Spontaneous striatal activity reflects disease states and symptom dimensions in schizophrenia

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1. Introduction:
Striatal dysfunction is thought to be fundamental in schizophrenia1 and is assumed to underlie a variety of psychotic phenomena1,3: Striatal dopamine transmission is elevated in patients especially during prodromal and psychotic states and correlates with positive disease symptoms, which respond to anti-dopaminergic drugs. These increased dopamine levels have also been observed in schizophrenic patients at rest. Thus, spontaneous resting state neuronal activity in the striatum might be altered in patients with schizophrenia during both acute state of psychosis and state of remission and be associated with severity of symptoms. However, in-vivo evidence for altered spontaneous striatal activity in patients has not been reported yet.

2. Questions:
(1) Is spontaneous striatal activity changed in schizophrenia?
(2) Are potential changes modulated by psychosis, i.e. are potential changes in spontaneous striatal activity different during acute state of psychosis and during psychotic remission?
(3) Are potential changes related to symptom dimensions?

3. Methods:
Measurement: BOLD-fMRI during a 10-minute rest period (rest-fMRI). Participants: Patients with schizophrenia (n=21) and healthy controls (n=21). Patients were assessed during psychosis (SP, n=21) and during psychotic remission (SPR, remaining n=13) (see Tab. 1). Data analysis: Independent component analysis (ICA, GIFT-toolbox) was performed to identify the basal ganglia network (BGN). Outcome measures: (1) intensities of the BGNs spatial maps (connectivity / degree of coactivation), (2) power spectra of BGNs time course (level of coherent activity).

3.3. Spectral power of time courses of synchronous activity was changed during psychotic remission.

4. Results:
4.1. ICA revealed a basal ganglia network (BGN), which comprised the dorsal/associative and the ventral/limbic striatum.

4.2. Spatial maps of co-activation reflect psychosis and psychotic remission in distinct parts of the striatum.

4.3. Spectral power of time courses of synchronous activity was changed during psychotic remission.

5. Conclusion:
These data reveal spontaneous activity in the striatum as a neuronal mechanism reflecting disease states and symptom dimensions in schizophrenia. We suggest that the regional shift of increased spontaneous co-activity from ventral to dorsal striatum reflects both an elevated striatal dopamine function and the corresponding shift from non-psychotic flexible to psychotic habit-like behaviour.

6. References:

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