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Mechanisms of apoptosis induced in immune cells directly or indirectly by virus infection

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Apoptosis of immune cells is an important factor in pathogenesis of certain viral diseases. Specifically those viruses, which target directly immune cells, e.g., lymphocytes - manifest often, at least in part, with increased cell death with consequent immune deficits. Typically in the diseases like HIV infection, the virus targets CD4+ T cells, which then undergo apoptosis or activation induced cell death at an increasing rate. Other viruses, e.g., some herpetic viruses are known to infect immune cells, but the role of this infection in the immune response of the host is not clear. The potential of other viruses, such as HCV, to target directly immune cells is still being discussed. Several pathways, which can serve as targets for virus-induced dysregulation of immune cells, can have a significant effect on apoptosis. One of the important ones is mediated by Akt kinase. This kinase is regulated mainly by phosphorylation of two sites - Ser473 and Thr310. The phosphorylation status of these sites dictates downstream signaling through GSK3beta, which manifests by phosphorylation of its Ser9 site. We show that several viruses mentioned above exert significant effects on this pathway, which, in turn, correlates with the rate of apoptosis of T lymphocytes.

Biography

Pavel Bostik completed his MD at Charles University School of Medicine in Prague, Czech Republic in 1990 and his PhD at FMHS in Czech Republic. He conducted his Postdoctoral studies at the University of Iowa, School of Medicine. He subsequently worked at the Emory University School of Medicine in Atlanta, GA until 2009. He is the Vice Dean for Research at the FMHS and Professor at Charles University School of Medicine in Hradec Kralove, Czech Republic. He has published more than 50 papers in reputed journals. His focus is in the effect of viral infections on intracellular signaling in T cells.

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