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Unravelling molecular mechanisms in transcription-coupled nucleotide excision repair

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Stellingen

behorend bij het proefschrift getiteld

Unravelling molecular mechanisms in transcription-coupled nucleotide excision repair

1. CSB, CSA, and UVSSA are equally important for the recruitment of the TFIIH complex to DNA damage-stalled RNAPII (this thesis; chapter 2).
2. Recruitment of the CRL4^{CSA} complex to DNA damage-stalled RNAPII is not sufficient to catalyze RNAPII ubiquitylation (this thesis; chapter 3).
3. ELOF1 is a transcription-coupled DNA repair factor that promotes CSB-dependent repair and functions in a second pathway that protects cells against DNA damage during replication, which becomes particularly important when canonical TCR fails (this thesis; chapter 3).
4. Proximity-dependent biotin identification is a promising method to identify potential regulators of TCR (this thesis; chapter 4).
5. The inability to remove transcription-blocking DNA lesions does not explain the severe phenotype seen in Cockayne syndrome (Nakazawa et al. 2020; Nakazawa et al. 2012).
6. Contradictory to previous suggestions, not all components that are required for TCR have been identified (Gregersen and Svejstrup 2018; de Boer and Hoeijmakers 2000).
7. The recently developed strand-specific CHIP-seq (TCR-seq) method enables a genome-wide quantification of TCR-mediated repair, which was not possible before (Nakazawa et al. 2020).
8. Genome-wide CRISPR screens will not only enable the identification of novel DNA repair factors but will also shed light on the connections between pathways involved in the cellular response to genotoxic stress (Olivieri et al. 2020).
9. Partner dance sport should be a compulsory element of primary education.