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Perspective

The DNA Damage Checkpoint Pathways: Down Regulation of the Signal and Adaptation

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DESCRIPTION

In order to prevent cell death or the uncontrolled multiplication that is a hallmark of the malignant state, cellular proliferation must be closely managed. The integration of both positive and negative impulses allows for this control. The so-called "checkpoints," which are signal-transduction routes specialized for either aberrant or imperfectly constructed cellular structures, are among the negative controls. These structures may result from the cell's own error-prone internal machinery (such as replication fork collapse), the metabolic environment (such as DNA hydrolysis and oxidation), or they may be brought on by outside forces (e.g. carcinogens).

Cell-cycle progression is inhibited or slowed down by checkpoint pathways until these 'abnormalities' are properly corrected or put together. One such surveillance method that "checks" the integrity of the genome is a DNA-damage checkpoint. This pathway's activation has a number of biological effects that follow. They also involve effective correction of the error in addition to cell-cycle delays and the development of a transcriptional program. The DNA-checkpoint pathway includes sensors, transducers, and effectors, just like all other signal-transduction pathways. The sensors start the checkpoint signal after monitoring DNA for structural flaws. This signal is amplified and transmitted further by transducers. Effectors are in charge of the biological effects of activating the route. As with other signal-transduction pathways, the DNA damage checkpoint pathways exhibit the phenomenon of signal down regulation or adaptation in the on-going presence of the starting signal.

Although checkpoint proteins are highly conserved from yeast to human cells, the fundamental structure of these pathways has remained constant across the course of evolution. DNA-damage checkpoints are thought to be crucial for carcinogenesis in human cells some of these pathways' elements are mutated in uncommon human disorders linked to a higher risk of developing cancer. However, there is minimal evidence connecting mutation of other DNA-damage checkpoints with sporadic human tumours, with the notable exception of the often mutated p53. It will be crucial to ascertain the entire significance of these pathways in cancer.

Mainly on the budding yeast (*Saccharomyces cerevisiae*) model system, with a recent emphasis on breakthroughs in the field of DNA damage checkpoint research. We provide comparisons when appropriate with analogous findings in other model systems. We focus on recent hypotheses about transduction of the damage signal, DNA damage detection, and how checkpoint pathways affect DNA repair. Lastly, a brief discussion is made on cell-cycle restart and adaptation in the presence of irreversible DNA damage. Fission yeast genes and proteins (*Schizosaccharomyces pombe*) are superscripted with the letter F in this review due to the significant nomenclature confusion that exists between the two main yeast systems.

Protein Polymer Poly (ADP-ribose) polymerases catalyse the common post-translational change known as ADP-Ribosylation (PAR) at DNA lesions (PARPs). Many biological processes, including chromatin reconfiguration, DNA Damage Response (DDR), transcriptional control, apoptosis, and mitosis are regulated by this alteration. DNA lesions can activate PARP1, which acts as a DNA damage sensor, producing PAR chains that act as docking platforms for biochemically complex DNA repair components. We will gain a better knowledge of the biological functions of this particular post-translational modification by highlighting molecular insights into PARylation recognition, the expanding significance of PARylation in DDR pathways, and the functional relationship between PARylation and ubiquitination.

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