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p53 and Cell Cycle-Regulated Protein Expression in Small Intestinal Cells after Fast Neutron Irradiation in Mice

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The involvement of the p53 gene in apoptosis of many cell types towards g-radiation is well established. However, little information is available on the relationship between p53 status and cells ability to undergo apoptosis following exposure to fast neutrons. The aim of this study was to characterize the apoptotic pathway traveled by neutrons in mouse intestinal crypt cells. Each mouse received whole body doses of 0.25Gy to 8Gy fast neutrons and were sacrificed 0, 4, 6, 12, 24, 48, and 72hr respectively after irradiation. Apoptosis of crypt cells and expression of p53, cyclin A, cyclin B, cyclin D, and cyclin E were measured. The apoptosis in crypt cells was maximal at 4 and 6 hours after irradiation, showing a gradual decline at 24hours. The highest frequency of apoptosis was seen at a 1Gy dose and then declined gradually beyond a 2Gy dose with high levels of damage. In immunoblot analysis, apoptosis was confirmed to be dependent on p53 function after fast-neutron irradiation. In addition, cyclin B1, cyclin D, and cyclin E were overexpressed in intestinal cells after fast neutrons irradiation and their immunoreactivities were increased strongly in round and oval cells of laminar propria in villi core and crypts. The results of the current study suggest that apoptosis in crypt cells shows a time- and dose-dependent increase after fast-neutron irradiation. In addition, fast neutron-induced apoptosis in mouse intestinal crypt cells appears to be related to the increase in functional p53 proteins to a level sufficient to initiate apoptosis and up-regulation of cell cycle-regulated proteins, which may lead to resistance to DNA damage through cell cycle arrest, is involved deeply in protection of gastrointestinal cells after low doses of fast-neutron irradiation.

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