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COMMENTARY

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Biological Targets of Radiation-Generated Free Radicals

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Description

The harmful effects of ionizing radiation on living organisms are well known. The damage is mainly oxidative, and not all changes caused by radiation are equally important to the body. Only damage to vital molecular targets commonly identified as DNA and lipids will impair the body's normal physiological functioning, while most other targets, such as proteins, will be repaired or replaced without adverse effects.

Perhaps, the most extensively studied potential targets of ionizing radiations are the polyunsaturated lipids. There are two reasons for this. First, lipid integrity is important for maintaining cell and organelle membranes, preserving their identity, and providing both a barrier to the external environment and the placement of channels responsible for the selective transport of solutes into and out of cells and compartments. A second reason is the easy oxidizability of unsaturated lipids, especially in condensed phases such as micelles, where one interaction with ●OH (Hydroxyl Radical) can lead to the generation of 100 peroxides. In theory, such chains can cause significant membrane damage from a minor event, such as a reaction with a free radical. There is virtually no evidence for chain oxidation of lipids in biological systems. Rather, attempts to test their possibility have suggested that membrane lipids are not the primary target of radiation-generated radicals.

In an important series of experiments, it was confirmed that the viability of murine fibroblast cells exposed to X-rays was not affected by a fivefold increase in the content of polyunsaturated lipids. In other studies, the formation of lipid peroxides in Sp2/0-Ag and U937 cells under the influence of gamma radiation showed that lipid oxidation was observed at a barely detectable level, demonstrating the effective protection of membranes by the constituent proteins and other components that act as chain breaker to prevent any extensive lipid oxidation.

With respect to DNA, theoretical and experimental evidence indicates that although DNA is clearly a vital biological target, it is not the initial reaction site of primary ●OH generated by radiation. In eukaryotic cells, most of the nucleic acid is in the form of chromatin, where it forms DNA-protein nucleosomes. Numerous studies have shown that nucleosomal DNA is protected from radiation damage by its associated histones and other proteins. Radiolysis of DNA-histone complexes showed that the yields of damaged amino acids were the same as in irradiated single histones, demonstrating that the proteins are effective protective agents for DNA. Significant damage to DNA bases was not observed even at high doses of radiation. In the more complex bacteriophage T4 system, the primary targets of radiation-generated ●OH were proteins, not DNA. In cultured Sp2/0 murine myeloma cells exposed to 60Co γ-rays, DNA fragmentation did not occur immediately after gamma-irradiation, but only after a significant delay and showed a pattern of regular enzyme-mediated strand breaks rather than random breaks associated with direct effects of DNA radiolysis.

The apparent relative resistance of lipids and DNA to direct exposure to ionizing radiation or radical water breakdown products leaves proteins as the primary initial site of damage. The radical responsible for damage under normal physiological conditions was identified as ●OH because eaq- is rapidly absorbed by physiological oxygen. Based on bulk alone, proteins make up about 70% of the organic mass of cells, they are the most common molecules attacked by ●OH. The intracellular molar concentration of proteins is high, 5 to 10 mmol for

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50–25 kDa proteins, and the rate of their reaction with ●OH is usually diffusion controlled with k values among the highest reported, typically >1010 M−1 s−1. Thus, there is a high probability that in living organisms exposed to ionizing radiation, proteins will sustain a large proportion of the initial damage. While the remaining ●OH will react with other biomolecules, the main result is also the formation of aliphatic C-centered radicals, which must be repaired by the same antioxidants as the proteins damaged by the reaction.