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Clustered DNA lesions: From Chemistry, Formation, DNA repair and biological importance

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In cells and tissues there is a constant radiolytic attack that has exogenous or endogenous (intracellular) oxidative origin. During these attacks, although not in all cases, the primary damage is being induced by reactive oxygen species (ROS) and reactive nitrogen species (RNS). Examples of such species are the hydroxyl radical (OH·), O2·-, singlet oxygen (1O2) and peroxynitrite to others. Intracellularly, ROS are primarily created in mitochondria as a natural byproduct of natural metabolism of oxygen but also from cells of the immune system such as macrophages. Human tissues have developed through millions of years of evolution several sophisticated mechanisms such as radical scavengers, antioxidant enzymes catalase and superoxide dismutase and elaborate DNA repair mechanisms. The oxidatively-induced DNA damage consists of a variety of lesions of small to high importance and dangerous for the cell i.e., isolated base lesions or single strand breaks (SSBs) to complex lesions like double strand breaks (DSBs) and other non-DSB oxidatively-induced clustered DNA lesions (OCDLs). In this presentation, I will discuss the current status of knowledge and evidence on the chemistry and formation mechanisms, involvement of intracellular oxidative stress and DNA damage in human pathology and possible use of these parameters as stress biomarkers. At the same time, I will discuss controversies related to potential artifacts inherent to specific methodologies used for the measurement of oxidatively-induced DNA lesions in human cells and tissues.

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