Exposure to Radiofrequency Fields and Heat Shock Protein (Hsp) Expression in the Rat Brain

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The expression of heat-shock proteins (HSP) had been observed after exposure to low-level radiofrequency fields (RFR) exposure in worms and in human endothelial cells . These data have been hypothetically linked at best to stress, and at worse to cancer. Further studies failed to confirm these RFR bioeffects, while very few experiments have looked at the expression of Hsp in mammals exposed to RFR. Earlier however, changes in hsp70 mRNA were reported in the brain on rats exposed to a GSM-900 signal. In this study, we have investigated the effects of a single exposure or repeated exposures to two mobile phone-related RFR signals (GSM-1800 MHz and UMTS) on Hsp25 and Hsp70 expression in rat brains.

Groups of 8 male Wistar rats were exposed to RFR using a loop antenna at a brain averaged specific absorption rate (BASAR) of 2.6 W/kg. BASAR characterisation was performed as previously described at 900 MHz . Rats were submitted to either a single 2-hour exposure or repeated exposures (two hours/day, five days/week, four weeks). Controls for restraining stress, cage-control and positive-control rats were included. Rats were trained to the setup to avoid stress. At the end of exposure, rat brains were fixed, frozen, and coded. 10 μm sections were prepared and stained with anti-Hsp25 and anti-inducible Hsp70 antibodies. The cerebral cortex and the hippocampus were analysed. The significance of differences between groups was evaluated using the Kruskal-Wallis test.

Repeated exposures to UMTS induced an increased expression of Hsp25 and Hsp70 in the retrosplenial cortex and in the hippocampus. A drop in the amount of both Hsp was observed in both the cortex and the hippocampus after a single exposure to GSM-1800, while repeated exposures to GSM-1800 increased Hsp25 expression in the motor and retrosplenial cortex and Hsp70 in the motor cortex. Decreased expression of Hsp, as observed after single exposure to GSM-1800, has been described after treatments with Benzo[a]pyrene or TNFα, but its physiological significance is not clearly established. Our results suggested that repeated exposures to UMTS and GSM-1800 are capable of increasing HSP expression. According to the experimental evidence of the carcinogenic potential of low-level RFR, increase in HSP expression is unlikely to be linked to cancer. We hypothesised that repeated exposures to mobile phone signals may be perceived as a stress and may participate in an adaptative process in the rat brain.

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