



**Molecular Dissections of DNA replication and Trans-lesion
DNA synthesis in Eukaryotes**

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Genome instability is a hallmark feature of tumor cells and many genetic disorders. Function of the eukaryotic genome solely depends upon efficient and accurate replication of DNA strands and precise removal of various errors in DNA by multiple mechanisms. DNA polymerases (pols) play essential roles in DNA replication, repair and recombination. The budding yeast *S. cerevisiae* is known to possess at least eight and *H. sapiens* possess at least fifteen pols. While pols like δ , ϵ and α are essential for cell survival as they carry out bulk of DNA synthesis, others are not. Pol η (Rad30) is one among them; however, absence of this leads to a variant form of the human genetic disorder Xeroderma Pigmentosum. Pol η is capable of error-free bypassing UV-induced TT dimer, in absence of this polymerase; a different or unknown pol with low fidelity is presumably utilized, leading to an increased level of UV-induced mutations. How do cells manage the actions of different pols to ensure that the right pol gets to the right place at right time? Considering the fact that the replication machinery is extremely dynamic, recruitment of a wrong polymerase will be deleterious to the organism as it can result in mutations or genome rearrangements. My talk will be focused on two classes of DNA pols and their functions at the moving and stalled replication fork.