ALS SLA
CANADA

APRIL 24-25, 2026
TORONTO

ALS EnCouRage CANADA

PROGRAMME

Welcome to ALS EnCouRage Canada 2026!

In 2023 and 2024, ALS Canada was fortunate to be invited as a contributor to an initiative created by our friends at the MND Association called MND EnCouRage UK. Through our participation, we have been able to witness the incredible impact that EnCouRage had on early-career researchers and members of the MND community in the UK. ALS Canada is proud to bring this exciting concept to the Canadian community in 2026 through the inaugural **ALS EnCouRage Canada**.

Over two days of programming, our hope is for early-career researchers to feel that they are joining a community that believes deeply in the value of their ideas, their curiosity, and their potential to shape the future of ALS research in Canada and beyond.

On Day 1, ECRs will have the opportunity to strengthen how they communicate their work, by refining presentations, receiving constructive feedback, and building confidence alongside peers and mentors who are invested in their growth. This day is about building skills, learning from others who have been there before, forming new peer relationships, and support in a collaborative environment.

Day 2 invites something equally important: connection. By opening the conversation to people affected by ALS, we hope to ground research in lived experience and remind us all why this work matters. We'll hear how research is understood beyond the lab, and how meaningful, accessible communication can help bridge the gap between discovery and impact.

ALS research is challenging, complex, and often forging new and uncertain paths. It is also driven by collaboration, shared learning, and community. We hope this meeting sparks new ideas, builds lasting relationships, and reminds ECRs that they are not working in isolation, while providing the community a sense of the passion, intelligence and heart that is working feverishly towards a future without ALS. We are all part of a growing, supportive network working together toward a common goal.

Thank you to all participants for bringing your time, energy, and passion to ALS EnCouRage Canada! We are excited to learn with you, and from you, over the days ahead.

Dave, Carolina & the ALS Canada Team

Dr. David Taylor,
Chief Scientific Officer

Carolina Jung,
Manager, Research Engagement & Impact

Day 1 | Friday, April 24

McIntosh Room

9:00 – 10:00 AM	BREAKFAST & REGISTRATION
10:00 - 10:30 AM	Opening Remarks & Faculty Introductions
10:30 - 12:00 AM	Breakout Groups (45 minutes each) **Breakout Group #1** – Dr. Heather Durham & Dr. David Taylor *Be memorable: communicating your science with impact* **Breakout Group #2** – Monique Dannell & Carolina Jung *Science in the spotlight: media, social, and public communications*
12:00 - 1:00 PM	LUNCH
1:00 - 2:10 PM	Early-Career Researcher Presentations & Workshop *Presenters will be divided into four groups of four. Speaking order will be randomized.* **Group 1 Individual Presentations** (3 minutes per speaker) **Feedback Workshop** (15 minutes) **Group 2 Presentation + Feedback Workshop** **Open Group Reflections** (10 minutes)
2:10 - 2:30 PM	BREAK
2:30 - 3:40 PM	Early-Career Researcher Presentations & Workshop **Group 3 & 4 Presentations + Feedback Workshops**
3:40 – 4:55 PM	Faculty Presentations (15 min each) **Building a successful career path in academia** – Dr. Chantelle Sephton **Building a successful career path in industry** – Kristiana Salmon **If at first you don't succeed...** – Dr. Richard Robitaille & Dr. Christine Vande Velde **The superpower of thinking big** – Dr. David Taylor
4:55 - 5:00 PM	Closing Remarks
6:00 - 9:00 PM	ALS Ignite Dinner
9:00 PM +	ECR Workshop & Networking space available

Day 2 | Saturday, April 25

McIntosh Room | Community Ambassador Streaming from 10:20 – 4:00 PM

7:30 – 8:30 AM	BREAKFAST
8:30- 8:45 AM	Opening Remarks
8:45 – 9:00 AM	Team Introductions
9:00 - 10:00 AM	A Scientific Paper Sleuthing
10:00 - 10:20 AM	BREAK
10:20 – 11:00 AM	Group 1 & 2 Presentations
11:00 - 12:00 PM	Faculty Scientific Talks **Damaged proteins take center stage in ALS** - Dr. Martin Duennwald **Maintaining nerve-muscle contacts to keep moving in ALS** - Dr. Richard Robitaille **Harnessing metabolic flexibility in ALS** - Dr. Chantelle Sephton **Beyond stress granules: new insights into TDP-43 and ALS** - Dr. Christine Vande Velde
12:00 - 1:00 PM	BREAK
1:00 – 1:40 PM	Group 3 & 4 Presentations
1:40 - 2:40 PM	Research in Real Life: Lived-Experience Perspectives Angie Leroux, Shawn Penno, Jason Ritchie *Moderated by Kelsie Snow*
2:40 - 3:00 PM	BREAK
3:00 - 3:45 PM	ALS Scientific & Medical Expert Panel
3:45 – 4:00 PM	Open Discussion: Integration of Research and Community
4:00 PM	Wrap up & Thank You

Presentation Groups

Speakers were divided into four groups of four. Speaking order was randomized.

GROUP 1	1. Isabel Rea 2. Bryan Kartono 3. Veronica Grybas 4. Jeremy Slayter, MD
GROUP 2	5. Antoine Desmeules 6. Jaya Bansal 7. Rafsanjany Kushol, PhD 8. Yasmeen Alshehabi
GROUP 3	9. Jacqueline Palik 10. Simon Alvado 11. Jennifer Soriano, PhD 12. Daniel Knight
GROUP 4	13. Mariam Choughari 14. Emmaley Hunter 15. Pedram Parnianpour, PhD 16. Sarah Lépine

Early-Career Researcher Presentations

(listed in alphabetical order by last name)

Yasmeen Alshehabi, University of Waterloo

A novel genetic approach to treating ALS using antisense oligonucleotides delivered via non invasive nanodiscs

Antisense oligonucleotides (ASOs) are promising therapies for genetic diseases like ALS. However, ASO delivery to the brain remains challenging due to the protective blood-brain barrier. Consequently, current delivery methods are highly invasive. For example, Tofersen is an FDA-approved ASO therapy for SOD1 ALS that must be injected directly into the spinal cord. To address this, we developed nanodiscs that can deliver ASOs to mouse brains following intravenous injection. I am optimizing these nanodiscs to increase the amount of ASOs they can carry to the brain and periphery. ALS is also genetically complex. The VCP gene alone has over 50 disease-causing mutations. Because VCP is an essential protein, lowering all VCP may be toxic. However, targeting 50 mutations may not be feasible. I am identifying shared DNA changes among VCP patients to develop a few ASOs that selectively degrade mutant VCP. This approach could also be applied to other ALS genes.

Simon Alvado, Université de Montréal

Why nerve-muscle contacts are destroyed in ALS?

Amyotrophic Lateral Sclerosis (ALS) is a severe neurodegenerative disease, affecting neurons that control muscles leading to their paralysis and ultimately breathing failure. A key feature of ALS is the early and progressive loss of the special connections between the neurons and the muscles. The reason why these specialized contacts are destroyed in ALS remains unclear. However, we discovered that they are destroyed and restored repeatedly before their definitive demise. We are convinced that this instability contributes to their fate, in other words, to their definitive demise. Thus, the project goal is to unravel the underlying properties of nerve-muscle contacts to understand the reasons why they are not repaired properly and sustainably in ALS. Understanding how this repair process works will help us identify potential therapeutic targets to maintain the nerve-muscle contact and preserve movement

Jaya Bansal, University of Calgary

Assessing Glymphatic Function as a Biomarker of ALS Using DTI-ALPS

The human brain has a cleaning network known as the glymphatic system, active at night to keep the brain properly functioning. It flushes out harmful proteins and other waste that builds up. However, if this system is impaired, toxic proteins can collect, spread, and damage brain cells (neurons), leading to diseases such as Amyotrophic Lateral Sclerosis (ALS). How the glymphatic system is altered in ALS remains under investigation.

To address this need, our research is assessing the utility of Diffusion Tensor Imaging-Along the Perivascular Space (DTI-ALPS), a safe brain scan that measures glymphatic activity. Using the largest multi-centre ALS cohort to date, our study will explore whether glymphatic changes are linked to the severity of the condition and how ALS changes over time. If confirmed, this work could enable a simple brain scan to track ALS progression, helping provide clearer information for patients and improving treatment development for researchers.

Mariam Choughari, Université de Montréal

When Neurons Lose Control

G3BP1 is an important protein that helps cells survive during stressful conditions by forming structures called stress granules. In amyotrophic lateral sclerosis (ALS), the normal behavior of these stress granules is disrupted. This happens partly because another protein, TDP-43, moves from its usual location in the nucleus of the cell to the cytoplasm in most patients. When TDP-43 is lost from the nucleus, it can no longer perform some of its normal functions, including protecting G3BP1. As a result, G3BP1 becomes more likely to be degraded. We also discovered a brain-enriched microRNA that binds to the same region of G3BP1 as TDP-43. When TDP-43 is missing from the nucleus, this binding site becomes exposed, allowing the microRNA to bind and promote the degradation of G3BP1. In this way, TDP-43 normally competes with this microRNA to maintain G3BP1 stability. Together, our findings reveal a new mechanism linking TDP-43 loss of function to stress granule dysregulation in ALS.

Antoine Desmeules, Université Laval

Understanding how brain and spinal cord cells use alternative energy source in ALS

Amyotrophic lateral sclerosis (ALS) is a fatal disease primarily affecting voluntary movement. Many people living with ALS also show changes with how their bodies produce energy from sugars and fats. In this project we tested whether a ketogenic diet (high in fat and low in sugar), which uses ketone bodies as a primary source of fuel instead of glucose, could prove beneficial to recover energy balance and slow disease progression. Using a familial mouse model of ALS carrying a mutation in the FUS gene, we found that a four-week ketogenic intervention improved motor and cognitive function along with disease-associated pathology. A ketogenic diet also normalized energy uses in a sex-dependent manner and improved the health of the cell's powerhouse, the mitochondria. Ongoing work focuses on how a ketogenic diet affects different cell types in the brain and spinal cord to find new metabolism-based targets for therapy.

Veronica Grybas, University of Ottawa

A protein modification called SUMOylation may contribute to TDP-43 localization

TDP-43 is a protein normally found in the nucleus that moves to the cell body when a cell is stressed. In ALS and under chronic cell stress TDP-43 persists in the cell body, preventing it from performing its function in the nucleus. The link between TDP-43 mislocalization and stress is unknown, but it may be a process called SUMOylation that modifies TDP-43 in response to cell stress. We hypothesize that SUMOylation acts as a safeguard that becomes critical when cells face additional challenges. Initially, we tested aging as a stressor in mice with impaired TDP-43 SUMOylation, however this did not produce robust effects. We therefore introduced a more ALS-relevant stressor, the C9ORF72 expansion, to determine whether combining impaired SUMOylation with disease-associated stress unmasks ALS-like changes. Complementary studies in stem cell-derived neurons will help identify human-specific effects. This will enhance our understanding of TDP-43 mislocalization, leading to new therapeutic targets.

Emmaley Hunter, Queen's University

Eye Tracking Reveals Vulnerability of Extra-Motor Neural Circuits in Amyotrophic Lateral Sclerosis

Amyotrophic lateral sclerosis (ALS) is increasingly recognized as a multisystem disorder. Functions beyond movement, such as energy balance and alertness, are often affected before motor symptoms appear and contribute significantly to disease burden. Importantly, these functions rely on brain regions that overlap with the oculomotor system: widespread neural circuits that direct and control the eyes. We investigated how oculomotor control is affected by ALS using a simple, video-based eyetracking task. A group of people living with ALS and healthy volunteers watched short clips of naturalistic scenes while their eye movements, pupil size, and eyeblinks were measured. Compared to the healthy volunteers, the ALS group exhibited a smaller pupil size at baseline, weaker pupil responses to changes in brightness, and longer eyeblinks. These findings support the view of ALS as a multisystem disorder and highlight eye tracking as a promising, noninvasive tool for monitoring non-motor symptoms and disease progression.

Bryan Kartono, University of Toronto

Repurposing an Approved Cancer Drug to Repair Nerve Cell Function in ALS

Amyotrophic lateral sclerosis (ALS) is a devastating neurodegenerative disease, and mutations in the C9orf72 gene are its most common genetic cause. In many patients, reduced levels of the C9orf72 protein are thought to contribute to disease by weakening the normal function of nerve cells. Increasing evidence suggests that problems at the connections between nerve cells occur early, before neurons begin to die. In this study, we explored whether an already approved brain-penetrant cancer drug could be repurposed to protect these vulnerable connections. Using genetically modified neurons lacking C9orf72 and patient-derived stem cell neurons, we found that neuronal structure and communication were impaired. Remarkably, treatment with this drug restored these defects, improving communication between neurons and stabilizing their structure. As this drug is already approved for clinical use and can reach the brain, this approach may offer a faster path to developing new treatments for ALS and related disorders.

Daniel Knight, Carleton University

Can DNA Aptamers Block Protein Aggregation in ALS?

In ALS, a protein called TDP-43 has shown to form harmful clumps, or aggregates, in about 97% of cases and is believed to play a central role in the disease. My work focuses on developing small molecules of DNA, called aptamers, to bind a specific region of TDP-43 to prevent the formation of these toxic aggregates. To select the most effective aptamers, we use a method called SELEX. This process can be compared to testing billions of keys in a lock: from a vast group of potential DNA sequences (the keys), only those that best fit the target region of TDP-43 (the lock) will prevail. After one year, we have completed this SELEX process and identified 16 promising aptamers. We are currently evaluating their ability to block TDP-43 aggregation and will soon study how they function in biological systems, with the goal of advancing ALS research and therapeutic development.

Dr. Rafsanjani Kushol, University of Alberta

AI-Driven Multi-Modal MRI Fusion for ALS Classification with Clinical Integration

A major challenge in diagnosing Amyotrophic Lateral Sclerosis (ALS) is the average delay of nearly a year before confirmation, which postpones vital care and clinical trial access. While MRI and artificial intelligence (AI) show promise, current models struggle to identify subtle brain changes and overcome artifacts and noise inherent in hospital scanners. Furthermore, most existing tools analyze only a single type of MRI, limiting their ability to capture the full picture of the disease. This project will develop a robust AI system that combines multiple MRI modalities (T1, T2, FLAIR, and diffusion MRI) with clinical data, including motor and cognitive scores. By integrating these complementary sources, the framework aims to classify ALS more accurately. The model will also be designed to work reliably across hospitals and even when some data are missing, helping shorten the diagnostic journey and improve patient selection for clinical trials.

Sarah Lépine, McGill University

Studying ALS mutations in stem cell-derived neurons to find new treatments

Many people with ALS show changes in a protein called TDP-43, suggesting it plays an important role in the disease. In my research, we wanted to understand how mutations in the gene encoding this protein disrupt motor neuron function. Using stem cells, we generated human motor neurons carrying mutations found in some people with ALS. We found that these neurons were less active and more vulnerable to cellular stress. By analyzing changes in gene expression, we identified molecular signatures linked to these mutations and used them to search an online database for compounds that might reverse these changes. This approach allowed us to prioritize promising candidates for laboratory testing and identify one compound that improved neuron survival and firing activity. Our next goal is to better understand how this compound works and explore whether it could represent a potential treatment avenue for ALS.

Jacqueline Palik, University of British Columbia

Histopathologic Correlates of High-Strength MRI Abnormalities in ALS

Certain individuals living with ALS show distinct changes on MRI scans, particularly along the corticospinal tract, the major nerve pathway that controls voluntary movement. These imaging findings have been associated with earlier disease onset and faster progression, yet the exact tissue changes responsible for this remains unclear. To investigate this, I study donated brain and spinal cord tissue from individuals with ALS. The tissue first undergoes MRI scanning at both conventional field strength (3T) and ultra-high field (9.4T), producing exceptionally detailed images. The same regions are then examined under the microscope to identify cellular and molecular changes, including motor neuron loss, inflammation, iron accumulation, and abnormal protein deposits such as TDP-43. By directly linking MRI findings to the underlying biological changes, this research aims to clarify what these scan abnormalities truly represent and help develop MRI tools that can better detect and monitor ALS progression during life.

Dr. Pedram Parnianpour, University of British Columbia

Enhancing ALS Clinical Trial Efficiency Using Longitudinal Measures of Brain Function

Amyotrophic lateral sclerosis (ALS) disrupts the brain's communication networks as nerve cells gradually lose function. Many emerging therapies aim to restore synaptic function, but clinical trials need sensitive tools to detect whether treatments are working. My research evaluates resting-state functional MRI (rs-fMRI), a non-invasive brain imaging method that measures how different brain regions communicate while a person is at rest. Using longitudinal data collected across multiple ALS centres, I examined how brain function changes over time and calculated how many participants would be needed to detect meaningful treatment effects in clinical trials.

We found that specific measures of brain connectivity are relatively reliable and highly sensitive to progression, requiring relatively small sample sizes compared with other MRI biomarkers. This work provides quantitative guidance for selecting efficient imaging endpoints and aims to help design faster, more informative clinical trials to accelerate the development of new ALS therapies.

Isabel Rea, University of Calgary

The role of the gut-blood-CNS axis in sex differences and pathogenesis of ALS

Men are twice as likely to develop ALS, with earlier onset and faster progression compared to women. Unfortunately, mechanisms underlying these sex differences are unclear. Recently, the gut microbiome (the ensemble of microorganisms that mediate the immune, metabolic and nutritional functions of the gut) has been implicated in ALS pathogenesis. Using ALS mouse models and a cohort of human ALS patients, the Nguyen lab has identified gut bacteria and metabolites that are responsible for the differential disease trajectory in males vs. females. However, whether the peripheral immune system mediates gut microbiome-driven sex differences is unknown. From both human and ALS mouse blood, I have identified sex differences in the composition and gene expression of the peripheral immune system that may contribute to accelerated disease in males or protection in females. Overall, this work implicates the gut-blood-CNS axis in sex differences in ALS, identifying immune pathways that may inform sex-specific therapeutics.

Dr. Jeremy Slayter, Dalhousie University

Measuring What Matters for People with ALS: Building an Evidence-Based Map to Improve the Measurement of Clinically Important Aspects of Life

ALS isn't just about muscles; it changes how people live, making it tough to move, connect with others, speak clearly or even think. It's important that ALS research looks at all these different effects and has helpful ways to measure them. This research considered what's most important for people with ALS and put together a map of the tools and what we know about those tools. Now, researchers and doctors can pick the best tools to measure more aspects of life and even create new tools to better understand what it is like to live with ALS. Next, we'll get together as a national group of people with ALS, doctors and researchers to pick the tools we think are most important and show what life with ALS is really like. This will help future studies focus on what patients care about most.

Dr. Jennifer Soriano, University of Toronto

Amyotrophic Lateral Sclerosis Bulbar Dysfunction Index–Remote: Responsiveness to change

Monitoring changes in bulbar function (i.e., speech and swallowing) of individuals with amyotrophic lateral sclerosis (ALS) is crucial to patient care, particularly when presence of bulbar dysfunction has been associated with deceased quality of life and shorter life expectancy. The ALS Bulbar Dysfunction Index – Remote (ALSBDI-R) was developed to measure and monitor bulbar function of individuals with ALS. Current evidence on ALSBDI-R demonstrated moderate to excellent interrater and test–retest reliability, with evidence supporting face, content, and construct validity. However, the psychometric responsiveness and meaningful change of the tool have not yet been established. Thus, the primary aim of the project is to establish the responsiveness and meaningful change of the ALSBDI-R. Sixty-four individuals with ALS participated in two data collection sessions approximately 6-months apart. We hypothesize that ALSBDI-R is responsive to changes in bulbar symptoms, thereby enhancing the range of assessment tools available for monitoring bulbar function.

Faculty & Presenters

Angie Leroux

My name is Angie Leroux and I'm in the diagnostic grey area between PLS and UMN dominant ALS. I have a degree in Science (major in Microbiology) and a teaching degree. I have always loved Science and I am fascinated and hopeful with the research being done by the participants of this conference. Before going on long term disability, I taught middle school and I miss my career more than I can say. I am an ALS Canada Ambassador and I am thankful for this opportunity.

Shawn & Sabina Penno

Shawn was diagnosed in September of 2023 and Sabina has been by his side supporting him in every way. He continued to work as a Community Health Worker until the end of February when he stopped doing client care but continued office work for OH&S and union work. Sabina works as a Long Term Care Aid, and is well aware of what ALS looks like. We both work to advocate for more investment in care, research and mental health, hence why we are excited to meet and inspire early career researchers in this summit to excel in this field and move the needle forward to find the cure!!

Jason & Christina Ritchie

Small business owners Jason and Christina Ritchie have been married for 22 years, raising their two adult children from their home in Mississauga, ON. In the summer of 2023, Jason began noticing changes in his gait, causing difficulty with everyday activities. Dismissing the signs as "nothing serious" for the remainder of 2023, he eventually sought medical advice in early 2024 after a few months of worsening symptoms. Aggressive investigations by a team of medical professionals lead to Jason's limb onset ALS diagnosis in June 2024. Since then, he's been focused on advocacy and fundraising. Jason is a member of the Board of ALS Canada, attends Walk to End ALS events, and is a two time rider in ALS Canada's Revolution Ride. Jason has been a participant in the clinical trials of two experimental compounds, as well as a study subject in the observational trials Capture ALS, and the ALS Research Collaborative (ARC) at the ALS Therapy Development Institute (TDI) in the USA.

Kelsie Snow

Kelsie Snow is the Project Manager of the Alberta ALS Research Network, a collaboration between the ALS Society of Alberta and the province's top ALS clinicians and researchers. She stepped into ALS advocacy in June 2019, when her husband, Chris, was diagnosed with familial ALS (SOD-1, A4V) and given 6-12 months to live. Instead, Chris enrolled in Phase 3 of the Valor trial for the ASO tofersen and lived for more than 4 years, making countless core memories with his family before he passed away in September 2023. During Chris' illness, the Snows chose to live out their lives publicly to raise ALS awareness and funds for research. Both trained journalists, Kelsie and Chris chronicled their journey on Kelsie's blog, in her podcast and through numerous radio, television and newspaper stories and appearances. In partnership with Chris' employer, the Calgary Flames, the Snows' initiatives have raised more than $600,000 CAD for research on familial ALS at the University of Miami's Miller School of Medicine in Florida, a world-first study testing the use of focused ultrasound to open the blood brain barrier for more effective delivery of ALS medications at Sunnybrook Health Sciences Centre in Toronto, and the Canada-wide data collection study CAPTURE ALS, headquartered in Alberta. Kelsie lives in Calgary with her children, Cohen (14) and Willa (11).

Nick Cole, PhD

Nick completed his PhD in St. Andrews University in Scotland, UK before research postdocs in St. Andrews, Dundee and Sydney Australia. Nick has a background in muscle development and physiology, limb development and MND/ALS research. Nick began his own research laboratory in The University of Sydney, modelling ALS/MND in zebrafish before helping to establish the MND Research Centre at Macquarie University, Sydney. Nick returned to the UK with his family (and dog 'Vegemite') to take up his position as Head of Research at the MND Association in 2018. Nick is a keen Kitesurfer and previous Guiness world record holder after his 'kitethereef' MND fundraiser in 2015. As Head of Research, Nick's role involves leading the team responsible for The MND Associations International Symposium on MND/ALS and event for early career researchers (Encourage UK). He also oversees the Research Communications team and represents the research directorate of the MND Association at various events, which includes standing in for the Director of Research as required. He is also a member of the the International Alliance Scientific Advisory Council and the UK Clinical Studies Group.

Monique Dannell

Monique received her Bachelor of Arts with an Honours in Communications and Media from the University of Ottawa. With experience specializing in not-for-profit communications, Monique works at the ALS Society of Canada as the Manager, Digital Communications and Content Strategy. She leads ALS Canada's digital content strategy, overseeing the organizations social media channels, eNewsletter, and community blogs. With a passion for community engagement and human connection through storytelling, Monique focuses on utilizing diverse digital platforms to uplift community voices, raise awareness on critical issues, and share credible information in digestible, compelling ways.

Martin Duennwald, PhD

Dr. Duennwald received his PhD from the Max-Planck Institute in Cologne Germany in molecular cell biology. He then trained as a postdoc in Dr. Susan Lindquist's lab at the Whitehead Institute/MIT in Cambridge, Massachusetts where he developed yeast novel models and deciphered how defects in cellular protein quality control contribute to protein misfolding and the associated cytotoxicity. He then led his own research team as principal investigator at the Boston Biomedical Research Institute to continue research on protein misfolding in neurodegenerative disease. Joining the faculty of the Department of Anatomy and Cell Biology at the Schulich School of Medicine and Dentistry at the University of Western Ontario, where he is now an Associate Professor, Dr. Duennwald's lab focuses on impaired cellular protein quality control in ALS.

Heather Durham, PhD

Dr. Heather Durham obtained her Honours BSc in Physiology and Pharmacology and MSc in Pharmacology from the University of Western Ontario and PhD in Pharmacology from the University of Alberta. After postdoctoral studies in the Department of Pharmacology and Therapeutics of McGill, she continued studies at the Montreal Neurological Institute and joined the faculty of the Department of Neurology and Neurosurgery, achieving the rank of Professor. She is currently Professor Emerita with a post-retirement position. Her laboratory has focused on the study of motor neuron diseases and other rare disorders. Administratively, she has held several positions including Director of the Graduate Program in Neurological Sciences, Associate Dean of Graduate and Postdoctoral Studies, and Chair of the Neuro lab safety committee. Dr. Durham has been highly involved with charitable organizations, professional societies and government agencies, including the Scientific and Medical Advisory Committee and the Board of Directors of ALS Canada; the Medical Advisory Committee for MDA; the Board of Directors of the Society of Toxicology of Canada; and the Biological Chemical Defence Review Committee for the Department of National Defence. She is currently a member of the Board of Directors of ALS BC. She currently resides in Victoria, while maintaining a presence in Montreal.

Carolina Jung

Carolina obtained a Bachelor of Science with an Honours Specialization in Interdisciplinary Medical Sciences from Western University in 2021. She further advanced her education by obtaining a certificate in Biomedical Communication and Visualization from the University of British Columbia. With experience in laboratory work and knowledge translation, she has been working at ALS Society of Canada since 2021, now as Manager, Research Engagement and Impact. At ALS Canada, Carolina leads research communications and provides key support to the Research Grant Program, as well as contributing across multiple initiatives that drive meaningful impact for the ALS community. Carolina remains dedicated to her passion for accessible and timely scientific and medical communication, empowering people affected by ALS to make informed decisions, and elevating the visibility and recognition of ALS research across Canada.

Richard Robitaille, PhD

Dr. Richard Robitaille's current research explores the roles of glial cells in the regulation of function of neurons and the neuronal contacts. His work focuses particularly on the contribution of glial cells in the demise of neuromuscular synapse in ALS, using neuromuscular preparations from rodent models of ALS and samples from patients. His work led to an ongoing Phase 2a clinical trial testing a strategy that help stabilize neuromuscular synapses. The current goals are to develop novel neuromuscular-based biomarkers, understand the mechanisms underlying neuromuscular demise and identify new therapeutic targets with potential clinical applications.

Kristiana Salmon

Kristiana Salmon is an Associate Medical Director, Clinical Development at Satellos Bioscience. In this role, she is responsible for the design, execution, and scientific oversight of clinical trials across all phases of drug development. Prior to joining Satellos, Kristiana previously held the position of Clinical Scientist, Clinical Development at QurAlis, where she supported multiple clinical-stage programs in ALS and FTD, and consulted for pre-clinical and clinical-stage biotechs. Prior to her industry roles, she spent over a decade at the Montreal Neurological Institute-Hospital, with extensive clinical and research experience in ALS, adult neuromuscular diseases, and rare neurogenetic conditions. Internationally, Kristiana has played a leading role in advancing initiatives to improve genetic counselling and testing for ALS, most recently leading the development of global best practice recommendations. Kristiana received her Bachelor of Science in Microbiology at McGill University and her Executive MBA at Concordia University, both in Montreal.

Chantelle Sephton, PhD

Dr. Chantelle F. Sephton received her Bachelor's of Science in Biochemistry from the University of Saskatchewan in Saskatoon (2002) and her PhD in Psychiatry from the University of Saskatchewan (2007). She did her postdoctoral studies at the University of Texas (UT) Southwestern Medical Center at Dallas under the mentorship of Dr. Gang Yu in the Department of Neuroscience (2007-2014). She started her lab at Université Laval in the CERVO Brain Research Centre in 2014 and is currently an Associate Professor in the department of psychiatry and neuroscience. Dr. Sephton's research interests are focused on the mechanism of action of post-transcriptional regulation of RNA by RNA-binding proteins. Several RNA-binding proteins including TDP-43 and FUS are mutated in some patients with familial amyotrophic lateral sclerosis (ALS). Mutations in these proteins result in their cytoplasmic mislocalization and aggregation, which are thought to contribute to neurodegeneration in these diseases. Stemming from her post-doctoral work and the discovery of the biological functions of disease-linked RNA-binding proteins, her current work now focuses on understanding the mechanisms by which disease-associated mutations of TDP-43 and FUS influence RNA metabolism and how this may lead to neurodegeneration.

David Taylor, PhD

Dr. Taylor completed a PhD in ALS research at McGill in 2006, followed by two postdoctoral fellowships at EPFL, Lausanne, and University of Toronto. He joined ALS Canada in 2012 and for over a decade his work has included; managing ALS Canada's national and global research portfolios, guiding the organization's research strategy, facilitating collaboration between Canadian and international ALS researchers, advocating with government for the needs of Canadians living with ALS, and engaging with industry to highlight Canada as a top destination for clinical development. His role has also provided an opportunity to be involved in, or leading numerous panels, committees, and meetings of global ALS/MND significance, with impact that will ultimately accelerate a better day, sooner, for Canadians living with ALS. Passionate about ALS research, Dave has delivered hundreds of presentations across Canada and internationally, participated in countless media interviews and is incredibly driven to engage in mutually beneficial opportunities for knowledge exchange.

Christine Vande Velde, PhD

Dr. Christine Vande Velde is Full Professor in the Department of Neurosciences at the Université de Montréal and Université de Montréal Hospital Research Center (CRCHUM). Her research is centered on understanding the underlying pathological mechanisms that lead to the fatal neurodegenerative diseases amyotrophic lateral sclerosis (ALS) and frontotemporal dementia (FTD) and the exploitation of that knowledge for biomarker and therapeutic development. Dr. Vande Velde is the current Scientific Director of the Robert Packard Center for ALS Research at Johns Hopkins and a member of the Scientific and Medical Advisory Council of ALS Canada.

ALS ADVANCE NATIONAL MEETINGS

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