Corrosion and Damage Mechanisms in Retrieved Long-Term TKA Femoral Components

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Introduction: The release of metal debris and ions has raised concerns in joint arthroplasty. Metal debris and ions can be generated in THA through metal-on-metal wear and mechanically assisted crevice corrosion (MACC) at modular junctions [1]. More recently, inflammatory-cell induced corrosion (ICIC) was identified as a possible source of metal debris and/or ions [2]. MACC has been shown to occur at modular junctions in TKA, however little is known about the prevalence of other sources. The purpose of this study was to determine the sources of metallic debris and ion release in long-term implanted (in vivo > 15y) TKA femoral components. Specific attention was paid to instances of ICIC as well as damage at the implant-bone interface.

Methods: 1873 retrieved TKA components were collected from 2002-2013 as part of a multi-center, IRB-approved retrieval program. 52 CoCr femoral condyles were identified as long term TKA (Average: 17.9±2.8y). These components were mainly revised for loosening, PE wear and instability. 40/52 of the components were primary surgeries. Components were examined using optical microscopy to confirm the presence of 5 damage mechanisms (PE failure, MACC corrosion of modular tapers, corrosion damage between cement and backside, third-body wear, and ICIC). Third-body wear was evaluated using a semi-quantitative scoring method where a score of 1 had minimal damage and a score of 4 had severe damage. Polyethylene components were semi-quantitatively scored using the Hood method [3] and CoCr components were scored similarly to quantify metal wear. The total area damaged by ICIC was quantified using photogrammetry. Images were taken using a digital SLR with a calibrated ruler in the
same focal plane. Using known pixel dimensions, the ICIC damaged area was calculated. Cement was removed from the backside of components by boiling in Toluene, an organic solvent. Components were boiled for 2 to 3 hours dependent on the amount and thickness of cement. This was followed by ultrasonication for 10 minutes and drying overnight. Investigation was conducted by visual inspection and confirmed with microscopy.

**Results:** Surface damage indicative of corrosion and/or CoCr debris release was identified in 92% (n=48) of the components. Third-body wear was the most prevalent damage mechanism identified in 77% (n=40/52; Figure 1) of these components. ICIC was identified in 38% (n=20/52, Figure 2) of the components. The polyethylene damage scores were predominantly a score of 4 out of a maximum score of 4 (89%). The corresponding femoral components had moderate to severe damage scores, with 39% with a score of 2, 37% scoring 3 and 22% scoring 4 out of a maximum score of 4. The total ICIC damaged area was an average of 0.11 ± 0.12 mm² (Range: 0.01-0.46mm²). 44/52 components were cemented (n=85%). 27 of these components did not have a backside coating and were chosen for preliminary investigation. Fretting and corrosion at the backside and cement interface was observed in 11 out of 27 components (41%). This was observed as either discoloration, staining, fretting scars or blackened debris, found on the backside of the bearing surface, posterior and anterior of the implant.

![Bar chart](image)

**Figure 1:** Third body wear, ICIC damage and cement interface damage were the most prevalent of the 5 observed damage modes.
Discussion: In this study, we sought to identify mechanisms that could lead to the release of CoCr debris/ions in TKA. Five different mechanisms of potential metal release were observed. The most prevalent were third-body wear and ICIC damage. However, the clinical implications remain unclear for several mechanisms because none of the devices were revised due to adverse local tissue reactions or biologic reactions to CoCr. Although we documented the prevalence of each damage mechanism, the quantity of metal removal was not investigated, warranting future studies.

Significance: This study sought to identify the prominent modes of metal and ion release within long-term (in-vivo > 15 years) implanted CoCr femoral components.

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