## SUMMARY OF THE 2024 NAVIGATOR RECIPIENTS

In all forms of neurodegenerative diseases or spinal cord injury, a damaging signal can travel down the long axons (the “highways”) from the synapse to the cell body (the “signal interpreter”), leading to the breakdown of the neuron. This signal is mediated by a protein called Dual leucine zipper kinase (DLK). As a result, inhibitors of DLK have been developed to protect neurons after spinal cord injury and are now being tested in the context of neurodegenerative diseases such as amyotrophic lateral sclerosis (ALS), Alzheimer’s disease, and Parkinson’s disease (AD and PD, respectively). DLK drives these harmful signals through a pathway also known to be activated in Huntington disease (HD). Remarkably, no studies have yet investigated whether inhibiting DLK could be beneficial in HD. The Martin, Sanders, and Alpaugh Labs will address this by exploring the role of DLK in HD using genetic approaches to reduce DLK in HD mouse models and HD patient-derived neurons.

375 Hagey Blvd, Suite #114, Waterloo, ON N2L 6R5 • Tel: 519-749-7063 • Fax: 519-749-8965
info@huntingtonsociety.ca • www.huntingtonsociety.ca • Charitable Registration Number: 11896 5516 RR0001