

Interference from GSM Cell Phones with the Production of Stress Hormones in Healthy and Lewis Lung Carcinoma-bearing Mice: Effectiveness of a Protective Device *

B.J. Youbicier-Simo, J.C. Lebecq, J. Giaimis, M. Bastide

Laboratoire d'Immunologie et Parasitologie (MENRT UA 2413), Faculté de Pharmacie, Université Montpellier-1, 34060 Montpellier cedex 2, France

The present study was aimed at determining possible influence of GSM cell phone radiation, either as a whole (MW + ELF fields) or with substantially mitigated MW power (i.e. mainly restricted to its ELF component) on the production of stress hormones in healthy, as well as tumor-bearing mice. We also tested the capacity of a protective device to counteract stressogenic effects induced by the cell phone radiation.

The source of electromagnetic (EM) stimulus was a commercial 900 MHz GSM digital cell phone (SAGEM, France) with 2W maximum power output. When necessary, grounded copper gauze (Soulas & Cie, Montreuil Sous-Bois, France), with sufficiently fine mesh diameter (350 μm) and permeable to ELF fields, was used to attenuate the power of MW involved in the cell phone EM spectrum. The magnitude of MW and ELF fields was measured and showed the highest power in close proximity to the cell phone case and aerial. The study was initiated with 4 week-old mice of the C57BL/6 j RJ strain (Janvier, Le Genest-St-Isle, France). From 4-5 weeks of age, the mice were housed under 22 ± 2 °C and 12 L- 12 D with lights on from 0800 to 2000. Irradiation lasted 15 weeks, from 5 to 20 weeks of age. Lewis lung carcinoma 1 (LLC1) cells were injected in the thigh muscle of the right hind leg at the beginning of the 12th week of irradiation (17 weeks of age) and the tumor was allowed to grow during 3 weeks (from 17 to 20 weeks of age). Six groups of mice were introduced in the study. The sham-irradiated and saline-treated control group was exposed under switched off cell phone and was inoculated with the saline. The tumor group was exposed under switched off cell phone and was inoculated with LLC1 cells. The cell phone + tumor group was exposed under operating cell phone, i.e. under whole EM spectrum and was inoculated with LLC1 cells. The cell phone + protective device + tumor group was exposed under operating cell phone equipped with the protective device and was inoculated with LLC1 cells. The cell phone + copper filter + tumor group was exposed to operating cell phone while being sheltered from MW by means of grounded copper filter and was inoculated with LLC1 cells. The cell phone + protective device copper + filter + tumor group was exposed to operating cell phone with the protective device installed while being sheltered from MW by means of grounded copper filter, and was inoculated with LLC1 cells. After 15 weeks of irradiation, blood samples were collected by retro-orbital puncture between 1000 h and 1200 h and plasma samples were stored at - 20 °C until assayed for adrenocorticotrophic hormone (ACTH) and corticosterone. Afterwards, the mice were sacrificed by anesthetic overdose and were immediately autopsied for the inspection of metastatic foci at the surface of the lungs.

In the second experiment, the protocol was exactly the same as in experiment 1, except that the schedule for blood sampling was slightly modified. Blood samples were collected after 2 and 5 weeks of irradiation, to check the influence of the cell phone radiation on the production of stress hormones (ACTH and corticosterone) in healthy mice before implantation of the tumor. Another sampling point was performed 3 weeks after tumor development, i.e. after 15 weeks of irradiation, to evaluate

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the impact of the cell phone radiation on ACTH and corticosterone levels in mice suffering from Lewis Lung carcinoma. Afterwards, the mice were sacrificed by anesthetic overdose and were immediately autopsied for the inspection of metastatic foci at the surface of the lungs.

Before tumor implantation, i.e. after 2 and 5 weeks of irradiation, the cell phone radiation, either as a whole or with depleted MW, induced significant decrements in plasma ACTH and corticosterone levels, relative to placebo. The protective device allowed partial restoration of corticosterone levels and total restoration of ACTH levels. In sham-irradiated mice receiving tumor cells, slightly reduced ACTH and significantly decreased corticosterone levels were observed. This tumor-induced depletion of stress hormones was potentiated by whole or MW-deprived cell phone radiation. The protective device induced the restoration of ACTH and corticosterone to levels comparable to those observed in the controls, while only partial restoration of corticosterone levels was observed. Irradiation had no impact on the number of lung metastases.

Taken together, these findings indicate that chronic exposure under GSM cell phone radiation, either as a whole or restricted mainly to its ELF component, was stressful for healthy mice and synergistic with cancer to deplete stress hormones. These stressful effects were partly or totally reversed by the protective device. The cell phone radiation did not affect the formation of lung metastases.