SES09.3

Neurocognitive dimensions characterizing first-time psychotic natients

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Background: Assessment of neurocognitive dysfunction in schizophrenia is hampered by the multitude of diagnostic tools available and the ad-hoc use of concepts for cognitive impairments measured by the given set of neuropsychological tests.

Aims: To identify the main dimensions of a neurocognitive assessment battery for patients with first episode psychosis and to evaluate their relationship to sex, age, education, diagnosis and symptoms.

Methods: Eight neuropsychological tests were given to 219 patients with first episode psychosis three months after start of therapy or at remission, whichever occurred first.

Results: The factor analysis indicated five dimensions: Working Memory (WM), Verbal Learning (VL), Executive Function (EF), Impulsivity (Im) and Motor Speed (MS). The index scores were weakly related to sex, age, diagnosis and GAF symptom and function scores. The MS score was significantly higher for men than for women, and the WM and VL scores were significantly correlated with years of education.

Conclusion: The study indicates that the tests measure five fairly independent dimensions that were only weakly related to age, sex, diagnosis or symptoms.

SES09.4

Relationship between positive, negative and cognitive symptoms

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Objectives: Different syndromes have been described in psychosis. Among these are positive, negative, disorganization, manic and depressive. These may have different pathophysiological backgrounds and precipitating factors. The aim of this communication was to analyze the literature and contribute empirically in an attempt to clarify the relationships between the symptom profiles and neurocognitive factors.

Method: Factor analysis and other relevant studies were compared for consistency of findings. From an early intervention study for patients with first episode psychosis (the TIPS study) over 200 patients were examined using PANSS and neuropsychological tests. Factor analysis revealed 5 neurocognitive dimensions.

Results: There is an overall tendency that positive symptomathology seems not correlated with neurocognitive impairment, while disorganization and especially negative dyndrome is. Genetic studies suggest a genetic contribution to variation in disorganization, and to a lesser extent to negative and manic dimensions. A confounding factor is that side effects of traditional antipsychotics may give secondary negative symptoms.

Conclusions: Disorganization and negative syndrome seem correlated with neurocognitive impairment. Clinical and scientific implications will be discussed.

SES09.5

Mapping positive and negative symptoms using brain imaging

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The neurophysiological basis for positive and negative symptoms in patients with schizophrenic psychoses is still largely unknown. The brain imaging techniques MRI, fMRI and PET have introduced new possibilities to explore the localization of brain regions and systems involved in the manifestation of specific psychopathological phenomena, PET and fMRI recording of regional blood flow changes in patients with schizophrenia have demonstrated increased flow in superior and medial temporal cortex during the experience of auditory hallucinations. Reduced flow in prefrontal cortical regions has been reported in subjects with negative symptoms. Disorganized and paranoid symptoms appear to have more wide spread and diverse blood flow correlates. In general, few studies have been performed and most of them lack analyses of reliability and reproducibility. Molecular PET imaging of neuroreceptors and transporters using specific radioligands have so far demonstrated few correlations to psychopathological dimensions. However, PET studies of drug-induced dopamine release have demonstrated positive relationships to aspects of positive symptomalogy. More systematic studies in patients experiencing specific aspects of psychopathology should be performed with several imaging modalities in the same patient to explore specificity and reproducibility of changes observed.

S33. Psychological treatments in affective disorders: recent developments

Chairs: E.S. Paykel (GB), C.B. Pull (L)

S33.1

Cognitive therapy for bipolar disorders

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Background: The efficacy and effectiveness of brief psychological therapies such as cognitive therapy (CT) are well established for unipolar disorders. However, treatment studies have always excluded individuals with bipolar disorders, so relatively little is known about the utility of psychological approaches in this disorder. This paper explores the feasibility and efficacy of using CT as an adjunct to usual treatment in bipolar disorders by reviewing key studies on cognitive therapy (CT) in bipolar disorders and reporting on an MRC pilot study.

Results: There are only five published studies available, and the largest sample is only 69 subjects. Some of these studies have focussed on selected areas such as medication adherence or monitoring and management of the prodromal phase, whilst others have used a more traditional CT approach tackling key problems that make the individual vulnerable to relapse. Despite the small literature, the data suggest that approaches targeted only at prodromes are more effective in reducing manic or hypomanic relapses rather than depressive relapse. A more refined CT approach used in the MRC pilot study appeared to be beneficial in improving functioning, treating bipolar depressive symptoms and preventing full manic relapse.

Conclusions: The use of CT in subjects with bipolar disorders requires modifications, is more complex than in unipolar disorders

and requires a high level of therapist expertise. However, given the considerable efficacy-effectiveness gap observed with medication alone, CT and other evidence-based brief therapies have an important role to play in the treatment of individuals with bipolar disorder.

S33.2

Cognitive therapy in relapse prevention in unipolar depression

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This paper reports a study designed specifically to investigate relapse prevention by cognitive therapy. 158 subjects with residual depressive symptoms after antidepressant treatment for major depression were randomised to receive clinical management plus antidepressant alone, or with 20 sessions of cognitive therapy over 5 months with two booster sessions. Subjects were followed up and treatment controlled for a further year. Cognitive therapy significantly reduced relapses from 47% to 29%. Effects on remission were smaller and symptom ratings affected were predominantly those of negative cognitions. Social adjustment was improved. Extreme thinking in either negative or positive direction predicted later relapse and was modified by cognitive therapy. Costs of other treatment were reduced by cognitive therapy, but did not fully compensate for therapy costs, giving a net cost per relapse avoided. This study is now one of five controlled trials in which cognitive therapy after the acute episode has been found to reduce relapse or recurrence in depression.

S33.3

Cognitive behaviour therapy versus pharmacotherapy in unipolar depression

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Both, cognitive behaviour therapy and pharmacotherapies are well-established treatments of unipolar depression. There are several large-scale studies available which demonstrate the efficacy of each of these treatments in comparison to control conditions. Looking at the direct comparison of psychotherapy and pharmacotherapy does not yet show a clear picture. This presentation will discuss results of own research on this topic. In particular, I shall discuss the question of efficacy in relation to dropouts, to side effects, to short-term and long-term response rates, as well as the question of combination treatment. I'll also address the question of severity of depressive symptomatology and the appropriateness of cognitive behaviour therapy and/or pharmacotherapy. In addition, I'll present some recent approaches of cognitive behaviour therapy with inpatients, with minor depression, with older depressed patients, and cognitive behaviour therapy with depressed stroke patients.

S33.4

Interpersonal psychotherapy in depression

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Interpersonal Psychotherapy (IPT) was developed by Klerman, Weissman et al. for the treatment of depressive disorders, in association with, or without pharmacotherapy.

The procedures and strategies of IPT have been described in detail by its authors. As such, IPT may be considered a manual-based psychotherapy.

IPT is practical rather than theoretical It is based on the asssumption that depressed patients have difficulties in interpersonal relationships. The difficulties are identified as belonging to one out of four major problem areas, i.e. grief, life transitions, interpersonal disputes, interpersonal deficits. IPT is designed to help depressed patients to solve these problems and to improve their depression.

The authors will describe the basic principles underlying IPT and provide information on recent developments in IPT.

In addition, the authors intend to present recent data concerning controlled trials that have been completed to assess the efficacy of IPT in depression.

S34. Disturbances of the self construct in schizophrenia

Chairs: K. Vogeley (D), P. Falkai (D)

S34.1

Perceiving the self in schizophrenia: Abnormalities in the awareness of action

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It has been proposed that an impairment in 'self-monitoring' underlies certain symptoms associated with schizophrenia¹. Recently, the self-monitoring mechanism has been interpreted in terms of a forward model of the sensorimotor system2. Forward models predict the sensory feedback from self-produced movements, thereby enabling us to recognise the sensory consequences of our own actions. It is proposed that an impairment in this predictive mechanism might give rise to certain psychotic symptoms. If self-produced sensations are interpreted as being generated by an external source, then thoughts might be interpreted as external voices (auditory hallucinations) and self-produced movements might be interpreted as externally generated (delusions of control or passivity phenomena). Psychophysical studies have shown that self-produced tactile stimulation is perceived as less intense than externally produced stimulation, which might be due to the sensory predictions made by a forward model³. Functional neuroimaging studies have demonstrated that such perceptual attenuation is mediated by somatosensory cortex and the anterior cingulate cortex: these areas are activated less by a self-produced tactile stimulus than by an identical stimulus when it is externally produced⁴. Evidence suggests that the cerebellum is involved in generating the prediction of the sensory consequences of movement⁵. Furthermore, psychotic patients with auditory hallucinations and passivity phenomena show no perceptual attenuation of self-produced sensory stimulation⁶. This supports the proposal that these symptoms are associated with an impairment of the functioning of the forward model.

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