

Mast cells acquire MHCII from dendritic cells during skin inflammation.

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Abstract

Mast cells (MCs) and dendritic cells (DCs) are essential innate sentinels populating host-environment interfaces. Using longitudinal intravital multiphoton microscopy of DC^{GFP}/MC^{RFP} reporter mice, we herein provide in vivo evidence that migratory DCs execute targeted cell-to-cell interactions with stationary MCs before leaving the inflamed skin to draining lymph nodes. During initial stages of skin inflammation, DCs dynamically scan MCs, whereas at a later stage, long-lasting interactions predominate. These innate-to-innate synapse-like contacts ultimately culminate in DC-to-MC molecule transfers including major histocompatibility complex class II (MHCII) proteins enabling subsequent ex vivo priming of allogeneic T cells with a specific cytokine signature. The extent of MHCII transfer to MCs correlates with their T cell priming efficiency.

Importantly, preventing the cross talk by preceding DC depletion decreases MC antigen presenting capacity and T cell-driven inflammation. Consequently, we identify an innate intercellular communication arming resident MCs with key DC functions that might contribute to the acute defense potential during critical periods of migration-based DC absence.

Involved units

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