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Original Research

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Effect of static and alternating magnetic fields on acetylcholinesterase and monoamine oxidase activities in the brain of mice

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ABSTRACT

Background: Exposure to magnetic fields (MFs) can affect the release of neurotransmitters, such as acetylcholine, serotonin and dopamine in the brain. **Objective:** The aim was to study some biochemical changes associated with exposure to low frequency static and alternating MFs at a flux density of 3 mT. **Animals and Methods:** Forty male mice were included in this study. Thirty mice were exposed to a combination of static and alternating MFs at a flux density of 3 mT for the duration of 1-3 weeks. Ten mice were taken as a control group. Activities of acetylcholine esterase (AChE) and monoamine oxidase (MAO) enzymes were determined in whole brain homogenates of these mice. **Results:** The activities of both enzymes were significantly increased in mice exposed to the MFs compared with the control group. These changes were more apparent after 3 weeks of exposure and were associated with some behavioral manifestations. No mortality or body weight changes were reported after MF exposure. In the present study, the enzymatic activities of AChE and MAO-A and -B were found to be significantly increased in brains of mice exposed to low frequency MFs. **Conclusion:** Exposure to MF causes changes in the activities of brain enzymes and these changes could affect the behavior of the animals exposed to MF.

KEY WORDS: Acetylcholinesterase, behavioral changes, brain enzymes, low frequency magnetic fields, monoamine oxidase

INTRODUCTION

In modern societies, electric power lines, electric appliances, and occupational technology are sources for different magnetic fields (MFs) including extremely low frequency (ELF) MFs [1]. Exposure to these MFs could have various clinical [1-4] and behavioral [5,6] health problems.

The effects of ELF-electromagnetic fields (EMFs) on biological systems such as different enzymes, nucleic acids, cell proliferation, etc., have been investigated [7,8]. It was suggested that, the interaction site for ELF-MFs is the cell membrane which could affect the movement of biological messages among cells [9] including synaptic chemical signal transmission operating with neurotransmitter (NT) substances [10]. It was believed that by interfering with cellular communication mechanisms, MFs can trigger changes in hormones, NTs, immune system as well as cancer promoter molecules at the cell surface [11].

NTs are molecules that transmit information from one nerve cell to another. They are considered as "behavioral codons" which means that we can ascribe the expression of specific behavior to the action of a specific chemical agent at the brain [12]. It was suggested that, the effect of EMFs on the brain was chemical rather than electrical and so EMF can affect the release of NTs such as acetylcholine (Ach), serotonin and dopamine in the brain [11]. Exposure to MF was found to alter both turnover and receptor reactivity of monoaminergic [13] and cholinergic [14] systems.

In addition, evidence from studies of MF exposure has revealed several biochemical changes in the nervous system [15]. These changes may involve alterations either in

neural activity or in the activity of enzymes that catabolize the nets; example of these enzymes are acetylcholine esterase (AChE) and monoamine oxidase (MAO)[16]. Recently, *in vitro* study, Fathi and Farahzadi [17] found that when the AChE enzyme exposed directly to EMF field, there was a dramatic decrease in its activity. They concluded that EMF application affects the AChE activity and these effects might be related to structural changes in the secondary and tertiary structures of the enzyme. In addition, the results of previous studies on the effect of EMF on MAO [18-20] and AChE [21-24] enzymatic activities in the brain of different animal species were controversial.

Thus, the aim of the present work was to study the changes in the enzymatic activities of AChE and MAO (A and B) enzymes in the brain of mice after exposure to combined low frequency static and alternative MFs. In addition, the behavioral changes of these mice were observed and recorded daily throughout the study.

ANIMALS AND METHODS

Animals

Forty male albino mice of 6 weeks age and of 25 ± 2 g each were housed in plastic cages and were maintained under a 12-h light/12-h dark cycle. They had *ad libitum* access to food and water. During their stay in the respective housing conditions, they were removed daily from their cages for cleaning the cages and renewing their food and water supply. After 2 weeks of habituation to the laboratory environment, the animals were divided randomly into two groups; the control group (Group I) of 10 mice subjected to the same conditions as the experimental group without exposure to MF, and the experimental group (Group II) of 30 mice which were exposed to both alternating and static MF (3 mT for 35 min/day) for 1 week (Group IIa, 10 mice), 2 weeks (Group IIb, 10 mice) and 3 weeks (Group IIc, 10 mice).

The Procedures were approved by the Ethical Committee of the Medical Research Institute.

MF chamber

The MF producing system was manufactured in Department of Biophysics, Medical Research Institute, Alexandria University. It consisted of two Helmholtz coils "A" and "B"; each has a diameter of 23.7 cm and 2100 turns with copper wire gauge No. 18 (diameter 1.024 mm) with a resistance of 21.7 ohms/1000 m. The two coils were separated by a distance of 11.5 cm in which the animals were placed. One coil was connected directly to alternating current (220 V/50 Hz) to produce the alternating MF. The other coil was connected to direct current to produce the static MF. The total magnetic flux density in the area where the animals were placed was 3 mT. The sham exposed mice were placed in the same placed but without currents in the coils. The cages were made of plastic (non-magnetic) and were isolated from coils to avoid vibration and heating.

Methods

a. Behavioral changes of mice were observed and recorded daily by one experimenter in both the control and experimental groups.
b. At the end of the exposure period of each group, the animals were weighed and then were sacrificed by cervical dislocation. The whole brain was rapidly removed from each mouse, dissected and washed with cold saline and then immediately homogenized in cold homogenizer. The whole brain homogenates in phosphate buffer (PB) were used for determination of enzymatic activities of AChE and MAO (A and B) enzymes. Protein concentrations in brain homogenates were measured using bovine serum albumin as a standard.
    1. The activity of AChE (EC 3.1.1.7) was determined in whole brain homogenate and calorimetrically, using Ach iodide as a substrate [25] based on the colorimetric reaction:
    Acetylthiocholine iodide → AChE *thiocholine + acetate*
    thiocholine + dithiobisnitrobenzoic acid → *5-thio-2-nitro-benzoat (yellow)*
    200 ul of whole brain homogenate (20 mg/ml) were added to 1.8 ml PB (0.1 M disodium phosphate, pH 8.0) in a cuvette to which 70 μl of 5, 5dithiobis-2-nitrobenzoate (0.01M, 3.96 mg/ml) were added as a chromogen. Then 13 μl of the substrate (Ach iodide; 075 M, 21.67 mg/ml) were added and changes in the absorbance (at 412 nm) were recorded for at least 6 min.
    2. The activities of MAO (EC 1.4.3.4) enzymes (A and B) were determined calorimetrically using serotonin (5-hydroxytryptamine [5-HT]) as a substrate for MAO-A and benzyl amine as a substrate for MAO-B.
        - Determination of MAO-A activity [26]: 1 ml of whole brain homogenate (50 mg/ml) were added to a glass stoppered centrifuge tube containing 1 ml of 5-HT (645.3 μM/L, 2.5 μg/ml) and 9 ml PB (0.2 M disodium phosphate, pH 7.2). A standard tube was prepared without the addition of the brain homogenate. After incubation in shaking water bath (37°C for 60 min), 1 ml of 0.1% 1-nitroso-2-naphthal (in 95% ethyl alcohol) and 1 ml of freshly prepared nitrous acid reagent (0.2 ml of 2.5% $NaNO_2$ in 5 ml of 2N H_2SO_4) were added. After incubation in a water bath (for 5 min at 55°C), 5 ml ethylene dichloride were added, the tubes were shaken and then centrifuged at low speed. The absorbance of the aqueous supernatant was measured at 540 nm in a spectrophotometer using PB (0.2 M, pH 7.2) as a blank.
        - Determination of MOA-B activity [27]: 600 μl of whole brain homogenate (50 mg/ml) were added to a glass tube containing 750 μl PB (0.2 M, pH 7.2) and 150 μl of 0.008 M (1.2 μmol/ml) benzyl amine in the same buffer. A control tube was prepared similarly, but the substrate (benzyl amine) was added after the incubation. After incubation in a water bath (37°C for 3 h.) 1.5 ml of 60% perchloric acid and 1.5 ml of cyclohexane were added to test and control tubes.

After stirring with glass rods at room temperature for 15 min, the tubes were centrifuged for 10 min at 2000 rpm. The absorbance of the cyclohexane extract for the test tube against that of the control tube was measured at 250 nm with a 10 mm light path.

Statistical Analysis

The data were collected and analyzed using SPSS 11.5 software package (SPSS Inc., Chicago Ill, USA). As the distribution of the data was not normal, groups were compared using non-parametric tests (Kruskal–Wallis ANOVA for comparison of multiple groups and Mann–Whitney U-test for comparison of two groups). The data are presented as mean ± standard error and a value of $P < 0.05$ was considered to be statistically significant.

RESULTS

In general, there were no significant body weight changes or deaths among the experimental and control groups during the period of the experiment.

1. Behavioral changes: Manifested as taming behaviors as well as decreased activities - were observed among mice of Group II. After 3 weeks of exposure to EMF, mice did not object to being stroked and handled, they can safely be left loose on the table. Reactions to handling by the experimenter were grossly reduced and they were accustomed to handling with less fear during loading and unloading to cages with sluggish movements.
2. Biochemical findings: The AChE activity (μmol/min/g protein) in whole brain homogenates of mice exposed to MF for 1 week (Group IIa) did not differ from that of the control group. After 2 weeks of exposure (Group IIb), the activity became non-significantly higher than that of the control group. The activity became markedly and significantly higher than that of the control group only after 3 weeks of exposure (Group IIc) [Table 1].

The MAO-A activities (μmol/min/g protein) in whole brain homogenates were significantly increased starting from the 1st week of exposure to MF. The increase became more marked and significant after 2 and 3 weeks (Groups IIb and IIc) of exposure to MF than that of the control group. The level after 3 weeks exposure (Group IIc) was similar to that after 2 weeks of exposure (Group IIb) [Table 2].

The behavior of the MAO-B activities (μmol/min/g protein) in whole brain homogenates was similar to that of MAO-A, where the activities were significantly higher in all groups exposed to MF compared with the control group, starting from the 1st week of exposure. On the other hand, the level in Group IIc decreased and was significantly lower than that in Group IIb, but still significantly higher than that of the control group [Table 3].

DISCUSSION

Effects of MFs on the mammalian nervous system have been reported to include alteration in NT/neurohormone turnover and release [28]. The results of the present study support these findings where exposure to combined MFs caused a significant

Table 1: Statistical analysis of AChE activity (μmol/min/g protein) in brain homogenates of mice exposed to combined magnetic field of 3 mT compared with the control group

	Group I (control group)	Group II (exposed groups)		
		Group IIa (1 week)	Group IIb (2 weeks)	Group IIc (3 weeks)
Number	10	10	10	10
Range	0.062-0.087	0.064-0.081	0.064-0.096	0.075-0.101
Mean±SD	0.073±0.008	0.073±0.006	0.079±0.011	0.087±0.009
F (P)		5.402* (0.004)		
P_1		0.954	0.162	0.001*
P_2			0.146	0.001*
P_3				0.051

$P<0.001$ was considered as significant, *F*: *F* test (ANOVA), P_1: *P* value of LSD test between Group I and other groups, P_2: *P* value of LSD test between Group II (a, b and c), P_3: *P* value of LSD test between Group II (b and c), LSD: Least significant difference, SD: Standard deviation, AChE: Acetylcholine esterase

Table 2: Statistical analysis of MAO-A activity (μmol/min/g protein) in brain homogenates of mice exposed to combined magnetic field of 3 mT compared with the control group

	Group I (control group)	Group II (exposed groups)		
		Group IIa (1 week)	Group IIb (2 weeks)	Group IIc (3 weeks)
Number	10	10	10	10
Range	0.16-0.19	0.21-0.29	0.43-0.59	0.43-0.56
Mean±SD	0.17±0.01	0.26±0.03	0.48±0.04	0.49±0.04
F (P)		219.986* (<0.001)		
P_1		<0.001*	<0.001*	<0.001*
P_2			<0.001*	<0.001*
P_3				0.632

$P<0.001$ was considered as significant, *F*: *F*-test (ANOVA), P_1: *P* value of LSD test between Group I and other groups, P_2: *P* value of LSD test between Group II (a, b and c), P_3: *P* value of LSD test between Group II (b and c), LSD: Least significant difference, SD: Standard deviation, MAO: Monoamine oxidase

Table 3: Statistical analysis of MAO-B activity (μmol/min/g protein) in brain homogenates of mice exposed to combine magnetic field of 3 mT compared with the control group

	Group I (control group)	Group II (exposed groups)		
		Group IIa (1 week)	Group IIb (2 weeks)	Group IIc (3 weeks)
Number	10	10	10	10
Range	0.21-0.38	0.38-0.56	1.11-1.33	0.68-0.92
Mean±SD	0.30±0.06	0.45±0.07	1.20±0.07	0.79±0.08
F (P)		333.305* (<0.001)		
P_1		<0.001*	<0.001*	<0.001*
P_2			<0.001*	<0.001*
P_3				<0.001*

$P<0.001$ was considered as significant, *F*: *F*-test (ANOVA), P_1: *P* value of LSD test between Group I and other groups, P_2: *P* value of LSD test between Group II (a, b and c), P_3: *P* value of LSD test between Group II (b and c), LSD: Least significant difference, SD: Standard deviation, MAO: Monoamine oxidase

increase of AChE and MAO-A and -B enzymatic activities; both enzymes are involved in turnover and regulation of NTs in the central nervous system and peripheral organs.

It has been suggested that, under the excitatory effects of the MFs and their induced potential on the plasma membrane, the release of the NTs could be stimulated to mediate the functional activities of the excited neurons [29]. Furthermore, it has been postulated that, the microelectrophoretic motion induced in the cell membranes by MFs may affect the membrane transport of cations such as calcium [30]. Thus, any exogenous agent that affects the flow of Ca^{2+}, either into or out of neurons, could potentially have a major impact on neural function [31].

It has been found that; exposure to MF increased the release of Ach NT from cholinergic vasodilator nerve endings [32]. In addition, Shalaby *et al*. [35] found that dopamine and serotonin activities were increased in brain homogenates of mice exposed to combined MFs.

In the present study, the enzymatic activities of AChE and MAO-A and -B were found to be significantly increased in brains of mice exposed to low frequency MFs. This increase was observed after 1 week and became more apparent after 3 weeks of exposure. In agreement with our results, Kaminski *et al*. [18] found that the activity of MOA in the brain and liver homogenates of guinea pigs was significantly increased after exposure to the electric field. Furthermore, Dolgacheva *et al*. [19] have reported an increase in the MAO activity (up to 174%) in rat brain after exposure to EMF. On the other hand, Lyshove group [20] observed a decrease in the activity of MAO in Wister rat brain cortex after irradiation.

Regarding AChE enzyme, Dimberg [21] has reported a similar finding in mice exposed to MF. The MF treatment resulted in increases in the brain cortical activities of AChE enzyme. On the other hand, Stegemann *et al*. [22] found a decrease in the AChE enzymatic activities in rat bone marrow cells after 2 h on which static MF was applied at 37°C. At 27°C, an increase in the enzymatic activity was detected after 3.5 h of the same static MF. These contradictory results between the present and previous studies could be due differences in the animal species, the organs studied, type and/or duration of exposure to MF.

Exposure to MF leads to increased release of NTs in the brain to mediate the functional activities of the neurons [22]. These NTs include Ach [32], dopamine and serotonin [33]. AChE enzyme is responsible for the regulation of the NT Ach concentration at cholinergic nerve synapses [34]. Furthermore, MAO enzymes are known to play a role in the regulation of NTs in mammalian central nervous system, including regulation of synaptic concentration of serotonin, dopamine, norepinephrine and other catecholaminergic NTs [35]. Accordingly, we can suggest that the increase in the enzymatic activity of AChE and MAO enzymes found in the present and previous studies [18,21,22] could be a compensatory mechanism i.e., increased levels of AChE and MAO-A and -B may not be a direct effect of MF, but it may be a feedback mechanism as a consequence of increased levels of their corresponding substrates (i.e. Ach and serotonin and dopamine) - to counteract the effects of increased release of NTs after exposure to MF.

These changes in the brain enzymatic activity could affect the behavior of the animals exposed to MF [6,36,37]. This was demonstrated in the present study as some behavioral changes - manifested as taming behaviors as well as decreased activities - observed among mice exposed to MF. Kanno *et al*., [38] demonstrated that acute repetitive trans-cranial magnetic stimulation (rTMS) can result in functional changes in the cortex with affection of the serotonergic and dopaminergic neuronal systems in rats, which may have therapeutic implications for emotional disorders. Recent studies have shown that TMS affects mood and improves symptoms in patients with major depression and other psychological disorders [6,39]; U.S. Food and Drug Administration has cleared a TMS system for therapeutic use [6].

CONCLUSION

This study showed that exposure to MF causes changes in the activities of brain enzymes and these changes could affect the behavior of the animals exposed to MF. These findings open the gate for further studies on the effect of exposure to MF on the release of different NTs and their regulating enzymes that could have clinical and therapeutic impacts.

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