

# 特別講演のお知らせ

## How bone cells distinguish between catabolic and non-catabolic mechanical stimuli

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**Abstract:** Lack of mechanical stimulation cause bone loss through activation of bone resorbing osteoclasts by mechanosensitive bone cells. In contrast, bone cells subjected to physiological mechanical loads inhibit osteoclast formation and activity. How bone cells distinguish between catabolic and non-catabolic mechanical stimuli remains a mystery. Previously we found that physiological mechanical stimulation may reduce the stimulating effect of cytokines on osteoclastogenesis. How inflammatory factors, such as cytokines, affect the response of bone cells to (un)loading also remains to be discovered.

Low intensity mechanical stimuli corresponded to active Piezo1, SERCA2, and inhibition of bone resorption. We also found lower protein levels of Ezrin in cultured human bone cells that stimulated osteoclast formation upon supraphysiological. Ezrin regulates membrane tension and is activated by inflammation. Membrane tension and inflammation both affect ephrin receptors (EPH). We have tested the effect of unloading versus a 5 min stimulation with shear stress of physiological magnitude, known to inhibit osteoclast formation *in vitro*, on EPH activity in cultured human primary bone cells. Our results suggests that daily mechanical stimuli rapidly inactivate multiple EPHs. Ongoing experiments need to elucidate whether the activity of EPHs is modulated by mechanical (un)loading of bone *in vivo*, by overloading and inflammatory factors *in vitro*, and whether there is a causal relation between activation of certain EPHs and stimulation of osteoclastogenesis.

Date: 5<sup>th</sup> November (Tue), 2024 17:00-18:30

Venue: 歯学部棟6階セミナー室624

本講演は博士課程授業科目の「研究方法論（基礎・応用）の授業に出席したとみなす講演会」となります。  
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**Organizer: Hiroshi KAMIOKA**

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