ECNP SEMINAR

9-11 November 2012

Nafplio, Greece

National and Kapodistrian University of Athens, Medical School, 1st Department of Psychiatry, Eginitio Hospital
Introduction

The European College of Neuropsychopharmacology (ECNP) was established in 1987 on the initiative of scientists and clinicians working in Europe in the convergent disciplines in neuropsychopharmacology and related neurosciences.

ECNP aims to widen knowledge in regard to central nervous system disorders, and to increase awareness, recognition and improvement of the treatment of these disorders. To fulfil this aim ECNP organises, amongst others, yearly the ECNP Congress that comprises at least 3 plenary lectures, 28 symposia and 6 educational update sessions. The latter sessions target issues such as updates on evidence-based treatment and new developments in the preclinical area that influence the clinical field. The annual meeting attracts more than 7,000 participants and is considered to be the largest event in neuropsychopharmacology in Europe.

ECNP also supports on an annual basis participation of 100 young psychiatrists and researchers in an intensive three-day Workshop in Nice. Other activities of ECNP include the journal European Neuropsychopharmacology that promotes scientific knowledge along with publishing consensus statements. These consensus statements are products of an annual meeting with delegates from the scientific community in neuropsychopharmacology (scientists and clinicians), European regulators and industry in which discussion about issues such as use of placebo, guidelines for long-term maintenance are discussed. In addition, since 2009 ECNP organises a summer school of neuropsychopharmacology in Oxford and since 2012 a child and adolescent school of neuropsychopharmacology in Venice.

Finally, ECNP organises seminars, as the one you have been invited to participate, in areas where there are less opportunities for psychiatrists to participate in international meetings. So far, ECNP has organised this meeting in Poland, Estonia, Turkey, Bulgaria, Slovak Republic, Hungary, Czech Republic, Moldova and Romania. Interaction is the keyword at these meetings and they have proved very successful both for the participants and for the faculty.

Please see the ECNP website (www.ecnp.eu) where you can find information about the above initiatives and additional information.

I look forward to a fruitful and inspiring meeting in Greece!

Celso Arango, MD
Chair ECNP Educational Committee
## Program

### Friday 9 November 2012

Arrival of experts and participants  
19:00 Welcome and Dinner

### Saturday 10 November 2012

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
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</thead>
<tbody>
<tr>
<td>09:00-09:15</td>
<td>Introduction to the programme, Celso Arango, Spain</td>
</tr>
<tr>
<td>09:15-10:00</td>
<td>Treatment of acute psychoses, Celso Arango, Spain</td>
</tr>
<tr>
<td>10:00-10:45</td>
<td>Focus on Where We Draw the Line on Issues such as Delusions and Obsessions, Behavioral Addiction and Treatment Resistant OCD, Joseph Zohar, Israel</td>
</tr>
<tr>
<td>10:45-11:15</td>
<td>Coffee break</td>
</tr>
<tr>
<td>11:15-12:00</td>
<td>The functional anatomy of mood disorders revised, Antonio Drago, Italy</td>
</tr>
<tr>
<td>12:00-12:30</td>
<td>How to give a talk, Celso Arango, Spain</td>
</tr>
<tr>
<td>12:30-13:30</td>
<td>Lunch</td>
</tr>
<tr>
<td>13:30-15:00</td>
<td>Groups Round 1</td>
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<tr>
<td></td>
<td>Group A</td>
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<tr>
<td></td>
<td>J. Zohar &amp; G.N. Papadimitriou</td>
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<td></td>
<td>Group B</td>
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<td></td>
<td>C. Arango &amp; D. Dikeos</td>
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<td></td>
<td>Group C</td>
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<tr>
<td></td>
<td>A. Drago, I. Zervas &amp; N. Kokras</td>
</tr>
<tr>
<td>15:00-15:15</td>
<td>Break</td>
</tr>
<tr>
<td>15:15-15:45</td>
<td>How to prepare a scientific paper, Celso Arango, Spain</td>
</tr>
<tr>
<td>16:00-21:00</td>
<td>Cultural Event: Short Boat Trip to Bourtzi and Dinner</td>
</tr>
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</table>

### Sunday 11 November 2012

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
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<tbody>
<tr>
<td>09:00-10:30</td>
<td>Groups Round 2</td>
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<td>Group B</td>
</tr>
<tr>
<td></td>
<td>J. Zohar &amp; G.N. Papadimitriou</td>
</tr>
<tr>
<td></td>
<td>Group C</td>
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<tr>
<td></td>
<td>C. Arango &amp; D. Dikeos</td>
</tr>
<tr>
<td></td>
<td>Group A</td>
</tr>
<tr>
<td></td>
<td>A. Drago, I. Zervas &amp; N. Kokras</td>
</tr>
<tr>
<td>10:30-11:00</td>
<td>Coffee Break</td>
</tr>
<tr>
<td>11:00-12:30</td>
<td>Groups Round 3</td>
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<tr>
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<td>Group C</td>
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<td></td>
<td>J. Zohar &amp; G.N. Papadimitriou</td>
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<td>Group A</td>
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<td>C. Arango &amp; D. Dikeos</td>
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<td></td>
<td>Group B</td>
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<tr>
<td></td>
<td>A. Drago, I. Zervas &amp; N. Kokras</td>
</tr>
<tr>
<td>12:30-13:30</td>
<td>Lunch</td>
</tr>
<tr>
<td>13:30-14:30</td>
<td>Preparation for plenary session</td>
</tr>
<tr>
<td>14:30-15:30</td>
<td>Plenary Session</td>
</tr>
<tr>
<td></td>
<td>14:30 - 14:50 Group A Presentation &amp; Discussion</td>
</tr>
<tr>
<td></td>
<td>14:50 - 15:10 Group B Presentation &amp; Discussion</td>
</tr>
<tr>
<td></td>
<td>15:10 - 15:30 Group C Presentation &amp; Discussion</td>
</tr>
<tr>
<td>15:30-15:45</td>
<td>Time to fill out the evaluation forms and preparation of the awards ceremony</td>
</tr>
<tr>
<td>15:45-16:00</td>
<td>Awards Ceremony and Concluding Remarks</td>
</tr>
</tbody>
</table>
Celso Arango, MD, PhD is a psychiatrist and Associate Professor of Psychiatry at the University of Maryland in Baltimore and the Universidad Complutense in Madrid. He is also Head of the Child and Adolescent Department of Psychiatry at Hospital General Universitario Gregorio Marañón. Dr. Arango is the Scientific Director of the Spanish Psychiatric Research Network with 25 centers and more than 400 researchers. He is also Coordinator of the Child and Adolescent First-Episode Psychosis Study (CAFEPS) funded by the Spanish Ministry of Health (with eight centers in Spain) and the Child and Adolescent Neuropsychiatry Network funded by the European College of Neuropsychopharmacology (ECNP). He has written more than 230 peer-reviewed articles, 6 books, and more than 35 book chapters. Many of his articles and book chapters have focused on the neurobiology of early-onset and first-episode psychoses as well as the safety of psychiatric medications in pediatric patients. In addition, his group has shown how patients with a first psychotic episode experience greater losses of gray matter than expected and a correlation of gray matter loss with antioxidant status. Dr. Arango has participated in more than 59 competitively funded research projects, as Principal Investigator in 43 of them, including projects with international funding (Stanley Foundation, NARSAD, European Commission, etc.) and several clinical drug trials. He is also coordinator of several multicenter projects that assess multiple prognostic factors and treatment in early-onset psychosis, and is currently participating in five EU projects funded by the VII Framework.

Lecture Topic:
Treatment of acute psychoses

The first episode of psychosis is a critical period in the course of each patient’s illness and perhaps the most important opportunity for therapeutic intervention. The first experience of the patients with the psychiatric system should be less traumatic as possible. The treatment provided in the emergency setting should not jeopardize long-term objectives. This also includes that whenever is possible the patients is given the option to choose among different recommended treatments. Randomized controlled trials show no difference between different antipsychotics in terms of efficacy for the short-term acute treatment of psychosis. Main differences between antipsychotics are more markedly in side effects. This is even more important for pediatric patients that seem more vulnerable to some of these side effects. In the acute setting benzodiazepines are sometimes of great help. For the treatment of mania many different therapeutic options have shown to be effective. Second generation antipsychotics are used more frequently nowadays to treat acute mania. Patients usually need lower doses than used with more chronic patients. Recovery is a multidimensional process, improving psychotic symptoms is not the most difficult task for the clinician. Engaging the patient with a good therapeutic alliance, reducing the risk of lack of adherence and provide the proper psychoeducation are more difficult tasks that influence the long-term prognosis.
Joseph Zohar
Department of Psychiatry, Chaim Sheba Medical Center, Tel Hashomer, Israel

Dr. Zohar is director at the division of Psychiatry at Chaim Sheba Medical Center, Israel, and Chief Psychiatric Advisor to the Israeli Ministry of Defence. Dr. Zohar is currently President of the European College of Neuropsychopharmacology (ECNP), Chair of the International College of Obsessive-Compulsive Spectrum Disorders (ICOCS). He is Chair of the Israeli consortium on PTSD, and a board member for the International Master in Affective Neuroscience. Dr. Zohar has been honored with several awards, including the Fogarty International Research Fellowship Award (1984), the A.E. Bennet Award for Clinical Research (1986 and 2002), ECNP – Lilly Neuroscience Award for Clinical Research (1998), and the WFSBP Award for Excellence in Education (2001). Dr. Zohar has authored more than 250 papers, has written or edited 14 books focusing on refractory depression, OCD and post-traumatic stress disorder, and was the founding associate editor of CNS Spectrums and of the World Journal of Biological Psychiatry. Dr. Zohar is considered a world expert on posttraumatic stress disorder, and has recently received funding from the American National Institute of Mental Health (NIMH) to explore the potential of hydrocortisone in the immediate aftermath of trauma, as a preventive measure against the development of PTSD.

Lecture Topic:
Focus on Where We Draw the Line on Issues such as Delusions and Obsessions, Behavioral Addiction and Treatment Resistant Obsessive-Compulsive Disorder

At times, a treatment dilemma arises in severe OCD, deriving from diagnostic ambiguity; many of the very severe ego-syntonic obsessive compulsive patients may be erroneously diagnosed as schizophrenic, while they are actually severe OCD that should be treated with antiobsessive medication and not antipsychotic. The prevalence of OCD amongst schizophrenic patients ranges from 10-25% and has a negative effect on the prognosis for those substantial proportion of schizophrenic patients. Preliminary data implies that for this subset of patient (the schizo-obssessive patients) a combination of antipsychotic and antiobsessive medication might be useful. Another area of potential overlap with OCD is with behavioral addiction. The origin of addiction is from the Latin verb "addicere", which means "to enslave". The association with substance was a later addition to the equation. Actually, the border between substance addiction and "behavioral addition" on one hand, and OCD and "behavioral addiction" on the other hand is not straightforward. When examining substance addiction and behavioral addiction from the phenomenological perspective, the similarity is quite impressive; both behaviors (substance addiction and behavioral addiction) are characterized by developing tolerance, experiencing withdrawal effects and the urge (which is quite irresistible) to perform this behavior despite the negative consequences. Indeed, many patients talk about being "addicted" to the rituals and to their pattern of thinking. Endophenotype represents intermediary constructs between complex disorders and genotypes, and may track more closely to biological constructs, hence presenting improved targets for treatment interventions. Along these lines, compulsivity, like impulsivity, may represent an important endophenotype for impulse control disorders, substance use disorders and OCD. Behavioral addiction as a common denominator of OCD and pathological gambling will be presented, accompanied by the relevant studies. The dimensional approach will be brought forward, and its implication for development of alternative strategies for diagnosis (and treatment) will be discussed.
Antonio Drago
Institute of Psychiatry, University of Bologna, Bologna, Italy

Dr. Drago graduated from the Medical School of Padua, Italy, completed an internship in Psychiatry at the same University and started the residency in 2002. After finishing a Master in Affective Neuroscience at the University in Maastricht, The Netherlands in 2005, he obtained his Ph.D. at the University of Modena, Italy with a thesis on the genetic investigation methods of psychiatric phenotypes. From 2006 he works at the Institute of Psychiatry in Bologna, Italy where he is part of the genetic investigation unit for mood disorders as a research fellow. He attended the ECNP School in pharmacology in 2011 and he actively collaborates with international groups of research in genetics. Stronger collaborations are with the University of Biologia in Barcelona and with the Statistic Unit in Medical Genetics at the University of Cardiff. He is recipient for international travel awards from the ECNP and other international scientific societies. He is editor of Neuropsychopathology and Conference Papers in Biology. During the last years he focused in the functional anatomy of mood disorders, stressing the boundaries between the brain structures and the genetic background that cause the mood imbalances.

Lecture Topic:
The functional neuroanatomy of mood disorders revised

The last 50 years have been witness to a diversity of theories, each one claiming to have the key to the aetiology of mood disorders, based on the narrow perspective of its own discipline (genetic, social, psychological or biochemical). However, in the last decade or so, thanks to technological advances, major leaps have been made in our understanding of the workings of the brain. It has become increasingly clear that both psychosocial and biological factors are highly relevant and far from contradicting each other, they are inextricably linked in the genesis of this multifaceted condition. An overview of the principal ways through which neurological information flows within the brain allows for the identification of critical nods that base the pathophysiology of mood disorders. This perspective can be enriched by the genetic and neuroimaging data that have been gathering in the last years. The limbic system was identified as playing a role in the experience of emotion in the early 1930s and in 1937 James Papez described the ‘system of emotion’, a major pathway of the limbic system, connecting a group of brain structures surrounding the brainstem (the cingulate gyrus, hippocampus, the hypothalamus and the anterior thalamic nuclei). The emergence of neuroimaging techniques, magnetic resonance imaging (MRI), positron emission tomography (PET) and functional fMRI, established the importance of the ‘neurocircuit of emotion’ which has been expanded to include other important brain areas and in particular the prefrontal cortex (PFC). These brain sites and their connections, which have been widely studied, are responsible for maintaining emotional stability and their malfunction is considered central to the pathophysiology of mood disorders. In particular, the way the neurological information is treated in the thalamo-cortical and spino-thalamic connections may be related to some key issues in the mood disorders’ symptomatology. Consistently, the associative areas of the basal ganglia gained relevance as relevant structures whose dysregulation may determine some of the most relevant clinical features of bipolar and unipolar disorders.
Local Organizing Committee

National and Kapodistrian University of Athens, Medical School,
1st Department of Psychiatry, Eginitio Hospital

Professor George N. Papadimitriou

Prof. George N. Papadimitriou, M.D. is Professor and Chairman of the First Department of Psychiatry at Athens University Medical School in Eginition Hospital, Athens, Greece. Prof. Papadimitriou held clinical and research posts in Greece, Belgium and the USA. His main research interests are genetics, psychopathology, psychopharmacology, psychoendocrinology and sleep. He has authored more than 200 papers in international and 100 in Greek peer-reviewed scientific journals and books. He has been involved as principal and co-investigator on a number of international multicenter research projects, including EU and ESF projects. He has served on the executive committees and advisory boards of several national and international societies, including the European College of Neuropsychopharmacology (ECNP) and the World Federation of Societies of Biological Psychiatry (WFSBP). He holds the office of Vice-President of the Hellenic Association of Biological Psychiatry. He is editorial advisor in the Greek editions of Harvard Review of Psychiatry and American Journal of Psychiatry. He serves on the editorial boards of several other international peer-reviewed journals, including Neuropsychobiology, European Psychiatry, Bipolar Disorders, World Journal of Biological Psychiatry, etc. Prof. Papadimitriou is an independent reviewer of the European Accreditation Committee (EACIC) in Central Nervous System and was the Chairman of the XVIIIth World Congress on Psychiatric Genetics, held on October 2010 in Athens.

Assoc. Professor Dimitris Dikeos

Dimitris G. Dikeos is an Associate Professor of Psychiatry at the 1st Department of Psychiatry of Athens University Medical School, Athens, Greece and a Visiting Research Associate at the Division of Psychological Medicine, Institute of Psychiatry, King’s College London, London, UK. He is the Director of the Sleep Research Unit of Athens University at Eginition Hospital. His research activities have focused on psychiatric genetics, sleep research, psychopharmacology and clinical studies in psychiatry. He has participated in various Multicentre Research Programmes in Europe and the U.S.A. such as: European Science Foundation, European Collaborative Studies of Affective Disorders, Johns Hopkins Genetic Epidemiology Schizophrenia Program, Meta-analysis of Sleep Laboratory Studies on Tolerance and Rebound Insomnia with Rapidly Eliminated Hypnotics, Maudsley Family Study, European Collaborative study by the Group for the Study of Resistant Depression, International Multicentre Study “FACTOR”, The Schizophrenia Psychiatric Genome-Wide Association Study, etc. He is or has been member of various scientific and professional Societies and Boards, as well as member of the Executive Committees of the Hellenic Sleep Research Society (President), the International Neuropsychiatric Association (President-elect), the Athens Medical Society, the Hellenic Society for the Advancement of Psychiatry and Related Sciences. He has also served as member of the Editorial Board of the “Archives of Hellenic Medicine” and is a reviewer in many international Journals. Dr. Dikeos is the author or co-author of more than 100 full publications, out of which 50 articles in SCI Journals (h-index=17), among which: American Journal of Medical Genetics, British Journal of Psychiatry, Current Opinion in Psychiatry, International Clinical Psychopharmacology, Journal of Psychosomatic Research, Molecular Psychiatry, Nature Genetics, Psychiatric Genetics, and Science.
Assoc. Professor Iannis Zervas

Iannis Zervas MD is Associate Professor of Psychiatry at the University of Athens Medical School and Director of the Women’s Mental Health Clinic at Eginition University. He has received his MD and his doctorate degree from the University of Athens and was trained in Psychiatry and Consultation Liaison Psychiatry at Stony Brook University Hospital on Long Island, NY. While in the past his research focused mostly on ECT, over the past decade he has been involved in psychiatry of the female reproductive cycle and his research has mostly focused on mood and anxiety of the perinatal and the perimenopausal phases. He has published over 100 articles and book chapters and has co-edited a Greek textbook on Consultation Liaison Psychiatry. Over the years he has translated, co-translated and edited translations for over fifteen books on various psychiatric topics. His most recent research projects have focused on antepartum anxiety and depression, stress effects on women with multiple sclerosis, and the relation between insomnia and depression in perimenopause.

Dr. Nikolaos Kokras

Dr Nikolaos Kokras is a clinical & research associate at the Departments of Psychiatry and Pharmacology at the University of Athens Medical School. He received his MD from the Aristotle University of Thessaloniki and was trained in Psychiatry at the South London & Maudsley NHS and the 1st Department of Psychiatry, Eginition hospital. He holds an MSc in Affective Neuroscience from the University of Maastricht and a PhD in Experimental Psychopharmacology from the University Of Athens. He has attended several psychopharmacology schools, including the ECNP School of Neuropsychopharmacology and the BAP series of seminars in preclinical psychopharmacology. He has published several articles in the field of gender/sex differences in psychopharmacology.
Treatment of Acute Psychoses
Celso Arango
Hospital General Universitario Gregorio Marañón,
Madrid, Spain
carango@hggm.es
Greece, November 2012

Index

- Review of first episode studies
- Treatment in the acute setting
- Treatment of acute mania
- Special Population: children and adolescents
- Discussion

The first episode of psychosis is a **critical period** in the course of each patient’s illness and perhaps the most important opportunity for therapeutic intervention
Placebo-Controlled First-Episode Maintenance Trials

<table>
<thead>
<tr>
<th>Study</th>
<th>Relapse Rate (%)</th>
<th>A value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kane et al, 1982</td>
<td>41 (7/17)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Crow et al, 1988</td>
<td>62 (41/66)</td>
<td>0.002*</td>
</tr>
<tr>
<td>McCreadie, et al (1989)</td>
<td>57 (4/7)</td>
<td>NS</td>
</tr>
<tr>
<td>Hogarty and Ulrich, 1998</td>
<td>64</td>
<td>N/A</td>
</tr>
</tbody>
</table>

Note: The period between onset of index episode and hospital admission is taken into account.

Olanzapine vs Haloperidol

- Time to Study Discontinuation for Any Reason of Subjects with First Episode Psychosis in the 12-Week Acute Treatment Phase of a Long-term Comparison of Olanzapine and Haloperidol:

- No significant difference between treatment groups in time to discontinuation (p=0.06, log rank test).

Primary Outcome: All-Cause Treatment Discontinuation

- For each category above, the comparison of quetiapine vs olanzapine and quetiapine vs risperidone met the a priori level of noninferiority (20%) at P=0.05.
European first episode (EUFEST) Study

Time to treatment discontinuation for any cause

First-Episode Patients: Lower Medication Doses Than Multi-Episode Patients

<table>
<thead>
<tr>
<th>Study</th>
<th>Mean Modal Daily Dose (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lieberman et al 2005</td>
<td>Haloperidol: 4.4, Olanzapine: 9.1</td>
</tr>
<tr>
<td>Schooler et al 2005</td>
<td>Haloperidol: 2.9, Risperidone: 3.3</td>
</tr>
<tr>
<td>Robinson et al 2006</td>
<td>Olanzapine: 11.8, Risperidone: 3.9</td>
</tr>
</tbody>
</table>

Treatment goals in the emergency setting

- Reducing acute symptoms
- Minimising risk of harm
- Calming agitation
- Improving role functioning

Achieving these goals must not be at the expense of long-term treatment objectives

Arango & Bobes 2004
Patient requirements and preferences in the acute setting

- Receive a rapid and accurate diagnosis
- Be offered a choice of treatment
- Benefit from a good therapeutic alliance
- Receive verbal rather than physical interventions
- Receive oral medication

Recovery is a multidimensional process

- Quality of life
- Control of symptoms
- Social and family interactions
- Relapse prevention
- Daily functioning
- Improved cognition
- Return to work
- Overcoming stigma

Treatment Options for Acute Mania

- Classical antipsychotics
- Atypical antipsychotics
- Lithium
- Valproate
- Carbamazepine
- Combinations
- Benzodiazepines
- ECT
Change in weight over time by treatment group olanzapine/quetiapine

Arango et al, 2009

Metabolic side effects in young people treated with second-generation antipsychotics

<table>
<thead>
<tr>
<th></th>
<th>At risk adverse</th>
<th>Baseline</th>
<th>6 month</th>
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<tbody>
<tr>
<td>RIS</td>
<td>22.7%</td>
<td>36.4%</td>
<td></td>
</tr>
<tr>
<td>OLZ</td>
<td>15.0%</td>
<td>60.0%*</td>
<td></td>
</tr>
<tr>
<td>QTP</td>
<td>12.5%</td>
<td>20.8%</td>
<td></td>
</tr>
</tbody>
</table>

* p=0.05

Significant weight gain
Defined as > 0.1 increase in body mass index (BMI) z-score (adjusted for age and gender) at 6 months
RIS: 50%
OLZ: 75%
QTP: 29%
*p=0.01

Fraquas et al, J Clin Psychiatry 2008

SATIETY study design

Treatment with all antipsychotics was associated with changes in metabolic parameters at 12 weeks

All values refer to mean change from baseline (p value)

<table>
<thead>
<tr>
<th></th>
<th>Aripiprazole (n=41)</th>
<th>Olanzapine (n=46)</th>
<th>Quetiapine (n=30)</th>
<th>Risperidone (n=15)</th>
<th>Untreated (n=15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (kg)</td>
<td>4.44</td>
<td>8.54</td>
<td>6.96</td>
<td>5.34</td>
<td>0.19</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>2.40</td>
<td>4.12</td>
<td>2.52</td>
<td>2.45</td>
<td>0.35</td>
</tr>
<tr>
<td>Waist (cm)</td>
<td>5.40</td>
<td>8.35</td>
<td>5.27</td>
<td>5.10</td>
<td>0.70</td>
</tr>
<tr>
<td>Glucose (mg/dl)</td>
<td>0.54</td>
<td>3.14</td>
<td>2.64</td>
<td>1.14</td>
<td>0.69</td>
</tr>
</tbody>
</table>

Comer CJ, et al JAMA 2009;302;1765-1773
Treating first-episode patients

The most difficult task is not getting them to respond to treatment, but getting them to continue to take medication.

The course of an acute episode of psychoses can be directed towards successful treatment outcomes by...

- Prompt intervention with agents that are well tolerated
- Initiating a programme of long-term therapy (including social services, psychoeducation, accessibility to health facilities and intervention with family is possible) to maintain and build upon the initial success of treatment
- Consider polypharmacy in the acute treatment of bipolar disorders
- Ensuring a positive experience in the acute setting and establishing an interactive therapeutic alliance
Focus on Where We Draw the Line on Issues such as Delusions and Obsessions, Behavioral Addiction and Treatment Resistant OCD

Joseph Zohar
ECNP President

Department of Psychiatry
Chaim Sheba Medical Center
Tel Aviv University
Israel

OCD and Schizophrenia

**DSM-IV**

Some individuals manifest symptoms of both obsessive-compulsive disorder and schizophrenia, warranting both diagnoses.

**DSM-IV**

If another Axis I disorder is present, the content of the obsessions or compulsions is not restricted to it; eg, paranoid delusions in schizophrenic-paranoid type.
Is There Room for a New Diagnostic Subtype—The Schizo-obsessive Subtype?

Zohar, J (1997) Editorial
CNS Spectrums; 2(3): 49-50

“Schizo – obsessive“ subtype

Actually this term has been used, by many clinicians, around the globe.

Is there enough evidence to support “schizo-obsessive” as a schizophrenia subtype?

From a dimensional point of view

Addiction is:

♦ Craving state prior to behavioral engagement
♦ Impaired control over behavioral engagement
♦ Continued behavioral engagement despite adverse consequences

OCD & Addiction
<table>
<thead>
<tr>
<th></th>
<th>SUD</th>
<th>PG</th>
<th>OCD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Interference with major area of life function</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Tolerance</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Withdrawal</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Repeated unsuccessful attempts to cut back or quit</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

**Repetitive behaviours**

**Symptom**

**Syndrome**

**Behavioural dimension**

**OCD**

**Addiction**

Is it possible that OCD and Addiction are actually different presentations of the same underlying disorder?
 Syndromes appearing clinically distinct may result from the same etiology.

The Future of Psychiatric Diagnosis
Phenotype vs. endophenotype

Ten Species in One. DNA barcoding reveals cryptic species in the neotropical skipper butterfly. Hebert et al, PNAS, 101: 14813-17, 2004

Can an Endophenotypical Approach Help?
RDoC classification
Data from genetics, brain circuits and clinical neuroscience will yield bio-signatures

What Are Endophenotypes?

- Endophenotypes in psychiatry (Gottesman and Gould, 2003)
  - Define mediating factors between genes and disorders
  - More genes involved, greater complexities of phenotypes and genetic analyses

Possible tools to explore endophenotype

- Family aggregation
- Pharmacological dissection
- Pharmacological challenge
- Cognitive challenge
- Brain structure
- Brain Circuitry
- Epigenetic tools

Brief Report

Impaired Cognitive Flexibility and Motor Inhibition in Unaffected First-Degree Relatives of Patients With Obsessive-Compulsive Disorder

Samuel R. Chamberlain, M.A.
Naomi A. Fineberg, M.B.B.S.
Lara A. Menzies, B.A.
Andrew D. Blackwell, Ph.D.
Trevor W. Robbins, Ph.D.
Barbara J. Sahakian, Ph.D.

Objective: Obsessive-compulsive disorder (OCD) is highly heritable; attempts to delineate genetic contributors have met with limited success. There is an ongoing search for intermediate cognitive brain markers (endophenotypes) that may help clarify genetic contributions. The aim was to assess inhibition control processes in unaffected first-degree relatives of OCD patients for the first time with objective tests.

Method: The radial-incremental radial-horizontal shift, stop-signal, and Cambridge Gamble tasks were administered to 20 unaffected first-degree relatives, 20 OCD patient probands with washing/skipping symptoms, and 20 healthy matched comparison subjects without a family history of OCD.

Results: Unaffected first-degree relatives and OCD patient probands showed cognitive inflexibility (radial-horizontal set shifting) and motor impulsivity (stop-signal reaction time). Depression making Cambridge Gamble task was intact.

Conclusions: Deficits in cognitive flexibility and motor inhibition may represent cognitive endophenotypes for OCD. Such measures will play a key role in understanding genetically influenced endophenotype associations for OCD and related spectrum conditions.

Brain Circuitry

OCD has:

**Specific** brain circuitry:

Prefrontal cortex - temporal cortex - thalamus - basal ganglia

**Orbitofrontal Dysfunction in Patients with Obsessive-Compulsive Disorder and Their Unaffected Relatives**

Researchers identified abnormally reduced activation of several cortical regions, including the orbitofrontal cortex, during reversal learning in OCD patients and their clinically unaffected close relatives, supporting the existence of an underlying **endophenotype** for this disorder.
The functional anatomy of mood disorders revised

Antonio Drago
University of Bologna
Institute of Psychiatry
antonio.drago@unibo.it

Systems

- Thalamocortical
- Basal Nuclei
- Lymbic system

evidence

Reduced Natural Oscillatory Frequency of Frontal Thalamocortical Circuits in Schizophrenia

Table 1. Description of Participants

<table>
<thead>
<tr>
<th>Clinical Variable</th>
<th>Healthy Control Subjects (n=20)</th>
<th>Patients With Schizophrenia (n=20)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), y</td>
<td>31.7 (7.5)</td>
<td>32.8 (6.5)</td>
<td>.00</td>
</tr>
<tr>
<td>Malefemale, No.</td>
<td>16/4</td>
<td>13/7</td>
<td>.31</td>
</tr>
<tr>
<td>Positive and Negative</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Syndrome Scale score, mean (SD)</td>
<td>18.0 (6.3)</td>
<td>21.5 (5.6)</td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>General</td>
<td>38.7 (10.8)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
**Priority Communication**

Proton Spectroscopic Imaging of the Thalamus in Treatment-Naive Pediatric Obsessive–Compulsive Disorder*

Kate Dimond Fitzgerald, Gregory J. Moore, Lori Anne Paulson, Carol M. Stewart, and David R. Rosenberg

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>OCD patients (n = 11)</th>
<th>Comparison subjects (n = 11)</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>age (years)</td>
<td>11.1 ± 2.9 (9.0–13.5)</td>
<td>11.0 ± 3.0 (9.0–13.5)</td>
<td>0.09</td>
<td>.927</td>
</tr>
<tr>
<td>Number of subjects</td>
<td>4</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>6</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>43.4 ± 11.2 (25.0–64.0)</td>
<td>41.2 ± 11.2 (20.9–61.0)</td>
<td>.431</td>
<td>.64</td>
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<tr>
<td>Weight (kg)</td>
<td>46.1 ± 11.3 (25.0–64.0)</td>
<td>40.0 ± 11.3 (20.9–61.0)</td>
<td>.94</td>
<td>.32</td>
</tr>
<tr>
<td>Age at onset of first clinical presentation (years)</td>
<td>8.3 ± 2.3 (6.0–14.75)</td>
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<td>--</td>
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</tr>
<tr>
<td>Duration of Illness (years)</td>
<td>2.6 ± 2.0 (0.0–6.0)</td>
<td>--</td>
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<td></td>
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<tr>
<td>Total SSRS</td>
<td>24 ± 9 (3–57)</td>
<td>24 ± 9 (3–4)</td>
<td>1.0</td>
<td>.314</td>
</tr>
</tbody>
</table>

*Patient was not on psychiatric drug use. Data are provided as mean ± SD unless otherwise indicated. Total SSRS: 1 (highest to 5 lowest) measure of parent/observer report of psychiatric and obsessive-compulsive functioning, indexed (total).  

**Note:** This patient was scoring neurocognitive test scores.
ORIGINAL INVESTIGATION

Multimodal psychodynamic psychotherapy induces normalization of reward related activity in somatoform disorder

MORITZ DE GRECK1, LISA SCHEIDT1, ANNETTE F. BÖLTER1, JÖRG FROMMER2, CORNELIA ULBRICH2, EVA STOCKMANN1, BORIS ENZ3, CLAUS TEMPELMANN1, THELO HOFFMANN1 & GEORG NORTHOFF1

Methods

We investigated 20 patients (gender: 12 females, eight males; handedness: 19 right-handed, one left-handed; mean age = 42.5, SD = 18.0). All patients suffered from a somatoform disorder as ascertained by the Structured Clinical Interview for DSM-IV (German version: SKID (Wittchen et al. 1997)).
The Dynamics of Change in Striatal Activity Following Updating Training

Simone Köhn,1,2 Florian Schmiedek,1,2 Hannes Noack,3 Elisabeth Wenger,7 Nils C. Badzak8,3 Ulman Lindenberger,8 and Martin Lövén1,2

A healthy sample consisting of 40 adults (Mage = 33.3, SDage = 1.3; range: 20–30) was recruited through flyers and word-of-mouth recommendations circulated in Berlin. Participants were first matched on sex and global cognitive performance (Digit-Forward Substitution Test, Hachinski, 1987) and then randomly assigned to either one of two groups: response inhibition, N = 20 (15 males; 5 females; Mage = 33.3, SDage = 1.3; range: 20–29), or action control, N = 20 (15 males; 5 females; Mage = 33.3, SDage = 1.3; range: 20–30). Our previous experience with

Increases from scan 1 (session # 1) to scan 2 (session # 7)
Decreases from scan 2 (session # 7) to scan 3 (session # 57)
Task related activation (all loads > baseline all time points)

Brain blood flow changes in depressed patients treated with interpersonal psychotherapy or venlafaxine hydrochloride: preliminary findings.

Martin SD, Martin E, Bai S, Richardson MA, Royal B

Affinity Research Unit, Cherry Knowle Hospital, Ryhope, Sunderland, SR2 0NB, England, UK.

METHODS: Twenty-eight men and women aged 30 to 53 years with a DSM-IV major depressive episode, a 17-item Hamilton Rating Scale for Depression (HAM-D) rating of 18 or higher, and antidepressant-naïve for at least 6 months were studied. After baseline (Washboard-methylphenylpyridone-aziridine scan, 1T-Magnetic resonance imaging, and psychometric ratings, patients were assigned to different treatments. Thirteen patients had 1-hour weekly sessions of IPT from the same supervised therapist (E.M.). Fifteen patients took 37.5 mg twice-daily of venlafaxine hydrochloride. Single-photon-emission computed tomography scans and ratings were repeated at 6 weeks.
RESULTS: Both treatment groups improved substantially, more so with venlafaxine (mean [SD])
HAM-D scores at pretreatment: IPT, 27.7 [7.7], and venlafaxine, 22.4 [3.3]; and posttreatment:
IPT, 16.2 [7.1], and venlafaxine, 10.9 [8.6]. No patients had structural brain abnormalities. On
analysis with statistical parametric mapping 98, the venlafaxine group showed right posterior
temporal and right basal ganglia activation (P = .01), while the IPT group had limbic right posterior
cingulate and right basal ganglia activation (P = .01).

**Hold Your Horses: Impulsivity, Deep Brain Stimulation, and Medication in Parkinsonism**

Michael J. Frank,1* Johan Sanz-Ortiz,1* Ahmed A. Mostofa,1 Scott J. Sherman1

*Hold Your Horses: Impulsivity, Deep Brain Stimulation, and Medication in Parkinsonism
Michael J. Frank et al.

Science 318, 1309 (2007);
DOI: 10.1126/science.1146157

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**Conflict-Modulated RT's**

**A** Conflict-Modulated RT's

**Correct Trials**

**B** Conflict-Modulated RT's

**Error Trials**

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Modulation of Subthalamic Alpha Activity to Emotional Stimuli Correlates with Depressive Symptoms in Parkinson’s Disease

Julius Huebli, MD, Thomas Schonecker, MD, Sandy Segeit, MD, Christof Brücke, MD, Gerrit-Heike Schreider, MD, Andreas Kuziora, MO, Katarina Vortov, MO, and Andrea A. Kuhn, MO

Department of Neurology, Charité – University Medicine Berlin, Germany
Department of Psychology, City University, London, United Kingdom

Twelve consecutive PD patients (age 60 ± 7 (MEAN ± SD); 4 women) undergoing stereotactic neurosurgery for implantation of STN DBS electrodes at the Department of Neurosurgery, Charité were

FIG. 1. Mean decrease in alpha power during 1 to 2 seconds after stimulus onset (recorded from n = 12 patients/24 contact pairs). Values are expressed as percentage change in relation to baseline activity (2 seconds before stimulus onset). A one sample t test confirmed a significant decrease in alpha power for pleasant (PLS, \( *P < 0.001 \)) and unpleasant stimuli (UNPLS, \( *P < 0.01 \)) but not for neutral stimuli (NTR, n.a.) compared with baseline activity. The mean decrease in alpha power for pleasant stimuli was also significantly greater than for neutral stimuli (\( *P < 0.05 \)).

Changes of brain activation pre- post short-term psychodynamic inpatient psychotherapy: An fMRI study of panic disorder patients

Manfred E. Brunet^, Rudolf Stark^, Heng Pan^, David Silbersweig^, Sylvia Dietrich^*^

Table 4

<table>
<thead>
<tr>
<th></th>
<th>Positive</th>
<th>Neutral</th>
<th>Negative</th>
</tr>
</thead>
<tbody>
<tr>
<td>PD (n = 9)</td>
<td>58.31 (19.82)</td>
<td>37.96 (17.27)</td>
<td>75.00 (22.02)</td>
</tr>
<tr>
<td>C (n = 18)</td>
<td>58.33 (21.35)</td>
<td>45.00 (26.92)</td>
<td>65.51 (28.06)</td>
</tr>
</tbody>
</table>

PD: panic disorder patients; C: controls; S.D.: standard deviation.
How to prepare a scientific presentation
Celso Arango

Before you start
• What does the audience already know about your topic?
• What are their interests?
• Why are you giving presentation?

Before you start
• What is your desired outcome?
• How much time do you have?
• What are key points?
Common Causes of Ineffective Presentations

- Failure to prepare the talk
- Confusing structure/not giving take home messages
- Gaps in logic
- Poorly designed slides
- Poor delivery

Organizing a Presentation

i. Outline
ii. Problem and background
iii. Design and methods
iv. Major findings
v. Conclusion and recommendations

Making slides

- Main points only
- One idea per slide
- Short words, few words (5 per line)
- Strong statements: active voice
The start
• Let audience know what they are going to hear
• Let them know how the presentation will be organized

Start broad, get specific, and end broad
The middle is your original contribution
Start with the biggest questions and get progressively more specific
Focus now on conclusions

Introduction
• Context
• Study question
• Relevant knowledge on issue
Major findings

- Text and or table/graph
- One slide for each
- Message should be unambiguous

Audience attention curve

Conclusion and Recommendations

- Key points
- Implications
- One slide for each message
Formal aspects

- Avoid using all capital letters because it's really hard to read!
- Dark letters against a light background (or the opposite) work
- Avoid some colour combinations (red-green)

Formal aspects

- Choose style that supports the tone
- Apply the same style to each slide
- Don't Say It, Show It

Be consistent!

Formal aspects

- Every slide should have a heading.
- Lists should contain no more than 3-4 items
- Limit text blocks to no more than two lines each.
- Be careful with the pointer!
ECNP Seminar: 9-11 November 2012 Nafplio, Greece

**Formal aspects**

Type size should be 20 points or larger:
- 18 point
- 20 point
- 24 point
- 28 point
- 36 point

*References can be in 14 point font*

---

**And do not forget to.......**

Relax
Listen to what you are saying
Pace and time yourself

---

**And do not forget to.......**

Face the audience
Never underestimate your audience!
With time you will enjoy.....
How to prepare a scientific presentation

• “Tell me and I will forget, show me and I will remember, involve and I will understand”
How to prepare a manuscript

Celso Arango

Categories

- Original research (focus of this talk)
- Reviews (invited vs. not invited)
- Case reports/series
- Letter to the editor

The Journal

- Does the article fit the aims and scope of the Journal?
  - Choose before writing
  - General vs. subspecialty journal
  - Read the table of contents of potential journals
  - Examine several articles in potential journals
  - Which journals will you cite in your article?
Sequence

• The syndrome of the blank screen
• Figures, tracings, tables
• Methods and Results
• Discussion and Introduction
• Abstract and Title

Tables and Figures

• Do before writing
• Exceed 1 sheet: redraw
• If small: move data to text
• Should be able to stand alone

Methods

• Draft can be made while doing the study
• Enough information for an experienced investigator to repeat your work
• Avoid tiresome detail
• Tables preferred to long list of numbers or statistics
**Methods**

- Refer to data (Fig. X, Table Y)
- Do not repeat numbers in Tables
- Include ethics information (with Ethics Committee approval and i.e.)
- Include complete statistics section

**Discussion**

- First paragraph
  - State major findings

- Last paragraph
  - "In summary..." (2-3 sentences)
  - "In conclusion..." (biggest message, return to Intro, avoid speculation, avoid "need more work")

- Middle paragraphs
  - Base each on a major result

- Always focus on your results

- Explain what is new without exaggerating

- Never discuss prior work without reference to your work (but do not forget appropriate identification of prior research)
Discussion

• Refer Tables and Figures
• Do not repeat results
• Include limitations section

Introduction

• Keep it short. In most cases 3 graphs make it.
  • 1. Why the study is interesting (broad)
  • 2. Why did we do it? (specific)
  • 3. Hypothesis

Abstract

• Is your visiting card
• In most cases make the editor to send the ms to reviewers or reject it.
• Some numbers, but not in excess
• Determines if paper will be read
• Is distributed freely in databases
• Avoid acronyms
Title
- Max information in least words
- The title is an invitation to read the paper
- Use catchy titles
- State results

Keywords
- Make them easy for indexing and searching! (if you want to be cited)

References
- Cite the Journal you are submitting the paper to
- Reviewers may be selected from your references
- Use editing programs
- Relevant and recent
The context

- Need stretch of several hours
- Avoid distractions: phone, e-mail
- Ideas come while writing

First draft

- Write as quickly as possible
- As if thinking out loud
- Get everything down
- Ignore spelling, grammar, style
- Correct and rewrite only when the whole text is on paper
- Do not split the manuscript among the co-authors

Introduction

- Context
- Study question
- Relevant knowledge on issue
Major findings
- Text and or table/graph
- One slide for each
- Message should be unambiguous

Formal aspects
- Avoid ambiguity
- Concise: Least words, short words, one word vs many
- Strengthen transition between sentences

Formal aspects
- Check narrative flow: tell a story that the reader wants to read from start to end
- Smooth transitions
- Writing improves in proportion to deletion of unnecessary words
- Keep sentences short
Formal aspects

• After the second draft send ms to your coauthors
• After the suggestions have been incorporated leave it for some time a re-read

Formal aspects

• If you do not have time to check the spelling you may have not had time to check the quality of your experiments......

Formal aspects

• Prepare article, figures and table according to the journal’s ‘Guide for Authors’
• Adherence to the style of the journal is crucial
• Check references
• Check and double check your work
Authorship

- Decided as early as possible
- The journal has instructions on who should/should not be an author
- Basically all authors should have done a major contribution to the study

Authorship

- Approval of final version must be obtained from all coauthors before submission
- The first author is primarily responsible for collecting and analyzing data, and writing

Authorship

- The manuscript is not under consideration elsewhere and will not be submitted elsewhere until a final decision has been made by the journal
- All funding sources must be acknowledged
- All conflicts of interest should be reported
Peer Review

- Authors write
- Reviewers comment
- Editors decide
- Readers read (only what they like)

Peer Review

- Peer review helps to determine the significance, contribution to what is already known and originality of research
- Most journals reject some paper prior to peer review (on basis of Editor's own evaluation)
- Usually 2-3 reviews sought (per manuscript)

Possible Decisions

- Reject (up to 90-95% in good journals, do not give up!)
- Major revisions required (it will be reviewed again, may be rejected)
- Minor revisions needed (usually accepted)
- Accepted (congratulations! Enjoy and celebrate!)
Response to the editor

- Reviewers are (almost) always right. Editor is always right.
- Response to all the comments in a nice and polite way
- Thank the reviewers for their contribution

When the study is negative

- If your result is not as expected, you should understand the reason. It may be something really new. (Must find out why it did “not work” in the expected way!)

“Scientists are rated by what they finish, not by what they attempt”
Surely you were aware when you accepted the position, Professor, that it was publish or perish!
### List of Participants

<table>
<thead>
<tr>
<th>Antrian, Virginia</th>
<th>Lyrakos, Dimitrios</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anyfanti, Helen</td>
<td>Mavridis, Thodoris</td>
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<tr>
<td>Balaris, Dimitris</td>
<td>Mousios, Dimitrios</td>
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<td>Chatzimanolis, Pythagoras</td>
<td>Nika, Stella</td>
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<td>Christopoulos, Athanasios</td>
<td>Novais, Ashley</td>
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<td>Dalla, Christina</td>
<td>Oikonomou, Elina</td>
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<td>Dasoukis, Ioannis</td>
<td>Papakonstantinou, Anastasios (DNA)</td>
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<td>Dimitraka, Maria (DNA)</td>
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<tr>
<td>Liapis, Christos</td>
<td>Ziakas, Dimitris (DNA)</td>
</tr>
</tbody>
</table>

### Abstracts

**Antrian, Virginia**  
**Agrarian crisis and social pathology - the suicides of farmers:** The purpose of this study, conducted by the Research Centre of the Greek Society of the Academy of Athens, is the understanding and interpretation of the complexity of the causes (biological, psychological and social) of the suicide of the farmers. Quantitative data are obtained from the Police and forensic services and the qualitative study involves interviews with families and professionals. At the present stage, the pilot study was held in Aitolokarnania for the period 1994-2008, the period which follows the great rural crisis and reaches the limits of today's great financial crisis.

**Anyfanti, Helen**  
**Pregabaline in chronic benzodiazepine abuse: cognitive impact:** Pregabalin was used as monotherapy in discontinuation of chronic benzodiazepine abuse and dependence. Patients were assessed with clinical and neuropsychological measures, at base-line, after benzodiazepine discontinuation and at a 2-months follow-up. A matched control group was assessed at baseline. After benzodiazepine discontinuation, patients exhibited significant improvement in clinical measures as well as visuospatial memory and learning. However, there were no changes in cognitive flexibility. Several issues remain open for future research: Do benzodiazepine - addicted patients have “trait” executive deficits? Do those traits persist after benzodiazepine discontinuation? Are those deficits affected by pregabalin? Should such deficits be addressed pharmacologically in benzodiazepine discontinuation?

**Balaris, Dimitris**  
**Long acting antipsychotics in schizophrenia and bipolar disorder treatment:** In this presentation, data on long acting antipsychotics with regards to treatment of schizophrenia and bipolar disorder will be presented. Patients with both disorders commonly present low insight and poor compliance. Long acting injectable antipsychotics are a safe and effective alternative treatment option. Advantages and disadvantages of long acting treatment in schizophrenia and bipolar disorder in comparison with oral treatment options will be discussed as well as the design of a future survey in planning aiming to assess the use of long acting antipsychotics in Greek patients.
Chatzimanolis, Pythagoras

**Therapeutic Management of Depression in Elderly:** Depression in elderly persons is widespread, often undiagnosed, and usually untreated. The current system of care is fragmented and inadequate, and staff at residential and other facilities is often ill-equipped to recognize and treat aged patients with depression. Because there is no reliable diagnostic test, a careful clinical evaluation is essential. Depressive illness in later life should be treated with antidepressants that are appropriate for use in geriatric patients. A comprehensive, multidisciplinary approach, including consideration of electroconvulsive treatment in some cases, is important. The overall long-term prognosis for elderly depressed patients is good.

Christopoulos, Athanasios

**Treatment of bipolar disorder in Greece:** this presentation will be about the bipolar disorder and its treatment in Greece. Pharmacological interventions regarding the bipolar disorder are the focus of particular research and numerous international guidelines exist regarding the management of this disorder. I would like to refer to the variability of treatment with mood stabilisers, antipsychotics and antidepressants, the combination of them which depends on the phase of the disorder and discuss how this subject could be better investigated in Greek patients.

Dalla, Christina

**Sex Differences in Animal Models of Depression:** Women are more prone to depression than men and may have a differential response to antidepressants. However, antidepressant tests have been mainly validated on male animals. In our preclinical studies, we apply models of depression and tests of antidepressant activity to male and female rats. We have shown that female rats exhibit enhanced “depressive-like” symptomatology and we further present data uncovering the sex-dependent antidepressant response and the role of estrogens in the behavioral and neurochemical profile of female and male rats during models of depression. These studies contribute to gender-based therapies for affective disorders

Dasoukis, Ioannis

**Benzodiazepines & Alzheimer’s Disease:** Observations on elderly patients that are under medication with benzodiazepines or misuse benzodiazepines may indicate a protective effect on Alzheimer's disease. There are indications of lower prevalence of dementia on this population despite the cognitive impairment that benzodiazepines cause to the elderly patients. This could be a plan for a future epidemiological study to investigate if there is difference in Alzheimer's incidence between benzodiazepines users and non-users in the Greek population.

Dimitraka, Maria

**Using antipsychotic agents and cognitive enhancers in older patients with serious mental illness:** There is evidence of significant cognitive and functional deficits commonly seen in elderly patients with schizophrenia which amplify comorbidity with dementia. The purpose of the study is to assess the pharmaceutical approaches in long term inpatients with serious mental illness. Participants were aged 55 years or more from two long term psychogeriatric units of the psychiatric hospital of Attica. One-hundred and three Greek long term inpatients were recruited with a mean age of 71 years old. Schizophrenia was present in 60.2% of them whereas 7.8% had a mood disorder and 32% a mental disorder due to general medical condition. Long term mental disorder along with long term hospitalization leads to severe deterioration of both cognitive function and daily skills abilities.

Dimitrakopoulos Stefanos

**Aripiprazole treatment of persistent bruxism:** A 63-years-old woman with a 40-years long history of refractory obsessive-compulsive disorder (OCD) was receiving fluoxetine 40 mg/day. Fluoxetine was replaced by escitalopram up to 60 mg/day, and, within 6 weeks, patient’s symptoms improved moderately. However, the patient began to complain of involuntary jaw movements of grinding and clenching her teeth all day long. Escitalopram was decreased to 40 mg/day for the next 4 weeks without improvement. Fluoxetine up to 60 mg/day substituted escitalopram for another 4 weeks, with amelioration of her OCD symptoms. However, her bruxism persisted unabated. Aripiprazole 10 mg/day was then added and, within 1 week, her bruxism subsided.
Dimitriadis, Giorgos
**Biological Basis of Love and Schizophrenia:** From ancient times, many have connected the concept of love with the concept of madness. It is known that the first signs of schizophrenia appear at late adolescence or early adulthood. At that very time, when we usually fall in love easily, frequently and with great intensity the psychopathological features of psychosis also make their first appearance. Is there a connection between the biological basis of love and the biological basis of schizophrenia?

Giannopoulos, Panagiotis
**Anxiety and Medication Phobia:** A 44 years old woman presented to psychiatric services with a chief complaint of “extreme anxiety”. She had a history of a major depressive episode at the age of 22. At that time she had a trial of sertraline, to which she responded well and continued receiving sertraline for 15 months. After stopping the medication she suffered from anxiety and she had several panic attacks. It was considered that she had a relapse of depression. Then she had successive trials of sertraline, paroxetine, venlafaxine, fluoxetine and escitalopram, along with benzodiazepines. The patient however discontinued all proposed medications because of side effects and medication phobia. She refused every new medication. Such clinical cases raise the question of how patients with medication phobia and intolerable side effects can be successfully treated.

Giannopoulou, Chara
**Mirtazapine in Oncology:** Two clinical cases are presented, in which patients suffering from cancer and treated with chemotherapy exhibited episodes of nausea and vomiting. Treatment with low dose mirtazapine, 7.5mg and 15mg respectively, resulted in rapid dissolution of those symptoms in both patients. Mirtazapine, being a 5-HT3 receptor antagonist is suspected to have a clear antiemetic effect and thus can be a safe choice in oncology patients suffering from nausea and vomiting, irrespectively of being depressed or not. Possible synergistic effects on mood can be considered beneficial and thus, it is discussed whether mirtazapine should be considered first line antidepressant in oncology patients instead of the most commonly used selective serotonin re-uptake inhibitors.

Kafetzopoulos, Vasileios
**The involvement of an intact prefrontal cortex- hippocampal circuit in the forced swim test in male and female rats:** The prefrontal cortex (PFC) and the hippocampus are involved in the pathophysiology of depression; however, the contribution of the circuit formed by their connection is unclear. We investigated the role of the PFC-hippocampus circuit in depression by lesioning in rats the relay nucleus reuniens (RE) and thus disrupting the PFC-hippocampus connection. RE lesion reduced immobility and enhanced swimming duration in the forced swim test, while sex differences in antidepressant response were present in lesioned animals. The integrity of the PFC-hippocampus circuit is necessary for the expression of depressive behaviour. Disrupting the PFC-hippocampus circuit also affects the sex-differentiated response to SSRIs.

Kanelli, Stamatina
**Rare ophthalmological side effect from tricyclic antidepressant:** Clomipramine inhibits the serotonin and noradrenaline pump, increasing the availability of these neurotransmitters in the synapse. Its anticholinergic action can cause sedation, xerostomia, constipation and blurred vision. Other usual side effects involving eyes are adjustment disorder and an increased intraocular pressure. Our patient with a seven year history of obsessive-compulsive disorder was admitted to our clinic already treated with clomipramine at least five years, lately with a dose of 225 mg daily. During his stay, the oral administration of the drug was discontinued, followed by intravenous injection with a gradual increase in the dose of 25mg, 25mg every five days. The intravenous administration was stopped at 150 mg. Our patient received the drug only for about 20 days and the treatment was stopped due to dizziness, blurred vision, fatigue and xerostomia. In the ophthalmological control, we observed reduction of visual acuity to 3/10 non adjusted on both eyes, horizontal nystagmus, unstable diplopia, mild disturbance in colour perception and abnormal pupillary reflex on both eyes, compatible with optic neuritis. Administration of clomipramine was interrupted resulting in rapid disappearance of clinical symptoms and restoration of visual acuity to 10/10 on both eyes, as the ophthalmological examination revealed at that time and also six weeks afterwards.
Karakitsou, Nefeli

Treatment of cancer patients with serotonin-noradrenaline reuptake inhibitors: In this project, careful concern should be given to the diagnostic criteria of a ‘major depressive episode’ used for this special group of patients. Firstly, there is an overlap of neurovegetative symptoms (insomnia, decreased appetite, etc.) due to cancer and its treatment and due to psychological components. Secondly, it may be difficult to differentiate normal grief and sadness due to illness with pathologic depression. Since the experience of SSRIs is wider it would be interesting to examine the use of SNRIs. Another challenge will be the selection of the sample considering the wide range in the spectrum of patients with cancer.

Kleisas Spyros

Treatment dilemmas in an acutely suicidal bipolar type-II patient: Mrs. C., a 72 years old female patient presents in private practice with nausea, vomiting, tremor and diarrhea, reportedly for a week. She seems dehydrated and confused. She has been receiving lithium for the past 20 years together with antipsychotics, antidepressants and anxiolytics. Her previous psychiatrist had increased the lithium dose one month earlier, and currently she was lithium intoxicated. Lithium was discontinued and her kidney function returned to normal. A week later she started showing depressive mood, difficulty in sleeping, and a very intense suicidal ideation. In the past she had committed two suicide attempts: one when she was an adolescent and one when she was 68 years old, both were benzodiazepine overdoses. She had a sister that committed suicide when she was 28 and a niece (her sister’s child) who also committed suicide when she was nineteen.

Kokras, Nikolaos

Depression, antidepressants and surrogate endothelial markers: Commonly prescribed psychotropic medications, such as antipsychotics and antidepressants are known to increase prolactin serum levels to a greater or lesser degree. The current standard of practice is to leave patients with asymptomatic hyperprolactinemia untreated. However emerging evidence suggests that moderate and perhaps even small increases in prolactin levels may significantly aggravate specific arterial characteristics and thus increase the risk of adverse cardiovascular events. In this open label flexible dose observational study, patients are recruited if suffering from major depression and being treated with a combination of a selective serotonin re-uptake inhibitor and an atypical antipsychotic. Assessment of surrogate markers of preclinical atherosclerosis, arterial stiffening and endothelial and microcirculatory function as well as prolactin levels assays take place at baseline and following a mean treatment duration of four months.

Komporozos, Angelos

Tiagabine in the treatment of alcoholism: There is evidence that GABAergic anticonvulsants can be efficacious in the treatment of alcohol dependence and in the prevention of alcohol relapse because these agents act on the substrate that is involved in alcoholism. In this randomized, open pilot study, we aimed to investigate the efficacy and tolerability of tiagabine as adjunctive treatment of alcohol-dependent individuals during the immediate post-detoxification period and during a 6-month follow-up period following alcohol withdrawal. Although a steady improvement in terms of psychopathology, craving and global functioning was observed in both groups throughout the study, subjects on tiagabine improved significantly more. The relapse rate in the tiagabine group was lower than in the control group. Tiagabine was well tolerated and only a minority reported some adverse effects in the beginning of treatment. Results from this study suggest that tiagabine is a safe and effective medication for the management of alcohol dependence when given adjunctively to a standard psychotherapy treatment.

Liapis Christos

Evaluation of the impact of revascularization on cognitive and psychological status and on the quality of life in patients with carotid stenosis: The estimation of the anatomical and pathophysiological causality links between cerebral blood flow alternations and patients’ cognitive performance as well as their mental status, opens a new, wide, pioneering and inter-disciplinary medical field of research and clinical practice, by pointing out the emerging benefits of carotid revascularization techniques, in neurological, biochemical and neuroimaging terms. Within this frame, further clinical research on delirium attracts interest, specifically at the point of studying the possibility of a coexisting underlying vascular pathology that worsens the cognitive and functional decline often demonstrated in
old aged patients with preceding clinical manifestations of delirium. Such a hypothesis can propel following interventions from the Health Policy domain, aiming at the improvement of the quality of life of these patients.

Lyrakos, Dimitrios
Dysfunctional remembered parenting in oncology outpatients affects psychological distress symptoms in a gender-specific manner: Evidence suggests that gender differences appear in a variety of biological and psychological responses to stress and perhaps in coping with acute and chronic illness as well. Dysfunctional parenting is also thought to be involved in the process of coping with stress and illness; hence, the present study aimed to verify whether dysfunctional remembered parenting would influence psychological distress in a gender-specific manner in patients suffering from cancer. Further on, an on-going cross-sectional study aims to evaluate the use of antidepressants, benzodiazepines and other psychotropics in patients with and without a dysfunctional remembered parenting, when confronted with the stressor of a severe somatic disease.

Mavridis, Thodoris
Role of neuroestrogens in the development of cognitive deficits and affective disorders and attribution of those behavioral deficits to neurochemical and molecular alterations in both sexes: It is anticipated that inhibition of neuroestrogen synthesis in the brain with an aromatase inhibitor will cause or aggravate specific behavioral indices that simulate depressive symptoms including anxiety, excess fear and learning deficits in male and female rats. Subsequently it is expected that the distinct behavioral indices related to depressive symptomatology and stress vulnerability caused by aromatase inhibitors will be matched with specific changes in neurotransmission (due to rapid and genomic effects of neuroestrogen) and in neuroplasticity (due to long lasting changes of neuroestrogen) in a sex-specific manner.

Mousios, Dimitrios
Dilemmas in Schizophrenia treatment: In this presentation, it will be attempted to critically appraise the schizophrenia treatment guidelines. Furthermore, a review on the newest data on schizophrenia prodromal symptoms and data for patients at high risk to develop psychoses will be presented. With regards to acute psychoses, recommendations for pharmacological treatment and what type, but also it will be discussed the use of electroconvulsive treatment as a biological therapy, its mechanism of action and its indication when the pharmacological treatment fails.

Nika, Stella
Sertindole and impaired atrioventricular function, in the absence of Q-T prolongation: The congenital channelopathies, the LQT Syndrome, and the increased risk for syncope, seizures, and sudden cardiac death. LQTS is a genetically heterogeneous disorder most often inherited in an autosomal dominate mode and emotions could be a triggering factor for lethal dysrhythmia.

Novais, Ashley
Unraveling the biological role of neudesin: behavioral impact of neudesin ablation: We aim at investigating if the ablation of neudesin (Nenf), a putative non-canonical neurotrophic factor, impairs brain function, namely behavior. We evaluated the i) acquisition of early postnatal age specific neurological functions by analysing behavioral developmental milestones of nenf-null mice vs. control littermates; ii) emotional and cognitive behavioral dimensions in adult neudesin-null mice. We found that nenf-null mice display i) latency in the acquisition of maturity for particular developmental milestones; ii) an anxious-like phenotype, as assessed in the elevated plus maze and light dark box. We will next characterize the monoaminergic neurotransmission system in target brain regions i) involved in the control of movement and body position and ii) in anxiety, to further explain the behavior phenotype observed in nenf-null mice.

Oikonomou, Elina
Neuropsychiatric manifestations in patients with multiple sclerosis (MS) treated with fingolimod: Several neuropsychiatric disorders such as depression, anxiety, psychosis, sleep disturbances and cognitive impairment were reported in patients receiving medications to treat MS. Fingolimod is a recently approved new class of disease modifying oral medication used in the treatment of relapsing forms of MS. The aim of the study is to investigate
the presence of neuropsychiatric symptoms in patients with MS receiving fingolimod. A control group of MS patients with a similar degree of disability will be used in the study. Patients prescribed fingolimod from August 2012 are identified from the admission records at the Neurology Department. Demographics, MS disease history, psychiatric history including medication history are obtained on initial visit. Subjects are assessed with a number of questionnaires on initial visit and at 6-month and one year follow up appointments.

**Papakonstantinou, Anastasios**

**Resolving drug-induced hyperprolactinemia in patients receiving antipsychotics with aripiprazole adjunct treatment:** Sexual dysfunction receives limited attention in the care of patients with mental illness. It is estimated that 30-80% of patients with schizophrenia suffer from sexual dysfunction, making it an important cause of poor quality of life. Early recognition and prompt treatment of the sexual dysfunction caused by psychotropic drugs (due to chronic hyperprolactinemia), can reduce the adverse effects of treatment, thus strengthening adherence. There is a serious risk of medication discontinuation by patients who discover their inability to have a normal sex life. The purpose of this study is to investigate whether the addition of aripiprazole can restore the high levels of prolactin and/or improve the hormonal side effects in patients who are already on antipsychotic monotherapy and have drug-induced hyperprolactinemia and/or hormonal side effects.

**Papalias, Elias**

**Interpersonal Violence and Alcohol abuse:** Substances with psychotropic actions may play a significant role in the appearance of interpersonal violence and alcohol is certainly involved in such behaviours. It is known that 58% of men and 30% of women consume alcohol before committing a crime. Interestingly, international studies report that 40% of violence victims have also consumed alcohol. However alcohol use and potential abuse may present differences across different societies and cultures. In this study, we are investigating whether in Greece violent behaviour presents similar associations with alcohol abuse as to what is reported in international studies.

**Pappas, Dimitrios**

**Human Aggression:** Aggression works. Like most animal behaviours, aggression is the refined and tested product of more than 3 billion years of trial and error. The fact that aggression works, is not what immediately comes to mind when the psychiatrist is confronted with a threatening patient. In this presentation we will discuss the pharmacological interventions – treatments of aggressive behaviours primarily seen in adulthood. Also, a discussion will follow on the treatment of aggression in schizophrenia and schizoaffective disorder, in intermittent explosive disorder, in borderline personality disorder, in Alzheimer’s disease and related dementias, in traumatic brain injury and of intimate partner violence.

**Polissidis, Alexia**

**The cannabinoid CB1 receptor antagonist SR141716 diminishes the effects of amphetamine on locomotor activity and dopamine and glutamate in the nucleus accumbens in vivo:** The aim of the present study was to investigate the effects of coadministration of SR141716 (SR), a CB1 receptor antagonist, and the psychostimulant d-amphetamine on locomotor activity and extracellular dopamine and glutamate levels in the nucleus accumbens in rats. Our results showed that d-amphetamine per se induced hyperlocomotion and increased dopamine and glutamate. When coadministered with SR, a reduction in d-amp-induced hyperlocomotion was observed and increases in dopamine and glutamate levels in the nucleus accumbens were reversed. This study provides evidence for the role of the endocannabinoid system in d-amphetamine-induced behavioral and neurochemical effects in vivo, and on a greater scale, psychostimulant addiction.

**Sardis, Matthaios**

**A case of treatment-resistant obsessive - compulsive disorder:** A clinical case is presented of a 61-year-old male patient with a history of obsessive – compulsive disorder with obsessions about contamination and doubt. He presented four months ago with depression-like symptoms and especially chronic fatigue, hypersomnia, psychomotor retardation, loss of interest and decreased activity. He is currently under treatment with sertraline 200mg, amitriptyline 50mg and modafinil 100mg daily with poor and unstable results.
Sarrigiannidis, Alexios

Sleep disorders in psychiatric patients: Disorders of sleep (insomnia, hypersomnia, etc.) are part of the diagnostic criteria for various psychiatric conditions, but psychiatrists are not adequately trained in clinical practice at acquiring a thorough sleep disorders history and evaluating a patient’s sleep problem. Conditions such as narcolepsy that have a high comorbidity with psychiatric disorders are still under-recognised. An increased awareness of sleep disorders is necessary and should be provided in psychiatry training.

Selakovic, Mirjana

Relationship of stress, cannabis use and first psychotic episode: Several studies have associated cannabis use and stressful life events with first psychotic episode (FPE). We examined the relationship between stressful life events such as service in the army, cannabis use and FPE. Twenty soldiers who were hospitalized in the Military hospital for a FPE were studied and compared with 20 patients who were hospitalized in a public hospital for FPE, in the same time period. One of our conclusions is that the combination of the service in the army and the cannabis use do not have a more negative impact on the onset of FPE than what they have each one by itself.

Skokou, Maria

Treatment of depression in schizophrenia: Depression is common in schizophrenia, as well as insomnia and circadian sleep/wake disruptions. Studies on the efficacy and safety of available antidepressants are relatively lacking, while sleep disorders in the context of schizophrenia have only recently attracted research attention. In the present observational study the efficacy of agomelatine will be measured, in a sample (n=30) of stabilized patients with schizophrenia and depressive symptoms. The severity of depression will be measured by applying the Calgary Depression Scale for Schizophrenia, and the severity of insomnia will be assessed by using the Athens Insomnia Scale. Data regarding adverse reactions will also be collected.

Stachtea, Xanthy

Psychophysiological phenomena and pharmacological interventions: the paradigm of latent inhibition and prepulse inhibition: The comparison and evaluation of modern psychophysiological techniques using physiological and biological parameters is of major research interest. An ongoing project consists of psychophysiological monitoring with behavioral paradigms of Latent Inhibition and Prepulse Inhibition of startle. The present study is designed with the aim to delineate cognitive deficits in major psychopathologies such as schizophrenia by measuring the performance of patients and healthy volunteers in computerized applications of the above mentioned paradigms. Pharmacological interventions will be discussed.

Stefanakis Giorgos

Treatment Resistant Obsessive – Compulsive Disorder: A Case Report: A 27 year old male patient suffered from obsessive – compulsive disorder for the last 10 years. Pharmacologically has he been treated with various SSRI’s, and tricyclic antidepressants, along with benzodiazepines, lithium and pregabalin. However all pharmacological interventions proved inefficient in reducing symptomatology. In addition, the patient was also treated with cognitive behavioral therapy for two years with moderate results. He has also undergone 2 treatment cycles of electroconvulsive treatment, with 7 and 14 sessions respectively.

Tarasidis, Christos

Inter-racial and inter-individual differences in antidepressant action and side effects: Subjective clinical experience claims that there are differences of the medication effects and side effects on patients of different ethnicity. Different recommendations are given from treating physicians to their patients about the usage of antidepressant medication. Physicians recommend the usage of SSRI’s on the evening, however patients often develop insomnia as a side effect. On the contrary, some patients prefer to take SSRI’s on the evening because they feel tired after. Clinical experience also highlights that duloxetine, whereas has good efficacy and few side effects on patients from Central Europe, it has more side effects on Greek patients. Examples like this highlight possible inter-racial and inter-individual differences. The value of genetic enzyme analysis to get better results on the therapy will also be discussed.
Tsopelas, Christos
Polypharmacy in Schizophrenia treatment: The aim of the present research is to assess the prevalence of polypharmacy in patients who are hospitalized in the Psychiatric Hospital of Attica. Data were collected from a randomly selected group of 305 patients with a diagnosis of Schizophrenia. After 3 weeks of hospitalization 49.9% of patients were on either first generation antipsychotics (FGA) or second generation antipsychotics (SGA), while 38.8% were on a combination of FGA and SGA and 11.3% were receiving more than two antipsychotics with the use of long acting antipsychotics in 27.3% of patients. Despite treatment guidelines, and despite the general acceptance of monotherapy as the preferred practice, antipsychotic polypharmacy continues to increase.

Ziakas Dimitris
Distinguishing between treatment-resistance and non-compliance in deciding clozapine initiation in schizophrenia: A clinical case is presented of a 45 year-old female patient with nearly 20 years of psychopathology who has been on several treatment regimens: haloperidol, chlorpromazine, aripiprazole with insufficient and inadequate response given that she constantly suffered from auditory hallucinations and persecutory delusional ideas. She was diagnosed as “treatment-resistant schizophrenia” and she was admitted with the aim of initiating clozapine. However, upon detailed examination, it was revealed that she had not had a proper antipsychotic trial with an adequate dose for an adequate duration. The question of when should patients be considered truly treatment-resistant is discussed.
Nafplio is a seaport town in the north end of the Argolic Gulf in Peloponnese, Greece. It was named after Nafplios, son of Poseidon, and home of Palamidis, a hero of the Trojan war. During the Greek War of Independence (1821-1830), the 3rd National Assembly decided in 1827 that Nafplio would be the first official capital of the Hellenic State, and Kapodistrias the First Governor. Following his assassination in 1831, the Bavarian King Otto of Greece arrived in Nafplion and then decided to move the capital to Athens in 1834.