of 4x30s cycling sprints with 4 minutes of active recovery between sets. The UST protocol consisted of 3 sessions per week for 2 weeks, with each session consisting of 4x30s sprints on a 10% slope. During the 4-minute recovery participants walked back to the bottom of the hill. A separate group of participants (not included in the n=17) completed 2 sessions of SIT and 2 sessions of UST in a randomized order. VO2peak did not significantly change in either group after training compared to the pre-training values. There was a small effect size with greater change in UST compared to SIT. There
were no changes in time to exhaustion in the control group. TTE increased by ~3% in the SIT group and ~11% in the UST group with a significantly different change between groups. There was a small effect size between SIT and UST, and a large effect size between CON and UST, with greater changes in UST. Ventilatory threshold was significantly increased after training in both SIT and UST, while it remained unchanged in CON. There was no difference between the magnitude of change SIT and UST. MPO was similar between SIT and UST. The drop in power from sprints 1-4 was significantly different after UST but not SIT (UST session 1: 26 ± 4%, session 6: 14 ± 4%, p = 0.001, SIT session 1: 23 ± 11%, session 6: 17 ± 5%, p = 0.18). There was a large effect size for power drop between groups with a greater improvement in UST. Blood lactate was significantly higher after sprints when compared to the baseline in both sprints. Blood lactate was significantly higher after the first sprint in SIT compared to UST. However, there was no significant difference in any other sprint. The results of this study show that similar adaptations occur following SIT compared to UST. UST had larger effect sizes for power drop, TTE, and VO2peak.

Sandvei et al. (56)

The purpose of this investigation was to compare the effects of eight weeks of SIT to eight weeks of MICT on insulin sensitivity and cholesterol profile. Twenty-three participants were sex and VO2max matched and randomized into two groups, SIT or MICT. Both groups performed 3 sessions per week for 8 weeks. The MICT sessions consisted of 30-60 minutes of 70-80% max HR running, increasing 5 minutes per week. The SIT sessions consisted of 5-10x30s near maximal sprints on a 5-8% incline with 3 minutes of recovery between sets. A 6-minute rest period was incorporated at the halfway point of the SIT sessions. All participants completed a test protocol consisting of a treadmill VO2max test, body composition tests, and a glucose tolerance test.
VO2max significantly increased by 5.3 ± 1.8% (p < 0.05) and by 3.8 ± 1.6% (p < 0.05) in SIT and MICT respectively after training with no difference between groups. Glucose concentration was lower at 2 hours after 8 weeks of SIT compared to pre-training tests. Glucose response was unchanged in MICT after 8 weeks of training. Eight weeks of SIT training improved insulin sensitivity while 8 weeks of MICT did not. The results of this study show that SIT training is more effective with less total work at improving insulin sensitivity and cholesterol profile after 8 weeks compared to MICT training of the same duration.
III: METHODS

Subjects

Fifteen men volunteered to participate in this investigation and were randomly assigned to complete sprint interval training (SIT) with continuous blood flow restriction (BFR), intermittent BFR, and no-BFR. Participants had no known cardiovascular, pulmonary, metabolic, muscular, and/or coronary heart disease, or regularly used prescription medications. All participants were aerobically active for at least 6 months at the time of testing (1). Participants visited the laboratory on 5 separate occasions: 1 familiarization + baseline visit, 3 randomly ordered testing visits (continuous BFR, intermittent BFR, and no-BFR), and a follow-up VO$_{2\text{max}}$ testing visit after the completion of the 3 SIT protocols.

Experimental Design

A randomized, repeated measures, within-group design was used for this study. All subjects completed a SIT protocol that consisted of 2, 30 second sprints with 2 minutes of passive recovery between sprints. To achieve continuous BFR, the cuff was applied immediately prior to the start of sprint 1 and removed immediately after the 2nd sprint. For intermittent BFR, the cuff was applied immediately following the first sprint and removed immediately prior to the next sprint (i.e., applied only during the rest break). All testing procedures were performed using a cycle ergometer and performed at the same time of day (± 2 h). During each testing session, muscle oxygenation, sprint decrement score (Sdec), peak power output (PP), total work performed (TW), distance, RPE, and muscle mitochondrial function were determined.
Procedures

Baseline Visit

Following a 5-minute warmup on a stationary cycle ergometer (Corval 400, Groningen, The Netherlands), participants completed 3, 3-second maximal voluntary isometric contraction (MVIC) muscle actions on the isokinetic dynamometer at a joint angle of 90 degrees (180 degrees corresponds to full extension at the knee). Participants were instructed to push as “hard and fast” as possible against the lever arm once the verbal cue of “Go” is given. Each MVIC was be separated by 30 seconds of rest and the highest torque produced of the three attempts was used for further analyses.

After the MVIC tests, participants performed an incremental test to volitional exhaustion on a cycle ergometer (Corval 400, Groningen, The Netherlands) to determine peak oxygen consumption (VO$_{2\text{max}}$). Each participant was fitted with a heart rate monitor (Polar H10, Polar Electro Oy, Kempele, Finland) and a silicone facemask (7450 V2, Hans Rudolph, Inc., Shawnee, KS) with a two-way non-rebreathing valve which was connected to a metabolic measurement system (TrueMax 2400, ParvoMedics, Sandy, UT). Prior to each use, the metabolic gas analyzer was calibrated with gases of known concentration (16% O$_2$, 5% CO$_2$, and N$_2$ bal) and calibrated for airflow with a 3 L syringe as per the manufacturer’s recommendations. Mask size was determined using the manufacturer provided sizing gauge and was secured using the manufacturer provided five-strap headgear.

The graded exercise test began at an initial work rate of 90 W and increased by 30W every 3 minutes. The test concluded when the participant could no longer maintain a pedaling cadence of 70 ± 5 rpm for a duration of 5 seconds despite strong verbal encouragement. The highest 30-s average breath-by-breath oxygen consumption rate was recorded as VO$_{2\text{max}}$. A test
was classified as valid if the maximal volume of oxygen consumption ($\dot{V}O_{2\text{max}}$) met two of four criteria: respiratory exchange ratio $\geq 1.0$, heart rate within 10 beats per minute of age-predicted max, maximal rating of perceived exertion, and/or plateau in oxygen consumption.

**Sprint Interval Protocol**

On testing visits 1-3, subjects performed a standardized warm-up on a cycle ergometer at a self-selected pace and resistance for 5 minutes. Following the warmup, participants received a rest period of approximately 5 minutes during the placement of assessment devices. Participants then completed 2, 30 second all-out sprints ($2 \times 30$s) at a resistance that corresponded to 7.5% body mass on a mechanically braked cycle ergometer (894 E, Monark, Vansbro, Sweden). Both sprints were initiated from a rolling start with participants instructed to progressively increase to their maximal cadence 3 seconds prior to the start of each sprint. Each sprint was separated by 2 minutes of passive recovery. Distance, peak power output (PP), and total work (TW) was recorded using the Monark software (Monark, Vansbro, Sweden) for each sprint. $S_{\text{dec}}$ was determined as total work from both sprints relative to the first sprint, which had the best performance, and expressed as a percentage (8,28,64). TW was determined as the product of force (in newtons) and distance covered in joules.

**Blood Flow Restriction**

To achieve continuous and intermittent BFR, a 12-cm wide rapid cuff inflator (Hokanson Rapid Cuff Inflator; Hokanson Inc., Belleview, WA, USA) was applied at 60% of the lowest amount of pressure needed to completely occlude the posterior tibial artery blood flow as indicated by a portable color mode ultrasound-imaging device (GE Logiqe, USA) and a multi-frequency linear-array probe (12L-Rs; 5-13MHz; 38.4 mm field-of-view). The pressure was initially applied at 30mmHg and progressively inflated over a 60 second period until target
pressure was achieved. For the continuous BFR condition, the cuff was inflated immediately prior to the first sprint and remained inflated until the completion of the 2nd sprint. For the intermittent BFR condition, BFR was applied immediately following the 1st 30-second sprint during the 2-minute rest period and was rapidly deflated immediately prior to the start of the 2nd sprint.

**Near-Infrared Spectroscopy**

Muscle oxygenation was assessed using near-infrared spectroscopy (NIRS) (PortaLite, Artinis Medical Systems, Arnhem, The Netherlands). The NIRS device was placed on the vastus lateralis muscle of the dominant leg at 70% of the distance from the anterior superior iliac spine to the lateral aspect of the patella. The NIRS device was secured to the leg using double-sided tape and covered with colored athletic stretch tape to prevent contamination from ambient light. NIRS data acquisition included a standard differential pathlength factor of 4 sampled at 50 Hz and filtered (4th order, zero lag Butterworth filter) using LabVIEW (National Instruments, Austin, TX) software (50,75). Skin and adipose tissue thickness were measured at the site of application of the NIRS device prior to the testing sessions using a portable brightness mode (B-mode) ultrasound imaging device (GE Logiq E, USA) and a multi-frequency linear array probe (12LRs; 5-13MHz; 38.4mm field-of-view). Skin and subcutaneous tissue thickness were the closest value less than half the distance between the source and the detector.

Changes in muscle tissue oxygenation, oxyhemoglobin (O$_2$Hb), deoxyhemoglobin (HHb), and total hemoglobin (THb) were reported as change from the baseline measure (collected during the first two minutes of NIRS measurements in a supine position) in micromolar units (µM) (12) and examined across time using optical densities from two continuous wavelengths (760 and 850 nm). Tissue saturation index (TSI, expressed in % and
calculated as \( \frac{[O2Hb]}{[O2Hb] + [HHb]} \times 100 \) was used to assess muscle reoxygenation levels. The value for TSI was determined from the last 5 seconds of each sprint.

**Muscle Mitochondrial Function**

Muscle oxygen consumption (m\(\text{VO}_2\)) was measured as described in previous investigations (52–54). Briefly, baseline muscle oxygenation was determined on each visit prior to the warmup over a 2-minute period. Resting m\(\text{VO}_2\) was determined by rapidly inflating the blood pressure cuff to at least 250 mmHg for a 30-second period to achieve total arterial occlusion. The NIRS rate constant \(k\) was determined as the constant m\(\text{VO}_2\) recovery rate following a 15-second submaximal (30% MVIC) isometric leg extension muscle action. To measure m\(\text{VO}_2\) recovery, a series of brief (5-10s) total arterial occlusions (full cuff inflation) were applied beginning at 5s and increasing to 10s (occlusions 1-5: 5s on/5s off; occlusions 6-10: 5s on/10s off; occlusions 11-15 10s on/20s off). The slope of m\(\text{VO}_2\) for each occlusion was then fit to a monoexponential curve for final determination of \(k\). Tissue oxygen saturation was maintained above 30% to avoid low oxygen tensions. The NIRS time constant \(t\) was the inverse of the rate constant \(k\).

To normalize the NIRS signal, an ischemia/hyperemia calibration was applied following the series of total arterial occlusions. A 5-second 30% MVIC was performed on the isokinetic dynamometer and immediately followed by a 3–6-minute total arterial occlusion at 250mmHg of pressure. The end value before the total arterial occlusion was released represented 0% oxygenation and the peak hyperemic response after the cuff was deflated represented 100% oxygenation.

**Statistical Analyses**

Separate 3 (Condition [Continuous BFR, Intermittent BFR, No-BFR]) x 2 (Time [Sprint
1, 2]) repeated measures ANOVAs were used to analyze PP, TW, RPE, $S_{dec}$, TSI, $O_2$Hb, HHb, and THb. The NIRS rate constant $k$ and the time constant $t$ were derived from $mVO_2$ measured prior to (pretest) and immediately after (posttest) the sprint protocol and analyzed using separate 3 (Condition [Continuous BFR, Intermittent BFR, No-BFR]) x 2 (Time [Pre, Post]) repeated measures ANOVAs. Greenhouse–Geisser corrections were used if sphericity was not met according to Mauchly’s test of sphericity. Significant interactions were decomposed, when appropriate, using Bonferonni-corrected repeated measures ANOVAs. Partial eta squared effect sizes ($\eta^2_p$) were calculated for each ANOVA. All statistical analyses were performed using IBM SPSS v. 27 (Armonk, NY) and an alpha of $p \leq 0.05$ was considered statistically significant for all comparisons.
IV: RESULTS

Peak Power

There was a significant ($p = 0.013, \eta_p^2 = 0.304$) Condition × Time interaction that was decomposed into follow-up paired samples $t$-tests and repeated measures ANOVAs. Follow-up analyses indicated that peak power decreased from Sprint 1 to Sprint 2 for CBFR ($866.0 \pm 151.8$ to $641.5 \pm 141.4$ W), IBFR ($875.3 \pm 169.4$ to $669.1 \pm 143.5$ W), and NoBFR ($887.2 \pm 168.7$ to $761.7 \pm 128.8$ W). Furthermore, during Sprint 2 peak power was lower for CBFR ($p = 0.003$) and IBFR ($p = 0.001$) compared to NoBFR, but there were no differences during Sprint 1 (Fig. 1AB).

Total Work

There was no significant ($p = 0.306, \eta_p^2 = 0.091$) Condition × Time interaction but there were significant main effects for Condition ($p < 0.001, \eta_p^2 = 0.566$) and Time ($p < 0.001, \eta_p^2 = 0.855$) for total work. Specifically, collapsed across Time, total work was lower for CBFR (14,320.7 ± 769.1 J) than IBFR (15,548.0 ± 840.5 J) and NoBFR (15,915.4 ± 771.5 J). Additionally, collapsed across Condition, total work decreased from Sprint 1 (17,835.6 ± 966.2 J) to Sprint 2 (12,687.2 ± 675.2 J) ($p < 0.001$) (Fig. 2AB).

Ratings of Perceived Exertion

There was no significant ($p = 0.566, \eta_p^2 = 0.046$) Condition × Time interaction or main effect for Condition ($p = 0.844, \eta_p^2 = 0.014$). There was, however, a significant main effect for Time ($p < 0.001, \eta_p^2 = 0.709$). Specifically, collapsed across Condition, RPE increased from Sprint 1 (8.5 ± 0.3) to Sprint 2 (9.7 ± 0.1) ($p < 0.001$) (Fig. 3AB).
Sprint Decrement

There was a significant \((p = 0.043, \eta_p^2 = 0.230)\) one-way repeated measures ANOVA across Condition, but, there were no significant follow-up Bonferroni-corrected paired samples \(t\)-tests. Thus, sprint decrement was similar for CBFR (84.3 ± 1.7 %), IBFR (86.1 ± 1.5%) and NoBFR (87.2 ± 1.1%) (Fig. 4AB).

\[\text{VO}_{2\max}\]

\(\text{VO}_{2\max}\) was assessed prior to and re-evaluated after the completion of all 3 conditions (CBFR, IBFR, and NoBFR). The paired samples \(t\)-test indicated that \(\text{VO}_{2\max}\) increased from 37.6 ± 6.0 ml•kg•min to 39.6 ± 6.1 ml•kg•min \((p < 0.001)\) following the 3 SIT protocols.

Hemoglobin Changes

\[\Delta\text{HHb from Baseline}\]

There was a significant \((p < 0.001, \eta_p^2 = 0.504)\) Condition × Time interaction that was decomposed into follow-up paired samples \(t\)-tests and repeated measures ANOVAs. Follow-up analyses indicated that \(\Delta\text{HHb}\) increased from Sprint 1 to Sprint 2 for CBFR (14.2 ± 9.4 to 19.8 ± 7.5), IBFR (10.2 ± 8.2 to 13.4 ± 8.6), and NoBFR (10.0 ± 9.6 to 11.6 ± 8.8). Furthermore, during Sprint 2, \(\Delta\text{HHb}\) was greater for CBFR \((p = 0.020, p = 0.002)\) than IBFR and NoBFR, but there were no differences during Sprint 1 (Fig. 5AB).

\[\Delta\text{O}_2\text{Hb from Baseline}\]

There was no significant \((p = 0.653, \eta_p^2 = 0.038)\) Condition × Time interaction or main effect for Condition \((p = 0.319, \eta_p^2 = 0.099)\). There was, however, a significant main effect for
Time ($p = 0.004$, $\eta_p^2 = 0.539$). Specifically, collapsed across Condition, ΔO2Hb increased (smaller $\Delta$) from Sprint 1 (-9.9 ± 1.7) to Sprint 2 (-7.6 ± 1.6) (Fig. 6AB).

ΔTHb from Baseline

There was no significant ($p = 0.696$, $\eta_p^2 = 0.032$) Condition × Time interaction. There were, however, significant main effects for Condition ($p = 0.005$, $\eta_p^2 = 0.386$) and Time ($p = 0.002$, $\eta_p^2 = 0.604$). Specifically, collapsed across Time, ΔTHb was greater for CBFR (8.4 ± 2.0) than IBFR (2.4 ± 1.5) and NoBFR (0.7 ± 1.9). Additionally, collapsed across Condition, ΔTHb increased from Sprint 1 (1.6 ± 1.4) to Sprint 2 (6.1 ± 1.4) (Fig. 7AB).

TSI

There was no significant ($p = 0.096$, $\eta_p^2 = 0.206$) Condition × Time interaction or main effect for Condition ($p = 0.981$, $\eta_p^2 = 0.002$). There was, however, a significant main effect for Time ($p = 0.007$, $\eta_p^2 = 0.470$). Specifically, collapsed across Condition, TSI increased from Sprint 1 (52.9 ± 2.3) to Sprint 2 (55.3 ± 1.9) (Fig. 8AB).

Mitochondrial Functioning

NIRS Rate Constant, K

There was no significant ($p = 0.822$, $\eta_p^2 = 0.018$) Condition × Set × Time interaction, Set × Time interaction ($p = 0.298$, $\eta_p^2 = 0.098$), Condition × Time interaction ($p = 0.933$, $\eta_p^2 = 0.002$), or Condition × Set interaction ($p = 0.909$, $\eta_p^2 = 0.009$). There was a significant main effect for Time ($p = 0.012$, $\eta_p^2 = 0.452$) but not Condition ($p = 0.239$, $\eta_p^2 = 0.301$) or Set ($p = 0.341$, $\eta_p^2 = 0.101$). Specifically, collapsed across Condition and Set, $k$ increased from prior to
the sprints (3.5 ± 0.36) to after the completion of the two sprints (5.3 ± 0.8) \((p = 0.012)\) (Fig. 9AB).

**Time Constant, \(t\)**

There was no significant \((p = 0.626, \eta_p^2 = 0.042)\) Condition \(\times\) Set \(\times\) Time interaction, Set \(\times\) Time interaction \((p = 0.443, \eta_p^2 = 0.054)\), Condition \(\times\) Time interaction \((p = 0.815, \eta_p^2 = 0.018)\), or Condition \(\times\) Set interaction \((p = 0.153, \eta_p^2 = 0.157)\). There was a significant main effect for Set \((p = 0.014, \eta_p^2 = 0.439)\), but not Condition \((p = 0.530, \eta_p^2 = 0.056)\) or Time \((p = 0.731, \eta_p^2 = 0.011)\). Specifically, collapsed across Condition and Time, \(t\) decreased from Set 1 \((23.6 \pm 3.2)\) to Set 2 \((19.2 \pm 2.1)\) \((p = 0.014)\).
V: DISCUSSION

The results of the present study indicated that SIT, with and without continuous or intermittent BFR, elicited reductions in PP and TW from Sprint 1 to Sprint 2, that were associated with similar increases in perceived effort across sprints. Additionally, Sdec was similar following each SIT protocol and VO2max increased following the completion of the 3 SIT protocols. There were also no differences in mitochondrial function among the 3 SIT protocols that increased following each acute intervention. Despite these similarities across time, PP was lower for CBFR and IBFR than NoBFR during Sprint 2; TW was less for CBFR than IBFR and NoBFR (collapsed across time), ΔHHB that was greater for CBFR than IBFR and NoBFR during Sprint 2, and ΔTHb was greater for BFR than IBFR and NoBFR. Collectively, SIT with or without continuous or intermittent BFR was effective at eliciting potent acute physiological changes that may result in positive cardiovascular-related adaptations (e.g., aerobic capacity, mitochondrial function).

Sprint Performance

In the present study, there were similar reductions in TW from Sprint 1 to Sprint 2 for all 3 conditions, although TW was lower (collapsed across time) during CBFR than IBFR and NoBFR. Additionally, PP decreased from Sprint 1 to Sprint 2 but decreased to a greater extent during CBFR (25.5 ± 11.9%) and IBFR (23.4 ± 9.3%) than NoBFR (13.4 ± 8.6%). There were, however, no differences in Sdec, which was similar among all 3 conditions. These findings were partially consistent with previous investigations (33,60,62,75) that have examined sprint performance following BFR and non-BFR conditions. For example, like the present investigation, PP decreased to a greater extent for IBFR than NoBFR during the fourth and fifth
sprints across 5 maximal 10s sprints performed on a cycle ergometer (33). There were, however, no differences in TW between IBFR (46.8 ± 2.9 W/kg) and NoBFR (49.5 ± 2.4 W/kg) (33). Additionally, there were no differences in PP or TW between IBFR (1147 ± 171 W; 67.1 ± 9.8 kJ, respectively) and NoBFR (1149 ± 179 W; 68.3 ± 10.4 kJ) following 4, 30s maximal sprints when performed at a constant pedaling cadence (68). Furthermore, TW was similar between NoBFR (40 ± 25 kJ) and CBFR (30 ± 16 kJ) across 10s maximal effort sprints to exhaustion on an arm ergometer (76). Contrarily, following 10s maximal effort sprints to exhaustion on an arm ergometer, TW was lower for CBFR (42 – 67 kJ) than NoBFR (162 ± 81 kJ) (75). Thus, in conjunction with previous investigations (33,68,75,76) that have examined BFR and NoBFR, there are inconsistencies among sprint performance that may reflect the heterogeneity among investigations including sample (men or women), number of sprints (3-45+), sprint duration (5s - 30s), hypoxic conditions (local versus systemic), occlusion pressures (individualized or absolute), and modalities (arm cycling, leg cycling, or running).

Despite differences in PP and TW among conditions, there were no differences in RPE that increased across time for each sprint. Like the present findings, RPE has been shown to be similar between normobaric systemic hypoxia (17.7 ± 1.4) and control (16.8 ± 1.6) conditions following 3 sets of 5 × 10s shuttle sprints at maximal intensity (25). Additionally, RPE increased similarly between NoBFR and IBFR across 5 × 30s maximal cycling sprints (62). Furthermore, like RPE, in this investigation there were no differences in Sdec among conditions that was consistent with the findings of Willis et al. 2018. Collectively, these findings, in conjunction with previous investigations (25,33,60,62,68,75,76) indicated that there were discrete differences among sprints performance during BFR and non-BFR conditions, but perception of effort was not affected by the application of BFR.
**Muscle Oxygenation**

The present study examined muscle oxygenation kinetics using NIRS to identify the physiological mechanisms associated with SIT with and without continuous or intermittent BFR. The results of the present study indicated that all 3 SIT protocols (NoBFR, CBFR, and IBFR) elicited changes in these parameters (ΔHHb, ΔO2Hb, ΔTHb, & TSI) across time, while there was a greater increase in ΔHHb during Sprint 2 with CBFR and ΔTHb was greater (collapsed across time) for CBFR. ΔHHb, ΔO2Hb, and ΔTHb measure changes in deoxygenated hemoglobin + myoglobin (oxygen usage), oxygenated hemoglobin + myoglobin (oxygen delivery), and total hemoglobin + myoglobin (blood volume), respectively, that increase with exercise intensity and metabolic stress (4). Collectively, these parameters can be used to track changes in regional muscle oxygenation (TSI) that reflects the balance between muscle oxygen delivery and muscle oxygen usage (47). Therefore, in the present study, the changes among these NIRS parameters for all 3 conditions suggested that all conditions were metabolically demanding and may facilitate aerobic adaptations. The larger changes in ΔHHb and greater overall effect for ΔTHb for CBFR indirectly suggested that it may elicit superior acute physiological adaptations relative to IBFR and NoBFR. These findings were consistent with the application of BFR across other exercises whereby volume is potentially reduced but the physiological adaptations are comparable or even superior with BFR (6,17).

**NIRS Rate Constants and Time Constants**

The NIRS-derived rate and time constants have been applied to make inferences regarding muscle mitochondrial function, demarcating higher versus lower fitness individuals, and is sensitive to training induced aerobic adaptations (35,54). Specifically, an increase in the
rate constant (k) derived from the mono-exponential decay curve of oxygen consumption may represent increased mitochondrial functioning, and/or biogenesis. The time constant (t) is inversely related to the rate constant and provides a measure of the time required for oxygen consumption to return to near baseline levels. In the present study, k and t increased similarly when assessed immediately prior to and after each of the 3 SIT protocols. Acutely, these findings suggested that all 3 protocols were associated with similar acute increases in muscle mitochondrial function that may facilitate chronic adaptations (e.g., increased aerobic capacity). Together, these findings indicated that each sprint condition may be a viable modality to elicit potent aerobic adaptations. In partial support of these findings, VO2max increased by 5.3% following the 3 SIT protocols, although there was no control group to allow for comparison analyses. Our acute findings, however, were corroborated by those of Mandic et al 2022 that reported a 10.3% increase in VO2max following 18 SIT sessions that consisted of 3 × 30s maximal effort sprints on a cycle ergometer. Collectively, the results of the present study elicited acute increases in k and t following 3 SIT sessions totaling 6 sprints over the course of up to 4 weeks that may facilitate beneficial adaptations in muscle mitochondrial function.
VI: CONCLUSION

The results of the present study indicated that SIT with and without BFR elicited reductions in PP and TW that were associated with similar increases in RPE and $S_{dec}$ across sprints. Furthermore, $VO_{2\text{max}}$ increased following the completion of the 3 SIT protocols, and there were no differences in mitochondrial functioning among the 3 SIT protocols which increased. All 3 SIT protocols were also associated with changes in $\Delta HHb$, $\Delta O2Hb$, $\Delta THb$, and TSI from Sprint 1 to Sprint 2, although $\Delta HHb$ and $\Delta THb$ were more pronounced for CBFR. Thus, all 3 SIT protocols were sufficient to induce acute physiological adaptations related to muscle oxygen usage, but that response may be superior with CBFR than IBFR or NoBFR. Collectively, the findings of the present study indicated that applying BFR to maximal aerobic exercise is capable of eliciting similar acute physiological adaptations albeit achieved with less work.
February 8, 2022

Dear Ethan Hill:

On 2/8/2022, the IRB reviewed the following submission:

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<th>Type of Review:</th>
<th>Initial Study, Categories 4 and 7</th>
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<tr>
<td>Title:</td>
<td>The Acute Effects of Continuous and Intermittent Blood Flow Restriction on Sprint Interval Performance and Muscle Oxygen Responses</td>
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The IRB approved the protocol from 2/8/2022.

In conducting this protocol, you are required to follow the requirements listed in the Investigator Manual (HRP-103), which can be found by navigating to the IRB Library within the IRB system. Guidance on submitting Modifications and a Continuing Review or Administrative Check-in are detailed in the manual. When you have completed your research, please submit a Study Closure request so that IRB records will be accurate.

If you have any questions, please contact the UCF IRB at 407-823-2901 or irb@ucf.edu. Please include your project title and IRB number in all correspondence with this office.

Sincerely,

[Signature]

Katie Kilgore
Designated Reviewer
Hello Aaron,

Context - your thesis was part of the IRB project. Within the Huron system, this may also be visible.

Let me know if you need anything else.

Ethan C. Hill, Ph.D, CSCS, EP  
Assistant Professor, School of Kinesiology and Physical Therapy  
Assistant Scientist, Florida Space Institute  
University of Central Florida  
12484 University Boulevard 320L  
Orlando, FL 32816  
Email: ethan.hill@ucf.edu  
Office: 407-823-1183
APPENDIX B:
MEDICAL AND ACTIVITY HISTORY QUESTIONNAIRE
Confidential Medical Health and Activity Questionnaire

Participant # __________
(investigator will assign you a number)

When was your last physical examination? _____/_____/_______

1. Have you ever been hospitalized? If yes, please explain. [ ] N/A
   
   Year of Hospitalization: __________________________
   Reason for Hospitalization: __________________________
   __________________________
   __________________________
   __________________________

2. List any chronic (long-term) illnesses that have caused you to seek medical care. [ ] N/A

3. Have you undergone major surgery within the previous 16 weeks? If yes, please explain. [ ] No

4. Have you ever had (or do you have now) active malignant disease or cancer? If yes, please explain. [ ] No
5. Please place a check in the appropriate box.

**Symptoms or Signs Suggestive of Disease**

- Are you a male over age 45 or female over age 55?
- Have you experienced any unusual pain or discomfort in your chest, neck, jaw, arms, or other areas that may be due to a heart problem?
- Have you experienced unusual fatigue or shortness of breath at rest, during usual activities (e.g., climbing stairs, carrying groceries, walking briskly), or during mild or moderate exercise?
- Have you had any problems with dizziness or fainting?
- When you stand up, do you have difficulty breathing?
- Do you have difficulty breathing while sleeping?
- Do your ankles swell (ankle edema)?
- Have you ever experienced an unusual or rapid heartbeat or fluttering of the heart?
- Have you experienced severe pain in your legs while walking?
- Have you ever been told by a doctor that you have a heart murmur?
- Has anyone in your family died before the age of 40 (excluding accidental death)?
- Are you a cigarette smoker, quit smoking within the past 6 months, or are exposed to tobacco smoke?
- Do you have a history of drug or alcohol dependency?
- Has your doctor ever said you have a heart condition and that you should only do physical activity recommended by a doctor?
- Are you physically inactive (perform little physical activity on the job or during your leisure time)?
- Do you feel any pain in your chest when you do physical activity?
- Are you ever bothered by racing or your heart?
- Do you ever notice abnormal or skipped heartbeats?
- Do you ever have any arm or jaw discomfort, nausea, or vomiting associated with cardiac symptoms?
- Do you ever have difficulty breathing?
- Do you ever experience shortness of breath?
- Have you ever had any tingling or numbness in your arms or legs?
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<td></td>
<td></td>
<td>Claudication</td>
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**Cardiovascular Disease**

<table>
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<tr>
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<th>No</th>
<th>Condition</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>Peripheral vascular disease</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Hypercholesteremia (high cholesterol)</td>
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<td></td>
<td></td>
<td>Cerebrovascular disease</td>
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<td></td>
<td></td>
<td>Coronary artery disease</td>
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<td></td>
<td></td>
<td>Aortic stenosis</td>
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<tr>
<td></td>
<td></td>
<td>Congestive heart failure</td>
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<td></td>
<td></td>
<td>Atrial fibrillation</td>
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<tr>
<td></td>
<td></td>
<td>“Heart block”</td>
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<td></td>
<td></td>
<td>Myocardial infarction (Heart attack)</td>
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<td></td>
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<td>Hypertension</td>
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<td></td>
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<td>Heart pacemaker</td>
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<td></td>
<td></td>
<td>High blood pressure</td>
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<td></td>
<td></td>
<td>Heart murmur</td>
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**Pulmonary**

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<tr>
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<th>No</th>
<th>Condition</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>Chronic obstructive pulmonary disease</td>
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<td></td>
<td></td>
<td>Asthma</td>
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<td></td>
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<td>Interstitial lung disease</td>
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<td></td>
<td></td>
<td>Emphysema</td>
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<td></td>
<td></td>
<td>Metabolic disorder</td>
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<tr>
<td></td>
<td></td>
<td>Diabetes mellitus (type 1, type 2)</td>
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<td></td>
<td>Diabetes insipidus</td>
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<td></td>
<td></td>
<td>Thyroid disorders</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>No</td>
<td>Condition</td>
<td>Comments</td>
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<td>Renal disease</td>
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<td>Liver disease</td>
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<td></td>
<td>Immunodeficiency disorder</td>
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<tr>
<td></td>
<td></td>
<td>Interstitial lung disease</td>
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<td>Emphysema</td>
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<td>Metabolic disorder</td>
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<td>Thyroid disorders</td>
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<td></td>
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<td>Renal disease</td>
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<td></td>
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<td>Liver disease</td>
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<tr>
<td></td>
<td></td>
<td>Immunodeficiency disorder</td>
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</tbody>
</table>

- Do you have a bone or joint problem that could be made worse by a change in your physical activity?
- Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?
- Are you presently taking any nutritional supplements or ergogenic aids?
- Has a health care practitioner ever denied or restricted your participation in sports for any reason?
- Are you pregnant?
- Is there a chance that you may be pregnant?
- When was the first day of your last menstrual cycle (period)?
- Do you know any other reason why you should not do physical activity?
Please check any of the medications or supplements that you currently take regularly. Also give the name of the medication.

<table>
<thead>
<tr>
<th>Type of Medication</th>
<th>Name of the Medication</th>
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</thead>
<tbody>
<tr>
<td>Heart medicine</td>
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<tr>
<td>Blood pressure medicine</td>
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<td>Blood cholesterol medicine</td>
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<tr>
<td>Hormones</td>
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<td>Birth control pills</td>
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<td>Medicines for breathing or lungs</td>
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<tr>
<td>Insulin</td>
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<tr>
<td>Other medicine for diabetes</td>
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<tr>
<td>Arthritis medicine</td>
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<tr>
<td>Medicines for depression</td>
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<td>Medicine for anxiety</td>
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<td>Thyroid medicine</td>
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<td>Medicines for ulcers</td>
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<tr>
<td>Painkiller medicine</td>
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<td>Allergy medicine</td>
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<td>HIV/AIDS medicine</td>
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<tr>
<td>Hepatitis medicine</td>
<td></td>
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<tr>
<td>Other medicines or supplement</td>
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</tbody>
</table>

6. Please check any of the medications or supplements that you currently take regularly. Also give the name of the medication.
7. Are you allergic to any medications? If yes, please list medications and reaction. □ No

8. Please list any allergies, including food allergies that you may have? □ N/A

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INTERNATIONAL PHYSICAL ACTIVITY QUESTIONNAIRE

We are interested in finding out about the kinds of physical activities that people do as part of their everyday lives. The questions will ask you about the time you spent being physically active in the last 7 days. Please answer each question even if you do not consider yourself to be an active person. Please think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise, or sport.

Think about all the vigorous and moderate activities that you did in the last 7 days. Vigorous physical activities refer to activities that take hard physical effort and make you breathe much harder than normal. Moderate activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal.

PART 1: JOB-RELATED PHYSICAL ACTIVITY

The first section is about your work. This includes paid jobs, farming, volunteer work, course work, and any other unpaid work that you did outside your home. Do not include unpaid work you might do around your home, like housework, yard work, general maintenance, and caring for your family. These are asked in Part 3.

1. Do you currently have a job or do any unpaid work outside your home?
   □ Yes
   □ No  →  Skip to PART 2: TRANSPORTATION

The next questions are about all the physical activity you did in the last 7 days as part of your paid or unpaid work. This does not include traveling to and from work.

2. During the last 7 days, on how many days did you do vigorous physical activities like heavy lifting, digging, heavy construction, or climbing up stairs as part of your work? Think about only those physical activities that you did for at least 10 minutes at a time.

   _____ days per week
3. How much time did you usually spend on one of those days doing vigorous physical activities as part of your work?

   ____ hours per day
   ____ minutes per day

4. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do moderate physical activities like carrying light loads as part of your work? Please do not include walking.

   ____ days per week

   No moderate job-related physical activity  ➔ Skip to question 6

5. How much time did you usually spend on one of those days doing moderate physical activities as part of your work?

   ____ hours per day
   ____ minutes per day

6. During the last 7 days, on how many days did you walk for at least 10 minutes at a time as part of your work? Please do not count any walking you did to travel to or from work.

   ____ days per week

   No job-related walking  ➔ Skip to PART 2: TRANSPORTATION

7. How much time did you usually spend on one of those days walking as part of your work?

   ____ hours per day
   ____ minutes per day

**PART 2: TRANSPORTATION PHYSICAL ACTIVITY**

These questions are about how you traveled from place to place, including to places like work, stores, movies, and so on.

8. During the last 7 days, on how many days did you travel in a motor vehicle like a train, bus, car, or tram?

   ____ days per week

   No traveling in a motor vehicle  ➔ Skip to question 10

9. How much time did you usually spend on one of those days traveling in a train, bus, car, tram, or other kind of motor vehicle?


Exercise Physiology Intervention and Collaboration Lab

_____ hours per day
_____ minutes per day

Now think only about the bicycling and walking you might have done to travel to and from work, to do errands, or to go from place to place.

10. During the last 7 days, on how many days did you bicycle for at least 10 minutes at a time to go from place to place?

_____ days per week

☐ No bicycling from place to place ➔ Skip to question 12

11. How much time did you usually spend on one of those days to bicycle from place to place?

_____ hours per day
_____ minutes per day

12. During the last 7 days, on how many days did you walk for at least 10 minutes at a time to go from place to place?

_____ days per week

☐ No walking from place to place ➔ Skip to PART 3: HOUSEWORK, HOUSE MAINTENANCE, AND CARING FOR FAMILY

13. How much time did you usually spend on one of those days walking from place to place?

_____ hours per day
_____ minutes per day

PART 3: HOUSEWORK, HOUSE MAINTENANCE, AND CARING FOR FAMILY

This section is about some of the physical activities you might have done in the last 7 days in and around your home, like housework, gardening, yard work, general maintenance work, and caring for your family.

14. Think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do vigorous physical activities like heavy lifting, chopping wood, shoveling snow, or digging in the garden or yard?

_____ days per week

☐ No vigorous activity in garden or yard. ➔ Skip to question 16

15. How much time did you usually spend on one of those days doing vigorous physical activities in the garden or yard?
hours per day

minutes per day

16. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do moderate activities like carrying light loads, sweeping, washing windows, and raking in the garden or yard?

___ days per week

☐ No moderate activity in garden or yard.  

Skip to question 18

17. How much time did you usually spend on one of those days doing moderate physical activities in the garden or yard?

___ hours per day

___ minutes per day

18. Once again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do moderate activities like carrying light loads, washing windows, scrubbing floors and sweeping inside your home?

___ days per week

☐ No moderate activity inside home  

Skip to PART 4: RECREATION, SPORT AND LEISURE-TIME PHYSICAL ACTIVITY

19. How much time did you usually spend on one of those days doing moderate physical activities inside your home?

___ hours per day

___ minutes per day

PART 4: RECREATION, SPORT, AND LEISURE-TIME PHYSICAL ACTIVITY

This section is about all the physical activities that you did in the last 7 days solely for recreation, sport, exercise or leisure. Please do not include any activities you have already mentioned.

20. Not counting any walking you have already mentioned, during the last 7 days, on how many days did you walk for at least 10 minutes at a time in your leisure time?

___ days per week

☐ No walking in leisure time.  

Skip to question 22

21. How much time did you usually spend on one of those days walking in your leisure time?

___ hours per day

___ minutes per day
22. Think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do **vigorou**s physical activities like aerobics, running, fast bicycling, or fast swimming in your leisure time?

___ days per week

☐ No vigorous activity in leisure time → **Skip to question 24**

23. How much time did you usually spend on one of those days doing **vigorou**s physical activities in your leisure time?

___ hours per day

___ minutes per day

24. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do **moderate** physical activities like bicycling at a regular pace, swimming at a regular pace, and doubles tennis in your leisure time?

___ days per week

☐ No moderate activity in leisure time → **Skip to PART 5: TIME SPENT SITTING**

25. How much time did you usually spend on one of those days doing **moderate** physical activities in your leisure time?

___ hours per day

___ minutes per day

**PART 5: TIME SPENT SITTING**

The last questions are about the time you spend sitting while at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading or sitting or lying down to watch television. Do not include any time spent sitting in a motor vehicle that you have already told me about.

26. During the last 7 days, how much time did you usually spend **sitting** on a **weekday**?

___ hours per day

___ minutes per day

27. During the last 7 days, how much time did you usually spend **sitting** on a **weekend day**?

___ hours per day

___ minutes per day

This is the end of the questionnaire, thank you for participating.
14. I have answered these questions honestly and have provided all past and present health and exercise information to the best of my knowledge.

Yes  No

_____________  ______/_____/______
Participant #  Date
APPENDIX C: FLOW CHART FOR MUSCLE MITOCHONDRIAL PROTOCOL
1. 250mmHg Cuff inflation for 30 seconds
2. 30 second rest
3. Inflation for 30 seconds
4. 30 second rest
5. 30% MVIC for 5 seconds
6. 3-6 minute inflation
7. 1-3 minute rest
8. 30% MVIC for 15 seconds
9. Inflations 1-5, at 5 seconds occlusion each inflation
   5 seconds of rest between each inflation
10. Inflations 6-10, at 5 seconds occlusion each inflation
    10 seconds of rest between each inflation
11. Inflations 11-15, at 10 seconds occlusion each inflation
    20 seconds of rest between each inflation
12. 30 seconds of rest
13. Repeat steps 1-11
**Figures 1 and 2** Individual (1) and mean ± SD (2) values for peak power (PP). PP was determined as the highest power output (W) generated over the course of each sprint performed with continuous blood flow restriction (CBFR), intermittent blood flow restriction (IBFR), and no blood flow restriction (NoBFR). There was a significant interaction for PP that was decomposed into follow-up separate 3 × 1 ANOVAs.

*Significant (*p* ≤ 0.05) simple main effects for time (Sprint 1 > Sprint 2).
†Significant (*p* ≤ 0.05) simple main effect for Condition for Sprint 2 (CBFR and IBFR < NoBFR).
Figure 3 Individual Total Work

Figure 4 Mean Total Work

**Figures 3 and 4.** Individual (1) and mean ± SD (2) values for total work (TW). TW in joules (J) was determined as the product of force (in newtons) and distance covered, with continuous blood flow restriction (CBFR), intermittent blood flow restriction (IBFR), and no blood flow restriction (NoBFR). There was no significant interaction, but there were significant main effects for Time and Condition.

*Significant ($p \leq 0.05$) main effect for Time, collapsed across Condition (Sprint 1 > Sprint 2).
‡Significant ($p \leq 0.05$) main effect for Condition, collapsed across Time (CBFR < IBFR and NoBFR).
Figures 5 and 6. Individual (1) and mean ± SD (2) responses for ratings of perceived exertion (RPE), measured after each sprint with continuous blood flow restriction (CBFR), intermittent blood flow restriction (IBFR), and no blood flow restriction (NoBFR). There was no significant interaction, but there was a significant main effect for Time.

*Significant ($p \leq 0.05$) main effect for Time, collapsed across Condition (Sprint 2 > Sprint 1).
Figure 7 Individual Sprint Decrement

Figure 8 Mean Sprint Decrement

**Figures 7 and 8.** Individual (1) and mean ± SD (2) values for sprint decrement ($S_{\text{dec}}$). $S_{\text{dec}}$ was calculated as total work covered from both sprints relative to the best sprint and expressed as a percentage. There was a significant one-way repeated measures ANOVA across Condition, but there were no significant follow-up Bonferroni-corrected paired samples $t$-tests. Thus, $S_{\text{dec}}$ was similar among all 3 conditions.
In individual (1) and mean ± SD (2) responses for ΔHHb, determined during the last 5 seconds of each sprint with continuous blood flow restriction (CBFR), intermittent blood flow restriction (IBFR), and no blood flow restriction (NoBFR). There was a significant interaction for ΔHHb that was decomposed into follow-up separate 3 × 1 ANOVAs.

*Significant (*p* ≤ 0.05) main effect for Time, collapsed across Condition (Sprint 2 > Sprint 1)
†Significant (†*p* ≤ 0.05) main effect for Condition (CBFR > IBFR and NoBFR)
In Figures 11 and 12, individual (1) and mean ± SD (2) responses for ΔO2Hb, determined during the last 5 seconds of each sprint with continuous blood flow restriction (CBFR), intermittent blood flow restriction (IBFR), and no blood flow restriction (NoBFR). There was no significant interaction, but there was a significant main effect for Time.

*Significant ($p \leq 0.05$) main effect for Time, collapsed across Condition (Sprint 2 > Sprint 1)
Figures 13 and 14. Individual (1) and mean ± SD (2) responses for ΔTHb, determined during the last 5 seconds of each sprint with continuous blood flow restriction (CBFR), intermittent blood flow restriction (IBFR), and no blood flow restriction (NoBFR). There was no significant interaction, but there were significant main effects for Condition and Time. *Significant (p ≤ 0.05) main effect for Time, collapsed across Condition (Sprint 2 > Sprint 1) † Significant (p ≤ 0.05) main effect for Condition, collapsed across Time (CBFR > IBFR and NoBFR)
Figures 15 and 16. Individual (1) and mean ± SD (2) responses for TSI, determined during the last 5 seconds of each sprint with continuous blood flow restriction (CBFR), intermittent blood flow restriction (IBFR), and no blood flow restriction (NoBFR). There was no significant interaction, but there was a significant main effect for Time.

*Significant ($p \leq 0.05$) main effect for Time, collapsed across Condition (Sprint 2 > Sprint 1)
Figure 17. Pre-sprints (▼) and post-sprints (□) recovery curves for muscle oxygen consumption (mVO₂).
REFERENCES


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48. Popov, DV, Swirkun, DV, Netreba, AI, Tarasova, OS, Prostova, AB, Larina, IM, et al. Hormonal adaptation determines the increase in muscle mass and strength during low-


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