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ORIGINAL ARTICLE

Short-time high-intensity exercise increases peripheral BDNF in a physical fitness-dependent way in healthy men

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Abstract

BDNF is associated with brain health and positively modulated by exercise; however, the influence of physical fitness status on BDNF is incipient. This study investigated the BDNF response after acute-exercise sessions performed at low, moderate, and high intensities and the relationship between physical fitness status and BDNF response. Twenty-eight men, divided according to physical fitness status (<50th or >50th percentile for VO_{2max}), performed three randomised acute exercise sessions at low (90% of VT1), moderate (midpoint between VT1-VT2), and high (midpoint between VT2-W_{max}) intensities until exhaustion or for up to 60 min. Lactate and BDNF were determined pre and post-exercises. For BDNF, there were main effects of time ($p = 0.003$) and interaction ($p < 0.001$), showing an increase post high-intensity exercise ($p < 0.001$). Changes in BDNF presented differences between conditions ($p < 0.001$) with greater increase in high-intensity compared with the others ($p = 0.003$). For lactate, there were main effects of time ($p < 0.001$), condition ($p < 0.001$), and interaction ($p < 0.001$) with greater concentration in high-intensity. High-intensity exercise exhibited inverse correlation between the changes in BDNF and lactate (r=−0.38, $p = 0.044$). There was significant correlation between BDNF and VO_{2max} for moderate ($r = -0.57$, $p = 0.002$) and a trend for high-intensity condition ($r = -0.37$, $p = 0.050$) and when evaluating BDNF according to physical fitness level, it was observed that subjects with lower physical fitness levels had greater increases in BDNF in short-time high-intensity exercise ($p = 0.041$). In conclusion, short-time high-intensity exercise seems to be more efficient in increasing BDNF concentration, and physical fitness level influences this response, as healthy individuals with lower physical fitness levels were more responsive.

Keywords: *BDNF, neurotrophin, lactate, brain health, high-intensity training*

Highlights

- Acute high-intensity aerobic exercise induces increase in BDNF levels.
- Physical fitness level should be considered for increasing BDNF concentrations since it appears to be physical fitness-dependent.
- The expression of serum BDNF is likely reliant on intensity of exercise.
- Individuals with lower physical fitness level were more responsive after high-intensity aerobic exercise.

Introduction

Chronic exercise improves cardiovascular and metabolic health and the ability of exercise to promote brain health has recently been demonstrated (Hillman, Erickson, & Kramer, 2008; van Uffelen, Chin, Hopman-Rock, & van Mechelen, 2008). Brain-derived neurotrophic factor (BDNF) is an important and abundant neurotrophin in the brain, directly associated with development, regeneration, survival, and maintenance of neurons and its positive modulation during training is well documented in the literature, especially in the elderly population (Byun

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& Kang, 2016; Vaughan et al., 2014) and patients with neuropathology (Kim et al., 2014; Kimhy et al., 2015; Lin et al., 2015; Nascimento et al., 2014).

Recently, some studies have shown the acute and chronic effects of different kinds of exercise, such as strength, high-intensity intermittent, moderate-intensity, and continuous exercise, on speed and cognition accuracy (McMorris & Hale, 2012), executive processes and oxygenation (Tempest, Davranche, Brisswalter, Perrey, & Radel, 2017), information processing in the central nervous system (Kamijo et al., 2004), neurocognitive performance (Olson et al., 2016), and regulation of neurotrophin expression, mainly BDNF (Cabral-Santos et al., 2016; Santos, et al., 2016; Walsh, Edgett, Tschakovsky, & Gurd, 2015).

Our group investigated the impact of different aerobic exercise intensities (moderate-intensity continuous exercise – 70% of maximum velocity at VO_{2max} (vVO_{2max}); and high-intensity intermittent exercise – 100%vVO_{2max}) with volume equalised (5 km) in healthy young men and observed a greater increase in BDNF levels for high-intensity intermittent exercise compared to moderate-intensity continuous exercise (Santos, et al., 2016). In a similar line, Saucedo-Marquez et al (Saucedo Marquez, Vanaudenaerde, Troosters, & Wenderoth, 2015), investigated the effectiveness of two high-intensity exercise protocols, both protocols lasted 20 min, on the kinetics of serum BDNF before, during, and after continuous exercise (70% of maximal work rate) and high-intensity interval training (90% of maximal work rate performed at 1:1) in active men, and observed that both protocols evoke similar BDNF kinetics with higher concentrations post-exercise session; however, high-intensity interval training facilitated superior BDNF levels when compared with continuous exercise.

These previous studies corroborate the idea of the benefits imposed by high-intensity exercise on BDNF modulation; however, none of them considered different physical fitness levels. Given that this metabolic *status* directly influences several outcomes, such as cardiovascular risk factor development, responsiveness to training, and immunometabolic profile, the purposes of this study were: 1) to investigate the peripheral BDNF response after an acute exercise session performed at low, moderate, and high intensities; 2) to study the BDNF response according to physical fitness status at different exercise intensities. We hypothesised that high-intensity exercise would promote greater secretion of BDNF levels than low and moderate-intensity aerobic exercise, independent of physical fitness.

Methods

Participants

Twenty eight men (age: 28.8 ± 5.6 years; body mass: 75.8 ± 9.9 kg; $VO_{2maxmean}$: 50.5 ± 8.8 $mL.kg^{-1}.min^{-1}$ (or 3.9 ± 1.2 $L.min^{-1}$)) were recruited for the study and the characteristics of the subjects in all exercise sessions are presented in Table 1. Initially, all participants were classified as physically inactive ($n = 10$) (<600 METS), physically active ($n = 9$) (600–2999 METS), or well-trained ($n = 9$) (>3000 METS) using the International Physical Activity Questionnaire (IPAQ). All participants were required to complete all exercise sessions. This study was approved by the local Ethics Committee (CAAE: 31168714.6.0000.5402) and the research was conducted according to the 2013 Revision of the Declaration of Helsinki. Healthy men were included, without any health disorders, such as cardiorespiratory and osteoarticular diseases, and who had not used any ergogenic substances or medicines for at least six months prior to the study. Written informed consent was obtained from all participants prior to participation.

Experimental design

Four acute exercise sessions were performed on different days, with a 48 h recovery interval between sessions, under controlled conditions (mean temperature = 22.1°C; mean relative humidity: 55%; mean barometric pressure: 731.3 mmHg) between 8:00 am and 12:00 pm. On the first visit to the laboratory, anthropometric and body composition measurements were assessed, and the participants performed a maximal incremental test on a cycle ergometer (Inbrasport CG-04, Embramed, Porto Alegre, Brazil) to determine maximal oxygen uptake. Posteriorly, three acute exercise sessions were performed on alternate days at low (<60% VO_{2max} – 90% of VT1), moderate (60–75% VO_{2max} – midpoint between VT1 and VT2), and high (>90% VO_{2max} – midpoint between VT2 and W_{max}) intensities, in random order. Blood samples were collected pre (rest) and immediately post-exercise session to analyze the BDNF and lactate concentrations. Rating of perceived exertion (RPE) and heart rate (HR) were recorded immediately post-exercise. All participants were instructed not to exercise the day before each experimental session, and not to consume alcohol, caffeine, and/or stimulants of any kind during the 24 h before the tests. Additionally, the participants ate a standard breakfast, with energy intake fixed at 25% of the estimated daily energy needs for each participant, composed of toast, cottage cheese, and yogurt (≈50% carbohydrate, 35% fat, and 15% protein). The

Table 1. Individual characteristics, by mean and standard deviation, at maximal incremental test and each aerobic intensity.

	Incremental	Low	Moderate	High
Age (years)	28.8 ± 5.6	–	–	–
Body mass (kg)	75.8 ± 9.9	–	–	–
Workload (watts)	214.6 ± 55.3	96.1 ± 36.3	129.9 ± 44.0$^{£}$	182.6 ± 49.2$^{£,\#}$
VO_{2mean} ($mL.kg^{-1}.min^{-1}$)	50.5 ± 8.8	30.8 ± 6.1	37.5 ± 8.4$^{£}$	47.4 ± 9.3$^{£,\#}$
VO_{2mean} ($L.min^{-1}$)	3.9 ± 1.2	2.4 ± 0.8	2.9 ± 1.0$^{£}$	3.6 ± 1.2$^{£,\#}$
HR_{mean} (bpm)	188 ± 10	139 ± 15	160 ± 17$^{£}$	180 ± 11$^{£,\#}$
T_{lim} (minutes)	–	58 ± 7	46 ± 17$^{£}$	13 ± 13$^{£,\#}$

HR_{max}: maximal heart rate; T_{lim}= time limit; £= statistically significant difference from low intensity; #= statistically significant differences from moderate intensity.

participants were instructed to eat the breakfast two hours before the acute session tests.

Maximal incremental test

Initially, the physical fitness level was classified by the International Physical Activity Questionnaire (IPAQ) to determine the initial workload of the maximal incremental test. The initial workload was 35 watts for sedentary individuals, 70 watts for the physically active individuals, and 105 for the well-trained individuals, and this was increased by 25 watts every 3-minutes until exhaustion. Participants were instructed to cycle at a constant speed (70–90 rpm) throughout the test (Caputo & Denadai, 2008). Criteria to stop the maximal test were gas exchange ratio > 1.1, HR_{max}> 90% of the maximum expected for age and rating of perceived exertion (RPE) >18. The maximum workload (W_{max}), maximal oxygen uptake (VO_{2max}), and ventilatory thresholds (aerobic and anaerobic thresholds) were assessed by a breath-by-breath gas analyzer (Quark PFT, Cosmed®, Rome, Italy). VO_{2max} was assumed as the highest 30-sec mean observed during the incremental test and the ventilatory thresholds were determined by experienced researchers given that the first (or aerobic) ventilatory threshold (VT1) was determined by the first inflection point between ventilation (Ve) and workload, and/or the first increase in oxygen equivalent ($VE{\cdot}VO_2^{-1}$) vs. workload (Binder et al., 2008). The second (or anaerobic) ventilatory threshold (VT2) was assessed using the second increase in VE vs. workload, and/or the non-linear increase in carbon dioxide equivalent ($VE{\cdot}VCO_2^{-1}$) vs. workload (Binder et al., 2008).

Aerobic exercise protocols at different intensities

The exercise sessions started with 5 min of warm-up on a cycle ergometer at 30% of W_{max} for all intensities. Randomly, three sessions were performed at low (<60% VO_{2max}– 90% of VT1), moderate (60–75% VO_{2max} – midpoint between VT1 and VT2), and high (>90% VO_{2max} – midpoint between VT2 and W_{max}) intensities until exhaustion or for up to 60 min (Binder et al., 2008).

Blood samples and analysis

In all sessions, 25ul of blood were collected from the ear lobe to determine lactate concentration ([La]) pre and immediately post-exercise. The blood samples were stored in plastic tubes containing 50ul of sodium fluoride at 1% and analyzed in a lactate analyzer (Yellow Springs 1500 Sport).

Additionally, 10 ml of blood were collected by peripheral puncture from the forearm vein pre and immediately post-exercise sessions. The blood samples were immediately allocated into vacutainer tubes (Becton Dickinson, BD, Juiz de Fora, Brazil) containing ethylenediaminetetraacetic acid (EDTA) for plasma separation and into dry vacutainer tubes for serum separation. The tubes were refrigerated for 1 h until centrifugation at 3000 rpm for 15 min at 4°C, and plasma and serum samples were stored at −20°C until analysis. BDNF was analysed using enzyme-linked immunosorbent assay (ELISA) with a commercial kit (R&D System, Minneapolis, MN, USA) according to the manufacturer's guidelines. Sensitivities of the ELISA kit are 1500–23.4 $pg.mL^{-1}$ with an intra-assay coefficient of variation (CV) of 1.3%. The BDNF concentration post-exercise was adjusted for plasmatic volume using the hematocrit determination.

Statistical analysis

Initially, the sphericity was confirmed according to the Mauchly's W test and Greenhouse–Geisser correction and a 3 × 2 repeated measures analysis of variance (RMANOVA) with the Bonferroni adjustment for multiple comparisons was used to compare BDNF and lactate concentration. When an interaction (condition x time) was observed, a Bonferroni post hoc test was conducted and the partial eta-

square for *ANOVA* was presented for main effect of time (η^2). In addition, when an interaction was observed, the changes were calculated for each intensity (Δ = Post-value minus Pre-value) and a repeated measures analysis was performed. Next, to compare BDNF changes according to the physical fitness level at low, moderate, and high intensities, the subjects who were above the median VO_{2max} ($\geq$49.7 mL.kg.min^{-1}) were classified as having higher physical fitness. The participants who were below the median (<49.7 mL.kg.min^{-1}) were classified as having lower physical fitness, due to the absence of cut-off points. Subsequently, data normality was verified using the Shapiro–Wilk test and data are presented as median and interquartile range, since the data presented non-parametric distribution. Thus, the relationship between physical fitness and BDNF concentration was verified through the Spearman correlation. Next, the BDNF percentage of changes was calculated (Δ% = post-value minus pre-value divided by pre multiplied by 100) and the comparison according to physical fitness at three different intensities was conducted by the Mann–Whitney test. Besides that, was verified the relationship between changes (Δ%) of BDNF and lactate for each intensity. Statistical significance was set at $P < 0.05$ and the data were analyzed using the Statistical Package for Social Sciences 17.0 (*SPSS* Inc. Chicago. IL. USA).

Results

Table 1 shows the individual characteristics, as mean and standard deviation, for the maximal incremental test and by intensities. The standard breakfast offered to the participants had an energy intake of approximately 605 kilocalories according to estimated daily energy needs.

The mean of oxygen uptake (VO_2) presented a statistically significant difference between conditions ($F = 115.590$, $p < 0.001$, $\eta^2 = 0.87$). The Post-hoc showed a significant difference between intensities, with greater increases in the high intensity compared to both low and moderate intensities ($p < 0.001$), and moderate greater than low intensity ($p < 0.001$). Workload (W) presented a statistically significant difference between conditions ($F = 340.139$, $p < 0.001$, $\eta^2 = 0.93$). The Post-hoc showed a significant difference between intensities, with greater increases in the high intensity compared to both low and moderate intensities ($p < 0.001$), and moderate greater than low intensity ($p < 0.001$). For heart rate, there was a statistically significant difference between conditions ($F = 87.444$, $p < 0.001$, $\eta^2 = 0.76$). The Post-hoc showed a significant difference between intensities, with greater increases in the high intensity compared to both low and moderate intensities ($p < 0.001$), and moderate greater than low intensity ($p < 0.001$). Finally, the time limit presented a statistically significant difference between conditions ($F = 133.367$, $p < 0.001$, $\eta^2 = 0.83$). The Post-hoc showed a significant difference between intensities with lower duration in the high intensity compared to both low ($p < 0.001$) and moderate intensities ($p = 0.001$), and moderate lower than low intensity ($p = 0.001$). It is important to mention that, when analyzing the energy expenditure according to exercise intensities, lower expenditure was observed in high intensity (477.2 ± 391.0 kJ) compared to moderate (1861.6 ± 796.7 kJ) and low (1836.3 ± 647.0 kJ) intensities ($p < 0.001$); however, between moderate and low intensities there were no differences (*data not shown*).

Table 2 presents the BDNF and lactate concentrations pre and post-acute aerobic exercise sessions in healthy men (data are mean and standard deviation) and the comparison between low, moderate, and high intensities in the absolute changes.

For BDNF, there was a main effect of time ($F = 10.311$, $p = 0.003$, $\eta^2 = 0.28$) and statistically significant interactions ($F = 9.676$, $p < 0.001$). The Bonferroni's Post hoc showed an increase Post-exercise only for high intensity ($p < 0.001$) and significantly greater than moderate ($p = 0.002$). For BDNF changes, there was a statistically significant difference between conditions ($F = 9.677$, $p < 0.001$, $\eta^2 = 0.27$). The Post-hoc showed a significant difference

Table 2. Metabolic and inflammatory parameters concentrations pre and post-acute aerobic exercise session in healthy men (data are mean and standard error of measurement (SEMeas)).

		Pre	Post	Δ
BDNF (pg.mL^{-1})	*Low*	33440.85 ± 6229.58	34900.17 ± 6908.31	1459.31 ± 3317.28
	Moderate	28169.05 ± 4674.63	32793.15 ± 5198.64	4624.11 ± 3081.61
	High	26673.37 ± 4896.58	43542.48 ± 6774.00*	16869.12 ± 3196.77£,#
Lactate (mmol.L)	*Low*	1.89 ± 0.13	2.72 ± 0.34	0.82 ± 0.31
	Moderate	1.80 ± 0.16	4.35 ± 0.54*,£	2.57 ± 0.52£
	High	1.94 ± 0.22	11.20 ± 0.68*,£,#	9.25 ± 0.59£,#

Δ= absolute difference between post and pre session;*= statistically significant difference from Pre; £= statistically significant difference from low intensity; #= statistically significant differences from moderate intensity.

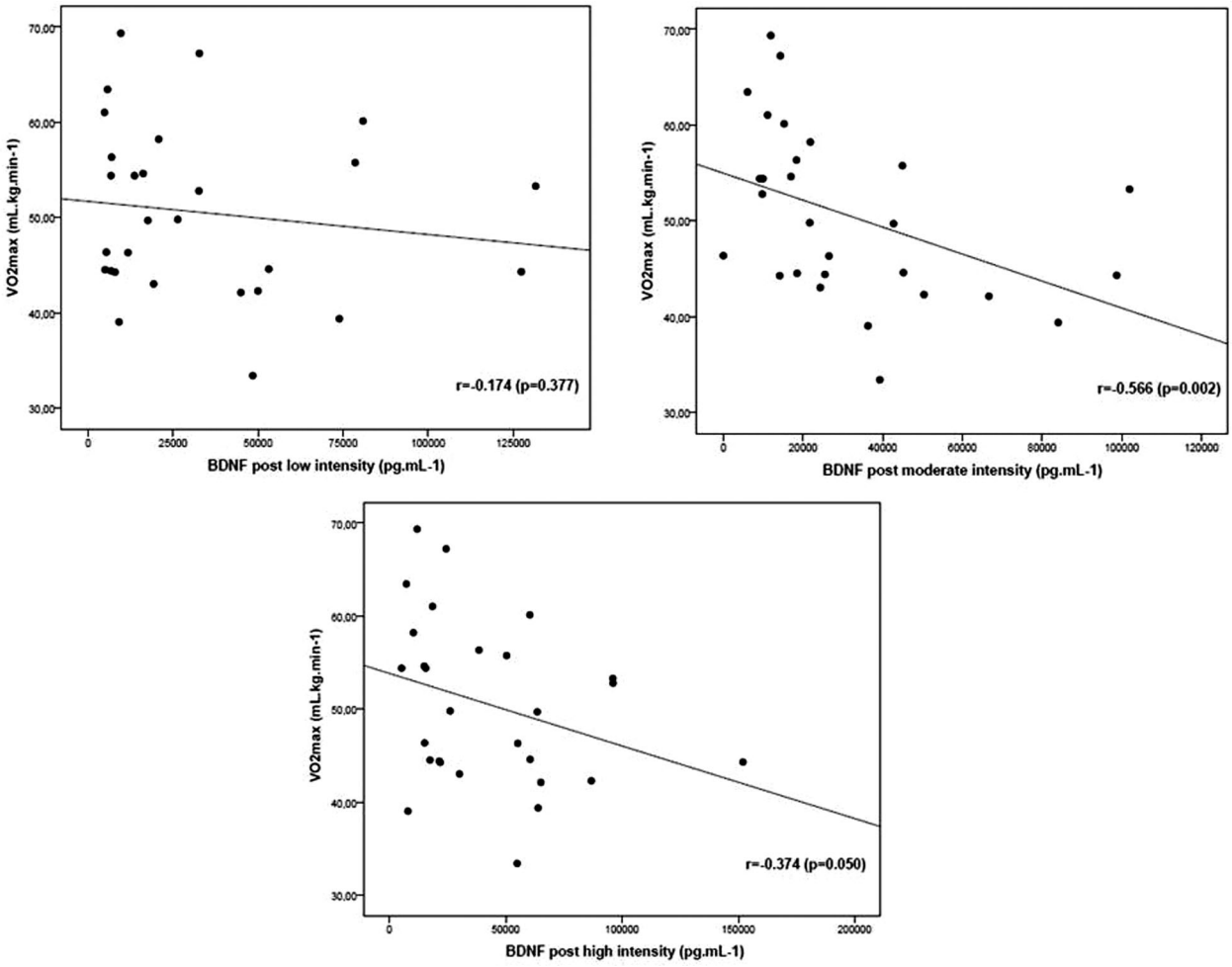

Figure 1. Relationship between increased BDNF post-session and maximal oxygen uptake (VO_{2max}) at (**A**) Low, (**B**) Moderate and (**C**) High intensities.

between intensities with greater increases in the high intensity compared to both low and moderate intensities ($p = 0.003$), and moderate greater than low intensity ($p = 0.003$).

For lactate concentrations, there was a main effect of time ($F = 109.179$, $p < 0.001$, $\eta^2 = 0.80$), significant differences between conditions ($F = 107.890$, $p < 0.001$), and statistically significant interactions ($F = 166.790$, $p < 0.001$). Lactate was greater for all three intensities post-exercise compared to rest ($p < 0.001$), with high intensity greater than low and moderate conditions ($p < 0.001$); and moderate greater than low intensity ($p < 0.001$). For lactate changes, there was a statistically significant difference between conditions ($F = 166.266$, $p < 0.001$, $\eta^2 = 0.86$) with a greater increase for high intensity in relation to both moderate and low intensities ($p < 0.001$), and moderate greater than low intensity ($p = 0.002$).

The RPE post-exercise was greater for high intensity (17.5 ± 2.1 points) compared to moderate (14.2 ± 2.3 points) and low intensities (11.4 ± 2.4 points) ($F = 48.405$, $p < 0.001$, $\eta^2 = 0.64$).

Figure 1 presents the relationship between physical fitness and BDNF concentration. Regarding the relationship post-exercise, there was a negative and statistically significant correlation between BDNF and VO_{2max} only for moderate intensity ($r = -0.57$, $p = 0.002$) and a trend to significant correlation for high intensity ($r = -0.37$, $p = 0.050$), but no correlation was observed for low intensity ($r = -0.17$, $p = 0.388$). Besides that, the BDNF changes (Δ%) were correlated with lactate changes (Δ%) for each intensity verifying an inverse relationship between variables at high-intensity ($r = -0.38$, $p = 0.044$) whereas no significant correlation was observed at low ($r = 0.03$, $p = 0.882$) and moderate ($r = 0.12$, $p = 0.54$) intensities.

Figure 2 shows the difference between BDNF changes according to physical fitness (<50th or > 50th percentile for VO_{2max}) at low, moderate, and high intensities.

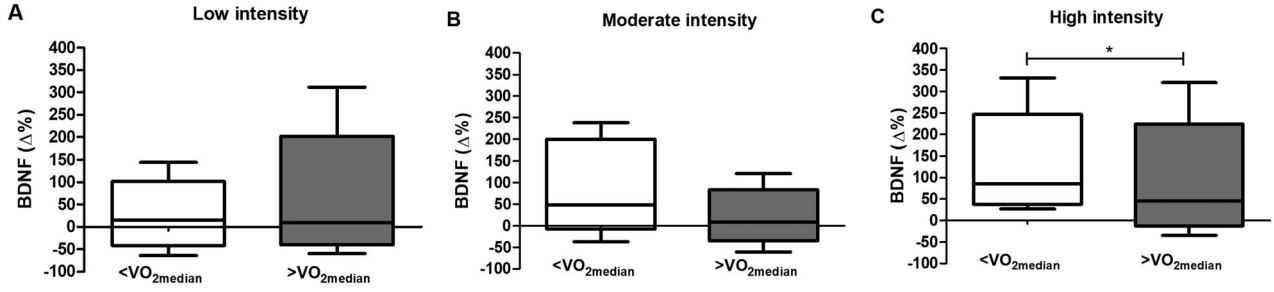

Figure 2. Differences between BDNF changes by fitness condition groups at (**A**) Low, (**B**) Moderate and (**C**) High intensities.

When verifying the BDNF response at different intensities, according to physical fitness, there was a statistically significant difference only for high intensity ($p = 0.041$) with greater increases in the subjects with lower physical fitness.

Discussion

The main findings were: 1) higher-intensity exercise with a short-time induced greater BDNF levels in relation to low and moderate-intensity followed by higher lactate concentration and RPE; 2) the BDNF concentrations post-exercise were inversely associated with VO_{2max} at moderate and high intensities and; 3) when considering the physical fitness status for each aerobic exercise, the individuals with lower physical fitness (<49.7 mL.kg.min^{-1}) exhibited greater BDNF changes, mainly after high-intensity with a short-time, when compared with well-trained individuals with better physical fitness.

Studies have investigated the BDNF kinetics after different exercise protocols, demonstrating a gradual increase mainly during high-intensity compared to low-intensity exercise, with peak concentrations immediately-post exercise; however, this increase returned to baseline quickly (Saucedo Marquez et al., 2015; Schmidt-Kassow et al., 2012). In the present study, we demonstrated a similar response regarding the acute effect, given that only the high-intensity exercise with a short-time was able to significantly increase the BDNF levels.

In special populations, such as patients with chronic diseases and disability, BDNF concentrations increase following an exercise of low to moderate-intensity (between 50 and 70% VO_{2max}), while healthy subjects, in agreement with the present study, seem to demonstrate more benefits after high-intensity exercise protocols (>75% VO_{2max}) (Knaepen, Goekint, Heyman, & Meeusen, 2010). Therefore, these previously cited studies suggested that the effort-intensity is an important variable to induce BDNF changes.

In this sense, Schmolesky et al. (Schmolesky, Webb, & Hansen, 2013), investigated the effects of an aerobic exercise session in healthy males performed on a cycle ergometer at different intensities (80% or 60% of heart rate reserve) and durations (20 or 40 min) on serum BDNF, and found no significant differences between the four exercise conditions when considering both the intensity and duration main effects, and the intensity X duration interaction. However, when comparing high-intensity exercise performed for 40 or 20 min, the subjects who exercised at high-intensity for 40 min were more likely to experience significant increases in serum BDNF levels (>2.7 times) compared to high-intensity for 20 min.

In our findings we observed a significant increase in BDNF only after high-intensity with a short-time, however future studies should be conducted with different volumes, with maximal and supramaximal exercises, to investigate the influence of volume in high-intensity exercise. In this line it is important to highlight that although these findings were observed in a high-intensity exercise, with short duration and low energy expenditure, the protocol presented higher oxygen uptake. Therefore, we assume that greater mean VO_2 may explain why high-intensity exercise may be better than lower intensities to improve BDNF concentrations and health parameters in a healthy population; however, more studies with equalised duration and energy expenditure should be conducted in order to certify the effects of intensity.

Sobral-Monteiro-Junior et al (Sobral-Monteiro-Junior et al., 2019) have been suggested an alternative neurobiological pathway linked with exercise-released lactate. The authors proposed that lactate is able to cross the blood brain barrier and the neurons may utilise it as energy supply as well as signaling molecule, principally stimulating of gene expression BDNF, favouring the neuroplasticity. The present study, we found that high-intensity exercise showed higher BDNF and lactate levels; however, when considered the percentage of changes, an inverse correlation was

observed for the same intensity, which leads us to assume that not only exercise intensity, but also other factors (i.e. adaptation and responsiveness to training) may influence BDNF changes.

Although several studies in the literature have investigated the effects of exercise intensity on BDNF levels, to our knowledge this was the first study to verify the influence of physical fitness level at different intensities. It is important to mention that individuals with lower physical fitness (<49.7 mL.kg.min^{-1}) showed greater BDNF concentration after high-intensity when compared with well-trained individuals with greater physical fitness.

Regarding the relationship between physical fitness level, training *status,* and BNDF concentration, a recent study conducted by Hebisz et al. (Hebisz, Hebisz, Murawska-Cialowicz, & Zaton, 2018) investigated the acute changes in serum BDNF during and after a sprint interval training session and the long-term effects after 6 months of training with sprint interval training in mountain bike cyclists. The authors did not observe any acute changes in serum BDNF concentrations when performing the sprint interval exercise test pre-intervention. However, after 2 and 6 months of training, the exercised-group presented decreased BDNF levels 10 min following the first set and 60 min after the final set of sprints when compared with baseline levels. These findings are in agreement and explain, at least in part, our results, which show that the group with better physical fitness levels, probably due to a greater and better training frequency, demonstrated lower production and release of BDNF after short-time high-intensity exercise.

In this line, Knaepen et al (Knaepen et al., 2010) conducted a systematic review about the neuroplasticity and exercise-induced response of BDNF levels. The authors emphasise that healthy subjects (trained and athletes) exhibit lower BDNF levels, indicating more effective clearance (i.e. a higher disappearance rate), with less stored or circulating BDNF in the periphery. In this perspective, a recent study with mice evidenced that long-term physical exercise increased BDNF mRNA in dentate gyrus of the hippocampus, parallel with significant alterations in the levels of TrkB, a BDNF receptor, in astrocytes (Fahimi et al., 2017). Therefore, the positive effects associated with physical exercise on neuroplasticity appear to be mediated by BDNF-TrkB signaling and, according to the literature, chronic exercise training and overexpression of BDNF can increase the levels of truncated and non-truncated TrkB receptors (Kim et al., 2015; LeMaster et al., 1999). These studies support our hypothesis that better physical fitness levels may be associated with greater BDNF sensitivity, via increasing TrkB receptor, making it necessary to lower production and release of BDNF after exercise.

Some limitations should be mentioned regarding the present study, such as the different durations of each aerobic exercise, given that the effort time may influence the BDNF responses. In addition, future studies should be performed with extreme physical fitness groups in order to identify the impacts of low and high maximal oxygen uptake on BDNF changes.

In conclusion, acute short-time high-intensity aerobic exercise seems to be more efficient in increasing BDNF levels in healthy subjects. In addition, the physical fitness level should be considered when analyzing this response, as individuals with a lower physical fitness level were more responsive, mainly after high-intensity aerobic exercise.

Disclosure statement

No potential conflict of interest was reported by the authors.

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