NATIONAL ACADEMY OF SCIENCES

JAMES VAN GUNDIA NEEL
1915–2000

A Biographical Memoir by

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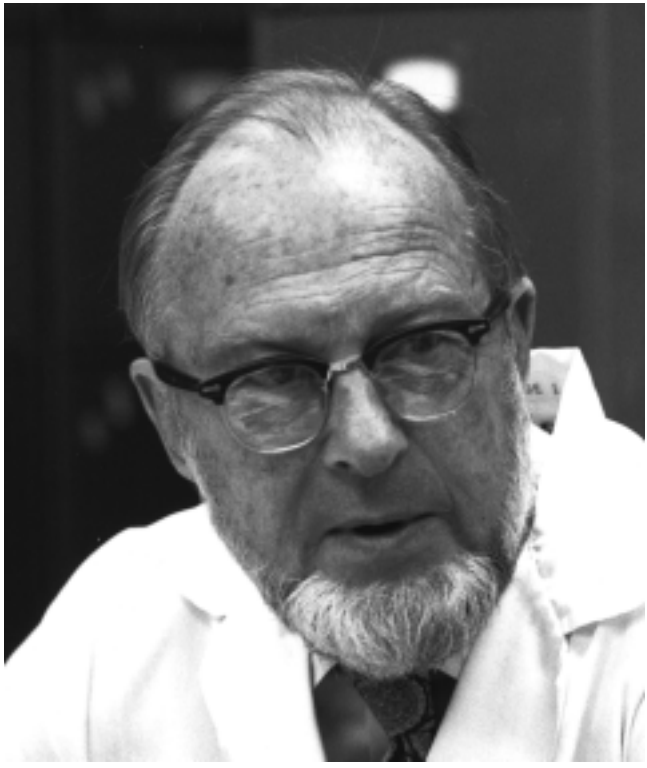
Biographical Memoirs, VOLUME 81

PUBLISHED 2002 BY
THE NATIONAL ACADEMY PRESS
WASHINGTON, D.C.



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James V. Neel

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March 22, 1915–February 1, 2000

BY WILLIAM J. SCHULL

ARGUABLY, GENETICS—particularly human genetics—was the most dynamic of the biological sciences in the second half of the twentieth century. It is widely acknowledged that one of the world's leading contributors to the latter discipline was James V. Neel. Some have called him the father of modern human genetics. Jim, as his colleagues knew him, was born in Hamilton, Ohio, on March 22, 1915, to parents comfortably placed, if not economically well off. An assured middle-class upbringing came to an end, however, with the death of his father when he was 10. His mother and her three children then moved from Detroit, where the family had been residing, to Wooster, Ohio, and it was here that he came of age. The times were parlous, and as a result of the Great Depression and the death of his father, a college education was no longer assured. Fortunately, the community his mother selected was the home of the College of Wooster, a small but outstanding liberal arts college to which he won a scholarship. Jim's career directions were not fixed when he entered college. Once enrolled, however, he came under the influence of Warren Spencer, an inspiring teacher and highly regarded *Drosophila* population geneticist and soon saw genetics as the direction he

would pursue. After graduation from Wooster he enrolled in the University of Rochester, where he was the first American graduate student of Curt Stern.

Soon after receiving his Ph.D. in 1939, he accepted a position as instructor in zoology at Dartmouth College, but before his teaching duties began he set out for Edinburgh to attend the VIIth International Congress of Genetics. The latter would be interrupted by the onset of World War II. When this occurred, the Americans present sought to return to the United States as quickly as possible. Not all were lucky. Some, like his colleague Charles Cotterman, booked passage on the British passenger ship *Athenia*, which was torpedoed on September 3, and it sunk with the loss of more than 100 lives. Jim was on the American freighter *City of Flint*, one of the vessels that came to the aid of the *Athenia*. Cotterman was among the passengers the *City of Flint* saved. Even before this near debacle occurred, however, Jim's interest had begun to shift to human genetics for which he had reasoned a medical degree would be important and had set his sights on such at the University of Rochester's Medical School. His progress toward this goal would be hastened by the acceleration of medical education during the war and would be eased financially by support from the Cramer Fund at Dartmouth, the Carnegie Foundation, and enlistment in the Army's Specialized Training Program (ASTP). He was awarded an M.D. in 1944, and in the following two years completed his internship and residency at Strong Memorial Hospital.

Upon completion of his medical training in 1946 he was called to active service in the U.S. Army Medical Corps. Soon thereafter, when President Harry Truman directed the National Academy of Sciences to undertake long-term studies of the health effects on the survivors of exposure to the atomic bombing of Hiroshima and Nagasaki, he was

one of five individuals (the others being Austin Brues, Paul Henshaw, Melvin Block, and Frederick Ulrich) the Academy sent to Japan to assess the needs and feasibility of the studies Truman had directed. Neel's involvement would not end with this assessment. He would serve as the first director of the agency charged with the studies, the Atomic Bomb Casualty Commission, and would design and initiate a survey to assess the genetic damage. He hinged this survey on a special provision in Japan's postwar rationing system. This provision made it possible for women, upon registering their pregnancies with the local government, to obtain rationed food to sustain themselves and their unborn offspring through gestation, and clothing for the infant once the child was born. When these mothers-to-be enrolled their pregnancies with the municipal authorities, they were also registered in the survey Neel had designed.

Implementation of this strategy was formidable. The economic circumstances in Japan were severe. Housing was scarce; food and clothing were rationed; and transportation, public or private, was limited. Personnel—American and Japanese—had to be recruited, including not only physicians to perform the examinations but also clerks to interview the prospective parents and to manage the records. Moreover, these individuals had to be motivated and impressed with the importance of each individual task, no matter how menial it might seem. These difficulties not withstanding and through perseverance and percipience, Jim would prevail, and a program would ensue. He would guide this undertaking—the largest, most comprehensive effort to assess the mutagenic effect of ionizing radiation on human beings that has yet occurred—for over a half century.

Jim's commitments at the time were greater than merely an involvement in the Japanese studies. Before his recall to

service in 1946, he had accepted a position in the University of Michigan's Heredity Clinic, where one of his colleagues was the aforementioned Charles Cotterman. Jim's first task there was to develop a research program in human genetics. Initially, he chose to focus this research on the estimation of the rate of spontaneous mutation of genes associated with a series of dominantly inherited diseases and on the mode of inheritance of several blood dyscrasias, an interest that had begun while he was still at Rochester. It was then that he deduced the genetic relationship between sickle cell anemia and the sickling trait and postulated the mode of inheritance now universally accepted. When Linus Pauling and his colleagues (1949) showed that sickle cell anemia was a molecular disease, Jim initiated a series of electrophoretic studies of families resident in Michigan; to further the understanding of the frequency of abnormal hemoglobins in Africa, he developed a working relationship with the Liberian Institute of Tropical Medicine.

In 1956, upon the retirement of Dr. Dice, who had been the director of the Institute of Human Biology of which the Heredity Clinic was a part, the university established a Department of Human Genetics. Jim was its founding chairman, and through his efforts it would become one of the stellar such departments, nationally and internationally. From its beginning, he wanted his department to have the breadth of knowledge and skills to approach genetic issues on the broadest possible front—from the biochemical, to the cytogenetic, to the immunological, to the epidemiological. He recruited to this end and steadfastly sought to establish and maintain a research milieu that fostered individual creativity, one in which his colleagues could reach their full potential. His success in this respect is attested by the scientific prominence his colleagues, past and present,

have achieved and the students and postdoctoral fellows the department has trained.

The year 1956 was a noteworthy one in Neel's career in other respects as well. The results of the radiation studies in Japan were presented at international conferences in Japan and Europe, a monograph on neurofibromatosis was published, and the first of a series of studies of the life experiences of the children of consanguineous marriages that would extend over a decade commenced in Japan. When these studies began, little was certain about the effects of consanguineous marriages. It was known that the children of related parents were more likely to be homozygous for a rare gene than were children whose parents were not related to one another. If the gene's effects were harmful when homozygous, the children of related parents would be expected to exhibit these deleterious consequences more often than the children of unrelated parents. This knowledge rested largely on studies of children selected because they were known to have a rare inherited disease. It was not known, however, how common these deleterious effects would be among children of related parents who had not been chosen with a view toward some specific health outcome.

The earlier study of the effect of ionizing radiation on a pregnancy outcome in Hiroshima and Nagasaki had identified several thousand children whose parents were related. Analyses of the data collected at or shortly after the birth of these children revealed that congenital defects were more common when the parents were related, and more of the children died in the first year of life than would be expected normally. The new studies were aimed at extending these observations over a longer period of time. Again, the preliminaries and logistics were challenging, but Jim proved to be an adept, patient advocate and organizer. He recruited the faculties of several Japanese universities and initiated a

series of meetings with the local municipal and educational authorities, parent-teacher associations, and the medical community to seek approval of the study and understanding of its objectives. However, other logistic problems existed. There was a need to train contactors to solicit the participation of the study cases, and a means found to transport the child (and parents, if they wished to accompany the child) to the clinic. All this planning sought to serve parental and social needs and to enhance the value of the examination to the children as well as their families.

Out of this effort came the most complete body of data available on the biological consequences of being the child of consanguineously related parents and a better appreciation of the relative magnitude of the health risks involved. Intriguing as these studies were in their own right, they were not tangential to the search for radiation-induced mutations. The aim of the latter search was not merely to count newly arisen mutations but also to estimate their long-term health impact. Because mutations can lurk in a population for generations before manifesting themselves, it had to be determined how genetic variability was maintained through this period before manifestation. Several competing theories existed but few human data to provide guidance as to which of these was correct. Finally, Newton Morton, James Crow, and Hermann Muller (1956) indicated how studies of the children of consanguineous marriages might contribute to the estimation of this "load" and to an assessment of the relative importance of these competing hypotheses.

This need to know how genetic variability is maintained stimulated a great deal of theoretical, experimental, and epidemiological research, but the populations that were being studied were generally more culturally advanced than those thought to characterize much of human evolution. Jim sought

to observe humans in a more ancestrally "natural" state. Thus began his quest for less technologically acculturated populations in South America. He recognized that these populations were not unacculturated in terms of the day-to-day circumstances of their lives, but they did dwell under conditions much more like human aboriginal ones than those generally prevailing. He reasoned that a study of their lives might provide insight into the general nature of human ancestral selective pressures, with consequences for human health (1958). As a consequence, much of Jim's work in the Amazon concerned biomedically relevant phenotypes. His intent was to compare the health profiles of hunting and gathering communities with those of the industrialized world. He was intrigued by the thought that a genotype might be beneficial in one environment but not in another. Indeed, it was this notion that gave rise to his much imitated argument that diabetes today was a "thrifty" genotype made disadvantageous by environmental changes (1962, 1982).

Jim was also interested in the evolution of responses to infectious organisms. It was known that infectious diseases such as smallpox and measles devastated aboriginal New World populations. But why? Were they inherently more susceptible or did the answer lie elsewhere? An obvious way to address the first of the alternatives was to study the susceptibility of populations suddenly exposed to what is typically a rather benign disease in populations with centuries of exposure to the same agent. Measles is such a case and epidemics of this viral disease have occurred with high rates of lethal complications in Amerindians. An ethically defensible way to examine the question of susceptibility would be to vaccinate isolated previously unexposed populations to gauge their reactions to the vaccine that, in the process, would also protect them from the actual disease. Jim was

planning to do this when an epidemic arose near, and even in, the villages they were about to study. Their plan gave way to an effort to limit the epidemic and minimize its health costs.

The Amazonian studies centered on all the factors contributing to population structure, including the determination of patterns of mate selection, mortality and fertility, and the estimation of effective population size and selection coefficients, as well as other parameters, such as admixture. They were surprisingly elegant, given the technology of the time, the complex logistics, and the need to coordinate substantial numbers of collaborators and government officials. While it was not possible then to document DNA variation very directly or exhaustively, limited genotyping was possible by blood typing and protein electrophoresis. Nonetheless, the global nature of many polymorphisms was demonstrated, but locally unique or "private" variants were also discovered. The overall level of variation was considerably higher than had been expected, raising questions about how that variation was maintained. Eventually these studies would embrace about 35 Yanomama villages and at least 20 other tribes in South and Central America. The result was a formidable set of data that along with the thousands of samples collected elsewhere around the world since then, has been influential in shaping our perception of human genetic diversity. The continued existence of 15,000 or so samples collected 30 or more years ago ensures that this scientific legacy will be profitably mined for many years to come.

As these studies were unfolding, Jim's interest again turned to the estimation of the frequency of radiation-induced mutation. Oliver Smithies's (1955) demonstration of the value of starch gel electrophoresis in characterizing inherited protein variability opened a new investigative door,

and the growing number of electrophoretically recognizable protein differences among individuals offered an opportunity Jim was quick to seize. Most of these proteins can easily be studied in blood specimens, but if this approach was to succeed, tens of thousands of tests would be needed, and the feasibility of a study of this scale was not clear. Demonstration of feasibility meant the identification of a suitable study group and the acceptability of the study to the survivors of the atomic bombings and their children. These concerns could only be resolved through a pilot study and in 1972 one was begun. When this study was terminated in 1975, it was clear that a full-scale investigation was technically feasible and acceptable to the population of interest. When the latter began in Hiroshima and Nagasaki in 1976, the aim was to examine each participating child for rare electrophoretic variants of 28 proteins of the blood plasma and red cells, and a subset of these children for deficiency variants of 10 of the red-cell enzymes.

When either such variant was encountered and before it could be attributed to mutation, the possibility of a technical error had to be excluded and then blood samples from both parents had to be examined for the presence of a similar variant. If the variant is not found in one or the other parent and if an error in assigning parentage is improbable, it presumably represents a new mutation. To establish parentage (since a priori the probability that the putative parents might not be the real parents is several orders of magnitude larger than the probability of a new mutation) some 11 different red-cell antigenic systems and the major histocompatibility phenotypes (the HLA system) were used to search for evidence that the putative parents were not the actual parents of the child. Although such testing does not prove parentage (it can only exclude falsely identified parents), the battery used was sufficiently large

that the a priori probability of failing to detect a falsely identified parent was approximately the same as the a priori probability of a new mutation.

When this study terminated in the 1980s, three probable structural mutations had been seen in 667,404 locus tests on 13,052 children born to parents whose average combined gonadal dose was about 0.47 Sv, and three in 466,881 locus tests on 10,609 children whose parents received less than 10 mSv. The mutation rates in the two groups of children were almost identical; the values are 0.60×10^{-5} mutations per generation in those who were the offspring of parents receiving more than 10 mSv of gonadal exposure, and 0.64×10^{-5} in those whose parents received less than 10 mSv. The confidence intervals for these two estimates, that is, the probable range in which the "true" value lies, were 0.2-1.5 and $0.1\text{-}1.9 \times 10^{-5}$, respectively. In addition, one probable "deficiency" mutant was seen in 60,529 locus tests on children whose parents, one or both, received more than 10 mSv of radiation, but none among the 61,741 tests on the children of distally exposed parents. Thus, when the results of the studies of structural and activity variants were combined after more than 1,256,000 biochemical tests, four mutants were seen among the children of parents receiving more than 10 mSv, and three among those whose parents received less than 10 mSv.

Despite the inconclusive results this was a landmark study integrating evolutionary, population, and molecular genetics and a clever study designed to address a question of importance to contemporary public health and to give a perspective on evolutionary biology. Moreover, in the measurement of mutation rates it shifted the focus from crude phenotypes (the product of a complex web of gene-environment interactions) to the immediate product of gene action. Nonetheless, the study had two significant limita-

tions. First, despite the enormous amount of work involved, a sample of a million and a quarter locus tests was marginally adequate to detect the level of mutational damage thought to be most likely. Second, while the number of functional human genes was uncertain, it appeared to be no larger than 50,000 and if only 28 or so of these were studied how likely is it that they would be representative of the totality? Neither of these issues seemed likely to be resolved with the technology then available.

Two new techniques of promise had appeared on the horizon, however: gene sequencing and two-dimensional electrophoresis. In the late 1970s both of these approaches had their strengths and their limitations, and it was unclear which to pursue, if only one could be pursued. Jim chose to champion two-dimensional electrophoresis. He and his colleagues immediately turned to the standardization of the purely biochemical aspects of the technique, and the proof that the technique would work. However, the two-dimensional separation of DNA results in 500 or more recognizably discrete products. Analysis of the difference between two samples in the distribution of these products defies easy visual examination. This fact led to a substantial investment in automated methods of pattern recognition (see, e.g., Skolnick and Neel, 1986). As was his wont, Jim immersed himself in this technology until he could persuade himself that he could contribute to its furtherance. Scarcely eight weeks before his death, he was still so engaged. He and a colleague, Junichi Asakawa, were summarizing their joint study of the utility of two-dimensional electrophoresis in the estimation of radiation-induced mutation rates.

Research was not the sole function of the Department of Human Genetics; teaching was no less important. When in the late 1950s the National Institute of General Medical Sciences instituted a pre- and postdoctoral training pro-

gram in genetics, Jim was asked to serve as the chairman of the Genetics Training Grant Committee, a position he would hold from 1958 through 1963. In this position he and his committee did much to codify the standards that would guide this program for several decades. Ironically, students, generally unaware of his role in the establishment of the program that supported many, were often wary of Jim. His accomplishments, prestige, and no-nonsense demeanor were intimidating. They feared he would be unreasonably demanding and insensitive. But as they soon realized, this was not the case. He was demanding but sympathetic. He sought to encourage all students to be thoughtful and critical, not only of their own work but that of others, including their mentors as well. Through subtle probing he invariably managed to bring out the best in a student.

Neel's contributions to human genetics are legion and it is difficult to discern a single thread that connects all aspects of his research career. If a thread exists, however, it is the phenomenon of mutation. His interest began at Dartmouth College, was whetted by his association with Philip Ives and Ernst Hadorn, and continued throughout his long connection with the studies in Japan. In the pursuit of this fundamental biological process he demonstrated an admirable capacity to incorporate new technologies and new ideas as these became available. His interest focused not merely on the frequency of mutation, whether spontaneously occurring or induced, but also upon the biochemistry of the process, the manifestation of mutations when present in a single dose, and the factors that govern the persistence or loss of new mutations at the population level.

Neel was elected to membership in the National Academy of Sciences (1963), American Philosophical Society (1965), American Academy of Arts and Sciences (1971), Institute of Medicine (1972), and the Royal Society of Medi-

cine (1992), as well as other honorary societies. He received numerous awards, among these being the Lasker Award of the American Public Health Association (1960), Allen Award of the American Society of Human Genetics (1965), National Medal of Science (1975), Medal of the Smithsonian Institution (1981), and the Silvio Conte Award (1991). Among the many named lectureships he gave were the Galton Lecture (University College, London), the Cutter Lecture (Harvard), Harvey Lecture (Harvey Society), the Russel Lecture (University of Michigan), the Jacobson Lecture (University of Newcastle-upon-Tyne), the Baker Lecture (Pennsylvania State University), and the Joshua Lederberg Distinguished Lecture (Rockefeller University). His colleagues recognized his many contributions to human genetics through electing him to numerous presidencies, among them those of the American Society of Human Genetics (1953-54), International Society of Genetic Epidemiology (1991-93), and the Sixth International Congress of Human Genetics (1981). He served on many editorial boards and in a consultative capacity for countless national and international agencies. Among these editorial boards were those of *Blood, Perspectives in Biology and Medicine, Proceedings of the National Academy of Sciences, Behavioral Genetics, Mutation Research, Journal of Molecular Evolution, Clinical Genetics, and Genetic Epidemiology,* to mention only a few. The agencies he aided included the National Institutes of Health, Department of Energy, Environmental Protection Agency, National Council on Radiation Protection and Measurements, Veterans Administration, Pan American Health Organization, and the World Health Organization. He received honorary degrees from his alma maters, the College of Wooster (1959) and Rochester University (1974), as well as the Medical College of Ohio (1981).

This recitation of his scientific vision and professional

achievements is a limited measure of the man. He was much more. His curiosity and interests constantly amazed his colleagues and fellow academics. He was not only an exceptionally able clinician who, despite his administrative responsibilities, periodically took on the management of a clinical ward but also a great human biologist in the premodern sense of that calling. He was also an avid orchidist and a collector of butterflies. More important, he was a person of enormous personal integrity, sensitivity, and compassion. He was deeply concerned with the lot of his fellow kind as his autobiographical book *Physician to the Gene Pool* (1994) compellingly testifies. He was truly a "man for all seasons." Above all, however, he was devoted to his family: his wife, Priscilla Baxter, and his three children, Frances, James, Jr., and Alexander. His concern for them was always foremost.

Jim was not without his foibles. For example, although his career spanned an era extending from the mechanical desktop calculator (the Monroe, Marchant, or Frieden) to the electronic marvel that now exists, he was never comfortable with these devices. He reluctantly used the calculator but never adapted to its electronic counterpart. When he needed the services these could provide, such as e-mail or word processing, he turned to his children or to his secretaries. He preferred to continue to write his manuscripts in longhand and saw no hardship in this. Some of his resistance to gadgets may reflect an anecdote he told on himself. As a doctoral student, to save money so that he might attend the international congress in Edinburgh he decided to type his doctoral dissertation himself. This proved a more traumatic experience than he had anticipated and he swore off such machines. Be this as it may, when Jim relinquished the chairmanship of the department in 1981, unlike many of his age peers who settled quickly and comfortably into

the role of a senior scientist, he remained totally involved, and kept abreast of new developments in genetics with a fervor his younger colleagues envied. Although his was a full life by any accounting, his death leaves contemporary human genetics and modern clinical medicine much the poorer, and his friends and associates deprived of a concerned and willing source of counsel.

Several months after James Neel's death his family, colleagues, and friends found themselves involved in a controversy. Allegations were made that his involvement in the studies of the Xavante and Yanomama of Brazil and Venezuela stemmed largely, if not solely, from his interest in eugenics, and that he had consciously and unethically imported a measles epidemic into the Venezuelan outback to further his interest in the biology of immune response to exogenous infectious pathogens (Tierney, 2000). It was alleged that this epidemic led to the deaths of hundreds, if not thousands, of individuals who were ill prepared immunologically to cope with the new virus. Callously the individuals responsible for these allegations ignored the fact that the epidemic began before Neel and his colleagues were in the field. Informed of the raging epidemic before his departure from the United States, Neel brought gamma globulin and measles vaccine from pharmaceutical companies in the United States to combat the spread of the disease. However, even this effort was diminished. He was accused of bringing a virus less suitable to the situation than was then available. The salutary aspect of this sordid affair was the promptness with which all of those scientists who knew Jim or were aware of his work rose to his defense. Clinicians, geneticists, virologists, all spoke on his behalf. A reasonable society could reach only one conclusion. The charges are baseless, wholly unwarranted, and mendaciously cruel. The reasons that prompted these allegations may never

be fully known, but whatever their bases they do no credit to the individuals who availed themselves of this opportunity to pursue their own agendas.

REFERENCES

Morton, N. E., J. F. Crow, and H. J. Muller. 1956. An estimate of the mutational damage in man from data on consanguineous marriages. *Proc. Natl. Acad. Sci. U. S. A.* 42:855-63.

Pauling, L., H. A. Itano, S. J. Singer, and I. C. Wells. 1949. Sickle cell anemia: A molecular disease. *Science* 110:543-48.

Skolnick, M. M., and J. V. Neel. 1986. An algorithm for comparing two-dimensional electrophoretic gels, with particular reference to the study of mutation. In *Advances in Human Genetics*, vol. 15, eds. H. Harris and K. Hirschhorn, pp. 55-160. New York: Plenum Press.

Smithies, O. 1955. Grouped variations in the occurrence of new protein components in normal human serum. *Nature* 175:307-308.

Tierney, P. 2000. *Darkness in El Dorado: How Scientists and Journalists Devastated the Amazon.* New York: W. W. Norton.

SELECTED BIBLIOGRAPHY

James Neel was the author of more than 600 scientific articles and the author, coauthor or editor of no less than 12 books or monographs. It is a challenging task to select from his extensive publications those that all would construe as seminal. Any selection must reflect, and perhaps unduly, one's personal preferences. Geneticists would undoubtedly select one set, human biologists another, and clinicians still a third. But these differences merely reflect the breadth of his interest and competence.

1944

With W. N. Valentine. Hematologic and genetic study of the transmission of thalassemia (Cooley's anemia, Mediterranean anemia). *Arch. Int. Med.* 74:185-96.

1947

The clinical detection of the genetic carriers of inherited disease. *Medicine* 26:115-53.

1949

The inheritance of sickle cell anemia. *Science* 110:64-69.

1951

With H. F. Falls. The rate of mutation of the gene responsible for retinoblastoma in man. *Science* 114:419-22.

1952

The study of human mutation rates. *Am. Nat.* 86:129-44.

1954

With W. J. Schull. *Human Heredity*. Chicago: University of Chicago Press.

1955

On some pitfalls in developing an adequate genetic hypothesis. *Am. J. Hum. Genet.* 7:1-14.

1958

The study of natural selection in primitive and civilized populations. *Hum. Biol.* 3:43-72.

1962

Diabetes mellitus: A "thrifty" genotype rendered detrimental by "progress"? *Am. J. Hum. Genet.* 14:353-62.

1964

With F. M. Salzano, P. C. Junqueira, F. Keiter, and D. Maybury-Lewis. Studies on the Xavante Indians of the Brazilian Mato Grosso. *Am. J. Hum. Genet.* 16:52-140.

1970

Lessons from a "primitive" people. *Science* 170:815-22.

1973

"Private" genetic variants and the frequency of mutation among South American Indians. *Proc. Natl. Acad. Sci. U. S. A.* 70:3311-15.

1978

The population structure of an Amerindian tribe, the Yanomama. *Ann. Rev. Genet.* 12:365-413.

1982

The thrifty genotype revisited. In *The Genetics of Diabetes Mellitus,* eds. J. Köbberling and R. Tattersall, pp. 137-47. New York: Academic Press.

1986

With A. A. Awa. Cytogenetic "rogue" cells: What is their frequency, origin, and evolutionary significance? *Proc. Natl. Acad. Sci. U. S. A.* 83:1021-25.

1990

With S. E. Lewis. The comparative radiation genetics of humans and mice. *Ann. Rev. Genet.* 24:327-62.

1991

With W. J. Schull. *The Children of Atomic Bomb Survivors: A Genetic Study.* Washington, D. C.: National Academy Press.

1994

Physician to the Gene Pool. New York: John Wiley.