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Axe Neurosciences
Centre de recherche du CHUM
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Statut universitaire / University status

Professeure sous octroi agrégée, Département de neurosciences, Faculté de médecine, Université de Montréal

Appartenance à d'autres groupes / Affiliation with other groups

Membre associé, Groupe de recherche sur le système nerveux central (GRSNC) du FRQS

Formation / Training

B.Sc., Génétiques, University of Manitoba, Winnipeg, MB, Canada, 1993-1997
Ph.D., Biochimie et biologie moléculaire, University of Manitoba, Winnipeg, MB, Canada, 1997-2001
Stage postdoctoral, Neuroscience, Ludwig Institute for Cancer Research, UCSD, San Diego, CA, États-Unis, 2001-2007

Orientations de la recherche

- Étude de la dégénérescence des motoneurones dans le système nerveux, particulièrement dans le cas de la sclérose latérale amyotrophique (SLA).
- Utilisation des modèles de la souris et des rats transgéniques, des approches complémentaires et multidisciplinaires sont utilisées in vivo (culture cellulaire et souris) et in vitro (mitochondrie isolé). Les technologies incluent des outils de biochimie, biologie cellulaire, protéomique, immunocytochimie et de microscopie confocale et électronique.

Principaux projets en cours

- Contributions de SOD1 et mitochondries à la pathogénèse de la SLA.
- Contributions de granules de stress et TDP-43 à la SLA.

Research orientations

- Study of motor neuron degeneration, especially in the context of amyotrophic lateral sclerosis (ALS), also known as Lou Gehrig's Disease. We use transgenic mouse and rat models, cell culture, biochemistry, cell biology, proteomics, immunocytochemistry, confocal and electron microscopy.
- Utilization of transgenic rat and mouse models. Complementary and multidisciplinary approaches are used in vivo (cell culture and mice) and in vitro (isolated mitochondria). Technologies include aspects of biochemistry, cell biology, proteomics, immunocytochemistry, confocal and electron microscopy.

Current research projects

- Contribution of SOD1 and mitochondria to ALS pathogenesis.
- Contribution of stress granules and TDP-43 to ALS.

Publications choisies / Selected publications

Arbour, D., Vande Velde, C. and Robitaille, R. (2017). New perspectives on amyotrophic lateral sclerosis: the role of glial cells at the neuromuscular junction. *J Physiol*, 595 (3): 647-661.

Chiasseau, M., Cueva Vargas, J. L., Destroismaisons, L., Vande Velde, C., Leclerc, N. and Di Polo, A. (2016). Tau Accumulation, Altered Phosphorylation, and Missorting Promote Neurodegeneration in Glaucoma. *J Neurosci*, 36 (21): 5785-98.

Pickles, S., Semmler, S., Broom, H. R., Destroismaisons, L., Legroux, L., Arbour, N., Meiering, E., Cashman, N. R. and Vande Velde, C. (2016). ALS-linked misfolded SOD1 species have divergent impacts on mitochondria. *Acta Neuropathol Commun*, 4 (1): 43.

Aulas, A., Caron, G., Gkogkas, C. G., Mohamed, N. V., Destroismaisons, L., Sonenberg, N., Leclerc, N., Parker, J. A. and Vande Velde, C. (2015). G3BP1 promotes stress-induced RNA granule interactions to preserve polyadenylated mRNA. *J Cell Biol*, 209 (1): 73-84.

Aulas, A. and Vande Velde, C. (2015). Alterations in stress granule dynamics driven by TDP-43 and FUS: a link to pathological inclusions in ALS? *Front Cell Neurosci*, 9: 423.

Pickles, S., Arbour, N. and Vande Velde, C. (2014). Immunodetection of outer membrane proteins by flow cytometry of isolated mitochondria. *J Vis Exp* (91): 51887.

Pickles, S., Cadieux-Dion, M., Alvarez, J. I., Lecuyer, M. A., Peyrard, S. L., Destroismaisons, L., St-Onge, L., Terouz, S., Cossette, P., Prat, A. and Vande Velde, C. (2013). Endo-MitoEGFP mice: a novel transgenic mouse with fluorescently marked mitochondria in microvascular endothelial cells. *PLoS One*, 8: e74603.

Pickles, S., Destroismaisons, L., Peyrard, S. L., Cadot, S., Rouleau, G. A., Brown, R. H., Jr., Julien, J. P., Arbour, N. and Vande Velde, C. (2013). Mitochondrial damage revealed by immunoselection for ALS-linked misfolded SOD1. *Hum Mol Genet*, 22: 3947-59.

Aulas, A., Stabile, S. and Vande Velde, C. (2012). Endogenous TDP-43, but not FUS, contributes to stress granule assembly via G3BP. *Mol Neurodegener*, 7: 54.

Pickles, S. and Vande Velde, C. (2012). Misfolded SOD1 and ALS: zeroing in on mitochondria. *Amyotroph Lateral Scler*, 13: 333-40.

McDonald, K. K., Aulas, A., Destroismaisons, L., Pickles, S., Belec, E., Camu, W., Rouleau, G. A. and Vande Velde, C. (2011). TAR DNA-binding protein 43 (TDP-43) regulates stress granule dynamics via differential regulation of G3BP and TIA-1. *Hum Mol Genet*, 20: 1400-10.