Acute Aerobic Exercise Decreases a Neurophysiological Response to Emotionally Negative Stimuli

Original Research

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Abstract

Introduction: A growing body of literature suggests that aerobic exercise results in improved mental and emotional well-being. Despite the clear benefits of aerobic exercise on mental well-being, the physiological mechanisms through which exercise benefits emotion processing is currently unclear.

Methods: Twenty participants were tested in a randomized crossover design that consisted of one exercise session and a control session (no exercise) scheduled one week apart. The exercise session consisted of an acute (30 minutes) bout of aerobic exercise (i.e., running on a treadmill at 75-85% max heart rate). The current study applied an established neurophysiological measure of emotion processing - the electroencephalogram (EEG) Late Positive Potential (LPP) component of the Event Related Potential (ERP). The LPP ERP amplitude was measured in response to a series of randomly presented emotionally negative and emotionally neutral picture stimuli. Self-report mood measures were also administered. The possibility that salivary alpha amylase (sAA) and cortisol would be related to changes in the LPP ERP after exercise was also examined.

Results: It was shown that relative to the baseline condition, aerobic exercise decreased the amplitude of the LPP response to negative pictures. Participants also self-reported a significant decrease in total mood disturbance following exercise. Although cortisol and sAA were significantly different from baseline measures, neither measure was related to the LPP ERP amplitude.

Conclusions: The findings suggest that acute aerobic exercise has a neuroprotective effect against emotionally negative stimuli.

Key Words: ERP, Late Positive Potential, POMS, Mood

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Introduction

A growing body of literature suggests that aerobic exercise improves mental and emotional well-being\(^1\),\(^2\). Relative to low-intensity exercise, high-intensity exercise results in a greater reduction in anxiety sensitivity and only high intensity exercise diminishes fear and anxiety related somatic arousal\(^3\). In a group of moderately depressed males, moderate- and high-intensity exercise was able to improve depression levels\(^4\). Interval aerobic training also reduced depressive symptoms in older adults\(^5\). The effect of aerobic exercise on emotion processing is robust and even extends to clinical populations. For example, a 3-month running intervention
resulted in significantly decreased depression, anxiety, and stress scores in adolescents and adults with diagnosed mood disorders.

Despite the clear benefits of aerobic exercise on emotion processing and mood, the physiological mechanisms through which exercise benefits emotion processing is currently unknown. Several theories have been proposed including mood changed through increased endorphins (i.e., the “runner’s high”), increased temperature (the thermogenic hypothesis), increased mitochondrial function, altered mTOR signaling, altered neurotransmitter levels, and changes in the stress-sensitivity of the HPA axis. It is not clear from these theories, however, how direct neural processing is altered after exercise to improve mood and emotion processing. One potential direct neurophysiological marker is the Electroencephalogram (EEG) Event Related Potential (ERP). A neurophysiological marker of decision making, the ERP P300 component, has already been shown to be a useful indicator of changes in cognitive processing after acute soccer exercise. In this study, reaction time was significantly faster during Stroop (interference task) performance and the P300 ERP amplitude was significantly larger during the task.

In agreement with the idea that the ERP is a potentially useful marker of neurophysiological changes after exercise, the current study employed the ERP late positive potential (LPP) component of the visual ERP to index emotion changes after exercise. The LPP ERP is established as a sensitive measure of attention to emotionally-charged visual stimuli. Induction of the LPP is thought to serve as a neurobiological correlate of motivated attention to stimuli of adaptive significance (sex, death, etc.). In support of this idea, the LPP is shown to be important for the memory formation of emotional events. Simultaneous fMRI and EEG recording show that the LPP is generated by an extensive network of cortical and subcortical regions with some differential activation by picture valence category (positive vs. negative).

The current study applied the LPP ERP measure to assess neural changes to emotionally negative and emotionally neutral picture stimuli after an acute (30 min) bout of high-intensity aerobic treadmill exercise or a control condition. We hypothesized that, relative to a control condition, high-intensity aerobic exercise would decrease the amplitude of the LPP response to negative pictures. We also assessed self-reported mood measures after the exercise and control condition. We predicted that, consistent with previous studies, acute high-intensity aerobic exercise would improve self-reported mood. Finally, since the HPA axis has been implicated in emotion processing changes after exercise and cortisol and noradrenaline also modulate neural responses to emotional picture viewing, we tested the idea that cortisol and salivary alpha amylase (sAA, a biomarker of norepinephrine), would correlate with any observed changes in the LPP after exercise.

Methods

Participants
Twenty undergraduate students (10 women; mean±SD: age = 20±3) were recruited from Nova Southeastern University (NSU) for course credit. We were unable to analyze EEG data from one participant due to excessive movement interference. Participants were asked to refrain from eating or drinking 1 hour prior to participation. All participants were right-handed and had normal or corrected-to-normal vision. Testing procedures were carried out according to a protocol approved by the Institutional Review Board (IRB) of the university. Exclusion criteria included a history of cardiovascular disease, having a psychiatric condition, or taking psychiatric medication.

Protocol
Participants were tested in a randomized crossover design that consisted of one exercise session and one control (i.e., no exercise) session scheduled one week apart. All testing took place in the afternoon between 2:30-4:30 pm in order to control for the circadian variation of cortisol. Physical characteristics such as weight, height, age, and sex were recorded. Then, all subjects filled out a demographic form, including questions about fitness habits. Saliva was then collected for a baseline measure. Next, the participants were exposed to either the treadmill exercise condition or control condition. Three additional saliva samples were taken after the exercise and the control condition (+1, 30, and 60 min). A stopwatch was used to ensure consistent timing throughout the experiment between participants. Following the +1 min saliva collection, participants filled out self-report mood assessments. Participants were then fitted with an electrode cap and the second saliva sample was taken (+30 min). Finally, the participants underwent the EEG testing procedures followed by the final (+60 min) saliva sample collection.

**Exercise and Control Procedures**
In the exercise condition, subjects were asked to run on a treadmill for 30 minutes (following a 5-minute warm-up). The speed of the treadmill was adjusted to keep each participant at 75-85% of their maximum estimated heart rate. Heart rate was measured using a Polar heart rate monitor (Polar USA). During the control session, subjects were required to stand for 40 minutes in order to control for orthostatic effects in the exercise condition.

**Biomarkers**
Saliva samples were collected from each participant through passive drool into polyethylene tubes. Immediately after collection, the sample tubes were stored in a -20°C freezer. Prior to biomarker quantification, the saliva samples were thawed, vortexed, and centrifuged at 3000 rpm (0.9 x g) for 15 minutes.

**Cortisol**
Saliva samples were run in duplicate and quantified via a human cortisol enzyme immunoassay (EIA) kit per the manufacturer’s instructions (Salimetrics LLC, USA). The samples were immediately read in a BioTek ELx800 plate reader (BioTek Instruments, Inc., USA) at 450 nm with a correction at 630 nm. All samples were within the detection ranges indicated in the cortisol immunoassay kit, and the variations of sample readings were within the expected limits. Final concentrations for the biomarkers were generated by interpolation from the standard curve in µg/dL.

**Salivary Alpha Amylase (sAA)**
Saliva samples were run in duplicate and quantified via a human Kinetic Enzyme Assay Kit per the manufacturer’s instructions (Salimetrics LLC, USA). The samples were immediately read in a BioTek ELx800 plate reader (BioTek Instruments, Inc., USA) at 405 nm. All samples were within the detection ranges indicated in the assay kit, and the variations of sample readings were within the expected limits. Final concentrations for the biomarkers were generated via absorptivity over two min and generated in U/mL of activity.

**Emotional stimuli**
Emotional responses were elicited by visual stimuli from the International Affective Picture System (IAPS) (Lang, Bradley, Cuthbert, 1997). The IAPS contains positive and negative pictures that are rated to be high on valence and arousal relative to neutral pictures. There were 105 trials and each trial began with a 400 ms randomized presentation of either a negative (n=35) neutral (n=35) or positive (n=35) IAPS picture followed by 3000 ms of a black screen. The IAPS normative ratings were used to select the emotional category of each picture. Independent picture sets were used for each time-point and the order was counter-balanced across participants. The
picture sets were matched on normative valence and arousal ratings for each picture category. Following the picture presentation, a black screen was on for the rest of the trial. All pictures were presented on a 23-inch LCD monitor with a vertical refresh rate of 60 Hz. A central fixation point was present in the center of the screen throughout the experiment. As a manipulation control (to ensure that the participants were attending to the pictures) participants were asked to rate the valence of each picture during the 3000 ms period following picture presentation. The pictures were rated on a 1-9 scale in accord with the normalized scores - 1 being most negative and 9 being most positive. Picture presentation and timing were controlled through the use of Presentation software (Neurobehavioral Systems, LLC).

Electrophysiological recordings
EEG assessment was conducted using Contact Precision Instruments' Psychlab EEG amplifying and recording equipment (Contact Precision Instruments, Cambridge, MA). EEG activity was recorded with a cap fitted with pure tin cup electrodes at Fz, Cz, Pz, C3, C4, O1, and O2 (Electro-Cap International, Eaton, OH) placed in accordance with the International 10–20 System. Eye movements and eye blinks were monitored via tin electrodes (Electro-Cap International, Eaton, OH) placed above and at the outer canthus of the left eye. Signals were referenced to linked electrodes attached to earlobes. Electrode impedance was maintained at less than 5 kΩ. Procedures for infection control specified by the Society for Psychophysiological Research were followed in attaching and removing electrodes (Putnam, Johnson, & Roth, 1992). The EEG amplifier was set at a gain of 30,000 and the sampling rate of the EEG was 500 Hz. High pass filters were set to .1 Hz and low pass filters were set to 40 Hz. A 60 Hz notch filter was active. The data were analyzed offline with Psylab8 software (Contact Precision Instruments, Cambridge, MA). For the ERP analyses, 1000 ms of raw EEG data were epoched to the respective stimulus presentation including a 100 ms pre-stimulus baseline. The LPP was measured as the average voltage between 300–800 ms following picture onset. Trials in which the EOG exceeded ±75 µV were excluded from the final averaged ERP. The ERP trials we also visually examined and individually rejected at each electrode location for any additional observed artifact (e.g. blocking, movement, alpha).

Mood Assessments
Anxiety
The State-Trait Anxiety Inventory (STAI) are two 20-item scales used to measure state and trait anxiety. We used the state sub-scale as our measure of state anxiety and the trait sub-scale as our measure of trait anxiety. The instrument has been used extensively in research and clinical practice. Spielberg et al. (1983) report internal consistency coefficients for young adults to be 0.93 for state anxiety and 0.92 for trait anxiety. Test-retest reliability coefficients range between 0.65 and 0.75. Moreover, it has been validated as an accurate measure of anxiety in adults and convergent and discriminant validation has been exhibited when compared with other measures.

Mood
The Profile of Mood States (POMS) was used to measure acute and ongoing mood. The 65 items include six scales assessing anger-hostility, confusion-bewilderment, depression-dejection, fatigue-inertia, tension-anxiety, and vigor-activity in addition to a composite score of total mood disturbance. McNair et al., (1992) report internal consistencies varying from 0.84 for the confusion-bewilderment scale, 0.95 for the vigor–activity scale, and 0.74 for depression-dejection scale.

Statistical Analysis
In order to examine the effect of picture category on the visual LPP across electrodes in the exercise vs. control condition, a 2 (session) x 2 (picture category)
repeated measures (RM) Analysis of Variance (ANOVA) was carried out. Consistent with previous LPP work \cite{19,20} the average of electrode locations Cz and Pz were analyzed. Cortisol and sAA were assessed by a 2 (session) \times 4 (collection time). A correlation analysis was also conducted to observe a possible relationship between Cortisol and/or sAA and LPP measures. The effect of exercise of self-reported mood measures was examined through paired samples t tests comparing the exercise to the control condition. All calculations were conducted using an SPSS statistical package (version 19, SPSS inc., IBM Company). In instances where the sphericity assumption was not met, the reported p-values associated with the F statistics were adjusted via Greenhouse–Geisser. All reported p values have an a priori significance level of \( p<0.05 \).

Results

LPP and Picture Analyses

Figure 1 presents the grand average LPPs for the two picture categories (neutral and negative) separated by condition (exercise vs. control). The LPP measure is an average of electrode locations Cz and Pz (where the LPP is most prominent). A repeated measures analysis of variance (ANOVA) revealed a main effect for picture type \((f(1,18)=31.88, p < 0.01)\). As expected, negative pictures evoked a larger LPP than neutral pictures. There was also a main effect of session \((f(1,18)=4.58, p < 0.01)\). There was not a significant time \(x\) session interaction. Follow up t tests showed that, relative to the control condition \((M = 4.94, SD = 6.88)\) exercise \((M = 1.85, SD = 2.27)\) significantly reduced the LPP to negative pictures \((t(18) = 2.13, p = 0.03)\).

![Figure 1](image)

**Figure 1.** Grand average LPPs to the two picture categories (neutral and negative) separated by picture type. The LPP measure is an average of electrode locations Cz and PZ (where the LPP is most prominent). As expected, negative pictures evoked a larger LPP than neutral pictures. Exercise significantly reduced the LPP to negative pictures. * indicates \( p < 0.05 \).

Self-Reported Mood

As shown in figure 2, paired samples t tests on the POMS total mood disturbance score revealed that, relative to the baseline condition \((M = 0.90, SD = 16.96)\) acute exercise significantly reduces total mood disturbance \((t(19) = 2.27, p < 0.05)\). Significant subcomponents of the POMS that were significantly different between exercise and control session included depression (exercise \(M = 0.65, SD = 1.39\), control \(M = 0.21, SD = 3.77\); \(t(19) = 2.38, p < 0.05\)) and vigor (exercise \(M = 19.25, SD = 6.26\), control \(M = 15, SD = 4.86\); \(t(19) = -3.09, p < 0.01\)). Trait anxiety at baseline was within the expected norms \((M = 33.35, SD = 6.38)\) \cite{21}. Fig 2 also shows that state anxiety was not significantly affected by exercise (control \(M = 30.4 SD = 10.13\), exercise \(M = 28.5 SD = 8.25\), \(p > 0.05\)).
Figure 2. Total self-reported mood disturbance (POMS) is significantly reduced after acute exercise (top left). Subcomponents of the POMS that were significantly different after exercise included depression and vigor (top right). State anxiety was not significantly affected by exercise (bottom). * indicates p < 0.05.

Biomarkers
As shown in figure 3, for sAA measures there was a significant main effect of time (F(3, 57) = 4.94, p = 0.004) and a main effect of session (F(1, 19) = 6.09, p = 0.02). There was also a significant time x session interaction (F(3, 57) = 3.08, p = 0.04). For cortisol there was a significant main effect of time (F(3, 57) = 2.98 p = 0.04). There was also a significant time x session interaction (F(3, 57) = 5.85, p =0.001). Follow up t test comparisons showed that the sAA levels were significantly higher in the exercise condition compared to the control condition 1 minute post-exercise (t(19) = 2.69, p = 0.02). Cortisol levels were significantly higher in the exercise condition compared to the control condition 1 minute post-exercise (exercise M = 0.23 SD = 0.21, control M = 0.13 SD = 0.07; t(19) = 2.24, p = 0.04) and 30 minutes post-exercise (exercise M = 0.23 SD = 0.19, control M = 0.11 SD = 0.06; t(19) = 2.62, p = 0.02). Correlation analyses did not reveal any relationship between LPP measures, sAA, and cortisol response to exercise (all p’s < 0.05).
Figure 3. Salivary Alpha Amylase (sAA) measures reflect the level of sympathetic arousal. Follow up analyses to significant RM ANOVA showed that sAA levels were significantly different between control and exercise at 1 min post-exercise and cortisol levels were significantly different between control and exercise at 1 and 30 mins post-exercise * indicates p < 0.05.

Discussion
The results of our study show that, relative to a baseline condition, an acute bout of aerobic exercise improves neurophysiological and self-report measures of emotion processing and increases physiological arousal. As expected, aerobic exercise acutely increased sympathetic arousal (measured through sAA) and HPA axis activity (measured through cortisol); however, these measures did not relate to the LPP ERP amplitude.

Our neurophysiological LPP ERP findings suggest that exercise decreases the brain’s response to emotionally negative stimuli. Although many cortical and subcortical brain regions are involved in LPP generation, the ventrolateral prefrontal cortex and insula are particularly associated with LPP amplitude in response to unpleasant pictures 22. Notably, these are areas that are also critical to subjective emotional experiences and emotion regulation 23-25. Our findings suggest that the exercise exposure, at least acutely, alters the activation of these neural systems.

This study also expands on previous work which tested an ERP measure of attention (the P300 response) during a Stroop Color Word Conflict Task following an acute indoor soccer match 8. However, in this study, an acute exercise treadmill condition did not show any influence of the ERP compared to control. The authors suggest that the influence on the ERP in the acute soccer, but not acute treadmill condition,
might be due to the fact that soccer is a cognitively engaging form of aerobic exercise. The current study directly builds upon this work by showing that acute exercise alone appears to exert benefits to emotion processing networks, without the need to overtly prime or engage these networks.

The self-report mood finding in our study is in agreement with previous studies which show an improvement in mood following acute exercise \textsuperscript{26-29}. Results from our study suggest that the overall change in mood (decrease in total mood disturbance) is driven primarily by an increase in vigor and a decrease in depressive symptomatology. This is notable since a review of exercise and mood suggests that exercise could be a significant adjuvant treatment for clinical depression \textsuperscript{30}. There was no influence of acute aerobic exercise on the state anxiety measure or the anxiety subcomponent of the POMS. The STAI Trait Anxiety measure showed that the current sample did not have high levels of anxiety at baseline (the average measure was within the expected norm for this age group). Therefore, it is not certain what effect, if any, acute aerobic exercise would have on anxiety in a clinical population. Further, it is possible that the increased activation of the sympathetic nervous system during exercise does not allow for an amelioration in anxiety (at least acutely).

Changes in both the autonomic nervous system (ANS) and the hypothalamic-pituitary-adrenal (HPA) axis were examined. Increased energy requirements induce the ANS and the HPA axis to release catecholamines and glucocorticoids into the blood stream, respectively. In humans, the major glucocorticoid, cortisol, is a particularly crucial biochemical mediator of changes in cognitive function \textsuperscript{31}. The salivary alpha amylase data suggests that the fast-acting sympathetic nervous response is significantly elevated by exercise (+1 min post-exercise), but quickly returns to baseline levels. Cortisol, which is more sensitive to psychological stress, was elevated at baseline- indicating anticipatory arousal. Of note, exercise did not significantly increase cortisol relative to this baseline measure; however, the levels were maintained with exercise for 30 minutes. In the control condition the cortisol levels decreased with exercise onset. Although changes in HPA axis functioning has been suggested to relate to mood changes after exercise, a correlation analysis did not reveal a relationship between LPP measures and cortisol response to exercise\textsuperscript{7}. A relationship between ANS activity (measured through sAA) and the LPP amplitude was also not found.

In summary, the current study examined the effects of acute aerobic exercise on self-report and neurophysiological measures of emotion processing. It was discovered that, relative to a control condition, acute aerobic exercise (at 75-85% max heart rate) results in a decrease in mood disturbance and a decrease in the LPP ERP response to emotionally negative images. Although cortisol and sAA were significantly different from baseline measures, neither measure related to the LPP ERP amplitude. Combined, these data suggest that acute aerobic exercise may decrease the neurophysiological response to negative information. We are planning on carrying out a follow up study that investigates the possibility that long-term exercise can be used to inoculate the brain against emotional insults.

**Media-Friendly Summary**

An acute bout of aerobic exercise blunts one’s perception of negative visual images.

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**Author Contributions**
JT, SS, and JA designed the study; SS and RP performed the experiments; JT, SS, RP, and JA, analyzed and interpreted the data. JT and JA wrote and edited the manuscript. All authors discussed the results and interpretations.

Reference


