Methods: We conducted an exhaustive PubMed literature search followed by double-screening, double-extraction, and cross-checking to identify all whole-brain, case-control fMRI activation studies of mood, anxiety, and anxiety-related disorders in order to construct a large-scale meta-analytic database of primary studies of these disorders. We then employed multilevel kernel density analysis (MKDA) with Monte-Carlo simulations to correct for multiple comparisons as well as ensemble thresholding to reduce cluster size bias to analyze primary fMRI studies of mood, anxiety, and anxiety-related disorders followed by application of triple subtraction techniques and a second-order analysis to elucidate the disorder-specificity of the previously identified neural features.
Results: We found that participants diagnosed with mood, anxiety, and anxiety-related disorders exhibited statistically significant ($p < .05 – 0.0001$; FWE-corrected) differences in neural activation relative to healthy controls throughout the cerebral cortex, limbic system, and basal ganglia. In addition, each of these psychiatric disorders exhibited a particular profile of neural features that ranged from disorder-specific, to category-specific, to transdiagnostic.
Conclusions: These findings indicate that psychiatric disorders exhibit a complex profile of neural features that vary in their disorder-specificity and can be detected with large-scale fMRI meta-analytic techniques. This approach has potential to fundamentally transform neuroimaging investigations of clinical disorders by providing a novel procedure for establishing disorder-specificity of observed results, which can be then used to advance our understanding of individual disorders as well as broader nosological issues related to diagnosis and classification of psychiatric disorders.

Disclosure of Interest: None Declared

EPP0873

Increased and decreased cortical thickness and unaltered amygdala nuclei in patients at clinical-high risk of psychosis

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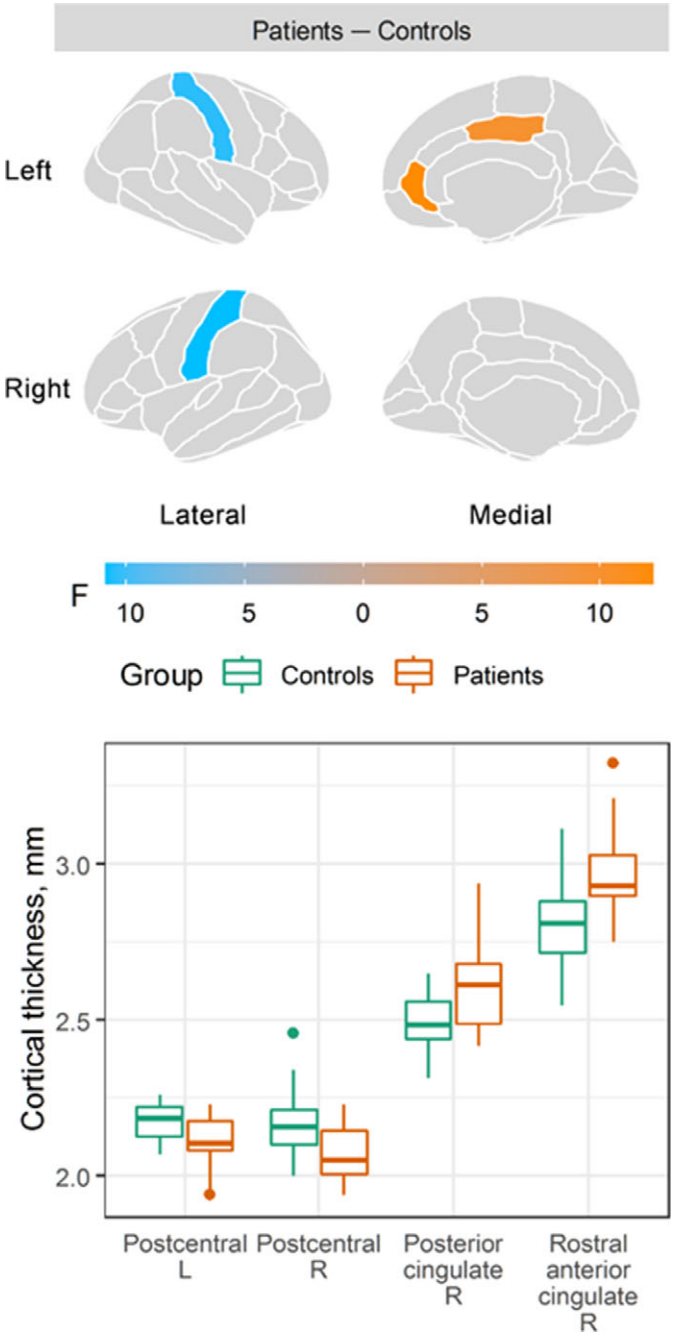
Introduction: There is much evidence of grey matter alterations in subjects at clinical-high risk of psychosis (CHR). Although, to the best of our knowledge, no studies have analyzed both cerebral cortex and amygdala nuclei morphometry alterations in CHR individuals.
Objectives: The aim of the study was to explore cortical thickness and amygdala nuclei morphometric characteristics in CHR patients.
Methods: Nineteen right-handed male patients (17-24 years, mean age 21.1 ± 2.1) fulfilling CHR criteria and 20 matched healthy controls (18-24 years, mean age 21.1 ± 1.8) underwent T1-weighted structural MRI at 3T Philips scanner. Images were processed via FreeSurfer 7.0. Cortical thickness (according to Desikan atlas) and volumes of 9 separate amygdala nuclei bilaterally were compared between groups. The morphometry data, SOPS, HDRS (Hamilton Depression Rating Scale) scores and chlorpromazine equivalents were included in correlation analysis.
Results: Compared to healthy controls, patients showed decreased cortical thickness in the left [$F(1, 36) = 10.8$, $p = 0.002$; Cohen's $d = -1.1$, 95% CI: −1.8 to −0.4] and right [$F(1, 36) = 10.5$, $p = 0.003$; Cohen's $d = -1.0$, 95% CI: −1.7 to −0.3] postcentral gyri, and increased cortical thickness in the right posterior cingulate [$F(1, 36) = 9.9$, $p = 0.003$; Cohen's $d = 1.0$, 95% CI: 0.3 to 1.6] and the right rostral anterior cingulate gyri [$F(1, 36) = 12.2$, $p = 0.001$; Cohen's $d = 1.1$, 95% CI: 0.4 to 1.8]. No changes in any amygdala nuclei were detected. No correlations between altered cortical thickness, HDRS, SOPS or chlorpromazine equivalents were revealed.
Image:



Conclusions: The current findings suggest that volumetric characteristics of amygdalar complex are unaffected in the CHR state. The results have some inconsistency with our previous findings (Tomyshev *et al*. Psychiatry Res Neuroimaging. 2019; 289 26-36), which revealed only a decrease in cortical thickness in CHR individuals. However, the cross-sectional design of the current study and the lack of correlations between cortical thickness and clinical symptoms do not allow to conclude definitely whether the revealed higher cortical thickness can represents some resilience mechanisms, which will be elucidated via further research.
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EPP0874

Voxel-based morphometric imaging in first-episode psychosis: interrogating the role of familial liability

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Introduction: Neuroanatomical abnormalities are reported in psychotic disorders compared to healthy controls; nevertheless, less is known about the role of familial liability to psychosis in morphological brain changes.
Objectives: Using an exploratory voxel-based morphometry (VBM) analyses of the whole brain, we evaluated differences on GMVs across the whole brain among first-episode psychosis (FEP) patients, community-controls, and healthy siblings of patients to interrogate the role of familial liability.
Methods: Data were retrieved from a study (STREAM) conducted in Ribeirão Preto/SP Brazil. We included 71 first-episode psychosis patients (67.6% males, mean age±SD: 18.7±10.8), 24 unaffected siblings of patients (37.5% males, mean age±SD 30.8±10), and 36 controls (71.9% males, mean age±SD: 10±10.5). All magnetic resonance imaging (MRI) scans were acquired on a 3T Philips scanner. VBM data were processed using Statistical Parametric Mapping (SPM) software in MATLAB the MNI coordinate system. We performed exploratory voxel-wise comparisons of GMVs among the three groups using an analysis of covariance (ANCOVA) model in SPM. Results were considered significant if they retained significance after family-wise error (FWE) correction for multiple comparisons ($p<0.05$). All the analyses were adjusted for age, sex, education in years, and total brain GMV.
Results: The whole-brain exploratory analyses revealed no significant findings at the $p<0.05$ level (FWE-corrected). However, pairwise comparisons revealed significant changes betweeen FEP patients and their unaffected siblings. In particular, FEP patients had decreased volumes in the right side of the following regions (FEW = 0.047): superior temporal cortex, Rolandic operculum, insula, Heschel's gyrus, supramarginal gyrus, superior temporal pole, hippocampus, parahippocampal gyrus, fusiform gyrus, amygdala, olfactory, inferior frontal operculum, cerebellum, posterior and medial orbital frontal cortex, rectus, medial temporal, medial frontal, and putamen. FEP patients also showed decreased volumes on the left side of the following regions (FWE 0.049): frontal superior medial gyrus, superior frontal gyrus, frontal middle part, caudate, anterior cingulate cortex, thalamus, and pallidum. Patients also showed widespread reduced GMV in various GMVs regions compared to controls at FWE<0.05. However, no difference was found between siblings and controls (FWE: >0.05).
Conclusions: The study of healthy siblings of patients with heritable illnesses could help in the understanding of the contribution of genetic background and environmental factors to illness state and predisposition. Differences between patients and their siblings could be attributed to the disease state, considering that the unaffected sibling group and unrelated healthy control group did not differ. We will next evaluate biological and environmental contributors to the reported differences.

Disclosure of Interest: None Declared

EPP0875

Global signal topography of the depressive syndrome in bipolar disorder

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Introduction: Previous findings show that the depressive state is characterized by a peculiar suppression of the resting state functional connectivity (rsFC) anti-correlation between resting-state networks (e.g., Default Mode Network) and task-positive networks (e.g., Sensory-Motor Network) in favor of an abnormal positive rsFC pattern. This suggests a large-scale functional disbalance in adaptively switching the attentional focus from an internal-oriented cognitive modality to an external-oriented processing modality. Yet, according to further evidence, such a functional inversion is primarily driven by the global signal (GS) (i.e., by an abnormal large-scale topographical reconfiguration) in major depressive disorder (MDD). However, it is not clear if similar alterations may affect bipolar disorder (BD) in depressive phase.
Objectives: Investigation of the global topography of the depressive syndrome as a potential transnosographic endophenotype and evaluation of the GS on generating differences between groups.
Methods: We compared large-scale rsFC patterns in a group of healthy controls (HC) (n=70) and a group of patients with BD (n=70) during a depressive episode. In order to investigate the impact of the GS, we further performed all analyses both with and without GS regression (GSR).
Results: Compared to HC, patients with an ongoing major depressive episode exhibit specific resting-state changes that are only observed when analysis is performed without regressing GS. Patients were found to exhibit an (i) abnormally strong GS contribution within an extended cluster comprising regions known to be part of highly interconnected hubs (i.e., *transmodal networks*) and showing functional relations' core along the cortical midline and a (ii) diminished influence of the GS in correspondence of