Short communication

Effect of long-term swimming exercise on somatosensory evoked potentials in rats

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Abstract

The study investigated whether long-term swimming exercise prevents age-related changes in rat somatosensory evoked potentials (SEPs) and somatosensory cortex (SC) morphology. A total of 25 9-month-old rats were assigned to an exercise or control group. The exercise group swam 1 h/day five times weekly for 1 year. The results showed that long-term exercise prevented age-related changes in SEPs and SC morphology.

Most biological functions deteriorate with age. Even though life expectancy appears to be universal and genetically determined in all species, it can be modified by external factors such as stress, nutrition, environmental conditions and physical activity [2,10]. In recent years, research has focused on the identification of contributing factors that potentially alter the rate and extent of degenerative change in aging. Findings have suggested that regular exercise may reduce age-related degenerative changes in neurological parameters such as cognitive function, electroencephalographic (EEG) activity, reaction time and event-related potentials (ERPs) [2–5]. The mechanisms by which physical exercise affect the age-related changes of CNS structure and function are undoubtedly complex and are not well understood.

The recording of somatosensory evoked potentials (SEPs) yields valuable information about CNS function, and studies have shown that reliable changes occur in SEPs as age advances [4,9]. On the structural side, light microscopy investigations have revealed approximate but reliable information about morphological changes in the rat cortex with aging [13,14,16].

To our knowledge, the effects of short- and long-term training on SEPs have not yet been studied in the rat. Our aim was to find out whether long-term physical training delays the onset of age-related changes in SEPs and somatosensory cortex (SC) morphology in this animal model. We also sought to determine whether short-term exercise produces electrophysiological effects.

The study was approved by the university’s Committee for the Use of Animals in Research and followed the guidelines established by the European Communities Council Directive. A total of 25 9-month-old male Wistar albino rats weighing 250–350 g were randomly assigned to either the control (n=13) or the exercise (n=12) group. Standard rat chow and tap water were provided ad libitum, the animals were housed at 23±2°C under a 12/12-h dark/light cycle, and both groups were handled equally often. The exercise group swam 1 h/day (always between 09:00 and 11:00 h) five times weekly over 1 year. The
swimming was done in two 100×50×50-cm tanks of tap water that were kept at 32–34°C.

SEP recordings were done at three time points in both groups, namely, before the exercise program started (baseline), at 3 months and at 12 months. In the exercise group, the recordings at 3 and 12 months were done 48 h after swimming. All animals were deprived of food for 24 h prior to SEP recordings. Ether anesthesia was used, and all recording data were collected within 15 min after the loss of the corneal reflex and pain response. Rectal temperatures were maintained between 35 and 37°C, by keeping the animals warm using a heating table when necessary.

Cortical SEPs were recorded from a needle electrode inserted subcutaneously over the hind limb projection area of the somatosensory cortex. The active electrode was placed 9 mm anterior and 2 mm lateral to the bregma [12]. A reference electrode was placed 20 mm anterior to the bregma at the midline, and the grounding electrode was attached to the animal’s tail [6]. SEPs were recorded using a Nihon Kohden (MEM-4104) Neuropack Four EMG/EP measuring system, with a filter set to bandwidth 20–3000 Hz. The contralateral posterior tibial nerve was stimulated in the swimming group (0.00318 mA, 0.2 ms, the repetition rate was 3 Hz, and we used an intensity sufficient to produce a definite twitch of the big toe (~2.4–5 mA). For each set of recordings we took the mean of 200 responses, and did this at least twice to ensure response reproducibility. Hind limb transcutaneous stimulation evoked electrical activity with prominent first-positive (P1), first-negative (N1) and second-positive (P2) components. A negative wave was expressed as an upward deflection. Peak latency and peak-to-peak amplitude were measured for each component in order to quantify the effects of exercise on neuronal responses to somatosensory stimulation in the aging brain.

After the final SEP recordings were completed, each rat was euthanized with 1 g/kg urethane injected intraperitoneally. The brain was immediately removed, fixed, dehydrated and embedded in paraffin. Serial sections (20-μm thick) were then cut in the coronal plane and stained with Cresyl Violet. The three anatomically and physiologically distinct regions of the rat primary SC (parietal 1 (Par 1), forelimb (FL) and hind limb (HL)) [20] were examined using a monitorised Zeiss-Axioplan light microscope. A total of five sections from each rat’s brain were quantitatively analyzed by counting the neurons and glial cells in the cortical layer (I–VI) in an unbiased counting frame (2500 μm²) under the ×40 objective [13,19]. The neuron-to-glia ratio (N : G) was calculated by dividing the total number of neurons by the total number of glial cells.

Statistical analysis was performed using the SPSS for Windows software package. The data were distributed normally and are presented as means±S.D. We used Student’s t-test to compare the two groups’ data, and the paired t-test to make comparisons within the groups. We also applied 2 (swimming and control groups) ×3 (data sets collected at three time points) repeated measures analysis of variance. P-values less than 0.05 were considered significant.

From baseline to 12 months, the groups registered similar mean body and heart weight gains, but the mean heart weight-to-body weight ratio was significantly higher in the swimming group (0.00318±0.00058) than in the control group (0.00274±0.00052) (P<0.05).

Table 1 lists all the measured latency and amplitude data for each group. In the recordings done at baseline and at 3 months, there were no significant differences in latency and amplitude findings between or within the groups, and the data for both time points were similar. However, at 12 months the control group’s N1 latency was significantly longer, and their N1-P1 and P1-N2 amplitudes significantly lower than the values recorded previously. These findings also differed significantly from the exercise group results at 12 months. Apart from this, the control group P1 and N2 latencies were similar to those in the first two data sets. In the exercise group, none of the SEP component measurements at 12 months differed statistically from previously recorded values.

The results of the histologic examination of the somatosensory cortex are summarized in Table 2. The N : G was significantly higher in the exercise group than in the

<table>
<thead>
<tr>
<th>Table 1</th>
<th>SEP latencies and amplitudes of control (n=13) and exercise (n=12) groups*</th>
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<tbody>
<tr>
<td></td>
<td>Control</td>
</tr>
<tr>
<td></td>
<td>Baseline 3 Months 12 Months</td>
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<tr>
<td>Latencies (ms)</td>
<td></td>
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<tr>
<td>N1</td>
<td>10.63±0.44 10.80±0.34 11.24±0.64$^4$</td>
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<tr>
<td>P1</td>
<td>12.81±0.75 12.74±0.54 13.31±0.91</td>
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<tr>
<td>N2</td>
<td>16.75±1.24 16.78±1.05 16.29±1.01</td>
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<tr>
<td>Amplitudes (μV)</td>
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<tr>
<td>N1-P1</td>
<td>1.17±0.49 1.19±0.37 0.63±0.16$^4$</td>
</tr>
<tr>
<td>P1-N2</td>
<td>1.25±0.42 1.42±0.30 0.64±0.26$^4$</td>
</tr>
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*Values are mean±S.D., significant difference within groups $^4$ P<0.05, $^{**}$ P<0.01, $^{***}$ P<0.001, significant difference between groups * P<0.05, ** P<0.01, *** P<0.001.
controls in all regions except layer I in Par 1, layer I–II in FL and layer III in HL.

Our main aim was to investigate whether long-term exercise delays the onset of age-related changes in SEPs and SC morphology in rats. In addition, we looked at the impact of short-term physical training on SEPs. Short-term exercise produced no changes in either group’s SEP recordings, but long-term physical training did prevent the age-related changes that have been documented in rat SEPs and SC morphology. Similar findings have been reported in human studies regarding short-term physical activity [1,11].

Brain bioelectric activity measurements (EEG, ERPs and visual, somatosensory and auditory evoked potentials (EPs)) provide valuable information about aging [4,9]. Research has proven that advancing age is accompanied by slowing of dominant brain rhythms on EEG, and by prolonged latency and decreased amplitude in ERPs and EPs [4,15]. These characteristic findings are considered to reflect a reduction in CNS inhibitory abilities, and may be linked to lower neuron and synapse density, loss of dendritic spines, impaired cerebral circulation, and changes in neurotransmitter synthesis and degradation [2].

As expected, at 12 months we noted significant age-related changes in the control rats’ SEPs. Control N1 latency was longer, and the N1-P1 and P1-N2 amplitudes were lower than previous values, while the rats that had exercised exhibited no statistically significant electrophysiological changes. However, delay in peak latency and decreased amplitude can occur with any change that affects the sensory pathway from the sensory receptors to the somatosensory cortex, but this was not the case in our study group. Nevertheless we did not find any significant changes in P1 and N2 latencies in our control group. These potentials may be affected in later stages of aging process.

Regarding SC tissue changes, light microscopic examination showed that the control group had a significantly lower mean N : G than the exercise group. These findings are in accordance with those of previous studies that investigated changes in neuronal and glial cell populations in the aging brain [13,14,16]. Exercise might play a preventive role in age related neuronal loss and glial density increase. The later could be due to the reduction of synaptic density with age.

The impact of physical exercise on age-related changes in CNS structure and function is not well understood, but several mechanisms have been proposed to explain this complex relationship. Animal and human studies have both proven that aerobic exercise is associated with permanent structural changes in the brain [7,17], and with elevated biogenic amine levels, enhanced oxygen transport and utilization [8,18]. These mechanisms are not mutually exclusive. Rather, these and other exercise-related processes likely work together to decelerate neuronal degeneration.

In line with other published evidence, our findings suggest that a sedentary lifestyle contributes to physiological and structural CNS deterioration during aging. It seems that regular exercise may effectively slow the rate of functional decline as the body ages. It is currently unclear at what age range exercise effects on neuronal structure and function are greatest. However longitudinal and cross-over designed studies are necessary to clarify the effect of life-long physical activity on age-related functional decline.

References


