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1975	B.A., Biochemistry, Harvard University, Cambridge, MA
1980	Ph.D., Cell & Developmental Biology, Harvard University
1982	M.D., Harvard Medical School, Boston, MA
1985	Resident, Internal Medicine, Brigham & Women's Hospital, Boston, MA
1987	Fellow, Molecular Biology, Whitehead Institute, Massachusetts Institute of Technology, Cambridge, MA
1987-1991	Assistant Investigator, Howard Hughes Medical Institute, University of Michigan, Ann Arbor, MI
1990-1993	Associate Professor, Internal Medicine (with tenure) and Biological Chemistry, University of Michigan
1991-1994	Associate Investigator, Howard Hughes Medical Institute, University of Michigan
1993-1999	Professor, Internal Medicine (with tenure) and Biological Chemistry, University of Michigan
1994-1999	Investigator, Howard Hughes Medical Institute, University of Michigan
1995-1999	Henry Sewall Professor of Internal Medicine, University of Michigan
1999-present	Director, Vaccine Research Center, National Institutes of Health, Bethesda, MD

Honors

1992	Midwest American Federation for Clinical Research Young Investigator Award
1992	American Society for Clinical Investigation
1995	Association of American Physicians
1996	ASBMB-Amgen Scientific Achievement Award
1996	Harold W. Siebens Lecturer on Molecular Medicine, Mayo Clinic, Rochester, MN
1996	Sir Henry Hallett Dale Visiting Professorship in Clinical Pharmacology, The Johns Hopkins University
1998	Institute of Medicine-National Academy of Sciences
1998	Elkin Distinguished Investigators Cancer Lectureship Series, Emory University School of Medicine
1998	Kaiser Lecturer in Biomedical Sciences, John A. Burns School of Medicine, University of Hawaii

Fas Ligand (CD95L) and Cancer Gene Therapy

Recent progress in understanding the genetic basis for human cancer has led to a variety of new perspectives regarding the pathogenesis of these diseases. A gene therapy clinical trial employing an expression vector/lipid complex encoding a major histocompatibility complex (MHC) class I protein, HLA-B7, was tested. This stimulated local anti-tumor responses which were useful in the generation of effector cells. We have further explored the cellular and molecular basis of immune suppression in malignancy. One mechanism associated with inhibition of immune function and induction of lymphoid apoptosis involves the CD95-CD95 ligand (CD95L) system. In vivo gene transfer of CD95L inhibited the growth of $CD95^+$ tumor cell lines, as expected. Unexpectedly, marked regression was observed after CD95L gene transfer into a colon carcinoma line which does not express CD95, caused by a potent inflammatory reaction that was induced. Our findings suggest that gene transfer of CD95L generates apoptotic and proinflammatory responses which can induce regression of both $CD95^+$ and $CD95^-$ tumors. More recently, we have begun to define the molecular basis for the suppression of inflammation by tumors and in immune-privileged sites. Our data indicate that TGF-β suppresses the proinflammatory effects of CD95L. Because both CD95L and TGF-β1 inhibit T cell function, we suggest that these cytokines contribute to the development of immunologic tolerance. This information may be useful in regulating responses to tumors. In summary, advances in the understanding of molecular immunology and gene delivery have provided new insights into molecular genetic strategies that may be applied to the treatment of cancer.