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UV induced DNA damage repair in bacteria

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The "Dogma" that has been accepted for many years in the field of mutagenesis and lethal of effect in bacteria was non-selective in the nature of its repair mechanism, as well as, non-special and rather primitive. We studied the influence of a temporary specific inhibition of post-radiation macromolecular syntheses and of preliminary UV irradiation on the kinetics of accumulation of fixed mutations, that is mutations insensitive to MFD, in UV-irradiate *B. subtilis* cells. From experimental results, it is deduced that the entry of pre-mutagenic lesion into a round of replication, initiated before irradiation, is not a fixing event in UV mutagenesis. For performance of fixation, the proceeding of replication, initiated after irradiation, and protein synthesis are necessary. In irradiate cells incubated in medium with lowered concentration of nitrogen sources, the anti-mutagenic activity of UVR-dependent repair system competes with the process of fixation for pre-mutagenic lesions and reduce the efficiency of mutagenesis. The most efficient fixation and mutagenesis occur at high concentration of nitrogen sources in post-radiation medium, when the manifestation of anti-mutagenic activity appears to be blocked. The possible nature of a process leading to mutation fixation in the detection of specific pathway-dependent mechanisms being conferred only to the acquired post-irradiation metabolism. It had also long been believed that both pre-mutagenic as well as pre-lethal lesions are same pathways function until the adaptive repair mechanisms come into actions. However, this simplistic sight, in which DNA repair performs, could not explain how bacterial cell recognize and trigger different repair pathways cascades responses.

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