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HERPES ZOSTER: EXOGENOUS BOOSTING, PROGRESSIVE IMMUNITY AND THE DILEMMA OF MASS VARICELLA IMMUNIZATION

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Herpes zoster (HZ) is a painful disease caused by the reactivation of the varicella zoster virus (VZV) when the cell mediated immunity (CMI) acquired after primary varicella infection goes down (e.g., with ageing). Hope-Simpson formulated (1965) the exogenous boosting hypothesis (EBH), according to which further infective exposures to VZV may boost CMI, resulting in a protective effect against HZ. The EBH, which has received a number of field confirmations, offers surprisingly consistent explanations to a number of regularities, such as thezoster puzzle (more HZ in countries where varicella circulation is slower) and the widespread increase in HZ even in the absence of any immunization. Inclusion of the exogenous boosting hypothesis in VZV transmission models predicts a large and prolonged transient wave in natural HZ incidence following mass varicella immunization. The fear of this HZ boom is a main responsible of the current stall of varicella vaccination in Europe. In this talk, I summarize a number of recent results from a couple of projects i coordinated on the subject, based on a model incorporating a further noteworthy Hope-Simpsons hypothesis, stating that each VZV re-exposure progressively raises CMI protection against HZ after each new episode of re-exposure to VZV. This progressive immunity model fits well available European HZ data, suggesting that the mechanism may be critical in shaping HZ patterns, and supplies, once the issue of parametric identifiability is properly handled, fairly stable estimates of the critical biological parameters governing reactivation. The model suggests counter-intuitive implications of varicella immunization in relation to vaccine-related HZ and the epidemiology of HZ after varicella elimination. I conclude by discussing the challenges for future VZV research.

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