

Inflammatory Markers Involved In Failed Total Joint Replacements

FRIDAY
20 OCTOBER 2017

2.30 PM TO 3.30 PM

SEMINAR ROOM
LEVEL 2 MD 10
DEPT OF ANATOMY

MS RITA HAMEISTER

Graduate Student
Department of Anatomy
Yong Loo Lin School of Medicine
National University of Singapore

Aseptic loosening is a major cause of joint replacement failure and therefore of clinical and economical relevance. It is commonly thought that wear products such as metal ions and particles which originate from failed implants induce inflammation that finally results in bone loss and implant loosening. Cobalt-alloy wear debris has been reported to induce hypoxia-like conditions *in vitro*. Hypoxia has a critical role in many diseases and is known to be interdependent with inflammation but this has not been studied extensively in the context of joint replacement surgery. Thus, this project aims to elucidate on some of the cellular and molecular events in relation to hypoxia and inflammation in failed joint replacements. Using molecular and histological techniques, hypoxia-associated factors, the macrophage response and the vascular ultrastructure were investigated in patients' hip capsular tissue. Taken together, our results suggest the presence of hypoxia and inflammation. Macrophages were confirmed to release hypoxia-inducible factor-1 α , vascular endothelial growth factor and nitric oxide. A cell-line based approach will be used to study the cell viability and function of macrophage- and osteoblast-like cells upon exposure to metal wear or an environment of deprived oxygenation. Ultrastructural changes in both cell types were observed after exposure to metal ions. Preliminary results further suggest a metal ion-induced hypoxia-like response in osteoblasts.