

22nd nanobiofluids seminar

2026 Mar 5th, 15:00-16:00

Conference Room (Room 134), 1st Floor, Bldg. No.1

<https://www.infront.kyoto-u.ac.jp/en/access/>

[Zoom link](#)

Protein crosslinking reactions governing organ stiffening: Molecular mechanisms and control strategies for fibrosis



Hideki Tatsukawa, Ph.D.

Assistant Professor

Graduate School of Pharmaceutical Sciences,
Nagoya University

Abstract

Tissue injury typically triggers inflammation followed by repair, and wounds usually heal completely. However, when damage is severe or repetitive, the repair program can break down, leading to excessive accumulation of the extracellular matrix (ECM) and progressive tissue stiffening. Similar maladaptive repair can also occur under chronic inflammatory conditions, resulting in fibrosis and scarring. Fibrosis is a common pathological feature shared by multi-organ diseases such as liver cirrhosis, kidney failure, and pulmonary fibrosis, and is estimated to account for approximately ~ 45% of deaths. In this seminar, I will focus on protein crosslinking enzymes that catalyze the formation of isopeptide covalent bonds between proteins, and introduce our analyses of the molecular mechanisms by which these enzymes contribute to fibrotic pathogenesis through macrophage phenotypic changes, fibroblast activation, and qualitative alterations in ECM properties, together with potential control strategies targeting these processes.

Biography

Dr. Tatsukawa is a molecular pathobiology researcher at the Graduate School of Pharmaceutical Sciences, Nagoya University (Cellular Biochemistry), with a background in enzymology. He received a B.Sc. in Chemistry from Tokyo University of Science (2003), an M.Sc. in Medical Science from Tokyo Medical and Dental University (2005), and a Ph.D. in Medicine (2009). In recent years, he has also served as a JST PRESTO researcher in a program aiming to establish a fundamental understanding of organismal changes associated with aging. His current research focuses on the molecular mechanisms by which aging accelerates fibrotic disease progression, with particular emphasis on qualitative ECM remodeling and the molecular basis of immune–stromal cell interactions.

Host: Hirofumi Shintaku, shintaku@infront.kyoto-u.ac.jp