Radioprotection of the Brain White Matter by Mn(III) N-Butoxyethylpyridylporphyrin-Based Superoxide Dismutase Mimic MnTnBuOE-2-PyP5+

Supplementary Data

Files in this Data Supplement:

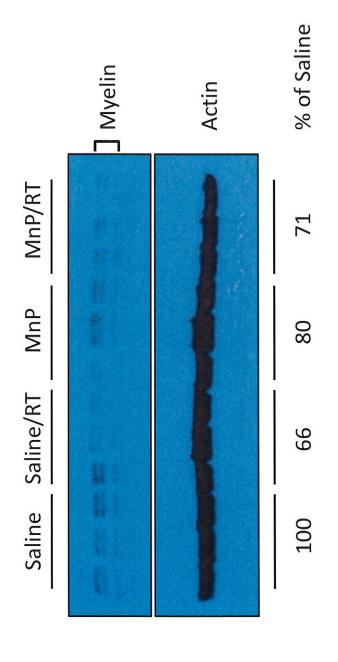
- Supplemental Figure 1- Supplemental Figure 1: Western analysis of myelin within the corpus callosum. Signal
 intensity of myelin and actin were quantitated by Image J, averaged within groups, then normalized to the saline

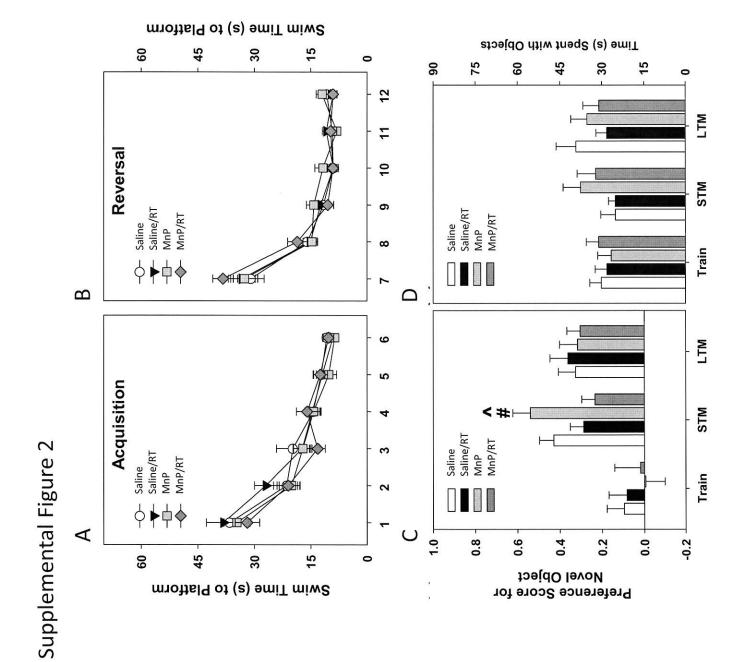
- intensity of myelin and actin were quantitated by Image J, averaged within groups, then normalized to the saline group.

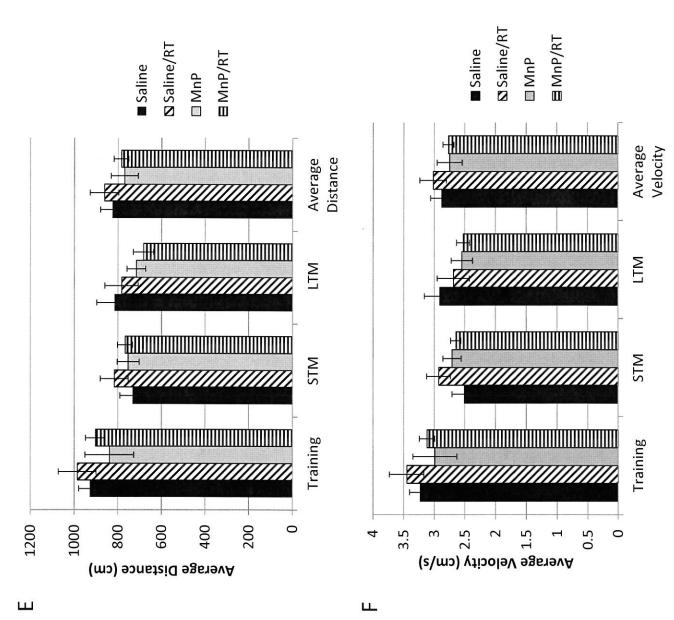
 Supplemental Figure 2- Supplemental Figure 2: Neurocognitive analysis of mice by Morris watermaze (A and B), novel object test (C and D), and fear conditioning (E and F). Tests were performed as described (29). (G and H) Distance and velocity during the novel object training and test periods and overall averages.

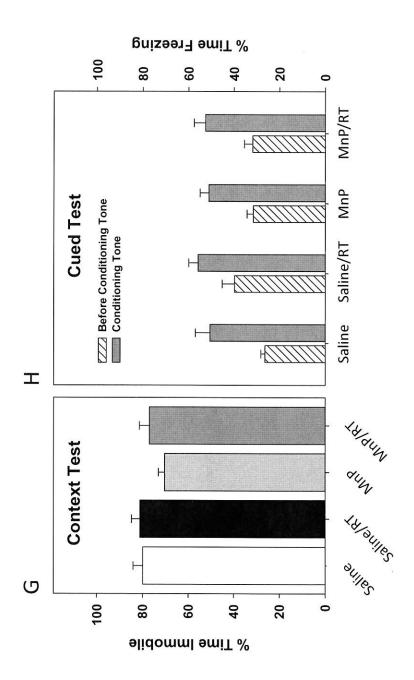
 Supplemental Figure 3- Supplemental Figure 3: Effects of radiation and MnTnBuOE-2-PyP5+ in tumor cells. (A) LN-18 and (B) LN-229 glioblastoma cell lines treated with or without 50 nM MnTnBuOE-2-PyP5+ in clonogenic survival assays prior to various doses of radiation.

 Supplemental Figure 4- Supplemental Figure 4: Model of differential actions of MnTnBuOE-2-PyP5+ in causing the apoptosis of tumor during radiotherapy, while suppressing radiation injury to surrounding normal tissue. Such effects are due to the differential redox environment of tumor vs normal cell, in particular much lower ability of cannec cell to remove cytotoxic hydrogen peroxide. Consequently, much higher levels of hydrogen peroxide already exist in tumor cell, and are greatly enhanced upon radiation. In normal cells and tissues, porphyrins suppress cycling inflammatory responses that may result from radiation also.









Supplemental Figure 3

