T147. Higher complexity of EEG signal as a state marker in first episode psychosis compared to individuals with at risk mental state for psychosis and healthy controls.

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Background: Psychosis disrupts the stability and self-regulation of brain systems. Non-linear analyses such as complexity estimators applied to electroencephalography (EEG) are particularly relevant to study chaotic neural activity during psychosis (Fernandez *et al.* 2013). Various studies in schizophrenia have shown that complexity estimators vary across stages of the disease. Importantly, several studies have shown increased complexity estimators in first episode psychosis (FEP) compared to healthy controls, thus indicating a higher level of chaotic activity in early psychosis (Fernandez *et al.* 2013). However, it is still unknown if increased complexity is a state marker of FEP or a trait marker of the risk of psychosis. In order to address this issue, we studied the complexity of EEG signal in patients with FEP, compared to individuals with at risk mental state for psychosis (ARMS) and healthy controls (HC).
Methods: Patients and HC were recruited within the Basel FePsy Study for the early detection of psychosis (Riecher-Rössler *et al.* 2009). We included 47 patients with FEP, 67 with ARMS and 29 HC using the FEPSY study criteria, based on the Basel Screening Instrument for Psychosis (BSIP) (Riecher-Rössler *et al.* 2008). Symptoms of psychosis were assessed by BPRS and SANS. All the participants underwent a 19 channels resting state EEG recording at their inclusion in the study. After EEG pre-processing, the complexity of EEG signal was estimated with the Lempel Ziv Complexity (LZC). The LZC is a non-parametric measure in a one-dimensional signal, which counts the number of distinct substrings and the rate of their recurrence before computing it in a Z-score. The LZC was compared with an ANOVA with the group as between factor and the electrode location as within factor.
Results: The analysis showed a main effect of group, indicating that LZC was similar in ARMS and HC but was higher in FEP. The analysis also showed a main effect of electrode location on LZC but no interaction between electrode and group on LZC. Correlation analysis revealed a significant correlation between LZC and age.
Discussion: To our knowledge, this is the first study measuring EEG complexity in people with ARMS in comparison with FEP and HC. We showed a similar EEG complexity in people with ARMS and HC, but an increased EEG complexity in FEP patients. This result can possibly be interpreted as a higher level of neural chaotic activity in FEP patients. There was no gradation of LZC across HC, ARMS and FEP, which could eliminate this measure as a risk marker for psychosis. Since only FEP patients elicit an increased LZC, such a pattern could rather be considered as a state marker, specific for psychosis in comparison with ARMS and HC.

T148. Mismatch negativity (MMN) in psychosis following traumatic brain injury (PFTBI)

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Background: Patients who develop psychotic symptoms following a traumatic brain injury (PFTBI) are relatively rare (i.e., ~10%). Circuitry implicated in the neurogenesis of psychotic symptoms overlaps with circuitry typically affected by traumatic brain injury (TBI), however the neurobiological precursors and/or processes in the development of PFTBI remain unknown. Auditory mismatch negativity (MMN) is an event-related potential that indexes the preattentive detection of changes in the acoustic environment. Although impoverished MMN has been shown across a range of disorders, MMN deficiency has recently been described as a "break-through biomarker" in the prediction of disease risk/symptom onset. We sought to determine whether PFTBI patients demonstrate impoverished MMN akin to patients with schizophrenia as further evidence for the MMN biomarker for psychosis. We expected that MMN amplitude would be significantly reduced in both psychotic cohorts relative to TBI patients without psychosis and non-clinical controls.
Methods: Duration (100ms vs 50ms standard) and frequency (1050 Hz vs 950 Hz standard) MMN was measured at frontal sites where the difference waveform was maximal (F3, FZ, F4) in: nine PFTBI patients, nine TBI patients without psychosis, 17 schizophrenia patients without TBI, and 17 nonclinical controls.
Results: PFTBI and schizophrenia patients demonstrated significantly reduced amplitudes to both duration and frequency MMN at all three electrode sites relative to nonclinical controls as expected. However, TBI patients demonstrated statistically comparable amplitudes to both the psychotic cohorts and to the nonclinical controls (with the exception of frequency amplitude at F3 where TBI amplitudes were significantly larger than in schizophrenia).
Discussion: This is the first empirical event-related potential study in dually-diagnosed PFTBI. In support of a biomarker for psychosis, impoverished MMN to both duration and frequency deviants is apparent in PFTBI, akin to schizophrenia. Although MMN amplitude in TBI patients without psychosis was not significantly different from psychotic patients on the majority of contrasts, we note that peak amplitude was largely variable in TBI, with the mean looking more like nonclinical controls. Future work should determine the variability of MMN amplitude in TBI according to injury profile/lesion location, and follow TBI patients with reduced MMN amplitude to determine risk for transition to psychosis.

T149. C9ORF72 Repeat expansions that cause frontotemporal dementia are detectable among patients with psychosis.

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Background: A pathologic hexanucleotide repeat expansion in the regulatory region of C9orf72 causes frontotemporal dementia (FTD) or amyotrophic lateral sclerosis (ALS). In addition to features of dementia, behavioral abnormalities can be present among mutation carriers. It is uncertain whether a proportion of individuals clinically diagnosed with psychoses bear pathologic C9orf72 expansions in the absence of dementia.
Methods: We screened for C9orf72 repeat expansions among individuals diagnosed with functional psychoses such as schizophrenia (SZ)/schizoaffective disorder (SZA), and described the clinical features of individuals with the mutation.
Results: Pathogenic C9orf72 repeat expansions were detected in two pairs of related individuals following a survey of 740 participants in a psychiatric genetic research study. The mutation carriers included two siblings with schizophrenia, another unrelated individual with schizoaffective disorder and her non-psychotic mother. All the mutation-bearing patients with SZ/SZA had severe, florid illness, but did not provide a history suggestive of dementia or ALS.
Discussion: In conclusion, a small proportion of patients with SZ/SZA could bear C9orf72 repeat expansions. The patients showed atypical psychotic features without the typical cognitive features of dementia.