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HP1β-dependent recruitment of UBF1 to irradiated chromatin occurs simultaneously with CPDs

Lenka Stixová[1], Petra Sehnalová[1], Soňa Legartová[1], Jana Suchánková[1], Tereza Hrušková[1], Stanislav Kozubek[1], Dmitry V Sorokin[1,2], Pavel Matula[1,2], Ivan Raška[3], Aleš Kovařík[1], Jaroslav Fulneček[1] and Eva Bártová[1*]

Abstract

Background: The repair of spontaneous and induced DNA lesions is a multistep process. Depending on the type of injury, damaged DNA is recognized by many proteins specifically involved in distinct DNA repair pathways.

Results: We analyzed the DNA-damage response after ultraviolet A (UVA) and γ irradiation of mouse embryonic fibroblasts and focused on upstream binding factor 1 (UBF1), a key protein in the regulation of ribosomal gene transcription. We found that UBF1, but not nucleolar proteins RPA194, TCOF, or fibrillarin, was recruited to UVA-irradiated chromatin concurrently with an increase in heterochromatin protein 1β (HP1β) level. Moreover, Förster Resonance Energy Transfer (FRET) confirmed interaction between UBF1 and HP1β that was dependent on a functional chromo shadow domain of HP1β. Thus, overexpression of HP1β with a deleted chromo shadow domain had a dominant-negative effect on UBF1 recruitment to UVA-damaged chromatin. Transcription factor UBF1 also interacted directly with DNA inside the nucleolus but no interaction of UBF1 and DNA was confirmed outside the nucleolus, where UBF1 recruitment to DNA lesions appeared simultaneously with cyclobutane pyrimidine dimers; this occurrence was cell-cycle-independent.

Conclusions: We propose that the simultaneous presence and interaction of UBF1 and HP1β at DNA lesions is activated by the presence of cyclobutane pyrimidine dimers and mediated by the chromo shadow domain of HP1β. This might have functional significance for nucleotide excision repair.

Keywords: DNA-damage response, DNA repair, Irradiation, Live-cell studies, Nucleolus, UBF1