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Involvement of Crk adaptor proteins in T cell signaling

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Adaptor proteins participate in signal transduction from the T cell antigen receptor by simultaneously interacting with signaling effector molecules and receptor subunits or receptor-associated molecules. By recruiting enzymes and other molecules to the receptor site adaptor proteins promote molecular events essential for signal propagation.

The Crk adaptor proteins possess an N-terminal SH2 domain and one or two C-terminal SH3 domains. They have been linked to signaling from a large variety of receptors, and were found to interact with multiple effector molecules that regulate cell growth, differentiation, transformation, and apoptosis.

We found that CrkII interacts with tyrosine phosphorylated ZAP-70 in TCR engaged T cells by SH2-mediated binding of pTyr 315 in the ZAP-70 interdomain B region. In addition, T cell activation induced the formation of a trimolecular complex that includes CrkII, the PI3K regulatory subunit, p85, and the Cbl protein.

Recently we found that CrkII serves as an in vivo substrate for peptidyl-prolyl *cis-trans* isomerases (PPIases) and that its in vivo conformation and activity are regulated by cyclophilin A, a member of the immunophilin family of PPIases.

Our studies suggest the existence of a potential synergism between the effects of PPIase inhibitors on the calcineurin-NF-AT- and the CrkII-regulated signaling pathways during the early phase of the T cell activation response

Biography

Noah Isakov holds the Joseph H. Krupp Chair in Cancer Immunobiology. He is a Professor of Immunology and Head of the Shraga Segal Department of Microbiology and Immunology at the Faculty of Health Sciences and the Cancer Research Center, Ben Gurion University of the Negev, Beer Sheva, Israel

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