

# Application of iPS cell technologies to cartilage regeneration and disease modeling

●Lecturer: Prof. Noriyuki TSUMAKI

(Dept. of Cell Growth and Differentiation  
Center for iPS Cell Research and Application, Kyoto University)

●Date: November 11<sup>th</sup> (WED), 2015 from 5:30 p.m.

●Place: Lecture room 2, Medical Education & Library Building 3F.

**//ATTENTION//**

There is another seminar at  
Lecture room 4.


当日は第4講義室でもセミナーが  
あります。ご注意ください。

●講師: 妻木 範行 教授

(京都大学 iPS 細胞研究所 増殖分化機構研究部門)

●日時: 平成27年11月11日(水) 17:30~

●場所: 医学教育図書棟3階 第2講義室



Primordial cartilage serves as skeletal templates during development that sustain the embryo bodies. It gives rise to two types of cartilage, growth cartilage and articular cartilage, after birth. Growth cartilage is where the bone grows in children, and its dysfunction due to genetic mutations cause dwarfism and skeletal malformation, conditions called skeletal dysplasia. Articular cartilage covers the ends of bones and provides shock absorption and lubrication to diarthrodial joints. Injury and degeneration of articular cartilage cause joint pain during motion, leading to osteoarthritis in adults. The conditions that compromise growth cartilage or articular cartilage are poorly understood, and curative drugs are not available. iPS cell technologies are beginning to be used to study these cartilage diseases. We have been developing a method in which human iPS cells (hiPSCs) are differentiated toward chondrocytes, the cells that constitute cartilage. We are generating effective and safe hiPSC-derived chondrocytes as regenerative medicine technology to treat defects in articular cartilage and sustain healthy joint function. The goal is to use these chondrocytes in clinical tests. In a separate project, we have generated hiPSC-derived chondrocytes from patients with skeletal dysplasia. FGFR3 chondrodysplasia such as achondroplasia is caused by a gain-of-function mutation in the FGFR3 gene. We found that chondrocytes derived from hiPSCs generated from patients suffering from FGFR3 chondrodysplasia produce abnormal cartilage that reproduces the pathology of the diseases and thus offers an iPSC-based disease model.

●Inviter: Prof. T. Era (Dept. of Cell Modulation)/幹細胞誘導学分野 江良 択実 教授

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