−大学院歯学独立研究科−
第 133 回 中 間 発 表 会 プ ロ グ ラ ム
第 41 回 テ ー マ 発 表 会 プ ロ グ ラ ム

大学院学生等が、これまでの研究成果を発表します。
どなたでも聴講できますので、多数の参加をお待ちしております（聴講申込不要）

場　所：実習館２階　総合歯科医学研究所セミナー室
日　時：2024 年 7 月 17 日（水）17 時 15 分　開会

−2024 年 7 月 17 日（水）−

No.	発表区分・予定時間	演題名・発表者	審査委員
	17:15	開会挨拶　平岡研究科長	
1	［テーマ］ 17:20〜17:30 司会：田口 教授	「顎骨の脆弱化が口腔に与える影響の探索」 大滝 紘史　硬組織疾患制御再建学講座　臨床病態評価学	ー
2	［中 間］ 17:30〜18:00 司会：中道 准教授	「The effect of the vitamin-D receptor in osteoblastic cells on calcium and phosphate imbalance disorders 骨芽細胞におけるビタミン D 受容体がカルシウム・リン酸不均衡障害に及ぼす影響」 刘 子洋　硬組織疾患制御再建学講座　硬組織機能解析学	主査：平賀教授 副査：北川教授 ：芳澤教授

問合わせ先：本館 2 階　学事室（大学院）内線 2012

発表内容の要旨(課程博士)
Abstract of Presented Research (For the Doctoral Course)

学籍番号 Student ID No.	ID#G 2214	入学年 Entrance Year	2022 年 Year
(ふりがな)	りゅう しょう		
氏名 Name in Full	刘 子洋		
専攻分野 Major Field	硬組織機能解析		
主指導教員 Chief Academic Advisor	中道 裕子		
発表会区分 Type of Meeting	(中間発表会) / 大学院研究科発表会 ・ 松本歯科大学学会 Midterm Meeting / Graduate school research meeting presentation /The Matsumoto Dental University Society		

演題名 / Title of Presentation

The effect of the vitamin-D receptor in osteoblastic cells on calcium and phosphate imbalance disorders
骨芽細胞におけるビタミンD受容体がカルシウム・リン酸不均衡障害に及ぼす影響

発表要旨 / Abstract

Among cells that compose bone, osteoblasts and osteocytes (osteoblastic cells) express the highest levels of the vitamin-D receptor (VDR). A number of studies including Nakamichi et al. (JBMR 2017) reported that, VDR in osteoblastic cells does not obviously affect calcium (Ca) and phosphate (Pi) metabolism or bone turnover under physiological conditions. In addition, our research demonstrated that supra-physiological doses of 1,25-dihydroxyvitamin D_3 (1,25D) induced hypervitaminosis D, characterized by enhanced bone resorption, soft tissue calcification, and elevated serum Ca and Pi levels, all of which were mediated by VDR in osteoblastic cells (Endocrinology 2020, JSBMB 2023). Near-physiological doses, however, suppressed bone resorption via VDR in osteoblastic cells (JBMR 2017). These findings suggest the role of VDR in osteoblastic cells varies in a vitamin D status-dependent manner, yet the underlying mechanisms remain unclear.

To further explore the role of VDR in osteoblastic cells under different vitamin D conditions, we employed a hypervitaminosis D model and also an adenine-induced chronic kidney disease (CKD) model, which is usually accompanied by 1,25D deficiency. Unexpectedly, both conditions resulted in suppressed bone formation and increased serum sclerostin, an antagonist of the Wnt signaling pathway. Both the models also showed increased bone resorption, serum Ca x Pi, and fibroblast growth factor 23 (FGF23) levels. Notably, serum parathyroid hormone (PTH) levels decreased in hypervitaminosis D model but increased in CKD mice.

VDR ablation in osteoblastic cells mitigated most of the changes induced by hypervitaminosis D, including soft tissue calcification, increased bone resorption, and elevated serum levels of Ca x Pi, FGF23, and sclerostin, while also rescued the reduced bone formation. Conversely, VDR deletion in osteoblastic cells exacerbated CKD symptoms, such as increased bone resorption, cortical porosity, and elevated serum levels of Ca x Pi, PTH, FGF23, and sclerostin.

These findings suggest that VDR in osteoblastic cells is involved in disease progression in hypervitaminosis D but involved in protection against disease progression in kidney failure. This study highlights the critical roles of VDR in osteoblastic cells and its downstream sclerostin in calcium and phosphate imbalance disorders.