

ALLERGY, VITAMINS

# ONCE MORE: CALCIUM AND IGE

18.06.2010

Here comes the most [striking connection](#) between calcium and IgE that updates some earlier posts here

The fact that the regulation of intracellular  $Ca^{2+}$  concentration is different in various T-cell effectors may offer the opportunity to target key intermediates ... to inhibit specifically the functions of one given T-cell subset. It is classically admitted that stimulation through the T-cell receptor (TCR) induces inositol triphosphate production that binds to its receptors on the membrane of the endoplasmic reticulum (ER) leading to the release of ER  $Ca^{2+}$  stores into the cytosol ... We show that mouse Th2 but not Th1 cells expressed Cav1.2 and Cav1.3 channels. Th2 cells transfected with Cav1AS had impaired  $Ca^{2+}$  signaling and cytokine production, and lost their ability to induce airway inflammation on adoptive transfer ... These results indicate that Th2 cells selectively express Cav1 channels that may be efficiently targeted in T lymphocytes to prevent experimental asthma.

## Addendum Aug 10, 2010

another first: [Ca<sup>2+</sup> signaling in DCs](#)

The NCKX blocker 3',4'-dichlorobenzamyl (DBZ) reversed the inhibitory effect of 1,25(OH)<sub>2</sub>D<sub>3</sub> on the LPS-induced increase of  $Ca^{2+}$ . Expression of the costimulatory molecule CD86 was down-regulated by 1,25(OH)<sub>2</sub>D<sub>3</sub>, an effect reversed by DBZ.

## Addendum Nov 21, 2013

from Saito, Hirohisa, Teruko Ishizaka, and Kimishige Ishizaka. "Mast Cells and IgE: From History to Today." In *Allergy international* 62, no. 1 (2013)

For FcεRI-dependent activation of mast cells and basophils, extracellular Ca<sup>2+</sup> is absolutely required for the release of both pre-formed (such as histamine) and newly generated (such as cysteinyl leukotriene; cys-LT) mediators.

Really forgot that, [it is even in Wikipedia](#).

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