



Article abstract

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DCs metabolize sunlight-induced vitamin D3 to 'program' T cell attraction to the epidermal chemokine CCL27

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During adaptive immune responses, dendritic cells activate T cells and endow them with specific homing properties. Mechanisms that 'imprint' specific tropisms, however, are not well defined. We show here that 1,25(OH)₂D₃, the active form of vitamin D₃, signaled T cells to express CC chemokine receptor 10, which enabled them to migrate to the skin-specific chemokine CCL27 secreted by keratinocytes of the epidermis. In contrast, 1,25(OH)₂D₃ suppressed the gut-homing receptors $\alpha 4 \beta 7$ and CCR9. Vitamin D₃, the inactive prohormone naturally generated in the skin by exposure to the sun, was processed by dendritic cells and T cells to the active metabolite, providing a mechanism for the local regulation of T cell 'epidermotropism'. Our findings support a model in which dendritic cells process and 'interpret' locally produced metabolites to 'program' T cell homing and microenvironmental positioning.

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