BAGADILICO PROFESSORS INSTALLED

On March 16th two well-known Bagadilico scientists were installed as professors during a ceremony in Lund University's main hall. Here, Jia-Yi Li and Cecilia Lundberg tell the stories of how they ended up in a professorial robe on that special day.

JIA-YI LI

“I was born in 1958 in a small village in Shanxi province in central China. During my childhood, as well as during my university time, I lived in southwest China, more precisely the Sichuan province. I graduated from Luzhou Medical College in 1982. After several years of working as a lecturer and researcher, I came to the University of Gothenburg in 1990 and received my doctorate there in 1995. The first years as a postdoctoral researcher I stayed in Gothenburg and in 1999 I became an associate professor. In 2001 I moved to Lund and ever since I have worked at the Wallenberg Neuroscience Center, first as a research assistant and since 2005 as a university lecturer. Since 2011, I am a professor of neuroscience.”

CECILIA LUNDBERG

“I was born in 1966 in Uppsala. When I was five the family moved to Gothenburg, where I grew up. In 1988, I began to study medicine in Lund and I started my graduate studies at the Department of Medical Cell Research a couple of years later. After my dissertation, I continued my studies as a postdoctoral fellow at Harvard Medical School in Boston. After nearly three years in the U.S., I returned to Lund and commenced to set up my research team at the medical faculty. I continue to teach future physicians and biomedical scientists and in 2010 I became professor of neurobiology. Today, I work as a research leader, teacher and vice dean of the medical faculty.”
The fifth Parkinson Café saw two skilled clinical researchers take center stage. With their professional paths often crossing, Oskar Hansson and Elisabet Londos worked in comfortable tandem as they inspired a direct dialogue with the audience. As the Parkinson Café is now becoming a regular fixture in people’s calendars the many recurring visitors seemed unafraid to take the floor as they showered the duo with questions.

Neurologist Oskar Hansson kicked off proceedings with his talk on the importance of developing improved biomarkers for Parkinson’s and other neurodegenerative disorders. Disease development in the parkinsonian brain is likely to occur up to fifteen years before serious symptoms start to show. With effective and safe tools for early diagnosis new opportunities for pre-emptive therapies would be made possible. However, today’s diagnostic methods do not allow for secure diagnosis at this stage. Oskar Hansson aims to rectify this research gap with his own studies in biomarkers.

- Our overarching research goals are threefold. First, we want to identify and validate accurate and cost-effective blood-based biomarkers for early identification of those at high risk of developing Parkinson’s disease in primary care. Secondly, we aim to develop diagnostic algorithms using advanced imaging and cerebrospinal fluid-markers to diagnose diseases earlier and more accurately in specialist care. Finally, this would lead us to better understand the underlying pathology and early development of Parkinson’s disease so that we can begin to develop relevant new drug targets for clinical trials, Oskar told the audience. Café visitors followed up with questions on the different advantages early diagnosis might bring.

- Well, besides from the immediate benefits of being able to set in symptomatic treatments early and the possibilities for testing new therapies there is also an advantage for the patient in just knowing their diagnosis. It will perhaps give peace of mind as the uncertainty may be wearing on the patient. Also, you will be able to avoid a number of unnecessary examinations that are likely to add further stress to the patient’s life.

Oskar Hansson continued to speak on the exciting research avenues that may result from a new generation of biomarkers. New disease modifying treatments that aim to “catch” the disease at an earlier stage are likely to have a better chance of slowing down or stopping brain cell death in Parkinson’s disease.

- Despite a large number of completed clinical trials, there are still no treatments that halt the development of Parkinson’s disease. New disease modifying treatments are likely to be most effective if started before patients have obvious symptoms, that is before the nerve cell damage is already severe and irreversible, making the biomarkers for early diagnosis crucial to future drug development and clinical trials. Given that animal models of Parkinson’s disease often do not reflect the underlying disease mechanisms in humans adequately, we must intensify our efforts to study people affected by these diseases, with the aim to identify these early disease mechanisms in order to develop new and effective therapies.

The “Unknown” Disease

The second talk of the night was delivered by Elisabet Londos, a clinician and researcher stationed at Skåne’s University hospital in Malmö. Her long-time experience in interacting with patients at the neuropsychiatric clinic was reflected in her calm and gentle way of addressing the crowd. With pedagogic prowess she educated the audience on her specialty, Lewy Body Dementia (LBD). Straight away pointing out that the use of the word dementia was unfortunate, it meaning ‘without soul’. LBD, a disease in the borderland between Parkinson’s and Alzheimer’s disease, is a relatively new concept even though the pathology of Lewy bodies has been known to scientists for almost a hundred years. One of the hallmarks of Parkinson’s and Alzheimer’s, Lewy bodies contain a protein, alpha-synuclein, which
is toxic to neurons. Depending on where in the brain the alpha-synuclein damages cells you get different clinical expressions.

**LBD is an umbrella** term describing both Parkinson's disease dementia and dementia with Lewy bodies. The early symptoms differ, but reflect the same underlying biological changes in the brain. Over time, people with both diagnoses will develop very similar cognitive, physical, sleep, and behavioral symptoms. Elisabet went on to discuss the intricacies of diagnosing and treating the disease.

- **Today we cannot** visualize alpha-synuclein or Lewy bodies in animal models or in humans. But we can map the resulting lack of dopamine via imaging technology. Also, there is now intensive research to safely be able to measure alpha-synuclein in the spinal fluid, as Oskar mentioned. As of yet no drug exists that can affect the alpha-synuclein or the Lewy bodies. But the secondary deficiencies of signal substances can be treated in different ways. When we have established a patient's total picture of cognitive symptoms and perhaps hallucinations, it is important to find a balanced approach with different drugs. Then you can usually alleviate the hallucinations and not have to deal with so-called neuroleptics, which can be very inappropriate and dangerous for these patients.

**In a recent** painstaking study, Elisabet described how her office was cluttered with boxes filled with application papers, her research group teamed up with English and Norwegian scientists. The trial tested memantine for LBD patients. Originally a drug developed for Alzheimer’s, memantine proved an effective therapy also for LBD patients, many of them showing improved functions across the board. Even though the findings of the study were clearly exciting Elisabet Londos returned to the fact that when it comes to treating LBD patients there is no one-size-fits-all therapy strategy.

- **It is important** that LBD patients come to specialists that have the skill-set to set out an individual treatment plan. Often, patients are misunderstood because they do not act as the common dementia patient. Their memory and general intellectual ability is often good but they have difficulties in range estimation and spatial awareness. They can also be slow and show a general difficulty in getting things done. Most patients also have good disease awareness and do not tell others about symptoms like hallucinations or unpleasant dreams. We need to get better at catching these patients and offering them health care suited to their specific needs. There is a lot of room for improvement here in my opinion.

The café visitors listened carefully as Elisabet Londos talked about a disease unknown to most of them. Quite a few of the symptoms, though, seemed to be shared by some of the Parkinson’s patients in the audience. As the evening drew to a close the two researchers were bombarded with a battery of questions during the final discussion. The energy in the room seemed to only pick up as the sun began setting outside the windows of the Belfrage hall on the top floor of the high rise BMC-building.
On March 16-17th ScandModis held its 12th Scandinavian Meeting. The annual get-together has become the biggest annual meeting on Parkinson’s disease in Scandinavia. This year’s event brought together around 150 participants, focussing on the genetics, pathophysiology, current and future treatment of Parkinson’s disease and tremor diagnostics. Bagadilico partners Parkinson’s Movement brought a full squad to Stockholm to cover the event for their increasing following. Jon Stannford reports.

This year’s Scandmodis meeting “From Diagnosis to Management of Parkinson’s disease”, sponsored by Abbott and Orion and held in Stockholm 16-17th March, featured a range of top PD specialists from Scandinavia and beyond. Visiting the Scandmodis meeting for the first time (this was the 12th annual meeting) was a revelation. Although a short meeting, so much good science is packed into such a short space of time that this is fast becoming a compulsory meeting on the northern European circuit.

Parkinson’s Movement were grateful for the opportunity to attend the meeting and made good use of their time by interviewing several of the scientists. And it was a pleasure to see old friends from Bagadilico there too.

Everybody takes home different messages from any scientific meeting but, for me, there were a number of specific highlights. It was a pleasure to hear Thomas Gasser from Tübingen talk about the genetic aspects of Parkinson’s disease, an area that everyone acknowledges but far fewer actually understand. As with all good speakers, Gasser made a complex subject seems simple. He stripped away a lot of the mysticism in genetics, reducing the many risk factors essentially
to pathways - alpha synuclein aggregation and mitochondrial dysfunction. Pretty much all the known genes influence one or other of those two pathways. And more genes are being discovered.

I often feel that tremor, although one of the more obvious symptoms, is perhaps the least well understood. This misunderstanding often extends from diagnosis. In the early stages, Parkinson’s tremor can be difficult to distinguish from essential tremor when its amplitude is small. Jan Raethjen provided a direct approach to these distinctions. It was a welcome refresher course and a reminder of the skill set that is needed when walking along that shady boundary between art and science that is diagnostics.

The highlight of the following day, and again I make no apology for what is a personal choice, was Dag Nyholm’s advocacy for duodopa in the debate over the relative merits of duodopa and DBS for patients with advanced motor fluctuations. The clinical data looks strong but in the end patient opinions may well sway the decision beyond the science itself. Patients on the whole tend to favour invasive procedures rather less than their neurologists.

Sending a Message. The Parkinson’s Movement is a patient-driven organization with a growing influence in the global Parkinson community. At Scandmodis they continued their role as a key information nexus between patients and researchers.