

Inhibiting diacylglycerol kinase (DGK) for asthma treatment

A treatment for asthma where diacylglycerol kinase zeta is inhibited to target both inflammatory mediators and non-immune effectors linked to asthmatic responses.

Inventor
[Taku Kambayashi](#)

STATE OF DEVELOPMENT

- *In vivo* genetic targeting of DGK ζ in mice leading to reduced lung resistance and tissue dampening

REFERENCE MEDIA

- Joshi et al. [Sci Signal, 2013, 6\(303\): ra102.](#)

DESIRED PARTNERSHIPS

- Co-development

Problem

Asthma is a respiratory disease triggered by inflammatory mediators from T helper 2 (Th2) cells and non-immune effectors, such as smooth muscle shortening, which causes airway obstruction during an asthmatic attack. Treatments targeting inflammatory mediators do not always lead to a reduction in airway hyper-responsiveness. Thus, alternate ways to target both inflammatory and non-immune effectors are needed to control asthma.

Solution

Dr. Taku Kambayashi has identified a target within an intracellular signaling pathway that, unlike current therapies, can target both inflammatory mediators and non-immune effectors for asthma therapy. Inhibiting diacylglycerol (DAG) kinase zeta (DGK ζ) enhances DAG signaling, which can increase downstream signaling effectors. As shown in the figure, enhanced DAG signaling can increase Th1 T cell differentiation, rather than Th2, increase surfactant secretion, reduce smooth muscle contraction, and inhibit mast cell degranulation, all of which can relieve asthmatic responses. *In vivo* rodent models in which DGK ζ is genetically or pharmacologically inhibited, show that when these mice were challenged with a molecule that induces airway hypersensitivity, lung resistance and pulmonary inflammation was dampened compared to controls. Thus, DGK inhibition is a promising target for asthma treatment.

Advantages

- Targets both inflammatory and non-immune effectors to control asthma
- Specific targeting against diacylglycerol kinase zeta

Application

- Asthma treatment

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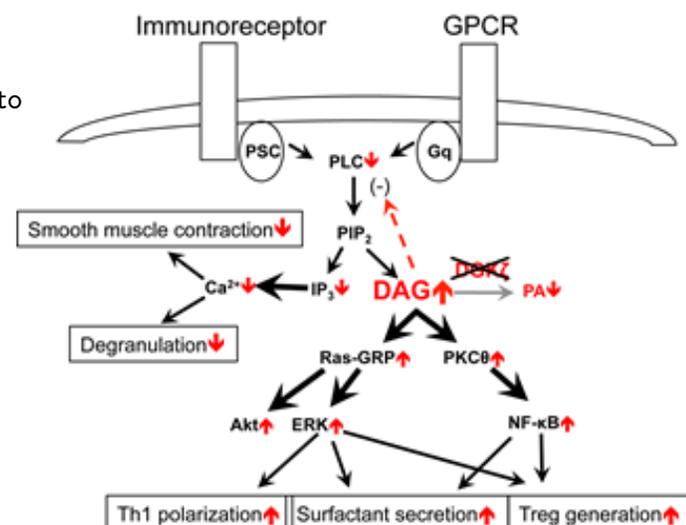


Image retrieved from Figure 1 of disclosure.