INTRODUCTION:
Femoroacetabular impingement (FAI) is recognized as a pathomechanical process that leads to hip osteoarthritis (OA) [1-3]. It is hypothesized that mechanical stimuli are more prominent in an impinged joint at a higher range of motion. Adverse loading conditions can impose elevated mechanical stimuli levels at the articulating surfaces and underlying subchondral bone, which has a predominant mechanical role in early OA [4]. Despite past attempts to characterize mechanisms by which cam FAI (spherical femoral head-neck deformity) leads to OA, very little has been done to integrate clinical patient-specific data to determine stresses and strains within the symptomatic hip joint. The aim of this research is to determine the levels of mechanical stimuli within the hip, examining the effects of severe cam FAI on the onset of OA, using patient-specific biomechanics data, CT data, and finite element analysis (FEA).

METHODS:
Joint reaction forces were obtained from inverse dynamics, calculated from joint kinematics and kinetics, for a dynamic squat motion of patients with cam FAI [5]. Patients were control-matched for gender, BMI, and physical activity level. Pelvic CT radiographs were compiled and segmented into 3-D models using 3D-DOCTOR (Able Software Corp, MA). Two segmented symptomatic and two control-matched models were then resurfaced using SolidWorks (Dassault Systèmes, MA) to minimize geometric artefacts. Alpha angles of the femur models were verified with the CT radiographs to ascertain accuracy. A cartilage layer of varying thickness was created using an offset method from the acetabulum. The models were imported into ANSYS (ANSYS, PA) for FEA and meshed with tetrahedral SOLID187 elements. Bone was modeled as a linear elastic orthotropic material [6]; and cartilage as a linear elastic isotropic material [7]. The reaction forces for patients and controls were applied onto their respective models, where two quasi-static loading scenarios were considered: (1) stance and (2) maximum force endured during the impinged squat. The femur was oriented with respect to the acetabulum according to the squat interval. Maximum shear stress was analyzed to determine the adverse loading conditions within the joint and strain energy density (SED) was determined to examine its effect on the initiation of bone remodeling [8].

RESULTS:
For the two patients with severe cam FAI, changes in max-shear stress in the cartilage was marginal for both when going from the standing to squatting position. However, significantly higher max-shear stresses were found behind the acetabular cartilage layer, localized at the antero-superior acetabulum during the squat position, as seen in Figure 1. Marginal increases in SED were noticed from stance to squat.

Figure 1: Left hip sagittal view of FAI Patient 1 indicating the max-shear stress distributions during the squat position. The arrow denotes the peak stress point (16.86 MPa) at the antero-superior acetabulum, situated behind the cartilage layer.

For the control subjects, mechanical stimuli were found to be well distributed on the cartilage layer and at the acetabulum during the standing and squatting positions. During the squat position, marginal increases in max-shear stresses were observed at the antero-superior cartilage and acetabulum. No significant changes in SED were noticed. Table 1 summarizes the peak mechanical stimuli observed for the cam patients and control subjects for the two quasi-static loading scenarios.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Alpha Angle (deg)</th>
<th>Max Shear Stresses Carritlage (MPa)</th>
<th>Max Shear Stresses Acetabulum (MPa)</th>
<th>Max SED Acetabulum (J/cm²)</th>
<th>Max Shear Stress Carritlage (MPa)</th>
<th>Max Shear Stresses Acetabulum (MPa)</th>
<th>Max SED Acetabulum (J/cm²)</th>
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<tbody>
<tr>
<td>FAI 1</td>
<td>45 acet, 85 renal</td>
<td>3.05 3.00 1.00</td>
<td>2.52 2.00</td>
<td>0.03 0.01</td>
<td>3.05 3.00 1.00</td>
<td>2.52 2.00</td>
<td>0.03 0.01</td>
</tr>
<tr>
<td>FAI 2</td>
<td>45 acet, 85 renal</td>
<td>3.05 3.00 1.00</td>
<td>2.52 2.00</td>
<td>0.03 0.01</td>
<td>3.05 3.00 1.00</td>
<td>2.52 2.00</td>
<td>0.03 0.01</td>
</tr>
<tr>
<td>Case 1</td>
<td>45 acet, 85 renal</td>
<td>3.05 3.00 1.00</td>
<td>2.52 2.00</td>
<td>0.03 0.01</td>
<td>3.05 3.00 1.00</td>
<td>2.52 2.00</td>
<td>0.03 0.01</td>
</tr>
<tr>
<td>Case 2</td>
<td>45 acet, 85 renal</td>
<td>3.05 3.00 1.00</td>
<td>2.52 2.00</td>
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</table>

DISCUSSION:
The resultant mechanical stimuli were found to be much higher at the squatting position for the cam FAI patients, where the joint was at its most impinged state. Squatting orients the deformity into the antero-superior acetabulum, increasing contact with the articular cartilage and labral regions, thus resulting in higher peaks behind the cartilage on the acetabular surface. The resultant magnitudes agree with a recent study of an idealized parametric model using a similar stand-to-sit motion [7]. However in that study, only peak von Mises stresses in the cartilage were reported as opposed to the entire joint assembly.

The resultant location of the peak max-shear stresses and SED, the antero-superior regions of the acetabulum, corresponds well with the region of initial cartilage degradation and early OA observed during open surgical dislocation [2-3].

Though the resultant magnitudes are not representative of actual stresses experienced in vivo, they are beneficial indications of relative hip joint reactions due to cam FAI. The inclusion of muscle vectors and soft tissues may provide a more accurate result of hip joint contact stresses, yet may also increase the resultant magnitudes.

Due to the relatively low elastic modulus of cartilage, articular cartilage transfers the loads to the acetabulum, amplifying the stimuli reaching the subchondral bone. With the cartilage layer transferring the load onto the acetabulum, it is hypothesized that elevated levels of mechanical stimuli could increase the rate of bone remodeling [8] in the subchondral bone, causing stiffening of the subchondral plate [4] and consequently accelerating the onset of OA. Since mechanical stimuli results are unique to their patient-specific loading parameters and conditions, it would be difficult to determine a patient-specific threshold to provoke bone remodeling at this stage.

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REFERENCES:

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