Biomolecular Sciences Institute

School of Integrated Science and Humanity

Seminar Announcement

Different mechanisms for lesion bypass by translesion DNA polymerases

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In humans 17 DNA polymerases have been identified, of which only five in the A- or B-family are dedicated to normal DNA replication and the other 12 are specialized DNA polymerases for repair and translesion DNA synthesis (TLS). The specialized DNA polymerases mainly belong to the X and Y families, but a few are in the A and B families and homologous to the high-fidelity replicases. These TLS DNA polymerases in different structural families act on lesion bypass at two stages by different mechanisms. Human DNA pol η is a typical translesion synthesis polymerase and recognizes the major UV lesion, and it can accurately incorporate nucleotide opposite a UV-induced pyrimidine dimer, thus constituting the first stage of TLS. Deficiency of DNA pol η causes the variant form of Xeroderma Pigmentosum (XPV), characterized by sunlight-induced pigmentation changes and a highly elevated incidence of skin malignancies. DNA pol ζ is a B-family member and appears to play a role in the second stage of lesion bypass that is primer extension immediately after DNA lesions. How the replicative polymerase-like TLS polymerases recognize and bypass lesions remains a puzzle. Using biochemical and structural approaches, Dr. Wei Yang's lab finds that these specialized polymerases use different mechanisms to avoid "road blocks" and carry out translesion DNA synthesis with errors. Dr. Yang will present her latest results on discerning different mechanisms of TLS essential for cell survival.

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11:00 a.m. to 12 p.m.

Room: PG5-153 at MMC, MSB-105 via Polycom at BBC

This event is co-sponsored by the Biochemistry Ph.D. Program Seminar Series, Department of Chemistry and Biochemistry and the Biomolecular Sciences Institute.

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