

# Effects of radiofrequency field exposure on glutamate-induced oxidative stress in mouse hippocampal HT22 cells.

[Kim JY](#)<sup>1,2</sup>, [Kim HJ](#)<sup>1</sup>, [Kim N](#)<sup>3</sup>, [Kwon JH](#)<sup>4</sup>, [Park MJ](#)<sup>1</sup>.

## Author information

### Abstract

#### **PURPOSE:**

To define the impact of radiofrequency (RF) under in vitro experimental Alzheimer's disease conditions, we investigated the effect of RF radiation on glutamate-induced oxidative stress in mouse hippocampal neuronal HT22 cells.

#### **MATERIALS AND METHODS:**

Cell survival rate was measured by MTT and trypan blue exclusion assays. Cell cycle distribution, cell death, and ROS production were analyzed using flow cytometry. Expression of proteins was analyzed by Western blot.

#### **RESULTS:**

RF exposure alone had a marginal impact on cell proliferation, however significantly enhanced glutamate-induced cytotoxicity in HT22 cells. Glutamate augmented the subG1 fraction of cell cycle, annexin/propidium iodide positive cell population, and expression of cleaved poly (ADP ribose) polymerase, which were further increased by RF exposure. Glutamate induced reactive oxygen species (ROS) generation and RF exposure further upregulated it. N-acetylcysteine (NAC) treatment completely abrogated glutamate- and RF-induced ROS production followed by cell death and restored cell proliferation in HT22 cells. Finally, glutamate phosphorylated c-Jun N-terminal kinase (JNK) and RF increased this event further. Treatment with NAC and inhibitor of JNK decreased JNK phosphorylation and restored cell proliferation, respectively.

#### **CONCLUSIONS:**

Our results demonstrate that RF exposure enhanced glutamate-induced cytotoxicity by further increase of ROS production in HT22 cells.