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Editorial

Siegfried Kasper (Chief Editor)

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Editorial

Dear colleagues,

I am delighted to introduce to you our last issue of 2015 featuring up-to-date research on suicidal behaviour and personality disorders as well as treatment options and biomarkers of impulse control disorders.

At the outset, a review by Richard-Devantoy and coworkers presents results on the **association between memory deficits and vulnerability to suicidal acts**. Analyses revealed that long-term memory and working memory were more impaired in patients with a history of suicide attempt and that autobiographical memory was less specific and more general in suicide attempters. The authors conclude that memory may play a significant role in the risk of suicidal acts.

Pompili and colleagues set out to investigate the **association between concentrations of lithium in drinking water and local rates of suicide** between 1980 and 2011. The authors indeed found an inverse relationship between trace lithium concentrations and suicide rates, particularly in women, between 1980 and 1989.

By using both evoked and induced event-related oscillations (EROs) and source localization (sLORETA), Andreou and coworkers examined **reward processing during a gambling task in borderline personality disorder** (BPD). Results showed reduced theta-band responses to negative feedback in BPD as well as disturbed evoked responses depending on feedback magnitude in the theta and high-beta frequency range in different brain regions.

Premkumar and colleagues set out to explore the **relation of positive and negative schizotypy to altered attention to rejection** and investigated healthy individuals with high, moderate and low neuroticism scores. Results showed that positive and negative schizotypy related divergently to attention to rejection with positive schizotypy attenuating, but negative schizotypy increasing rejection-related mobilization of attention.

Liu and coworkers explored the **association between *BDNF Val66Met* polymorphism and susceptibility to both obsessive-compulsive disorder** (OCD) **and Tourette syndrome** (TS). Results supported the involvement of the polymorphism as a common genetic susceptibility to both disorders showing specific gender trends.

Tong and colleagues investigated the **association between polymorphisms of the gene encoding dopamine beta hydroxylase (*DBH*) with attention deficit hyperactivity disorder** (ADHD). The authors indeed found an association of a *DBH* gene variant, *rs129882*, which confers risk to ADHD, with reduced in vitro gene expression. It is discussed that reduced DBH expression would be consistent with decreased conversion of dopamine to noradrenaline and thus with a relative hypo-noradrenergic state in ADHD.

Ozbaran and coworkers examined the **efficacy, safety and tolerability of combined methylphenidate and atomoxetine pharmacotherapy in children and adolescents with ADHD**. Results revealed irritability, appetite reduction, palpitations and headache as most common side effects and an improvement in symptoms in the majority of the patients.

Van Rooij and colleagues set out to explore the **influence of genetic variants within the serotonin pathway on response inhibition in adolescents with ADHD**. Although whole-brain analyses demonstrated large scale neural activation differences in the medial frontal and temporal/parietal regions of the brain's inhibition network between the variants of the *HTR1B* and *5HTT* genes. Activation in these regions was associated with response inhibition performance, but not with ADHD diagnosis or severity.

In their brief report, Bloch and coworkers examined the **effects of methylphenidate therapy on prosody in adult ADHD**. The authors found a prosodic change as an effect of methylphenidate only in a cognitive task, but not in an emotional task, and only in female patients. Prosodic changes are discussed as possible objective and accessible dynamic biological markers of treatment responses.

Yours sincerely,
Siegfried Kasper, MD
Chief Editor