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Vitamin C protects against ionizing radiation damage to goblet cells of the ileum in rats

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Summary

The aim of the present study was to evaluate the radioprotective effect of vitamin C on gamma-radiation-induced damage to goblet cells of the ileum. Thirty male Wistar albino rats weighing between 250 and 300 g were randomized into the following study groups: I, control; II, single dose radiation treated; III, two dose radiation treated with a 4-day interval between doses; IV, single dose radiation treated with vitamin C; V, two dose radiation treated with vitamin C. Each group contained six animals. The rats in groups IV and V were given a daily dose of 100 mg/kg of vitamin C for 14 and 18 days, respectively. During the vitamin C administration period, the rats in group IV were exposed in the abdominal area to a gamma-ray dose of 5 Gy on day 10 and group V was exposed to same dose of radiation on days 10 and 14. Irradiation and treatment groups were decapitated 4 days after exposure to single or two dose irradiation and ileum tissues were removed for light and electron microscopic investigation. Single or two dose gamma-irradiation caused a marked intestinal mucosal injury in rats. Radiation produced increases in the number of goblet cells. Using transmission electron microscopy, extensions in the area between the cells, disorders in the microvilli, mitochondrial damage and endoplasmic reticulum (ER) cisternae dilatation were observed. Antioxidant treatment with vitamin C prior to irradiation provided protection against intestinal damage.
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Introduction

In organisms exposed to long-term ionizing radiation for acute and chronic diseases, accompanying tissue and cell damage may develop, depending on the dose and exposure time (Weiss et al., 1990). More than half of all cancer patients receive radiation therapy at some period during the course of their disease. However, radiation therapy of cancer continues to be dose-limited by the tolerance of critical surrounding normal tissues.

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The intestine is an important dose-limiting organ during radiation therapy of tumors in the pelvis or abdomen. Intestinal radiation toxicity (radiation enteropathy) is, by convention, classified as early or delayed, depending on when it occurs relative to the time of radiation therapy (Wang et al., 2006). It was previously believed that the severity of intestinal radiation toxicity depended directly on cell death in intestinal crypts. This view has been supplanted by the recognition that radiation-induced changes in cellular function and alterations secondary to cell death contribute substantially to the intestinal radiation response (Denham et al., 2001; Denham and Hauer-Jensen, 2002). Because these functional and secondary changes develop over time after radiation exposure, they are particularly promising targets for interventions aimed at preventing or reducing intestinal radiation toxicity.

The gastrointestinal tract is covered by a mucous layer secreted by goblet cells, which arise from pluripotent stem cells present at the base of crypts (Cheng and Leblond, 1974). Goblet cells reside throughout the length of the intestine and are responsible for the production and maintenance of the protective mucous blanket by synthesizing and secreting high molecular weight mucins (Cheng, 1974; Cheng and Leblond, 1974; Forstner, 1978). Other components within the mucus gel include water, electrolytes, sloughed epithelial cells and secreted immunoglobulins. This produces a physical and chemical barrier that protects the epithelium from physical damage by luminal contents, guards against bacterial invasion, regulates epithelial hydration and interacts with secreted immunoglobulin A to produce antibody and antitoxin effects. Much is known about the biochemical nature of mucins and the qualitative distribution of goblet cells in the small intestine (Kemper and Specian, 1991; Specian and Oliver, 1991). However, a kinetic analysis of goblet cell dynamics of rat small intestine in the process of restitution of surface epithelium subjected to radiation injury has to our knowledge not so far been reported.

The effects of radiation are caused mainly by the generation of reactive oxygen species (ROS). These ROS interact with biological molecules producing toxic free radicals leading to lipid peroxidation and DNA damage (Jagetia and Reddy, 2005). Lipid peroxidation has important effects on biological membranes and studies on lipid peroxidation can provide important information about detrimental effects of gamma-radiation. Apart from the lipid peroxidation, ROS can also alter the balance of endogenous protective systems, such as glutathione and enzymic antioxidant (SOD, CAT and GPx) defence systems (Prasad et al., 2005). The endogenous antioxidant defences are inadequate to reduce the radiation-induced free radicals. Appropriate antioxidant intervention may inhibit or reduce free radical toxicity and thus offer protection against radiation. Several dietary antioxidants have been reported to decrease free radical attack on biomolecules (El-Habit et al., 2000). Numerous studies have examined the radioprotective effects of antioxidant free radical scavengers, which protect the cell and its organic constituent molecules from free radical damage.

Vitamin C (ascorbic acid) has been reported to be an effective antioxidant and free radical scavenger and, *in vivo* and *in vitro* conditions, reduce oxidative and free radical-induced damage to DNA and membranes in biological systems (Wilson, 1983). Vitamin C functions as a free radical scavenger of active and stable oxygen radicals and has been shown to protect several biological systems against ionizing radiation. The radioprotective effect of ascorbic acid seems to be due to its interactions with radiation-induced free radicals (Duschesne et al., 1975). Ascorbic acid pre-treatment inhibited the radiation-induced elevation in lipid peroxidation (Jagetia, 2004). It protected the mice against radiation-induced sickness, reduced the mortality and improved the healing of wounds after exposure to whole body gamma-radiation (Mallikarjun Rao and Jagetia, 2004).

Radioprotective effects of vitamin C have been restricted to pro-oxidant properties in high doses. No study has been reported so far on the radioprotective activity of vitamin C on ileum goblet cells. The aim of the present study was to evaluate the radioprotective effect of vitamin C on gamma-radiation-induced damage to ileum goblet cells.

Material and methods

Animals

Thirty male Wistar albino rats weighing between 250 and 300 g were randomized into the following study groups: I control; II, single dose radiation treated; III, two dose radiation treated; IV, single dose radiation treated with vitamin C; V, two dose radiation treated with vitamin C. Each group contained six animals. All animals were kept under standardized conditions: temperature between 22 and 24 °C, relative humidity 50–60%, and 12 h light:dark cycle. They were fed a standard laboratory diet with free access to food and water. Animal care was performed according to the Guide for the

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