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Short Communication

DNA repair by polymerase δ in *Saccharomyces cerevisiae* is not controlled by the proliferating cell nuclear antigen-like Rad17/Mec3/Ddc1 complex

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ABSTRACT. DNA damage activates several mechanisms such as DNA repair and cell cycle checkpoints. The *Saccharomyces cerevisiae* heterotrimeric checkpoint clamp consisting of the Rad17, Mec3 and Ddc1 subunits is an early response factor to DNA damage and activates checkpoints. This complex is structurally similar to the proliferating cell nuclear antigen (PCNA), which serves as a sliding clamp platform for DNA replication. Growing evidence suggests that PCNA-like complexes play a major role in DNA repair as they have been shown to interact with and stimulate several proteins, including specialized DNA polymerases. With the aim of extending our knowledge concerning the link between checkpoint activation and DNA repair, we tested the possibility of a functional interaction between the Rad17/Mec3/Ddc1 complex and the replicative DNA

polymerases α, δ and ε. The analysis of sensitivity response of single and double mutants to UVC and 8-MOP + UVA-induced DNA damage suggests that the PCNA-like component Mec3p of *S. cerevisiae* neither relies on nor competes with the third subunit of DNA polymerase δ, Pol32p, for lesion removal. No enhanced sensitivity was observed when inactivating components of DNA polymerases α and ε in the absence of Mec3p. The hypersensitivity of *pol32*Δ to photoactivated 8-MOP suggests that the replicative DNA polymerase δ also participates in the repair of mono- and bi-functional DNA adducts. Repair of UVC and 8-MOP + UVA-induced DNA damage via polymerase δ thus occurs independent of the Rad17/Mec3/Ddc1 checkpoint clamp.

Key words: Checkpoint; DNA replication; DNA repair; Proliferating cell nuclear antigen; Rad17/Mec3/Ddc1; *Saccharomyces cerevisiae*

