the normal cells proliferation after treatment of X-ray, however, it can enhance the potential damage effect of X-ray on tumor cells. It suggested that RA have a radioprotective effect on normal cells and can kill the tumor cells as well. In further study, we will investigate its related mechanism at the molecular and genetic level to demonstrate the different effect of RA on tumor and normal cells by low LET radiation.

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3 - 87 In Vivo Evidence of Localization of DNA Damage Proteins within Apoptotic Bodies

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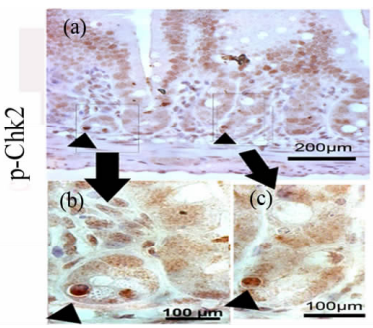

Fig. 1 p-Chk2 (Thr 68) is also localized in apoptotic bodies.

Previous studies have revealed that H2AX is phosphorylated in chromatin flanking DNA double strand breaks DSBs induced by ionizing radiation and is required for the recruitment of the repair machinery to the damaged foci. Therefore, the function of H2AX is believed to be associated primarily with DNA damage repair. In addition to its role in DDR, γ-H2AX was recently identified as an early marker of apoptosis in TNF-related apoptosis-inducing ligand (TRAIL)-treated tumor cell lines[1,2]. In this report, we provide in vivo evidence of H2AX phosphorylation in apoptotic crypt cells that is morphologically distinct from the DNA damage response (DDR) focal pattern. Activated DNA damage response proteins were also identified in apoptotic bodies showed in Fig. 1, where, arrow head indicate apoptotic bodies in crypts that strong positive for p-Chk2 at 1 d after irradiation. The DNA damage response is dramatically enhanced in apoptotic cells compared with non-apoptotic cells.

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