NEWSLETTER
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ecnp matters

Call for papers

Until 15 March 2011 it is possible to submit papers for poster presentation at the 24th ECNP Congress. You are invited to visit the ECNP website for instructions on the preparation and submission.

The papers will be reviewed for presentation at the congress and for publication in the congress supplement to the journal *European Neuropsychopharmacology*. Please note: young scientists whose paper is accepted for presentation **and** publication, may benefit from free registration at the congress.

Call for brainstorming sessions

At the 24th ECNP Congress, ECNP members again have the opportunity to organise a brainstorming session on a topic of their choice in the field of neuropsychopharmacology and related sciences. The sessions will be held on Sunday, Monday and Tuesday from 07.45-08.45 hours.

If you are interested, please send your proposal in accordance with the guidelines – see the member pages on the ECNP website – to Paris2011@ecnp.eu. The deadline for submission of proposals is 15 March 2011.

Call for applications: ECNP Fellowship and Travel Award 2011

Young scientists are invited to apply for the ECNP Fellowship and Travel Award 2011. The application period is open until 15 March 2011.

Please visit the ECNP website for further information on these awards and to check if you match the application criteria.

25th ECNP Congress: call for symposium proposals

The Scientific Programme Committee (SPC) of the 25th ECNP Congress invites you to submit proposals for a full symposium.

ECNP celebrates its 25th anniversary during the 25th ECNP Congress. Therefore, the setup of the scientific programme will be changed slightly. Instead of the usual five lectures, a symposium proposal will now consist of four lectures.

More information on the requirements for a symposium proposal, as well as the submission pages, can be found on the ECNP website. Please also take into account the details of the selection criteria used by the SPC to establish the scientific programme.

The deadline for submissions is 15 March 2011.

25 years ecnp

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Impressions from the 23rd ECNP Congress in Amsterdam

Report from the Scientific Programme Committee

Michel Hamon, France
Chair

As it has been each year since the beginning of this enterprise 23 years ago, it was a great challenge for the Scientific Programme Committee (SPC) to set up a really innovative and attractive scientific programme for the 23rd ECNP Congress, which was held this year in Amsterdam. Fortunately, we had major 'winning cards' to help us face such a challenge.

First, all of us in the SPC were highly motivated and did our very best to build up a scientific programme of great topicality in preclinical and clinical neuropsychopharmacology, with the aim of promoting as much as possible translational research and fruitful exchange and dialogue between basic neuroscientists and clinicians. I am especially grateful to the whole SPC group for their generous, effective and clever contributions to making our enterprise a success.

Second, the great number of proposals for symposia that were submitted by scientists, mostly from Europe but also from overseas, allowed us to select the very best in all domains relevant to neuropsychopharmacology. Selection was indeed difficult because most proposals were really excellent. For our choice, the best of science was our priority, but special care was also taken to balance time and space between clinical and preclinical sciences, and to respect fair participation of speakers from as many countries as possible, with the aim of reaching the highest percentage of women among them. Indeed, for the ECNP Congress in Amsterdam this participation rate was close to 30%, and ECNP will continue to make every effort to increase this percentage up to a level really reflecting the major contributions of female scientists and clinicians in neuropsychopharmacology research.

Third, the staff at ECNP Office, particularly Maria Vrijmoed-de Vries and her assistant Petra Hoogendoorn, played, of course, a pivotal role in setting up the optimal conditions for the SPC to select topics, speakers, chairs and moderators for the symposia, educational tracks and other sessions. I am especially grateful for the professionalism and warm cordial collaboration of the whole ECNP team in Utrecht, without whom nothing would be possible.

Thus, with such 'winning cards', the ECNP Congress in Amsterdam was a great success, and still the largest European meeting dedicated to the neurosciences. More than 7,200 delegates from 104 different countries not only registered at the congress, but also participated in the ECNP scientific programme and the 13 satellite symposia supported by pharmaceutical companies. Indeed, all the rooms were full during the whole congress, from the very first to the very last symposium, and it was not exceptional to see participants entering with additional chairs when scientific sessions had already started, because of the difficulty getting to the few remaining free seats in the middle of the room. Clearly, the superb quality of presentations and discussions by the faculty speakers and moderators was the reason for this success, and special thanks have to be addressed to them all.

Also the poster sessions were densely attended each day, with enthusiastic discussions and exchanges in spite of the limited space available. The three plenary lectures on hot topics – *Epigenetics* by Isabelle Mansuy, *Biomarkers of Alzheimer's disease* by the ECNP Award winner Kaj Blennow, and the fascinating *Hippocampus*, by György Buzsaki – were really excellent and also attracted a huge number of attendees.

For the first time this year, a breakfast meeting with young scientists had been organised with each of these prestigious speakers the day after their lecture. This initiative, which will be refined for the next year, is in line with the top-priority objective of ECNP to ensure the future of neuropsychopharmacology by providing support to PhD students and postdoctoral fellows involved in the field. Thus, as at previous congresses, we had four Target Expert Meetings (TEMs) in Amsterdam 2010 on *addiction, child and adolescent neuropsychopharmacology, neurological disorders* and *psychotic disorders and antipsychotics*, in which young senior scientists had been invited for thorough discussions and exchanges, hopefully ending with the setting up of new European collaborative projects.

Another innovative initiative for the 23rd ECNP Congress was the webcasting of presentations for which we received speakers' agreement. Already for this very first time, two out of the three plenary lectures, five out of the six educational update sessions and seventy five presentations for symposia can be watched on the ECNP website, thereby making a large part of the congress available *free* for those who could not be in Amsterdam.

Last but not least, I would like to emphasise that contacts with the press were very positive thanks to the three delegates, Anna Wirz-Justice, Philippe Fossati and Jim van Os, who clearly explained to journalists the key importance of neuropsychopharmacological research for better therapeutic and preventive management of psychiatric diseases. Amsterdam 2010 was therefore a *'grand cru'*, thanks to all these fantastic people who performed so well for its success.

Rendez-vous is now taken for the next challenge: the 24th ECNP Congress, 3-7 September 2011 in Paris.

ECNP Summit on the future of CNS drug reseach in Europe

Alexander Schubert, The Netherlands
Executive Director

The withdrawal of major pharmaceutical companies from CNS drug research in Europe this year triggered alarm throughout the field. If, as is looking increasingly possible, this signals the beginnings of a wholesale retreat by industry from the CNS arena, the implications for European neuroscience are very worrying indeed. As one of the leading associations for neuropsychopharmacology and psychiatry in Europe, ECNP felt that urgent action was called for.

Approaches were made to the key players in these developments to open a discussion on the issue. A meeting in July with Andrzej Rys, Director of Public Health and Risk Assessment at DG SANCO, proved especially instructive and positive. Another meeting, also very constructive, followed with Thomas Lönngren and his team at the European Medicines Agency in October.

From these meetings the idea of an 'all-hands' summit of interested parties evolved – a meeting in which all those groups affected across all dimensions of this complex problem could come together, achieve a common understanding of the challenge and work towards a long-term solution.

It was decided that the best vehicle for such a summit would be the annual ECNP Consultation Meeting in Nice. President-elect Guy Goodwin and past-president David Nutt were assigned the organisation of the event by the Executive Committee, and set about developing a participant list of 60 carefully selected stakeholders and designing the two-day programme for 6 and 7 March.

The result promises to be a highly productive meeting, with senior representatives from the European Commission, regulatory bodies, the scientific community and industry committed to participating. Further reports on the meeting and future activities being planned by ECNP to address the threat to European neuroscience will follow in *ECNP Matters*, as well as in our monthly *E-News*.

The programme of the 2011 ECNP Summit on the Future of CNS Drug Research in Europe can be found on www.ecnp.eu.

Big Pharma and Neuroscience: what's at stake?

On behalf of the Executive Committee:
Guy Goodwin, United Kingdom, *president-elect*
David Nutt, United Kingdom, *past-president*

In February of 2010 GlaxoSmithKline announced its plans to withdraw from pain and depression discovery research, with the closure of its major facilities in Verona, Italy, and Harlow, United Kingdom. In March AstraZeneca announced it would be pulling out of schizophrenia, bipolar, depression and anxiety. In July 2010 MSD announced the closure of six research operations in Denmark, Germany, the Netherlands and Scotland, with no CNS research left in Europe. In September Abbott Laboratories announced it would be eliminating 800 positions in the Netherlands and Germany. What is going on?

The question is especially puzzling because neuroscience is one of Europe's great academic success stories, with real strides being made every year in our understanding of brain structure, movement, thinking, memory and emotion. The importance of this research, moreover, is only growing as the burden of brain disorders in the region increases. As ECNP-funded studies have found, brain disorders now account for over 40% of the total disease burden in Europe. And this is not, it should be pointed out, due to treatment costs, which remain a fraction of those documented for other common disorders like diabetes and heart disease, but because of high prevalence, early onset, and high levels of associated disability. Why then do more and more major pharmaceutical companies see psychiatry as such an unpromising target?

The reasons are complex, but some key themes can be identified. To begin with, the identification and development of new treatments for brain disorders are particularly demanding as compared with other diseases. And on top of this, the criteria for proof of clinical efficacy and safety are unusually challenging, and satisfactory reimbursements sometimes too difficult to obtain. The encouragement of generics by some countries has further undermined the economics of commercial drug innovation. This also raises the question whether it's even realistic to expect profit-driven pharmaceutical companies to shoulder the long-term scientific investments needed to address Europe's changing mental health landscape. A different model is clearly needed.

The stakes here are high. First are the public health consequences. The disengagement of drug companies from basic research into psychiatric disorders poses a serious threat to the translation of important new insights into innovative and improved medicines. If, as seems likely, the discovery of and access to newer and better medicines are curtailed, this can only be viewed with grave concern. Second is the impact on Europe's research infrastructure, which has the potential to be extremely damaging. With funding and career options narrowed, many young people will be discouraged from choosing neuroscience as a career, with long-term consequences for research and health care.

But if a new model is needed, what might that model be? It will entail a new dynamic between academia, clinical practitioners, industry and the regulatory agencies – one in which therapeutic innovation and progress are prioritised irrespective of short-term economic considerations, and in which researchers, drug developers, clinicians and regulatory bodies work together to optimise public health and the control of psychiatric disorders. Some of the groundwork for this has already been laid by European Commission-led Innovative Medicines Initiative to encourage public-private partnership in European drug discovery. But this is only the beginning.

The shape of this new model will be the central theme of our upcoming 'Summit on the Future of Drug Discovery in Europe' on 6-7 March 2011, in Nice, France – to map out, even if in outline, a long-range rescue plan for European CNS drug research.

The summit is part of a larger ECNP strategy to address this issue – the first such campaign ECNP has ever launched – to change opinions and affect public health and research policy directly. The entire Executive Committee is behind the campaign, as is the European Brain Council and our other sister organisations. But at the risk of some melodrama, this is life and death.

interview

Maria Vrijmoed-de Vries, The Netherlands
Editor

Interview with the winner of the 2010 ECNP Neuropsychopharmacology Award in Clinical Research: Kaj Blennow, Sweden

Kaj Blennow was born in Malmö, Sweden. Currently, he is professor in clinical neurochemistry, chief physician at the Clinical Neurochemistry Laboratory at the Sahlgrenska University Hospital and heads the research group 'Neurochemical Pathogenesis and Diagnostics' at the University of Gothenburg, Sweden.

By training you are a specialist in psychiatry and in clinical (neuro)chemistry. Could you elaborate a bit on this interesting combination?
In the sixties with governmental support, Prof. Carl-Gerhard Gottfries M.D. *(note ed: founding member and former president of ECNP from 1987-1989)* started a Department of Psychiatry and Neurochemistry in Göteborg.

When I came to work there during my training as a psychiatrist, I got so interested in the neurochemistry aspects of brain diseases that I did my PhD in psychiatry, but with a focus on neurochemistry under the supervision of Gottfries. I am no longer working as a psychiatrist, however, I have a lot of interactions both with psychiatrists and neurologists and I find my knowledge on (psychiatric) patients extremely helpful both in directing the focus and in understanding of the outcome of my research.

You moved from psychiatry to neurology, i.e. Parkinson's and Alzheimer's disease, and presently back to psychiatric disorders such as schizophrenia, depression and addiction. You are also involved in basic neuroscience. By combining all aspects within neuropsychopharmacology, did you find (a) common factor(s) between psychiatric and neurological disorders?
To me the common factor is that neurology and psychiatry both deal with 'brain disorders'. The pathophysiological findings from neurology such as disturbances in synaptic functions and degeneration of (part of) neurons are probably common to many brain disorders.

Initially, studies were performed on post-mortem brains of patients. The findings of these studies already indicated some common pathophysiological changes, including disturbances in synaptic functions. By definition these findings were done at the end stage of the disease making the interpretation of 'common' factors sometimes less solid, i.e. less related to the initiating disease pathogenesis.

Fortunately the possibilities of modern technology allowed the development of increasingly more sensitive biochemical essays to measure small proteins in the human cerebrospinal fluid (CSF) making it possible to detect metabolic changes that are a direct result of changed processes in the brain. At our department we have been able to study the molecular pathogenesis of both neurodegenerative disorders such as Alzheimer's disease and psychiatric disorders such as schizophrenia and depression in patients who consented to a 'CSF-tap'. We have already found several biomarkers that not only may help us to detect diseases such as Alzheimer's in the prodromal phase, but should also provide insight in the (individual) effects of medication and even direct to the development of new pharmaceutical entities.

A recent paper in Nature Reviews/Drug Discovery addresses an interesting aspect of continuous interest to ECNP: the perspectives of academia, industry and regulators on biomarkers for Alzheimer's disease. Could you tell us more about this?
The paper is a spin-off of the discussions with Harald Hampel in Germany. One part is an effort to control and diminish the between-laboratory variability of the biomarker essays in Alzheimer's disease that I had developed with my team. This part is called the Alzheimer's Association QC program for CSF biomarkers.

At that time, we also realised that the biomarkers for disease detection, treatment effect(s) and/or drug development can only be of importance when the other stakeholders, such as regulatory agencies and the pharmaceutical industry, next to the academic institutions, patients and their doctors, acknowledge the validity of the findings as well as support the discovery and development of even better biomarkers and innovative treatments.

Together we have developed the pyramid as depicted in the picture on page 5 that shows the translation of biomarkers from being general risk-measuring tools to individual, disease-modifying treatments.

In relation to the above, what is your opinion about the closure of neuroscience labs in Europe by the industry?
Europe has a long tradition in neuroscience and contributed to the major developments in this area. The 'why' of the closure is the most important, but a difficult question to answer.

In over 30 years the treatment of Alzheimer's disease developed from 'nothing' to the promise of disease-modifying agents. This is the result of solid clinical biochemical research. The first spin-off, ß-amyloid modifying drugs show great promise. We are not sure whether ß-amyloid changes are the cause of Alzheimer's disease, but it surely plays an important role in the pathogenesis.

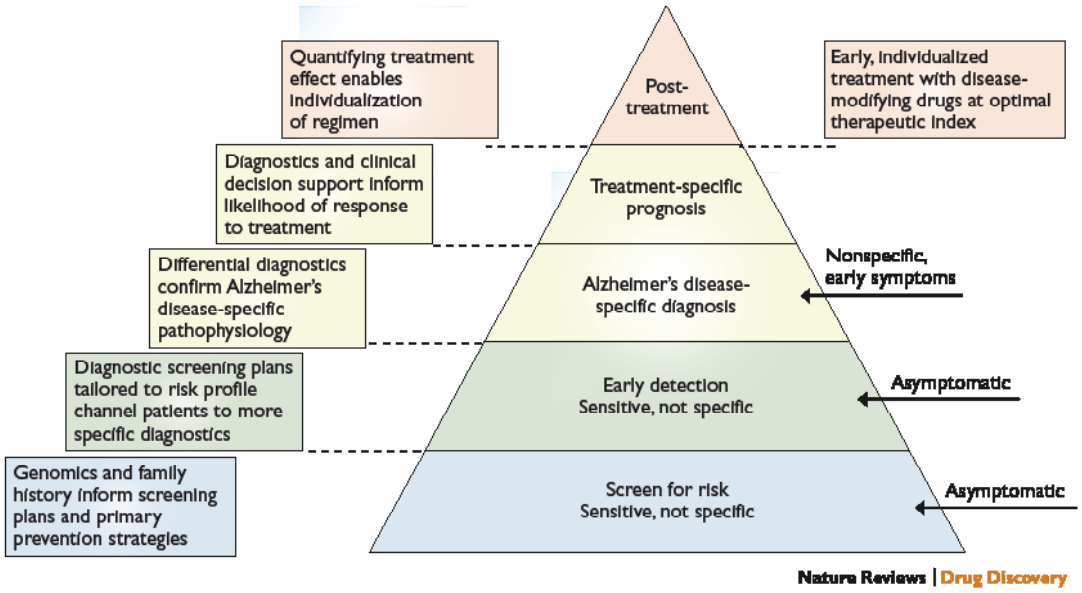
Neuropsychopharmacology also needs new targets for drug development. This has proven to be difficult. The current types of monoamine-directed drugs are over 50 years old,

and it should be noted that both antipsychotics (chlorpromazine) and antidepressants were found merely by chance. Even if these types of drugs ameliorate the clinical symptoms, their mode of action may be unrelated to the central disease pathogenesis. A comparison can be made with acetylcholine esterase inhibitors (which improve cognition in Alzheimer's disease), or dopaminergic drugs (which improves extrapyramidal symptoms in Parkinson's disease), but do not affect the actual causes of these brain disorders. The newer drug variants have been developed along a safe track, i.e. without any risk-taking. I believe research groups and the pharmaceutical industry should work together on leads outside dopamine and serotonin. The focus should be to learn more on the pathogenesis and thereby establish new treatment targets. Such knowledge could come from proteomics studies, but also the discovery of novel genes that are involved in the disease process may provide new leads.

ECNP is always interested to learn ways in which young scientists can be attracted and stimulated to involvement in neuropsychopharmacology. How is this organised in your lab?
Today, at least in Sweden, it is less attractive to become a scientist, especially for an MD, due to a variety of reasons. One aspect is that the financial prospects are not great for a young researcher, when compared to just becoming a physician. Further, research often has to be done in one's spare time. Last, I believe that a pre-doctoral education training programme bringing together students from different specialities (such as chemists, medical doctors, biologists and physicists) would be important to stimulate a broader interest in research.

As a long-standing member of ECNP (since 1988) may I challenge you to do some fortunetelling on the future of neuropsychopharmacology?
I believe neuropsychopharmacology should learn more from other disciplines. Especially, I would like to see clinically oriented longitudinal studies combining neurochemistry, neuroimaging (MRI, PET), neuropsychology and genetics, aiming at gaining more detailed knowledge on the pathogenesis of this group of disorders. This could in the end lead to new leads for treatment strategies.

For the congress, I would like to support the development of a symposium where one person would give a review on a topic, after which two other persons would present the pro's and con's and concluded by an overall discussion. In a small group setting this is done at the ECNP Targeted Expert Meetings, but I believe there are topics of interest to a larger group of scientists that could well be dealt with in the setting of a symposium at the ECNP Congress.



Hampel H, Frank R, Broich K et. al. Biomarkers for Alzheimer's disease: academic, industry and regulatory perspectives. Nat Rev Drug Discov. 2010 Jul;9(7):560-74 (permission for use asked).

Translation of biomarkers
The translation of biomarkers from being research tools that can be used in clinical trials, to being aids in regulatory decision-making and to being commercial diagnostics that can aid clinical decision-making facilitates the efficient selection of different patient populations:

- *When used as research tools, biomarkers can identify a large population of individuals (that is, the base of the pyramid) who may benefit from low-cost and safe primary prevention strategies.*
- *Diagnostic biomarkers can be used in smaller populations with early forms of the disease which, when proven by a definitive diagnosis, warrants a more expensive and riskier therapeutic intervention that is tailored to their individual pathophysiology and monitored for efficacy (that is, the pinnacle of the pyramid). This role of biomarkers for diagnostics in individualisation of therapy is also enabled by information technology, known as 'clinical decision support'.*

Different biomarkers such as genomics and molecular imaging will each be useful in a diagnostic flow. This begins with risk assessment (enabling primary prevention) to screening (for early detection and early intervention), to diagnosis and prognosis (for staging and best choice of therapy) and finally to monitoring treatment effect (for true individualization of treatment). This flow starts from tests with high sensitivity but low specificity and low cost to those with increasing specificity and value with potential for longitudinal quantification. Patients will enter at different points in this diagnostic flow (from the bottom to the top of the pyramid) according to the manner in which they present; the two main options being with or without symptoms.

Report of the ECNP Award Jury 2010

Tomas Hökfelt, Sweden
Chair

The winner of this year's award is Kaj Blennow, professor in clinical neurochemistry, University of Gothenburg, and senior consultant (överläkare), Neurochemical Laboratory, Sahlgren's Hospital, Gothenburg, Sweden.

A pioneer in the development of biomarkers for Alzheimer's disease, Kaj Blennow has made very significant contributions with relevance for both patient care and drug development for Alzheimer's disease, one of the most prevalent and devastating psychiatric disorders. He has also extended his neurochemical innovations to other fields of neuropsycho-pharmacology, such as schizophrenia and depression.

Kaj Blennow is recognised as one of the world's leading researchers into Alzheimer's disease.

Report from the 2010 ECNP-EPA Seminar in Neuropsychopharmacology

Pavel Mohr, Czech Republic
President Czech NeuroPsychopharmacological Society

From 22 to 24 April 2010, the European College of Neuropsychopharmacology (ECNP) in cooperation with the Czech Neuropsychopharmacological Society (CNPS) organised a seminar for young clinicians and researchers in Trest, Czech Republic.

One of the main ECNP missions is supporting young psychiatrists and scientists. In addition to the annual spring ECNP Workshop in Nice, summer School of Neuropsychopharmacology in Oxford, and ECNP Research Grant for Young Scientists, ECNP regularly organises seminars in neuropsychopharmacology in the countries of Eastern and Central Europe. The successful series of events has been so far held in Poland, Estonia, Turkey, Bulgaria, Romania, Slovakia and Hungary. This year it took place in the Czech Republic.

The seminars are always run by the top experts in the field. The teachers of the Czech seminar were a member of the ECNP Executive Committee, Celso Arango from Madrid who is also chair of the ECNP Educational Committee, and a member of the ECNP Scientific Programme Committee 2010-2011, Alessandro Serretti from Bologna. Unfortunately, Sven Ove Ögren from Stockholm could not attend; his trip was cancelled due to an 'act of God' named Eyjafjallajökull. The local tutors were Lucie Bankovska-Motlova, Jiri Horacek, and Pavel Mohr.

The participants highly appreciated all introductory lectures, Celso Arango's presentations 'How to prepare a manuscript' and 'How to prepare a scientific presentation' and Alessandro Serretti's talk 'Gene-environment interaction' were used to demonstrate some methodological pitfalls and possible solutions. However, the main focus of the seminar was in the interactive work in groups led by the faculty members. The total number of 27 participants, young researchers and clinicians, presented and discussed their projects. The quality of papers presented in the groups varied, reflecting different experience in research and presentation skills. For some of the participants it was their very first oral presentation in English. Nevertheless, the feedback was always kind and positive, aiming to encourage and improve research work. The tutors also appreciated the fact that trainees were able to prepare their PowerPoint presentations overnight. The informal but working atmosphere at the Trest Castle was further reinforced by a joint trip to the nearby Renaissance town and Castle of Telc.

The three best projects were selected from both groups and their authors presented them to the plenary forum. There was a tendency to support and encourage those who did not have extensive foreign experience. The top three projects were chosen without ranking order and their authors were invited to attend the 23rd ECNP Congress in Amsterdam to present a poster with their own data. Selected unanimously were:

- Michaela Fujaková from Prague Psychiatric Center: 'A potential antipsychotic effect of group II/III metabotropic glutamate receptor agonist LY379268 on behavioural changes and quantitative EEG in ketamine model of psychosis – an animal study'.
- Monika Klirova from Prague Psychiatric Center: 'Clinical response of neuronavigated rTMS in the treatment of auditory hallucinations'.
- Alena Machalová from Department of Pharmacology, Medical Faculty of the Masaryk University in Brno: 'Effects of modafinil in preclinical experiments'.

The ECNP Seminar in Neuropsychopharmacology not only awarded the winners but mainly rewarded all participants with invaluable experience and inspiration. Great thanks for smooth organisation should go to Iva Dobruska from the co-organising CNPS.

Reports on the ECNP Targeted Expert Meetings (TEMs) 2010

TEM Addiction

Rainer Spanagel, Germany
Coordinator

Progress in understanding the genetic basis of addiction is derived from both preclinical studies in animals and human studies. In this TEM we discussed in a multidisciplinary group of researchers from 12 different countries a new translational approach for the integration of data sets that derive from forward genetics in animals and genetic association studies including genome wide association studies (GWAS) in humans.

The aim of forward genetics in animals and association studies in humans is to identify mutations (e.g. SNPs) that produce a certain phenotype, i.e. 'from phenotype to genotype'. The repertoire of forward genetics includes the generation of random mutations in an organism, either by radiation or by a chemical mutagen such as N-ethyl-N-nitrosourea (ENU), and then through a series of breeding of subsequent generations, isolating individuals with a phenotype relevant for addictive behaviour. Most powerful however, in terms of forward genetics, is combined QTL analysis and gene-expression profiling in recombinant inbreed rodent lines or genetically selected animals for a specific phenotype, e.g. high versus low drug consumption. By Bayesian filtering genomic information from forward genetics in animals are then combined with data from a meta-analysis of GWAS on a similar addiction-relevant phenotype. This integrative approach generates a robust candidate gene list that has to be functionally validated by means of reverse genetics in animals, i.e. 'from genotype to phenotype'.

We conclude that studying addiction-relevant phenotypes and endophenotypes by this convergent functional genomics approach will allow us to pin down the genetic determinants of addictive behaviour. A state of the art review paper has now been submitted to the ECNP journal *European Neuropsychopharmacology* and a symposium on this topic will be presented at the 24th ECNP Congress in Paris, 3-7 September 2011. We are really grateful to ECNP for organising this high-calibre two-day TEM meeting. All 30 attendees benefitted enormously!

TEM Child and Adolescent Neuropsychopharmacology

Jan Buitelaar, The Netherlands
Coordinator

The Targeted Expert Meeting on Child and Adolescent Neuropsychopharmacology was focused on Attention Deficit Hyperactivity Disorder (ADHD).

The first series of three presentations discussed new molecular pathways in ADHD and their basic, translational and clinical implications. Barbara Franke reviewed the results of the association studies of classic candidate genes, linkage studies and the newer generation of genome-wide association studies (GWAS). Although GWA studies in ADHD have reported few so far, if any, genome-wide significant findings, many interesting and potentially useful genetic variants may be found among the top findings. These do not support the involvement of the classic neurotransmitter genes (e.g. of the dopaminergic, serotonergic or noradrenergic neurotransmission) but genes involved in basic processes like cell division, adhesion (especially via cadherin and integrin systems), neuronal migration, and neuronal plasticity. One of the newer candidate genes is CDH13. The GWAS results point further to the relevance of shared genetic factors across multiple psychiatric disorders as well as shared genes between psychiatric disorders like ADHD and somatic conditions such as diabetes mellitus and cardiovascular disease. Recent papers also indicate that in an as yet unknown number of cases ADHD is due to major rare variants such as copy number variations. Strategies for the future are to perform whole genome or exon sequencing to understand the prevalence of the rare variations, to perform translational studies in animal and cell models, and intensify bioinformatic analyses to integrate the various findings. Phil Asherson drew upon further implications of these newer findings, and Klaus-Peter Lesch illustrated the value of using knock-out models of these newer candidate genes in ADHD and of studying the functional consequences of these genes in an imaging genetics approach.

The second series of presentations was about brain imaging in ADHD and its translational aspects. Kerstin Konrad first commented on some practical issues, such as that the sample of ADHD children recruited for brain imaging studies may not be representative of the larger clinical population of ADHD patients, since more severe patients tend not to be selected or tend to drop out. Findings of brain anatomy indicate widespread abnormalities in structural brain development with both regional decrease and increase of grey matter and decrease of white matter. Most of the differences appear to be stable over age. A new idea is that functional abnormalities such as attentional lapses and other cognitive impairments in ADHD are due to an abnormal default-mode network (DMN), i.e. a DMN that stays activated while performing on a task or interferes with task performance. Most exciting are newer data of patterns of abnormal connectivity between neural systems in ADHD, and beneficial effects of both medication and intensive cognitive training on these connectivity problems. Sarah Durston stressed the importance of making brain imaging findings clinically meaningful. She showed that different cognitive subtypes of ADHD (deficits in control, reward, timing and speed-accuracy tradeoff) had a distinctive brain imaging profile. Nikos Makris made a close connection to this presentation in his talk about a neural systems analysis of ADHD in adults.

The third component in this TEM was about the use and clinical applications of animal models of ADHD. Terje Sagvolden extensively discussed the merits of the spontaneous hypertensive rat (SHR) model of ADHD and explained the Developmental Dynamic Theory of ADHD that emphasises impairment of reinforcement delay gradients. Jeffrey Glennon provided a critical appraisal of the SHR and argued that we need a variety of animal models that reflect the clinical heterogeneity of ADHD. Trevor Robbins discussed a neural-systems approach based on a refined dissection of the various fronto-striatal circuits in animals and functional psychopathology approaches in humans.

All presentations gave rise to lively and interesting discussions and debate, and to various new ideas for research and translation studies. These will be described in a road map that identifies critical gaps in current knowledge and present priorities in research.

Continued on page 8

TEM Neurological Disorders

Hans Lassmann, Austria
Coordinator

On behalf of
Pia Maria Amato, Italy; Bernhard T. Baune, Australia; John De Luca, USA; Anthony Feinstein, USA; Nanna Figved, Norway; Massimo Filippi, Italy; Alexandra Kutzelnigg, Austria; Walter Pirker, Austria and Tjalf Ziemssen, Germany.

Multiple sclerosis (MS) is the most common neurological disease of young adults in the Western world. It is a chronic inflammatory disease, which primarily leads to focal demyelinating lesions in the brain and spinal cord. Thus, MS is a neurological disease and patients are in general seen and treated by neurologists. However, patients also suffer from cognitive impairment and a large spectrum of psychiatric syndromes. These aspects of the disease are frequently neglected in patient care and only a little research has been performed to define these conditions, to determine their pathogenetic mechanisms and to develop and validate their treatment. The aim of this TEM 2010 was to discuss the state of the art of our knowledge and to identify areas of future research in this area.

Cognitive impairment in MS
Cognitive impairment is a very frequent clinical problem in MS patients. In contrast to classical dementia, cognitive disturbances in MS patients mainly affect the speed of information processing, executive functions, perceptional processing and memory acquisition. Its differentiation from fatigue is difficult and requires extensive neuropsychological testing. Particularly severe cognitive impairment is seen in a subset of patients with childhood onset of MS.

Several studies have addressed the structural correlates of cognitive impairment seen by MRI or pathology. These studies identified multiple potential structural substrates. They include focal white matter lesions, in particular in the frontal white matter, the thalamus and hippocampus, diffuse injury in the global white matter as well as cortical demyelination or atrophy. Correlations between single MRI parameters and clinical severity are weak, but can be improved by the use of composite parameters. The situation is further complicated by compensatory changes, seen in functional MRI especially during early phases of the disease. In addition, pre-existing cognitive reserve diminishes clinical severity of cognitive disturbances. An unresolved question deals with cognitive dysfunction in patients at the very early stages of the disease. In such patients cognition is impaired even in the absence of structural lesions seen by MRI or pathology. It is, thus, likely that the inflammatory process in the nervous system itself may lead to functional disturbance of cognition. Potential candidates for such effects are pro-inflammatory cytokines, which are able to induce sickness behaviour in experimental animals.

Even less is known regarding the therapy of cognitive impairment in MS. Current anti-inflammatory or immunomodulatory therapies show some effects, which seem to be related to the anti-inflammatory action. Pharmacotherapy with cholinesterase inhibitors has been tested in few studies with inconsistent results. Cognitive rehabilitation revealed positive effects, but only few studies met sufficient quality standards.

Neuropsychiatric syndromes in MS
Neuropsychiatric syndromes in MS patients include depression, bipolar disorder, euphoria, pseudobulbar affect and in rare instances psychosis.

Depression is most frequent and depressive episodes occur in 25 to 50% of the patients. One problem in the diagnosis of depression in MS patients is the overlap of somatic symptoms due to depression with somatic symptoms caused by focal MS lesions in the brain. These include fatigue, change in appetite, weight loss or weight gain, altered sleep and mentation. The importance of neuropsychiatric syndromes in MS is highlighted by the fact that about 15% of MS patients are first seen by a psychiatrist before the disease is diagnosed and that MS patients have a seven times higher suicide rate compared to the normal population. The pathogenetic basis of depression in MS is largely unknown. MRI shows some correlations with focal lesion load (pre-frontal and supra-insular), brain atrophy (hippocampus; CA2/3, dentate gyrus) and white matter lesions, which may disturb the connection between amygdala and pre-frontal cortex. As with cognitive disturbances, pro-inflammatory cytokines and dysfunction of the hypothalamic-adrenal axis were also discussed, but these concepts largely reside on a speculative basis.

Regarding therapy the situation is rather unsatisfactory. It is generally suggested that established treatments for psychiatric diseases should be applied in MS patients, when necessary. Controlled clinical studies, which prove their efficacy in MS patients, are nearly non-existing. Furthermore, it is unclear whether these treatments may interfere with the disease process of MS itself.

Conclusions:
This TEM clearly identified major future needs in MS research in the areas of cognitive impairment and neuropsychiatric syndromes. The importance of these topics for the patients is undisputed, but current knowledge on the underlying mechanisms and treatment is sparse. Much more intense interdisciplinary interaction between neurologists, psychiatrists and basic scientists is mandatory to advance this field in the future.

TEM Psychotic Disorders and Antipsychotics

Shitij Kapur, United Kingdom
Coordinator

The last decade has seen an explosion in new information in three areas related to psychotic disorders and antipsychotics: genetics, cognition and early intervention. The pur-

pose of this TEM was to take stock of these developments and identify the real advances and imminent challenges.

Patrick Sullivan opened with a lecture on the genetics of schizophrenia and highlighted how the emerging data from the Genome-Wide Association Studies and the new and replicable 'copy number variants' will likely overwrite the findings from the previous decade's many case-control studies. Dan Rujescu presented some of the latest findings with CNV related to those identifiable genes that can be plausibly linked to neurobiology, while Florence Thibaut exemplified potential translational opportunities based on genetic findings.

The section on cognition discussed the emerging preclinical, clinical and commercial interest in this field. Terry Goldberg opened by reviewing the history of cognition in schizophrenia, but also the blind alleys and the confounded results from inadequate designs. He communicated the impression that most current atypical antipsychotics had cognitive-enhancing effects. Dwight Dickinson showed how the vast array of schizophrenia deficits could be conceptualised as pointing to a major single underlying 'latent variable' and showed how it was becoming possible to link these to the emerging genetics. Daniel Umbricht showed evidence of electrophysiological deficits and discussed how these might be biomarkers for early drug development. While the genetics and imaging discussions highlighted the significant new knowledge and sophistication of technology, it was also acknowledged that both areas were not as yet ready for immediate clinical application.

Finally, it was in the area of early intervention that the meeting acknowledged true clinical advances. Philip McGuire provided evidence on the predictive power of the prodrome concept and the biological validation of the concept by demonstration of distinctive brain dysfunction. Peter Falkai demonstrated evidence that non-somatic interventions (exercise) were powerful modifiers and Merete Nordentoft demonstrated how large-scale intervention was possible and led to enduring changes even after the intervention was over.

The presentations were followed by active discussions and with the impression that while genetics and cognition demonstrated potential, early intervention and prodromal studies were changing care on the ground.

Highlights of the 2010 ECNP School of Neuropsychopharmacology

Maria Vrijmoed-de Vries, The Netherlands
Editor

Once more the organisers of the (second) ECNP School of Neuropsychopharmacology, Guy Goodwin and his personal assistant Lucy Curtin, in collaboration with the staff of the ECNP Office, made it possible for 41 young psychiatrists from 20 different European countries to participate at the school on grants from ECNP, based on the recommendation of members of the ECNP Board of National Societies.

On Sunday evening Joseph Zohar, at that time president-elect, opened the school with a presentation on what is ECNP, what can ECNP do for the young scientists and what ECNP expects from them now and in the future.

Over the following four and a half days experts and previous school participants from seven different European countries presented a broad spectrum of lectures on diagnosis and treatment of brain disorders and methodology of clinical trail analyses, case history workshops and sessions on how to enhance national training experience. The latter sessions were new this year and consisted of participants from the 2009 edition of the school presenting how they have used their experience from the school in setting up training sessions at their home institutes, followed by an interactive discussion moderated by two of the senior speakers of that day. The aim of the sessions was to show the participants how and stimulate them to translate the school experience into training possibilities at their home institutes.

As a follow-up of the evaluation from last year, ECNP has asked for and received accreditation for the school by the European Accreditation Committee in CNS (EACIC). Twenty-four participants applied for CME points. From their evaluation it is obvious that the ECNP School of Neuropsychopharmacology fills a need in the international training of promising young psychiatrists.

If you feel you or one of your co-workers fulfil the profile of a participant, you are welcome to fill out the application form and forward it by e-mail to the member of the ECNP Board of National Societies in your country. The form and additional details can be found on www.ecnp.eu.

interview

Alexander Schubert, The Netherlands
Editor

Interview with the president of ECNP: Joseph Zohar, Israel

On Monday 31 August, Joseph Zohar formally took over as ECNP's ninth president. His involvement with the organisation now spans 20 years. Having chaired the organising committee of the ECNP Congress in Jerusalem, Israel, in 1994, he joined the Executive Committee in 1995. Amongst his many roles at ECNP, he served as secretary from 1998 to 2004, vice-president from 2004 to 2007 and president-elect from 2007 to 2010, as well as chair of the ECNP Educational Committee for more than a decade.

In his day job, Joseph is professor of psychiatry at Tel Aviv University, director at the Division of Psychiatry in Chaim Sheba Medical Center, medical director of the Israeli Center of Obsessive-Compulsive Disorder, and chairman of the Israeli Consortium on Post-Traumatic Stress Disorder. He is the author of 250 scientific papers and editor or author of 14 books on refractory depression, obsessive-compulsive disorder and post-traumatic stress disorder. Amongst many other awards, he also won the ECNP Neuropsychopharmacology Award in clinical research in 1998.

How did you first become involved in ECNP?
I returned to Israel in 1987 after three years at the National Institute of Mental Health in Bethesda, Maryland, and I was looking for the kind of larger research environment I had known in the United States. I came across an advertisement for the ECNP Congress in Stockholm. It looked promising, so I submitted an abstract, and it was accepted. The congress turned out to be exactly what I wanted: pan-European in scale, with a good mix of high-quality clinical research and basic science. It was an approach and a community I immediately felt at home in, and I've come to every meeting since.

What's your view of ECNP's role and function as an organisation?
ECNP has done very well in making the congress the largest meeting for brain research in Europe, attracting psychiatrists, neurologists and psychologists. Thanks to the success of the congress, ECNP is in a financially stable situation. Now we have the opportunity – and, actually, the obligation – to go beyond cutting-edge science at the ECNP Congress and to become more broadly relevant – being a source of reliable, evidence-based information for anyone interested in the brain and diseases of the brain, and encouraging dialogue between different stakeholders.

Being broader also means moving beyond our traditional territory of researchers and trying to join – and, of course, influence – larger conversations about brain science and mental health, among policy-makers, physicians, health economists, industry and the general public. In other words, trying to increase our impact beyond the ECNP heartland, especially Eastern Europe, to make sure that these areas also have the opportunity to participate in the rich variety of activities ECNP offers. This, to me, along with focusing on young colleagues and brain researchers, would be truly fulfilling the ECNP mandate.

Where do you see the big challenges for neuropsychopharmacology lying in the next five to ten years?
We've recently witnessed the shutting down of some major neuroscience research centres in Europe, such as GSK, AstraZeneca and MSD. The exact reason for the closures obviously vary from company to company. But behind them lies a familiar mix of problems: the stigma of psychiatric illness, despair at the potential of neuroscience to develop new breakthrough treatments, the targeting of 'soft medicines' by governments in their attempts to control rising health care costs, and the image of the pharmaceutical industry. The challenge is to communicate what drugs can deliver and also how important better tolerability can be in terms of compliance, and how this alone can favourably change the course trajectory. This has to be addressed through better communication, balanced education and focused outreach.

When you look back in three years' time, what would you like to have achieved for ECNP?
I'd like to have secured the foundations of the organisation while moving forward. This would mean retaining the ECNP Congress's leading role in the field, continuing to build on our educational activities, updating the bylaws and developing a code of ethics, all aimed at strengthening our base. It would also mean working to secure the future of the field: encouraging more young scientists, clinicians and physicians to make a career in neuroscience; promoting common standards of excellence in countries with fewer resources for research by promoting scientific exchange. If ECNP can play a leading role in the discussion around neuroscience and pharmacology, by providing those engaged in the debate – whether governments, the media, patients or the public – with the best information available, I'd see that as a major achievement. In other words, to make ECNP *the* organisation in Europe that fosters and communicates knowledge about the brain and brain disorders.

Closing thoughts?
The world's changing at an incredible rate. The challenge for ECNP is to help neuroscience, brain research and our understanding of mental disorders keep up. ECNP is at the centre of this drama. I hope that all our members help in meeting this challenge, and help the organisation to continue translating pre-clinical knowledge into a better understanding of brain mechanisms and consequently a better quality of life for the patients. I'm confident they will.

New at the 23rd ECNP Congress:

Young Researchers' Breakfasts

Celso Arango, Spain
Chair Educational Committee

ECNP is always innovating and a clear example of that is the initiative that took place for the first time at the 23rd ECNP Congress in Amsterdam: the Young Researchers' Breakfasts. The purpose of this activity is to allow junior researchers in neuropsychopharmacology to spend an entire hour with an established senior leader in the field.

The senior researchers who are invited to participate are those giving a plenary lecture during the congress, so the junior researchers have an opportunity to ask more detailed questions about the work presented. Another major objective is to hear first hand recommendations of a successful researcher to those who are starting their careers: the good and the bad, what worked for them and what did not, what they would not do again and what they would have done differently, and the keys to their success, to name a few.

This year the activity took place at 7.45 hours, while the neurons are still fresh, with a nice breakfast provided by ECNP. The young researchers who attended the meeting were able to discuss their concerns and interests with Kaj Blennow and Isabelle Mansuy, and mingle with other young researchers in different areas of expertise. The breakfasts were also attended by members of the ECNP Executive Committee, which provided them with a good opportunity to hear what young researchers expect from ECNP and, as our president always says, what we expect from them!

The ECNP Young Researchers' Breakfasts are open to any young researcher attending the congress and of course to those who have received any of the multiple awards that ECNP gives to young researchers. All you need to do is pre-register (free of charge). More information will become available on the ECNP website www.ecnp.eu the second quarter of 2011.

Looking forward to seeing you all next year at the Young Researchers' Breakfasts that will take place at the 24th ECNP Congress in Paris!

Members' reception and members' lounge

David Nutt, United Kingdom
Past-president

For years the Executive Committee has been considering how to improve the value of ECNP to our 1,000 members and why only around 20% come to the annual ECNP Congress. This year we introduced two initiatives to improve the situation.

The first was the members' reception just before the opening ceremony. This allowed members to meet with each other, the officers and the ECNP Office team before taking their reserved seats at the front of the auditorium. The turnout was pleasingly high and I had a strong sense that people were delighted to have this opportunity to catch up with old friends in this way.

The other innovation was to provide a lounge with drinks, snacks and internet access where members could find a place to sit, meet, work or even just relax away from the hurly-burly of the congress. This was also a great success, perhaps too much so, as the power sockets and coffee were over-subscribed, something we have noted and will rectify next year!

I hope all of you who used these new initiatives found them of benefit and do let us know if there are any other ways we can reward the loyalty of members and make your attendance at the annual congress even more enjoyable.

Poster certificates

In the past years, poster presenters inquired whether it would be possible to receive a certificate to prove to their grant committee that their poster was presented at the ECNP Congress. At the 23rd ECNP Congress, this new features was introduced.
The poster certificates were available for the presenting authors and could be picked up shortly after the poster session on the day the poster was presented. This time, around 27% of the 770 presenting authors picked up their certificate. ECNP anticipates this number will increase at the upcoming congresses once more poster presenters become aware of this possibility.

Participants' experiences

Li-Tzu Li, Taiwan
"I would like to see more basic research that could be applied to clinical so that you can see the potential of basic research."

Cleo Crunelle, The Netherlands
"I liked the topic on pain, useful for clinicians. I joined the Young Researchers' Breakfast, a very good initiative of ECNP. The ECNP Congress is the best congress, especially for young scientists."

Award winners 2010

ECNP Neuropsychopharmacology Award
Kaj Blennow, Sweden

ECNP Lifetime Achievement Award
Moussa Youdim, Israel

ECNP Fellowship Award
Alessandra Berry, Italy
Annette Brühl, Switzerland
Kim Kuypers, The Netherlands
Ciara McCabe, United Kingdom
Carmen Moreno Ruiz, Spain
Olivia O'Leary, Ireland

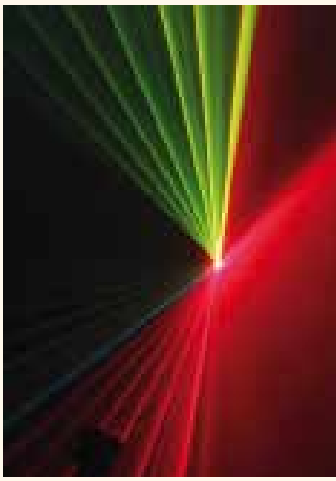
ECNP Poster Award

P.2.c.037	Elena Akimova, Austria
P.5.d.007	Sylvie Bourgoin, France
P.3.d.004	Philipp Csomor, Switzerland
P.1.e.002	Marta Gawlowska, Poland
P.1.d.008	Hideaki Hara, Japan
P.7.a.009	Maria Dorota Majewska, Poland
P.2.e.024	Agnieszka Remlinger-Molenda, Poland
P.4.b.016	Cristina Stern, Brazil
P.6.e.001	Ruth van Holst, The Netherlands
P.3.f.009	Karin Weigelt, The Netherlands

ECNP Travel Award

P.2.c.037	Elena Akimova, Austria
P.3.b.009	Péter Z. Álmos, Hungary
P.1.e.005	Matthijs Bossong, The Netherlands
P.2.a.016	Livia A. Carvalho, United Kingdom
P.2.d.006	Esther Castillo-Gómez, Spain
P.6.b.007	Claudio D'Addario, Italy
P.2.a.023	Jessica Eccles, United Kingdom
P.1.a.018	Agnes Feher, Hungary
P.2.b.013	Alvaro L. García-García, Spain
P.4.b.005	María S. García-Gutiérrez, Spain
P.2.h.002	Javier Gilabert-Juan, Spain
P.1.a.024	Kelli Hiio, Estonia
P.1.c.044	Srdjan Joksimovic, Serbia
P.3.a.010	Ivan Koychev, United Kingdom
P.2.b.010	Oliver Leske, Germany
P.6.f.001	Maartje Luijten, The Netherlands
P.6.b.001	Devesh Mishra, Sweden
P.1.c.029	Francisco Navarrete, Spain
P.1.c.061	Tatjana Nikolic, Serbia
P.4.e.004	Mykhaylo Oros, Ukraine
P.2.b.003	Evelin Painsipp, Austria
P.6.d.024	Katrin Preller, Switzerland
P.7.a.005	Marta Rapado-Castro, Spain
P.2.e.024	Agnieszka Remlinger-Molenda, Poland
P.3.c.001	Arne Risselada, The Netherlands
P.1.c.018	Harriet Schellekens, Ireland
P.5.a.016	Fatma Simsek, Turkey
P.1.c.006	Olga Solovieva, Russia

Other impressions



ECNP-supported symposium at the 51st SCNP Annual Meeting, 12-14 April 2010, Gothenburg, Sweden

Ole A. Andreassen, Norway
Chair of the symposium

The 51st Annual Meeting of the Scandinavian College of NeuroPsychopharmacology (SCNP; www.scnp.org) took place in Gothenburg, Sweden from 12 to 14 April 2010. There were more than 200 participants from all Nordic countries, and the scientific programme ranged from basic research to clinical treatment guidelines.

ECNP supported an educational symposium, 'Drugs in development', which enabled us to have an excellent programme. It was moderated by very well-known researchers in the field, Arvid Carlsson, Sweden, and Hans-Jürgen Möller, Germany.

First, Arvid Carlsson described a new therapeutic agent for Huntington's disease. Then Ole Kristian Kleivenes presented a new approach for schizophrenia treatment. Markus Heilig lectured about new treatment options in alcohol addiction. Finally, a new perspective of immune factors in psychotropic drug development was reviewed by Robert Dantzer.

The seminar was the most attended symposium, and facilitated a very interesting discussion. Overall, it was a well-balanced symposium, with basic and clinical presentations and a high degree of translation. Further, Arvid Carlsson who co-founded SCNP more than 50 years ago, showed with his enthusiasm and novel new data that neuropsychopharmacology research provides a lifelong opportunity for creative minds. The symposium gave an excellent update of important new approaches in drug development, as well as promising new treatment candidates for clinical use.

SCNP will have their next annual meeting on 12-14 April 2011 in Oslo, Norway.

Drugs in development
Chairs: Arvid Carlsson, Sweden
Hans-Jürgen Möller, Germany

Dopamine stabilizers – an update
Arvid Carlsson, Sweden

mGlu2/3 receptor agonists and other drugs in the pipeline
Ole Kristian Kleivenes, Sweden

New treatment options for alcohol addiction
Markus Heilig, USA

Potential utility of immunomodulators in psychiatry
Robert Dantzer, USA

interview

Maria Vrijmoed-de Vries, The Netherlands
Editor

Interview with the winner of the 2010 ECNP Lifetime Achievement Award: Moussa Youdim, Israel

Moussa Youdim was born in Teheran, Iran on 28 February 1940. Currently he is still active in his professorships at the Technion-Israel Institute of Technology and Technion-Rappaport Faculty of Medicine, Haifa, Israel, as well as at distinguished institutions in the USA, Hong Kong, South Korea and Italy.
Moussa Youdim is married and has three children and grandchildren, two from his second wife.

If you look back at the more than 45 years of your involvement in neuropsychopharmacology, what do you consider for yourself the most interesting discovery or research?
I find it hard to select the most interesting discovery or research. I would think the discovery of monoamine oxidase B inhibitors as anti-Parkinson drugs, mainly because we do not know what the function is of this enzyme in the brain, while we do know more about monoamine oxidase A: this regulates the metabolism of serotonin and noradenaline, which are implicated in depressive illness.
I would sooner refer to the fact that I have been, and still am, in a privileged position to play a major role in an interdisciplinary, international environment directed towards the development of diagnostic tools, i.e. biomarkers for brain diseases – especially Parkinson's disease – and from there the development of (potential) disease-modifying drug treatments.

If I was to mention a major achievement I would call it the successful translation of basic work from the laboratory to the clinic and subsequently to drug development. I.e. it is most rewarding that I have been able to conceptualise and develop multimodal neuroprotective and neurorestorative drugs with disease-modifying activities for Parkinson's and Alzheimer's disease as future treatment for these complex disorders.
One such drug is on the market: rasagiline as the first neuroprotective disease-modifying therapy in Parkinson's disease. Two drugs are now in development: a novel multifunctional neuroprotective anti-Alzheimer drug, ladostigil, and the neuroprotective-neurorestorative agent M30 series for Alzheimer's disease, Parkinson's disease and possibly ALS. The latter are brain-permeable iron chelators, brain-selective MAO inhibitors and cholinesterase inhibitors.

Your research includes not only neurodegenerative disorders, but also alcohol addiction and diabetes: is there a common factor?
The common factor is the neurodegeneration and there are very similar cascades of molecular evets that lead to the demise of the neuron. It occurs in alcohol addiction and diabetes as a result of the same cytotoxic process: oxidative stress that leads to cell death. There is a similar relationship with diabetes and Alzheimer's disease and obesity. However, no such connection has been found with Parkinson's disease. We do not know if rasagiline would be effective in alcoholics.

It is great that you are still active in the scientific arena and in addition you have quite a number of professorships all over the world. How are you managing this?
I believe India and China will play a major role in the future of neuropsychopharmacology and I think we, the Western world, should want them to play a part in it!

On a regular basis I spend one month at universities in Hong Kong with visits to China, and two months in South Korea. I have a position of Distinguished World Central Professor at Yonsei University in Seoul, where I do a lot of teaching and direct several research projects in the Department of Biology with Young J. Oh.

In addition I establish collaborations on drug development on neurodegenerative diseases for Varinel Inc. (our own company) as part of the Israeli and South Korean business alliance. Teva Pharmaceutical Industries Ltd is Israel's largest, international generic and innovative pharmaceutical company and I still collaborate with them. They developed rasagiline based on the original research of my colleague John Finberg and myself.

In addition, I still have my own lab with at present only female (!) scientists whom I can completely trust to leave alone when I am travelling. Students in Israel are in general much older and more serious than in the rest of the world due to our obligatory military service. Silvia Mandel and Orly Weinreb have achieved international recognition for their works in my centre.

Your name is attached as (co-)developer of a couple of drugs like the neuroprotective disease modifying drug rasagiline. How does that feel?
It is wonderful. The neuroprotective disease-modifying picture is so complicated and it really is great that it all turned out to really work. It is the dream of every scientist that his basic work will be beneficial for mankind.

I was once woken up at four o'clock in the morning from Dallas, USA, with the news that Parkinsonian patients had had a great clinical response to rasagiline. They wanted to bless me. This result is what you always dream of, however, I can assure you the adrenaline from that message kept me well awake for the rest of the day.

What is your view about the pull out of neuroscience by some pharmaceutical companies?
I think it is a big mistake. There is still so much to gain in the area of neuroscience.

It is unfortunate that Lilly had to halt the development of semagacestat in Alzheimer's disease. Although the intended effect of semagacestat was set in general terms to delay the onset of severe AD and thereby help preserve cognitive and executive functioning and in turn improve patient quality of life, it only works as a gammy secretase inhibitor on one aspect of the amyloid-ß complex. Therefore Lilly continues with the Phase III development of solanezumab, a humanised antibody that binds to ß-amyloid and expects to halt the progress and in the future prevent the development of Alzheimer's disease via that route.

One could also look at neurorestauration, neuromodulation, and drugs that work on the cell cycle for example by monitoring the development of neurons using stem cells with or without drugs. Also pharmacogenetics in Alzheimer's and Parkinson's disease show a promising future. This may be a new opportunity for the smaller innovative companies. They are more careful and often more dedicated to a specific goal. Here the initial development may take place up to and including Phase II.

How is the situation of young scientists in neuropsychopharmacology in Israel?
As you may know very well Israel is endowed with many outstanding neuropsychopharmacologists. In Israel the inhabitants are over-educated and many leave the country to work elsewhere. For those who stay not many jobs are available, so often they become independent entrepreneurs. This latter group is very successful and, like my group, not governmental. From there the small biotech companies emerge that may end up being as big as Teva.

I had to retire at 68; however my centre at Technion continued as one of the nine centres-of-excellence and financially supported by alumni, a situation that is not common in Israel and unique in the world. In this way and together with obtaining research grants from the USA, I am still able to provide young scientists with a chance to develop expertise in neuropsychopharmacology.

If you look at the future what, if you would speculate, will be the greatest innovation in the treatment of in particular Parkinson's and Alzheimer's disease?
I do not have a prediction, more a belief that there will be multimodal drugs that would possess disease-modifying activity, such as we see in AIDS and cancer treatment, in particular the use of specific poly-pharmacology.

We have developed the multimodal neuroprotective drug ladostigil, as a derivative of rasagiline, for Alzheimer's disease possessing cholinesterase inhibitor moiety. In laboratory and animal studies ladostigil has anti-Parkinson, anti-Alzheimer and anti-depressant activity – a very interesting combination since we know that Alzheimer patients do have predisposition to depressive illness and 40% of such patients have Lewy Body disease and extrapyramidal disorder. Ladostigil has now entered phase II controlled clinical studies in Alzheimer's disease.

Also the costs of medicine should be tackled as well as regulatory agencies' resistance to change. Having a single drug possessing more than one pharmacological action would keep the cost lower than giving two or three drugs. It may even have less side effects, since there will not be drug/drug or metabolite/metabolite interaction.

How does it feel to be awarded a lifetime achievement award?
It came as a big surprise. It is an honour and (again ☺) wonderful to be recognised in one's profession by my peers. I developed drugs when nobody believed in it. I was fortunate that I could put my subconscious dream ('serendipity') with some luck into action.

The biggest gratification, however, comes from the patients!

Note from the editor: the Scientific Programme Committee is pleased to announce that Moussa Youdim will be a plenary speaker at the 24th ECNP Congress, 3-7 September 2011, Paris, France

Report of the ECNP Award Jury 2010

Tomas Hökfelt, Sweden
Chair

The winner of the 2010 award is Moussa B. H. Youdim, Finkelstein professor of life sciences and professor of pharmacology, director of the Eve Topf and US National Parkinson Foundation at the Centers of Excellence for Neurodegenerative Diseases Research and Teaching and the Rappaport Family Faculty of Medicine at the Technion-Institute in Haifa, Israel.

Over nearly 50 years in the field, Moussa Youdim has gained a worldwide reputation for research in Parkinson's disease and Alzheimer's disease.

He is especially known for his key role in establishing the importance of the monoamine oxidase A and B and their connection to abnormal brain iron metabolism and the role of brain iron in the pathogenesis of these diseases and in brain function and dysfunction. Moussa Youdim has pursued this research into the clinic, with the development of novel medicines for neurodegenerative diseases.

ECNP Symposium at the 33rd Annual Meeting (CCNP/CAN joint meeting), 14-17 May 2010, Ottawa, Canada

Marco Leyton, Canada
President CCNP

The 2010 CCNP Annual Meeting went very well, not least because it was a joint conference with the Canadian Association of Neuroscience (CAN). As a result, attendance at the meeting was much greater than the norm. Also enhanced was attendance at the ECNP symposium, 'The endocannabinoid system during pre- and postnatal development'. It received a very positive response.

The endocannabinoid system during pre-and postnatal development: critical requirement and vulnerability to insult
Symposium proposer: Ester Fride, Israel †
Chair: Rafael Maldonado, Spain

Endocannabinoids: friends or foes of human reproduction
Mauro Maccarrone, Italy

Wiring of the foetal brain requires endocannabinoids
Andreas Zimmer, Germany

Adolescence: a time of vulnerability to cannabinoids
Daniela Parolaro, Italy

Critical role of the cannabinoid CB1 receptor in cognitive impairment
Rafael Maldonado, Spain

ECNP-supported educational symposium at the 43rd Meeting of the Polish Psychiatric Association, 23-26 June, Poznan, Poland

Janusz K. Rybakowski, Poland
Chair of the symposium

The topic of the ECNP-supported educational session at the 43rd Meeting of the Polish Psychiatric Association was 'Atypical antipsychotic drugs: pharmacological properties and their use in schizophrenia and mood disorders'. The session was organised in collaboration with the Psychopharmacology Section of the Polish Psychiatric Association and was chaired by Janusz K. Rybakowski, president of the section, and Jan Jaracz, the vice-president. The session was well attended and after each presentation a lively discussion ensued.

In the first talk, Adrian Newman-Tancredi presented new mechanisms contributing to the pharmacological profile of atypical antipsychotic drugs. The attention has increasingly focused on combining D2 receptor blockade with 5-HT1A receptor activation. Activation of serotonin 5-HT1A receptors prevents extrapyramidal symptoms (EPS) induced by dopamine D2 receptor blockade, favors dopaminergic neurotransmission in frontal cortex, has beneficial influence on mood and, in rodents, opposes NMDA receptor hypofunction-induced cognitive and social interaction deficits. Third-generation antipsychotics such as aripiprazole, perospirone, bifeprunox, lurasidone and cariprazine combine partial agonism at 5-HT1A receptors with antagonism (or partial agonism) at D2 receptors. They are anticipated to provide therapeutic benefits against a broader range of symptoms of schizophrenia, are essentially free of EPS and exhibit little or no interaction at sites potentially involved in side-effects such as weight gain, metabolic disorders or autonomic disturbance.

The second presentation, by René Kahn, involved a one-year comparison between treatment with a conventional antipsychotic (haloperidol in low doses) and atypical antipsychotics such as amisulpride, olanzapine, quetiapine and ziprasidone in patients with recent-onset schizophrenia, performed within the framework of the EUFEST (the European First Episode Schizophrenia Trial) project. René Kahn is the first author of the main article on EUFEST published in *The Lancet* (2008, 371, 1085). The Polish contribution, with Janusz Rybakowski as country coordinator to the EUFEST, was substantial, since the number of recruited patients from Poland made up nearly one fifth of the whole sample (94 out of 498 patients). It was found that discontinuation rates, global outcome and motor side-effects were in favour of atypical antipsychotics, compared with haloperidol in first-episode schizophrenia.

Jose Goikolea elaborated on the mood-stabilising properties of atypical antipsychotics. Mood-stabilising action involves efficacy both in the acute phase and in long-term prophylaxis, ideally in both poles of the illness or at least improving one pole without worsening the other. All atypical antipsychotics have shown themselves to be efficacious in acute mania. First-generation antipsychotics are also efficacious in mania but involve a higher risk of switch to depression. All clinical guidelines agree in recommending second-generation antipsychotics as first-line treatments of acute mania. Olanzapine was the first atypical antipsychotic to show some efficacy in bipolar depression and currently quetiapine has produced high-quality evidence demonstrating a positive effect on bipolar depression. Olanzapine, quetiapine, aripiprazole and risperidone have shown efficacy both in monotherapy and as add-on treatments to lithium or valproate to prevent affective relapses. Also, a positive study with ziprasidone as an adjunctive treatment has recently been published. According to these trials, prophylactic effect of atypicals in the long term is mainly based on prevention of mania, except for quetiapine, which shows a similar ability to prevent mania and depression.

Atypical antipsychotic drugs: pharmacological properties and the use in schizophrenia and mood disorders
Chairs: Janusz K. Rybakowski, Poland
Jan Jaracz, Poland

Pharmacology of atypicality of antipsychotic drugs
Adrian Newman-Tancredi, France

Are atypical antipsychotics better in schizophrenia than conventional ones?
René Kahn, The Netherlands

Mood-stabilising properties of atypical antipsychotics
Jose M. Goikolea, Spain

ECNP Scientific Gathering at the 7th FENS Forum of European Neuroscience, 3-7 July 2010, Amsterdam, The Netherlands

Michel Hamon, France
Chair ECNP Scientific Programme Committee
Hans-Ulrich Wittchen, Germany
Co-chair of the symposium

Although effective treatments have become available over the past 50 years and despite considerable attempts to implement improved methods of recognition, short and long-term care, depression still ranks among the most challenging health care problems worldwide. According to the latest epidemiological data, depression ranks as number one of all medical clinical disorders in terms of the 'disability-adjusted life years' burden in Europe and Northern America, accounting for almost 10% of the total disease burden of all mental, neurologic and somatic diseases. This constitutes a considerable increase in comparison to previous estimations in 90s, attributed partially to a higher incidence of depression, particularly in the young and increased life expectancy and thus longer time spans spent with the disease in the elderly. In addition, however, this increase also reflects significant ongoing failures in the provision of prevention, treatment and care even in the most developed mental health care systems. Particular critical failures are (a) the lack of effective primary preventive interventions, (b) the failure to recognise and diagnose a considerable proportion of all patients suffering from the disease, and (c) the failure to ensure that depression patients are treated earlier and that an optimal treatment is provided according to state-of-the-art guidelines.

Extensive and multifaceted basic, clinical and epidemiological efforts are under way to identify with increasing sophistication the predicted multiple pathways and interactions between genetic, other neurobiological and environmental

factors that are involved in the onset and progression of depressive disorders in the young and the old. Such improved knowledge is expected to open up a range of targets for improved intervention strategies including improved medication and cognitive-behavioural strategies. Recent extensive neuroimaging investigations have demonstrated that severe depression is associated not only with functional alterations of neural circuitries involved in information processing and emotional regulation, but also with anatomical changes in the brain. In particular, decreased volume of the hippocampus, reduction of dendritic arborisation, loss of astrocytes and neurons, and deficiency in adult neurogenesis in this limbic area have been consistently reported in depressed patients. Furthermore, signalling pathways involved in such regression phenomena appeared closely related to those involved in neurodegenerative diseases, and cognitive deficits in depressed patients can be confounded with prodromal symptoms of such diseases, adding to increasing clinical epidemiologic evidence of a close connection of classical neurodegenerative diseases and depression.

Because of the increasing evidence of close links between neurobiological features of depression and those underlying neurodegenerative disorders, ECNP has organised a Scientific Gathering at the 7th FENS Forum on Sunday 4 July 2010 from 18.45 to 20.15, to review evidence with regard to possible shared mechanisms and processes. The set-up of the large-scale forum meeting allowed thorough and fruitful scientific exchanges between neuroscientists participating in the 7th FENS Forum and representatives of ECNP.

Hans-Ulrich Wittchen focussed on the evolution and determinants of depressive disorders across the life span, and compared clinical-epidemiological incidence patterns of depression with those of neurodegenerative diseases *sensu stricto*, especially Alzheimer's disease and Parkinson's disease as well as their overlap (comorbidity). Despite considerable evidence for an overlap of both groups of conditions in the elderly, he found no indications for a potential role of neurodegenerative mechanism in depression overall. This suggests that there might be specific depressive sub-phenotypes in which exploration of neurodegenerative mechanisms might be particularly useful.
Bill Deakin gave an overview of the clinical features of depression, with consideration to the most recent development in genetic approaches aimed at identifying candidate genes involved in vulnerability/resistance personality traits and reviewed their potential relations to mechanisms of neurodegeneration.

Philip Fossati discussed the most recent neuroimaging and neuropsychological features of depression in the context of the 'neurodegenerative hypothesis' of the disease.

Eero Castrén presented a critical overview of the relationships between depression-associated behavioural deficits and neuroplastic phenomena in specific brain areas, as inferred from relevant animal models.

The gathering was indeed a great success. The auditorium was almost full with hundreds of people. We believe this format is excellent and should be continued, possibly linked to having explicit outreach activities to stimulate young scientists to join us!

Is depression a neurodegenerative disorder?
Chairs: Bill Deakin, United Kingdom
Hans-Ulrich Wittchen, Germany, secretary ECNP

Depression over the life span: a major societal burden and research challenge
Hans-Ulrich Wittchen, Germany

Clinical phenotype/genotypes of depression
Bill Deakin, United Kingdom

Neuroimaging and neuropsychological evidence of cognitive deficits in depression
Philip Fossati, France

Neuronal plasticity, neurotrophic factors and depression
Eero Castrén, Finland

General discussion

XVIII Annual Conference of the Bulgarian Psychiatric Association

Luchezar G. Hranov, Bulgaria

The 18th Annual Conference of the Bulgarian Psychiatric Association (BPA) was held from 5 to 7 November 2010 in the famous spa resort Hissar, situated in the geographic centre of the country. It gathered an impressive number of more than 400 attendees out of the 630 Bulgarian psychiatrists. For the first time in quite a long period there were a lot of authors' papers for presentation. The main topics of the conference were schizophrenia, generalised anxiety disorder, somatisation, suicide, pharmacotherapy of depression, psychoses and dementias, brain stimulation, standards and funding of mental healthcare in Bulgaria.

Among the guest lecturers at the conference were the respected ECNP members Stuart Montgomery and Zoltán Rihmer, who spoke on contemporary findings in the fields of pharmacotherapy of depression and suicidology. Academician Mila Vlaskovska presented innovations in the creation and production of antipsychotics, and the most-awarded Bulgarian neurologist Latchezar Traykov discussed contemporary developments in diagnosis and treatment of depression. Gabor Gazdag from Hungary held a lecture and a workshop on ECT. The attendance was impressive at all times.

Among the posters was the work of Georgi Hranov and Naomi Fineberg on obsessive-compulsive disorder and tics, supported by the ECNP Research Grant for Young Scientists.

It was an inspiring and satisfying scientific event held in beautiful natural surroundings and bearing new hopes for the future of psychiatric research and practice in Bulgaria.

ECNP Symposium at the CINP Biennial International Congress, 6-10 June 2010, Hong Kong

Robert H. Belmaker, Israel
Chair of the symposium

The ECNP symposium titled 'The yin and yang of bipolar disorder' at CINP Hong Kong was held on Thursday 10 June in the morning, the last day of the conference, but it was extremely well attended. The audience included numerous psychiatrists from China, Korea, Japan, Indonesia and India who might not otherwise been exposed to these ECNP speakers. It is hoped that this will be a step toward further ECNP/CINP cooperation for the benefit of world neuropsychopharmacology.

The yin and yang of bipolar disorders
Chairs: Guy Goodwin, United Kingdom
Robert Belmaker, Israel

Early diagnosis and intervention in bipolar disorder
Michael Bauer, Germany

Genes and environmental vulnerability factors in bipolar disorders
Stéphane Jamain, France

Neurobiological dysfunctions in bipolar disorders
Guy Goodwin, United Kingdom

The role of typical antipsychotics in the treatment of bipolar disorders
Eduard Vieta, Spain

Passed away

Prof. Tatiana B. Dmitrieva, M.D.
Serbsky National Research Center
Social and Forensic Psychiatry,
Moscow, Russia

Meetings national societies

Czech NeuroPsychopharmacological Society (CNPS)
53rd Czech-Slovak Psychopharmacological Conference
5-9 January 2011, Jesenik Spa, Czech Republic
Information: www.cnps.cz

Swiss Society of Biological Psychiatry (SSBP)
31st Annual meeting – The ageing brain and psychiatry
28 January 2011, Lausanne, Switzerland
Information: www.ssbp.ch

German Association of Neuropsychopharmacology and Pharmacopsychiatry (AGNP)
27th Symposium of the Arbeitsgemeinschaft für Neuropsychopharmacology and Pharmacopsychiatry
5-8 October 2011, Munich, Germany
Information: www.agnp.de

Meetings related organisations

3rd European Neurological Conference on Clinical Practices (ENCCP)
28-30 January 2011, Lisbon, Portugal
Information: www.paragon-conventions.net/enccp2010

10th International Conference on Alzheimer's & Parkinson's Diseases
9-13 March 2011, Barcelona, Spain
Information: www.kenes.com/adpd

19th European Congress of Psychiatry (EPA)
12-15 March 2011, Vienna, Austria
Information: www.epa-congress.org

4th World Congress on Women's Mental Health
16-19 March 2011, Madrid, Spain
Information: http://www2.kenes.com/iawmh/Pages/Home.aspx

9th Göttingen Meeting of the German Neuroscience Society
23-27 March 2011, Göttingen, Germany
Information: www.nwg-goettingen.de/2011

Advancing Drug Discovery for Schizophrenia
9-11 May 2011 New York, USA
Information: www.nyas.org/schizophrenia2011

34th annual meeting Canadian College of Neuropsychopharmacology (CCNP)
20-24 May 2011, Montreal, Canada
Information: www.ccnp.ca

XXVth International Symposium on Cerebral Blood Flow, Metabolism, and Function &
Xth International Conference on Quantification of Brain Function with PET
24-28 May 2011, Barcelona, Spain
Information: www2.kenes.com/brain/pages/home.aspx

Ninth international conference on Bipolar Disorder
9-11 June 2011, Pittsburgh, USA
Information: www.9thbipolar.org

15th World Congress of Psychiatry
18-22 September 2011, Buenos Aires, Argentina
Information: www.wpa-argentina2011.com.ar

50th ACNP Annual Meeting
4-8 December 2011, Waikoloa, Hawaii
Information: www.acnp.org

12th International Stockholm/Springfield Symposium on Advances in Alzheimer Therapy
9-12 May 2012, Stockholm, Sweden
Information: www.siumed.edu/cme/alzheimer

Calendar of ECNP Meetings

Congresses

24th ECNP Congress	3-7 September 2011	Paris, France
25th ECNP Congress	13-17 October 2012	Vienna, Austria
26th ECNP Congress	5-9 October 2013	Barcelona, Spain
27th ECNP Congress	30 August-3 September 2014	Helsinki, Finland
28th ECNP Congress	29 August-2 September 2015	Amsterdam, The Netherlands
29th ECNP Congress	17-21 September 2016	Vienna, Austria
30th ECNP Congress	2-6 September 2017	Paris, France
31st ECNP Congress	6-10 October 2018	Barcelona, Spain
32nd ECNP Congress	7-11 September 2019	Copenhagen, Denmark

For further information:

Organising secretariat 24th ECNP Congress
Colloquium Brussels
6, Avenue E. Van Nieuwenhuyse
1160 Brussels, Belgium
T: +32 2 777 0188
F: +32 2 779 5960
organisingsecretariat@ecnp2011.eu

Scientific secretariat
ECNP Office
P.O. Box 85410
3508 AK Utrecht, The Netherlands
T: +31 30 253 8567
F: +31 30 253 8568
www.ecnp.eu

Workshops

3-6 March 2011	ECNP Workshop on Neuropsychopharmacology for Young Scientists in Europe, Nice, France Topics: - Molecular neuropsychopharmacology - Behavioural pharmacology - Clinical neuropsychopharmacology - Variable topic ⟶ Schizophrenia: towards new drug targets
15-18 March 2012	ECNP Workshop on Neuropsychopharmacology for Young Scientists in Europe, Nice, France - Variable topic ⟶ Depression towards new drug targets
7-10 March 2013	ECNP Workshop on Neuropsychopharmacology for Young Scientists in Europe, Nice, France - Variable topic ⟶ Anxiety towards new drug targets

Stand-Alone Meeting

6-7 March 2011	ECNP Summit on the future of CNS drug research in Europe, Nice, France

Regional Meeting

14-16 April 2011	11th ECNP Regional Meeting, St. Petersburg, Russia

Seminar

7-29 April 2011	ECNP-EPA Seminar, Estonia

School

3-8 July 2011	ECNP School of Neuropsychopharmacology, Oxford, United Kingdom

Consultation Meetings

18-20 March 2012	ECNP Consultation Meeting, Nice, France
10-12 March 2013	ECNP Consultation Meeting, Nice, France

For further information:

Secretariat
ECNP Office
P.O. Box 85410
3508 AK Utrecht
The Netherlands
T: +31 30 253 8567
F: +31 30 253 8568
secretariat@ecnp.eu
www.ecnp.eu

For regular updates on ECNP initiatives please visit www.ecnp.eu

ecnp matters

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Edited by
Maria Vrijmoed-de Vries, The Netherlands

Contributors
Celso Arango, Spain
Kaj Blennow, Sweden
Ligia Maria Bohn, The Netherlands
Jan Buitelaar, The Netherlands
Robert H. Belmaker, Israel
Cleo Crunelle, The Netherlands
Guy Goodwin, United Kingdom
Michel Hamon, France
Petra Hoogendoorn, The Netherlands
Tomas Hökfelt, Sweden
Luchezar G. Hranov, Bulgaria
Shitij Kapur, United Kingdom
Hans Lassmann, Austria
Marco Leyton, Canada
Li-Tzu Li, Taiwan
David Nutt, United Kingdom
Pavel Mohr, Czech Republic
Janusz K. Rybakowski, Poland
Rainer Spanagel, Germany
Alexander Schubert, The Netherlands
Maria Vrijmoed-de Vries, The Netherlands
Suzanna Tjoa, The Netherlands
Hans-Ulrich Wittchen, Germany
Moussa Youdim, Israel
Joseph Zohar, Israel

Graphic Design
Pieter Hoogeveen, Design and Art Direction
The Netherlands

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Teewes Textpresse Grafische Bedrijven
The Netherlands

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European College of Neuropsychopharmacology

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24th ECNP Congress

15 March 2011 deadline for:

- **Call for papers**
- **Call for applications**
  Fellowship and Travel Awards
- **Early registration**

Paris

3-7 September 2011

Visit www.ecnp.eu for further information

25th ECNP Congress

Call for symposium proposals

Deadline: 15 March 2011

Vienna

13-17 October 2012