

Comprehensive Seminar

Understanding the mechanism of selection and turnover of polymerases during Translesion Synthesis

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All life forms depend on DNA replication to faithfully convey genetic information to daughter cells. Often during replication, the replicative polymerases stall upon encountering a lesion. One of the damage tolerance pathways utilised by the cells is 'Translesion Synthesis' (TLS), which helps the replication machinery to progress uninterruptedly by bypassing the lesion by usage of error-prone polymerases. On encountering damage, the TLS polymerases gain access to the stalled fork and trade place with the replicative polymerases to synthesise past the lesion. As TLS polymerases are error-prone cells tend to displace them off the genome shortly after synthesis past the lesion and replicative polymerases are recruited back to re-establish the replisome. Our study aims to unravel the mechanism of polymerase-switch and explore the corresponding changes in the replication fork associated with it. We also aim to address whether the choice of recruitment of the TLS polymerase is random or if specific biases exist depending on the encountered damage. In addition, we are attempting to determine the kinetics of recruitment and resolution of different TLS polymerases during damage bypass.

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2:30 PM (Tea / Coffee 2:15 PM)

CR-4, TIFR-H