Herpes Simplex Virus Type 1 Glycoprotein E Is Required for Axonal Localization of Capsid, Tegument, and Membrane Glycoproteins

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Abstract

Herpes simplex virus type 1 (HSV-1) glycoprotein E (gE) promotes cell-to-cell spread at basolateral surfaces of epithelial cells (2). The mouse retina model and the rat SCG neuron culture were utilized to determine the gE mutant virus spread phenotype. During infection of the mouse or rat eye, virion components travel from retina ganglion cells to optic nerve and optic tract indicating anterograde spread; however, virus can also infect the endplates of autonomic neurons that innervate the ciliary body and iris to control pupil size (4), and it represents retrograde spread. Virion components travel from postsynaptic to presynaptic neurons in a retrograde direction to infect regions of the brain that are distinct from those infected by anterograde spread in the optic nerve (1, 3). The gE-null virus failed to travel into brain not only through anterograde but retrograde pathway. It indicates that gE-null virus is defective in axonal localization. Two FcγR- mutants, NS-gE264 and NS-gE380 virus were constructed. NS-gE380 virus failed to spread from retina to optic nerve, while NS-gE260 spread intact. Therefore, axonal localization and IgG Fc receptor activity involve overlapping but distinct gE domains. Besides, according to in vitro rat SCG neuron model, the site of defect in gE-null virus anterograde spread is prior to axon terminus; however, the site of defect in gE-null virus retrograde spread remains unclear (2).

References