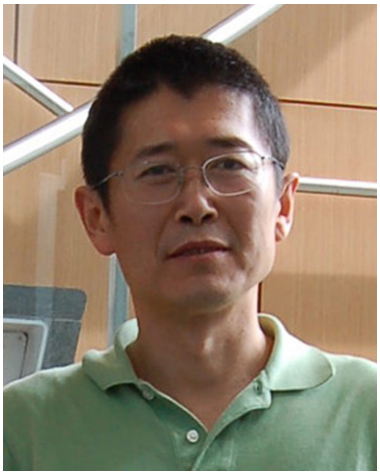
徳島大学研究クラスター講演会

◆ 選定クラスター「要介護リスクを高める骨粗鬆症・関節リウマチにおける骨代謝制御機構とその破綻のエピゲノム解析」
（代表：歯学域　口腔顎顔面矯正学分野　井澤 俊）



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■ 日時：平成30年11月21日 (水)　17:00~18:30

■ 場所：藤井節郎記念ホール（蔵本キャンパス）

“Fat talks to Bone”

While obesity has been considered beneficial for skeletal health recent studies suggest the opposite. Because there is little mechanistic insight as to how fat, per se, regulates the skeleton, we generated "fat-free" (FF) mice completely lacking visible visceral, subcutaneous and brown fat. Due to robust osteoblastic activity, trabecular bone volume is enhanced 400-500% in these animals. As expected, FF mice are diabetic but normalization of glucose tolerance fails to alter their skeletal phenotype indicating their enhanced bone mass does not reflect the metabolic syndrome. Importantly, the skeletal phenotype of FF mice is completely rescued by transplantation of adipocyte precursors or various fat depots indicating adipocyte products regulate bone growth. Confirming such is the case, transplantation of fat derived from adiponectin and leptin double knockout mice, unlike that obtained from their WT counterparts, fails to normalize FF bone. Thus, fat suppresses bone formation and decreased adiposity may greatly enhance bone mass due to a paucity of adiponectin and leptin. These observations challenge the concept that obesity improves skeletal health and provides insight as to why lipodystrophic patients are osteosclerotic.

(REF) Blood Adv, 2: 2467- 77, 2018; JBMR, 33: 1114-25, 2018; Nat Med, 22: 1203-05, 2016; Cell Rep, 11: 1625-37, 2015; J Cell Biol, 208: 125-36, 2015

＊本講演会は、大学院特別講義を兼ねています。大学院生の皆様も、ぜひご来聴ください。

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