Hope-simpson's progressive immunity hypothesis as a possible explanation for herpes zoster insidence data

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Abstrak

Varicella-zoster virus (VZV) is the causative agent of both varicella (chickenpox) and herpes zoster (HZ) (shingles). After varicella infection, the virus remains dormant in the host's dorsal ganglia and can reactivate due to waning cell-mediated immunity, causing HZ. Exposure of varicella-immune persons to VZV may boost the host's immune response, resulting in a protective effect against HZ. In this study, we used mathematical models of VZV transmission and HZ development to test the biological hypothesis of "progressive immunity," originally proposed by Hope-Simpson (Proc R Soc Med. 1965;58:9-20), that cell-mediated protection against HZ increases after each episode of exposure to VZV. Predictions from a model incorporating such a hypothesis were compared with those of other concurrent models proposed for explaining HZ epidemiology. The progressive immunity model fits significantly better the age profile of HZ incidence for Finland (years 2000-2006), Italy (2003-2005), Spain (1997-2004), and the United Kingdom (1991-1992), suggesting that this mechanism may be critical in shaping HZ patterns. The model thus validated is an alternative to VZV models currently used to evaluate the impact of mass immunization programs for varicella and therefore extends the range of tools available to assist policy-makers with the present decision paralysis on the introduction of vaccination.