Allyson Caisley

Danvers, IL

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Junior
Biological Sciences

Faculty Mentor: Dr. Aaron Stoker, Orthopaedic Surgery; Dr. James Cook, Orthopaedic Surgery

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The Effect of Passage on the Metabolic Profile of Osteoarthritic Chondrocytes

Allyson B Caisley, Spencer E. DeLucia, Anna N. Sellentrup, James L. Cook, and Aaron N Stoker

Introduction

Clinically, there is significant patient to patient variability in the progression and development of Osteoarthritis (OA). Previous studies have shown that there is also patient-to-patient variability in the metabolic responses of OA chondrocytes during initial monolayer culture. This study was designed to determine the effect of culture passage on the metabolic responses of OA chondrocytes, by comparing biomarker production levels of cells at passage 0 (P0) and passage 1 (P1). We hypothesized that when comparing P0 to P1, high producing cells at P0 would be high producers at P1, correlations between biomarkers at P0 would be maintained at P1, and that there would be strong positive correlations between P0 and P1 for relative biomarker production levels.

Methods

With IRB approval and informed patient consent, chondrocytes were recovered from 10 patients undergoing TKA surgery. Once the cells in P0 and P1 were 90% confluent, media was changed and collected 3 days later for analysis of inflammatory and degradative biomarkers. Cells were grouped into upper and lower 50% production groups at P0, and a Mann-Whitney Rank Sum test was used to determine significant differences between groups at P1. A Kruskal Wallace test was used to group biomarkers into high, mid, and low producing groups at P0 and P1. A Pearson's correlation was used to identify correlations between biomarkers at P0, P1 and between P0 and P1.

Results

When group at P0 biomarker levels, there was not a significant difference between groups at P1, indicating that the cells did not maintain this stratification in biomarker production P1. There was more variability in biomarker production level observed at P0 compared to P1. There were few similar biomarker production correlations observe between P0 and P1.

Discussion

Data from this study suggest that there is significant change in the metabolic output of OA chondrocytes in vitro after just one passage and patient-to-patient variability is lost. Going forward, these passage-based changes may be used to identify key cellular pathways associated with the pathobiology of OA.