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Cellular targets of EBV-encoded nuclear antigens (EBNAs) and their involvement in virus-induced transformation

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EBV is one of the most highly transforming viruses known. The mechanism of transformation is not understood in detail, but it is clear that the virus exploits the normal signaling pathways of the B lymphocyte. The six growth transformation associated viral proteins promote cell proliferation and protect from apoptosis. DNA damage, oncogene activation and other types of cellular stress induce p53 dependent G1 arrest or apoptosis. Primary EBV infection of human B-lymphocytes leads to a striking increase of p53 expression with no induction of apoptosis. LCLs maintain p53 in the wild type configuration. We found recently that MDM2, a negative regulator of p53, binds to EBNA-5 and forms a tri-molecular complex with p53. This leads to the maintenance of p53 expression in LCLs at a high level, but in a transcriptionally inactive state. Like other DNA tumor viruses, EBV induces an Sphase in its natural host cells. We have found that EBNA-6 binds to mitochondrial ribosomal protein MRPS18-2 (S18-2). The interacting of EBNA6 with S18-2, a pRb binding protein, may inhibit the association of pRb with E2F1 competitively and thereby facilitate the entry of EBV infected B-cells into the Sphase. Recently we have found that an overexpression of the human S18-2 protein leads to immortalization of the rat primary fibroblasts (REFs). Immortalized cells expressed embryonic stem cell markers that were not present on the control REFs. We have found, using microarray techniques, that 18IM cells and REFs differ dramatically in gene expression, despite minor changes of the genome methylation pattern.

Biography

Elena Kashuba has completed her PhD in Chemistry from Institute of Physical Chemistry of NASU, Kiev, Ukraine, and PhD in Tumor Biology from Karolinska Institutet, MTC, Stockholm, Sweden. She is an Associated professor at MTC, Karolinska Institutet. She has published 58 papers in reputed journals in the fields of Chemistry and Tumor Biology.