Forrest Shreve: 1878–1950

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Forrest Shreve was born in Easton, Maryland, on July 8, 1878. He completed his grade and high school training in this small tidewater town, and was graduated from The Johns Hopkins University with the A.B. degree in 1901. He immediately began working toward a Ph.D., choosing to investigate the life history of *Sarracenia purpurea* and becoming deeply interested in the ecology of plants. During the final year of his doctoral training he served as instructor in phanerogamic botany at Cold Spring Harbor and received the Ph.D. degree from Johns Hopkins in June 1905.

During 1905–06 Dr. Shreve held an Adam T. Bruce fellowship as a postdoctoral investigator from The Johns Hopkins University and spent that year, and shorter periods on two later occasions, at the New York Botanical Garden's tropical station at Cinchona, Jamaica. His experiences there gave him an insight of considerable scope into the intricacies of tropical plant ecology. Thirty years later he continued to compare floristic conditions in Jamaica with those occurring along the west coast of Mexico.

For two years (1906–08) Dr. Shreve was associate professor of botany at Goucher College and in collaboration with M. A. Chrysler and F. H. Blodgett devoted much time and effort to a botanical survey of Maryland. This task resulted in the publication of a 533-page report, entitled *Plant Life in Maryland,* under the authorship of the three men.

In 1909 Dr. Shreve married Edith Coffin Bellamy and moved to Tucson, Arizona, to accept an appointment to the staff of the Desert Laboratory of the Carnegie Institution of Washington, a connection he maintained until his retirement in 1947. In 1928 he was placed in charge of the Desert Investigations of the Carnegie Institution and began planning a series of taxonomic and ecological investigations of the floras of all the major desert regions in North America. For four years he felt impelled to work on projects initiated prior to his appointment as head of the Desert Laboratory before devoting his energies to the comprehensive survey of the desert vegetations.

In 1932 he began intensive work on the floristics of the Sonoran Desert, a natural desert region occupying parts of Arizona, California, Sonora, and Baja California. He chose to ignore both state and international boundaries in conducting the study and to recognize instead lines of demarcation drawn by the vegetational assemblages peculiar to, and characteristic of, that particular desert area. He planned to make the observations on the floristics of the region himself, but to enlist others to carry on the taxonomic investigation of its flora. He pushed the project forward with vigor and imagination until gathering war clouds in the late 1930s hampered field, laboratory, and herbarium work. By that time he had accumulated nearly all of the data he needed to complete his account of the vegetational groups he felt he could recognize with assurance. He completed his book covering that aspect of the work, but unfortunately was not permitted to see it through final publication.

Forrest Shreve was a man of quiet demeanor, a bit difficult to know well, but loyal to his friends, kindly in his support, and possessed of a great patience with his associates and employees. He early developed, and retained until his death, an interest in the publication of papers and reports dealing with plants and in several societies whose members shared his own enthusiasm for botany. He was one of the group of men who organized the Ecological Society and he served as secretary-treasurer of that organization from 1915 to 1919. He was its president in 1921. As editor of *Plant World* from 1911 until 1919, he did much to enhance the value of that journal. He was a member of the Association of American Geographers, serving as vice president in 1940, and of the Association of Pacific Coast Geographers, serving as president in 1942. He maintained membership in several other societies supporting work in botany and he contributed to their treasuries when financial difficulties overtook them. He collected plant specimens and kept a herbarium that grew slowly but constantly over a period of nearly half a century and donated his carefully annotated specimens to the herbarium of the University of Arizona when he retired. This herbarium was particularly rich in the ferns of Jamaica.

Papers dealing with plants, mostly ecological in emphasis, began to appear under his authorship in 1906 and continued to be published, up to a hundred in number, at fairly regular intervals until his retirement in 1941. In addition to *The Plant Life of Maryland* and his posthumous book on the floristics of the Sonoran Desert, he was author or co-author of four other books, including *The Distribution of Vegetation in the United States as related to Climatic Conditions.* Many of his papers were illustrated with halftones made from photographs of superb quality, taken with a heavy, cumbersome view camera using $8'' \times 10''$ glass plates.

When death overtook Forrest Shreve on July 19, 1950, only a part of his plan for recording the vegetational characteristics of desert areas in North America had been completed. Following his survey of the Sonoran Desert, he had done a good deal of work in Chihuahua, Mexico, and several papers from the pens of Shreve, C. A. Weatherby, I. M. Johnston, and

[1] The authors submitted separate accounts of Forrest Shreve, and the editors have combined them with the authors' permission.

probably from others, stand as a memorial to his vision and perseverance, for he worked under the growing burden of impaired health. In spite of this handicap he accomplished much and encouraged others to carry on the work he could not finish. He was a leader in the field of desert ecology, for his understanding of desert life and desert problems was founded on long experience, keen observation, and an analytical mind. He became almost a part of the desert he studied and knew so well.

Technical Papers

The Question of Extraneural Growth *in vivo* of Poliomyelitis Virus

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The cultivation of poliomyelitis virus in cell suspensions of various tissues (*1–3*) (skin, muscle, intestine, kidney, testis, etc.) has been interpreted as invalidating the assumption of obligate neurocytotropism of this virus in the intact animal, an assumption which Syverton and his associates explicitly state is "no longer tenable." There is a serious fallacy in inferring from *in vitro* growth of a virus in particular tissues that the same tissues are capable of supporting growth *in vivo*. It has yet to be shown in the living animal that cells of skin, muscle, kidney, or testis, in contrast to those of the nervous system, either support growth of poliomyelitis virus or display specific lesions, or, indeed, lesions of any kind, during the early stages of poliomyelitis. Certainly, dermatitis, enteritis, nephritis, and orchitis are not features of the clinical picture of the disease. Enders himself, in whose laboratory the first successful cultures of the virus were made on extraneural tissues, has made no such claim. On the contrary, he has stated (*1*), in discussing the factors influencing multiplication of viruses and rickettsiae in tissue cultures, that:

> The results of many studies with different viruses, however, have made it clear that the degree of pathogenicity exhibited by an agent for the intact animal is frequently not correlated with its capacity to increase in cultures prepared from the tissues of such an animal; . . . [and] Extracellular inhibitory mechanisms present in the living body may be eliminated in cultures, thus permitting multiplication.

There is some reason to believe that these remarks may well apply to the case of poliomyelitis virus. The method used, with various modifications, by Enders and others, in the cultivation of poliomyelitis virus, is that of Maitland and Maitland (*5*), using one of Hanks' salt mixtures and Simms' ox blood serum ultrafiltrate. Preliminary washing of the tissue appears to be important, both in the original preparation and in subcultures. The salt solutions depart widely from normal mammalian interstitial fluid in respect to electrolyte composition. The importance of electrolytes, at certain critical concentrations, in promoting the attachment of virus to host cell has recently been noted by Puck and his associates (*6*). The part played by ox blood serum ultrafiltrate in virus cultivation also appears to be critical. Simms (*7*), who introduced this material for tissue culture, found that normal tissue and serum contain several factors that affect cell growth and metabolism, one of which is inhibitory, one (A) stimulative, one (B) causative of fat granule production, one (C) degenerative, and one (D) causing cohesion of cells. The ultrafiltrate contains only A, removes B, C, and D from cells and counterbalances the inhibitory factor.

The presence of poliomyelitis virus in the intestine in the disease has been offered by Evans and Green (*8*), and more recently by Syverton and his associates, as evidence in support of extraneural growth of the virus, presumably on the cells of the oral and intestinal mucous membranes. The usual lack of signs of inflammation in these membranes early in the disease is suggestive contrary evidence. An alternative explanation, based on the characteristic neurotropism and axonal conduction of the virus, has been demonstrated by us in recent experiments (*9*), which showed that the virus is excreted into pharynx and gut as early as 3 days after neural exposures in which primary exposure of the pharyngeal or intestinal surfaces was rigorously excluded. At this time virus was demonstrable in the regional ganglia (*10*). In other experiments heavy exposures of the gastrointestinal tract, in which the oropharyngeal surfaces did not participate, were not followed, after the immediate postexposure period, by continuing excretion of virus such as might have been expected if the mucosal epithelium had become infected. In a single instance, excretion of virus began later, at the time when paralytic symptoms appeared, an indication of a neural source. In recent experiments, as yet unpublished, we found that nontraumatic oropharyngeal application of the virus was followed by the appearance at 2 days of specific lesions and at 3 days of recoverable virus from regional peripheral ganglia, whereas no evidence of infection of the CNS had appeared then nor for several days later. The experiments indicate an almost immediate entry and centripetal passage of virus through the superficial nerve fibers to the ganglia, without any lag such as