INTRODUCTION:
Quadricipitates central activation failure (CAF) occurs frequently following anterior cruciate ligament reconstruction (ACLr) and lingers beyond the rehabilitation period. CAF impairs the ability to fully activate the quadricipitates and has been demonstrated to alter lower extremity biomechanics. Neuromuscular fatigue similarly reduces volitional activation and neuromechanical control strategies within the affected muscle. In healthy persons, fatigue has been shown to impair quadricipitates central activation, with this effect being magnified in the presence of muscle weakness. How the quadricipitates respond to neuromuscular fatigue following ACL injury/reconstruction remains unknown. With individuals returning to activity following ACLr likely experiencing quadricipitates weakness, CAF, and neuromuscular fatigue, understanding how these three impairments interact seems vital so that strategies to combat the potentially hazardous consequences can be developed to better protect against graft failure upon return to activity.

This study examined the effects of neuromuscular fatigue on quadricipitates weakness and CAF following ACLr. Additionally, this study determined the influence of neuromuscular fatigue on knee joint biomechanics following ACLr.

METHODS:
Seventeen individuals 7-10 months following ACLr (10 male, 7 female; age: 21.4±4.73years; height: 1.75±0.08m; mass: 76.5±11.85kg) and 16 healthy, control subjects (5 male, 11 female; age: 23.38±4.11years; 1.71±0.08m; mass: 68.2±10.17kg) participated. Subjects had quadricipitates strength (maximum voluntary isometric contraction [MVIC]) and the central activation ratio (CAR) recorded pre- and post-fatigue. Maximal fatigue was induced via sets of eight double-leg squats. There was no limit to the number of squats an individual could complete. Knee sagittal and frontal plane biomechanics were recorded while subjects performed a dynamic landing activity pre- and post-fatigue. The dynamic landing consisted of a double leg take-off, followed by a single-leg landing on a force platform (Figure 1) located one meter away. Immediately upon landing, subjects aggressively laterally hopped to the opposite side. Data were analyzed via a standard inverse dynamics analysis. Statistical analysis consisted of 2x2 (time x group) repeated measures ANOVAs.

RESULTS:
Both groups demonstrated smaller knee flexion angles (initial contact [IC]: \(p=0.018\); peak stance [PS]: \(p=0.002\)) (Figure 1) and moments (\(p<0.001\)) (Figure 2) post-fatigue. Both groups also landed with less knee abduction (IC: \(p=0.005\); PS: \(p=0.017\)) and smaller abduction moments (\(p=0.024\)) following fatigue. The ACLr group was less flexed at PS (\(p=0.009\)) and experienced a smaller flexion moment than controls regardless of fatigue state (\(p<0.001\)). Following fatigue, all subjects (ACLr and control) demonstrated significantly lower MVIC (\(p<0.001\)) and CAR (\(p=0.003\)) (Table 1) values. No group differences were detected for either MVIC (\(p=0.13\)) or CAR (\(p=0.17\)).

Table 1. Quadricipitates strength and central activation ratio data (mean ± standard deviation).

<table>
<thead>
<tr>
<th>ACLr</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-Fatigue</td>
</tr>
<tr>
<td>MVIC (Nm/kg)</td>
<td>2.03±0.57</td>
</tr>
<tr>
<td>CAR</td>
<td>0.82±0.11</td>
</tr>
</tbody>
</table>

DISCUSSION:
Subjects demonstrated less knee flexion at IC and PS with concurrent reductions in the external knee flexion moment following fatigue. This sagittal plane biomechanical profile has been demonstrated previously in healthy adults following lower extremity neuromuscular fatigue. While a more extended knee position may protect against collapse of the lower extremity on landing, increases in both the knee extension angle and moment are suggested contributors of non-contact ACL injury. As such, this adaptive strategy may be hazardous.

Both ACLr and control subjects demonstrated smaller knee abduction angles and moments following fatigue, which was unexpected based on previous research. However, differences in the fatigue protocol employed between this and previous studies may account for these discrepancies.

Both groups demonstrated quadricipitates weakness and CAF following fatigue with control subjects concurrently altering knee joint biomechanics. These biomechanical alterations may prove injurious and have been linked to non-contact ACL injury risk, confirming the need to consider fatigue-resistant training within non-contact ACL prevention programs. Further, the reductions in CAR as a result of fatigue confirm fatigue is, at least in part, centrally mediated and strategies to train these central control components ought to be included within fatigue-resistant training. That ACLr subjects demonstrated potentially injurious mechanisms prior to fatigue suggests that the reconstruction and/or rehabilitation processes are not sufficiently reducing the biomechanical risk factors for re-injury when individuals return to activity.

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