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EFFECT OF PROLONGED EXPOSURE OF MICE TO GSM CELL PHONE RADIATION ON NEUROGENESIS IN THE HIPPOCAMPUS AND ON BLOOD LEVELS OF STRESS HORMONES AND VALIDATION OF THE EFFECTIVENESS OF A COMPENSATION OSCILLATOR. G. Alonso¹, B.J. Youbicier-Simo². ¹Laboratoire de Biologie des Neurones, UMR 5101 CNRS, CCIPE, 141 rue de la Cardonille 34094 Montpellier Cedex, France. ²Tecnolab Research Center, ZAC de la Thalie, Av. de l'Europe, 71100 Chalon sur Saone, France. * Tecno AO technology.

OBJECTIVE: To determine whether GSM cell phone radiation interferes with both the secretion of stress hormones and the proliferation of progenitor nerve cells in germinative areas of the adult brain, and to prevent induced effects if any.

BACKGROUND: Progenitor nerve cells actively proliferate within the brain of adult mammals, including man (Gould et al., 1999, PNAS, 96:5263-7), likely due to elevated levels of blood corticosterone (Cameron and McKay, 1999, Nat. Neurosci., 2:894-97). This active cell division mainly occurs in three germinative areas of the brain including the subventricular zone of the lateral ventricle (SVZ), the dentate gyrus of the hippocampus and white matter. Knowing that stress inhibits the proliferation of hippocampal progenitor cells (Lemaire et al., 2000, PNAS, 97:11032-7) and that electromagnetic fields interfere with cell proliferation (Velizarov et al., 1999, Bioelectrochem. Bioenerg., 48:177-80; Macias et al., 2000, Bioelectromagnetics, 21:272-86) and are stressful (Youbicier-Simo et al., 1997, Bioelectromagnetics, 18:514-23: Daniells et al., 1998, Mutat. Res., 399:55-64), we undertook a pilot study 1) to assess potential influence of GSM cell phone radiation on both the proliferation of progenitor nerve cells in brain germinative areas and the circulating levels of stress hormones (ACTH, corticosterone), and 2) to test the effectiveness of a compensation oscillator designed to prevent radiation-induced dysfunction. METHOD: The radiation source was a 900 MHz GSM cell phone (SAGEM, France) with 2 W maximum power output. It was placed horizontally with the battery downwards, 4 cm underneath the cage containing the mice. EMF intensity measured on the floor of the cage ranged from 6 to 18 V/m for microwaves and from 70 to 90 dBpT for extremely low frequency magnetic field. The study was initiated with three weekold C57BL mice held under under 22 + 2 °C, 12L-12D_{19h-07h}, with free access to feed and water. The irradiation schedule consisted in sending 3 min. phone calls continuously during the experimental session. From the age of 4 to 15 weeks, three groups of 15 mice each experienced different irradiation conditions: the control group was exposed to switched off cell phone; the exposed group was submitted to operating cell phone; the protected group was submitted to operating cell phone with the compensation oscillator installed. Five mice from each group were injected i.p. with the cell proliferation marker bromodeoxyuridine (BrdU) (3 x 5 mg in 0.2 ml 0.1 N NaOH). Ten mice remaining in each group were bled for the measurement of plasma levels of stress hormones i.e. adrenocorticotropic hormone (ACTH) and corticosterone. RESULTS: With respect to irradiation, quantitative estimation of cell proliferation indicated no inter-group difference in the numerical density of proliferating cells (BrdU-labeled) within the SVZ and white matter. On the other hand, a slight decrease (-25%) was observed in the hippocampal dentate gyrus of mice in the exposed group as opposed to controls, whilst mice in the protected group displayed normal nerve cell proliferation rate. ACTH and corticosterone levels were significantly decreased in the exposed group as compared with the controls (P < 0.05). On the other hand, mice in the protected group showed normal levels of ACTH, while corticosterone levels remained as low as in the exposed group.

CONCLUSION: Together, these data indicate that prolonged exposure of mice to GSM cell phone radiation moderately but significantly altered hippocampal neurogenesis and contemporaneously decreased blood levels of stress hormones. The compensation oscillator allowed restoration of disrupted biological parameters.

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