

Mapping the Hidden Triggers of Jaw Joint Arthritis at the Cellular Level

Researchers map early molecular and cellular changes in the jaw joint that may trigger temporomandibular joint osteoarthritis

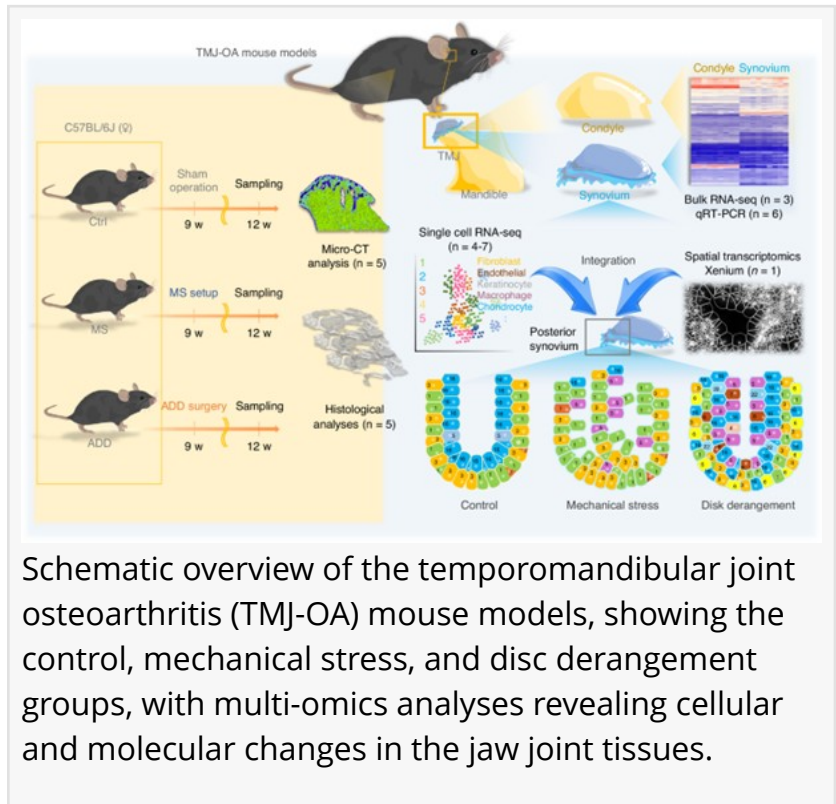
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Temporomandibular joint (TMJ) osteoarthritis affects millions of people, yet the earliest molecular events behind the disease remain poorly understood. Researchers have now mapped how mechanical stress and disk displacement trigger cellular changes in the jaw joint's synovium using advanced transcriptomic technologies. Their findings reveal inflammatory and fibrotic responses that may drive disease onset. The work provides a high-resolution cellular atlas of TMJ tissues, offering insights that could guide future therapies aimed at preventing joint degeneration.

Temporomandibular joint disorders (TMDs) affect a large portion of the global population and are a common source of chronic jaw pain and difficulty in chewing or speaking. Among these conditions, temporomandibular joint osteoarthritis (TMJ-OA) is the most prevalent degenerative disease of the jaw joint, marked by progressive cartilage damage, inflammation, and structural changes in surrounding tissues. Although TMJ-OA shares similarities with osteoarthritis in other joints such as the knee, its biological mechanisms remain less understood because far fewer clinical samples and studies are available for TMJ-OA.

To address this gap, researchers used advanced genomic and imaging technologies to investigate the earliest molecular responses that occur in the jaw joint under stress. By studying two experimental mouse models—one mimicking abnormal mechanical stress and the other simulating articular disc displacement—the team examined how these conditions affect the



Schematic overview of the temporomandibular joint osteoarthritis (TMJ-OA) mouse models, showing the control, mechanical stress, and disk derangement groups, with multi-omics analyses revealing cellular and molecular changes in the jaw joint tissues.

synovium, a soft tissue lining that plays an important role in joint health. Their findings were published in Volume 18 of the [journal International Journal of Oral Science](#) on March 12, 2026.

The research was led by Associate Professor and Vice Director Fumiko Yano from the Department of Biochemistry, Graduate School of Dentistry, Showa Medical University, Japan.

To better understand how TMJ-OA begins, the team designed a comprehensive experimental framework combining several cutting-edge methods, including histological analysis, bulk RNA sequencing, single-cell RNA sequencing, and spatial transcriptomics. These approaches allowed the researchers to study gene activity and cellular interactions across thousands of individual cells while also mapping where those cells were located within the joint tissue. The models simulated two common triggers of TMJ-OA: mechanical stress caused by malocclusion and inflammation resulting from articular disc derangement.

The analyses revealed striking structural and molecular changes in the joint tissues. In both models, the researchers observed degeneration of cartilage and abnormal remodeling of the underlying bone. In particular, the synovial tissue surrounding the articular disc displayed signs of inflammation, fibrosis, and metabolic shifts. Mechanical stress promoted adipogenic changes in the synovium, while disc displacement triggered fibrotic thickening and hyperplasia of the synovial lining. These tissue-level changes were accompanied by activation of genes linked to inflammation and cartilage degradation.

At the cellular level, the study identified diverse populations of fibroblasts, endothelial cells, macrophages, and keratinocyte-like cells interacting within the synovial environment. Single-cell sequencing revealed that fibroblast clusters communicated with immune and vascular cells through signaling pathways associated with inflammation and mechanotransduction. Spatial transcriptomics further showed that inflammatory markers and matrix-degrading enzymes were concentrated in the posterior synovium of the articular disc, suggesting that this region may act as an early hotspot for disease initiation.

“By integrating single-cell and spatial transcriptomic technologies, we were able to visualize how mechanical stress and structural changes reshape the cellular landscape of the temporomandibular joint,” explains Dr. Yano. “This approach allowed us to identify molecular signals and cell-to-cell interactions that may trigger the earliest stages of TMJ-OA.”

The findings also highlighted specific molecular pathways that could serve as potential therapeutic targets. For instance, the researchers observed activation of inflammatory signaling networks and endothelial Notch signaling in the synovial microenvironment. These pathways are known to regulate tissue remodeling and inflammation, suggesting that they may contribute to joint degeneration when persistently activated.

“Our study provides a high-resolution map of the cellular responses occurring in the synovium during early TMJ degeneration,” adds Dr. Yano. “Understanding these mechanisms may help

researchers design targeted strategies to prevent or slow the progression of the disease.”

Beyond advancing fundamental knowledge of TMJ biology, the study could have broader implications for joint disease research. The integrated methodological framework developed by the team can be applied to other musculoskeletal disorders to better understand how mechanical stress and inflammation reshape tissue microenvironments. In the short term, the research offers a valuable reference for scientists studying jaw joint disorders.

Over the longer term, insights from this work could support the development of early diagnostic markers or therapies aimed at preventing irreversible cartilage damage, potentially improving the quality of life for people suffering from chronic TMJ pain and dysfunction.

Reference

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Website: <https://www.showa-u.ac.jp/en/>

About Associate Professor Fumiko Yano from Showa Medical University, Japan

Dr. Fumiko Yano is currently working as an Associate Professor and Vice Director in the Department of Biochemistry at the Graduate School of Dentistry at Showa Medical University, Japan. She completed her Ph.D. in Medicine from the University of Tokyo in 2006. Her research focuses on the molecular mechanisms of musculoskeletal diseases, regenerative medicine, and joint biology. She has served as a delegate of the Japanese Society for Regenerative Medicine since 2018. She has authored 76 scientific publications and holds two patents. Her work has received more than 3,774 citations, reflecting her significant contributions to biomedical and translational research.

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Yini Bao
IJOS Editorial Office
2885546461 ext.
ijos@scu.edu.cn

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