Ocular infections by Herpes simplex virus are among the most prevalent and serious eye disease in developed nations. Numerous vaccines have been developed to combat against herpes simplex virus type 1 (HSV-1)-associated eye disease (1). So far, these vaccines have failed to prevent HSV-1 replication in the eye or the subsequent establishment of latency in the trigeminal ganglia. On the other hand, recent studies have shown that IL-12 initiates the development of cell-mediated immunity following infection with certain infectious agents. This activity of IL-12 makes it a good candidate as a vaccine adjuvant (2). IL-12 is a heterodimeric cytokine composed of 35-kDa (p35) and 40-kDa (p40) subunits (3, 4). To improve the vaccine efficacy against infection from ocular HSV-1 infection and reduce virus explant reactivation in trigeminal ganglia, Osorio et al (5) have constructed recombinant HSV-1 viruses expressing IL-12p35 or IL12p40 as an adjuvant. Assessment of adjuvant effects based on survival, severity of eye disease, virus clearance, virus explant reactivation, antibody response, T cell responses, and cytokines expression, revealed that codelivery of IL12p35 induces higher neutralizing antibody titers, lymphocyte proliferation, CTL activities, IFN-γ production, and less explant reactivation in the trigeminal ganglia. These results indicate that IL-12p35 can enhance the protective immune response against ocular HSV-1 challenge, and therefore may serve as a potential adjuvant in ocular HSV-1 vaccination strategies.

References: