The central message of our study is that post-polio syndrome might be associated with a chronic low-level poliovirus infection caused by the virus type that – at the time of the initial event – had infected each patient. That is, the possibility exists that poliovirus remains in a “quiescent state” in the body for a long period of time.

The conclusion is derived from the study of a cohort of Italian PPS patients (112) and their family members (51), together with non-polio controls (71), and aged polio survivors with “stable polio” (18). Nearly three quarters of PPS patients appear to harbor “poliovirus remnants” (in other words polioviral genomes and low-level virus activity), while survivors with stable polio very rarely harbor poliovirus.

The identity of the poliovirus remnants is being clarified by genome sequencing using novel techniques in collaboration with Konstantin Chumakov, one of the best poliovirus experts of the U.S. Food and Drug Administration. It is believed, but not completely proven yet, that the poliovirus forms persisting in polio survivors represent “mutated” (that is, genetically changed) derivatives of the virulent polioviruses that were infecting the patients at the time of the acute polio.

It is thought that slow virus infection of the nervous and muscular cells may be responsible for the slowly progressive loss of neural and muscular cells and chronic inflammation. A further conclusion of the study is that poliovirus remnants are not transmitted from PPS patients to their family members. Thus, these poliovirus remnants are not dangerous to the population, nor represent a possible form of reinfection for poliomyelitis.

Finally, the possible infectious etiology of PPS calls for an “effective cure and prevention.” So far, in fact, anti-inflammatory drugs and other treatments have failed in this field. A possible remedy is seen in the administration of intravenous human immunoglobulins (i.e., antibodies derived from blood donors; a form of “passive” immunotherapy). These antibody preparations also contain anti-poliovirus antibodies.

Our work brings the attention of clinicians and the pharmaceutical industry to the need of antiviral agents for treating PPS. New antiviral compounds are being developed for picornaviruses (the virus family that comprises polioviruses), some of them having activity against polioviruses. These novel antivirals might be tested in PPS patients, provided that the pharmaceutical industry is willing to design and finance clinical trials.

Quantitative methods for evaluating the possible efficacy of these drugs are already available at a number of clinical centers worldwide. Investigations in this field could lead to significant progress as seen over the last few years in the successful therapy of cases with chronic liver infection due to hepatitis C virus.