Only a few studies examined the olfactory identification ability in adolescents at-risk for schizophrenia and suggested smell identification deficits as a risk marker for schizophrenia. These studies included adolescents at clinical as well as at genetic risk for schizophrenia. None of these studies focused on children at genetic risk for schizophrenia. Therefore, we investigated the olfactory identification ability in children of parents with schizophrenia in comparison to children of parents without a psychotic disorder. As we are also interested in the specificity of the olfactory impairments to schizophrenia, we included children of parents with bipolar disorder. We hypothesize that children at genetic risk for schizophrenia would have the most severe smell identification deficits and that children of bipolar disorder patients would have less severe deficits than the at-risk for schizophrenia group but more severe than the group of children without a psychotic parent.
Methods: Participants - The olfactory identification ability was assessed in 202 children of schizophrenia patients ('children at familial risk for schizophrenia') in relation to that of 200 children of parents without a psychotic disorder ('controls'). In addition, we also assessed the B-SIT in 120 children of bipolar disorder patients ('children at familial risk for bipolar disorder'). All children were 7 years of age at the time of assessment and they were part of the Danish High Risk and Resilience Study – VIA7.
Brief Smell Identification Test - The Brief Smell Identification Test (B-SIT) contains 12 items that need to be scratched and sniffed. The test has excellent reliability (> 0.80) and demonstrates agreement for abnormal olfaction comparing B-SIT with the San Diego Odor Identification Test (SDOIT). A maximum score of 12 reflects intact olfactory identification functioning. B-SIT has been conducted in patients with neurodegenerative disorders (Parkinson's disease and Alzheimer's disease) and can be used for individuals above 5 years of age.
Statistics - We will use analysis of covariance (ANCOVA) for analysis of the B-SIT total scores with 'diagnosis of parent' as the independent variable and age and sex as covariates for the three groups.
Results: Analyses will be performed within the next 3 months so can be presented in April 2018.
Discussion: Conclusion and discussion cannot be drawn at this time.

F206. A TRANSLATIONAL STUDY OF BEHAVIOR, BRAIN STRUCTURE AND GENE PATHWAY IN ERBB4 KNOCKOUT MICE AND FIRST-EPISODE TREATMENT-NAÏVE PATIENTS WITH SCHIZOPHRENIA

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Background: The current study was to explore how disruption of specific molecular circuits in the cerebral cortex may cause large-scale brain structure deficits and behavior changes via a translational study in conditional Erbb4 mutant mice and patients with schizophrenia.
Methods: We conducted prepulse inhibition (PPI) and brain structural and diffusion magnetic resonance imaging (MRI) scans in 27 mice with ErbB4 knockout in parvalbumin (PV) interneurons and 23 age, sex-matched controls. Real-time quantitative polymerase chain reaction was used to assess the levels of five GABA-related transcripts in brain regions. We also measured structural and diffusion MRI and cumulative contribution of risk alleles in the GABA pathway genes using polygenic risk scores (PRS) in first-episode treatment-naïve schizophrenic patients (N=117) and age, sex-matched healthy controls (N=86).
Results: ErbB4 knockout mice displayed behavioral deficit of PPI, as well as gray and white matter impairment in right sensorimotor cortical-striatal networks. We found significant correlations between gray matter volumes (GMVs) of the somatosensory cortex and PPI as well as GAD1 mRNA expression in controls but not in knockout mice. These findings were confirmed in a human sample where we observed significantly decreased gray and white matter impairment in sensorimotor cortical-striatal networks in schizophrenics. The PRS of GABA-pathway genes also displayed a negative correlation with the GMVs of the somatosensory cortex in patients.
Discussion: Our study identified ErbB4 ablation induced prepulse inhibition deficits and GABAergic dysregulation in sensorimotor cortical-lateral striatal networks. We propose that ErbB4 signaling participates in sensorimotor gating dysfunction in schizophrenia by getting involved in somatosensory cortex deficits and GABAergic dysfunction.

F207. SCHIZOTYPY AND SENSORY GATING: A 6-MONTH-OLD EEG STUDY

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Background: Schizotypal traits are present in the general population and are distributed along a continuum, with the clinical disorder schizophrenia found at its extremity (Claridge, 1997). Schizotypy is a dimension of personality within the general population, which has been found to be elevated among schizophrenia-spectrum patients (Brosey and Woodward, 2015) and their first-degree relatives (Morenzo-Izco et al., 2015). One hypothesis to account for the sensory deficits observed across the spectrum suggests a difficulty in the inhibition of irrelevant sensory input, such as the secondary beep in the paired-click paradigm.
Sensory gating describes the pre-attentional habituation of responses to repeated sensory input, for example, auditory tones. This gating mechanism is used to distinguish between important and irrelevant information (Hall et al., 2011) and is typically explored using the paired-click paradigm and analysed using the P50 event-related potential component. This can be observed approximately 50-milliseconds following the presentation of an auditory stimulus and is a highly established biological trait of schizophrenia, with abnormalities displayed in the P50 component all throughout the schizophrenia-spectrum.
Methods: This research aimed to observe whether the 6-month-old offspring of mothers with schizotypic traits display abnormalities in the P50 event-related component when explored using the paired-click paradigm. The paired-click paradigm was used to highlight the sensory-gating abilities of fifty-three 6-month-old infants during 15-minutes of continuous sleep. The mothers of the infants completed the Short Form of the Oxford and Liverpool Inventory of Feelings and Experiences, which was used to determine their personality dimension scores, and identify schizotypic traits. Participants were categorized into one of three groups: infants of controls mothers, infants of intermediate mothers, and infants of schizotypic mothers.
Results: It was predicted that the 6-month-old infants of mothers who demonstrate schizotypy scores would demonstrate different amplitudes compared to those of control mothers. This research found a significant generalized difference between the P50 component for the paired-clicks in the right hemisphere of the brain ($F(1,51)=5.34$, $p=.025$), and a significant latency effect was observed in the frontal regions ($F(1,51)=5.41$, $p=.024$). A significant between-subjects effect was observed centrally ($F(2,50)=3.71$, $p=.031$); suggesting there are significant differences between the ways each group distinguished the paired-clicks. Infants of schizotypic mothers showed an increase in activation compared to other groups. An interaction was observed in the left hemisphere between the paired-clicks and each identifiable group ($F(2,50) = 3.45$, $p = .039$). In addition to the P50 a significant slow wave effect was also observed across the left ($F(1,51)=8.38$, $p=.006$) and right ($F(1,51)=7.81$, $p=.007$) posterior regions; a latency effect in the left ($F(1,51)=5.47$, $p=.023$), and a distinction in mean amplitude in the right ($F(1,51)=7.25$, $p=.010$).
Discussion: Schizotypy is viewed as a risk factor for schizophrenia, which is present in the general population, and is present on the schizophrenia-spectrum. The 6-month-old infants of mothers showed an increase in activity centrally, demonstrating that the infants' P50 amplitudes were influenced by their mothers' schizotypy status. This finding is consistent with the developmental hypothesis of

schizophrenia, however longitudinal studies will be required to determine whether a sensory gating deficit is a valid predictor of the later onset of schizophrenia.

F208. COGNITION, POSITIVE SYMPTOMS, AND INTERNET USE FOR MENTAL HEALTH IN PEOPLE WITH PSYCHOSIS

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Background: People with severe mental illness are increasingly using digital resources for mental health, including social media and online interventions. However, individuals' ability to engage with or benefit from such resources may be impaired by deficits in cognition and insight, and experiences of psychotic symptoms, including paranoia about cyber-security or motives of others in online social interactions. This study aimed to explore the association between cognition, positive symptoms, and internet use for mental health information in adults with psychosis.
Methods: This study used baseline data collected as part of a broader research program investigating a digital recovery-focused intervention for psychosis. Participants completed a questionnaire on their existing internet use, both in general and for mental health information, and a range of cognitive and functioning measures. Cognitive variables included premorbid IQ, estimated using the Wechsler Test of Adult Reading, and composite scores for processing speed, working memory, and executive functioning. The Positive and Negative Syndrome Scale was also administered, with five items used to examine the relationship between mental health-related internet use and psychopathology: Delusions, Grandiosity, Suspiciousness & Persecution, Unusual Thought Content, and Lack of Judgment & Insight. Logistic regressions were used to identify unique predictors of internet use for mental health information, controlling for age and frequency of general internet use.
Results: 179 adults with psychosis (mean age = 39.82 years; range = 18–65; SD = 11.0) took part in this study, of whom 157 (87.7%) were regular internet users. Of these, 107 (68.2%) reported regularly using the internet for mental health information, with 33 (20.9%) doing so daily, 28 (17.7%) weekly, and 46 (29.3%) monthly or less. General websites were most commonly used for this purpose (n = 92; 58.6%), followed by video streaming sites (n = 62; 39.5%), social networking sites (n = 52; 33.2%), and forums (n = 34; 21.7%). When age and frequency of general internet use were controlled for, use of any type of website for mental health information was predicted by lower scores on Grandiosity (Exp(B) = .675, 95% CI = .513, .886, p = .005); mental health-related social media use was significantly predicted by lower estimated premorbid IQ (Exp(B) = .964, 95% CI = .937, .991, p = .010); lower scores on Unusual Thought Content predicted use of both video networking sites (Exp(B) = .629, 95% CI = .403, .981, p = 041) and forums (Exp(B) = .576, 95% CI = .379, .876, p = .010) for mental health information; while use of general websites for mental health information was not uniquely predicted by any cognitive or symptom variables.
Discussion: While internet use for mental health information is now common among people with severe mental illness, the presence of psychotic symptoms may inhibit such information-seeking behaviour, particularly when using interactive websites such as video streaming sites and forums. Cognitive functioning may also affect how online sources of mental health information are selected. However, using general websites for mental health information is common regardless of cognition and symptom severity, with implications for how such resources should be designed.

F209. TRANSCRANIAL DIRECT CURRENT STIMULATION (TDCS) IN A NON-CLINICAL POPULATION AS A MODEL FOR TREATMENT OF AUDITORY VERBAL HALLUCINATIONS IN SCHIZOPHRENIA

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Background: We used transcranial direct current stimulation (tDCS) in a non-clinical population to simulate a model of tDCS-treatment for auditory verbal hallucinations (AVH) in schizophrenia. In tDCS, a low current is induced via two electrodes attached to the scalp. The anode and cathode typically up- and downregulate neuronal activity, respectively. It was suggested that AVH arise due to two main neuronal pathways: hyper-activity in the language areas in the temporo-parietal cortex and hypo-activity of the cognitive control areas in the dorsolateral prefrontal cortex. Accordingly, it was further hypothesized that by reducing activity in the temporo-parietal cortex through cathodal tDCS and simultaneously increasing neuronal activity in the dorsolateral prefrontal cortex with anodal tDCS, AVH could be reduced. Patients with schizophrenia, particularly those with AVH show additionally deficits on language and cognitive control tasks, which are known to draw on temporo-parietal and dorsolateral prefrontal cortex regions, respectively. In order to test the model we thus reversed the electrode montage in non-clinical participants and tested whether they would show similar deficits as schizophrenia patients. In addition, the healthy participants underwent magnetic resonance spectroscopy (MRS) to test whether, in accordance with the model, glutamate levels increase under the anode, and decrease under the cathode area.
Methods: Eighteen participants were recruited in a convenience sample (7 male/ 11 female) with a mean age of 26 years. They were tested twice with a mean interval of 8 days. In one session they received real 2mA tDCS for 20 min, while in an MRI scanner (GE 750, 3T). The other session was a sham stimulation control. The order of real/sham stimulation was counterbalanced and stimulation was double-blind. In each session, MRS was measured using a PRESS sequence (TE=35ms, 1500ms) before and after stimulation. MRS data were acquired from two voxels, one in the left dorsolateral prefrontal cortex (22ml) and one in the left temporo parietal cortex (25ml), right underneath anode and cathode electrodes, respectively. MRS data were analyzed using LCModel software; water-scaled estimates for glutamate and glutamine combined (Glx) are reported herein, with N-Acetylaspartate (NAA) and creatine (Cre) inspected to ensure stability of the Glx measure. Glx levels were subjected to a 2x2x2 ANOVA with the within-participant factors Stimulation (real vs sham), Stimulation area (dorsolateral prefrontal cortex versus temporo-parietal cortex), and Time (Pre and Post stimulation).
Results: Two datasets where excluded from analysis due to poor spectral quality. As expected, NAA (F1,16=.809, p=.382) and Cre (F1,16=.005, p=.944) did not show significant changes. There was a trend for Glx to be higher during real as compared to sham stimulation (main effect Stimulation F1,16=3.867, p=.067) and for Glx to be higher after than before stimulation (main effect Time F1,16=1.396, p=.078).
Discussion: Glx was increased during real compared to sham tDCS, and before compared to after stimulation. This could indicate that tDCS overall changes neuronal firing thresholds. However, we did not observe the expected three-way interaction of reduced glutamate levels in the dorsolateral prefrontal cortex and increased glutamate levels in the temporo-parietal cortex. This could be due to the relatively small sample. However, the present data analysis is preliminary and we aim to report findings for a larger dataset.