BIOMEDICAL ENGINEERING
COLLEGE OF ENGINEERING AND APPLIED SCIENCES

RESEARCH OPPORTUNITIES FOR UNDERGRADUATE students

APPLICATION DEADLINE: April 3, 2026

PROJECT TITLE: Cell-Specific Mitochondrial Dysfunction and Intercellular Transfer Following Traumatic Brain Injury

Physical Requirement :
No requirements

Project's Technical Skills Requirement :
No requirements

Project's Available Positions : 1

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Project Description

Introduction: Traumatic brain injury (TBI) is a major cause of disability and is associated with long-term neurological complications, including neurodegenerative diseases such as Alzheimer’s disease and chronic traumatic encephalopathy. TBI triggers a cascade of molecular and metabolic events that are amplified by interactions between different brain cell types. While the initial injury is significant, secondary processes such as mitochondrial dysfunction, metabolic alterations, neuroinflammation, and protein aggregation largely determine long-term outcomes. Mitochondria are key regulators of cellular function and are implicated in injury-induced neurodegeneration. Following TBI, mitochondrial bioenergetic and metabolic dysfunction is associated with neuroinflammation and disease progression. In addition to intracellular changes, mitochondria can be transferred between cells, altering the function of recipient cells. However, the cell-specific dynamics, timing, and mechanisms of mitochondrial involvement in injury progression remain poorly understood.
Project Description: This project aims to determine how traumatic brain injury induces cell-specific mitochondrial dysfunction and how intra- and extracellular mitochondria contribute to neurodegeneration. We hypothesize that injury-driven dysregulation of mitochondrial bioenergetic and metabolic

functions promotes neuroinflammation, neuronal damage, and disease progression. To test this, we will use a human three-dimensional triculture brain model composed of neurons, astrocytes, and microglia with fluorescently labeled mitochondria. Aim 1 will define injury-induced intracellular and extracellular mitochondrial alterations. We will determine the temporal profile of mitochondrial dysfunction, assess cell-specific bioenergetic and metabolic changes, quantify intercellular mitochondrial transfer, and evaluate the release and neurodegenerative potential of extracellular mitochondria. Aim 2 will investigate the molecular mechanisms of mitochondrial reuptake. We will determine whether mitochondrial internalization is mediated through integrin ?1 and annexin 2 pathways and assess how modulation of these pathways affects mitochondrial transfer and neurodegeneration progression.
The student involved in this project will participate in the development and analysis of the human three-dimensional brain model, including cell culture, controlled injury experiments, and molecular and imaging-based assessments of mitochondrial function and neuronal health. The student will also contribute to data analysis, integrating cellular, molecular, and metabolic measurements to understand injury progression. A basic understanding of cell culture and molecular biology techniques is recommended.

The expected outcome of this work is to define cell-specific mitochondrial mechanisms that drive secondary neurodegeneration following traumatic brain injury. This work will provide mechanistic insight into how mitochondrial dysfunction and intercellular transfer contribute to disease progression and may identify targets for therapeutic intervention.