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RESEARCH AREA

Our research aims to better understand the interplay between the cardiovascular system and the nervous system. Specifically, we are focusing on the connection between myocardial infarction/heart failure and major neurodegenerative diseases including Alzheimer's disease, and amyotrophic lateral sclerosis/frontotemporal dementia (ALS/TFD). We are particularly interested in molecular mechanisms that underlie cell death in these diseases including endoplasmic reticulum mitochondrial signalling and intracellular transport. We have shown damages to these cellular processes in some forms of dementia and ALS/FTD, and these are also involved in some cardiovascular diseases. We are currently investigating this further with special focus on therapeutic routes for correcting these types of damages.

TECHNIQUES AVAILABLE IN THE LAB

We apply advanced cell- and molecular biological methods to our research, thus we use various molecular cloning methods, cell line cultivation, protein isolation and separation, and detection of protein-protein interactions. We complement our cellular studies with a variety of fluorescent histological examination methods. We also use the most advanced super-resolution and electron microscopic imaging techniques to follow changes in our samples in the size range of a few tens of nanometres. We develop our own computer programs for the analysis of microscopic images.

SELECTED PUBLICATIONS

Markovinovic, A., Martín-Guerrero, S. M., **Mórotz, G. M.**, Salam, S., Gomez-Suaga, P., Paillusson, S., Greig, J., Lee, Y., Mitchell, J. C., Noble, W., & Miller, C. C. J. (2024). Stimulating VAPB-PTPIP51 ER-mitochondria tethering corrects FTD/ALS mutant TDP43 linked Ca^{2+} and synaptic defects. **Acta Neuropathol Commun 12(1):** 32.

Mórotz, G. M., Martín-Guerrero, S. M., Markovinovic, A., Paillusson, S., Russell, M. R. G., Machado, P. M. P., Fleck, R. A., Noble, W., & Miller, C. C. J. (2022). The PTPIP51 coiled-coil domain is important in VAPB binding, formation of ER-mitochondria contacts and IP3 receptor delivery of Ca^{2+} to mitochondria. **Front Cell Dev Biol 10:** 920947.

Gomez-Suaga, P., **Mórotz, G. M.**, Markovinovic, A., Martín-Guerrero, S. M., Preza, E., Arias, N., Mayl, K., Aabdien, A., Gesheva, V., Nishimura, A., Annibali, A., Lee, Y., Mitchell, J. C., Wray, S., Shaw, C., Noble, W., & Miller, C. C. J. (2022). Disruption of ER-mitochondria tethering and signalling in C9orf72-associated amyotrophic lateral sclerosis and frontotemporal dementia. **Aging Cell 21(2):** e13549.

Mórotz, G. M., Glennon, E. B., Greig, J., Lau, D. H. W., Bhembre, N., Mattedi, F., Muschalik, N., Noble, W., Vagnoni, A., & Miller, C. C. J. (2019). Kinesin light chain-1 serine-460 phosphorylation is altered in Alzheimer's disease and regulates axonal transport and processing of the amyloid precursor protein. **Acta Neuropathol Commun 7(1):** 200.

Mórotz, G. M., De Vos, K. J., Vagnoni, A., Ackerley, S., Shaw, C. E., & Miller, C. C. (2012). Amyotrophic lateral sclerosis-associated mutant VAPBP56S perturbs calcium homeostasis to disrupt axonal transport of mitochondria. **Hum Mol Genet 21(9):** 1979–1988.