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Cellular Responses to DNA Damage Integrating Autophagy and DNA Repair Pathways for Survival

Gillian Lipsey*

Department of Medical Sciences, Griffith University, South East Queensland, Australia

Commentary

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***For Correspondence:** Gillian Lipsey Department of Medical Sciences, Griffith University, South East Queensland, Australia

E-mail: gillianlipsey162@gmail.com

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DESCRIPTION

The main issue with modern anticancer treatment is resistance to chemotherapy and radiation therapy. Despite much research, the therapeutic result is still far from ideal. Autophagy is one of the many variables that cause drug resistance in cancer cells, and its role is becoming more and more clear. The term "Autophagy" refers to a process of cellular self-digestion in which specific cytoplasmic components can be taken up and eventually broken down in autophagolysosomes to provide the cells with building blocks and nutrients.

Under some conditions, such as hypoxia and growth factor detoxification, digestion can be activated in response to stressors that affect cellular homeostasis. Since maintaining cellular metabolic balance is autophagy's primary physiological function, imbalanced respiration has been connected to a variety of illnesses, including cancer. It's interesting to note that autophagy has both a pro and an anticancer influence on cancer. For example, digestion can prevent the growth of tumours by regulating the division of cells and the generation of oxygen species that are reactive. However, in situations when nourishment is scarce, autophagy could inject the tumour cells sufficient nutrients to sustain tumour growth, which improves the proliferation of the tumour.

Anticancer therapy also exhibits this ambiguous behaviour autophagy has been established to improve the cytotoxicity of chemotherapy medications by causing autophagy cell death. However, phagocytosis is also connected with drug resistance, since it has been observed that inhibiting autophagy can make tumour cells more susceptible to the induction of cell death by anticancer drugs.

This article will address a number of issues pertaining to future drug development including the dual roles that autophagy plays in carcinogenesis and chemotherapy. It will also classify autophagy inducers and inhibitors that are implemented in anticancer treatment.

Inherent, biological, and therapeutic therapy stressors can all cause Deoxyribonucleic acid (DNA) damage, including those brought on by radiation or chemotherapy. This review will mostly focus on DNA Double Strand Breaks (DSBs), which are the deadliest type of DNA damage. DSBs can be caused directly by irradiated DNA bases or indirectly by reacting with reactive oxygen species caused by DNA damaging agents like radiation.

In order to preserve genomic integrity, mammalian cells that detect DNA damage initiate the DNA-damage response, an evolutionarily conserved pathway that senses the damage, transduces a signal through a series of post-translational modifications, and finally results in DNA damage repair. The main kinase in the DNA-damage response is Ataxia Telangiectasia Mutated (ATM). Histone H2AX is phosphorylated by ATM at the damaged DNA locations, which is attracted by DSBs. The factors linked with DSB repair are recruited by H2AX.

The cells with the DNA damage time to relieve pain Atms phosphorylate CHK1 and CHK2 in addition to H2AX. This leads to the p53/p21WAF1-dependent cell cycle regulatory to be activated. Furthermore, decomposition is triggered to supply the fundamental biochemical elements required for functioning vital cellular processes. The quick reactions to DNA damage are cell cycle arrest DNA repair, and degradation. Cells may experience senescence, permanent cell cycle prevent, or premature death through apoptotic or mitotic catastrophe if the DNA damage is too great and or persistent. Thus, DNA repair and autophagy are essentially short-lived, acute processes that come preceding senescence or cell death, which are biological processes that include the cytostatic or cytotoxic departure of living cells from the cell population.