The Effect of Acute Aerobic Exercise on Spontaneous Brain Activity in Children

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Abstract

The present study examined the immediate effect of a single session of sub maximal exercise on brain activation in children. Twelve 9- to 11-year-old boys pedaled 30 minutes on a cycle ergometer at ~65% of their maximum heart rate maximum. Electrophysiological activity was recorded before physical exercise, and 10-, 20- and 30 minutes post-exercise. The results indicated that relative spectral power in the alpha1 band (8-10 Hz) decreased from 10 to 20 minutes post-exercise, and that relative spectral power in the alpha2 band (10-12 Hz) increased 20 and 30 minutes post exercise when compared to pre-exercise measurements. These concomitant changes occurring in the alpha1 and in the alpha2 bands are suggestive of an increased in attentional vigilance. The present results also suggest that a single session of sub maximal exercise produces changes in the spontaneous electro-cortical activity of the brain that last at least 30 minutes post-exercise.

Keywords: Quantitative EEG; Acute exercise; Children; Brain activation

Introduction

Physical activity (PA) and aerobic fitness positively relate to brain and cognitive development [1-3]. More active children routinely outperform their sedentary peers on standardized and experimental measures of academic and neuropsychological function [4]. Furthermore, cross-sectional and longitudinal studies reveal differences in brain structure [5,6] and function [4] between more active/higher-fit children and less active/lower-fit children. Although interest in the long-term effects of PA/exercise on brain structure and function is rapidly growing, fewer efforts have been directed toward understanding the influence of acute exercise on pediatric brain function.

One way to investigate the transient influence of aerobic exercise on brain function is electroencephalography (EEG). EEG can be recorded spontaneously while the participant is sitting, making this method ideal for evaluating brain function in paediatric populations [7]. Recorded from sensors placed on the scalp, spontaneous EEG activity is believed to reflect rhythmic oscillations of temporally synchronous neurons in the cerebral cortex [8]. These oscillations are typically decomposed into frequency bands (delta: 0-4 Hz, theta: 4-8 Hz, alpha1: 8-10 Hz, alpha2: 10-12 Hz, beta1: 12-20 Hz and beta 2: 20-30 Hz) and the power associated with each band reflects its individual contribution to the overall spectrum.

Accumulating research attributes a certain degree of functional significance to each frequency band. For example, power in the delta frequency is modulated by sleep stages, whilst increasing energy in the theta band is associated with drowsiness [9]. On the other hand, higher frequency oscillations such as alpha and beta occur mostly during wakefulness. In general, alpha rhythm is indicative of a state of relaxed wakefulness whereas beta activity indicates higher alertness [10]. A reduction of power in the slower bands combined with an increase of power in the faster bands is believed to reflect heightened vigilance [9].

Few paediatric exercise studies examined changes in EEG, and only one evaluated changes in spontaneous EEG in children [11]. Specifically, Schneider and Colleagues evaluated spontaneous EEG activity in 10 preadolescent children immediately before and after a 15-minute bout of moderate intensity cycling. The authors observed a general increase in alpha activity, which they located to the posterior parietal lobe and precuneus. Furthermore, these changes in alpha power were correlated with self-reported changes in calmness and relaxation. Similar to adolescent [12], and adult studies [13-15] evaluating alpha activity, Schneider and colleagues concluded that acute aerobic exercise yields a calming effect on paediatric brain function.

It should be noted, however, that Schneider and colleagues did not decompose alpha activity into lower (alpha1) and upper (alpha2) bands. Tonic changes in alpha frequency may reflect different processes whether they occur in the lower or in the upper alpha frequency band, and failure to analyze sub-bands separately may obviate important effects [16]. Specifically, although increasing power within the alpha 1 band (8-10 Hz) is associated with calmness-relaxation, drowsiness, and fatigue [17], increasing power within the alpha2 band (10-12 Hz) has been associated with cortical priming and increased vigilance [18], as well as brain maturation [19]. Thus, further inquiry is warranted to clarify the effects of acute exercise on spontaneous brain activity.

Accordingly, the primary aim of this study was to gain a more refined understanding of exercise-induced changes in spontaneous brain activity by evaluating multiple frequencies (delta, theta, alpha, beta) and frequency sub-bands (alpha1, alpha2, beta1, beta2). As previous paediatric research has only measured spontaneous EEG immediately prior to and following exercise, the current study also aimed to gain a greater understanding of the duration of acute exercise-induced alterations in spontaneous brain activity. To fulfill this aim, EEG activity was evaluated at multiple time points following exercise. Because paediatric studies show that event-related cortical activity during tasks requiring vigilance (sustained attention) is altered following acute exercise [20], we hypothesised that power within the alpha1 band would decrease after exercise and that power within the alpha2 band would increase. As previous paediatric research demonstrates exercise-induced alterations in event-related cortical activity 20-30 minutes post exercise [20] we predicted that tonic EEG alterations would persist at 20 and 30 minutes post-exercise.

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Method

Participants

Seventeen preadolescent boys ages 9-11 were recruited to participate in the current study. Only boys were included because of known gender difference in the development of EEG responses [21]. Data from five participants were discarded: two participants were non-compliant; for two participants, the electrodes did not properly remain on the scalp; and for one participant, data were contaminated with muscle artefacts. The remaining twelve participants did not differ demographically from those who were excluded (p's > .5). Retained participants had a mean age of 10.83 years (SD = 0.74 years), a height of 1.45 m (SD = 0.07 m), a weight of 37.17 kg (SD = 5.81 kg) and a body mass index of 17.64 kg/m^2 (SD = 2.08 kg/m^2). All participants engaged in regular physical activity three or more days a week and were free of neurological disorders, learning disabilities and history of head injury as verified by a health history questionnaire completed by guardians.

Procedure

The experimental procedure lasted ~2.5 hours. Participants and their parents were first familiarized with the laboratory, experimental equipment and procedures. Following familiarization, each participant's parent read and signed an informed consent form (which was approved by the Institution’s ethics committee of research in health sciences), and each child provided verbal assent. Following consent/assent, participants were fitted with an electrode-cap. EEG activity was recorded while participants sat still in a chair with their eyes open. Following the baseline EEG recording, participants completed an acute bout of aerobic exercise on a cycle ergometer. Following exercise, participants were then given 10 minutes for heart rate to resume to normal and to control for perspiration. EEG activity was again recorded at 10, 20, and 30 minutes post-exercise, while participants sat still in a chair with their eyes open (Supplementary Figure).

Exercise protocol

Exercise was performed on a cycle ergometer (Ergomeca GP 440, La Bayette, France) and exercise intensity was continuously monitored with a heart rate monitor (Polar Electro Oy, Finland). The bout of exercise consisted of a three minute warm up followed by 30 minutes of steady-state cycling and ended with a three minute cool down for a total of 36 minutes. During the three minute warm up, workload was progressively increased until the participant reached an intensity corresponding to ~65% of his age-predicted maximum heart rate. This intensity was chosen based on previous research in children demonstrating changes in brain function following exercise at this intensity [20]. Participants were encouraged to maintain target intensity for 30 minutes and cycle intensity was adjusted periodically to ensure maintenance of target heart rate. The mean heart rate reached for the group averaged throughout the exercise session was 137.00 beats per minute (SD = 5.67 beats per minute).

EEG recording

Data were recorded from eight gold disc electrodes (F3, F4, C3, C4, P3, P4, O1, and O2; Grass-Telefactor, West Warwick, RI, USA), arranged according to the international 10-20 system [9]. All electrodes were referenced to electrodes places on the earlobes (A1, A2) and an additional electrode (Fz) served as the ground. Two additional electrodes placed above and below the eyes were used to measure electro-occulographic (EOG) activity. All impedances were kept below 5 kΩ, and impedance measurements were performed before baseline and before each post-exercise EEG recording. All EEG recordings were collected while participants sat comfortably on a chair in an electromagnetic isolated room, and EEG signals were amplified 20,000 times with a Neurodata amplifier system (model 15A54; Grass-Telefactor, West Warwick, RI, USA). High and low pass filters were set to 0.01 and 100 Hz (6 dB/octave) respectively, and signals were digitized at 256 Hz via Gamma software (Grass-Telefactor, West Warwick, RI, USA).

EEG reduction

Data reduction was performed offline via Brain Vision Analyzer version 1.05 (Brain Products GmbH, Munich, Germany). High- and low-pass filters were applied to the digitized data, forming a 1–40 Hz (24 dB/octave) band-pass. The first 30 seconds of each three-minute recording were rejected to eliminate state transitions. The remainder of the data were segmented in 150 epochs of 1 second. Semi-automatic artefact rejection was performed on each segment using the following criteria: the absolute difference between two sampling points or between the maximum and the minimum could not exceed 150 µV, the amplitude could not exceed 150 µV or fall below -150 µV, and the difference within an interval of 100 ms could not be lower than 0.5 µV. Because the number of artefact-free segments varied from one participant to another, 60 artefact-free epochs were randomly selected for the analyses. Fast Fourier Transformations (FFT) with a 10% Hanning window were performed on each segment to determine spectral power (µV^2) and averaged for the following frequency bands: delta (1–4 Hz), theta (4–8 Hz), alpha1 (8–10 Hz), alpha2 (10–12 Hz), beta1 (12–20 Hz), and beta2 (20–30 Hz). Mean absolute power values for each electrode and for each frequency band were converted to relative power by dividing the power at each electrode of each band by the sum of the power at an electrode for all frequency bands.

Statistical Analyses

All statistical analyses were performed via SPSS version 18 (SPSS inc., Chicago, IL). A 4 × 6 × 4 × 2 (time of recording × frequency band × scalp region × laterality) repeated measures ANOVA was conducted to evaluate differences in spectral power after exercise relative to baseline. Post-hoc analyses adjusted with Sidak’s corrections were applied to the interactions that included the factor time. An alpha level of .05 was used for all statistical tests.

Results

Omnibus analysis failed to reveal any 3- or 4-ways interactions, however, a time of recording × frequency band interaction was observed (F [3.84, 42.25] = 3.73, p < .05, η^2 = .25). Post-hoc analyses revealed an effect of time for alpha1 and alpha2 bands, with relative spectral power decreasing significantly for the alpha1 band from 10 minutes post-exercise (M = 24.52, SD = 9.13) to 20 minutes post-exercise ((M = 21.92, SD = 8.08), t (11) = 3.70, p < .05, d = .28). Although not significant (p = .17), relative spectral power for the alpha1 band tended to remain lower, even 30 minutes post-exercise (Figure 1). For the alpha2 band, relative power significantly increased at 20 minutes (M = 17.96, SD = 8.14) and 30 minutes post-exercise (M = 17.47, SD = 7.30), relative to baseline (M = 15.70, SD = 7.43), (t (11) = 3.34, p < .05, d = .29 and t (11) = 3.35, p < .05, d = .24). No significant effect was found for any of the other frequency bands (p's > .05), and no interactions were observed for time of recording or frequency band with any other factor (region, laterality); suggesting a global effect of acute exercise on brain activity (Figure 1).
Discussion

The aim of the present study was to investigate the nature and duration of alterations in spontaneous brain activity following an acute bout of sub-maximal aerobic exercise. To do so, brain activity was recorded in twelve preadolescent boys, immediately prior to exercise and at 10, 20, and 30 minutes post-exercise. We predicted that power within the alpha1 band would decrease after exercise and that power within the alpha2 band would increase. The present results partly support our hypothesis as we observed a reduction of relative spectral power for the alpha1 band (8-10 Hz) and an increase for the alpha2 band (10-12 Hz). Contrary to our predictions, changes in alpha2 power were not present during the first post-exercise recording (10 minutes), but emerged at 20 minutes and continued at 30 minutes post-exercise.

At first glance the lack of neuroelectric alterations at 10 minutes post-exercise may seem surprising, considering that Schneider and colleagues [11] demonstrated changes immediately following exercise. One possibility is that splitting alpha activity into lower (alpha1) and upper (alpha2) bands may have reduced our ability to detect a more general alpha effect. Differences in the length of exercise may also account for the current discrepancy. Previous research suggests that more than one mechanism is responsible for alpha alterations following exercise, and that they each have a different time course [22]. As such, an initial short-lasting mechanism may be followed by a relative ‘silent period’ before the emergence of an additional and perhaps longer-lasting mechanism. As we were unable to measure EEG immediately following exercise, we may have missed an initial short-lasting mechanism, detecting only a second or longer-lasting mechanism. Although speculative, this finding does fit with dominant psychobiological models of acute exercise [23]. However, further research is warranted to elucidate the mechanisms underlying exercise-induced alpha alterations in children.

The functional interpretations of the current changes in brain function are less speculative, however. Specifically, the observed reduction in relative spectral power for the alpha1 band and the augmentation of relative spectral power for the alpha2 band is indicative of an increase in vigilance/arousal. This is important for several reasons. First, the current results help explain the discrepancy between dominant psychobiological models, which predict exercise-induced cerebral arousal (Reticular Activation, Hypo-frontality) and prior studies, which interpret alpha alterations as calming. Second, it suggests that an acute bout of aerobic exercise may be simple and cost-effective means to help children maintain vigilance.

It is important to note that we did not observe changes in neighboring frequency bands such as theta or beta. Indeed, aerobic-exercise EEG studies in adults have observed changes in a myriad of frequencies [24]. However, in the only other study to evaluate spontaneous EEG activity following acute exercise in preadolescent children, [11] failed to observe any significant modulation in beta or theta activity, although the authors did note a trend for beta activity. Furthermore, research examining adolescents [12] also found alterations in alpha activity, without observing concomitant changes in other frequency bands. Future research comparing children, adolescents, and adults will be well positioned to evaluate the similarities and differences of exercise-induced changes in spontaneous brain activity across the lifespan.

Another aim of the present study was to evaluate the duration of spontaneous EEG alterations following acute exercise. We observed a reduction in relative spectral power from 10 minutes to 20 minutes post-exercise for the alpha1 band and an increase in relative spectral power at 20 and 30 minutes post-exercise in the alpha2 band, relative to baseline. These results highlight the importance of evaluating brain activity at multiple time points following exercise and of evaluating frequency sub-bands. The current results also agree with previous adult studies [13-15] demonstrating spontaneous EEG alterations 30 minutes following exercise, and paediatric studies [4,20] demonstrating ERP.
altered by aerobic exercise. Together, these findings suggest that the beneficial influence of acute exercise on functional brain activity may be the result of the increased metabolic demands of exercise, as well as the activation of the sympathetic nervous system.

Conclusions and Limitations

In sum, the current results add important information regarding the nature and duration of alterations in spontaneous brain activity induced by acute aerobic exercise in children. These results both further and help explain discrepancies in the extant literature by demonstrating a differential modulation of sub-bands following exercise, and by demonstrating that these alterations are present at least 30 minutes post-exercise. Although meritorious, the current study is not without limitations. First, the current sample size is relatively small, and participants served as their own controls. Second, it could be argued that the modifications in EEG activity that we observed 20 and 30 minutes after exercise are the result of passage of time and are unrelated to the acute bout of exercise. This is unlikely, however, given the convergence with the results of Schneider and colleagues [11] and the fact that the frequencies investigated in the present experiment show stability over time [26]. Irrespective of these limitations, the current results add important information to the developmental and exercise-cognition literatures, and provide an impetus for further inquiry.

References