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Effects of cycling exercise on vigor, fatigue, and electroencephalographic activity among young adults who report persistent fatigue

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Abstract

We previously reported that 6 weeks of exercise training had positive effects on feelings of vigor and fatigue among college students who reported persistent fatigue. Here we examined whether transient mood changes after single sessions of exercise would mimic those chronic effects and whether they would be related to changes in brain activity measured by electroencephalography (EEG). Feelings of vigor were higher after both low- and moderate-intensity exercise during Weeks 1, 3, and 6 compared to a control condition. Feelings of fatigue were lower after low-intensity exercise during Weeks 3 and 6. Posterior theta activity accounted for about half the changes in vigor. Studies that manipulate mood, EEG activity, or both during exercise are needed to determine whether EEG changes after exercise are causally linked with mood.

Descriptors: Aerobic exercise, Mood, Theta activity

Feelings of fatigue or low energy are a burden on public health. Persistent fatigue has an estimated point prevalence of 20% among adults in the United States and worldwide (Wessely, Hotopf, & Sharpe, 1998). It is a common reason for doctor visits and is often treated inadequately by health care providers (Lange, Cook, & Natelson, 2005). The cumulative evidence suggests a role for physical activity as an adjuvant in the primary and secondary prevention of persistent fatigue. Population-based studies show that physical activity is associated with lower odds that people will report feelings of fatigue and low energy (Puetz, 2006). Clinical trials of chronic exercise among sedentary adults with fatigue-related medical conditions showed improvement in feelings of energy and fatigue that compares favorably with effects of cognitive–behavioral interventions or treatment with drugs (Puetz, Beasman, & O'Connor, 2006; Puetz, O'Connor, & Dishman, 2006).

We recently reported positive effects on feelings of vigor and fatigue during 6 weeks of exercise training among inactive college students who complained of persistent fatigue (Puetz, Flowers, & O'Connor, 2008). Vigor was increased similarly regardless of exercise intensity, but fatigue was decreased only after low-intensity training. Results were most pronounced during the last week of training and were independent of changes in fitness.

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Those results suggested that chronic effects of exercise training on feelings of persistent fatigue or low energy might result from a gradual accumulation over several weeks of repeated, acute responses to each exercise session. To our knowledge, this possibility has not been investigated. We report here ancillary analyses of the aforementioned trial to examine the pattern of change in transient feelings of vigor and fatigue in response to single, repeated sessions of low-intensity or moderate-intensity exercise.

We further examined whether the expected changes in fatigue and vigor after acute exercise would be related to changes in brain activity measured by electroencephalography (EEG). Although the chronic results of the trial were positive, they did not clarify whether the effects of physical activity on feelings of vigor and fatigue were plausibly explainable by neurobiological mechanisms or whether they might have been partly artifact, influenced by experimental demand (i.e., responses according to expectations of a trial's goals) that bias participants' responses to self-report measures having transparent content (Morgan, 1997). Putative health benefits of physical activity are commonly known, and it is not feasible to blind participants to their participation in an exercise program. Hence, demonstration that participants' transient responses to acute exercise are consistent across time and are related to biological markers plausibly linked to feelings of vigor or fatigue would bolster the evidence to support real psychological benefits of exercise for people who report persistent fatigue.

PSYCHOPHYSIOLOGY

A substantial literature, including studies involving moderate submaximal and maximal exercise intensities (e.g., Hall, Ekkekakis, & Petruzzello, 2007; Petruzzello, Hall, & Ekkekakis, 2001; Petruzzello & Tate, 1997), is consistent with the theoretical view that hemispheric asymmetry in frontal alpha activity is both a neural mediator and a moderator of affective experience (Coan & Allen, 2004). The importance of EEG oscillatory activity in other frequencies and at other scalp locations for understanding feelings of energy or fatigue is uncertain. In a recent study of female college students without complaints of energy or fatigue problems, Woo, Kim, Kim, Petruzzello, and Hatfield (2009) reported that feelings of vigor were elevated above resting levels after 30 min of moderately intense treadmill running. That effect was attenuated after controlling for EEG activity in delta, theta, and alpha frequency bands measured at a right (F4), but not left (F3), anterior sensor location. The authors' interpretation was that the increase in vigor was explainable by a reduction in behavioral withdrawal processes, consistent with the theoretical view that asymmetrical activation of right anterior brain regions is a predominant feature of the neural circuitry of unpleasant affective experience (Davidson, 2001). However, although activity in each frequency band was increased at the right anterior site and decreased at the left anterior site, only the reduction in theta activity at the left anterior site (F3) was statistically different after exercise compared to rest.

An alternative theoretical position postulates that variations in posterior brain regions can be influential in the arousal dimension of affect (Heller, 1993). Although this theory has not yet been tested with people suffering from fatigue, predictions about increased left-hemisphere activity among people classified with high anxious apprehension relative to those classified with high anxious arousal have been supported (Heller, Nitschke, Etienne, & Miller, 1997). The potential relevance to the present work is that fatigue often accompanies anxious apprehension (Nitschke, Heller, Palmieri, & Miller, 1999), and other theorists have linked persistent fatigue to a dysregulation of arousal in the central nervous system (Pfaff, 2006).

We previously reported on the cumulative evidence indicating that acute exercise typically increases EEG activity in delta, theta, alpha, and beta frequencies regardless of recording site (Crabbe & Dishman, 2004), and we hypothesized that the increased metabolic arousal of exercise might elicit diffuse increases in cortical EEG activity via thalamic-cortical or thalamic-reticular-cortical circuitry (Steriade, Gloor, Llinás, Lopes de Silva, & Mesulam, 1990). In the study reported here, we were particularly interested in alpha and theta responses measured at both anterior and posterior sites because of three additional types of evidence. First, neurofeedback that elevates theta activity relative to alpha activity in the midline parietal region has been shown to increase feelings of energy (Raymond, Varney, Parkinson, & Gruzelier, 2005). Second, research on attention and executive cognitive functions such as working memory has revealed the importance of understanding dissociations between tonic and phasic changes in alpha and theta frequencies, especially in anterior–posterior neural networks (Klimesch, Freunberger, Sauseng, & Gruber, 2008). It was important to evaluate whether similar findings would be obtained for feelings of energy or fatigue after physical exertion. Third, viewing affective pictures that are rated as moderately or highly arousing has resulted in increased left frontal theta activity and larger bilateral increases at posterior parietal and occipital sites, with mixed and variable influences on alpha activity (Aftanas, Varlamov, Pavlov, Makhnev, & Reva, 2002).

The purpose of this report, therefore, was to examine whether acute sessions of exercise repeated during 6 weeks of training would result in transient changes in feelings of vigor and fatigue (i.e., "right now" feelings assessed immediately before and after each exercise session) that would be similar to the chronic effects (i.e., more enduring feelings indexed using a "past week, including today" time frame) that we reported earlier (Puetz et al., 2008). Further, we examined whether the expected changes in vigor and fatigue after acute exercise would be related to concurrent changes in brain activity measured by EEG. Our goal was not to test neural correlates of emotional responding (e.g., Crabbe, Smith, & Dishman, 2007) or neural predictors of mood responses after exercise (e.g., Petruzzello et al., 2001) but, rather, to test whether acute exercise would modify feelings of energy and fatigue and whether those effects would be mediated by altered alpha and theta activity at anterior or posterior parietal and occipital sites. We also examined whether the effects would differ either according to exercise intensity or across the weeks of training.

Materials and Methods

Participants

Healthy male and female college students between the ages of 18 and 35 were recruited from the University of Georgia via electronic mail sent to several campus groups. Recruitment procedures and participant characteristics have been described in detail elsewhere (Puetz et al., 2008). Participants were paid $60 as an incentive to participate in one of the conditions for 6 weeks. Controls also were offered participation in a 6-week supervised exercise program that started after their participation in the control condition was completed. An a priori statistical power analysis (D'Amico, Neilands, & Zambarano, 2001) showed that a sample of 36 participants would provide a statistical power of ≥ 0.80 for the main and interaction effects of the study design assuming a two-tailed α value of .05, a high correlation across repeated measures ($r = .80$), and an expected Cohen's d effect size of 0.60 for both EEG (Crabbe & Dishman, 2004) and feelings of fatigue and energy (Puetz et al., 2008).

Exclusion criteria were as follows: (a) the absence of persistent fatigue ($\geq$ 30 days), defined as a raw score of 17 or higher on the vitality scale of the SF-36 Health Survey; a raw score of 16 is approximately one-half standard deviation below U.S. population norms (Ware, 1993). Patients with chronic fatigue syndrome would have a longer chronicity of symptoms ($\geq$ 6 months vs. $\geq$ 30 days) and SF-36 vitality scores likely to be 1–2 standard deviations lower than the participants in the present study (Hardt et al., 2001). (b) The presence of contraindications to maximal exercise based on professional guidelines (American College of Sports Medicine, 2006). (c) A physically active lifestyle, defined as a weekly energy expenditure greater than one-half standard deviation below college-aged norms as measured by the 7-Day Physical Activity Recall questionnaire (Blair et al., 1985; Dishman & Steinhardt, 1988). (d) The self-reported use of any antidepressant medication within the last month. (e) Scores at or above a cutoff suggesting any of the following DSM-IV psychiatric disorders measured by the Psychiatric Diagnostic Screening Questionnaire (Zimmerman & Mattia, 2001a, 2001b): generalized anxiety disorder, panic disorder, social anxiety disorder, major depressive disorder, and substance abuse.

Design and Procedures

Thirty-six participants were randomly assigned to one of three conditions: moderate intensity aerobic exercise, low intensity aerobic exercise, or no-treatment control. Blocked randomization, which assured 12 participants in each condition, was performed using Research Randomizer (www.randomizer.org).

Following randomization, the week before the intervention began, mood, physical activity, and medication/nutritional supplement data were obtained. Week-to-week changes in the use of medications and nutritional supplements were monitored with a questionnaire created for use in this investigation. Participants in each condition subsequently visited the laboratory on 18 occasions over a 6-week period. Laboratory visits occurred three days per week. Each laboratory visit took place at approximately the same time each day. In addition to the 18 testing sessions, aerobic fitness testing occurred the week before the start and the week after the end of the 6-week study period.

Aerobic Fitness Testing

During aerobic fitness testing, all participants performed an incremental exercise test on an electronically braked, computer-driven cycle ergometer (Lode BV, Groningen, The Netherlands) in order to measure peak oxygen consumption (VO_{2peak}). The purpose of the VO_{2peak} test was to examine changes in fitness over the course of the study. Test results also were used to ensure that subsequent submaximal exercise training bouts were completed at an exercise intensity that was equal for each participant relative to his or her peak oxygen consumption and that the two exercise conditions differed so that participants were below ventilatory threshold during the low-intensity condition and above ventilatory threshold during the moderate-intensity condition.

Participants were fitted to the cycle ergometer and provided with standardized, taperecorded instructions for providing overall ratings of perceived exertion using a 6–20 scale (Borg, 1998). After inserting a mouthpiece for collecting expired gases, the participants performed a 5-min warm up at 25 W. The initial work rate for the exercise test was 50 W and the work rate continuously increased at a rate of 24 W per minute until the participant reached volitional exhaustion (Storer, Davis, & Caiozzo, 1990). Pedal rate was maintained at 50–70 rpm throughout the test and was verified by an automated revolution counter. An integrated metabolic measurement system (Parvo Medics TrueOne 2400) was used to measure ventilation, oxygen consumption, and carbon dioxide production and calculate respiratory exchange ratio measures every 15 s. Heart rate was measured continuously using a Polar S120 heart rate monitor (Polar Electro Oy, Kempele, Finland). Heart rate, perceived exertion, and work rate were recorded during the last 10 s of every minute during the test of peak oxygen consumption. Peak oxygen consumption was defined as the highest VO_2 value when two of three following criteria were satisfied: (a) respiratory exchange ratio ≥ 1.10, (b) heart rate within 10 beats per minute of age-predicted maximum (i.e., 220–age), or (c) rating of perceived exertion ≥ 18. All participants satisfied at least two of the three criteria. Ventilatory threshold (Beaver, Wasserman, & Whipp, 1986) was 60.8% (95% confidence interval [CI] = 57.6%–64.0%) of peak VO_2 and did not differ between groups, $F(2,26) = 0.807$, $p = .457$, $\eta^2 = .058$, across time, $F(1,26) = 3.0$, $p = .095$, $\eta^2 = .103$, or between groups across time, $F(2,26) = 0.206$, $p = .815$, $\eta^2 = .016$. Differences between and within groups ranged from 5% to 8%, which did not exceed the standard error of the test.

Intervention Conditions

Moderate-intensity exercise condition. During the 6-week training intervention, the participants in the moderate-intensity aerobic exercise condition performed 20 min of exercise at 75% VO_{2peak} on a cycle ergometer. Participants warmed up by cycling at 25 W for 5 min. Then participants cycled against a resistance that produced an intensity of 75% VO_{2peak} for 20 min. During the 4th and 14th minute of exercise, VO_2, heart rate, and perceived exertion data were collected. Based on the oxygen consumption measurements, an investigator made necessary adjustments to the work rate in order to maintain an intensity of 75% VO_{2peak} throughout the remainder of the exercise bout. The typical adjustment was a reduction in pedal resistance, resulting in a reduction in associated power output to account for the slow component rise in VO_2 that occurs during exercise at intensities exceeding ventilatory threshold. Intensity exceeded or equaled (1 participant) ventilatory threshold for all participants. After the 20-min exercise bout, participants cooled down by cycling at 25 W for 5 min. During the entire 30-min period, pedal rate was maintained at approximately 60 rpm. Participants completed all exercise sessions in a small room with the door closed in the presence of a single investigator who supervised the exercise. Social interactions were purposefully kept to a minimum in order to minimize possible effects of social interactions on mood and standardize the exercise setting.

Low-intensity exercise condition. The participants assigned to the low-intensity exercise condition performed 20 min of exercise at 40% VO_{2peak} on a cycle ergometer. Procedures and measures were identical to those previously described for the moderate intensity aerobic exercise condition except participants cycled at 40% VO_{2peak} for 20 min. Intensity was below ventilatory threshold for all participants.

No-treatment control condition. The participants assigned to the no-treatment control condition sat quietly on a cycle ergometer. Procedures and measures were identical to those previously described for the aerobic exercise conditions except participants sat on the cycle ergometer for 30 min instead of performing a 5-min warm up, 20-minute training session, and 5-min cool down. This group also was asked to maintain their current physical activity level for the duration of the study.

Adherence to exercise conditions. The overall adherence rate to the assigned condition for the three groups combined was 98.3% and did not differ between the groups, $F(2,33) = 2.03$, $p = .15$, $\eta^2 = .11$. The moderate intensity exercise, low intensity exercise, and no-treatment control groups completed an average of 97.2%, 98.3%, and 99.4% of the 18 prescribed sessions, respectively.

Outcome Measures

Mood States. Vigor and fatigue mood states were measured immediately before and 10 min after the acute exercise or no-treatment control conditions during the 1st (Week 1), 9th (Week 3) and 18th (Week 6) laboratory visit using the 30-item Profile of Mood States–Short Form (POMS-SF; McNair, Lorr, & Droppleman, 1992). Participants rated the intensity of 30 mood items on a 5-point Likert-type scale ranging from *not at all* to *extremely*. Participants were asked to respond as to how they felt "right now."

EEG measurement. Four minutes of eyes-closed continuous EEG data were recorded prior to and after conditions of exercise or quiet rest according to standard procedures (Pivik et al., 1993; Pizzagalli, 2007). The timing of postcondition EEG assessments started $\sim$ 6 min after exercise stopped. EEG data were recorded vertex referenced using a 256-sensor Geodesic Sensor Net and NetAmps 200 amplifiers (Electrical Geodesics; EGI, Eugene, OR). The sensor net was adjusted until all pedestals were properly seated on the scalp (i.e., not sitting on thick mats of hair that could result in bridging between sensors; e.g., Greischar et al., 2004). Individual sensor impedances were adjusted until they were below 50 kΩ (Ferree, Luu, Russell, & Tucker, 2001). In addition, an electrolyte bridge test was conducted between all pairs of sensors prior to recording (Tenke & Kayser, 2001), and, if there was evidence of bridging, sensors were adjusted until bridging was no longer evident (this was rarely required). Data were sampled at 500 Hz with an analog filter bandpass of 0.1–200 Hz.

Data Reduction

EEG data screening. Data were preprocessed following recommendations (with minimal modification) made by Junghofer, Elbert, Tucker, and Rockstroh (2000). Raw data were visually inspected off-line for bad sensor recordings. Bad sensors were interpolated (no more than 5% of sensors for any subject) using a spherical spline interpolation method as implemented in BESA 6.0 (MEGIS Software, Gräfelfing, Germany). Data were transformed to an average reference and digitally filtered from 0.5 to 58 Hz (12 db/octave rolloff, zero-phase) and notch-filtered at 60 Hz (4 Hz width). Eyeblink and cardiac artifact correction was achieved by using the Independent Component Analysis (ICA) toolbox in EEGLAB 4.515 (Delorme & Makeig, 2004) under Matlab (Version 7.0, MathWorks, Natick, MA). ICA allows artifact removal without spatially distorting the data by using higher-order statistics to produce temporally independent signals (Onton, Westerfield, Townsend, & Makeig, 2006). Independent components representing saccades, blinks, and heart rate artifact were removed according to published guidelines (Jung et al., 2000). Data were then transformed to an 81-sensor virtual surface Laplacian montage using BESA to improve spatial resolution (Nunez & Srinivasan, 2006). The surface Laplacian is the second spatial derivative of the scalp potential recorded at the sensors, which accentuates neural activities associated with radially oriented superficial cortical sources, the signals that are best measured with EEG (Nunez & Srinivasan, 2006).

EEG data reduction. After artifact removal and before transformation to the frequency domain, the mean and linear trends were removed using Matlab. The data were then fast-Fourier transformed to absolute power ($\mu V^2/Hz$) using a moving Hanning window (50% overlap) on 10-s segments of the data and averaged over each minute. Intraclass correlation coefficients (ICC-2) were high (mean ICC-2 = .96, range: .722–.997) for each minute of data obtained prior to each of the three acute exercise or control conditions; therefore, each minute was averaged across the 4 min. Because of nonnormality, a natural log transform was performed, with FFT power expressed as $\log_n(\mu V^2/Hz)$ averaged across four frequency bands: theta (4–7 Hz), alpha (8–13 Hz), low beta (13–20 Hz), and high beta (20–30 Hz). To evaluate which scalp locations were most active, grand average top-meridian plots were constructed in BESA for each frequency band. These plots were visually inspected for regions of highly active sensor groupings, and then mean power was calculated within these regions. Two sensor groupings (anterior and posterior) consistently emerged from the visual inspection of the top meridian plots and were therefore used for subsequent statistical analyses (see Figure 1). Channels included in each frequency band are as follows: anterior theta, low beta, and high beta = 11 sites centered on Fpz and AFz; posterior theta and high beta = 15 sites centered on Oz; posterior alpha = 10 sites centered on P3 and P4; posterior low beta = 26 sites centered on POz and Oz. ICC values within these regions were high (ICC-2 $>$ .90).

Data Analysis

The primary dependent measures were mood ratings of vigor and fatigue and EEG power. Results did not differ according to hemisphere, so, as for mood ratings, EEG effects were tested for anterior and posterior regions using a series of 3 (group: moderate- and low-intensity exercise, quiet rest control) $\times$ 2 (time: pre- vs. post-condition) $\times$ 3 (week: Week 1, Week 3, Week 6) mixed model analyses of variance (RM-ANOVA) with time and week repeated (SPSS Windows Version 15, Chicago, IL). Sphericity adjustments were made using Huynh-Feldt ε. After determining directional effects of change in each group, hypothesis testing was conducted by RM-ANCOVA using the precondition score as a time-varying covariate. Bonferroni corrected simple contrasts of adjusted means were used to decompose significant effects (two-tailed). Effect sizes associated with the F statistics are expressed as η^2.

To examine whether changes in brain activity mediated changes in mood, EEG power was added as a time-varying covariate in each RM-ANCOVA for mood (Coan & Allen, 2004). This resulted in a 3 (group: moderate- and low-intensity exercise, quiet rest control) $\times$ 3 (week: Week 1, Week 3, Week 6) RM-ANCOVA controlling for precondition differences on the mood scores and for change scores (post–pre) of EEG activity. For this model, statistical findings were of interest if the addition of EEG as a covariate changed the mood model results from a statistically significant F value to an insignificant F, which would indicate that the EEG response accounted for some of the changes in mood (i.e., partially mediated the effect of experimental condition; MacKinnon, Fairchild, & Fritz, 2007).

Results

Mood

For vigor, there was a Group $\times$ Time effect, $F(2,30) = 9.238$, $p = .001$, $\eta^2 = .38$, but not a Group $\times$ Time $\times$ Week effect, $F(4,60) = 1.97$, $p = .11$, $\eta^2 = .12$, indicating differences between the groups in change after each session (see Figure 2). Vigor was increased after low-intensity, $F(1,9) = 9.32$, $p = .014$, $\eta^2 = .51$, and moderate-intensity, $F(1,11) = 7.26$, $p = .021$, $\eta^2 = .40$, exercise and was reduced after the control condition, $F(1,10) = 8.88$, $p = .014$, $\eta^2 = .47$. The Group $\times$ Time effect was not changed after controlling for precondition scores, $F(2,29) = 8.77$, $p = .001$, $\eta^2 = .38$. Contrasts indicated that feelings of vigor were higher after both low-intensity exercise ($p = .002$) and moderate-intensity exercise ($p = .032$) compared to the control condition and that vigor was similar after moderate- and low-intensity exercise ($p = .629$; see Figure 2).

For fatigue, there was a Group $\times$ Time $\times$ Week effect, $F(4,60) = 2.84$, $p = .032$, $\eta^2 = .16$, that was quadratic ($p = .015$; see Figure 3). Fatigue was reduced after low-intensity exercise during the last two sessions (Time $\times$ Week quadratic effect),

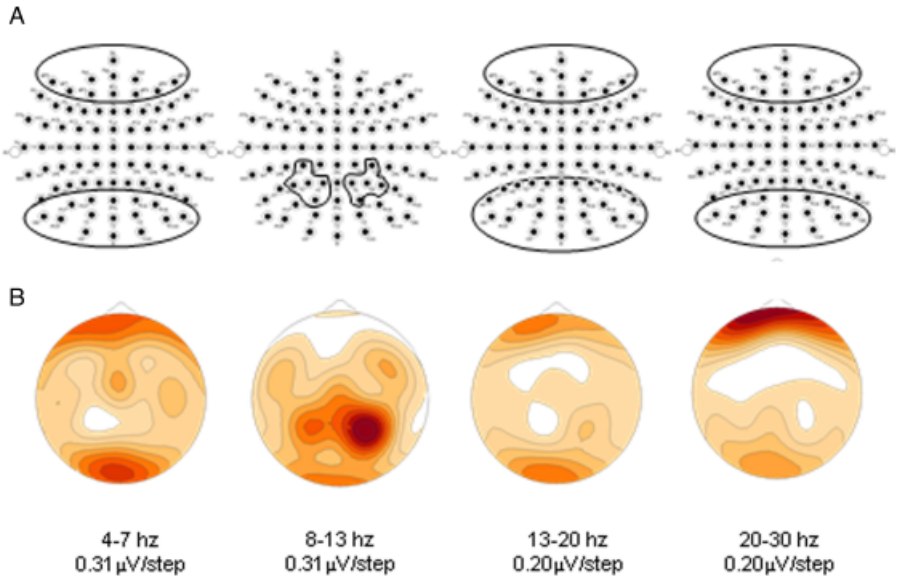

Figure 1 (A) Top-down maps depicting the channels that were included in the analysis for each frequency band. (B) Top meridian plots of grand average FFT power (averaged across group, time, and week) for each frequency band across the 4-min data collection period. These plots indicate that there might be anterior/posterior mirroring of alpha power, although we used the surface Laplacian transformation, which should serve to minimize such effects (Nunez & Srinivasan, 2006).

$F(1,9) = 5.20$, $p = .048$, $\eta^2 = .37$, but was unchanged after moderate-intensity exercise (p values $> .145$) and the control condition ($p > .440$). The Week $\times$ Group effect was attenuated slightly after controlling for precondition scores, $F(4,59) = 2.40$, $p = .060$, $\eta^2 = .14$. Contrasts indicated that fatigue was lower after low-intensity exercise compared to both the moderate-intensity and control conditions during Sessions 2 ($p = .016$) and 3 ($p = .05$) but not Session 1 ($p = .395$).

EEG Power

There were no significant main or interaction effects for EEG activity in the anterior channels (all p values $\geq .51$, all η^2s $\leq .03$), indicating that EEG activity was not different between groups or across time over anterior sites. However, significant Group $\times$ Time effects were detected for EEG activity in posterior regions for all frequency bands (all F ratios ≥ 6.00, all p values $\leq .006$, all η^2s $\geq .27$). There were no Group $\times$ Week or Time $\times$ Week effects (all η^2s $\leq .05$), indicating activity in all frequency bands was different between groups after each session, but not across weeks.

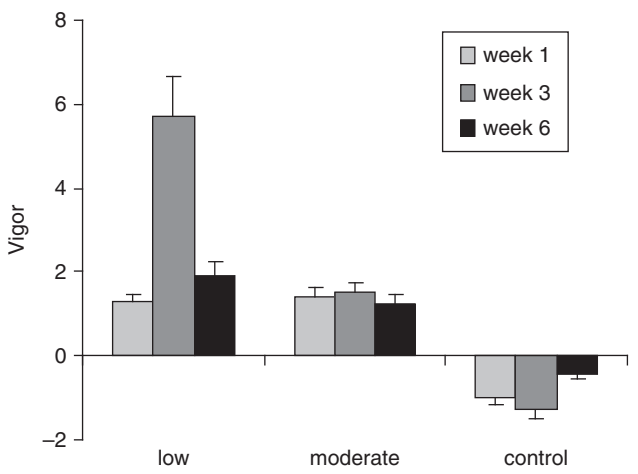

Figure 2. POMS-SF vigor change scores (post- minus precondition) $\pm$ *SEM* for moderate- and low-intensity cycling exercise and the control condition.

Alpha activity was increased after low-intensity exercise, $F(1,11) = 6.23$, $p = .03$, $\eta^2 = .36$, but activity in other frequencies was unchanged after low-intensity exercise (all F ratios ≤ 1.74, all p values $\geq .214$, all η^2s $\leq .14$). Conversely, activity in all frequencies was decreased after moderate-intensity exercise and the control condition (all F ratios ≥ 9.44, all p values $\leq .01$, all η^2s $\geq .36$) with the exception that alpha activity was unchanged after the control condition, $F(1,12) = 2.35$, $p = .15$, $\eta^2 = .18$.

Results were not different for group effects (all F ratios ≥ 5.56, all p values $\leq .008$, all η^2s $\geq .26$) and Group $\times$ Week effects (all F ratios $\leq .89$, all p values $\geq .474$, all η^2s $\leq .052$) after controlling for precondition values. Contrasts indicated that activity in all frequencies was higher after low-intensity exercise than after the control condition (all p values $\leq .033$). Alpha activity after low-intensity exercise was also higher than after moderate-intensity exercise ($p = .047$). Otherwise, activity did not differ between exercise conditions or between the moderate-intensity and control conditions (all p values $\geq .314$).

Relationship between Changes in EEG Power and Mood

The higher vigor observed after both low- and moderate-intensity exercise repeated during Weeks 1, 3, and 6, $F(2,29) = 8.77$, $p = .001$, $\eta^2 = .38$, was partially mediated by change in theta activity (4–7 Hz) in the posterior region (see Figure 4): adjusted model, $F(2,29) = 2.78$, $p = .08$, $\eta^2 = .17$. EEG activity in all other regions and frequency bands did not account for the change in feelings of vigor (all p values $\leq .05$ for all adjusted models). The lower fatigue observed after low-intensity exercise during Sessions 2 and 3, $F(4,59) = 2.40$, $p = .060$, $\eta^2 = .14$, was

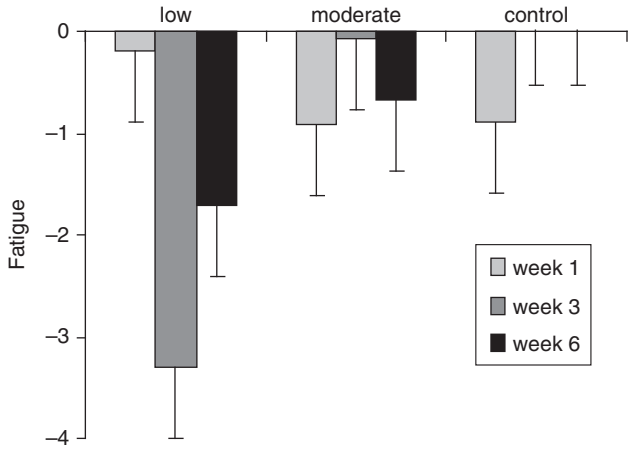

Figure 3. POMS-SF fatigue change scores (post- minus precondition) ± *SEM* for low- and moderate-intensity cycling exercise and the control condition.

not explained by change in EEG activity (p values $\leq .065$ and $\eta^2 \geq .139$ for all adjusted models).

Discussion

There were two important outcomes of the study. First, transient increases in feelings of vigor and decreases in feelings of fatigue experienced after each exercise session of low or moderate intensity paralleled results of our prior report that chronic exercise training led to improved feelings of vigor and fatigue experienced during the preceding week (Puetz et al., 2008). Reduced fatigue occurred only after low-intensity exercise and only during the second and third exercise sessions, suggesting that feelings of vigor are more sensitive to the effects of acute exercise than are feelings of fatigue among young people who report persistent fatigue. Second, and most importantly, half of the effect of exercise on feelings of vigor was mediated by changes in occipital-parietal brain activity in the theta frequency range, suggesting that exercise-induced modifications in mood are not simply a function of experimental demand characteristics. The significance of these findings for understanding the relationships between exercise and its effects on mood and specific cortical brain activity is discussed next.

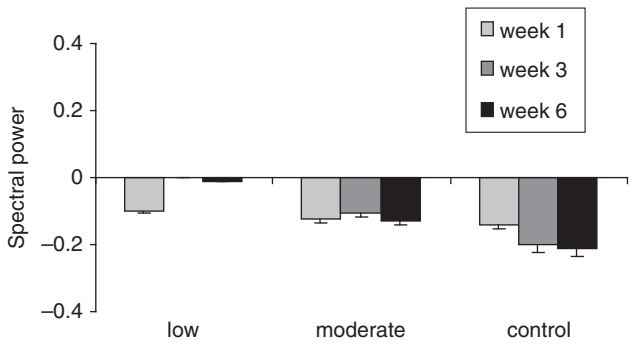

Figure 4. Change scores (post- minus precondition) for posterior theta-band spectral density and low-intensity and moderate-intensity cycling exercise and the control condition.

People without clinical mood disorders rank exercise near the top among the behaviors they use to self-manage their moods (Thayer, Newman, & McClain, 1994). Even short bouts (i.e., 10 min) of walking for exercise in natural settings are accompanied by feelings of increased energy (Thayer, 1987). To our knowledge, however, this is the first experimental report indicating that acute exercise improves transient feelings of energy and fatigue in people who report persistent fatigue. Our participants' scores on other scales of the POMS were in the normal range for college students and were not affected by exercise (data not shown). Hence, our results do not appear to represent an artifact of participant expectancies of general benefits of exercise for improving mood. Despite suggestions that the mood-enhancing effects of exercise may be slow to develop among persons less experienced with an exercise routine (Hoffman & Hoffman, 2008), the present findings offer further promise for exercise as a clinical tool to combat the deleterious public health costs of persistent fatigue and low energy (e.g., Lange et al., 2005; Puetz, 2006).

Previous research used the POMS to assess the effects on mood of acute exercise in young women without persistent fatigue. During short bouts (~ 8 min), higher-intensity exercise (cycling at 100 W) led to increases in subjective fatigue, whereas vigor was increased after low-intensity exercise (cycling at 25 W) among female medical students (Steptoe & Cox, 1988). In another sample of young women, fatigue was increased and vigor was decreased similarly after each of two maximal exercise tests separated by 1 month (Pronk, Crouse, & Rohack, 1995). Among middle-aged women and men without cardiopulmonary or metabolic diseases, 20 min of self-selected, moderate intensity treadmill exercise were followed by increased vigor and decreased fatigue among regular exercisers but not among those who had not exercised regularly during the preceding 6 months (Hoffman & Hoffman, 2008). Women and men who were receiving treatment for major depressive disorder reported reductions in fatigue as well as an increase in vigor after 30 min of moderately intense (60%–70% maximal heart rate) treadmill exercise (Bartholomew, Morrison, & Ciccolo, 2005). Only the changes in vigor were greater than those reported after a similar period of quiet rest. In another study of middle-aged women and men diagnosed with depression or anxiety disorders, 20 min of cycling exercise were followed by decreased feelings of fatigue (measured by a subjective exercise experiences scale) when the intensity was 50% of maximal heart rate reserve (~ 58 W), but fatigue was unchanged after a self-selected intensity that was higher (~ 66 W; Knapen et al., 2008). An interesting question for future research is whether transient feelings of vigor after early sessions of low- or moderate-intensity exercise might alter people's awareness or interpretation of their fatigue after exertion and subsequently contribute to reductions in persistent feelings of fatigue or low energy.

Most studies and literature reviews of the effect of acute exercise on brain electrocortical activity measured by EEG have emphasized, or have been restricted to, reports of changes in alpha frequency band activity (e.g., Kubitz & Pothakos, 1997). The common premise has been that increased alpha activity reflects a state of decreased cortical activation indicative of fatigue, relaxation, or decreased anxiety (Boutcher & Landers, 1988; Pineda & Adkisson, 1961). Alpha activity in response to exercise may be related to increased activity in somatosensory afferents (Youngstedt, Dishman, Cureton, & Peacock, 1993). This idea is consistent with evidence, including the data we report here, of increased alpha activity measured at central and temporal sites

that overlay portions of the sensorimotor cortex that receive afferent feedback during exercise. Sensorimotor alpha has been called the mu rhythm (Kuhlman, 1978), and neuroimaging data indicate that it is directly associated with brain activation in the sensorimotor cortex (Ritter, Moosmann, & Villringer, 2009). Alpha activity in the present study was increased only after low-intensity exercise, and that change did not account for feelings of vigor or fatigue after exercise.

Despite the historical focus on alpha activity, a systematic review of the exercise literature showed that acute exercise is accompanied by increases in EEG activity in all the measured frequency bands regardless of recording site (Crabbe & Dishman, 2004). A pattern of nonspecific changes in frequency bands associated with exercise would be consistent with research on the neuronal and neurophysiological architectures supporting oscillatory brain activity. For instance, alpha and theta frequencies traditionally are considered to recruit different neuronal pools when measured with scalp-level recordings (Pizzagalli, Oakes, & Davidson, 2003; Schürmann & Basar, 1994), and to index unique cognitive processes or states of the nervous system (Pfurtscheller, 1992). However, recent work at the microscopic (cellular) level indicated a direct link between relative amounts of alpha/theta composition in the EEG and thalamocortical inputs in the same neuronal architecture (see Hughes & Crunelli, 2005). Thalamocortical neurons can generate alpha/theta oscillations after the activation of either metabotropic glutamate receptors (mGLUR) or muscarinic acetylcholine receptors (mAchR) (Hughes et al., 2008). In cat lateral geniculate nucleus preparations, pharmacological activation of mGLUR1a leads to increased alpha activity. Deactivation at the same synapses leads to a dose-response-related decrease in the idling frequency of these neurons and, therefore, a relative increase in amount of theta activity (Hughes et al., 2004). Activation of mGLURs is also associated with increased high threshold burst firing in the alpha/theta frequency range in the motor and somatosensory regions of the thalamus (Hughes et al., 2008). This work suggests that a decrease in thalamocortical input via mGLUR or mAchR activation may determine relative alpha/theta concentrations in the spontaneous EEG. Also, activation of afferent inflow from carotid and cardiopulmonary baroreceptors into cardiovascular centers in the brain stem is alerting and increases indices of arousal, including hippocampal theta activity and cortical EEG activity in the insular cortex, a cardiovascular–limbic interface (Morgane, Galler, & Mokler, 2005; Oppenheimer, 1992; Spyer, 1989; Yu & Blessing, 2000). In humans, acute exercise increases cerebral blood flow in both the thalamus and insular cortex (Williamson, McColl, Mathews, Ginsburg, & Mitchell, 1999).

Generalized effects of exercise on attention or arousal (Magnie et al., 2000) can plausibly influence brain electrocortical activity in several frequencies and loci, and it is plausible that brain cortical systems are altered generally in response to the increased metabolic arousal of physical exertion and regulation of physical fatigue by the brain (Nielsen, Hyldig, Bidstrup, Gonzalez-Alonso, & Christoffersen, 2001; Nybo & Nielsen, 2001; Nybo & Secher, 2004). Other research also implicates thalamocortical feedback in modification of oscillatory brain activities associated with exercise via sympathetically mediated changes in epinephrine and norepinephrine at higher intensities of exercise that elicit mechanoceptive and nociceptive signaling (Stock et al., 1996). The extent to which specifically cortico-thalamic input and associated neurocircuity are regulated or modified, or both, by varying intensities of exercise, therefore, would be a valuable avenue to pursue in subsequent research.

Most prior studies that assessed nonspecific EEG activity associated with feelings of energy after acute exercise focused on frontal cortex activity (e.g., Petruzzello et al., 2001; Woo et al., 2009). Researchers recently have begun to examine more brain regions, reporting varying mood-related effects (e.g., increased calmness, reduced psychological strain) of different intensities of running on EEG alpha, beta, and gamma activity measured at anterior, temporal, parietal, and occipital sites among experienced runners (Schneider et al., 2009). Smit, Eling, Hopman, and Coenen (2005) reported that 40 min of moderately intense cycling exercise were followed by increased feelings of general activation and increased EEG power in low beta frequencies measured with eyes closed at the midline parietal region. There were no effects of exercise on theta or alpha frequencies at either frontal or parietal sites.

Similarly, in the present report, there were no between-groups effects on EEG activity measured over frontal cortex. Rather, all effects were associated with activity over posterior brain regions. The present finding of associations between mood and posterior cortical activations is consistent with previous reports. For instance, Heller and colleagues have reported increases in self-ratings of arousal and cardiovascular and sympathetic nervous system activity related to electrocortical activation in parietal and temporal regions (Heller, 1990; Heller et al., 1997; Heller, Nitschke, & Miller, 1998).

There are many possible reasons for the difference between previous studies and the present report on distributions of brain activations associated with exercise and changes of mood, one of which may be the study here of persons reporting persistent fatigue. Additionally, EEG theta power may vary in scalp topography as a function of caffeine sensitivity (Retey et al., 2006), which may be mediated by a polymorphism of the adenosine A_{2A} receptor gene (Retey et al., 2007). Such findings are also of interest given numerous investigations reporting a link between theta activity and alertness or feelings of fatigue (e.g., Lal & Craig, 2001). Caffeine sensitivity and its relationship to exercise-induced changes in EEG and mood have not been investigated. In our prior quantitative review of brain electrocortical activity during and after exercise, we analyzed a dozen potential moderators of the effects of acute exercise on EEG alpha activity. Several variables were found to significantly moderate the effect, including exercise duration, EEG recording latency from the end of exercise, time of day, whether the eyelids were opened or closed, the body position during EEG recording, whether the EEG reference scheme was reported, age, and whether the study did or did not include a control condition (Crabbe & Dishman, 2004).

Differences between previous reports and the present study might also be attributed to EEG quantification schemes. Neural activity recorded at the scalp at any one sensor and at any single time point contains activity from multiple neural generators (Dien, Beal, & Berg, 2005). Locations of sources in the brain, therefore, cannot be directly inferred from the spatial distribution of EEG activity without first performing a source-space transformation. For the present report we used the surface Laplacian transformation, which has the desirable properties of requiring few underlying assumptions for its computation (Nunez & Srinivasan, 2006) but still being proportional to the dura potential (Srinivasan, Nunez, Tucker, Silberstein, & Cadusch, 1996). On the one hand, this transformation accentuates superficial neural activities that are oriented radially with respect to the sensors, which are the signals best measured with EEG. On the other hand, the surface Laplacian attenuates deep, nonfocal

sources that can be either the cause of diffuse noise when examining cortical activity or be related to true brain activations (Nunez & Srinivasan, 2006). The quantification scheme used here, therefore, was conservative with respect to the signals measured, which may have resulted in the attenuation of real neural activations that are difficult to discern from nonspecific neural or system noise, or both. The best way to resolve this issue in future investigations is by using multiple imaging modalities to collect brain activity data from the same participants (e.g., Dale & Halgren, 2001). Such multimodal neuroimaging work has the potential to more fully elucidate how brain activity mediates the relationship between changes in mood and exercise.

REFERENCES

Aftanas, L. I., Varlamov, A. A., Pavlov, S. V., Makhnev, V. P., & Reva, N. V. (2002). Time-dependent cortical asymmetries induced by emotional arousal: EEG analysis of event-related synchronization and desynchronization in individually defined frequency bands. *International Journal of Psychophysiology*, *44*, 67–82.

American College of Sports Medicine. (2006). *ACSM's guidelines for exercise testing and prescription* (7th ed). Philadelphia: Lippincot Williams & Wilkins.

Bartholomew, J. B., Morrison, D., & Ciccolo, J. T. (2005). Effects of acute exercise on mood and well-being in patients with major depressive disorder. *Medicine and Science in Sports and Exercise*, *37*, 2032–3037.

Beaver, W. L., Wasserman, K., & Whipp, B. J. (1986). A new method for detecting anaerobic threshold by gas exchange. *Journal of Applied Physiology*, *60*, 2020–2027.

Blair, S. N., Haskell, W. L., Ho, P., Paffenbarger, R. S. Jr., Vranizan, K. M., Farquhar, J. W., et al. (1985). Assessment of habitual physical activity by a seven-day recall in a community survey and controlled experiments. *American Journal of Epidemiology*, *122*, 794–804.

Borg, G. (1998). *Borg's perceived exertion and pain scales*. Champaign: Human Kinetics.

Boutcher, S. H., & Landers, D. M. (1988). The effects of vigorous exercise on anxiety, heart rate, and alpha activity of runners and nonrunners. *Psychophysiology*, *25*, 696–702.

Coan, J. A., & Allen, J. J. (2004). Frontal EEG asymmetry as a moderator and mediator of emotion. *Biological Psychology*, *67*, 7–49.

Crabbe, J. B., & Dishman, R. K. (2004). Brain electrocortical activity during and after exercise: A quantitative synthesis. *Psychophysiology*, *41*, 563–574.

Crabbe, J. B., Smith, J. C., & Dishman, R. K. (2007). Emotional & electroencephalographic responses during affective picture viewing after exercise. *Physiology & Behavior*, *90*, 394–404.

Dale, A. M., & Halgren, E. (2001). Spatiotemporal mapping of brain activity by integration of multiple imaging modalities. *Current Opinion in Neurobiology*, *11*, 202–208.

D'Amico, E. J., Neilands, T. B., & Zambarano, R. (2001). Power analysis for multivariate and repeated measures designs: A flexible approach using the SPSS MANOVA procedure. *Behavior Research Methods*, *33*, 479–484.

Davidson, R. J. (2001). Toward a biology of personality and emotion. *Annals of the New York Academy of Sciences*, *935*, 191–207.

Delorme, A., & Makeig, S. (2004). Eeglab: An open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *Journal of Neuroscience Methods*, *134*, 9–21.

Dien, J., Beal, D. J., & Berg, P. (2005). Optimizing principal components analysis of event-related potentials: Matrix type, factor loading weighting, extraction, and rotations. *Clinical Neurophysiology*, *116*, 1808–1825.

Dishman, R., & Steinhardt, M. (1988). Reliability and concurrent validity for a 7-d re-call of physical activity in college students. *Medicine and Science in Sports and Exercise*, *20*, 14–25.

Ferree, T. C., Luu, P., Russell, G. S., & Tucker, D. M. (2001). Scalp electrode impedance, infection risk, and EEG data quality. *Clinical Neurophysiology*, *112*, 536–544.

Greischar, L. L., Burghy, C. A., van Reekum, C. M., Jackson, D. C., Pizzagalli, D. A., Mueller, C., et al. (2004). Effects of electrode density and electrolyte spreading in dense array electroencephalographic recording. *Clinical Neurophysiology*, *115*, 710–720.

Hall, E. E., Ekkekakis, P., & Petruzzello, S. J. (2007). Regional brain activity and strenuous exercise: Predicting affective responses using EEG asymmetry. *Biological Psychology*, *75*, 194–200.

Hardt, J., Buchwald, D., Wilks, D., Sharpe, M., Nix, W. A., & Egle, U. T. (2001). Health-related quality of life in patients with chronic fatigue syndrome: An international study. *Journal of Psychosomatic Research*, *51*, 431–434.

Heller, W. (1990). *The neuropsychology of emotion: Developmental patterns and implications for psychopathology* (*Vol. 1*). Hillsdale, NJ: Lawrence Erlbaum Associates.

Heller, W. (1993). Neuropsychological mechanisms of individual differences in emotion, personality, and arousal. *Neuropsychology*, *7*, 476–489.

Heller, W., Nitschke, J. B., Etienne, M. A., & Miller, G. A. (1997). Patterns of regional brain activity differentiate types of anxiety. *Journal of Abnormal Psychology*, *106*, 376–385.

Heller, W., Nitschke, J. B., & Miller, G. A. (1998). Lateralization in emotion and emotional disorders. *Current Directions in Psychological Science*, *7*, 26–32.

Hoffman, M. D., & Hoffman, D. R. (2008). Exercisers achieve greater acute exercise-induced mood enhancement than nonexercisers. *Archives of Physical Medicine and Rehabilitation*, *89*, 358–363.

Hughes, S. W., & Crunelli, V. (2005). Thalamic mechanisms of EEG alpha rhythms and their pathological implications. *The Neuroscientist*, *11*, 357–372.

Hughes, S. W., Errington, A., Lorincz, M. L., Kékesi, K. A., Juhász, G., Orbán, G., et al. (2008). Novel modes of rhythmic burst firing at cognitively-relevant frequencies in thalamocortical neurons. *Brain Research*, *1235*, 12–20.

Hughes, S. W., Lörincz, M., Cope, D. W., Blethyn, K. L., Kékesi, K. A., Parri, H. R., et al. (2004). Synchronized oscillations at alpha and theta frequencies in the lateral geniculate nucleus. *Neuron*, *42*, 253–268.

Jung, T. P., Makeig, S., Humphries, C., Lee, T. W., McKeown, M. J., Iragui, V., et al. (2000). Removing electroencephalographic artifacts by blind source separation. *Psychophysiology*, *37*, 163–178.

Junghofer, M., Elbert, T., Tucker, D. M., & Rockstroh, B. (2000). Statistical control of artifacts in dense array EEG/MEG studies. *Psychophysiology*, *37*, 523–532.

Klimesch, W., Freunberger, R., Sauseng, P., & Gruber, W. (2008). A short review of slow phase synchronization and memory: Evidence for control processes in different memory systems? *Brain Research*, *1235*, 31–44.

Knapen, J., Sommerijns, E., Vancampfort, D., Sienaert, P., Pieters, G., Haake, P., et al. (2008). State anxiety and subjective well-being responses to acute bouts of aerobic exercise in patients with depressive and anxiety disorders. *British Medical Journal*, *43*, 756–759.

Kubitz, K. A., & Pothakos, K. (1997). Does aerobic exercise decrease brain activation? *Journal of Sport and Exercise Psychology*, *19*, 291–301.

Kuhlman, W. N. (1978). Functional topography of the human mu rhythm. *Electroencephalography and Clinical Neurophysiology*, *44*, 83–93.

Lal, S. K. L., & Craig, A. (2001). A critical review of the psychophysiology of driver fatigue. *Biological Psychology*, *55*, 173–194.

Lange, G., Cook, D. B., & Natelson, B. H. (2005). Rehabilitation and treatment of fatigue. In J. DeLuca (Ed.), *Fatigue as a window to the brain* (pp. 301–316). Cambridge, MA: MIT Press.

MacKinnon, D. P., Fairchild, A. J., & Fritz, M. S. (2007). Mediation analysis. *Annual Review of Psychology*, *58*, 593–614.

Magnie, M. N., Bermon, S., Martin, F., Madany-Lounis, M., Suisse, G., Muhammad, W., et al. (2000). P300, n400, aerobic fitness, and maximal aerobic exercise. *Psychophysiology*, *37*, 369–377.

McNair, D. M., Lorr, M., & Droppleman, L. F. (1992). *Edits manual for the profile of mood states*. San Diego, CA: Edits/Educational and Industrial Testing Service.

Morgan, W. P. (1997). Methodological considerations. In W. P. Morgan (Ed.), *Physical activity and mental health* (pp. 3–32). Washington, DC: Taylor & Francis.

Morgane, P. J., Galler, J. R., & Mokler, D. J. (2005). A review of systems and networks of the limbic forebrain/limbic midbrain. *Progress in Neurobiology*, *75*, 143–160.

Nielsen, B., Hyldig, T., Bidstrup, F., Gonzalez-Alonso, J., & Christoffersen, G. R. J. (2001). Brain activity and fatigue during prolonged exercise in the heat. *Pflügers Archives: European Journal of Physiology*, *442*, 41–48.

Nitschke, J. B., Heller, W., Palmieri, P. A., & Miller, G. A. (1999). Contrasting patterns of brain activity in anxious apprehension and anxious arousal. *Psychophysiology*, *36*, 628–637.

Nunez, P. L., & Srinivasan, R. (2006). *Electric fields of the brain: The neurophysics of EEG*. New York: Oxford University Press.

Nybo, L., & Nielsen, B. (2001). Perceived exertion is associated with an altered brain activity during exercise with progressive hyperthermia. *Journal of Applied Physiology*, *91*, 2017–2023.

Nybo, L., & Secher, N. H. (2004). Cerebral perturbations provoked by prolonged exercise. *Progress in Neurobiology*, *72*, 223–261.

Onton, J., Westerfield, M., Townsend, J., & Makeig, S. (2006). Imaging human EEG dynamics using independent component analysis. *Neuroscience and Biobehavioral Reviews*, *30*, 808–822.

Oppenheimer, S. M. (1992). Cardiovascular effects of human insular cortex stimulation. *Neurology*, *42*, 1727–1732.

Petruzzello, S. J., Hall, E. E., & Ekkekakis, P. (2001). Regional brain activation as a biological marker of affective responsivity to acute exercise: Influence of fitness. *Psychophysiology*, *38*, 99–106.

Petruzzello, S. J., & Tate, A. K. (1997). Brain activation, affect, and aerobic exercise: An examination of both state-independent and state-dependent relationships. *Psychophysiology*, *34*, 527–533.

Pfaff, D. (2006). *Brain arousal and information theory: Neural and genetic mechanisms*. Cambridge, MA: Harvard University Press.

Pfurtscheller, G. (1992). Event-related synchronization (ERS): An electrophysiological correlate of cortical areas at rest. *Electroencephalography and Clinical Neurophysiology*, *83*, 62–69.

Pineda, A., & Adkisson, M. A. (1961). Electroencephalographic studies in physical fatigue. *Texas Reports on Biology and Medicine*, *19*, 332–342.

Pivik, R. T., Broughton, R. J., Coppola, R., Davidson, R. J., Fox, N., & Nuwer, M. R. (1993). Guidelines for the recording and quantitative analysis of electroencephalographic activity in research contexts. *Psychophysiology*, *30*, 547–558.

Pizzagalli, D. A. (2007). Electroencephalography and high-density electrophysiological source localization. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds). *Handbook of psychophysiology* (3rd ed, pp. 56–84). Cambridge, UK: Cambridge University Press.

Pizzagalli, D. A., Oakes, T. R., & Davidson, R. J. (2003). Coupling of theta activity and glucose metabolism in the human rostral anterior cingulate cortex: An EEG/PET study of normal and depressed subjects. *Psychophysiology*, *40*, 939–949.

Pronk, N. P., Crouse, S. F., & Rohack, J. J. (1995). Maximal exercise and acute mood response in women. *Physiology and Behavior*, *57*, 1–4.

Puetz, T. W. (2006). Physical activity and feelings of energy and fatigue: Epidemiological evidence. *Sports Medicine*, *36*, 767–780.

Puetz, T. W., Beasman, K. M., & O'Connor, P. J. (2006). The effect of cardiac rehabilitation exercise programs on feelings of energy and fatigue: A meta-analysis of research from 1945 to 2005. *European Journal of Cardiovascular Prevention & Rehabilitation*, *13*, 886–893.

Puetz, T. W., Flowers, S. S., & O'Connor, P. J. (2008). A randomized controlled trial of the effect of aerobic exercise training on feelings of energy and fatigue in sedentary young adults with persistent fatigue. *Psychotherapy and Psychosomatics*, *77*, 167–174.

Puetz, T. W., O'Connor, P. J., & Dishman, R. K. (2006). Effects of chronic exercise on feelings of energy and fatigue: A quantitative synthesis. *Psychological Bulletin*, *132*, 866–876.

Raymond, J., Varney, C., Parkinson, L. A., & Gruzelier, J. H. (2005). The effects of alpha/theta neurofeedback on personality and mood. *Cognitive Brain Research*, *23*, 287–292.

Retey, J. V., Adam, M., Gottselig, J. M., Khatami, R., Durr, R., Achermann, P., et al. (2006). Adenosinergic mechanisms contribute to individual differences in sleep deprivation-induced changes in neurobehavioral function and brain rhythmic activity. *Journal of Neuroscience*, *26*, 10472–10479.

Retey, J. V., Adam, M., Khatami, R., Luhmann, U. F. O., Jung, H. H., Berger, W., et al. (2007). A genetic variation in the adenosine a2a receptor gene (adora2a) contributes to individual sensitivity to caffeine effects on sleep. *Clinical Pharmacology and Therapeutics*, *81*, 692–698.

Ritter, P., Moosmann, M., & Villringer, A. (2009). Rolandic alpha and beta EEG rhythms' strengths are inversely related to fmri-bold signal in primary somatosensory and motor cortex. *Human Brain Mapping*, *30*, 1168–1187.

Schneider, S., Askew, C. D., Diehl, J., Mierau, A., Kleinert, J., Abel, T., et al. (2009). EEG activity and mood in health orientated runners after different exercise intensities. *Physiology & Behavior*, *96*, 709–716.

Schürmann, M., & Basar, E. (1994). Topography of alpha and theta oscillatory responses upon auditory and visual stimuli in humans. *Biological Cybernetics*, *72*, 161–174.

Smit, A. S., Eling, P. A., Hopman, M. T., & Coenen, A. M. (2005). Mental and physical effort affect vigilance differently. *International Journal of Psychophysiology*, *57*, 211–217.

Spyer, K. M. (1989). Neural mechanisms involved in cardiovascular control during affective behaviour. *Trends in Neuroscience*, *12*, 506–513.

Srinivasan, R., Nunez, P. L., Tucker, D. M., Silberstein, R. B., & Cadusch, P. J. (1996). Spatial sampling and filtering of EEG with spline Laplacians to estimate cortical potentials. *Brain Topography*, *8*, 355–366.

Steptoe, A., & Cox, S. (1988). Acute effects of aerobic exercise on mood. *Health Psychology*, *7*, 329–340.

Steriade, M., Gloor, P., Llinás, R. R., Lopes de Silva, F. H., & Mesulam, M. M. (1990). Report of IFCN Committee on Basic Mechanisms. Basic mechanisms of cerebral rhythmic activities. *Electroencephalography and Clinical Neurophysiology*, *76*, 481–508.

Stock, C., Baum, M., Rosskopf, P., Schober, F., Weiss, M., & Liesen, H. (1996). Electroencephalogram activity, catecholamines, and lymphocyte subpopulations after resistance exercise and during regeneration. *European Journal of Applied Physiology*, *72*, 235–241.

Storer, T. W., Davis, J. A., & Caiozzo, V. J. (1990). Accurate prediction of VO_{2max} in cycle ergometry. *Medicine and Science in Sports and Exercise*, *22*, 704–712.

Tenke, C. E., & Kayser, J. (2001). A convenient method for detecting electrolyte bridges in multichannel electroencephalogram and event-related potential recordings. *Clinical Neurophysiology*, *112*, 545–550.

Thayer, R. E. (1987). Energy, tiredness, and tension effects of a sugar snack versus moderate exercise. *Journal of Personality and Social Psychology*, *52*, 119–125.

Thayer, R. E., Newman, J. R., & McClain, T. M. (1994). Self-regulation of mood: Strategies for changing a bad mood, raising energy, and reducing tension. *Journal of Personality and Social Psychology*, *67*, 910–925.

Ware, J. E. (1993). *Sf-36 health survey: Manual and interpretation guide*. Boston, MA: The Health Institute, New England Medical Center.

Wessely, S., Hotopf, M., & Sharpe, M. (1998). *Chronic fatigue and its syndromes*. Oxford: Oxford University Press.

Williamson, J. W., McColl, R., Mathews, D., Ginsburg, M., & Mitchell, J. H. (1999). Activation of the insular cortex is affected by the intensity of exercise. *Journal of Applied Physiology*, *87*, 1213–1219.

Woo, M., Kim, S., Kim, J., Petruzzello, S. J., & Hatfield, B. D. (2009). Examining the exercise-affect dose–response relationship: Does duration influence frontal EEG asymmetry? *International Journal of Psychophysiology*, *72*, 166–172.

Youngstedt, S. D., Dishman, R. K., Cureton, K. J., & Peacock, L. J. (1993). Does body temperature mediate anxiolytic effects of acute exercise? *Journal of Applied Physiology*, *74*, 825–831.

Yu, Y. H., & Blessing, W. W. (2000). Carotid and cardiopulmonary chemoreceptor activity increases hippocampal theta rhythm in conscious rabbits. *American Journal of Physiology*, *278*, R973–R979.

Zimmerman, M., & Mattia, J. I. (2001a). The psychiatric diagnostic screening questionnaire: Development, reliability and validity. *Comprehensive Psychiatry*, *42*, 175–189.

Zimmerman, M., & Mattia, J. I. (2001b). A self-report scale to help make psychiatric diagnoses the psychiatric diagnostic screening questionnaire. *Archives of General Psychiatry*, *58*, 787–794.

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