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Review

Global genome and transcription-coupled nucleotide excision repair pathway in prokaryotes

Manoj Thakur[1]* and Kalappa Muniyappa[2]

[1]*Department of Botany, Sri Venkateswara College, University of Delhi, Benito Juarez Marg, Dhaula Kuan, New Delhi 110 021, India*

[2]*Department of Biochemistry, Indian Institute of Science, Bengaluru 560 012, India*

**Corresponding author (Email, manojthakur@svc.ac.in)*

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In all cells—from bacteria to humans—nucleotide excision repair (NER) is a highly conserved, versatile DNA repair pathway that is responsible for the removal of a wide variety of DNA helix-distorting lesions arising from both endogenous and exogenous sources. In many organisms including bacteria, fungi, animals, and plants, NER occurs through two sub-pathways: the global genome NER (GG-NER) pathway and the transcription-coupled NER (TC-NER) pathway. Although essential factors and basic steps involved in NER have been identified, mechanisms and stages of their assembly process are not well understood. In this review, we summarize recent literature about protein interaction networks that manifest during initial stages of bacterial NER pathway while highlighting some of the key functional studies.

Keywords. Mfd; nucleotide excision repair; transcription-coupled repair; UV irradiation; UvrABC excinuclease; UvrD

1. Introduction

A plethora of DNA-damaging agents both from endogenous sources such as reactive oxygen species and exogenous sources such as ultraviolet light, polycyclic aromatic hydrocarbons, mycotoxins, and ionizing radiation can directly distort the structure of DNA double helix and/or break DNA strand(s) (Chatterjee and Walker 2017). These lesions, if not repaired in a timely and efficient manner, can result either in mutations or block DNA-based processes, which will compromise the viability of cells (Sale *et al*. 2012; Kreuzer 2013; Thakur and Muniyappa 2023). Thus, bacteria have evolved DNA-damage response (DDR) mechanism(s) and encode DNA-repair proteins/enzymes to recognize and repair a wide diversity of DNA lesions to safeguard the integrity of their genomes (Friedberg 2003; Morita *et al*. 2010; Kreuzer 2013). These distinct DNA-repair mechanisms include direct reversal of damage, nucleotide excision repair (NER), mismatch repair (MMR), base excision repair (BER), homologous recombination (HR) mediated repair, non-homologous end-joining (NHEJ), and interstrand crosslink repair (ICL) (Hoeijmakers 2001; Morita *et al*. 2010). An in-depth discussion on MMR, BER, HR, NHEJ, and ICL pathways is outside the scope of this review. Here, we discuss recent literature on protein interaction networks that manifest during the initial stages of the bacterial NER pathway obtained from a variety of settings, including biochemical, biophysical, structural, and *in cellulo*-based techniques and/or approaches.

2. Overview of NER pathway

NER in prokaryotes entails two pathways, often described as the global genome repair (GGR) pathway that functions throughout the genome and a transcription-coupled repair (TCR) pathway which specifically repairs lesions that block transcription by RNA polymerase (RNAP) on the coding strand of expressing

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