INHIBITING ANTERIOR TIBIAL TRANSLATION AND AXIAL TIBIAL ROTATION PROTECTS THE ANTERIOR CRUCIATE LIGAMENT AT THE EXPENSE OF FEMORAL ARTICULAR CARTILAGE

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INTRODUCTION

The vital players involved in non-contact anterior cruciate ligament (ACL) injury mechanisms are anterior tibial translation and axial tibial rotation [1]. While the inhibition of anterior tibial translation and axial tibial rotation is suggested to reduce the risk of sustaining ACL failure during landing, it is not yet known whether this inhibition can also lead to greater femoral cartilage damage. This study sought to examine the femoral cartilage damage profile upon simulated landing impact of the porcine knee joint with and without the inhibition of anterior tibial translation and axial tibial rotation.

METHODS

Twelve porcine hind legs (pig age ~2months; weight ~40kg) were procured at a local abattoir (Primary Industries, Singapore) and prepared according to a previous protocol [2]. We adapted a similar testing platform and impact protocol [2] but introduced additional fixtures for inhibiting anterior tibial translation and axial tibial rotation to assess their effects on the extent of femoral cartilage damage. The specimens were divided into 3 groups: (I-) no *I*mpact, (I+) *I*mpact, (I+I) *I*mpact with *I*nhibition. The impact test was ceased when either a significant compressive force drop was observed (ACL failure) or a visible bone fracture was present. ACL failure was confirmed via dissection. Femoral cartilage damage was examined using histology and microCT. Osteochondral explants were extracted from both medial and lateral femoral condyles, and were subjected to formalin fixation prior to microCT scans (SMX-100CT, Shimadzu, Japan) and, thereafter, used for histology using procedures previously described [2]. One-way ANOVA was performed between test groups to identify any differences in Mankin scores, cartilage thickness and volume. All significance levels were set at p=0.05.

RESULTS AND DISCUSSION

Group I+ underwent ACL failure while group I+I remained ACL-intact. The femoral cartilage in both groups generally revealed cartilage disruption at superficial-middle zones (Figure 1A), but the Mankin scores were substantially higher in group I+I than in group I+ (Figure 1B). The femoral cartilage thickness in both groups was notably reduced at the lateral condyle, compared to the control group (Figure 2A). The cartilage volume was not observed to vary significantly between impact groups (Figure 2B).

The purpose of this study was to examine whether the inhibition of anterior tibial translation and axial tibial rotation will promote femoral cartilage damage. One key limitation of this study was the use of porcine specimens to investigate post-traumatic cartilage damage. Both the porcine and human knee joints were found to incur ACL failure upon excessive impact compression [2,3], which indicated similar failure mechanisms. Our study suggested that the inhibition of anterior tibial translation and axial tibial rotation prevents the sliding of the tibiofemoral contact upon a landing impact and thus minimizes ACL strain. However, this may focus a majority of the compressive impact onto the contact region, which can induce cartilage lesions and deformation, especially at both medial and lateral femoral condyles.

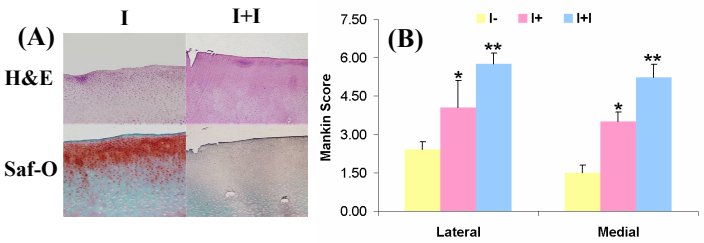

Figure 1: (A) Histological images of I and I+I groups; (B) Comparison of Mankin scores between groups at both condyles.
*Significant difference compared to I- ($p<0.05$)
**Significant difference compared to I- and I+ ($p<0.05$)

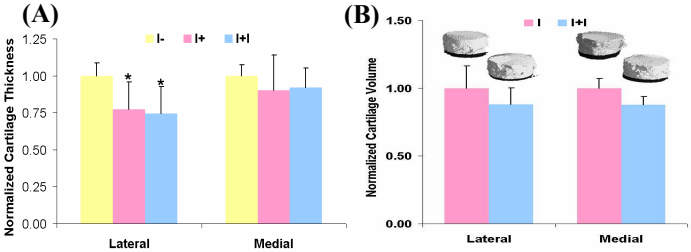

Figure 2: Comparison of normalized (A) cartilage thickness and (B) volume between groups at both condyles. *Significant difference compared to I-

CONCLUSIONS

Collectively, our results delivered that the inhibition of anterior tibial translation and axial tibial rotation can protect the ACL during impact landing, but does not necessarily protect the femoral cartilage from sustaining compressive damage. Our findings further demonstrated that the femoral cartilage damage was considerably greater during inhibition, which may increase the risk of developing osteoarthritis at the femoral condylar contact regions.

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