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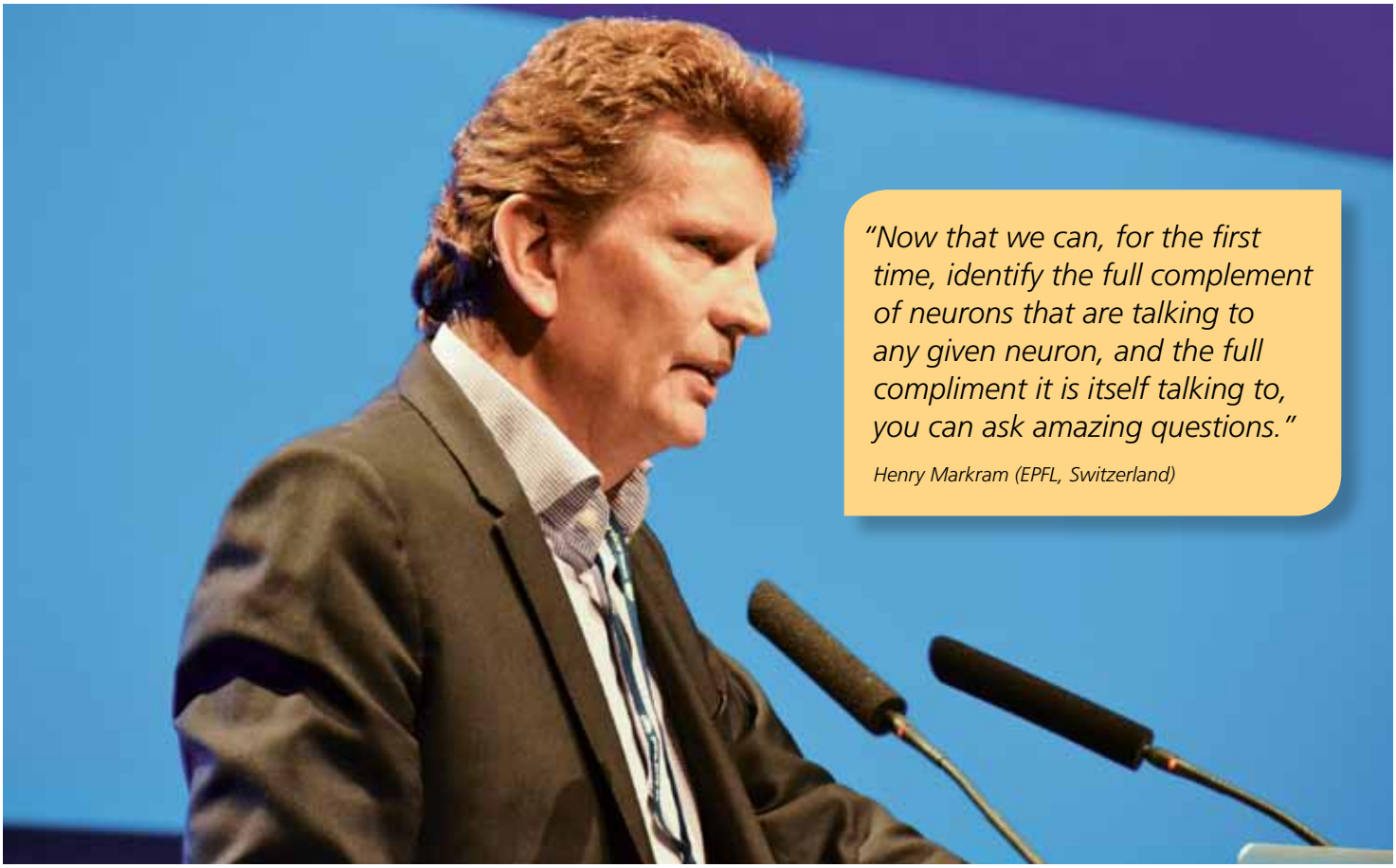
ECNP DAILY NEWS

# Dissecting the Human Brain Project



*"Now that we can, for the first time, identify the full complement of neurons that are talking to any given neuron, and the full compliment it is itself talking to, you can ask amazing questions."*

*Henry Markram (EPFL, Switzerland)*

Previously only a matter of fiction, the construction of a virtual human brain that will revolutionise and unify our understanding of the world's most complex organ is now a prospect gathering incredible momentum, delegates heard in Saturday evening's packed-out Keynote Lecture.

Understanding of the human brain is one of the greatest challenges facing 21st century science, yet a major hindrance in our knoweldge of the brain has been the fragmented nature of brain research and the data it produces, being enormously productive but ultimately unsystematic.

The new initiative, known as the Human Brain Project, stemmed from a €1 billion, ten-year grant from the Future and Emerging Technologies (FET) Flagship Programme, a new endeavour launched by the European Commission that encourages visionary and 'mission-oriented' research that has the potential to offer ground-breaking information technology that will benefit European society and industry.

Founder and Director of the project Henry Markram, a Professor of Neuroscience at the École Polytechnique Fédérale de Lausanne (EPFL) in Switzerland, shared the goals of this staggering project in the lecture, beginning with what he believed the take home message would be in the genesis of the Human Brain mission.

"It is time to begin a unification of our understanding of the brain and its diseases," said Professor Markram. "Now, many scientists and clinicians of course argue at this point whether this is the time. There are many reasons for arguments against it. The main reason of course is an obvious one: how could we possibly begin towards a unified understanding of the brain, when we just don't know enough? We don't have enough data.

"But there are two fundamental problems with that argument. The first one is that actually we are facing a wall of data – an exponentially growing wall of data ... and we have no plan of what to do with it. We deal with it piece by piece, and we have no coherent plan of how to approach all of the data that is generated. Far too much data is being generated for us to even cope with, but it is still nowhere near enough to build a model of the brain, and to begin a unified model."

Mapping the brain experimentally is, as Professor Markram noted, an impossible task in most if not all people's expert opinion, thus he stressed that we would be realistic about the challenges that we are facing, and instead usher in more "predictive neuroscience", looking anatomically, structurally and mechanistically.

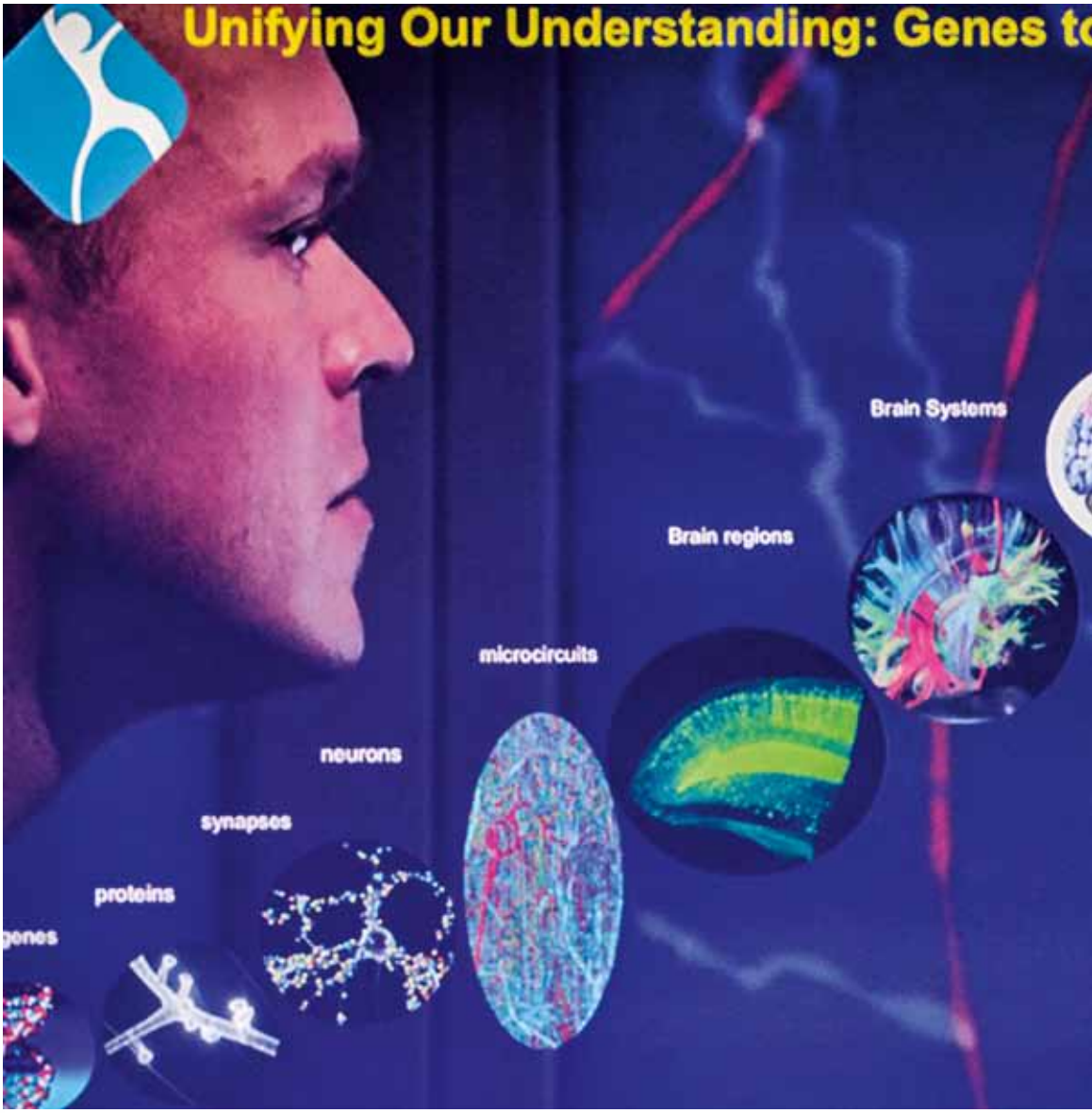
To arrive at the stage where a human virtual brain is a real possibility, there of course has been an incredible amount of work and development to establish both a core understanding, and future perspective, in how the project be realised. Much of this knowledge has stemmed from a precursor initiative, the Blue Brain Project. Professor Markram described

*Continued on page 2*

LIVE FROM BARCELONA

**Keynote Lecture** The Human Brain Project

# Dissecting the Human Brain Project




*Continued from page 1*

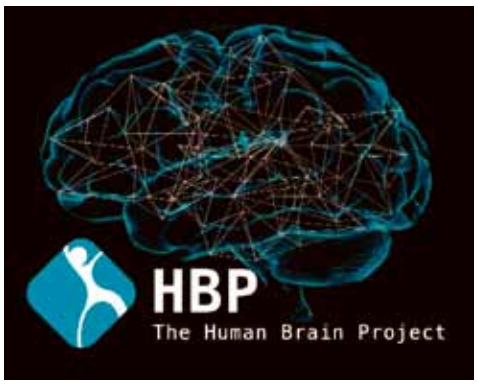
some of the remarkable journey thus far, saying: "We collected and formed about 20,000 experiments on single cells and pairs of cells. We found out synaptic connections and how these synaptics actually worked, and drew in three dimensions 100s of cells, of every cell type, almost obsessively.

"We recorded the cells, and even extracted the cytoplasm to get the gene expression in every cell. It was a huge amount of data. As a neuroscientist it was a very exciting adventure, but I still didn't see a picture of how this all fits together. What was even more remarkable was that I started to do a calculation, and I realised that after 15 years of obsessively studying this microcircuit, I managed to collect probably only a thousandth of a percent of the data you would need to build a model."

This precious, time-consuming yet limited data (in the scope of the whole brain paradigm) was not sufficient to build the model of the microcircuit that Professor Markram set out to achieve, thus his team determined a need to develop an algorithm that was data driven, using as much as they could bundle together, in order to better capture the activity. That way, even with missing links in the chain, albeit with a few assumptions, a model could be harnessed to recreate the features of millions of neurons.

Using significant computational power, the team has had impressive results thus far: "We discovered a lot of fundamental rules about how the morphology shaped electrical behaviour, how the ion channel distribution shaped electrical behaviour, but the single most important thing is that when anyone ever publishes something new about an ion channel in that neurons, we integrate that and use it as a constraint," said Professor Markram. "And what we learned was that if you add biology to this approach, then modelling becomes easier. You actually have to fit less."

Of course to extrapolate mapping out to the whole brain, an incredible resource in computing is required, and the Human Brain Project estimates that mapping of just one individual neurons would require the computing power of a laptop.[1] Thus a significant part of the project will be research into neuromorphic or 'brain-like' supercomputers that can combine microelectronics with the cognitive flexibility of human intelligence.




"Ultimately what we need to know is how many genetic cell types there are in the brain," continued Professor Markram. "It's the single most important data set that we need if we want to understand the brain and unify all our knowledge around it.

"If we had that information, you could actually just throw in these neurons randomly and rearrange

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them until they reproduce staining maps, and you would have a derived composition of cells in the brain, and then you can translate the predictions of the morphology, the electrical types, the receptors, the synapse types. There is an almost endless cascade of predications you could make about how the brain is structured if we have that data set, but we don't have that data set yet... it's one of the priorities in the Human Brain Project.

"The next challenge is the connectome, i.e. how do you connect these neurons. This is where it becomes interesting, and this is where we will actually show you that it is experimentally impossible to map the human brain... It takes about a million dollars and one year to map one pathway. It would take 3000 years and $3 billion just to map this piece of the brain about the size of a pinhead, let alone every age, every brain region, every species, gender or genetic variation, and every disease. That's not going to happen. So with this tiny bit of data that we have, we thought how can we make a prediction?"

Professor Markram added that another absolutely fundamental principle concerned with how the brain is wired together is that one neuron does not project to another neuron. A neuron projects to a region with lots of other neurons, and principle allowed the development of an algorithm that actually predicts the connectome. "In a series of steps, we could actually eliminate all those structural connections that are not viable and do not fit the synapse rule," he said. "Basically this algorithm allowed us to come up with a prediction that we could validate."

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>
> *Henry Markram (EPFL, Switzerland)*

> *"There is an almost endless cascade of predications you could make about how the brain is structured."*
>
> *Henry Markram (EPFL, Switzerland)*

Looking ahead to the 10-year plan for the Human Brain Project, it has been mapped to three phases that have specific goals which should ensure momentum is maintained when moving the project forward. The initial phase (2.5 years) will see the 'ramp-up' of ICT technology, developing the platforms that should, by the end of the phase, be ready to harness by internal and external researchers alike. In phase two, the so-called 'operational' period (4.5 years), focus will be placed on data collection, while still constantly building and developing the platforms, as well as feeding back their value with demonstration of efficacy.

The final three years of the proposed timeline, referred to as the 'sustainable' phase, should maintain the activities and technological improvements already established, with unified efforts to ensure further financial support is garnered to push the project – and its advancements so far – into the next era.

With an incredible burden of mental health in Europe, an ageing population and pharmaceutical constraints, the Human Brain Project has the potential of being a potent and revolutionary medical tool, offering insight and investigation into the deepest minutiae of the human brain hitherto inaccessible with the tools at our disposal.

The Human Brain Project will be a service, available to collaboratively access and study models worldwide, allowing the answer of revolutionary questions that, it would appear, are nearly endless. "Now that we can, for the first time, identify the full complement of neurons that are talking to any given neuron, and the full compliment it is itself talking to, you can ask amazing questions," said Professor Markram in closing.

**References**

1) EPFL. The Blue Brain Project. (Available online at http://bluebrain.epfl.ch – accessed April 2013)

LIVE FROM BARCELONA

CBT in psychosis

# Psychotherapy in psychosis


A session that examined cognitive behaviour therapy (CBT) in psychosis took place yesterday afternoon, with Tilo Kircher (University of Marburg, Germany) introducing speakers Mark Van der Gaag (Parnassia, Den Haag, the Netherlands) and Carlijn de Roos (GGZ Rivierduinen, Leiden, the Netherlands) to explore the topic in detail. A relatively underdeveloped area of psychotherapy, the intention of this session was to highlight the effectiveness of CBT in various facets of psychosis, such as reducing hospitalisation times and the likelihood of relapse, improving well-being, cognitive status and patient compliance.

"Psychotherapy in schizophrenia is mentioned in basically all of the national and international treatment guidelines, such as the NICE guidelines and the German guidelines," began Professor Kircher, "but it is not so much implemented into clinical practice that we, who run this session, think it should be.

"If we think about psychosis and psychotherapy, it might not be the first thing that comes to your mind in a pharmacology context. But if you look at the risk factors, the story is different. We know that there is a high genetic risk for schizophrenia and psychosis (about 50%) and the other 50% is environmental factors. The most important of these environmental factors are obstetric complications and childhood maltreatment and urban upbringing, migration and cannabis abuse. And some of these environmental factors are amenable to psychotherapy treatment."

While these environmental factors might benefit from talking therapies, Professor Van der Gaag spoke in further detail about recent developments in both positive and negative symptoms of psychosis and schizophrenia. Speaking of the relatively new appreciation of the unifying features of different psychiatric disorders, he said: "We are starting to treat more psychotic symptoms in non-psychotic disorders, and we are also starting to treat more comorbid disorders in people with psychosis."

> *"We are starting to treat more psychotic symptoms in non-psychotic disorders, and we are also starting to treat more comorbid disorders in people with psychosis."*
>
> *Mark Van der Gaag (Parnassia, Den Haag, the Netherlands)*

The origin of the principles of CBT go back a long way, explained Professor Van der Gaag: "It all started in first century AD, with Epictetus, who was a Greek philosopher, who said that people are not disturbed by the things that happen to them, but the views that they take about them. I think that this is still very true; of the people that, for instance, are traumatised by disaster, only a small proportion will develop PTSD. Most people are resilient to it and don't develop psychopathology. "Is [CBT] effective? Yes, it is effective in many disorders, including psychotic disorders. It is in many guidelines all over the world."

This notion led to the model of antecedence, wherein the appraisal of an occurrence, whether internally or as witnessed in the surrounding environment, will elicit a particular emotional or behavioural response. Professor Van der Gaag cited an instructive example to demonstrate how CBT can achieve a reduction in the thought processes tied up with psychosis and related disorders: upon waking up in the middle of the night and hearing the stairs squeaking, the noise could either be ascribed to the household pet (in which case one would go back to sleep), or to an armed intruder (in which case one would either confront or escape the presumed intruder). "In both cases, it is waking up and hearing a squeaking on the stairs," he said. "But depending on the appraisal, your emotional and behavioural response will be quite different. Therapy and CBT focus on that. We try to find alternative appraisals if they have disastrous or catastrophic appraisals. They have to do behavioural

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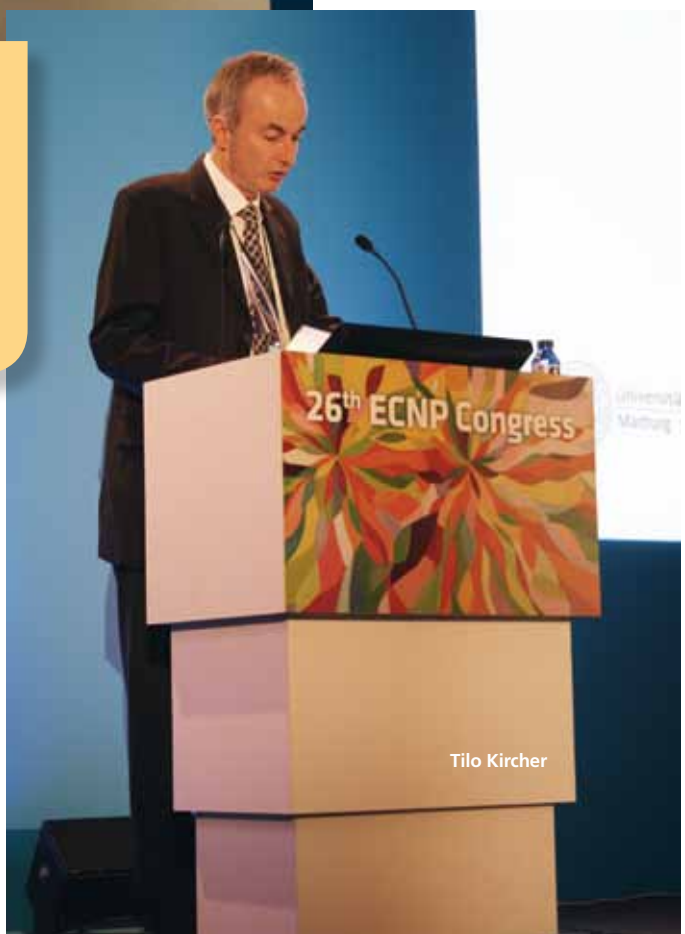



Tilo Kircher

PLENARY LECTURES

PL.03 **Understanding pain in somatoform disorders** Auditorium Monday 7 October **11:00-11:45**

# Piecing together the fibromyalgia puzzle

Fibromyalgia is a clinically well-characterised condition which causes widespread chronic pain and fatigue, but there is still a pressing need to better understand its functional and morphological characteristics, delegates will hear during this morning's Plenary Lecture at the Congress.

Speaking to *ECNP Daily News*, Claudia Sommer (University of Würzburg, Germany) described the research that her team has been undertaking in order to uncover the physical basis of the syndrome.[1] "We had the idea that something might be wrong with the peripheral nerves in these patients," she began.

To study this further, a number of techniques were employed to look at the small peripheral nerve, including quantitative sensory testing (QTS), a technique that measures the detection threshold for warmth and cold, as well as innocuous and noxious touch. "We found that the patients are less sensitive to warmth and cold, so it needs to be warmer and colder for them to notice than for controls," she said. "And of course they are more sensitive to pressure on their muscles, which is a very well known phenomenon. This was an internal control for us; that our patients do not differ from regular fibromyalgia patients."

In additional tests, the investigators utilised pain-related evoked potentials, a neurophysiological method that measures the afferent fibres from the face, hand or foot (wherever stimulated) to the brain. In contrast to routine evoked potentials, the method uses a special electrode that is able to measure the small A-delta nerve fibres – sensory fibres that respond to stimuli such as cold and pressure, as well as fast/first nociception.

Professor Sommer continued: "Here, we found that the response to the electrical stimulation of these fibres in the fibromyalgia patients was lower. Wherever we stimulated – foot, hand or face – it was lower compared to healthy controls and compared to the group of patients with clinical depression that we had recruited as an extra control."

Furthermore, skin biopsies from subjects' legs were harnessed in order to gauge the number of nerve fibres in the epidermal layers of the lower leg and upper thigh, the results clearly demonstrating that, on average, fibromyalgia patients possessed lower numbers of nerve fibres than both controls and depressive patients.

The link between physical pain and depression is a familiar one, but depression is not necessarily a feature of fibromyalgia, thus the study was expanded to compare subjects with unipolar depression versus healthy controls. "The reason why we recruited this control group with depression but no pain was that many patients with fibromyalgia syndrome have depression, and there is an overlap between chronic pain and depression," said Professor Sommer. "We know that, but we wanted to be sure that we are not measuring things related to the depression."

At the present time, the number of different drugs that are used for the treatment of fibromyalgia are relatively few. While antidepressants work for some patients, the basis for their effect is not understood, and they may not address all associated symptoms. "We don't really know why they work," said Professor Sommer. "We know that they have an effect on the pain which is independent of their effect on depression. In some of the clinical trials that were done with fibromyalgia patients, depression was an exclusion criterion, so only patients without depression could be included, and these patients responded to the antidepressants – they had less pain. So the effect is different.

"There are many theories: it could be that antidepressants are strengthening the descending pain inhibitory system, which works with noradrenaline and serotonin; or that the antidepressants silence some types of sodium channels. These are the main theories, i.e. that they work independently from the antidepressant effect."

Bringing this work together with other research areas such as genetics and inflammation is something which Professor Sommer hopes will allow for greater understanding of the subgroups within the fibromyalgia diagnosis. "So far, we have shown that something is wrong with these small nerve fibres," she said, adding: "of course, we would like to know what is wrong, and why.

"The 'what' needs other techniques, in particular neurophysiological techniques, showing what these fibres are actually doing, what their electrical properties are. This is not something that our group is doing, but we know that other research groups in the world are doing this at the moment."

Professor Sommer continued: "Then, why are these fibres degenerating or becoming diseased? There are lots of possibilities that could be integrated with a general concept on chronic widespread pain and fibromyalgia, starting with things that happen in childhood. With genetics and stressful influences later in life, that might change the organism's balance of hormones, cytokines, and so on, which could then damage the nerve fibres. In this body of theory of how this could all fit together, there are many points at which we can now start to do further studies."

> *"There are lots of possibilities that could be integrated with a general concept on chronic widespread pain and fibromyalgia... In this body of theory of how this could all fit together, there are many points at which we can now start to do further studies."*
>
> *Claudia Sommer (University of Würzburg, Germany)*

**Claudia Sommer will give her Plenary Lecture 'Understanding pain in somatoform disorders,' this morning from to: 11:00-11:45 in the Auditorium.**

**References**

1. Uçeyler N et al. Small fibre pathology in patients with fibromyalgia syndrome. Brain. 2013, March 9 [Epub ahead of print]

JUNIOR SCIENTISTS SYMPOSIUM

S.12 **New targets for treating depression and anxiety** Room J Monday 7 October **09:00-10:40**

# Could sleep hold therapeutic answers for anxiety and depression?

The relationship between sleep and depression remains unclear, with insomnia being common while some individuals may suffer hypersomnia. Kara Panetta (University of Dundee, UK) will speak during this morning's Junior Scientists symposium, presenting recent work on the topic of the interplay of histamine and serotonin, both known to have some involvement with the wakefulness cycle. Speaking to *ECNP Daily News* ahead of the congress, she described the motivations for her meticulous cell clamping studies and how she is taking her work forward.

Ms Panetta's findings in histamine and serotonin have emerged from research into novel sleep therapies – an effort to develop drugs that result in fewer side effects than those presently available. Referring to how this could be applied to the study of anxiety and depression, Ms Panetta said: "I come at it from a sleep point of view. There is some suggestion that if you are restricted in certain parts of your sleep, it can either prevent or lead to depression. One study suggests that if you prevent patients from experiencing REM sleep, it actually produces for the next day an antidepressant-like effect.

"In my presentation in Barcelona, I am going to be talking about histamine and how it can influence [serotonin neurons]. The serotonin neurons themselves in the brain have a wide range of functions. The most important and perhaps most well known is the role they play in our emotional state. When there is a dysregulation in serotonin neurons, we can experience periods of depression or anxiety, which obviously are two big problems in psychiatry. For me, I was most interested in how serotonin neurons are involved in regulating our sleep and wake cycle. It is currently expected that serotonin neurons function to promote wakefulness in humans and animals."

Ms Panetta then went on to describe her own studies, which arose from the well-known observation that antihistamine medication causes drowsiness; this effect is most prominent in first generation antihistamines, which are known to cross the blood-brain barrier (whereas the second generation do not and hence do not cause drowsiness). Upon entering the brain, first generation antihistamines target the H1 histamine receptor subtype. Ms Panetta continued: "These antihistamines will stop or inhibit the H1 receptor from working. Normally histamine, like serotonin, promotes wakefulness. So if you stop this particular receptor, you stop the promotion of wakefulness and that is why we get drowsy."

Ms Panetta initially investigated the modulatory role of the H1 receptor by taking recordings from serotonin neurons in the dorsal raphe nucleus. However, a literature search led Ms Panetta to the finding that three of the four histamine receptor subtypes in the brain, namely H1, H2 and H3, occurred within the dorsal raphe nucleus. By investigating all three histamine receptor subtypes, Ms Panetta was able to exclude the involvement of the H2 and H3 receptors in modulation of serotonin neurons, while forming an understand of exactly how histamine bound to H1 receptors. She demonstrated the modulatory capability of histamine on serotonin neurons and, moreover, that histamine tone is present in the dorsal raphe nucleus, by performing whole-cell voltage clamp recordings from putative serotonin neurons while applying selective ligands to witness their effect on histamine conductance. "Initially I applied histamine and checked that I could block it," she said. "You can do this by using a neutral antagonist or an inverse agonist. A neutral antagonist binds to the same receptor that the histamine molecule would and it blocks histamine; it can either compete directly or do it in a non-competing way. An inverse agonist binds to a receptor which can be active without the presence of the histamine molecule; so we term it as being either spontaneously active or constitutively active. What that tells us is that if you give an inverse agonist and you see the opposite of what you would normally see with the agonist (in this case histamine) it suggests that these receptors are spontaneously active."

> *"Spontaneous turnover of histamine [affects] serotonin neurons. Because they are so closely linked, it is possible that the brain has its own regulatory mechanism to prevent too much serotonin firing."*
>
> *Kara Panetta (University of Dundee, UK)*

The purpose of this work, Ms Panetta explained, was to target both serotonin and histamine in an effort to develop a more effective drug therapy for sleep disorders. Whilst blocking each individually proved effective in increasing sleep, blocking both did not result in any additive effect. She said: "I think the reason for that is possibly because of what I've discovered – that this spontaneous turnover of histamine is affecting serotonin neurons. Because they are so closely linked, it is possible that the brain has its own regulatory mechanism to prevent too much serotonin firing. So it is good in a way to have an explanation for why we were having problems developing a drug that was hitting both of these targets."

Ms Panetta's current *in vivo* studies extend her single-cell *in vitro* work. She said: "I have done recordings from whole animal serotonin neurons in a similar fashion to my previous work. The animal is anaesthetised, so there is a bit of conflict there, but it is certainly allowing us to gain more of an insight into what these drugs are doing when you have the entire brain intact. To an extent, this supports my *in vitro* studies, however I believe that due to the presence of other regulatory inputs to the dorsal raphe nucleus being intact (a very important one being the noradrenergic drive from the locus coeruleus), the effect is not always as clear as what I had seen *in vitro*."

**Ms Panetta will give her presentation as part of the Junior Scientists symposium, 'New targets for treating depression and anxiety: preclinical and clinical studies', at 09:00-10:40 today in Room J.**

PRECLINICAL RESEARCH

S.16 **Towards treatment of cognitive deficits in Down syndrome** Room J Monday 7 October **14:30-16:10**

# Cognitive restoration in Down syndrome

Down syndrome (DS) is the most common form of intellectual disability in the population,[1] but cutting-edge research is paving the way in hopefully offering treatment that can recover impaired cognitive function, delegates will hear this afternoon in a comprehensive session dedicated to the disorder.

One of the most proposed theories for the cause of DS has been an imbalance between inhibitory and excitatory neurotransmission in the brain, and indeed many of the presentations during the ECNP session will focus on receptors and inhibitory/excitatory approaches that are hoped to offer therapeutic power in this arena.

Speaking to *ECNP Daily News*, Benoît Delatour (Université Pierre et Marie Curie, Paris, France) began by discussing his previous work in Alzheimer's and how it led him to new research in DS. "Down syndrome is a trisomy of chromosome 21, and on this chromosome you have the APP [amyloid precursor protein] gene, a precursor of the protein beta amyloid, which is key in Alzheimer's disease," he said. "Most of the people with Down syndrome that are in their 50s develop Alzheimer's disease, so that is the original connection for me to begin to work in this field."

Dr Delatour stressed that while at the current time there are no clinical treatment options available that can address the huge learning, memory and attention deficits associated with DS, the therapeutic potential of pharmacological neurotransmission intervention has gathered momentum thanks to several studies in specifically engineered, Ts65Dn mice (a genetic model for DS). "Some people began to use antagonists against GABA, an inhibitory neurotransmitter in the brain," he said.

Many of these studies were able to rescue cognitive phenotypes in mice – as well as impaired brain plasticity. While extremely promising results, the GABA antagonists themselves had a risk of promoting seizures and epilepsy, due to the increased constraints on the inhibitory system (especially at high doses). With this in mind, Dr Delatour and his team identified another approach which they hoped would not cause side effects: "We decided to use inverse agonists instead, which are targeted against a specific subunit for the GABA-A receptor, and are known to be linked specifically to learning and memory functions," he said.

This inverse agonist, α5IA, targets the α5 subunit of the GABA-A receptors, and in the study it was shown to increase the learning capacity of Ts65Dn mice, when compared to wild type controls. "Using the drug at specific dosages, we showed that the inverse agonist was able to reverse deficits in at least two different learning and memory paradigms," continued Dr Delatour.

The first of these tests was a standard Morris water maze, in which previously-impaired Ts65Dn mice were able to perform as well as wild type controls if they were given the inverse agonist. Similarly, the study also employed an object recognition task in which two objects, one familiar and one novel, were presented to both groups. "The naturally tendency of the mouse – if it has a good memory – is to investigate and explore the novel object: this is a normal response," said Dr Delatour.

While untreated Ts65Dn mice were impaired in the task, a single injection of inverse agonist before testing was able to promote improved memory and learning functionality, and crucially, the drug did not cause any side effects. "We did not see differences in terms of anxiety or general locomotor activity when comparing placebo versus treated mice," said Dr Delatour.

> *"These effects are not acute; they are observed in the long term, and they can be sustained by the fact that the drug promotes some restoration of the brain morphology and activity in the long term."*
>
> *Benoît Delatour (Université Pierre et Marie Curie, Paris, France)*

"Also, we did an extensive study after chronic administration of the drug to check whether it induces lesions in various organs, and this was not the case. There was a previous paper from a German group showing that various metabolites from the drugs could promote the formation of crystals that could be deleterious, but we did not see this using our formulation."

Looking into the mechanism of action of the drug, and its positive effects on cognition, Dr Delatour's group determined that the inverse agonist was able to enhance the activity of so-called immediate early genes (IEGs) – genes that are normally activated following the types of training or learning paradigms used in the mouse models. He added: "And also we did a study at the level of gene expression, using a transcriptomatic approach, and we showed that several dysregulated gene pathways that are normally observed in trisomic mice were restored to normal levels following treatment with the drug.

"So we have at least two different mechanisms showing that the drug can act on immediate early genes and also on gene expression. That could be the basis of long term effects of the drug, because we also have preliminary data showing that after washout, several weeks after ending the treatment, we still have beneficial effects of the drug in terms of cognition. Therefore these effects are not acute; they are observed in the long term, and they can be sustained by the fact that the drug promotes some restoration of the brain morphology and activity in the long term."

Looking to the next steps, i.e. human trials, Dr Delatour pointed out that while several companies are now recruiting for studies using similar drugs, it would still be some time before data is available from

*Continued on page 8*

PRECLINICAL RESEARCH

**Towards treatment of cognitive deficits in Down syndrome** Room J **Monday 7 October** 14:30-16:10

# Cognitive restoration in Down syndrome

*Continued from page 7*

clinical trials. However, he added that at least safety profiles for the compounds had already been mostly obtained, thanks to previous work by David Nutt, who explored the use of these types of drugs for the alleviation of cognitive impairment caused by alcohol consumption. "It at least means that all the toxicity studies have been performed [in order] to show that the drug does not have side effects and can be safely used in humans," said Dr Delatour in closing.

**Dr Delatour will delve into his work with selective GABA-A inverse agonists for cognitive restoration during his presentation in the session 'Towards treatment of cognitive deficits in Down syndrome', held at 14:30–16:10 this afternoon in Room J.**

**Reference**

1) J Braudeau et al. Specific targeting of the GABA-A receptor a5 subtype by a selective inverse agonist restores cognitive deficits in Down Syndrome mice. Journal of Psychopharmacology. (2011); 25(8): 1030–1042

*"Using [5IA] at specific dosages, we showed that the inverse agonist was able to reverse deficits in at least two different learning and memory paradigms."*

*Benoît Delatour (Université Pierre et Marie Curie, Paris, France)*

LIVE FROM BARCELONA

**The 2013 Neuropsychopharmacology Award**

# Celebrating pioneering work in brain regeneration therapy

The ECNP Neuropsychopharmacology award recognises innovative and distinguished research achievements in neuropsychopharmacology and related fields. Alternating between basic science and clinical research each year, recipients of the award are invited to present a plenary lecture at the ECNP Congress, as well as the submission of a review article for publication in *European Neuropsychopharmacology*.

The recipient of the 2013 Award is Anders Björklund (Wallenberg Neuroscience Centre, Lund University, Sweden) for his work in novel therapeutic targets for neuroprotection and disease modification in Parkinson´s disease.

"Over the last decades Professor Björklund and his team at Lund University have pioneered the use of cell transplantation in Parkinson's disease, and they played a key role in the exploration of stem cells for brain repair," said ECNP Award Committee Chair, Julien Mendlewicz as he introduced the award during the Keynote Session on Saturday evening. "I invite all of you to come and listen to this truly very exciting and pioneering lecture."

Stepping up to the podium to accept his award, Professor Björklund expressed his gratitude: "Thank you very much; I am deeply honoured to be selected for this award. I am particularly happy also that brain repair is recognised by ECNP. It is field that is not traditional neuropsychopharmacology, but is increasingly more important, and I think that with the fantastic development that ECNP is showing, my field is going to be more of focus. It is very much the future of how we can approach damage in the brain, and 'brain damage' is not just what may be the result of trauma but something that we live with in many ways.

"I'm also happy, as a founding member of the board of the European Brain Prize, that the ECNP has formed a partnership with the European Brain Prize foundation. This is something that we are very happy to see, and we hope that this is going to become a long term friendship and collaboration."


**Anders Björklund**

In the lecture, held yesterday afternoon, Professor Björklund outlined the search for treatments that can interfere or modify the disease mechanism underlying Parkinson's disease. "The current treatments are symptomatic, that is to say that if you take L-dopa – which is the standard treatment for Parkinson's – it is capable of alleviating motor symptoms, and helping patients to move better, and in fact in the early status of the disease the drug treatment is quite effective; it can help patients over several years," he told *ECNP Daily News*.

"However, it doesn't affect the underlying progressive degeneration of neurons which is the main cause of disability. So this means that the next generation in treatment of Parkinson's, and it is the

*"Thank you very much; I am deeply honoured to be selected for this award. I am particularly happy also that brain repair is recognised by ECNP. It is field that is not traditional neuropsychopharmacology, but is increasingly more important."*

*Anders Björklund (Wallenberg Neuroscience Centre, Lund University, Sweden)*

LIVE FROM BARCELONA

## The 2013 Neuropsychopharmacology Award


*"There is a clear possibility that restoring Nurr1 function will also restore functionality in surviving but dysfunctional neurons, which is an interesting therapeutic opportunity."*

*Anders Björklund (Wallenberg Neuroscience Centre, Lund University, Sweden)*

same in other relative neurodegenerative diseases like Alzheimer's for example, is to find ways of interfering with the underlying disease."

These interventional strategies fall under three broad categories of neuroprotective treatments, disease modifying treatments (slowing the progression of the disease) and 'restorative' treatments (those which can restore functionality in already affected patients), and Professor Björklund discussed two avenues of his work engaged in finding effective Parkinson's treatments within these categories. "The first is that we've generated in rodents, primarily in rats, a condition where we increase the level of the disease-causing protein which is called alpha-synuclein," he said.

Elevated levels of the protein can cause adverse cellular changes, impacting mechanisms that include the genetic machinery, for example. "We are particularly interested in these dopamine neurons that are central to Parkinson's pathology, and we have, in collaboration with colleagues, identified one such gene-regulating factor that is directly affected by alpha-synuclein when it is present in increased amount. It is called Nurr1," said Professor Björklund.

Nurr1 (Nuclear receptor related 1 protein) is a transcription factor that has been identified in animal models as a key mechanism for dopamine neuron development and survival, thus when Nurr1 is dysfunctional, so too will be the development of the neurons. "What has put Nurr1 into focus for Parkinson's research is that it is seen to be down regulated or affected in Parkinson's patients," added Professor Björklund.

"In ongoing Parkinson's disease, if you analyse the dopamine neurons affected by the disease, Nurr1 is present at abnormally low levels, and when that happens in animals we know that this means that a broad set of genes are affected in terms of reduced expression. The expression of many a number of genes is dependent on Nurr1, so there is an impact of both the function of a cell and its ability to resist damage and toxicity."

**Anders Björklund (right) accepts his award from ECNP Award Commitee Chair, Julien Mendlewicz**

Professor Björklund stressed that evidence points towards Nurr1 as being able to individually tip the balance of Parkinson's disease – reduced levels leading to cells that will be more vulnerable to alpha-synuclein-induced toxicity, and above normal levels blocking toxic processes almost completely. In fact it is likely that Nurr1 offers all three kinds of positive effects for the disease: protection, modification and restoration.

He added: "The synthesis of dopamine, its storage vesicles, and its release mechanism and the reuptake mechanism are all regulated by Nurr1. So there is a clear possibility that restoring Nurr1 function will also restore functionality in surviving but dysfunctional neurons, which is an interesting therapeutic opportunity. The fact that the patients for many years have more neurons than they actually make use of, and if one can restore functionality and actually restore function in the dysfunctional cells, it means that they will recover function that previously has been lost. So that would again be a direct restorative mechanism without having to generate any new neurons."

With these observations in mind, Nurr1 is emerging as an interesting therapeutic target for drug research, being broadly classed as 'drugable' owing to its relatively straightforward administration to the brain and to the neurons themselves.

As Professor Björklund underlined, it still remains to be seen whether Nurr1 therapy will have the same benefit in human trials, but it is clear that if this promising avenue of research does come to fruition, it will offer a revolution for Parkinson's disease patients.

"There is ongoing development in pharmaceutical companies and in biotech companies that are activating Nurr1. There are several projects where candidate drugs are now being tested as Nurr1 –activating drugs, and in the next period one can expect trials in patients."

He continued: "There are now at least four companies we're aware of that are at various stages to take this towards the clinic. And one can hope perhaps that at least one of these approaches will reach patients. But you know it's famous these days how slow it is just to get the approval for patient studies, and then of course the first round has to be safety focussed. But let me say in the next couple of years I think one can be hopeful that clinical trials will be started."

LIVE FROM BARCELONA

ADHD in adults

# Treat me differently: ADHD pharmacotherapy in adults


Adult attention deficit-hyperactivity disorder (ADHD) is now a well-recognised diagnosis, whose clinical profile naturally differs from the symptomatology of childhood ADHD. In a session held yesterday that was dedicated to the understanding of the adult form of the disorder, Esther Sobanski (Central Institute of Mental Health, Mannheim, Germany) spoke of how the psychopathology in adults is distinct, and the new treatment strategies that aim to address this.

Dr Sobanski commented how, while it was once thought that individuals recovered from ADHD as they moved into adulthood, more often than not symptoms actually persist. Like many disorders, persistence of ADHD is associated with a development in the manifestation of symptoms as well as comorbid conditions, and ADHD in adulthood is associated with a tendency toward risky or sensation-seeking behaviours such as dangerous driving and a general proneness to accidents. Speaking to *ECNP Daily News*, she added: "We found in a clinical sample of adults with ADHD a lifetime prevalence of other psychiatric disorders of 77.1% compared to 40% in age and gender matched population-based controls. Common comorbid disorders in adults with ADHD include mood, anxiety and substance use disorders."

With an increasing awareness of the persistence of ADHD throughout life comes a necessity for improved tailored therapy to address the evolution of patients' needs in adulthood. Dr Sobanski spoke of our current knowledge of medications and talking therapies, while emphasising the questions that urgently need addressing. "Pharmacotherapy with available medications, including stimulants and non-stimulants, has been shown to be highly effective with effect sizes up to 1 in meta-analyses for reduction of ADHD core symptoms, which are similar to effect sizes for medication treatment found in children with ADHD. Some studies further show that stimulants and non-stimulants can reduce ADHD-associated symptoms, like emotional dysregulation or sleep problems, and that pharmacologically treated ADHD-patients with residual ADHD core symptoms have additional benefit from disorder-oriented psychotherapy. Preliminary and recent research has shown that medication can have an impact beyond ADHD symptoms and can also improve psychosocial functioning like managing on-road driving traffic. However... only every second to fourth patient profits from stimulant therapy [and] only every 3rd to 5th patient benefits from non-stimulant therapy.

> *"Awareness and specific treatment of adult ADHD needs to be increased throughout Europe... Future therapy studies should not only address the impact of treatment on ADHD core symptoms but also on psychosocial functioning."*
>
> *Esther Sobanski (Central Institute of Mental Health, Mannheim, Germany)*

"Some first studies show that medication adherence and persistence to treatment are issues that need to be addressed for optimal treatment outcome. Pharmacological treatment approaches and the evaluation of treatments that target not only ADHD core symptoms but also co-morbid psychiatric disorders like alcohol use or social phobia are needed. This is due to the high comorbidity of ADHD with these disorders and due to interaction of the disorders in terms of disorder severity, outcome and psychosocial functioning. Future studies should also address special and currently still neglected populations, like prison inmates, as all available data show that the prevalence of ADHD is high in the population of prisoners, who currently in most cases do not receive specific pharmacological treatment of their ADHD symptoms."

Dr Sobanski concluded her discussion with *ECNP Daily News* by explaining that ADHD in adults can be tackled only when treatment protocols are tested in a diverse set of individuals with the condition, in order to improve coping and social functioning in everyday situations. She highlighted the importance of drug testing by which to establish the best pharmacological tools in the treatment battery for ADHD, saying: "Awareness and specific treatment of adult ADHD needs to be increased throughout Europe. Available data point to a diagnostic prevalence of adult ADHD in clinical samples of less than 0.5%. Treatment algorithms need to be established for ADHD and co-morbid psychiatric disorders, and for ADHD with treatment failures to stimulant and non-stimulant treatment approach. Future therapy studies should not only address the impact of treatment on ADHD core symptoms but also on psychosocial functioning. They should also investigate the impact of treatment adherence and persistence on treatment outcome and how to ensure treatment adherence and persistence."

LIVE FROM BARCELONA

**CBT in psychosis**

# Psychotherapy in psychosis

*Continued from page 4*

experiments to find out how reality is and if it is diverted from how they thought it would be, or congruent with alternative explanations."

Professor Van der Gaag explained that trivial antecedents, when combined with a dramatic interpretation, can result in fear and avoidant behaviour (such as not wanting to go out alone). These thought patterns are fed by experiences of the past, such as bullying in school, and suffering from one mental disorder – or a symptom thereof, such as hallucinations – can make an individual more susceptible to others. "Hallucinations seem to be quite a pluripotent predictor of all kinds of psychopathology," he said. "Once you hear voices, the odds ratio for also having PTSD is about 26, depression about 16, addiction 5, social phobia 8. These are quite high odds ratios, especially when you compare them with cannabis use (3) and urban upbringing (about 1.4). It is also the other way around: if you have a person who is depressed, the odds ratio for them also hearing voices is about 4.5. So having depression is also a risk factor for psychotic experiences, and it is the same for anxiety (about 3). A lot of this psychopathology is mixed up and that is something to be aware of."

> *"Is [CBT] effective? Yes, it is effective in many disorders, including psychotic disorders. It is in many guidelines all over the world."*
>
> *Mark Van der Gaag (Parnassia, Den Haag, the Netherlands)*

Outlining some of the effective techniques that are applied in hallucinations, Professor Van der Gaag noted competitive memory training (COMET), metacognitive training, treating post-traumatic stress syndrome in psychotic disorders, treating demoralisation and negative symptoms, virtual reality exposure treatment, and prevention and treatment of subclinical psychosis, as effective treatment strategies. He stressed that treatments are increasingly proving both effective and safe, as well as dealing with comorbid disorders such as anxiety, depression and PTSD.

LIVE FROM BARCELONA

**The ECNP Media Award**

# *Brain: The Inside Story* receives ECNP honour

Launched last year, the ECNP Media Award was established to recognise exemplary contributions in any medium to creating awareness of brain disorders and making the underlying scientific concepts accessible to the general public. This year the award was presented to Monica di Luca (University of Milan, Italy), for the travelling exhibit, Brain: The Inside Story. The exhibit was organised collaboratively by the American Museum of Natural History (USA), with Codice, Idee per la cultura and Comune di Milano in Italy; Guangdong Science Center in Guangzhou, China; and Parque de las Ciencias in Granada, Spain. Alongside the honour itself, the award also features a €5,000 prize.

Introducing the award was ECNP President Joseph Zohar, who said: "Last year, we started the ECNP Media Award in order to open up a channel to the public. The idea of this Media Award is to highlight those who contribute to destigmatising disorders of the brain in any type of medium within Europe. This is our mission as a college, and I am very pleased to announce it for the second time. This is an exhibition that took place in Spain and now it is in Milan. More than half a million visitors have been to see this exhibition."


Daniela Tardito accepting the Media Award on Saturday evening

> *"The idea of this Media Award is to highlight those who contribute to destigmatising disorders of the brain in any type of medium within Europe. This is our mission as a college, and I am very pleased to announce it for the second time."*
>
> *Joseph Zohar (Tel Aviv University, Israel)*

Curated to appeal to all ages, the exhibit gives visitors an insight into the cutting edge of neuroscience, incorporating imaginative artworks and interactive models to demonstrate the functions of healthy brains as well as study and treatment methods for diseases of the brain. Exploring basic and higher cognitive functions of the brain such as sensory and emotional processing, reasoning, and memory, the exhibit also emphasises the dynamic structural and functional plasticity of the brain. Finally, visitors can witness the latest exploratory techniques, such as brain-computer interfacing, deep brain stimulation and transcranial magnetic stimulation.

Accepting the Media Award on behalf of Dr di Luca, who organised the Milan exhibition, was Daniela Tardito (University of Milan, Italy). Reading a message from Professor di Luca, she said: "We are very proud that this great brain exhibit is here in Milan. The exhibit starts just a few days from now on October 17, so we are now in the hectic final phases. The exhibit brings the latest neuroscientific research to all, highlighting the brain's amazing ability to rewire itself in response to experience, disability or disease states, and showcasing new technology that we as scientists use to study the brain. Interactivity, puzzles, brain scanning imaging and spectacular models will be included in the largest brain exhibit we have ever had in Italy in an area of more than 1,000 square metres. The award honours not only me as the presenter but all the people that have been involved in such as enterprise for more than 18 months now. Please accept my sincere thanks and I wish you a successful meeting!"

> *"The exhibit brings the latest neuroscientific research to all, highlighting the brain's amazing ability to rewire itself in response to experience, disability or disease states, and showcasing new technology that we as scientists use to study the brain."*
>
> *Monica di Luca (University of Milan, Italy).*

**Brain: The Inside Story is showcasing in the Museum of Natural History in Milan, Italy, from October 17, 2013 untill April 13, 2014.**


# ECNP Certificate

A European-wide qualification for junior researchers in the science and treatment of disorders of the brain.

To find out more, please visit:
**www.ecnp.eu/certificate**

ECNP *neuroscience applied*

EDUCATION

E.03 **Binge eating obesity is a food addiction** Room M2 Monday 7 October **09:00-10:40**

# Sizeable debate over obesity addiction

A session dedicated to exploring the arguments in favour of and against the addiction hypothesis in obesity takes place this morning at ECNP Congress. In an interview with *ECNP Daily News*, Hisham Ziauddeen (University of Cambridge, UK), whose work involves the exploration of the neurobiological basis for eating behaviour, spoke of the problems of the addiction model and the place that he hopes his pharmacological work will have in dealing with obesity in the future.

Dr Ziauddeen's current work focuses on the role of dopaminergic systems in food reward, with particular emphasis on antipsychotic-induced weight gain. "The opioid work that we have been doing is a collaboration with GlaxoSmithKline," he began. "This is a set of trials of a new opioid antagonist drug. The hypothesis is that the drug is likely to change how much people are motivated towards food and their hedonic experience of food and consequently eat less and lose weight. However while we have seen effects on food intake in the lab, only a small subgroup of people who have a more active version of the opioid receptor that this drug targets, lost weight. We are currently investigating this further."

Dr Ziauddeen has written about the conceptual problems of the food addiction model in two separate papers on obesity. [1][2] Summarising their message, he said: "Both of the papers that we've written essentially say that there isn't really evidence for a food addiction syndrome, let alone one that applies to obesity. Our point of view is essentially one that is widely accepted, which is that obesity is quite a complex syndrome and that there are several pathways to obesity.

"If food addiction has any role to play at all in obesity, it is likely to play a very small one. So far, in humans, definitely, we don't have any evidence to say that this is a real entity. There are multiple psychological, metabolic, neurological and environmental factors that all feed in to this complex pathway, at the centre of which lies energy balance. Ultimately, obesity results from taking in more energy than you actually need. How you get to that excess intake over expenditure can happen in multiple ways."

> *"I think the way that most people are looking at pharmacotherapy in obesity is that, given that it is a complex problem with multiple causes, you have to use treatments that target the multiple causes."*
>
> *Hisham Ziauddeen (University of Cambridge, UK)*

Dr Ziauddeen's most recent work considers the use of CNS biomarkers signifying the metabolic, cognitive and behavioural changes that take place in obesity in the early development of obesity drugs. [3] However, the multiple strands from which obesity emerges means that pharmacology offers the potential for successful therapy only as part of a broader strategy. "There are so many complex factors that feed into it," he said. "If we take a system that is very close to the cause, for example the appetite control system (because whatever you want to consider in terms of environmental and social factors,

they all ultimately increase intake via the appetite and eating system, of which the reward system is a critical part), if you want to treat that system hard enough to stop all the other influences having an effect, that is going to be very difficult. It can be very difficult to safely target a system that is that close to intake without causing side-effects or causing other problems.

"I think the way that most people are looking at pharmacotherapy in obesity is that, given that it is a complex problem with multiple causes, you have to use treatments that target the multiple causes. Obesity pharmacotherapy will be one potential treatment, but it will not be used on its own. In fact, the official guidelines from the Food and Drug Administration and the European Medicines Agency say that if you are trialling an anti-obesity drug, it is made very clear that it is supposed to be used as an add-on treatment. If you conduct your trials, you should at least have some sort of exercise or lifestyle intervention alongside the treatment."

Identifying the mechanisms that can lead an individual to consistently over-consuming is a key step towards better weight management, and Dr Ziauddeen highlighted the work of John Blundell (Institute of Psychological Sciences, University of Leeds, UK) in investigating the role of non-fat tissues in regulation of intake. "Fat mass doesn't actually consume that much energy; it is fat-free mass (i.e. largely your muscle mass) that determines how much energy you need. Usually when people gain weight, they tend to also gain fat-free mass. One of the things [Professor Blundell] is exploring is the role of fat-free mass in determining intake and the role it has in maintaining overall weight or obese status. I think that is a very interesting question, because the answer is not going to come to us by just looking at fat."

> *"Pharmacotherapy is very good at the individual subject level, but when we are thinking on the population level we need to think about public health strategy."*
>
> *Hisham Ziauddeen (University of Cambridge, UK)*

Dr Ziauddeen concluded by stressing the role of research in influencing and forming future health policy. "There are several efforts already going on. Here at Cambridge we have the Behavioural Health Research Unit which is a Department of Health-funded body that is trying to find evidence for the role of environmental factors in promoting unhealthy behaviours, such as overconsumption and smoking etc., to see how that can inform policy decisions. Ultimately, we will have to think about making major environmental changes to accompany whatever other insights we get, to make a difference at the population level. Pharmacotherapy is very good at the individual subject level, but when we are thinking on the population level we need to think about public health strategy."

**Dr Ziauddeen will present, 'Obesity and the brain: how convincing is the addiction model?' as part of the session entitled 'Binge eating obesity is a food addiction', taking place this morning at 09:00-10:40 in Room M2.**

**References**

1. Ziauddeen H, Fletcher PC. Is food addiction a valid and useful concept? Obes Rev. (2013); 14(1):19-28.
2. Ziauddeen H, Farooqi IS, Fletcher PC. Obesity and the brain: how convincing is the addiction model? Nat Rev Neurosci. (2012); 13(4):279-86.
3. Ziauddeen H, Fletcher PC. Central nervous system biomarkers for antiobesity drug development. Drug Discov Today. (2013); pii: S1359-6446(13)00282-1.

BRAINSTORMING @ ECNP

BS.7 **Alcohol, drugs and violence: neuropsychopharmacology in court** Room M1 Tuesday 8 October **07:45-08:45**

# Neuropsychopharmacology in the courtroom

The use of neuropsychopharmacology in legal cases of delinquency will come under scrutiny in a brainstorming session dedicated to the topic tomorrow morning at ECNP Congress. Robbert J Verkes (Radboud University Medical Centre, Nijmegen, the Netherlands) will speak at the session together with Adrian Raine (University of Pennsylvania, USA). Dr Verkes spoke to *ECNP Daily News* ahead of the session, outlining the ethical, methodological and dissemination issues that urgently need to be resolved in order for judges, juries and the public to be properly informed about the possibilities and drawbacks of current scientific knowledge.

Neuroscience techniques are increasingly used in order to form cases in court, yet Dr Verkes was wary about the validity of such practise on the scale of n=1. Summarising the motivation for the brainstorming session, he said: "We know that antidepressants can have violent behaviour as a side effect. We know that this is an association in the general community, but how does it apply to individuals? That is one thing I want to discuss with other people in the brainstorming session. More and more, for instance, neuroimaging images are presented in court, but does it make sense to do this?"

While the use of expert witnesses has been exploited in the past, Dr Verkes highlighted the register of experts that is now in place in the Netherlands. "Experts have to apply to be in that register," he said. "I think it is very good, because previously people were used as experts who were said to be a professor in something, which makes their statements authoritative, but they might have been a professor in a field too far away from clinical neuroscience."

> *"We know that antidepressants can have violent behaviour as a side effect. We know that this is an association in the general community, but how does it apply to individuals?"*
>
> *Robbert J Verkes (Radboud University Medical Centre, Nijmegen, the Netherlands)*

Dr Verkes' work involves the study of the effects of different illicit drugs on social behaviour and impulsiveness. In this way, his work contributes to the understanding of the effects of drugs and to distinguish the realities from the myths of drugs use. He has also been involved in a court case in the Netherlands, where the court requested an experimental study of an individual suspect in order to investigate the claim that a murder was precipitated by the administration of a selective serotonin reuptake inhibitor. He said: "It could be that the perpetrator showed violent behaviour only after starting with antidepressants. What are the issues involved in these kinds of experiments and how can we interpret the results?"

Dr Verkes noted that, interestingly, legal penalties in the Netherlands may well be increased if illicit drugs are demonstrated to have been involved in violent crimes. He said: "While that is easy to say, how is it operationalised? We can measure blood levels or urine levels of drugs, but how do we show that a particular individual was under the influence of drugs at the moment of the delict? And if one asks which drugs are important, then politicians would say, 'All drugs.' This is not very well informed by science! I think we, as experts from the field of neuropsychopharmacology, have to inform and to set these kinds of things in a proper framework."

The ethical basis for considering prescription drugs differently from drugs of abuse in delinquency cases is based on the principle of 'culpa in causa', an expression denoting that the blame in a crime is extended to the defendant's choice to create a situation whereby the crime is more likely to occur. Dr Verkes continued: "With illicit drugs, you are putting yourself in a position of risk – that is the opinion – and then the penalty is enhanced. But when prescribed drugs are involved, it is not culpa in causa – it is not your responsibility.

"Alcohol appears very often in court in violent behaviour. I have analysed all cases in the Netherlands for the last 15 years where the prescription of drugs was used in the defence, and it appeared that when alcohol was also involved the judge is saying that it is culpa in causa because of the alcohol. But this I think is too short an argument, because the leaflets that come with e.g. antidepressants only state that it is not advised to use them with alcohol. This is not proper information. You cannot then say that it is culpa in causa, because the individual had to have known that when he used alcohol and antidepressants together it would clearly enhance the risk for aggressiveness. Proper information to patients is needed, but also to the general public, the media, and to people involved in these cases."

> *"Proper information to patients is needed, but also to the general public, the media, and to people involved in these cases."*
>
> *Robbert J Verkes (Radboud University Medical Centre, Nijmegen, the Netherlands)*

**Dr Verkes will provide expert input alongside Dr Raine in the brainstorming session, 'Alcohol, drugs and violence: neuropsychopharmacology in court', chaired by Miro Jakovljevic (University Hospital Centre Zagreb, Croatia), taking place on Tuesday morning at 7:45-08:45 in room M1.**

INTERVIEWS

The ECNP Office

# Interview: **Godelieve Escartín**

The fundamentals required to organise the yearly ECNP Congress are vast, thus a successful, engaging and memorable meeting requires a great deal of work from talented teams behind the scenes. The ECNP Office is as the centre of each congress, tasked with the organisation, logistics and planning of wide-ranging list of aspects that include the congress venue itself, finances, the scientific programme, on-site publications and information, and overseeing their organisational partners Colloquium Brussels who in turn take care of registration, hotel reservations and onsite management.

The ECNP Office is also at the forefront of the yearly calendar activities, including the workshops, schools, seminars and other associated meetings, as well as the social media outlets, website communications and networking projects.

This year's ECNP Congress Project Manager, Godelieve Escartín, spoke to *ECNP Daily News* to describe the work her and the ECNP team have been doing to make sure the Barcelona Congress goes off with a bang.


**How early does planning have to start for each ECNP Congress, and what steps are taken?**

We start three years before the congress with details such as the budget, the contract with the venue, setting up of the scientific programme, starting the promotion of the congress and making divisions of the sessions rooms, exhibitions and general areas at the venue to make best use of it. One and a half years before the congress we start with all the remaining details, and this does not finish until the congress takes place.

**With such a wealth of suitable venues across Europe, how is the city for each ECNP Congress decided?**

We work with four rotating venues: Barcelona, Paris, Amsterdam and Vienna. These venues are very suitable for our congresses. As we have five main scientific sessions in parallel there are not many congress centres we can use. Furthermore the hotel capacity of a city is also important, as well as the possibility to easily travel to the city as we have around 6000 participants coming from all around the world.

**Could you tell us more about the typical process of planning that takes place between the ECNP Office and the other committees?**

The ECNP Office cannot organise the congress without the help of the members of the ECNP Committees. These people all work voluntarily for ECNP and are the experts in the field. The Scientific Programme Committee (SPC) is in charge of creating the scientific programme of the congress. Seventeen members of the SPC with all different expertise make a complete programme with the input they receive from the call for symposia proposals. At the office we arrange that all speakers and chairs are invited, all required information is gathered and that everything goes smoothly at the congress.

Besides the SPC, the Executive Committee also gives ideas to improve the congress in all aspects. Other committees, like the Scientific Advisory Panel (SAP), are in charge of the review of all scientific abstracts for poster presentations together with the Field Editor.

**The ECNP has so much more to offer than the Congress itself, so how much emphasis (and in what way) will be placed on making sure Barcelona communicates the wider reach of the College's activities?**

During the year we promote not only the congress but also all other activities we have, like the schools, workshop, seminars, ECNP certificate and membership, amongst others. At the congress we try to make participants aware of all other activities we have by giving away flyers, give information at the ECNP Plaza, show an ECNP promotion video and of course all information can also be found on our website.

**Refer to page 19 for the full list of ECNP Committee and ECNP Office members.**

ECNP INITIATIVES

## ECNP Travel and Poster Awards

# Congratulations!










## to Sunday's ECNP Travel Award and ECNP Poster Award winners

Congratulations to Sunday's ECNP Travel Award and ECNP Poster Award winners.

Both initiatives recognise outstanding contributions by junior scientists at the ECNP Congress. Specifically, the Poster Award identifies poster presentations that are truly exceptional, as determined by a dedicated and distinguished team of senior judges. Similarly, the Travel Award – established to encourage junior scientists to attend the congress – highlights the best abstracts by junior scientists in Europe, accepted for publication and presentation in at the congress. The abstracts are judged by a scientific review committee.

Both awards offer a grant of €500 and a commemorative certificate, and the winners will see their posters on permanent display in the Award area of the congress, as well as being published on the ECNP website.

### Travel Award winners

| | | |
|---|---|---|
| Albert Ferrés-Coy | Spain | P.2.a.010 |
| Sonia Torres-Sanchez | Spain | P.2.a.015 |
| Jelena Vrublevska | Latvia | P.2.b.028 |
| Andreas Hahn | Austria | P.2.b.044 |
| Nikolaos Kokras | Greece | P.2.b.045 |
| Daria Smirnova | Russia | P.2.b.060 |
| Marin Jukic | Israel | P.2.c.011 |
| Dina Popovic | Spain | P.2.d.012 |
| Tevfik Kalelioglu | Turkey | P.2.d.018 |
| Shimon Burshtein | Israel | P.2.d.025 |
| Philippa Rock | United Kingdom | P.2.d.031 |
| Kamilla Miskowiak | Denmark | P.2.d.033 |
| Malgorzata Maciukiewicz | Poland | P.2.d.034 |
| Chiara Fabbri | Italy | .2.f.014 |
| Clara Lopez-Sola | Spain | P.4.b.005 |
| Carla Nasca | Italy | P.4.c.007 |
| Lior Carmi | Israel | P.4.d.001 |
| Elizabeth Rickenbacher | Portugal | P.4.e.011 |
| Bruno MDC Godinho | Ireland | P.5.c.002 |

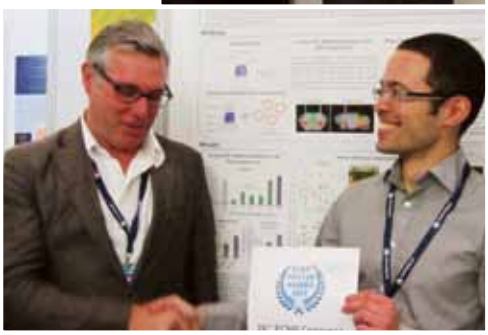
### Poster Award winners

| | | |
|---|---|---|
| Andreas Hahn | Austria | P.2.b.044 |
| Marin Jukic | Israel | P.2.c.011 |
| Esther Via | Spain | P.4.b.001 |
| Bruno Godinho | Ireland | P.5.c.002 |









## Word jumble

### Unjumble the words to solve the clues...

Main component of amyloid plaques — A E A B T

Frontal lobe region linked to speech production — C A R O B

Inner ear structure important for hearing — O E A C C H L

Discoverer of classical conditioning — P L O V V A

"One cannot step twice into the same river" — L I T C H E S A U R

Almond shaped nuclei, part of the limbic system — M Y L D A A A G

A large division or section of the brain — B L O E

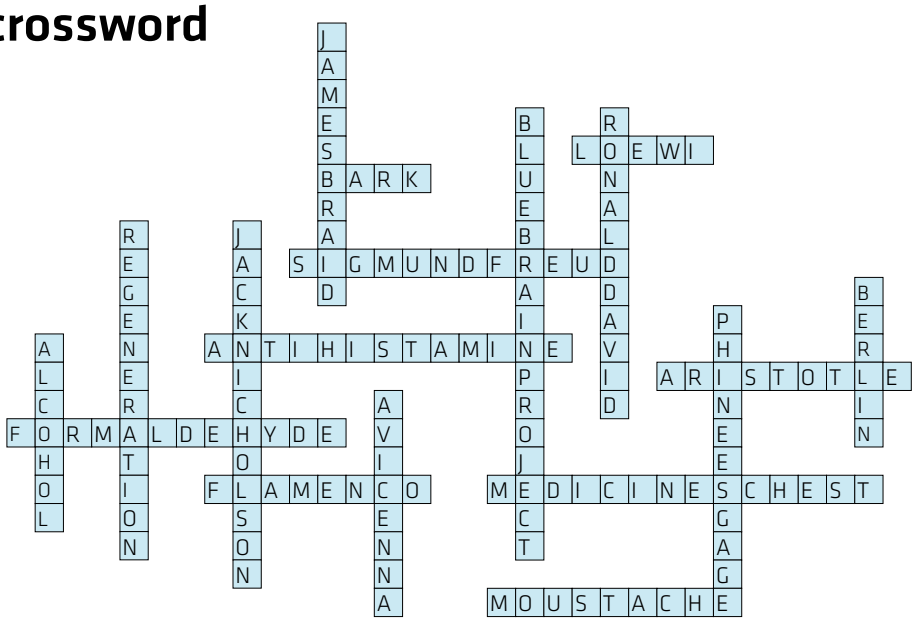
Partial or complete loss of memory — N E M A I A S

Star-shaped glial cell — S T R A Y C O A T

Answers for the word jumble can be found on page 18 of *ECNP Daily News* Issue 3, available on Tuesday


## Answers for yesterday's crossword

1 James Braid
2 Blue Brain Project
3 Ronald David
4 Loewi
5 Bark
6 Regeneration
7 Jack Nicholson
8 Sigmund Freud
9 Berlin
10 Phineas Gage
11 Alcohol
12 Antihistamine
13 Aristotle
14 Avicenna
15 Formaldehyde
16 Flamenco
17 Medicine Chest
18 Moustache



## ECNP CALENDAR OF EVENTS

| | |
|---|---|
| **2014** | |
| **6-9 March** | ECNP Workshop on Neuropsychopharmacology for Young Scientists in Europe, Nice, France |
| **4-6 April** | **ECNP Seminar,** *Veles, Macedonia* |
| **6-11 April** | ECNP School of Child and Adolescent Neuropsychopharmacology, Venice, Italy |
| **8-10 May** | **ECNP Seminar,** *Croatia* |
| **18-21 October** | **27th ECNP** *Congress, Berlin, Germany* |
| **14-16 November** | **ECNP Seminar,** *Serbia* |
| **2015** | |
| **29 Aug–1 Sept** | **28th** *ECNP Congress, Amsterdam, The Netherlands* |
| **2016** | |
| **17-20 September** | **29th ECNP** *Congress, Vienna, Austria* |
| **2017** | |
| **2-5 September** | **30th ECNP** *Congress, Paris, France* |
| **2018** | |
| **6-9 October** | **31st ECNP** *Congress, Barcelona, Spain* |
| **2019** | |
| **7-10 September** | **32nd ECNP** *Congress, Copenhagen, Denmark* |

**For regular updates on ECNP initiatives please visit:**
**www.ecnp.eu and www.ecnp-congress.eu**


## Stay informed, stay connected

In addition to this newsletter, ECNP offers a variety of other news and media channels designed to keep you at the forefront of our latest activities, initiatives and developments:

### Websites (www.ecnp.eu | www.ecnp-congress.eu)
The ECNP websites provide a myriad of information on matters related to our organisation. Follow links to sign up for e-bulletins and news updates.

### Message from the President
A monthly personal e-message from the President.

### E-news
Monthly overview of latest news within ECNP.

### Talk of the Month
Short video talks by distinguished scientists, aimed at making ECNP science more accessible to the general public.

### Facebook (www.facebook.com/myECNP)
Find ECNP on Facebook to subscribe to the news feed and join meeting 'events' throughout the year.

### Twitter (twitter.com/ECNPtweets)
Follow ECNP on Twitter to receive the latest news and updates, hot off the presses!

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**David Nutt** United Kingdom, *past-president*
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**Nicoletta Brunello** Italy, treasurer

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**Wim van** den Brink *The Netherlands*

Editor-in-Chief *European Neuropsychopharmacology*

**Michael Davidson** Israel

Executive Director

**Alexander Schubert** The Netherlands

**Scientific Programme** Committee 26th *ECNP Congress*

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**Celso Arango** Spain, chair *Educational Committee*

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