POLYETHYLENE DAMAGE ON MODULAR, UNCONSTRAINED Tibial COMPONENTS:
DO ALL RETRIEVED INSERTS SHOW EVIDENCE OF BACKSIDE MICROMOTION AND EDGE LOADING?

**Introduc**tion: Two design features common to many contemporary total knee replacements (TKR) are 1) modularity between the metal tibial baseplate and polyethylene insert, and 2) coronally unconstrained (flat-on-flat) tibio-femoral articulations. Modularity provides surgeons the choice of different polyethylene thickness, which aids soft tissue balancing. Coronally flat articulations allow relatively unconstrained internal/external femoral rotation during functional activities, aiding in extensor mechanism function. However, the long-term wear performance of these design features has recently been questioned.

Interface micromotion and damage on the backside of retrieved modular polyethylene inserts has led to the suggestion that the modular capture mechanism degrades with physiologic loading.[2] Studies of tibial-femoral motions during dynamic activities suggest that coronally flat components are vulnerable to medial-lateral lift-off and edge-loading, decreasing the femoral contact area and increasing polyethylene surface stress.[5] However, backside damage severity varies greatly between TKR designs, and some modular capture designs appear more susceptible to disruption and backside damage. In addition, few retrieved inserts from contemporary TKR’s have shown polyethylene damage consistent with lift-off and edge loading.

This study presents articular and backside surface damage measurements from retrieved modular polyethylene inserts of the same design with coronally flat-on-flat articulations. It was hypothesized that 1) backside damage would increase with time in-situ, and 2) articular damage would be located closer to the medial or lateral edge with a high incidence of delamination if substantial edge loading occurred in-vivo.

**Methods:** Thirty seven posterior cruciate ligament retaining TKR’s of the same design (Series 7000, Stryker Howmedica Osteonics) were retrieved at autopsy (n=12) after 41±21 (1-74) months in-situ and at revision TKR (n=25) after 26±21 (1-71) months in-situ. Reason for revision included loose patellar, patellar resurfacing(20%), and supracondylar fracture(4%). Patient age and weight averaged 71±(54-83) years and 86±(57-115) kg, respectively. All components were implanted by the same surgeon and consecutively retrieved over an eight year period. Polyethylene inserts were ≥6 mm thick with a full peripheral rim press-fit and supplemental anterior locking wire capture mechanism. The Series 7000 tibial baseplate has a non-polished surface finish at the modular interface. All tibial, femoral and patellar components were fixed with cement.

Articular and backside surface damage were evaluated using light microscopy. Eight different damage modes were visually assessed and the number of inserts with each damage mode was determined. For articular damage, the circumference of the medial and lateral damage regions were digitized and the damage size (% of articular surface area) was measured using a published digital image analysis technique.[3] The area centroid was calculated and it’s location on the medial and lateral plateau determined relative to the medial-lateral (ML location) and anteroposterior (AP location) edges of the insert, with a value of 50% representing a central location. An ML location of <33% was measured using a published digital image analysis technique.[3]

**Discussion:** In agreement with the stated hypothesis, the backside damage score increased with time in-situ. However, the damage morphology suggests that axial compression of the polyethylene insert against the baseplate produced the observed damage rather than interface micromotion. This finding is consistent with mechanical testing of autopsy retrieved Series 7000 tibial components showing that inserts with the greatest amount of insert micromotion have the lowest amount of backside damage and the shortest duration of function.[4] This type of backside damage appears clinically benign in this cemented TKR prosthesis and does not support the notion that this modular locking mechanism becomes increasingly unstable with physiologic loading.

Concerns of high contact stresses associated with edge-loading were unsupported by these retrievals. Evidence of damage modes associated with reduced contact area and high contact stresses were not observed. Delamination was not substantial and the damage areas were located on the central third of the articular surface (Table 1). The rotated damage patterns are consistent with the surgical alignment of the femoral component and the femoral external rotation that occurs with knee flexion after TKA.[3] Similarly, damage located on the posterior interface of the articular surface correlates well with the trend toward more backside damage to this region. This suggests that compressive forces from contact at the articular surface are transmitted to the backside surface. Condylar lift-off, if it occurs, does not appear to substantially impact polyethylene damage in these coronally flat-on-flat articulations.

When assessing the performance of contemporary TKR’s, it is imperative that design features associated with modularity and the tibial-femoral articulation in successful TKR’s remain distinct from the material and design factors previously shown to contribute to wear and implant failure.

**Articular Surface Damage:** Articular damage covered 48±16% and 47±14% of the medial and lateral surfaces, respectively. The most frequent damage modes were burnishing and scratching. Delamination occurred on 4 (11%) inserts, but involved <2% of the articular surface. Damage patterns were internally rotated, with the lateral damage centroid located significantly more posterior than the medial damage centroid (paired t-test, p<0.05). The ML location was greater than 33% for every insert (Table 1), and therefore not one insert had a damage area centroid located in the medial or lateral third of the articular surface. There were no significant correlations between articular damage area and in-situ time or average backside damage score (Spearman Correlation, p>0.05).

**Backside Damage Score**

![Fig. 1: Backside damage score was correlated (r=0.48) with in-situ time. Regression: average backside score = 1.6 + (0.05 * months in-situ)](image)

**Table 1:** Location of medial and lateral damage area centroid.

<table>
<thead>
<tr>
<th>Location</th>
<th>Medial</th>
<th>Lateral</th>
</tr>
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<tbody>
<tr>
<td>AP location</td>
<td>49.4±7.0%</td>
<td>45.6±7.0%</td>
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<tr>
<td>AP range</td>
<td>32.7% to 64.9%</td>
<td>33.2% to 64.9%</td>
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<tr>
<td>ML location</td>
<td>42.7±4.3%</td>
<td>43.4±3.9%</td>
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<tr>
<td>AP range</td>
<td>35.3% to 52.8%</td>
<td>34.5% to 49.3%</td>
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