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In vitro Interaction of Poliovirus with Cytoplasmic Dynein

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Key Words
Poliovirus · Dynein · Neuroblastoma cells · Axonal transport

Abstract
Objective: Poliovirus (PV) enters the host by the oral route and can infect the central nervous system (CNS) by two mechanisms: crossing the blood-brain barrier and traveling along the nerves from the muscle to the spinal cord. In the latter mechanism, the PV receptor, CD155, and the motor protein, dynein, have been implicated in the transport of PV to the CNS. In this work we analyzed the possible interaction of PV with dynein. ***Methods:*** PV was bound to a Sepharose 4B beads and they were used to analyze the interaction of PV with cytoplasmic proteins from neuroblastoma cells by affinity chromatography and Western blot. ***Results:*** The interaction with cytoplasmic dynein was observed only when the Sepharose beads bound to PV were used and not in the control ones, where proteins from uninfected cells were coupled. ***Conclusion:*** These preliminary results open the possibility that PV uses the dynein directly in its retrograde axonal transport.

The three serotypes of poliovirus (PV) belong to the *Picornaviridae* family [1, 2] and they are small, non-enveloped viruses with a single-strand and positive polarity RNA genome [1, 3]. Only 1–2% of the patients infected with any of the three serotypes of PV have paralytic poliomyelitis, mainly in the lower limbs caused by the destruction of motor neurons present in the central nervous system (CNS) [2, 4].

PV infection occurs through the oral route and the virus infects the digestive tract, especially the lymphatic tissue present in Peyer's patches and tonsils. Later, the virus reaches and replicates in the mesenteric and deep cervical lymph nodes and it can be spread to other tissues by the bloodstream [5].

There are two hypotheses to explain the CNS infection by PV. The clinical features suggest that PV can cross the blood-brain barrier (BBB) [5, 6], since the amount of PV present in the brain of both transgenic CD155+ (TghPVR) or non-transgenic mice inoculated intravenously is similar to the amount of anti-transferrin receptor antibody, suggesting that the transport to the brain is effective. This process is independent of the presence of the CD155 molecule or the PV strain [7] and its mechanism is still unknown.

The other hypothesis is based in the capability of PV to move through the nerves from the neuromuscular junction to the neurons of the spinal cord (SC) [8] as sug-

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