JOYCE MASSEY TBI INNOVATION FUND
2015 TBI Funded Research Projects
Funding was awarded based on the potential to impact the way TBI is diagnosed and treated during the initial “golden hours” of care.

IMPROVING TBI-INDUCED SYNAPTIC CHANGES

THE TEAM
Leslie Satin, PhD
Pharmacology
Seth Wescott, PhD
Pharmacology

THE RESEARCH

HISTORICAL STUDIES
TREAT USING GLUTAMATE RECEPTOR ANTAGONISTS
UNSUCCESSFUL CLINICAL TRIALS
Decrease glutamate excitotoxicity
Neurons damaged or killed by excessive stimulation by neurotransmitter glutamate

TRAUMATIC BRAIN INJURY
TRI.setParameter("text", "Discovering TBI-induced changes in neurotransmission and reversing these changes with novel therapeutic treatments")

OUR INNOATIVE APPROACH
DYSFUNCTIONAL GLUTAMATE RECEPTORS
Mediate synaptic transmission
Contrary to historical studies

DEPRESSES NEUROTTRANSMISSION
SELECTIVELY REVERSE WITH NOVEL THERAPIES
1. Cyclosporine A
2. Memantine
3. Branched Chain Amino Acids

COMPETITIVE ADVANTAGE

ONE-OF-A-KIND IN VITRO MODEL
Utilizes one of the few TBI models where cellular/molecular studies of synaptic function and neurotransmitter receptor trafficking can be done.

NOVEL TBI THERAPY
There are currently no effective therapies for TBI patients. Cyclosporine A, memantine and branched chain amino acids could be new therapies for TBI.

NOVEL NEUROTTRANSMISSION THEORY
Contrary to previous studies, the team’s recent data reveal that neurotransmission is depressed after TBI, not overstimulated. This places U-M in an advantageous position to develop new TBI therapies.

COMMERCIALIZATION ROADMAP

INVESTIGATIONAL NEW DRUG (IND) regulatory pathway
LICENSE TECHNOLOGY/ THERAPY
POTENTIAL PARTNERS
Drug companies

PROJECT MILESTONES

Identify the molecular changes that underlie the early & persistent suppression of glutamate receptor synaptic transmission following TBI

Use patch clamp electrophysiology, cell imaging techniques to determine changes in glutamate receptors after TBI

Test novel therapeutic agents post-injury to determine if they selectively reverse TBI-induced changes in glutamate receptors