

Ph.D. Dissertation Defense Announcement
Mechanical and Aerospace Engineering Department
University of Texas at Arlington

**MULTISCALE INJURY BIOMECHANICS AND NEURONAL
COMMUNICATION IN BRAIN**

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Abstract

Traumatic brain injury (TBI) and similar acute physical insults induce malfunction in neuronal communication at the cellular level through coupled alterations in tissue mechanics, cellular microstructure, and electrophysiology. This dissertation develops a multiscale framework to examine how neuronal morphology governs structural connectivity, local field responses, and subsequent electrical communication under rate-dependent mechanical loading and pulsed electromagnetic exposure. The computational growth model encompasses cell bodies, neurites, and mature synaptic contacts and investigates how cellular growth parameters alter developmental trajectories, process-level responses, and the final topology of neuronal networks. This distinction establishes a robust structural baseline for subsequent injury simulations. The obtained geometry is then subjected to two loading pathways. In the rate-dependent mechanical loading pathway, finite element (FE) analyses resolve local deformation and reconstruct neurite strain and cell-body distortion from nodal displacement to map them into the electrophysiological formulation. Strain-dependent geometry and membrane-property changes are coupled with action potential (AP) signal propagation and synaptic recruitment, enabling communication deficits to be associated with localized injury rather than bulk deformation alone. In the pulsed high-power microwave (pHPM) exposure pathway, local electromagnetic fields are translated to volumetric absorption and specific absorption rate (SAR), transferred to transient thermal FE analyses, and coupled to temperature-sensitive cell-membrane kinetics and synaptic efficacy. These mechanisms produce non-uniform alterations in neuronal firing, signal amplitude, recruitment, and transmission. The major contribution is a reusable structure–field–function framework in which neuronal geometry connects development, FE field quantities, and network electrophysiology. It enables mechanistic comparison of distinct exposure modalities, identifying how similar macroscopic impact conditions can yield different communication outcomes due to cellular heterogeneity and local network topology.