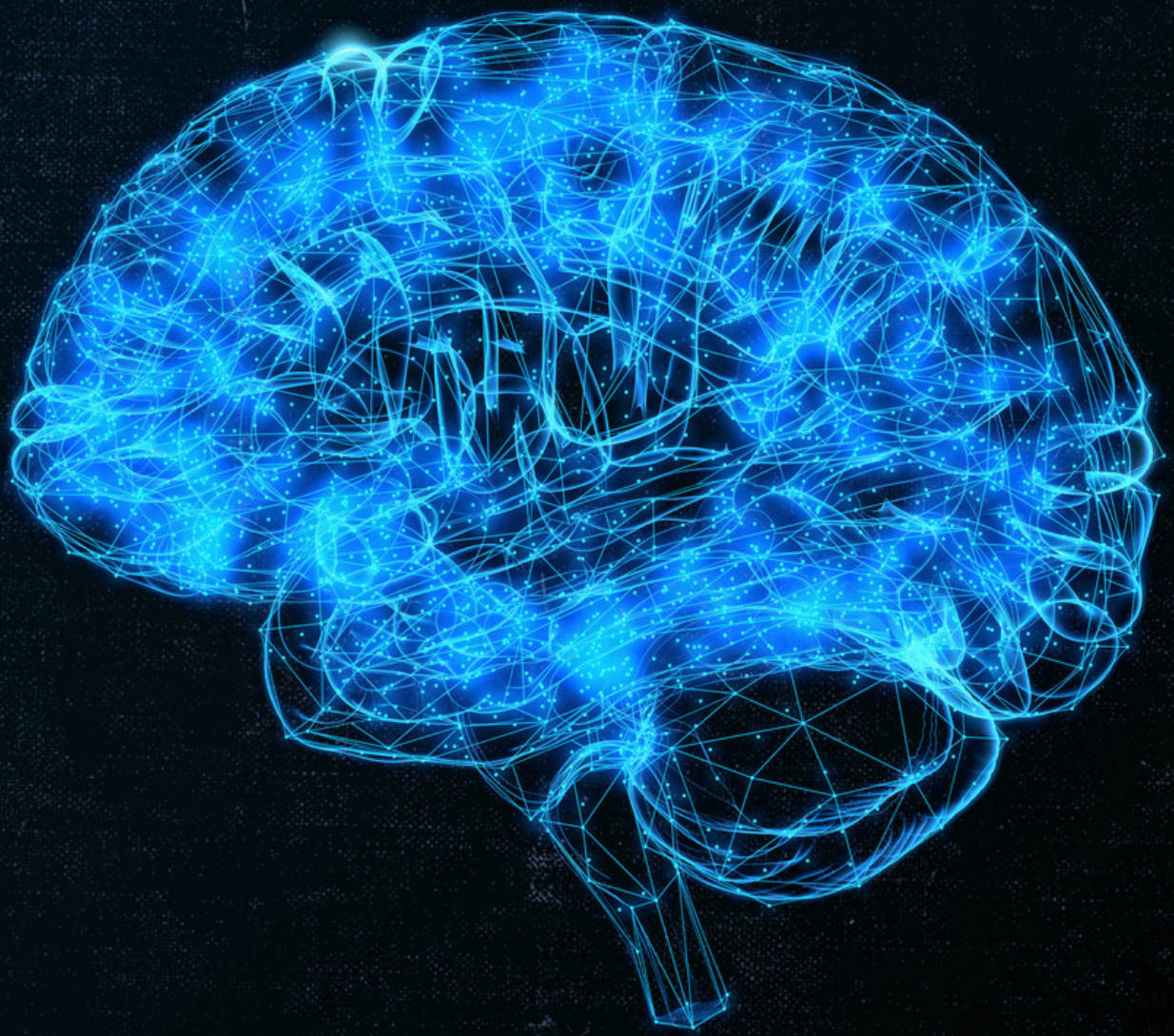
Huntington Society of Canada
HUNTINGTON
Société Huntington du Canada

HELIX

PATHS TO HD BREAKTHROUGHS



The 2025 Huntington Society of Canada Research Report

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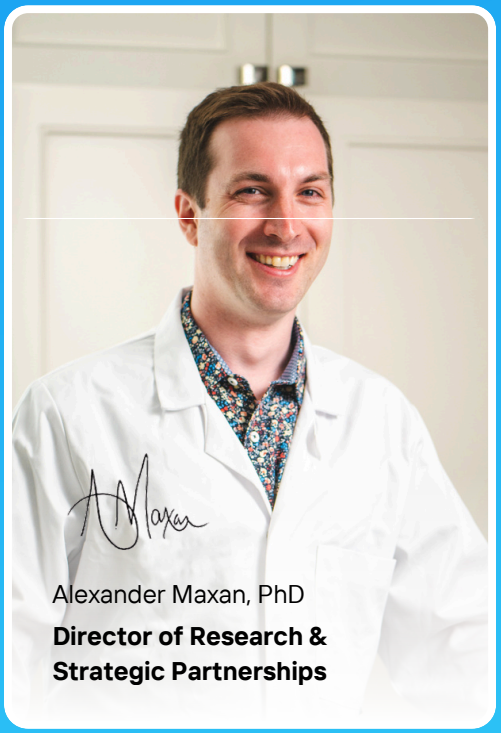


Alexander Maxan, PhD

Director of Research & Strategic Partnerships

A Message to the Community

As I sit down to share this report with you, my heart is still buzzing from the energy of our recent National Conference. What an unforgettable gathering it was with participants coming together in person to share stories, laughter, and that unbreakable sense of hope that defines our community. Hearing directly from our Navigator Award recipients was nothing short of inspiring; their curiosity in pushing research forward reminded me why we do this work every single day. And the updates from HDBuzz? They brought the science to life in a way that made complex breakthroughs feel within reach, sparking conversations that lingered long after the sessions ended.

These moments of connection mean the world to our Huntington disease (HD) community, especially as we navigate the latest news from the clinical trial landscape. I know many of you have been following recent developments closely. The encouraging signals emerging from ongoing studies hint at real, tangible progress toward slowing this disease. At the same time, our need for transparency in reporting on relevant and meaningful data is crystal clear. While challenges and uncertainties remain part of the journey, every step forward builds on the foundation of perseverance demonstrated by the HD community. For families waiting, donors who give so generously, and every volunteer who lends their time, these updates are much more than just data points.

Your unwavering support, whether through donations, a raised voice in advocacy, or simply showing up for one another, fuels every grant, trial, and breakthrough. From the bottom of my heart, thank you for being part of this resilient community. Change is happening thanks to you.

Thank you for being part of this journey, and here's to an even better 2026.

NAVIGATOR Research Program

The NAVIGATOR Program has been providing funding to support HD researchers since 2003. We are grateful to the River Philip Foundation and Brain Canada for their generous support. The River Philip Foundation and Brain Canada (through the Canada Brain Research Fund) each committed $600,000 for a total of $1.2 million, translating into **four NAVIGATOR projects per year in 2025 and 2026.** We hope you will continue to follow the Huntington Society of Canada for more research updates and announcements.

2024 RECIPIENTS

Left to right:
Dr. Melanie Alpaugh, Dr. Shaun Sanders, Dr. Dale Martin

Dr. Shaun Sanders (University of Guelph), **Dr. Melanie Alpaugh** (University of Guelph), **Dr. Dale Martin** (University of Waterloo)

Project Title: DLK as a novel therapeutic target to treat Huntington Disease

Project Summary: This proposal seeks to validate the mitogen activated protein kinase kinase kinase (MAP3K) dual leucine-zipper kinase (DLK; MAP3K12) as a therapeutic target in HD. DLK is an upstream regulator of the MAPK cJUN N-terminal kinases (JNK2 and 3) that initiates pro-degenerative signaling in neurons in response to injury and neuronal stress. DLK inhibition is protective in neuronal injury models and promotes neuronal connectivity. Conversely, several forms of neurodegeneration, both chronic and acute, are initiated by DLK, including ALS, Alzheimer's disease, Parkinson's, and injury. These findings have sparked great interest in targeting DLK-dependent signaling therapeutically as a neuroprotective strategy, and direct inhibitors are actively being developed. Thus, inhibition of DLK likely prevents neuronal degeneration in HD as well, but this has never been explored. We will assess the therapeutic potential of targeting DLK in HD mouse and human neurons.

NAVIGATOR Research Program

2025 RECIPIENTS

Dr. Blair Leavitt (University of British Columbia), **Dr. Lynn Raymond** (University of British Columbia), **Dr. Aurélie de Rus Jacquet** & **Dr. Francesca Cicchetti** (Université Laval), **Dr. Alex Parker** (CHUM)

Dr. Blair Leavitt

University of British Columbia

Project title: Targeted DNA methylation editing to ameliorate Huntington's disease phenotypes in human induced pluripotent stem cells

Project summary: Huntington's disease (HD) is a brain disorder caused by a single, specific mutation in the DNA sequence of the huntingtin gene (HTT). This mutation produces an abnormal, toxic mutant huntingtin protein (mHTT) that causes a progressive loss of cells in the brain and leads to the development of HD symptoms. Many factors, including naturally-occurring chemical modifications of DNA, impact levels of mHTT. In turn, mHTT levels affect the onset, progression, and severity of HD patient symptoms. One type of chemical DNA modification, called DNA methylation (DNAm), alters the activity of HTT in brain cells and may affect the loss of brain cells that occurs in HD. In this proposal, we will alter DNAm at HTT to selectively reduce the quantity of toxic mHTT protein produced from HTT, and will evaluate how lowering toxic mHTT using this approach improves the health of cells carrying the HD mutation. These studies will use specialized human cells highly similar to the brain cells affected in HD, enabling accurate assessment of how altering HTT DNAm improves cellular HD symptoms. This work will increase our understanding of the impact of DNAm levels on toxic mHTT abundance and measure how altering DNAm levels at HTT improves cellular HD symptoms, and will evaluate the utility of altering DNAm as a novel approach for therapeutic mHTT lowering in HD.

Dr. Lynn Raymond

University of British Columbia

Project title: Neurofeedback-driven restoration of cortical connectivity in HD

Project Summary: HD is traditionally viewed as a disorder of the deep brain (the striatum), but mounting evidence shows that even before clear symptoms appear, HD alters how different parts of the brain communicate, particularly in motor regions. In HD mouse models, neurons in cortical motor areas not only lose connection with other regions but also activate in a disorganized manner, impairing movement control. In our study, we will first confirm these circuit changes, and investigate whether and how they correlate with performance in a fine motor control task (pulling a lever for water reward). Then we will test two ways to help them "rewire" the affected motor area: one teaches mice to boost the correct brain signals by giving them water rewards whenever they succeed (neurofeedback training), and the other uses direct stimulation to trigger those same cells on a fixed schedule (optogenetic stimulation). By comparing these approaches, we hope to restore coordinated activity in the affected motor region in the cortex and improve the animal's performance on the motor task. If successful, this could lead to simple, noninvasive brain-training programs for people with early-stage HD.

Since 2003, HSC has funded **37 research awards** to **24 investigators** at **16 institutions** across **7 provinces**, providing **over $7.5 million** in HD research funding.

37	24	16	7	$7.5+
Research Awards	Investigators	Institutions	Provinces	Million

NAVIGATOR Research Program

2025 RECIPIENTS

Dr. Alex Parker

Centre Hospitalier de l'Université de Montréal,

Project title: Stimulation of axonal regeneration and repair in Huntington's disease

Project summary: HD is a neurodegenerative disorder marked by progressive motor dysfunction, cognitive decline, and early death. Despite advances in understanding its genetic basis, effective therapies are limited. Current efforts mainly focus on reducing mutant huntingtin (HTT) levels, but axonal degeneration, a key factor in neurodegeneration, remains inadequately addressed. Our project aims to validate SAC2/INPP5F as a novel therapeutic target for HD. SAC2, a lipid phosphatase involved in regulating endosomal dynamics and the PTEN/PI3K/mTOR pathway, shows promise in ALS research for maintaining axonal integrity. In ALS models, including C. elegans and zebrafish, SAC2 knockdown has been shown to provide protection against axonal degeneration and motor dysfunction. We have observed these protective effects in our ALS models. Additionally, other research teams have found that SAC2 knockout (KO) induces highly potent axonal regeneration phenotypes in human cellular axotomy models. Preliminary data from our research using htt-1 KO and PolyQ Huntington C. elegans models also indicate that SAC2 knockdown reduces axonal degeneration and enhances neuronal integrity. Our approach aims to stimulate axonal regeneration and repair mechanisms in models of HD, translating our promising ALS findings to HD and paving the way for new therapeutic strategies. By targeting SAC2, we aim to enhance existing gene therapies designed to reduce mutant HTT, offering a new strategy for preserving neuronal function in HD. This proposal seeks funding to validate SAC2 as a therapeutic target in HD and to develop small-molecule inhibitors and antisense oligonucleotides (ASOs) as potential therapeutic candidates

The number of applications for NAVIGATOR funding has increased 111% since 2017

Dr. Aurélie de Rus Jacquet & Dr. Francesca Cicchetti

Université Laval

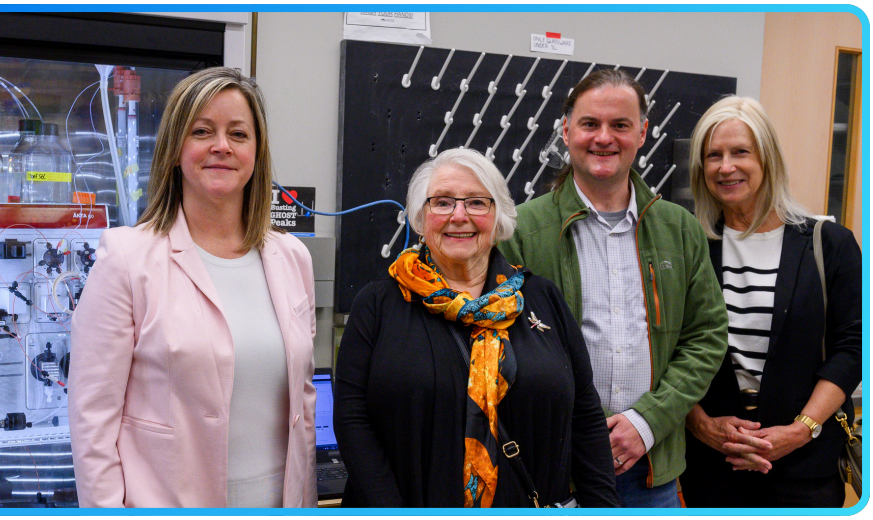
Project title: Brain rejuvenation: an innovative approach to Huntington's disease therapy

Project summary: HD is a neurodegenerative disorder caused by a single defective gene known as huntingtin (HTT). This disease is characterized by uncontrolled movements, a decline of thinking ability as well as psychiatric problems. The life span of affected individuals is usually 10 to 30 years after the appearance of symptoms. In this disease, an abnormal form of a protein referred to as mutant Huntingtin (mHtt) accumulates in brain cells disrupting their normal function and eventually leading to their death. Recently, the Cicchetti group utilized a model system called "parabiosis" in which mice are surgically sutured to create the joint circulatory system and has provided exciting new evidence showing reduced pathology in the diseased (HD) mice that were surgically paired to normal mice. This study demonstrated the involvement of circulating factors from healthy blood in diluting/ameliorating disease pathology. Building on this new data, we designed a project which aims to identify the beneficial elements contained in healthy blood and to understand how they ameliorate brain health. To achieve this goal, we will isolate human plasma proteins capable of migrating from the blood into the brain because they will likely be the most neuroprotective factors. We will then evaluate if these proteins improve the survival and function of neurons and non-neuronal cells in a dish but also in a mouse model of HD. This project will help us identify circulating factors in healthy plasma that may prevent the death of brain cells and that therefore could be useful in treating various disease features. Additionally, outcomes of this study may encourage the transfusion of healthy human plasma as a potential therapeutic strategy to treat HD.

Since 2003, the NAVIGATOR program has awarded over $4.5 million to fund Huntington disease research

Meet Dr. Patrick O'Donoghue:
Pioneering Hope for Huntington Disease

Dr. Patrick O'Donoghue, PhD, is the first-ever endowed HSC Research Chair at Western University's Schulich School of Medicine & Dentistry. **Appointed in November 2024 for a five-year term, Dr. O'Donoghue is leading groundbreaking work to tackle HD.** With a strong background in biophysics and chemistry from the University of Illinois and Yale University, he joined Western in 2013 and has since become a tenured associate professor. His role as Research Chair allows him to focus on innovative therapies that could change lives for HD families across North America.



A Fresh Approach to Targeting HD

HD is caused by a faulty gene that leads to harmful build-ups (called aggregates) of a protein called huntingtin in the brain. These clumps damage brain cells over time, causing symptoms that worsen progressively. Dr. O'Donoghue's lab is developing a new type of treatment using tiny molecules called transfer RNAs (tRNAs). Think of tRNAs as "editors" in the body's protein-making process; they can tweak how proteins are built. His team has created special tRNAs that "rewrite" parts of the faulty huntingtin protein, reducing its levels and stopping those damaging clumps from forming. Early tests in cell models (like those mimicking HD) show these tRNA "medicines" can cut harmful protein build-ups by up to 50% without hurting healthy cells. This approach isn't just for HD, it could help with other genetic diseases like certain cancers or muscle disorders, but Dr. O'Donoghue is laser-focused on accelerating HD treatments.

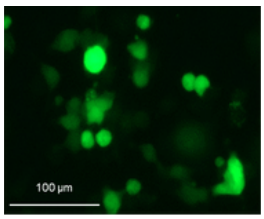
Milestones and Momentum in Just One Year

Since starting as Research Chair, Dr. O'Donoghue has hit the ground running, thanks to HSC funding.

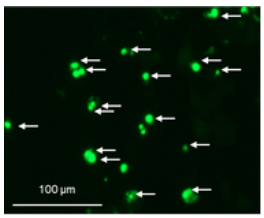
Highlights include:

- ***Securing Major Grants:*** **Over $1.9 million** from sources like the Canadian Institutes of Health Research (CIHR) and Natural Sciences and Engineering Research Council (NSERC) to expand HD studies. The HSC Chair position helped unlock these larger awards.

- ***Key Publications:*** **Four new research papers,** including a standout one in Molecular Therapy: Nucleic Acids (2025) showing how tRNA therapies suppress huntingtin clumps.



Normal Huntingtin

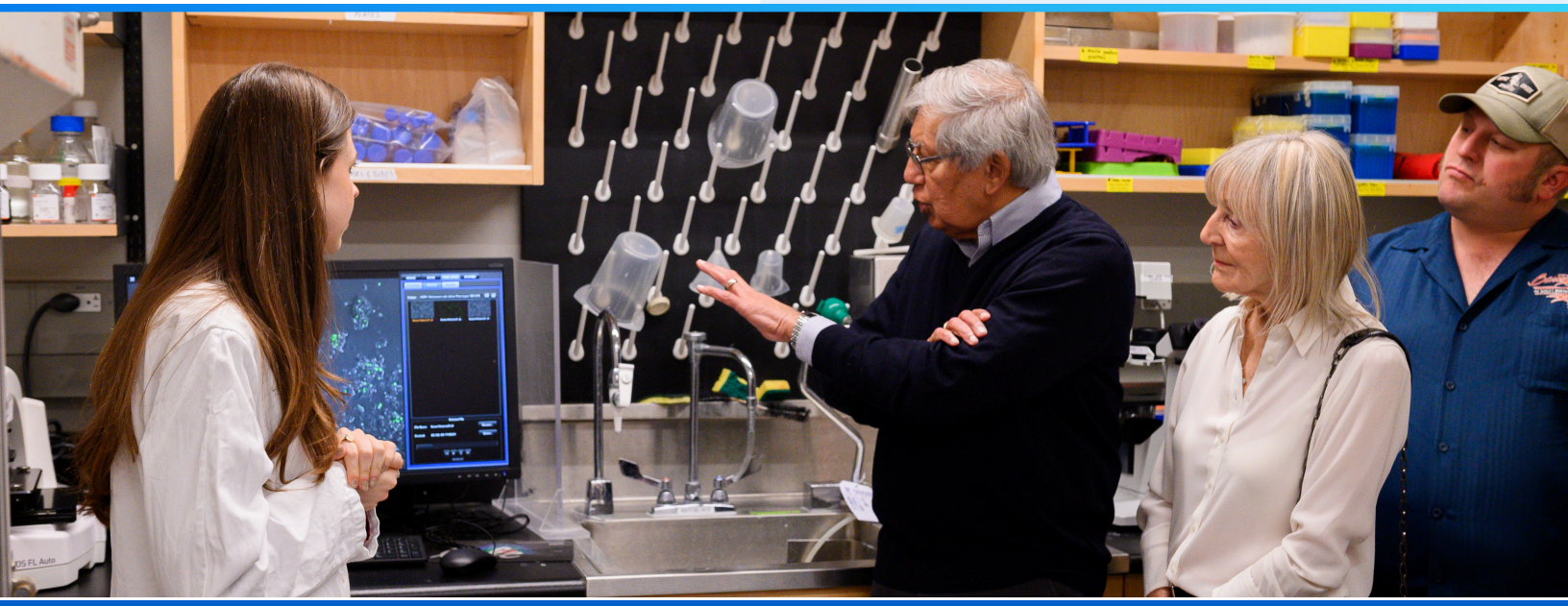


Huntingtin aggregates

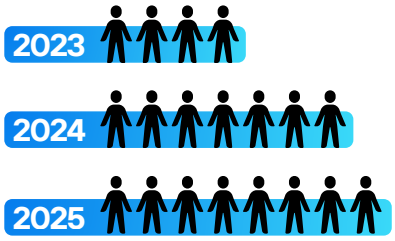
- ***Growing the Team:*** **From four PhD students to six, plus a postdoctoral fellow, with plans for two more recruits.** This builds Western's training mission, inspiring the next generation of scientists. New team members like MSc candidates Kira Dempsey and Milan Mammen, and postdoc Dr. Sarah Schultz, are optimizing tRNA delivery to brain cells.

- ***Community Connections:*** Dr. O'Donoghue has shared his work with us, **presenting to the HSC Board (March 2025), the London chapter (May 2025), and hosting a lively site visit in April.** These outreach efforts bridge lab discoveries with real hope for patients and families.

- ***Looking Ahead:*** **This funding from HSC has doubled Dr. O'Donoghue's research time by easing his teaching load, allowing faster progress in the lab.** His work aligns with HSC's goal of preventing, detecting, and treating HD, with potential for local, national, and global impact. As he notes, "HSC's leadership in raising awareness and resources is critical. I'm deeply grateful for donors who make this possible."



Undergraduate Student Summer Fellowship



In 2023, we launched our inaugural Undergraduate Student Summer Fellowship, which was met with encouraging interest and support. Thanks to Brain Canada for matching our funding this year, we were able to fund **eight fellowships that enabled young up-and-coming researchers to spend the summer working in HD research labs, bringing our total fellowship recipients to 19.**

HERE ARE THE EIGHT OUTSTANDING RECIPIENTS FOR 2025:

Brianna Adams | Memorial University of Newfoundland

Supervisor: Matthew Parsons

Project title: Synaptic activity as a driver of DNA damage and somatic expansion in Huntington's disease

Alexandra Bérubé Ebacher | Université Laval

Supervisor: Francesca Cicchetti

Project title: Shedding light on the huntingtin protein

Isabella Bu | Western University

Supervisor: Peter Stathopulos

Project title: Investigating S-nitrosylation of MCUB in mitochondrial calcium dysregulation and Huntington's disease

Maya Klepfish | McMaster University

Supervisor: Ray Truant

Project title: The use of AI-generated peptides with the CK2 kinase catalytic domain to restore huntingtin N17 phosphorylation

Yusra Kureshi | University of Waterloo

Supervisor: Dale Martin

Project title: Optimizing nanodiscs to improve antisense oligonucleotide delivery in the treatment of Huntington disease.

Christiana Lammers | University of Guelph

Supervisor: Melanie Alpaugh

Project title: Evaluating DLK activity in brain samples from Huntington's disease patients

Shannon Pulido | Memorial University of Newfoundland

Supervisor: Matthew Parsons

Project title: Synaptic activity as a driver of DNA damage and somatic expansion in Huntington's disease

Fatma Slim | Université Laval

Supervisor: Aurélie de Rus Jacquet

Project title: Vascular innervation and blood-brain barrier dysfunction in Huntington's disease using human iPSC-derived vascularized brain organoids

Clinical Fellowship

At the Huntington Society of Canada (HSC), we want to ensure that each Canadian with HD receives appropriate medical expertise, treatment options, support services, and any other help needed. Supporting the training of the next generation of HD specialists in neurology and neuropsychiatry is an important part of our plan to ensure that these critical elements in the day-to-day management of HD are accessible and available. **Here are the three clinical fellowship recipients supported by HSC in 2025:**

NEUROLOGY

Dr. Falen Fernandes
University of Alberta
Supervisor: Dr. Justyna Sarna

NEUROLOGY

Dr. William Leduc
University of Alberta
Supervisor: Dr. Justyna Sarna

NEUROPSYCHIATRY

Dr. Caroline Dance
University of British Columbia
Supervisor: Dr. Andrew Howard

Updates on Clinical Trials

Recent clinical trial updates bring renewed hope to the HD community, showcasing progress in both experimental disease-modifying and symptomatic approaches. **Huntingtin-lowering therapies, designed to reduce the harmful protein driving HD progression, are showing promising early results, with ongoing studies refining their potential to slow or halt the disease.** Meanwhile, symptomatic treatments are advancing to better manage motor, cognitive, and psychiatric challenges, aiming to enhance quality of life for those living with HD. While no disease-modifying therapies have yet been approved for HD, many of the different approaches summarized on the following pages offer hope for the future. Here are updates from clinical trials conducted over the past year:

SYMPTOMATIC THERAPIES

Sage Therapeutics completed the Phase 2 DIMENSION trial evaluating dalzanemdor, an oral NMDA receptor positive allosteric modulator, in Huntington disease. **Dalzanemdor**, also known as **SAGE-718**, is an oral drug that acts as an NMDA receptor positive allosteric modulator. The drug was designed to enhance synaptic activity and cognitive function by modulating NMDA receptor signaling. The DIMENSION trial assessed changes in cognitive and functional performance over 12 weeks as its primary and secondary endpoints, respectively. The results indicated that dalzanemdor did not improve outcomes compared to placebo, despite earlier Phase 2 SURVEYOR results suggesting potential cognitive benefits. Following these findings, Sage Therapeutics discontinued further development of SAGE-718 and closed the long-term open-label Phase 3 PURVIEW study.

Prilenia Therapeutics conducted the Phase 3 PROOF-HD trial to evaluate **pridopidine** for Huntington disease. This oral drug acts as a selective sigma-1 receptor agonist that supports neuronal survival and signaling. Several previous trials have assessed changes in total functional capacity (TFC) and the composite Unified Huntington's Disease Rating Scale (cUHDRS) as primary and secondary endpoints, respectively. No study of pridopidine has met the endpoints; however, Prilenia believes exploratory analysis showed potential benefits for participants not taking antidopaminergic medication. Although the European Marketing Authorization Application was recently rejected, Prilenia is continuing to explore a path forward for pridopidine.

SOM3355 is an oral small molecule with mild β1-adrenergic blocking and dual VMAT1/2 inhibitory activity, developed for treatment of chorea in HD. In a Phase 2b randomized controlled trial of 140 HD patients (placebo, 400 mg, 600 mg daily), the 600 mg group achieved significant improvement in chorea among patients not receiving antipsychotics. In this subgroup, mean UHDRS-Total Maximal Chorea (TMC) improved compared with placebo. SOM3355 was safe and well tolerated. These findings supported FDA Orphan Drug Designation and a positive EMA CHMP orphan drug opinion in 2025. **SOM Biotech** is preparing a Phase 3 trial to confirm efficacy.

Updates on Clinical Trials

DISEASE-MODIFYING THERAPIES

SPK-10001, originally sponsored by Spark Therapeutics, is now fully integrated into the **Roche** portfolio under the name **RG6662.** It is a gene therapy target to downregulate both mutant and normal HTT proteins, but unlike Roche's other pipeline candidate, tominersen, it is delivered via a one-time infusion procedure straight to the brain. The drug is currently in an ongoing phase 1/2 trial in adults aged 25 to 65, with locations in Ohio and Massachusetts currently recruiting. The first part of the study will involve single-dose escalations until the most optimal dose for safe treatment is found, and the latter part of the study will involve RG6662 or placebo random assignment to participants with a follow up for 5 years. The study aims to be completed by 2035.

Tominersen is an antisense oligonucleotide (ASO) that targets HD by binding to the gene encoding for mutant huntingtin protein, limiting its production and lowering its levels in the brain. It is currently in an ongoing phase 2 clinical trial with the objective to evaluate the safety, efficacy and biomarkers compared to placebo in participants with early manifest and prodromal HD. The route of administration is by 'intrathecal injection,' where a needle is inserted in the lower back, delivering the drug/placebo into the cerebrospinal fluid (CSF) that will deliver the drug to the brain. Trial participants were initially split into three groups to receive one of the following: 60mg or 100mg of tominersen or an equal amount of placebo once every 4 months for a total of 16 months. The trial has since been updated to continue testing only the higher 100 mg dose, following interim data favouring its potential clinical benefit. The main endpoints from the clinical trial include observed side effects, changes in mutant HTT protein levels in the CSF, changes in daily activity function, and changes in brain magnetic resonance imaging results. The study aims to be completed in 2027. Canadian study sites include Edmonton and Montreal.

PTC Therapeutics announced the results of the PIVOT-HD Phase 2a study on **PTC518**, an oral small-molecule splicing modulator that targets huntingtin mRNA (mHTT) to reduce the expression of huntingtin protein (HTT). The study demonstrated dose-dependent lowered HTT levels in blood cells over a 12-week period in early HD patients with no treatment-related adverse effects. Since completion of the Phase 2 trial, **Novartis** has reached a global license and collaboration agreement with PTC Therapeutics. Novartis will assume responsibility for the drug, now called **votoplam**, and its development, manufacturing, and commercialization. Novartis is currently completing the analysis of the Phase 2 study and has mentioned an upcoming Phase 3 trial to gather more data supporting the efficacy and safety of votoplam.

Wave Life Sciences Ltd. announced the results of the phase 1b/2a SELECT-HD trial studying safety, tolerability, pharmacokinetics, and pharmacodynamics of **WVE-003**, an allele-selective antisense oligonucleotide (ASO) for Huntington disease (HD). The treatment included single-administration (30mg, 60mg, and 90mg) and multiple-administration (30mg) doses. After the 24th week, the study demonstrated lowered mHTT in cerebrospinal fluid (CSF). This indicates that the dose of WVE-003 required to achieve effective results was less than originally estimated. Canadian study locations include Edmonton, Ottawa, and Montreal.

AMT-130 is an AAV5-based gene therapy carrying an artificial microRNA targeting HTT mRNA. In a randomized, controlled Phase 1/2 trial of early-HD patients, participants received low-dose AMT-130, high-dose AMT-130, or sham surgery. At 36 months, the high-dose cohort achieved the primary endpoint: their mean composite UHDRS (cUHDRS) score declined –0.38 versus –1.52 in matched natural history controls, corresponding to a 75% slower progression. Total Functional Capacity (TFC) decline was slowed by ~60%. No new therapy-related serious adverse events were reported. **UniQure** initially intended to submit a Biologics License Application to the FDA in early 2026 under the accelerated approval pathway; however, recent feedback from the FDA has indicated that they may not be as open to external control group comparisons used in the trial as they have previously indicated. UniQure will continue to develop AMT-130, but the estimated timeline is now unclear.

DISEASE-MODIFYING THERAPIES

SKY-0515 is an orally available RNA splice modulator that reduces production of huntingtin and PMS1 proteins. In Phase 1, daily oral dosing at 3 mg or 9 mg for 84 days showed dose-dependent biomarker effects. The 9 mg group achieved a mean 62% reduction in plasma mutant huntingtin protein from baseline, compared with 29% in the 3 mg group; placebo showed no change. SKY-0515 effectively penetrated the CNS and was well tolerated at all dose levels. Based on these results, in June 2025, **Skyhawk** launched the FALCON-HD Phase 2/3 trial, a multicenter randomized study in approximately 120 Stage 2–3 HD patients in Australia and New Zealand, testing three oral doses of SKY-0515 versus placebo over at least 12 months.

Alnylam Pharmaceuticals is currently recruiting participants for a phase 1 clinical trial to evaluate **ALN-HTT02** in adult patients with Huntington Disease. ALN-HTT02 is an RNA interference targeting HTT mRNA to reduce the huntingtin protein. It is planned to be systematically delivered to silence the gene. As Alnylam Pharmaceuticals is conducting the first in-human trials to determine the drug's safety, tolerability, pharmacokinetics, and pharmacodynamics, the data is not yet available. Canadian study sites include Edmonton, Montreal, Ottawa and Vancouver.

VO-659 is an allele-selective antisense oligonucleotide designed to bind expanded CAG repeats in mutant HTT mRNA. In a Phase 1/2a trial with intrathecal dosing every four weeks in early manifest HD and mild SCA1/SCA3 patients, the 40 mg cohort demonstrated an average 28% reduction in CSF mutant huntingtin protein at Day 85 compared to baseline. Importantly, CSF neurofilament light chain (NfL) levels remained essentially unchanged through Day 85. A few participants at higher doses experienced transient radiculitis, but no new serious adverse events were attributed to VO-659. **Vico** reports that VO-659 has a long CNS half-life, implying potential for only 1–2 intrathecal injections annually. Based on this data, the company is engaging regulators to plan a Phase 2 trial.

Collaborations & Alliances

HD-COPE

We are also proud to spotlight HD-COPE, a globally recognized international volunteer group composed of individuals and families with lived experience of Huntington disease. HD-COPE plays a vital role in shaping the future of HD research by providing essential feedback to pharmaceutical companies during drug development and to academic researchers worldwide. This unique perspective ensures that the voices of those who are the most affected guide scientific progress toward safe, effective, and meaningful treatments. We're always welcoming new members who want to contribute their insights and help drive change. **If you're interested in joining this impactful community, please reach out to** amaxan@huntingtonsociety.ca.

iGEM

We are thrilled to highlight our collaboration with the McMaster iGEM team, a group of innovative undergraduate researchers who reached out earlier this year to deepen their understanding of Huntington disease (HD) through the lens of lived experience and community needs. Their 2025 project focused on developing a novel diagnostic tool using synthetic biology, and our discussions about prevalence, family impacts, and gaps in care directly shaped their Integrated Human Practices approach. These insights helped refine their solution to better address real-world challenges, from equity in access to practical usability. At the 2025 iGEM Jamboree in Paris, the team proudly earned McMaster's first-ever Gold Medal, placed Top 5 in Best Diagnostics Project & Measurement, and fully met the gold criteria for Integrated Human Practices. These milestones were made possible, in part, by the Huntington Society of Canada's input. We're grateful for this meaningful partnership and look forward to future opportunities to inspire the next generation of HD researchers.

Collaborations & Alliances

CIHR-IA Dementia Research & Innovation Funders Alliance

We are excited to share our active involvement in the CIHR Institute of Aging's Dementia Research and Innovation Funders Alliance, a collaborative platform launched in November 2023 to unite Canadian funders and stakeholders in advancing brain health and dementia research. As a steering committee member, we contribute to aligning national investments, promoting capacity building, and mobilizing knowledge to support Canada's National Dementia Strategy. Through the alliance, we've partnered with leading dementia-related organizations, including the Alzheimer Society of Canada, ALS Canada, Parkinson Canada, Brain Canada, the Centre for Aging + Brain Health Innovation, Healthcare Excellence Canada, the Public Health Agency of Canada, and many more to conduct a comprehensive, nation-wide analysis of funding priorities and gaps. This mapping exercise documents current investments; identifies emerging needs and disparities in areas like prevention, care innovation, and equity; and informs a unified "research and innovation action plan" to maximize impact and address underserved domains in dementia research.

Our collective efforts are bridging silos, amplifying voices from affected communities, and paving the way for more targeted, transformative funding across the country.

HSC Research Council

Dr. Mahmoud Pouladi

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University of British Columbia

Dr. Tamara Maiuri

Dept. of Biochemistry & Biomedical Sciences
McMaster University

Dr. Rachel Harding

The Structural Genomics Consortium
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Dr. Tiago Mestre

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Huntington Society of Canada
HUNTINGTON
Société Huntington du Canada

Vision

A world free from Huntington disease

Mission

To improve the quality of life for those affected by Huntington disease

Huntington Society of Canada

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