INTRODUCTION:
Anteromedial Gonarthrosis (AMG) is a distinct phenotype of osteoarthritis first described by White et al in 1991 [1]. Within this pattern of disease, the anterior third of the medial tibial plateau exhibits full thickness cartilage loss. The middle third has damaged partial thickness cartilage, and the posterior third has retained cartilage, which is seen on macroscopic visual assessment, using an Outerbridge grade [2], to be normal.

This study investigates the cellular and molecular features of progressive severities of cartilage damage within this phenotype.

METHODS:
Ten medial tibial plateau specimens were collected from patients undergoing unicompartmental knee replacements (Figure 1). The cartilage within the region of macroscopic damage was divided into equal thirds: T1 (most damaged), to T3 (least damaged). The region of macroscopically undamaged cartilage was taken as a 4th sample, N. The specimens were prepared for histological (Haematoxylin and Eosin, and Safranin-O) and immunohistochemical analysis (Type I and II Collagens). The same regions were snap frozen in liquid nitrogen and immunoassays were undertaken for Type I and II Collagens and GAG content. Real time PCR compared gene expression between regions T and N.

RESULTS:
The histology, graded using the Osteoarthritis Research Society International Cartilage Histology Assessment System (OCCHAS) [3] showed there was a decrease in grade across the four regions, with progressively less fibrillation between regions T1, T2 and T3 (p<0.001) (Figure 2). Region N was histologically normal.

The GAG immunoassay showed decreased levels with increasing severity of cartilage damage (p<0.001) (Figure 3). There was no significant difference in the Type II Collagen content or gene expression between regions (Figure 4).

DISCUSSION:
The GAG and Type II Collagen assays produced predictable results in keeping with the literature. The presence of Type I Collagen in end stage osteoarthritis is documented in the literature, however, its presence in histologically normal cartilage is an area of contention. We have shown its presence in histologically normal cartilage, using several laboratory techniques, in the AMG phenotype.

This increase in Type I Collagen may represent very early changes of the cartilage matrix within the osteoarthritic disease process. It may be a possible therapeutic target for disease modification or treatment. Further work will involve a similar detailed analysis of normal cartilage in human knee joints.

REFERENCES: