



Editorial

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EDITORIAL

Dear colleagues,

I am pleased to present to you our seventh issue of 2014 featuring up-to-date research on biomarkers and treatment strategies in schizophrenia and unipolar depression.

Following their review of literature on the disease concepts of **schizophrenia (SCZ)**, **autism disorder** and **mental retardation**, Waltereit and colleagues analyse selected **mechanisms shared in the neurodevelopmental pathways and synaptic plasticity**. Their results on psychopathological constructs support the existence of three distinct clinical entities, but also show important associations. Similarly, common mechanisms especially in global and social cognition could be disclosed.

By using wrist-actigraphy recordings, a sleep diary and standardized psychometric scales, Afonso and co-workers set out to examine sleep patterns, self-reported sleep quality and quality of life in **SCZ** outpatients. Their results show that **SCZ patients sleep more at night, but have poorer sleep efficiency and self-reported sleep quality** than healthy controls. Self-reported quality of life was also lower in SCZ. The authors found two disturbed patterns of sleep–wake phase in SCZ: an **advance sleep-phase syndrome and an irregular sleep–wake rhythm**.

In a functional magnetic resonance imaging (fMRI) study, Choi and colleagues examined the neural basis of the predictive and experiential components of anticipatory pleasure in SCZ. Results suggest that the **augmentation of hedonic enjoyment between the predictive and experiential stages of anticipatory pleasure is reduced in SCZ** because of **diminished activity in the reward-related regions during the prediction of pleasure**. Moreover, during cued-emotional experiences of anticipatory pleasure, SCZ patients seem to have difficulties in the integration of emotional information.

Schreiner and colleagues explored the **differences in outcomes for 1083 patients with SCZ treated with risperidone long-acting treatment (RLAT) or oral antipsychotics (oAP)**. The authors found that, at baseline, RLAT patients had higher symptom severity, greater functional impairment, and poorer compliance. In addition, RLAT patients showed a more positive clinical global impression increase and higher functional improvement than oAP patients.

The **relationship between serum levels of prolactin and the inflammatory status** in drug-naïve, first-episode **SCZ** patients with normal weight was investigated by Song and co-workers. Higher serum levels in SCZ were found for PRL, IL-1 β , IL-6 and TNF- α when compared to the control group. Within the SCZ group, positive relationships were found between serum levels of PRL and serum levels of IL-1 β , IL-6 and TNF- α with TNF- α being the strongest predictor among the three cytokines for serum levels of prolactin.

Wang and colleagues set out to explore the association between CMYA5 with SCZ and major depressive disorder (MDD) and genotyped 16 SNPs within the CMYA5 gene and performed case–control studies. In addition to SNPs that were specifically associated with SCZ or MDD, the authors found **one risk haplotype of rs16877109-rs3828611 (G-G)** that was **associated with both SCZ and MDD**. These results are interpreted as supporting the idea that specific alleles and haplotype in the CMYA5 confer risk for both SCZ and MDD.

The association between individual symptoms or symptom profiles with low BDNF levels in MDD was investigated by Bus and co-workers. The authors tested items of standardized diagnostic interviews and symptom scales individually in separate multiple regression analyses with serum BDNF levels as the dependent variable. Results revealed only the item “loss of interest” of the Composite International Diagnostic Interview to be associated with higher serum BDNF levels. The authors conclude that **decreased BDNF levels in MDD cannot be attributed to a specific symptom or symptom cluster**.

In an open-label trial, Berlim and colleagues explored the clinical utility of **deep transcranial magnetic stimulation (DTMS) as an augmenting strategy for treatment-resistant depression (TRD)**. After undergoing 4 weeks of daily high-frequency DTMS treatment over the left dorsolateral cortex, patients with TRD **showed response and remission rates of 70.6 and 41.2%**, respectively. Also, depression, anxiety, and suicidality ratings as well as quality of life ratings were improved.

Finally, Lai and co-workers investigated the efficacy and tolerability of ketamine given by rapid

intravenous infusion over 2–5 min in a double-blind, placebo-controlled, crossover design pilot study with four patients with TRD. Three of four patients achieved antidepressant response, for two subjects the greatest improvement was found at the highest dose received. Moreover, rapid infusion over 2 min led to adverse psychotomimetic effects, which also increased proportionately with ketamine dosage. The

authors conclude that the results do **not support the rapid intravenous infusion over 2 min for ketamine dosing in depression.**

Yours sincerely,

Siegfried Kasper, MD
Chief Editor