Newsletter 88

December 2012

bcn

BCN - SCHOOL FOR BEHAVIOURAL AND COGNITIVE NEUROSCIENCES

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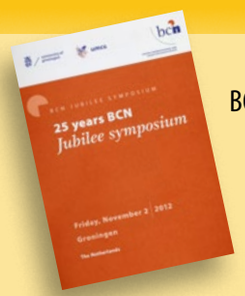

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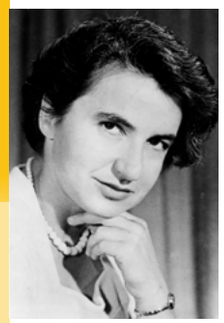


Interview Rosalind Franklin Fellowship with Dr. ir. Ineke Ganzeveld and Geert Jan Arends

You have the brains and ideas, but not the position to realise those ideas. What do you do? Well, if you are not already part of this university, and provided that you are a woman, you could apply for a Rosalind Franklin Fellowship (RFF) at the University of Groningen. The RFF programme offers excellent female scientists the opportunity to become a professor with your own research group if you do well in the programme. After a maximum of six years, achievements will be evaluated, and if all criteria are fulfilled you will be promoted to Associate Professor. With only twenty spots available every two years, it is a tough competition to become part of this prestigious programme. The good news is that the RFF is awarded with a Marie Curie COFUND from the European Commission. This means that in the coming RFF round, eight extra excellent scientists can get started.

One of the people involved in the RRF programme is Dr.ir. Ineke Ganzeveld. Ganzeveld is the secretary of the central RFF Committee. This committee checks if the correct procedures and criteria were followed by the RFF committees of each faculty by the nomination of candidates. They also evaluate every RFF round. Another person important for RFF is Geert Jan Arends. Arends is the project manager of European Grants and was involved in the application for the Marie Curie COFUND.

You might wonder why it is of interest to Brussels to finance a programme like the RFF. The European Commission aims to strengthen the research capacities of Europe. This combines well with RFF's opportunity of starting your own research group. Says Arends: "The impact of the Marie Curie programme was recently analysed. This analysis showed that there are not enough support mechanisms for people later on in their research career". So what does COFUND do to overcome this problem? As indicated by Arends: "COFUND co-finances existing fellowship programmes, this way the research capacity is increased and the European Commission does not have too much administrative trouble". Furthermore RFF fits well in with Brussels' wish to solve the gender gap. The gender gap is the lack of gender diversity at the highest levels of organisations. Arends explains: "The gender issue is a very big topic for the European Commission. You need to discuss it in all your pieces to show that you are working on it". However, a verdict of the Commissie Gelijke Behandeling (Committee of Equal Treatment), in the case where 12 associate professors were promoted to full professors, ruled that the University of Groningen discriminated based on gender (verdict no. 2011-198). You probably noticed that these two things contradict each other. Arends: "The main problem with applying for a COFUND was that the European Commission wishes to solve the gender issue, but is not allowed to support discriminating programmes". So how was this problem solved? Arends describes: "The solution we choose was to ask for funding to help the underrepresented gender to move up in the university". This means that if men were underrepresented in some fields of research, they are also invited to apply for an RFF, however Arends notes that "it turned out that in all fields females are underrepresented". Based on the verdict of the Commissie Gelijke Behandeling, the RFF

>> CONTINUATION OF THE INTERVIEW WITH DR. IR. INEKE GANZEVELD AND DRS. GEERT JAN ARENDS

programme decided to adjust their advertisements. Says Ganzeveld: "Advertisements are now phrased so as to be gender neutral, but we do primarily ask females to apply". With gender neutral advertisements and a critical look to all applications, men are not excluded anymore from applying, but Ganzeveld adds: "We do have the policy that if candidates are equally suitable, we prefer the female candidate".

Rosalind Franklin Fellows

So far so good, but you might want to know now why the RFF programme was originally started, what it is aiming for and if it reaches its aims. The RFF programme was originally started at the Faculty of Mathematics and Natural Sciences in 2002 by the former dean, Prof. dr. Douwe Wiersma. Ganzeveld explains why Wiersma started the RFF programme: "Wiersma noticed that a lot of female talent was lost, so five nonspecific tenure track positions with criteria for promotion were created". The advertisements were highly successful and a lot of applications were received from all over the world. The Board of the University recognised this success and the need for such a programme, and extended it to the whole university. This indicates that the RFF programme aims not to lose any talented people; RFF also tries to increase diversity at the top of the university by offering excellent female scientist a good position with good prospects.

> *At the moment we are at 20% female professors.*

One of the great advantages of the RFF programme is that from the beginning your application is evaluated based on your own achievements and future plans. Ganzeveld says: "One of the reasons that people apply for the fellowship is that they have the prospect of moving higher up into a permanent position based on their own efforts, which is not the standard at Dutch universities. Normally you are dependent on a position opening up and fitting in the exact profile". When you apply for an RFF, your application is sent to the RFF committee of your (future) faculty. They check if you meet the criteria [see textbox] and ask internationally recognised experts in your field of research to provide more information about you. Based on this information, they decide whether or not you are one of the most excellent candidates. If you are, you will be invited to give a presentation about your future research and are asked to meet your possible new work environment. After this stage, a new shortlist is created and based on this you can be nominated for an RFF. Ganzeveld adds: "The central committee checks if all procedures were conducted correctly and the candidates fulfil all criteria. Subsequently they advise the Board on the nominations, who can formally acknowledge the RFFs".

Criteria for Rosalind Franklin Fellowships

- A PhD with postdoctoral experiencew in different research institutions
- Publications in first-rate international scientific journals
- Experience in supervising research projects
- Ability to successfully compete for external research funding
- Affinity to teaching
- International recognition
- The potential to develop into a leader who guides and inspires

The criteria are tough; as a result it is not easy to get an RFF. Ganzeveld says: "It is sometimes the idea that every female who comes around will get a fellowship. That is absolutely not true and also impossible with only twenty positions every two years". So with its strict selection criteria, RFF wishes to show that they are not just a tenure track programme for female scientists, but rather that RFF is a programme of high quality for ambitious scientists. This is also confirmed by Arends, who says that "RFF is a prestigious programme, it delivers true quality and being a Rosalind Franklin Fellow can be seen as a true label of quality". When it comes down to the working reality, no real differences exist between 'normal' tenure trackers and RFF, however Arends adds that "as an outsider, I do notice some pride in the fact that people are RFFs".

However, being proud of being in a certain position is not a guarantee that something actually works. According to Ganzeveld the programme works well: "Most of the Rosalind Franklin Fellows have been promoted to the position of Associate Professor and stayed at this university. And the development of the current fellows looks promising". So the RFF programme works from the perspective of losing less talent. Furthermore, it also helps in increasing the diversity at the top of the university – as Ganzeveld notes, "at the moment we are at 20% female professors".

In conclusion, the RFF programme does really well. The programme was awarded with a COFUND to increase the number of positions for excellent female scientists to 28 in the next round. The diversity at the top of the university is increasing and less talent is lost. It is as Arends says: "The Rosalind Franklin Fellowship programme truly is a role model for other universities".

■ BY RENSKE BOSMAN

> HEAD OFFICE MATTERS

Meetings of BCN

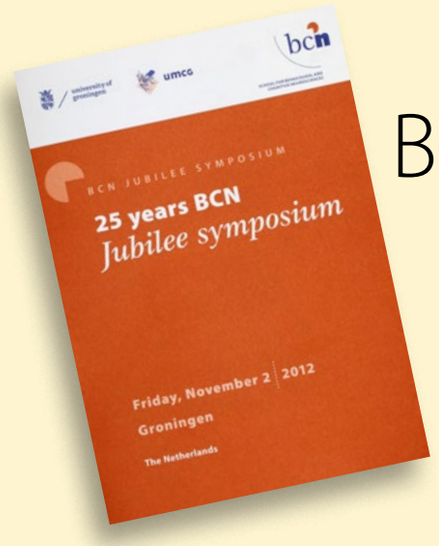
On September 27th Prof. John Duncan was awarded the Heineken Prize for Cognitive Science for his integral work on selective attention and general intelligence which has resulted in many important concepts in the cognitive neurosciences. At the same time Dr. Floris de Lange received the Heineken Young Scientists Award for cognitive neuroscience for his research on visual perception and motor imagery.

This offered BCN an excellent opportunity to invite both scientists to Groningen for a lecture. A symposium featuring both Heineken Prize winners was organized by Sander Martens, a former student of John Duncan. It was great to see that BCN showed much interest, and a large audience attended the symposium. Given that cognition research is an important topic in BCN, it is reassuring to see so many people showing up at relevant occasions. This made me conclude that we have to keep organizing meetings and symposia on our main topics.

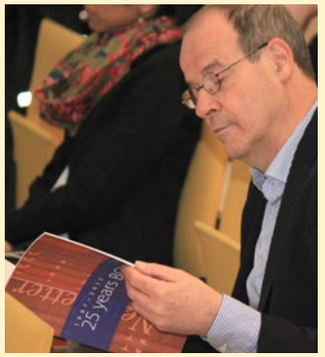
The general idea is that if any BCN member sees an occasion to invite an excellent scientist who could give a lecture which would be interesting for a large BCN audience, then please invite the scientist and organize a general BCN lecture, symposium, and/or master class.

This was the same policy followed for the BCN 25th anniversary symposium: We asked the five faculties of BCN to identify the “scientist of their dreams” for a lecture at the BCN 25th anniversary symposium. Since BCN took the financial responsibility for the symposium, the faculties were not modest with their proposals, and together we organized a very interesting and challenging symposium. This was an exquisite opportunity to celebrate the state of the art of our fields, and it was a pleasure to see so many BCN members at the event – and also at the party afterwards, where we celebrated the anniversary of BCN with great music and drinks. It was a fine toast to our successes, and an inspiring way to lead us into the future. I look forward to seeing all of you at the next BCN-wide event.

■ BY PROF. ERIK BODDEKE



BCN 25th Anniversary Symposium and Party



SPEAKERS


Sybrand Poppema


Erik Boddeke


Thomas Metzinger

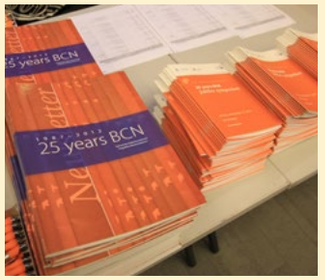

Markus Schlosser


Michael Spivey


Ben Maassen


Anne Bertolotti

>> CONTINUATION BCN 25TH ANNIVERSARY SYMPOSIUM AND PARTY



SPEAKERS


Peter Paul De Deyn


Craig Heller


Lambert Schomaker


Raja Parasuraman


Wiebo Brouwer


Katherine Gardiner (Chair)

>> CONTINUATION BCN 25TH ANNIVERSARY SYMPOSIUM AND PARTY

> INTRODUCING A NEW BCN MEMBER

Thomas Kantermann

It's 7.30 in the morning and the alarm goes off. I'm in shock, and a little angry. Why me? Why so early? Later, I try to wake up by drinking coffee, but it doesn't help. I get agitated by the noise coming from the radio that my girlfriend had turned on and feel miserable by the thought of cycling to the Bernoulliborg. At about 11.30 to 12.00, I finally feel awake. My mood improves and life brightens up. This is an ordinary day for me. According to dr. Thomas Kantermann, I'm not alone in my suffering. I am what is called a *late chronotype*. Apparently, more than 80 percent of the population needs an alarm to get up in the morning. Kantermann is currently involved in a project from the Chronobiology Department at the University of Groningen called 'OnTime – How to fix a (broken) "circadian clock', where he searches" for easily applicable tools and guidelines for people like me in order for us to get a better night's sleep. Let's meet him.

> Blue light is what our internal clock is synchronised to.

Kantermann studied Biology and Psychology as a minor, and from early on he started working on human subjects, which is uncommon for biologists. His supervisor at the time was not only a biologist but also a physician. So she had access to human material; brains and that kind of stuff. For his degree Kantermann studied the limbic system in Gerbils and thereafter he worked in an anatomy institute in Rostock, Germany. There, he worked with human fetal brains studying the limbic system and how changes in the amygdalae related to birth defects.

His Ph.D. in Munich deviated from his previous research topics and turned out to lay the path for his future career. There he studied shift work, and how the Daylight Savings Time transitions affect health and sleep, considering especially the impact on the internal circadian clock. Since then, he hasn't let the topic go.

In your Ph.D. thesis, you measured the effects of Daylight Savings Time and shift work on circadian entrainment. What were the outcomes of this study? What did you reveal?

The basic idea to do this was that shift work and Daylight Savings Time are transitions in clock time, which lack equivalent parallel changes in sun time. Especially Daylight Savings Time is nothing else than a social convention. The whole nation says: "Now we are going to work an hour earlier". So you don't change the sun time, we just pretend it is earlier now. The construct of Daylight Savings Time is sort of like shift work, but only a minor shift. Shift work is the extreme: A shift of eight hours or more. These are excellent cases for studying circadian entrainment under real life conditions.

So we have this internal clock in our brains, which synchronises our bodies to sun time, the change of light and darkness. This is called entrainment. And if this system is stable and properly functioning then we have high quality sleep and we're being in good health. And everything that interferes with this entrainment interferes with our health. Daylight Savings Time is a minor disturbance. Nevertheless, a huge number of people report that they feel uncomfortable in spring change and feel better after the autumn change.

How do you study it? Are there sociological or physiological measures or questionnaires?

We had a basic protocol at that time. We had daily sleep diaries and activity devices. People wore these for four weeks before and for four weeks after one Daylight Savings Time transition in both spring and autumn. Hence, we measured variables before and after each change. We were interested in how long it takes our participants to adjust to the new time/photoperiod. We know from previous studies – the Munich group has a huge database with entries from over a 100.000 people – that sleep time aligns with the progression of dawn. We are a bit earlier during summer and a bit later during winter. So like animals in the wild, we nicely synchronise to this photoperiod. Previous studies reported that people would adjust after one or two weeks to

>> CONTINUATION OF THE INTERVIEW WITH THOMAS KANTERMANN

summertime, so we monitored people a bit longer, and we found that especially later chronotypes don't even adjust after four weeks.

So there is a big difference in the effects between chronotypes?

Yes, so on the one hand we found that Daylight Saving Time does disrupt your seasonal adjustment. People undergo a change in the photoperiod across March – about one hour in dawn time – and due to the change in clock time we need to go through this whole process again. This is especially troublesome for later chronotypes. The common argument people have is: "Well, we have more light in the evenings". But what challenges late chronotypes is that it makes them a bit later, because more light in the evenings makes us later. Thus they need to get up early in the morning, while falling asleep later – a circadian vicious cycle.

> *Not sleeping well is like having too small shoes.*

If you'd be on a board that could change the policy on this, what would you do?

I'm still interested in the effects. For a scientist, Daylight Savings Time makes a brilliant population to study. We can investigate entrainment in large numbers of people. Health-wise I'd say, clearly, abolish it! It's ridiculous. There are plenty of studies that show there are no gains money-wise. We don't save electricity, rather we spend more energy. And there are studies that show increased risk of cardiovascular effects. It clearly appears to be a stressor. So I'd say abolish it, but as a scientist I say, keep it for a couple of years so we can do nice studies. However, it is a political decision, we have 25 countries in the EU and they'd have to vote with one voice, so it's an either/or: Either everyone says "abolish it" or no one, so there is practically no chance of a change. I am in contact with people from the European Parliament and learned that currently there is hardly any ambition among the EU countries to put this issue on the EU agenda. Clearly, we have to inform and increase awareness in people and push our politicians to take action.

From your Ph.D. onwards, what are the highlights of your career?

I had a wonderful time in the UK, at the University of Surrey. There I had the chance to follow up on my Ph.D. work, after I secured funding from a German funding agency (DFG). I was interested in the question: To what extent does the direction of shift work rotation affect cardiovascular risk? What are the prerequisites for good valid data to answer this question? And what can we say about the internal clock being involved in this? I had the opportunity to do this in the UK and Belgium. In Belgium there was a steel company that had those two rotations of shift schedules that I was most interested in, so this was quite a unique opportunity to study different shift systems, at the same location and in workers with comparable workload. What we found is that at this company, going forward in a fast changing schedule turned out to be worse for the cardiovascular system than working in a slow backward rotating system – results contradicting official shift scheduling guidelines.

This seems like applicable research. Have there been any policy changes since your results?

Not yet. It's hard to change people's habits and way of thinking once they have managed to find their (even if not optimal) way to cope with a certain situation, especially when something argues against the common regulations. But we will continue this work and I hope that it will be heard. I'm regularly invited to meetings where health physicians and employees come together. I can tell from these meetings that knowledge on the mechanisms is highly wanted and warranted. Hence, I am confident that these results are being heard and adapted.

So what are you doing now?

I've had the great opportunity to work on a STW-funded project called 'OnTime – How to fix a (broken) circadian clock'. My previous studies on shift work are important for a specific subpopulation, but OnTime is tackling a problem that concerns the whole population. OnTime is about decreasing everyday chronic sleep deprivation. There are 11 more OnTime projects, it is a Dutch consortium. There are other groups in Amsterdam, Eindhoven, Rotterdam and Leiden, working on particular aspects, like bipolar diseases, or working with mice models. All are concerned with getting more information on entrainment. We want to help individuals with different chronotypes, age and sex groups, and older and younger people to get a better sleep. We want to find simple solutions that are easily applied in a whole population. In addition, our work is supported by industry partners in the Netherlands.

Not sleeping well is like having too small shoes. If you go for a run with too small shoes, you can have your run but it's no fun and there are noticeable consequences to your feet. This is what late chronotypes suffer from, especially on their workdays. There is nothing wrong with their biology; rather it is the social system. We want to help people to get their proper shoes.

Are sleeping problems really a population-wide problem?

We know that over 80% of the population needs an alarm clock on workdays. When you talk to people and say that over 80% of the people need an alarm clock they will say "OK? Where is the problem?" This appears as nothing new, but from a chronobiological perspective, everyone knows the consequences just as you can feel it. There is nothing worse than sleep restriction. This is even worse the later your chronotype is. It is a mass population wide sleep experiment and we

>> CONTINUATION OF THE INTERVIEW WITH THOMAS KANTERMANN

want to help especially later chronotypes to get off their alarm clocks and to decrease their social jetlag. This social jetlag is especially pronounced in the adolescence between the age of 14 and 20 years, where we are biologically the latest chronotypes. Everyone knows how terrible school times can be. We know that children that age should get up at 10.00 or 11.00 to be fully awake and functional. In Germany, some schools start at 7.15 AM, so this is quite in the middle of their subjective night. Just as an example, there is good data showing that the later the chronotype, the worse your marks are. The more challenged you are. Such facts cannot be in the interest of any political system.

With what kinds of solutions has OnTime come up so far?

Firstly, we can give early types light in the evenings and late types more light in the mornings and less light in the evenings. Less light here doesn't mean putting them in the darkness but helping them receive primarily less blue light. The blue light is what our internal clock is synchronised to. We try to find ways to implement this easily. What you could think of, for instance, is sleeping with your curtains open. Or, you could dim your mobile phone and computer screens in the evenings (or even not use them at all). Modern displays are high on blue light. Studies are coming that sitting in front of a computer before sleep can prevent the increase in melatonin and thus delay your sleep phase. OnTime is a first-of-its kind mixture of applied science and finding out mechanisms.

Diet is another part of the equation. We work on this. We plan to have diet protocols. It could be simple things as not having a big meal before you go to sleep; giving proper signals to metabolism. There is some evidence from rodent work that feeding time has an effect but there is not yet conclusive data from humans. Also we will measure temperature. Our internal clock naturally adjusts to seasonal changes, but in our modern world we have air conditioning and heating. Because of these artificial temperature regulations, the environmental temperature is the same all year round, so we restrict ourselves from this seasonal entrainment. Bringing this back is one potent way, but by far the most important variable is light.

So what is your opinion on things like a melatonin pill?

They do work. For instance, if you want to prepare yourself for a trip to New York, it works nicely, but we wouldn't advise it for a whole population. This is not curing the problem, it is relieving the symptoms.

Back to BCN, why did you choose to join?

What I very much like is the interdisciplinary approach. I joined BCN in June this year, shortly after I started here at the RUG; I was kindly introduced by Deniz Baskent. I'm extremely impressed about how much is going on, the seminars, high quality of speakers, researchers and output. I am happy to be a member now and look forward very much to making interesting and lasting contacts.

And something more personal, what are your interests besides science?

Music! I play guitar and write songs. I used to play in a band, but I didn't manage to establish something here, due to lack of time (which I know is a bad excuse). Also, I like walking and cycling.

Do you still have a lot of time for these kinds of things or is science taking up all of your time?

Well, yes it is.

Do you mind?

No, not at all. My friends and family call me a workaholic. I can understand what they mean, but I myself feel to be in a happy position to have made my personal interests my work. I don't recognize my work as work. This is why I don't mind it. I like observing people, the psychology, just to study life. This has always intrigued me. Of course there are things that are connected to this work that aren't nice, such as paperwork, or too much time behind the computer, instead of spending the time with other people. But overall it's great fun. Especially being here in Groningen, where many pioneers of Chronobiology come from. It is an honor to continue the line of work here.

Do you apply the research to yourself?

As an academic you can more or less arrange your own sleep, fortunately. I am a rather early type. And a rather shorter sleeper, thus this fits quite well. But it certainly influences your lifestyle, to be aware of what entrains your system.

What would you advise late chronotypes like me?

If you have trouble getting up in the morning, try to get more light before noon. I mean real outdoor light. So take the bike instead of the bus. Have your lunch outside. Or if you'd wake up far too early, and can't enjoy the evenings, going to the cinema with friends or something, do it the other way around. Wear your sunglasses in the morning. Seek more light in the evenings. You can very nicely shift your sleep by playing around with light. If you like to find out more about your chronotype then just visit www.thewep.org. And, more about OnTime you can find under www.clocks-ontime.nl.

Thank you very much and good luck with your work.

■ BY ROBIN MILLS

> *You can very nicely shift your sleep by playing around with light.*

Brain power - beyond the age of 50

André Aleman is a professor of Cognitive Neuropsychiatry, and has published three books in recent years. His first book *Hallucinations - The Science of Idiosyncratic Perception* was published in 2008. In early 2011 his second book *Hersenspinsels* followed. This autumn, Prof. Aleman's newest book *Het seniorenbrein* ("The senior brain") was published, sold over 5000 copies within two weeks, and was recently acquired by a major publisher in Germany.

Please tell us a little bit about your current research.
My current research focuses on the cognitive and neural bases of apathy. I will investigate this primarily in schizophrenia, a disorder that I have been studying for 15 years now. But I will expand this to mild cognitive impairment (MCI) and depression. In both conditions apathy is also a huge clinical and scientific problem. In fact, together with the Department of Neurology, we have already started our investigation in MCI by preparing a research protocol that has been submitted to the medical ethical committee.

How does your book "Het seniorenbrein" ("The senior brain") connect to your research? Why did you decide to write a book about the aging brain?
I have been involved in research into cognitive aging from the time of my master thesis and onwards. As healthy aging is the primary research topic of the UMCG, I thought it would be good to take aging into account in my current studies. While I was taking stock of the literature, I thought that I might as well write a book about this topic for the general public. After all, aging is something that affects us all...

Is the audience for the book a scientifically oriented one or could it appeal to anyone who happens to come across your book in a bookstore? And when one is worried about getting older, do you think it can help to read books such as "Wij zijn ons brein" ("We are our brain") and "Het seniorenbrein" ("The senior brain")?
My recent book is for the general public, who need not have scientific training. I think it can indeed help people that worry about aging because they will be better informed. In addition, I show that besides decline, there are also cognitive functions that improve during aging, such as general knowledge and emotional stability. And I explain what can help you keep your brain sharp and healthy: physical and mental exercise.

Why should we read your book?
If you want to have an overview of key findings pertaining to cognitive brain aging, based on recent scientific research, this is the book for you.

What was the most interesting finding you came across when doing research for the book?
A study that showed that the brain's volume (grey matter) increased after a year of aerobic exercise training in elderly people, whereas it decreased in the control group which did only stretching and toning.

Do you think that research about the aging brain is something that needs more focus?
A stronger interaction between molecular approaches (e.g. regarding neurotransmitters such as dopamine) and systems neuroscience approaches (e.g., functional brain scans) would help the field move ahead.

Are there any plans yet to write another book?
Not really. But I do have some ideas and I think there is enough room for new books on the human brain, aimed at a general audience (even though a flurry of such books have been published in recent years). I especially think these books should contain more psychology, besides neuroscience, as this will really help us understand better how we think, feel and behave.

■ BY ANNIKA LUCKMANN
■ PHOTOS: SANDER MARTENS

Interview with Prof. John Duncan

First of all, congratulations for winning the Heineken Prize for Cognitive Science! Could you give us a brief introduction to your background and your research interests that lead to where you are today?

I did my first degree in Oxford in 1970-1973, jointly in Psychology and Physiology. This was a great degree programme, directly encouraging students to link mind and brain. Though very tempted by neurophysiology, through reading the work of Donald Broadbent and through my tutorials with Pat Rabbitt, I was imprinted on the (at that time) new psychology of information processing, reaction time and cognition. I went on to doctoral work under Pat, still in Oxford, and then decided on a postdoc with Mike Posner at the University of Oregon.

It's sometimes hard to appreciate just how many great ideas Mike Posner has had. In 1976 I went to work with him not because of the "Posner paradigm", which he was just inventing, but because of the previous Posner paradigm, same-different matching, which at that time was one of the central methods of cognitive psychology. During my time in Oregon, Mike was also just beginning to think that the time was coming for a real marriage of behavioural and neurophysiological studies of cognition. As I remember, I thought he was slightly crazy… but eventually I did get the message. Now I have spent my lifetime trying to bring together the psychology and neuroscience of higher cognitive functions – especially attention and intelligence.

Not only are you a very productive researcher, but you also manage to bridge multiple disciplines to address theoretical questions from different directions. What, to you, is the greatest challenge in multi- and inter-disciplinary work?

Probably not losing track of the details of the different experiments and methods – exactly how different methods work, what they can do and what their limitations are. Of course, this is not just the greatest challenge of interdisciplinary work, but of seniority - as the size of your research group grows, there is an inevitable struggle to hold on to all the important details, and the diversity of different methods and levels of analysis adds another layer of challenge to that.

Like any other researcher, you strive to share your findings and ideas with your peers in scientific journals. In addition to that, though, you also take the time to communicate the fascination of cognitive science to laymen. Do you think this is an important aspect of being a successful researcher?

I think it's important and that it should be done, though of course, it isn't for everybody. In my own case, I found that writing a popular science account of my work was valuable not just for communicating to the layperson, but for communicating to myself. Shaping things into a story for a popular book absolutely forces you to look at a bigger picture – at how the different parts of a topic or discipline fit together – and though I can't say my book is a bestseller, at least I learned something from it myself!

>> CONTINUATION OF THE INTERVIEW WITH PROF. JOHN DUNCAN

Your 2010 book "How Intelligence Happens" targets non-scientists and discusses the aspects of intelligence from different perspectives. You give a very accessible introduction to the fields of classic experimental psychology à la Spearman, brain imaging and the complex involvement of the frontal lobes, problem solving computer software and the importance of cognitive enclosures, and cognitive biases that defy our intelligence. Which of these disciplines do you expect to have the greatest potential to tell us more about human intelligence in the next ten years?

In the shortish term, I hope that the critical thing will be no one discipline, but their integration. After two decades of cognitive neuroscience, I think the time is approaching for serious theoretical integration of mind and brain stories – for example, how critical cognitive events unfold through the dynamics of neural populations. What we need is a marriage of the theoretical sophistication of information-processing models with detailed neurophysiological data.

In the medium term, I feel sure that some time, all that we do in cognitive neuroscience will be supplanted by the invention of a method for accurate, real-time measurement of localized neural activity in the human brain. When that happens there will be no more fMRI and measurements of haemodynamics, no more EEG/MEG and attempts to reconstruct neural activity from measurements at the surface of the head, probably much less animal neurophysiology except for experiments requiring intervention in network activity. It's surely a matter more of when than if… and when it happens, everything will change overnight.

> *The great excitement of research is that you never know where the next critical discovery will be.*

In your book, you describe the impressive feats of human intelligence and then show how we are all susceptible to cognitive biases. Even though we like to see ourselves as rational beings, we often fail to be rational. Could you say that intelligence is the capacity to overcome cognitive biases?

Well, it must certainly help! Though in the book I also argue that some of the same mechanisms that give us our intelligence also give us our biases. To move clearly from one step to the next in a line of thought requires focus, with only one small part of a problem considered at once. But focus also means that we find it terrifyingly easy to leave important information out of consideration, basing beliefs and actions in politics, religion, emotional disputes just on the half of the story that fits our biased or preconceived view.

I was surprised that you ended your book by bringing up the well-known philosophical question of whether the human mind is capable of understanding the human mind. Do you think this will ever be a practical problem or merely an interesting thought experiment?

In principle I don't see any conundrum in the question of a mind understanding itself. Why would it be hard for an information-processing device to represent principles and details of anything, including itself? What I meant to say at the end of the book is that it seems certain there are limits to our ability to conceive and understand all things – not especially ourselves. If we look at any other animal from our perspective, the limits of their realm of understanding are always obvious. Is it likely we do not have such limits ourselves? And will we always lack the broader perspective that would be needed to see them?

What projects are you currently involved in and what is the research question that currently captivates you most?

The great excitement of research is that you never know where the next critical discovery will be. This is all the more true when you are looking at different levels simultaneously – at the behavioural experiments of cognitive psychology, studies of patients with brain lesions, neuroimaging, single cell physiology. I'd love to think that something exciting will come from our studies of neural population dynamics in the monkey frontal lobe, but it could be our new attempts to teach adults and children how to "bullet-point" their thoughts and improve mental organization. Or something totally unexpected, like a recent fMRI experiment from one of my students suggesting how large-scale brain networks switch the whole mental landscape – an experiment I never for a moment thought could work. It was interesting that, in their presentations of their work, several of the 2012 Heineken laureates emphasized this critical aspect of scientific discovery – pursuing something sensible but always with your eyes open for the significance of the totally unexpected. Given the preliminary state of our own field, I think we can expect the unexpected indefinitely. Thank heavens!

■ BY FLORIAN SENSE
■ PHOTOS: MICHIEL HOOIVELD AND SANDER MARTENS

Interview with Dr. Floris de Lange

Congratulations for winning the Heineken Young Scientists Award 2012 for Cognitive Science! How does it feel to be awarded with such a prestigious prize?

It is really a great honour! I had first heard about the Heineken Awards when Stanislas Dehaene, my postdoc supervisor at the time, and my all-time hero, won the (senior) Heineken Prize for Cognitive Science four years ago. I already knew about the Heineken prizes and how prestigious they were, so I was really excited when I heard I was selected for the award.

According to the jury, "Dr. de Lange displays intellectual depth, and an understanding of virtually all areas of cognition, making him one of the most talented cognitive scientists currently working". How does it feel to get such a compliment? And how do you see yourself?

Of course these are very flattering words. And it is nice to get such a "pat on the back", being told that you're doing a good job. I suppose I see myself as someone who is curious about how the brain works, and how it implements cognition. I get most excited about trying to find out principles of neural function that apply to multiple cognitive domains. This is one of the reasons why I have been working on seemingly different topics: perception, action, mental imagery, decision-making. It would be great if we could connect these domains with a set of unifying principles. My working hypothesis is that each cortical area is trying to predict its input. Hopefully this could be one of those principles.

What goals do you want to achieve with your research?

Thinking really big, I of course want to find out how the mind arises from the interactions between neurons in different brain areas. More specifically (and modestly), I currently zoom in on perception, and perceptual decision-making. All the percepts we have are ultimately the product of decisions taken by the brain (which is perhaps most obvious when studying bistable stimuli, where the decision alternates between two interpretations of the stimuli). Understanding how these decisions are made in cortical ensembles, and how they are governed by both bottom-up factors (external stimuli) and top-down factors (what we find relevant and what we expect), are the current goals of my research.

> *Honestly, I think being a researcher is the best job in the world.*

What kind of projects do you plan to work on in the near future?

So far I have mostly looked at how "priors" (expectations that we have about the world) influence sensory processing. Here we experimentally induced "priors" by implicit learning of regularities (stimulus A is mostly followed by stimulus B). In the near future, we are going to look at multimodal perception, to see how unimodal signals are integrated. Here, information in one modality (e.g, vision) could also be a "prior" for

>> CONTINUATION OF THE INTERVIEW WITH DR. FLORIS DE LANGE

sensory processing in another modality (e.g., auditory). Somewhat similary, we're investigating whether our language system also constrains and biases visual perception. This is an old idea (known as the Sapir-Whorff hypothesis), which is currently being revived. I am also getting more interested in consciousness, and the extent to which it is necessary for predictive processes to occur.

> *Be critical, but don't let it spoil your enthusiasm.*

Are there also things that you do not like about being a researcher?

Honestly, I think being a researcher is the best job in the world. Why? Because we have (almost) total freedom to ask the questions we're interested in, and then to try to find the answer to them. That said, there are of course potential drawbacks to a career in science. For example, it is not a 9-to-5 job. This of course doesn't mean that you always have to work, but it is a bit like having your own shop: you want it to do well, and sometimes something needs to be done in the evenings or on weekends (for example, when scanner time is available). This can be a drawback. Also the publication pressure and the fact that science is sometimes a rat race are potential drawbacks, e.g. when it pushes scientists to compete rather than collaborate. Luckily the Donders Institute in Nijmegen, where I work, is a very sociable and collaborative place. I learn a lot from my collaborators and students every day.

What you achieved already is really impressive. Do you also have the time to do things that are not related to doing research? (In other words, what do you like to do in your leisure time?)

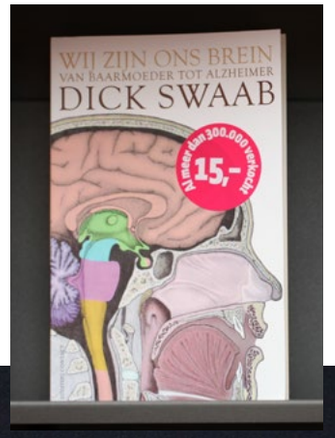
Luckily I still have time to do other things as well. I like making bike tours, cooking, exploring big cities, occasional photography and watching French or Italian movies from the 60's and 70's. I used to also make wine and have chicken, but those are things that I have given up on.

Is there something that you could recommend to the students of the BCN?

That's a difficult one. Perhaps: Be critical, but don't let it spoil your enthusiasm. Dare to be creative in finding new ideas, but also be rigorous when translating them to actual research. For me, it took time to find such an "actor/critic balance". Also, a book that is more than 100 years old but which I still warmly recommend is "Advice for a Young Investigator" by Ramon Y. Cajal, in which he discusses the "do's and don't's" of being a researcher. It's still surprisingly accurate!

■ BY RICCARDA PETERS
■ PHOTOS: MICHIEL HOOIVELD AND SANDER MARTENS

"Wij zijn ons brein" ("We are our brain")
Discussion with Prof. Dick Swaab



In September, the DwarsDiep Groningen invited several speakers to debate the position of Dick Swaab that "we are our brain". Next to Swaab himself, the retired Professor of Psychiatry Herman van Praag, Professor of Cognitive Philosophy Marc Slors, and the journalist Asha ten Broeke participated. Swaab sold about 350.000 copies of his controversial book "Wij zijn ons brein" ("We are our brain"), which was published in September 2010. The popularity of the topic was supported by the fact that the tickets for the debate evening were sold out after only three weeks and a screening of the event was also sold out in no time. I myself had read the book two years ago and was excited to see Dick Swaab discussing the book in person.

The evening started with a short introduction of the topic by Hans Harbers who led the debate. Then Dick Swaab was asked to give a short presentation about his view and beliefs. He gave a number of examples on how the brain can trick us into believing we perceive something that is not actually there, like a sailor who only sees water for days and suddenly believes he sees an island with a city on it. This phenomenon is due to under stimulation of the brain, and is comparable to the sensation of a high pitched beeping sound in Tinnitus.

Swaab then turned to the topic of religion, since he and Van Praag had been discussing it quite often in the past and cannot seem to find a common ground. Swaab is an atheist while Van Praag is religious and had apparently called Swaab abnormal in the past. Swaab explained that in today's society, fewer and fewer people believe in God, and that it is even less in academia. One of his arguments was that fewer than 1% of Noble Prize winners believe in God and therefore, it is anything but abnormal to be an atheist. Van Praag countered this argument by saying that people in academia just do not feel comfortable saying they do believe in God due to strong criticism. The religious debate went on for a while until Marc Slors was invited to the podium. (More about Slors' views can be found in the interview with him below.)

Finally, Asha Ten Broeke, who is a young journalist writing for both scientific and women's magazines, was invited to join the discussion. Her contribution focused on gender identity, and was against Swaab's claim that gender identity is already determined in the uterus, which he states in his book. Ten Broeke supports the view that it is not your genes but your environment that determines your gender and identity. Swaab countered Ten Broeke by saying that the whole nature or nurture debate is long solved by the fact that it is an interplay between the two that determines gender and other factors such as personality and sexual preference.

After all the debating, some questions from the audience were answered, and the evening was closed by Habers. I really enjoyed myself during the debate but thought that the evening was a little too broad as the speakers were talking about so many different topics, such as free will, religion, gender identity and of course the statement that we are our brain.

■ BY ANNIKA LUCKMANN
■ PHOTOS: ARINE MENTINK AND SANDER MARTENS

"Wij zijn ons brein" ("We are our brain") continued: Interview with Prof. Marc Slors

Please tell us a little bit about who you are and why you were part of the debate evening.
I am professor of philosophy of mind at Radboud University; I work in the Philosophy Department, but I also teach in and collaborate with the Faculty of Social Science, where I teach psychology students and students of cognitive neuroscience. I work on various aspects of the mind-brain problem, but the past couple of years I have focused on social cognition. About two years ago, I became irritated with the fact that many neuroscientists gained public popularity by defending poorly developed philosophical positions, especially on free will, without knowledge of the relevant literature. At the same time I was frustrated by the fact that philosophers were entirely defensive when it came to free will and hardly willing to see that the neuroscientific data are, indeed, very interesting. I decided to write a book for a wider audience on this, which was published in April this year (Dat had je gedacht: Brein, bewustzijn en vrije wil in filosofisch perspectief, Amsterdam: Boom). I take it that that is why I was invited.

You yourself state that you do not see a problem in the title of Swaabs book "Wij zijn ons brein". Why do you think so many discussions evolved out of this title and why do you not think that it is an issue to say that we are our brain?
Opposition to the idea that we are our brain stems largely from the fact that the brain is a biological organ. If we are our brains, this can easily be taken to mean that we are nothing but biological beings – i.e. that the mental, moral or spiritual dimensions of our beings are reduced to biological, chemical, and ultimately physical ones. In order to avoid such a reduction, many believe the only way to go is to admit that we are more than our brains. The problem here is twofold: 1. many people, including neuroscientists, only have a vague notion of what 'reduction' means and 2. the idea that we are 'more' than our brains is usually implicitly interpreted in quantitative terms, i.e. as implying that there is something like an immaterial soul. By getting both issues straight, we can save our mental, moral, and spiritual dimensions – and our free will, for that matter – within the framework of a scientific worldview. I find that important because I do not want vital notions such as free will to be dependent upon a worldview that is contested by science.

1. Reduction can be understood in terms of the 'stuff' minds are made of – soul-stuff or material stuff. But a much more interesting notion of reduction (the one developed in the philosophy of science) is in terms of translation: I can reduce my mentality, my freedom, my spiritual dimensions only if I can translate a mentalistic description of myself in neuroscientific terms. The first kind of reduction is implied by 'I am my brain'. But not the second. In fact the second kind of reduction is a scientific myth: no serious scientist will claim that we can translate statements about what I believe, desire, and value entirely and without loss of meaning in neuroscientific terms. There are very good scientific and philosophical reasons to doubt whether this will ever be possible, even in principle.

2. Hence, I can admit that I am my brain and still reject that I can be reduced to biological processes. Thus it is not necessary to think that we are 'more' than our brains in quantitative terms (i.e. in terms of thinking we are immaterial souls). We are 'more' than our brains in qualitative terms.

You say that, free will is about making choices. What exactly do you mean and are those choices conscious?
For me to be able to act out of free will, I need 1) options to choose from and 2) I need to pick the option that I want to pick (i.e. I do not want to choose randomly or under peer pressure or as a result of psychopathologies). 1) is the domain of the philosophical free will vs. determinism debate. 2) is what neuroscientist talk about. They claim that I do not pick the options, rather my unconscious processes or genes are responsible for my choices. What I think is wrong in this line of reasoning is that I am also my genes and my unconscious processes, not merely my consciousness. Choices made unconsciously are also my choices. Whether or not they are free choices depends not on whether they are caused consciously, but on whether they fit my identity – my values, my longer term plans, my ideals.

On the evening of the debate, there was a lot of discussion about religion. Are you yourself religious?
I used to be religious but now I am no longer. In general I dislike fanaticism, both religious and atheist fanaticism.

> *I am also my genes and my unconscious processes, not merely my consciousness.*

■ BY ANNIKA LUCKMANN
■ PHOTO: FLEUR JONGEPIER

Interview with a VENI and Marie Curie Integration grant winner

Dr. Tjakko van Ham recently won two grants: A VENI grant and the Marie Curie Integration grant. The VENI grant provides a maximum of €250,000 to researchers who received their PhD fewer than three years before applying for the grant, and is intended by the NWO to support talented researchers at the beginning of their career. The additional Marie Curie Integration grant is supposed to facilitate the research projects of researchers returned from abroad, and provides an additional €100,000 over a four-year period. Winning these grants has put van Ham in a good position to pursue his research interests, and the BCN newsletter used this opportunity to talk to van Ham to find out more about what these interests are.

Please introduce yourself. What's your background and how did you get to where you are now? Where do you want to go from here?

My name is Tjakko van Ham, I was born in Arnhem and studied at the University of Utrecht (fundamental biomedical sciences/biology). My first research work in the lab (at the Hubrecht Institute), made me decide to go abroad to work for a biotech startup company in San Diego about 10 years ago. Working and living in San Diego really blew my mind, the science is fantastic. Although I worked at a company, it was run mostly by academics who held faculty positions at UCSD, and it felt like a research lab. I went to seminars and conferences across the street at UCSD, the Salk Institute, or the Scripps. You'd see hot-shot professors surfing at six in the morning before work. After this experience, I figured that doing a PhD programme was the next logical step for me, and although I considered getting my PhD abroad, it seemed to me that doing a PhD project in the Netherlands would be best. After that, however, I decided I would definitely return to the US as a postdoc. I wanted to work on functional genetics in the zebrafish, but through a change of fate I finally ended up working on functional genomics in C. elegans. The project involved C. elegans genetics to find new genes in age-related neurodegenerative diseases, with Ellen Nollen (at the time a postdoc in the lab of Ronald Plasterk). Halfway through my PhD, she took an offer from the UMCG for a Rosalind Franklin position and I decided to join. When I was close to finishing my thesis, it was time to choose and visit some labs in the US to join as a postdoc. I had to make a very tough call in deciding on an offer from a well known lab specializing on Alzheimer's and Parkinson's; joining this lab would have been the most logical choice from a career point of view. I finally chose a position at Massachusetts General Hospital in Boston (USA) in a lab, ran by a chemist who pioneered zebrafish chemical screening a little over 10 years ago. Genetics in C. elegans is amazing, but when I heard about his chemical biology approach in zebrafish and had a good experience visiting his lab, I made up my mind. Working in that lab indeed was quite special. I learned more than I could imagine, I had complete freedom in choosing my projects and scientific direction, and I had a great time. In the three years I stayed there, I did the work that forms the basis for my current line of research. That's basically how I got where I am now. What's next? Of course I have a lot of plans, but I bet it's going to look different than I can imagine now a few years down the road. It would be nice if in the next three years I would find out some new biology of how the immune system interacts in the brain, and some small molecules with neat phenotypes, to work out pathways controlling immune responses. On a little longer timeline, I hope I can link some of these findings to mechanisms of brain disease.

What exactly have the grants be awarded for? Can you tell us more about the funded project?

A VENI grant from the NWO is a personal grant to promote scientific talent. They are awarded within three years of obtaining a PhD, and grantees can freely choose

>> CONTINUATION OF THE INTERVIEW WITH A VENI AND MARIE CURIE INTEGRATION GRANT WINNER

> *Within the first week of development zebrafish share the immune cell lineages we have.*

their subject. The other grant I received is a Marie Curie Career Integration Grant. This European grant allows researchers who worked abroad to continue their own line of research in their home country for four years. The projects are very similar scientifically, although the Marie Curie grant is focused more on the potential for collaborations and networks, local as well as (inter)national etc. The goal for both grants is to investigate how different immune responses, in particular cells like astrocytes and microglia but also macrophages, affect pathogenic processes in the brain. We only came to know quite recently how versatile and important these cells are. It is clear they are involved in many brain diseases, but the circumstances under which they are protective and when they actually can cause harm are unclear. To study this I wanted to really observe how it happens in living brains, and zebrafish are perfectly suitable to do this. Another important reason to use zebrafish is the possibility of carrying out small molecule screens. The lab where I spent the last three years, Randall Peterson's lab at Massachusetts General Hospital in Boston, specialized in chemical biology and drug discovery in the zebrafish. Zebrafish larvae are small enough to swim around in a well of a 96 wells plate, and can live for days surviving on nutrients supplied by the yolk sac. They absorb chemicals dissolved in the surrounding water very well. By using optically transparent plates and automated microscopes you can screen for many different phenotypes, screening up to a thousand chemicals per week. There are many recent examples where this approach yielded small molecules affecting phenotypes ranging from cancer to behaviour. Some of these drugs are very close to testing in clinical trials, a remarkable achievement if you realize they were discovered within the last five years. Part of my proposal is to use small molecular screenings to find new drugs that alter immune cell responses in neurodegeneration.

If I understand correctly, your research involves microscopy in living zebrafish. Can you tell us more about your technique and the insight you hope to gain from this line of research?

Zebrafish develop rapidly; within a day after fertilization they have blood circulation and a functioning innate immune system. Within the first week of development they share the immune cell lineages we have, such as neutrophils and macrophages, but also microglia and astrocytes, very important in the brain. Because they develop an adaptive immune system and T cells a little later, we can use this to distinguish between effects of the two. Because the young fish are transparent, which lets us use different fluorescent proteins to mark different cell types, you can really observe what these cells are doing in the brain, how they interact. To do this we use confocal and multiphoton imaging. Using this "video-microscopy" we can image straight into the brain of anaesthetized young zebrafish without harming the animal. I guess it is not that different from how people who first studied brain diseases literally a century ago spent many many hours behind the microscope peeking into brains, people like Alois Alzheimer. But instead of looking at fixed brain slices, I'm looking at movies of the different cells involved. So in a way a crossover between Ilya Mechnikov, who first observed phagocytic cells alive, and looking at diseased brains.

What I hope to learn is to be able to distinguish different responses of immune cells occurring when brain cells die, and how these contribute to recovery or progression of the disease process. I also plan to carry out drug screens in zebrafish to find small molecules that control such responses. Such drugs will help understand these responses and the effect they have, by acting as a handle to control them. Ideally they would serve as candidate drugs to ultimately treat neurodegenerative diseases. One last thing is, once you have a drug and a cool phenotype, you can identify the target and figure out the molecular pathway. This can take many years, but there are some recent examples from zebrafish where this was done within only a few years…

What do you think sets your project apart from others? What do you think convinced the committee to fund your project?

Zebrafish are more and more often used as a model for biology and medicine, and I'm sure I wasn't the only one including zebrafish in my proposal. I think for me it was important that I had a strong proof of principle; my work from the Peterson lab formed the groundwork for my proposal. Another thing is that in stem cell research and medical fields such as cardiology, immunology, and cancer biology, zebrafish are an established model system with a strong track record of important findings. What I'm doing is a little more novel, but I'm not sure if this worked to my advantage. Figuring out what immune responses do in the brain and whether and how they contribute to disease is still a big question. It's hard to tell why my project was picked, since you don't know the contents of other proposals. Initially, your CV is important of course, for example the papers you published. In fact if you don't spend time working abroad you can really reduce your chances, something I don't always agree with. In addition, you must be convincing and really believe in your research questions and approach. But in the end they want to see potential to bring your own, original line of research to the next level.

■ BY FLORIAN SENSE

Interview with a VIDI winner

In July 2012, the Dutch Organization for Scientific Research (NWO) awarded 94 researchers a VIDI grant. The VIDI grant gives excellent researchers the opportunity to set up their own research line and build up their own research group. One of these researchers was Dr. Reinoud Gosens from the Department of Molecular Pharmacology.

> *Seeing that asthma is a major health problem and that some of my family members are affected by this illness, I decided to choose this project.*

Reinoud Gosens completed his studies in Pharmaceutical Sciences with honours at the University of Groningen and received his PhD from the same university in 2004, for work done investigating the role of airway smooth muscle phenotypic plasticity in airway remodeling in asthma. After being a postdoc fellow at the University of Manitoba, Canada, he was awarded the Marie Curie Outgoing International Fellowship from the European Community in 2005, and the VENI fellowship in 2008 for further research on this topic in Groningen. Currently he is an Assistant Professor in Translational Pharmacology at the University of Groningen, where he focuses on mechanisms that regulate structural remodelling in asthma.

First of all – congratulations for winning the VIDI grant for your project. Could you tell us something about your research?

Thank you very much for the congratulations. The research that I do is focused on obstructive lung diseases. The main cause of these diseases are airflow obstructions that lead to shortness of breath and other symptoms these patients experience. Generally, there are three causes of symptoms: narrowing of the airways caused by constricted muscles, an inflammation of the airways and, as a result of a chronic inflammation, the so-called remodeling of the airways. During this process the inflammation-caused wound becomes fibrotic and thickens, which leads to scar tissue formation within the airways. This results in persistent symptoms and chronic progression of the disease. Currently, these patients are prescribed anti-inflammatory drugs which relax the muscles and open the airways. At present there is, however, no therapy available to cure this chronic remodeling. This is not only a problem for lung diseases, but for most chronic diseases.

My research is focused on the mechanisms that regulate these chronic responses. I am trying to identify which pathways direct these types of pathological responses and whether these pathways can be used for drug intervention, so that it might be possible to stop the progression of the disease and even reverse some damage that has been done. To achieve this, I am focusing on a group of developmentally regulated genes. Some literature suggests that the same genes that coordinate development in utero become reactivated during tissue damage. It was always believed that these genes were switched off after birth, but it has now become clear that these are necessary to put the normal architecture of the damaged tissue back in shape. But somehow this repair is not adequate during chronic disease processes.

So, the remodeling of the tissue is drifting into the wrong direction.

Yes, exactly. Personally, I see remodeling as an inadequate repair response. And this inadequate repair response happens because there is tissue damage on top of earlier tissue damage, which does not allow the wound to repair itself. Another explanation could be improper lung development in utero, which could impair lung function and repair later in life. For example, a mother who smokes exposes her fetus to the risk of impaired lung and airway development. This increases the risk of developing asthma, which might be explained with these developmental pathways.
So, the basic idea behind my project is to find out where in these processes the developmental pathways play a role and whether we can try to modulate them in order to improve the outcome.

That sounds indeed very interesting. Can you briefly explain how you try to answer these questions?

The research project has three aspects. First, we are trying to mimic this type of injury in cell culture studies in vitro. We are able to do this by taking those cells that are responsible for the repair, injure them, and then look at whether these developmental pathways are reactivated. The strength that we have in a cell culture system in terms of modulation is very high. Based on these models we might have an idea of which developmental paths or genes are most important.

>> CONTINUATION OF THE INTERVIEW WITH A VIDI WINNER

The second component of the project are in vivo studies, which we do mostly in mice. We aim to identify those genes that are important for the disease and for the functional repair both in mice that show structural remodeling, and in mice that have impaired lung development from birth on. These processes are similar to those inhumans. On the basis of this, we will do functional in vivo studies. We will try to modulate the expression of one particular gene that might be important by using transgenic mice. This technology is very advanced nowadays, so that you can knock-down or over-express a certain gene in a specific kind of tissue or even at a particular time point. This is quite a necessary step, because without it one would not be able to gain the insight in the relevance of the gene that you need. Through a collaboration with the Pulmonology Department at the UMCG we are then able to validate the genes that we find in tissue specimens of asthma patients.

The final step is to take the information from the transgenic model to experimental drugs. Here, we will modulate the repair responses simply by giving the mouse a drug via inhalation to see if the processes are returning to normal.

The ultimate goal of this project is to identify drug targets. We will try to come up with a few drug targets that are important for tissue repair in the lungs, and these may provide clues for drug development.

I saw that you started your PhD here in Groningen in 2000, during which time you already looked at processes in the airway smooth muscles in asthma. What made you enter this field, and where does your fascination about this topic come from?

First, when I started to study pharmacology, I was most attracted to the understanding of how drugs work and, consequently, mainly interested in the pharmacology courses. Quite soon I knew that I wanted to do my research project in this department and received a few project proposals. Part of the research in this department was done on lung diseases. Seeing that asthma is a major health problem and that some of my family members are affected by this illness, I decided to choose this project. I enjoyed this research so much that I decided to continue with my PhD in this field when the opportunity arose instead of working as a pharmacist, which would have been another option.

So, you grew into the topic.

Yes, I was not "predetermined" to do asthma research. It is a sort of combination of events that just happened. If there would not have been asthma research in this department I probably would have done something different.

Why do you think were you considered for the VIDI grant? What makes your research so valuable?

I like to believe that it is because of the research itself, but I think it is because of several components. First, I think that the aspect that early life exposures affect the susceptibility of diseases in later life generally is a very active area of research that attracts a lot of interest. Even though this project is specifically focused on asthma, the aspect of the developmental pathways is a very general phenomenon that can be applied to several diseases. I think that the committee appreciated that the research can be applied to multiple fields. Second, the project also has to be feasible. It is important to lay out the way you will approach your goal. A third component is, of course, the person behind it. NWO wants to see a person that already has a solid background in that particular field, who already started their own research line, and who has the potential to use this grant to not only build a research programme but also to develop more active research. That does not mean, of course, that the projects that are not chosen are not important. You have to be realistic and consider that the differences between the applicants are very small. The combination of a grant with potential and a researcher with potential are the factors that are looked at.

So, on the basis of that, do you have any specific advice for people who want to apply for funding?

For the grant itself, make sure that is has a very clear focus. Start with B and then go back to A. In other words, in every sentence that you write, the idea of the grant has to be worked towards that endpoint. A very easy pitfall is to take too many side paths. Make sure your project is feasible and make sure you have it read by your peers. They can always give you advice on things that you may not see. For personal development, it is essential in my area of research to continue your postdoc career abroad and gain demonstrable international experience. Especially for a VENI grant, this personal development is important and the CV is a very strong component of the overall judgment. Also try to find something that is new and not a logical extension of your previous research, but nevertheless find something that suits you. A last piece of advice is to attend the workshops organized by the University and NWO. They do provide you with very specific information on how you should write a grant application.

> *Start with B and then go back to A.*

Thank you, I will definitely keep that in mind for future applications. Thank you very much for this interview.

■ BY CHRISTINA CORDES
■ PHOTO: CHRISTINA CORDES

First medical Summer School on the 'Ageing Brain'

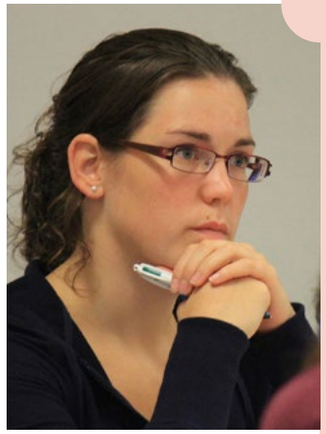
From Sunday, August 26th to Saturday, September 1st, 2012, the NEU4EU Network 'Ageing Brain' organized the first Tranlational Neurosciences Summer School 'Ageing Brain' at the University Medical Center Groningen (UMCG). The theme of the course was on neurodegenerative diseases, novel detection and imaging technologies, and treatments developed for these diseases. Lectures were given by scientists from the University of Groningen and Twente (the Netherlands) and from five other foreign universities, including the Universities of Ghent, Göttingen, British Columbia (Vancouver), Copenhagen and Uppsala. Twenty research master and PhD students from more than 10 countries from all over the world participated in the 7-day long course.

Besides an interesting educational programme, a committee of six medical students from the UMCG organized an exciting social programme that showed the participating students many aspects of typical Dutch student life. The course was concluded with a visit to the legendary city of Amsterdam.

■ BY MICHIEL HOOIVELD
■ PHOTOS: MICHIEL HOOIVELD

Participants of the NEU4EU Translational Neurosciences Summer School 'Ageing Brain'

NEU4EU Network Ageing Brain



Sietske Berghuis

'Besides the educational programme, there was plenty of time for the nice social programme. The Summer School started on Sunday, August 26th, with a welcoming party at the guesthouse with a lot of pizza and a quiz on medical facts, neurology, and of course facts about Groningen. At the end of each day, after listening to several lectures and working on assignments, students and lecturers went out for dinner. We enjoyed delicious dinners in different restaurants in Groningen. We ate pancakes on a boat and had a nice BBQ together. On Tuesday we went out to see fireworks celebrating 'Bommen Berend'. Additionally, we climbed the Martini tower, went bowling and to the movies, and completed the week with a sightseeing trip to Amsterdam! In short, it was an awesome week!'

Francisco Javier Cano Navarro

'For me, participating in the Summer School 'Ageing Brain' was a great experience. First of all, it was exciting to arrive in a new country and get together with people from so many different countries. Amazing how something simple like an interest in neuroscience research can bring so many people together. In addition, it was nice to see how neuroscience research is performed in other regions of the world. The lectures by leading researchers were very interesting and thought-provoking. To see how the brain normally functions and to see what goes wrong when the brain fails through ageing or disease are great motivations to do research in this area. I learned important lessons, and I think the programme provided me new chances to do research in novel research areas.'

From trip to treatment – the science of psychedelic drugs

You will rarely see me get up early and bike through the rain to make it to church on time. I made an exception for the Interdisciplinary Conference on Psychedelic Research (ICPR2012), a two-day event hosted in the Moses and Aaron Church in Amsterdam. One of the invited speakers was Ruud Kortekaas from the NeuroImaging Center in Groningen. Here I outline some of the talks I attended.

ICPR is organized by the OPEN foundation, which is an interdisciplinary foundation aiming to facilitate research on psychedelics. The atmosphere is clearly different from what you would expect at a regular conference. The audience is pretty diverse and the media coverage is impressive which says something about a great public interest for this topic in the Netherlands. The talks were broad, covering fields such as philosophy, anthropology, psychology, and neuroscience.

I was particularly interested in the two sessions on the neuropharmacology of psychedelic drugs. The first talk was by Dr. Robin Carhart-Harris (Imperial College London). Together with colleagues, he completed an fMRI study of the brain on psilocybin, the psychedelic compound in magic mushrooms. Just as the drug lights up your senses, you might expect that the brain would be full of activations. Actually the opposite happened: Carhart-Harris found decreases in cerebral blood flow in the thalamus, posterior cingulate cortex (PCC) and medial prefrontal cortex (mPFC). These deactivations were also correlated with the reported intensity of the drug's effects. The deactivated regions are typically thought to integrate information. Carhart-Harris suggested that psychedelics might decrease the connectivity of the brain's connector regions, which leads to a state of unconstrained cognition. The mPFC is often overactive in patients with depression. Both psilocybin and several other psychedelic drugs such as MDMA and ketamine seem to deactivate this region.

Vivid memories

Both Mendel Kaelen (PhD candidate at Imperial College London, and a former student at Groningen University) and Carhart-Harris presented on-going research on memory and MDMA, also known as ecstasy. The results point towards some therapeutically useful effects of MDMA. The drug appears to make negative memories feel less painful and enhance memories of positive episodes. In the MRI scanner, participants were asked to think of some of their most positive and negative memories. The memories were rated for vividness during the scan and later in the day. Participants in the MDMA group rated positive memories as more positive and vivid compared to the placebo group. There was also more activity in visual cortex. Mendel Kaelen is currently looking at the effective connectivity between visual and memory related regions. He presented modeling data (dynamic causal modeling) which show that during MDMA effects input from the visual cortex drives activation in the parahippocampal region. It seems like enhanced sensory processing provides input that makes memory recall more detailed and vivid. After reading this, you might be worried about how participants responded to drugs in the MRI scanner. It

>> CONTINUATION FROM TRIP TO TREATMENT – THE SCIENCE OF PSYCHEDELIC DRUGS

can seem like a claustrophobic place, but participants described it as “a nice warm buzzing tube”, “a cocoon for blissed-out meditation” and the annoying sounds from the scanner “turned into a choir of chanting monks”.

The dualist drug

Dr. Ruud Kortekaas (University Medical Center Groningen) outlined his plans for an ambitious study. Together with Profs Schoevers and Aleman he plans to investigate the effects of ketamine on patients with treatment-resistant depression. Ketamine is a medically approved dissociative anesthetic and is also used as a party drug. One of the curious effects of ketamine is the dissociative effect – it literally separates the mind from the body. It is often described as a trip deep into the mind where the external world seems distant.

> *One of the curious effects of ketamine is the dissociative effect - it literally separates the mind from the body.*

Kortekaas presented a study protocol, which involves 52 patients and will look at the effects of three weeks of treatment over a period of twenty weeks in total. The patients in the experimental group will take low doses of oral ketamine, and all patients will continue with their on-going treatment as well. Previous studies have already shown that patients with depression improve within a few hours after ketamine administration. For some, the effects persist for more than a week. Up until now, the doses of ketamine have been quite high and given by injections. This means that the patients have to come to a hospital and that they will be tripping for an hour or so. In the study planned by Kortekaas, the goal is to see if positive effects can be achieved with a much lower amount of ketamine given as a pill on a daily basis. If that is the case, ketamine would be a serious alternative to conventional antidepressants.

I am curious about the outcome of the ketamine study. One issue that kept coming up during the conference was whether the actual psychedelic experience is necessary for an effective treatment. Perhaps the trip reflects some form of plasticity or underlying restructuring of the brain that is crucial for the effectiveness of the treatment. For instance, Dr. Matthew Johnson (John Hopkins University, USA) found that the effect of psilocybin on mood and attitudes was related to the rating of the psychedelic experience rather than the amount of drugs received. In other words: having an eye-opening, mind-blowing, spiritual experience mattered more than the dose of the drug that facilitated it. I wonder if the psychedelic experience itself reflects that neural changes are taking place. Or can you have the same effects without the trip? To me, the psychedelic effect could be essential for the treatment, but the trip is also a challenge for this field of research. You could argue that psychedelic studies rarely are double blind: if you are in the experimental condition, you will know. The study by Kortekaas will definitely contribute to this debate, because the ketamine dose given is too low to have any psychedelic effects. If the treatment is successful, it will be a lot easier to rule out a placebo effect.

■ BY BARBARA NORDHJEM
■ PHOTOS: RENÉ PASSET AND MARCO REEUWIJK

Ruud Kortekaas

Barbara Nordhjem

> If Diana has any ideas, she should feel free to give me a call!

> ALUMNICOLUMN

Scientific decision making

About five years ago, Diana Koopmans called me with the question whether I was interested in attending a master class to be given by Whee Ky Ma – a former math and physics student of the University of Groningen who had moved to the US to obtain postdoctoral training in theoretical neuroscience. At that time, I was a PhD student with Jos Roerdink (Computer Science) and Frans Cornelissen (Experimental Ophthalmology), working on questions related to visual perception and data visualization. When Diana called me, I was in the midst of finishing up a paper and my answer was something along the lines of "Ehm, I am kind of busy with this paper deadline right now and I already attended the master class by Prof. Hans Colonius last week. I think I'll pass on this one". However, since the topic of the master class seemed interesting and since I'm a talented procrastinator, it didn't take Diana a lot of effort to make me change my mind. Unbeknownst to both of us, her phone call not only had the effect that I spent a morning in this master class a few days later, but also that I would say goodbye to the Netherlands two years later.

The master class was mostly an introduction to neural modeling using the mathematical concept of "population codes". Whee Ky explained to us how the brain can perform optimal cue combination (i.e., multiply two probability distributions) by simply summing the activities of populations of neurons. While I had always thought the gap between behaviour and neural coding was astronomical, the concept of population codes seemed to offer an elegant way of closing this gap. I was intrigued by this, and decided to apply this framework to model my own behavioral data. I kept in contact with Whee Ky through email and he taught me not only about population coding, but also a bit about Bayesian models of behaviour.

About half a year after the master class, I got an email from him telling me that he was soon going to start his own lab "here in the US" and that I might be a suitable postdoc candidate. I was excited, but found it somewhat suspicious that he was so vague about the location. It turned out that "here in the USA" was Baylor College of Medicine in Houston, Texas. Although I had strong reservations about the location, after some more correspondence and a visit to his lab, the opportunity to work with him seemed too good to reject, so I decided to take it. When I joined his lab in October 2009, the idea was that I would work on optimal observer modeling of problems that we both thought were interesting, and we would test these models on data from psychophysical experiments. Looking back on it, our backgrounds matched perfectly for this rather unspecific goal: I was experienced in setting up psychophysical experiments but didn't really have a clue about (proper) optimal observer modeling, while Whee Ky had excellent mathematical skills but was at that time not very experienced with setting up experiments.

Soon after I started working in Houston, I found myself working on a dozen or so different projects. Two years later I realized that it is apparently much easier to come up with interesting problems to work on than it is to finish them in time. As a result, two years have become three, and three years have become three-years-and-two-months. But it has been worth it, because eventually we got a lot of work done and we currently have a steady stream of papers coming out. In January of next year I will move back to Europe, where I will do a postdoc in the lab of Daniel Wolpert in Cambridge. Whereas I probably won't miss Houston life, I will certainly miss the playful atmosphere and friends in the Ma Lab. However, since there are still plenty of unfinished projects, I expect to keep in touch with the lab for quite a while after moving. I don't know where I will end up after my next postdoc adventure, but if Diana has any ideas, she should feel free to give me a call!

■ RONALD VAN DEN BERG

New editors wanted!

Do you enjoy reading the Newsletter? If so, why not join our enthusiastic editorial team and make it even better? Regardless of whether you're a master student, PhD student, postdoc, or principle investigator, it's a great way to expand your network, improve your English writing skills, and be actively involved in BCN. Interested? Send an e-mail to Sander Martens, s.martens@med.umcg.nl!

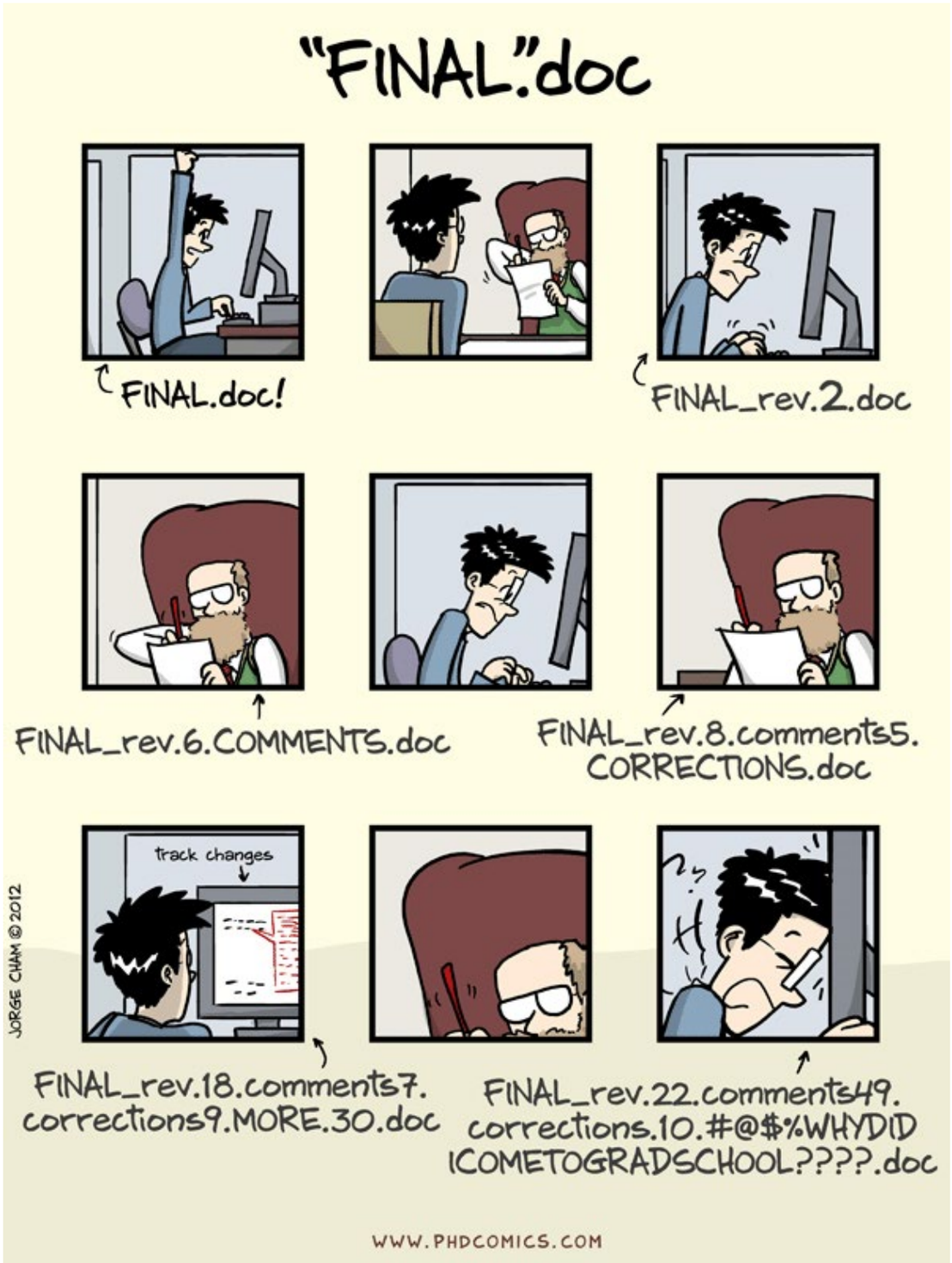
> UPCOMING BCN EVENTS

December 17-20, 2012
BCN Matlab course

Februari 7, 2013
BCN New Years Meeting / Poster Presentation
Location: Bernoulliborg

March 14 & 15, 2013
BCN Retreat

June 19 & 20, 26 & 27, 2013
BCN Statistics course



> ORATIONS

De grijze massa ingekleurd

ORATIE
R.C. Oude Voshaar
TITEL
De grijze massa ingekleurd
LEEROPDRACHT
Ouderenpsychiatrie
DATUM
9 oktober 2012

De beste bezuiniging in de hedendaagse gezondheidszorg is het investeren in een goede psychiatrische ouderenzorg. Dat stelt prof.dr. Richard Oude Voshaar, hoogleraar ouderenpsychiatrie, tijdens zijn oratie. Als gevolg van de vergrijzing komen er steeds meer mensen met meerdere lichamelijke ziekten tegelijkertijd, ook wel multimorbiditeit genoemd. Ouderen met multimorbiditeit doen een groot beroep op onze gezondheidszorg. Ongeveer de helft van deze ouderen kampt met psychiatrische stoornissen (vooral depressie en angst), wat gepaard gaat met een verdubbeling van de medische zorgkosten. Deze extra kosten kunnen sterk verminderd worden door goede psychiatrische zorg.

De afdeling psychiatrie van het UMCG heeft de ouderenpsychiatrie niet voor niets tot één van haar speerpunten gemaakt. Inmiddels is een volledig zorgprogramma ouderenpsychiatrie vormgegeven. Daarmee wordt niet alleen een impuls gegeven aan de kwaliteit van (psychiatrische) ouderenzorg in de regio, maar ook een stevig fundament gelegd voor het opleiden van de ouderenpsychiater van de toekomst. Deze nieuwe generatie ouderenpsychiaters zal zich meer dan voorheen moeten bekwamen in het samenspel tussen lichamelijke en psychiatrische ziekten, en de hierop aangepaste psychotherapeutische behandelingen.

Hoewel psychiatrische behandelingen steeds effectiever worden, kunnen veel ouderen met depressieve en angstklachten nog niet adequaat geholpen worden. Een belangrijk probleem is dat de meeste behandelingen die worden aangeboden, rechtstreeks uit de zorg voor volwassenen zijn overgenomen of enkel zijn getest bij relatief jonge en fysiek gezonde ouderen. Kennis over de behandeling van lichamelijk kwetsbare ouderen met psychiatrische problemen, dus de patiënten die dagelijks de spreekkamer van de afdeling ouderenpsychiatrie bezoeken, is beperkt voorhanden. De onderzoekslijn van Oude Voshaar zal zich specifiek inzetten om de psychiatrische problematiek bij lichamelijk kwetsbare ouderen en ouderen met beginnende cognitieve stoornissen te verbeteren. Met behulp van het Noord-Nederlandse LifeLines onderzoek (een groot bevolkingsonderzoek) en de Nederlandse Studie naar Depressie bij ouderen (NESDO) zal hij kijken naar de effecten van lichamelijke kwetsbaarheid en cognitief functioneren op de diagnostiek en het beloop van depressie en angst.

Om deze kennis te kunnen vertalen naar de dagelijkse praktijk, zal Oude Voshaar investeren in het routinematig meten van de dagelijkse zorg. Tevens zal hij heel gedetailleerde studies verrichten binnen individuele patiënten, ook wel ideografisch onderzoek genoemd. Op die manier kunnen algemene verbanden vertaald worden naar hun betekenis voor een individu. Zo hoopt Oude Voshaar de grijze massa preciezer in te kleuren en het leven van ouderen met depressie en angst weer kleur te geven.

The magic of logic

ORATIE
B.P. Kooi
TITEL
The magic of logic
LEEROPDRACHT
Logica en argumentatietheorie
DATUM
23 oktober 2012

De oratie van prof.dr. Barteld Kooi gaat over logica, het vakgebied dat zich bezighoudt met redeneringen. Logica levert de middelen om correcte redeneringen te onderscheiden van incorrecte. Mensen hebben een aardige intuïtie voor welke redeneringen kloppen en welke niet. De noodzaak voor een betere studie van de logica ontstaat pas als we geconfronteerd worden met een logische paradox. Met schijnbaar kloppende redeneringen komen we bij een tegenspraak terecht. We hebben ergens een foutje gemaakt, maar waar? Welke redeneerstap die we normaal gesproken onproblematisch achten deugt eigenlijk niet? En waarom niet? En waarom andere stappen juist wel?

Aan de hand van een goocheltruc waarin een goochelaar ons een geheim ontfutselt lijkt te hebben, zal Kooi uitleggen hoe we kunnen laten zien dat een redenering klopt. Aan de hand van een mislukte goocheltruc zal hij uitleggen hoe we kunnen laten zien dat een redenering niet klopt. Op die manier ontstaat een duidelijk beeld hoe formele logica eigenlijk in elkaar steekt en welke vervolgvragen voor de logicus van belang zijn.

Ten slotte laat Kooi zien hoe we, dankzij een goed begrip van de logica, bepaalde onmogelijk lijkende breinbrekers te lijf kunnen door de cruciale stappen van de oplossing te verhelderen met goede logische analyse.

■ EVELYN KUIPER-DRENTH, OP BASIS VAN PERSBERICHTEN VAN DE RIJKSUNIVERSITEIT GRONINGEN

> PROMOTIONS

Social stress in adolescent rats: adult behavioral and neurobiological consequences

PROMOVENDUS
J. Viddal Mollon
PROEFSCHRIFT
Social stress in adolescent rats: adult behavioral and neurobiological consequences
PROMOTOR
Prof.dr. J.M. Koolhaas

Langetermijngevolgen van sociale stress in de adolescentie
Pesten op scholen wordt gezien als een groot probleem. Het wordt door de slachtoffers ervaren als uiterst stressvol en kan de kwaliteit van hun leven – ook later – ernstig beïnvloeden. Jose Vidal Mollon onderzocht bij ratten de individuele verschillen in de langetermijngevolgen van sociale stress tijdens de adolescentieperiode.

Vidal Mollon maakte gebruik van zowel de Wistar rat als de Wildtype Groningen rat. Allereerst onderzocht hij in hoeverre sociale stress tijdens adolescentie van invloed is op angst op volwassen leeftijd. De dieren werden daartoe getest in een sociale vermijdingstest en een water-conflicttest. Naast deze gedragstesten onderzocht hij ook een aantal neurobiologische markers zoals neurogenese en serotonerge neurotransmissie, die in het algemeen geassocieerd worden met stemmingsstoornissen.

De resultaten kunnen als volgt worden samengevat:

- Wistar ratten die tijdens de adolescentie een sociaal verlies hebben ondergaan vertonen op volwassen leeftijd meer gegeneraliseerde sociale angst en vermijdingsgedrag dan Wildype Groningen ratten. Deze angst blijft echter beperkt tot situaties die nog enige relatie hebben met de context van de stressvolle ervaring tijdens de adolescentie.
- Individuele eigenschappen zijn bepalend voor de lange termijn gevolgen van pesten. De keuze van de rattenstam is daarbij cruciaal voor de validiteit van diermodellen.
- Het verhoogde niveau van sociale angst in de Wistar ratten is niet gereflecteerd in veranderde gehaltes en turnover van monoamines in het centrale zenuwstelsel zoals dat is gemeten met behulp van HPLC.
- Sociale stress tijdens de adolescentieperiode leidt tot een verhoging van neurogenese en overleving van nieuw gevormde neuronen in de hippocampus van Wistar ratten.
- De water-conflicttest is heel geschikt voor het bepalen van de delicate balans tussen sociale angst en de motivatie voor het verkrijgen van eerste levensbehoeftes in omgevingen die in verschillende mate geassocieerd kunnen worden met het sociale verlies.

Het sociale stressmodel dat Vidal Mallon ontwikkelde, biedt vele mogelijkheden voor verder onderzoek naar de gevolgen van stress tijdens de adolescentie.

Jose Vidal Mollon (Spanje, 1979) studeerde psychologie aan de universiteit van Valencia. Zijn promotieonderzoek deed hij aan de Rijksuniversiteit Groningen bij de vakgroep dierfysiologie, afdeling biologie. Inmiddels werkt hij als docent psychologie aan de RUG. Hij promoveerde op 7 september 2012.

Early life influences, sex differences and stress vulnerability. The impact of maternal separation on adult stress sensitivity in rats

PROMOVENDUS
H.J. Hulshof
PROEFSCHRIFT
Early life influences, sex differences and stress vulnerability. The impact of maternal separation on adult stress sensitivity in rats
PROMOTORES
Prof.dr. J.A. den Boer
Prof.dr. P.G.M. Luiten

Stress en stemmingsstoornissen
Dat vrouwen vaker last hebben van stemmingsstoornissen (zoals depressie) dan mannen, heeft er wellicht mee te maken dat ze gevoeliger zijn voor stress. Het precieze verband tussen stemmingsstoornissen en stress is nog onopgehelderd, maar er zijn aanwijzingen dat de verstoring van slaap en/of de aanmaak van nieuwe hersencellen hierbij een rol speelt.

Henriëtte Hulshof bracht dit verband nader in kaart. Ze zette een experiment op waarin vrouwelijke en mannelijke ratten werden blootgesteld aan verschillende vormen van stress. Vrouwelijke ratten bleken veel hogere stresshormoonspiegels te ontwikkelen dan mannelijke ratten, onafhankelijk van het soort of de ernst van de stress waaraan ze werden blootgesteld. De gevolgen hiervan waren echter kleiner dan verwacht. Vrouwtjesratten sliepen na stressvolle ervaringen niet slechter dan mannetjesdieren. Ook wat betreft de aanmaak van nieuwe hersencellen vertoonden de

>> CONTINUATION PROMOTIONS

mannetjes- en vrouwtjesdieren geen significante verschillen.

Ook onderzocht Hulshof of ratten anders op stress reageren wanneer ze kort na hun geboorte enige tijd bij hun moeder worden weggehaald. Anders dan werd aangenomen, verstoorde dit de normale (hersen)ontwikkeling niet en verhoogde dit de gevoeligheid voor stress evenmin. De ontwikkeling van stemmingsstoornissen is waarschijnlijk het resultaat van een complexe interactie tussen de erfelijke eigenschappen, sekse, pre- en postnatale ontwikkelingsfactoren en stresservaringen in het latere leven, suggereert de promovenda. In het onderzoek naar het ontstaan van stemmingsstoornissen zou deze interactie meer aandacht moeten krijgen.

Henriëtte Hulshof (Stadskanaal, 1982) studeerde medische biologie aan de Rijksuniversiteit Groningen (RUG). Ze verrichtte haar onderzoek aan de onderzoeksgroep Moleculaire Neurobiologie van de RUG en de afdeling Psychiatrie van het Universitair Medisch Centrum Groningen (UMCG). Het onderzoek werd mede gefinancierd door de farmaceutische firma's Organon en Schering-Plough. Zij promoveerde op 10 september 2012.

Stress and cognition. Mechanisms regulating memory and empathy

PROMOVENDUS
P. Atsak
PROEFSCHRIFT
Stress and cognition. Mechanisms regulating memory and empathy
PROMOTORES
Prof.dr. B. Roozendaal
Prof.dr. C.M. Keysers

'Cannabissysteem' in de hersenen helpt herinneringen te verdringen
Het endocannabinoïde systeem in onze hersenen speelt wellicht een rol bij het 'verdringen' van herinneringen, zo blijkt uit het onderzoek van Piray Atsak. Dit biedt wellicht aanknopingspunten voor een nieuwe behandeling voor het posttraumatisch stress syndroom (PTSS). En misschien verklaart het ook waarom mensen die traumatische ervaringen hebben doorgemaakt relatief veel cannabis gebruiken.
Niet al onze herinneringen zijn even sterk. Emotionele en traumatische levenservaringen worden beter opgeslagen in ons geheugen dan alledaagse gebeurtenissen. Ook is bekend dat stress, bijvoorbeeld tijdens een examen of een sollicitatiegesprek, het oproepen van informatie uit ons geheugen kan verminderen. Het was al bekend dat de stresshormonen glucocorticoïden en adrenaline de verwerking van herinneringen via een 'langzaam mechanisme' (genexpressie) beïnvloeden.

Recent onderzoek toonde aan dat er ook een snel mechanisme moet zijn. Piray Atsak bracht de details hiervan in kaart. Zij stelt vast dat het endocannabinoïde systeem, een snelwerkend lipide-systeem in de hersenen, onmisbaar is bij deze snelle geheugeneffecten van glucocorticoïden. Het endocannabinoïde systeem is vooral bekend vanwege de psychoactieve effecten van cannabis. De bevindingen kunnen niet alleen leiden tot nieuwe inzichten bij het bestrijden van emotionele of traumatische geheugenprocessen bij mensen met een posttraumatische stressstoornis, maar kunnen wellicht ook verklaren waarom het gebruik van cannabis zo hoog is in mensen met traumatische levenservaringen.

Piray Atsak (Turkije, 1981) studeerde biologie te Izmir. Ze verrichtte haar onderzoek aan de afdeling Neurowetenschappen van het Universitair Medisch Centrum Groningen (UMCG). Het onderzoek werd mede gefinancierd door NWO en de Jan Kornelis de Cock Stichting. Atsak werkt inmiddels als postdoc aan de Radboud Universiteit Nijmegen. Zij promoveerde op 1 oktober 2012.

Wayfinding and accessibility for visually impaired people. Opportunities and challenges

PROMOVENDUS
E.M. Havik
PROEFSCHRIFT
Wayfinding and accessibility for visually impaired people. Opportunities and challenges
PROMOTOR
Prof.dr. A.C. Kooijman

De inrichting van straten en pleinen volgens het Shared Space concept kan voor blinden en slechtzienden een probleem vormen omdat zij te weinig houvast hebben voor hun oriëntatie. Onderzoeker Else Havik van het Universitair Medisch Centrum Groningen pleit ervoor om al in de vroege ontwerpfase van Shared Spaces rekening te houden met mensen met een visuele beperking. Concrete aanbevelingen uit het onderzoek van Havik zijn door Koninklijke Visio, expertisecentrum voor slechtziende en blinde mensen, verwerkt in een gids die deze maand verschijnt.

Het Shared Space-concept wordt in steeds meer steden en dorpen in Nederland toegepast bij de inrichting en het gebruik van straten en pleinen, onder andere in Haarlem, Drachten, Enschede, Tiel en Zwolle. In een Shared Space-gebied ontbreekt vaak een traditionele indeling in rijbanen, fietspaden en trottoirs, en zijn er

>> CONTINUATION PROMOTIONS

geen stoplichten en zebrapaden meer. Het ontwerp beoogt dat voetgangers, fietsers en gemotoriseerd verkeer rekening houden met elkaar in een gebied waar iedereen te gast is en niemand de overhand heeft. De zorgen die verschillende belangenorganisaties hebben geuit over de veiligheid van blinden en slechtzienden in Shared Spaces vormden de aanleiding voor het onderzoek van Havik.

Havik heeft knelpunten geïnventariseerd die blinden en slechtzienden kunnen tegenkomen in Shared Spaces. “Het ligt heel genuanceerd,” legt Havik uit. “Veel voorkomende knelpunten zijn het ontbreken van een duidelijk onderscheid tussen rijbaan en voetgangersgebied en het ontbreken van herkenbare oversteekplaatsen. Dit maakt het voor slechtzienden en blinden moeilijk om zich te oriënteren.”

Vijfentwintig vrijwilligers met een visuele beperking namen deel aan een onderzoek van Havik om ervaringen in Shared Spaces te verzamelen. Zij liet de mensen opdrachten uitvoeren om hun weg te vinden in Shared Spaces in Haren en Muntendam, en in twee traditioneel ingerichte gebieden. Alle plekken waren onbekend voor de deelnemers. Oriëntatie bleek het grootste knelpunt te zijn in de Shared Spaces, maar de ene locatie gaf duidelijk meer problemen dan de andere. “Het merendeel van de blinde deelnemers kon zich in een van de Shared Spaces niet voorstellen er zelfstandig een route te leren lopen,” vertelt Havik.

Op grond van haar onderzoek doet Havik aanbevelingen voor het ontwerp van Shared Spaces. Het belangrijkste is dat in een vroege

fase al rekening wordt gehouden met het gebruik van deze openbare ruimtes door slechtzienden en blinde mensen. “Elementen die belangrijk zijn voor deze doelgroep kunnen dan worden meegenomen in het ontwerp. Denk aan duidelijke oriëntatiepunten en gidslijnen, bijvoorbeeld door zorgvuldige plaatsing van straatmeubilair en het aanbrengen van een voelbaar verschil in de ondergrond,” aldus Havik. Naast de gids “Shared Space voor blinde en slechtziende mensen. Een uitdaging voor ontwerpers. Aandachtspunten voor een toegankelijke openbare ruimte” die Koninklijke Visio uitgeeft, heeft het centrum veel expertise en adviseurs die geraadpleegd kunnen worden.

Else Havik (Haren, 1978) studeerde psychologie in Groningen. Zij voerde haar onderzoek uit bij het Laboratorium Experimentele Oogheelkunde van de afdeling Oogheelkunde van het UMCG en de Koninklijke Visio, expertisecentrum voor slechtziende en blinde mensen, en was aangesloten bij het Onderzoeksinstituut BCN. Financiering voor het onderzoek werd verkregen van ZonMw Inzicht, Professor Mulderstichting, Stichting Blindenhulp, Stichting Novum en het College voor Zorgverzekeringen (CVZ). Zij promoveerde op 10 oktober 2012.

Maternal brain involvement in (pre) eclampsia. Pathophysiology and long-term consequences

PROMOVENDUS
M.J. Wiegman
PROEFSCHRIFT
Maternal brain involvement in (pre)eclampsia. Pathophysiology and long-term consequences
PROMOTORES
Prof.dr.J.G. Aarnoudse
Prof.dr. M.J. Cipolla

Nader inzicht in hersenveranderingen bij zwangerschapsvergiftiging
Zwangerschapsvergiftiging (pre-eclampsie) komt voor bij vijf tot zeven procent van alle zwangerschappen in Nederland. De zwangere vrouw krijgt dan een hoge bloeddruk en er komen eiwitten in haar urine terecht. In ernstige, zeldzame gevallen treedt vervolgens eclampsie op: door vochtophoping in de hersenen kan de patiënte zwangerschapsstuipen ontwikkelen.

UMCG-promovendus Marjon Wiegman onderzocht in een diermodel hoe pre-eclampsie kan leiden tot eclampsie. Ze stelt vast dat gedurende de zwangerschap de kleine hersenbloedvaten meer vocht doorlaten wanneer de bloeddruk stijgt. Door pre-eclampsie lijken de bloedvaten in de hersenen bovendien minder goed op hoge bloeddruk te kunnen reageren. Dit onderzoek vergroot het inzicht in de kwetsbaarheid van de hersenbloedvaten gedurende de zwangerschap. De inzichten kunnen wellicht helpen eclampsie en andere hersengerelateerde complicaties tijdens de zwangerschap beter te behandelen of zelfs te voorkomen.

Ook stelt Wiegman vast dat vrouwen die eclampsie hebben doorgemaakt rapporteren dat zij in het dagelijks leven meer beperkingen ervaren van hun gezichtsvermogen. Of dit te maken heeft met de hersenveranderingen die optreden na eclampsie, is nog onduidelijk. Het is overigens niet waarschijnlijk, stelt Wiegman, dat de hersenveranderingen na eclampsie rechtstreeks worden veroorzaakt door vochtophoping in de hersenen. Mogelijk speelt aanleg voor het krijgen van hart- en vaatziekten op latere leeftijd hierbij een grotere rol.

Marjon Wiegman (Groningen, 1985) studeerde Geneeskunde te Groningen. Ze verrichtte haar onderzoek aan de afdelingen Obstetrie & Gynaecologie en Medische Biologie van het Universitair Medisch Centrum Groningen (UMCG) en de University of Vermont (Burlington, Verenigde Staten). Het onderzoek werd mede gefinancierd door de Junior Scientific

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Masterclass Groningen en de Jan Kornelis de Cock-stichting. Wiegman is inmiddels in opleiding tot dermatoloog in het UMCG. Zij promoveerde op 15 oktober 2012.

Echoes from a stressful past. Effects, pathways and adaptive value of maternal stress in birds

PROMOVENDUS
R. Henriksen
PROEFSCHRIFT
Echoes from a stressful past. Effects, pathways and adaptive value of maternal stress in birds
PROMOTOR
Prof.dr. A.A.G. Groothuis

Invloed moederlijke stress op haar nakomelingen
Rie Henriksen onderzocht bij kwartels welke invloed warmtestress tijdens de zwangerschap bij de moeder heeft op de stresshormonen in haar kuikens.

Het is bekend dat stress bij de moeder tijdens zwangerschap en eivorming van invloed is op de hormoonsamenstelling in hun nakomelingen. Maar wat gebeurt er als de stress waarmee de moeder te maken had niet optreedt in de omgeving van de jonge nakomelingen? Hebben ze er dan last van dat ze 'niet adequaat zijn voorgeprogrammeerd'? De factor warmtestress bij de moeder – die lijdt tot verhoging van het stresshormoon corticosteron, dat ook weerspiegeld is in het eiplasma – bleek van minder invloed op de kuikens dan de daadwerkelijke warmtestress die zij als kuiken buiten het ei ondervonden. Blijkbaar hebben de kuikens een mechanisme waarmee zij effecten van invloeden tijdens hun embryonale periode teniet kunnen doen als de omgeving niet 'matcht' met die tijdens hun prenatale periode.

Henriksen maakte duidelijk dat stress bij het moederdier tijdens de eivorming in vogels de groei, fysiologie en gedrag van de nakomelingen kan beïnvloeden via een veranderende samenstelling van het ei. Haar resultaten bij vogels kwamen deels overeen met de zoogdierenliteratuur, wat aantoont dat vogels een uitstekend alternatief model zijn voor studies over maternale stress.

Haar bevinding dat postnatale stress effecten van prenatale maternale stress tegen kan gaan geven aan dat stress bij het moederdier de nakomelingen zou kunnen voorbereiden op een stressvolle postnatale omgeving en ondermijnt daarom het algemene idee dat stress ervaren gedurende het hele leven cumulatief leidt tot een slijtage van fysiologische systemen.

Rie Henriksen (Denemarken, 1981) studeerde biologie aan de aan de universiteit van Kopenhagen. Haar promotieonderzoek deed zij aan de RUG, bij de vakgroep Behavioural Biology, dat deel uitmaakt van het Centre for Behaviour and Neurosciences. Het werd gefinancierd door het Oostenrijkse wetenschapsfonds. Inmiddels werkt zij als postdoc in Zweden. Zij promoveerde op 19 oktober 2012.

The role of lipocalin 2 in Alzheimer's disease and depression

PROMOVENDUS
J.P. Naudé
PROEFSCHRIFT
The role of lipocalin 2 in Alzheimer's disease and depression
PROMOTORES
Prof.dr. J.A. den Boer
Prof.dr. P.G.M. Luiten
Prof.dr. U.L.M. Eisel

Nieuw ontdekt eiwit betrokken bij ziekte van Alzheimer
Het nieuw ontdekte eiwit Lipocaline 2 speelt een rol in het ziekteproces van de ziekte van Alzheimer en bij depressiviteit bij ouderen. Omdat Lipocaline 2 goed te meten is in het bloed van patiënten, is hiermee wellicht een methode gevonden om alzheimer in een zeer vroeg stadium op te sporen. Mogelijk biedt de ontdekking ook aangrijpingspunten om nieuwe

>> CONTINUATION PROMOTIONS

medicijnen tegen alzheimer en depressie bij ouderen te ontwikkelen. Dat blijkt uit het promotieonderzoek van Pieter Naudé. Naudé identificeerde Lipocalin2 in gekweekte hersencellen van muizen. Het is een eiwit dat een rol speelt in de ontstekingsreactie van alzheimer. Het bleek verhoogd aanwezig in hersenweefsel dat is aangetast door de ziekte van Alzheimer, evenals in het hersenvocht en het bloed van patiënten met deze ziekte.

Onderzoek naar de betrokken moleculaire mechanismen toonde aan dat Lipocaline 2 de dood van zenuwcellen bevordert, wanneer hersenweefsel in contact komt met het voor alzheimer kenmerkende giftige eiwit amyloïd.

Klinisch onderzoek liet zien dat een toename in Lipocaline 2-concentraties niet alleen voorkomt bij alzheimer, maar ook samenhangt met depressieve symptomen in ouderen. Dit zijn aanwijzingen dat Lipocaline 2 wellicht kan helpen alzheimer en depressie bij ouderen in een vroeg stadium op te sporen. Nader onderzoek naar Lipocaline 2 kan een medicijn tegen deze aandoeningen mogelijk dichterbij brengen.

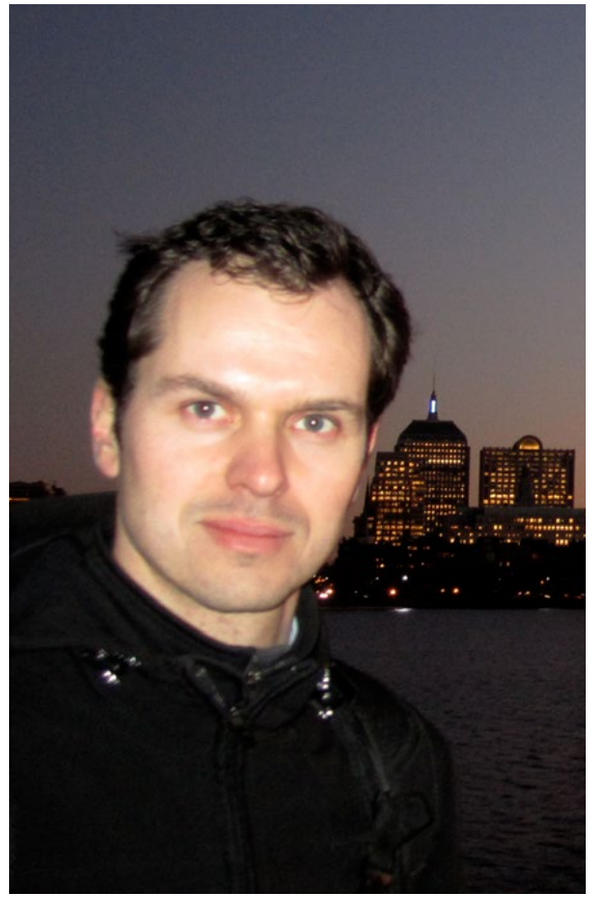
Pieter Naudé (Zuid Afrika, 1982) studeerde Biomedisch Wetenschappen en Farmacologie te Pretoria. Hij verrichtte zijn onderzoek aan de afdeling Biologische Psychiatrie van het Universitair Medisch Centrum Groningen (UMCG), de onderzoeksgroep Moleculaire Neurobiologie van de Faculteit Wiskunde en Natuurwetenschappen van de Rijksuniversiteit Groningen en binnen onderzoeksschool BCN. Het onderzoek werd mede gefinancierd door de Jan Hendrik de Cock Stichting. Naudé werkt inmiddels als postdoc in het UMCG. Hij promoveerde op 24 oktober 2012.

Stem cell based generation of midbrain dopaminergic neurons. Towards cellular tools to study Parkinson's disease

PROMOVENDUS
R.A. Roessler
PROEFSCHRIFT
Stem cell based generation of midbrain dopaminergic neurons. Towards cellular tools to study Parkinson's disease
PROMOTOR
Prof.dr. H.W.G.M. Boddeke

Nader inzicht in effectiviteit 'huidstamcellen' tegen parkinson
Uit huidcellen gereprogrammeerde stamcellen kunnen helpen de ziekte van Parkinson te onderzoeken en behandelen, blijkt uit het onderzoek van Reinhard Roessler.

De ziekte van Parkinson is een van de meest voorkomende neurodegeneratieve aandoeningen. Bij deze ziekte sterft een groep neuronen in de middenhersenen, de zogenaamde dopaminerge neuronen, geleidelijk af. Dit leidt tot motorische problemen (tremoren, stijfheid, een trage, schuifelende gang), maar ook tot depressie en dementie.
Ongeveer 25 jaar geleden werd geprobeerd ter behandeling van de ziekte van Parkinson dopaminerge neuronen te implanteren die waren verkregen uit de hersenen van geaborteerde foetussen. Om praktische en ethische redenen is deze behandelmogelijkheid echter geblokkeerd. Sinds 2006 is bekend dat huidcellen gereprogrammeerd kunnen worden tot een soort embryonale stamcellen, de zogenaamde 'geïnduceerde pluripotente stamcel' (IPS-cel). De ontwikkeling van deze technologie werd onlangs bekroond met de Nobelprijs. Inmiddels kunnen uit de huidcellen ook dopaminerge neuronen geproduceerd worden. Stamcellen uit foetussen zijn dus niet meer nodig. Een ander voordeel is dat eigen huidcellen van de patiënt kunnen worden gebruikt, zodat de ingezette stamcellen niet als lichaamsvreemd worden herkend en het afweersysteem van de patiënt niet onderdrukt hoeft te worden.



Het onderzoek van Roessler laat zien dat de uit IPS-cellen verkregen dopaminerge neuronen vrijwel identiek zijn aan primaire dopaminerge neuronen. Het lijkt erop dat de IPS-cellen (en wellicht de directe conversie van huidcellen tot dopaminerge neuronen) hernieuwde interesse zullen opwekken voor celtransplantatie als therapie voor parkinsonpatiënten.

Reinhard Roessler (Duitsland, 1979) studeerde Biochemie te Berlijn. Hij verrichtte zijn onderzoek aan de afdeling Neurowetenschappen van het Universitair Medisch Centrum Groningen (UMCG). Het onderzoek werd mede gefinancierd door de Hazewinkel-Behringer Stichting en de Jan Cornelius de Cock Stichting. Roessler gaat binnenkort als onderzoeker werken aan het Whitehead Institute for Biomedical Research in Cambridge, Massachusetts. Hij promoveerde op 29 oktober 2012.

The good and the bad of stress. Implications for memory and adaptive processes

PROMOVENDUS
R. Wichmann
PROEFSCHRIFT
The good and the bad of stress. Implications for memory and adaptive processes
PROMOTORES
Prof.dr. B. Roozendaal
Prof.dr. G.J. ter Horst

Stresshormonen reguleren ook geheugen voor positieve ervaringen
De meeste mensen verbinden de werking van stresshormonen vooral met negatieve ervaringen en hoe we die onthouden. Promovenda Romy Wichmann stelt vast dat stresshormonen ook een cruciale rol spelen bij het verbeteren van het geheugen voor positieve en lonende ervaringen. Zij onderzocht dit mechanisme in verschillende delen van het emotionele brein.

Niet alle ervaringen worden even goed opgeslagen in ons geheugen; vooral belangrijke en emotionele gebeurtenissen worden goed onthouden. Stresshormonen zoals glucocorticoïden en adrenaline komen vrij tijdens en na zulke gebeurtenissen en zijn onderdeel van de neurobiologische processen die emotionele ervaringen in ons geheugen opslaan. Wichmann verrichtte proefdieronderzoek aan deze neurobiologische circuits en mechanismen.

De cruciale rol van stresshormonen op emotioneel leren is vaker onderzocht maar tot nu toe vooral bij angstregulerende leertaken. Nu blijken de nucleus accumbens en de amygdala hersenkernen ook betrokken te zijn bij het leren van positieve en lonende ervaringen. Dit betekent dat alleen de aanwezigheid van stresshormonen na een prikkel onvoldoende is om die prikkel als stressvol – in negatieve zin – te kenmerken. Het is een kwestie van timing en dosering.

Romy Wichmann (Schwerin, Duitsland, 1980) studeerde Biomedische Wetenschappen aan de Universiteit van Marburg. Zij verrichtte haar promotieonderzoek bij de Afdeling Neurowetenschappen, Sectie Anatomie, van het Universitair Medisch Centrum Groningen (UMCG) en in het kader van het onderzoeksinstituut BCN. Het onderzoek werd medegefinancierd door de Hazewinkel Behringer Fonds, de Jan Kornelis de Cock Stichting en de Dobberke Stichting. Wichmann wordt postdoc onderzoeker aan MIT in Cambridge, VS. Zij promoveerde op 29 oktober 2012.

■ EVELYN KUIPER-DRENTH, OP BASIS VAN PERSBERICHTEN VAN DE RIJKSUNIVERSITEIT GRONINGEN

> ONE CAN ALSO LEARN FROM "STELLINGEN"

Berglucht is niet alleen heilzaam voor de longen, maar ook voor de geest.

> Marjon Wiegman

In tegenstelling tot hetgeen de Engelse uitdrukking "As the twig is bent, the tree's inclined" (Alexander Pope, English poet) suggereert, hebben (negatieve) gebeurtenissen vroeg in het leven niet altijd grote gevolgen voor de toekomst.

> Henriëtte Hulshof

Als promovendus werkend aan stressonderzoek krijg je te maken met het Droste-effect.

> Henriëtte Hulshof

When you consider doing a PhD, you are standing at the top of the cliff, when you start, you jump off and if you manage to finish it, you know you built your wings on the way down. Adapted from Ray Bradbury

> Piray Atsak

The crucial question about cannabis is rather a dose issue than its usage.

> Piray Atsak

If the brain was so simple we could understand it, we would be so simple that we couldn't

> Romy Wichmann

Je mist meer dan je meemaakt. Helemaal niet erg. -Martin Bril

> Else Havik

> COLOPHON

This newsletter is published by the School for Behavioural and Cognitive Neurosciences

Frequency
4 x a year

Publishing Office
BCN Office (FA33), A. Deusinglaan 1, 9713 AV Groningen,
050 363 4734

Editorial Staff
Michiel Hooiveld, m.h.w.hooiveld@umcg.nl
Evelyn Kuiper-Drenth, Copy Editor, BCN Office,
050 3634734, e.t .kuiper-drenth@umcg.nl
Sander Martens, Editor-in-chief, s.martens@umcg.nl
Kashmiri Stec, Copy Editor, k.k.m.stec@rug.nl

Editors
Renske Bosman, r.c.bosman@student.rug.nl
Christina Cordes
Léon Faber, l.g.faber@rug.nl
Emily de Hartog, e.a.de.hartog@student.rug.nl
Annika Luckmann, a.luckmann@student.rug.nl
Robin Mills, r.mills@student.rug.nl
Kathi Müller, a.k.mueller@rug.nl
Riccarda Peters, riccarda_p@gmx.de
Florian Sense, floriansense@googlemail.com

Contributors
André Aleman, a.aleman@umcg.nl
Geert Jan Arends, g.j.arends@rug.nl
BCN PhD Council Committee, bcnphdcouncil@umcg.nl
Sietske Berghuis, s.a.berghuis@student.rug.nl
Erik Boddeke, BCN Director, h.w.g.m.boddeke@umcg.nl
John Duncan, John.Duncan@mrc-cbu.cam.ac.uk
Ineke Ganzeveld, k.j.ganzeveld@rug.nl
Reinoud Gosens, r.gosens@rug.nl
Tjakko van Ham, t.j.van.ham@rug.nl
Thomas Kantermann, t.kantermann@rug.nl
Diana Koopmans, d.h.koopmans@umcg.nl
Floris de Lange, floris.delange@donders.ru.nl
Francisco Javier Cano Navarro
Mark Slors, marc.slors@phil.ru.nl
Dick Swaab, d.f.swaab@nin.knaw.nl

Layout
Dorèl Extra Bold, eddy@dorelextrabold.nl

Photos/illustrations
Piray Atsak, Ronald van den Berg, Erik Boddeke, Christina Cordes, Tjakko van Ham, Michiel Hooiveld, Henriëtte Hulshof, Fleur Jongepier, Thomas Kantermann, Annika Luckmann, Sander Martens, Arine Mentink, Pieter Naudé, René Passet, Marco Reeuwijk, Reinhard Roessler, Romy Wichmann, Marjon Wiegman, www.phdcomics.com; http://www.rug.nl/news-and-events/

Deadline for the next edition: 18 January 2013