NATIONAL ACADEMY OF SCIENCES

JOHN FRANKLIN ENDERS

1897—1985

A Biographical Memoir by

THOMAS H. WELLER AND FREDERICK C. ROBBINS

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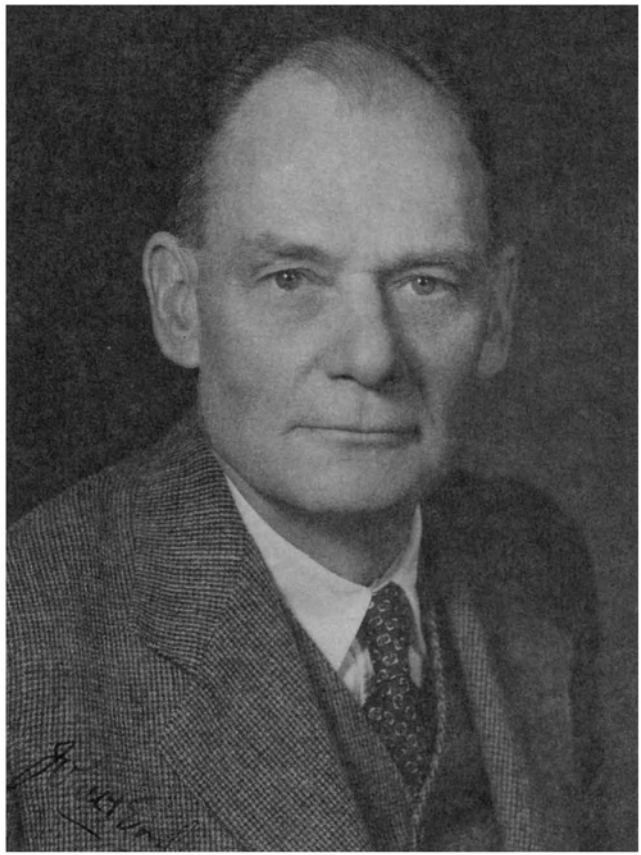

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John Flanders

JOHN FRANKLIN ENDERS

February 10, 1897–September 8, 1985

BY THOMAS H. WELLER
AND FREDERICK C. ROBBINS

THE INVESTIGATIVE CAREER of John Enders comprised three phases. For eight years he focused on pathogenic bacteria, in particular the pneumococcus. Switching in 1939 to the study of viruses, he refined tissue culture techniques for the study of viruses in vitro and made significant discoveries regarding mumps. This work prepared the way for the cultivation in 1949 of the polio viruses in non-nervous tissues, for which he was the corecipient of the 1954 Nobel Prize in Physiology or Medicine. Never one to rest on his laurels, Enders turned his focus to measles. This work led to the eventual production of a measles vaccine. By 1959, however, his research focus had shifted once again, this time to the problem of viral host-cell resistance and viral oncogenesis—the subject of the final segment of a magnificently productive investigative career.

EDUCATION AND EARLY CAREER

John Franklin Enders was born February 10, 1897, in West Hartford, Connecticut. His father headed the Hartford National Bank, and he was the first of four children in a family whose economic means were, as he once observed, "above average." Raised in a family whose business centered on finance and trade, he had little contact with science as a

boy. An uncle, a retired physician, who often visited the house, however, acquainted his nephew with certain aspects of science and medicine. The family also maintained contacts with luminaries in the field of literature, in particular handling the financial affairs of Mark Twain, whose spotless white suits impressed the young lad when the famous author visited.

Enders first attended the Noah Webster public grammar school in Hartford and in 1912 entered St. Paul's boarding school in Concord, New Hampshire, from which he graduated in 1915. Of the studies required, he wrote, "I preferred in the main certain of the so-called humanities—Latin, French, German, and English literature, although biological subjects always proved highly attractive. In mathematics and physics I encountered difficulties which were surmounted in a most mediocre fashion [and] only after great effort."[1]

In 1915, at the age of eighteen, he entered Yale University with no definite academic objectives. After two years there he enlisted in the Naval Reserve and learned to pilot aircraft. First as an ensign and then as a lieutenant, he served as a flight instructor at Pensacola, Florida, for three years. This experience influenced his mode of travel in later life which, if at all possible, was by train or boat—not by plane.

Free of economic pressures, Enders spent seven years after receiving his B.A. degree from Yale in 1920 seeking a suitable career. Starting with a real estate venture, he found business dull. He next considered a career as a teacher of English and moved from Yale to enroll in the Harvard Graduate School of Arts and Sciences. Receiving an M.A. degree in 1922, he spent the next three years exploring three different thesis topics in the field of philology, with little enthusiasm.

[1] Unpublished autobiographical note prepared in 1953 and now on file in the archives of the National Academy of Sciences.

MICROBIOLOGY AT HARVARD AND THE ZINSSER INFLUENCE (1925–1946)

At this juncture Enders was living in a boarding house in Brookline, Massachusetts, where he shared lodgings with several young Harvard medical students and instructors. Among them was Dr. Hugh Ward, an instructor in Hans Zinsser's Department of Bacteriology and Immunology. "We soon became friends," Enders wrote, "and thus I fell into the habit of going to the laboratory with him in the evening and watching him work. I became increasingly fascinated by the subject—which manifestly gave him so much pleasure and about which he talked with such enthusiasm—and so eventually decided to change the direction of my studies."[2]

It was Ward who introduced Enders to microbiology and to Hans Zinsser, the magnetically attractive, charismatic man who was a major influence in determining John Enders's career. In 1927 he married Sarah Frances Bennett. That same notable year he began a doctoral program in bacteriology under Hans Zinsser at the Harvard Medical School—the move that initiated his illustrious scientific career. In 1930 he completed the requirements for the Ph.D. degree in biology under Zinsser, presenting as his doctoral thesis evidence that bacterial anaphylaxis and hypersensitivity of the tuberculin type are distinct phenomena.

In a memorial tribute to Zinsser, Enders wrote movingly of life in his department. During staff lunches the conversation, led by Zinsser, would become animated. "Literature, politics, history, and science—all he discussed with spontaneity and without self-consciousness. Everything was illuminated by an apt allusion drawn from the most diverse

[2] John F. Enders, "Personal recollections of Dr. Hugh Ward," *Austral. J. Exp. Biol.* 41:(1963):381–84.

sources, or by a witty tale. Voltaire seemed just around the corner, and Laurence Sterne upon the stair. . . . Under such influences, the laboratory became much more than a place just to work and teach; it became a way of life."[3]

In this congenial environment Enders slowly progressed up the academic ladder. He was an instructor from 1930 to 1935 and an assistant professor from 1935 to 1942.

Late in 1939, Dr. Alto E. Feller and Thomas H. Weller (then a senior medical student) undertook a research project under Enders cultivating vaccinia virus in roller cultures of chicken tissues. Even then Enders had developed characteristic and well established patterns in the laboratory. He would arrive in the middle of the morning carrying a simple lunch. His first priority was always to review any new observations. Although at the time he had no technician and only rarely participated actively in work at the bench, he delighted in looking at cultures and analyzing new data and knew exactly what was going on in his laboratory.

In those days, too, resources were limited and his entire research budget amounted to two hundred dollars a year. His junior associates spent much of their time, therefore, washing, plugging, and sterilizing glassware. Yet such menial tasks did not seem a chore, for as they worked Enders would lead wide-ranging discussions in the Zinsserian tradition—heady and stimulating interactions for a fourth-year medical student. (Parenthetically, the personal magnetism so evident in interactions with small groups was not evident when he lectured to large groups. Though he found them an unpleasant obligation, Enders always crafted his lectures to medical students with great care, then delivered them in a soft, uninflected, and almost apologetic tone.)

[3] "Hans Zinsser in the Laboratory: Address by John F. Enders at the Memorial Service for Hans Zinsser, Harvard Medical School, October 8, 1940," *Harvard Medical Alumni Bulletin* 15(1940):13–15.

BOSTON CHILDREN'S MEDICAL CENTER (1947–1972)

In 1940 Hans Zinsser died, initiating a difficult period for Enders that was compounded by the unexpected death of his wife, Sarah Frances, from acute myocarditis in 1943. From 1940 to 1942 he served as interim head of the Department and in addition—with faculty departing during the war years—augmented his own teaching duties. In 1942 he was made associate professor and in 1943 his administrative duties, for which he had little liking, terminated with the appointment of his contemporary, Dr. J. Howard Mueller, as permanent chairman of the Department.

Enders could then expand his own research on mumps, and with additional funding from military sources, he was able for the first time to employ a personal technician and a succession of junior associates. Yet under Mueller, who began work at six in the morning, the lifestyle of the Department was not that of Enders.

In 1946 Dr. Charles A. Janeway and Dr. Sydney Farber asked Enders to establish a laboratory for research on infectious diseases at the Boston Children's Hospital. He accepted and in 1947 was allocated four rooms on the second floor of the long-vacant Carnegie Building. Thus began his long and productive association with the Children's Medical Center in Boston, where, until 1972, he was chief of the Research Division of Infectious Diseases. Thereafter, though his contact with Harvard Medical School was limited to one or two lectures a year, he was promoted to full professor in 1956 and named University Professor in 1962—a title he held until 1967, when he became University Professor Emeritus.

Enders's unique personal magnetism in the laboratory arose from the pleasure he took in discussions with his associates. Deeply interested in medical problems, he would make astute observations regarding different diagnoses of a

puzzling case. He had a remarkable capacity to identify and exploit significant findings and, in a low-key manner, could stimulate junior associates to further productive endeavors.

Casual visitors viewed Enders as a quiet, somewhat reticent individual of great personal charm. Stoop-shouldered, he moved slowly about the laboratory, usually with a pipe in his mouth. He had a sly sense of humor and the occasional facetious remark was accompanied by a half-grin and a twinkle in his eyes. Though he lived comfortably his personal tastes were simple, and his frugality in the laboratory became legendary.

Most observers thought Enders impervious to honors, but his true intimates recognized a highly competitive spirit behind his humble façade. Once he had obtained fully convincing scientific data, therefore, there were no delays in publication.

As Enders's reputation spread his laboratory became increasingly attractive, and though the number of associates he accepted remained small, he was an influential force in the training of a generation of virologists. In 1967 when a symposium was arranged in honor of his seventieth birthday, more than a hundred associates and assistants from all over the world attended.

THE PRIVATE MAN

In 1951, eight years after the death of Enders's first wife, with whom he had two children—John Ostrom Enders II (deceased 1982) and Sarah Enders Steffian—he married Carolyn B. Keane. Known to a host of friends as "Carol," she proved a constant source of support, participating socially and scientifically in the subsequent events of her husband's life.

An autobiographical note written in 1953 lists carpentry,

photography, and gardening among Enders's avocations, but his major nonscientific interests were fishing and playing the piano.[4] The family spent summers in the Enders's compound at Waterford, Connecticut, from which they launched powerboat outings on Long Island Sound in search of striped bass. Enders himself made a pilgrimage each summer to his brother's fishing club in New Brunswick. If they were successful, salmon packed in ice would arrive at the laboratory.

Playing the piano was for the most part a private matter for Enders, and his interests ranged from Bach to Joplin. One exception, however, was the annual Christmas party he held at his home for his laboratory staff, and which regularly concluded with Enders at the piano, accompanying Christmas carols.

SCIENTIFIC CONTRIBUTIONS

Pathogenic Bacteria and the Pneumococcus (1929–1937)

During the first segment of Enders's long and prolific career he focused on pathogenic bacteria and, in particular, the pneumococcus. Throughout the thirties he published eighteen papers—both alone and with various collaborators—that demonstrated the relationship between virulence and the capacity of encapsulated Type III pneumococci to grow at elevated temperatures. He (and, concurrently, Oswald T. Avery and Walter F. Goebel) identified a new form of pneumococcal polysaccharide as an acetyl polysaccharide. He obtained evidence that serum complement expedited phagocytosis of pneumococci. His final paper of this period appeared in 1937 and recorded that inactive mixtures of pneumococci and homologous antisera regained virulence on dilution.

[4] Unpublished biographical note by Enders prepared in 1953.

Rickettsiae, Tissue Cultures, and Viruses (1937–1947)

The second phase of Enders's research—dealing with viruses—began in 1937 when he accepted the young epidemiologist, William McD. Hammon, as a doctoral degree candidate. When an epizoötic disease developed in their local stock of kittens, Hammon and Enders began an investigation. They published their findings in a series of papers describing malignant panleucopenia of cats, which established a viral etiology, and described procedures for the immunization of cats.

At the same time Enders was investigating the kinetics of inactivation of herpes simplex virus by specific antisera and with Dr. Morris F. Shaffer developed an indicator system using counts of foci on the chorio-allantoic membrane of the developing chick embryo.

As Zinsser's illness progressed, Enders also assumed responsibility for ongoing research on rickettsial diseases. Together with Zinsser and Harry Plotz he grew typhus rickettsiae in tissue culture in sufficient quantities to produce a vaccine. Resolving the laboratory problem posed by the lack of susceptible experimental animals, he, Dr. P. T. Liu, and Dr. John C. Snyder showed that European typhus rickettsiae produce a fatal infection in irradiated white mice.

During this period Enders utilized roller tissue cultures to propagate several viruses. With Dr. Feller and Thomas Weller he reported, in 1940, the prolonged growth of vaccinia virus, and with Dr. Harold E. Pearson in 1941, of influenza A virus. Enders and Dr. Alfred L. Florman then investigated the influence of antiserum and complement on the growth of vaccinia virus in roller cultures and the persistence of this virus in Maitland-type cultures. A leading proponent of cultural techniques in the study of viruses, Enders contributed the definitive chapter on the propagation of viruses

and rickettsiae in tissue culture in the first (1948) edition of T. M. Rivers's classic *Viral and Rickettsial Infections of Man.*

Because mumps had been a major cause of days lost from duty in the armed forces during World War I, authorities in Washington at the beginning of World War II requested that Enders study the problem. Collaborating with Drs. Sydney Cohen and Lewis W. Kane, and with Mrs. Jeanette H. Levens, he began studying immunity in mumps.

At that time, although the viral etiology of mumps had been established using monkeys as the host, there were no diagnostic tests or techniques to assess susceptibility. Developing a complement fixation test that satisfied these deficiencies, Enders's group demonstrated that the intradermal injection of killed virus elicited a response in those previously infected. But their attempts to culture the virus and experiments directed at producing active or passive immunity proved less productive.

When, in 1945, Karl Habel reported the growth of mumps virus in embryonated hen's egg, Enders and Levens's confirmatory experiments showed the virus present in high titer in infected amniotic fluids, and that the infected fluids agglutinated erythrocytes. This meant that both infectivity and neutralizing antibody could be titrated.

In 1947 Enders's laboratory at the Children's Hospital became functional; Dr. Weller initiated virological studies; and in 1948 Dr. Frederick C. Robbins joined the group. Weller wanted to attempt growing varicella virus using cultures of human cells, but Enders suggested that he first try to develop a system to propagate a known agent: mumps virus. Slightly modifying the classical, Maitland-type culture, Weller maintained the tissue component for long periods by changing the nutrient fluids at three- to five-day intervals. In this way an egg-adapted strain of mumps virus was propagated in vitro for the first time and it was shown that viral replication

could be assayed by hemagglutinins elaborated in vitro. Jeanette Levens, under Enders's direction, showed that Influenza A virus behaved in a similar fashion.

Poliomyelitis Virus (1948–1952)

In March 1948, Weller attempted to isolate varicella virus in a comparable system using human embryonic skin and muscle tissue as the tissue phase. A few unused cultures were inoculated with Lansing strain poliomyelitis virus. After twenty days in culture and three changes of medium, intracerebral inoculation of the fluids into mice resulted in paralysis of all inoculated. Serial passage in vitro was readily accomplished. At Enders's suggestion, Robbins—who was interested in using tissue cultures to identify a viral etiology of infantile diarrhea—used cultures of intestinal tissue obtained at the autopsy of a premature human infant and obtained similar results.

The potential significance of these observations was such that the laboratory directed its subsequent principal efforts to the study of poliomyelitis. First it was determined that Type I poliomyelitis virus could be similarly cultivated and supported in vitro by completely differentiated, nonnervous tissue (human foreskin). It was also noted early that the polio viruses, when propagated in cultures, induced degenerative changes in the cells in which they grew. The virus could be detected, therefore, by both metabolic and morphologic changes—a phenomenon Enders termed the "cytopathic effect."

Armed with these observations the researchers were able to assay virus in vitro and assess the neutralizing capacity of antisera. Thus a tissue culture could replace the experimental animal—usually a monkey. In June 1949 Robbins used the culture system successfully to isolate polio viruses from patients and a number of nonpolio enteroviruses from clin-

ical cases of nonparalytic "polio." In the meantime Weller, concentrating on long-term propagation of the polio viruses, managed to obtain attenuated strains exhibiting decreased virulence. Dr. Arne Svedmyr and Ann Holloway, Enders's assistant, developed a complement fixation test for poliomyelitis using concentrated infected culture fluids as antigen.

In the decade following the war, Enders's spectacularly successful poliomyelitis team disbanded. In May 1952 Robbins left to accept a chair in pediatrics at Western Reserve University, while Weller departed in June 1954 to chair the Department of Tropical Public Health at Harvard.

The Measles Vaccine (1954–1960)

Though poliomyelitis was the primary focus of the research effort in Enders's laboratory between 1948 and 1952, the researchers also explored other illnesses of possible viral etiology. In 1950 Weller and Enders collaborated with Ms. M. Buckingham and Dr. J. J. Finn in a study that demonstrated that a Coxsackie virus was the etiologic agent of epidemic pleurodynia. With Dr. Franklin A. Neva, Enders described viruses isolated from patients with an unusual exanthematous illness—agents later classified as Echo 16 virus.

The measles segment of Enders's research began in 1954 when he suggested that Dr. Thomas C. Peebles, a research fellow, attempt to isolate the agent of this disease. In roller cultures of human kidney cells inoculated with acute-phase throat washings or blood from cases of rubeola, they observed unique changes with syncytial giant-cell formation. Serial passage was accomplished in cultures of human or monkey kidney cells. This cytopathogenicity was neutralized by convalescent-phase measles sera. The researchers found a complement-fixing measles antigen in harvested culture fluids.

With Peebles and Dr. Kevin McCarthy, a research fellow

from Liverpool, Enders explained the irregular results achieved by earlier investigators attempting to infect monkeys. Monkeys held for a period in captivity often showed serologic evidence of a prior spontaneous infection, but monkeys first proven seronegative who were inoculated with measles virus inevitably developed the disease.

In collaboration with Drs. Milan V. Milovanovic and Anna Mitus, Enders showed that cultures of human amnion cells supported growth of measles virus and that the virus could be propagated in chick embryos. Working with Dr. Samuel L. Katz, he showed that the egg-adapted virus could be grown in cultures of chicken cells. By 1958 Enders, Katz, and Dr. Donald N. Medearis had sound evidence that a strain thus propagated became attenuated and that monkeys inoculated with the attenuated strain produced an antibody response with no viremia or recognizable disease.

Enders immediately turned all the resources of his laboratory to the task of developing a measles vaccine based on the attenuated, avianized strain, and the results of their labors were published in a series of papers in the *New England Journal of Medicine*'s July 28, 1960 issue. Three significant papers derived from the work of Enders's laboratory. Parallel clinical trials were carried out in Denver, New Haven, Cleveland, and New York. The combined findings involved 303 vaccinated children. A mild, modified infection resulted from these vaccinations. The vaccine virus did not spread, and protection was induced. This classic group of papers provided the basis for studies that led to the licensing of the measles vaccine in 1963 in the United States.

About this time, Kevin McCarthy recovered measles virus from the lungs of a patient diagnosed as having Hecht's giant-cell pneumonia, and it was shown that the measles virus was the etiologic agent. Enders—in collaborative studies with Anna Mitus, Dr. William Cheatham, and others—recovered

rubeola virus from two other fatal cases of giant-cell pneumonia that had not exhibited clinical evidence of measles. The researchers subsequently observed that the rubeola virus persisted in the respiratory tracts of children with leukemia who had had measles to produce a giant-cell pneumonitis. They also obtained evidence that, in children with leukemia, measles vaccine might induce a chronic giant-cell pneumonia.

Though his cultivation of poliomyelitis viruses garnered Enders, Weller, and Robbins the 1954 Nobel Prize, Enders later wrote that this work on measles was more personally satisfying to him and more socially significant.

Virus and Host Cell: Interferon and Viral Oncogenesis (1959–1976)

By 1959 Enders's group had once again shifted the focus of their investigations, first to problems of viral host-cell resistance, then to viral oncogenesis—the subject of the final segment of Enders's investigative career.

Dr. Monto Ho observed that viral inhibitory substances were present in cultures infected with an avian-adapted Type II polio virus, substances later recognized as "interferon," as described by A. Issacs and J. Lindenmann in 1957. Enders, with Dr. Edward DeMaeyer, demonstrated that interferon was present in cultures infected with rubeola virus. He and Dr. Ion Gresser then showed that Sindbis virus also induced the production of interferon and that primary and established cultures of human amnion cells differed in their ability to produce interferon.

Although simian vacuolating viruses (SV_{40}) had been found in polio vaccines before Dr. Harvey M. Shein and Enders's 1962 report (*Proc. Soc. Exp. Biol. Med.* 109:495–500), evidence that SV_{40} would multiply in cultures of human cells was inconclusive. Enders obtained viral multiplication in cul-

tures of several human tissues with both cell degeneration and apparent stimulation of cell proliferation occurring. In three related papers Enders described the appearance of chromosomal abnormalities in transformed cells and showed that immunofluorescence demonstrated persistent virus in some transformed cells. Hamster renal cells transformed by SV_{40}, furthermore, produced adenocarcinomatous tumors when introduced into the cheek pouch of hamsters. Enders and Dr. Albert Sabin then showed that cells so transformed exhibited new, specific, SV_{40}-tumor complement-fixing antigens.

Continuing these SV_{40} studies, Dr. George Diamandopoulos and Enders found that—in contrast to earlier results—an apparent *absence* of the viral genome in the transformed cells was associated with an increased oncogenic potential when hamster lung and liver cells were exposed to SV_{40}. Cultured transformed hamster cells, when X-irradiated or exposed to colchicine—showed polynucleate giant-cell formation.

Enders and his associates next examined the thesis that cellular resistance to viral infection in vitro might reflect a barrier at the cell surface. With Dr. John M. Neff he showed that, though naturally resistant, cultures of hamster and chick embryo cells would support growth of the poliovirus if they were fused in the presence of Sendai virus killed by irradiation or beta-propriolactone.

The relationships binding virus and host cell were explored with Dr. George Miller (1969). Using human placental cells as a feeder layer, the two had established continuous cell lines of human leukocytes, which they co-cultivated with X-irradiated cells of an EBV-infected line of leukocytes.

At the age of eighty Enders retired from laboratory work but continued to follow the literature avidly. Ever clear-minded, he enjoyed discussions with scientific visitors to his

home to the end—as he had throughout the fifty years in his laboratory. On the evening of September 8, 1985, John Enders died quietly at his summer home in Waterford, Connecticut, as he sat reading T. S. Eliot aloud to his wife and daughter.

MATERIAL FOR THIS MEMOIR derived from several sources, including our personal files and autobiographical summaries prepared by Dr. Enders, one of which, a short summary prepared in 1953, is now on file in the Academy archives. Mrs. Carolyn Enders kindly provided additional material and access to bound volumes of Dr. Enders's publications.

MAJOR AWARDS AND DISTINCTIONS

1953 Election to National Academy of Sciences
1953 Passano Award
1954 Lasker Award
1954 Nobel Prize in Physiology or Medicine
1955 Charles V. Chapin Medal
1955 Gordon Wilson Medal
1961 *TIME* Man of the Year
1962 Robert Koch Médaille, Germany
1963 Presidential Medal of Freedom, United States
1967 Foreign Member, Royal Society of London
1981 Galen Medal of the Worshipful Society of Apothecaries, London.

Honorary doctoral degrees from thirteen universities.

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