a person's HL and clinical outcomes. So far, there are no reviews, which investigate HL in individuals at-risk for psychosis. The aim of the current review is to assess how individuals at risk of developing a first episode of psychosis gain access to, understand, evaluate and apply risk-related health information.
Methods: A mixed-methods approach was used to analyze and synthesize a variety of study types including qualitative and quantitative studies. Search strategy, screening and data selection have been carried out according to the PRISMA criteria. The systematic search was applied on peer-reviewed literature in PUBMED, Cochrane Library, PsycINFO and Web of Science. Studies were included if participants met clinical high risk criteria (CHR), including the basic symptom criterion (BS) and the ultra-high risk (UHR) criteria. The UHR criteria comprise the attenuated psychotic symptom criterion (APS), the brief limited psychotic symptom criterion (BLIPS) and the genetic risk and functional decline criterion (GRDP) Furthermore, studies must have used validated HL measures or any operationalization of the HL's subdimensions (access, understanding, appraisal, decision-making or action) as a primary outcome. A third inclusion criterion comprised that the concept of HL or one of the four dimensions was mentioned in title or abstract. Data extraction and synthesis was implemented according to existing recommendations for appraising evidence from different study types. The quality of the included studies was evaluated and related to the study results.
Results: The search string returned 10587 papers. After data extraction 15 quantitative as well as 4 qualitative studies and 3 reviews were included. The Quality assessment evaluated 12 publications as "good", 9 as "fair" and one paper as "poor". Only one of the studies assessed HL with as primary outcome. In the other studies, the five different subdimensions of HL were investigated as a secondary outcome respectively mentioned in the paper. "Gaining Access" was examined in 18 of the 22 studies. "Understanding" has been assessed in 7 publications. "Appraise" was examined in 9 studies. "Apply decision making" and "Apply health behavior" were investigated in 1 of 8 studies. Since none of the included publications operationalized neither HL nor the subdimensions of HL with a validated measure, no explicit influencing factors could be found.
Discussion: Quantitative and qualitative evidence indicates that subjects at-risk for psychosis describe a lack of understanding about their state and fear stigmatization that might lead to dysfunctional coping strategies, such as ignoring and hiding symptoms. Affected subjects are eager to be informed about their condition and describe favoured channels for obtaining information. The internet, family members, school personnel and GP's play a crucial role in gain access to, understand, evaluate and apply risk-related health information. The results clearly highlight that more research should be dedicated to HL in individuals at risk of developing a psychosis. Further studies should explore the relation between HL and clinical outcomes in this target population by assessing the underlining constructs with validated tools.

F168. PSYCHOTIC EXPERIENCE AND ADOLESCENT BRAIN TRAJECTORY: EVIDENCE FOR STRUCTURAL ALTERATIONS IN DOPAMINERGIC REGIONS

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Background: Psychotic Experiences (PE) are often reported by children and adolescents and have a bidirectional association with mental disorders, both increasing subsequent risk for mental disorders and being more frequently reported among subjects with a current psychiatric diagnosis. The brain developmental trajectories associated with PE in children and adolescents and how PE may evolve to a full blown mental disorder remain largely unknown. We assessed PE effect on subcortical and cortical measures over a 3-year follow-up in a community cohort of children and adolescents.
Methods: This study is part of the Brazilian High-risk Cohort, a multi-site longitudinal study. A total of 2,512 youths (6–12 years old, mean age at baseline 9.7 years, SD = 1.92; 53,1% male) completed the baseline assessment and 2,012, the 3-year follow-up (T1). PE were assessed at the two timepoints with The Community Assessment of Psychic Experience (CAPE). A confirmatory factor analysis (CFA) was used to generate a latent score for PE for baseline and follow-up, yielding good model fits.
A subset of the sample (n=809) was scanned in a 1.5T scanner on either baseline or follow-up resulting in 1183 MRI scans. Structural images were processed using Freesurfer. Subcortical volumes for the amygdala, hippocampus, caudate, putamen and pallidum were entered as a dependent variable in a linear mixed effects model (lme) with age, sex, CAPE and CAPE by age interaction as fixed effects and site and subject as random effects. The same model was applied in a mass-univariate analysis for cortical thickness measure (CT). A smoothing kernel (FWHM = 10 mm) was applied to CT before statistical testing. False Discovery Rate was used to control for multiple comparisons in the mass-univariate analysis with a $p<0.05$ threshold.
Results: CAPE was significantly related to the right putamen (Beta: -0.30 p:0.03), right caudate (Beta: -0.32 p: 0.03) and left caudate (Beta: -0.32 p:0.02). The age by CAPE interaction was significant for the three regions (right putamen Beta: 0.001 p:0.04, right caudate Beta:0.002 p: 0.03 and left caudate Beta: 0.001 p:0.03).
CAPE and CAPE by age interaction terms showed no effect on cortical thickness after correction for multiple comparisons.
Discussion: PE report was associated with lower subcortical volumes of the caudate and putamen, regions of the striatum. The striatum receives important dopaminergic neurotransmission and have been previously implicated in the pathogenesis of schizophrenia. In line with the hypothesis in schizophrenia, the PE experienced by children and adolescents may relate to a dopaminergic imbalance, possibly due to developmental structure alterations of the striatum and its connections. Interestingly, neither the hippocampus nor the amygdala were related to PE. Taken together with the lack of findings related to cortical thickness, the results presented here suggests a probable dopaminergic role on PE in young people with no current psychotic disorder.

F169. BRAIN CONNECTIVITY DURING PSYCHOLOGICAL STRESS IN PATIENTS WITH SCHIZOPHRENIA

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Background: It is commonly accepted that in most patients with schizophrenia external factors act on genetic predisposition to produce active psychotic symptoms. In fact, we showed that patients with schizophrenia have an abnormal brain activation and peripheral autonomic response to psychological stress. We sought to characterize the brain connectivity networks of such response in schizophrenia.
Methods: We studied the pattern of brain connectivity in relation to mental arithmetic stress paradigm in 21 patients and 21 healthy subjects aged 18 to 50 years, using 3T-fMRI. A period of 6 minutes of resting state acquisition (PRE) were followed by a block design with three 1-minute CONTROL task (one digit sum), 1 minute STRESS task (two digit subtraction) and 1 minute rest after task (POST). Pairwise Pearson correlations were calculated between 90 regions of interest. Data were analyzed with MATLAB and SPSS software.
Results: Patients with schizophrenia showed a lower connectivity network between fronto-temporal limbic areas compared with control subjects during control and stress task.

Moreover, we observed a great variability of link density during resting state in patients but not in controls, and it diminishes in response to task.
Discussion: Patients present abnormalities in networks related to stress response showing an alteration in fronto-temporal connectivity, and a poor and random modulation of these networks at rest. Current and previous findings suggest abnormal fronto-temporal connectivity that ultimately would lead to psychotic symptoms emergency in response to an environmental stressor and, even, could be related to hypervigilance and misattribution feeding into the paranoid cognition characteristic of patients with schizophrenia.

F170. SCHIZOPHRENIA POLYGENIC RISK SCORE ASSOCIATED WITH LEFT TEMPORAL GYRIFICATION

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Background: Brain structural changes in schizophrenia are thought to arise in part from genetic liability, as shown in studies of twins and siblings. Polygenic risk scores (PGRS) derived from large-scale genome-wide association studies (GWAS) have allowed to use measures of genetic liability calculated from large numbers of individual single nucleotide polymorphisms (SNPs). Initial studies on PGRS and structural imaging have, however, failed to provide clear associations. We used three separate measures of brain morphometry (voxel-based morphometry, cortical thickness, and gyrification) in a sample of healthy subjects to associate them with PGRS for schizophrenia in order to test the hypothesis that gyrification, a putative indicator of early brain development.
Methods: We analysed high-resolution MRI scans (3 Tesla, T1-weighted MPRAGE, 1x1x1mm resolution) from n=153 healthy subjects with not current or previous psychiatric condition recruited from the local community. DNA from each subject was analysed using the PsychChip, and polygenic risk scores were calculated for schizophrenia, as well as bipolar disorder and major depression (for assessment of relative specificity of the schizophrenia PGRS). MRI data were pre-processed with the CAT12 toolbox (dbm.neuro.uni-jena.de/cat12) for analysis using a) voxel-based morphometry (VBM), b) cortical thickness, and c) gyrification (calculated using the absolute mean curvature approach (Luders et al., NeuroImage 2006). We initially used $p<0.001$ uncorr. on the peak-level and performed correction for multiple-comparisons on the cluster level.
Results: We found a negative correlation of the schizophrenia polygenic risk score with gyrification in the left anterior superior cortex (i.e. the higher risk score loading the lower local gyrification), which was significant at the cluster-level for FWE correction ($p<0.047$). There was not such significant finding for positive correlations, nor for any of the VBM or cortical thickness analyses. Also, there was not significant association (positive or negative) with major depression or bipolar disorder PGRS in any of the three morphometry analyses.
Discussion: Our findings suggest that SNP-based genetic risk for schizophrenia is associated with left temporal gyrification, a putative indicator of early brain development, which again might be affected by multiple schizophrenia risk genes regulating cortical formation and connectivity. Furthermore, our findings are consistent with the notion of specificity for both morphometric marker (i.e. gyrification, but not VBM or cortical thickness) as well as diagnosis (with negative findings for major depressive and bipolar disorder risk scores). PGRS might impact on early developmental markers of brain structure (and possibly function), rather than overall liability-related variance.

F171. ALTERED DIFFUSIVITY IN THE BRAIN OF PATIENTS WITH SCHIZOPHRENIA: A DIFFUSION WEIGHTED MAGNETIC RESONANCE IMAGING STUDIES WITH PUBLIC NEUROIMAGING DATA

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Background: In recent decades, numerous in vivo brain imaging studies utilizing diffusion weighted MRI (dMRI) technique have focused on altered diffusivity in brains of patients with schizophrenia. However, the literature has not reached at consistent consensus despite a few interesting and promising results. In this study, we investigated whether or not various measures of dMRI (FA, AD, RD, and TR) are altered in patients with schizophrenia by comparing them in both patients and healthy controls with public neuroimaging data from SchizConnect (http://schizconnect.org).
Methods: The final data set was consisted of 121 schizophrenia patients and 119 healthy controls. After verifying 161 anatomical regions of interest (ROIs), we estimated the mean value and standard deviation of fractional anisotropy (FA), axial diffusivity (AD), radial diffusivity (RD), and trace (TR) in each ROI among the healthy controls. After that, we calculated the Z-score of each single ROI in every individual brain of both patients and healthy controls. The Z-score information of each person is then integrated into two location-independent measures. One is the total number of "abnormal" lesions, in which the absolute Z-score is above the cut-off value estimated by the Bonferroni correction, and the other is the largest absolute Z-score. After all, by using Welch two-sample t-test, we compared these two measures between the groups of patients and healthy controls.
Results: The number of abnormal lesions was notably increased in patients group, in terms of RD ($p=0.01063$) and TR ($p=0.009329$). Meanwhile, no statistically significant differences related to FA and AD were observed. On the other hand, it was found that the largest absolute Z-score was elevated in patients group, in terms of AD ($p=0.03371$), RD ($p=0.0001762$), and TR ($p<0.00001$). Otherwise, no significant differences related to FA were observed.
Discussion: In this study, we found a few remarkable differences of familiar measures, especially TR, between brains of patients with schizophrenia and healthy controls. This suggests that there should be some subtle changes in the brains of patients with schizophrenia, including microstructural destruction.

F172. INDIVIDUAL PREDICTION OF RISK IN ADOLESCENT OFFSPRING OF PARENTS WITH SCHIZOPHRENIA OR BIPOLAR DISORDER: A MACHINE LEARNING NEUROIMAGING STUDY WITH A CROSS-STAGE VALIDATION

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Background: Schizophrenia (SZ) and bipolar disorder (BD) are severe psychiatric disorders that are not easily distinguishable based on clinical measures. Offspring of patients with SZ or BD have a tenfold increased risk of developing the disorder as well as an increased risk for other severe mental disorders. Reliable identification of these subjects might allow for early recognition and intervention, which have been shown to be beneficial for