

## FIRST PERSON

# First person – Pamela Martino Adami

First Person is a series of interviews with the first authors of a selection of papers published in Journal of Cell Science, helping early-career researchers promote themselves alongside their papers. Pamela Martino Adami is first author on 'Perturbed mitochondria–ER contacts in live neurons that model the amyloid pathology of Alzheimer's disease', published in JCS. Pamela conducted the research described in this article while a PhD student in Dr Laura Morelli's lab at Fundación Instituto Leloir–IIBBA CONICET, Argentina. She is now a Postdoctoral Research Fellow in the lab of Dr Alfredo Ramirez at the University of Cologne, Germany, investigating the biochemical mechanisms and signaling pathways responsible for the onset of Alzheimer's disease.

### How would you explain the main findings of your paper in lay terms?

There are certain structures inside the cell, called mitochondria and the endoplasmic reticulum, which can associate physically to regulate very important cellular functions, like lipid production. In this paper we have shown that in young neurons in possession of a mutated gene (amyloid precursor protein, APP) that causes a heritable form of Alzheimer's disease, this 'association' is impaired, and it has a negative impact on the production of lipids that make up the membranes within the neurons, like the mitochondrial membrane. When mitochondria have disrupted membranes, they do not produce all the energy they should do, and this situation has detrimental consequences for the neurons: they rely on properly functioning mitochondria when they are involved in memory-related tasks.

### Were there any specific challenges associated with this project? If so, how did you overcome them?

We had many difficulties concerning funding and equipment. Argentina has serious problems with financing research and innovation, and so the budget is low. We needed to perform specific microscopy techniques, and neither the equipment nor the knowledge was available in our country, so I had to apply for international fellowships to go abroad. Fortunately, both the International Society for Neurochemistry (ISN) and the International Union of Biochemistry and Molecular Biology (IUBMB) awarded me with fellowships which made two short stays in the USA possible for me, so that I could perform all the live imaging experiments there.

### When doing the research, did you have a particular result or 'eureka' moment that has stuck with you?

I remember how happy I was the day we received the analysis of the lipidomics data and saw that some key phospholipids were downregulated in the transgenic neurons, just like we expected – it meant that our hypothesis was true! We celebrated and just after that we started to write the manuscript.

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Pamela Martino Adami

### Why did you choose Journal of Cell Science for your paper?

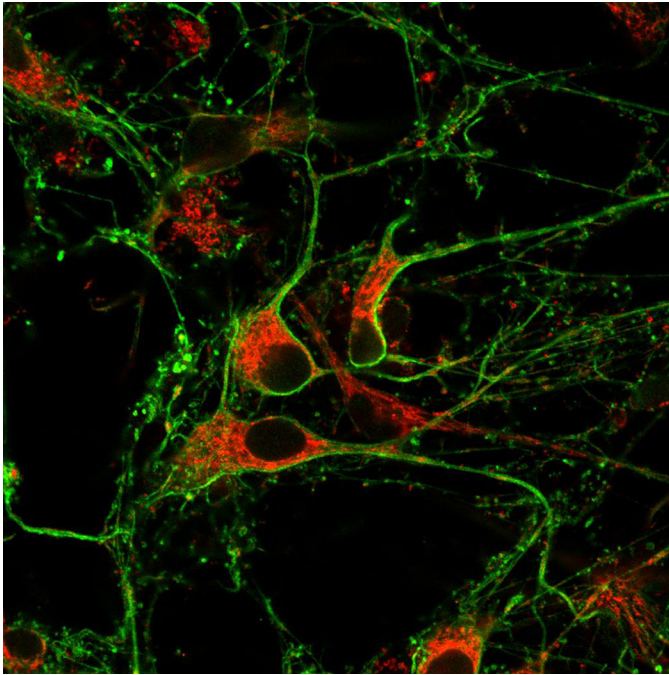
Because we wanted our results to be published in a high-quality journal that could reach a broad cell biology audience, and not only neuroscientists.

### Have you had any significant mentors who have helped you beyond supervision in the lab? How was their guidance special?

I think that every member of my former lab has helped and advised me, not only about science but also about matters of life, and made the days during my PhD great. I have also met other colleagues during this period, both in Argentina and abroad, who have encouraged and inspired me. They have all absolutely contributed to shaping me into the person I am now.

### What motivated you to pursue a career in science, and what have been the most interesting moments on the path that led you to where you are now?

I have always been a curious person who tries to understand how and why things work. While I was at school, I took part in the National Biology Olympics, an experience that made me decide to pursue a major related to biology and medicine. After graduating college, I enrolled in a PhD program focused on Alzheimer's disease. The reason for this was seeing my grandmother suffering from a neurodegenerative disease, without the possibility of any



Primary hippocampal neuronal cultures (7 days *in vitro*) labeled for  $\beta$ III-tubulin (green) and the mitochondrial protein Tom20 (red).

therapeutic approach that could change the course of it. At that moment I decided to make my own tiny contribution to expand the current knowledge of neurodegenerative diseases.

#### Who are your role models in science? Why?

Luis Federico Leloir, an Argentine physician and biochemist who received the 1970 Nobel Prize in Chemistry, and former director of the institute where I undertook my PhD studies. Even though his laboratory, located in an underdeveloped country, often lacked financial support and had second-rate equipment, he and his team were able to produce outstanding scientific research that has led to significant progress in understanding the congenital disease galactosemia. He even spent the Nobel Prize money directly on research. Besides, he was known as a very humble person.

#### What's next for you?

I am currently doing my postdoc in Germany focusing also on Alzheimer's disease, but working with human samples. My goal is to be able to generate key data for the patient care decision-making process and medical education, which could help future patients plan their future while they are still capable, and also conduct preventive behavior in order to delay the onset of dementia.

#### Tell us something interesting about yourself that wouldn't be on your CV

I love traveling, drawing, singing and riding horses. I would love to learn to sing opera!

#### Reference

Martino Adami, P. V., Nichtová, Z., Weaver, D. B., Bartok, A., Wisniewski, T., Jones, D. R., Do Carmo, S., Castaño, E. M., Cuellar, A. C., Hajnóczky, G. et al. (2019). Perturbed mitochondria-ER contacts in live neurons that model the amyloid pathology of Alzheimer's disease. *J. Cell Sci.* **132**, 229906. doi:10.1242/jcs.229906