Commentary: The Polio Model—Does it apply for polio?

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Nielsen et al. conducted a retrospective, observational study of hospital record data from 1940 to 1953 in Denmark to evaluate the deductions of the old polio model. The authors defined polio as those patients ‘with a discharge diagnosis of non-paralytic polio, paralytic polio, suspected polio or primary lymphocytic meningitis’. Severity was graded progressively from non-paralytic polio, to paralytic polio, respiratory paralysis, and death. The authors found the polio model’s prediction about the impact of age, sibship size, and birth order on polio incidence and severity to have only limited support. They concluded that a model emphasizing intensity of exposure as a risk factor for severity better explained the epidemiology of polio.

The investigators in this article explored some very interesting territory in the history of polio. While the article offers a valuable historical perspective, it fails to address the polio model in light of more recent virological findings. Since the period of the epidemics studied in this article, we now know of three poliovirus serotypes, and dozens of other non-polio enteroviruses (NPEV) potentially capable of causing paralytic polio-like syndromes. Moreover, our general understanding of the aetiology, clinical presentation, and epidemiology of wild poliovirus transmission has substantially expanded. Certainly, the overall picture of the epidemiology of poliomyelitis is much more complex than the older polio model once indicated.

Today, the incidence of aseptic meningitis caused by polioviruses in many developed countries approaches zero because wild poliovirus transmission has been interrupted and oral polio vaccine (OPV) in many of these countries has been discontinued. It would be difficult to quantify to what extent NPEV contributed to the aseptic meningitis burden in the pre-vaccine era, but we know that NPEV certainly played a role. Seasonal variation of aseptic meningitis caused by NPEV exists as it does for aseptic meningitis caused by polioviruses. The same conclusions may apply for NPEV-caused paralytic poliomyelitis. Large paralytic outbreaks of NPEV occur sporadically around the world. Significant overlap exists between the findings of clinical syndromes caused by NPEV and those caused by polioviruses. Indeed, try to understand these findings with laboratory investigations is daunting.

We have learned from the polio eradication initiative in Latin America that as much as 44% of Guillain-Barre Syndrome cases had residual paralysis >60 days, a phenomenon previously thought to be limited to paralytic poliomyelitis. Field experience in the global polio eradication initiative demonstrates that the clinical distinction between Guillain-Barre Syndrome and acute paralytic poliomyelitis may often be very challenging, highlighting the need to identify the causative agent.

Furthermore, neurovirulence varies by serotype. Paralysis is more likely to occur with poliovirus type 1 infection, followed by type 3, and type 2. Geographical distribution varies by specific genotypes. The distribution of many of these geographic-specific genotypes is well known. Wild poliovirus type 1 found on the Indian sub-continent might be imported to other parts of the world in a matter of hours.

Indeed, in the last 30 years our epidemiological and virological knowledge of polio has expanded to the extent that the world is now poised to eradicate indigenous wild poliovirus transmission. However, enormous challenges confront this global effort. The efforts to eradicate smallpox pale in comparison to those required for polio. The successful eradication of smallpox was based on the facts that all infections were clinically evident, that the spread of the virus was limited, and that a strategy of vaccination around recognized cases could break global chains of transmission. This is not the case for polio, in which 99% of infections are clinically inapparent. Limited vaccination around recognized cases is not effective for polio. Huge efforts to implement high-quality mass immunization campaigns are required in large geographical areas where the virus is known to be circulating.

Therefore, it is clear that our virological rather than our clinical understanding of wild poliovirus transmission will be the most critical factor in its control and eventual eradication. Defining the reservoirs of the last chains of transmission by genotype should lead to the successful implementation of the appropriate strategies. In the absence of virological data, the authors should be commended for their efforts to expand our historical perspective about a disease that hopefully will become extinct in the near future.

References

