such as d-amphetamine (AMPH) that increase cortical DA could improve cortical function in SZ subjects while impairing cortical function in HC. We examined the effect of AMPH in SZ and HC subjects during an auditory click-train paradigm, assessing EEG dynamical complexity to complement our prior investigations using traditional spectral measures.

Methods: Single dose AMPH was administered in a double-blind, placebo controlled cross-over fashion to 8 patients with schizophrenia and 8 matched controls performing the steady state auditory evoked potential (SSAEP) task. Sample Entropy was used as a measure of EEG dynamical complexity.

Results: During placebo, the SSAEPs task reduced EEG complexity in all participants, consistent with synchronous oscillations during the auditory stimulus, with the expected greater reductions in HC compared to SZ. However, this complexity reduction in HC was attenuated with AMPH, while schizophrenia patients appeared to have enhanced reductions with AMPH.

Conclusions: Using Sample Entropy as a measure of complexity in cortical activation, our findings provide preliminary evidence that increasing cortical dopamine may enhance reductions in complexity in schizophrenia while impairing such reductions in healthy subjects, consistent with an inverted-U shaped relationship between cortical activation and dopamine levels. Results will be discussed in terms of possible neurophysiologic effects of dopamine and therapeutic relevance for schizophrenia.

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801. Early Auditory Gamma Band Response in Healthy Controls and Individuals with Schizophrenia at First Hospitalization and 18 Month Follow Up

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Background: Deficits in the EEG early auditory gamma band response (EAGBR) have been noted in schizophrenia by some authors. Herein we examine the EAGBR at first hospitalization and at 18 month follow up in subjects with schizophrenia and controls.

Methods: 71 controls and 37 subjects with schizophrenia at first hospitalization underwent a P300 auditory paradigm with 61 channel EEG recording. Wavelet transformation with a complex Morlet provided the basis for evoked power and intertrial phase locking measures. The mean response from central sites in the 40-55 Hz, 70-90 ms, post stimulus interval was used for comparisons. 15 of the controls and 16 of the patients repeated the protocol at 18 months.

Results: Controls showed a trend towards higher intertrial phase locking and evoked power at time 1 though not significantly (p = 0.1). There was no significant change in the patient population from time 1 to time 2. There were no correlations between measures and clinical parameters (PANSS, total and subscales).

Conclusions: The trend level deficit in EAGBR while intriguing is non significant and consistent with variable reports of abnormalities in schizophrenia. Further analysis may clarify if it constitutes a neurophysiologic deficit in a subpopulation. There is no evidence for significant deterioration over 18 months early in the course of schizophrenia.

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Background: Emotional dysfunction is a critical clinical feature of schizophrenia, and there has been extensive work suggesting that the amygdala may play a critical role in this dysfunction. Neuroimaging studies have often demonstrated under-recruitment of the amygdala in response to negative emotional stimuli, but many studies have found intact recruitment or even greater activation of the amygdala by patients.

Methods: We conducted a meta-analysis of 35 neuroimaging studies investigating amygdala activation in patients with schizophrenia during a negative emotional manipulation. We used bootstrapping in combination with classic effect-size meta-analytic procedures to assess patient-control differences in amygdala activation and to evaluate a number of putative moderators of these between group differences.

Results: We demonstrate that patients with schizophrenia show significant (P=0.002) but modest under-recruitment of bilateral amygdala (mean effect=-0.22 SD). However, we also demonstrate that under-recruitment is dependent on the use of a neutral vs emotion interaction contrast and is not observed if patients and controls are compared in a negative emotional condition only (P=0.009, FDR corrected).

Conclusions: These findings demonstrate that patients with schizophrenia show a reduction in amygdala activation relative to control participants when a neutral vs emotion interaction contrast is used to compare activation between groups. However, given that patients do not exhibit underactivation of the amygdala when compared to control participants in a negative emotional condition directly, our findings suggest that amygdalar processing of negative emotional stimuli is intact—rather, patients exhibit abnormal overactivation of the amygdala in response to affectively neutral stimuli.

803. Neural Circuitry and Social Cognition in First Onset Schizophrenia

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Background: Schizophrenia is characterized by difficulties in social and emotional function. In this study we assessed whether emotion processing is impaired from the first onset schizophrenia. We tested whether cognitive behavioral measures of emotion processing relate to functional outcomes on the one hand, and to deficits in neural circuitry on the other.

Methods: First onset patients (n=61) and matched healthy controls were assessed with a standardized set of protocols (1). Clinical testing included a structured diagnostic interview and PANSS symptoms ratings. Cognitive testing provided accuracy and reaction time for facial emotion identification. Gamma synchrony was quantified from EEG recordings taken during a facial emotion processing task, with both masked (nonconscious) and unmasked (conscious) conditions. For functional outcomes we assessed social functioning, quality of life and negativity bias.

Results: At first onset, schizophrenia patients were distinguished by poor...