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CORR Insights

CORR Insights[®]: A Novel System Improves Preservation of Osteochondral Allografts

Charles J. Malemud PhD

Where Are We Now?

here is a relative deficiency in approved drugs for the treatment of human osteoarthritis when compared to the significant biopharmaceutical commitment for researching medical therapies for rheumatoid arthritis. Because osteoarthritis is a slowly progressive musculoskeletal disorder, clinical symptoms

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All ICMJE Conflict of Interest Forms for authors and *Clinical Orthopaedics and Related Research*[®] editors and board members are on file with the publication and can be viewed on request. generally emerge after significant damage to joint articular cartilage has already occurred. However, when inflammation becomes a component of the osteoarthritis process, which often is the case, the destruction of articular cartilage becomes more prominent and highly aggressive. Additionally, repair of articular cartilage in osteoarthritis is inefficient, owing to the loss of chondrocytes by cell death. It is also likely that the vitality of the remaining resident chondrocytes become compromised as well.

Where Do We Need To Go?

To counteract the relative paucity for developing new therapeutics for osteoarthritis, one strategy that has leaped to the forefront of osteoarthritis research involves employing the relatively novel approach of grafting chondrocyte progenitor cells on to the lesional surface of osteoarthritis articular cartilage. The purpose of this engrafting is primarily to provide a fresh source of viable articular chondrocytes which may be able to suppress cartilage extracellular matrix protein degradation as well as providing a cellular source for promoting cartilage repair to the damaged articular cartilage surface [1]. Thus, a cell-based therapy for human osteoarthritis may eventually result in a complimentary approach to any new pharmaceutical-based therapies for osteoarthritis.

How Do We Get There?

With respect to developing a cellbased therapy for osteoarthritis it has been proposed that a "bank" of stored chondrocytes be routinely available for osteochondral allografting. However, chondrocyte vitality must be stabilized in these stored human chondrocytes if they are eventually to be used for osteochondral allografting in human osteoarthritis. The experimental study using canine chondrocytes reported by Cook and colleagues in this issue of $CORR^{(R)}$ was conceived to achieve just such a purpose. In their study, canine

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C. J. Malemud PhD (🖂)

Department of Medicine, Case Western Reserve University School of Medicine, 2061 Cornell Road, Cleveland OH 44106, USA e-mail: cjm4@cwru.edu

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osteochondral allografts were preserved for 28 or 60 days using either a conventional tissue bank storage standard or the newly-developed Missouri Osteochondral Allograft Preservation System (MOPS). The results of this study indicated that although infection rates during storage between the two preservation systems were comparable,

osteochondral allografts maintained in MOPS retained significantly greater chondrocyte viability at day 60. Although extending the use of MOPS employed in this canine model of osteochondral allografting to the repair of large cartilage defects characteristic of human osteoarthritis remains a goal yet to be fully achieved, improving chondrocyte viability by storage of the cells in MOPS appears to be a step in the right direction.

Reference

1. Malemud CJ. Repair of injury to articular cartilage with chondrocyte progenitor cells. *Rheumatology (Sunnyvale)*. 2013;3:2.