Optical imaging of mouse articular cartilage using the glycosaminoglycans binding property of fluorescent-labeled octaarginine

Toshitaka Oohashi, Kiichi Inagawa, Keiichiro Nishida, Yoshifumi Ninomiya
Okayama University Graduate School of Medicine, Dentistry and Pharmaceutical Sciences, Okayama, Japan

*Contact author: oohashi@cc.okayama-u.ac.jp

Keywords: Optical imaging, Articular cartilage, Arthritis

Objective: The aim of the current study was to examine the cartilage-specific binding property of polyarginine peptides (R4, 8, 12, and 16) and specifically to test octaarginine peptides for the optical imaging of articular cartilage in experimentally-induced arthritis in mice.

Methods: Four rhodamine-labeled polyarginine peptides each with a different-length arginine chain were injected into the knee joints of mice.

Results: Fluorescent signals were specifically detected in the cartilage pericellular matrix from the surface to the tide mark but were completely absent in the calcified layer or bone marrow. The number of arginine residues significantly influenced peptide accumulation in articular cartilage, with R8 accumulating the most. The fluorescent signal in the femoral condylar cartilage diminished when it was treated with Ch'ase ABC. R8 accumulation was significantly decreased in the degenerative cartilage of CAIA mice, and this was demonstrated both histologically and in 3D-reconstruction image by OPT.

Conclusion: R8 may be a useful new experimental probe for optical imaging of normal and arthritic articular cartilage.

REFERENCE

The application of elastin haploinsufficiency mice on lung disease with aging

Yuichi Shimizu*, Ayako Koga, Yoshitaka Ai, Risa Nonaka, Hiroshi Wachi, Yoshiyuki Seyama
Department of Clinical Chemistry, Hoshi University School of Pharmacy and Pharmaceutical Sciences

*Contact author: m843@hoshi.ac.jp

Keywords: Elastin, lung, Aging

Objective: Elastin (ELN) is a highly insoluble extracellular matrix (ECM) protein and the core protein of the elastic fibers that impart resilience to elastic such as skin, lungs, ligaments, and arterial walls. Loss of elasticity is observed in a range of serious diseases or age-related lesions, such as arteriosclerosis, emphysema, or chronic obstructive pulmonary disease (COPD). However, COPD is predisposed the increase of incidence, experimental model of COPD with aged mice is not still established. The purpose of this study was to investigate the expression of elastic fiber related protein with experimental lung disease in elastin haploinsufficiency mice.

Methods: ELN+/+ or ELN+/- was treated with elastase in nose to induce lung disease. The elastic fiber related mRNA and protein expression in lung was determined by RT-PCR and Western blot assay, respectively. Tropoelastin expression in BALF was also determined by Western blot assay.

Results: The mRNA expression of senescence maker protein 30 (SMP 30) significantly reduced in retire mice and ELN+-. The mRNA and protein expression of tropoelastin decreased and increased on the 3rd day after treatment with elastase in ELN+/+ and ELN+-, respectively. Increase of tropoelastin expression in BALF was observed in both ELN+/- treated with elastase and human lung disease such as COPD.

Conclusions: In this study, our data show that the heterozygous mutation mouse in elastin gene showed phenotype same as aged mouse and experimental model of lung disease with elastase showed similar data with human lung disease such as COPD. These results suggest that ELN+/- is very useful for the study of novel diagnostic procedures or new therapeutic approaches for the patients with lung disease.