combination treatment arm and two monotherapy treatment arms with flexible dosage of oral amisulpride 400–800 mg/day and oral olanzapine 10–20 mg/day. Sample size was calculated to $n = 3 \times 101$, assuming an effect-size of 0.50 with a power of 90 % and a two-sided *t* test with significance level of 0.025.
Results: Recruiting for this trial has started June 2012. Up to January 17th, 2013 42 patients have been randomized. The end of the trial is expected to be in March 2015 (last patient out).
Conclusion: We expect results to enhance evidence for rational approaches towards antipsychotic combination treatment.
Policy of full disclosure: None.

S-07 Dimensional concepts of psychosis

S-07-001
Dimensions of psychotic symptoms and brain physiology

W. Strik (University Hospital of Psychiatry, University of Bern, Bern, Switzerland), Sebastian Walther, Alexander Wopfner, Katharina Stegmayer, Daniela Hubl, Helge Horn and Thomas Dierks

Abstract: Classical descriptions of schizophrenia are based on the concept of dissociation of higher brain functions, i.e. between Thinking, Feeling and Will (E. Bleuler). This implies a relative independence of the respective brain functions and, in a brain-physiological view, interacting but structurally and functionally distinct brain systems. The dimensional approach to psychotic symptoms is appealing to study such a concept, due to the often supposed continuity from symptoms to normal, and to the distinct symptom domains. In principle, dimensions allow quantifying the degree of deviation from normal, and to rigorously refer them to a respective normal brain function. In a systems-physiological view, normal brain functions can be understood as the emergent property of brain systems in a functional, homeostatic balance. In this perspective, deviations can be described as an imbalance between the inhibitory and the excitatory components of the respective circuitry, resulting in a dysfunctional inhibition or disinhibition of the output, along with a reduced reactivity to external inputs. Although a systematic dimensional approach has not been possible to be realized in DSM-5 due to insufficient data and practical reasons, in psychiatric neuroscience it is increasingly applied. Based on brain imaging results obtained with a dimensional approach to brain physiological changes during psychotic symptoms, evidence for the existence of three specific symptom dimensions related to the classical descriptions and to specific brain circuitries will be shown.
Policy of full disclosure: None.

S-07-002
Thought and language disorders in psychosis

T. Kircher (Philipps-University Marburg Department of Psychiatry, Marburg, Germany)

Abstracts: Speech and language disorders, such as concretism and formal thought disorder (FTD) are core symptoms of Schizophrenia. We will review clinical rating scales of FTD and introduce a new, validated scale. Further, brain imaging data will be reviewed, relating speech and language dysfunctions, such as FDS and concretism in Schizophrenia to neural networks. The impact of genetic variance and NNDA receptor blockage with Ketamine on brain activation will be reviewed with a particular stress on speech and language paradigms.
Policy of full disclosure: None.

S-07-003
Dysfunctions of the motor system in schizophrenia

S. Walther (University Bern, Dept. of Psychiatry, Bern, Switzerland)

Objective: Schizophrenia patients suffer from a variety of motor symptoms, including parkinsonism, catatonia, neurological soft signs, abnormal involuntary movements and psychomotor slowing.
Methods: Literature review of prevalence rates and presentation of own results.
Results: Parkinsonism and abnormal involuntary movements are intrinsic to schizophrenia, but may also be evoked by antipsychotic treatment. Reduced motor activity is associated with negative symptoms, catatonia and psychomotor slowing. Furthermore, 40 % of schizophrenia patients are impaired in gesture performance, which is related to executive and basic motor function. Mild motor disturbances are found in the majority of patients, while severe dysfunctions are limited to a minority. Our neuroimaging studies suggest that hypokinesia is caused by defective cortico-subcortical motor loops in schizophrenia. Taken together, a dimensional approach to schizophrenia motor symptoms seems promising. A purely descriptive assessment of motor signs is preferred over theory-laden categorization. Using objective motor parameters allows finding neural correlates of abnormal motor behaviour.
Conclusion: The motor dimension of schizophrenia is linked to distinct disturbances in the cerebral motor system. Targeted modification of the defective motor system might become a relevant treatment option in patients suffering from schizophrenia with predominant motor features.
Policy of full disclosure: None.

S-07-004
The limbic system and positive symptoms of schizophrenia

A. Heinz (Psychiatry and Psychotherapy Charité, Berlin, Germany), Schlagenhauf, Florian; Friedel, Eva; Hägele, Claudia

Abstract: It has long been suggested that dopaminergic dysfunction plays a key role in the pathogenesis of schizophrenia. Here, we describe empirical studies in animals and in humans that model dopaminergic neurotransmission as a signature for reward prediction errors and suggest that increased phasic dopamine release during acute psychosis attributes salience to otherwise irrelevant stimuli and can thus contribute to delusional mood. We show how this idea has originally been tested on incentive delay tasks, in which a conditioned, reward predicting cue, appears to be drowned in increasingly noisy ventral striatal neurotransmission. We further describe how reversal learning tasks can be applied to directly model reward prediction errors and linked to ventral striatal activation. This ventral striatal activation as a neuronal signature for reward prediction errors seems to be dysfunctional in unmedicated patients suffering from schizophrenia. However, it appears to be absolutely necessary to strictly model behavioural performance, because unmedicated patients with acute psychosis can fail to understand task structure and hence lack of functional activation does not necessarily reflect a biological impairment but rather simply results from not performing the task as required. The same is true for studies with medicated patients, where blockade of dopamine D2 receptors by neuroleptic medication profoundly alters ventral striatal activation in schizophrenia. Our studies show that ventral striatal dysfunction is a key aspect of acute psychosis, while altered prefrontal activation patterns appear to be associated with the severity of psychosis rather than schizophrenia per se.
Policy of full disclosure: None.