N O R M A N D A V I D S O N 1916 – 2002

A Biographical Memoir by HENRY A. LESTER AND AHMED ZEWAIL

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Biographical Memoirs, VOLUME 86

PUBLISHED 2005 BY THE NATIONAL ACADEMIES PRESS WASHINGTON, D.C.



Morman Davidson

NORMAN DAVIDSON

April 5, 1916–February 14, 2002

BY HENRY A. LESTER AND AHMED ZEWAIL

N ORMAN DAVIDSON WAS BORN in Chicago. He earned a bachelor's degree in chemistry at the University of Chicago in 1937 and completed another bachelor of science degree at the University of Oxford in 1939 as a Rhodes scholar. In 1941 he completed his Ph. D. in chemistry at the University of Chicago.

Before and during World War II, he worked on the problem of purifying plutonium for the Manhattan Project at the University of Southern California, at Columbia University, and finally at the University of Chicago. He also had a brief stint as a researcher at the Radio Corporation of America.

Norman Davidson's career as an independent scientist was entirely at the California Institute of Technology (Caltech) and covered the period from 1946, when he was appointed instructor in chemistry, to his death in 2002. Norman made important contributions sequentially in three quite different fields. From 1946 until about 1960 he worked in physical and inorganic chemistry. From about 1960 till about 1980 he was a founder of nucleic acid molecular biology; and from then until 2002 he made numerous contributions to molecular aspects of neuroscience. Norman was admired by his many students and colleagues. His students organized symposia for his sixtieth, seventieth, seventy-fifth, and eightieth birthdays. The format was simple: present one's own science. Norman sat in front, with his yellow pad, and took in every word.

He is survived by his wife, Annemarie Davidson, of Sierra Madre, California; by four children, Terry Davidson of Poway, California; Laureen Agee of Mammoth Lakes, California; Jeff Davidson of Cayucos, California; and Brian Davidson of Walnut Creek, California; and by eight grandchildren. Norman rarely used his middle name, Ralph.

CONTRIBUTION BY A. ZEWAIL

1946-1960: PHYSICAL CHEMISTRY

Norman Davidson made significant contributions to physical chemistry before he shifted his efforts to biophysical chemistry and to biology. Perhaps these contributions can be classified into two major areas. One is theoretical and involves the work on thermodynamics and statistical mechanics that culminated in his classic text book (1962), which was based on his course for first-year graduate students. The preface states, "The statistical mechanics of dilute systems of independent particles at equilibrium is a subject which is essentially fully developed. The practicing chemist should be able to apply this theory with assurance and accuracy to calculate the thermodynamic properties of substances in the ideal-gas state from molecular structure data." In 2003 this book received the accolade of republication as a Dover paperback edition.

The other area is experimental. Norman and his group were among the leaders in developing the shock-tube method for kinetics of reactions. Stimulated by the work in 1920 of

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Einstein on the dispersion of sound velocity, Norman studied the rate for the reaction $N_2O_4 \Leftrightarrow 2NO_2$. The work was scholarly and of highest quality, but Norman himself realized that the methodology needed to be advanced to one that is capable of better time resolution and cleaner chemical products.

It is not widely known that Norman was early in the development of flash photolysis and its applications. Flash photolysis began after World War II in 1949 at Cambridge University with the work of R. G. W. Norrish and G. Porter, who used intense flashes of light to create free radicals that could be studied spectroscopically. Together with G. Herzberg and D. A. Ramsay at the National Research Council of Canada, they used the method to study ClO, SO, CH₃, and others. In the process of developing flash photolysis around 1950, Norman tackled one of the most elementary yet complex reactions-the dissociation and recombination of iodine. This is fundamental to chemical change-how is a bond broken and reformed? Although the dissociation reaction involves only two atoms, the recombination was thought to involve "three body collisions." Norman found, from the kinetics, that the reaction occurs as a succession of twobody collisions, one between iodine atom (I) and iodine molecule (I_9) to form a relatively stable complex (I_9) , and a second between I₃ and I to form a pair of I₂ molecules. The kinetics in those days was of microsecond to millisecond resolution, and these elementary steps could not be resolved directly in real time. Forty years later Norman was pleased and thrilled that we were able to freeze in time I₃ complexes with femtosecond time resolution in femtochemistry experiments involving the collision of halogen atom and diatomic molecules. The lifetime of such complexes was short, tens of picoseconds, but Norman's earlier inference was, as usual, insightful and correct. Norman remarked to H.A L., "If I knew such experiments were on the horizon at Caltech, I would have stayed in chemistry."

TRANSITION TO MOLECULAR BIOLOGY

Norman's work from 1946 to 1960, he wrote, "is completely unrelated to molecular biology, but it resulted in my being elected to membership in the National Academy of Sciences in 1960. This kudo was very useful in my promotion at Caltech and my independence to shift fields from time to time" (2002,2). Indeed, Norman's audacity in switching to new fields played a large role in his ability to influence science so broadly and through so many young colleagues.

Norman was influenced by Linus Pauling, who directed Caltech's Chemistry Division during Norman's early years on the faculty. Pauling believed that chemists could make fundamental contributions to biology, and in 1951 he defined the α -helix and the β -sheet. Pauling attempted to solve the structure of DNA; and soon after the 1952 publication of Watson and Crick's model, J. D. Watson spent a year at Caltech. During the 1950s, other founders of molecular genetic biology who worked at Caltech included Howard Temin, Renato Dulbecco, John Cairns, Alex Rich, Jerome Vinograd, Robert Sinsheimer, and Max Delbruck. The Meselson-Stahl experiment, published in 1958, was conducted just down the hall from Norman's office.

Davidson believed that one contributed to molecular biology primarily through published papers and through excellent students. He wrote very few review papers and no textbooks in this field, in contrast to physical chemistry, where a textbook can be timely even after 40 years! However, in late 2001 he felt it appropriate to sum up his work since about 1960 with a prefatory chapter in the *Annual Review of Biochemistry* (2002,2). Published after his death, the chapter provides Norman's own clear views about the work that he considered most noteworthy.

Norman wrote of his transition to molecular biology as follows:

Some time around 1958 or 1959, I was thinking about switching to biologyrelated research. . . . I learned that ion channels were selective for either sodium ions or for potassium ions. This fascinated me because I knew from my undergraduate analytical chemistry course how difficult this separation was. . . . [I told] Bernard Katz about my interest in doing something chemical about ion channels. He advised me to forget about it because the density of ion channels in the squid axon was only about 1 per μm^2 , and it would be impossible to isolate a sufficient quantity to do anything chemical. He was of course right because before recombinant DNA and cloning came along it was not possible to do anything other than electrophysiological studies. . . . I decided that the field most suitable for biochemical studies was DNA. (2002,2)

NORMAN'S STUDIES ON DNA

With his students William Dove and James Wetmur, Norman developed fundamental facts about the effects of ionic strength and divalent cations on DNA hybridization and denaturation (1962, 1968). These ideas are still in use today, providing the foundation for hybridization-based phenomena such as Northern and Southern blots. With Tetsuo Yamane, Norman used his chemistry background to exploit the Hg⁺ ion as a probe for isolating characterizing DNA.

James Wang and Norman developed the chemistry and biology of closed circular DNA (1966). With Ronald W. Davis, Norman helped to develop electron microscopy as a technique for visualizing regions of single- and doublestranded DNA (1968) This team was the first to physically map a mutant genome (i.e., a deletion of the phage lambda). There followed a decade when electron microscopy was the dominant technique for high-resolution studies of nucleic acid interactions. Philip Sharp, who joined Norman's lab in 1971, studied details of the antibiotic resistance factors and their interactions with host chromosomes. They discovered that insertion sequences, which were usually of length 1-4.5 kb (and this abbreviation was introduced by Norman Davidson), contained a palindromic sequence at each end, flanking the genes for transposition (1972). Madeline Wu and Norman developed a way to employ antibodies to localize protein-DNA binding sites in the electron microscope. "We used a chemical method to attach the hapten dinitrophenyl to the protein that was attached to the DNA (for example, the protein that was bound to the two ends of adenovirus-2 DNA). By then adding an antibody to dinitrophenyl and, if necessary, a second antibody, we could observe the protein at each end" (2002,2).

STUDIES ON RNA

Madeline Wu and Norman also developed ferritin labeling to visualize tRNA molecules. Beginning in 1972 he concentrated increasingly on RNA, especially the retroviruses. He and Welcome Bender, working with SV40, used the poly(A) tail to map the 3' end of the mRNA molecule.

Norman understood that studies of cDNA derived from mRNA were an appropriate way to assess complexity in a genome. He settled on *Drosophila* as a model system, and he showed his usual excellent scientific taste in devoting his efforts to molecules and topics that remain important to this day. With Eric Fyrberg, Norman cloned all six *Drosophila* actin genes and found homologies to both *Dictyostelium* (which served as the original probe) and vertebrate cytoskeletal actins (1976). Ronald L. Davis came to Norman's lab to study the *Drosophila dunce* gene, which encodes a cAMP phosphodiesterase. Norman and Davis cloned and sequenced this gene and discovered some aspects of its alternative splicing.

Norman played a role in recruiting Eric Davidson (no relation) to Caltech in 1971. Eric Davidson continued to study the arrangement of DNA sequences, sequence complexity, and selective transcription throughout the 1970s. These experiments interacted with Norman's developments in the fields of nucleic acid hybridization kinetics and electron microscopy. Norman and Eric had a close intellectual relationship.

CONTRIBUTION BY H. A. LESTER

NEUROSCIENCE

Norman wrote, "[About 1980] I felt that it was a good time to go back to my earlier interest in neurobiology. *Drosophila* was not a good organism for this work because its neurons are too small for patch clamping, which was a highly developed skill for vertebrate cells. My colleague in the Biology Division at Caltech, Dr. Henry Lester, was interested in learning molecular biology, so we teamed up and collaborated up to the present" (2002,2). Norman and I published our first joint paper in 1985, when Norman was 69 years old; it was Norman's 291st paper. The group eventually published a total of 93 papers together. Cesar Labarca joined the Caltech group in 1986 and played a key role in the DNA manipulations of many of these studies.

ION CHANNELS AND RECEPTORS

Davidson believed that cloning genes for ion channels would open up new vistas for neuroscience; so the Caltech group began studying cDNA clones for the classically defined ion channels, the nicotinic acetylcholine receptors, and sodium channels, the latter in collaboration with William Catterall at Seattle and Robert Dunn in Toronto. In the 1980s the group conducted several studies that first isolated these clones, then used *Xenopus* oocytes (after the key publication by Eric Barnard and Ricardo Miledi) and mammalian cells to express the functional channels. The expression systems, which are still used in many labs today, had several roles. First, one wished to prove function. Second, one wished to verify that one had all the clones for certain multi-subunit proteins. And one also conducted many mutagenesis studies to define important functional roles for individual amino acids.

Early colleagues on those studies included Mike White, Alan Goldin, Reid Leonard, Lei Yu, Pierre Charnet, and Doug Krafte. Norman's skills at DNA manipulations and his delicacy with RNA were vital to the experiments, which occurred before the days of molecular kits and PCR. There were spirited competitions with the lab of Shosaku Numa in Kyoto, especially as Numa teamed up with Bert Sakmann in Göttingen to perform physiological studies on the sitedirected mutants. The Caltech group was usually the runnerup in those races. Norman was particularly pleased with the physical chemical elegance of a study that defined the permeation pathway of the nicotinic receptor by manipulating the millisecond interruptions that occurred when the openchannel blocker, QX-222, bound (1988). In Na channels Norman played a key role in the discovery that single amino acid changes could dramatically affect such functional properties as voltage dependence and inactivation (1990,1). Terry Snutch joined the group to contribute particularly on the diversity of calcium channel genes, as did Nathan Dascal, Joel Nargeot, and John Leonard (1990,3).

Ion channels were in the air elsewhere at Caltech. Seymour Benzer's *Drosophila* lab had previously worked on the *shaker* mutant and generated evidence that it was a K⁺ channel. After Benzer's former students Mark Tanouye (then on the Caltech faculty) and especially Lily and Yuh Nung Jan (at the University of California, San Francisco) had separately cloned this K⁺ channel, Norman's group conducted several structure-function studies, in collaboration with Tanouye and separately. Later, in the 1990s, Norman's group worked with Kai Zinn and his student John Bradley to clone, express, and study cyclic nucleotide-gated channels.

Norman was particularly taken with the idea of cloning by functional expression, which used his skills at nucleic acids to the fullest. In 1987 Norman's postdoctoral fellow Hermann Lübbert used antisense suppression to find a partial cDNA clone for the receptor now termed serotonin 5-HT2C, in collaboration with Paul Hartig and Beth Hoffman. Upon reading of the Caltech partial clone (1987), Richard Axel at Columbia promptly sent Norman a bottle of champagne. Then David Julius and Axel went on, using an even better expression cloning technique, to find the entire functional cDNA. This was probably the second G protein-coupled receptor cloned (the first having been found by Rich Dixon, Brian Kobilka, Marc Caron, Bob Lefkowitz, Cathy Strader, and their colleagues at Merck and Duke). Norman's appetite for the G protein pathway was thus whetted, and Mel Simon's lab at Caltech, which cloned many of the G protein subunits, became active collaborators.

In 1992 Nathan Dascal from Tel Aviv University and Wolfgang Schreibmayer from the University of Graz arrived as sabbatical visitors and set about expression cloning the gene for the cardiac G protein-activated inwardly rectifying K⁺ channel. Roughly at the same time, the Jans at the University of California, San Francisco, and the Caltech group found the gene, now termed GIRK1 or Kir3.1 (1993). For several years after that the Caltech group studied the functional activation of this channel. The questions revolved around activation by the G α subunits vs. the G $\beta\gamma$ subunits. The availability of expression systems enabled several labs to determine that the major activation occurred via the G $\beta\gamma$ subunits. However, the α subunits also clearly played a role, and these interactions are not yet settled. Paulo Kofuji and Craig Doupnik joined these studies and performed elegant experiments on the role of the regulators of G protein signaling (RGS) proteins, which nicely tuned up the kinetics of the Gi-coupled pathway (1997).

NEUROTRANSMITTER TRANSPORTERS

John Guastella joined the lab in 1988 to take a rather new direction: neurotransmitter transporters. The collaboration included Baruch Kanner of the Hebrew University, who had obtained a partial sequence for the GABA transporter. Norman designed degenerate oligonucleotide probes, and John isolated a cDNA clone, designated GAT-1, which in Xenopus oocytes caused the uptake of [³H]GABA (1990,2). Shortly afterward Susan Amara and coworkers used an expression strategy to clone a noradrenaline transporter. The substantial regions of sequence homology between these two transporters then allowed many investigators to clone additional transporters.

As usual, we were fascinated by the opportunity that an isolated expressible clone provided for functional studies, so between 1992 and 1998 the Caltech group adapted voltageclamp techniques to dissect mechanistic details of the GABA and serotonin transporters. Excellent postdocs, including Sela Mager and Michael Quick, deduced turnover rates and substrate binding orders. They also made pioneering observations that GAT1 could be modulated via membrane trafficking. Eventually Chi-Sung Chiu made knock-in mice with GFP fusions to GAT1, and he counted GAT1 molecules

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using quantitative microscopy: There are about 1,000 GAT1 molecules per μ m² in a presynaptic nerve terminal (2002,1).

Beginning in the mid-1990s Norman saw clearly that X-ray crystallography would furnish the key answers to outstanding questions in ion channel and transporter function. He began to study overexpression for this purpose, while also engaging in the next phases of his career.

SYNAPTIC PLASTICITY

Erin Schuman came to Caltech in 1993. She had helped to show that nitric oxide as a second messenger could cause long-term potentiation (LTP). "I was fascinated by this. Neuronal nitric oxide synthase (nNOS) knock-out mice still expressed LTP. This suggested that endothelial NOS (eNOS), which despite its name was known to occur in the dendrites of hippocampal neurons, was the contributing enzyme for LTP. Furthermore, it had been shown that an inhibitor of myristoylation would inhibit LTP" (2002,2). Norman therefore engineered an adenovirus containing the signal sequence of CD8 (which occurs on the cell membrane of T cells) fused to the eNOS gene. With this gene, eNOS expression on the cell surface of neurons in rat hippocampal slices was not blocked by a myristoylation inhibitor, and tetanically induced LTP was expressed (1996).

Dr. Schuman's lab then helped to show that brain-derived neurotrophic factor (BDNF) could induce long-lasting enhancement of synaptic transmission. Norman, Erin, and I, with postdoctoral fellow Yong-Xin Li, followed up by studying the effects of BDNF on E18 neurons in culture. We showed that BDNF could enhance synaptic transmission between a synaptically connected cell pair. We made a dominant negative TrkB by deleting the intracellular portion of the gene and fusing GFP to the C terminus as a marker for expression. We then observed that only when the presynaptic cell was infected could we observe an inhibition of the BDNF enhancement of transmission. Thus, at least for short-term activation the effect of BDNF is presynaptic (1998).

Norman continued to study cAMP-dependent LTP, using organotypic cultures from E18 rats, with Tzu-Ping Yu. Surprisingly she observed LTD with a mixture of Sp-cAMPS and the GABA receptor inhibitor, picrotoxin. Norman was following up this observation until a week before his death.

A TYPICAL DAY WITH NORMAN, 1983-2002

A typical day for Norman started at about 7:15, with tennis at the Athenaeum (Caltech's faculty club). Norman kept in excellent physical shape with a combination of athletics and dietary discipline, until arthritis crippled him after the age of 80.

Later in the morning Norman enjoyed presiding at a "club" meeting. Norman knew how to run a sizeable research group; he termed our subgroups "clubs" and organized meetings every two weeks. There was the GIRK Club, the Culture Club, the Slice Club, and many others. The lab attracted a wonderful series of scientists, born in 26 different countries between the years 1920 and 1980.

At these meetings Norman discussed expression tactics to understand the molecules we and others around the world were discovering and studying. Norman loved gene transfer, and he experimented with all appropriate techniques as they appeared: simple transfection, vaccinia virus, adenovirus, Sindbis virus, lentivirus, and adeno-associated virus. Norman particularly enjoyed discussing antisense RNA, and he paid attention to the rapid advances in siRNA that occurred in the last three years of his life.

Early on, students and postdocs had desks in both labs, Norman's in the sub-basement of the Crellin building in Chemistry and mine on the third floor of the Kerckhoff

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building in Biology. In 1991 we confirmed this intellectual merger with a full physical merger, and Norman and I occupied adjoining offices on the third floor of Kerckhoff.

Norman's copious memos issued from beloved yellow memo pads. They were usually clipped to photocopied papers, annotated in two colors for emphasis, and often with a selfdeprecating heading such as "Enthusiasm of the moment dept." These memos sent some lab members on to a series of experiments that lasted only a week and were abandoned. Some of those experiments lasted a month and produced interesting data, or a year and were followed by a paper. But former colleagues report that a few of those memos led to an entire career of satisfying research.

Norman also attended Molecular Biology Lunch at the Athenaeum every Monday, and he kept the conversation focused on science. The yellow pads also came to seminars. Norman paid attention to every word and typically asked the most incisive question (in an appropriate memorial, the seminar room has been renamed Norman Davidson Hall). Norman and his yellow pad then dined with the speaker. Norman noticed the food only when it was unusually bad, and he again kept the conversation focused on science.

Norman's day was not yet finished. He usually had a phone conversation with a colleague. My children, who were born just when Norman and I started our partnership, simply expected him to phone at bedtime. When they became teenagers, we got phones for them, a phone for Margaret and me, and a phone for Norman.

Saturdays were half workdays for Norman, but Sunday nights were reserved for an excursion to a cinema and a modest restaurant. Norman and Annemarie took these excursions with several generations of young Caltech faculty, and Norman kept up with the latest ideas and trends during these excursions.

AMGEN

Norman was an original member of Amgen's scientific advisory board (in 1980), and he kept up contact with the company in Thousand Oaks, California, until just before his death. He was well regarded for excellent advice, explanations, and career guidance. Many papers from Amgen thank Norman for comments on the manuscript.

AWARDS AND LEADERSHIP POSITIONS

Davidson's awards included the Peter Debye Award by the American Chemical Society in 1971, the California Scientist of the Year in 1980, the Dickson Prize for Science in 1985, the Robert A. Welch Award in Chemistry in 1989, the National Medal of Science in 1996, and a McKnight Senior Investigator Award in Neuroscience (1997-1999). He was a member of the National Academy of Sciences for 42 years, a fellow of the American Academy of Arts and Science since 1984, and held an honorary doctorate from the University of Chicago.

Despite his primary commitment to bench research, Norman held key leadership positions. He served two terms as executive officer at Caltech, in the 1960s for the Division of Chemistry and in the 1990s for the Division of Biology. He also served as first chair of the faculty at Caltech in the 1960s and briefly as interim chair of the Division of Biology in 1989. On the national scene he was a founding member of the National Advisory Council to the Human Genome Institute.

WE THANK Annemarie Davidson, Judith Campbell, Eric Davidson, and Philip Sharp for help with this memoir.

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