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1965–1969	B.S., Biology, University of Detroit
1969–1973	Ph.D., Microbiology, Rutgers University, Rutgers, NJ
1973–1975	Postdoctoral Fellow, Biochemistry, Stanford University, Stanford, CA
1975–1980	Assistant and Associate Professor, Department of Microbiology, School of Medicine, University of Connecticut
1980–1984	Professor, Department of Microbiology, School of Medicine, State University of New York at Stony Brook, Stony Brook, NY
1984–1996	Elkins Professor, Department of Molecular Biology, Princeton University, Princeton, NJ
1989–present	Investigator, Howard Hughes Medical Institute
1996–present	Elkins Professor and Chair, Department of Molecular Biology, Princeton University

Honors

l986–1988	Research Professor, American Cancer Society
1987	W.P. Rowe Award for Excellence in Virologic Research, National Institutes of Health
1993–present	Fellow, American Academy of Microbiology
1994–present	Editor in Chief, *Journal of Virology*
1996–present	Member, National Academy of Sciences
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Use of DNA arrays to probe the biology of human cytomegalovirus

Human cytomegalovirus (HCMV) is a ubiquitous herpesvirus that can cause life-threatening disease in immunocompromised individuals, and it is the leading viral cause of birth defects. We have employed DNA arrays corresponding to cellular and viral genes to study the interaction of the virus with its host cell and to investigate the composition of the HCMV particle.

To identify virus-induced changes in cellular gene expression, RNA was prepared at various times after infection of human fibroblasts with HCMV, and biotinylated RNA probes were prepared and hybridized to an Affymetrix oligonucleotide array corresponding to about 6,500 human genes. We indentified 258 cellular mRNAs whose steady-state level was increased or decreased by a factor of at least 4 during the early phase of HCMV infection. We are now working to identify the mechanism by which these changes are induced and to investigate their physiological consequences. A constituent of the HCMV particle induces many if not all of the changes in cellular RNA levels. This viral glycoprotein interacts with an unknown cell-surface receptor and presumably initiates a signal cascade that significantly alters cellular gene expression. One of the pathways induced by infection includes the cyclooxygenase 2 gene and generates prostaglandins, which are potent second messages that induce a variety of cellular responses. High doses of a cyclooxygenase 2 inhibitor inhibit viral replication, suggesting that HCMV benefits from the induction of this pro-inflammatory pathway.

HCMV virus particles were found to contain RNA. To determine whether this RNA is encoded by the virus, 33P-labelled cDNA was prepared from virion RNA, and used to probe an array containing all 208 open reading frames encoded by the HCMV genome, each represented by an approximately 300-bp, PCR-generated DNA. This analysis revealed that five virus-coded mRNAs are present in virions. These mRNAs are produced during the late phase of infection and packaged into virus particles. They can then function at the start of a new round of infection, as soon as the viral envelope fuses with the host cell membrane.