the action of antipsychotic drugs. A common functional polymorphism (rs6295) in the promoter region of the human 5-HT1A receptor gene has been reported. This polymorphism may be useful in identifying psychopathology and phenotypic characteristics associated with altered function of the human 5-HT1A receptor.

The aim of this study was to determine whether genetic variants for these receptor influence the functional morphological characteristics of brain in schizophrenia.

63 patients with schizophrenia were genotyped for the functional variant in the promoter region of 5-HT1A receptor (rs6295) and for polymorphisms for 5-HT2A (rs6313) and serotonin transporter-SERT (rs4795541). The subjects were investigated by 18fluoro-deoxyglucose (18FDG) positron emission tomography (PET) in the resting state, magnetic resonance imaging (MR) and functional magnetic resonance (fMR) with 2-back test activation paradigm. Voxel-based-morphometry (VBM) was used to detect the differences in the density of grey and white matter. The neuroimaging data were treated by the use Statistical Parametric Mapping (SPM5) with genetic variants as the factor.

The polymorphism in 5-HT1A receptor was associated with the functional morphometric characteristics in cortical regions in projection areas of serotonergic system.

Our findings identify an important genetic factor predicting functional and structural characteristics in schizophrenia. Future research would test the role of HT1A polymorphism in the interaction with 5HT2A and SERT on morphological characteristics within the context of antipsychotic effects.

This research was supported by grant NR9324 (IGA MZCR) and by the project 1M0517 (MSMT).

S48.04

Serotonin-1A agonists as a cognitive enhancer in schizophrenia: Clinical evidence

T. Sumiyoshi [1], T. Uehara [1], T. Matsuoka [1], Y. Higuchi [1], T. Ito [1], H.Y. Meltzer [2]. [1] *Department of Neuropsychiatry, University of Toyama Graduate School of Medicine and Pharmaceutical Sciences, Toyama, Japan* [2] *Department of Psychiatry, Vanderbilt University Medical Center, Nashville, TN, USA*

Background and Aims: Postmortem and PET studies indicate increased serotonin (5-HT)-5-HT1A receptor density in frontal and temporal cortices in schizophrenia, suggesting up-regulation secondary to diminished 5-HT1A-receptor stimulation. We previously conducted a series of pilot studies of the effects of the addition of tandospirone, a 5-HT1A partial agonist and azapirone derivative, to ongoing treatment with small to moderate doses of typical antipsychotic drugs, on cognitive function in patients with schizophrenia. The addition of tandospirone (30 mg/day), but not placebo, for 4 to 6 weeks was found to improve executive function and verbal learning and memory.

Methods and Results: We have conducted a randomly-assigned placebo-controlled double-blind study to investigate the ability of the addition of buspirone to enhance cognitive function in subjects with schizophrenia treated with atypical antipsychotic drugs (AAPDs). Buspirone, 30 mg/day, outperformed placebo in improving the performance on a measure of attention/speeded motor performance and index of general cognitive function. The distinct cognition-enhancing ability of buspirone suggests its usefulness for patients who have large deficits in attention in spite of treatment with AAPDs.

Conclusions: The findings from these clinical studies indicate 5-HT1A receptors are a promising target for the management of psychotic symptoms and cognitive disturbances of schizophrenia. This concept has prompted the development of novel antipsychotic compounds with agonist actions at 5-HT1A receptors, e.g. F156063, SLV313, SSR181507, and bifeprunox. Evidence from basic studies with these drugs suggests an optimal balance of activity at 5-HT1A and dopamine-D2 receptors is required to gain cognitive benefits, which deserves further investigations.

Free Communications

FC02.01

New psychotherapeutic package for PTSD- patients without marked personality disorders

I.D. Note [1], E.M. Grosse [2]. [1] *Hôpital Sainte Marguerite, Marseille, France* [2] *Hôpital de la Conception, Marseille, France*

Cognitive models suggest that PTSD becomes persistent when individuals process the trauma in a way that leads to a sense of threat, which arises as a consequence of excessively negative appraisals of the trauma and its sequelae, and of a disturbance of autobiographical memory characterised by poor elaboration and contextualization, strong associative memory and strong conceptual priming. Nevertheless, despite adequacy and pertinence of the cognitive model of PTSD, recent neurobiological evidence shows that emotions can be experienced without cortical interpretations of stimuli, and clinical evidence indicates that experiences can be stored as isolated affective fragments that function later to distort cognition. This suggests that cognitive therapies are based on a limited model of mental functions that sometimes must be supplemented by broader approach, combined with classical cognitive therapy. EMDR for instance may be a specific treatment for non-cognitive driven and primary emotions, derived from direct activation of the amygdala. The actual impact of CBT on PTSD may be considered a result of the well known efficacy of those treatments on comorbid personality disorders or unipolar depression, which are often associated with PTSD. However, the usually high failure rate in treating PTSD along the lines of CBT may be due to its inefficacy on primary emotions, not-linked to cognitive dysfunction. Therefore, a combination of treatments targeting primary emotional disorders as well as secondary affective disorders, linked to cognitive distortions, may enhance efficacy of therapeutic management in PTSD.

FC02.02

PTSD Symptoms: Results of trauma or correlates of psychosocial characteristics?

B.S. Rosenthal [1], W.C. Wilson [2]. [1] *Social Sciences Department, City University of New York-York College, Jamaica, NY, USA* [2] *School of Social Work, Adelphi University, Garden City, NY, USA*

Background and Aims: Reports in recent literature regarding PTSD note that: 1) defining symptoms do not always accompany exposure to a potentially traumatic event and 2) symptoms are often observed in the absence of experience with traumatic events. The question arises: is manifesting PTSD symptoms principally a function of experiencing traumatic events or of unrelated psychosocial characteristics.

Methods: Data on three sets of variables [PTSD symptoms (Intrusive Experiences and Defensive Avoidance), potentially traumatic experiences (victim of assault, witness of assault, experiencing domestic violence, experiencing interpersonal loss, injured in an accident) and psychosocial characteristics (availability of adult confidant, sense of personal efficacy, emotional reactivity)] were collected by

https://doi.org/10.1016/j.eurpsy.2008.01.226 Published online by Cambridge University Press