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Mutation of UL24 impedes the dissemination of acute herpes simplex virus 1 infection from the cornea to neurons of trigeminal ganglia

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INTRODUCTION

Herpes simplex virus 1 (HSV-1; human herpesvirus 1) replicates in epithelial cells and then infects the ends of sensory neurons that form neuronal ganglions (Rota et al., 2007). HSV-1 persists in neurons as a life-long latent infection and may reactivate, often following stress, resulting in recurrent infections (Rota et al., 2007). In healthy individuals, symptoms typically vary from cold sores to viral keratitis and in rare cases infections can lead to a life-threatening encephalitis (Whitley & Rota, 2001). In people who are immunocompromised, e.g. people living with AIDS, neonates or stem cell recipients, the symptoms are often severe (Fatemi et al. & Schwartz, 2007). Many questions remain regarding the spread of infection within the host, which has hindered the development of treatments to prevent recurrent infections.