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## **Abstract:**

Resident memory CD8<sup>+</sup> T cells (Trms) are essential for protecting barrier nonlymphoid tissues (NLTs) against reinfection, yet the involvement of dendritic cells (DCs) in this process and the nature of Trm-DC interactions within these tissues remain poorly understood. Our study demonstrates that upon reactivation, memory CD8<sup>+</sup> T cells located in the skin-independently of circulating memory counterparts-initiate the infiltration and maturation of plasmacytoid DCs (pDCs) in the tissue. This, in turn, promotes the maturation of conventional type 1 DCs (cDC1s) through type I IFN (IFN-I) signaling in a pDC-dependent manner. Depletion of pDCs or blocking IFN-I signaling disrupts this axis, severely impairing Trm-driven protection against secondary infections with vaccinia virus (VACV) in the skin. Notably, this pDC-dependent, IFN-I-mediated pathway is also essential for Trm-mediated protection against secondary respiratory infections with influenza A virus (IAV). Our findings uncover a crucial collaboration between Trm, pDCs, and cDC1s, offering new insights for enhancing vaccines.