Chronic exposure to radiofrequency electromagnetic radiation deregulates key functional proteins in the rat hippocampus

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Worldwide, the number of mobile phone subscribers has reached 8.65 billion in 2022 [1]. Exposure to radiofrequency electromagnetic radiation (RF-EMR) emitted from mobile phones is suspected to be a cause of common fatigue and headache along with more severe effects including behavioral abnormality and cognitive impairment [2,3]. These altered pathophysiological responses to RF-EMR might have resulted from deregulated proteome in the hippocampus. However, there are very few studies on RF-EMR exposure-induced proteome changes in the hippocampus of experimental animal models. Global proteome profiling of the hippocampus tissues from animals exposed to RF-EMR and controls will provide precise information about the effect of RF-EMR at the molecular level.

In this study, we attempted a global proteome profiling study of rat hippocampus exposed to radiofrequency electromagnetic radiation for 20 weeks (for 3 hrs/day for 5 days/week) to identify deregulated proteins and western blot analysis for validation. As a result, we identified 358 hippocampus proteins, of which 16 showed deregulation (log2(exposed/sham) ≥±1.0, p-value<0.05). The majority of these deregulated proteins are grouped into three clusters sharing similar molecular pathways. A set of four proteins (Succinate-semialdehyde dehydrogenase: Aldh5a1, Na⁺ K⁺ transporting ATPase: Atp1b2, plasma membrane calcium transporting ATPase: PMCA, and protein S100B) presenting each functional pathway were selected for validation. Western blot analysis of these proteins, in an independent sample set, corroborated the mass spectrometry findings. Aldh5a1 involve in cellular energy metabolism, both Atp1b2 and PMCA are responsible for membrane transport, and protein S100B has a neuroprotective role. In conclusion, we present a deregulated hippocampus proteome upon radiofrequency electromagnetic radiation which might influence the healthy functioning of the brain.