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Short Communication

Oxidative DNA Damage: Affecting Signal Transduction Pathways and Transcription Rates

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DESCRIPTION

Numerous neuropathologies include oxidative and nitrooxidative stress as a major contributing factor in their pathogenesis. The effects of Reactive Oxygen and Nitrogen Species *(ROS/RNS)* on the synthesis of oxidized proteins and lipid peroxidation within neurons have been the subject of the research [1]. However, oxidative DNA damage and its impact on neuronal function are another result of oxidative stress that has been understudied in the field of neuroscience. The idea that DNA damage is less hazardous in fully differentiated neurons compared to other cell types because neurons do not undergo cellular division is one possible explanation for the dearth of neuroscience research in the domain of oxidative DNA damage.

Normal cellular metabolism generates reactive oxygen and nitrogen species, and excessive generation is reduced by the potent endogenous antioxidant systems found in neurons [2]. However, the antioxidant defenses are frequently outmatched, and unchecked *ROS/RNS* can result in oxidative DNA damage. When a highly reactive oxygen or nitrogen species interacts with a DNA base, different base lesions are produced, which causes oxidative DNA damage. 7,8-dihydro-8-oxo-2'-deoxyguanosine (8oxo-dG), which is created at the 8th carbon of the guanine base, is the most abundant oxidative DNA lesion. The 2,6-diamino-4-oxo-5-Formamidopyrimidine (FapyG), which likewise forms on the guanine base, and the 7,8-dihydro-8-Oxoadenine (oxoA), which forms on the adenine base, are examples of additional lesions. The importance of 8oxo-dG and FapyG is highlighted by the relative abundance of these lesions, but there is also room to investigate less common lesions, like oxoA and others, and to critically evaluate the relative incidence of these lesions in neurons compared to other cell types.

As previously indicated, oxidative damages can result in altered DNA transcription and signal transduction, which can affect cells' ability to operate. Repair of oxidative DNA damage is predominantly carried out through the Base Excision Repair (BER) mechanism. Depending on the lesion of interest, several glycosylases can cleavage the damaged base. 8-Oxoguanine DNA Glycosylase-1 *(OGG1)* is the main glycosylase in charge of cleaving 8oxodG [3].

Nth Like DNA Glycosylase 1 *(NTHL1)* and Neilike DNA Glycosylases 1 and 2 *(NEIL1* and *NEIL2)* are two other glycosylases that can detect 8oxodG, although their main mode of recognition is other pyrimidine lesions. Thymine-DNA Glycosylase (TDG) and Uracil-DNA Glycosylase (UNG) are two more DNA glycosylases that identify and cleave non-guanine lesions.

The appropriate glycosylase removes the base, creating an abasic site that is identified and processed by AP Endonuclease 1/Ref-1 (APE1/ Ref-1). DNA Polymerase (Pol) may recognize and fill the abasic site with the proper base through template-directed synthesis because APE1/Ref-1 processes the ends on the DNA backbone. APE1/Ref-1 serves as an endonuclease, but it also has a redox role that controls transcriptional activity by reducing a number of transcriptional factors. Nudix hydrolase 1 (also known as *MTH1*) hydrolyzes oxidized bases in the nucleoside triphosphate pools to prevent integration of a damaged base into a nucleic acid, which is another enzyme that reduces oxidative DNA damage [4].

Research is currently being done on various neuropathologies to determine the relative contributions of these various parts of the base excision repair pathway to prevent the harmful effects of oxidative DNA damage. Here, we will briefly address new research on the effects of DNA-damaging chemotherapeutics on sensory neurons and examine the role of oxidative DNA damage in different disorders of the peripheral and central nervous systems.

CONCLUSION

Since post-mitotic cells, like neurons, lack effective alternative methods to repair the damage, such as replication proofreading and mismatch repair, the repair of oxidative DNA damage through activation of the BER pathway is crucial. According to the information presented here, there is substantial evidence linking oxidative DNA damage to neuropathologies and weaker

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evidence suggesting that DNA damage plays a role in the development or progression of various disorders affecting the peripheral and central nervous systems.

To better understand how oxidative DNA damage affects transcriptional patterns to affect neuronal function, more research is required. Similar to this, more research is required to determine the causal role of oxidative DNA damage in various neurodegenerative illnesses and to investigate treatments that can encourage DNA repair and possibly result in advancements in clinical patient care.

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