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DNA Damage Response

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Editorial

The survival of organisms depends on the accurate transmission of genetic information from one cell to its daughters. Such faithful transmission requires not only extreme accuracy in replication of DNA and precision in chromosome distribution, but also the ability to survive spontaneous and induced DNA damage while minimizing the number of heritable mutations [1,2].

To accomplish this delicacy, cells have evolved surveillance systems that monitor chromosomal shape and coordinate cell-cycle progression and repair. The genetic regulation of cell-cycle transitions in response to DNA damage was first discovered in Escherichia coli's SOS DNA damage response pathway1 and in humans in ataxia telangiectasia cells that lack the ataxia telangiectasia mutant gene.

Later, this regulation was discovered in yeast, and the name checkpoint was coined to describe the yeast pathway. DNA damage checkpoints were originally thought to be non-essential regulatory processes that regulate cells' capacity to pause the cell cycle in response to DNA damage, giving time for repair. Recent research reveals, however, that the checkpoint pathway's historical definition is insufficient to fully explain its function.

These pathways have been shown to control the activation of DNA repair pathways, the composition of telomeric chromatin and the movement of DNA repair proteins to sites of DNA damage transcriptional programme activation telomere length and, in some cell types for reasons that are unknown, induction of cell death by apoptosis. As a result, it is now obvious that the checkpoint is a subroutine that governs a multidimensional response and is part of the wider DNA damage response mechanism.

Furthermore, some checkpoint genes are required for cell and organism survival showing that these pathways are more than just surveyors of occasional harm, but are integral parts of cellular function. The condition ataxia telangiectasia is an illustration of the physiological relevance of these pathways. Individuals with two mutant ATM genes may experience a variety of issues, including loss of motor control due to Purkinje cell loss, immunological deficiencies, and an increased risk of cancer.

Because ATM is a key signalling protein in the DNA damage response, cells without it are unable to carry out many of the biological responses to DNA damage. Because many chromosomal structural errors arise during normal cell duplication, AT patients face several issues even when they are not exposed to DNA-damaging substances. Errors in DNA replication, such as double-strand breaks caused by halted replication forks, necessitate the DNA damage response pathway's attention.

Aside from these inevitable faults, cells live in a highly reactive chemical environment in which reactive species such as free radicals are by-products of cellular metabolism or results of the cells' presence in an oxygen-rich environment. These reactive organisms react with DNA, causing additional issues to be resolved. ATM has been suggested to play a role in the cellular response to oxidative stress, which might be especially significant for long-lived, terminally differentiated cells like Purkinje cells. Furthermore, DSBs are controlled components of VDJ recombination and meiosis, not just natural occurrences. ATM is also required to respond to these developmentally regulated DNA changes, emphasising its significance in organism physiology.

The recent discovery of links between 'checkpoint' pathways and DNA repair, as well as their physiological consequences on cells, motivated us to reconsider checkpoint proteins' function in the overall response to DNA damage. We'll focus on data from mammalian cells since that's where a model of the DNA damage response is starting to take form. Human and yeast DNA damage response systems are quite similar, and we'll try to exploit these similarities to fill in the gaps in our understanding of the mammalian system [3-5].

Conflict of Interest

None.

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