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# Protein kinase CK2 controls T - cell polarization through dendritic cell activation in response to contact sensitizers

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## Abstract

Allergic contact dermatitis (ACD) represents a severe health problem with increasing worldwide prevalence. It is a T - cell - mediated inflammatory skin disease caused by chemicals present in the daily or professional environment. $NiSO_4$ and 2,4 - dinitrochlorobenzene (DNCB) are 2 chemicals involved in ACD. These contact sensitizers are known to induce an up - regulation of phenotypic markers and cytokine secretion in dendritic cells (DCs; professional APCs), leading to the generation of $CD8^+$ Tc1/Tc17 and $CD4^+$ Th1/Th17 effector T cells. In the present study, using a peptide array approach, we identified protein kinase CK2 as a novel kinase involved in the activation of human monocyte - derived DCs (MoDCs) in response to $NiSO_4$ and DNCB. Inhibition of CK2 activity in MoDCs led to an altered mature phenotype with lower expression of CD54, PDL - 1, CD86, and CD40 in response to $NiSO_4$ or DNCB. CK2 activity also regulated proinflammatory cytokine production, such as TNF - α, IL - 1 β, and IL - 23 in MoDCs. Moreover, in a DC/T cell coculture model in an allogeneic setup, CK2 activity in MoDCs played a major role in Th1 polarization in response to $NiSO_4$ and DNCB. CK2 inhibition in MoDCs led to an enhanced Th2 polarization in the absence of contact sensitizer stimulation.