

## Body fat predictive of acute effects of exercise on prefrontal hemodynamics and speed

James Crum, Ph.D.<sup>a,§,\*</sup>, Flaminia Ronca, Ph.D.<sup>b</sup>, George Herbert<sup>a</sup>, Estela Carmona<sup>a</sup>, Isla Jones<sup>a</sup>, Uzair Hakim<sup>c</sup>, Mark Hamer, Ph.D.<sup>b</sup>, Joy Hirsch, Ph.D.<sup>c,d,e,f</sup>, Antonia Hamilton, Ph.D.<sup>a</sup>, Ilias Tachtsidis, Ph.D.<sup>c</sup>, Paul W. Burgess, Ph.D.<sup>a</sup>

<sup>a</sup> Institute of Cognitive Neuroscience, University College London, London, UK

<sup>b</sup> Institute of Sport Exercise and Health, University College London, London, UK

<sup>c</sup> Department of Medical Physics and Biomedical Engineering, University College London, London, UK

<sup>d</sup> Brain Function Laboratory, Department of Psychiatry, Yale School of Medicine, New Haven, CT, USA

<sup>e</sup> Department of Neuroscience, Yale School of Medicine, New Haven, CT, USA

<sup>f</sup> Department of Comparative Medicine, Yale School of Medicine, New Haven, CT, USA

<sup>§</sup> Institute of Cognitive Science, University of Colorado, 1777 Exposition Dr, Boulder, CO, USA

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### 1. Introduction

Considering the well-established positive effects that physical exercise has on physiological systems, such as reducing body fat, lowering the risk of diabetes, breast cancer, colon cancer, heart disease and stroke, among many others (Kyu et al., 2016), together with the fact that the mind is realized through a physiological system (i.e., the brain), it is plausible to assume that physical activity might have similar beneficial effects on cognition. Research linking physical movement and exertion with cognition has gained increasing interest, with the last two decades seeing a marked increase in the frequency of investigations adopting cognitive neuroscientific methods (e.g., Herold et al., 2020a,b, 2021; see Pontifex et al., 2019, for review). A historical starting point for theorising in this field is Yerkes and Dodson's (1908) postulation that the interaction between arousal and performance follows an inverted-U curve, according to which performance increases as arousal increases, but only to a certain point: Too much arousal for too long detracts from performance. In information-processing terms, Kahneman (1973)

argued that increases in physiological arousal increases the availability of cognitive resources for tasks, and it is this effect which mediates the relationship between physical activity and cognitive performance. Theories accounting for the multidimensional interrelationships between arousal, cognition, and performance are referred to as 'cognitive-energetical' and 'allocatable resources' theories (e.g., McMorris and Graydon, 1996; Sanders, 1983; see McMorris, 2016, for review of historical accounts of the study of brain-body interactions related to exercise). More specifically, these approaches predict that task performance should improve if there is a greater availability of resources, because this facilitates the efficiency with which they can be allocated to the cognitive operations required of a task (e.g., Hockey, 1997). However, a competing account, termed the 'reticular-activating hypofrontality model' (Dietrich and Audiffren, 2011), is predicated on the distinction between control and automatic cognitive processes and, more specifically, on the idea that acute bouts of aerobic activity have differential effects on automatic and controlled information-processing systems in the brain. Namely, exercise is so inherently taxing on the systems

\* Corresponding author. Institute of Cognitive Neuroscience, UCL (University College London), 17 Queen Square, London WC1N3AZ, UK.  
E-mail address: [James.Crum@Colorado.edu](mailto:James.Crum@Colorado.edu) (J. Crum).

supporting movement and balance that any upregulation of resources is preferentially allocated to these systems, reducing the resources available for controlled processing whilst exercising. That is, exercise should result in worse performance on tests of controlled processing if the availability of cognitive resources is constrained by the motor and visual demands of bodily movement.

### 1.1. Acute effects of exercise and intensity on cognition

However, predictions from this perspective have been inconsistent with findings from studies examining the acute effects of exercise on cognitive control processes, and with studies of cerebral oxygenation and blood flow (see [Rooks et al., 2010](#), for review). More specifically, cognitive neuroscientific studies have suggested that there are acute effects of exercise on multiple types of cognition from just a single bout, such as improvements in response time and accuracy ([Hillman et al., 2003, 2009](#); [Kamijo et al., 2009](#); [Kamijo et al., 2007](#); [O'Leary et al., 2011](#); [Pontifex et al., 2013](#)), including cognitive control or 'executive functions' ([McMorris, 2016](#)). Executive function is an umbrella term used in cognitive neuroscience to refer to a group of cognitive faculties that enable people to adapt new ways of behaving, such as inhibition, planning, prospective memory, analogical reasoning, and so forth—all of which are supported to a large degree by brain structures in the frontal lobes of the brain, particularly the prefrontal cortex (PFC). Interestingly, the acute nature of exercise-induced improvements to executive functions, and cognition more broadly, means that these effects remain for a limited window of time, with meta-analyses suggesting that although these effects can last up to 2 h, the largest effects fall within a 15–20-min period after exercise ([Chang et al., 2012](#); [Lambourne and Tomporowski, 2010](#)). In addition, the type of exercise and its intensity have also varied in this domain of research. Specifically, exercise interventions typically consist of aerobic exercises, which involve continuous activities such as walking, running, and cycling, where the intensity can be defined into three categories: mild, moderate, and vigorous. More specifically, mild, moderate, and vigorous exercise are specified as <40% maximum oxygen uptake (VO<sub>2</sub>max), 40–79% VO<sub>2</sub>max, and ≥80% VO<sub>2</sub>max, respectively ([McMorris, 2016](#)). A meta-analysis of exercise intensity studies has shown that, during and just after moderate exercise, the 'speed of processing' across executive functions generally improves, with some reductions in accuracy depending on the type of task and intensity ([Smith et al., 2010](#); see [McMorris and Hale, 2012](#) for review). For example, [Kamijo et al. \(2004\)](#) showed that behavioural changes were optimal when exercise intensity was between low and vigorous (i.e., medium intensity condition).

### 1.2. Hemodynamics, task performance, and current limitations

So, the evidence is largely trending towards the interpretation that acute exercise increases the availability of cognitive resources, and potentially the efficiency with which they are allocated to mental tasks, but the neurophysiological mechanisms driving these interactions between physical activity and cognition are less well understood in humans. One explanation involves considering the chief factor on which both exercise and cognitive functioning directly depend: hemodynamics. Exercise is itself a cognitive task, and all cognitive tasks depend on brain regions which need to receive oxygen; therefore, the upregulation of cerebral blood during physical activity might largely explain the positive effects of exercise on cognition (but see [Lucas et al., 2012](#)). Meta-analytic findings have supported the idea that cerebral blood flow increases during exercise ([Yu et al., 2021](#)), particularly when the activity is moderate to heavy ([Rooks et al., 2010](#)). More specifically, this upregulation consists of both increases in cerebral blood velocity, as measured by transcranial doppler ultrasound ([Lupetin et al., 1995](#)), and oxygenation, as measured by functional near-infrared spectroscopy (fNIRS). See [Ando \(2016\)](#) for further review on cerebral oxygenation.

However, what remains unclear is whether these exercise effects on

hemodynamics are global across the PFC, in that subregions are recruited which are typically not active during a task, or more specific to the role of particular subregions in the task(s) of interest. These relations are unclear in part because previous research has obtained only partial coverage of the PFC, such that activity is measured only from one hemisphere (e.g., [Yanagisawa et al., 2010](#)) or there are few channels over a single bilateral subregion (e.g., [Endo et al., 2013](#)). This can create some ambiguity in interpreting results, but coverage of multiple PFC subregions on both hemispheres should be able to better address this problem; this was therefore one of the aims of the present study. Another limiting factor in the investigation of exercise-induced changes in hemodynamic activity in the brain is the possibility that observed changes in HbO<sub>2</sub> do not reflect information processing. More specifically, the upregulation of extra-cerebral blood flow and artifacts of motion increase the noise in signals purporting to show activation changes in the brain, potentially washing out the neural signatures of the task ([Ekkekakis, 2009](#)). In addition, cerebral oxygenation is particularly modulated by respiration demands ([Zhang et al., 2019](#)). However, deoxygenated haemoglobin (HbR) signals are less affected by systemic confounds ([Dravida et al., 2017](#)), especially in fNIRS paradigms involving marked changes in arterial CO<sub>2</sub> due to changes in respiration ([Scholkmann et al., 2013a, 2013b](#)). A recent methods paper by [Pinti et al. \(2019\)](#) discusses how some of these physiological confounds can be accounted for when estimating single-subject design matrices by having collected additional data, such as heart and respiration rates ([Pinti et al., 2019](#)).

Neuroimaging experiments investigating exercise effects can also reduce the dangers to data validity at the level of experimental design: namely, measuring cognition immediately before and after exercise rather than during it using a repeated-measures design. For example, [Endo et al. \(2013\)](#) used this approach to investigate the effects of different aerobic demands on response inhibition using the Stroop task. They found improvements in response inhibition (15 min post-exercise) were coupled with increases in PFC activation compared to measures taken before exercise. [Faulkner et al. \(2016\)](#) later replicated these findings using the same task and exercise (cycling) at 30 min post-exercise. Researchers using other tasks have also linked acute improvements on executive function tasks with increases in cerebral oxygenation to the PFC ([Byun et al., 2014](#); [Hyodo et al., 2012](#); [Yanagisawa et al., 2010](#)). These findings contradict the predictions of the 'reticular-activating hypofrontality' model ([Dietrich and Audiffren, 2011](#)) of acute exercise. Instead, they are more consistent with the idea that increases in physiological arousal increase the availability of oxygenated haemoglobin for cognitive resources ([Kahneman, 1973](#)), regardless of their modulatory nature. However, it is possible that the issues above with design and methodology might have influenced the findings in favour of this interpretation. Addressing these shortcomings in exercise research on the PFC will bolster greater confidence in the link between physical activity, hemodynamic changes, and cognitive performance.

### 1.3. The present study

If exercise induces changes in the availability of cognitive resources via changes in oxygenated haemoglobin, then this 'availability' might show a generalized recruitment of PFC subregions; however, if greater availability also engenders greater efficiency in satisfying the resource demands of cognitive functions, then resource consumption might appear more region-specific on individual task conditions (e.g., attentional monitoring, attentional mode). Therefore, the aim of this study was to examine the acute effects of exercise on behaviour and the PFC. More specifically, we investigated whether performing executive function tasks shortly after vigorous exercise would result in activation changes that are specific to the operational demands of the PFC subregions typically supporting these tasks, hypothesizing also that global changes in local hemodynamics across tasks will correspond with

improvements in behavioural performance, specifically decreases in response times. Secondly, we sought to discover whether the post-exercise haemodynamic changes and changes in cognitive performance could be predicted by individuals' bodily or physiological characteristics, such as cardiopulmonary fitness levels, age, birth sex, and body fat.

## 2. Method

### 2.1. Participants

One-hundred and six participants were randomly allocated into an exercise group ( $n = 106$ ; 73% male; age:  $39 \pm 9$  years; weight  $84.0 \pm 19.7$  kg; height  $173.6 \pm 20.2$  cm), and a control group ( $n = 27$ ; 97% male; age:  $43 \pm 6$ ; weight  $89.4 \pm 18.6$  kg; height  $177.7 \pm 9.4$  cm). Sample size was determined from pilot data indicating the likely effect size for these cognitive tasks and this equipment and analysis protocol (Pinti et al., 2020). All participants completed a physical activity readiness questionnaire (PAR-Q; Pescatello, 2014) to screen for eligibility to undergo exercise test to volitional exhaustion and provided written informed consent prior to participating in the study. Participants were excluded from the study if they presented any injury or illness that prevented them from exercising to exhaustion, if they had a neurological condition or if responded "yes" to any of the questions on the PAR-Q. Ethical approval was granted by the University College London Research Ethics Committee in line with the declaration of Helsinki.

### 2.2. Cognitive tasks

While the exercise group completed neurocognitive testing before and after exercise, the control group completed neurocognitive testing before and after resting, and then underwent exercise testing after the cognitive testing was complete to gather their fitness data. The experimental design (Fig. 1) consisted of a time manipulation (neurocognitive testing before and after exercise) and cognitive manipulations within each neurocognitive testing session.

These data were collected whilst participants completed a set of cognitive tasks on a computer screen. The cognitive tasks were created in PsychToolbox, MATLAB (Mathworks, Natick, MA) to test aspects of executive function (inhibition and attention). The response inhibition task consisted of two conditions: Go and No-Go. The Go condition was a simple reaction time (SRT) task in which participants were required to respond (press the spacebar) each time an image appeared on the screen between instances of a fixation cross (jittered). The No-Go condition was the same as the previous one, with the exception that participants were instructed to not respond to specific images, particularly kittens: "Do not 'shoot' the kittens" (Fig. 2).

There was also an attention task which measured people's ability to maintain stimulus-oriented vs. stimulus-independent modes of attending. This task (called the Alphabet Test) was first published by Gilbert et al. (2005), and subsequently used many times in this lab to provoke activations in rostral PFC and surrounding brain areas (see



Fig. 2. Example trials of the Go/No-Go condition of the response inhibition task (images: Public Domain) Stimuli of kittens represented No-Go trials.

Burgess and Wu, 2013 for review). The Alphabet Task had different conditions, which required participants to respond in particular ways depending on whether they were attending to stimuli on the computer screen (i.e., exogenous attending) or to stimuli independent of it—i.e., in their minds only (i.e., endogenous attending). More specifically, the first of these conditions was a stimulus-oriented thought (SOT; also known as exogenous attending) task in which participants pressed one of two keys depending on whether a given letter in the alphabet that was presented on the screen contained any curved lines. The next condition was a stimulus-independent thought (SIT; also known as endogenous attending) condition in which they were required to respond in the same way as the previous task to each letter in the alphabet, but no letters were shown on the screen; they needed to represent them mentally (Fig. 3). See Burgess et al. (2007) for more background on SOT/SIT tasks.

The last set of cognitive tasks involved memory conditions (Fig. 4). First, in the encoding condition, participants were shown a series of images of urban backgrounds with either a picture or description of a person and their vocation. Participants were given two potential jobs to choose from using the arrow keys and were asked to decide the job of the stimulus. The next condition was a recognition task in which participants were shown just the backgrounds of the previous task, as well as some new ones, and needed to decide if they have seen them before. The final condition was a source memory task in which participants were required to recall whether the jobs they were shown in the encoding phase were presented as images or descriptions.

These cognitive tasks were chosen on the grounds that they would, on existing evidence both from our lab and others, potentially show changes in the prefrontal brain regions we were recording from. Thus, the endogenous vs exogenous (also known as a SO/SI attending switch task), called the Alphabet Task, has been previously shown to activate rostral PFC in particular (approximating area 10), with simple reaction time and exogenous attending mode tasks typically activating more anterior medial rostral PFC regions, and endogenous (SI) conditions typically activating more lateral rostral PFC regions (Burgess et al., 2007). Inhibition tasks are often found to activate, in standard cognitive neuroscience experiments, lateral inferior aspects of prefrontal cortex, and sometimes also orbitofrontal regions (see e.g. Zhang et al., 2017). Source memory tasks typically activate rostral aspects of prefrontal cortex (PFC), but also lateral and superior lateral PFC (BA 8, 9 and 46; see e.g. Turner et al., 2008). Thus together, *a priori*, this battery of tasks



Fig. 1. Testing sessions. After participants in the exercise group completed several cognitive tests (1), they engaged in a battery of strength tests, including press-ups, hand-grip strength, and countermovement jump height (2). Next, they were required to complete a 15-min bleep test of aerobic fitness, namely a VO<sub>2</sub>max test (3) and, after a cooldown period, completed similar cognitive tests (4).

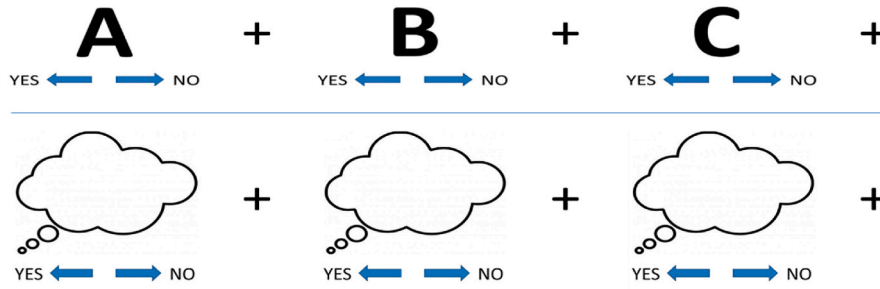


Fig. 3. Example trials of the exogenous (stimulus-oriented thought) and endogenous (stimulus-independent thought) attending conditions in the upper and lower rows, respectively.

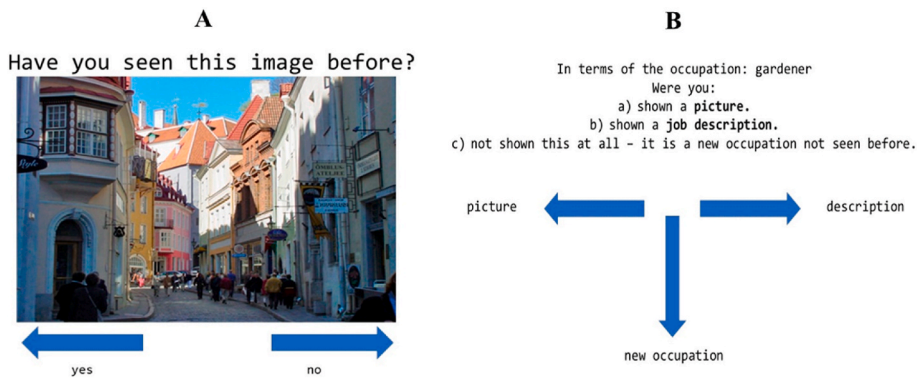


Fig. 4. Example trials of the recognition (A) and source (B) memory conditions. After being shown stimuli in the encoding task, participants indicated whether they recognised stimuli and recalled the source of nature of encoding information (e.g., picture vs. description), respectively.

enabled us to maximise our chances of detecting exercise-related wherever they might occur in prefrontal cortex, within the constraints of our method and the time available. The order of the administration of the tasks was kept the same for each participant and each testing session, to avoid risking complex individual differences in strategy development or effort according to task order (this can be a special issue when testing executive functions, see Burgess, 1997; Burgess and Stuss, 2017 for further explanation). The order was: Baseline, SRT, Inhibition, SO/SI attending mode control, Source Memory, Baseline.

After the first cognitive testing session, participants completed four strength tests and a  $VO_{2max}$  test (described below) whereas the control group passively viewed a video for 30 min, which was equivalent to the amount of time required for the exercise group to complete the fitness tests. Immediately after terminating the  $VO_{2max}$  test on a treadmill, participants walked for 3 min at 6 km/h, they were allowed a drink of water only and then returned immediately to the neurocognitive testing room to repeat the same cognitive tests. Fitting the fNIRS cap required 5 more minutes, while the participant sat quietly, giving a total recovery time of about 10 min between the end of the  $VO_{2max}$  test and the start of the second time of testing on the cognitive test battery. The second round of cognitive tests had counterbalanced blocks containing different stimuli. Behavioural, fNIRS, and systemic data were collected again.

### 2.3. Fitness and strength testing

Participants were instructed not to eat large meals for up to 2 h before testing, and to not consume caffeine for 24 h prior to testing. Anthropometric data (height and weight) were recorded prior to exercise testing. Body composition was analysed via bioelectrical impedance on a Tanita MC980MA (Tanita Cooperation, Tokyo, Japan). The exercise testing consisted of a series of strength tests and a  $VO_{2max}$  test. All exercises were demonstrated first by the researchers and participants

were allowed familiarization trials. Participants first warmed up on a cycle ergometer at 60 rpm for 5 min, at a rate of perceived exertion of 6/10. Four minutes rest were given between strength tests to prevent fatigue. Participants completed a push-up test, a triple hop jump, a counter-movement jump (CMJ) and a hand grip test. For the push-up test, participants were instructed to complete as many push-ups as possible in 1 min without breaking form. Next, for the triple hop jump test, participants were instructed to cover the farthest distance possible by hopping three times on one leg without stopping or stumbling. Three jumps were completed per leg, the farthest distance was recorded. For the CMJ, participants were instructed to place hands on hips, squat down and immediately jump up with straight legs maintaining hands on hips. Three CMJs were performed using a contact platform (Chronojump-Biosystems, Barcelona, Spain), the highest jump was recorded. Hand grip strength was measured as the best of three trials with a T.K.K. 5001 grip strength dynamometer (Takei Scientific Instruments Co., Niigata, Japan). After the strength tests, participants completed a Bruce protocol test (Myers et al., 2000) on a treadmill (h/p/cosmos, Nussdorf, Germany). The protocol begins with 3 min walking (2.6 km/h) with no incline; every 3 min thereafter, the incline and speed of the treadmill are increased. Throughout the test, the participant is encouraged to continue exercising until volitional exhaustion, at which point the test is terminated, the treadmill returns to level at walking pace, the participant is instructed to walk slowly (2.6 km/h) and in silence for 3 min to recover fully. Breath-by-breath gas analysis and heart rate (HR) were gathered through the Vyntus CPX Metabolic Cart (Vyair Medical, Chicago, USA) throughout the test. The anaerobic threshold was determined using the v-slope method, as the point of departure from linearity of carbon dioxide output ( $VCO_2$ ) plotted against oxygen uptake ( $VO_2$ ) (Wasserman et al., 1994).  $VO_{2max}$  (ml/kg/min) was determined as the highest recorded  $VO_2$  value.  $VO_{2max}$  was identified if the gas analysis showed a plateau in the  $VO_2$  values, if respiratory exchange ratio (RER)



> 1.13 and if heart rate max reached approximately 220-age. Participants who did not reach these criteria were excluded from the study. VO<sub>2</sub>max values were recorded as ml/min/kg, VO<sub>2</sub>max rating was determined as per the American College Sports Medicine normative values (ACSM, 2014; Pescatello et al., 2014), where scores are categorized into percentiles according to age and gender. See Table 1 for fitness test outcomes of the exercise and control groups.

2.4. Signal acquisition and regions of interest (ROIs)

Functional NIRS signal acquisition of hemodynamics was acquired using a 16-fiber (22-channel configuration: 8 sources & 8 detectors) continuous-wave fNIRS system (LIGHTNIRS, Shimadzu Corp., Kyoto, Japan) sampled at a rate of 13.33 Hz at three wavelengths of light (780, 805, and 830 nm). A light-emitting diode probe (Daiso Crop., Hiroshima, Japan) was used to achieve an orthogonal connection between the fNIRS optodes and scalp (i.e., to displace hair in the cap). Due to the large sample size and need for rapid-testing, digitization was based on a single subject. Anatomical locations of optodes in relation to standard head landmarks, includinginion and top center (Cz) and left and right tragi, were determined using a Patriot 3D Digitizer (Polhemus, Colchester, VT) and linear transform techniques (Fig. 5). Researchers were then carefully trained to place the cap in the same way for each participant according to this configuration.

Montreal Neurological Institute (MNI) coordinates (Mazziotta et al., 2001) for each channel were obtained using NIRS-SPM software (Ye et al., 2009; https://www.nitrc.org/projects/nirs\_spm/) with MATLAB (Mathworks, Natick, MA). The anatomical coverage of the channel configuration was corresponded with three bilateral ROIs (Table 2): rostral PFC (BA10), dorsolateral PFC (BA46/9), and ventrolateral PFC (BA44/45/47). These ROIs were specified a priori based on neuroimaging and neuropsychological research on frontal lobe functions (for reviews see Knight and Stuss, 2013; Shallice and Cooper, 2011).

2.5. fNIRS signal processing

Signal processing of the fNIRS data was carried out in accordance with the quality control standards suggested by Yücel et al. (2021). Data collection errors reduced the sample size of fNIRS dataset (n = 90), in that participants were excluded if they did not have enough behavioral data to match the fNIRS data or if their fNIRS data were too noisy or compromised in some way. The pre-processing of raw fNIRS signals was conducted according to the particular recommendations of Pinti et al. (2019) using functions developed in HOMER2 (https://homer-fnirs.org).

Table 1  
Fitness test outcomes for exercise and control groups.

Variable	Group	
	Exercise Group	Control Group
	M	M
N (male, female)	65, 27	16, 2
Height (cm)	173.6 ± 20.2	177.7 ± 9.3
Weight (kg)	84.0 ± 19.7	89.4 ± 15.6
Fat %	25.2 ± 7.8	24.0 ± 5.0
Age (years)	39 ± 8	43 ± 6
Push-ups per minute (reps)	23.4 ± 16	No data
Counter-Movement Jump height (cm)	23.7 ± 7.7	No data
Counter-Movement Jump power (W)	874 ± 236	No data
Hand Grip (W)	46.2 ± 12.5	No data
VO <sub>2</sub> max at Anaerobic Threshold (ml/min/kg)	27.3 ± 6.6	30.7 ± 7.1
VO <sub>2</sub> max (ml/min/kg)	36.9 ± 9.1	40.1 ± 7.2
Superior	2	0
Excellent	12	1
Good	21	3
Fair	12	2
Poor	14	4
Very Poor	31	2
Heart Rate max (bpm)	170 ± 49	160 ± 51

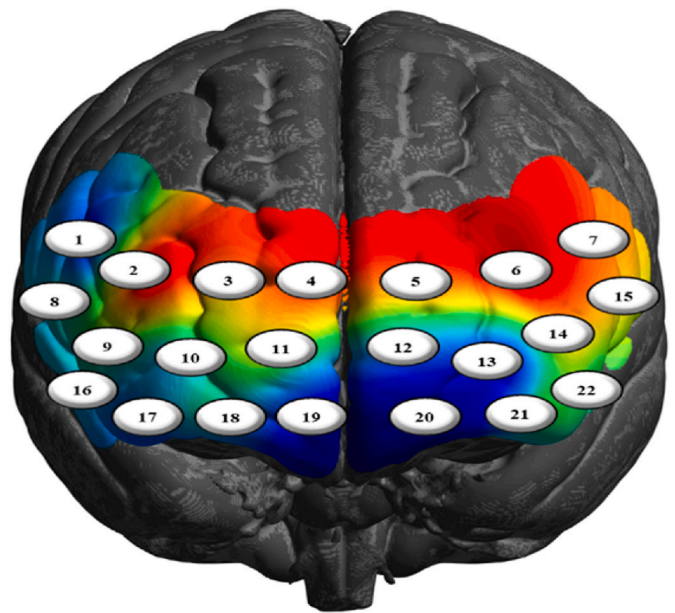


Fig. 5. Channel-specific locations of the 22-channel (8 sources & 8 detectors) configuration overlaid onto a model brain mesh of the PFC with example activation.

Table 2  
Channels, coordinates, and anatomical regions.

Channel #	Anatomical Region	BA <sup>a</sup>	Coordinates <sup>b</sup>
1	Right Superior Frontal Gyrus	9	53, 26, 30
2	Right Middle Frontal Gyrus	46	41, 49, 23
3	Right Rostral PFC	10	25, 62, 21
4	Right Rostral PFC	10	4, 65, 21
5	Left Rostral PFC	10	-19, 63, 21
6	Left Middle Frontal Gyrus	46	-37, 52, 22
7	Left Superior Frontal Gyrus	9	-51, 28, 29
8	Right Inferior Frontal Gyrus	44	60, 15, 16
9	Right Inferior Frontal Gyrus	45	51, 43, 7
10	Right Rostral PFC	10	36, 60, 5
11	Right Rostral PFC	10	17, 68, 5
12	Left Rostral PFC	10	-8, 68, 5
13	Left Rostral PFC	10	-32, 62, 4
14	Left Inferior Frontal Gyrus	45	-47, 46, 7
15	Left Inferior Frontal Gyrus	44	-58, 17, 17
16	Right Inferior Frontal Gyrus	47	54, 30, -6
17	Right Rostral PFC	10	45, 51, -8
18	Right Rostral PFC	10	28, 64, -7
19	Right Rostral PFC	10	3, 67, -7
20	Left Rostral PFC	10	-22, 66, -8
21	Left Rostral PFC	10	-41, 54, -7
22	Inferior Frontal Gyrus	47	-51, 36, -3

Note.  
<sup>a</sup> BA = Brodmann's Area.  
<sup>b</sup> Coordinates are based on the MNI system and (-) indicates left hemisphere.

Specifically, raw voltage intensities were converted from.OMM format to.TXT and, then, into.NIRS format. Next, these data were converted into optical density (OD) signals. Then, motion-artifact correction was conducted using wavelet convolution (iqr = 1.5), with a differential pathlength factor (DPF) that is conventionally used for continuous-wave fNIRS [6, 6, 6]. To further reduce sources of noise in the OD signals and isolate task-related components, these signals were temporally smoothed using a band-pass filter (FIR: order 1000) [0.01 0.4 Hz] to remove extracerebral, systemic effects. The cleaned OD signals were then converted into changes in concentrations of oxygenated hemoglobin (HbO<sub>2</sub>), deoxygenated hemoglobin (HbR), and total hemoglobin (HbO<sub>2</sub> + HbR) using the modified Beer-Lambert Law (see Dirnagl and

Villringer, 1997). To further account for variance attributable to physiological confounds in the data (Tachtsidis and Scholkmann, 2016), HR signals were extracted from the fNIRS data. More specifically, a band-pass filter [0.8 1.6 Hz] was used on each channel for each participant to isolate the HR components. Next, the ten channels of each participant (~1/2 channel configuration) that showed the HR component most clearly via visual inspection were averaged to produce a HR timeseries that spanned across all experimental blocks. These HR signals were included as additional parameters in the single-subject design matrices.

The data were down-sampled to 5 Hz using spline interpolation (Cohen, 2017) and a general-linear model (GLM) analysis—using SPM and NIRS-SPM functions—fitted the predicted models to the observed data, yielding beta estimates for each parameter of the single-subject design matrices. The second-level analysis of the group data used a random-effects approach via summary statistics (Friston et al., 2007; Poldrack et al., 2011) and was channel-based. After false-discovery rate (FDR) correction ( $q < 0.05$ ; Singh and Dan, 2006) was carried out, the group effects of each HbO<sub>2</sub> and HbR contrast for each channel were then projected onto a 3-D brain mesh using the sample digitization coordinates that were normalized to standard MNI space via linear interpolation. Lastly, although all signal analyses were conducted on both HbO<sub>2</sub> and HbR, the interpretation of the results was based on research suggesting that HbR signals are less affected by systemic confounds (Dravida et al., 2017), especially in fNIRS paradigms involving marked changes in arterial CO<sub>2</sub> due to changes in respiration (e.g., exercise, speaking, etc.), because such changes alter the HbO<sub>2</sub> signal to a greater degree than HbR in these cases (Scholkmann et al., 2013a, 2013b).

### 3. Results

As discussed above, the study of exercise using cognitive neuroscience methods is challenged by the facts that (1) exercise upregulates blood flow to extra-cerebral areas; (2) motion from physical activity introduces artifacts (i.e., noise) into hemodynamic signals; and (3) respiration and cerebral oxygenation are closely interrelated. These issues are exacerbated when the intensity of exercise is vigorous; therefore, we used a repeated-measures design to assess changes in brain activation. However, practice effects (i.e., learning) are of some concern for this type of paradigm, so the following analyses aimed to pick apart these effects from those of acute exercise on the brain, starting with the behavioral data of the experimental and control groups, followed by neuroimaging results within- and between-groups, and then predictive interrelations with physical parameters.

#### 3.1. Exercise improved information-processing speed

Descriptive statistics of group median response times (RTs) are shown in Table 3. There were improvements in cognitive performance. In the exercise group, RTs significantly changed after exercise compared to before exercise for all tasks except simple RTs (Table 4). More specifically, Wilcoxon signed-rank tests showed that RTs decreased (i.e., faster performance) for the inhibition and attentional mode conditions and increased for the memory conditions ( $p$ 's < .01). For the executive function tasks in which speed was a key factor (i.e., inhibition and attentional conditions), there was an average increase in speed by 21.18%. There were no significant changes in accuracy. However, the control group had similar increases and decreases in speed—albeit to a lesser extent—in the second neurocognitive testing session, namely RTs decreased in the attentional mode conditions and increased in the memory ones ( $p$ 's < .01), with no significant differences in the go/no-go task. The pre- and post-exercise RTs differed by an average of 16.87%, suggesting that these behavioural changes were likely learning effects from the repeated-measures design, so the question was whether the differences in RT observed in the exercise group were significantly different from that of the control group. To examine this, we summated

**Table 3**  
Descriptive statistics of response times (RTs).

Task	Pre		Post	
	<i>Mdn</i>	<i>Mad</i>	<i>Mdn</i>	<i>Mad</i>
Simple RTs	0.31	0.03	0.29	0.03
Inhibition (No-Go)	0.73	0.06	0.66	0.06
<b>Exogenous (SOT)</b>	0.67	0.16	0.42	0.12
<b>Endogenous (SIT)</b>	0.57	0.22	0.38	0.16
<b>Encode</b>	2.52	0.65	3.00	0.67
<b>Recognition</b>	1.44	0.34	1.82	0.27
Source	2.15	0.42	2.22	0.31
<b>Control Group</b>				
Task	Pre		Post	
	<i>Mdn</i>	<i>Mad</i>	<i>Mdn</i>	<i>Mad</i>
Simple RTs	0.30	0.04	0.32	0.03
Inhibition (No-Go)	0.71	0.06	0.68	0.05
<b>Exogenous (SOT)</b>	0.63	0.11	0.42	0.10
<b>Endogenous (SIT)</b>	0.61	0.16	0.39	0.12
<b>Encode</b>	2.39	0.53	3.29	0.61
<b>Recognition</b>	1.38	0.21	1.91	0.22
Source	1.88	0.39	2.28	0.42

Note. Medians (*Mdn*) and mean absolute deviations (*Mad*) of pre- and post-executive function tasks in the Experimental and Control Groups.

**Table 4**  
Difference descriptive statistics of response times (RTs).

Task	Exercise Group		Control Group	
	Z	p	Z	p
Simple RTs	-1.77	= 0.08	1.5	0.13
Inhibition (No-Go)	-6.95	<.01*	-1.59	0.11
<b>Exogenous (SOT)</b>	-8.11	<.01*	-3.72	<.01*
<b>Endogenous (SIT)</b>	-6.58	<.01*	-2.9	<.01*
<b>Encode</b>	6.38	<.01*	3.72	<.01*
<b>Recognition</b>	6.57	<.01*	3.72	<.01*
Source	2.58	= 0.01*	3.16	<.01*

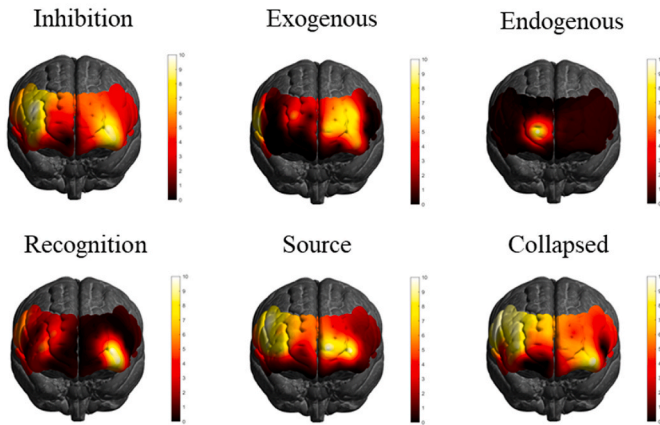
Note. Within-group differences between pre- and post-exercise RTS in the exercise and control groups (\*) indicates a significant statistical difference. Negative and positive Z values represent the direction of change in RTs, such that negative values indicate a decrease in RTs (i.e., increase in speed).

the RTs for each condition of the inhibition and attention tasks for participants' pre- and post-exercise scores. Then, the percent change between sessions was computed for each participant of each group, resulting in composite values of percent change, which were then compared between groups. This showed a significant difference,  $t(106) = 2.04, p = .04$ . In other words, 4.31% of the observed decrease in RTs on speed-intensive tasks in the exercise group was due to the acute effects of exercise. So, exercise appears to have increased participants' information-processing speed on these executive tests; however, it is plausible that other tests might show different effect sizes.

#### 3.2. PFC hemodynamics: pre-versus post-exercise

Within-brain statistical comparisons for the response inhibition, attentional mode, and memory tasks (i.e., subtracting their pre-exercise equivalents) showed significant increases in activation in several PFC subregions (Fig. 6). See Tables 4 and 5 for channel-based statistics. More specifically, inhibition showed significant activation changes in bilateral dorsolateral (BA46/9), ventrolateral (BA44/45), and rostral (BA10) PFC, with the greatest change occurring in right dorsolateral (BA46) PFC (channel 2),  $t(89) = 7.94, p < .01$ . For the attentional mode tasks, the SIT condition (endogenous attending) solely recruited right rostral PFC (channel 11),  $t(89) = 3.46, p < .01$ . The SOT condition (exogenous attending) showed significant activation changes in right ventrolateral (BA44), left dorsolateral (BA46), and bilateral rostral (BA10) PFC

**Acute Exercise Effects**



**Fig. 6.** Within-group hemodynamic changes in the PFC (HbR) as an acute effect of exercise for the inhibition, attentional mode, and memory conditions ( $\alpha < .05$ , FDR corrected), with the ‘Collapsed’ image showing these changes across all tasks. Greatest activation changes are represented in white and bright yellow, with little to no effects represented in dark red and black, respectively (t values of the images are scaled from 0 to 10+).

**Table 5**  
Channel-based Comparisons (HbR signals) of the Acute Effects of Exercise (corrected).

Contrast	Channel	t value	p	BA <sup>a</sup>	Coordinates <sup>b</sup>	
<b>Inhibition</b>	1	5.59	<0.01	9	(53, 26, 30)	
	2	7.94	<0.01	46	(41, 49, 23)	
	3	3.85	<0.01	10	(25, 62, 21)	
	4	3.33	<0.01	10	(4, 65, 21)	
	5	3.43	<0.01	10	(-19, 63, 21)	
	6	3.77	<0.01	46	(-37, 52, 22)	
	7	2.52	= 0.02	9	(-51, 28, 29)	
	8	4.46	<0.01	44	(60, 15, 16)	
	9	6.63	<0.01	45	(51, 43, 7)	
	10	7.08	<0.01	10	(36, 60, 5)	
	13	4.98	<0.01	10	(-32, 62, 4)	
	14	4.06	<0.01	45	(-47, 46, 7)	
	15	2.62	= 0.01	44	(-58, 17, 17)	
	17	5.22	<0.01	10	(45, 51, -8)	
	18	3.60	<0.01	10	(28, 64, -7)	
	20	4.19	<0.01	10	(-22, 66, -8)	
	21	5.64	<0.01	10	(-41, 54, -7)	
	22	3.05	<0.01	47	(-51, 36, -3)	
	<b>Endogenous (SIT)</b>	11	3.46	= 0.01	10	(17, 68, 5)
	<b>Exogenous (SOT)</b>	3	3.10	= 0.01	10	(25, 62, 21)
		5	3.32	<0.01	10	(-19, 63, 21)
		6	4.10	<0.01	46	(-37, 52, 22)
	8	5.01	<0.01	44	(60, 15, 16)	
	12	3.87	<0.01	10	(-8, 68, 5)	
	13	3.72	<0.01	10	(-32, 62, 4)	
	21	3.47	<0.01	10	(-41, 54, -7)	

*Note.*  
<sup>a</sup> BA = Brodmann’s Area.  
<sup>b</sup> Coordinates are based on the MNI system and (-) indicates left hemisphere.

(Table 5).

In addition, the recognition memory condition showed significant activation changes in bilateral rostral PFC (BA10), with peak activation in an area of left BA10,  $t(89) = 6.98, p < .01$  (channel 21). The source memory condition showed significant activation changes in bilateral ventrolateral (BA44), right dorsolateral (BA46/9), and bilateral rostral PFC, with the greatest change occurring in left BA10 (channel 12),  $t(89) = 5.98, p < .01$ . Collapsing across all blocked tasks (as a single contrast: post-exercise > pre-exercise), showed significant activation changes in all PFC subregions, bilaterally (Table 6), including BA46/9, BA44/45,

**Table 6**  
Channel-based Comparisons (HbR signals) of the Acute Effects of Exercise (corrected).

Contrast	Channel	t value	p	BA <sup>a</sup>	Coordinates <sup>b</sup>	
<b>Recognition</b>	1	3.67	<.01	9	(53, 26, 30)	
	3	3.48	<.01	10	(25, 62, 21)	
	10	4.43	<.01	10	(36, 60, 5)	
	13	6.42	<.01	10	(-32, 62, 4)	
	21	6.98	<.01	10	(-41, 54, -7)	
<b>Source</b>	1	5.14	<.01	9	(53, 26, 30)	
	2	3.40	<.01	46	(41, 49, 23)	
	3	3.63	<.01	10	(25, 62, 21)	
	4	2.80	= .01	10	(4, 65, 21)	
	8	3.32	= .04	44	(60, 15, 16)	
	9	4.35	<.01	45	(51, 43, 7)	
	10	3.23	<.01	10	(36, 60, 5)	
	12	5.98	<.01	10	(-8, 68, 5)	
	13	4.54	<.01	10	(-32, 62, 4)	
	15	2.90	= .01	44	(-58, 17, 17)	
	22	2.35	= .04	47	(-51, 36, -3)	
	<b>Collapsed</b>	1	7.23	<.01	9	(53, 26, 30)
		2	7.28	<.01	46	(41, 49, 23)
3		4.89	<.01	10	(25, 62, 21)	
4		5.54	<.01	10	(4, 65, 21)	
5		3.79	<.01	10	(-19, 63, 21)	
6		4.12	<.01	46	(-37, 52, 22)	
7		3.15	<.01	9	(-51, 28, 29)	
8		7.96	<.01	44	(60, 15, 16)	
9		4.00	<.01	45	(51, 43, 7)	
10		6.86	<.01	10	(36, 60, 5)	
11		2.81	= .01	10	(17, 68, 5)	
12		5.19	<.01	10	(-8, 68, 5)	
13		5.76	<.01	10	(-32, 62, 4)	
14		2.77	= .01	45	(-47, 46, 7)	
15		3.45	<.01	44	(-58, 17, 17)	
17	4.37	<.01	10	(45, 51, -8)		
20	4.54	<.01	10	(-22, 66, -8)		
21	8.67	<.01	10	(-41, 54, -7)		
22	2.27	= .03	47	(-51, 36, -3)		

*Note.*  
<sup>a</sup> BA = Brodmann’s Area.  
<sup>b</sup> Coordinates are based on the MNI system and (-) indicates left hemisphere.

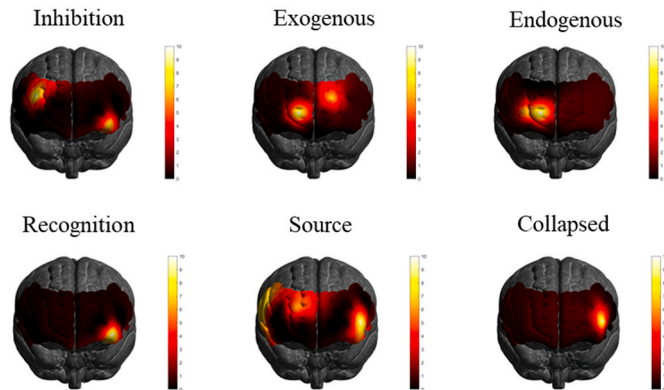
and BA10, with the greatest general change in activation occurring in left BA10,  $t(89) = 8.67, p < .01$ .

**3.3. PFC hemodynamics of acute exercise: control group comparisons**

To assess the degree to which these PFC subregions are indeed affected by exercise, and not by some test-retest effect, comparisons of brain activity between the control group and the exercise group were examined (Fig. 7). More specifically, the activation changes between testing sessions that were unique to the control group were subtracted from those of the exercise group, yielding significant differences for the response inhibition, attentional mode, and memory tasks (Table 7). Namely, inhibition showed significant activation differences in right dorsolateral (BA46) and left rostral (BA10) PFC, with the greatest difference observed again in right BA46,  $t(106) = 4.10, p < .01$ . For the attentional mode tasks, the SIT condition (endogenous attending) showed a significant difference in right rostral PFC,  $t(106) = 4.83, p < .01$ . The SOT condition (exogenous attending) showed significant differences in bilateral rostral PFC,  $t(106) = 5.08, p < .01$ . In addition, the recognition memory condition showed significant activation differences in left rostral PFC,  $t(106) = 3.63, p = .01$ . The source memory condition showed significant activation differences in right dorsolateral (BA9), and bilateral BA47, left BA45, right BA44, and bilateral rostral PFC, with the greatest difference observed in the left BA45,  $t(106) = 6.57, p < .01$ . Collapsing across all blocked tasks showed a significant group difference in left ventrolateral (BA45) PFC,  $t(106) = 2.85, p = .04$ . There were no significant differences in sex.



**Group Effects (Exercise > Control)**



**Fig. 7.** Between-group differences in PFC hemodynamics (HbR) for the inhibition, attentional mode, and memory conditions ( $\alpha < .05$ , FDR corrected), with the ‘Collapsed’ image showing the overall group difference across tasks. Greatest activation changes are represented in white and bright yellow, with little to no effects represented in dark red and black, respectively (t values of the images are scaled from 0 to 10+).

**Table 7**  
Between-Group Channel-based Comparisons (HbR signals) (corrected).

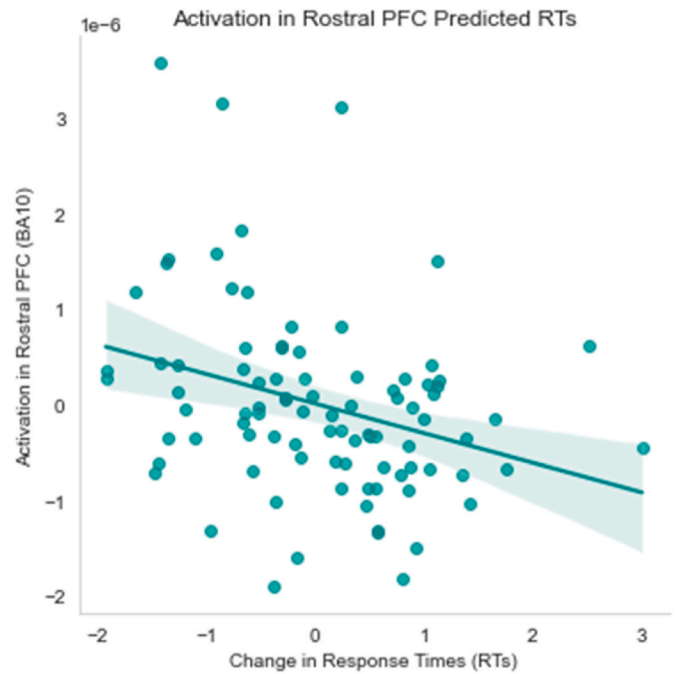
Contrast	Channel	t value	p	BA <sup>a</sup>	Coordinates <sup>b</sup>	
Inhibition	2	4.10	<0.01	46	(41, 49, 23)	
	21	3.33	= 0.01	10	(-41, 54, -7)	
Endogenous (SIT)	10	3.20	= 0.01	10	(36, 60, 5)	
	11	4.83	<0.01	10	(17, 68, 5)	
Exogenous (SOT)	5	3.29	= 0.01	10	(-19, 63, 21)	
	11	5.08	<0.01	10	(17, 68, 5)	
Recognition	21	3.63	= 0.01	10	(-41, 54, -7)	
Source	1	4.43	<0.01	9	(53, 26, 30)	
	3	4.17	<0.01	10	(25, 62, 21)	
	4	2.70	= 0.02	10	(4, 65, 21)	
	8	4.63	<0.01	44	(60, 15, 16)	
	14	6.57	<0.01	45	(-47, 46, 7)	
	16	2.71	= 0.02	47	(54, 30, -6)	
	21	2.76	= 0.02	10	(-41, 54, -7)	
	22	4.28	<0.01	47	(-51, 36, -3)	
	Collapsed	14	2.85	= 0.04	45	(-47, 46, 7)

*Note.*  
<sup>a</sup> BA = Brodmann’s Area.  
<sup>b</sup> Coordinates are based on the MNI system and (-) indicates left hemisphere.

**3.4. Predictive interrelations between the PFC, behaviour, and fat percentage**

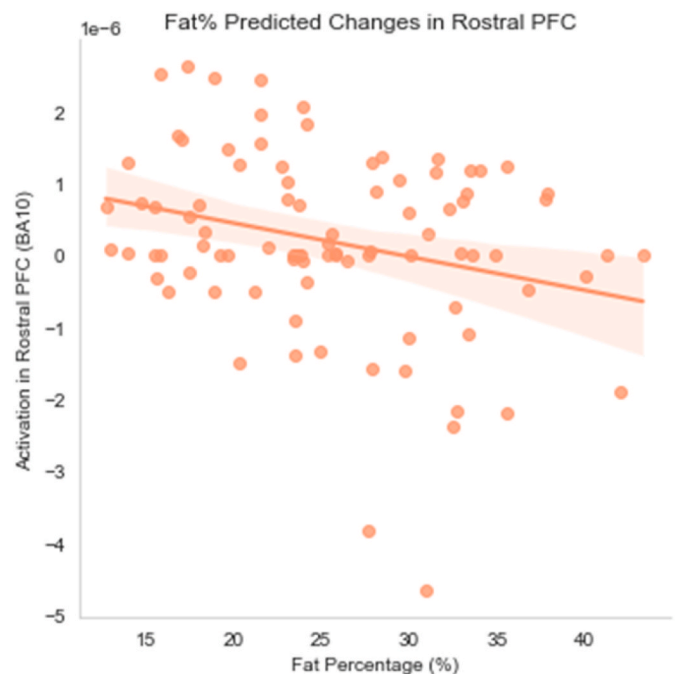
Interestingly, the exercise-related activation changes within bilateral rostral PFC significantly predicted the behavioral changes in speed (RTs) that were found across all executive function tests (3.1),  $R^2 = 0.22$  ( $R^2$  adjusted = 0.12),  $F_{(10, 79)} = 2.25$ ,  $p = .02$ , with channel 13 (left BA10) in particular emerging as a statistically significant predictor,  $t(79) = -2.74$ ,  $p = .008$  (Fig. 8). That is, the greater the activation in rostral PFC, the more negative the values of RT changes (i.e., the greater the behavioral change in the direction towards faster responding).

Multiple linear regression was again used to test if the physiological indices of fitness reported in Table 1 were predictive of the activation changes in all PFC subregions (rostral, dorsolateral, and ventrolateral PFC) that were present across executive function tasks. More specifically, the beta values of the channels forming these subregions reported in Table 5 were mean centered and used to predict values of weight, fat percentage, visceral fat, BMI, and  $VO_{2max}$  in turn. The model that tested whether fat percentage was predictive of the exercise effects on rostral (BA10) PFC was the only statistically significant model,  $R^2 = 0.20$  ( $R^2$



**Fig. 8.** General activation of rostral PFC after exercise was negatively associated with participants’ response times (RTs; seconds) on the executive tests in which speed was a principal component. That is, greater activation of rostral PFC (y-axis) predicted lower RT change values (x-axis), where negative values represent increases in post-exercise RTs, accounting for 22% of this behavioral effect.

adjusted = 0.10),  $F_{(10, 79)} = 2.00$ ,  $p = .04$ . That is, the channels comprising bilateral BA10 accounted for 20% of the variance in participants’ fat levels (Fig. 9). Channel 11 was the only significant



**Fig. 9.** Fat percentage was negatively associated with general rostral PFC activation after exercise. That is, participants’ body fat percentages (x-axis) accounted for 20% of the effect of exercise on brain activity (y-axis) in rostral PFC across all executive function tests; lower fat percentages were corresponded with greater activation.



predictor,  $t(79) = -2.26$ ,  $p = .03$ , indicating that there was a strong negative relationship with fat percentage. In other words, lower fat percentages in participants were most predictive of acute exercise-related increases in right BA10 (channel 11) during general executive functioning. Models for the two other PFC ROIs did not reach significance for the physiological indices of fitness.

In addition to rostral PFC being linked to fat percentage—rather than dorsolateral and ventrolateral PFC subregions—there was further consistency with this in terms of fat percentage also being linked to the behavioral changes that were found as an acute effect exercise (Fig. 10). Namely, fat percentage also positively predicted behavioral changes in speed (RTs) across all executive function tests,  $R^2 = 0.07$  ( $R^2$  adjusted = 0.08),  $F(1, 88) = 7.28$ ,  $p = .008$ .

#### 4. Discussion

Investigating the influence of vigorous exercise on behaviour and the PFC revealed several acute effects. Namely, the exercise group showed improved performance in terms of speed. Participants who exercised had a ~5% decrease in RTs when accounting for the percent change between the two groups. Collapsing across all experimental task conditions showed significant changes in all PFC subregions, bilaterally, with the greatest change in activation occurring in left rostral (BA10) PFC. The observed effects in the PFC across all executive function tests might most closely represent broad changes in information-processing speed. This wider recruitment of PFC subregions is what would be expected of features that are shared across executive function tasks, such as implementation speed—the speed with which the cognitive operations in the PFC can be implemented. Consistent with this interpretation was the finding that these exercise-related activation changes in the PFC significantly predicted the behavioural changes in the exercise group. Interestingly, the effects of exercise on left rostral PFC showed the strongest negative relationship with changes in RTs (Fig. 8). This relation was negative because percent changes in RTs indicating faster responses were negative values: post-minus pre-exercise. This finding can

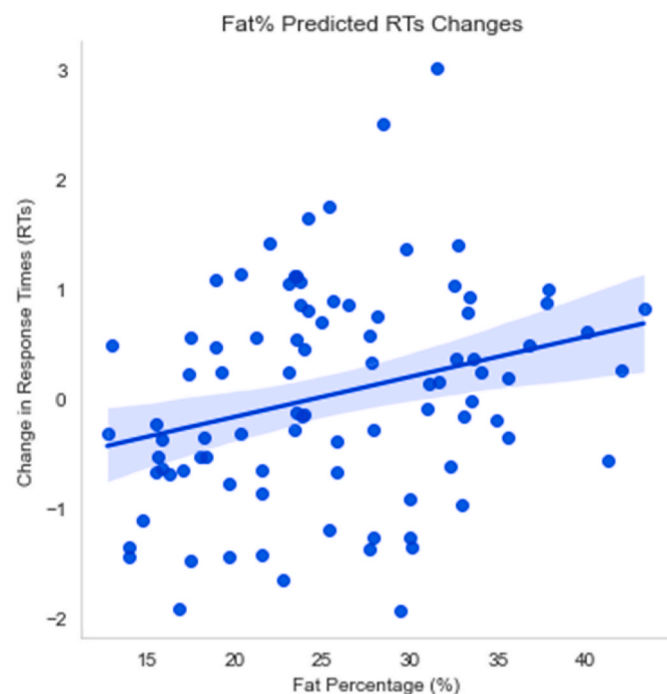


Fig. 10. Fat percentage (x-axis) was positively associated with the average change in participants' response times (RTs; seconds) across tasks, accounting for 8% of the behavioral effect of acute exercise. That is, greater fat percentages were corresponded with less behavioral improvement in performance speed.

be framed as positive in that increases in activation predicted increases in speed. One interpretation at a higher level of explanation could be that exercise upregulated the systems in the brain that facilitate executive functions, resulting in a heightened state of mental 'preparedness'.

Although the average change in activation across all tasks exhibited a more global-like effect, assessing the influence of vigorous exercise on specific executive function tasks showed effects which were more region specific. Namely, the individual executive functions that the experimental tasks were developed to index, such as response inhibition, exogenous and endogenous attending, and recognition and source memory, showed significant activation changes in the PFC after exercise. These changes were largely specific to the subregions that are typically implicated in these executive function tasks. For example, one PFC subsystem of interest was the 'attentional gateway' (Burgess et al., 2007). This is a dynamic mechanism for which rostral PFC (BA10) is functional specialized. Specifically, it operates in an executive capacity in that it modulates the selection and sustainment of stimulus-oriented and stimulus-independent information processing. In the present study, the stimulus-independent thought (SIT) condition showed a significant change in activation that was over the lateral surface of right rostral PFC. In the stimulus-oriented thought (SOT) condition, there were significant activation changes in bilateral rostral (BA10), right ventrolateral (BA44), and left dorsolateral (BA46) PFC (Table 5). These results are very much in line with the literature on the neural basis of these modes of attending (Burgess et al., 2003; Burgess et al., 2007; Gilbert et al., 2005; Gilbert et al., 2006; Gilbert et al., 2007).

Another executive function of interest was response inhibition. A go/no go task was used to assess this executive function. There was a significant change in activations during this task in bilateral dorsolateral (BA46/9), ventrolateral (BA44/45), and rostral (BA10) PFC, with the greatest change occurring in right BA46. Activation of right BA46 is a common finding of response inhibition tasks that contain strong monitoring elements (Criaud and Boulinguez, 2013). This is because go/no-go tasks likely require as much attentional monitoring as response inhibition—if not more. More specifically, the key cognitive demand placed on participants in this type of task is the need to check stimuli to verify that a given stimulus is something to which one should *not* respond; in this case, participants were instructed to not respond if they were shown a kitten. Therefore, this finding is also in line with the cognitive neuroscientific literature. In addition, PFC activation during recognition and source memory tasks after exercise showed significant changes in bilateral BA10, and in bilateral BA44, right BA46/9, and bilateral BA10, respectively. Between-group comparisons with the control group according to the same task contrasts suggested that the within-group findings were related to the effects of exercise, in that they showed highly similar patterns of activation. For example, the greatest difference in activation between the two groups during the no-go condition was in right BA46, which reflects the large within-group finding that there was a marked change the consumption of attentional monitoring resources in BA46. Regarding the attentional mode task, there was again a significant difference in right BA10 during the SIT condition and bilateral BA10 during the SOT condition. Between-group comparisons of recognition memory also showed the same peak activation pattern as the within-group contrast, namely left BA10 was most active during recognition in the second neurocognitive testing session. However, activation in left BA45 represented the major difference between groups during the source memory task, which was not the area of peak activation in the within-subjects contrast, raising the question of whether at least some within-groups channels were not solely influenced by exercise.

Perhaps the most surprising findings were that participants' body fat percentages predicted both the overall (across tasks) behavioural and neuroscientific effects described above. More specifically, fat percentage accounted for 22% of the effect of exercise on RTs and 20% of the effect of exercise on the rostral PFC; lower body fat percentage predicted greater changes in PFC activation and faster task performance. That is,

participants with higher body fat percentages showed relatively smaller changes in hemodynamics and behaviour. So, of the several physiological indices of fitness reported in Table 1, fat percentage appears to be the variable that is most closely linked to how exercise affects brain functioning and behavior post-exercise. This could mean that body fat interferes in some metabolic way with the ability of the body to efficiently allocate changes in resource availability due to exercise. This remains to be investigated, but the present findings highlight such broader implications. Among these are topics relevant to obesity (see Sui and Pasco, 2020, for review) and obesity across the life span (Liang et al., 2014); one interesting direction, for example, is macronutrient intake and the intake of highly processed foods (Muth and Park, 2021).

Importantly, how might the exercise-brain effects be interpreted within the theoretical resource framework described above? Recall that the type of exercise intensity in the present study was vigorous ( $\geq 80\%$   $\text{VO}_2\text{max}$ ). There is some evidence showing significant decreases in PFC oxygenation during vigorous levels of exercise (Ando et al., 2011), so researchers have tested whether executive function performance is preserved under these conditions by varying the degree to which participants experienced hypoxia during exercise—where fractions of inspired oxygen were manipulated and pulse oximetric saturation was monitored. For example, Ando et al. (2013) found that the response times of participants on a response inhibition task decreased during these conditions. So, oxygenation is relatively decreased during vigorous exercise, and yet cognitive performance is nonetheless sometimes improved. Potentially key to explaining this is the term ‘relatively’, in that oxygenation might be lower during heavy physical activity compared to moderate exercise, but not with respect to baseline levels prior to exercise. That is, although cerebral oxygenation might vary depending on exercise intensity, any amount of exercise will upregulate blood flow and, therefore, potentially contribute to brain function. The present study was consistent with this idea. Its findings suggested that the metabolic changes that occur during exercise cascade into post-exercise behaviour and thought in a way that is largely positive. This evidence supports the idea that there is potentially an optimal window of time ( $\sim 30$  min) in which cognitive functions might benefit from such physical activity (see Chang et al., 2012; Lambourne and Tomporowski, 2010). These benefits generally manifest as a global change in activation across PFC subregions, which resonates with the postulation that exercise leads to widespread increases in cerebral blood flow—increases in the availability of metabolic resources for cognitive functions (Kahneman, 1973). The concomitant change in processing speed via RTs is consistent with the idea that this upregulation of hemodynamics translates into behaviour.

In addition, contrasts that isolated individual operations of cognitive control showed effects that were rather specific. That is, the hemodynamic resources that exercise modulated were consumed by the subregions which are typically implicated in the function of interest. It therefore makes sense to extend our current cognitive neuroscientific understanding of exercise hemodynamics by suggesting that exercise increases not only the global availability of metabolic resources upon which the brain can draw but also the degree and efficiency with which they are allocated and consumed. Thus, it is unlikely that the neural patterns of activation that indicate these features will show the same neural effects across many different cognitive tasks, especially when using tasks that tap functionally specialized subsystems in the brain. This means that the patterns of activation one would expect after exercise depend on the unique cognitive demands of the tasks being used. Complex cognitive tasks can therefore be used to delve further into the empirical rudiments of efficiency in the modulation of resource availability. However, this might only be the case for the lateral surfaces of the PFC, which has highly specialized, domain-general functions. It is plausible that the medial surfaces of the PFC would show similar effects, but hemodynamic data were collected only from these bilateral areas. The penetration depth of fNIRS limited the investigation of other areas of the brain, unfortunately.

Although using a repeated-measures design to study the acute effects of exercise addresses the significant issue of physiological confounds during exercise, it raises the possibility of learning or repetition effects. A control group was used to help address this—to help separate the effects that were unique to exercise and those that were not. As discussed above, the results showed between-group differences suggesting that exercise affected the brain and behaviour over and above learning, which was represented by changes in the control group between the first and second neurocognitive testing sessions; however, a limitation of the present work was that the size of the control group was relatively small. Ideally, almost one hundred participants should have been recruited for this group to match the experimental one. Such matching is challenging in practice when the experimental sample is quite large and tested in rapid succession, but future studies might interleave data collection across groups rather than sequentially. In addition, long-term changes in cognition, behavior, brain, and fitness were not measured, so inferences about the effects of repeated sessions of exercise could not be made; this was an additional aim of the study in its original design, but the onset of a global pandemic precluded it. It therefore makes sense for future research within this vein to investigate the chronic effects of exercise on PFC systems, such as the rostral attentional gateway and attentional monitoring. Interestingly, there might be important ergonomic implications of this research. For example, there are many vocations in which people are required to engage in complex cognitive tasks, such as needing to make decisions quickly, direct attention appropriately, and recall memories accurately. Exercise might be relevant to certain professions, and different professions might benefit from different types and intensities of physical activity. So, future investigations might consider an ergonomic-oriented line of neuroscientific research.

In sum, evidence is converging from behavioral neuroscience, exercise psychology, and human neuroimaging suggesting that even a single bout of exercise has acute, positive effects on people. However, the nature of the precise neurocognitive and metabolic mechanisms into which these benefits translate remains somewhat unclear. Cognitive neuroscientific studies have been attempting to address this by investigating exercise-related changes and interrelationships between cerebral oxygenation, hemodynamic upregulation, and cognitive performance, with the present study falling within this vein of research. There are several important contributions of this work. Namely, it was designed to have more spatial coverage of bilateral PFC subregions to assess multiple executive functions. It has also taken greater care in addressing issues of systemic confounds. For example, it assessed the effects of exercise on the brain shortly after exercise rather than during it and used a control group. This is because neuroimaging during physical activity has not yet overcome several methodological issues (e.g., extracerebral blood flow washing out signals, motion artifacts, etc.), and it was this concern that formed the rationale for accounting for heart rate from extracerebral sources. HbR was also used for interpretation due to how marked changes in respiration affect  $\text{HbO}_2$  (Scholkmann et al., 2013a, 2013b). To our knowledge, this is the first time the acute effects of exercise on executive functions, such as those which rostral PFC (BA10) support, have been investigated using fNIRS on a group of over one-hundred people. Functional neuroimaging data do not measure that which is available in the brain, but rather reflects the use and allocation of metabolic resources during cognitive tasks, and we found that these aspects of hemodynamics were largely spatially sensitive to the operations responsible for satisfying task demands. An additional important methodological lesson from our findings is that one should perhaps be cautious about generalising across studies about the prefrontal cortex brain regions that are affected by physical exercise until we have a great deal more evidence: If patterns of prefrontal cortex activation detected in exercise neuroscience studies depend on the precise task used, then since the variety of tests of executive functions typically used is rather limited, the aggregated results across many studies in the literature may come to reflect substantially the preponderant use of a particular paradigm. Hence it would be a good idea for the field to examine physical

exercise-induced prefrontal cortex changes from a wide range of different types of task and, as here, using a repeated-measures design with a control group. Future research might also further investigate the rather surprising finding that body fat has a predictive role in the extent to which acute exercise affects behavioural performance and neural activity in the PFC, and our findings add to the current debate on the role of visceral fat upon cognition (Guo et al., 2020).

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## CRedit authorship contribution statement

**James Crum:** Data curation, Formal analysis, Investigation, Methodology, Software, Visualization, Writing – original draft, Writing – review & editing. **Flaminia Ronca:** Conceptualization, Investigation, Supervision, Writing – original draft. **George Herbert:** Investigation. **Estela Carmona:** Investigation. **Isla Jones:** Writing – review & editing. **Uzair Hakim:** Writing – review & editing. **Mark Hamer:** Writing – review & editing. **Joy Hirsch:** Writing – review & editing, Supervision. **Antonia Hamilton:** Supervision, Writing – review & editing. **Ilias Tachtsidis:** Supervision, Writing – review & editing. **Paul W. Burgess:** Conceptualization, Formal analysis, Project administration, Supervision, Writing – original draft, Writing – review & editing.

## Declaration of competing interest

The authors declare no competing financial interests.

## Data availability

Data will be made available on request.

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