**Supporting information**

**Supplementary Table. 1.** **Donor and sample information.**The table includes descriptions of sample names, cell counts, sample IDs, donor status, OA grade, donor demographics (age, sex, BMI), and detailed sampling locations.

**Supplementary Table. 2.** **Supplementary Table. 2: supplemental table of gene names.** This table provides a comprehensive list of abbreviations and full names for reference.

**Supplementary Fig. 1. Quality control indices of scRNA-seq:** (a) Statistical plots of nFeature\_RNA, nCount\_RNA, and percent.mt showing that the proportion of mitochondrial genes in the sample cells meets the standards. (b) Principal component analysis (PCA) of highly variable genes using the "RunPCA" function. (c) Selection of significant principal components using the "ElbowPlot" function and noise reduction through permutation tests. (d) Heatmap displaying the expression of differentially expressed genes in each cluster.

**Supplementary Fig. 2. Sample information:** (a) Information on 8 patients from the GSA-Human database and 8 patients from the Orthopedics Department of Peking University Shenzhen Hospital. (b) Abbreviations, full names, and marker genes of major cell subtypes. (c) t-Distributed Stochastic Neighbor Embedding (tSNE\_2) and Uniform Manifold Approximation and Projection (UMAP) plots showing cell clustering results of the scRNA-seq. (d) Annotation of cell populations based on known cell markers using the SingleR package. (e) Genes related to each single-cell type and their expression in different cell types inferred by SingleR.

**Supplementary Fig. 3. The violin plot revealed the expression of major class markers.**

**Supplementary Fig. 4. The expression levels of major class markers shown on UMAP plots.**

**Supplementary Fig. 5. Expression of major markers of cell subtypes:** (a) Expression of differentially expressed genes in subtypes of synovial fibroblasts and meniscus chondrocytes. (b) Expression of differentially expressed genes in immune cell subtypes. (c) Expression of differentially expressed genes in endothelial cell subtypes.

**Supplementary Fig. 6. Cross-talk analysis of OA-related pathways:** (a) Cross-talk analysis of IGF Pathway, PTN Pathway, MK Pathway, and FGF Pathway in different groups. (b) Receptor-ligand interaction analysis in Normal Meniscus, Degenerated Meniscus, Normal Synovium, and Degenerated Synovium.

**Supplementary Fig. 7. Cross-talk analysis in cartilage regulatory pathways.**Cross-talk analysis of ANGPTL Pathway, and PROS Pathway in different groups.

**Supplementary Fig. 8. Cross-talk analysis in cartilage regulatory pathways.** Cross-talk analysis of PDGF Pathway in different groups.

**Supplementary Fig. 9. Cross-talk analysis in neovascularization pathways.** Cross-talk analysis of VISFATIN Pathway, and VEGF Pathway in different groups.

**Supplementary Fig. 10. Cross-talk analysis in neovascularization pathways.** Cross-talk analysis of SEMA3 Pathway in different groups.

**Supplementary Fig. 11. Cross-talk analysis in macrophage polarization pathways.**Cross-talk analysis of GALECTIN Pathway, and CXCL Pathway in different groups.

**Supplementary Fig. 12. Cross-talk analysis in macrophage polarization pathways.** Cross-talk analysis of TGFβ Pathway in different groups.

**Supplementary Fig. 13. Cross-talk analysis in inner meniscus samples.** Cross-talk analysis of key osteoarthritis pathways, including the insulin-like growth factor (IGF), pleiotrophin (PTN), fibroblast growth factor (FGF), and Midkine (MK) pathways; cartilage regulatory pathways, such as angiopoietin-like protein (ANGPTL), protein S (PROS), and platelet-derived growth factor (PDGF) pathways; neovascularization pathways, including VISFATIN, vascular endothelial growth factor (VEGF), and semaphorin 3 (SEMA3) pathways; and macrophage polarization pathways, such as the GALECTIN pathway, CXCL pathway, and transforming growth factor beta (TGF-β) pathway, in inner meniscus samples.

**Supplementary Fig. 14.** **Functional enrichment of HALLMARK TNFA SIGNALING VIA NFKB. Described by GSEA and scMetabolism in normal and degenerated meniscus and synovium samples:** (a) In CXCL14+ fibroblasts (CXCL14+ Fibs) and synovial sublining fibroblasts (SSFs), inflammation-related gene sets are highly expressed in normal synovial samples compared to normal meniscus samples. In contrast, these gene sets show higher expression levels in degenerated meniscus samples than in degenerated synovial samples. When considering synovial and meniscus samples as a whole, these inflammation-related gene sets are highly expressed in inflammatory meniscus and synovial samples in SSF, Fibroblast\_1 (Fib\_1), Chondrocyte\_1 (Ch.1), and Chondrocyte\_4 (Ch.4) cells. (b) Conversely, in CXCL14+ Fibs, synovial lining fibroblasts (SIFs), and Chondrocyte\_2 (Ch.2) cells, the inflammation-related gene set "HALLMARK TNFA SIGNALING VIA NFKB" is expressed at lower levels in degenerated samples.

**Supplementary Fig. 15. Functional enrichment of HALLMARK HYPOXIA:** **(a) and HALLMARK APOPTOSIS(b). Described by GSEA and scMetabolism in normal and degenerated meniscus and synovium samples.**

**Supplementary Fig. 16. GO\_BP Analysis of Degenerated (Meniscus and Synovium) vs. Normal (Meniscus and Synovium):** (a) Compared to normal samples, GO\_BP terms related to inflammatory injury, such as "response to reactive oxygen species," were downregulated in Ch2 cells and *CXCL14*+ Fibs in degenerated samples. (b) GO\_BP terms related to ATP metabolic process and OA anti-inflammatory repair were upregulated in Ch2 cells and *CXCL14*+ Fibs in degenerated samples compared to normal samples.

**Supplementary Fig. 17. Functional enrichment analysis reveals differences in fibroblast subtypes:** (a) Functional enrichment of HALLMARK TNFA SIGNALING VIA NFKB, and HALLMARK INTERFERON GAMMA RESPONSE in Fib\_1 and Fib\_2 cells from normal and degenerated meniscus and synovium samples, described by GSEA and scMetabolism. (b) GSEA enrichment analysis of HALLMARK EPITHELIAL MESENCHYMAL TRANSITION and HALLMARK\_TNFA\_SIGNALING\_VIA\_NFKB in Fib\_2 cells from Normal and Degenerated samples in meniscus and synovium.

**Supplementary Fig. 18. EdU staining was used to assess cell proliferation on the lower surface of the meniscus in both the Normal and Degenerated groups. The spatial distribution of proliferating cells was evaluated in human meniscus samples with different clinical symptoms and radiographic grades (Kellgren-Lawrence grades 0, 3, and 4).**