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hat is ECNP about? It is applied neuroscience in the broadest and best sense, and it has put multidisciplinarity and cross-talk at the heart of the organisation's mission and personality. As we grapple with the grand challenges of brain health in the twenty-first century, the mission is more important than ever.

Most people will know ECNP from the annual ECNP Congress, now the largest applied neuroscience meeting in Europe. This remains our most important activity and the upcoming 26th ECNP Congress in Barcelona (5-9 October 2013) promises another outstanding banquet of the latest and best in the science and treatment of disorders of

the brain - from molecules to the clinic - from around the world. As we predict to host close to 7,000 participants (the average number in the last five years), it's truly a unique chance for psychiatrists, neuroscientists, neurologists and psychologists to come together for five days of exceptionally stimulating exchange and discussion. For me, it's still by far the most vibrant and productive meeting of the year.

But ECNP's activities extend beyond the congress, and encompass a wide range of programmes and initiatives in research coordination (ECNP network initiative (ENI), congresses), education (ECNP School), clinical and preclinical training (Nice workshop), public information (ECNP Media Award), advocacy

summit, and patient outreach (DepNet). Together they make ECNP the largest noninstitutional supporter of applied neuroscience in treatment, research and education in Europe.

The following pages have been designed to give you a snapshot of some of those programmes and initiatives, and what ECNP is doing to advance the science of the brain, promote better treatment, and enhance brain health. As you don't want to miss the biggest meeting in Europe on applied neuroscience, come and join us in Barcelona on 5-9 October.

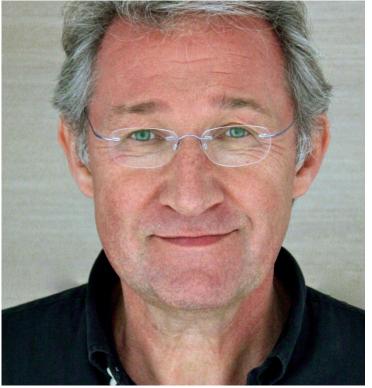
> Joseph Zohar **ECNP President**

Looking ahead to Barcelona

ver the last two and a half decades the ECNP Congress has established itself as the place where all aspects of brain research – from foundational basic science to breakthroughs in the clinic - come together.

Continuing its unique focus on multidisciplinarity and translation, the congress this year turns its spotlight on a host of new and exciting topics. "We have a packed scientific schedule," says Scientific Programme Committee Chair Wim van den Brink (Amsterdam Institute for Addiction Research, University of Amsterdam, the Netherlands). "The content this year really zeros in on what's most exciting in the field, including areas such as mitochondrial dysfunction in Alzheimer's disease, the neurobiology of persistence in antisocial behaviour, epigenetics and the relationship between sex-hormone fluctuations and vulnerability in neuropsychiatric disorders. Other areas of great interest include immunogenetics in psychiatry, pain processing, brain imaging, the role of reward, computational models in addiction, ADHD, how glutamate can affect mental disorders and treatment updates for schizophrenia, bipolar disorder and depression. It's an excellent selection."

"The interactive Educational Sessions have always been a special feature of the ECNP Congress and are for



Wim van den Brink, Chair Scientific Programme Committee

The 26th ECNP Congress, on 5-9 October in Barcelona, Spain, will build upon last year's quartercentury milestone with an even more exhilarating and cians, but it has implications which are comprehensive programme of applied neuroscience and clinical insight.

me an important part of what makes it such a useful meeting. This year we have a very strong line-up, with sessions on cognitive behaviour therapy in patients with schizophrenia with and without post-traumatic stress disorder, the treatment of addiction in patients with comorbid psychiatric disorders, pharmacotherapy and the developing brain, and a debate on binge eating and obesity as a food addiction."

Another priority this year, Professor van den Brink explains, will be challenging participants to think outside of their usual areas of expertise. So the Keynote Lecture, which formally opens the congress, will be delivered by Henry Markram (Switzerland), director of the paradigmshifting Human Brain Project (page 2). And continuing last year's highly successful formula, there will be six plenary lectures to provide stimulating new perspectives on the field.

"This year, the first of what will be a regular highlight of the congress, we'll have a lecture by the winners of the Brain Prize, Christine Petit (France) and Karen Steel (UK), who'll discuss the journey from genes to therapy in deafness (page 5). It's something which is far away from where we are as clinifascinating for psychiatry," said Professor van den Brink. "We come across

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Keynote Lecture Neuropsychopharmacology through computational simulation: the Blue Brain Project Saturday 5 October

Henry Markram, the Keynote Lecturer at this year's ECNP Congress, will take delegates inside the Human Brain Project. A bold initiative to develop nothing less than a computational model for the human brain, this €1-billion project stands to revolutionise our understanding and treatment of the world's most complex organ.

n 2005, Professor Markram, who is director of the Brain and Mind Institute at Switzerland's École Polytechnique Fédérale de Lausanne (EPFL), launched a project with IBM to create a virtual brain in a supercomputer. By reverse-engineering brain activity down to the molecular level, Professor Markham sought to build a synthetic model of brain function that could shed new light on brain disorders.

The project, which began by simulating a rat cortical column, was enormously complex. A rat's brain has about 100,000 columns of some 10,000 neurons each; mapping one neuron required the computing power of a laptop. But it was so successful that it prompted a much bigger initiative, the Human Brain Project, an international collaboration to take computer modelling to the next level by creating a complete virtual brain.

The project so impressed the

European Commission it was awarded a €1 billion grant by the Future and Emerging Technologies (FET) Flagship Programme, a new initiative of the Commission's to encourage visionary and 'mission-oriented' research that has the potential to offer groundbreaking benefits for European society and industry. Coordinated by EPFL, the Human Brain Project involves over 80 institutions in 20 countries.

In some respects the project is all about timing. Understanding the human brain is one of the great challenges facing 21st century science, yet progress has been hampered by the fragmented nature of brain research and the data it produces. The cornerstone of the Human Brain Project is the game-changing convergence of biology and information and communication technologies (ICT), with the evolution in imaging modalities that can observe the brain finally coming together with incredible increases in computing power, now bolstered by the Internet and cloud computing. This convergence has created the possibility of integrating data from these different domains into a unified picture of the brain as a single multilevel system

"What we are proposing is to establish a radically new foundation to explore and understand the brain, its diseases, and to use that knowledge to build new computing technologies," said Professor Markram. The potential benefits for diagnosis and treatment are enormous, including revolutionary improvements in personalised medicine and new paradigms for drug testing and discovery in which the simulated computational brain will supersede animal models.

"What makes the Human Brain project so exciting," said ECNP president Joseph Zohar, "is the way it brings together very different technologies, and opens whole new treatment horizons. For a multidisciplinary organisation like ours, focused on the science and treatment of brain disorders, it's a great story."

The challenges, nevertheless, are formidable. The computational capacity required is a thousand times more powerful than that of today. Hence,

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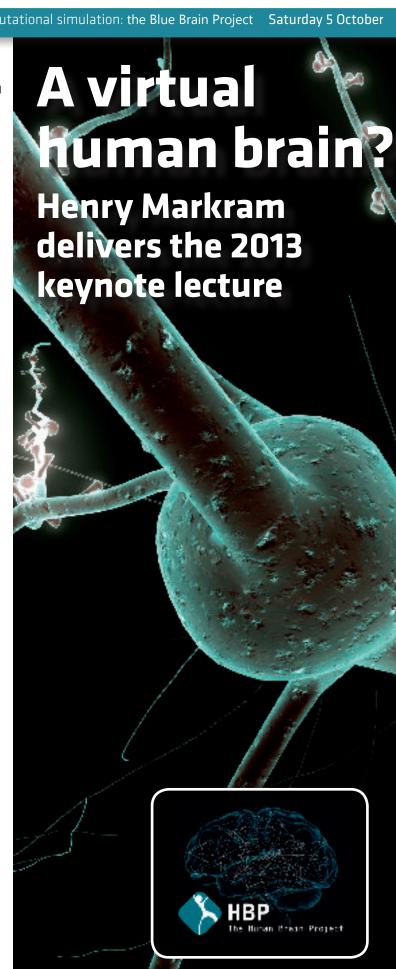
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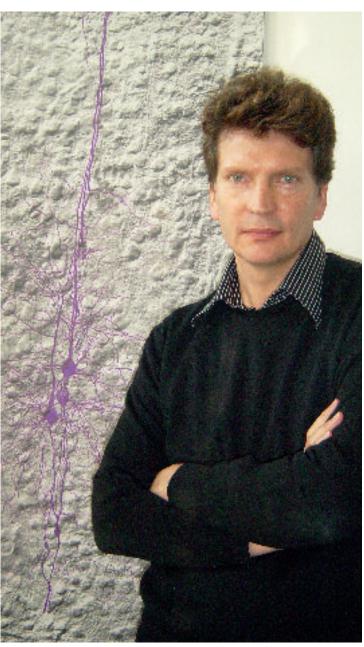




developing computing systems that can handle the workload will be a critical priority, with a focus on neuromorphic or 'brain-like' supercomputers that can combine technological improvements in microelectronics with the cognitive flexibility of human intelligence.

Planned across a 10-year period (2013-23), the progression of the project has been mapped to three phases. The initial phase (2.5 years) will see the 'ramp-up' of ICT technology, developing the platforms to harness the data. During phase two, the so-called 'operational' period (4.5 years), the focus will be on data collection, while still constantly building and developing the platforms. The final three years of the proposed timeline, referred to as the 'sustainable' phase, will 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

Looking forward, the first goal will be data, specifically the generation and interpretation of strategically selected data from mouse and human studies and accelerate research on the brain (e.g. multi-level view of the brain, causal chain of events from genes to cognition, uniqueness of the human brain, body ownership, language, emotions, consciousness, theory of mind), its diseases (e.g. from symptom-based to biologically-based classifications, unique biological signatures of diseases, early diagnosis and preventative medicine, optimised clinical trials, efficient drug and other treatment, personalised medicine) and future computing (e.g. supercomputing as a scientific instrument, supercomputing as a commodity, new software for multiscale and interactive supercomputing, new hardware from neuromorphic computing, intelligent tools for managing and mining massive data, human-like intelligence). The second will be theory, placing the spotlight on the brain's ability to acquire, represent and store information, with a focus on the mathematical principles of how different levels of the brain are organised. The third will be to develop the ICT platforms required, themselves split into six separate platforms that will focus on neuroinformatics, brain simula-



Henry Markram

tion, high-performance computing, medical informatics, neuromorphic computing and neurorobotics. The final goal is application, the use of the established platforms research to trigger a wave of new research interest across the different areas of fundamental neuroscience, especially around those unique capabilities, such as language, that distinguish humans from other animals.

Commenting on the concept to develop a virtual human brain, ECNP Scientific Programme Committee chair Wim van den Brink stressed that it was "truly fascinating." He continued: "The size of the project, the ambition of its goals, and the historic levels of funding it's received make it very exciting. But a project as massive and potentially groundbreaking as this will

also spark debate. One person may come out of the Keynote Session and say 'this is a great thing for neuroscience, and a great thing for people with mental disorders', and another may say 'this is wasted money.' So let's have that debate."

With a prevalence of mental health in Europe, an ageing population and a pharmaceutical industry that is pulling back on brain research, revolutionary new tools are needed to investigate the brain and address the impact of brain disorders. The Human Brain Project is a massive, bold step in this direction.

Henry Markram, 'Neuropsychopharmacology through computational simulation: the Blue Brain Project', Keynote Lecture, Saturday 5 October 18.00-19.30

The 26th ECNP Congress 5–9 October 2013 Barcelona

Looking ahead to Barcelona

Continued from page 1

a lot of people with deafness and, as a consequence of it, paranoid and depressed feelings and social defeat. Based on their basic research, this promises contributions from a very different area."

"But we'll also have Angela Vincent (UK)," he continued, "speaking about the autoimmune processes in encephalopathy and the role of autoimmunity in causing psychiatric disorders. The whole issue of whether immune processes play a part in the development of psychotic disorders has been revitalised by wonderful miraculous cures seen in people with psychotic disorders who turned out to have encephalopathy."

This year will also see development of a number of initiatives introduced in more recent meetings, including the Scientific Cafés, which were integral to promoting the atmosphere of exchange, debate and networking that so marked the 2012 congress. "I went to one on deep brain stimulation and there was a very active discussion on technical issues regarding the targets that we should be heading for, and also issues such as whether it's ethical to do brain surgery, is it reversible and what are the risks?" added Professor van den Brink.

Junior Scientists symposia will also make a welcome return, having been extremely well attended last year, which Professor van den Brink emphasised was a clear indicator that ECNP is heading in the right direction in terms of its support for junior researchers. "If we actually spread them out into the existing seminars, which wouldn't be always easy, it would lose the feeling of vitality within the organisation," he said.

In a similar vein, ECNP Networks symposia have been stepped up to offer better-tailored sessions for those looking to join in projects of interest, in turn nurturing new professional relationships and teamwork. Building on the previous Targeted Expert Meetings (TEM) symposia, the ECNP Networks symposia allow both current research and new scientific ideas to be communicated and discussed.

Alongside all of these different pillars of the scientific programme, Professor van den Brink underlined that there is always much more going on in the background of ECNP. "The meeting is very much not just the meeting; ECNP is doing much more. We will utilise the congress as a platform to show what else the ECNP has to offer."

How the ECNP Congress works

Every ECNP Congress has **five** parallel tracks, covering the spectrum of interests:

- Clinical treatment track, for evidence-based treatment
- Clinical research track, for clinical research issues
- Interface research track, link between preclinical and clinical research
- Preclinical research track, for basic and preclinical research
- Educational track, for interactive educational update sessions

Each track has two sessions a day, at 09.00-10.40 and 14.30-16.10, with four speakers each. In addition there are:

Six plenary lectures, two per day on Sunday, Monday and Tuesday. These include the lectures by the winner of the 2013 ECNP Neuropsychopharmacology Award (Sunday 6 October 13.30-14.15) and the winners of the 2012 Brain Prize (Tuesday 8

- October 13.30-14.15).
- Morning Brainstorming sessions - small, focused sessions organised by ECNP members on a topic of their choice, and open to everyone.
- The Keynote Session on Saturday 5 October, 18.00-19.30, which includes the Keynote Lecture - this year by Henry Markram of the landmark Human Brain Project - and awards presentations. It is followed by the Welcome Reception.
- Poster Sessions, from
 Sunday until Tuesday, 11.4513.30. These are grouped
 into eight topics, with two
 or three topics displayed
 every day. (During these
 sessions, lunch is available.)
- Scientific cafés, five cafés per day (Sunday, Monday and Tuesday) from 16.10– 16.40. These are informal, topic-focused networking opportunities for participants sharing a common interest.



Karen Steel and Christine Petit to deliver the 2013 Brain Prize **Plenary Lecture**

dentifying the molecular basis of deafness puts us on a road to developing novel therapies for both early and late onset cases of hearing loss. Karen Steel (King's College London, UK) and Christine Petit (College de France, and Institut Pasteur, Paris, France), joint winners of the 2012 Brain Prize, spoke to ECNP Matters ahead of their Plenary Lectures at the ECNP Congress to discuss their exemplary work in elucidating many of the ways in which deafness comes about.

Deafness has many causes, including genetic factors that influence the deterioration of certain structures in the auditory system. Understanding the underlying molecular mechanisms of deafness has formed the foundation of therapeutic development and continues to do so. "For deafness in particular, because the number of sensory cells in the ear is very small, genetics is the best way of finding molecular mechanisms rather than any biochemi-

cal approach, because there is so little material in each ear to study," said Professor Steel. "So, genetics has been extremely effective in identifying the essential molecules involved in normal hearing processes, which, when you have a mutation in that particular gene, lead to deafness. It is a way of that ran from 1997 to 2000, although the mouse getting access to the critical molecules involved."

Advancements in molecular tools to study genetics, coupled with the sequencing of the mouse genome, have greatly invigorated genetics over recent years. Mouse models, generated by targeted mutation, are used extensively to bridge the gap between phenotypic expression and genes involved in deafness, as Professor Petit explained: To understand the various forms of human deafness, in the absence of possible direct observation of the cochlea, the auditory sensory organ, and the phenotype – to find out exactly what was wrong most frequent target of the gene defect, we extensively rely on multidisciplinary analyses, including

morphological, biochemical, electrophysiological and biophysical studies, of the engineered corresponding mutant mice. Together, we have thus been able to decipher some molecular

networks underlying known cochlear functions, to clarify how some cochlear structures operate. and even to discover cochlear structures and to decipher their roles in sound processing."

Generating these mouse models has involved large coordinated efforts that began over a decade ago, using ENU (N-ethyl-N-nitrosourea) to create random point mutations throughout the genome; these mice were then bred with wildtype mice and the offspring were screened for hearing and balance function. "I have initiated two separate programmes aimed at finding new mouse mutants affecting hearing," said Professor Steel. "The first one was an EC-funded programme and mutants that we got we are still studying today. It really laid the foundations for a lot of further work. That programme involved two large mutagenesis programmes that were just getting started: one in Munich and the other in Harwell (near Oxford in the UK). This was an EC-funded programme that covered five different groups in four European countries: Germany, UK, France and Israel.

"Once we got the abnormal animals and could breed from them, we were able to characterise the with the ear – and then go through this process of positional cloning, which means identifying the mutation, based on finding its location on a particular part of a chromosome. From that, we identified the mutations involved in 24 different

Continued on page 6



2013 Neuropsychopharmacology Award Lecture Therapeutic targets in Parkinson's disease Sunday 6 October

Revolutionary promise for Parkinson's disease

ach year ECNP honours a prestigious and distinguished researcher within the field of translational and applied neuroscience with the ECNP Neuropsychopharmacology Award. Amongst other prizes, the 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 2013 award lecture will be given by Anders Björklund (Wallenberg Neuroscience Centre, Lund University, Sweden) whose lecture will discuss novel therapeutic targets for neuroprotection and disease modification in Parkinson's disease.

"The general background of my talk is the search for treatments that can interfere or modify the disease

mechanism underlying Parkinson's disease," Professor Björklund told ECNP Matters. "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

"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 same in other relative neurodegenerative diseases like Alzheimer's for example,

can help patients over several

is to find ways of interfering with the underlying disease."

These methods of intervention fall under three broad categories: Neuropro-

tective treatments,
disease modifying
treatments (slowing
the progression
of the disease)
and 'restorative'
treatments, which can
restore functionality in already

In his plenary lecture, Professor Björklund will discuss two avenues of his work engaged in finding effective Parkinson's treatments within these three therapeutic categories. "The first is that we've generated in rodents, primarily in rats, a condition where we increase the level

affected patients.

Elevated levels of the pro-

of the disease-causing protein

which is called alpha-synucle-

in," he said.



Anders Björklund

tein 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 amounts. It is called Nurr1,"

Plenary Lecture 2012 Brain Prize winners

Continued from page 5

mutants that had hearing or balance problems. We identified altogether 13 different genes. Of those 13 genes, nine of them were completely novel. They were all of course good candidate genes for involvement in human deafness, and some of those have now gone on to be associated with human deafness as well."

Professor Steel also initiated the Mouse Genetics Program, funded by the Sanger Institute and Wellcome Trust. "We took advantage of a different large scale mutagenesis project that Bill Skarnes [Sanger Institute, UK] started. He was targeting genes in ES [embryonic stem] cells that you can use to make mouse mutants from. Over the years, he had been building up a library of ES cells, each with a different gene targeted. He now has over 13,000 of the mouse genes – two thirds of mouse genes targeted in ES cells. When we started, he was only just beginning this project. We took those ES cells and we generated mouse mutants from them, and because they were targeted we knew which genes were affected. Then we put in place a screening programme that tested for signs of many different

"We've now screened about 600 of these mouse lines (we've made 900 at the Sanger Institute). Although I've recently moved to King's College London, the project is going ahead at the Sanger Institute and I am still involved with it, particularly the auditory screening. This project has directly led to the establishment of a large international consortium called the International Mouse

Phenotyping Consortium (IMPC), which involves labs around the world following this same procedure." Using this screen, Professor Steel has identified twelve new genes involved in normal hearing processes, all candidates for human hearing loss.

Professor Petit's work has addressed mainly the genetic basis of human deafness. As a pioneer of this field, she has brought up over 20 novel causative genes. She said: "What we have contributed most, so far, is the deciphering of the normal molecular physiology of hearing and molecular pathophysiological pathways of hereditary early onset

forms of deafness in humans. Considering the possible continuity between the early and the late onset forms of deafness, in terms of underlying pathogenic processes, we are now in a position to use what we have learned about early onset forms to address the pathogenesis of the late-onset forms. Improving

our understanding of the molecular physiology of normal hearing and deafness can lead to knowledge that can be of direct benefit to patients, both in terms of clinical assessment and treatment.

"Having deciphered the pathogenic processes

of the various deafness forms, we are now in a position to conclude whether or not the auditory prostheses (hearing aids and cochlear implants, which are presently the only way to improve or restore hearing) could be beneficial, or will have no effect – or even, for some forms of deafness, could be detrimental to residual hearing. This last scenario calls for the development of alternative approaches to prevent or alleviate hearing impairment. Rooting my genetic approach on human deafness, it naturally follows that one of my objectives is to prevent, alleviate or to cure

deafness. Having clarified a network of molecular interactions, you can intervene on some proteins of the pathways that are more favourable targets for therapeutic approaches than others."

Pharmacological agents and gene therapy are, according

to Professor Petit, two of the most promising lines of investigation when it comes to helping patients in the short to middle term. Together with José-Alain Sahel (Centre Hospitalier National d'Ophtalmologie des Quinze-Vingts,

"Improving our understanding of the molecular physiology of normal hearing and deafness can lead to knowledge that can be of direct benefit to patients, both in terms of clinical assessment and treatment."

Christine Petit (College de France, and Institut Pasteur, Paris, France)

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 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."

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 abovenormal 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

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

"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)

> 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

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 vari-

> ous 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 focused. But let me say

in the next couple of years I think one can be hopeful that clinical trials will be started."

Anders Björklund, 'Novel therapeutic targets for neuroprotection and disease modification in Parkinson's disease,' 2013 ECNP Neuropsychopharmacology Award Plenary Lecture, Sunday 6 October 2013, 13.30-14.15

Paris, France), she is moving towards gene therapy in retinitis pigmentosa in Usher-1 syndrome, a hereditary condition that affects both hearing and sight. "When I was conducting my research, I always had in mind whether or not our results could be useful for patients," she said. "During my ECNP talk, I will discuss one deafness form for which we have recently found a way to prevent the worsening of hearing impairment, in the corresponding mouse model. We now plan to move to clinical trial. In terms of therapy, we are very much focused on the possibility of using existing drugs to alleviate or prevent hearing impairment; there is evidence that some can be efficient. We are also developing gene therapy approaches.'

The study of mouse models can also lead to the uncovering of unsuspected particular features in some human deafness forms. "In humans, hearing is the sense of the communication. It is a necessary condition to oral language acquisition, conversational exchanges and to enjoy music," explained Professor Petit. "From an in-depth analysis of the processing of some sound physical parameters



in the mouse models of human deafness, we can anticipate that although hearing threshold is only moderately elevated in some patients, speech intelligibility and music perception are likely to be very seriously affected in patients. This leads us to proceed with a more detailed analysis of auditory perception in these patients."

Emphasising the impact of basic discoveries on improving clinical diagnosis and action, Professor Petit noted: "It is a real contentment to consider the medical impact of our basic research, regarding the elucidation of deafness causes, the reevaluation of the meaning of some auditory tests, the changes in the way of exploring hearing impairment in humans, and the therapeutic perspectives. My lab is deeply involved in the transfer of newly gathered basic knowledge to clinicians."

Karen Steel and Christine Petit. 'Deafness: on the road from genes to therapy,' Brain Prize Plenary Lecture, Tuesday 8 October 2013, 13.30-14.15

Plenary Lecture Prefrontal cortex-based circuits: relevance for antidepressants and antipsychotics Sunday 6 October

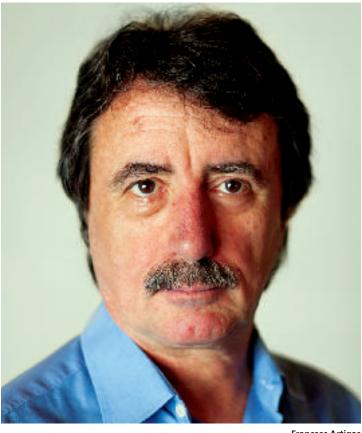
Exciting times for antidepressant therapies

he field of pharmacotherapy in depression has undergone something of a quiet revolution over the past decade. Francesc Artigas (Barcelona Institute of Biomedical Research, Spain), who will be giving a plenary lecture on the role of antidepressants and antipsychotics in prefrontal cortical circuits at the ECNP Congress in October, shared his optimism about the future of therapy in a recent interview with ECNP Matters — optimism, not only in identifying new mechanisms, but also in the effective collaboration between academia and industry.

Classical treatments for depression are widely known to require a few weeks to start taking effect, during which patients are highly vulnerable. But new discoveries have the potential to assign this period of limbo to the history books. "Everyone accepts that antidepressants are only effective in about two-thirds of patients," said Professor Artigas. "However, there are a couple of really exciting observations of the last decade which have really changed our views about how depression can be treated. One is the finding that ketamine, which is an NMDA [N-methyl-D-aspartate] receptor antagonist, is actually effective in a subset population of patients who are refractory to practically every pharmacological treatment.

"The other finding is the electrical manipulation of the ventral subdivision of the prefrontal cortex (Brodmann area 25): deep brain stimulation [DBS], as used in drugresistant Parkinsonian patients. Actually, DBS is very effective in subsets of patients that are refractory to all kinds of treatments. These two different findings have really shaken the world of research in depression, not because these interventions can be routinely used but because they definitely show that more rapid and effective treatments can be obtained. Now, basic and clinical researchers are frantically trying to find out why these treatments are so effective. I would say that probably half of the neuropharmacology people all over the world are working in one or the other."

Even so, moving from clinical research to clinical reality takes time. New drugs, Professor Artigas estimated. will take a decade or more to ar-



Francesc Artigas

rive into the hands of patients. These drugs adopt a different mechanism to the current monoamine system targets, and include agonists of metabotropic glutamate receptors, chemical analogues of ketamine, amongst others. Hence, glutamate modulation within the prefrontal cortex, either via ionotropic or metabotropic receptor

binding, could be the key to improving the efficacy of antidepressant therapy. Meanwhile, new drugs are still based on multiple monoamine targets, incorporating advances in this field.

Clinical research takes money too, as much as it takes time. While the financial crisis has impacted every sector of work, Professor Artigas noted additional factors that have led to the brain research

dip: "First, when investing in non-CNS diseases, companies get more revenue because the brain is very complicated! The pharmacology of the brain cannot

be approached in the same way as the pharmacology of, say, cancer or diabetes, because one of the most important differences between the brain and the rest of the body is the complexity of the human brain.

"So, if you understand how a cancer cell works you will understand that particular type of cancer. If you

"I think we should abandon the idea of a single drug being effective in all depressed patients. Many people support that there should be genetic characterisation, to see which drugs are more effective for that particular patient."

Francesc Artigas (Barcelona Institute of Biomedical Research, Spain)

know how a hepatocyte works you will understand the liver. But if you understand how a single neuron works, you will not know anything

about the brain. This enormous complexity of the brain means that large research efforts need to be made, requiring larger investments than other diseases to identify new targets. And then, there is a long way from a target to a medicine, and many potential targets at preclinical level never translate into new medicines."

He continued: "In addition to that, there is one important problem with regulatory aspects. In general, since we have treatments for virtually all psychiatric illnesses, the regulatory agencies ask for new drugs to be very safe. That probably means that, sometimes, drugs that could provide better efficacy are abandoned because of potential side effects. That is a very important problem too."

But new efforts are working to overcome these problems. Effective research is borne out of collaboration, and new EU initiatives strive to shorten the link between scientific innovation and economic growth in many fields, including drug development. Working together, the journey from basic research to new drugs ought to be less tough. "I am involved in a EU project called NEWMEDS, which is part of a new initiative from the European Union, the IMI (Innovative Medicine Initiative)," said Professor Artigas.

"These are projects in different fields in which drug companies and academic groups work together. This is an excellent initiative to shorten the delay of the translation of basic data to industrial products. This can improve the joint effort between academic people and industrial people in search of new targets and new medicines."

The conveyor belt of such translational efforts must pass through animal modelling before reaching the stage

of human testing. While these models can never be mimics, they nevertheless serve as useful analogues of disorders, as Professor Artigas explained: "We don't have any really reliable models for depression or for schizophrenia. We attempt to make the animal behave in a certain way, in which the current drugs, antidepressants and antipsychotics, are effective to treat the alterations of any

kind produced in the model. Chronically stressed animals are not really depressed animals. Animals treated with phencyclidine or ketamine are not schizophrenic animals.

"But these models are useful in terms of trying to see what brain areas or circuits and what neurochemical elements are involved in the response to the drug. They are good for that, but they are not good for trying to understand the pathophysiology of the illness, because of course the rat or the mouse cannot mimic the changes in the brain of a depressed or schizophrenic person. We should know the limitations of these models. but we should work with these models because there is no other way to reliably test new drugs."

A broad goal of new medicines is to address individual symptoms that make up complex disorders such as depression and that differ from

patient to patient. Environmental and biological factors play a role, and Professor Artigas outlined his treatment of depression," he said. "A large number of studies have defined individuals having one or another

"If you understand how a single neuron works, you will not know anything about the brain. This enormous complexity of the brain means that large research efforts need to be made, requiring larger investments than other diseases to identify new targets."

Francesc Artigas (Barcelona Institute of Biomedical Research, Spain)

vision for genetics in future patient assessment. "The genetic factors are indeed very important for the

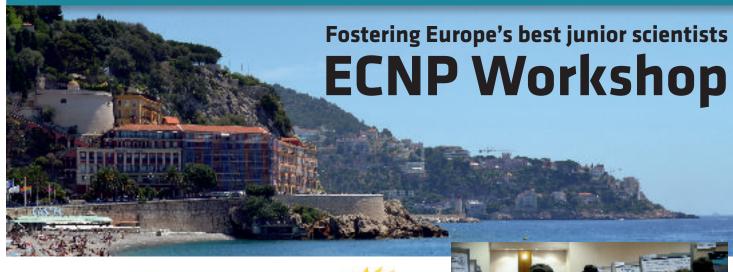
gene polymorphism that respond with higher or lower efficacy to certain drugs. These are really key issues for

the future. I think we should abandon the idea of a single drug being effective in all depressed patients. Many

people support that there should be genetic characterisation, to see which drugs are more effective for that particular patient. This has been shown in many different studies – there is one very classical study showing that a polymorphism of the serotonin transporter gene makes some people respond better than others. So it seems very clear."

Francesc Artigas, 'Prefrontal cortex-based circuits: relevance for antidepressant and antipsychotic drug action,' Plenary Lecture, Sunday 6 October 2013, 11.00-11.45

ECNP Meetings for Junior Scientists in Europe



he objective of the ECNP Workshop, held annually in Nice, France, is to promote and encourage the scientific development of European (pre)doctorates and residents in neuropsychopharmacology. To facilitate this, a number of senior experts in the field are brought together with approximately 100 junior scientists from Europe for a three day programme rich with informative exchange and discussion of various topics within the domain of applied and translational neuroscience.

By promoting and encouraging the development of junior scientists seeking to pursue research careers in neuropsychopharmacology, the ECNP Workshop aims to stimulate high-quality experimental and clinical research in mental disorders and their

The ECNP Workshop Committee is responsible for the content of the programme, the selection of the faculty and the selection of the participants. The latter occurs on the basis of submitted papers before a certain deadline.

The Workshop focuses on three recurring

topics, molecular neuropsychopharmacology, behavioural pharmacology and clinical neuropsychopharmacology, as well as one variable topic. Within each of these core topics, two distinguished scientists address specific themes, after which there are short presentations by five junior scientists, who are invited to present their research.

Warkshop

In addition, all participating junior scientists are required to present a poster.

- For those selected, there is no registration fee and travel and accommodation will be substantially covered by ECNP.
- The 16 best poster presenters will speak in the Junior Scientists symposia at the 26th ECNP Congress.

Junior Scientists symposia, Sunday 6 October, 14.30-16.10, and Monday 7 October, 09.00-10.40





Florian Riese

David Nutt introduces the 2012 Fellowship Award winners

Comprised of a multinational team that includes graduates of the Workshop (to represent the preclinical multidisciplinary component) and the ECNP Schools, the panel will be rotated with fresh graduates on a regular basis to ensure that it can always remain very closely related to the needs of current juniors.

As chair of the panel, Florian Riese (Psychiatric University Hospital Zurich, Switzerland) shared the aims of J-MAP, as well as touching upon the general initiatives that ECNP has in place to ensure a better and more inclusive future for its junior members.

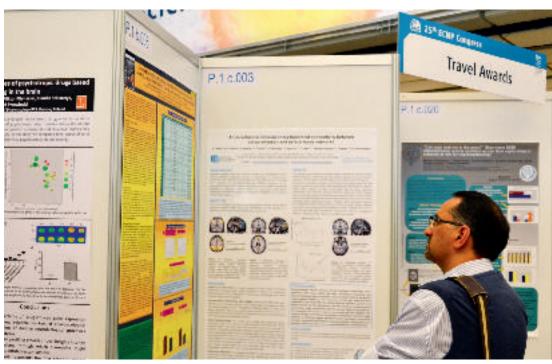
"So far ECNP has already invested a lot by organising, for example, the Schools on neuropsychopharmacology, the workshop and also by the new initiative, the ECNP certificate," said Dr Riese. "But one area where there was room for improvement was that while people would go and actually make use of these activities, they would then disappear again from the ECNP network. We want to change that."

By organising places to network that have a more accessible atmosphere, J-MAP hopes the junior members will feel more comfortable, and make more connections that will encourage them to return to subsequent meetings and other activities.

"We've only been recently founded so we don't have a track record of achievement yet, but we hope to get several activities up and running at the ECNP Congress in Barcelona," continued Dr Riese. "We plan on organising several scientific cafés that are directed towards junior members on the topics of how to get published, how to get grants and how to get your career going both in academia and in industry."

On the Sunday night during the 26th congress in Barcelona, the panel is also planning an informal networking event known as 'Science on the Rocks', due to be held in a local bar. With emphasis on a relaxed, friendly atmosphere in which to discuss all avenues of their careers, the event will feature an informal lecture by ECNP past-president David Nutt.

"We also hope to create an ongoing communication platform so that people who, for example, attended the schools and workshops can continue or



Travel Awards posters during the 25th Congress in Vienna

maintain their professional relations to the other participants," said Dr Riese.

He continued: "I think what really makes you come back would be that you expect to meet friends there, or your professional and social network, so I think if junior members also start to come back every year that would be a really great achievement."

Crucially, he added that the panel has no intention of creating an exclusive junior member experience.

"To actually have exposure to senior members is I think a unique opportunity. That is also the impression I get about ECNP – that senior members are very welcoming and open to junior members approaching them, and wanting to become part of the ECNP network."

Florian Riese (Psychiatric University Hospital Zurich, Switzerland)

Support for Junior Scientists

Junior scientists and clinicians are the lifeblood of the field. Encouraging their professional development is one of ECNP's key aims, and we have developed a range of programmes and benefits to address their challenges and needs:

- The ECNP Schools three week-long programmes of intensive training for 50 junior clinicians, one in general neuropsychopharmacology (annually), one in child and adolescent neuropsychopharmacology (biennially), and one in Old Age Neuropsychopharmacology (biennially).
- The ECNP Workshop an annual threeday interactive workshop for 100 junior scientists.
- The 16 best poster presenters at the Workshop are selected for the two Junior Scientists symposia at the ECNP

Congress, one in the preclinical research track, one in the interface research track.

- The travel, registration and accommodation costs of all participants of the Schools and Workshops are substantially funded by ECNP.
- A range of prizes and awards, including six Fellowship Awards (€1,500), 40 ECNP Travel Awards (€500), and three ECNP Seminar Awards (€500).
- Free registration at the ECNP Congress for junior scientist poster presenters whose abstracts are accepted for publication in the congress supplement.
- €100 registration for non-poster-presenting junior scientists if accompanied by an ECNP member.
- The ECNP Certificate a unique, European-wide qualification for junior researchers in the science and treatment of disorders of the brain.

Quite the opposite, as a core aim of the organised events is to provide a more relaxed atmosphere in which junior members can feel comfortable enough to then approach senior members for discussion of topics or to seek advice. "All senior members are very much invited to all activities of J-MAP as well, but I think it makes a difference if you are a junior member, and maybe slightly shy, to feel you are on 'home ground' rather than a territory where seniors clearly dominate," explained Dr Riese.

He added: "To actually have exposure to senior members is I think a unique opportunity. That is also the impression I get about ECNP – that senior members are very welcoming and open to junior members approaching them, and wanting to become part of the ECNP network."

The financial constraints faced by iunior members is another key component in their overall attendance, and ECNP is already doing a lot to support juniors with initiatives such as travel awards and fellowship awards that offer a cash prize to a selection of outstanding junior members, their work being permanently displayed in poster format during the congress.

Dr Riese continued, underscoring the importance of interaction between pre-clinical and clinical research by ECNP: "I think this not done in other places to a similar extent. I am a medical doctor and I think it's really important, especially for the clinical scientists, that we get this kind of exposure and do not allow the generation of knowledge to be taken out of our hands by other professions. That naturally occurs, but we do play a role in that."

He added: "I think that can be a focus of J-MAP as well. It's not an exercise for its own right; it serves a true purpose."

J-MAP Members

- Florian Riese, Switzerland; chair
- Covadonga M. Díaz-Caneja, Spain
- Kfir Feffer, Israel
- Rajna Knez, Croatia
- Kara Panetta, United Kingdom
- Olga Paravaya, Belarus

Plenary Lecture The future of molecular pharmacology and drug discovery in psychiatry Tuesday 8 October

GPCRs and the pathways to better tre

he genomic revolution has given us insight into the byzantine complexity of cellular development and communication processes, and, more recently, multiple risk loci have been identified to be common to various psychiatric conditions. While these findings inform us about cellular and molecular processes, delivering significant advances in medical treatment by selectively targeting these particular loci will represent a challenge that may seem overwhelming. Marc Caron (Duke University Medical Center, Durham, NC, USA) spoke to ECNP Matters about his approach of characterising molecular pathways downstream of receptor activation in order to tease apart the ways in which neurotransmitter activity is mediated, a tried and true approach for drug development, but with a new

The days when we might have considered simple therapeutic targets emerging directly from genomewide analysis of psychiatric disorders – discovering, say, 'the gene for schizophrenia' - are well and truly over. "My reading of this, and I think any cell biologist would agree with this, is that these are diseases of homeostatic control of cell function - the life of the cell." said Professor Caron. "I call this, to use a crude expression, 'cell biology gone amok', because essential cellular processes have been disrupted. Whether you have a mutation in this or that particular gene, [the outcome] also depends on what other genetic makeup you have. That will be the thing to dictate whether you will develop schizophrenia, or major depression, or autism - it is not potentially that particular gene, but how that particular gene might contribute along with the rest of your genetic make-up."

While the genetic com-

monalities between different psychiatric disorders could help us to more finely categorise them, Professor Caron and his colleagues have taken a different approach. "I am not a clinician, so I talk from the position of a basic scientist. The way that these disorders have been treated is basically by treating the systems (serotonin, dopamine, glutamate) where the manifestations are," he said. "Where do the symptoms emerge? From the contribution of the hundreds or thousands of mutations, and the weakest system flares up, giving defects in serotonin, in glutamate, in dopamine systems. That is what we have been treating, albeit with limited success for many years. Many of these compounds target what we work on, so admittedly it's a biased view. But many of these drugs target, directly or indirectly, GPCRs [G-protein coupled receptors).

When a GPCR is activated, it activates heterotrimeric G-proteins and generates secondary messengers or modulates ion channels. Cataloguing the evolution in our understanding of GPCRs over the past two decades, which has led to the discovery that GPCRs may communicate by engaging signalling proteins other than G-proteins, Professor Caron continued: "As the receptor remains in an arrestin mediated signal. The

and eventually the signal, which occurred through the G-protein, is turned off. What we have realised, since initial observations we made in 1996 and later on in around 2000, is that this agonistliganded receptor complex that is phosphorylated, associated with an arrestin, is capable on its own to activate other signalling pathways. These signalling pathways had not previously commonly been associated with GPCR activation. They are more of the kind that tyrosine kinase growth factor receptors activate, like the MAP-kinase pathway

"The interesting thing is that over the years it has been shown that these two signalling pathways have two main properties that distinguish them. G-protein signalling is usually rapid, but it turns off rapidly. Arrestin mediated pathways are slightly slower (although they happen quite fast too) but they are more protracted, lasting for minutes and sometimes for hours (when we do this in the lab in cells). Invariably, when it's been looked at in a handful of receptors, they appear to serve different cellular func-

He continued: "What you get from a G-protein mediated signal is different cellularly from what you get from an

activated through a G-protein by a given ligand or hormone or neurotransmitter, while the same ligand will inhibit the arrestin pathway – and vice versa. So you can have a ligand that activates preferentially, say, a G-protein – and there are very few compounds that are completely one or the other; they usually have a mix of activity. You can have another compound that activates model allowed Professor mostly arrestin, but inhibits the G-protein. We call this bias signalling, or functional selectivity."

This is a long departure

from the notion that GPCRs simply signal through G-proteins. More importantly, it presents an untapped opportunity to the research community, given that 30 to 50 percent of drugs target GPCRs. "There are a few examples where it has been shown that Gprotein activation by a given receptor mediates the beneficial effect of a drug, whereas the ar-



biased signalling of these

receptors is the best char-

acterized. This is now a very

well-studied concept. You can

ments of proteins that interact

with these receptors, and they

will change the readout from

Creating a genetic mouse

a ligand.'

vary the intracellular comple-

Caron and his colleagues to study the function of arrestins. "These animals did not respond as usual to amphetamine," he said. The response of rodents to amphetamine has been used for decades as a pharmacological model to assess the efficacy of antipsychotics. "All clinically effective antipsychotics to date interact with the D2 dopamine receptor; they interact with lots of other receptors too, but they all must interact with D2 receptors. When we gave amphetamine to the mice that had no arrestin, we saw little to no response to amphetamine. So we started looking to see if there were

D2 receptor. "We found out that the

restin mediates the side effect of a drug, and vice versa," said Professor Caron. "There are many, many ways to

pathways downstream of the

other potential signalling

"Whether you have a mutation in this particular gene or that particular gene, [the outcome] also depends on what other genetic make-up you have. That will be the thing to dictate whether you will develop schizophrenia, or major depression, or autism."

Marc Caron (Duke University Medical Center, Durham, NC, USA)

activated form, it gets desensitised – phosphorylated. Then, the phosphorylated receptor binds to arrestin molecules.

second thing is that it has been shown by several of our colleagues in the field that the same receptor can be

atment response



Register online: www.ecnp-congress.eu

D2 receptor was capable of engaging a pathway that was not canonical for GPCRs. The arrestin downstream of D2 receptors is capable of interacting with a protein kinase called Akt, which is upstream of another kinase called GSK-3. Some of the antipsychotics appear to inhibit that pathway preferentially, and we have subsequently done genetic manipulations to eliminate various components of the pathway, which mimic the action of antipsychotics."

Professor Caron is currently exploring several ways in which these pathways can be modulated. Using synthesised compounds

selective for D2 receptors (in collaboration with Jian Jin and Brian Roth of the University of North Carolina at Chapel Hill, United States), Professor Caron's team are studying those that are capable of acting through either the G-proteins or the arrestin pathway. Compounds selective for D2 receptor/arrestin interactions act as antipsychotics in animal models but seem to have lower side effects. "Another approach is that we are eliminating some of the components, like arrestin, in every dopamine D2 receptor-expressing neurons, to try to map which neuronal pathway is really responsive to antipsychotics.

"The last approach is that we have generated mutants of the D2 receptors which are only capable of functioning through the G-protein or through the arrestin pathway. We are reconstituting these mutated receptors along with the wild-type receptors into genetically modified mice that have had their endogenous D2 receptors knocked out selectively in various neurons. We will be able to tell, both biochemically and behaviourally, what behaviour you get if you have a receptor that can only signal through the G-protein or through the arrestin nathway "

With a cautious reminder that this work is still at the

proof-of-concept stage, Professor Caron described how he is coupling it with the expression of a biochemical tool – a component of the ribosomal machinery – that will be expressed along with the receptor in a cell-specific pattern. This will allow the isolation of messenger RNAs transcribed in vivo (in mice) in response to the selective activation of one pathway or the other. Hence, this work will circle back to its beginnings in the hope of identifying novel targets that will be selective for the D2 receptor in the neurons that are mediating the effects of antipsychotics.

Professor Caron concluded: "Thus, our combined

approaches should allow us. first, on the basis of the new concept of GPCR functional selectivity, to identify leads to develop more selective and efficacious antipsychotics; second, to identify previously unappreciated cell specific targets for therapy. Is it going to work? I don't know, but we are very hopeful! It seems like an interesting approach worth exploring since all currently available drugs were developed without these new concepts in mind."

Marc Caron, 'The future of molecular pharmacology and drug discovery in psychiatry,' Plenary Lecture, Tuesday 8 October 2013, 11.00-11.45

ECNP Projects and Initiatives Ambassadors

ECNP Ambassadors

ince its inception 26 years ago, ECNP has striven to not only cultivate applied and translational neuroscience research in Europe, but to also promote it across all European countries. This mantra has been a central pillar in many activities such as Regional meetings and Seminars, as well as in the recruitment of participants for the Congresses, Workshops and Schools from all corners of the region. The result is now a strong and vibrant support base, not just in Western Europe, but Eastern and Southern Europe as well.

Following in the footsteps of this initial work. ECNP has now launched a new initiative, ECNP Ambassadors, to draw in countries even further afield, many of which lack the infrastructure to sustain a national society, and so have fallen outside the Advisory Board of National Societies programme.

A senior brain researcher or clinician in his or her region, each ambassador will help to coordinate seminars and mini-symposia, to nominate junior scientists for the ECNP Schools and encourage application for the ECNP Workshop, and act as representative and point of contact for ECNP in his or her home country. Each ambassador will serve a term of two years

The group has its own newsletter and meets every year to plan activities and welcome new members at the ECNP Congress.

Contact details for the current list of ECNP Ambassadors can be found on the ECNP website.

Armen Melik-Pashayan	Yerevan State Medical University after M.Heratsi	Yerevan, Armenia
Luchezar Hranov	Teaching and Research Hospital, Neurology and Psychiatry "Sveti Naum"	Sofia, Bulgaria
Miro Jakovljević	University Hospital Center Zagreb	Zagreb, Croatia
Elmars Rancāns	Riga Stradins University	Riga, Latvia
Robertas Bunevicius	Lithuanian University of Health Sciences	Palanga, Lithuania
Eugenia Sinita	IMSP Clinical Hospital of Psychiatry	Chisinau, Moldova
Maria Ladea	Al Obregia Hospital	Bucharest, Romania
Peter Morozov	Russian National Medical Research University	Moscow, Russia
Gorica Djokic	Neuropsychiatric disorder Institute	Belgrade, Serbia
Rok Tavcar	University Psychiatric Hospital	Ljubljana - Polje, Slovenia
Oguz Karamustafalioglu	University Hospital for Active Treatment in Sisli Etfal Teaching and Research Hospital	Istanbul, Turkey
Eva Chkonia	Department of psychiatry and drug abuse of the Tbilisi State Medical University	Tbilisi, Georgia
João Bessa	Life and Health Science Research Institute of the Universidade do Minho	Braga, Portugal
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Photo by Albertane, Flickr

Plenary Lecture Understanding pain in somatoform disorders Monday 7 October

Getting to the root of pain in fibromyalgia

ibromyalgia is a condition characterised by widespread chronic pain and fatigue that has a significant negative impact on quality of life. While it is sometimes classed as a somatic syndrome, new findings have indicated physiological differences between sufferers and control subjects. Claudia Sommer (University of Würzburg, Germany), who has contributed greatly to the redefinition of the condition, will speak at this year's ECNP Congress about her recent work. In a recent interview with ECNP Matters, she described the progress so far in unearthing functional and morphological characteristics of fibromyalgia and the road to better aetiological understanding that lies ahead.

The origin of fibromyalgia remains unclear, but its neuropathic nature is becoming increasingly evident. Both pervasive and persistent, it represents a significant healthcare burden. Uncovering the physical basis of the disorder is the first step towards improving its treatment, and Professor Sommer and her colleagues are working to bring about such a conceptional shift. "We had the idea that something might be wrong with the peripheral nerves in these patients," began Professor Sommer, describing her recently published research.1 "We used several methods to look at the small peripheral nerve, and one method was quantitative sensory testing (QTS), which involves measuring the detection threshold for warmth and cold, but also for touch - 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. 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."

Along with this, the group used two further tests, corroborating the notion that small nerve fibres were affected in fibromyalgia. "We used pain-related evoked potentials, a neurophysiological method that measures the afferent fibres from the face, hand or foot (wherever you stimulate) to the brain," continued Professor Sommer. "In contrast to routine evoked potentials, this method uses a special electrode. We measured the small nerve fibres called the A-delta fibres



Claudia Sommer

[sensory fibres that respond to stimuli such as cold and pressure, as well as fast/first nociception]. Here, we found that the response to the electrical stimulation of these fibres in the fibromyalgia patients was lower. Wherever

we stimulated – foot, hand, 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."

The third test involved the analysis of skin biopsies from

subjects' legs. Nerve fibres in the epidermis were counted in the lower leg and upper thigh, and on average fibromyalgia patients possessed lower numbers of nerve fibres than both controls and depressive patients.

Ten subjects with unipolar depression effectively formed a second control group alongside healthy controls. The link between physical pain and depression is a familiar one, but depression is not necessarily a feature of fibromyalgia, so teasing the two apart was a valid objective. Professor Sommer explained: "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; we know that. We wanted

"So far, we have shown that something is wrong with these small nerve fibres.
Of course, we would like to know what is wrong, and why."

Claudia Sommer (University of Würzburg, Germany)

to be sure that we are not measuring things related to the depression. This is why we recruited this group with depression, but no pain."

The number of different drugs that are used for the treatment of fibromyalgia are relatively few. Antidepressants work for some patients, although the basis for their effect is not understood, and they may not address all associated symptoms. Moreover, antidepressants may well have different modes of action in depression and fibromyalgia. "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, that they work independently from the antidepressant effect."

Bringing this work together with other research areas such as genetics and inflammation will hopefully 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." said Professor Sommer. "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.

"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."

Claudia Sommer, 'Understanding pain in somatoform disorders,' Plenary Lecture, Monday 7 October 2013, 11.00-11.45

References

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ECNP Certificate



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

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- The presentation of results in an abstract and poster at the ECNP Congress

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Registration and tultion are covered by ECNP



european college of neuropsychopharmacology

ECNP Awards Media Award

aunched in 2012, the ECNP Media Award was established to recognise exemplary contributions in journalism, literature, dance, film, theatre or other media to destigmatising disorders of the brain. Spanning all European languages and countries, the media award gives credit to those who create awareness, make the science of brain disorders accessible to the general public, and clarify the underlying concepts. Alongside the honour itself, the award also features a €5,000 prize.

The Science Media Centre, London

The first ECNP Science Media Award now sits proudly on the desk of the Science Media Centre (SMC), an independent UK organisation whose mission it is to disseminate accurate, evidence-based scientific information. to the media for the benefit of the public and policymakers. Ed Sykes, who recently took over from Claire Bithell as Head of Mental Health at the SMC, spoke to ECNP Matters about the Centre's work and the issues that emerge when a big story hits the headlines

we can convince to

better. If there aren't

stand up and speak, the

scientists lined up to be

"[The SMC] originally started up because of the debacle with the MMR vaccine and mad cow disease," said Dr Sykes. "It was back in around 2000, when there was a House of Lords Science and Technology Select Committee that wanted to come up with ways of improving how the public got their information from the media." The idea of an independent press office

emerged, which led to the establishment of the SMC in 2002. In line with a growing awareness of mental health issues, a dedicated post for mental health and neuroscience was introduced in 2010.

London, UK)

"We want the public, as well as government, to hear the views of the experts," said Dr Sykes. "Our general policy is that we think experts should be able to speak out. They should explain their thinking as the experts to the public, the media, and everyone. Then the government should decide, with the evidence, whether they are going to say yes or no. That is when it

ECNP Media Award



(Left to right) Fiona Fox, David Nutt and Guy Goodwin at the ECNP Summit Report on the Future of CNS Drug Research in Europe

Below: The ECNP Media Award is presented during the 25th ECNP Congress Keynote Session works well."

The improved dialogue between journalists and scientific experts is bringing about parallel improvements in press reportage. Addressing the tactics that consumers can adopt to find the best information in the melee of a freshly breaking story, Dr Sykes said: "What we try to encourage, and what we think the public should look towards as their gold standard, is to give much more credence to the articles that are written by the science. environment and health specialists than the general news reporters."

The SMC offer training courses to introduce scientists to the media process, as well as publishing advisory guides to help scientists address complex questions when engaging with the public. The model has been so successful that sister organisations have been set up in Australia, New Zealand, Canada and Japan.

But getting more experts to engage is the crux, as Dr Sykes explains: "The more of those who we can convince to stand up and speak, the better. If there aren't scientists lined up to be interviewed then the media will start going to people who don't have the expertise, who don't necessarily know what they are talking about."

ECNP has worked with the SMC on a number of press briefings, to ensure the Centre has access to the best experts not just in the UK, but from

Continued on page 19



Plenary Lecture Autoimmune processes in encephalopathy Monday 7 October

"Antibodies have been identified

in certain forms of epilepsy, rare

cases of dementia and in a few

Angela Vincent (Nuffield Department of Clinical

Neurosciences, Oxford University Hospitals Trust, UK)

case reports of patients who

present with psychosis."

Autoimmunity and psychiatric disorders

s one of the enlightening and informative plenary lectures at this year's congress in Barcelona, Angela Vincent (Nuffield Department of Clinical Neurosciences, Oxford University Hospitals Trust, UK) will discuss autoimmune processes in encephalopathy, and the promise for treatment in psychiatric disorders.

"There are now well-recognised forms of

encephalitis that are associated with antibodies to important membrane proteins, namely ion channels, receptors and associated proteins," Professor Vincent communicated in an abstract for her lecture. "The antibodies are directed to voltage-gated potassium channel complex proteins, particularly

LGI1 and CASPR2, NMDA-receptors, AMPA, GABAb mide or other second-line therapies. and Glycine receptors. Antibodies to the water channel aquaporin-4 (AQP4) are present in a rare but relapsing disease, neuromyelitis optica, that can be confused with multiple sclerosis.

"Although originally described in neurological syndromes such as neuromyelitis optica, limbic encephalitis, NMDAR-antibody encephalitis or progressive encephalomyelitis with rigidity and

myoclonus, there is little doubt that these antibodies can cause partial forms of disease without clear evidence of an encephalopathy. For instance, NMDAR and VGKC-complex antibodies have been identified in certain forms of epilepsy, rare cases of dementia and in a few case reports of patients who present with psychosis."

She continued: "The most important aspect

of these newlydescribed conditions is that the patients respond well to immunotherapies, particularly plasma exchange, steroids and intravenous immunoglobulins; in some it is necessary to use the CD20 targeting monoclonal antibody Rituximab and/or cylcophospha-

"There are now several studies in progress looking for these or other autoantibodies in patients with different forms of psychiatric disease, principally psychosis. A few published studies indicate that perhaps 5-10% of patients with psychiatric diagnoses will have antibodies to one of the known targets, but further work is required to identify whether these patients respond to immunothera-



Angela Vincent

pies, and to identify other potential target antigens in this important group of diseases."

Angela Vincent, 'Autoimmune processes in encephalopathy: a promise for treatment of psychiatric disorders' Plenary Lecture, Monday 7 October, 13.30-14.15

Reference

Vincent A. Bien CG, Irani SR, Waters P. (2011) Autoantibodies as-sociated with diseases of the CNS: new developments and future challenges. Lancet Neurology 10:759-72

DO NOT MISS.

the 'Science on the Rocks' event, held in downtown Barcelona on Sunday 6 October during the 26th **ECNP Congress.**

With emphasis on a relaxed, friendly atmosphere for junior scientists, the event will encourage interaction with peers and discussion in a casual



setting. The event will also feature an informal lecture by David Nutt.

More information will be available via the ECNP website nearer the time of the event.

Projects and Initiatives ECNP Medicines Chest

Taking drug development off the shelf

oo frequently, drugs under development within pharmaceutical companies are shelved before their full potential can be explored. Whatever the reason, this early termination of compounds leaves academic institutions and clinical researchers with poor access to these potentially beneficial drugs and, in many cases, completely unaware that they even existed.

To address this issue the **ECNP Medicines Chest has** been initiated, hoping to act as an intermediary between companies and researchers, placing emphasis on the continued development of drugs rather than focussing on whose hands they are kept in. As the initiative progresses, the Medicines Chest will update its database to allow quick and simple reference to drugs that have already been identified as useful research compounds, as well as allowing researchers and companies to submit suggestions themselves.

As project manager of the initiative, Gavin Kilpatrick (UK)

is overseeing the Medicines Chest in its efforts to promote better collaboration between clinical researchers and pharmaceutical companies, and in ensuring better access is granted to existing compounds.

"What I'm doing is reaching out to pharmaceutical companies and saying 'look, we've got this initiative, we know you've got this compound and we think you're not doing anything with it would you be willing to make it available?"" Dr Kilpatrick told ECNP Matters. "I think it sort of chimes with the current situation where there is more of a movement by the pharma industry in supporting open innovation and moving the competitive phase later in development.

"Many of the companies can see the advantage of making these compounds available, in the sense that these early experimental medicine studies can be done, and then if there are some interesting results that point in the direction we might go in terms of new medicines then the whole community can then potentially build upon those."

Although it is of course possible in many cases to identify and obtain a drug of interest more independently, Dr Kilpatrick underlined the inherent benefits that working directly with pharmaceutical companies could bring. He said: "It's very difficult and

them. So the aim would be to get their safety package and toxicology package and even the drug substance itself if possible."

While in some cases there will still be a reluctance by companies to enter into agreement with academic development programmes as the benefits of 'sharing' their product may not be immedi-

"You could envisage a situation where in the longer term, pharma companies make available a lot of their assets they are not taking any further forward, for the benefit of the whole community."

Gavin Kilpatrick (Project Manager, ECNP Medicines Chest)

can be quite expensive to pick up compounds afresh, and do toxicology work and get them synthesised etc. So the reason to go to pharma companies is to get their support. They'll have made quite an investment in these compounds, but they're on the shelf and so don't have much value to

ately apparent. However, with the help of the Medicines Chest, companies are more able to visualise the bigger picture and how the development of their drugs externally can then be re-fed back into their future plans.

"For example you might find a drug that is an an-

tagonist of receptor 'X', but the compounds that were developed were then shelved because they didn't work in depression for example - their original target," said Dr Kilpatrick. "But then an academic researcher shows that it may help people who are dependent on alcohol [for example], and you're then at a phase where the pharma industry is able to build on that, either by developing that compound further, or others, than can be used in that indication."

As such, Dr Kilpatrick reiterated that there is now some shift in the attitudes of pharmaceutical companies in terms of moving the competitive phase of drug discovery to later in the timeframe of development, allowing more time for investigation of drug efficacy in different therapeutic targets first.

Although the Medicines Chest is still in the early stages, as time progresses Dr Kilpatrick is confident that as more initial companies get involved, the better the knock-on effect will be for encouraging other companies to follow-suite. "Once one or two of them buy into the idea and help, and once people know that they've done that, they will get a sort of reputational kick out of that. Then I think other companies would follow," he said.

He added: "I think it's very forward thinking, and really a nice idea, and something

that ultimately doesn't just have to be in the CNS and neuropsychopharmacology area. You could envisage a situation where in the longer term, pharma companies make available a lot of their assets they are not taking any further forward, for the benefit of the whole community."

Further information on the ECNP Medicines Chest, including the current list of drugs within the initiative, can be found on the ECNP website. Suggestions for new or alternative compounds should be addressed to ecnp-medicineschest@ecnp.eu

ECNP Awards Media Award

ECNP Media **Award**

Continued from page 16 around Europe. "We've been able to see the SMC, and its value, from the inside," said ECNP Executive Director Alex Schubert. "It's an impressive organisation, with a reach - because of the influence of the London media market – way beyond the UK. We'll continue to work with them to ensure the public and media are served with high-quality science about disorders of the brain."

Brain: The Inside Story

The 2013 winner has taken the ECNP Media Award in a different direction. Described by Live Science as "an interactive sensory feast that both surprises and stimulates," Brain: The Inside Story exhibition is a multicountry collaboration that adopts the



Joseph Zohar (left) presents the 2012 ECNP Media Award to Science Media Centre representatives Claire Bithell and Fiona Fox

latest interactive multimedia technology in installations that explore the brain and how it works, bringing visitors up to the cutting edge of neuroscience. The ECNP citation commended the exhibition's creativity in engaging participants in the drama and wonder of brain function and its vital contribution to explaining brain function and demystifying brain disorders.

Originating at the American Museum of Natural History in New York, the exhibition is currently on display

in Granada, Spain, and will travel to Guangzhou, China and Milan, Italy. It will be at Milan's Museo di Storia Naturale from October 2013 until March 2014. The ECNP Media Award will be presented at the Keynote Session of the 26th ECNP Congress.

ECNP Publications

ECNP TALK OF THE MONTH

Taking it to the next level

n 2011 ECNP launched its 'Talks of the Month' - short, 10-to-15-minute videoed talks on core themes and topics in the field delivered by key opinion leaders. The talks, high-quality summaries of key indications and developments in the field, were a key part of ECNP's campaign to provide accessible and reliable evidencebased information and insights about the brain and brain disorders to patients and members of the wider public.

The first, released in June of that year, was by Educational Committee chair Celso Arango on 'Child and adolescent neuropsychopharmacology'. To date there have been 20 talks, on subjects ranging from basic neuroscience to specific disorders, and from epidemiology to treatment strategies and public health.



The format was simple and production values were basic. Nevertheless, the talks have attracted hundreds and hundreds of viewers. "They give a great opportunity both to patients but definitely also for psychiatrists to see new developments in that area," said Wim van den Brink, ECNP Scientific Programme Committee chair. So it was decided to take them to the next level. The result is a whole new look and feel, and even more accessible for viewers

Nine new talks are scheduled for release over the coming months:

- Mary Baker, The societal challenges in Europe
- Nicolett Brunello, Antidepressant drugs
- Ed Bullmore, Imaging brain networks in psychiatry
- Bill Deakin, Neuroinflamma-

- tion and the possibility of preventing schizophrenia
- Guy Goodwin, Bipolar depression
- Gitte Moos Knudsen, Molecular brain imaging in movement disorders and Alzheimer's disease
- Rafael Maldonado, Addiction: a brain disease
- Barbara Mason, Can We Treat Cannabis Dependence?
- Jack Price, Induced pluripotent stem cells and in vitro models of neurodevelopmental disorders

The talks can be seen on the ECNP YouTube channel 'myECNP'.



Register online: www.ecnp-congress.eu

Projects and Initiatives ECNP Certificate



ECNP Certificate

he ECNP Certificate is a new initiative that brings together all of the college's junior researcher programmes into a European-wide qualification backed by ECNP. At its core, the ECNP Certificate serves as recognition of the efforts and commitment of junior researchers in the field of applied and translational neuroscience with each candidate requiring to demonstrate a level of knowledge and skills that includes the ability to undertake either an original research project or effectively disseminate knowledge on the science and treatment of disorders of the brain by writing a scientific literature review or by organising an educational event.

Underpinning each certificate is one-to-one mentoring by an ECNP Fellow – experienced, senior members of the college from a number of wideranging backgrounds and international locations that are available to share their expertise and guidance, as well as giving feedback. Unlike PhD or Master's Thesis structures, mentors do not offer close supervision, and in fact it is preferred that they be located in a different country to their candidate, with quarterly (typically) catch-ups on the most substantive matters.

To be eligible for one of the 20 (approximately) certificate places in

each rotation candidates must have participated in at least one ECNP Workshop, School or Seminar, or attended another ECNP-affiliated course. Once this is complete, an ECNP Fellow is then chosen under the guidance of the

Certificate committee, and the project can begin.

Typically spanning six to twelve months, the project should culminate in a piece of original work or meaningful contribution that both the mentor and the Certificate Committee deem to be fit for accreditation and exhibition in poster format at the ECNP Congress. Once all these steps have been completed, the certificate itself will be bestowed upon candidates within the Keynote Session of the congress.

Although still in its early stages, the ECNP Certificate programme has already attracted candidates. "The big challenge now is to get the word out, and to get people signed up," Alex Schubert, Executive Director of FCNP told FCNP Matters

The ECNP Certificate comes with

no registration or tuition cost, and while it does not contain a grant, candidates can work remotely from their mentors, negating the need to travel, and once they complete the project they will be given free registration to exhibit their work at

the ECNP Congress. When one examines the journey that ECNP Certificate candidates

will take, each participant will pro-

gress through a chain of events that begins with attendance at a School, and ends with recognition of their work in an internationally renowned, world-leading congress. As such, the ECNP Certificate is a vital component in a wider collection of initiatives that ECNP has developed in order to foster and promote the work of junior scientists in European applied neuroscience. "We are really trying to develop careers," stressed Dr Schubert. "We're going into kind of 'hyperdrive' on this issue. It's really now an overriding priority for us to develop and promote junior scientists."

More information

on the ECNP Certificate can be found on the ECNP website. To apply, prospective candidates should send the following documents to

ecnp-certificate@ecnp.eu:

- A letter of application, indicating which ECNP Workshop or School, seminar, or affiliated course, vou have attended
- A short description of your research plan (1-2 pages A4), including timelines and preferred mentor
- A curriculum vitae

ECNP Meetings Schools

ECNP Schools

he ECNP Schools were established to encourage excellence in clinical neuropsychopharmacology in junior practitioners and to contribute to the continuing improvement of the field's high standards of practice.

Currently there are three ECNP Schools. The first is the ECNP School of Neuropsychopharmacology, held annually each year. The second and third ECNP Schools are rotated each year, alternating between Child and Adolescent Neuropsychopharmacology and Old Age Neuropsychopharmacology respectively.

In order to enrol, European participants (within five years of having received their defining qualification) must be nominated by ECNP Ambassadors or members of the ECNP Advisory Board of National Societies, who will assess their career potential. The ECNP Schools have capacity for a maximum of 50 junior psychiatrists, thus only two to three candidates from each country will be considered. Once accepted, ECNP will substantially cover travel and accommodation costs.

ECNP School of Neuropsychopharmacology

Participants at the ECNP School of Neuropsychopharmacology receive intensive training in all aspects of neuropsychopharmacology, from fundamental practices and the use of medications in individual indications, all the way to good clinical practice and optimal treatment and algorithms.

The 2013 School in Oxford, UK (7-12 July), will cover a multitude of topics over the course of its five-day programme, including bipolar disorder diagnosis and treatment in the short and long term, antidepressants, meta-analysis methodology, antipsychotics and the management of resistant schizophrenia, anxiolytics and the duration of untreated illness in anxiety disorder.

Alongside several small workshops, the programme also covers more general topics that include how to organise a meeting, how to manage relationships with pharmaceutical companies, and a discussion on where to take the School of Neuropsychopharmacology in the future.

ECNP School of Child and Adolescent Neuropsychopharmacology

The ECNP School of Child and Adolescent Neuropsychopharmacology was established to encourage and disseminate excellence in clinical neuropsychopharmacology among child and adolescent psychiatrists. The first edition of this School was held in Venice, Italy, from 19-24 February 2012.

Participants were offered an interactive week with an international faculty of experts in basic and clinical paediatric neuropsychopharmacology. The six-day programme tackled topics that included gene-environment interplay in developmental psychopathology, antipsychotics, pharmacological intervention in autism spectrum disorder, eating disorders, antidepressants, bipolar disorder, ADHD, suicide and debates as to whether medication should be considered a first line intervention in







Continued on page 22

ECNP Meetings Schools

ECNP Schools

Continued from page 2

severe child and adolescent psychopathology.

In addition, the School played host to workshops and discussions of ethical issues, risk management, and practical or clinical guidelines pertaining to children and adolescents.

The next ECNP School of Child and Adolescent Neuropsychopharmacology will be held from 6–11 April, 2014 in Venice, Italy.

ECNP School of Old Age Neuropsychopharmacology

April 2013 saw the first ECNP School of Old Age Neuropsychopharmacology (in Venice, Italy) open its doors to a new class of junior psychiatrists. This newly established ECNP School offers an interactive week of intensive training with an international faculty of experts in basic and clinical old age psychiatry, with special emphasis placed on neurobiology and neuropsychopharmacological management.

Topics addressed during the lectures and workshop cases included the art of old age psychiatry, cognitive examination, electroconvulsive therapy in the clinic and research, co-morbidity, Alzheimer's disease and vascular dementia.



The next ECNP School of Old Age Neuropsychopharmacology is planned for 2015.

See the ECNP website for more information on the past and future schools.

The 26th ECNP Congress Barcelona

'Why I look forward to the ECNP Congress'

s this year's annual ECNP
Congress in October draws
closer, ECNP will be hosting
a number of video shorts from longstanding members that will outline
their individual perspectives on what
makes the Congress so special to
them

The first video of the series, now available on both the ECNP Congress website and the ECNP YouTube channel, has been delivered by David Nutt (Edmond J Safra Chair of Neuropsychopharmacology and Director of the Neuropsychopharmacology Unit in the Division of Brain Sciences at Imperial College London, UK).

"I love the ECNP annual meetings," began Professor Nutt. "I think they are one of the highlights of the scientific year in Europe. I really would like to encourage

as many people to come as possible, because over the years we've built these meetings to be something for everyone. If you're a clinician you can go to the educational track and collect a phenomenal amount of CPD points in presentations given by some of the great experts in the world.

"If you're a basic scientist you will find there are colleagues there who are working at the cutting edge of the particular disciplines you are in, and they will be able to share with

"I love the ECNP annual meetings. I think they are one of the highlights of the scientific year in Europe. I really would like to encourage as many people to come as possible."

David Nutt (Imperial College London, UK)

you their exciting new findings. And if you are a translational scientist like myself you will benefit greatly from rubbing shoulders with the pre-clinical scientists, with the clinical



David Nutt shares his thoughts on what makes the ECNP Congress so special

researchers, and also the clinicians."

Professor Nutt concluded: "That really sums up the essence of this meeting, which is a family of people interested, fascinated and dedicated to the study of the brain and how we

can improve the treatment of patients with psychiatric disorders."

Stay tuned to the ECNP Congress website (www.ecnp-congress.eu) or subscribe to the 'My ECNP' channel on YouTube for all upcoming videos ahead of the Congress in Barcelona.

ECNP CALENDAR OF EVENTS

2013		
7-12 July	ECNP School of Neuropsychopharmacology, Oxford, UK	
4-5 October	ECNP Targeted Network Meetings, Barcelona, Spain	
5-9 October	26 th ECNP Congress, Barcelona, Spain	
2014		
6-9 March	ECNP Workshop for Junior 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	27 th 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	29 th 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	32 nd ECNP Congress, Copenhagen, Denmark	

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



Register online: www.ecnp-congress.eu

Stay informed, stay connected

n 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!

ECNP Committees

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