

**Cerebral Blood Flow and Immediate and Sustained Executive Function Benefits Following  
Single Bouts of Passive and Active Exercise**

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## Abstract

Passive exercise occurs when an individual's limbs are moved via an external force and is a modality that increases cerebral blood flow (CBF) and provides an immediate postexercise executive function (EF) benefit. To our knowledge, no work has examined for how long passive exercise benefits EF. Here, healthy young adults (N=22; 7 female) used a cycle ergometer to complete three 20-min conditions: passive exercise (via mechanically driven flywheel), a traditional light intensity (37 W) "active" exercise condition (i.e., via volitional pedalling) and a non-exercise control condition. An estimate of CBF was obtained via transcranial Doppler ultrasound measurement of middle cerebral artery blood velocity (MCAv) and antisaccades (i.e., saccade mirror-symmetrical to a target) were completed prior to and immediately, 30- and 60-min following each condition to assess EF. Passive and active exercise increased MCAv; however, the increase was larger in the latter condition. In terms of antisaccades, passive and active exercise provided an immediate postexercise reaction time benefit. At the 30-min assessment, the benefit was observed for active but not passive exercise and neither produced a benefit at the 60-min assessment. Thus, passive exercise provided an evanescent EF "boost" and is a finding that may reflect a smaller cortical hemodynamic response.

**Keywords:** *antisaccade; cognition; cortical hemodynamics; oculomotor; vision*

## 1. Introduction

Executive function (EF) is a high-level cognitive construct essential for activities of daily living and includes the core components of inhibitory control, working memory and cognitive flexibility (Diamond, 2013). A growing literature has shown that each core component of EF is improved following a single bout of aerobic and/or resistance training (for meta-analyses see, Lambourne and Tomporowski, 2010; Chang et al., 2012; Ishihara et al., 2021). This benefit has been linked to increased cerebral blood flow (CBF) (Tari et al., 2020), biomolecule concentrations (e.g., catecholamines, brain-derived neurotrophic factor (BDNF)) (for reviews see, Zouhal et al., 2008; Knaepen et al., 2010), and resting state functional connectivity (Schmitt et al., 2019), that improves the efficiency and effectiveness of frontoparietal EF networks.

One notable meta-analytic finding is that **a single bout** of moderate (e.g., ~40-60% of heart rate reserve: HRR) to heavy (>60% of HRR) **intensity exercise produces the largest and most reliable postexercise EF benefit** (Chang et al., 2012). It is, however, important to recognize that positive benefits have been reported for very-light (<30% of HRR) to light (30-50% of HRR) intensity exercise. For example, Kamijo et al. (2007) reported that flanker task (i.e., an executive task of response inhibition) reaction times (RTs) assessed immediately following 20-min of light intensity cycle ergometry were shorter than a non-exercise baseline condition (cf. Morris et al., 2020). Similarly, our group examined EF (via the antisaccade task – see details below) prior to and immediately following separate sessions of cycle ergometry involving a continuum of power outputs and observed that for each intensity postexercise antisaccade RTs were shorter than their pre-exercise counterparts (Tari et al., 2020; Heath et al., 2018; Petrella et al., 2019; Tari et al., 2021). As well, Shirzad et al. (2022) contrasted pre- and postexercise antisaccade RTs in conditions involving a traditional “active” light intensity aerobic exercise

protocol (i.e., participants actively pedalled a cycle ergometer for 20-min at a light intensity) with a same duration “passive” exercise condition wherein the cycle ergometer flywheel was mechanically driven and did not require volitional muscle activation. Notably, **due to the lack of volitional muscle activation, passive exercise did not result in elevated ventilation ( $\dot{V}_E$ ) or gas exchange variables (e.g., oxygen consumption:  $\dot{V}O_2$ ; carbon dioxide production:  $\dot{V}CO_2$ ) associated with active exercise (see Fisher et al., 2015; Trinity and Richardson, 2019).** **Consequently,** passive exercise increases CBF independent of the metabolic and “intensity” demands of active exercise (e.g., Doering et al., 1998). Specifically, passive exercise increases CBF due to the activation of mechanoreceptive muscle afferents to the primary somatosensory cortex that increase cardiac output and stroke volume (Nóbrega and Araujo, 1993). In contrast, active exercise primarily increases CBF due to  $CO_2$  production, increased diffusible molecules (e.g., nitric oxide: NO), and vascular deformation (Smith and Ainslie, 2017) related to volitional muscle activation. Shirzad et al. reported that CBF, as estimated via functional transcranial Doppler ultrasound (TCD) blood flow velocity of the middle cerebral artery (MCAv), increased during active and passive exercise conditions – albeit the magnitude was larger in the former – and both conditions produced an immediate postexercise reduction in antisaccade RTs. In other words, results indicated that an intensity-independent increase in CBF provides an immediate EF benefit.

A second meta-analytic finding in the active exercise literature relates to the persistence of a postexercise EF benefit. Lambourne and Tomporowski (2010) concluded the benefit is observed within the first 15-min postexercise but not thereafter, whereas Chang et al. (2012) concluded that benefits are “[...] observed following 11–20 min of delay, and smaller positive effects are evident following 20 min of delay” (p. 91). It is, however, important to recognize that

emergent literature indicates that active exercise EF benefits are more persistent. Johnson et al. (2016) reported that healthy older adults (>55 years of age) that completed 10- and 30-min sessions of moderate-intensity aerobic and resistance exercise exhibited improved EF as assessed via the Stroop interference task (i.e., executive task of response inhibition) at intervals 30- and 60-min postexercise. As well, Joyce et al. (2009) and Hung et al. (2013) observed that healthy young adults who completed 30- and 20-min of moderate-intensity active cycle ergometry exhibited improved EF performance via stop-signal (i.e., executive task of response inhibition) and Tower of London tasks (i.e., executive task of planning and working memory) at intervals 52- and 80-min postexercise, respectively. Last, our group (Shukla and Heath, 2022) had healthy young adults complete a 20-min single-bout of heavy intensity aerobic exercise via active cycle ergometry and observed an EF benefit that persisted up to 47-min postexercise. Hence, there is evidence to assert that a postexercise EF benefit persists for longer than that reported in earlier meta-analyses.

Here, we examined for how long a single bout of active light intensity exercise and passive exercise elicit a postexercise EF benefit. This represents a salient question because it provides a basis to evaluate the persistence of EF benefits incurred independent of the metabolic requirements of active exercise. To that end, healthy young adults completed separate 20-min sessions of cadence-matched cycle ergometry in an active light intensity condition and a passive exercise condition wherein the ergometer flywheel was mechanically driven at 70 rpm. In addition, a non-exercise control condition was employed. During all conditions, TCD was used to measure MCAv and evaluate baseline to steady state changes in CBF. EF was assessed prior to and immediately following exercise cessation (i.e., immediate-post) and at intervals 30-min (i.e., 30-post) and 60-min (i.e., 60-post) postexercise (see **Figure 1**). CBF was measured during

each intervention (i.e., control, passive exercise, active exercise) and not during each EF assessment given that exercise-induced changes in CBF return to baseline levels in as little as 5 min following exercise cessation (González-Alonso et al., 2004; Goodall et al., 2012).

The EF task entailed pro- and antisaccades completed in separate blocks. Prosaccades require a goal-directed eye movement (i.e., saccade) to an exogenously presented target and are mediated largely independent of top-down executive processes via retinotopic projections to the superior colliculus (Wurtz and Albano, 1980). In contrast, antisaccades require a saccade mirror-symmetrical to a target and result in longer RTs (Hallet, 1978) and more variable endpoints (Gillen and Heath, 2014) than their prosaccade counterparts. The behavioural ‘costs’ of antisaccades reflect the executive demands of response inhibition and vector inversion (i.e., 180° spatial transformation) (for review see Munoz and Everling, 2004) and are mediated via EF networks (Neggers et al., 2012; Chen and Machado, 2016) that show task-dependent changes following single bouts of exercise (e.g., Schmitt et al., 2019). Thus, when combined with prosaccades, antisaccades provide a hands- and language-free basis to identify subtle and exercise-specific EF changes. Given previous work (Shirzad et al., 2022), we predicted that active and passive exercise conditions – but not the control condition – would increase CBF and produce an immediate postexercise reduction in antisaccade (but not prosaccade) RTs. In terms of the 30- and 60-postexercise assessments, it may be that the increased metabolic demands of active exercise provide the neurophysiological environment (i.e., a larger CBF increase) resulting in active exercise producing an EF benefit that is more persistent than passive exercise.

## 2. Materials and Methods

### 2.1 Participants

Twenty-two English-speaking individuals (7 female, age range 19–26 years) from the University of Western Ontario community volunteered to participate in this study with sample size determined *a priori* using G\*Power (Faul et al., 2007, 2009) based on an effect size derived from previous work examining pre- to postexercise changes to antisaccade RTs ( $\alpha=0.05$ , power = 0.90, pooled effect size  $d_z = 0.76$ ) (Shirzad et al., 2022). The present study was based on work that investigated immediate postexercise EF benefits (Shirzad et al., 2022); however, the participants recruited here are independent of that prior work. Participants were self-reported right-hand dominant (i.e., “What hand do you 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 reported that they did not take medication that may affect metabolic, cardiac, respiratory, or hemodynamic responses to exercise, were free from illness on the days of testing and had no history of SARS-CoV-2 (e.g., Hampshire et al., 2022). It was requested that participants abstain from strenuous exercise and not consume alcohol or caffeine 12 hours prior to data collection, that they get 8 hours of sleep on the night prior to each session of data collection, and that they present to the lab in a hydrated state (i.e., consume 555 ml of fluid 1 hour prior to data collection) – all participants reported compliance with these requests. Prior to data collection participants read a letter of information approved by the Health Sciences Research Ethics Board, University of Western Ontario (ID: 120491) 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 were deemed to be physically able to exercise as determined via the 2020 Physical Activity Readiness Questionnaire (PAR-Q+) and were classified as recreationally active via the Godin Leisure-Time Exercise Questionnaire (GLTEQ). The average GLTEQ

score was 56 (SD = 22; Range: 29 – 110) – a result indicating that all participants were recreationally active.

## 2.2 Apparatus and Procedure

### 2.2.1 Passive, active and control conditions

This investigation involved three conditions (passive exercise, active exercise, and control) completed on separate days between 9:30 am and 11:00 am. The ordering of conditions was counterbalanced; however, given that 22 participants were entered into this study we acknowledge the passive exercise condition served as the first condition for eight participants, whereas the control and active exercise condition served as the first condition for seven participants each. For all conditions, participants sat on an upright active-passive cycle ergometer (E-PAT AP; Healthcare International, Langley, WA, USA) equipped with a mechanically driven flywheel. Participants were positioned with their feet secured to the ergometer pedals via Velcro™ straps and their legs achieved approximately 85% of full extension at the end of an active/passive pedal stroke. Participants were fitted with a TCD probe (Neurovision 500M, 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. TCD provides a valid proxy for a direct measure of changes to CBF (Bishop et al., 1986). All conditions were preceded by a 2-min resting baseline wherein participants sat stationary on the ergometer. During the passive exercise condition pedal cadence was mechanically determined for a 2-min warm-up at 40 rpm followed by a step-transition to 70 rpm for 20-min after which a 2-min cool-down at 40 rpm was initiated. In this condition, participants did not actively engage their leg muscles (as confirmed via electromyography; see **Supplementary Figure 1**) during each pedal



189 revolution and this condition did not increase ventilation ( $\dot{V}_E$ ) or gas exchange variables (i.e.,  
 190  $\dot{V}O_2$  and  $\dot{V}CO_2$ ) (see **Supplementary Figure 2**) indicating the low likelihood that participants  
 191 actively engaged their leg muscles during the passive exercise condition. In the active exercise  
 192 condition, the same timeline as the passive exercise condition was used (i.e., baseline, warm-up,  
 193 intervention, cool-down); however, in this condition the 2-min warm-up and cool-down involved  
 194 a self-generated cadence of 40 rpm with resistance set to 15 W, whereas the 20-min intervention  
 195 involved a self-generated cadence of 70 rpm with resistance set at 37 W. Thus, power output in  
 196 the active exercise session provided for light intensity exercise. A light intensity active exercise  
 197 condition was selected for three reasons: (1) pilot testing showed that light intensity is preferable  
 198 to no-load cycling because maintaining a constant pedal cadence in the latter task is attentionally  
 199 demanding and frequently resulted in an uncomfortable posture for participants, (2) light  
 200 intensity exercise provides a reliable postexercise EF benefit (Shirzad et al., 2022; Tari et al.,  
 201 2021), and (3) light intensity provides an increase in CBF reasonably comparable to passive  
 202 exercise (Shirzad et al., 2022). The control condition required participants sit on the same cycle  
 203 ergometer for 26-min (i.e., equivalent to the baseline, warm-up, intervention, and cool-down  
 204 during active and passive exercise conditions) without completing passive or active exercise and  
 205 participants chatted to the experimenters during this time. During both exercise conditions, a  
 206 metronome was set in time with the appropriate cadence. Heart rate was measured (Polar Electro  
 207 T34; Polar Electro Oy, Kempele, Finland) and blood pressure was assessed via a manual  
 208 sphygmomanometer and stethoscope (Welch Allyn FlexiPort reusable blood pressure cuff;  
 209 Welch Allyn Inc. Skaneateles Falls, NY, USA) secured to participants' left upper arm. TCD was  
 210 not measured following passive, active or control conditions given that MCAv returns to baseline  
 211 shortly after exercise cessation (i.e., < 5-min) (González-Alonso et al., 2004; Goodall et al.,

2012). In spite of this return to baseline, increased CBF leads to sustained mechanical and temperature changes to the brain's neural and glial networks (i.e., the hemo-neural hypothesis) (Moore and Cao, 2008).

### 2.2.2 *EF assessment*

EF was assessed via an oculomotor task completed prior to, immediately (immediate-post), 30-min (30-post) and 60-min (60-post) after the active exercise, passive exercise, and control conditions. The immediate-post assessment began ~4-min after each condition to ensure heart rate had returned to baseline (see also Tari et al., 2021; Shirzad et al., 2022). 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,  $1280 \times 960$  pixels; Dell 3007WFP, Round Rock, TX) was located 550 mm from the table's front edge. Participants placed their head in a head-chin rest, 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 nine-point calibration and validation of the viewing space was completed (i.e.,  $<1^\circ$  of error). All experimental events were controlled via MATLAB (R2018a; The MathWorks, Natick, MA) and the Psychophysics Toolbox extension (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 were presented on a black screen ( $0.1 \text{ cd/m}^2$ ) and included a  $1^\circ$  midline-located and luminance-matched ( $50 \text{ cd/m}^2$ ) red or green fixation cross presented at participants' eye level and targets (i.e., open white circle;  $2.5^\circ$  in diameter:  $127 \text{ cd/m}^2$ ) presented  $15^\circ$  (i.e., proximal target) and  $20^\circ$  (i.e., distal target) to the left and right of fixation and in the same horizontal plane. Fixation onset signaled participants to direct their gaze to its location and

fixation colour indicated the nature of the upcoming trial. Once a stable gaze was achieved (i.e.,  $\pm 1.5^\circ$  for 450 ms), a uniformly distributed randomized foreperiod (i.e., 1000 – 2000 ms) was introduced after which the fixation disappeared and a target appeared 200 ms thereafter (i.e., gap paradigm) and remained visible for 50 ms. For half of participants green and red fixation crosses indicated pro- (i.e., saccade to veridical target location) or antisaccade (i.e., saccade mirror-symmetrical to target location), respectively. For the other half of participants, the converse fixation-to-task colour mapping was used. **In advance of data collection for each assessment, participants were provided an instruction screen indicating the nature of the fixation colour and task (i.e., pro- vs. antisaccade) mapping.** For each oculomotor assessment (i.e., pre, immediate-post, 30-post and 60-post), 80 pro- and 80 antisaccade trials were presented in separate and randomly ordered blocks which included 40 trials to each target location (i.e., left, and right visual field) and eccentricity (i.e., proximal, and distal) (i.e., 160 total trials). **Further, post hoc analysis indicated that the random ordering of pro- and antisaccades resulted in each equally serving as the first trial block across all assessments.**

### *2.3. Data reduction, dependent variables, statistical analysis*

TCD data corrupted by signal aliasing and/or signal loss (e.g., a sudden head shift) were omitted (Terslev et al., 2017) and systolic MCAv were retained for analysis (Clyde et al., 1996) because they provide a valid assessment of TCD-based CBF through the MCA (Clyde et al., 1996; Rosengarten and Kaps, 1991) (note: we also report time-averaged mean velocity:  $\text{MCAv}_{\text{mean}}$ ). MCAv, heart rate and blood pressure were computed from the last minute of baseline and the last minute of each intervention (i.e., steady state) (**see Figure. 2**) and examined via 3 (condition: active exercise, passive exercise, control) by 2 (time: baseline, steady state) fully repeated measures ANOVAs ( $\alpha = 0.05$ ).

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 five-point central-finite difference algorithm was used to compute instantaneous velocities and acceleration. Saccade onset was determined when velocity and acceleration exceeded  $30^\circ/\text{s}$  and  $8,000^\circ/\text{s}^2$ , respectively. Saccade offset was determined when velocity fell below  $30^\circ/\text{s}$  for 40 ms. Trials involving a signal loss (e.g., an eye blink) were removed as were anticipatory responses ( $\text{RTs} < 50 \text{ ms}$ ) (Wenban-Smith and Findlay, 1991) and  $\text{RTs} > 2.5$  standard deviations of a participant- and task-specific mean (Gillen and Heath, 2014). Less than 11% of trials for any participant were omitted. Trials involving a directional error (i.e., a prosaccade instead of an instructed antisaccade and *vice versa*) were excluded from subsequent analyses because they are associated with planning mechanisms distinct from their directionally correct counterparts (DeSimone et al., 2014) and accounted for less than 6% of total experimental trials (i.e., 2% of prosaccade trials and 9% of antisaccade trials). Oculomotor dependent 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). Mean values were used in our ANOVA models given that RT, saccade duration and saccade gain data were not skewed (all  $g_1 < 1.00$ ,  $z_s < 1.96$ ,  $p_s > 0.05$ ) (Kim et al., 2013). Oculomotor dependent variables were examined via 3 (condition: active exercise, passive exercise, control) by 4 (time: pre-, immediate-post, post-30, post-60) by 2 (task: pro-, antisaccade) fully repeated measures ANOVAs ( $\alpha = 0.05$ ).

Interactions and appropriate main effects were decomposed via simple-effects (i.e., time by task ANOVAs and/or paired-samples t-test) ( $\alpha = 0.05$ ). Where appropriate, two one-sided test (TOST) statistics were used to determine whether means were within an equivalence boundary

(Lakens, 2017). Huynh-Feldt corrections for violations of sphericity are reported when necessary (i.e., degrees of freedom adjusted to one decimal place), and Bonferroni adjustments were made for the multiple comparisons required to assess changes in RT across time ( $\alpha = 0.017$ ).

### 3. Results

#### 3.1 Heart rate and blood pressure.

Heart rate produced main effects for condition,  $F(2,42)=32.76$ ,  $p<0.001$ ,  $\eta_p^2=0.61$ , time,  $F(1,21)=186.62$ ,  $p<0.001$ ,  $\eta_p^2=0.90$ , and their interaction,  $F(1.3,28.0)=84.47$ ,  $p<0.001$ ,  $\eta_p^2=0.80$ . Control condition baseline (76 bpm, SD=10) and steady state (76 bpm, SD=13) heart rate did not reliably differ ( $p=0.76$ ), whereas passive and active exercise condition heart rates increased from baseline (passive: 75 bpm, SD=11; active: 74 bpm, SD=11) to steady state (passive: 78 bpm, SD=12; active: 109 bpm, SD=14) ( $ps<0.04$ ). Participant-specific heart rate difference scores (steady state minus baseline) indicated a smaller magnitude change for passive (3 bpm, SD=6) than active (35 bpm, SD=13) exercise ( $p<0.001$ ). Last, the active exercise condition produced a steady state heart rate corresponding to 30% of participants' heart rate reserve (i.e., 112 bpm, SD=8) – a result supporting the light intensity active exercise classification used here.

Systolic and diastolic blood pressures yielded main effects of condition,  $F_s(2,42)=10.92$  and 3.46,  $ps<0.001$  and 0.04,  $\eta_p^2=0.34$  and 0.14, time,  $F_s(1,21)=39.25$  and 7.37,  $ps<0.001$  and 0.01,  $\eta_p^2=0.65$  and 0.26, and their interactions,  $F_s(1.6,33.1$  and  $2,42)=34.54$  and 7.94,  $ps<0.001$  and  $=0.001$ ,  $\eta_p^2=0.62$  and 0.27. For control and passive exercise conditions, systolic and diastolic blood pressure did not vary from baseline (control: 123 mmHg, SD=10 and 79 mmHg, SD=8; passive: 124 mmHg, SD=9 and 78 mmHg, SD=9) to steady state (control: 122 mmHg, SD=10 and 77 mmHg, SD=8; passive: 126 mmHg, SD=8 and 81 mmHg, SD=9) ( $ps=0.11$  and

0.20), whereas in the active exercise condition systolic (123 mmHg, SD=13) and diastolic (79 mmHg, SD=12) values increased from baseline to steady state (144 mmHg, SD=19 and 87 mmHg, SD=12) ( $p < 0.001$ ).

### 3.2 Transcranial Doppler ultrasound.

Systolic MCAv produced main effects for condition,  $F(2,42)=14.07$ ,  $p < 0.001$ ,  $\eta_p^2=0.40$ , and time,  $F(1,21)=110.19$ ,  $p < 0.001$ ,  $\eta_p^2=0.84$ , and their interaction,  $F(2,42)=59.99$ ,  $p < 0.001$ ,  $\eta_p^2=0.74$ . **Figure 2** shows that the control condition did not result in a baseline (96 cm/s, SD=14) to steady state (94 cm/s, SD=14) change in MCAv ( $p=0.25$ ), whereas passive and active exercise conditions produced a baseline (95 cm/s, SD=14 and 98 cm/s, SD=17) to steady state (104 cm/s, SD=12 and 127 cm/s, SD=26) increase ( $p < 0.001$ ). Participant-specific MCAv difference scores (steady state minus baseline) indicated the magnitude of the MCAv change was smaller during passive (8 cm/s, SD=7) than active (29 cm/s, SD=13) exercise ( $p < 0.001$ ). In addition, results for time-averaged mean velocity (MCAv<sub>mean</sub>) mirrored systolic MCAv; that is, passive (4 cm/s, SD=4) and active (8 cm/s, SD=10) exercise produced a baseline to steady-state increase in mean MCAv<sub>mean</sub> ( $p < 0.002$ ), whereas no change was observed in the control condition (-1 cm/s, SD=6) ( $p=0.53$ ). As well, steady-state MCAv<sub>mean</sub> was smaller for passive than active exercise ( $p=0.02$ ).

### 3.3 Oculomotor performance.

RT produced main effects for condition,  $F(2,42)=5.85$ ,  $p=0.006$ ,  $\eta_p^2=0.22$ , time,  $F(2.1,43.7)=3.67$ ,  $p=0.03$ ,  $\eta_p^2=0.15$ , and task,  $F(1,21)=127.23$ ,  $p < 0.001$ ,  $\eta_p^2=0.86$ , as well as time by task,  $F(3,63)=4.43$ ,  $p=0.007$ ,  $\eta_p^2=0.17$ , and condition by time by task interactions,  $F(6,126)=2.29$ ,  $p=0.04$ ,  $\eta_p^2=0.10$ . The three-way interaction was decomposed via time by task ANOVAs computed separately for each condition. All conditions yielded main effects of task,

$F(1,21)=114.82$ ,  $124.92$  and  $101.75$ , for control, passive and active conditions respectively,  $p<0.001$ ,  $\eta_p^2=0.85$ ,  $0.86$ ,  $0.83$ , indicating that prosaccade RTs (202 ms,  $SD=37$ ) were shorter than antisaccades (263 ms,  $SD=38$ ). The control condition did not elicit a time by task interaction,  $F(3,63)=1.50$ ,  $p=0.22$ ,  $\eta_p^2=0.07$ , whereas passive and active exercise did,  $F(3,63)=2.80$  and  $5.75$ ,  $p=0.047$  and  $0.002$ ,  $\eta_p^2=0.12$  and  $0.22$ . For passive and active conditions, **Figure 3** shows that prosaccade RTs at each post-assessment interval (i.e., immediate, post-30 and post-60) did not reliably vary from pre-assessment ( $p>0.25$ ). In turn, **Figure 3** shows that passive exercise condition antisaccade RTs at the immediate-post assessment were shorter than their pre-assessment counterparts ( $p=0.009$ ), whereas 30-post and 60-post values did not reliably differ from pre-assessment ( $p>0.20$ ). For the active exercise condition, **Figure 3** shows that antisaccade RTs at immediate-post and 30-post assessments ( $p<0.001$ ) – but not the 60-post assessment ( $p=0.51$ ) – were shorter than the pre-assessment. Put more directly, the three-way interaction indicated that passive and active exercise produced an antisaccade-specific reduction in RTs at immediate (i.e., passive and active exercise conditions) and 30-post (i.e., active exercise condition) assessments<sup>1</sup>. Last, for passive and active exercise conditions we computed antisaccade RT difference scores (pre-assessment minus immediate post-assessment) and observed that values did not reliably differ ( $p=0.63$ ); however, a TOST statistics indicated values were not within an equivalence boundary ( $t(21)=0.34$ ,  $p=0.63$ ).

Saccade duration and gain variability yielded main effects of task,  $F(1,21)=10.97$  and  $64.99$ ,  $p=0.003$  and  $<0.001$ , all  $\eta_p^2=0.34$  and  $0.76$ . Prosaccade durations were shorter (54 ms,  $SD=9$ ) and endpoints less variable (0.11,  $SD=0.05$ ) than antisaccades (saccade duration: 62 ms,

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<sup>1</sup>For RT data, we performed a supplementary condition by time by task by target eccentricity (i.e., proximal and distal) repeated measures ANOVA ( $\alpha=0.05$ ) to demonstrate that target location did not modulate RT in any experimental session. Results demonstrated that target eccentricity did not elicit any higher-order interactions ( $p>0.39$ ).

SD=17; gain: 0.20, SD=0.07). Notably, saccade duration and gain variability did not elicit main effects for condition nor time, nor any higher-order interactions involving these variables ( $p>0.35$ ).

### 3.4 Systolic MCAv and the immediate postexercise improvement in antisaccade RTs

Pearson  $r$  correlations for the baseline to steady state change in systolic and mean MCAv (i.e., steady-state minus baseline) and associated antisaccade RT differences scores computed separately for passive and active exercise conditions did not indicate a reliable relationship between an exercise-based increase in CBF and the magnitude of an immediate postexercise benefit to an oculomotor-based index of EF (all  $r(21)< -0.07$  and  $-0.25$  for passive and active exercise, respectively,  $p>0.77$  and  $0.26$ ).

## 4. Discussion

We examined whether 20-min single bouts of active and passive exercise differentially influence the persistence of a postexercise benefit to an oculomotor index of EF. Below, we first discuss the observed physiological differences between active and passive exercise before outlining their respective impact on EF.

### 4.1 Active and passive exercise differentially increase cerebral blood flow

Active exercise produced baseline to steady state increases in MCAv (29 cm/s), heart rate (35 bpm) and systolic (21 mmHg) and diastolic (8 mmHg) blood pressure. These findings are in line with an extensive literature reporting that active exercise initiates a rapid rise in CBF via CO<sub>2</sub> production and an associated increase in NO, cardiac output, stroke volume and vascular deformation to account for the metabolic demands of volitional muscle activation (Smith and Ainslie, 2017). Passive exercise also produced a baseline to steady state increase in MCAv (8 cm/s); however, the magnitude of this change was less than active exercise. In turn, passive



exercise produced a small change in heart rate (3 bpm) and a null change in systolic (1 mmHg) and diastolic (3 mmHg) blood pressure (for similar results see Shirzad et al., 2022; Chen et al., 2019; Christensen et al., 2000). As outlined in the Introduction, a passive exercise increase in CBF has been linked to the activation of mechanosensitive Group III muscle afferents and feedforward signals related to the passively moved limb(s) that stimulate the primary somatosensory and motor cortices to increase cardiac output and stroke volume via cerebral autoregulation (Nóbrega and Araujo, 1993; Gladwell and Coote, 2002; Amann, 2012). Accordingly, passive exercise provides a basis to evaluate a link between CBF and postexercise EF independent of the metabolic and intensity demands of volitional muscle activation.

#### *4.2 Active and passive exercise produce an immediate postexercise reduction in antisaccade RT*

Active and passive exercise produced an immediate postexercise reduction in antisaccade RTs and is a result that cannot be attributed to a practice-related performance benefit given that the non-exercise control condition showed that antisaccade RTs did not reliably differ from pre- to immediate post-assessment intervals. As well, the result cannot be linked to a strategy designed to reduce RT to enhance response accuracy (i.e., speed-accuracy trade-off) (Fitts, 1954) given that saccade durations and saccade gains did not differ across assessment intervals. Another important finding is that the immediate postexercise RT reduction was specific to antisaccades; that is, prosaccade RTs did not differ from the pre- to immediate postexercise intervals. This result demonstrates that the exercise conditions did not simply improve physiological and/or psychological arousal (see Ayala and Heath, 2021) or provide a general improvement in the speed of information processing (for review see, Chang et al., 2012). If that were the case, then immediate postexercise RTs for pro- *and* antisaccades would have shown a performance benefit. Instead, the antisaccade RT findings for the active exercise condition are consistent with a wealth

of evidence that a single bout of exercise provides an immediate and selective benefit to EF (for review see Diamond, 2013). Additionally, that passive exercise elicited a similar benefit supports previous work by our group (Shirzad et al., 2022) and when combined with the condition's baseline to steady state increase in systolic MCAv suggests that an increase in CBF independent of the metabolic demands of active exercise relates – and possibly contributes – to improved EF (i.e., the hemo-neural hypothesis) (Moore and Cao, 2008).

Two issues related to the immediate postexercise reduction in antisaccade RTs require addressing. First is our observation that light intensity active exercise improved EF. At initial glance this may represent a surprising finding given that the majority of the active exercise literature has reported that positive benefits are restricted to moderate to heavy intensity exercise (i.e., the inverted-U theory) (for meta-analysis see Chang et al., 2012). It is, however, important to recognize that the aforementioned meta-analysis was limited given that a paucity of work had directly examined EF benefits following light intensity exercise. With the growth of the exercise neuroscience literature there is now compelling evidence to assert that very light and light intensity active exercise benefits EF (Morris et al., 2020; Tari et al., 2021; Hyodo et al., 2021). The second issue relates to the magnitude of the postexercise reduction in antisaccade RTs across the active and passive exercise conditions. Given the potential relationship between CBF and EF, one might predict the active exercise condition's larger baseline to steady state increase in CBF would give rise to a larger reduction in postexercise antisaccade RTs. In addressing this issue, null hypothesis testing indicated that antisaccade RT difference scores for passive and active exercise conditions did not reliably differ; however, **Figure 3** qualitatively shows a larger difference score was observed in the latter condition and a two one-side test (TOST) statistic (for review of equivalence tests see, Lakens, 2017) indicated that values were not within an

equivalence boundary. Hence, an identified limitation of the present study is that we are unable to assert whether active and passive exercise produce an equivalent postexercise EF benefit.

#### *4.3 Active – but not passive – exercise produces a persistent reduction in antisaccade RTs*

The active exercise condition showed a selective improvement in antisaccade RTs at the immediate and 30-post – but not the 60-post assessment – a result matching an earlier study by our group employing a 20-min single bout of heavy intensity aerobic exercise (Shukla and Heath, 2018). Given the time required to complete each oculomotor assessment (i.e., ~17-min) the current results indicate that an EF benefit persisted up to ~47-min postexercise. Although the persistence of the EF benefit is longer than reported in meta-analyses (i.e., 15-20-min; see Lambourne and Tomporowski, 2010; Chang et al., 2012) it does accord previous work employing inhibitory control and planning tasks comparable to antisaccades (Joyce et al., 2009; Hung et al., 2013; Shukla and Heath, 2022). We therefore believe that results for our active exercise condition add importantly to the literature inasmuch as they demonstrate that the timeframe by which a single bout of light intensity exercise benefits EF is on par to that associated with moderate to heavy intensities.

The passive exercise condition showed a reduction in antisaccade RTs at the immediate postexercise assessment but not the 30-post or 60-post assessments. Accordingly, passive exercise improved EF for ~17-min – a timeframe that is less than the active exercise condition. In addressing this difference, we note that along with an increase in CBF, active exercise increases catecholamine (e.g., epinephrine, dopamine) and neurotrophin (e.g., BDNF) (Lambourne and Tomporowski, 2010; Zouhal et al., 2008; Knaepen et al., 2010) concentrations and supports improved resting state functional connectivity within EF networks for up to 30-min postexercise (Schmitt et al., 2019). These changes combined with increased CBF are

thought to benefit EF. To our knowledge, no literature has examined whether passive exercise induces a similar response. This is unlikely given that increased peripheral blood lactate associated with volitional muscle activation is a key signalling mechanism for an exercise-based increase in the aforementioned biomolecule concentrations and their putative impact on resting state functional connectivity (for review, Müller et al., 2020). Hence, it may be that passive exercise does not produce the requisite metabolic changes necessary to elicit a temporally persistent benefit to EF. Regardless, the passive exercise results add importantly to the literature inasmuch as they provide a timeline for understanding the persistence of passive exercise EF benefits and how such benefits may translate to individuals requiring passive exercise to account for limited (e.g., hemiparesis) or absent (i.e., spinal cord injury) mobility.

#### *4.4 Study Limitations*

We recognize that our study is limited by several methodological aspects. **First, we examined only healthy young adults deemed physically active. As a result, our findings cannot be extended across the continuum of low- to high-fit individuals, older adults, or to those individuals with compromised CBF and prodromal Alzheimer's disease.** Second, the single bout sessions of active and passive exercise were 20-min in duration. It is therefore unclear whether shorter or longer durations would produce similar postexercise EF benefits. Third, TCD does not take into consideration vessel diameter when assessing changes in MCAv. This is a possible limitation because the MCA is capable of dilation and constriction in response to changes in CO<sub>2</sub> concentration (Coverdale et al., 2015). To our knowledge, however, such changes have not been shown to influence the validity of TCD in evaluating exercise-related changes in MCAv. Fourth, subtle muscle contractions and/or an increase in ventilation may have been present during the passive exercise condition. This is a salient issue because any muscle contraction may evoke a

hemodynamic response and thus increase CBF. In addressing this concern, the supplemental data presented here demonstrated that the passive exercise condition did not elicit agonist muscle activation or increase ventilation ( $\dot{V}_E$ ) or gas exchange ( $\dot{V}O_2$ ,  $\dot{V}CO_2$ ) variables (Bell and Duffin, 2004) (see also **Supplementary Figure 1 and Supplementary Figure 2**). Fifth, we note that group mean RTs for control condition pro- and antisaccades showed a trend (albeit non-significant) for smaller values as a function of each successive post-assessment time frame – a result that could be interpreted to reflect a practice-related performance benefit. That said, a number of purpose-designed studies have reported that antisaccade RTs are immutable to practice effects (Dyckman & McDowell, 2005; see also Klein and Berg, 2001; Ettinger et al., 2003). Moreover, if a practice effect served as a viable account then it would be expected to assert an influence on pro- and antisaccade RTs across passive and active exercise condition as opposed to selectively influencing the control condition. Sixth, results did not indicate a reliable relationship between antisaccade RT difference scores and baseline to steady-state changes in MCAv (see also Tari et al., 2021). To better elucidate such a relationship, we recommend future work employ a larger sample size to better understand the putative relationship between CBF and postexercise EF. Last, we demonstrate that MCAv is differentially elevated during passive and active exercise and these modalities elicit temporally distinct EF benefits. However, it is likely that passive and active exercise elevated CBF via distinct mechanisms which may alter cerebral perfusion and cognition to different degrees. The present study is therefore unable to assert a causative link between elevated CBF and an EF benefit. In spite of these limitations, the present findings provide an important addition to the literature given that they provide a first demonstration of the temporal persistence associated with active light intensity exercise relative to passive exercise.

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490 **6. Declarations of Interest**

491 None.

492 **7. Research Data**

493 The datasets of the current study are available from the corresponding author upon reasonable  
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## Figure Captions

**Figure. 1.** Schematic of the timeline of control (green), passive exercise (blue) and active exercise (red) condition measurement-related events. The “gears” and “lightning bolt” icons next to the cycle ergometer symbol in the passive and active exercise conditions represent mechanical and volitional movement of the ergometer flywheel, respectively. The “eye” symbol in each condition represents an oculomotor assessment. The checkered and solid rectangles serve to represent the resting baseline and warm-up and cool-down periods, respectively, and the bottom grey rectangle indicates the period of time blood velocity was sampled via TCD. The bottom arrow serves to represent the timing by which each oculomotor assessment was initiated following the end of the control, passive exercise and active exercise conditions.

**Figure. 2.** The left panels depict an exemplar participant’s blood velocity through the middle cerebral artery during the first 24-min of the control (i.e., green), passive exercise (i.e., blue) and active exercise (i.e., red) conditions. Solid grey rectangles depict the warm-up period (i.e., 120 s – 240 s) and striated rectangles correspond to the time period from which baseline (i.e., 60 s – 120 s) and steady state (i.e., 1380 s – 1440 s) means were derived. The cool-down period is not represented in this figure. The right offset panels depict group (top) and participant specific systolic MCAv difference scores (i.e., steady state minus baseline) across all three conditions. Error bars in the top offset panel represent 95% between-participant confidence intervals for each condition.

**Figure. 3.** The left panels depict group mean reaction time (RT) for pro- (top panel) and antisaccades (bottom panel) with associated 95% within-participant confidence intervals at each oculomotor assessment for the control (i.e., green), passive exercise (i.e., blue) and active exercise (i.e., red) conditions. The right offset panels depict participant-specific pro- (top panel)

699 and antisaccade (bottom panel) difference scores for the immediate (post, i.e., post- minus pre-),  
700 30-post (30+, i.e., post-30 minus pre-) and 60-post (60+, i.e., post-60 minus pre-) assessments  
701 and error bars represent 95% between-participant confidence intervals. Notably, because of the  
702 multiple comparisons employed here, an asterisk indicates a reliable difference from pre-  
703 assessment RTs ( $\alpha=0.017$ ).



Figure 1

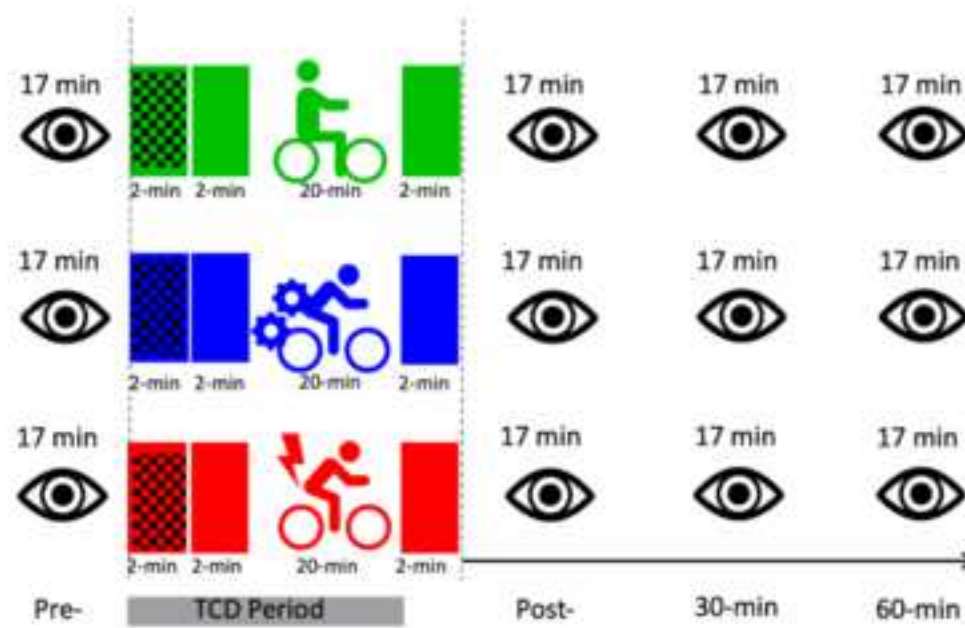


Figure 2

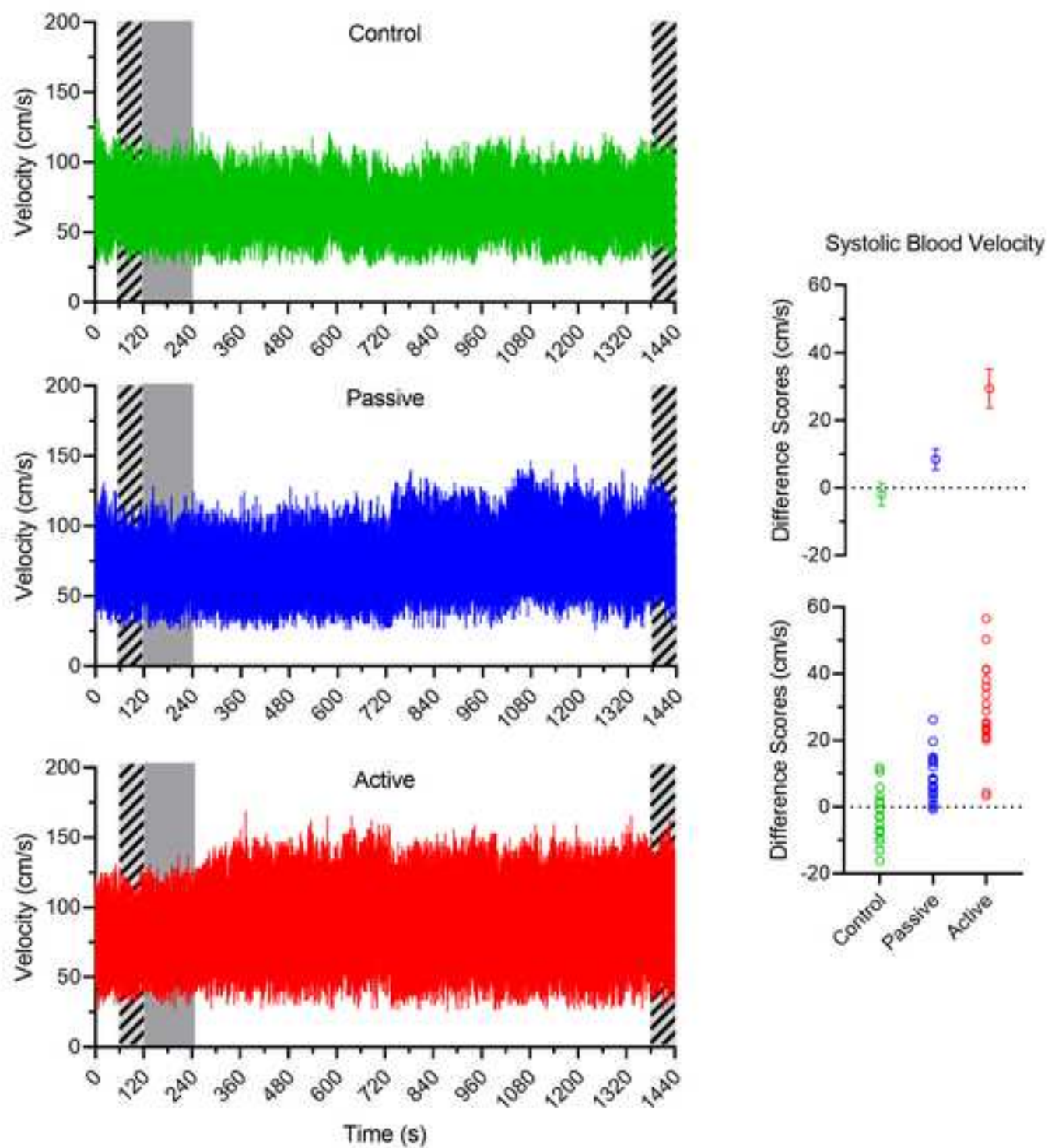
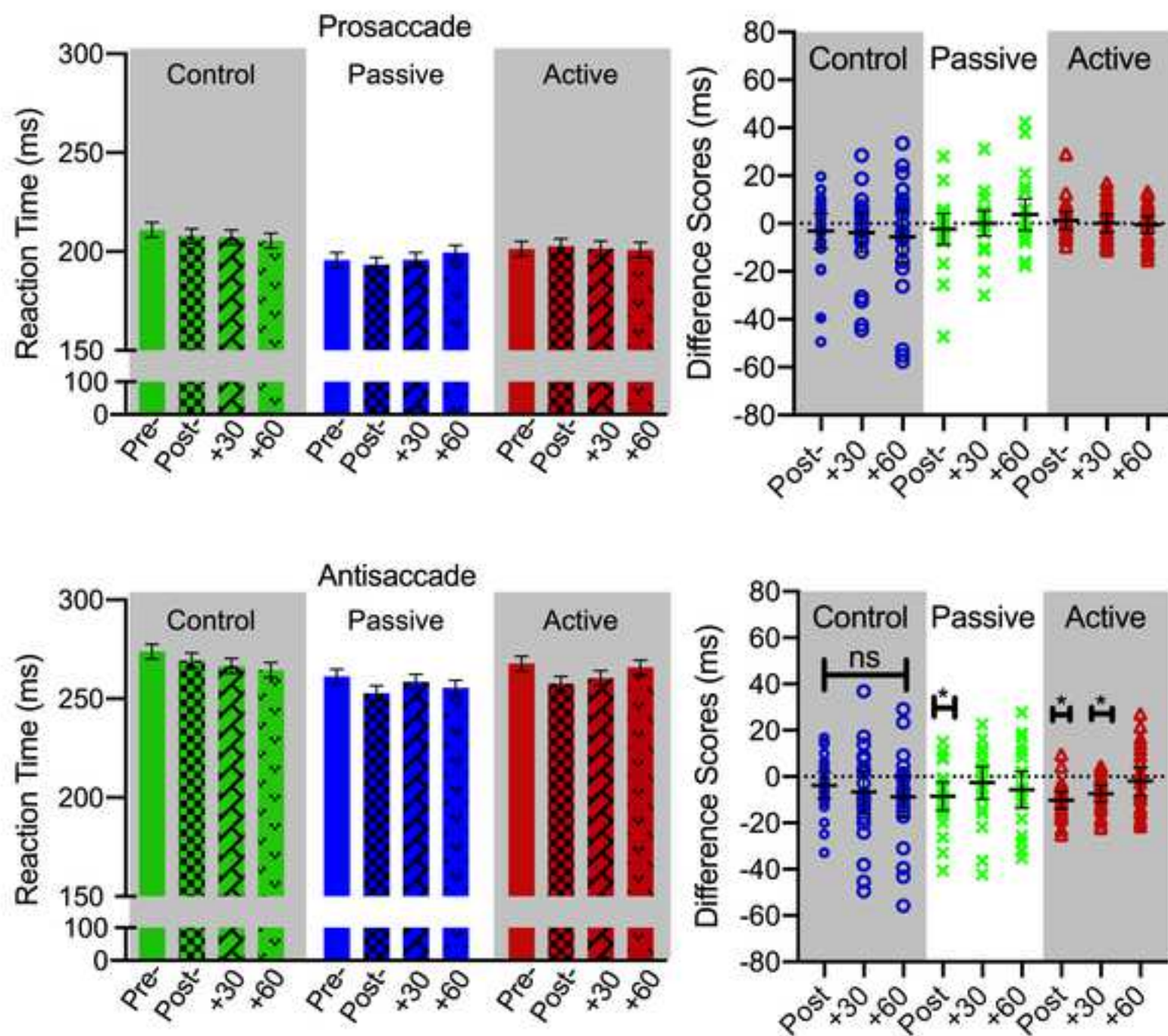


Figure 3



### **CRedit author statement**

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