The 45th Biomechanics Seminar

2023 年 11 月 8 日（水）15:00 - 17:00

京都大学 医生物学研究所 1 号館 1 階 共同セミナー室 3

MAP: http://www.kyoto-u.ac.jp/ja/access/campus/yoshida/map6r_b.html (Bldg. 31)

Bone Microstructure in Osteoarthritis

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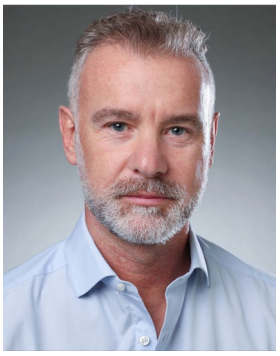
Thermomechanobiology: The Next Frontier for Treating Osteoarthritis

Prof. Dominique Pioletti



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Bone Microstructure in Osteoarthritis

Prof. X. Edward Guo (Columbia U, New York)

Osteoarthritis (OA) of the knee, a musculoskeletal disease characterized by degenerations in multiple joint tissues, including the articular cartilage and subchondral bone, is a major clinical challenge worldwide that currently has no cure. Traumatic knee injuries such as anterior cruciate ligament (ACL) tears predispose subjects to early onset of post-traumatic OA (PTOA), necessitating the development of effective disease-modifying therapies as total knee replacement surgeries have a limited lifetime. A significant knowledge gap remains in the pathogenesis of OA, while recent evidence suggests the critical role of subchondral bone microstructure and mechanics in OA development. Subchondral bone comprises the subchondral bone plate, a thin layer of cortical lamella, and the subchondral trabecular bone, composed of individual plate-like and rod-like trabeculae. Recent application of ITS showed that changes in the plate-and-rod microstructure of subchondral trabecular bone precede cartilage damage and are implicated in playing a role in disease pathogenesis. We will examine the connections between articular cartilage to subchondral bone plate and subchondral trabecular in human OA samples, a canine ACL transection model, and a guinea pig spontaneous OA model. The importance of bone microstructure in the early detection and intervention of OA will be emphasized.

Thermomechanobiology: The Next Frontier for Treating Osteoarthritis

Prof. Dominique Pioletti (EPFL, Switzerland)

Osteoarthritis is a painful joint disease characterized by a progressive degeneration of the tissues composing the osteochondral unit with no possibility to reverse its progression once its onset. We may have identified one factor favouring this irreversibility: dissipative properties, i.e., the capacity to convert mechanical energy into heat. During our previous study, we made the important observation that degenerated cartilage does not possess high enough dissipative properties to allow its self-heating during mechanical stimulation. The temperature of degenerated cartilage culminates only to a maximum of around 33°C during physiological loadings compared to 37°C for healthy cartilage thanks to its preserved self-heating capability. Mechanical as well as thermal stimuli might therefore play an essential role in cartilage homeostasis. By considering thermomechanobiology aspect in cartilage, we will propose an original approach which may have the potential to change the therapeutical approach in the treatment of early osteoarthritis.