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Kakoli Bose, J Cancer Sci Ther 2015, 7:10
http://dx.doi.org/10.4172/1948-5956.C1.054

Unraveling a novel adaptor-independent extrinsic cell death pathway

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Early protein E2 of human papillomaviruses (HPV), that are associated with cervical and anogenital cancers regulates viral DNA replication and transactivation of essential viral oncogenes. Apart from these functions, E2 protein from high risk virus types such as HPV-16 and -18 triggers apoptosis in host cell. Although the exact mechanism is unclear, recent literature suggests that in HPV-18 E2, the N-terminal transactivation domain directly interacts with procaspase-8, a component of Death Inducing Signaling Complex (DISC) in the extrinsic cell death pathway. This interaction bypasses the requirement of upstream adaptor proteins which are essentially required for DISC formation, thereby representing a novel adaptor-independent caspase activation pathway. In this work, we dissected the binding interface of E2-procaspase-8 and classical FADD-procaspase-8 interaction using an interdisciplinary approach employing techniques such as *in silico*, mutational, biochemical and biophysical analyses. Our results provide a molecular basis of this novel E2-procaspase-8 interaction and help in providing a model for E2-induced apoptosis in high risk HPV types. This information may be utilized in future studies to design E2 analogs so as to modulate procaspase-8 activation and hence promote apoptosis.

Biography

Kakoli Bose has completed her PhD from North Carolina State University, USA in Biochemistry and took Post-doctoral from Tufts University, Boston, USA. Currently she is working as a Principal Investigator in Advanced Centre for Treatment Research and Education in Cancer (ACTREC), India.

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