Review of: "Global and transcription-coupled repair of 8-oxoG is initiated by nucleotide excision repair proteins"

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Many in vitro systems have been established for the detection of nucleotide excision repair (NER) activities in cell and tissue extracts, but few investigators attempted to answer how NER factors could get access to helix-distorting lesions buried in compact chromatin structures. Earlier studies using minichromosomes as in vitro repair substrates have shown a high possibility that NER factors are able to disassembly local chromatin structures before their contact with DNA lesions. Although the DDB2 subunit of UV-DDB is critical to the initiation of UV-dependent NER because of its high affinity for UV-induced dipyrimidine photoproducts, this article showed the recruitment of DDB2 to 8-oxoG-containing sites and the recruitment of DDB2 preceded that of 8oxo-G DNA glycosylase. indicating a tight collaboration between NER factors and base excision repair (BER) machinery for the removal of a oxidized base damage with high mutation potential. Besides demonstrating the novel role of DDB2 in BER, results of this study also greatly advance our understanding on the approach of repair proteins to DNA lesions wrapped by chromosomal proteins, as authors conducted elegant experiments that revealed the ability of DDB2 to disassemble chromatin organization. Hence, the most significant contribution of this article to DNA repair community is that DDB2 is not only a key factor in sensing various types of DNA lesions but also in opening the path for the access of other repair proteins to lesion sites.