D. Carleton Gajdusek

1923–2008

BIOGRAPHICAL *Memoirs*

A Biographical Memoir by
David M. Asher
with Michel B. A. Oldstone

NATIONAL ACADEMY OF SCIENCES

D. CARLTON **GAJDUSEK**

September 9, 1923–December 12, 2008

Elected to the NAS, 1974

By David M. Asher
with Michael B. A. Oldstone

Carleton Gajdusek, the brilliant and colorful polymath, the charismatic but controversial medical scientist, was found dead on December 12, 2008, at age eighty-five. He died in his hotel room in Tromsø, Norway, where he was guest scientist at the University of Tromsø with the Faculty of Health Sciences. His death was attributed to complications of severe atherosclerotic-hypertensive cardiovascular disease, with chronic cardiac and renal failure exacerbated by recent acute intestinal bleeding and a fall.

It is impossible to categorize or summarize in a few words the several areas of biomedical research to which Carleton contributed; the term "geographic medicine" might best describe his entire body of work, but his interests were even broader than his enormous accomplishments, which encompassed not only research in infectious diseases, for which he is best known, but also anthropology, linguistics, and Pacific art.

Carleton was elected to the National Academy of Sciences in 1974 and shared with Baruch Blumberg the Nobel Prize for Physiology or Medicine in 1976, in recognition of his pivotal research demonstrating that certain degenerative diseases of the human nervous system—now called transmissible spongiform encephalopathies (TSEs) or "prion diseases"—that were once thought to be idiopathic are in fact transmissible "slow" infections.

Looking back, I see Carleton's life as falling into four periods: (1) his early years, when he was an idiosyncratic, peripatetic, world-traveling young prodigy, seeking out and describing other interesting diseases little known in the United States, an undoubtedly productive but unfocused research physician, almost a dilettante; (2) the early years at the National Institutes of Health (NIH), when he accomplished the seminal work for which he gained fame by showing that both kuru and Creutzfeldt-Jakob disease were slow

Years later, visiting an exhibit of Pacific Art at the Smithsonian Institution in Washington, I was surprised (though I probably should not have been) to find that the largest object on display was the spectacular front section of a long house from Papua New Guinea that Carleton had managed to acquire and ship to the United States.

infections—a class of diseases previously unknown in human medicine; (3) the later NIH years, after he received the Nobel Prize and became a highly acclaimed scientific celebrity, a period that ended abruptly with his arrest and imprisonment; and (4) the years after his release from prison, a time that he called "exile" and that some friends have termed his "dark" or "King Lear" years, in which—although in declining health—he continued to write, travel widely, lecture, and seek out colleagues.

The early years

Daniel Carleton Gajdusek was born on September 9, 1923, in Yonkers, New York, to Karl Gajdusek, an immigrant from rural Slovakia who became a successful butcher, and Ottilia Dobroczki, the daughter of an immigrant family of literary bent that originated from Debrecen, Hungary.

An extended visit at age seven to his father's family in their Slovakian village may have awakened Carleton's fascination with rural life in out-of-the-way places. His mother's sister Irene, an entomologist at the Boyce Thompson Institute for Plant Research in Yonkers nurtured his interest in the sciences, as did William J. Youden, another scientist at the Institute with whom Carleton remained a close friend until Youden's death many years later. His Aunt Irene also introduced Carleton both to the mysteries of infectious diseases—insect virology—and, through her studies of geographic and economic entomology in the Pacific, she introduced him to the importance of maintaining a global view of science. His earliest work in the laboratory began with summer research at the Boyce Thompson Institute during his high school years.

Carleton also read widely. Paul de Kruif's books on infectious diseases and Nicholas Mikluho-Maclay's descriptions of life in New Guinea and other Pacific Islands were especially influential in forming Carleton's later interests.

His aunt Irene kindled in Carleton an enduring love of museums, starting with the American Museum of Natural History and the Metropolitan Museum in New York City. He went on to become a lover and avid collector of Pacific art, eventually donating many beautiful objects to the Peabody Museum of Salem, Massachusetts. In 1969, while I was

an exchange fellow in the Soviet Union, I had the impressive experience of visiting with Carleton the Museum of Ethnography in Leningrad, where he consulted on the probable origin of an object from Papua New Guinea (Sepik River, as I recall). Years later, visiting an exhibit of Pacific Art at the Smithsonian Institution in Washington, I was surprised (though I probably should not have been) to find that the largest object on display was the spectacular front section of a long house from Papua New Guinea that Carleton had managed to acquire and ship to the United States. (More restrictive attitudes towards export of national cultural treasures developed only later.)

He shared with his younger brother Robert a voracious appetite for cinema, sometimes in his later years seeing several films in a week, usually in the company of sympathetic friends who suffered through screening of avant-garde films that were accessible to Carleton, but few others.

Carleton entered the University of Rochester in his native New York State in 1940, studying mathematics, chemistry, and physics with luminaries who included Victor Weisskopf and Curt Stern, in preparation for a career in medical research that began in 1943, when he entered the Harvard Medical School. In addition to his medical studies at Harvard, Carleton worked with John Edsall, the protein physical chemist, and James Gamble, the pioneer in electrolyte balance, at the Boston Children's Hospital.

After receiving his medical degree in 1946, Carleton served internship and pediatric residency at Babies Hospital, Columbia Presbyterian Medical Center, in New York City, followed by an additional year of pediatric residency at Children's Hospital, Cincinnati, during which he participated in a medical mission to Germany. He went on to a post-doctoral fellowship lasting about a year at the California Institute of Technology, where he worked with Linus Pauling, John Kirkwood, and Max Delbrück, and was greatly influenced by young investigators Gunther Stent, Benoit Mandelbrot, George Beadle, and others.

Two more years of clinical and research fellowship at the Boston Children's Hospital, followed by two years as a research fellow in the laboratory of John Enders, who was then successfully propagating polioviruses in cell cultures, prepared Carleton for a career in infectious diseases. During his time with Enders in Boston, Carleton was drafted into the Army, leading to an assignment in 1952 to the Walter Reed Army Institute of Research (WRAIR), where he came to the attention of Joseph Smadel. Smadel, who went on to become deputy director of NIH, recognized the importance of Carleton's intuitions,

trusted his scientific judgment, and supported him enthusiastically when others were somewhat skeptical.

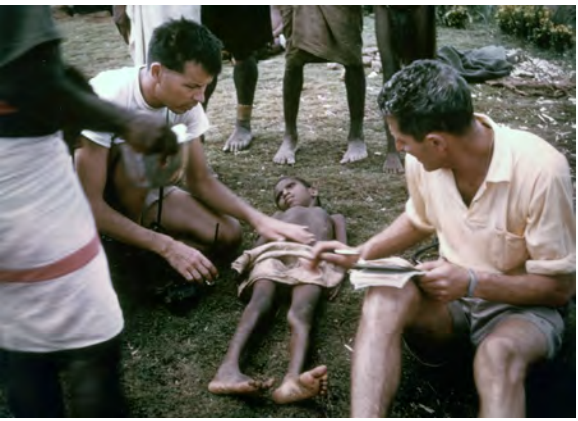
After leaving WRAIR in 1953, Carleton began a career in geographic infectious diseases in earnest, studying rabies, hemorrhagic fevers, and plague under the auspices of the Pasteur Institute in Tehran, research that led him to Afghanistan, Turkey, and ultimately to the Soviet Union, where hemorrhagic fevers were also a problem.

In Russia he was introduced to the mysterious Vilyuisk encephalitis, a chronic progressive disease of the brain in natives of the Yakut Autonomous Republic (now called Sakha) in Siberia, the cause of which remains unknown. He maintained his involvement in both Vilyuisk encephalitis and viral hemorrhagic fevers for the rest of his life.

One of Carleton's most unusual characteristics was the readiness with which he perceived, understood, and communicated newly emerging and important medical information. For example, based on observations and conversations during his travels, he was among the first US authorities to understand and describe in the medical literature Pneumocystis pneumonia in malnourished European babies, as well as the viral hemorrhagic fevers. When it became clear, many years later, that various rodents were reservoirs of hemorrhagic fevers in Europe and Asia, Carleton correctly predicted that similar viruses must also infect US rodents, and he successfully detected the first one—Prospect Hill Virus—in voles captured in his own Maryland backyard.

The years of Kuru and Creutzfeldt-Jakob disease

In 1955, perhaps during the pivotal moment of his scientific life, Carleton traveled to Australia to become visiting investigator in the Walter and Eliza Hall Institute of Medical Research, Melbourne, in the laboratory of MacFarlane Burnet, supported by the US National Foundation for Infantile Paralysis. He used the complement fixation technique to detect and characterize antibodies to antigens in the livers of people with hepatitis. The results, while creditable—finding low levels of antibodies to normal tissue antigens—failed to reveal a hepatitis virus. However, by chance, during the time Carleton was in Melbourne, samples of serum and a brain from patients with a puzzling progressive neurological disease arrived in the institute, sent by a local medical officer, Vincent Zigas, an ethnic Lithuanian and former Soviet citizen, but German-educated Baltic immigrant to Australia and self-styled "bush doctor," who suspected that the disease might be caused by some kind of infection. The samples had come from Fore



Gajdusek, left with Vincent Zigas examining a Fore child, 1957.

people in the Eastern Highland Region of Papua New Guinea, where they called the disease "kuru," meaning trembling.

The story of kuru, its first description in the medical literature by Carleton and Vincent Zigas in September 1957 and accounts of the friction between Carleton and Burnet, has been told several times. I was not involved then and cannot vouch for the accuracy of any individual account. I suppose that, taken together, they probably get the details more or less right. I do know that Carleton was both complimentary and grateful to Vin Zigas and other Australian colleagues (if not to Burnet, whom Carleton continued to respect greatly but who did not reciprocate until years later), and he was especially grateful to Fore participants in a study of kuru that he initiated in the spring of 1957 with Vin Zigas, leading to published descriptions of the disease in September of that year.

Carleton continued, with Vin, to consider, among other plausible etiologies, the possibility that kuru might be a previously unknown infectious disease. (They even conducted therapeutic trials with antibiotics.) But Carleton credited to William Hadlow, the American veterinary pathologist, the critical observation published in 1959 that kuru resembled scrapie, noting that scrapie was a slow infection spread among sheep and transmitted to goats, and that experimental transmission of scrapie required exposing animals of the same or closely related species to infected materials and observing the animals for a very long time before illness appeared. (Transmission of scrapie to rodents came only later.)

Strongly supported by Smadel, who had left WRAIR to occupy an influential position at NIH, Carleton was appointed chief of a new laboratory at NIH in 1958. NIH offered generous funding to extend Carleton's study of kuru and, later, of other subacute and chronic progressive neurological diseases, and it offered freedom to continue involvement in various other topics that interested him.

Smadel, recognizing Carleton's great scientific strengths but also a significant weakness (what we might now call a limited attention span), insisted that he appoint a deputy laboratory chief, Clarence Joseph Gibbs, Jr., a stay-at-home bench virologist who assumed responsibility for designing and managing most of the animal assays central to research in kuru and other TSEs of humans and animals. (Cell culture studies of TSEs Carleton entrusted to Nancy Rogers, but everything else—his anthropological and comparative cultural research—he managed himself.)

Gajdusek, approximately 1975.

Smadel's confidence proved justified: Carleton inoculated the first kuru brain suspension into the brain of a chimpanzee in February1963; inoculations with many other brain suspensions and other tissues from patients with kuru and other neurological diseases soon followed. Two chimpanzees became ill with progressive neurological disease in 1965. Histopathology of the two animals' brains revealed a remarkable similarity to that in brains of kuru patients, establishing that kuru, like scrapie, was an experimentally transmissible slow infection.

At first, Carleton's experimental transmission of kuru was received by the medical community as a fascinating example of research with an exotic disease that nobody was likely to encounter outside Papua New Guinea. However, Carleton suspected that was not the case. Igor Klatzo, a neuropathologist and collaborating investigator at NIH, had noticed early on that the pathology of kuru resembled that of Creutzfeldt-Jakob disease (CJD, called subacute spongiform encephalopathy in the United Kingdom). CJD, while relatively rare (about one diagnosed case per year in most surveys, but with an estimated lifetime risk of at least one in ten thousand) was well known to neurologists in the United States and other developed countries.

Another of Carleton's colleagues, Elisabeth Beck, a self-taught neuropathologist and expert in scrapie at the Institute of Neurology, Maudsley Hospital, in London, located a brain biopsy from a patient with CJD and sent a frozen sample to Carleton and Gibbs

at NIH. In 1966, shortly after my own arrival in the laboratory, Carleton and Gibbs inoculated a chimpanzee with that tissue; a year later the animal became ill with a TSE. Transmission of other cases of kuru and CJD were confirmed with chimpanzees, monkeys, and then other animals, establishing that TSEs (later including familial CJD, iatrogenic CJD, variant CJD, Gerstmann-Sträussler-Scheinker syndrome, and the fatal insomnia syndromes) affected humans throughout the world. The discovery led to a Nobel Prize for Carleton in 1976 and increased his scientific celebrity.

The Post-Nobel Prize years

Some recipients of the Nobel Prize may have successfully resisted the distraction of notoriety. Carleton did not, and he never tried. He reveled in every minute of his hard-won fame, spending less and less time involved with the every-day running of his research group at NIH, though he continued to provide overall direction and inspiration. He introduced new research topics in hemorrhagic fevers and retroviral illnesses, though those never approached the importance of his work with the TSEs. During the next twenty years he continued to travel and speak widely, received many other honors, and especially relished the accompanying "lavish meals" (Carleton's words) with ample wine and adulation. His curriculum vitae lists more than one hundred thirty honorary degrees, awards, plenary lectures, and other prestigious invited presentations in twenty-seven countries between the Nobel lecture he delivered in December 1976 and the spring of 1996, not to mention what must have been an even greater number of less formal professional events.

His colleagues became increasingly concerned about Carleton's surrender to surfeit, that he was harming his health. Gibbs, who was especially worried by our chief's rapidly increasing corpulence, arranged a serious heart-to-heart talk, suggesting that Carleton attempt to moderate his self-indulgence; Carleton replied curtly that the pleasures of trencher and vine were worth the risk of shortening life a little. With a brief interruption, Carleton continued to practice dietary excess for the rest of his life, but he nonetheless outlived Gibbs, his more abstemious colleague, by almost seven years.

It is impossible to attempt even a greatly reduced account of Carleton's life without commenting briefly on his unusual household: the large surrogate family he assembled by fostering children he met during working visits to Papua New Guinea and to other islands of Melanesia and Micronesia. Many of the children lived with him in the United States, a total of some twenty-nine boys and two girls, plus a number of others he helped in Papua New Guinea. He claimed to be not at all motivated by altruism (though he

did fund their education and even received an award for humanitarianism in 1987), but solely by the enjoyment of their company.

Clearly they provided him with companionship and also fulfilled his constant need for an attentive and admiring audience—a striking characteristic of Carleton's personality. His peculiar extended family and the destiny of several of the children have been described at least twice in scholarly literature, as well as in detailed journals that Carleton kept until the very end of his life. But there was also something darker (and illegal) going on in the household.

Last years

In early April of 1996, returning from a trip to Europe, Carleton was arrested at his home in Maryland and charged with sexually abusing a teen-aged boy who had lived with him and his other foster or adopted children several years earlier. (The story was, of course, immediately and widely covered in both local and national press; any Internet search engine can retrieve the details, and I will not repeat them.) Three devoted friends and colleagues put up considerable money for Carleton's bail. Carleton never returned to NIH, where he had worked for more than thirty-five years, signing a retirement request that Gibbs brought him the day before he pled guilty in February 1997.

At the end of April 1997, Carleton agreed to a sentence of up to thirty years in prison, with all but eighteen months suspended on the condition that he remain under state supervision for five years after release or, to avoid supervision, he leave the United States. The sentence was later reduced to one year of incarceration. On April 29, 1998, the same day he left the Frederick County Adult Detention Center, Carleton flew from Washington Dulles International Airport to Paris on an Air France plane, to avoid using a US carrier. He spent the rest of his life abroad.

While fully recognizing that he had broken laws of Maryland and offended accepted standards of behavior in the United States, Carleton emphatically and repeatedly rejected our norms as prudish and vociferously defended his behavior as natural, both historically and by the more liberal standards of many other countries today. While not persuaded that his personal behavior was acceptable, at least not in the United States—a country that generously supported Carleton's research, repeatedly rewarded his scientific accomplishments, and indulged his whims, but whose laws he flouted—I sadly came to agree with his friend Robert Gallo, who concluded simply that we should continue to be Carleton's "friend[s] and not his judge."

Whatever the details of Carleton's relationships with various members of his extended family were, it must be acknowledged that many of them continued to treat him with respect and affection for the rest of his life. Several of them adopted his family name and named their own children after him. Paul Brown, a close long-time colleague, recently inspected the final volume of Carleton's personal journal and reports[1] that the very last entries, probably written in Tromsø the day Carleton died, dealt with continuing the financial support for two of his boys, who were then grown men living far from Norway.

While he never again directed research and never returned to either the United States or the Pacific Islands he loved, Carleton was not ostracized in retirement and remained much sought after as an honored visiting scientist in a number of institutions in Europe and Asia. He divided most of his time among three: the Alfred Fessard Institute of Neurobiology, National Center for Scientific Research in Gif-sur-Yvette near Paris; the University of Amsterdam Academic Medical Center; and the University of Tromsø. He was visiting scientist in China at Sichuan University, the University of Petroleum, and institutions in Guangzhou, Guangxi, Shanghai, Beijing, and Shandong. He was also a guest in many other prominent academic institutions; his CV reveals that from 1998 until 2002 he visited eleven other countries in addition to the four noted above: Estonia, Germany, Greece, Hungary, Ireland, Israel, Italy, Russia, Slovakia, Spain, and Sweden.

Although he could no longer visit the Pacific, some of Carleton's extended family and former coworkers, including several from Papua New Guinea, managed to meet him in Europe, and many of us joined him in a festive eightieth birthday celebration on an estate outside Bologna in September 2003.

I last saw Carleton alive at a special meeting convened at the Royal Society in London in October 2007 to mark the fiftieth anniversary of his description of kuru and the gradual elimination of kuru after cannibalism ceased among the Fore people. Carleton, unable to walk for more than a few steps or to stand unassisted, but dynamic as always, expounded at length and forcefully about the origins and end of kuru and the lessons it provides for other diseases. My last encounter was to be in June 2009 at a memorial ceremony on a boat in the Tromsø Fjord, where a crowd of friends scattered Carleton's ashes.

Carleton's famous extended monologues were fascinating at first, and probably remained so to the many new admirers he continued to recruit over his last years. But they became increasingly tedious to his long-time colleagues as their novelty waned and he grew older, less organized, and more gratuitously unrestrained and obscene in speech. He disliked

1 P. Brown (see item 3 in the appendix).

hearing any kind of disagreement with his forceful analyses, but, throughout his life, he especially resented being told that something could not be done. That impatience, which undoubtedly contributed to his scientific success, encompassed issues great and small. The same furious energy Carleton applied to convincing NIH administrators to fund his ambitious research programs he focused one evening on a poor airline clerk who tried to inform our mutual friend, David Lang, an infectious-disease pediatrician and my mentor in pediatric residency, that it was too late in the day to fly from Vienna to Budapest. Carleton, having gone to the Vienna airport see Lang off, would have none of it and became so engaged in successfully proving the clerk wrong that he decided on the spot to fly to Budapest himself.

Carleton was equally impatient with his staff. He was absolutely scathing towards people of ordinary imagination and particularly those of us whom he considered overly cautious ("milquetoasts!"—Carleton's preferred spelling—he thundered) or who failed to pay sufficient attention to detail as he demanded. ("My goon staff has done it again!") His anger was often justified, but painful for us nonetheless.

On the other hand, especially in his earlier years, he could be surprisingly tolerant of our foibles and helpful when the situation arose. I remember with special fondness his short visit to the Soviet Union in the late spring of 1969. Having just completed two and a half years in Gibbs's section of Carleton's laboratory the previous winter, I was then working in the Ivanovsky Institute of Virology, Moscow, as an NIH fellow and member of the US-USSR Health Exchange—an appointment that Carleton had organized for me. Carleton arrived for a whirlwind tour of virology programs in Moscow and Leningrad, during which academician Boris Lapin invited us to visit the Primate Research Center in Sukhumi, a pleasant beach town in the "autonomous" region of Abkhasia, then in the Soviet Republic of Georgia (and now subject of an ongoing sovereignty dispute between Georgia and Russia).

Lapin was kind enough to arrange a short self-driving tour for Carleton from Sukhumi to Tblisi and further, through a corner of Azerbaijan to Erevan, Armenia. I was to share the driving. The vehicle we rented was a sturdy but clumsy Soviet Volga sedan with a stiff three-speed shifter on the steering column, and I was inexperienced with manual transmissions. The road through the Caucasus was narrow, twisting, rough, and hilly in places—no, it was mountainous. During my first inept turn at the wheel, it soon became clear that on steep declines, I was likely to burn out the brakes, the clutch, or both. Carleton might easily have insisted that he take over the driving, but he didn't. Instead

he encouraged me to persist and gave me a long tutorial on mountain driving that has served me since. Even after Carleton's lesson, I never became a skillful mountain driver, but I have yet to burn out a clutch or brakes. (On the same trip, Carleton's customary refusal to take "no" for an answer led us to attempt an unauthorized visit to an off-limits Armenian church claimed to be of historical importance, and we were promptly arrested. If police archives of Soviet Armenia survive, they preserve our jointly signed confession to a violation of USSR tourist laws.)

One of my most painful experiences after leaving the NIH has been responding to repeated expressions of sympathy from people who confide that it must have been terrible to work for Carleton, one even suggesting that I excise him from my CV. The NIH administration apparently did "air-brush" Carleton out of its public ceremonial history, offering none of the official recognition customarily accorded an NIH Nobel laureate after death. Carleton's final year at the NIH, as a criminal investigation proceeded, was indeed very unpleasant for everyone in his laboratory.

But I cannot conclude that most of my experience in Carleton's group was, on balance, bad. I believe that my time with Carleton and Gibbs—oddly mismatched personalities but effective scientific partners—ultimately enabled me to achieve my current position as chief of a small laboratory in the FDA, a modest but satisfying position that I have occupied for almost eighteen years and that would probably have been inaccessible to me had I followed the more traditional career path for a young pediatrician in the 1960s and not worked for Carleton. I believe that the same thing is also true, to some degree, for many investigators who trained or collaborated with Carleton—Michael Alpers, Elisabeth Beck, Paul Brown, Francoise Cathala, Larisa Cervenakova, Leon Epstein, Judith Farquar, Ralph Garruto, Joe Gibbs, Lev Goldfarb, Dmitry Goldgaber, Jaap Goudsmit, Robert Klitzman, Pawel Liberski, Colin Masters, Mark Miller, Pedro Piccardo, Mauricio Pocchiari, Robert Rohwer, Raymond Roos, Jiri Safar, Vin Zigas, and others. Our careers flourished, at least in part, because we had worked with Carleton.

Some aspects of the TSEs remain controversial even now. The puzzling nature of the self-replicating agents and the unknown origins of sporadic CJD and atypical forms of bovine spongiform encephalopathy and scrapie come to mind. But the validity of Carleton's major scientific contributions endure: the recognition that the human spongiform encephalopathies are infectious diseases and that their transmission can often be prevented by avoiding close contact with the agents. Daniel Carleton Gajdusek—the

fascinating, exasperating, and often bizarre but a brilliant and incredibly energetic and productive scientist—left those important discoveries to the world.

Reminiscences by Michael B. A. Oldstone

Idid not train with Carleton Gajdusek, but I retain a number of vivid memories of him. The first occurred when I was a first-year medical student at the University of Maryland. Carleton came on a Saturday morning to present one of the lectures on Frontiers of Medicine and Biology. His talk was about kuru and the Fore people of Papua New Guinea. He accompanied his talk with two slide projectors and a 16 mm movie camera, and the coordination of these instruments with his lecture was amazing, but not uncommon for him. Over the next several years I saw and heard him give updated presentations with the same video accompaniment.

During the summer following my first year in medical school, I was given the opportunity of work in the late Joe Smadel's laboratory at the Walter Reed Army Institute of Research. On occasion, Smadel and Gajdusek took me to lunch, and at times I got to visit Gibbs, Carleton, and their subhuman primate colony, where they were studying transmission of chronic neurologic diseases. Gibbs was careful, fastidious, and always with his notebook, while Gajdusek provided the emotion and advertising of the project under way.

As a young assistant professor at the Scripps Clinic and Research Foundation (later The Scripps Research Institute) in La Jolla, California, working on immune complex disease and discovering that antiviral antibodies previously not seen in persistent lymphocytic choriomeningitis and murine retrovirus infections of mice did actually occur, Carleton stopped several times to see me on his way back from New Guinea, bringing kidney tissues from kuru-infected persons so that I could look for immune complexes and thus seek antibodies specific to kuru. He usually spent the night at my house and we were visited until late hours of the night by senior faculty from the Salk Institute, who wanted to meet with Carleton. The net result was an exceptional salon for discussion of science, evolution, and infectious diseases. It was at these times that Carleton also preached to me the necessity for a totally independent career and obtaining the finances to support that career.

One of the last times I interacted with Carleton was when we were both invited to provide inaugural lectures at the dedication of the new medical school in Duluth, Minnesota. Our lectures were in the morning and strictly timed to be over by 12:30 pm,

when the Governor of Minnesota and his entourage were to arrive for lunch. Carleton spoke after me. Our talks were in a grand auditorium that also served, I believe, as a theater building. Despite exceeding his time and with continuous flashing of red lights on the podium, Carleton continued to speak, utilizing the slide and movie projectors. In desperation and just before 12:30, a member of the medical school committee threw a switch and the stage that Carleton occupied began to descend to the lower level. As I clearly remember, Carleton continued his talk as he slowly disappeared from view.

Many other sources summarize various aspects of the professional and personal life of D.C. Gajdusek. Here are several:

Alpers, M. P. (2010). Daniel Carleton Gajdusek, 1923–2008: an appreciation of his life and his love for Papua New Guinea. *PNG Med J* 53:54–64.

Asher, D. M. (2008). Kuru: memories of the NIH years. *Philos. Trans. R. Soc. B.* 363:3618–3625.

Brown, P. (2013). Transmissible spongiform encephalopathy: from its beginnings to Daniel Carleton Gajdusek, in *Prions and Diseases: Vol. 1. Physiology and Pathophysiology,* edited by Wien-Quan Zou and Pierluigi Gambetti. New York: Springer Science+Business Media, 1–19.

Gajdusek, D. C. (1976) D. Carleton Gajdusek - Autobiography. The Official Web Site of the Nobel Prize (http://www.nobelprize.org/nobel_prizes/medicine/laureates/1976/gajdusek-autobio.html) accessed April 12, 2013.

Masters, C. L. (2009) Ed. *Proceedings of a Celebratory Meeting on the Life and Legacy of D. Carleton Gajdusek by his Australian Friends and Colleagues*. Walter and Eliza Hall Institute of Medical Research, 24 Nov 2009. (A copy of these proceedings and primary archival materials related to D. Carleton Gajdusek are held by the Mandeville Special Collections Library, University of California at San Diego.)

Spark, C. (2007). Family man: the Papua New Guinean children of D. Carleton Gajdusek. *Oceania* 77:355–369.

Spark, C. (2009). Carleton's kids: the Papua New Guinean children of D. Carleton Gajdusek. *J. Pacific History* 44:1–19.

SELECTED BIBLIOGRAPHY

1952 With M. L. Robbins and F. C. Robbins. Diagnosis of herpes simplex infections by the complement fixation test. *JAMA* 149:235–240.

1956 Das epidemische hämorrhagische Fieber. [Epidemic hemorrhagic fever]. *Klin Wochenschr* 34:769–777.

1957 Pneumocystis carinii: etiologic agent of interstitial plasma cell pneumonia of premature and young infants. *Pediatrics* 19:543–565.

With V. Zigas. Degenerative disease of the central nervous system in New Guinea; the epidemic occurrence of kuru in the native population. *N. Engl. J. Med*. 257:974–978.

1959 With I. Klatzo and V. Zigas. Pathology of kuru. *Lab. Invest.* 8:799–847.

1964 The composition of musics for man or decoding from primitive cultures the scores for human behavior. *Pediatrics* 34:84–91.

1966 Wirth C. J. Gibbs and M. Alpers. Experimental transmission of a Kuru-like syndrome to chimpanzees. *Nature* 209:794–796.

With E. Beck, P.M. Daniel, M. Alpers, and C.J. Gibbs, Jr. Experimental "kuru" in chimpanzees. A pathological report. *Lancet* 2:1056–1059.

1967 With C. J. Gibbs, Jr., and M. Alpers. Transmission and passage of experimenal "kuru" to chimpanzees. *Science* 155:212–214.

1968 With C. J. Gibbs, Jr., D. M. Asher, and E. David. Transmission of experimental kuru to the spider monkey (Ateles geoffreyi). *Science* 162:693–694.

With C. J. Gibbs, Jr., D. M. Asher, M .P. Alpers, E. Beck, P. M. Daniel, and W. B. Matthews. Creutzfeldt-Jakob disease (spongiform encephalopathy): transmission to the chimpanzee. *Science* 161:388–389.

1969 With E. Beck, P. M. Daniel, W. B. Matthews, D. L. Stevens, M. P. Alpers, D. M. Asher, and C. J. Gibbs, Jr. Creutzfeldt-Jakob disease. The neuropathology of a transmission experiment. *Brain* 92:699–716.

1972 With C. J. Gibbs, Jr. Transmission of kuru from man to rhesus monkey (Macaca mulatta) 8 and one-half years after inoculation. *Nature* 240:351.

With C. J. Gibbs, Jr., N.G. Rogers, M. Basnight, and J. Hooks. Persistence of viruses of kuru and Creutzfeldt-Jakob disease in tissue cultures of brain cells. *Nature* 235:104–105.

With C. J. Gibbs, Jr. Transmission of scrapie to the cynomolgus monkey (Macaca fascicularis). *Nature* 236:73–74.

1975 With C. J. Gibbs, Jr. Familial and sporadic chronic neurological degenerative disorders transmitted from man to primates. *Adv. Neurol.* 10:291–317.

1976 With C. J. Gibbs, Jr. Survival of Creutzfeldt-Jakob-disease virus in formol-fixed brain tissue. *N. Engl. J. Med.* 294:553.

1977 With C. Bernoulli, J. Siegfried, G. Baumgartner, F. Regli, T. Rabinowicz, and C. J. Gibbs, Jr. Danger of accidental person-to-person transmission of Creutzfeldt-Jakob disease by surgery. *Lancet* 1:478–479.

Unconventional viruses and the origin and disappearance of kuru. *Science* 197:943–960.

With C. J. Gibbs, Jr., D. M. Asher, P. Brown, A. Diwan, P. Hoffman, G. Nemo, R. Rohwer and L. White. Precautions in medical care of, and in handling materials from, patients with transmissible virus dementia (Creutzfeldt-Jakob disease). *N. Engl. J. Med.* 297:1253–1258.

1978 With C. J. Gibbs, Jr., and R. Latarjet. Unusual resistance to ionizing radiation of the viruses of kuru, Creutzfeldt-Jakob disease, and scrapie. *Proc. Natl. Acad. Sci. U.S.A.* 75: 6268–6270.

1980 With C. J. Gibbs, Jr., H. L. Amyx, A. Bacote, and C. L. Masters. Oral transmission of kuru, Creutzfeldt-Jakob disease, and scrapie to nonhuman primates. *J. Infect. Dis.* 142:205–208.

With J. Goudsmit, C. H. Morrow, D. M. Asher, R. T. Yanagihara, C. L. Masters, and C J. Gibbs, Jr. Evidence for and against the transmissibility of Alzheimer disease. *Neurology* 30:945–950.

1981 With H. L. Amyx, C. J. Gibbs, Jr., and W. E. Greer. Absence of vertical transmission of subacute spongiform viral encephalopathies in experimental primates. *Proc. Soc. Exp. Biol. Med.* 166:469–471.

With C. L. Masters and C. J. Gibbs, Jr. Creutzfeldt-Jakob disease virus isolations from the Gerstmann-Sträussler syndrome with an analysis of the various forms of amyloid plaque deposition in the virus-induced spongiform encephalopathies. *Brain* 104:559–588.

1983 With Y. Kuroda, C. J. Gibbs, Jr., and H. L. Amyx. Creutzfeldt-Jakob disease in mice: persistent viremia and preferential replication of virus in low-density lymphocytes. *Infect. Immun.* 41:154–161.

1984 With P. Brown and R. G. Rohwer. Sodium hydroxide decontamination of Creutzfeldt-Jakob disease virus. *N. Engl. J. Med.* 310:727.

With P. A. Merz, R. G. Rohwer, R. Kascsak, H. M. Wisniewski, R. A. Somerville, and C. J. Gibbs, Jr. Infection-specific particle from the unconventional slow virus diseases. *Science* 225: 437–440.

1985 With C. J. Gibbs, Jr., A. Joy, R. Heffner, M. Franko, M. Miyazaki, D. M. Asher, J. E. Parisi, and P. W. Brown. Clinical and pathological features and laboratory confirmation of Creutzfeldt-Jakob disease in a recipient of pituitary-derived human growth hormone. *N. Engl. J. Med.* 313:734–738.

1986 With P. Brown, M. Coker-Vann, K. Pomeroy, M. Franko, D. M. Asher, and C. J. Gibbs, Jr. Diagnosis of Creutzfeldt-Jakob disease by Western blot identification of marker protein in human brain tissue. *N. Engl. J. Med.* 314:547–551.

With P. Brown, C. J. Gibbs, Jr., F. Cathala and R. LaBauge. Transmission of Creutzfeldt-Jakob disease from formalin-fixed, paraffin-embedded human brain tissue. *N. Engl. J. Med.* 315:1614–1615.

1987 With D. Goldgaber, M. I. Lerman, O. W. McBride, and U. Saffiotti. Characterization and chromosomal localization of a cDNA encoding brain amyloid of Alzheimer's disease. *Science* 235:877–880.

1990 With P. Brown, P. P. Liberski, and A. Wolff. Resistance of scrapie infectivity to steam autoclaving after formaldehyde fixation and limited survival after ashing at 360 degrees C: practical and theoretical implications. *J. Infect. Dis.* 161:467–472.

With L. G. Goldfarb, P. Brown, D. Goldgaber, R. M. Garruto, R. Yanagihara, and D. M. Asher. Identical mutation in unrelated patients with Creutzfeldt-Jakob disease. *Lancet* 336:174–175.

1991 With M. S. Godec, D. M. Asher, C. L. Masters, W. E. Kozachuk, R. P. Friedland, C. J. Gibbs, Jr., S. I. Rapoport, and M. B. Schapiro. Evidence against the transmissibility of Alzheimer's disease. *Neurology* 41:1320.

1992 With L. G. Goldfarb. Viliuisk encephalomyelitis in the Iakut people of Siberia. *Brain* 115:961–978.

1993 With C. J. Gibbs, Jr., D. M. Asher, P. W. Brown, and J. E. Fradkin. Creutzfeldt-Jakob disease infectivity of growth hormone derived from human pituitary glands. *N. Engl. J. Med.* 328:358–359.

1994 With P. Brown, L. Cervenakova, J. W. Boellaard, D. Stavrou, and L. G. Goldfarb. Identification of a PRNP gene mutation in Jakob's original Creutzfeldt-Jakob disease family. *Lancet* 344:130–131.

With P. Brown, C. J. Gibbs, Jr., P. Rodgers-Johnson, D. M. Asher, M. P. Sulima, A. Bacote, and L. G. Goldfarb. Human spongiform encephalopathy: the National Institutes of Health series of 300 cases of experimentally transmitted disease. *Ann. Neurol.* 35:513–529.

With C. J. Gibbs, Jr., D. M. Asher, A. Kobrine, H. L. Amyx, and M. P. Sulima. Transmission of Creutzfeldt-Jakob disease to a chimpanzee by electrodes contaminated during neurosurgery. *J. Neurol. Neurosurg. Psychiatry* 57:757–758.

1998 With P. Brown, R. G. Rohwer, B. C. Dunstan, C. MacAuley, and W. N. Drohan. The distribution of infectivity in blood components and plasma derivatives in experimental models of transmissible spongiform encephalopathy. *Transfusion* 38:810–816.

2001 With H. S. Lee, P. Brown, L. Cervenakova, R. M. Garruto, M. P. Alpers, and L. G. Goldfarb. Increased susceptibility to kuru of carriers of the PRNP 129 methionine/methionine genotype. *J. Infect. Dis.* 183:192–196.

2008 Kuru and its contribution to medicine. *Philos. Trans. R. Soc. B.* 363:3697–3700.