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A BOLD STRATEGY TO

STOP ALZHEIMER'S EARLIER

FOCUS: Fighting dementia by improving neuronal energy homeostasis.

Approximately 44 million people worldwide live with Alzheimer's disease (AD) or a related form of dementia, yet there are no effective treatments. It is believed that AD is caused by the accumulation of two proteins in the brain, amyloid beta and pTau.

"Much focus has been on preventing the buildup of amyloid beta and pTau," Dr. Trushina says. "Yet in the brains of people with AD, neuronal dysfunction appears long before amyloid and pTau become problematic, and even before cognitive symptoms become evident. Neurons in the brain are critically dependent on mitochondria-produced energy; problems

with energy homeostasis lead to neuronal dysfunction early in AD."

Dr. Trushina and her team have developed a small molecule therapeutic targeted at mitochondria in order to improve energy production and utilization in neurons.

"Scientists have traditionally avoided approaches involving mitochondria," Dr. Trushina says. "The metabolic system is so complex, the concern has always been that anything affecting it could have unpredictable consequences."


Dr. Trushina's lab has developed compounds that, in mouse models of

AD, improve glucose uptake and utilization, dendritic spine maturation, long-term potentiation, synaptic activity, cognitive function, and reduce amyloid deposition and pTau accumulation, oxidative stress and inflammation, ultimately blocking neurodegeneration.

In collaboration with the Harrington Discovery Institute, Dr. Trushina hopes to identify safe and efficacious preclinical and development small molecule candidates suitable for a Phase I clinical trial.

"After crawling for 10 years, Harrington is helping move this project at the speed of light!"

Harrington Discovery Institute

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