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SPORT AND EXERCISE PSYCHOLOGY

Distinct cortical haemodynamics during squat-stand and continuous aerobic exercise do not influence the magnitude of a postexercise executive function benefit

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ABSTRACT

A single bout of aerobic exercise benefits executive function (EF). A potential mechanism for this benefit is an exercise-mediated increase in cerebral blood flow (CBF) that elicits vascular endothelial shear-stress improving EF efficiency. Moderate intensity continuous aerobic exercise (MCE) asymptotically increases CBF, whereas continuous body weight squat-stand exercise (SSE) provides a large amplitude oscillatory response. Some work has proposed that an increase in CBF oscillation amplitude provides the optimal shear-stress for improving EF and brain health. We examined whether a large amplitude oscillatory CBF response associated with a single bout of SSE imparts a larger postexercise EF benefit than an MCE cycle ergometer protocol. Exercise changes in middle cerebral artery velocity (MCAv) were measured via transcranial Doppler ultrasound to estimate CBF, and pre- and postexercise EF was assessed via the antisaccade task. MCE produced a steady state increase in MCAv, whereas SSE produced a large amplitude MCAv oscillation. Both conditions produced a postexercise EF benefit that null hypothesis and equivalence tests showed to be comparable in magnitude. Accordingly, we provide a first demonstration that a single bout of SSE benefits EF; however, the condition's oscillatory CBF response does not impart a larger benefit than a time- and intensity-matched MCE protocol.

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KEYWORDS
Antisaccades; inhibitory control; middle cerebral artery velocity; shear-stress; transcranial Doppler ultrasound

Introduction

Executive function (EF) includes the core components of inhibitory control, working memory and cognitive flexibility and is a construct critical for activities of daily living (for review see, Diamond, 2013). Mounting evidence has reported that a single bout of aerobic and/or resistance exercise benefits EF (for meta-analyses, see Chang et al., 2012; Ishihara et al., 2021; Lambourne & Tomporowski, 2010; Ludyga et al., 2016) for up to 60 min postexercise (Hung et al., 2013, Shukla & Heath, 2022; Joyce et al., 2009). A candidate mechanism for this benefit is an exercise-mediated increase in cerebral blood flow (CBF). In particular, an increase in arterial carbon dioxide ($PaCO_2$) concentration arising from volitional muscle activation produces vascular deformation and an associated increase in CBF (Smith & Ainslie, 2017) that is thought to improve the efficiency of the local neural circuits supporting EF (i.e., the haemo-neural hypothesis) (Moore & Cao, 2008).

In demonstrating the role of CBF in mediating a postexercise EF benefit, Tari et al. (2020) measured middle cerebral artery velocity (MCAv) via transcranial Doppler ultrasound (TCD) to estimate CBF in healthy young adults in conditions involving (1) a 10 min single bout of moderate-to-heavy intensity aerobic exercise (via cycle ergometer) and (2) a 10 min non-exercise condition involving the inhalation of a higher-than-atmospheric concentration of CO_2 (i.e., hypercapnic environment). The hypercapnic condition was used because it increases CBF in response to elevated arterial $PaCO_2$ and reduced pH (Ainslie & Duffin, 2009; Hoiland et al., 2019). Tari et al. reported that exercise and hypercapnic conditions increased MCAv from baseline (~19%) and both conditions produced a 5% post-intervention EF benefit. Additionally, Shirzad et al. (2022) had healthy young adults complete a traditional "active" exercise condition (i.e., volitional pedalling of cycle ergometer at a light intensity of 37 W) and a "passive" exercise condition wherein the cycle ergometer flywheel was mechanically driven and did not require volitional muscle activation. Passive exercise was used because it stimulates type *III* mechanoreceptor afferent feedback to primary somatosensory and motor cortices that increase cardiac output, stroke volume and subsequently CBF (Nóbrega & Araujo, 1993). Shirzad et al. reported that active (25 cm/s, SD = 13) and passive (7 cm/s, SD = 7) exercise increased MCAv – albeit the magnitude was larger in the former condition – and both produced an equivalent magnitude (5%) postexercise EF benefit. Accordingly, the authors proposed that an increase in CBF independent of the metabolic costs and intensity demands of active exercise produce a reliable EF benefit. It is, however, important to note that not all work has reported a reliable relationship between CBF and EF. For example, Ogoh et al. (2014) reported that although healthy young adults showed improved EF when assessed *concurrently* with a 50-min exercise intervention such a benefit was not linked to an exercise-mediated increase in CBF. Thus, there remains some debate as to whether an increase in CBF represents a primary moderator of an exercise-mediated EF benefit.

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The majority of work examining a postexercise EF benefit and its putative relationship with increased CBF employed continuous aerobic exercise (i.e., treadmill running and cycle ergometry) (see Table 1 of meta-analysis by Ludyga et al., 2016). To our knowledge, no work has investigated whether continuous body-weight squat-stand exercise (SSE) promotes a similar – or potentially larger – postexercise EF benefit. This is a salient question because the steady state increase in CBF across continuous light to moderate intensity aerobic exercise (Ogoh & Ainslie, 2009) elicits a relatively constant elevation in mechanical shear-stress to the vascular endothelium – a stressor increasing biomolecule levels (e.g., nitric oxide: NO) that promote cerebrovascular function (Lucas et al., 2015), spatial navigation, memory and EF (Pertiwi et al., 2015). In turn, SSE elicits a distinct CBF response such that squatting produces a transient and large amplitude increase in CBF owing to a rapid rise in blood pressure and increase in cardiac output due to increased venous return from the lower limbs (Krediet et al., 2005). In contrast, standing produces a transient and large amplitude decrease in CBF due to a reduction in vascular resistance and cardiac output from a rapid shift in blood volume to the lower limbs (Wieling et al., 2007). When performed continuously, SSE exposes the vascular endothelium to a larger amplitude oscillatory shear-stress than continuous light to moderate intensity aerobic exercise (Harrison et al., 2006; Kuebler et al., 2003). Indeed, research involving a mouse model analogue for a chronic SSE protocol (Adami et al., 2018) reported that it provides an optimal intervention for cell proliferation within cognitive brain regions that may – in part – be supported via the increased bioavailability of NO. Notably, NO is an unconventional neurotransmitter because it is not stored in synaptic vesicles; rather, it is released for uptake upon synthesis and its release has been linked to improved neural function (for extensive review see, Paul & Ekambaram, 2011). Moreover, because NO bioavailability has been shown to be increased as a result of a shear-stress response arising from a 10- or 30-min single bout of aerobic exercise (e.g., Matsumoto et al., 1994) it has been proposed that the larger amplitude SSE oscillatory CBF response may provide the optimal short- and long-term training paradigm for brain health, cognition and EF (Larbi, 2021; Roberts, 2018). To our knowledge, however, no work has demonstrated whether the documented large amplitude oscillatory CBF response during SSE provides a putative single bout postexercise EF benefit and/or whether such a benefit is larger in magnitude than an MCE protocol.

Table 1. Means and standard deviations for baseline and steady state (SS) physiological and performance variables as a function of control, moderate continuous exercise (MCE) and squat-stand exercise (SSE) conditions.

	Control		MCE		SSE	
	Baseline	SS	Baseline	SS	Baseline	SS
HR	75 ± 8	75 ± 9	77 ± 9	148 ± 4	76 ± 8	146 ± 14
SBP	114 ± 12	114 ± 12	115 ± 9	135 ± 16	113 ± 12	134 ± 15
DBP	72 ± 7	73 ± 6	73 ± 6	82 ± 7	71 ± 8	82 ± 11
$MCAv_{sys}$	94 ± 16	94 ± 15	89 ± 18	128 ± 27	98 ± 19	125 ± 21
$MCAv_{\mu}$	59 ± 13	58 ± 11	56 ± 12	66 ± 14	59 ± 13	69 ± 11
PI	0.96 ± 0.17	0.97 ± 0.17	0.93 ± 0.15	1.39 ± 0.23	1.02 ± 0.16	1.28 ± 0.16
RPE	-	-	-	13 ± 1.2	-	13 ± 1.1
W	-	-	-	100 ± 35	-	-

Note: Heart rate (HR), systolic (SBP) and diastolic (DBP) blood pressure, peak systolic middle cerebral artery velocity ($MCAv_{sys}$), average middle cerebral artery velocity ($MCAv_{\mu}$), pulsatility index (PI) and Rating of Perceived Exertion (RPE). For the MCE condition, steady state power output (in watts: W) is reported..

The present work sought to determine whether a single bout of SSE provides an immediate postexercise EF benefit and whether the oscillatory CBF response associated with this protocol elicits an EF benefit distinct from moderate continuous aerobic exercise (MCE). Healthy young adults completed – on separate days – 15 min of MCE (i.e., 60% of heart rate reserve: HRR) and SSE, as well as a non-exercise control condition. During all conditions, MCAv was measured and EF was assessed prior to and immediately following each condition via the pro- and antisaccade task. Prosaccades require a saccade to the veridical location of an exogenously presented target and are largely implemented independent of EF networks via retinotopic projections to the superior colliculus (Wurtz & Albano, 1980). Work has shown that a single bout of exercise does not result in a decrease in prosaccade reaction times (RT) (for systematic review see, Zou et al., 2023) and is a result consistent with the view that a single bout of exercise does not elicit a general improvement to information processing (Chang et al., 2012). As well, there is no documented evidence from humans that an exercise-mediated increase in CBF – or any other exercise-mediated physiological change – influences the efficiency and effectiveness of downstream brain stem structures (e.g., superior colliculus). In contrast, antisaccades require a response mirror-symmetrical to a target and result in longer RTs (Hallett, 1978) and less accurate and more variable endpoints (Dafoe et al., 2007; Gillen & Heath, 2014) than their prosaccade counterparts. Human lesion and neuroimaging work, as well as non-human primate electrophysiology and transient cooling studies, have reported that antisaccade behavioural "costs" reflect the top-down EF components of inhibitory control and vector inversion (i.e., 180° spatial transposition of target location) (for reviews see Everling & Johnston, 2013; Munoz & Everling, 2004). As well, antisaccades show a single bout postexercise benefit (e.g., Shirzad et al., 2022, Tari et al. 2020; see also; Zou et al., 2023) and the EF networks supporting antisaccades are the same as those showing task-dependent changes following single bout and chronic exercise (Colcombe et al., 2004; Herold et al., 2020). Thus, the pro- and antisaccade responses used here provide a framework for identifying whether single bouts of MCE and SSE elicit a general information processing benefit (i.e., decreased pro- and antisaccade RTs) or a selective EF benefit (i.e., decreased antisaccade – but not prosaccade – RTs). In terms of research predictions, if a large amplitude oscillatory change in CBF imparts the physiological response optimizing an EF benefit, then the magnitude of a pre- to postexercise reduction in antisaccade RTs should be larger in the SSE than MCE condition. In turn, if the oscillatory CBF response associated with SSE does not mediate the magnitude of a single

bout postexercise EF benefit, then a postexercise reduction in antisaccade RTs should not reliably differ between SSE and MCE conditions.

Method

Participants

Twenty-two participants (10 female) with an average age of 23.4 years (SD = 2.6, range: 20–26 years) were recruited from the University of Western Ontario community. Sample-size was determined *a priori* using G*Power (ANOVA: Repeated measures, within factors) via an effect size derived from a previous study (Tari et al., 2023) examining control and exercise condition pre- to post-intervention pro- and antisaccade RT changes ($\alpha = 0.05$, power = 0.90, $\eta_p^2 = 0.32$). Participants self-reported being right-handed (i.e., "What hand do you write with?") with normal or corrected-to-normal vision, no history of smoking, cardiorespiratory disease, neurological/neuropsychiatric disease (including concussion) and reported not taking medication influencing cardiovascular, haemodynamic, or metabolic responses to exercise. Participants were requested to refrain from alcohol, caffeine and recreational drugs 12 hours prior to each session and were requested to get 8 hours of sleep the night before each session – all participants reported adhering to these requests. Data collection occurred between 9:00 am and 11:00 am in a hydrated state (i.e., ~555 ml of water consumed 30-min in advance of data collection) and participants were requested to eat a standard breakfast (e.g., bagel with banana or oatmeal with apple). Participants read a letter of information and signed a consent form approved by the Health Sciences Research Ethics Board, University of Western Ontario (HSREB #120489). The study was conducted in accord with the most recent Declaration of Helsinki with the exception that participants were not entered into a database.

Participants obtained a full score on the 2020 Physical Activity Readiness Questionnaire (PAR-Q+) (Warburton et al., 2011) indicating their ability to complete an exercise intervention and completed the Godin Leisure-Time Exercise Questionnaire (GLTEQ) (Godin, 2011). The group mean GLTEQ score was 56 (SD = 23, range = 21–110) indicating that all participants were recreationally active. Participants sat for approximately 15 min while they signed the consent letter and then completed the PAR-Q+ and GLTEQ.

Apparatus and procedure

Three conditions (MCE, SSE and control) were completed in random order on different days and separated by at least 48 hours. Once the PAR-Q+ and GLTEQ were completed, participants remained seated and were fitted with a TCD probe (Neurovision 500 M, Neurovision TOC2M; Multigon Industries, Elmsford, CA) coated in an aqueous ultrasound gel (Aquasonic Clear, Parker Laboratories Inc., Fairfield, NJ) and secured via headband to their right anterior temporal window to measure MCAv. A heart rate (HR) monitor (Polar Electro T34; Polar Electro Oy, Kempele, Finland) was secured to participants' chest and an automatic sphygmomanometer (Omron Blood Pressure Monitor, Omron Healthcare Inc., Lake Forrest, IL) was secured to their left arm to obtain blood pressure (BP). All conditions were preceded by a 5 min baseline wherein physiological variables (i.e., MCAv, HR and BP) were measured (see Figure 1 for timeline). In the MCE condition, participants sat on a cycle ergometer (Monark 818E Ergometer, Monark Exercise AB, Vansbro, Sweden) so that their legs achieved approximately 85% of full extension at the end of a pedal stroke. Once positioned, participants completed a 2.5 min warm-up at 30% of their HRR after which they exercised for 15 min at 60% of HRR at a metronome-paced cadence of 70 rpm. The duration and intensity were selected based on previous work showing that such a protocol provides a reliable postexercise EF benefit (e.g., Heath et al., 2018; Johnson et al., 2016; Samani & Heath, 2018). During this time, HR and pedal resistance were monitored by the experimenter to ensure a constant prescribed work rate. Subsequently, a cool-down was initiated for the same duration and intensity as the warm-up. For the SSE condition, participants first sat on the cycle ergometer and completed the same warm-up as the MCE condition; however, after the warm-up participants stepped down from the ergometer and completed intervals involving 1 min of continuous body weight, and depth-controlled, squat-stand manoeuvres at metronome paced frequency of one squat per 3 s (i.e., 0.33 Hz) followed by 1 min of rest (i.e., in upright stance). As such, a total of eight 1 min intervals of squat-stand were completed in the SSE condition. The SSE protocol was developed from pilot testing that manipulated the duration, frequency and number of SSE intervals, as well as the length of SSE rest intervals. The pilot testing was conducted to identify the protocol providing a HRR *and* Borg rating of perceived exertion (RPE) consistent with the MCE condition (i.e., 60% of HRR). In advance of data collection, the experimenter modelled the proper squat-stand procedure, that is, ~90° of knee flexion at the bottom (i.e., the squat position) and 180° of knee extension at the top (i.e., the stand position) of each cycle (Figure 1). Once the SSE intervention was completed, participants undertook the same cycle ergometer cool-down as the MCE condition. For the control condition, participants sat on the cycle ergometer for 20 min without exercising and engaged in casual conversation with the experimenters. For all conditions, TCD and HR data were continuously recorded, and BP was measured immediately following exercise cessation. At the end of MCE and SSE conditions, participants rated their perceived exercise intensity via the Borg RPE (scale: 6–20).

Executive function assessment

Oculomotor EF assessments were completed prior to (i.e., pre-intervention) and 5 min after (i.e., post-intervention) each condition (Figure 1). The 5 min post-intervention assessment was used based on previous work showing that it represents the timepoint wherein HR is less than 100 bpm (Dirk et al., 2020; Samani & Heath, 2018). For each assessment, participants sat on a height-adjustable chair in front of a table on which an LCD monitor (60 Hz, 8-ms response rate, 1280 × 960 pixels; Dell 3007WFP, Round Rock, TX) was located 550 mm from the table's front edge.

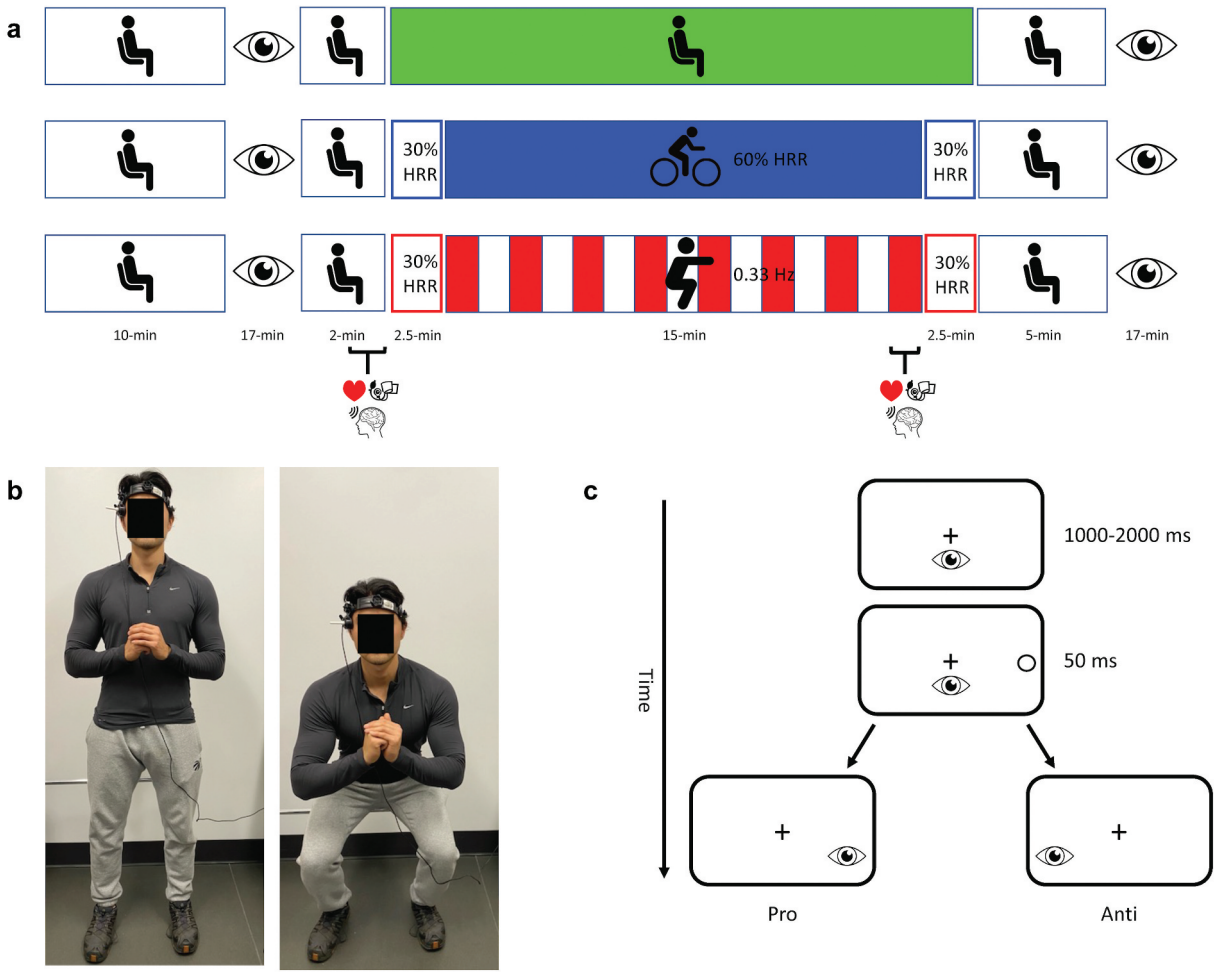

Figure 1. **Panel A** shows a timeline of experimental events in control (green), moderate continuous aerobic exercise (MCE: blue) and squat-stand exercise (SSE: hatched white and red) conditions. The eye icon represents when oculomotor assessments were completed (i.e., prior to and following each condition). The seated icon represents baseline and the capture of resting state heart rate (HR: i.e., icon of heart) blood pressure (BP: i.e., icon of blood pressure cuff) and transcranial Doppler ultrasound (TCD: i.e., icon of head/brain). Following baseline, the MCE and SSE conditions entailed a 2.5 min warm-up (i.e., 30% of heart rate reserve: HRR) followed by 15 min exercise interventions and a 2.5 min cool-down (i.e., 30% of HRR), whereas for the control condition participants remained in a seated position. During the last minute of all interventions (i.e., steady state), HR, BP and TCD were collected to provide a measure of steady state performance. **Panel B** shows the squat and stand manoeuvres used here and **panel C** shows a schematic of the timing of visual- and movement-related events in the prosaccade (i.e., pro) and antisaccade (i.e., anti) tasks.

Participants placed their head in a head/chinrest and the gaze location of their left eye was tracked via a video-based eye tracking system (EyeLink 1000 Plus; SR Research, Ottawa, ON, Canada) sampling at 1000 Hz. Prior to data collection, a 9-point calibration and validation of the viewing space was completed (i.e., <1° of error). All experimental events were controlled via MATLAB (R2018a; The MathWorks, Natick, MA, USA) and the Psychophysics Toolbox extension (v. 3.0) (Brainard, 1997; 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 were presented on a black screen (0.1 cd/m^2) and included a 1° midline-located white fixation cross (127 cd/m^2) presented at participants' eye level and targets (i.e., open white circle; 2.5° in diameter: 127 cd/m^2) 15° (i.e., proximal target) and 20° (i.e., distal target) to the left and right of fixation and in the same horizontal plane. Fixation onset signalled participants to direct their gaze to its location. Once a stable gaze was achieved (i.e., ±1.5° for 450 ms) a uniformly distributed randomized foreperiod (1000–2000 ms) was introduced after which a target appeared 200 ms thereafter and remained visible for 50 ms (i.e., overlap paradigm). Target onset cued participants to saccade to veridical (i.e., prosaccade) or mirror-symmetrical (i.e., antisaccade) target location (Figure 1). For each pre- and post-intervention assessment, separate and randomly ordered blocks of pro- and antisaccade trials were completed. Prior to a block, an instruction screen was presented indicating the nature of the to-be-completed task-type (i.e., pro- or antisaccade) with each block including 20 trials pseudo-randomly ordered as a function of target location (i.e., left and right visual field) and target eccentricity (i.e., proximal and distal) (i.e., 80 trials/block).

Data reduction, dependent variables and statistical analyses

TCD data corrupted by signal aliasing and/or signal loss (e.g., a sudden head shift) were omitted (Terslev et al., 2017) and peak systolic ($MCAv_{sys}$), peak diastolic ($MCAv_{dia}$) and mean ($MCAv_{\mu}$) MCAv were retained for analysis (Clyde et al., 1996).

Notably, previous work reported that $MCAv_{sys}$ provides a valid TCD-based measure for an exercise-based change in CBF (Willie et al., 2011). To provide an index of cerebrovascular resistance, Gosling's Pulsatility Index (PI) was calculated as: PI = ($MCAv_{sys}$ – $MCAv_{dia}$)/$MCAv_{\mu}$ (Beasley et al., 1979; Zuj et al., 2012). The PI index was measured because a single bout of resistance exercise increases carotid artery stiffness for up to 30 min postexercise; however, this increase has not been shown to influence MCAv resistance (Lefferts et al., 2014). As such, PI, $MCAv_{sys}$ and $MCAv_{\mu}$ provided a basis to document SSE and MCE baseline to steady state CBF resistance and/or flow changes compared to the control condition. Moreover, because oscillation amplitude increases endothelial shear-stress (Davies 2008), we computed participant-specific changes in MCAv amplitude for the SSE and MCE conditions. For the analysis of oscillation amplitude, MCAv were subjected to a dual-pass Butterworth filter with a passband of 0.1 to 1 Hz, and an average peak to trough difference was computed during steady state. $MCAv_{sys}$, $MCAv_{dia}$, $MCAv_{\mu}$, PI, HR and BP were computed from the last minute of baseline and the last minute of each intervention (i.e., steady state) (Figure 1) and examined via 3 (condition: control, MCE, SSE) by 2 (time: baseline, steady state) fully repeated measures ANOVAs ($\alpha = 0.05$). In turn, steady state MCAv oscillation amplitude for SSE and MCE conditions was contrasted via paired-samples t-tests.

For the oculomotor task, gaze position data were filtered offline using a dual-pass Butterworth filter employing a low-pass cut-off frequency of 15 Hz. A 5-point central-finite difference algorithm was used to compute instantaneous velocities and accelerations. Saccade onset was determined when velocity and acceleration exceeded 30°/s and 8,000°/s^2, respectively. Saccade offset was determined when velocity fell below 30°/s for 40 ms. Trials involving a signal loss (e.g., an eye blink) were removed as were anticipatory responses (RTs <50 ms) (Wenban-Smith & Findlay, 1991) and RTs >2.5 standard deviations of a participant- and task-specific mean (Gillen & Heath, 2014) (i.e., <8% of total trials). Trials involving a directional error (i.e., a prosaccade instead of an instructed antisaccade and *vice versa*) were excluded because they are associated with planning mechanisms distinct from their directionally correct counterparts (DeSimone et al., 2014) and accounted for less than 2% and 10% of trials for pro- and antisaccades, respectively. The low error rate observed here is attributed to the blocked presentation of trial-type and the use of an overlap paradigm (Olk & Kingstone, 2003). Oculomotordependent variables included RT (i.e., time from response cueing to saccade onset), saccade duration (i.e., time from saccade onset to saccade offset) and saccade gain variability (i.e., within-participant standard deviation of saccade amplitude/veridical target location). Oculomotordependent variables were examined via 3 (condition: control, MCE, SSE) by 2 (time: pre-, post-intervention) by 2 (task: pro-, antisaccade) fully repeated measures ANOVAs ($\alpha = 0.05$). Statistical models were validated for violations of sphericity and interactions and appropriate main effects were decomposed via simple-effects. Where appropriate, two one-sided test (TOST) statistics were used to determine whether means were within an equivalence boundary (Lakens, 2017). As advocated by Lakens, the effect size ($d_z = 0.55$) for the dependent means TOST statistics used here was based on previous work by our group showing the smallest effect size of significance for pre- to postexercise difference in antisaccade RTs (Tari et al. 2021) and was employed for all physiological and behavioural measures.

Results

Heart rate (HR), Borg rating of perceived exertion (RPE) and blood pressure (BP)

HR produced main effects for condition, $F(2,42) = 304.62$, $p < 0.001$, $\eta_p^2 = 0.94$, time, $F(1,21) = 921.11$, $p < 0.001$, $\eta_p^2 = 0.98$, and a condition by time interaction, $F(2,42) = 446.67$, $p < 0.001$, $\eta_p^2 = 0.96$. Table 1 shows that HR in the control condition did not reliably differ from baseline to steady state (t(21)=-0.62, $p = 0.55$, d_z=-0.13), whereas HR in MCE and SSE conditions increased from baseline to steady state (all t(21) = 37.14 and 22.06, ps=<0.001, $d_z = 7.91$ and 4.70). Participant-specific HR difference scores (steady state minus baseline) did not reliably differ between MCE (71 bpm, SD = 9) and SSE (70 bpm, SD = 15) conditions (t(21) = 0.38, $p = 0.71$, $d_z = 0.08$) and a paired-samples TOST statistic indicated that values were within an equivalence boundary (t(21) = 2.02, $p = 0.028$). As well, the percentage of HRR at steady state in MCE (59%: SD = 4) and SSE (58%, SD = 11) conditions indicated that participants exercised at the prescribed moderate intensity.

RPE for MCE (13.1, SD = 1.2) and SSE (13.4, SD = 1.1) conditions indicated a "somewhat exhausting" exercise intensity and a paired-samples TOST statistic indicated that values were within an equivalence boundary (t(21) = 2.35, $p = 0.014$).

Systolic and diastolic BP yielded main effects of condition, *F*s (2,42) = 16.07 and 3.78, ps < 0.001 and 0.03, $\eta_p^2 = 0.43$ and 0.15, time, *F*s(1,21) = 96.31 and 57.05, ps < 0.001, $\eta_p^2 = 0.82$ and 0.73, and condition by time interactions, *F*s(2,42) = 29.98 and 20.38, ps < 0.001, $\eta_p^2 = 0.59$ and 0.49. Table 1 shows that control condition baseline systolic and diastolic BP did not vary from their steady state counterparts (ts(21) = 0.07 and 1.08, ps = 0.94 and 0.29, $d_z = 0.02$ and 0.23). In contrast, Table 1 demonstrates that MCE and SSE condition baseline systolic (ts(21) = 7.08 and 8.99, ps < 0.001, $d_z = 1.51$ and 1.92) and diastolic (ts(21) = 5.42 and 8.57, ps < 0.001, $d_z = 1.15$ and 1.83) values were less than their steady state counterparts.

Peak systolic middle cerebral artery velocity (MCA_{sys}), average middle cerebral artery velocity ($MCAv_{\mu}$) and pulsatility index (PI)

Figure 2 presents unfiltered (left axis and colour-coded lines) and filtered (right axis and black lines) steady state MCAv for an exemplar participant. The figure shows that steady state MCAv was greater in MCE and SSE conditions than the control condition and the filtered data provide a clear demonstration that the SSE condition produced a large amplitude oscillatory response such that values increased and decreased by 51% in relation to squat sitting and standing, respectively. As well, the exemplar participant's MCAv oscillation frequency matched the condition's metronome-paced squat-stand-frequency. From this figure, it can also be

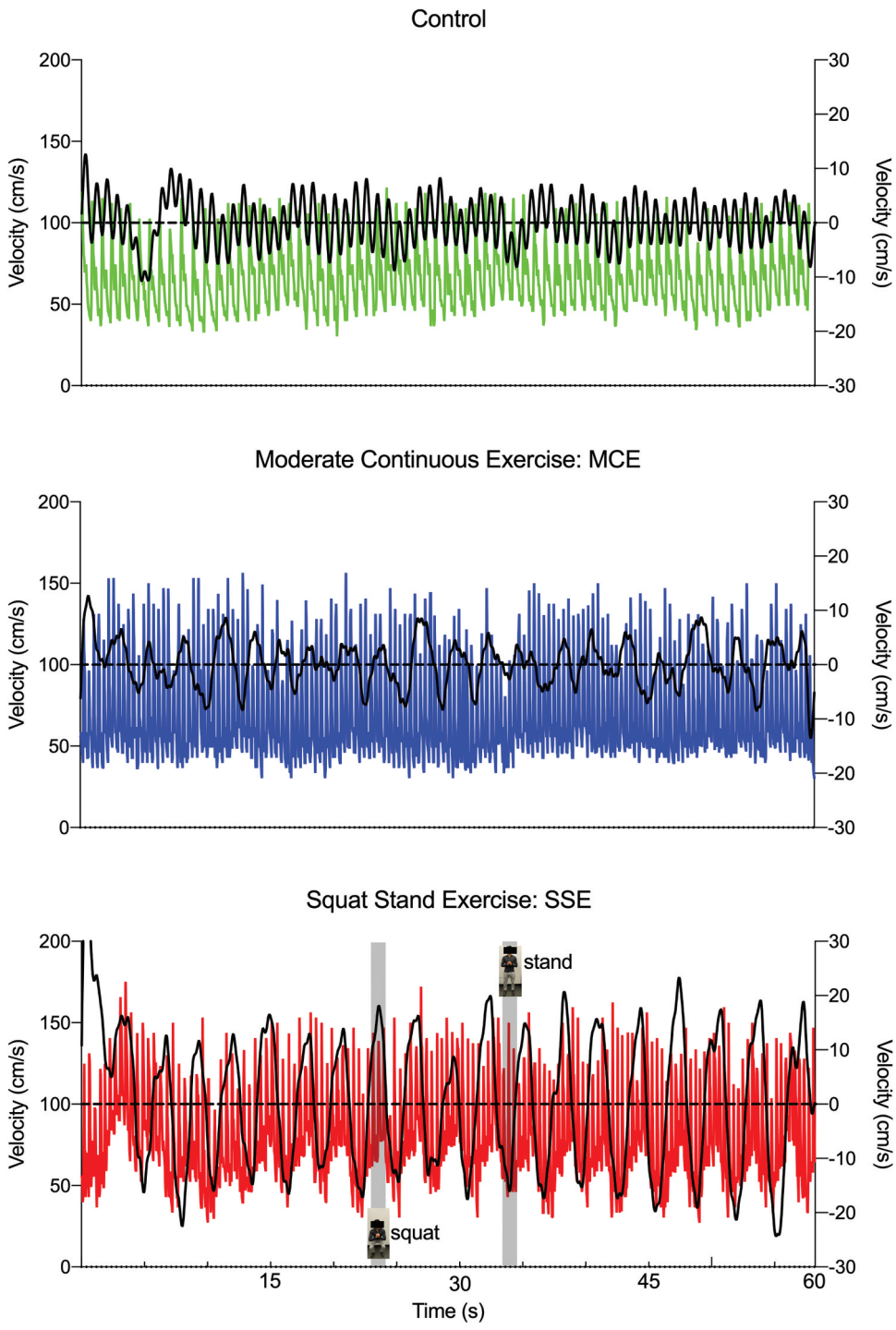

Figure 2. Unfiltered middle cerebral artery velocity (MCAv) plotted against the left axis for an exemplar participant as a function of control, MCE and SSE conditions and depicted in green, blue and red, respectively. For each condition, associated filtered MCAv (dual-pass Butterworth filter with a passband of 0.1 to 1 Hz) are plotted against the right axis and are presented as a black line. The unfiltered data provide a clear demonstration that MCAv for MCE and SSE conditions was increased relative to the control condition, and the filtered data show that the SSE condition produced an oscillatory response consistent with the condition's metronome-paced frequency.

observed that the MCE condition produced an observable oscillation frequency and is a finding that has been linked to exercise-based changes in BP due to cerebral autoregulation. Notably, however, Figure 2 shows that the oscillation amplitude is markedly less in MCE than SSE conditions (for comparable findings see, Labrecque et al., 2021; Lyngeraa et al. 2013).

In terms of quantitative analyses, $MCAv_{sys}$, $MCAv_{\mu}$ and PI produced main effects for condition, $F(2,42) = 38.92$, 12.27, 31.87, $ps < 0.001$, $\eta_p^2 = 0.65$, 0.37 and 0.59, time, $F(1,21) = 312.84$, 65.63 and 108.61, $ps < 0.001$, $\eta_p^2 = 0.94$, 0.76 and 0.84, and condition by time interactions, $F(2,42) = 92.08$, 29.74 and 58.72, $ps < 0.001$, $\eta_p^2 = 0.81$, 0.59 and 0.74. To decompose the interactions, we computed participant-

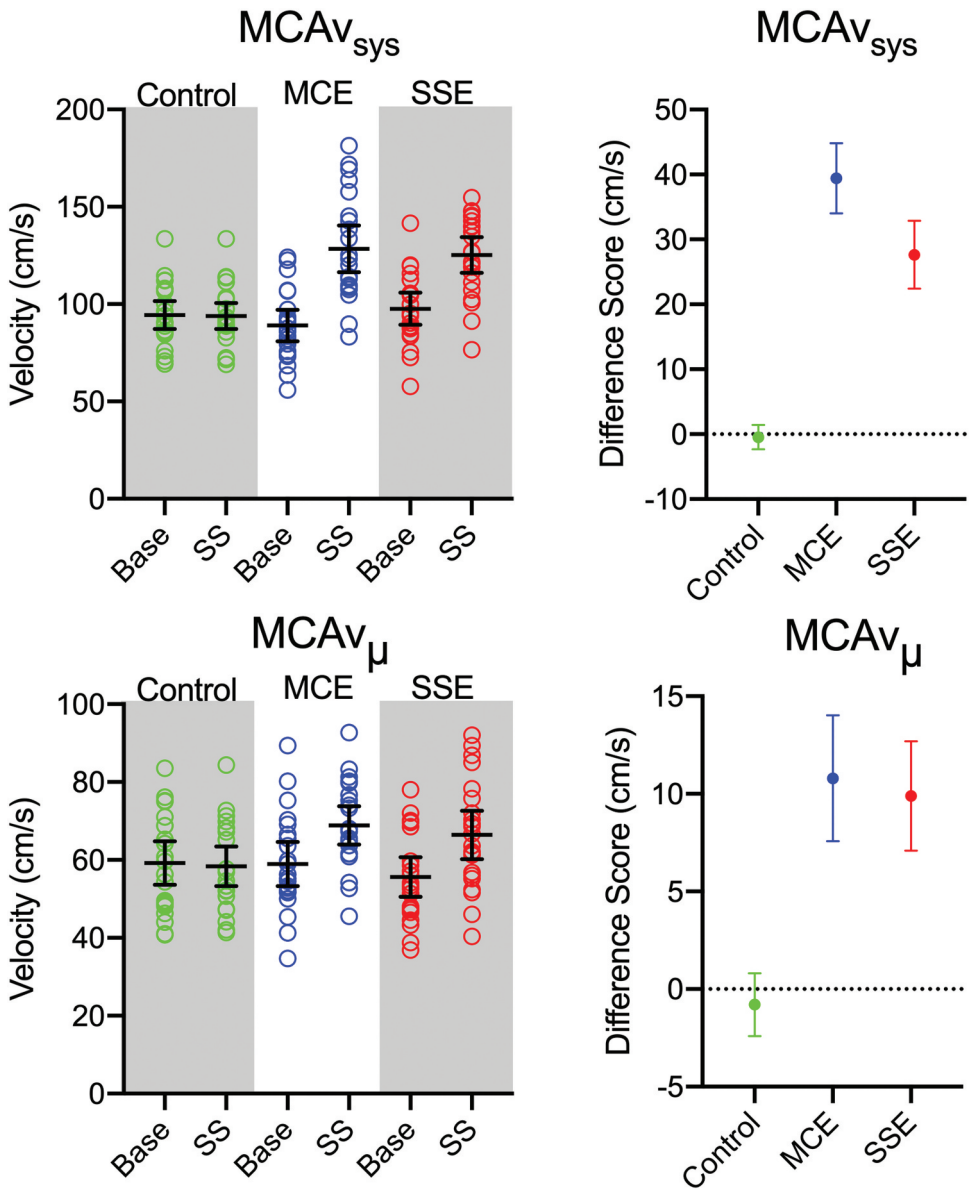

Figure 3. The left panels show control, moderate continuous aerobic exercise (MCE) and squat-stand exercise (SSE) condition participant-specific and group mean peak systolic ($MCAv_{sys}$) and average ($MCAv_{\mu}$) middle cerebral artery velocity at baseline (i.e., Base) and steady state (i.e., SS). The right panels show associated group mean $MCAv_{sys}$ and $MCAv_{\mu}$ difference scores (i.e., steady state minus baseline). Error bars represent 95% between-participant confidence intervals. For the right panels, the absence of overlap between an error bar and zero (i.e., horizontal dashed line) represents a reliable difference between baseline and steady state inclusive to a test of the null hypothesis.

specific difference scores (steady state minus baseline) for each condition and contrasted to zero via single-samples t-tests. Figure 3 shows that control condition *$MCAv_{sys}$*, $MCAv_{\mu}$ and PI values did not reliably differ from baseline to steady state (all ts(21)=-0.51, 1.03 and 0.14, ps > 0.31, d_z = 0.11, 0.22 and 0.03), whereas MCE (all ts(21) = 15.18, 6.97 and 9.71, ps > 0.001, d_z = 3.24, 1.49 and 2.07) and SSE (all ts(21) =11.03, 7.37 and 10.25, ps > 0.01, d_z = 2.23, 1.57 and 2.19) conditions produced a baseline to steady state increase in all variables.

To determine if cerebral haemodynamics varied between exercise conditions, we contrasted $MCAv_{sys}$, $MCAv_{\mu}$ and PI difference scores for MCE and SSE conditions via paired-samples t-tests. Table 1 and Figure 3 show that that $MCAv_{sys}$ and PI values were larger in the MCE than SSE condition, (ts(21) = 3.37 and 4.41, p = 0.003, d_z = 0.72 and 0.94); however, $MCAv_{\mu}$ did not reliably differ between conditions (t(21) = 0.47, p = 0.64, d_z = 0.10).

Results for oscillation amplitude showed that the magnitude of the MCAv change was larger in the SSE (80 cm/s, SD = 26) than control (44 cm/s, SD = 39) condition (t(21) = 3.92, p < 0.001, d_z = 0.84).

Executive function assessment: reaction time (RT), saccade duration and saccade gain

RT produced main effects for time, $F(1,21) = 37.58$, $p < 0.001$, $\eta_p^2 = 0.64$, and task, $F(1,21) = 95.10$, $p < 0.001$, $\eta_p^2 = 0.82$, and interactions involving condition by time, $F(2,42) = 4.80$, p =

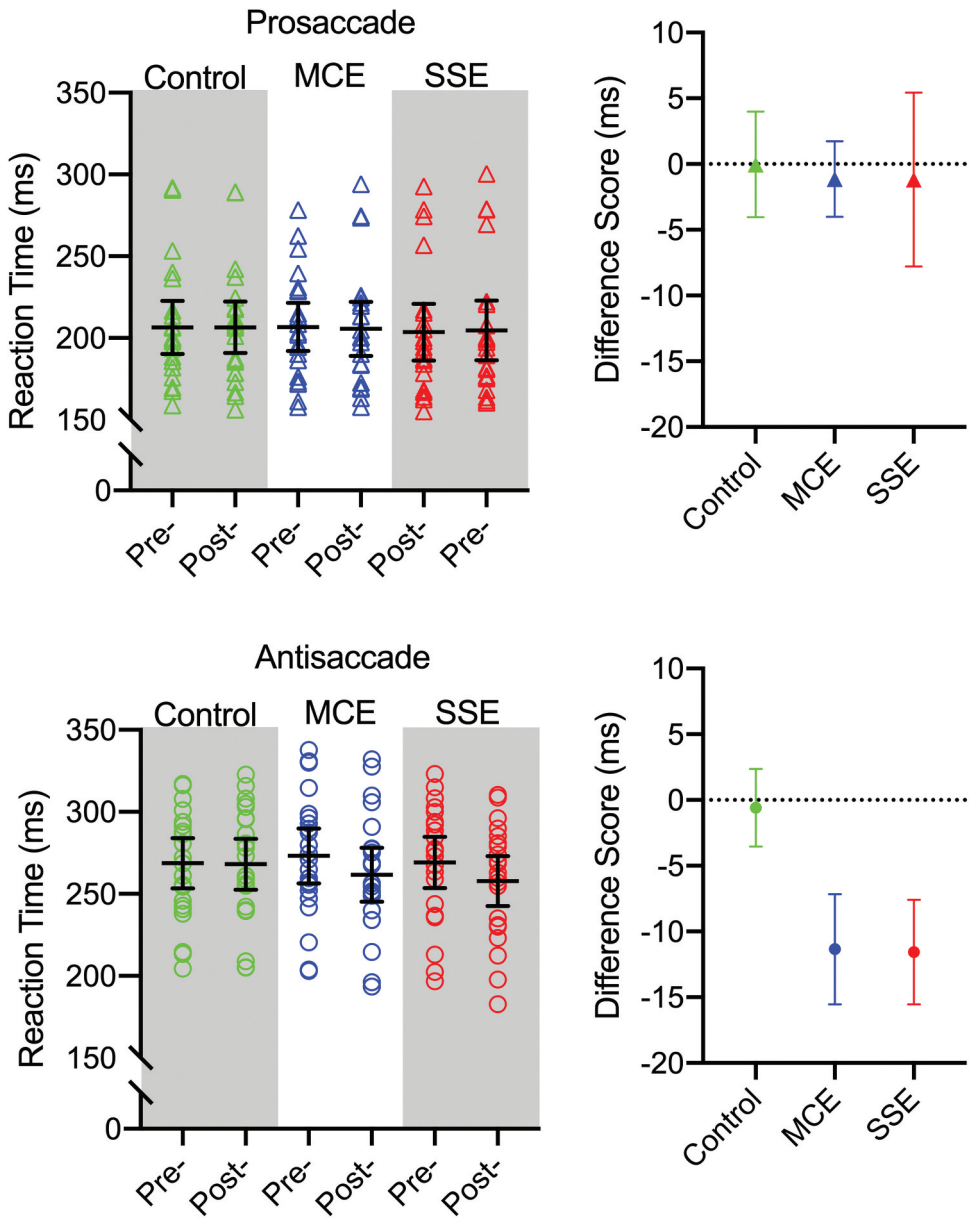

Figure 4. The left panels show control, moderate continuous aerobic exercise (MCE) and squat-stand exercise (SSE) condition participant-specific and group mean pro- and antisaccade reaction times at pre- and post-intervention assessments. The right panels show condition-specific group mean pro- and antisaccade reaction time difference scores (post-intervention minus pre-intervention). Error bars represent 95% between-participant confidence intervals. For the right panels, the absence of overlap between an error bar and zero (i.e., horizontal dashed line) represents a reliable pre- to postexercise RT difference inclusive to a test of the null hypothesis.

0.013, $\eta_p^2 = 0.19$, time by task, (1,21) = 17.23, $p < 0.001$, $\eta_p^2 = 0.45$, and condition by time by task, $F(2,42) = 3.87$, $p = 0.029$, $\eta_p^2 = 0.16$. The main panel of Figure 4 shows participant-specific and group mean pre- and post-intervention RTs and demonstrates the general finding that values for prosaccades (205 ms, SD = 36) were shorter than antisaccades (264 ms, SD = 35) across experimental conditions. In decomposing the highest-order interaction, we computed participant-specific pro- and antisaccade RT difference scores (post-intervention minus pre-intervention) and contrasted to a value of zero via single-sample t-tests (see offset panel of Figure 4). For the control condition, pro- and antisaccade values did not reliably differ from zero (ts(22)< 0.41, ps > 0.68, dz < 0.08). For the MCE and SSE conditions, prosaccade values did not reliably differ from zero (ts(22)<0.81, ps > 0.21, d_z <0.17), whereas antisaccade values were less than zero (ts(22) = 3.19 and 3.37, ps = 0.004 and 0.003, $d_z = 0.66$ and 0.70). In other words, MCE and SSE conditions produced a selective postexercise reduction in antisaccade – but not prosaccade – RTs. In addition, we contrasted antisaccade RT difference scores for MCE and SSE conditions via a paired-samples TOST statistic and observed that results were within an equivalence boundary (t(22) = 2.09, $p = 0.024$). Thus, the two conditions produced an equivalent magnitude post-intervention reduction in antisaccade RTs.

Saccade duration and gain variability produced main effects for task, Fs(1,21) = 14.18 and 27.92, ps < 0.001, $\eta_p^2 = 0.45$ and 0.57, such that prosaccade durations were shorter (57 ms, SD =

12) and endpoints less variable (0.12, SD = 0.08) than antisaccades (saccade duration = 72 ms, SD = 22; saccade gain variability = 0.25, SD = 0.06). Notably, neither variable elicited a higher-order interaction involving condition or time, Fs < 1.40, ps > 0.26, η_p^2 <0.07.

Relationship between MCAv metrics, RPE and antisaccade RT difference scores

We computed Pearson correlation coefficients relating all MCAv (i.e., $MCAv_{sys}$, $MCAv_{\mu}$ and PI) difference scores (i.e., steady state minus baseline) to antisaccade RT difference scores (postexercise minus pre-exercise) separately for MCE and SSE conditions. Results showed that for both conditions, $MCAv_{sys}$, $MCAv_{\mu}$ and PI difference score were not reliably related to their associated antisaccade RT difference scores (all $r < 0.24$ and 0.20 for MCE and SSE, respectively, ps > 0.27). We also correlated RPE values with antisaccade RT difference scores to determine if perceived exercise intensity influenced the magnitude of a postexercise EF benefit. Results for MCE ($r = 0.16$, $p = 0.47$) and SSE ($r = 0.10$, $p = 0.65$) conditions indicated that the variables were not reliably related.

Discussion

We sought to determine whether the distinct large amplitude CBF oscillatory response during SSE provides a postexercise EF benefit distinct from an MCE intervention. In addressing our results, we first outline HR, BP and cortical haemodynamic findings before turning our discussion to the postexercise EF changes.

Baseline to steady state cardiovascular and cortical haemodynamic changes to MCE and SSE

MCE and SSE conditions – but not the control condition – produced a baseline to steady state increases in HR and BP and are results linked to increased cardiac output due to the O_2 demands of the working muscles (for review see, Radak et al., 2013). Additionally, MCE and SSE cortical haemodynamics (MCA_{sys}, $MCAv_{\mu}$, PI) increased from baseline to steady state and is a well-documented response to exercise arising from increased $PaCO_2$, cerebral autoregulation, baroreflex and chemoreceptive control (for reviews see, Ogoh & Ainslie, 2009; Secher et al., 2008). Most notably, for the MCE condition, the baseline to steady state increase in MCAv entailed a distinct biphasic response, that is, MCAv progressively increased and then plateaued (Smith & Ainslie, 2017). In contrast, Figure 2 shows that the SSE condition produced a large amplitude oscillatory response such that squat and stand manoeuvres, respectively, increased and decreased MCAv, and the group mean MCAv oscillation amplitude was twofold greater in the SSE then MCE condition – a result that has been directly linked to directionally tuned changes in BP and cardiac output (Claassen et al., 2009; Labrecque et al., 2021; Panerai et al., 2021). Accordingly, the MCAv data provide a framework to evaluate whether a large amplitude CBF oscillatory response differentially influences a postexercise EF benefit.

Postexercise EF benefits in MCE and SSE conditions

A general finding from the oculomotor assessments was that MCE, SSE and control condition pre- and post-RTs for prosaccades were shorter than antisaccades. This expected finding is attributed to the fact that prosaccades are mediated via direct retinotopic projections to the superior colliculus (Wurtz & Albano, 1980), whereas the antisaccade RTs reflect the EF and time-consuming demands of response suppression and vector inversion (Munoz & Everling, 2004). The task-specific RTs therefore provide a basis to determine whether SSE and MCE conditions elicit a general information processing benefit (i.e., decreased pro- and antisaccade RTs) or a selective EF benefit (i.e., decreased antisaccade RTs only).

Both MCE and SSE conditions produced a 5% pre- to postexercise reduction in antisaccade – but not prosaccade – RTs. The RT findings are unrelated to a practice-related performance benefit given that values for the control condition did not vary from pre- to post-intervention assessments. Moreover, the RT benefit cannot be attributed to an implicit or explicit performance strategy (i.e., speed-accuracy trade-off) (Fitts, 1954) given that saccade duration and gain variability did not differ from pre- to postexercise assessments. In addition that the RT benefit was specific to antisaccades indicates that the exercise conditions did not simply improve physiological and/or psychological arousal (Ayala & Heath, 2021) or produce a general information processing benefit. Indeed, if that were the case then pro- *and* antisaccade RTs would have demonstrated a postexercise benefit. Instead, our results support a wealth evidence reporting that a single bout of aerobic exercise provides a selective postexercise EF benefit (for recent systematic reviews and meta-analysis, see Ishihara et al., 2021; Zou et al., 2023).

Our study's primary objective was to determine whether SSE elicits a postexercise EF benefit and whether the distinct CBF response for SSE and MCE conditions differentially influences the magnitude of a postexercise EF benefit. Recall that the basis for this comparison was that the large amplitude oscillatory CBF response associated with SSE increases endothelial shear-stress and is mechanism some work involving a chronic exercise manipulation has proposed to provide the optimal environment for improving cognition and EF (Green et al., 2018). Moreover, it had been proposed – but not empirically evaluated – that an SSE protocol provides the optimal environment for eliciting a single bout postexercise EF benefit (Larbi, 2021; Roberts, 2018). Of course, in the present work we evaluated whether a single bout of SSE provides a larger magnitude EF benefit than MCE and in doing so we equated conditions for absolute (i.e., HR and BP) and perceived (RPE) intensity. Given this experimental control, null hypothesis and TOST statistics indicated that the magnitude of the postexercise reduction in antisaccade RTs was equivalent between SSE and MCE conditions. Accordingly, we believe our results add importantly to the exercise neuroscience literature insomuch as they demonstrate that (1) a 15 min single bout of SSE provides a postexercise EF benefit equal to a duration- and intensity-matched MCE protocol, (2) the distinct CBF response associated with each exercise protocol does not differentially influence the magnitude of a postexercise EF benefit and (3) an SSE protocol

provides for economy of equipment (i.e., no need for cycle ergometer or treadmill) and space (i.e., can be completed within a small physical space) in providing a transient EF "boost".

At least two important issues require addressing. The first is that although SSE and MCE conditions produced an exercised-induced increase in MCAv and demonstrated a postexercise reduction in antisaccade RTs, neither condition showed a reliable relationship between our directed measures of MCAv (i.e., $MCAv_{sys}$, $MCAv_{\mu}$ and PI) and the postexercise reduction in antisaccade RTs. In accounting for this issue, we note that individual differences in an exercise pressor response and the associated differences in the absolute and perceived intensity of an exercise protocol may account for a null associative relationship between CBF reactivity and EF (Washio & Ogoh, 2023). Moreover, it is important to note that an increase in CBF may serve as one of a series of interdependent mechanisms supporting a postexercise EF benefit. As examples, increased brain-derived neurotropic factor (Knaepen et al., 2010) and catecholamine(Zouhal et al., 2008), Cathepsin B (De la Rosa et al., 2019) concentrations, and increased resting state functional connectivity (Schmitt et al., 2019) may serve as interactive mechanisms with increased CBF to support a single bout postexercise EF benefit. Hence, a consideration to be drawn from our work is that an increase in CBF during exercise may represent one of the number of interactive physiological changes that support a postexercise EF benefit. The second issue to address is that the SSE protocol provided minute-to-minute alternations between exercise and rest and thus entailed an exercise frequency comparable to high-intensity interval exercise (HIIE). This is an issue to address because some evidence has reported that a single bout of HIIE provides a larger postexercise EF benefit than MCE (Kovacevic et al., 2020; for review see; Hsieh et al., 2021). It is, however, important to recognize that HIIE does not elicit an oscillatory CBF response *within* an exercise interval, and that the protocol leads to a decrease in CBF over successive intervals due to a hyperventilation-induced reduction in arterial $PaCO_2$ and associated cerebral vasoconstriction (Whitaker et al., 2020). Accordingly, the SSE protocol used here is distinct from HIIE.

Study limitations

We recognize that our study is limited by several methodological traits. First, TCD does not account for changes in vessel diameter, and it is known that the MCA is capable of dilation and constriction in response to changes in $PaCO_2$ (Coverdale et al., 2015). Thus, it is not possible to assert that MCAv provided an absolute measure of CBF. Second, we recruited healthy and recreationally active young adults and it is therefore unclear whether older adults or persons with distinct cerebrovascular reactivity to exercise (i.e., chronic cerebral hypoperfusion) may show a similar EF benefit following MCE and SSE. Third, we used a single exercise duration and intensity and evaluated EF at one timepoint postexercise. It is possible that SSE elicits a dose–response relation distinct from MCE (Chang & Etnier, 2009) and may differentially influence the temporal persistence of a postexercise EF benefit (Shukla & Heath, 2022). Future work should address how such factors influence an SSE-based postexercise EF benefit. Last, the current work provided only a single bout of SSE and MCE, and we are thus unable to conclude whether chronic exposure to an SSE protocol – and associated large amplitude oscillatory CBF response – provides a long-term benefit to EF.

Conclusions

A single bout of SSE provides a large amplitude oscillatory CBF change distinct from the steady state increase associated with a moderate intensity MCE protocol. In spite of the condition-specific CBF response, SSE and MCE conditions did not differentially influence the magnitude of a postexercise EF benefit.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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Research data

The datasets of the current study are available from the corresponding author upon reasonable request.

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