

Short communication

P300 and long-term physical exercise

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Abstract

Electrophysiologic effects of physical exercise were investigated by comparing groups of individuals who engage in relatively low amounts of physical exercise (<5 h/week) to subjects who engage in relatively high amounts of aerobic exercise (>5 h/week). Event-related brain potentials (ERPs) were recorded using auditory and visual stimuli in separate oddball task conditions. P300 amplitude was affected by exercise frequency, such that increased amounts of exercise were associated with increased amplitude and somewhat more so for visual stimuli. No reliable exercise effects for P300 latency were observed, with little effect found for the other components. The findings suggest that a history of intensive physical exercise affects P300 amplitude. Theoretical mechanisms are discussed. © 1997 Elsevier Science Ireland Ltd.

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1. Introduction

Several reports have suggested that frequent physical exercise may have facilitating effects on general cognitive function (Bashore and Goddard, 1993; Tomporowski and Ellis, 1986) and affective state (Boutcher and Landers, 1988; Petruzzello and Landers, 1994; Youngstedt et al., 1993). For example, age-related differences in performance on some cognitive tasks are attenuated in subjects with high-compared to low-physical exercise (Dustman et al., 1984; Spirduso, 1980; Baylor and Spirduso, 1988), although strong effects are not always obtained (Blumenthal and Madden, 1988; Dustman et al., 1994). Despite the implication of these findings that exercise can contribute to intellectual performance and that such activities affect cognitive capability, the basis for these effects is not well understood (Dustman et al., 1994; Hatfield and Landers, 1987).

When such findings are considered in the context of the association between background electroencephalographic

(EEG) activity and event-related potentials (ERPs), it is not unreasonable to suppose that rigorous physical exercise could affect EEG activity and, therefore, cognitive ERPs (Basar and Stampfer, 1985; Basar et al., 1984; Pritchard et al., 1985). Indeed, a generally positive relationship of exercise on ERP and neuropsychological measures of mental processing speed has been reported (Dustman et al., 1990a; Geisler and Squires, 1992). However, because inter-subject variability of background EEG alpha-activity is related to individual variation in the P300 component (Jasiukaitis and Hakerem, 1988; Mecklinger et al., 1992; Intriligator and Polich, 1994, 1995; Polich and Luckritz, 1995; Polich, 1997), changes in background EEG from aerobic exercise may directly affect ERP variation.

Some evidence for this hypothesis has been obtained: assessment of subjects before and after exercise appears to produce an increase in alpha power across studies even though widely different electrophysiological methodologies were employed in these reports (Boutcher and Landers, 1988; Farmer et al., 1978; Hatfield et al., 1984; Kamp and Troost, 1978; Wiese et al., 1983; Hatfield and Landers, 1987). Dustman et al. (1990b) assessed the interaction between exercise and age by comparing groups of healthy

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young and older men such that half of the subjects in each group were in good aerobic condition, and half were in poor aerobic condition. Relative to low-fit men, high-fit subjects had better neurocognitive functioning and significantly greater amounts of alpha activity (8–10 Hz) regardless of age. These findings imply that exercise contributes to CNS function and superior cognitive performance (Dustman et al., 1994; Dustman et al., 1993) and are in accord with a previous study from the same laboratory (Dustman et al., 1985). An additional and particularly relevant report by Bashore (1989) found that response time and P300 latency from a complex visual information processing task were appreciably shorter in aerobically fit young men compared to a control non-exercise group. Analogous outcomes were obtained for elderly fit vs. non-fit men with increased P300 amplitudes also observed, suggesting that a history of exercise can contribute to the maintenance of mental capability with aging. These results are important in that they imply directly that exercise can affect P300 and related measures and, therefore, cognitive function.

Taken together, these findings suggest that long-term physical exercise may influence EEG activity and, perhaps, cognitive function. However, whether a history of physical exercise does affect ERP measures in this context has not been examined systematically. The present study was designed to assess this issue by comparing young adult exercisers who have engaged in high levels of physical exercise to control subjects who perform comparatively little exercise. Subjects were measured in a rested state that occurred during the course of their normal weekly schedule so that baseline ERP activity could be obtained, with both component amplitude and latency measures obtained. If exercise does affect the P300 ERP, low- and high-exercise groups should demonstrate different electrophysiological patterns. Although EEG data also were collected for some of these subjects, they are reported elsewhere because of differences in design and samples (Lardon and Polich, 1996).

2. Methods

2.1. Subjects

Individuals from the low and high exercise group were matched on age (34.7 vs. 30.0 years), educational level (16.3 vs. 14.7 years), and sex (8 male, 3 female in each group) to minimize general neurocognitive variation (Polich and Kok, 1995). However, the subject groups differed systematically in the average time spent engaged in vigorous physical exercise taken over the previous 3-year period (3.1 vs. 18.6 h/week): a factor that produced significant differences in resting heart rate as assessed manually at the wrist (67.6 vs. 53.8 beats/min, $P < 0.01$). Hence, the present comparison can be considered as evaluating the low and high ends of the exercise continuum, rather than 'none'

vs. 'high'. All subjects reported being free of neurological or psychiatric disorders, and provided informed written consent.

The 'low' exercise subject group (<5 h/week) was defined by the absence of any previous participation in high level sports activity and relatively minimal aerobic activities. It should be noted expressly that the low-exercise subjects engaged in some aerobic exercise each week, although much less than the high-exercise subject group. This was deemed desirable to preclude the possibility that any obtained effects could be attributed to just the difference between engagement in exercise and no exercise. The 'high' exercise subjects (>5 h/week) were recruited through personal contact and advertisements in local fitness centers. This group was defined by their life-long commitment to athletic endeavors via their participation in physically demanding competitive events (e.g., triathlon, bike racing, tennis, etc.), excelling at high school or college level sports (e.g., playing at the varsity, basketball, baseball, etc., level for 3 or more years), or having at least a 3-year history of performing vigorous, aerobic physical exercise (i.e., individuals who provided strong evidence of their personal commitment to aerobic physical exercise). Both groups were assessed during their typical weekly schedules, which included periods of exercise.

2.2. Recording conditions and procedure

Electroencephalographic (EEG) activity was recorded at the Fz, Cz, and Pz electrode sites, referred to linked earlobes, with a forehead ground and impedance at 10 k Ω or less. Additional electrodes were placed at the outer canthus and supraorbitally to the left eye to record electro-ocular activity. The filter bandpass was 0.016–30 Hz (3 dB down, 12 dB octave/slope). The EEG was digitized at 256 Hz for 768 ms with a 75-ms prestimulus baseline. Waveforms were averaged off-line, with trials on which the EEG or EOG exceeded +100 pV rejected, and all conditions recorded with eyes open.

ERPs were elicited in separate conditions. Auditory ERPs were obtained with binaural 1000 (standard) and 2000 (target) Hz tones presented at 60 dB SPL (9.9 r/f, 50-ms plateau). Visual ERPs were obtained with 2.5 cm wide black and white striped (standard) and 2.5 cm square black and white checked (target) patterns, which were viewed a distance of 70 cm to produce a visual angle of $\approx 2^\circ$ width, and an average intensity of 35 cd/m². The stimuli were presented in a random series once every 3 s. with a target stimulus probability of 0.20. All ERPs were recorded while the subjects eyes were open and fixated on a central dot placed on the visual stimulus screen. The subject indicated the occurrence of a target stimulus by pressing a button with the thumb of their preferred hand, and error rates were recorded. One ERP trial block consisting of at least 30 artifact-free target stimuli presentations was obtained for each stimulus modality.

3. Results

Greenhouse-Geisser corrections were applied to analyses of variance with repeated measures factors. Newman-Keuls comparisons were employed for post-hoc tests. Waveforms from each electrode and stimulus condition were analyzed with the P300 component defined as the largest positive going peak occurring for all electrode sites after the N100–P200–N200 complex, within a latency window between 250 and 500 ms. Amplitude was measured relative to the prestimulus baseline, with peak latency defined as the time point of maximum positive amplitude. The amplitudes of the N100, P200, and N200 components also were obtained. Because the standard stimuli did not yield any reliable results related to the exercise variable (see below), only the data from the target stimuli are illustrated.

3.1. Behavioral performance

The mean percentage of task performance errors taken across subjects was 0.55%. A 2-factor (2 groups \times 2 modalities) analysis of variance was applied to the error rates

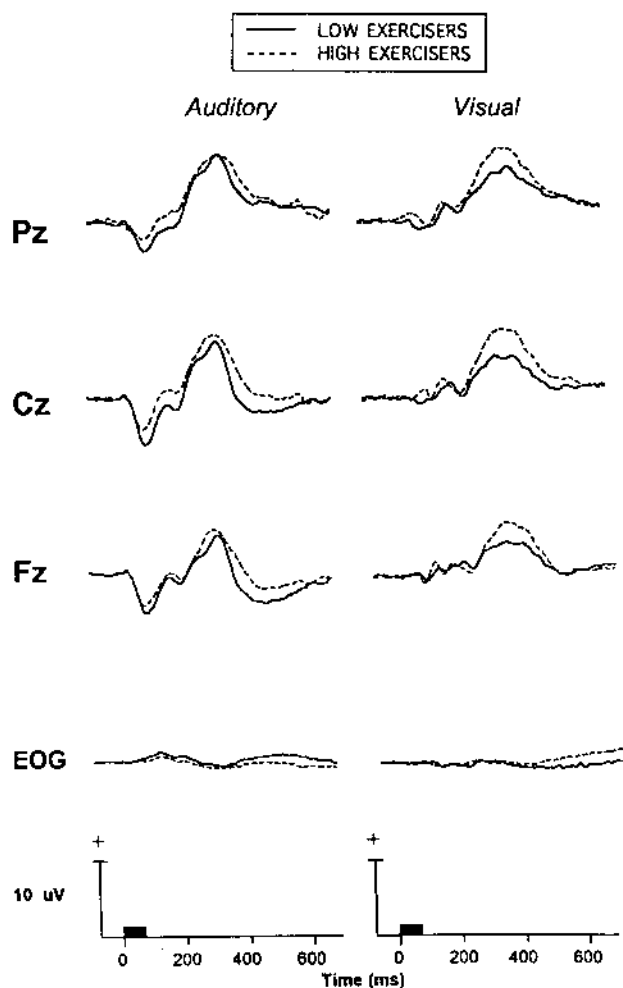


Fig. 1. Grand average ERPs for the low- and high- exercise subjects from the auditory and visual stimulus conditions ($n = 11/\text{group}$).

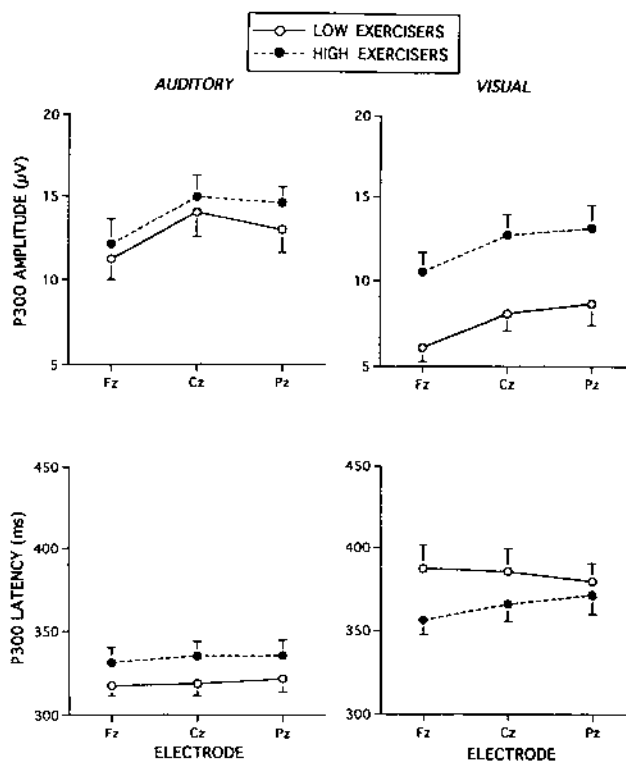


Fig. 2. Mean P300 amplitude (top) and latency (bottom) from the low and high exercise subject groups for the auditory and visual stimulus conditions.

from each subject and yielded no significant effects, although somewhat more errors were made for auditory compared to visual stimuli (0.91% vs. 0.18%). Thus, performance was excellent and did not differ across groups or experimental conditions.

3.2. P300 component

The grand average of the original ERPs for the target stimuli from each subject, modality, and recording site for each exercise group are presented in Fig. 1. The mean P300 amplitude and latency data from the target stimuli of each modality as a function of electrode site and for each group of subjects are illustrated in Fig. 2. The origins of the relatively small ERPs from the visual stimuli are unclear, but may be related to the moderate stimulus intensities employed (Covington and Polich, 1996; Polich et al., 1996). P300 measures were assessed with a 3-factor (2 groups \times 2 modalities \times 3 electrodes) analysis of variance applied to the component values from each subject in each condition.

P300 amplitude was smaller for the low- compared to high-exercise group, $F(1,20) = 4.5$, $P < 0.05$; larger for the auditory compared to visual stimuli conditions, $F(1,20) = 13.3$, $P < 0.01$; and increased from the frontal to parietal electrodes sites, $F(2,40) = 16.6$, $P < 0.001$. P300 latency was shorter for the auditory compared to visual stimuli conditions, $F(1,20) = 54.9$, $P < 0.001$. The exercise group and modality factors yielded a significant

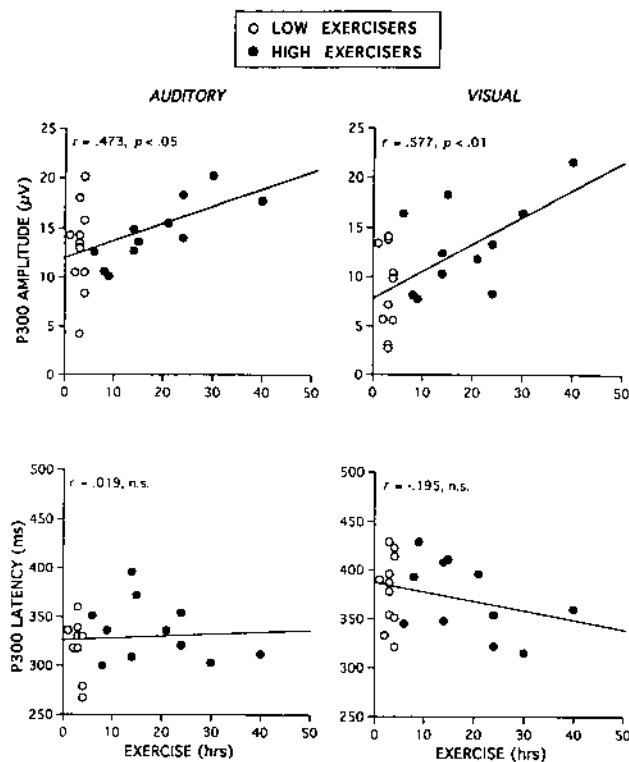


Fig. 3. Scattergrams and correlation coefficients of P300 amplitude (top) and latency (bottom) from the Pz electrode site with amount of exercise performed each week by each subject. The open circles (left side of each figure) represent the low-exercise (<5 h/week), and the closed circles represent the high-exercise (>5 h/week) subjects.

interaction, such that the peak latency from the low-exercise group increased more between the auditory and visual conditions than did the peak latency from the high-exercise group, $F(1,20) = 7.1$, $P < 0.02$. A significant interaction between the exercise group and electrode factors also was obtained, such that peak latency from the frontal to parietal electrode positions for the low-exercise group did not change, whereas peak latency for the high-exercise group increased, $F(2,40) = 3.9$, $P < 0.05$. A similar analysis applied to the P300 data from the standard stimuli revealed no effects for the exercise group factor.

Examination of ERP data from individual subjects of both exercise groups revealed substantial inter-subject variability within and between groups. To assess individual variation of exercise effects systematically, correlational analyses of the P300 from each modality condition with respect to the exercise variable were performed. These findings are summarized in the scattergrams for the relationship between P300 measures (Pz) and exercise amount presented in Fig. 3. In general, (1) P300 amplitude tended to be smaller for low- compared to high-exercise subjects, (2) considerable individual variation is present for both exercise groups, and (3) component size is significantly and positively correlated with exercise amount for both modalities. P300 latency does not appear to be strongly affected by exercise in general, with only a slight tendency to decrease

with increased amounts of exercise observed for the visual stimulus conditions.

3.3. N100, P200, N200 components

The other ERP component measures also were assessed with a 3-factor (2 groups \times 2 modalities \times 3 electrodes) analysis of variance applied to the data from each subject in each condition. For the target stimulus data, no significant effects involving the exercise group factor were obtained for any of the amplitude or latency data from these components. For the standard stimulus data, no amplitude effects involving the exercise group variable were found. However, the low-exercise subjects produced somewhat longer peak latencies than the high-exercise subjects for the N100 ($P < 0.10$), P200 ($P < 0.03$), and N200 ($P < 0.10$) components. Because of the absence of consistent or strong systematic exercise group differences for these components, they will not be considered further.

4. Discussion

Young adult low-exercise subjects demonstrated smaller P300 amplitudes than high-exercise subjects, although considerable inter-subject ERP variation for exercise effects was observed. P300 latency was not different between the exercise groups, but low-exercise subjects evinced larger intermodality peak latency effects than did high-exercise subjects. Generally little difference between exercise groups was observed for the other ERP components. Taken together, the present results suggest that exceptional amounts of physical exercise can alter the P300 ERP component from simple auditory and visual stimuli, but that these effects are variable across subjects and most evident only with very high amounts of weekly aerobic activity.

5. Theoretical considerations

Given the links between background EEG and cognitive ERPs outlined above (Basar and Stampfer, 1985; Basar et al., 1984; Jasiukaitis and Hakerem, 1988; Intriligator and Polich, 1994, 1995; Mecklinger et al., 1992; Polich, 1997; Polich and Luckritz, 1995; Pritchard et al., 1985), it seems likely that the effects of exercise observed for the present ERP data might originate from fundamental changes in baseline EEG that are produced by aerobic activity (Dustman et al., 1985, 1990a,b; Bashore, 1989; Bashore and Goddard, 1993; Geisler and Squires, 1992; Lardon and Polich, 1997). In this view, extended exercise helps to contribute to increased amounts of alpha-band activity and, therefore, increased P300 amplitude and decreased peak latency (Intriligator and Polich, 1994, 1995; Jasiukaitis and Hakerem, 1988). If this hypothesis is accurate, the present findings are in agreement with and extend previous reports that

found increased alpha power for high-fit relative to low-fit subjects (Dustman et al., 1985, 1990b; Lardon and Polich, 1996) and studies that assessed EEG activity before and after exercise in a variety of subject sport-populations (Boutcher and Landers, 1988; Farmer et al., 1978; Kamp and Troost, 1978; Hatfield et al., 1984; Wiese et al., 1983; Petruzzello and Landers, 1994). Furthermore, generally similar results for P300 latency from a complex visual stimulus task in a comparable young subject group have been reported by Bashore (1989), although the present study also obtained increased component amplitudes for the exercise relative to control subjects. Thus, it is reasonable to conclude that exercise does affect EEG and P300 values, but that the amount of exercise as well as the electrophysiological measurement parameters employed are important factors to consider when these variables are evaluated.

The underlying causes for the influence of physical exercise on the P300 ERP are far from clear, although speculation on the sources of these effects can be made. For example, it is straightforward to assume that physical exercise promotes cerebral blood flow (CBF) that could affect EEG measures (Dustman et al., 1990a,b; Geisler and Squires, 1992), but why such physiologic changes would affect specific EEG bands is uncertain (Bashore and Goddard, 1993; Dustman et al., 1993; Lardon and Polich, 1996). However, when a decrease in CBF occurs because of anoxia or hypoxia, an increase in delta and decrease in alpha and beta activity typically are observed (Chatrian, 1990; Keilaway, 1990; Kraaier et al., 1992; Niedermeyer, 1993). If physical exercise promotes increased CBF, an EEG spectral pattern opposite that of poor CBF might be obtained. Given that such EEG changes contribute to P300 components measures, the present study's findings can be viewed as suggestive support for the hypothesis that increased circulatory capacity contributes to ERP changes.

6. Conclusions

In an elegantly comprehensive review, Dustman et al. (1994) note that the findings from animal studies 'strongly suggest there is a positive relationship between physical exercise and CNS health' (p. 169), which occurs, at least in part, because of improved neurotransmitter functioning, preservation of dopaminergic cells, increased vascularization, and increased cell hypertrophy and complexity. These findings imply that if 'similar kinds of neurobiological changes occur in humans who frequently engage in aerobic exercise...these should outperform infrequent exercisers when compared on a variety of tasks that reflect CNS integrity' (p. 170). However, despite these findings in animal subjects, the ERP evidence for humans is not as convincing, perhaps because biological influences stronger than those resulting from exercise may be operative (for a review, see Polich and Kok, 1995). Even though the present study took pains to match the low- and high-exercise subjects on

such variables, the overall ERP differences between subject groups were variable (Fig. 3). Thus, additional effort is needed to determine if and how exercise, per se, contributes to appreciable changes in CNS function and how such changes affect the P300 component of the ERP.

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