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The anti-proliferative activity of IRF-1 depends on a phospho-dependent degradation switch

Nicole Clarke
University of Nottingham, UK

Interferon regulatory factor-1 (IRF-1) is an important anti-oncogenic transcription factor. A key component of IRF-1's tumor suppressor activity is the induction of apoptosis in cancer cells and many of its' gene targets are involved in the extrinsic or intrinsic apoptotic pathways. One area still lacking in terms of understanding IRF-1's activity is its' regulation by post-translational modifications. We have discovered that a novel phosphorylation event is required for proper IRF-1 function. The phosphorylation is required for the poly-ubiquitination and subsequent degradation of IRF-1. Mutation of the phosphorylation site or inhibition of the kinase results in a protein with increased stability that blocks processive transcription from target genes. Significantly, the IRF-1 phospho-mutant protein is defective in restricting the growth and proliferation of cancer cells in contrast to wild-type IRF-1. These data will impact on our understanding of interferon-regulated immune responses and the regulation of anti-oncogenic pathways in cells.

Biography

Nicole Clarke completed her Ph.D in Molecular and Cellular biology from Columbia University and postdoctoral studies at the Institut de Génétique et de Biologie Moléculaire et Cellulaire. Her research work is focused on elucidating tumor suppressor networks in cells and understanding how these pathways are affected by novel protein-protein interactions and posttranslational modifications. She has published in international journals and serves as an editorial board member for ISRN Molecular Biology.

Nicole.Clarke@nottingham.ac.uk