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TARGETING THE MEDIAL PREFRONTAL CORTEX TO AMELIORATE RADIATION-INDUCED NEUROBEHAVIORAL DEFICITS

Abstract

Future long-duration space exploration missions will involve travel outside of the protection of Earth's magnetosphere, exposing astronauts to protons and high energy and charge (HZE) particles through galactic cosmic rays (GCR) and solar particle events. Studies simulating space radiation exposure show that protons and HZE particles can damage multiple tissues, including the central nervous system (CNS), resulting in structural and functional changes to brain regions important for neurobehavioral function, such as the medial prefrontal cortex (mPFC). In the current study, we used a chemogenetic technique (designer receptors exclusively activated by designer drugs; DREADD) to determine the neurobehavioral consequences of silencing the mPFC following radiation exposure in irradiated animals. Male Long-Evans rats were implanted bilaterally with modified inhibitory (pAAV-hSyn-hM4D(Gi)-mCherry) G-protein coupled receptors, irradiated with oxygen ions (16O, 1000 MeV/n) at Brookhaven National Laboratory, and then tested with the social odor recognition memory test. In irradiated rats, acutely silencing the mPFC at specific times during behavioral testing attenuated recognition memory deficits. These data suggest that the mPFC is not only involved in radiation-induced deficits, including deficits in social odor recognition memory, but also that chemogenetically altering activity in this region can ameliorate some radiation-induced neurobehavioral deficits.