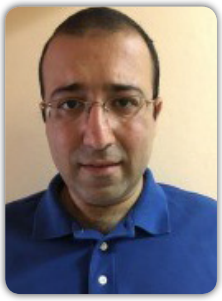

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The mysteries of S6K2 may shed light to Breast Cancer Therapy path

The divergence between S6 Kinase 2 (S6K2) and its homologue S6 Kinase 1 (S6K1) has displayed that the exclusive functions of S6K2 are very important mediators of tumor growth. Recent studies suggest that S6K2 complexes with B-Raf and PKCε to exert cancer cell survival. Also, indirect roles of S6K2 which involve interaction with Akt and PDCD4 to propagate cancer cell survival makes it an important therapeutic target. Also, centrosomal localization of a pool of S6K2 potentiates a proliferative role. Amplification and overexpression of *RPS6KB2* gene locus, which encodes S6K2 protein, is observed in breast cancer and is correlated with poor prognosis. Also, S6K2 expression is correlated with 4EBP1 and E2F1 expression in breast cancer. Also, breast cancer tissues display nuclear over-accumulation of S6K2 when compared to its normal counterparts.

Currently, the mechanisms which regulate the cellular levels of S6K2 are unknown. Also, there still remains new substrates of S6K2 to be unraveled. As the mysteries of S6K2 is solved, new stones are paved in the breast cancer therapy path.

Speaker Biography

Nurettin İlter Sever has completed his PhD in 2013 from The Ohio State University, USA. He is currently an assistant professor at Pamukkale University, Denizli, Turkey. He is currently establishing his laboratory and is a member of EACR.

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