

# **Exercise Intensity-Specific Changes to Cerebral Blood Velocity do not Modulate a Postexercise Executive Function Benefit**

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**Running Title:** Exercise and executive function

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## Abstract

Executive function is transiently improved (i.e., < 60-min) following a single bout of aerobic exercise. A candidate mechanism for this improvement is an exercise-mediated increase in cerebral blood flow (CBF). Further, it has been proposed that an increase in CBF across the continuum of increasing exercise intensities improves the *magnitude* of a postexercise executive function benefit (i.e., drive theory); however, this proposal has not been empirically tested. Here, participants completed four experimental sessions: a  $\dot{V}O_{2\text{peak}}$  test to determine cardiorespiratory fitness and estimated lactate threshold (LT), followed by separate 10-min sessions of light- (i.e., 25 W), moderate- (i.e., 80% estimated LT), and heavy-intensity (i.e., 15% of the difference between LT and  $\dot{V}O_{2\text{peak}}$ ) aerobic exercise. An estimate of CBF during exercise was achieved via transcranial Doppler ultrasound and near-infrared spectroscopy to quantify blood velocity (BV) through the middle cerebral artery and deoxygenated hemoglobin (HHb), respectively. Executive function was assessed before and after each session via the executive-mediated antisaccade task (i.e., saccade mirror-symmetrical to a target). Results demonstrated that BV increased in relation to increasing exercise intensity, whereas HHb decreased by a comparable magnitude independent of intensity. In terms of executive function, null hypothesis and equivalence tests indicated a comparable magnitude postexercise reduction in antisaccade reaction time across exercise intensities. Accordingly, the *magnitude* of CBF change during exercise does not impact the *magnitude* of a postexercise executive function benefit.

**Key Words:** *aerobic exercise; antisaccade; exercise intensity; transcranial Doppler; vision*

## 1. Introduction

Executive function entails high-level cognitive control supporting inhibition, updating and set-shifting (Miyake et al., 2000) – components essential for activities of daily living. Although evidence indicates that a single bout of aerobic or resistance exercise benefits executive function, the literature remains mixed with regards to the duration, intensity, participant fitness level, timing of assessment and component of executive function assessed (i.e., inhibitory control, working memory, cognitive flexibility) that provides the most optimal benefit (for meta-analyses see, Lambourne and Tomporowski, 2010; Chang et al., 2012). For example, Chang et al. reported 20-min of moderate intensity exercise is required to benefit executive function and that the largest positive benefit is observed 11-20-min postexercise, with a smaller benefit observed thereafter. In turn, Lambourne and Tomporowski concluded that an executive function benefit is observed up to 15-min postexercise. That said, more recent investigations have found that engaging in acute aerobic and resistance exercise for as little as 10 min improves executive function (Johnson et al., 2016; Samani and Heath, 2018) and that the benefit may persist for upwards of 60-min (Hung et al., 2013; Shukla and Heath, 2021; see also Joyce et al., 2009). Moreover, there is debate as to the mechanism(s) by which exercise improves executive function (Ludyga et al., 2016). Indeed, postexercise benefits have been linked to increases in catecholamine (Zouhal et al., 2008) and/or brain-derived neurotrophic factor (BDNF) (Knaepen et al., 2010) concentrations, as well as changes in resting state functional connectivity (Schmitt et al., 2019). As well, an exercise-mediated increase in cerebral blood flow (CBF) (Ide and Secher, 2000; Colcombe and Kramer, 2003; Querido and Sheel, 2007; Smith et al., 2010; Chang et al., 2012; Ludyga et al., 2016; Tari et al., 2020; but see Shoemaker et al., 2020) has been frequently linked to a postexercise executive function benefit. In particular, exercise onset produces a rapid

## Exercise and executive function

rise in CBF via CO<sub>2</sub> production, neurovascular coupling and increased systolic blood pressure (Smith and Ainslie, 2017). The increase in CBF renders mechanical- and temperature-based changes to the brain's neural and glial networks that enhance local neural circuits and improve information processing (i.e., the hemo-neural hypothesis) (Moore and Cao, 2008). Further, the link between CBF and executive function is indirectly demonstrated via studies showing that age- and/or disease-related decreases/disruptions to CBF impair executive function (Bertsch et al., 2009). As well, in a more direct examination, Tari et al. (2020) had participants complete a 10-min non-exercise session involving inhalation of a higher-than-atmospheric concentration of CO<sub>2</sub> (i.e., a hypercapnic environment) and separate 10-min sessions of aerobic exercise and a non-exercise, non-hypercapnic control condition. The hypercapnic condition was used because it increases CBF independent of the metabolic demands of exercise (for reviews see, Ainslie and Duffin, 2009; Hoiland et al., 2019). Tari et al. matched hypercapnic- and exercise-related changes in CBF via real-time transcranial Doppler ultrasound (TCD) monitoring of blood velocity (BV) through the middle cerebral artery (MCA) and showed that exercise and hypercapnic conditions produced equivalent magnitude pre- to postcondition improvements in executive function. In turn, the control condition did not demonstrate a pre- to postcondition change in executive function. The authors therefore proposed that an exercise-mediated improvement in executive function is – in part – supported via an increase in CBF.

The putative link between CBF and a postexercise benefit to executive function represents an interesting question for at least two reasons. First, drive theories assert a linear relationship between exercise intensity and executive function such that the benefit scales in relation to increasing intensity (Chang et al., 2012). In contrast, other work has reported a non-linear relationship such that light- to moderate-intensity exercise linearly improves executive

## Exercise and executive function

function and then asymptotes or decreases thereafter (i.e., heavy to very-heavy), whereas other work has reported a monotonic relationship (Chang and Etnier, 2009). As first identified by Lambourne and Tomporowski (2010), the discrepancy related to establishing a dose-response relationship is likely attributed to between-experiment differences in quantifying exercise intensity. Indeed, predicted maximum heart rate ( $HR_{max}$ ) and peak/maximum oxygen consumption ( $\dot{V}O_{2peak/max}$ ) are common taxonomies for quantifying exercise intensity; however, they do not ensure between-participant equivalence in physiological responses such as lactate threshold and the ability to maintain constant power at increasing intensities (Keir et al., 2016). Second, the CBF response to exercise is commonly reported as linear within light- to heavy-intensities (Smith and Ainslie, 2017), although the mechanisms regulating CBF are distinct. For light- and moderate-intensity, the increase in CBF is in large part attributed to increased arterial  $CO_2$  ( $PaCO_2$ ) – which induces hypercapnia – and systolic blood pressure (Hoiland et al., 2019). For heavy-intensity (i.e., beyond ventilatory threshold),  $PaCO_2$  gradually returns to baseline – or below – as exercise progresses; however, CBF is generally maintained due to increasing systolic blood pressure (Hoiland et al., 2019). Thus, the linear relationship between CBF and exercise intensity is consistent with drive theories of postexercise executive function improvements; however, to our knowledge, no work has directly examined whether exercise intensity-specific changes to CBF impact the magnitude of a postexercise benefit to executive function.

We examined pre- and postexercise executive function across 10-min single bouts of aerobic exercise (via cycle ergometer) at: 1) light-intensity (25 W), 2) moderate-intensity (80% of estimated participant-specific LT) and 3) heavy-intensity (15% of the difference between estimated participant-specific LT and  $\dot{V}O_{2peak}$ ). The 10-min exercise duration was chosen because it imparts a reliable postexercise executive function benefit (Samani and Heath, 2018)

## Exercise and executive function

and the defined moderate- and heavy-intensities were selected because they provide an absolute basis for between-participant equivalence in metabolic demands and because they show a linear relationship with CBF. During each exercise session, BV through the MCA and deoxygenated hemoglobin were measured via TCD and near infrared spectroscopy (NIRS) – measures providing a proxy for direct measures of CBF (Bishop et al., 1986, Madsen and Secher, 1999). Pre- and postexercise executive function was examined via the antisaccade task. Antisaccades require a goal-directed eye movement (i.e., a saccade) mirror-symmetrical to an exogenously presented target (i.e., 180° spatial transformation) and result in longer reaction times (RT) (Hallet, 1978) and more variable endpoints (Gillen and Heath, 2014a) than their prosaccade (i.e., saccade to veridical target location) counterparts. Extensive neuroimaging and single-cell-recording in non-human primates has attributed the antisaccade behavioural costs to the executive demands of response suppression and vector inversion (Munoz and Everling, 2004). What is more, the frontoparietal networks supporting antisaccades are the same as those showing task-dependent modulation following single bout and chronic exercise (Voss et al., 2010, Verbrugh et al., 2013). In turn, a single bout of aerobic exercise (i.e., 10 to 20-min) improves antisaccade reaction times for up to 47-min postexercise – a result attributed to improved executive function (Tari et al., 2020; Shukla et al., 2020, Shukla and Heath, 2021). Here, we sought to determine whether exercise intensity-related increases in CBF elicit a proportional postexercise improvement in executive function. Accordingly, if CBF and antisaccade – but not prosaccade – RTs increase and decrease, respectively (i.e., are inversely related), with increasing exercise intensity, then evidence would suggest that the magnitude of an exercise-mediated increase in CBF influences the magnitude of a postexercise benefit to executive function (i.e., a dose-response relationship). In contrast, if there is a threshold after which an increase in CBF

## Exercise and executive function

does not further benefit executive function, then the magnitude of a postexercise executive function benefit should be independent of intensity-specific changes in CBF. Moreover, prosaccades were evaluated at pre- and postexercise assessments because they are largely mediated independent of top-down executive control and provide a basis to determine whether a single bout of exercise globally improves oculomotor control (i.e., pro- and antisaccades) or selectively benefits an executive-mediated task (i.e., antisaccades).

## 2. Materials and Methods

### 2.1 Participants

Sixteen undergraduate and graduate students (5 female, age range 21–25 years) from the School of Kinesiology, University of Western Ontario volunteered for this study with sample size determined *a priori* based on the effect size derived from a paired-samples t-test contrasting pre- and postexercise antisaccade RTs ( $\alpha=0.05$ , power = 0.90,  $d_z= 1.30$ ) (Tari et al., 2020). Participants were self-reported right-hand dominant (i.e., “What hand do you use to write with?”), with normal or corrected to normal vision, no history of smoking and/or cardiorespiratory, metabolic, musculoskeletal, neurologic (including concussion), or neuropsychiatric disorder. Participants also reported that they did not take medication that may affect metabolic, cardiac, respiratory or hemodynamic responses to exercise. Prior to data collection, participants read a letter of information approved by the Health Sciences Research Ethics Board, University of Western Ontario and provided informed written consent. This study was conducted according to the most recent iteration of the Declaration of Helsinki with the exception that participants were not registered in a database. Participants obtained a full score on the 2019 Physical Activity Readiness Questionnaire (PAR-Q+) and completed the Godin

## Exercise and executive function

Leisure-Time Exercise Questionnaire (GLTEQ). The average GLTEQ score was 51 (SD = 21) indicating that all participants were recreationally active.

### 2.2 Experimental Overview

Four exercise conditions were completed on different days separated by at least 24 h with each condition performed between 9:30 and 11:00 am and participants were requested to consume approximately 2 cups of water one to two hours prior to data collection to provide a level of hydration. Participants exercised on a cycle ergometer (Velotron; RacerMate, Seattle, WA) with power output independent of pedal cadence, and cadence set at 70 rpm. In the first condition, participants completed an incremental ramp test to volitional exhaustion to determine peak oxygen consumption ( $\dot{V}O_{2peak}$ ) and LT. LT was defined as the  $\dot{V}O_{2peak}$  at which  $\dot{V}CO_2$  began to increase out of proportion to  $\dot{V}O_2$  with a systematic rise in minute ventilation-to- $\dot{V}O_2$  ratio and end-tidal  $PO_2$ , whereas minute ventilation-to- $\dot{V}CO_2$  ratio and end-tidal  $PCO_2$  were stable (Meyer et al., 2005). The subsequent three conditions entailed 10-min bouts of aerobic exercise (via cycle ergometer) at work-rates corresponding to 25 W (i.e., light-intensity), 80% of estimated LT (i.e., moderate-intensity), and 15% of the difference between individual-specific estimated LT and  $\dot{V}O_{2peak}$  (i.e., heavy-intensity). The exercise duration was based on work demonstrating that 10-min of aerobic exercise provides a reliable postexercise benefit to executive function (Samani and Heath, 2018; Tari et al., 2020; Shukla et al., 2021). For exercise intensity, 25 W was used as the work rate for a light-intensity because it corresponds to an intensity continuum frequently employed in the exercise literature (e.g., Takata et al., 1990; Shim et al., 2013). In turn, participant-specific moderate- and heavy-intensities were based around LT instead of the oft-used  $\dot{V}O_{2peak}$  because the former increases linearly with exercise intensity, whereas the latter does not (Keir et al., 2018). Hence, LT provides a consistent and linear basis to assign task-



specific and constant-intensity work rates. Moreover, the moderate and heavy-intensity work rates used here produce reliable immediate postexercise benefits to executive function (Heath et al., 2018; Petrella et al., 2019; Tari et al., 2020; see also Lambourne and Tomporowski, 2010; Chang et al., 2012; Ludyga et al., 2016).

### 2.3 Apparatus and Procedures

2.3.1.  $\dot{V}O_{2peak}$  condition. This condition measured participants' maximal  $O_2$  consumption and estimated LT. A confirmation ride (i.e.,  $\dot{V}O_{2max}$ ) was not completed because  $\dot{V}O_{2peak}$  is a reliable measure of maximal  $O_2$  consumption for individuals familiar with maximal exercise testing (see *Participants* section 2.1) (Poole and Jones, 2017). For this condition, an incremental ramp test to volitional exhaustion was performed with a work rate increment of 25 Watts (W) per minute. Strong verbal encouragement was given to facilitate peak effort. Participants wore a nose clip to prevent breathing from the nose, and a mouthpiece similar to breathing through a snorkel for breath-by-breath gas exchange analyses of  $O_2$  uptake and  $CO_2$  output. Air flow and volumes were measured via a bidirectional turbine of 100 mL dead space (VMM 110; Alpha Technologies, Laguna Hills, CA, USA) and pneumotach (model 4813; Hans Rudolph, Shawnee, KS, USA). Fractional concentrations of  $O_2$ ,  $CO_2$ , and nitrogen ( $N_2$ ) at the mouth were measured via mass spectrometry (AMIS 2000; Innovision ApS, Glamsbjerg, Denmark). To provide a profile of each breath, a peak-detection algorithm was used to determine end-tidal  $CO_2$  ( $P_{ET}CO_2$ ) and  $O_2$  ( $P_{ET}O_2$ ) pressures with inspired and expired gas volumes and durations time aligned at a sampling rate of 100 Hz. Heart rate was measured continuously by a heart rate monitor (Polar Electro T34, Kempele, Finland) using PowerLab (ML132/ML880, ADInstruments, Dunedin, New Zealand) and was calculated (using a 5 s rolling average) based on successive heart beats

## Exercise and executive function

(i.e., RR interval). Data were recorded using LabChart version 6.1 (Dunedin, New Zealand) on a separate computer.

Beaver et al. (1986) demonstrated that the  $\dot{V}O_2$  associated with lactate threshold (LT) does not differ from direct measures of LT in young healthy individuals. As such, we estimated LT via the V-slope method outlined by Schneider et al. (1993) and implemented in previous work by our group (Heath et al., 2018; Tari et al., 2020). LT was estimated from  $\dot{V}O_{2peak}$  data via visual inspection using standard gas exchange and ventilatory parameters and was determined as the  $\dot{V}O_2$  at which  $\dot{V}CO_2$  and  $\dot{V}_E$  began to increase out of proportion to  $\dot{V}O_2$ . This point was corroborated with the observation of an increase in end-tidal  $PO_2$ , while the  $\dot{V}_E$ -to- $\dot{V}CO_2$  ratio and  $P_{ET}CO_2$  were unchanged. Two exercise physiologists with experience in identifying LT evaluated each data set. If a discrepancy arose between the two investigators, a mean of the identified points was utilized. Once the  $\dot{V}O_2$  associated with LT was determined, 80% of that value was used to calculate power output for moderate-intensity exercise, whereas 15% of the difference between the estimated participant-specific LT and  $\dot{V}O_{2peak}$  defined the power output for a heavy intensity (see details below).

**2.3.2. *Light-, moderate- and heavy-intensity exercise.*** For the exercise conditions, participants sat on the same cycle ergometer as used in the  $\dot{V}O_{2peak}$  condition for 4 min to achieve a resting baseline and then pedalled for a 6 min warm-up at 25 W to achieve a physiological baseline “steady state”. Following baseline, the same work rate was maintained in the light-intensity condition, whereas a transition to 80% of the estimated participant-specific LT (i.e., moderate intensity), or 15% of the difference between the estimated participant-specific LT and  $\dot{V}O_{2peak}$  was completed (i.e., heavy intensity). In particular, the delay of the metabolic response (i.e., the time between ramp increment onset and  $\dot{V}O_2$  response) and the time spent at baseline during the

## Exercise and executive function

$\dot{V}O_{2\text{peak}}$  condition (i.e., 6 min) were subtracted from the total participant-specific end ramp exercise time and used to obtain participant-specific work rates (Keir et al., 2016, 2018; Heath et al., 2018). For moderate-intensity exercise, the cycle ergometer was programmed to transition from 25 W to a participant-specific wattage between 30 and 66 W (average=41, SD=15) for females, and 50 and 92 W (average=78, SD=12) for males. In the heavy-intensity condition, the cycle ergometer transitioned from 25 W to between 60 and 121 W for females (average= 78, SD=25) and between 108 and 148 W (average=127, SD=11) for males (see **Table 1**). The order in which the different exercise intensities were completed was counterbalanced.

**Table 1. Participant-specific  $\dot{V}O_{2\text{peaks}}$ , lactate thresholds and work rates across exercise conditions.**

Participant	Sex	Age	Height	Weight	$\dot{V}O_{2\text{peak}}$	LT	Light	Moderate	Heavy
1	F	24	160.02	61.23	1.79	0.65	25	30	71
2	F	25	167.64	74.84	1.54	0.88	25	30	60
3	M	20	175.26	86.18	3.23	1.49	25	90	120
4	M	24	175.00	79.38	3.35	1.60	25	81	131
5	M	24	172.00	79.38	2.94	1.66	25	66	126
6	M	24	172.72	58.97	2.24	1.35	25	76	114
7	M	22	187.96	92.98	4.05	2.15	25	50	125
8	F	25	157.48	46.26	1.60	1.00	25	44	78
9	M	21	175.26	68.03	2.97	1.65	25	81	131
10	F	21	152.40	63.50	1.72	1.15	25	37	60
11	F	21	157.48	49.89	2.23	1.60	25	66	121
12	M	24	182.88	86.18	2.35	1.43	25	89	133
13	M	22	170.18	65.77	3.17	1.53	25	83	139
14	M	22	182.88	78.92	3.24	1.64	25	76	108
15	M	23	180.34	83.91	3.30	1.52	25	75	120
16	M	22	180.34	77.11	2.91	1.59	25	92	148
$\mu$		22.75	171.87	72.03	2.66	1.43	25	67	112
SD		1.57	10.43	13.51	0.75	0.36	0	22	28

Note: Individual participants' sex (F: female; M: male), age (years), height (cm), weight (kg), peak oxygen consumption ( $\dot{V}O_{2\text{peak}}$ : L/min) and estimated lactate thresholds (LT) are listed as they correspond to work rates (W) for Light-, Moderate-, and Heavy-intensity exercise. Means ( $\mu$ ) and standard deviations (SD) are listed below.

## Exercise and executive function

For light-, moderate-, and heavy-intensity exercise conditions, transcranial Doppler ultrasound (TCD) (Neurovision 500M, Neurovision TOC2M; Multigon Industries, Elmsford, CA, USA) and near-infrared spectroscopy (NIRS) (Oxiplex TS, model 92505; ISS, Champaign, IL, USA) probes were used to measure: 1) blood velocity (BV) through the middle cerebral artery, 2) absolute cerebral deoxygenated hemoglobin (HHb), and 3) total hemoglobin concentration (THC). These measures provide a valid proxy for direct measures of CBF (Bishop et al., 1986). The NIRS probe was placed on the frons and the TCD probe was coated in an aqueous ultrasound gel (Aquasonic Clear, Parker Laboratories Inc., Fairfield, NJ, USA) and placed on the left anterior temporal window and the MCA was identified via criteria outlined by Aaslid et al. (1982). NIRS and TCD probes were secured via a headband. Heart rate (HR) was continuously measured, and blood pressure was taken at regular intervals (i.e., 3, 6, 9, 13, 16, and 19 min) via a manual sphygmomanometer and stethoscope (Welch Allyn FlexiPort reusable blood pressure cuff; Welch Allyn Inc. Skaneateles Falls, NY, USA) secured to participants' left upper arm. As well, ventilatory variables were attained as per the measures outlined in the  $\dot{V}O_{2peak}$  condition.

### 2.4 Oculomotor Assessment

Participants completed an oculomotor assessment (with which they were not familiarized) before and after light-, moderate-, and heavy-intensity exercise conditions. Participants sat at a height adjustable chair in front of a table with their heads placed in a head-chin rest. The gaze position of participants' left eye was measured via a video-based eye-tracking system (EyeLink 1000 Plus, SR Research, Ottawa, ON, Canada) sampling at 1000 Hz. Visual stimuli were presented against a black screen (0.1 cd/cm<sup>2</sup>) on a 30-inch LCD monitor (60 Hz, 8 ms response rate, 1280x960 pixels, Dell 3007WFP, Round Rock, TX, USA) that was 550 mm from the front edge

## Exercise and executive function

of the table and centered at participants' midline. Prior to data collection, a nine-point calibration of participants' viewing space was performed and followed by an immediate validation (i.e.,  $< 1^\circ$  of error for each point of the calibration space). Two additional monitors visible only to the experimenter provided gaze position data and trial-to-trial saccade kinematics. Computer events were controlled via MATLAB (R2018b, The MathWorks, Natick, MA, USA) and the Psychophysics Toolbox extensions (v 3.0) (Kleiner et al., 2007) including the Eyelink Toolbox (Cornelissen et al., 2002). The lights in the experimental suite were extinguished during data collection.

Visual stimuli included a midline located red ( $1^\circ$ : 50 cd/cm<sup>2</sup>) or green ( $1^\circ$ : 34 cd/cm<sup>2</sup>) fixation cross presented at participants' eye level, and targets (i.e., open white circles; 2.5° diameter: 127 cd/cm<sup>2</sup>) located 15° (i.e., proximal target) and 20° (i.e., distal target) to the left and right of fixation and along the same horizontal plane. The onset of a fixation cross signalled participants to direct their gaze to its location. Once a stable gaze was achieved (i.e.,  $\pm 1.5^\circ$  for 450 ms), a uniformly distributed randomized foreperiod between 1000 and 2000 ms was introduced after which the fixation cross disappeared for 200 ms (i.e., gap paradigm). Following the gap, a target was presented in one of four locations (i.e., left 15° or 20°; right 15° or 20°) for 50 ms. For half of participants, when the fixation cross was green, target onset cued a prosaccade (i.e., saccade to veridical target location) at target onset, whereas a red fixation cross indicated an antisaccade (i.e., saccade mirror-symmetrical to the target location) at target onset. For the other half of participants, the converse fixation and task-type mapping was used. Participants were asked to complete their responses "quickly and accurately". Pro- and antisaccades were completed in separate and randomly ordered blocks wherein 20 trials were pseudo-randomly presented at each target location (i.e., left and right visual field) and

## Exercise and executive function

eccentricity (i.e., proximal and distal) (i.e., 160 total trials). Following the pre-exercise oculomotor assessment, participants immediately began their appropriate exercise session. After the exercise session, participants began their postexercise oculomotor assessment when their heart rate dropped below 100 bpm (i.e., <4-min). Each oculomotor assessment required approximately 17-min to complete and the timeframe is well within the window (~47-min) wherein antisaccades have been shown to produce a reliable postexercise reduction in RT (Shukla and Heath, 2021).

### 2.5 Data Reduction, Dependent Variables, and Statistical Analyses

Ventilatory and NIRS data three standard deviations from a participant-specific mean were removed (Lamarra et al., 1987), data were then linearly interpolated on a second-by-second basis, time-aligned to the onset of an experimental session and averaged into 5-s time bins (Keir et al., 2015). TCD data corrupted by signal aliasing and/or signal loss (e.g., a sudden head shift) were omitted (Terslev et al., 2017) and systolic BVs were retained for analysis (Clyde et al., 1996). Systolic BV were analyzed given Rosengarten and Kaps' (2002) demonstration that it provides a valid measure for TCD-based measures of BV through the MCA.

For the oculomotor task, trials involving a signal loss (e.g., an eye blink) were removed. Trials involving anticipatory responses (RTs < 50 ms) (Wenban-Smith and Findlay, 1991) or RTs > 2.5 standard deviations of a participant- and task-specific mean were excluded, as were trials with amplitudes < 2° or > 2.5 standard deviations of a participant- and task-specific mean (Gillen and Heath, 2014b). Less than 9% of trials for any participant were omitted. Trials involving a directional error (i.e., a prosaccade instead of an instructed antisaccade or vice versa) were excluded from the analyses of RT and saccade gain (see below) because they are associated

## Exercise and executive function

with planning mechanisms distinct from their directionally correct counterparts (Munoz and Everling, 2004).

Ventilatory and hemodynamic dependent variables included  $\text{O}_2$  consumption ( $\dot{V}\text{O}_2$ ),  $\text{CO}_2$  output ( $\dot{V}\text{CO}_2$ ), ventilation ( $\dot{V}_E$ ),  $\text{P}_{\text{ETCO}_2}$ , THC, HHb, BV and systolic (SP) and diastolic (DP) blood pressure. Mean values were determined via the last minute of rest (i.e., baseline) and for the last minute of each exercise condition (i.e., steady state) (**Table 2**).

The aforementioned variables were analyzed via 3 (exercise condition: light-, moderate-, heavy-intensity) by 2 (time: baseline, steady state) fully repeated measures ANOVA ( $\alpha = 0.05$ ).

Oculomotor dependent variables included RT (i.e., time from response cueing to saccade onset), percentage of directional errors (i.e., prosaccade instead of an instructed antisaccade or *vice versa*), and saccade gain variability (i.e., within-participant standard deviation of saccade amplitude/veridical target location). Oculomotor dependent variables were examined via 3 (exercise condition: light-, moderate-, heavy-intensity) by 2 (time: pre-, postexercise) by 2 (task: prosaccade, antisaccade) fully repeated measures ANOVA ( $\alpha = 0.05$ ). **Data met underlying assumptions for repeated measures ANOVA and did not violate sphericity (i.e., Mauchly's  $p > 0.05$ ). For all reported main effects and interactions, effect sizes were calculated and significant main effects involving exercise condition and all interactions were decomposed via paired-samples t-tests. Where appropriate, two one-sided test (TOST) statistics were used to determine whether means were within an equivalence boundary (Lakens, 2017) and we employed an adjusted alpha level ( $\alpha = 0.025$ ) as per Lauzon and Caffo's (2009) recommendation. Additionally, for  $\text{P}_{\text{ETCO}_2}$  data power-polynomials (i.e., trend analysis; for review see, Pedhazur, 1997) were used to describe the variable's time-dependent change across each unfolding exercise intensity. Last, Pearson  $r$  correlations between antisaccade RT**

## Exercise and executive function

difference scores and hemodynamic difference scores (i.e., post- minus preexercise) were performed to assess whether a putative postexercise reduction in antisaccade RT is related to changes in THC, HHb or BV ( $p<0.05$ ).

### 3. Results

#### 3.1 Ventilatory and Hemodynamic Measures

Ventilatory variables.  $\dot{V}O_2$ ,  $\dot{V}CO_2$  and  $\dot{V}_E$  produced main effects of exercise condition, all  $F(2,30)>26.11$ ,  $ps<0.001$ , all  $\eta^2>0.64$ , time, all  $F(1,15)>107.03$ ,  $ps<0.001$ , all  $\eta^2>0.88$ , and their interactions, all  $F(2,30)>33.75$ ,  $ps<0.001$ , all  $\eta^2>0.69$ . **Figure 1** demonstrates that  $\dot{V}O_2$ ,  $\dot{V}CO_2$  and  $\dot{V}_E$  increased from baseline during light-, moderate-, and heavy-intensity conditions and intensity-specific difference scores (i.e., steady state minus baseline) showed that values increased from light- to moderate-intensity and from moderate- to heavy-intensity (all  $t(15)>3.20$ ,  $ps<0.006$ , all  $d_z>0.80$ ).



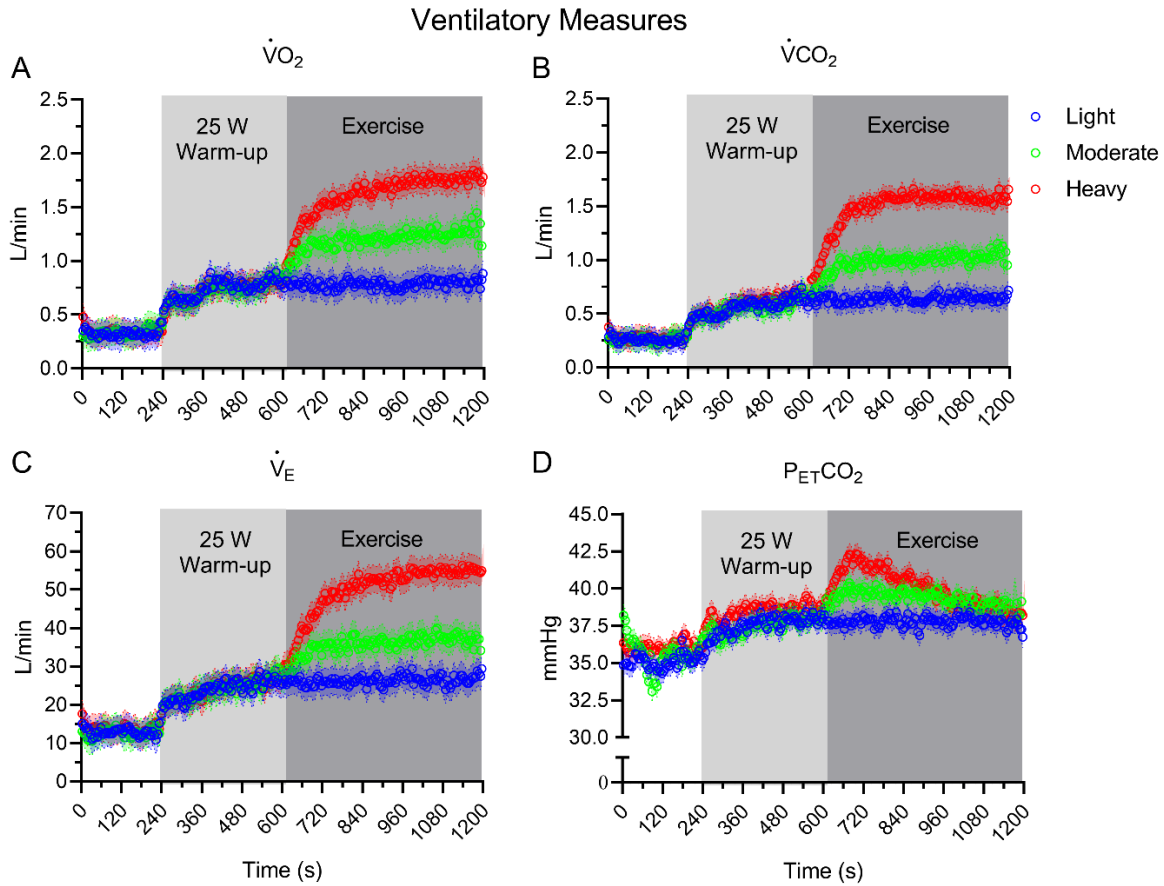


Fig 1. Group  $O_2$  consumption ( $\dot{V}O_2$ ) (**Panel A**),  $CO_2$  output ( $\dot{V}CO_2$ ) (**Panel B**), ventilation ( $\dot{V}_E$ ) (**Panel C**), and end-tidal  $CO_2$  ( $P_{ET}CO_2$ ) (**Panel D**) data for light-, moderate-, and heavy-intensity conditions at 5-s intervals with associated 95% between-participant confidence interval envelopes. The absence of between-condition overlap of error envelopes represents a reliable difference inclusive to a test of the null hypothesis. Light and dark grey rectangles depict the durations of a 25 W warm-up and exercise periods, respectively.

In other words,  $\dot{V}O_2$ ,  $\dot{V}CO_2$  and  $\dot{V}_E$  values increased with increasing exercise intensity.  $P_{ET}CO_2$  produced a main effect of time,  $F(1,15)=14.34$ ,  $p=0.002$ ,  $\eta^2=0.49$ , such that values increased from baseline to steady-state.  $P_{ET}CO_2$  did not elicit an exercise condition by time interaction,  $F(2,30)=1.63$ ,  $p=0.21$ ,  $\eta^2=0.10$ , indicating that the magnitude change of  $P_{ET}CO_2$  did not vary with exercise intensity. In considering  $P_{ET}CO_2$ , **Figure 1D** presents group means and associated 95% between-participant confidence envelopes for  $P_{ET}CO_2$  at decisecond increments of time

## Exercise and executive function

separately for each exercise intensity. In evaluating this figure, note that Cumming (2014) asserts that when the 95% confidence intervals associated with separate conditions do not overlap there is evidence to assert a reliable difference inclusive to a test of the null hypothesis, whereas overlap between condition-specific confidence intervals indicates the absence of a reliable between-condition difference. The figure shows that  $P_{ET}CO_2$  increased in relation to exercise intensity for approximately the first 2-min of exercise and that moderate- and heavy-condition gradually decreased to values commensurate with the light-intensity condition thereafter. Further, power-polynomials (i.e., trend analyses) were fit to  $P_{ET}CO_2$  values at successive 1-min intervals following exercise onset. The light-intensity condition was not associated with a reliable trend, all  $F(1,15) < 1.72$ ,  $p_s > 0.20$ ,  $\eta^2 = 0.10$ ; that is,  $P_{ET}CO_2$  did not vary with time. Moderate- and heavy-intensity conditions were best fit with cubic polynomials, all  $F(1, 15) = 10.01$  and  $27.92$ ,  $p_s < 0.01$ ,  $\eta^2 = 0.65$ , such that values increased during early exercise (i.e., up to 2-min), decreased during the middle phase of exercise (i.e., 3- to 6-min) and plateaued during the late phase of exercise (i.e., 7- to 10-min). Put more simply, moderate- and heavy-intensity  $P_{ET}CO_2$  elicited a time-dependent modulation.

Hemodynamic variables. THC did not elicit reliable main effects or interactions, all  $F(2,30) < 0.62$ ,  $p_s > 0.55$ , all  $\eta^2 < 0.04$ . HHb produced a main effect for time,  $F(1,15) = 60.65$ ,  $p < 0.001$ ,  $\eta^2 = 0.80$ : values decreased from baseline to steady-state; however, this change was not modulated by exercise intensity (i.e., null exercise condition by time interaction,  $F(2,30) = 2.10$ ,  $p = 0.14$ ,  $\eta^2 = 0.12$ ).

## Exercise and executive function

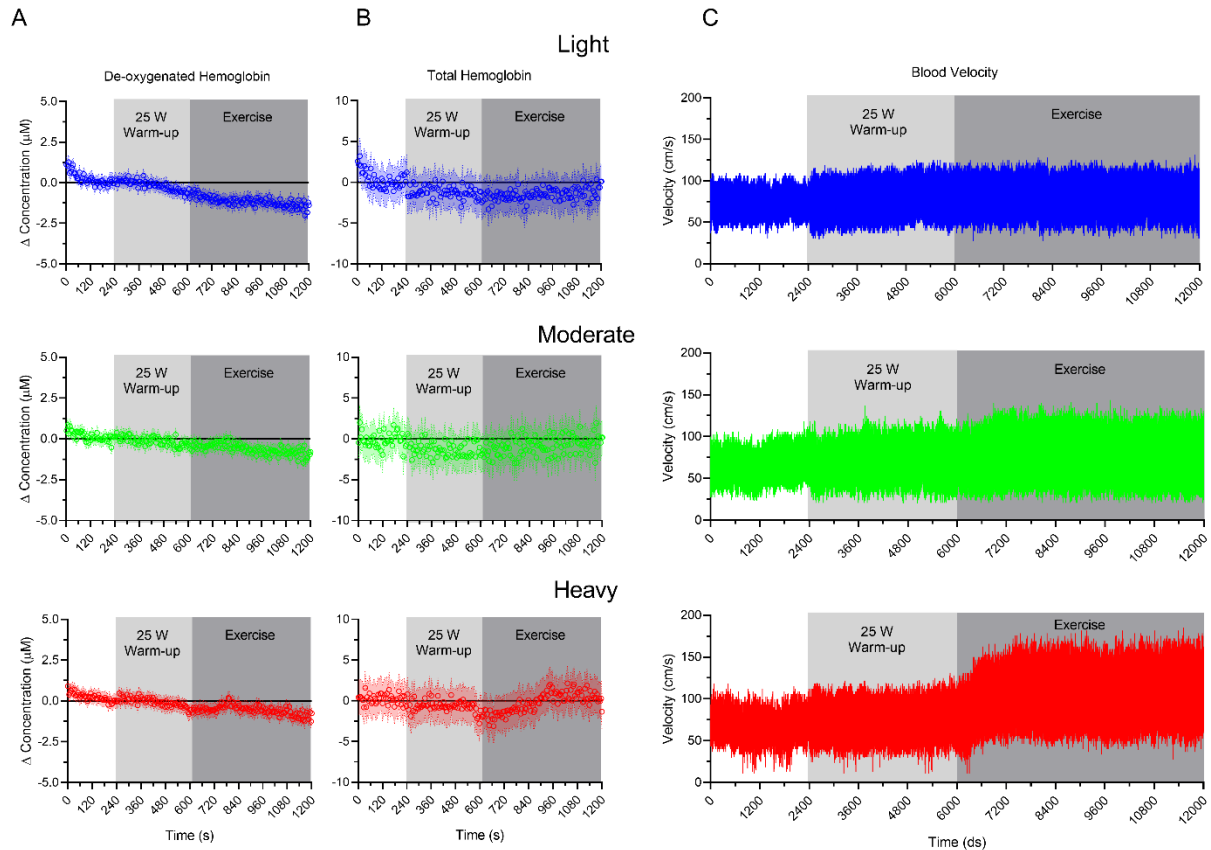


Fig 2. Normalized (i.e., zeroed to baseline) group average data for deoxygenated hemoglobin (HHb) (**Panel A**) and total hemoglobin (THC) concentrations (**Panel B**) via near-infrared spectroscopy (NIRS) presented as 5-s intervals with associated 95% between-participant confidence interval envelopes. **Panel C** depicts an exemplar participant's **blood velocity through** the middle cerebral artery via transcranial Doppler ultrasound (TCD). For all panels, light and dark grey rectangles illustrate the durations of a 25 W warm-up and exercise periods, respectively.

Table 2 demonstrates mean systolic BVs consistent with those documented by Bishop et al. (1986). BV produced main effects of exercise condition,  $F(2,30)=6.68$ ,  $p=0.004$ ,  $\eta^2=0.31$ , time,  $F(1,15)=481.73$ ,  $p<0.001$ ,  $\eta^2=0.97$ , and their interaction,  $F(2,30)=19.43$ ,  $p<0.001$ ,  $\eta^2=0.56$ . **Figure 2C** shows that BV increased from baseline to steady-state for all exercise conditions and intensity-specific difference scores (i.e., steady state minus baseline) indicated that the magnitude of this change increased from light- to moderate-intensity and from moderate- to heavy-intensity (all  $t(15)> -2.31$ ,  $ps<0.04$ , all  $d_z> -0.58$ ).

For blood pressure, DP produced a main effect of time,  $F(1,15)=12.93$ ,  $p=0.003$ ,  $\eta^2=0.46$ , showing an increase from baseline to steady state and this effect was not influenced by exercise intensity (i.e., null exercise condition by time interaction:  $F(2,30)=1.92$ ,  $p=0.16$ ,  $\eta^2=0.11$ ). SP produced main effects of exercise condition,  $F(2,30)=12.06$ ,  $p<0.001$ ,  $\eta^2=0.45$ , time,  $F(1,15)=96.10$ ,  $p<0.001$ ,  $\eta^2=0.87$ , and their interaction,  $F(2,30)=11.07$ ,  $p<0.001$ ,  $\eta^2=0.43$ . **Table 2** shows that SP increased from baseline to steady for all intensities; however, intensity-specific difference scores (i.e., steady state minus baseline) demonstrated that light- and moderate-intensity condition magnitudes **did not reliably differ** ( $t(15)=-1.84$ ,  $p=0.09$ ,  $d_z=-0.46$ ), and the former were less than the heavy-intensity condition (all  $t(15)>-2.97$ ,  $p_s<0.01$ , all  $d_z=-0.74$ ).

**Table 2. Mean baseline and steady state ventilatory and hemodynamic data for light, moderate and heavy-intensity exercise conditions.**

	Light		Moderate		Heavy	
	Baseline	Steady State	Baseline	Steady State	Baseline	Steady State
$\dot{V}O_2$ (L/min)	0.33 (0.07)	0.80* (0.20)	0.39 (0.17)	1.27* (0.55)	0.33 (0.10)	1.81* (0.55)
$\dot{V}CO_2$ (L/min)	0.26 (0.09)	0.64* (0.20)	0.27 (0.11)	1.03* (0.39)	0.27 (0.13)	1.59* (0.43)
$\dot{V}_E$ (L/min)	12.97 (3.60)	26.96* (5.47)	13.33 (3.39)	37.85* (10.64)	13.39 (4.10)	55.13* (13.92)
$P_{ET}CO_2$ (mmHg)	35.37 (3.76)	37.51* (3.67)	35.65 (4.97)	38.81* (5.43)	36.03 (5.11)	38.09* (6.18)
THC ( $\mu M$ )	49.20 (21.54)	47.78 (23.99)	50.22 (21.26)	50.01 (23.13)	50.69 (16.31)	51.51 (16.38)
HHb ( $\mu M$ )	11.44 (5.16)	9.85* (5.45)	11.88 (5.11)	10.97* (4.84)	12.19 (4.13)	11.41* (4.49)
BV (cm/s)	102.96 (11.58)	125.00* (13.74)	107.76 (16.15)	139.20* (20.22)	105.28 (13.70)	152.94* (18.25)
SP (mmHg)	116.94 (10.72)	127.00* (7.73)	119.63 (7.53)	136.81* (11.00)	117.50 (8.21)	147.56* (20.02)
DP (mmHg)	73.13 (7.48)	82.00* (6.57)	79.63 (11.78)	81.50* (6.63)	76.50 (7.75)	83.75* (11.02)

Note: Group mean data for  $O_2$  consumption ( $\dot{V}O_2$ ),  $CO_2$  output ( $\dot{V}CO_2$ ), ventilation ( $\dot{V}_E$ ), end tidal  $CO_2$  ( $P_{ET}CO_2$ ), total hemoglobin concentration (THC), deoxygenated hemoglobin (HHb), systolic blood velocity (BV), and systolic (SP) and diastolic (DP) blood pressure are presented with standard deviations in parentheses. Baseline corresponds to 180-240 s, and Steady State corresponds to 1140-1200 s during each condition. Steady State values marked with (\*) indicate a reliable difference ( $p<0.05$ ) from Baseline values.

## 3.2 Oculomotor Measures

**Reaction time.** Results produced main effects for exercise condition,  $F(2,30)=11.47$ ,  $p<0.001$ ,  $\eta^2=0.43$ , time,  $F(1,15)=7.45$ ,  $p=0.02$ ,  $\eta^2=0.32$ , and task  $F(1,15)=82.99$ ,  $p<0.001$ ,  $\eta^2=0.85$ , and a time by task interaction,  $F(1,15)=13.47$ ,  $p=0.002$ ,  $\eta^2=0.47$ . **Figure 3A** demonstrates that – in general – prosaccade RTs (215 ms,  $SD=34$ ) were shorter than antisaccade (286 ms,  $SD=42$ ) ( $t(15)= -9.11$ ,  $p<0.001$ ,  $d_z= -2.28$ ). In terms of the time by task interaction, prosaccade RTs were refractory to the exercise manipulation (all  $t(15)< -0.46$ ,  $ps>0.65$ , all  $d_z< -0.11$ ), whereas antisaccade RTs decreased from pre- to postexercise assessments ( $t(15)>2.94$ ,  $p<0.01$ , all  $d_z>0.74$ ).

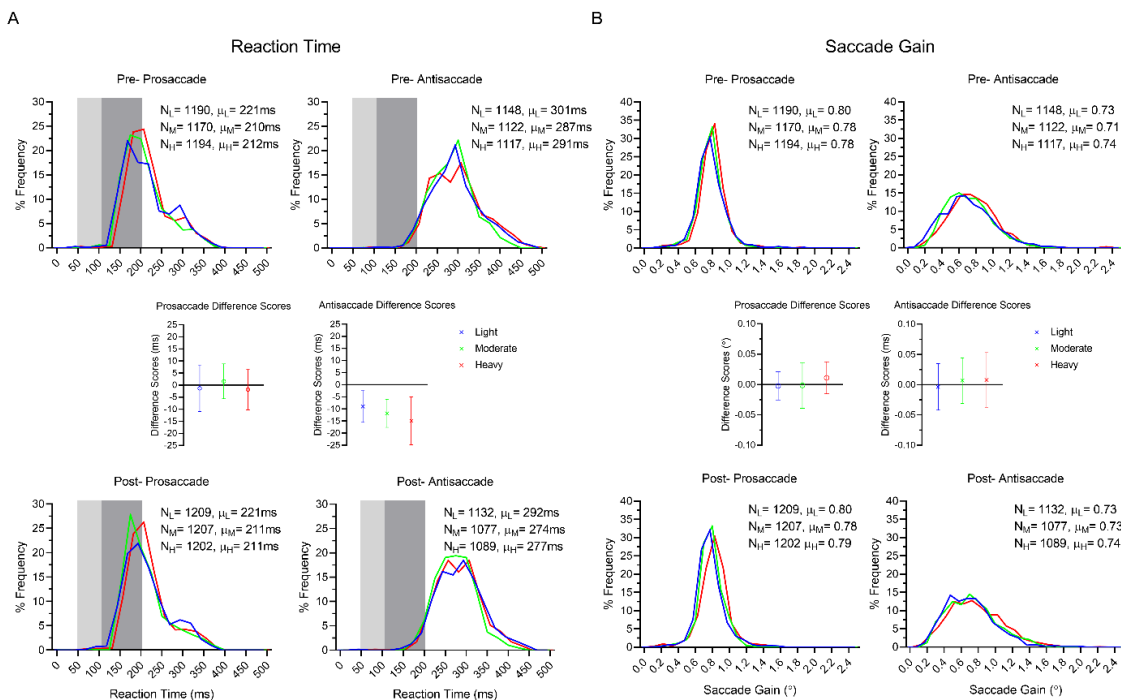


Fig 3. Pre- (top panels) and postexercise (bottom panels) pro- and antisaccade reaction time (RT) (**Panel A**) and saccade gain (**Panel B**) frequency distribution histograms for light- (L), moderate- (M), and heavy- (H) intensity exercise conditions. Light and dark grey rectangles in **Panel A** depict anticipatory saccades (i.e.,  $<100$  ms) and short-latency saccades (i.e.,  $100 < 200$  ms), respectively. Total number of trials (N) and mean ( $\mu$ ) RT and saccade gain values are reported for each condition. Inset panels show pro- and antisaccade group mean difference scores (i.e., post- minus pre-exercise) and associated 95% confidence intervals for RT and saccade gain.

## Exercise and executive function

Moreover, and given the nature of our primary research question, TOST statistics involving RT difference scores (i.e., post- minus pre-exercise) indicated that the **magnitude of the postexercise RT benefit was equivalent between the light- and moderate-intensity ( $t(15) = -2.43$ ,  $p=0.01$ ) and moderate- and heavy-intensity sessions ( $t(15) = -2.53$ ,  $p=0.01$ ), whereas the comparison between the light- and heavy-intensity session approached – but did not attain – a conventional level of statistical equivalence ( $t(15) = -2.05$ ,  $p=0.026$ )<sup>1</sup>.**

Directional errors and gain variability. Directional errors elicited a main effect for task,  $F(1,15)=18.60$ ,  $p=0.001$ ,  $\eta^2=0.55$ : prosaccades (2%,  $SD=2$ ) produced fewer errors than antisaccades (10%,  $SD=7$ ), and null task by time,  $F(1,15)=0.13$ ,  $p=0.72$ ,  $\eta^2=0.01$ , and exercise condition by task by time interactions,  $F(2,30)=0.46$ ,  $p=0.63$ ,  $\eta^2=0.03$ , indicated that pro- and antisaccade directional errors were not influenced by the exercise conditions used here. Results for saccade gain variability elicited a main effect of task,  $F(1,15)=33.31$ ,  $p<0.001$ ,  $\eta^2=0.69$ : prosaccade endpoints (0.12,  $SD=0.05$ ) were less variable than antisaccades (0.19,  $SD=0.06$ ) and this effect was not influenced by any higher-order interactions (i.e., null exercise condition by task by time interaction,  $F(2,30)=0.91$ ,  $p=0.41$ ,  $\eta^2=0.06$ ).

### *3.3 Correlation Between Antisaccade Difference Scores and Cortical Hemodynamics*

Pearson  $r$  correlations involving antisaccade RT difference scores at each exercise intensity and their associated baseline to steady-state changes in THC, HHb and BV were not statistically significant (all  $r(15) < -0.28$ ,  $ps > 0.30$ ); that is, the postexercise benefit to executive function did not vary with cortical hemodynamics.

## **4. Discussion**

Our results demonstrate that a postexercise benefit to executive function is not directly related to a change in CBF across light-, moderate- and heavy-intensity aerobic exercise. In outlining our

results, we first discuss intensity-specific ventilatory and hemodynamic changes before turning to their impact on postexercise executive function.

#### *4.1 Exercise Intensity-Specific Ventilation and Hemodynamic Changes*

$\dot{V}O_2$ ,  $\dot{V}CO_2$ ,  $\dot{V}_E$  and  $P_{ET}CO_2$  increased from baseline to steady state across all intensities and reflects an exercise-mediated change in venous  $CO_2$  concentrations associated with oxidative phosphorylation and/or ventilatory buffering (Ide and Secher, 2000; Thompson, 2010; Smith and Ainslie, 2017). Moreover, the magnitude of  $\dot{V}O_2$ ,  $\dot{V}CO_2$ , and  $\dot{V}_E$  baseline to steady state changes scaled with increasing intensity and demonstrates an adaptive response to the heightened metabolic demands of exercise at progressively heavier work rates (for review see, Ainslie and Duffin, 2009). In contrast, the magnitude of the  $P_{ET}CO_2$  baseline to steady-state change did not vary with intensity; however, **Figure 1D** shows that this reflects a time-dependent response for moderate- and heavy-intensities. In particular, intensity-specific scaling (i.e., light < moderate < heavy) was observed for the first 2-min of exercise and thereafter  $P_{ET}CO_2$  for moderate- and heavy-intensity conditions gradually diminished to values commensurate with light-intensity. This time-dependent decrease modulation has been attributed to moderate and heavy work rates stimulating peripheral chemoreceptor activity to increase ventilation to gradually reduce  $PaCO_2$ , and concomitantly  $P_{ET}CO_2$  during exercise (Subudhi et al., 2008).

THC did not vary from baseline to steady-state for any exercise intensity suggesting that the microvasculature in the area of investigation under the NIRS probe did not undergo observable increases in vessel diameter that would accommodate increased THC (for reviews see, Ainslie and Duffin, 2009; Hoiland et al., 2019). In contrast, BV and HHb increased and decreased, respectively, from baseline to steady state across all exercise intensities. The BV and HHb results reflect increased volumetric CBF (Hoiland et al., 2019) and  $O_2$  delivery from



baseline (for review see, Ainslie and Duffin, 2009; Hoiland et al., 2019; see also Kety and Schmidt, 1945) and is a well-defined consequence of exercise (Smith and Ainslie, 2017). In addition, BV – but not HHb – elicited an intensity-specific modulation such that values increased across the light- through heavy-intensity conditions. That HHb did not vary with intensity indicates that the metabolic demand for O<sub>2</sub> during exercise was exceeded by the rate of CBF (Ide and Secher, 2000). The intensity-specific scaling of CBF in relation to increasing exercise-intensity is consistent with literature examining similar exercise work rates (Smith and Ainslie, 2017) and is consistent with drive theories of CBF (Chang et al., 2012). Thus, the present findings add to the literature inasmuch as they demonstrate that CBF elicits an intensity-specific scaling across the LT-based moderate- and heavy-intensity conditions used here. Moreover, and regardless of the mechanism(s) associated with the change in CBF, the results support a framework to evaluate whether an intensity-specific change in CBF differentially influences a postexercise benefit to executive function.

#### *4.2 Oculomotor control: a single bout of exercise selectively benefits executive function*

Prosaccades produced shorter RTs, fewer directional errors and less variable endpoints than antisaccades. The prosaccade findings reflect their mediation via direct retinotopic motor maps in the superior colliculus (Wurtz and Albano, 1980) that operate with minimal top-down executive control (Pierrot-Deseilligny et al., 1995). In turn, the longer RTs, increased directional errors and endpoint variability of antisaccades reflect the demands of evoking a non-standard response (i.e., decoupling stimulus-response relations) via the activation of an extensive executive frontoparietal network (Munoz and Everling, 2004). What is more, antisaccades – but not prosaccades – were associated with a large magnitude postexercise RT reduction (average  $d_z=0.87$ ) that was independent of a pre- to postexercise modulation in directional errors or



## Exercise and executive function

endpoint variability – a result indicating that the postexercise reduction in antisaccade RT was unrelated to an implicit or explicit strategy designed to decrease planning time at the cost of increased response errors. Instead, results accord a growing literature indicating that a single bout of exercise selectively benefits an oculomotor task requiring top-down executive control (Samani and Heath, 2018; Heath et al., 2018; Petrella et al., 2019; Shukla et al., 2020; Tari et al., 2020; Shukla and Heath, 2021).

The light-, moderate- and heavy-intensity conditions produced an equivalent magnitude reduction in antisaccade RTs and is a result supported via null hypothesis and equivalence (i.e., TOST) testing. Although this result is inconsistent with drive-theories and/or work reporting that moderate- or heavy-intensity exercise provides the largest postexercise executive benefit (Chang et al., 2012) a candidate reason for this discrepancy is that previous work employed an absolute taxonomy for intensity classification (i.e.,  $HR_{max}$ ) or one that does not linearly increase with increasing power output (i.e.,  $\dot{V}O_{2peak/max}$ ) (Keir et al., 2016). Indeed, previous work by our group which used a LT-based framework for exercise intensity reported that young (Heath et al., 2018) and healthy older (Petrella et al., 2019) adults exhibit a comparable executive benefit across moderate (80% of LT), heavy (15% of the difference between LT and  $VO_{2peak}$ ) and very-heavy (50% of the difference between LT and  $VO_{2peak}$ ) intensities. Moreover, the most salient contribution of this work is the observation that the comparable postexercise executive function benefit was observed in spite of the fact that CBF increased with increasing exercise intensity. In addition, correlation analyses showed that antisaccade RT difference scores (i.e., post- minus pre-exercise) did not reliably relate to changes in CBF. Accordingly, a postexercise executive benefit does not scale in relation to an exercise intensity-specific change in CBF.

It is worth noting that evidence supporting an association between CBF and executive function remains mixed. For example, Shoemaker et al. (2020) reported that an increase in BV through the MCA was not associated with a postexercise improvement in executive function as assessed via a modified version of the pro- and antipointing paradigm (for outline of exemplar antipointing task see, Heath et al., 2009, 2012). In reconciling this discrepancy, we note that Shoemaker et al.'s postexercise assessment occurred immediately following a severe bout of exercise (i.e., >80% of heart rate reserve) that included a hypercapnic environment. As demonstrated in previous work, a single bout of severe intensity exercise does not reliably elicit a postexercise benefit to executive function due to the physiological and psychological costs of recovering from such a work rate (Sudo et al., 2017). As well, the present results do not exclude a possible role for CBF in a postexercise executive function benefit. Indeed, a 1% increase in functional hyperemia identified in sensory regions (i.e., primary somatosensory and visual cortices) has been shown to enhance stimulus detection times via mechanical- and temperature-based changes to local neural circuits (Drevets et al., 1995; McMains and Somers, 2004; Moore and Cao, 2008). In the present work, the light-, moderate- and heavy-intensity conditions increased BV from baseline by 22%, 30% and 46%, respectively. Thus, and in contrast to drive theories, it may be that only a small change in CBF is necessary to improve executive function postexercise and that intensity-specific modulations do not impart additive CBF-executive function benefits.

#### *4.3 Limitations and Future Directions*

We recognize that our study is limited by several methodological traits. First, we did not employ a non-exercise control condition to examine whether the pre- to postexercise reduction in antisaccade RTs might be attributed to a practice-related benefit. In addressing this issue, we

note that all previous studies by our group have shown that an exercise – but not control – condition elicits a post-condition benefit to antisaccade RTs (Samani and Heath, 2018; Heath et al., 2018; Petrella et al., 2019; Shukla et al., 2020; Tari et al., 2020; Shukla and Heath, 2021). Second, we examined executive function prior to and immediately following a 10-min single bout of aerobic exercise across light-, moderate-, and heavy-intensities. It is therefore unclear whether the increased CBF associated with moderate- and heavy-intensity provide a benefit that is temporally more persistent than the light-intensity condition. As well, it is unclear whether increasing the duration of the exercise interval would influence the extent to which an intensity-specific change in CBF improves executive function. Third, our results are specific to young healthy adults and cannot be extended to populations (i.e., older adults, individuals with neuropsychiatric disease, and/or individuals classified as low- or high-fit) that may demonstrate distinct reactivity to exercise (Chang et al., 2012; Ludyga et al., 2016). To address this issue, our group is currently examining the relationship between CBF and executive function in older adults experiencing cognitive decline and individuals with first episode psychosis. Fourth, a change in BV measured by TCD does not quantify vessel diameter. This is a potential limitation because the MCA is capable of dilation and constriction under certain physiological conditions (Coverdale et al., 2015). We therefore cannot directly assert a quantitative measure of CBF from the TCD data acquired here (Hoiland et al., 2019); however, that BV and HHb increased and decreased, respectively, during exercise indicates an increase in volumetric flow (for review see, Ainslie and Duffin, 2009; Hoiland et al., 2019). Last, a change in CBF combined with increased brain-derived neurotrophic factor (Knaepen et al., 2010) and catecholamine (Zouhal et al., 2008) concentrations coupled with enhanced frontoparietal resting state functional connectivity (Voss et al., 2010) has been linked to improved single bout postexercise executive function. It is

## Exercise and executive function

therefore possible that intensity-specific changes in the aforementioned variables across distinct participant groups, exercise durations and postexercise assessment intervals may differentially influence the magnitude of a postexercise executive function benefit.

### *4.4 Conclusion*

Our results provide convergent evidence that a 10-min single bout of exercise provides an immediate postexercise benefit to executive function. What is more, our findings demonstrate that the benefit is independent of exercise intensity-specific changes to CBF.

Exercise and executive function

## 5. Funding

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## 6. Disclosure Statement

The authors have no conflicts of interest to disclose.

## 7. Footnote

1. JASP (JASP Team, 2020) was used to compute Bayesian paired-samples t-tests (Cauchy=0.707) for between-intensity contrasts of antisaccade RT difference scores (i.e., post- minus pre-exercise). This approach was used as a supplement to TOST statistics because: (1) Bayesian statistics identify the hypothesis (i.e., null or alternative) that best predicts the data, and (2) Bayesian paired-samples t-tests provide a correction factor for multiple comparisons (see Westfall et al., 1997). Results produced Bayes factors ( $BF_{01}$ ) indicating that the null hypothesis for light- versus moderate-intensity, moderate- versus heavy-intensity, and light- versus heavy-intensity predicted the data 2.695, 3.084, and 1.583 times better than the alternative hypothesis and a robustness check indicated that the Bayes factors were robust to changes in the prior width. Hence, Bayesian and TOST procedures provide a basis to assert equivalent postexercise executive function across each exercise intensity.

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