



## David Baltimore



**Date of Birth** 7 March 1938

**Place** New York, NY (USA)

**Nomination** 17 April 1978

**Field** Biology

**Title** Professor, Nobel laureate in Physiology or Medicine, 1975

**Professional address**

California Institute of Technology

Pasadena, CA 91125 (USA)

### Most important awards, prizes and academies

**Awards:** First recipient of the Gustave Stern Award in Virology (1970); Warren Triennial Prize from the Massachusetts General Hospital (1971); Eli Lilly and Co. Award in Microbiology and Immunology (1971); National Academy of Sciences' United States Steel Award in Molecular Biology (1974); Gairdner Foundation Annual Award (1974); Nobel Prize in Physiology or Medicine (1975); National Medal of Science (1999); Warren Alpert Foundation Prize (2000). **Academies:** US National Academy of Sciences (1974); American Academy of Arts and Sciences (1974); Pontifical Academy of Sciences (1978); Chairman of the Board of Directors, American Association for the Advancement of Science (1980); Honorary Fellowship, American Medical Writers Association (1985); Foreign Member, The Royal Society, UK (1987); Honorary Membership, Alpha Omega Alpha Honor Medical Society (1987); Institute of Medicine (1988); Honorary Member, Japanese Biochemical Society (1991); Fellow, American Academy of Microbiology (1992).

### Summary of scientific research

Research in Dr. Baltimore's laboratory revolves around understanding aspects of the development and function of the immune system. His laboratory examines these issues at many levels – molecular, cellular and organismal – with the ultimate aim of integrating the various types of information. Present foci of activity include: 1) investigation of the NF- $\kappa$ B family of transcription factors and their controlling proteins with emphasis on the effects of ablating the mouse genes for these proteins; 2) extension of the studies on NF- $\kappa$ B to determine its role in neuronal function; 3) study of the role of the development and *c-abl* gene in cellular metabolism; 4) investigation of how memory T cells are set aside during an immune response.

### Main publications

Zarnegar B., He J.Q., Oganessian G., Hoffmann A., Baltimore D., Cheng G. (2004) Unique CD40-mediated biological program in B cell activation requires both type 1 and type 2 NF- $\kappa$ B activation pathways, *Proc. Natl. Acad. Sci. USA* 101, 8108-13; Schatz D.G., Baltimore D. (2004) Uncovering the V(D)J recombinase, *Cell* 116, S103-6, 2 p following S106; Lu W., Yamamoto V., Ortega B., Baltimore D. (2004) Mammalian ryk is a wnt coreceptor required for stimulation of neurite outgrowth, *Cell* 119, 97-108; Leung T.H., Hoffmann A., Baltimore D. (2004) One nucleotide in a kappaB site can determine cofactor specificity for NF- $\kappa$ B dimers, *Cell* 118, 453-64; Baltimore D. (2004) Science and the Bush Administration. *Science* 305, 1873; Qin XF, An DS, Chen IS, Baltimore D (2003) Inhibiting HIV-1 infection in human T cells by lentiviral-mediated delivery of small interfering RNA against CCR5, *Proc. Natl. Acad. Sci. USA* 100, 183-8; Porteus M.H., Baltimore D. (2003) Chimeric nucleases stimulate gene targeting in human cells, *Science* 300, 763; Porteus M.H., Cathomen T., Weitzman M.D., Baltimore D. (2003) Efficient gene targeting mediated by adeno-associated virus and DNA double-strand breaks, *Mol. Cell. Biol.* 23, 3558-65; Meffert M.K., Chang J.M., Wiltgen B.J., Fanselow M.S., Baltimore D. (2003) NF- $\kappa$ B functions in synaptic signaling and behavior, *Nat. Neurosci.* 6, 1072-8; Klausner R.D., Fauci A.S., *et al.* (2003) Medicine. The need for a global HIV vaccine enterprise, *Science* 300, 2036-9; Hoffmann A., Leung T.H., Baltimore D. (2003) Genetic analysis of NF- $\kappa$ B/Rel transcription factors defines functional specificities, *Embo J.* 22, 5530-9; Brown E.J., Baltimore D. (2003) Essential and dispensable roles of ATR in cell cycle arrest and genome maintenance, *Genes Dev.* 17, 615-28; Antov A., Yang L., Vig M., Baltimore D.,

Van Parijs L. (2003) Essential role for STAT5 signaling in CD25+CD4+ regulatory T cell homeostasis and the maintenance of self-tolerance, *J. Immunol.* 171, 3435-41.