EXOSOMES AS MEDIATORS OF NEUROINFLAMMATION AND NEUROTOXICITY: TRANSLATIONAL IMPLICATIONS FOR BIOMARKER DISCOVERY IN ENVIRONMENTALLY LINKED PARKINSONISM

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Abstract:

Emerging evidence indicates that a prion-like, cell-to-cell transfer of misfolded proteins contributes to the spreading of α-synuclein aggregates and its neurotoxicity in Parkinson’s disease, but the cellular mechanisms underlying disease propagation with respect to environmental neurotoxic chemical exposures are not well understood. This presentation will demonstrate that exposure to neurotoxic manganese in cell and animal models induces the exosomal release of misfolded α-synuclein and propagates cell-to-cell, which activates a sustained neuroinflammatory response that subsequently contributes to dopaminergic neurotoxicity. This presentation will also highlight the translational relevance of our findings using a highly sensitive high-throughput RT-QulC diagnostic assay to detect α-synuclein aggregates both in welders occupationally exposed to manganese and in Parkinson’s disease patients.

Host: Dr. Jason Cannon