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# Guest Editor

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**Dr Benjamin Bonavida**

Benjamin Bonavida earned his PhD at the University of California at Los Angeles (UCLA). He did his postdoctoral work in immunochemistry investigating synthetic polypeptides and nucleic acids at the Weizman Institute of Sciences in Rehovot, Israel with Drs Michael Sela and Sarah Fuchs. He then joined the Department of Microbiology and Immunology at the UCLA School of Medicine as an Assistant Professor. Currently, he is a Professor of Immunology at the Department of Microbiology, Immunology, and Molecular Genetics and the UCLA David Geffen School of Medicine. Dr Bonavida's earlier research at UCLA focused on the mechanism of cell-mediated cytotoxicity and with particular emphasis on natural killer (NK) cell-mediated cytotoxicity and the identification of NK-specific soluble cytotoxic factors. During the last decade, Dr Bonavida's research has been in the field of immunobiology of cancer and particularly on the molecular mechanisms underlying resistance of tumor cells to cytotoxic stimuli (cytotoxic cells, chemotherapeutic drugs, hormones, etc.). Earlier studies identified the development of cancer cell cross-resistance to apoptotic stimuli induced by various cytotoxic agents. Studies have identified gene products that regulate resistance to apoptosis and the demonstration that resistance can be overcome by sensitizing agents targeted at genes involved in resistance. Recent studies have also focused in the field of antibody-mediated immunotherapy in cancer. Earlier studies with Rituximab (anti-CD20 monoclonal antibody), currently used in the treatment of B-cell malignancies, have demonstrated Rituximab-mediated cell triggering and the modification of several intracellular survival pathways and the demonstration that combination treatment of Rituximab and a chemotherapeutic drug resulted in reversal of resistance and synergy was achieved. The studies have also identified several intracellular targets whose modification by pharmacological inhibitors resulted in the reversal of resistance when used in combination with chemotherapeutic drugs. Additional studies have also identified Rituximab-mediated sensitization to FasL and TRAIL via inhibition of the transcription repressor Ying Yang 1 (YY1), which regulates Fas and DR5 transcription and expression. Studies on Rituximab resistance have also been investigated with the development of Rituximab-resistant NHL clones and the demonstration that such clones are not triggered by Rituximab and cannot be sensitized; however, intervention with pharmacological inhibitors of the hyperactivated survival pathways reversed resistance to both chemotherapy and immunotherapy. The above studies have identified the clinical importance of the overexpressed transcription factor YY1 as well as the underexpressed expression of the tumor metastatic gene product Raf kinase inhibitor protein in cancer cells. The transcriptional regulation of these gene products and their relevance in the pathogenesis of cancer and malignancies are currently being investigated.