Highlights of the 25th European College of Neuropsychopharmacology (ECNP) Congress

Vienna, Austria, October 2012

In October 2012, the annual congress of the European College of Neuropsychopharmacology (ECNP) was held in Vienna, Austria. This report contains highlights of the five-day event, including information from podium presentations, educational symposia and the satellite symposia. The content is divided into two major themes: major depression and bipolar disorder. Other topics of interest are also briefly discussed.

ECNP 2012 Congress Report

Highlights from ECNP Sessions
Focusing on Depression

Several satellite symposia at ECNP 2012 dealt with major depression. The following review provides the highlights of these sessions.

Outstanding needs in depression and how multimodal antidepressants may solve them (Saturday, October 13). The first of the depression symposia to be presented at ECNP 2012 focused on identifying shortcomings of current depression therapy and discussing how multimodal antidepressants may help to address these shortcomings.

Dr. Yoram Barak, of Tel Aviv University in Israel, was the first of the speakers in this symposium. He reviewed data concerning the efficacy of current antidepressant options. In short, his thesis noted that there are excellent antidepressant treatment options available, but that substantial gaps remain unaddressed.

Of note, Dr. Barak stressed that overall health goes beyond that which is typically measured by efficacy endpoints in clinical trials. He used clinical-trial data to demonstrate how improvements in symptoms do not necessarily equate to improvements in function. The fact that so few clinical trials (less than 5%) evaluate patient functioning, Dr. Barak suggested, represents a substantial gap in our knowledge about the utility of antidepressants.

Dr. Eric Lenze, professor of psychiatry at Washington University in St. Louis, Missouri, presented the session on unsolved needs in depression, focusing on cognitive dysfunction. This is a clinically important deficit in depression, one that may be mistaken for attention deficit hyperactivity disorder (ADHD) in younger patients and mild dementia in older patients.

Cognitive deficits associated with depression affect not only concentration, but almost every domain of cognition, including processing speed, executive function, working memory, and episodic memory; these deficits can cause difficulties in every aspect of daily life. Dr. Lenze outlined the bi-directional relationship between depression and impaired cognitive function: depression itself may arise due to neurologic, neuroendocrine, inflammatory, or metabolic changes (which may also cause cognitive difficulties), but there is also the potential for depression itself to be neurotoxic and for cognitive dysfunction to be secondary to the disorder.
In older adults, Dr. Lenze explained, it may be more difficult to determine the cause of cognitive difficulties, as these patients are at higher risk for dementia and cerebrovascular disease and are more likely to be taking medications that can impair cognition (e.g., anticholinergics, hypnotic sedatives). While achieving sustained remission from depression may help to prevent cognitive decline, some antidepressants (e.g., paroxetine) have substantial anticholinergic effects. Even antidepressants with “cleaner” receptor-binding profiles can have a negative impact on cognition or, at best, exert a neutral effect on cognition.

Efforts to treat cognitive difficulties with specific pharmacotherapy have produced mixed results. A study of 130 older adults with major depression evaluated the effects of the cholinesterase inhibitor, donepezil, on cognitive performance, instrumental activities of daily living, and recurrences of depression over two years of maintenance treatment. The investigators observed that donepezil treatment was associated with modest improvements in cognition and activities of daily living, but that the donepezil group was 35% more likely to experience a recurrence of major depression compared to placebo.

To minimize the impact of cognitive dysfunction in depression, Dr. Lenze advocated minimizing the use of medications known to exacerbate cognitive dysfunction, increasing physical activity, improving diet, and promoting better sleep hygiene, smoking cessation, and reduced consumption of alcohol and/or other substances of abuse.

This symposium’s final speaker, Dr. Pierre Blier, psychiatrist at the University of Ottawa, in Ontario, discussed how the use of multimodal antidepressants (e.g., agents with combined receptor activity and reuptake inhibition) may help to overcome the shortcomings of currently available therapies.

Major depression is a highly heterogeneous disease, with different patients presenting with different sets of symptoms and complicating factors. In fact, Dr. Blier discussed how individual patients might have completely opposite symptomatology (see Figure 1 for illustration of this heterogeneity). Major depression also may be complicated by associated features of anxiety or psychosis, or by comorbid diagnoses.

Dr. Blier discussed how the various neurotransmitter systems targeted by psychotropic medications are all interconnected, with manipulation of one system often having substantial impact on another. For example, he reviewed how treatment with a selective serotonin reuptake inhibitor (SSRI) could impair norepinephrine transmission, but that this effect can be reversed through the blockade of serotonergic 5HT2A receptors. Another example is the inhibition of dopamine activity with the administration of an SSRI.

Recognition of the multi-receptor effects of antidepressant therapy provides a mechanistic rationale for the efficacy of adjunctive psychotropic medication with a complementary receptor-binding profile (e.g., atypical antipsychotics). Another potential approach to targeting multiple receptors for the treatment of depression is to use single agents that have substantial effects on more than one neurotransmitter pathway.

Dr. Blier presented the results of an eight-week, placebo-controlled trial of vilazodone, an agent that combines selective serotonin reuptake inhibition with serotonin 5HT1A agonism. In this study, vilazodone was associated with a significant difference in Montgomery–Åsberg Depression Rating Scale (MADRS) and Hamilton Depression Rating Scale (HAM-D) scores, evident as early as one week after treatment initiation.

The other multimodal agent Dr. Blier discussed was vortioxetine, which has a wide range of effects on relevant neurotrans-
Dr. Blier described how this agent’s multimodal profile of combined receptor activity and uptake inhibition could exert clinical effects on a wide range of symptoms. Along with direct effects of 5HT3 and 5HT7 antagonism, 5HT1B partial agonism, 5HT1A agonism, and SERT inhibition, vortioxetine also has indirect effects enhancing multiple neurotransmitter systems. These include increases in serotonin, noradrenaline, acetylcholine, dopamine and histamine. Altogether, these mechanisms may yield clinical effects such as improved mood and cognition, and reduced sexual dysfunction, anxiety and insomnia.

In a six-week proof-of-concept study, vortioxetine (5 mg or 10 mg daily) was associated with significant reductions in MADRS total score compared to placebo, with the differences reaching statistical significance as early as Week 2 (Figure 2). Vortioxetine also was studied in an eight-week trial with duloxetine or placebo among 453 elderly patients with recurrent major depression. Vortioxetine and duloxetine each demonstrated superior efficacy relative to placebo. In addition, vortioxetine showed superiority to placebo in cognition tests of speed of processing, verbal learning, and memory.

Dr. Blier concluded that, as major depression is such a heterogeneous disease, it is difficult to imagine that a single medication with a single mechanism of action will treat all patients effectively. There is a need for further study of medication combinations targeting multiple neurotransmitter pathways; multimodal single agents may also play a role, combining the potential benefits of medication combination with a more favorable side-effect profile.

**Overcoming the challenge of inadequate response in major depressive disorder (Saturday, October 13).** The second satellite symposium to be presented on the ECNP’s first day also involved a discussion of the shortcomings of antidepressant therapy and potential ways these might be addressed.

The symposium’s first speaker, Dr. George Papakostas from the Massachusetts General Hospital in Boston, discussed some of the key problems among patients treated with antidepressant therapy. According to data from the Sequenced Treatment Alternatives to Relieve Depression (STAR*D) trial, even if patients are systematically treated with successive lines of antidepressant therapy, approximately one quarter will never obtain a response to antidepressant medication and approximately one third will not achieve remission. In studies examining patients treated in a less-structured manner, response and remission rates are even lower: one study in an outpatient setting, for example, found approximately 20% of patients achieved remission with the first therapy, while overall cumulative remission was approximately 50%.

In addition to poor response and relapse rates, patients can have persistent problems with sleep, fatigue, anhedonia, and concentration. The prevalence of several of these persistent symptoms among patients with major depression who were treated to “remission” with antidepressant therapy are shown in Figure 3. In addition, Dr. Papakostas illustrated how the cognitive difficulties can span a number of domains, including mental acuity, word-finding, recall, and focus.

The importance of comorbidities was also discussed in this symposium. Dr. Papakostas provided additional data from the STAR*D trial, in which approximately two thirds of patients had at least one comorbid psychiatric condition (data included 16.1% of the sample who had two comorbid disorders and 20.2% who had two or more comorbid psychiatric disorders).

The next speaker, Dr. Michael Thase of the University of Pennsylvania in Philadelphia, also cited evidence from the STAR*D study, illustrating how each successive treatment strategy for major depression is less and less likely to achieve the desired outcome (i.e., response and remission).

Dr. Thase outlined the different pharmacologic strategies that currently exist to combat inadequate response in major depression.
depression, focusing on adjunctive therapy with atypical antipsychotics. He presented data from different clinical trials demonstrating the efficacy of aripiprazole, olanzapine, extended-release quetiapine, and risperidone as adjunctive therapy with antidepressants. Many of these studies were included in a 2009 meta-analysis involving 16 studies and 3,480 patients. The authors of this meta-analysis showed that adding an atypical antipsychotic to antidepressant therapy led to significantly higher rates of response and remission than the addition of placebo. The rates of response and remission were 44.2% and 30.7%, respectively, for antidepressant plus atypical antipsychotic, and 29.9% and 17.2%, respectively, for antidepressant plus placebo.

One key benefit of the addition of an antipsychotic is the relatively quick onset of action. Dr. Thase noted how, across the adjunctive antipsychotic studies, separation from the placebo arms occurred during the first one to two weeks of therapy. This is an important finding given the importance of obtaining a rapid response in critical clinical situations.

Dr. Thase highlighted some of the particular benefits of adjunctive antipsychotic therapy, including the potential for greater improvement in anxiety symptoms and insomnia with aripiprazole and extended-release quetiapine. These are symptoms that may persist even among patients who have responded to antidepressant therapy.

There are, however, several shortcomings associated with adjunctive antipsychotic therapy for major depression. Aripiprazole, for example, is associated with increased risk of restlessness, while olanzapine, quetiapine, and risperidone have well-known adverse effects on metabolic parameters (e.g., weight gain).

Dr. Thase concluded that several questions remain with respect to adjunctive therapy with atypical antipsychotics. These include determining the appropriate duration of adjunctive therapy among responders; whether or not benefits obtained are actually due to adjunctive effects or whether the antidepressant can be discontinued; determining the risks of tardive dyskinesia; and calculating the cost-benefit ratio of this strategy compared to other adjunctive agents (e.g., lithium).

The final speaker in this symposium was Dr. Koen Demyttenaere, from Leuven, Belgium. His presentation focused on what the future might hold for the management of major depression. One of the areas in need of improvement is to make randomized clinical trials (RCTs) more applicable to everyday practice. Most RCTs that evaluate antidepressants have been highly selective in their inclusion and exclusion criteria, with a specified range of symptom severity and restrictions on comorbidities. Among patients in one psychiatric practice, for example, 92% would not have been eligible for a clinical trial: 55% due to insufficient symptom severity and 37% due to comorbidity or suicidality. It is difficult to apply the results of clinical trials to clinical practice given these observations.

Dr. Demyttenaere likewise addressed a need for improvement in patient involvement in treatment decisions. Evidence has shown that patient definition of remission may be different from that of mental-health professionals. The criteria that patients have cited as defining remission include: a presence of positive mental health; general sense of well being; feeling in emotional control; feeling like usual self; and absence of depressive symptoms. Many of these criteria may not match well with the strict symptom-based criteria usually used in clinical trials. Research has also shown that outcomes are significantly better when the treatment a patient receives is the one he or she prefers. A patient’s outlook is also critical, an optimistic outlook resulting in greater improvement. The impact of these factors constitute determinants of response that, in some cases, are as important as the treatment selected.

In terms of therapy, Dr. Demyttenaere reviewed the various potential therapeutic strategies that are currently under investigation. These include novel agents targeting the same receptors as current therapies (e.g., the serotonin–norepinephrine reuptake inhibitor [SNRI] milnacipran), multimodal antide-
pressants targeting more than one known neurotransmitter pathway (e.g., vilazodone, vortioxetine), agents with even broader neurotransmitter targets (e.g., agomelatine, amitifadine) and nonpharmaceutical supplements (e.g., L-methylfolate).

**A new chapter in the history of depression (Monday, October 15).** The third satellite symposium included discussion of several novel topics. Dr. William Deakin, of the University of Manchester, United Kingdom, presented an update on the understanding of brain neurocircuitry in depression. Brain imaging studies have shown that depressed patients have an aberrant response to emotional stimuli (measured by responses to various face types [e.g., happy, sad, afraid]) that appears to be related to abnormalities in brain circuitry. Treatment with the SSRI antidepressant, citalopram, has been associated with changes in circuitry within the hippocampus and amygdala, which affected the way in which emotions were perceived by individuals with depression. The investigational melatonin agonist, agomelatine, has also been associated with changes in brain circuitry, albeit through different mechanisms and brain regions (ventral visual stream, frontal areas, and thalamus). This agent has also been associated with modulation of responses to faces expressing different emotions.

The second speaker, Dr. Raymond Lam from the University of British Columbia, also discussed the effects of agomelatine in major depression; he included a review of the current evidence in support of agomelatine in the context of rapidity of treatment response. Previous evidence has shown that patients who respond early (e.g., within the first two weeks) are more likely to go on to achieve remission. Assessment of the time to response has, however, been a problem with many clinical trials, which often do not have adequate frequency of follow-up during the first several weeks of therapy.

Among currently available agents, data suggest that mirtazapine may be associated with more rapid response than SSRIs or venlafaxine. However, this agent is also associated with higher incidence of adverse events. As Dr. Lam showed, data with agomelatine suggest that this agent is similarly associated with a rapid onset of efficacy. In an eight-week, multicenter, double-blind trial, patients with major depression were randomly assigned (1:1:1) to receive a once-daily dose of agomelatine 25 mg, agomelatine 50 mg, or placebo. Agomelatine was associated with significant improvements in mean scores on the 17-item Hamilton Depression Rating Scale (HAMD), the trial’s primary efficacy outcome measure (Figure 4). A statistically significant improvement for the 25 mg dose of agomelatine compared to placebo was observed at Week 1 after treatment initiation.

Dr. Sidney Kennedy, of the University Health Network in Toronto, was the third presenter in this symposium. He reviewed data with agomelatine from several comparative analyses in major depression that have examined efficacy in various domains. These included findings related to anhedonia, emotional blunting, anxiety symptoms, “clearer thinking,” sexual dysfunction, and functioning as measured by the Sheehan Disability Scale (SDS).

The symposium’s final speaker was Dr. Pierre-Michel Llorca from the University of Auvergne in Clermont-Ferrand, France. His presentation included a review of data from another study involving agomelatine, demonstrating that treatment of major depression with this agent leads to a higher proportion of patients with a morning chronotype compared to an evening chronotype (with evening-chronotype agomelatine responders switching to morning chronotype). This is thought to be of clinical relevance, as the evening chronotype is associated with a greater severity of baseline depression.

See appendix for complete reference listing.
Highlights from ECNP Sessions Focusing on Bipolar Disorder

Several sessions at the ECNP 2012 addressed bipolar disorder. The following review provides some highlights from two of these sessions.

Bipolar I disorder: improving clinical and functional outcomes (Sunday, October 14). The faculty of this symposium discussed how bipolar I disorder is a multifactorial disorder with impact on several domains other than mood. A substantial portion of the symposium was devoted to reviewing the receptor profile of asenapine and reviewing the existing clinical trial data in bipolar I disorder with this agent.

As explained by Dr. Ana Gonzalez-Pinto, professor of psychiatry at Santiago Apostol Hospital in Vitoria, Spain, bipolar disorder is a multifactorial illness, characterized by instability in mood, affect, and circadian rhythm, as well as cognitive dysfunction and functional impairment.

Dr. Gonzalez-Pinto showed the results of a study, among patients with bipolar I disorder, evaluating the impact of the number of episodes of mania on cognition. The investigators found that the number of manic episodes predicted poor cognitive performance across a broad spectrum of tests reflective of neuropsychological function, suggesting that the recurrence of mania may have a long-term neuropsychological impact.

Furthermore, Dr. Gonzalez-Pinto showed research demonstrating that patients with bipolar I and bipolar II disorders have substantially greater functional impairment compared to healthy controls across all key domains (autonomy, occupation, cognition, finances, interpersonal, and leisure).

One of the outstanding issues with respect to management, as discussed by Dr. Gonzalez-Pinto, is the low rate of functional recovery in bipolar I disorder. Even among patients who have achieved remission (e.g., at least six months asymptomatic), there is a large proportion who do not achieve functional recovery (i.e., clinical remission plus good social function [work, independence, social activities, and relationships]).

In a two-year, prospective, observational study of 1,656 patients with bipolar I disorder, 64% of subjects achieved clinical remission, but only 34% achieved functional recovery. Furthermore, increased number of episodes has also been correlated with worse functional outcomes.

Dr. Torgny Svensson, professor of pharmacology at the Karolinska Institute in Stockholm, Sweden, presented a summary of the characteristics of asenapine, a novel antipsychotic being evaluated for the treatment of bipolar disorder. He pointed out the similarities of asenapine with clozapine, in particular its relative affinity for 5HT₂ and 5HT₇ compared to D₂, and its impact on dopamine.
outflow in the prefrontal cortex (PFC), a potent antipsychotic effect and low propensity to cause extrapyramidal symptoms (EPS). Preclinical evidence also demonstrates that asenapine enhances dopaminergic and glutamatergic transmission in the prefrontal cortex, increases firing in the locus coeruleus, enhances prefrontal monoamine release, and may enhance prefrontal output of dopamine, norepinephrine, and serotonin when used in conjunction with a selective serotonin reuptake inhibitor (SSRI).

These characteristics suggest asenapine is an antipsychotic that would have a beneficial effect on the manic, depressive, and cognitive symptoms of bipolar disorder, Dr. Svensson concluded. In addition, these observations provide support for the use of asenapine as an adjunctive agent to SSRIs for the treatment of major depression and bipolar depression.

Asenapine has been studied for the acute treatment of manic or mixed episodes, as monotherapy or as adjunctive therapy with lithium or sodium valproate. Dr. Roger McIntyre, professor of psychiatry and pharmacology at the University of Toronto, presented the results of several of these studies. In the acute bipolar I setting, asenapine 10-20 mg/day has been compared with placebo in two separate three-week studies, with olanzapine 15 mg/day included as an active control arm. It should be noted that these studies were not powered to detect differences between the two active therapies. In both studies, asenapine and olanzapine each significantly reduced the Young Mania Rating Scale (YMRS) score and the Clinical Global Impression Scale for Bipolar Disorder (CGI-BP); starting as early as Day 2, both agents demonstrated significantly greater reductions compared to placebo.

In a post-hoc analysis of patients from the two three-week studies, investigators observed a significantly higher remission rate from depressive symptoms on Day 7 and Day 21 (as measured by the Montgomery–Åsberg Depression Rating Scale [MADRS]) among patients treated with asenapine compared to those treated with placebo (Figure 1). There was also a significant difference between asenapine and olanzapine noted at Day 7, but not Day 21. Subsequent pooled analyses of these patients showed no significant difference between asenapine and olanzapine in reducing manic symptoms (including amongst those with mixed episodes) over a total of 12 weeks and one year. In the Week 12 analysis, the investigators also noted that the proportion of patients who experienced an increase in severity of depressive symptoms was approximately twice as high among olanzapine-treated patients (5.0%) than among those treated with asenapine (2.3%).

The prevalence of EPS varied depending on the rating scale used. For example, on the Barnes Akathisia Rating Scale (BARS), the proportion of patients with a score of 3 or greater was higher among those in the placebo group than among those treated with asenapine (approximately 3% vs. 2%), while the proportions scoring 3 or greater on the modified Simpson-Angus Scale (MSAS) was substantially higher in the asenapine group (6%) than the placebo group (2%). The mean weight change from baseline to one year was +2.0 kg with asenapine and +4.5 kg with olanzapine.

As an adjunctive agent, asenapine has been shown to improve response and remission rates (as measured by the YMRS) in a 12-week study among 324 patients with acute mania. Response and remission rates were substantially higher at Week 12 among patients treated with adjunctive asenapine than among those treated with a mood stabilizer alone.

**FIGURE 1. Remission Rates for Depressive Symptoms**

<table>
<thead>
<tr>
<th>Patients with baseline MADRS ≥ 20</th>
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<tbody>
<tr>
<td>Asenapine (n = 45)</td>
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<tr>
<td>Placebo (n = 33)</td>
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<tr>
<td>Olanzapine (n = 54)</td>
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</tbody>
</table>

Remission = MADRS total score ≤ 12; post-hoc analysis.

\*p < 0.05, \**p < 0.01 vs. placebo, \†† p < 0.01 vs. olanzapine.

Are there relevant biomarkers of bipolar disorder? (Sunday, October 14). One of the educational symposia presented during ECNP 2012 focused on the relevance of biomarkers in bipolar disorder. Building on the results of post-mortem studies, researchers have identified several inflammatory and coagulation proteins that are abnormal among patients with bipolar disorder. Analysis of dorsolateral prefrontal cortex samples from MDD patients revealed alterations associated with energy metabolism and synaptic function, while analysis of post-mortem pituitary and hippocampal proteome profiles of BD patients showed dis-
rupture of hormones, core histones and mitochondrial pathways. Analyses of plasma from BD mania patients have shown hormonal dysregulation and disturbances in energy and lipid metabolism, as well as changes in inflammatory and coagulation proteins. Follow-up after patients experienced manic episodes has revealed marked suppression of plasma pro-inflammatory markers. At present, all of these biomarkers remain investigational, but the faculty noted that further molecular and functional investigations might reveal clinically meaningful markers that could then be used to direct future drug-target identification and discovery. Additionally, it is thought that peripheral biomarkers, such as BDNF, inflammatory cytokines and oxidative stress parameters, could eventually be used as adjunctive tools for staging bipolar disorder, since these appear to be differently expressed in early and late stages of the disorder.

Other Selected Topics of Interest at the 2012 ECNP

In addition to the specific sessions pertaining to major depression and bipolar disorder presented above, ECNP 2012 also included symposia that focused on additional topics. The highlights of some of these sessions are presented below.

Alcohol dependence. A satellite symposium entitled “Optimising Management of Alcohol Dependence – Rationale for a New Treatment Paradigm” included a review of data from clinical trials of nalmefene, a potential new therapy for alcohol dependence.\(^1\) The primary data came from three multicentre, double-blind, placebo-controlled Phase III studies including a total of approximately 2,000 subjects. These studies were designed to assess the efficacy and safety of nalmefene in reducing alcohol consumption. In the studies designed to test efficacy, nalmefene was found to be significantly more effective than placebo in reducing the number of heavy drinking days and total alcohol consumption. Reductions in liver enzymes (gamma-glutamyltransferase and alanine aminotransferase) were greater in the nalmefene group compared to placebo throughout all three studies. The most frequent adverse events were dizziness, insomnia, and nausea, which were felt to be mild and transient.

Social anxiety disorder. In an educational symposium specifically focusing on social anxiety disorder, topics included the role of the amygdala in the pathophysiology of the disorder, advances in neuroimaging, and an update on management.\(^2\)\(^-\)\(^5\) In terms of management, the combination of cognitive behavioral therapy in conjunction with pharmacotherapy has recently been found in a new study to show better results than either treatment alone. Among psychotropics, selective serotonin reuptake inhibitors (SSRIs), serotonin-norepinephrine reuptake inhibitors (SNRIs), monoamine oxidase inhibitors (MAOIs), benzodiazepines, and pregabalin have all been shown to be efficacious. In the event of treatment failure, switching to an alternative proven treatment is favored over increasing the dose. Augmentation of SSRIs is another option with evidence of efficacy.

Suicidal behavior. One of the educational update sessions at ECNP 2012 dealt with predicting and preventing suicidal behavior in mood disorders and schizophrenia.\(^6\)\(^,\)\(^7\) Dr. Mark Taylor presented data from a systematic review of risk factors for suicide in schizophrenia.\(^8\) The factors found to have a strong association included young age, male sex, unemployment, higher level of education, depression, anxiety, post-traumatic stress disorder (PTSD), positive psychotic symptoms, insight, physical illness, positive family history of suicide, personal history of suicide attempts or thoughts, and alcohol and drug misuse.

The effects of treatment for schizophrenia in preventing suicide cannot be overstated. Analysis of Finnish national registry data showed that the relative risk of suicide among people with schizophrenia who were not receiving treatment was more than 30 times that of treated individuals.\(^9\) The relative risk of overall mortality was more than 12 times higher among untreated individuals. In major depression, the difference between treated and untreated individuals in terms of suicide risk is considerably less. A recent meta-analysis of trials involving fluoxetine or venlafaxine in 2012 demonstrated that suicidal thoughts and behavior decreased over time for adult and geriatric patients randomized to these agents compared to placebo.\(^10\) In this study, no differences in suicide-related outcomes were detected between treatment and placebo for youths.

See appendix for complete reference listing.
Appendix: Complete Reference Listings

ECNP Sessions Focusing on Depression


ECNP Sessions Focusing on Bipolar Disorder


Future Directions in the Management of Mood Disorders


2. Leboyer M. Towards a better understanding of mood disorders and the importance of somatic comorbidities. Presented at ECNP 2012; Abstract #C.10.02.


Other Selected Topics of Interest at the 2012 ECNP

1. Mann K. A potential new treatment for alcohol dependence: results from nalmefene phase III clinical program. Presented at ECNP 2012; Abstract #C.12.03.

2. Iancu J. The amygdala story in social anxiety disorder Presented at ECNP 2012; Abstract #E.14.01.


6. Taylor M How to predict and treat suicidal behaviour in schizophrenia. Presented at ECNP 2012; Abstract #E.02.02.

7. Mann JJ. How to predict and treat suicidal behaviour in mood disorders. Presented at ECNP 2012; Abstract #E.02.03.

