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EDITORIAL

# Selected topics of the DGPPN Congress 2012

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This issue presents some selected topics from last year's DGPPN Congress in November 2012, which might be of interest for the broad readership of our journal. Bridging the area of expertise between Neurology and Psychiatry, Hellmann-Regen et al. [1] deal with depressive syndromes representing a common feature in several neurological disorders. The most prominent example is the post-stroke depression on the one hand, while on the other hand, depression itself is a risk factor for developing cardiovascular diseases and ischemia. Depressive symptoms are also often related to other neurological conditions such as epilepsy, multiple sclerosis and Parkinson's disease with impact on outcome and quality of life. Besides interesting findings of brain regions involved in the pathophysiology of this comorbidity, the authors also offer treatment recommendations. Deciphering the neuropathology of conversion of pre-clinical Alzheimer's disease (AD) to clinical AD may lead to the development of new therapeutic strategies. Thal et al. [2] compared clinical AD, non-demented preclinical AD and non-AD control cases with respect to neuropathological alterations. They defined clinical AD to exhibit late stages of neurofibrillary tangles, amyloid and neuritic plaque pathology. In contrast, preclinical AD displays early stages of these lesions, but early neurofibrillary tangles, granulovascular degeneration and amyloid angiopathy without amyloid plaques can also be detected in non-AD controls, representing a pre-amyloid plaque stage. Additionally, the presence of soluble/dispersible Aß aggregates in the neuropil plays a role in the conversion of preclinical AD to full AD.

The genetic background has an important influence on pathophysiological processes. In schizophrenia, heritability estimates amount from 60 to 80 %. Schwab and Wildenauer [3] update results from recent multistage genome-wide association studies, according to which a set of 8300 independent SNPs contribute to a schizophrenia liability of 32 %. Several of these genes have meanwhile been confirmed, such as ZNF804A, TCF4 (encoding a transcription factor), the MHC region, CACNA1C (encoding calcium channel, voltage dependent, L-type, alpha 1C subunit) and ITIH3-ITIH4 (encoding inter-alpha globulin inhibitors H3 and H4). Same applies to ANK3 (encoding ankyrin 3) and the MIR137, encoding a microRNA which is a regulator of neuronal development. Further studies combining larger samples with deep sequencing technologies will extend our knowledge on the genetic background of psychiatric disorders. Also, gene–environmental interactions have been deemed an important factor in the pathophysiology of schizophrenia. Malchow et al. [4] investigated the impact of familial load and cannabis abuse on volumes of subcortical brain regions in first-episode patients. Patients with a family history of schizophrenia combined with previous cannabis abuse showed smaller volumes of the bilateral caudate nucleus compared to all other patients, implicating an interaction between the genetic background and cannabis abuse as environmental factor. The identified brain region plays an important role in the corticothalamic-cortical circuit, and dysfunction of the caudate nucleus may lead to disinhibition of the thalamus in schizophrenia.

The neuropeptide oxytocin has been shown to be important for social cognition. Presenting direct versus averted gaze pictures to healthy probands, Montag et al. [5]

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in a fMRI study investigated the impact of the promoter region of the oxytocin receptor gene on individual differences in the recorded BOLD signal. A genetic variation of the gene modulated right amygdala activity with carriers of the heterozygous CT variant and showed higher activity than the TT group. In the literature, a pronounced amygdala activation has been observed in autistic individuals being confronted with direct gaze pictures in the fMRI setting. In adults with Asperger syndrome, Hermann et al. [6] detected intact automatic processing of novel metaphors. Van Elst et al. [7] review characteristics of autism spectrum disorder such as deficits in social cognition and competence as well as highly circumscribed interests and a strong desire for routines. The authors outline first treatment concepts for high-functioning autism that is associated with psychiatric diseases such as depression, anxiety, ADHD, psychotic symptoms or emotionally unstable syndromes. As a consequence of these symptoms, deficits in social cognition subsequently lead to social deficits and unemployment in high-functioning autism. Vogeley et al. [8] suggest to develop supported employment programs for these patients, comprising the adequate neuropsychological profile, adjustment to individual preferences plus workplace requirements, systematic communication and interaction training as well as instruction of colleagues and supervisors.

Besides psycho- and sociotherapy, in moderate physical aerobic exercise, first beneficial evidence for, e.g., increased hippocampus volume or improved cognition and negative symptoms, has been demonstrated recently. Bär et al. [9] review the prevalence of psychiatric diseases among elite athletes and examine the specific aspects of psychiatric disorders in the context of overtraining. Besides the amount of exercise, further research on other confounding environmental conditions in athletes is warranted.

Regrettably, we here could illustrate only a small selection of many highlights of last year's DGPPN Congress, and we hope that our readers will enjoy the widespread and multifaceted program of the upcoming Congress in November 2013 in Berlin.

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