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# Vitamin D contributes to mast cell stabilization

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## Abstract

**Background and aims:** Mast cells are the major effector cells in allergic disorders and many other inflammatory disorders. The mechanism of mast cell stabilization is not fully understood. Cumulative reports indicate that vitamin D (VitD) contributes to the homeostasis in the body. This study tests a hypothesis that VitD is required in the maintenance of the stability of mast cells.

**Methods:** The stability of mast cell lines, HMC1 cells, RBL-2H3 cells, p815 cells, and mouse bone marrow-derived mast cells (BMMC) was tested in the presence or absence of VitD3.

**Results:** Mast cells activated automatically in a VitD-deficient environment. Exposure to calcitriol in the culture increased the expression of VitD receptor (VDR) in mast cells. VDR formed complexes with Lyn in mast cells to inhibit the binding of Lyn to the  $\beta$  chain of Fc $\epsilon$ RI and MyD88, which decreased the phosphorylation of Syk, decreased the levels of MAPK and NF- $\kappa$ B. VDR bound to the promoter of TNF- $\alpha$  to decrease the acetylation of histone H3/H4, RNA polymerase II and OCT1 (a transcription factor of TNF- $\alpha$ ) at the promoter locus and repressed the expression of TNF- $\alpha$  in mast cells.

**Conclusions:** The data demonstrate that VitD is required to maintain the stability of mast cells. The deficiency of VitD results in mast cell activation.

**Keywords:** Lyn tyrosine kinase; Syk tyrosine kinase; calcitriol; mast cell; vitamin D.

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