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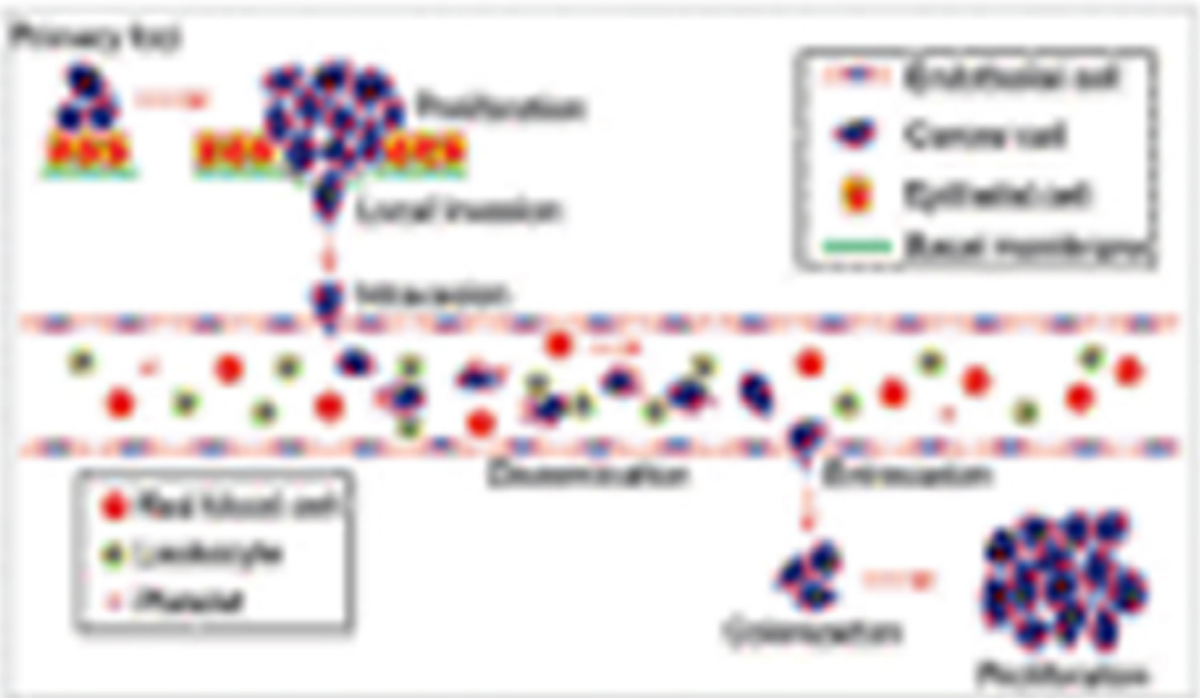

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Protective effects of sodium selenite supplementation against irradiation-induced damage in non-cancerous human esophageal cells

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Abstract. The administration of radioprotective compounds is one approach to preventing radiation damage in non-cancerous tissues. Therefore, radioprotective compounds are crucial in clinical radiotherapy. Selenium is a radioprotective compound that has been used in previous clinical studies of radiotherapy. However, evidence regarding the effectiveness of selenium in radiotherapy and the mechanisms underlying the selenium-induced reduction of the side effects of radiotherapy remains insufficient. To further investigate the effectiveness of selenium in radiotherapy, the present study examined the protective effects of sodium selenite supplementation administered prior to X-ray radiation treatment in CHEK-1 non-cancerous human esophageal cells. Sodium selenite supplementation increased glutathione peroxidase 1 (GPx-1) activity in a dose- and time-dependent manner. The sodium selenite dose that induced the highest GPx-1 activity was determined to be 50 nM for 72 h prior to radiotherapy. The half-maximal inhibitory concentration of sodium selenite in CHEK-1 cells was 3.6 μM. Sodium selenite supplementation increased the survival rate of the cells in a dose-dependent manner and enhanced the degree of cell viability at 72 h post-irradiation ($P<0.05$). Combined treatment with 50 nM sodium selenite and 2 gray (Gy) X-ray irradiation decreased the number of sub-G_1 cells from 5.9 to 4.2% ($P<0.05$) and increased the proportion of G_1 cells from 58.8 to 62.1%, compared with 2 Gy X-ray irradiation alone; however, this difference was not statistically significant ($P=1.00$). Western blot analysis revealed that treatment with 2 Gy X-ray irradiation significantly increased the expression levels of cleaved poly (ADP-ribose) polymerase (PARP; $P<0.05$). In addition, combined treatment with 50 nM sodium selenite and 2 Gy X-ray irradiation reduced the expression levels of cleaved PARP protein, compared with 2 Gy X-ray irradiation alone; however, this reduction was not statistically significant ($P=0.423$). These results suggest that 50 nM sodium selenite supplementation administered for 72 h prior to irradiation may protect CHEK-1 cells from irradiation-induced damage by inhibiting irradiation-induced apoptosis. Therefore, sodium selenite is a potential radioprotective compound for non-cancerous cells in clinical radiotherapy.

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Key words: sodium selenite, radioprotection, X-ray irradiation, esophageal cells, apoptosis, irradiation-induced damage

Introduction

Radiotherapy is one of the most common and effective treatments for cancer (1). Over 40% of patients with cancer require radiotherapy during the management of the disease (2). Although clinical radiotherapy treatment planning and delivery technologies have improved, the toxicity of radiotherapy to non-cancerous tissues and organs remains a problem (2,3). Thus, radioprotective compounds are crucial in clinical radiotherapy (3), and the administration of radioprotective compounds has been suggested as an approach for preventing radiation-damage in normal tissues (4,5).

Selenium is a trace element with a fundamental role in human biology (6). It detoxifies reactive oxygen species (ROS) produced by radiation treatment (4,7). In human antioxidant systems, selenium acts in the form of selenocysteine, which is incorporated into various selenoproteins (8,9). At least 25 selenoproteins have been identified in humans, including glutathione peroxidase (GPx), thioredoxin reductases, iodothyronine deiodinase and the selenoproteins P, W and R (10). Selenium exists in numerous chemical forms, of which the most studied are selenomethionine, sodium selenite, methylselenocysteine, 1,4-phenylenebis (methylene) selenocyanate and methylseleninic acid (9). Sodium selenite is the chemical form