



The History of Neuroscience in Autobiography Volume 7

Edited by Larry R. Squire

Published by Society for Neuroscience

ISBN: 0-19-539613-8

Robert Y. Moore

pp. 530–561

<https://doi.org/10.1093/acprof:oso/9780195396133.003.0012>



Robert Y. Moore

BORN:

Harvey, Illinois
December 5, 1931

EDUCATION:

Lawrence University, B.A. (1953)
University of Chicago, M.D. (1957), Ph.D. (1963)
Resident in Neurology (1963)

APPOINTMENTS:

Instructor in Anatomy to Professor Anatomy and Neurology,
University of Chicago (1959–1974)
Professor, Department of Neurosciences, University of California San Diego (1974–1979)
Professor and Chair, Department of Neurology, SUNY, Stony Brook (1979–1990)
Professor, Departments of Neurology and Psychiatry, University of Pittsburgh (1990–1996); Chair
of Neurology (1996–2000)
Love Family Professor of Neurology and Neuroscience, University of Pittsburgh (1998–2010)

HONORS AND AWARDS (SELECTED):

Markle Scholar in Medical Science (1964–1969)
M.D. (Honoris causa), University of Lund, Sweden (1974)
President, The Cajal Club (Society of Neuroanatomists) (1990–1992)
C.U. Ariens-Kappers Medal, Netherlands Institute for Brain
Research, Royal Netherlands Academy of Science (1991)
President, Society for Research on Biological Rhythms (1992–1994)
Farrell Prize in Sleep Medicine, Harvard Medical School (2010)
Member, President's Committee on the National Medal of Science (2006–2011)

Robert Moore became intrigued with the concept of localization of function in the brain, and its implications for medicine, as an undergraduate. After early work on the neurobiology of memory, he studied the organization of mammalian auditory and visual systems. This led to studies of the neural control of circadian timing, resulting in discovery of a direct retinohypothalamic tract specialized to mediate entrainment. Subsequent studies showed that selective ablation of the suprachiasmatic nuclei resulted in a loss of circadian function, evidence that the nucleus is a circadian pacemaker, and this has formed a basis for a continuing investigation of a brain circadian timing system. Description and analysis of the circadian timing system has continued in his laboratory along with extensive anatomical studies of the organization of brain systems producing monoamines. Over the last 10 years, he has extended this work to imaging studies of the dopamine systems in Parkinson's disease. Throughout his career, he has practiced and taught neurology, and care of patients has been an important component of his activities.

Robert Y. Moore

Parents and Early Years

My father, Raymon Irwin Moore, was born on a farm in Powder Springs, Georgia, on June 20, 1904. Powder Springs is north of Atlanta, close to the now prosperous town of Marietta. His father was Autry Cole (called "AC") Moore, born on the same farm in about 1865. My family had owned the farm since at least the Civil War, and it contained, not far from the farmhouse, the remains of trenches and fortifications built by Hood's army, the Confederates, for the Battle of Atlanta in 1864 during Sherman's march to the sea. I remember playing in them as a child. The family story, for which I have no proof, is that the Moores came to Georgia in the first group of prisoners with Oglethorpe (the prisoners were from debtor's prison and were transported to colonies as indentured servants) in the 1730s and had begun farming when released from their obligations. This was in an area of poor soil fertility, and their lives were ones of subsistence farming. My father was the fourth of five boys and, with no prospects of inheriting the farm, he was sent to a residential training school for the poor to obtain an education. After finishing high school, he moved to Chicago in the early 1920s to find employment. After some temporary jobs, he was taken on as a clerk by the Continental Illinois Bank and Trust Company. It was there he met my mother, Marie Louse Fisher, born on September 11, 1904, in Maquoketa, Iowa, a small farm town in eastern Iowa. My grandfather, Harry C. Fisher, was the son of German immigrants and owned the town lumberyard. He was the fire chief as well and had a siren on his car, very compelling for a young child. He also taught me to play golf, a lifelong but rarely fulfilled obsession.

I was born at the Ingalls Memorial Hospital in Harvey, Illinois, a southern suburb of Chicago. My parents lived in an apartment in Blue Island, Illinois, next door to my mother's maternal aunt and uncle, for 4 years after I was born, and my earliest memory is of a visit from the iceman. We had a real icebox kept cold by a block of ice delivered by a man with a black rubberized shawl over his back upon which a block of ice was held with great pincers. I have no other memories from that age, and we moved to Atlanta when I was 5 because my father was employed as a salesman by the Burroughs Adding Machine Company (a company that made a variety of business machines but was absorbed into Unisys subsequently). He was eager to return to the area of his home and we moved to an apartment in Atlanta just off Peach Tree Street. I remember the apartment and fragments of the neighborhood. It must have been integrated to some extent because

I remember playing often with a little “colored” girl. I attended kindergarten but failed and had to repeat it. I probably would be designated as having attention-deficit behavior now but such niceties were unavailable at the time and I suspect I was called “slow.” I had one frightening experience that I still remember vividly. On my way home from school I was stopped by a group of older boys, tied up, and taken to an abandoned building where one of the group dropped hot candle wax on my thigh. I screamed so loudly that the boys fortunately let me go. I reported this to my father, but the boys were apparently not local and could not be identified. From Atlanta, we moved to Detroit, quite a nice city in those days. We lived in an apartment near 9 Mile Road, north of the city center. It was a pleasant area away from heavy traffic and I learned to ride a bicycle. School was a several-block walk and I remember a problem with a teacher who objected to my left-handed writing. Neither she nor my parents were able to convince me to change. I did second and third grade there and my reading became sufficiently proficient that I could read fiction of some complexity. My father purchased a Modern Library edition of Mark Twain’s *Tom Sawyer* and *Huckleberry Finn* for me and I was enthralled, particularly with *Tom Sawyer* (*Huckleberry Finn* was a bit too deep for an 8-year-old). I read them over and over. At one point, my records indicated that I had read *Tom Sawyer* 56 times. The experience turned me into a reader.

My only sibling, Jim, was born in 1938 in Detroit but, as he was 7 years younger, we did not do much together until he was much older. One experience that started during our period in Detroit was a series of operations, performed at St Louis Children’s Hospital, to repair a congenital anomaly of the penis called hypospadias. These were excruciatingly painful and I dreaded the operations. My father was transferred again by his employer in 1939 and we moved to St. Louis, settling into an apartment on the then outskirts of the city. A few memories remain about the times; I was learning that we lived in a world and that things happening at a distance had direct effects on us. I remember the 1940 presidential campaign, Roosevelt and Willkie, well. All parents seemed to have strong feelings about FDR, pro or con and the election, in the midst of the very menacing start of World War II in Europe, was a subject of conversation among children as well. The events of the Pearl Harbor attack on December 7, 1941, seemed menacing, even for a newly 10-year-old. It seemed ominous. Rationing began, of gasoline in particular, and, at times, there was relatively little meat available. My family was not prosperous during this era, and we ate some things that were not food to my mind at the time. I became fascinated by sport; hours and hours passed with baseball, football, and a game called “corkball,” played with a small ball, slightly larger than a golf ball made of cork wrapped in leather, and a long thin bat. Its advantage was that four boys and girls could have a good game in a small area without risk of broken windows.

Our family finances were strained, and it was clear to me that I needed a source of income. An opportunity to sell magazines door to door came my way and I took it. Very rapidly I learned that interruption of the dinner hour by a magazine-carrying urchin was not greeted enthusiastically. The best time was afternoon, after school, and before the man of the house returned from work. The magazines sold for 10 or 15 cents and I received 30%. In time, this went so well that I became the distributor, employing other boys. My major entertainment in the summer months, besides playing with pals, was a thing called the "Knothole Gang." St Louis at that time had two baseball teams, the Cardinals and the Browns, and all games were played at Sportsman's Park. If one took a birth certificate to the ticket office, a membership card for the Knothole Gang was issued which permitted, for day games, admission to the game for the cost of tax on a ticket, 5 cents. After a modest walk from our apartment to a main road, a streetcar to the ballpark was 5 cents, popcorn at the game 10 cents so that, for 25 cents, a small boy could go to the baseball game with transportation, entertainment, and nourishment all included. I am now at about 1939 and 1940. In 1942, my father applied for a job with a company headquartered in Cleveland, the Snapout Forms Co., a maker of business forms, to head a new sales office in Chicago. It was a good time for jobs for men above age for military service, and my father had just missed both great wars as I did World War II and Vietnam. He and my mother were pleased to be able to return to Chicago. We settled in a suburb immediately northwest of the city, Park Ridge, for the first time in our own house. It was adjacent to a zone called the "Forest Preserve," a large area of preserved parkland surrounding the city of Chicago, a kind of public accomplishment that our current politicians would be incapable of making. My parents continued to live there until the late 1970s when both required nursing homes.

Education and Life in the Suburbs

The local schools in Park Ridge were quite good, but I was not a successful student, failing all courses in eighth grade. An IQ test was administered and the principal informed my parents that I was too bright to be failing in school and he was sending me to high school. For the first 2 years I managed a C+ average but, for no reason I can remember now, everything suddenly seemed easily accomplished and it was high grades from then on. My major preoccupations were sports and reading. In junior high school, we played basketball essentially year round, mixed with some baseball and football. In high school I tried out for the football team, but it was soon apparent to me, and the coach, that I was not big enough, strong enough, fast enough, or coordinated enough to succeed at that sport. There were no such requirements for cross-country running and that became my fall sport, followed by

indoor track in the winter and golf in the spring. In my last 2 years in high school there were few days when I went directly from school to home. In the summers from 12 to 16, I worked as a golf caddy. A friend who lived nearby would meet me at 5 a.m., and we walked the 1.5 miles to the golf course to be the first there as the work assigned by the caddy master was on a first-come, first-served basis. Being a caddy allowed us to use this very nice private course on Monday mornings, a help for my golf game. The job also provided a fine insight into the vagaries of human behavior. After I was 16, I worked in factories and hospitals in the summers. These were always interesting for many reasons and taught me a lot. I gave up factories after two summers with little interest in the monotony of factory work and a profound dislike, really abhorrence, of labor unions.

In my last 2 years of high school, I became increasingly interested in literature, but it was more than evident to me that I had no talent for creative writing. In searching for a surrogate activity, I came across literary criticism and this led me to attend Kenyon College, a small men's school at that time in Gambier, Ohio, well known for a program in that discipline. The education was excellent, but I was not happy with the location of the school in a town of 500 and an all-male student body. My best friend from high school was attending Lawrence College in Appleton, Wisconsin, and was happy there, so I transferred. I could not have made a better choice. Lawrence was, and continues to be, an excellent university with high standards and a faculty of very dedicated scholars and teachers. My education at Lawrence was a transforming experience. Classes were small and I became deeply interested in the brain through courses in biology and psychology. One professor in particular, John Bucklew, took an interest in me and taught me in individual tutorials over my last 2 years. These were largely devoted to reading and discussing a broad literature on brain function. I became fascinated by the concept of localization of function and that has been maintained. In my senior year I struggled with the choice between a Ph.D. or medical school. My mother and father were, of course, eager to have a doctor in the family, but I felt certain that I wanted to do research. I applied to a few Ph.D. programs and several medical schools and was accepted by all. At an interview at the University of Illinois School of Medicine, the interviewer suggested that I should consider the University of Chicago because I would be able to do both degrees there. I took the recommendation and have always been grateful for it. My last semester in college revealed a problem that has recurred intermittently ever since. In the late winter of 1953, I developed episodes of severe, unremitting fear. I was agitated, unable to sleep, and had great difficulty concentrating. Fortunately, I had no regular classes and my only unfulfilled requirement was a dissertation. Writing the dissertation was difficult, but I could do it and finished all that was needed to graduate. Shortly after the symptoms began, I visited home and my father took me to a psychiatrist who spent a few minutes

talking to me and recommended sedatives. The problem was what is now designated panic disorder; it was called acute anxiety attacks at the time. The symptoms gradually resolved, and I entered medical school without difficulty. Since that initial attack, I have had occasional recurrences, but none so severe. Instead, I have had intermittent episodes of depression. These all cleared with time, but I have certainly had repeated visits from what Dr. Samuel Johnson referred to as his "black dog." Since the development of the selective serotonin reuptake inhibitors (SSRIs), this has rarely been a problem.

My medical education at the University of Chicago was a very different experience from that of friends at other schools. Medical students were viewed as graduate students at Chicago and took preclinical courses designed as graduate courses. There was no clinical content for the most part and we saw no patients until the third quarter of the second year. In addition, there were elective courses in which any subject was allowed; a roommate took a course in art history. I took the opportunity to begin research and worked in the laboratory of Austin Riesen, a neuropsychologist who had discovered the effects of early visual deprivation on the development of the visual system in monkeys. As I found was typical of the University of Chicago, Riesen was perfectly happy to let me design my own experiment and conduct it with his funds and facilities. These were early days in the discovery of adrenal corticosteroids and Hans Selye's development of the concept of a stress syndrome with adrenal responses. My hypothesis was that adrenal responses would be necessary for aversive learning. I was taught how to do rat adrenalectomies by Dwight Ingle, a pioneer adrenal physiologist, and was able to do the experiment in the course of the quarter. Although the results were negative, I felt I had begun a career. My father was willing to support me for the summer and I went to work on another project with Howard Hunt, another neuropsychologist who had had some graduate training with B. F. Skinner and was introduced to the use of operant behavior paradigms. In all, I worked with Howard for about 6 years, summers, weekends, vacations and electives, and a year following graduation from medical school to complete a Ph.D.

The liberal program of the University of Chicago was again a blessing because my entire senior year in medical school was elective. My grades had been good except for gross anatomy, which I barely passed, and my junior year clinical grades had been outstanding. I was elected to Alpha Omega Alpha, as I had been to Phi Beta Kappa, and the medical school seemed perfectly happy to allow me to do as I pleased. I elected to do no clinical work and concentrated on research completing the work for my thesis during that year and a subsequent postdoctoral year. Thus, contrary to present programs, I was able to do an M.D.-Ph.D. in 5 years. My thesis was driven conceptually by the finding of Scoville and Milner that bilateral hippocampal lesions

in man produced a severe and permanent deficit in short-term memory. The hypothesis was that hippocampal lesions in animals would have the same effect and provide an animal model for memory research. Once again, I made a mistake in experimental design and chose to use a negative reinforcement paradigm and hippocampal ablation in cats. By this time I had become a very skilled animal neurosurgeon and the experiment was quite lovely but, again, negative (Moore, R.Y. Effects of some rhinencephalic lesions on retention of conditioned avoidance behavior in cats. *J. Comp. Physiol. Psychol.*, 57:65–71, 1964). Thus, this early venture into science was not rewarding with respect to positive outcomes but was rewarding in every other aspect. As time went on during my training, I became increasingly interested in neuroanatomy and began to collaborate with students working in the laboratory of W. D. Neff, a neuropsychologist studying the auditory system. Neff and an associate, Irving Diamond, did some anatomical studies and had an active lab in which I was taught anatomical techniques. I was a graduate assistant in a human neuroanatomy course my senior year in medical school and was allowed to give a number of the lectures in the course the following year. Neuroanatomy at that time was a course with 40 lectures and 90 hours of laboratory, all taught by one faculty member, a teaching load that would be derided now. It was evident to me from the beginning that teaching was the most efficient way to become broadly knowledgeable in a subject in a short time.

On entry to medical school, I had believed that the proper specialty for my interests would be psychiatry, but lectures on human behavior given by the Department of Psychiatry prompted further consideration. The department had become very Freudian, and I vividly remember one lecture in which it was proposed that one example of the success of psychoanalytic theory was the realization that asthma represented the calling out of a child for its mother. Even in my naiveté I knew that something was wrong with that idea. In that era, the first 2 years of medical school were basic science with little clinical correlation. One clinical experience available to all students was a weekly presentation of a patient by Dr. Douglas Buchanan. Buchanan was a Glaswegian educated at Cambridge who took neurological training at the famous National Hospital for the Epileptic and Insane, as it was then called, Queen Square, London. When he finished in 1931, there were few positions in England, and he was hired at the University of Chicago as a neurologist for children. Prior to 1927, the University of Chicago offered only 2 years of training for medical students who then went to Rush Medical College for the last 2 years. The University had planned for several years to open a 4-year medical school based on the recommendations of the famous “Flexner Report” that was quite critical of American medical education and made recommendations for an ideal medical school and curriculum. These included having a full-time faculty, both basic science

and clinical. To meet this objective, the University built its own hospital adjacent to the main campus and hired the faculty. Buchanan was one of these pioneers. The University of Chicago Hospitals and Clinics opened in 1927 and each of the general clinical services, medicine, surgery and pediatrics, was allotted a position for a neurologist. Child neurology was an unknown field at the time; pediatricians or general physicians cared for most children with neurological disorders. Through his vast experience at Chicago, and his skills as an educator, Buchanan became one of the three founders of child neurology, now a vigorous subspecialty. At the time I went to the University, he had trained more than a third of the child neurologists in the country. He was the most captivating teacher I have ever known. In addition to ward and clinic teaching, he made a Saturday morning presentation each week during the academic year in a lecture hall with 120 seats. The class was entirely elective, but the hall was filled every Saturday morning. After presenting a case and performing an examination, Buchanan would discuss it. These were far-ranging discussions, often with a good deal of history. What was evident to all was how much he cared about the patient and how deeply he was devoted to scholarship and education. In my clinical training, I was also captivated by the teaching of Richard B. Richter, whom I will discuss later. The two of them made a convincing argument for becoming a neurologist.

Following my postdoctoral year, I did a rotating internship at the University of Michigan with a focus on neurology. This was rigorous. Upon arrival, we were informed that vacations were too hard to schedule, but we would receive one month's salary (\$117) in lieu of vacation. We were on call every other night and spent a lot of time in the hospital on those nights. I lived in the hospital, in the intern's quarters, sharing a room. We were charged \$30/month rent and were required to pay for our own food. It was good to be young with no responsibilities. At the University of Chicago, I had learned about the work of C. Judson Herrick, an eminent neuroanatomist who made the standard classification of cranial nerves and wrote many papers and books. At Michigan I learned that he lived with his daughter, a dermatologist in Grand Rapids, and, even at an advanced age, was still writing. I had the opportunity to spend 2 months at St Mary's Hospital in Grand Rapids as an intern in pediatrics. I contacted his daughter to inquire whether I could meet the great man, and she invited me for dessert one evening and, subsequently, to his 90th birthday party later in the month. I was awed by his insight into structure–function relationships in brain and nearly overwhelmed by the birthday party held on a sunny autumn day and attended by great neuroscientists from around the world.

Despite the heavy schedule, I was bored by internship duties and went to the Department of Psychology to meet Robert Isaacson, a young faculty member, who wanted to do some work on brain and behavior but had not done animal surgery. We had a good experience together and the collaboration led

to a publication (Isaacson, R.L., Douglas, R.J., and Moore, R.Y. The effect of radical hippocampal ablation on acquisition of avoidance response. *J. Comp. Physiol. Psychol.*, 54:625–628, 1961).

During my internship, I was contacted by Chicago and offered a position on completion of my training in Michigan as instructor in the Department of Anatomy with my own laboratory, and funding to start, with half of my time to be spent as resident in Neurology. Thus began a career in research, teaching, and clinical medicine I have enjoyed ever since. As a special pleasure, I found that the lab I was assigned had been Herrick's. For the first 6 months I took my place as neurology resident. My laboratory was having some minor renovation, and I was ordering equipment and other essentials to start research. At that time, the summer and fall of 1959, I was the only resident in the program, a heavy load, and I was on night call every other night and every weekend. Somehow it did not seem so bad at the time. It helped not to be married. Another resident started in January 1960, and the strain was greatly relieved.

Neurology at that time was a Division in the Department of Medicine with three faculty members. Richard B. Richter was the chief. I have seen neurologists at work now for 50 years, all over this country and in Europe, but he was the best clinician I have ever seen. His style was deductive, not intuitive. Histories were precise and organized. His neurological examination was appropriate to the situation but always detailed, frequently lasting more than an hour. When one worked with him, he expected the same detailed approach, and I quickly caught on. He was a terror for medical students because he expected their work to be similarly performed, requiring from each student a detailed and organized history and examination and a deductive process formulating a differential diagnosis. He was not sympathetic to those who did not perform. When I responded to a question from him once about a rather weak medical resident applying to the neurology program with a statement that he was a very kind and diligent fellow, he responded, "Fortunately, that's not good enough." His research was in neuropathology and all of the hospital neuropathology was done in neurology.

One of my proudest moments as a trainee was when he asked me to join him in writing a paper (Richter and Moore, 1968). The other two faculty members were Sidney Schulman and Charles Barlow, both superb clinicians. Schulman was in the mold of Richter, precise, patient, deductive. Barlow, like Buchanan, was an intuitive neurologist who often took little time but nearly always reached the right conclusions. After my second year, Barlow left to become the Bronson Crothers Professor of Neurology at Boston Children's. This was shortly before Buchanan was scheduled to retire and, as I had spent more time than anyone in the program with Buchanan, I was asked to succeed Barlow on completion of my training and to take over the Child Neurology Division on Buchanan's retirement. It came with a great fringe benefit; the medical center had just built a new children's

hospital with incredible laboratory space and I moved into 3000 square feet of brand new space. At that time, there was a dispute among neurologists and pediatricians about the proper training of a child neurologist. Was the basic emphasis to be neurology or pediatrics? This was not decided until the late 1960s when a committee was appointed by the American Academy of Neurology to discuss and design a curriculum for training child neurologists. I was one of the committee and a final task for us was to have the cooperation and consent of the American Academy of Pediatrics for neurology to establish a special training program to be accredited by the Accreditation Council for Graduate Medical Education (ACGME). The head of the pediatric division of the ACGME at that time was F. Howell Wright, one of my regular squash partners and my children's pediatrician. I was delegated by our committee to discuss the situation with him, and that together with subsequent input from others, resulted in the decision that the regulation of training in child neurology would rest with neurology. I began my full-time appointment in January 1964, as Assistant Professor of Neurology, Pediatrics and Anatomy. Douglas Buchanan and Richard Richter both retired in 1966, initiating a new era without the founding chiefs with clinical duties of inpatient attending 4 months a year and outpatient clinic 3 half days a week.

A Career in Research

By the spring of 1960 my laboratory was equipped and ready to function, and I hired my first technician. Never having had that experience, and recognizing the importance of making a good choice, I sought advice from my neighbor on the first floor of the Anatomy Building, Isadore Gersh, one of the pioneers in posterior pituitary anatomy and function. Gersh was brief, stating, "You should hire a middle-aged woman who is an immigrant and a Roman Catholic." This characterized his technicians aptly and they were noticeably industrious, but I was puzzled. I told him I understood the middle age and immigrant status but, as Gersh was clearly Jewish, the reason for the Roman Catholic requirement eluded me. His reply was, "Bob, they work not only for you but for God." My first technician was a woman from Latvia who had lost her husband in World War II and spent 5 years in a refugee camp in Germany before coming to America. She was a gem as were all but one of the additional technicians hired using the same criteria. With a laboratory, equipment, and staff, it was time to begin research. My objective was to follow the example then being established by Walle Nauta with his work placing connectionist neuroanatomy into a strong functional context. Nauta had recently developed the most powerful anatomical tract tracing method available (Nauta, W.J.H., and Gyax, P.A. Silver impregnation of degenerating axons in the central nervous system: A modified technique. *Biotech. Histochem.* 1954; 29:91-93), and I began to attempt to use it. Despite long,

and intensive efforts, I was unable to stain degenerating axons and by the fall of 1960 I was quite discouraged. The 7th International Congress of Anatomy was held in New York that fall and funds permitted attendance. I took the train from Chicago to New York with the new experience of spending the trip in a roomette, a small space containing a seat, a toilet, and a bed that folded down from the ceiling at night. Shoes were placed at bedtime in a small cubicle and miraculously appeared in the morning shined. The train was called the 20th Century Limited, and in the morning it rolled along the Hudson River, a beautiful sight. I had never been to New York and that added to the pleasure of a wonderful experience with two high points. The first was meeting Walle Nauta. He gave a talk in one session and I waited after it to inquire whether I might ask him some questions about his method. This was at noon and, forsaking lunch, he sat down and asked me about my experiences with it. Learning of my frustration, he asked me to take some notes and for the next 2 hours he took me through the method in detail with numerous "if this goes wrong, you do that" and so on. When I returned to Chicago, all went as he had expected and I never had a problem with the method again. The second highlight of the trip was the meeting banquet. Somehow, the anatomists had managed to obtain exclusive use of the Metropolitan Museum of Art for the evening. As I was entering the museum, Nauta and another neuroanatomist of Dutch origin, Hans Kuypers, were also at the door. Nauta asked whether I would like to join them going through the museum and I agreed. Thus began my first lesson in Dutch art. Nauta was from a cultured family, and one aunt was a well-known art critic. For Nauta, the Rijksmuseum, the Stadeljk, and the Mauritshuis and others were familiar places. We spent nearly the whole evening with the Dutch collection. I have never known a scientist more gracious and unassuming than Nauta. We remained friends until his death.

With success using Nauta's method, I was ready to apply for funding and submitted two grants in 1961, "A Comparative Study of Tectothalamic Connections" and "Anatomical Studies of Brain Monoamine Neuron Systems." Both were funded in 1962 and were continuously funded, with evolving research topics, for many years. The study of tectothalamic connections was largely devoted to analysis of auditory pathways. The first paper from this presented a new analysis of projections from the inferior colliculus to the thalamus, particularly the medial geniculate body in the cat (Moore and Goldberg, 1963). Jay Goldberg, my oldest friend, was a student of Dewey Neff and we had collaborated as students. Following his Ph.D., he did a post-doctoral fellowship with Clinton Woolsey and Jerzy Rose at the laboratory of Neurophysiology, University of Wisconsin. Rose and Woolsey had done extensive studies of thalamocortical connections and Jay had discussed this at length with them. We concluded from our study that the medial geniculate body had two divisions that received auditory projections, a pars principalis (MGp) and pars magnocellularis (MGm), and another that was really part of

the pulvinar-posterior complex receiving no auditory input. The same organization was demonstrated in the monkey (Moore and Goldberg, 1966). We were concerned, however, about the shadow of Cajal. In the *Histologie*, Cajal described the fibers of the lateral lemniscus, the ascending pontine auditory pathway, bypassing the inferior colliculus and ascending directly to the medial geniculate. If true, this would render whatever projections came from the inferior colliculus as secondary. We set out to test Cajal's conclusion. This required producing a very small lesion transecting the lateral lemniscus but leaving the immediately adjacent fibers of the spinothalamic tract intact. It soon became evident that this was not possible using stereotaxic surgery. As I have described earlier, I had had extensive experience performing manual microsurgery and I began making lateral lemniscus lesions under direct vision by removing dorsal cerebellum to expose the lateral brainstem and inserting a microknife into the lemniscus. With some practice we obtained several brains with restricted lesions, and a number of controls. The result was a very clear demonstration that all lateral lemniscus axons terminate in the inferior colliculus, and none ascend to the geniculate. Again, the same organization was shown in the monkey (Goldberg and Moore, 1967). Thus, the inferior colliculus is an obligatory pathway in the ascending brainstem auditory system. From our material it was quite clear that Cajal had mistaken the spinothalamic tract for the lateral lemniscus, one of his few errors. There were other studies on the auditory system but, as I shall relate later, we shifted to an area where functional correlations were more readily explored, the visual system. My laboratory was moved in 1964 to the Wyler Children's Hospital, where additional space allowed more personnel and trainees.

Monoamines in the Brain

Part of my graduate work had been a study funded at first by the Wallace Company, developer of meprobamate (Miltown), a widely used tranquilizer. The mechanism of action was not known and Hunt thought it might act through sleep mechanisms. A new faculty member in the Department of Pharmacology, Alfred Heller, and another Hunt graduate student, John Harvey, and I began a study in which stereotaxic lesions were placed in various brain regions and, after recovery, a determination of sleeping time was made. Sleeping time was a standard method to quantify the effects of sedatives in which the experimental compound was injected intravenously, rats were placed on their backs, and the time until they first rolled over was measured. Our control was a short-acting barbiturate. We found a highly significant doubling of sleep time with both compounds with lesions ablating the septal area and the pontine tegmentum but not lateral hypothalamus (Harvey, J.A., Heller, A., Moore, R.Y., Hunt, H.F., and Roth, L.J. Effect of central nervous system lesions on barbiturate sleeping time in the rat. *J. Pharmacol. Exptl. Therap.*, 144:24-36, 1964). John Harvey and I had

spent an enormous amount of time on this and, to this day, I do not understand the results. Meprobamate soon was found not to be very effective and was supplanted as a tranquilizer of choice by chlordiazepoxide (Librium). We published the barbiturate results and went on to other projects. The major one was an attempt to demonstrate that brain monoamines were associated with specific pathways. The catecholamines were discovered in the early 20th century, but their presence in brain was not established until the work of Marthe Vogt (1954; Vogt, M. The concentration of sympathin in different parts of the central nervous system under normal conditions and after the administration of drugs. *J Physiol (Lond)*.1954; 123:451–481), and dopamine was not recognized as a separate transmitter until 1957. Serotonin was isolated and characterized in 1948 and shown in brain shortly thereafter. Early work on brain monoamines was hampered by the lack of efficient, reliable, and sensitive methods for assay.

In the mid-1950s, a spectrophotofluorometer was marketed and assay of all monoamines became routine (Udenfriend, S. Development of the spectrophotofluorometer and its commercialization. *Protein Sci.* 1995;4:542–551). Al Heller quickly understood the potential this had for study of brain monoamines and obtained one of the early instruments. During my postdoctoral fellowship, I had become proficient with stereotaxic surgery and we designed a study to test the hypothesis that monoamines in brain were associated with specific pathways. The fluorometric analyses at that time were only sensitive enough for either pooled regional concentration determinations or whole-brain levels. I had developed an interest in hypothalamus, particularly in the lateral hypothalamic syndrome. We had developed feeding techniques to obtain a high level of survival of these profoundly aphagic animals so that we could perform bilateral lesions in rats. As in the sleeping study, we placed other lesions in the septal area and lateral pontine isthmus region with cortical lesions as controls.

The first monoamine we were able to assay reliably was serotonin, and our first experiment had what, in our eyes, was a beautiful result. Lateral hypothalamic lesions produced decreases in whole-brain serotonin of about 50%, but the other lesions were not as effective (Harvey et al., 1963). In succeeding experiments, we showed that both serotonin and norepinephrine were affected and that all forebrain structures rostral to the lesion showed similar changes (Moore et al., 1965; Heller et al., 1966). I performed an experiment in which each of our lesion types were made in rats and the brains were prepared using the Nauta method. This showed degenerating axons in the vicinity of the lesions but none in the rest of the forebrain. My interpretation of this evidence was that the distant effects of our lesions were a consequence of transsynaptic changes, and I convinced my colleagues that this was correct. It was not. Subsequent work showed that the monoamine axons were very small and unmyelinated, below the resolution of the Nauta method. This was demonstrated almost immediately by the work of Swedish groups that developed the fluorescent histochemical, or Falck-Hillarp,

method (Falck, B., Hillarp, N.-Å., Thieme, G., and Torp, A. Fluorescence of catechol amines and related compounds condensed with formaldehyde. *J Histochem Cytochem.* 1962;10:348–354). With this, small axons below the limits of light microscope resolution are visualized readily with fluorescence microscopy because of diffusion of the signal. The origin of the fluorescence histochemical method and its history has been discussed in a recent paper by Tomas Hökfelt (Hökfelt, T. Looking at neurotransmitters in the microscope. *Prog Neurobiol.* 2010;90:101–118). Bengt Falck visited the University of Chicago for a lecture in early 1963. As soon as he presented the method, its power to study brain monoamine systems was evident and, after the lecture, I inquired whether I might visit him to learn it. He was most interested and I spent 6 weeks in Lund in the fall of 1963. Falck and his family greeted me warmly and I spent the last 3 weeks as a guest in their house. We became fast friends and have remained so since. Over the next 10 years, I visited Lund frequently carrying out a collaborative research program that was quite productive. In 1971, I did a sabbatical in Lund. This began in May and continued through the fall. On prior trips I had begun working with Anders Björklund, who was then a student in the laboratory. Bengt was not devoted to neuroscience studies but encouraged Anders and others to develop that field.

Adventures in the Rhythm Trade

In the 1960s, I did a study of retinal projection in collaboration with Marcel Frankel, a resident in ophthalmology, who had learned the technique of making small lesions in the retina with a laser (Moore, R. Y., Karapas, F., and Frenkel, M. Lateral geniculate projection from discrete retinal lesions in the cat. *Am. J. Ophthalmol.* 1966;62:918–925). Our study was done in cats with the Nauta method for staining degenerating axons, and it required me to learn the visual system in a detail I had not done previously. I found this fascinating and began to study the rest of the visual system in detail. As this proceeded, I began a collaboration with Julius Axelrod and Richard Wurtman to determine the central pathways of pineal control. This was not long after Axelrod had demonstrated the metabolic pathway for melatonin production, and Wurtman had come as a postdoctoral fellow to his laboratory with an interest in pineal function. My collaborator at Chicago, Al Heller, spoke with Axelrod at a FASEB meeting, and plans for a study between Chicago and the NIH were put in place. Axelrod's suggestion was that we could identify central pathways involved in pineal regulation.

Service for the Neuroscience and Academic Communities

My first service beyond local activities in Chicago was as a member of the Maternal and Child Health Program Project Review Committee of the

NICHD beginning in 1972. This was a broad committee and I was one of two neurologists serving. It introduced me to the review process and initiated my education in “committeeship.” The next year I was appointed to the Pediatric Subcommittee of the FDA Neuropharmacology Agents Advisory Committee. Our major charge was to review the use of antipsychotic drugs in children and provide a report. In 1976 I was appointed to the Psychobiology Study Section of the Division of Research Grants. This committee reviewed a very broad range of grants from standard brain–behavior studies to field studies of animal behavior. I remember one in which the major expense was the purchase of a Land Rover for a field study of primate behavior in Africa. Overlapping this, I served on a VA Career Development Committee that covered neurology, gastroenterology, and nephrology. I remember, with some feeling of chagrin, how confidently we reviewed applications that covered subjects that a substantial part of the committee had no real basis for critical judgment. It was a process of trusting your colleagues. The meetings were held at the VA Central Office on McPherson Square, and this location gave me my first experience with the elegance and institutional beauty of our capital. I have always enjoyed Washington, D.C. When I first went to Bethesda for meetings, it had the feeling of a small town. There were two hotels, the In Town and the Bethesdan. Restaurants were few and limited in scope. The changes since those times are amazing.

In 1980, I was appointed to a National Academy of Sciences Panel that was to study the Health Related Issues of Marijuana Use. The committee was chaired by Arnold Relman, Editor of the *New England Journal of Medicine*, and had a very diverse membership. I was the member assigned to cover neurological issues. The committee worked for 2 years to prepare a report of which we were all proud. Part of the work was public hearings, which were interesting. The report made news for a short period and was, I think, quite influential in the long run. Following this, I served for 4 years on the NIMH Board of Scientific Counselors. This was followed by 4 years on the Board of Scientific Counselors of NINDS, 2 years as Chair with a subsequent 4 years, 1992–1996, as Chair of the NIMH Board of Scientific Counselors. Service on these boards was both a rewarding and frustrating experience. When I first started the work, the NIH was in its heyday. Funds were abundant as was transient staff such as fellows. NIH scientists are reviewed every 4 years. In my first tour of duty on the NIMH board, the reviews were perfunctory, no changes were made in response to recommendations, and it was a pro forma process. Then money became tighter and new intramural research directors were under more pressure to justify the allocation of funds. The essential problem was, and is, how to deal with a tenured scientist who gradually loses productivity and whose work has diminishing impact. In the academic world this is accomplished by increasing teaching or administration. No such opportunities exist at NIH. In addition,

NIH scientists had been able in some circumstances to add other permanently appointed scientists. This resulted in some very large groups, particularly in clinical research. When these lost vitality, the problems were compounded. The NIH continues to struggle with this problem. I have served on a number of ad hoc panels and committees, advisory boards, and review boards. Among these was a 4-year appointment on the NASA National Space Biomedical Research Institute National Advisory Council. I have also served on 14 editorial boards, including a 25-year term of service on the Brain Research board. Although this has some rewards, done properly it requires a substantial commitment of time and intellectual energy.

Two experiences in industry have been informative. The first was as an advisor for the *Institute de Recherches Internationales Servier*. Servier is a French pharmaceutical company. The work was interesting, but the major benefit was many trips to Paris. On some occasions, they would call a meeting at short notice requiring me to travel on the Concorde. The second experience has been with the American company, Cephalon, Inc. I have consulted for the company from its founding in 1987 to the present. It went from the two founders in an office to an international company with annual sales exceeding \$2 billion. The difference between academic science and drug development is considerable, and the latter is critical for translation of academic science. Both are valuable, and understanding industry is increasingly important for the academic scientist.

I will recount two other advisory experiences. The first is a 12-year service, 1996–2008, on the Board of Directors of the National Sleep Foundation, a small foundation dedicated to increasing public knowledge of the importance of sleep and sleep disorders. The requirements for a foundation differ from any of the other advisory roles I have played. First, the knowledge needs to be packaged and distributed effectively. For this a cooperative interaction between scientists and clinicians and media experts is needed. Second, to exist and function, the foundation needs to raise funds. The Board oversees these, and I have been impressed by the commitment of uncompensated board members to the foundation. The second is service on the President's Committee on the National Medal of Science. I was appointed to the Presidential Commission by President George Bush in 2006 for a 3-year term, which was extended with a second term of 3 years in 2009. The National Medal of Science is given to eight scientists from all areas of science each year. Individuals are nominated and the nominations are reviewed by the committee that is comprised of 12 individuals. I am one of three biological scientists, and the only medical scientist on the committee. The challenge for us is selecting from the number of outstanding scientists nominated. The service on this committee is very gratifying, and we have the pleasure of attending the medal presentation by the President at the White House each year.

Serving on the Faculty of Several Institutions

I started on the faculty of the University of Chicago in 1959 and had every intention of finishing my career there. For me, my major appointment was in the Division of Neurology, even though I held appointments in Pediatrics and Anatomy. The Division prospered from 1966 through 1973 with increasing faculty and activity. The group worked together well and was very compatible. But, as happens with a young faculty, people were lured away by other institutions and one young woman tragically committed suicide after her fiancé died of an acute leukemia. As a Division, we were required to obtain approval for new appointments from the Chair of the Department of Medicine. There was, at that time, a new Chair who felt Neurology was not a major priority for that Department and refused to approve new appointments. As a consequence, the clinical load became overwhelming for those remaining and all, save the division head, left in 1973–1974. I received an offer from the University of California, San Diego, to join the Department of Neurosciences and agreed. We arrived in San Diego in July 1974. Our house was in La Jolla at the bottom of Mount Soledad and a half mile from the beach. The unvarying good weather was a real change from Chicago, and it took a couple of years to discern that the lack of seasons was a bit disconcerting. UCSD had a VA Hospital and what was called the “University Hospital.” Its previous incarnation had been as the San Diego County Hospital and this was how the public saw it. Consequently, the clinical load was rather light and much lighter than I experienced in Chicago. The advantage was increased lab time, and my years in San Diego were very productive. Another advantage was that recruiting students and postdocs to San Diego was much easier than to the south side of Chicago. I took up running for exercise and reached a level of running two marathons. About 2 years after we arrived, one of my sons was having some problems in school and I began investigating. He was using marijuana and not attending to school. In the course of reviewing his problem I found that the school curricula, both primary and secondary, did not have the rigor we had experienced at the University of Chicago Laboratory School. In addition, school funding in California was low then, as it is now. Finally, the level of cultural activity in San Diego was low; it was very much an “outdoor” society. With all of these issues, we decided to leave California in 1979. Two years before, I had been offered the Chair of Neurology at the University of Michigan but turned it down after long consideration. In 1979, I was interviewed, and subsequently offered the chair at the University of Wisconsin and the State University of New York at Stony Brook. Stony Brook presented an interesting challenge. The medical school was only 10 years old and had just completed building a new University Hospital. The Department of Neurology had not been formed and only two neurologists had been hired. There was new laboratory space and, thus, a founding chair would have responsibility for developing a clinical

service, hiring a faculty, and developing research programs. It was too intriguing to pass up. We opened the hospital in 1980 with 10 psychiatric beds and gradually increased the census to about 300 beds. In all, I spent 12 years at Stony Brook. By the end of that time, the department had 15 faculty members, a busy practice, multiple research grants covering both clinical and basic topics, a residency training program that was recruiting excellent American graduates, and it ran a substantial surplus. The other interesting component of this start-up operation was to establish a practice plan for the clinical departments. I learned an immense amount about the business of medicine and became Vice President of the plan. Over the next 10 years it became a business with revenue much in excess of \$100 million per year. But, by 1989, I felt the important challenges had been met, I was too deeply into administrative activities to actively run a laboratory, and it clearly was time to move on. I felt that it would be difficult to recruit a new chair with the old one looking over his or her shoulder, so I sought opportunities elsewhere.

I had served on an external advisory committee at the University of Pittsburgh for several years, and the Dean of the Medical School asked me what he could do to get me to come to Pittsburgh at each advisory committee visit. Early in 1990 he put the same question to me and I replied, "Give me something interesting to do." I was made Professor of Neurology and Psychiatry and Director of the Center for Neuroscience. The Center was without walls and its major functions were to establish and administer a graduate program that would serve all neuroscience faculty independent of departmental affiliation and to promote interaction among neuroscientists. This was not burdensome and went quite well. It left ample time for research. In 1992, I was asked to be Director of the Alzheimer Disease Research Center funded by NIA. This was to be temporary and only until a competing renewal of the grant was prepared. After this was completed I was asked by the President of the University to head a group of University of Pittsburgh faculty to work with a Carnegie Mellon University group to prepare a grant proposal for the RK Mellon Foundation to establish a Center for the Neural Basis of Cognition that would be a joint function of the two universities. We successfully raised \$12 million that allowed renovating space for the Center, the recruitment of new faculty, and establishing a cognitive neuroscience graduate program. All of these activities went well and the Center has been an enormous success. I resigned as Co-Director of the Center in 1996 when I agreed to become Chair of the Department of Neurology. The Department was in a crisis of leadership, and I was expected to get it on an even keel. Directing a clinical department is rarely an easy task, and it was not so on this occasion. At age 65 I was not interested in a long-term involvement and was able to withdraw in 2000.

In today's clinical departments, particularly in the cognitive specialties, the three most urgent problems are money, money, and money. The chairs

have become CFOs and the major attribute required for a clinical chair is financial probity. I took a sabbatical in 2000 in the MRC Cyclotron Unit, Hammersmith Hospital, London. In 1996 I had limited my clinical practice to movement disorders. The head of the MRC Unit, David Brooks, has done much of the important positron emission tomography (PET) work on Parkinson's disease. It was a very productive 6 months and London is a magnificent city. We lived in a mews apartment next to Hyde Park in Knightsbridge, two blocks from Harrods. When I returned, I became interested in developing research in movement disorders at Pittsburgh. A patient had endowed a chair for me, the Love Family Professorship, and with his encouragement, and help from the family of another patient, Michael Zigmond and I were encouraged to approach a foundation, the Scaife Family Foundation, with a proposal to develop a multidisciplinary program in neurodegenerative disease. After a long courtship, the Foundation agreed to provide \$10 million if it was matched. The University of Pittsburgh Medical Center graciously provided matching funds and the Pittsburgh Institute for Neurodegenerative Disease now exists in new laboratory space headed by a distinguished Parkinson's disease investigator, J. Timothy Greenamyre. And I have been gratefully free of any administrative duties for more than 5 years. In 2009, I retired from clinical practice, feeling that 50 years of neurology would suffice. I shall retire at the end of September 2010, and we plan to move to the San Francisco area to be near children and grandchildren.

Family

I have commented on my family origins but not on our life together nor on my own experiences as a husband and father. My mother was not a nurturing person and, as I later deduced, was given to intermittent depressive episodes of varying severity. As she grew older, and my recollections are most accurate for the period of her 40s and beyond, she became increasingly unable to interact with people, largely being by herself. In her early 50s, my Grandfather Fisher died suddenly of a myocardial infarct. This affected her greatly and she developed a delusion that people on the street were talking about her and saying, "There's the woman that killed her father." Other delusions and deep depression followed, and she was hospitalized and received electroshock therapy. She never recovered to any approximation of normal function and by her early 60s she required custodial care in a nursing home. Despite this, she was basically a kindly person but never played an important role in my life beyond the age of about 10.

My father was almost a complete opposite. He was open, friendly, and naturally gregarious. We spent a good deal of time together. He was especially important during the period of onset of my panic disorder. During my last undergraduate year, I developed episodes of anxiety that evolved into a

full panic disorder, a disorder not recognized at that time. Fortunately, I was largely engaged in independent study at school and could function some. My father provided understanding and support in a way hard to express. This continued throughout his active life. He was a major encouragement for me to pursue my objectives. After my mother was placed in a nursing home, he moved back to Georgia and purchased a small plot of land on the family farm from his sister and grew vegetables and fruit. That was the happiest I had ever seen him. His routine was to arise early and tend to the farm until about noon. After lunch he would begin "visiting," going around the area talking to friends and strangers. In the late 1970s, it became apparent that he was cognitively impaired and my brother and I placed him in the nursing home where my mother resided.

Over the years his siblings all died, save a younger brother who lived in Atlanta. I kept in touch with him until he and his wife died about 10 years ago. They were childless and I lost contact with other Georgia relatives. It was heartbreaking to watch his descent into profound dementia. He died in 1992 not knowing who he was or where he was. My mother remained intellectually intact, albeit with her depression, until she had a basilar artery thrombosis in 1986. My brother had chronic ulcerative colitis from the age of 7 with multiple hospitalizations. Nevertheless, he functioned well as a purchasing agent, married and had two children. In mood and interests he was very much like my father. He had a colectomy in 1983 and did exceptionally well, having the most active and healthy period of his adult life. In 1986, he developed angina and was scheduled for a stress test. He died of cardiac arrest during it at age 48. His son continues to live in the Chicago area and we communicate occasionally. His daughter attended college but did not graduate. She took a clerical job but was prone to depression. She simply disappeared one day without any communication and has never been heard from again. His wife left the area and we have not been in contact.

I was married to Colston Nauman, a graduate student with Neff, in 1959. She is from an old family in Harrisburg, Pennsylvania. We have three children, Betsy born in 1961, Matthew in 1963, and Joshua in 1965. Colston had an acute psychosis in 1967 and moved to live by herself, leaving me with the challenge of three children. She became well enough in a few months to take them for a few hours to a day at a time and that continued until they went to high school. Each time I have moved, she has followed so that her relations with the children are quite good. She is independently wealthy, which facilitates things. I was able to hire a woman for child care who lived with us, and the children did better than I would have predicted. In 1969 I married a former graduate student, Jean Kavanagh, and we had one son, Thomas Moore, born in 1972. About 6 weeks after birth he was found to have the inherited metabolic defect, phenylketonuria. The dietary treatment

for this was developed in the early 1960s and prognosis with dietary control was not known. Fortunately, Tom has done well and now works as a TV director in Los Angeles, but his early care and anxiety about his future put great strain on our marriage. The other children are also doing well. Betsy and Joshua both live in Silicon Valley and have good jobs in the information science business. Matthew lives in New York, works in interior design, largely for business, and has his own firm. He also teaches at the Fashion Institute of Technology. Only Tom is married. Jean left me in 1986, and I was married to an industrial toxicologist, Cynthia Driscoll in 1991. That did not work out either, and I am now married to Jane DeYoung. Jane was my departmental administrator in New York and we have been friends for 30 years. We were married in 1997 and have been splendidly happy since. We now live quietly in a Pittsburgh suburb, Fox Chapel, with our two Norwich terriers, Satchel and Sabrina.

Summation

I have long held that research university faculty are among the most privileged and pampered people in the world. They have few duties and almost unlimited time to study the scientific universe of their choosing. Tenure is obtained at a young age and their lives are secure. Compared to my father, who worked as a salesman in an environment of unease and uncertainty, I feel extremely fortunate. I have always looked forward to returning each day to my office and laboratory to indulge my curiosity. Teaching is one of the most pleasurable activities I know, both in the classroom and the laboratory. It is an exploration of knowledge with a group of intelligent, curious, and energetic people who ask only that you come prepared and present clearly. They enjoy it even more if you make it interesting. I have been able to travel the world through science. The most uncomfortable thing that happens in my academic life is a long, boring faculty meeting. Through my life I have had brief periods, such as on sabbatical, when I did not practice or teach neurology. It has always made me feel somewhat lost. There is, in my mind, no greater trust than that placed in a physician by a patient. One is asked to listen and understand. The process in neurology consists of taking and interpreting a history, the patient's story of illness. A physical examination is performed to verify hypotheses reached from the history. This information is then used to make a localizing diagnosis: what part of the nervous system is malfunctioning? With this it is hopefully possible to reach a judgment about the etiology and pathophysiology of the problem. Further information from the laboratory or imaging may be required. The final step is therapy and assessment of its efficacy. The process requires communication between patient and physician and, particularly, understanding and empathy from the physician.

Selected Bibliography

- Abrahamson, E.A., and Moore, R.Y. Lesions of suprachiasmatic nucleus efferents selectively affect rest-activity rhythm. *Mol. Cell. Endocrinol.*, 252:46–56, 2006.
- Abrahamson, E.A., and Moore, R.Y. The posterior hypothalamic area: Chemoarchitecture and afferent connections. *Brain Res.*, 889:1–22, 2001.
- Abrahamson, E.A., Leak, R.K., and Moore, R.Y. Suprachiasmatic nucleus projections to posterior hypothalamic arousal areas. *NeuroReport*, 12: 435–440, 2001.
- Abrahamson, E.E., and Moore, R.Y. Suprachiasmatic nucleus in the mouse: Retinal innervation, intrinsic organization and efferent projections. *Brain Res.* 916: 172–191, 2001.
- Aguilar-Roblero, R., Morin, L.P., and Moore, R.Y. Morphological correlates of circadian rhythm restoration induced by transplantation of the suprachiasmatic nucleus in hamsters. *Exper. Neurol.*, 130:250–260, 1994.
- Aguilar-Roblero, R., Shibata, S., Speh, J.C., Drucker-Colin, R., and Moore, R.Y. Morphological and functional development of the suprachiasmatic nucleus in transplanted fetal hypothalamus. *Brain Res.*, 580:288–296, 1992.
- Axelrod, J., Snyder, S.H., Heller, A., and Moore, R.Y. Light-induced changes in pineal hydroxyindole-0-methyltransferase: Abolition by hypothalamic lesions. *Science*, 154:898–899, 1966.
- Bjorklund, A., Moore, R.Y., Nobin, A., and Stenevi, U. The organization of tuberohypophyseal and reticulo-infundibular catecholamine neuron systems in the rat brain. *Brain Res.*, 51:171–191, 1973.
- Bohnen, N.I., Constantine, G.M., Mathis, C.A., and Studenski, S.A., and Moore, R.Y. Selective hyposmia and nigrostriatal dopaminergic denervation in Parkinson's disease. *J Neurol.*, 254:84–90 2007.
- Bohnen, N.I., Gedela, S., Herath, P., Constantine, G.M., and Moore, R.Y. Selective hyposmia in Parkinson disease: Association with hippocampal dopamine activity. *Neurosci. Lett.*, 447:12–16, 2008.
- Bohnen, N.I., Gedela, S., Kuwabara, H., Constantine, G.M., Mathis, C.A., Studenski, S.A., and Moore, R.Y. Selective hyposmia and nigrostriatal dopaminergic denervation in Parkinson's disease. *J. Neurol.*, 254:84–90, 2007.
- Bohnen, N.I., Kaufer, D.I., Hendrickson, R., Constantine, G.M., Mathis, C.A., and Moore, R.Y. Cortical cholinergic denervation is associated with depressive symptoms in Parkinson's disease and parkinsonian dementia. *J Neurol. Neurosurg. Psychiat.*, 78:641–643, 2007.
- Bohnen, N.I., Kuwabara, H., Constantine, G.M., Mathis, C.A., and Moore, R.Y. Grooved Pegboard test as a biomarker of nigrostriatal denervation in Parkinson's Disease. *Neurosci. Lett.*, 424:185–189, 2007.
- Bohnen, N.I., Studenski, S.A., Constantine, G.M., and Moore, R.Y. Diagnostic performance of clinical motor and non-motor tests of Parkinson disease: a matched case-control study. *Eur. J. Neurol.*, 15:685–691, 2008.
- Buysse, D.J., Nofzinger, E.A., Germain, A., Meltzer, C.C., Wood, A., Ombao, H., Kupfer, D.J., and Moore, R.Y. Regional brain glucose metabolism during morning and evening wakefulness in humans. *Sleep*, 27:1245–1254, 2004.

- Card, J.P., and Moore, R.Y. Neuropeptide Y localization in the rat suprachiasmatic nucleus and periventricular hypothalamus. *Neurosci. Lett.*, 88:241–246, 1988.
- Card, J.P., and Moore, R.Y. Organization of lateral geniculate-hypothalamic connections in the rat. *J. Comp. Neurol.*, 284:135–147, 1989.
- Card, J.P., and Moore, R.Y. The suprachiasmatic nucleus of the golden hamster: Immunohistochemical analysis of cell and fiber distribution. *Neurosci.*, 13: 415–431, 1984.
- Card, J.P., and Moore, R.Y. Ventral lateral geniculate nucleus efferents to the rat suprachiasmatic nucleus exhibit avian pancreatic polypeptide-like immunoreactivity. *J. Comp. Neurol.*, 206:390–396, 1982.
- Card, J.P., Brecha, N., and Moore, R.Y. Immunohistochemical localization of avian pancreatic polypeptide-like immunoreactivity in the rat hypothalamus. *J. Comp. Neurol.*, 217:123–136, 1983.
- Card, J.P., Brecha, N., Karten, H.J., and Moore, R.Y. Immunocytochemical localization of vasoactive intestinal polypeptide-containing cells and processes in the suprachiasmatic nucleus of the rat: Light and electron microscopic analysis. *J. Neurosci.*, 11:1289–1303, 1981.
- Card, J.P., Enquist, L.W., and Moore, R.Y. Neuroinvasiveness of pseudorabies virus injected intracerebrally is dependent on viral concentration and terminal field density. *J. Comp. Neurol.*, 407:438–452, 1999.
- Card, J.P., Riley, J.N., and Moore, R.Y. The motor trigeminal nucleus of the rat: Analysis of neuronal structure and the synaptic organization of noradrenergic afferents. *J. Comp. Neurol.*, 250:462–468, 1986.
- Cassels, D.E., and Moore, R.Y. Sympathetic innervation of the ductus arteriosus in relation to patency. *Chest*, 63:727–731, 1973.
- Cassone, V.M., Roberts, M.H., and Moore, R.Y. Melatonin inhibits metabolic activity in the rat suprachiasmatic nuclei. *Neurosci. Lett.*, 81:29–34, 1987.
- Cassone, V.M., and Moore, R.Y. Retinohypothalamic projection and suprachiasmatic nucleus of the house sparrow, *Passer domesticus*. *J. Comp. Neurol.*, 266: 171–182, 1987.
- Cassone, V.M., Speh, J.C., Card, J.P., and Moore, R.Y. Comparative anatomy of the mammalian hypothalamic suprachiasmatic nucleus. *J. Biol. Rhythms*, 3:71–91, 1988.
- Eichler, V.B., and Moore, R.Y. The primary and accessory optic systems in the golden hamster *Mesocricetus auratus*. *Acta Anat.*, 89:359–371, 1974.
- Fallon, J.H., and Moore, R.Y. Catecholamine innervation of the basal forebrain. III. Olfactory bulb, anterior olfactory nuclei, olfactory tubercle and piriform cortex. *J. Comp. Neurol.*, 180:533–544, 1978.
- Fallon, J.H., and Moore, R.Y. Catecholamine innervation of the basal forebrain. IV. Topography of the dopamine projection to the basal forebrain and neostriatum. *J. Comp. Neurol.*, 180:545–579, 1978.
- Fallon, J.H., and Moore, R.Y. Superior colliculus efferents to the hypothalamus. *Neurosci. Lett.*, 14:265–270, 1979.
- Fallon, J.H., Koziell, D.A., and Moore, R.Y. Catecholamine innervation of the basal forebrain. II. Amygdala, suprarhinal cortex and entorhinal cortex. *J. Comp. Neurol.*, 180:509–531, 1978.

- Fallon, J.H., Riley, J.N., and Moore, R.Y. Substantia nigra dopamine neurons: Separate populations project to neostriatum and allocortex. *Neurosci. Lett.*, 7:157–162, 1978.
- Fallon, J.H., Riley, J.N., Sipe, J.C., and Moore, R.Y. The islands of Calleja: Organization and connections. *J. Comp. Neurol.*, 181:375–396, 1978.
- Fuchs, J.L., and Moore, R.Y. Development of circadian rhythmicity and light responsiveness in the rat suprachiasmatic nucleus: A study using the 2-deoxy[1-¹⁴C] glucose method. *Proc. Nat. Acad. Sci. USA*, 77:1204–1208, 1980.
- Gall, C.M., and Moore, R.Y. Distribution of enkephalin, substance P, tyrosine hydroxylase, and 5-hydroxytryptamine immunoreactivity in the septal region of the rat. *J. Comp. Neurol.*, 225:212–227, 1984.
- Gao, B., and Moore, R.Y. Glutamic acid decarboxylase message isoforms in human suprachiasmatic nucleus. *J. Biol. Rhythms*, 11:172–179, 1996.
- Gao, B., and Moore, R.Y. The sexually dimorphic nucleus of the hypothalamus contains GABA neurons in rat and man. *Brain Res.*, 742:163–171, 1996.
- Gao, B., Fritschy, J.-M., and Moore, R.Y. GABA-A-receptor subunit composition in the circadian timing system. *Brain Res.*, 700:142–156, 1995.
- Gustafson, E.L., and Moore, R.Y. Noradrenaline neuron plasticity in developing rat brain: Effects of neonatal 6-hydroxydopamine demonstrated by dopamine- β -hydroxylase immunocytochemistry. *Dev. Brain Res.*, 37:143–155, 1987.
- Harvey, J.A., Heller, A., and Moore, R.Y. The effect of unilateral and bilateral medial forebrain bundle lesions on brain serotonin. *J. Pharmacol. Exp. Therap.*, 140:103–110, 1963.
- Harvey, J.A., Heller, A., Moore, R.Y., Hunt, H.F., and Roth, L.J. Effect of central nervous system lesions on barbiturate sleeping time in the rat. *J. Pharmacol. Exp. Therap.*, 144:24–36, 1964.
- Heller, A., and Moore, R.Y. Effect of central nervous system lesions on brain monoamines in the rat. *J. Pharmacol. Exptl. Therapeutics*, 150:1–9, 1965.
- Heller, A., Harvey, J.A., and Moore, R.Y. A demonstration of a fall in brain serotonin following central nervous system lesions in the rat. *Biochem. Pharmacol.*, 11:859–866, 1962.
- Heller, A., Seiden, L.S., and Moore, R.Y. Regional effects of lateral hypothalamic lesions on brain norepinephrine in the cat. *Intl. J. Neuropharmacol.*, 5:91–102, 1966.
- Heller, A., Seiden, L.S., Porcher, W., and Moore, R.Y. 5-Hydroxytryptophan decarboxylase in rat brain: Effect of hypothalamic lesions. *Science*, 147:887–888, 1965.
- Heller, A., Seiden, L.S., Porcher, W., and Moore, R.Y. Regional effects of lateral hypothalamic lesions on 5-hydroxytryptophan decarboxylase in the cat brain. *J. Neurochem.*, 13:967–974, 1966.
- Hemmendinger, L.M., and Moore, R.Y. Synaptic reorganization in the motor trigeminal nucleus of the rat following neonatal 6-hydroxydopamine treatment. *J. Comp. Neurol.*, 250:462–468, 1986.
- Johnson, R.F., Moore, R.Y., and Morin, L.P. Loss of entrainment and anatomical plasticity after lesions of the hamster retinohypothalamic tract. *Brain Res.*, 460:297–313, 1988.

- Johnson, R.F., Morin, L.P., and Moore, R.Y. Retinohypothalamic projections in the hamster and rat demonstrated using cholera toxin. *Brain Res.*, 462:301–312, 1988.
- Jones, B.E., and Moore, R.Y. Ascending projections of the locus coeruleus in the rat. II. Autoradiographic study. *Brain Res.*, 127:23–53, 1977.
- Jones, B.E., and Moore, R.Y. Catecholamine-containing neurons of the nucleus locus coeruleus in the cat. *J. Comp. Neurol.*, 157:43–51, 1974.
- Jones, B.E., Halaris, A.E., McIlhany, M., and Moore, R.Y. Ascending projections of the locus coeruleus in the rat. I. Axonal transport in central noradrenaline neurons. *Brain Res.*, 127:1–21, 1977.
- Kafka, M.S., Marangos, P.J., and Moore, R.Y. Suprachiasmatic nucleus ablation abolishes circadian rhythms in rat brain neurotransmitter receptors. *Brain Res.*, 327:344–347, 1985.
- Klein, D.C., and Moore, R.Y. Pineal N-acetyltransferase and hydroxyindole-0-methyltransferase: Control by the retinohypothalamic tract and the suprachiasmatic nucleus. *Brain Res.*, 174:245–262, 1979.
- Klein, D.C., Moore, R.Y., and Reppert, S.M. (Eds.) *The Suprachiasmatic Nucleus—The Mind's Clock*, Oxford Press, New York, 1991.
- Klein, D.C., Weller, J.L., and Moore, R.Y. Melatonin metabolism: Neural regulation of pineal serotonin: Acetyl coenzyme A N-acetyltransferase activity. *Proc. Nat. Acad. Sci. USA*, 68:3107–3110, 1971.
- Kromer, L.F., and Moore, R.Y. A study of the organization of the locus coeruleus projections to the lateral geniculate nuclei in the albino rat. *Neurosci.*, 5: 255–271, 1980.
- Kromer, L.F., and Moore, R.Y. Norepinephrine innervation of the cochlear nuclei by locus coeruleus neurons in the rat. *Anat. Embryol.*, 158:227–244, 1980.
- Leak, R.H., and Moore, R.Y. Identification of retinal ganglion cells projecting to the lateral hypothalamic area of the rat. *Brain Res.*, 770:105–114, 1997.
- Leak, R.K., and Moore, R.Y. Suprachiasmatic nucleus projections in the rat: Retrograde analysis. *J. Comp. Neurol.*, 433: 312–334, 2001.
- Leak, R.K., Card, J.P., and Moore, R.Y. suprachiasmatic pacemaker organization analyzed by viral transynaptic transport. *Brain Res.*, 819:23–27, 1999.
- Levitt, P., and Moore, R.Y. Noradrenaline neuron innervation of the neocortex in the rat. *Brain Res.*, 139:219–231, 1978.
- Levitt, P., Moore, R.Y., and Garber, B.B. Selective cell association of catecholamine neurons in brain aggregates *in vitro*. *Brain Res.*, 111:311–320, 1976.
- Levitt, P.R., and Moore, R.Y. Development of the noradrenergic innervation of neocortex. *Brain Res.*, 162:243–259, 1979.
- Levitt, P.R., and Moore, R.Y. Developmental organization of raphe serotonin neuron groups in the rat. *Anat. Embryol.*, 154:241–251, 1978.
- Levitt, P.R., and Moore, R.Y. Organization of brainstem noradrenaline hyperinnervation following neonatal 6-hydroxydopamine treatment in rat. *Anat. Embryol.*, 158:133–150, 1980.
- Levitt, P.R., and Moore, R.Y. Origin and organization of brainstem catecholamine innervation in the rat. *J. Comp. Neurol.*, 186:505–528, 1979.

- Lindvall, O., Bjorklund, A., Moore, R.Y., and Stenevi, U. Mesencephalic dopamine neurons projecting to neocortex. *Brain Res.*, 81:325–331, 1974.
- Loy, R., and Moore, R.Y. Anomalous innervation of the hippocampal formation by peripheral sympathetic axons following mechanical injury. *Exptl. Neurol.*, 57:645–650, 1977.
- Loy, R., and Moore, R.Y. Ontogeny of the noradrenergic innervation of the rat hippocampal formation. *Anat. Embryol.*, 157:243–253, 1979.
- Loy, R., Koziell, D.A., Lindsey, J.D., and Moore, R.Y. Noradrenergic innervation of the adult rat hippocampal formation. *J. Comp. Neurol.*, 189:699–710, 1980.
- Loy, R., Milner, T., and Moore, R.Y. Sprouting of sympathetic axons in the hippocampal formation: Conditions necessary to elicit ingrowth. *Exptl. Neurol.*, 67:399–411, 1980.
- Moga, M.M., and Moore, R.Y. Organization of neural inputs to the suprachiasmatic nucleus in the rat. *J. Comp. Neurol.*, 389: 508–534, 1997.
- Moga, M.M., and Moore, R.Y. Paraventricular thalamus lesions after circadian period and activity distribution in the blinded rat. *J. Bio. Rhythm Res.*, 31:212–219, 2000.
- Moga, M.M., and Moore, R.Y. Putative excitatory amino acid projections to the suprachiasmatic nucleus in the rat. *Brain Res.*, 743:171–177, 1996.
- Moga, M.M., Weis, R.P. and Moore, R.Y. Efferent projections of the paraventricular thalamic nucleus in the rat. *J. Comp. Neurol.*, 359:221–238, 1995
- Moore, R.Y. Catecholamine innervation of the basal forebrain. I. The septal area. *J. Comp. Neurol.*, 177:665–683, 1978.
- Moore, R.Y. Effects of some rhinencephalic lesions on retention of conditioned avoidance behavior in cats. *J. Comp. Physiol Psychol.*, 57:65–71, 1964.
- Moore, R.Y. Entrainment pathways and the functional organization of the circadian system. *Prog. Brain Res.*, 111:103–119, 1996.
- Moore, R.Y. Indolamine metabolism in the intact and denervated pineal, pineal stalk and habenula. *Neuroendocrinol.*, 19:323–330, 1975.
- Moore, R.Y. Neural control of the pineal gland. *Behav. Brain Res.*, 73:125–130, 1996
- Moore, R.Y. Organization of midbrain dopamine systems and the pathophysiology of Parkinson disease. *Parkinsonism Relat. Disord.*, 9: S65–S71, 2003.
- Moore, R.Y. Organization of the primate circadian system. *J. Biol. Rhythms*, 8:S3–S9, 1993.
- Moore, R.Y. Organum vasculosum lamina terminalis: Innervation by serotonin neurons of the midbrain raphe. *Neurosci. Lett.*, 5:297–302, 1977.
- Moore, R.Y. Pineal transplants to the anterior chamber of the eye: Evidence for functional reinnervation. *Exptl. Neurol.*, 49:617–621, 1975.
- Moore, R.Y. Retinohypothalamic projection in mammals: A comparative study. *Brain Res.*, 49:403–409, 1973.
- Moore, R.Y. Suprachiasmatic nucleus in sleep-wake regulation. *Sleep Med.*, 8: Suppl 3:27–33, 2007.
- Moore, R.Y. Suprachiasmatic nucleus, secondary synchronizing stimuli and the central neural control of circadian rhythms. *Brain Res.*, 183:13–28, 1980.

- Moore, R.Y. The geniculohypothalamic tract in monkey and man. *Brain Res.*, 486:190–194, 1989.
- Moore, R.Y. The organization of the human circadian timing system. *Prog. Brain Res.*, 93:101–117, 1992.
- Moore, R.Y., Abrahamson, E.A., and van den Pol, A.N. The hypocretin neuron system: An arousal system in the human brain. *Ital. Arch. Biol.*, 139:195–206, 2001.
- Moore, R.Y., and Bernstein, M.E. Synaptogenesis in the rat suprachiasmatic nucleus demonstrated by electron microscopy and synapsin I immunoreactivity. *J. Neurosci.*, 9:2151–2162, 1989.
- Moore, R.Y., and Bloom, F.E. Central catecholamine neuron systems: Anatomy and physiology of the dopamine systems. *Ann. Rev. Neurosci.*, 1:129–169, 1978.
- Moore, R.Y., and Bloom, F.E. Central catecholamine neuron systems: Anatomy and physiology of the norepinephrine and epinephrine systems. *Ann. Rev. Neurosci.*, 2:113–168, 1979.
- Moore, R.Y., and Card, J.P. The intergeniculate leaflet: An anatomically and functionally distinct subdivision of the lateral geniculate complex. *J. Comp. Neurol.*, 344:403–430, 1994.
- Moore, R.Y., and Danchenko, R.L. Paraventricular–subparaventricular hypothalamic lesions affect circadian function. *Chronobiol. Intl.*, 19:345–360, 2002.
- Moore, R.Y., and Eichler, V.B. Loss of a circadian adrenal corticosterone rhythm following suprachiasmatic lesions in the rat. *Brain Res.*, 42:201–206, 1972.
- Moore, R.Y., and Goldberg, J.M. Ascending projections of the inferior colliculus in the cat. *J. Comp. Neurol.*, 121:109–136, 1963.
- Moore, R.Y., and Goldberg, J.M. Projections of the inferior colliculus in the monkey. *Exptl. Neurol.*, 4:429–438, 1966.
- Moore, R.Y., and Gustafson, E.L. The distribution of dopamine-B-hydroxylase, neuropeptide Y and galanin in locus coeruleus neurons. *J. Chem. Neuroanat.*, 2:95–106, 1989.
- Moore, R.Y., and Halaris, A.E. Hippocampal innervation by serotonin neurons of the midbrain raphe in the rat. *J. Comp. Neurol.*, 164:171–184, 1975.
- Moore, R.Y., and Klein, D.C. Visual pathways and the central neural control of a circadian rhythm in pineal serotonin N-acetyltransferase activity. *Brain Res.*, 71:17–33, 1974.
- Moore, R.Y., and Leak, R.K. The suprachiasmatic nucleus. In Takahashi, J.S., Turek, F.W., Takahashi, J.S., and Moore, R.Y. (Eds.) *Circadian Clocks*, Plenum Press, New York, pp. 141–179, 2001.
- Moore, R.Y., and Lenn, N.J. A retinohypothalamic projection in the rat. *J. Comp. Neurol.*, 146:1–14, 1972.
- Moore, R.Y., and Rapport, R.L. Pineal and gonadal function in the rat following cervical sympathectomy. *Neuroendocrinol.*, 7:361–374, 1971.
- Moore, R.Y., and Sibony, P. Enkephalin-like immunoreactivity in neurons in the human pineal gland. *Brain Res.*, 457:395–398, 1988.
- Moore, R.Y., and Silver, R. Suprachiasmatic nucleus organization. *Chronobiol. Intl.*, 15:475–487, 1998.

- Moore, R.Y., and Smith, R.A. Postnatal development of a norepinephrine response to light in the rat pineal and salivary glands. *Neuropharmacol.*, 10:315–323, 1971.
- Moore, R.Y., and Speh, J.C. A putative retinohypothalamic projection containing substance P in the human. *Brain Res.*, 659:249–253, 1994.
- Moore, R.Y., and Speh, J.C. GABA is the principal neurotransmitter in the mammalian circadian system. *Neurosci. Lett.* 150:112–116, 1993.
- Moore, R.Y., Bhatnagar, R.K., and Heller, A. Anatomical and chemical studies of a nigro-neostriatal projection in the cat. *Brain Res.*, 30:119–135, 1971.
- Moore, R.Y., Bhatnagar, R.K., and Heller, A. Norepinephrine and DOPA decarboxylase in rat brain following hypothalamic lesions. *Intl. J. Neuropharmacol.*, 5: 287–291, 1966.
- Moore, R.Y., Bjorklund, A., and Stenevi, U. Plastic changes in the adrenergic innervation of the rat septal area in response to denervation. *Brain Res.*, 33:13–35, 1971.
- Moore, R.Y., Gustafson, E.L., and Card, J. P. Identical immunoreactivity of afferents to the rat suprachiasmatic nucleus with antisera against avian pancreatic polypeptide, molluscan cardioexcitatory peptide and neuropeptide Y. *Cell Tiss. Res.*, 236:41–46, 1984.
- Moore, R.Y., Halaris, A.E., and Jones, B.E. Serotonin neurons of the midbrain raphe: Ascending projections. *J. Comp. Neurol.*, 180:417–438, 1978.
- Moore, R.Y., Heller, A., Bhatnagar, R. K., Wurtman, R.J., and Axelrod, J. Central control of the pineal gland: Visual pathways. *Arch. Neurol.*, 18:208–218, 1968.
- Moore, R.Y., Heller, A., Wurtman, R.J., and Axelrod, J. Visual pathway mediating pineal response to environmental light. *Science*, 155:220–223, 1967.
- Moore, R.Y., Karapas, F., and Frenkel, M. Lateral geniculate projection from discrete retinal lesions in the cat. *Am. J. Ophthalmol.*, 62:918–925, 1966.
- Moore, R.Y., Speh, J.C. and Card, J.P. The retinohypothalamic tract originates from a distinct subset of retinal ganglion cells. *J. Comp. Neurol.*, 352:351–366, 1995
- Moore, R.Y., Speh, J.C. Serotonin innervation of the primate suprachiasmatic nucleus. *Brain Res.* 1010:169–173, 2004.
- Moore, R.Y., Speh, J.C., and Leak, R.K. Suprachiasmatic nucleus organization. *Cell Tiss. Res.*, 309:89–98, 2002.
- Moore, R.Y., Weis, R., and Moga, M.M. Efferent projections of the intergeniculate leaflet and the ventral lateral geniculate nucleus in the rat. *J. Comp. Neurol.*, 420:398–418, 2000.
- Moore, R.Y., Whone, A.L., and Brooks, D.J. Extrastriatal monoamine neuron function in Parkinson's disease: An 18F-dopa PET study. *Neurobiol. Dis.*, 29: 381–390, 2008.
- Moore, R.Y., Whone, A.L., McGowan, S., and Brooks, D.J. Monoamine neuron innervation of the normal human brain: An ¹⁸F-DOPA PET study. *Brain Res.*, 982:137–45, 2003.
- Moore, R.Y., Wong, S.L.R., and Heller, A. Regional effects of hypothalamic lesions on brain serotonin. *Arch. Neurol.*, 13:346–354, 1965.
- Moore, R.Y., Ziegler, B., and Bayer, S.A. Monoamine neuron innervation of the hippocampal formation: Alteration by neonatal irradiation. *Exptl. Neurol.*, 60: 318–326, 1978.

- Morin, L.P., Blanchard, J., and Moore, R.Y. Intergeniculate leaflet and suprachiasmatic nucleus organization and connections in the hamster. *Visual Neurosci.*, 8:219–230, 1992.
- Morin, L.P., Goodless-Sanchez, N., Smale, L., and Moore, R.Y. Projections of the suprachiasmatic nuclei and subparaventricular zone and retrochiasmatic area in the golden hamster. *Neurosci.*, 61:391–410, 1994.
- Mosko, S., and Moore, R.Y. Neonatal suprachiasmatic nucleus ablation: Absence of functional and morphological plasticity. *Proc. Nat. Acad. Sci. USA*, 75: 6243–6246, 1978.
- Mosko, S.S., and Moore, R.Y. Neonatal ablation of the suprachiasmatic nucleus: Effects on the development of the pituitary-gonadal axis in the female rat. *Neuroendocrinol.*, 29:350–361, 1979.
- Mosko, S.S., and Moore, R.Y. Neonatal suprachiasmatic nucleus lesions: Effects on the development of circadian rhythms in the rat. *Brain Res.*, 164:17–38, 1979.
- Mosko, S.S., and Moore, R.Y. Retinohypothalamic tract development: Alteration by suprachiasmatic lesions in the neonatal rat. *Brain Res.*, 164:1–15, 1979.
- Mosko, S.S., Erickson, G.F., and Moore, R.Y. Dampened circadian rhythms in reproductively senescent female rats. *Behav. Neural Biol.*, 28:1–14, 1980.
- Nofzinger E.A., Nichols T.E., Seltzer C.C., Price, J., Steppe D.A., Miewald J.M., Kupfer D.J., and Moore, R.Y. Changes in forebrain function from waking to REM sleep in depression: preliminary analysis of (18F)DG PET studies. *Psychiatry Res.*, 31:59–78, 1999.
- Nofzinger, E.A., Buysse, D.J., Miewald, J.M., Meltzer, C.C., Price, J.C., Sembrat, R.C., Ombao, H., Reynolds, C.F., Monk, T.H., Hall, M., Kupfer, D.J., and Moore, R.Y. Human regional cerebral glucose metabolism during NREM sleep in relation to waking. *Brain*, 125:1105–1115, 2002.
- Nofzinger, E.A., Mintun, M.A., Price, J., Meltzer, C.C., Townsend, D., Buysse, D.J., Reynolds III, C.F., Datchile, M.J., Matzzie, J., Kupfer, D.J., and Moore, R.Y. A method for the assessment of the functional neuroanatomy of human sleep using FDG PET. *Brain Res. Protocols*, 2:191–198, 1998.
- Nofzinger, E.A., Mintun, M.A., Wiseman, M.B., Kupfer, D.J., and Moore, R.Y. Forebrain activation in REM sleep: An FDG PET study. *Brain Res.*, 770: 192–201, 1997.
- Reuss, S., and Moore, R.Y. Neuropeptide Y-containing neurons in the rat superior cervical ganglion: Projections to the pineal gland. *J. Pineal Res.*, 6:307–316, 1989.
- Richter, R.B., and Moore, R.Y. Non-invasive central nervous system disease associated with lymphoid tumors. *Johns Hopkins School of Medicine Journal*, 122:271–283, 1968.
- Riley, J.N., Card, J.P., and Moore, R.Y. A retinal projection to the lateral hypothalamus in the rat. *Cell Tiss. Res.*, 214:257–269, 1981.
- Roberts, M.H., and Moore, R.Y. Localization of neuropeptides in efferent terminals of the eye in the marine snail, *Bulla gouldiana*. *Cell Tiss. Res.*, 248:67–73, 1987.
- Roberts, M.H., Bernstein, M.E., and Moore, R.Y. Differentiation of the suprachiasmatic nucleus in fetal rat anterior hypothalamic transplants *in oculo*. *Dev. Brain Res.*, 32:59–66, 1987.

- Shibata, S., and Moore, R.Y. Development of a fetal circadian rhythm after disruption of the maternal circadian system. *Dev. Brain Res.*, 41:313–317, 1988.
- Shibata, S., and Moore, R.Y. Development of neuronal activity in the rat suprachiasmatic nucleus. *Dev. Brain Res.*, 34:311–315, 1987.
- Shibata, S., and Moore, R.Y. Electrical and metabolic activity of suprachiasmatic nucleus neurons in hamster hypothalamic slices. *Brain Res.*, 438:374–378, 1988.
- Shibata, S., and Moore, R.Y. Neuropeptide Y and optic chiasm stimulation affect suprachiasmatic nucleus circadian function in vitro. *Brain Res.*, 615:95–100, 1993.
- Shibata, S., and Moore, R.Y. Neuropeptide Y and Vasopressin effects on suprachiasmatic nucleus neurons *in vitro*. *J. Biol. Rhythms*, 3:265–276, 1988.
- Shibata, S., Cassone, V.M., and Moore, R.Y. Effects of Melatonin on neuronal activity in the rat suprachiasmatic nucleus. *Neurosci. Lett.*, 97:140–145, 1989.
- Shibata, S., Newman, G.C., and Moore, R.Y. Effects of calcium ions on glucose utilization in the rat suprachiasmatic nucleus *in vitro*. *Brain Res.* 426:332–338, 1987.
- Shirakawa, T., and Moore, R.Y. Glutamate shifts the phase of the circadian neuronal timing rhythm in the rat suprachiasmatic nucleus in vitro. *Neurosci. Lett.*, 178:47–50, 1994.
- Smale, L., Cassone, V.M., Moore, R.Y., and Morin, L.P. Paraventricular nucleus projections mediating pineal Melatonin and gonadal responses to photoperiod in the hamster. *Brain Res. Bull.*, 22:263–269, 1989.
- Speh, J.C., and Moore, R.Y. Retinohypothalamic tract development in the hamster and rat. *Dev. Brain Res.*, 76:171–181, 1993.
- Squire, L.R., and Moore, R.Y. Dorsal thalamic lesion in a noted case of human memory dysfunction. *Ann. Neurol.*, 6:503–506, 1979.
- Steeves, T.D.L., King, D.P., Zhao, Y., Sangoram, A.M., Du, F., Bowcock, A.M., Moore, R.Y., and Takahashi, J.S. Molecular cloning and characterization of the human CLOCK gene: Expression in the suprachiasmatic nuclei. *Genomics*, 57:189–200, 1999.
- Stenevi, U., Bjorklund, A., and Moore, R.Y. Growth of intact adrenergic axons in the denervated lateral geniculate body. *Exptl. Neurol.*, 35:290–299, 1972.
- Stenevi, U., Bjorklund, A., and Moore, R.Y. Morphological plasticity of central adrenergic neurons. *Brain Beh. Evol.*, 8:110–134, 1973.
- Tarlov, E.C., and Moore, R.Y. The tecto-thalamic connections in the brain of the rabbit. *J. Comp. Neurol.*, 126:403–421, 1966.
- Thannickal T.C., Moore, R.Y., Nienhuis, R., Ramanathan, L., Gulyani, S., Aldrich, M., Cornford, M., and Siegel, J.M. Reduced number of hypocretin neurons in human narcolepsy. *Neuron*, 27:469–474, 2000.
- Thannickal, T.C., Siegel, J.M., Nienhuis, R., and Moore, R.Y. Pattern of hypocretin (orexin) soma and axon loss, and gliosis, in human narcolepsy. *Brain Pathol.* 13:340–51, 2003.
- Thomas, G.J., Moore, R.Y., Harvey, J.A., and Hunt, H.F. Relations between the behavioral syndrome produced by lesions in the septal region of the forebrain and maze learning in the rat. *J. Comp. Physiol. Psychol.*, 52:527–532, 1959.

- Watkins, W.B., Yen, S.S.C., and Moore, R.Y. Presence of b-endorphin-like immunoreactivity in the anterior pituitary gland of rat and man and evidence for the differential localization with ACTH. *Cell Tiss. Res.*, 215:577-589, 1981.
- Whone A.L., Moore R.Y., Piccini P.P., and Brooks D.J. Plasticity of the nigropallidal pathway in Parkinson's disease. *Ann. Neurol.* 53:206-13, 2003.
- Wilkes, M.M., Kobayashi, R.M., Yen, S.S.C., and Moore, R.Y. Monoamine neuron regulation of LRF neurons innervating the Organum vasculosum laminae terminalis and median eminence. *Neurosci. Lett.*, 13:41-46, 1979.