Over the last couple of years Malin Åkerblom has been part of a groundbreaking effort to map the role of microRNA in the brain, research that has moved the needle on our understanding of these microscopic molecules that help regulate gene expression. Her thesis showcases three major discoveries made by the group, shedding light on adult neurogenesis and glial cell function.

In one of three publications presented in the thesis she highlights the key role played by microRNA-124. In experiments with mice she shows that miRNA-124 is overexpressed when the stem cell begins its transformation towards neuron. However, when turning off the miRNA-124 the stem cell did not become a neuron, instead it developed into a glial cell. A further understanding of these fine-tuning functions of miRNA may put us one step closer to unveiling the mystery of neurogenesis.

Discoveries made in the other two studies, exploiting the unique qualities of miRNA, help further explain neuronal development in the adult brain as well as creating a new method for studying the behavior of microglia, the brain’s guardian cells.
Making sense of a sprawling research environment is a kaleidoscopic challenge. How does one see the forest and not just scattered trees, individually reaching for the most sunlight? How does one create a common identity from which to build trust and future collaborations? It seems clear that part of BAGADILICO’s further progress is yet obstructed by a lack of involvement of junior researchers. The challenges presented by the scientific and physical spread of the network demands proactive solutions to bridge the divide. Steps towards such a solution is now being taken, by the junior researchers themselves.
Under the tutelage of leadership consultant Thomas Sewerin a group of PhD's and post-docs have identified different key issues to be addressed in order to make communication flow better, top-down as well as bottom-up.

Cristopher Dunning, post-doc and one of the driving forces behind the junior researchers’ initiative, explains.

- From a young researcher’s perspective, I think many of us have seen a lack of communication and information, really. Some junior researchers might not even know they are associated to BAGADILICO, or at least in what capacity they are and how they can fit in. Most of this we have identified as a top-bottom problem; that the information wasn’t flowing down to us. A lack of common identity has meant that a lot of people have felt that they belonged only to their research groups, and that they might even be rivals in the scientific arena.

Identifying flaws in the BAGADILICO information structure is, of course, only a first step. So, how does one go about breaking down these barriers in order to create a common identity amongst research groups with sometimes competing objectives? There is, unsurprisingly, no quick fix.

The quartet spearheading this initiative - Chris Dunning, Irene Sebastianutto, Ludivine Breger and Thomas Padel – have listed a handful of first objectives to tie BAGADILICO's young researchers closer together.

- First off, we need to get more information readily available on who is actually part of BAGADILICO, what research they do and what groups they belong to. We also need more information on the specifics surrounding the different technical platforms in order to make them more accessible to all members. Lastly, we need to create social platforms, virtual and physical, where we can meet and communicate about social events, scientific collaboration, jobs advertised, conferences, etc., says Chris Dunning.

Ludivine Breger believes that new platforms for social interaction cannot be underestimated as a tool for promoting scientific collaborations in the longer term.

- Without social contact there can be no scientific alliances. They simply go together. If you want to facilitate collaboration between research groups that don’t meet going about their daily routines, it’s going to have to start through social events.

An opportunity for setting the tone for a more inclusive BAGADILICO is this year’s retreat, held in December. The junior researchers group has now taken command of this task and will, together with other PhD's and post-docs, arrange the event.

It’s probably safe to say it will have a different spin than previous years’ retreats.

ARE YOU A YOUNG RESEARCHER WITHIN BAGADILICO? JOIN THE FACEBOOK GROUP - CLICK HERE

“All junior researchers might not even know they are associated to BAGADILICO or at least in what capacity they are and how they can fit in”, says Chris Dunning
In Parkinson's disease, the dopamine-producing nerve cells that control our motor function waste away. The research to develop new treatments therefore often aims to save or restore these cells. In a new study from BAGADILICO affiliates, researchers are attacking the disease from a different angle through early activation of the brain’s defence mechanisms. Stimulating a specific protein, the Sigma-1 receptor, sets off a battery of self-healing effects that slow the progression of the disease and restore the lost motor function. The results have been produced in studies on mice, but clinical trials with patients are not far off.

By stimulating the versatile protein, the researchers were able to activate a number of the brain’s defence mechanisms simultaneously. Various pathways between nerve cells were reinforced, inflammation subsided, the production of nerve-protecting substances increased and dopamine levels rose. The results, which have been published in the journal Brain, show a marked improvement in motor symptoms. The protein is already known to be active in a number of protective processes in the brain, but has never previously been tested in connection with Parkinson’s disease. Various studies linked to stroke and motor neuron disease have already shown positive results and a biotech company in the US will soon begin clinical trials on Alzheimer’s patients. Already having the substances that stimulate this protein approved for clinical trials is a major advantage, according to BAGADILICO Coordinator Angela Cenci-Nilsson, head of the research team at Lund University.

“It is of huge benefit to us that these substances have already been approved and tested on humans. It means that we already know that the body tolerates this treatment and clinical trials for Parkinson’s disease could theoretically start straight away.”

Making use of the brain’s built-in defence mechanisms is still a fairly unusual idea in Parkinson’s research. However, Angela Cenci Nilsson believes that the number of targets for future treatments is increasing as we learn more and more about the complex effects of the disease on different structures in the brain.

“The motor improvements we have seen are disproportionately high compared with the recovery of dopamine levels. We believe this is because we have been able to protect the brain against a series of indirect consequences of the loss of dopamine. For example, damage to a number of other neural pathways and surrounding cells that lose synapses. This type of treatment could help to repair such secondary effects.”

The treatment was shown to be significantly more effective when it was started early on in the progression of the disease, before the most aggressive stage of nerve cell death had begun. As a future therapy it would therefore need to be applied shortly after diagnosis in order to produce maximum impact.

“Now we hope to be able to find further evidence of the positive effects of this treatment, in order to begin clinical trials as soon as possible,” concludes Angela Cenci-Nilsson.