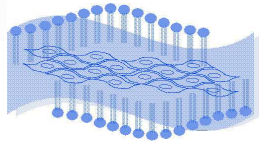




## Center for Biologically Inspired Materials and Material Systems (CBIMMS)



## Center for Biomolecular and Tissue Engineering (CBTE)

### SEMINAR

**Patrick S. Tresco, PhD**

Associate Professor of Bioengineering and Director of the W. M. Keck Center for Tissue Engineering, University of Utah

*“Mining the Borderland between Biomaterials and the Central Nervous System”*

My interests lie in understanding how materials can be used to restore function in damaged nervous tissue. Toward this end, we have examined fundamental behaviors of primary neurons, glia, and various stem cells following contact with well-characterized biomaterials that differ in their physico-chemical properties. Our results indicate that material surface chemistry, microtopography, surface curvature, and the manner in which ligands are presented have profound effects on primary neural cell behavior including changing cell attachment, morphology, cytoskeletal organization, matrix arrangement, and neurite outgrowth. In addition, several in vitro studies indicate that nanotopographic cues can transform adjacent neurite outgrowth inhibiting or reactive cell types into permissive substrates that promote directional outgrowth of regenerating neurons. Recently, while studying biocompatibility of chronic indwelling silicon microelectrode arrays we uncovered a possible explanation for why some classes of CNS devices have not yet reached the clinic. We observed persistent ED-1 immunoreactivity around silicon microelectrode arrays implanted in the adult rat brain and observed significant reductions in nerve fiber density and cell bodies in the tissue immediately surrounding the implants. Persistent ED-1 upregulation and neurodegeneration was not observed in microelectrode stab controls indicating that the chronic inflammatory phenotype and neuronal loss was not caused by the initial mechanical trauma, but was associated with the foreign body response. Our observations share features of diseases having a neuroinflammatory mediated neurotoxic component. In addition, we found that explanted electrodes were covered with ED-1/MAC-1 immunoreactive cells and released measurable quantities of MCP-1 and TNF- $\alpha$  in vitro. Together our findings suggest a mechanism for chronic recording failure that involves neuronal cell loss, which we speculate, is caused by chronic inflammation at the microelectrode brain tissue interface. We, further speculate that persistent macrophage activation at the biomaterial brain tissue interface may explain why ligand mediated bridging strategies have been difficult to translate in vivo.

**Thursday, March 8 – 203 Teer Building – 3:05–5:00 PM**