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Title: TAK1 protein kinase signaling

Research emphasis:

Dr. Ninomiya-Tsuji's group studies a protein kinase, TAK1, which is an intermediate of proinflammatory/stress signal transduction pathways. We found that TAK1 signaling not only mediates inflammatory responses but is also critically involved in cell survival and tissue integrity. Both hyper- and hypo-activations of TAK1 are associated with tissue injury and chronic inflammatory conditions. Accordingly, manipulation of TAK1 activity can be utilized to eliminate undesired cells such as tumors and also for preventing certain types of tissue injury and chronic inflammatory conditions .

Application :

- Characterization of genetically engineered mice
- Molecular and biochemical analyses of protein kinase signal transduction pathways
- Cell death
- Inflammation

Collaboration potential:

- Chronic inflammatory diseases
- Environmental chemical induced cell death/inflammation
- Protein kinase signaling

Selected publications: (limit 4)

Morioka, S., Sai, K., Omori, E., Ikeda, Y., Matsumoto, K., and Ninomiya-Tsuji, J. (2015). TAK1 regulates hepatic lipid homeostasis through SREBP. **Oncogene** *in press*.

Mihaly, S.R., Ninomiya-Tsuji, J.*, and Morioka, S*. (2014). TAK1 control of cell death†. **Cell Death Differ** 21, 1667-1676.*Co-corresponding authors; †Review Article

Morioka S, Broglie P, Omori E, Ikeda Y, Takaesu G, Matsumoto K, Ninomiya-Tsuji J (2014) TAK1 kinase switches cell fate from apoptosis to necrosis following TNFα stimulation. **J. Cell Biol.** 204, 607-623

Omori, E., Inagaki, M., Mishina, Y., Matsumoto, K., and Ninomiya-Tsuji, J. (2012). Epithelial transforming growth factor β-activated kinase 1 (TAK1) is activated through two independent mechanisms and regulates reactive oxygen species. **Proc. Natl. Acad. Sci. USA** 109, 3365-3370.