

# Epidemiology of Poliomyelitis in the United States and Its Recognition as an Infectious Disease from the Mid-19th Century to the Early 20th Century

Subjects: [Clinical Neurology](#)

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This study reviews the role of epidemiology in the United States in the late 19th and early 20th century, which led to recognition that poliomyelitis is an infectious disease and set the stage for subsequent developments in virology and immunology, the development of inactivated and live attenuated polio vaccines, and a dramatic worldwide decrease in poliomyelitis mortality and morbidity. Epidemiological studies in the United States were systematically reviewed from the mid-19th to early 20th centuries. Isolated cases and scattered small outbreaks of poliomyelitis in the mid-19th century led to epidemics of increasing size by the end of the century, causing public consternation, especially as the disease was considered “new” and had a predilection for young children. By the 1890s, the seasonal pattern of epidemics suggested that poliomyelitis might have an infectious etiology, but direct evidence of communicability or contagiousness was lacking, so an infectious etiology was not widely suspected until the early 20th century. Reports of bacterial isolations from spinal fluid and postmortem tissues suggested that poliomyelitis might be a bacterial disease, and simultaneous outbreaks of paralytic disease in humans and animals suggested a possible zoonotic basis. Although experimental studies showed that it was theoretically possible for flies to serve as vectors of poliovirus, and occasional cases of polio were likely caused by fly-borne transfer of poliovirus from human feces to human food, a fly abatement field trial showed convincingly that flies, whether biting or non-biting, could not explain the bulk of cases during polio epidemics. In conclusion, the early application of epidemiological evidence beginning in the late 19th century strongly suggested the infectious nature of the disease, distinct from previously identified conditions. Subsequent advances in virology and immunology from 1909 to 1954 proved that poliomyelitis was a viral disease with no natural animal host and made feasible the development of an inactivated trivalent poliovirus vaccine by Salk, and, subsequently, a live-attenuated trivalent poliovirus vaccine by Sabin.

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This review considers the emerging epidemiologic evidence from the mid-19th century to the beginning of the 20th century, which led to recognition in the United States that poliomyelitis is an infectious disease.

Isolated cases and small, geographically isolated outbreaks of probable poliomyelitis (“infantile paralysis”) were reported during the 18th and early 19th centuries <sup>[1]</sup>. These early cases were sufficiently characteristic to enable recognition of the disorder and to allow descriptions of typical clinical features but left those studying it puzzled

concerning its etiology. Because poliomyelitis did not show a clear pattern of communicability, an infectious etiology was not suspected.

By the end of the 19th century, epidemics of increasing size caused increasing public consternation, especially as the disease was considered “new” and had a predilection for young children. By the 1890s, the seasonal pattern of the epidemics suggested to multiple investigators that poliomyelitis might be infectious, but several factors confounded this growing consensus, and at the time there was certainly no proof of this idea. The late 19th-century outbreaks and epidemics of poliomyelitis provided a much clearer understanding of the clinical phenomenology of poliomyelitis, provided the beginnings of clinical–pathological correlation, allowed for the separation of poliomyelitis from other myopathies and other disorders causing acute or subacute weakness, and produced a rapid expansion in orthotic technologies and orthopedic surgeries to alleviate the resulting disabilities and deformities [2][3][4]. However, the contagiousness of poliomyelitis had long remained an open and contentious question, with inconclusive evidence of communicability even among those residing in close proximity (e.g., within families). In trying to elaborate an infectious theory of poliomyelitis, several misleading reports in the last decade of the 19th century led to consideration of whether poliomyelitis is a bacterial disease and whether it might be transmitted from animals to man—a zoonosis.

By the second decade of the 20th century, poliomyelitis was shown to be a viral disease [5][6], and the various putative bacterial causes were recognized to be merely contaminants. The early 20th century also saw the advent of the vector theory of poliomyelitis transmission, with increasing awareness that the poliovirus is excreted in feces, that poliovirus can be detected in sewage and in flies, and that flies can transmit poliomyelitis under experimental conditions. The vector-borne theory of poliomyelitis was abandoned by the mid-20th century after fly abatement field trials proved ineffective in modifying the course of poliomyelitis epidemics, but the zoonotic theory was not fully disproved until the latter half of the 20th century.

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