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APOPTOSIS AND INFLAMMATORY RESPONSES IN DIFFERENT BRAIN REGIONS OF RATS INDUCED BY HEAVY ION RADIATION AND DRAGON-1'S PROTECTIVE EFFECT

Abstract

Purpose: To investigate the nervous system apoptosis and inflammatory responses in different brain regions of rats after acute irradiation and the mechanism of traditional Chinese medicine-Dragon-1's protective effect. Methodology: The study included three groups of rats: groups of irradiated with or without administration of Dragon-1 and group of control. After adaptive breeding for 5 days, Wistar rats of model and drug group will be radiated by heavy ion at a dose of 7 Gy in head (rats of control group were not radiated) in National Institute of Radiological Sciences, Chiba, Japan. The cortex, hippocampus and striatum were then collected after 24h, 3 days and 7 days. We measured concentration of expression level of apoptotic markers (caspase-3, caspase-8), inflammatory cytokines (IL-1, IL-6) by ELISA. Results: Different regions of brain shown different sensitivities to heavy ions induced apoptosis and dragon-1 has protective effect. The sensitivity of cortex and hippocampus to heavy irons induced apoptosis were different (24 hours vs 3 days) and the drug Dragon-1 can decrease the increased caspase-8. The concentration of caspase-3 and caspase-8 were not stable in striatum with irradiation or not. But dragon-1 can keep them in a low level 24 hours after irradiation, as shown in figure 1. This may relate to its metabolism in vivo. The expression of inflammatory cytokines IL-1 and IL-6 increased in cortex and hippocampus 24 hours after irradiation but there's no significant difference except IL-6 in cortex. In striatum, the level of IL-1 and IL-6 were very similar to apoptosis, as shown in figure 2. The drug Dragon-1 can decrease the increased level ofIL-1 and IL-6. Conclusion: It was concluded that heavy ions radiation can induce high level apoptosis and inflammatory responses in different regions of brain at different times after radiation. The accumulation of these molecules may not directly cause neuron death or immune response.