NeuroCampus – Inside Out:

# Marina Romero-Ramos

## Group Leader at CNS Disease Modelling Group, Department of Biomedicine

Romero-Ramos' Lab investigates the underlying mechanisms of neurodegeneration related to α-synuclein aggreagation during Parkinson's disease with a focus on the immune component.

Marina Romero-Ramos. Photo: Karoline Klitgaard

**Can you describe your research in a nutshell?**
My team investigates the different immunological factors and players involved in the neurodegenerative process of Parkinson's disease. We do this with an underlying belief that the aggregation of α-synuclein plays a central role in the disease process. In our research method, we continuously translate back and forth between patients and animal models of the disease. We collaborate with clinicians at Aarhus University Hospital (AUH) who do PET-scans of people with Parkinson and other α-synuclein related brain diseases. We examine the same patients' immunological processes in blood and CSF and try to correlate this information with that obtained from the scans. This allows us to develop new hypotheses about how immune system influence the neural degeneration. We then study these hypotheses in our animal Parkinson's models at a molecular level.
One of our current research projects investigates the disease related changes associated with the CD163-receptor in collaboration with Drs. Søren Moestrup and Anders Etzerodt. This receptor appears to function differently in male and female patients, which potentially could be related to the differences in presentation and prevalence of Parkinson's between men and women.
For a different project, we collaborate with Drs. Per Borghammer and Nathalie Van Den Berge at AUH. In this project we investigate the hypothesis proposed by Per's team, that there exists two different subtypes of Parkinson's disease – namely a grain-first and a Gut-first type (Read the NeuroCampus Inside Out issue with Per Borghammer here for more information). Here the examination of underlying immunological mechanisms is of great relevance, since the brain-gut connection has plays crucial role in modulating the immune system and the immunological response during disease.

**What translational impact may your research have for people?**
Discovering new immunological processes and the involved immune cells are essential for finding novel therapeutic targets. This can improve the possibility of both treating and monitoring the disease as well as selecting the relevant type of therapy for the individual patients.

**How did you end up where you are today?**
I always wanted to be a scientist and originally did a degree in pharmacology back in Spain. While studying, I volunteered as a student assistant at a neuroscience lab since I was eager to do research. This eventually led me to pursue a PhD and a research career in neuroscience. I had the privilege of doing my education at various very good universities, e.g. UCLA (USA) with Dr. MF Chesselet, a respected Neuroanatomist, and at Lund University (Sweden), with Prof. Anders Björklund, a world leader in Parkinson's research.

**What does a (local) strong neuroscience research network mean for you and your research?**
I believe that a certain number of researchers working in related fields is essential for both technical support and scientific progression. A broad community is also key for our students as it provides important educational opportunities.
Collaboration across different neuroscience-related disciplines is such a natural part of our work that I do not even think of it as multidisciplinary- everyone is just in neuroscience. It is crucial for our projects that we have access to a lot of different knowledge and perspectives. Outside the field of neuroscience, we also engage in a lot of collaboration with the immunologists. like Dr. SR Palludan, experts in genetic research such as Dr. J. Kjems, and protein aggregation such as D. Otzen.

**If you had unlimited resources to conduct a big, multidisciplinary neuroscience project, what would you like to do?**
We just started a big project that I have actually had in mind for years, for which we could still use a lot more resources. The goal is to conduct several longitudinal studies of both animal models and human patients with α-synuclein-associated diseases, to learn about neuronal and immune changes in parallel and how these relates to α-synuclein. Aggregation of α-synuclein occurs in different brain diseases; e.g. Parkinson's, multiple system atrophy and Lewy Body dementia. However, the aggregation proceeds differently in each disease, and we want to examine whether immune disparities may also account for the differences between the diseases. With help from collaborators such as PH Jensen, we can use α -synuclein obtained from patients with each disease to model the disease in animals, and thus study the progression in patients and animals in parallel.
Longitudinal studies are crucial for our Parkinson's research, as the disease presents itself very differently as time progresses, with the progression being different for each patient. It is very likely that different immune cells and proteins are relevant at different stages of the disease, meaning that different therapeutics may similarly be relevant at different stages..
Thus, I believe it essential to study all the mechanisms *in vivo* over time in both humans and animal models.


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