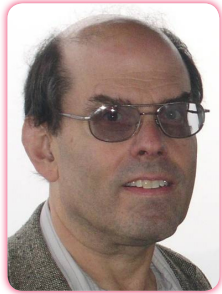


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Chronic interferon-gamma expression: Impact on the host

Interferon-gamma (IFN-g) affects both the innate and adaptive immune response. We have created a 162 nt substitution of the AU-rich element (ARE) in the 3'UTR of the IFN-g gene, resulting in a stabilized IFN-g mRNA and chronic low levels of circulating IFN-g protein. The ARE-deleted (ARE-Del) mice develop a lupus-like disease characterized by the presence of autoantibodies and glomerulonephritis. IFN-g induced gene expression remains high at 3, 6 and 12 weeks of age indicating that there is no desensitization to the effects of this cytokine. Furthermore, the changes in gene expression contribute to an altered serum metabolome by 12 weeks of age. Greater percentages of B cells are found in the lymph nodes and thymus with a decrease observed in splenic B cells. Marginal zone B cells and macrophages are absent but can be restored by elimination of TLR7 or the Type 1 IFN receptor. Overall, we demonstrate that chronic low serum IFN-g levels (30-40 pg/ml) promotes the development of SLE-like disease and suggest that IFN-gamma expression may contribute to disease in at least a subset of lupus patients.

Biography

Howard A Young obtained his PhD in Microbiology at the University of Washington and carried out Postdoctoral research at the NCI. He was a member of the Laboratory of Molecular Immunoregulation, NCI, from 1983 to 1989 prior to joining the Laboratory of Experimental Immunology in 1989. He was President, International Society for Interferon and Cytokine Research (2004-2005) and served as Chair of the Immunology Division of the American Society for Microbiology. He has also served as Chair of the NIH Cytokine interest Group and co-Chair of the NIH Immunology Interest Group. He is a two time recipient of the NIH Director's Award for Mentoring (2000, 2006) and in 2006 he received the National Public Service Award. In 2007, he was named Deputy Chief, Laboratory of Experimental Immunology.

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