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RESEARCH ARTICLE

Effects of sub-acute exposure to static magnetic field on iron status and hematopoiesis in rats

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ABSTRACT

The present work was undertaken in order to investigate the effects of static magnetic field (SMF) on hematopoiesis and iron metabolism in male rats. At thermoneutrality (25°C), the exposition of rats to SMF (128 mT, 1h/day for 5 consecutive days) induced an increase of hematocrit (+12%, $p<0.05$), hemoglobin (+6%, $p<0.05$) and mean corpuscular hemoglobin concentration (+9%, $p<0.05$). SMF exposure increased the plasma transferrin concentration (+25%, $p<0.05$) and the capacity of iron saturation in transferrin (+24%, $p<0.05$). However, the plasma iron level and the coefficient of transferrin saturation decreased (respectively 17% and 33%; $p<0.05$) in exposed rats. Our investigations suggested that SMF induced modifications in hematological and mineral parameters, indicating the development of hypoxia-like status associated with iron deficit in rats.

Key Words: Magnetic field, iron, hematopoiesis, rat

INTRODUCTION

In recent years, there has been a scientific debate regarding the effects of electromagnetic fields (EMF) on biological systems [1]. There is a growing concern about the increase in environmental pollution due to the emission of electromagnetic waves [2], although epidemiological studies have failed to find a correlation between magnetic fields (MF) at different intensities and the appearance of any particular pathology [3-4]. Events arising from exposure to static magnetic fields (SMF) may include alterations in cell membrane activity, and effects on nervous system and behavior of animals [5-6] and various enzyme systems [7]. Exposure to MF also caused an increase in locomotor activity, and a suppression of elaborated labyrinth behavior [8]. It may produce a variety of adverse effects such as headaches, sleep disturbances, modifications of electroencephalographic activity, as well as alterations in cognitive functions in both humans and animals [9-10]. MF increase the risk of various types of cancer, including leukemia, brain and breast tumors [11-14] and alter the function of reproduction and of the immune system [15]. Exposure to SMF increases the risk of Alzheimer's and Parkinson's diseases [16], and alters the blood-brain barrier by increasing its permeability to albumin [17], ions [18], metals and divalent elements [19]. However, it induces a weak change in the dimension and shape of the hemoglobin molecules and erythrocyte sedimentation rate (ESR) [20-26].

The present study performed an experimental approach to determine the effects of sub-acute exposure to SMF on hematological parameters and iron metabolism in rats.

MATERIALS and METHODS

Animals

Male Wistar rats (Pasteur Institute, Tunisia) weighing 100-150g at the time of experiments were housed at 25°C in a cage under a 12-12 h light/dark cycle, with free access to food and water. Treated rats (n=6) were exposed to SMF (128 mT; 1h/day for 5 consecutive days).

Exposure system

Lake Shore Electromagnets (Lake Shore Cryotronic, Inc, Westerville, Ohio, USA) are compact electromagnets suited for many applications such as magnetic resonance demonstrations. For the present experiment, we used an air gap of 15 cm. Water-cooled coils provide an excellent field for stability and uniformity when high power is required to achieve the maximum field capability for the electromagnet. An accurate pole alignment was achieved by precise construction of the air gap adjustment mechanism (Abdelmelek *et al.*, 2005) [6].

Blood sampling protocol

Blood samples were withdrawn via the Biotrol sampling catheter. Six blood samples (0.5 ml approximately/sample) were collected in vials containing EDTA for hematological investigations or heparin for biochemical studies.

Blood chemistry

Blood collected in heparinized chilled tubes was immediately centrifuged. Aliquots of plasma were frozen and stored at -80°C prior to biochemical analysis. Plasma iron was measured by using colorimetric methods according to manufacturer instructions (Biomaghreb, Réf.20061). Plasma transferrin was measured by using quantitative analysis of immunochemical reactions tanks to use nephelometer II of Bering (BN II), which offer an entirely automatic quantitative determination of proteins by means of nephelometry.

Hematological parameters were assayed by Medonic-precision instruments for hematology research (CA620).

The total capacity of metal saturation of transferrin (CTST) was reversely linked to store of iron: CTST (µmol/L) = transferrin (g/L). 25. The coefficient of the transferrin saturation of iron is an excellent index of iron transit and tissue supply: CST = plasma iron/CTST.

Data presentation and statistical analysis

Data are reported as the mean ± SEM. Differences between means were evaluated by one-way analysis of variance (ANOVA). Statistical significance of the differences between means was assessed by Student's t test. The level of significance was set at $p<0.05$.

RESULTS

As shown in Table 1, the exposure of rats to SMF (128 mT, 1h/day for 5 consecutive days) did not alter the number of red blood cells (RBC) ($7.52 \pm 0.15\ 10^6/mm^3$ vs $7.12 \pm 0.13\ 10^6/mm^3$, $p>0.05$) or white blood cells (WBC) ($11.41 \pm 0.41\ 10^3/mm^3$ vs $11.35 \pm 0.16\ 10^3/mm^3$, $p>0.05$). SMF decreased mean corpuscular hemoglobin

Table 1. Effect of sub-acute exposure to static magnetic field (128 mT, 1h/day, for 5 consecutive days) on blood parameters

	WBC ($10^3/mm^3$)	*Hb* (g/dl)	*RBC* ($10^6/mm^3$)	*Ht* (%)	*MCV* (μ3)	*MCHC* (G/dl)	*PLT* ($10^3/mm^3$)	*RDW* %
C	11.35 ± 0.16	12.16 ± 0.07	7.12 ± 0.13	34.1 ± 0.41	46.2 ± 0.79	37.7 ± 0.30	718 ± 24.10	20.85 ± 0.59
SMF	11.41 ± 0.41	12.92 ± 0.21*	7.52 ± 0.15	38.31 ± 0.95*	50.78 ± 0.34*	34.3 ± 0.48*	944 ± 49.89	6.78 ± 6.14*

WBC: White blood cells. **RBC:** Red blood cells. Hb: Hemoglobin. **Ht:** Hematocrit. **MCV:** Mean corpuscular volume. **MCHC:** Mean corpuscular hemoglobin concentration. **PLT:** Platelets. **RDW:** Red cell distribution width. **C:** Control. **SMF:** Static magnetic field. Values are means ± SEM. Calculated from n=6 in each group. *p<0.05, SMF vs C.

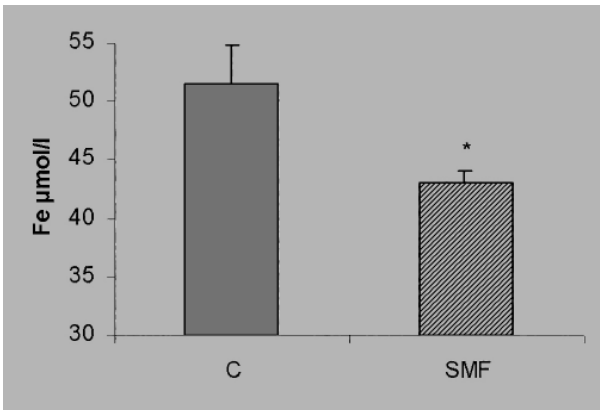

Figure 1. Effect of sub-acute exposure to static magnetic field (128 mT, 1h/day, for 5 consecutive days) on serum iron concentration. C: Control. SMF: Static magnetic field. Values are means ± SEM. Calculated from n=6 in each group. *p<0.05, SMF vs C.

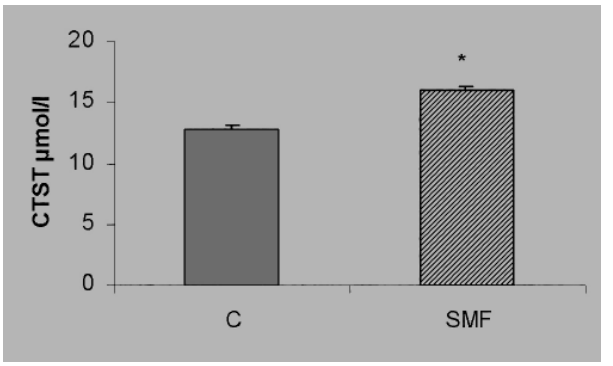

Figure 2. Effect of sub-acute exposure to static magnetic field (128 mT, 1h/day, for 5 consecutive days) on the coefficient of iron saturation of transferrin. C: Control. SMF: Static magnetic field. Values are means ± SEM. Calculated from n=6 in each group. *p<0.05, SMF vs C.

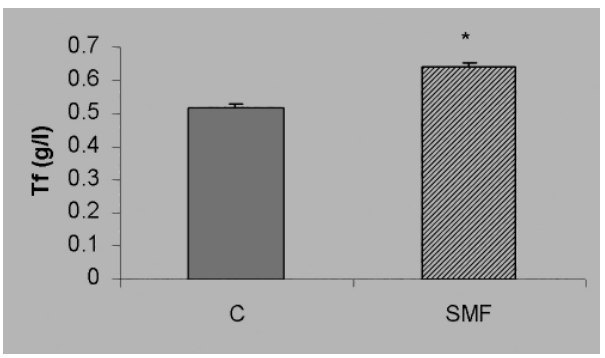

Figure 3. Effect of sub-acute exposure to static magnetic field (128 mT, 1h/day, for 5 consecutive days) on plasma transferrin level. C: Control. SMF: Static magnetic field. Values are means ± SEM. Calculated from n=6 in each group. *p<0.05, SMF vs C.

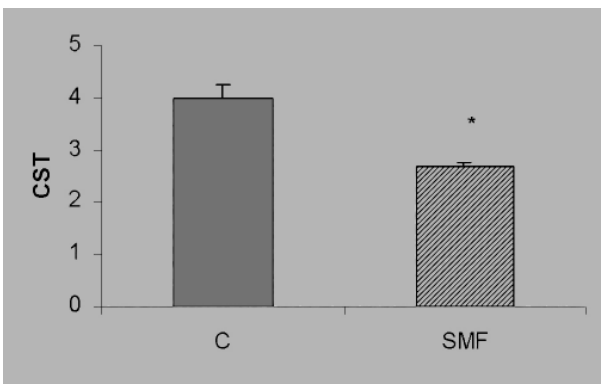

Figure 4. Effect of sub-acute exposure to static magnetic field (128 mT, 1h/day, for 5 consecutive days) on the total capacity of iron saturation of transferrin. C: Control. SMF: Static magnetic field. Values are means ± SEM. Calculated from n=6 in each group. *p<0.05, SMF vs C.

concentration (MCHC) (34.30 ± 0.48 g/dl vs 37.7 ± 0.30 g/dl, p<0.05) and red cells distribution width (RDW) (6.78 ± 6.14% vs 20.85 ± 0.59%, p<0.05). By contrast, SMF increased hematocrit (Ht) (38.31 ± 0.95% vs 34.10 ± 0.41%, p<0.05), hemoglobin (Hb) (12.92 ± 0.21 g/dl vs 12.16 ± 0.070 g/dl, p<0.05), and mean corpuscular volume (MCV) (50.78 ± 0.34 μ^3 vs 46.20 ± 0.79 μ^3, p<0.05). Exposure to SMF decreased the blood iron level (43.07 ± 0.91 µmol/L vs 51.48 ± 3.39 µmol/L, p<0.05) and the coefficient of the transferrin saturation of iron (CST) (2.69 ± 0.08 vs 4.00 ± 0.25, p<0.05) (Figures 1, 2). However, this treatment induced an elevation in plasma transferrin (0.64 ± 0.01 g/L vs 0.51 ± 0.01 g/L, p<0.05) and the total capacity of metal saturation of transferrin (CTST) (16.04 ± 0.35 µmol/L vs 12.87 ± 0.30 µmol/L, p<0.05), (Figures 3, 4).

DISCUSSION

In the present study, we report that SMF (128 mT, 1h/day for 5 consecutive days) produced a hypoxia-like status associated with iron deficit in the rat.

Our investigations showed that sub-acute exposure to SMF induced hemoglobin and hematocrit increase. This increase could be associated with the interaction between heme (iron) and SMF probably implicated in physiological defense. It is well known that hemoglobin is the blood protein for transport of oxygen within the human body. Oxygen is bound to the iron existing in the hemoglobin structure [21]. Hemoglobin is sensitive to MF; the morphological examination and the non-linear time course of the sedimentation in plasma indicated that MF increased cell aggregation and thereby enhanced ESR [22]. These data could be associated with a direct effect of MF of relatively high strengths leading to different hemoglobin conformations, accompanied by changes in intermolecular interactions without change in the intrinsic viscosity and shape [20]. In the present study, the increase in MCV and decrease in MCHC indicated the presence of hypochromic RBC in the forms of macrocysts. On the contrary, Mukewar and Baile [23] did not observe any modification in MCV of rats exposed to electric fields against a decrease in MCHC. SMF decreased the RDW; this parameter reflects the difference between the smallest and the biggest erythrocytes. Exposure to SMF can carry along agglutination of RBC due to an increase in MCV. Indeed, the increase in hemoglobin and hematocrit following sub-acute exposure to SMF may be explained by the installation of hypoxia-like status probably resulting from the oxygen binding impairment of hemoglobin or iron metabolism disruption.

Thus, interestingly, exposure to SMF decreased the serum iron level. This data is in accord with a previous study showing that exposure to electromagnetic field (40μT, 10 years) induced a decrease in blood iron in workers [24]. Similarly, Hachulla *et al.* [25] reported that iron was decreased in plasma of French population living near riverside high-voltage transmission lines. It is well documented that transferrin controlled transit of iron since intestinal enterocytes increase medullar erythroblasts and allowed recovery of iron after destruction of erythrocytes by macrophagic system [26]. The level of transferrin present in rats showed a reverse correlation with iron store. Production of iron increased when store decreased and this before appearance of anemia [26]. Previous data reported that ferritin was decreased in plasma of French population living near riverside high-voltage transmission lines [25]. However, the exposure of rats to SMF (128 mT, 1h/day for 5 consecutive days) increased the level of plasma transferrin. The amount of transferrin and ferritin allowed us to evaluate the CTST and CST that correspond respectively to total capacity of iron saturation in transferrin and coefficient of iron saturation in transferrin [26]. Indeed, exposure to SMF induced an increase in CTST, indicating a probable decrease of iron store [26]. By contrast, the decrease in CST is an excellent index of iron transit and tissue supply; it may reflect a decrease in iron delivery to erythropoiesis following EMF exposure [26]. The decrease in iron against the increase in transferrin may be explained by a decrease in the level of metal stock. This alteration can be related either to the leak of iron to other compartments or by the decrease in iron intestinal absorption. SMF exposure (67 mT 1h/day for 3 consecutive days) increased the calcium and iron content in frog sciatic nerve [19]. However, magnesium and copper levels remained constant. A previous study suggests that extremely low frequency (ELF) as electric fields can affect the nervous system and may have a specific effect on the risk of brain tumor. Change in blood-brain barrier, morphology, electrophysiology, neurotransmitter functions, cellular metabolism, calcium efflux and genetic effects have been reported in the brain of animals after exposure to ELF [27]. Moreover, the modification of peristaltic activity in the presence of several metal ions has been investigated *in vitro* in the rat intestinal muscle [28] by use of microscopic techniques, in order to determine the effect of aluminum, iron, chromium, and yttrium. Thus, divalent ions of these elements may operate differently on the mechanisms of intestinal absorption and contractions under SMF. Recently, Abdelmelek *et al.* [6] demonstrated that SMF induced sympathetic hyperactivity in rats; the high turnover of norepinephrine in the noradrenergic system could negatively influence the peristaltic activity implicated in the assimilation of divalent elements such as iron.

Our investigation demonstrated that sub-acute exposure to SMF (128 tm 1h/day for 5 consecutive days) induced a hypoxia-like status witch may be related to iron metabolism disruption. However, the mechanism of plasma iron deficit induced by SMF remains unknown.

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