S186. THE EFFECTS OF CHILDHOOD TRAUMA ON HIPPOCAMPAL VOLUME IN FIRST EPISODE PSYCHOSIS: DOES CORTISOL PLAY A ROLE?

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Background: Childhood trauma is one of the most important risk factors in psychosis. Mounting evidence is associating early trauma exposure with alterations in stress sensitive areas, like the hippocampus, and abnormal concentrations of the main stress hormone, cortisol. As hippocampus is a pivotal brain region in the hypothalamic–pituitary–adrenal (HPA) axis regulation of cortisol, better understanding the relationship between childhood trauma, hippocampus structure and cortisol concentration would help clarify how childhood trauma exposure can increase the risk of developing psychosis later on in life.

Methods: Brain structure was evaluated with a 3T MRI scan in 86 first episode psychosis patients (FEP) (49 of which positive for severe childhood trauma) (mean age: 27.8 SD ± 9.1 years). Hippocampal volume and the segmentation of the hippocampal subfields was obtained using FreeSurfer 6. Salivary cortisol samples were collected to measure cortisol levels at awakening (CAR). Initially two separate linear regression models were ran: 1) to predict hippocampal volume changes with childhood trauma as the independent variable and 2) to predict hippocampal volume changes with CAR as independent variable. Finally, we introduced CAR as moderator in the linear model 1 to explore whether it changed the relationship between childhood abuse and hippocampal volume.

Results: Individuals with psychosis and severe childhood abuse presented smaller volume of the right hippocampal head (β = -108.9, p = 0.027), particularly in subfields CA1, CA3, CA4 and in the right GC-ML-DG head (all significant at p < 0.05 with βs between – 110 and -90) (linear model 1). CAR did not predict changes in hippocampal volumes (linear model 2). However, when CAR was introduced the relationship between childhood abuse and hippocampal volume (linear model 1) it showed a moderator role. Indeed low levels of CAR were associated with an even further reduction in hippocampal volume in the right hippocampal head and particularly in subfields CA1, CA3 and CA4 (all significant at p < 0.05 and βs between – 150 and -180).

Discussion: These results suggest that exposure to childhood trauma has a long-term effect on the adult brain particularly in hippocampal areas related to the encoding and retrieval of memories. Importantly, low levels of CAR are associated with even smaller hippocampal head in patients who childhood trauma This is particularly important, because an abnormal hippocampal structure could alter the hippocampal feedback on the HPA axis leading to dysfunctional (lower) cortisol production, which in turn would amplify the impact on brain stress sensitive regions of further stress exposure.

S187. EXPLORING NEURODEVELOPMENTAL AND FAMILIAL ORIGINS OF NEUROLOGICAL SOFT SIGNS IN SCHIZOPHRENIA

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Background: The neurodevelopmental hypothesis is the most widely regarded framework for understanding the development of schizophrenia. One of the most commonly cited pieces of evidence for this theory is the presence of neurological soft signs (NSS) in individuals prior to the onset of psychosis. Increased NSS is also reported in unaffected individuals with a family history of schizophrenia, suggesting that NSS may also have a familial component. Although much research has implicated reduced grey matter volume (GMV) in association with these signs, a component of volume, known as gyration, has been poorly researched. Given that gyration develops predominantly in prenatal life it may be particularly susceptible to a neurodevelopmental abnormality. The aims of this study were to investigate the neurodevelopmental and familial underpinnings of NSS in schizophrenia. Specifically, we examined the brain structural correlates, at both the level of GMV and gyration, of NSS in individuals with schizophrenia, their unaffected relatives and healthy controls. We aimed to determine whether gyration better predicted NSS severity than GMV, and whether the relationship between brain structure and NSS were present in a step-wise manner across the diagnostic groups.

Methods: The sample consisted of individuals with schizophrenia (N=66), their unaffected relatives (N=27) and healthy controls (N=53). NSS was assessed with the Neurological Evaluation Scale (NES), and GMV and gyration were extracted from MRI using the FreeSurfer imaging suite. A series of analysis of covariance were used to compare NES scores and brain measures between the groups. Separate linear regression analyses were used to assess whether whole-brain GMV and gyration predicted NES above a covariate-only model. Moderation analyses were used to assess whether the relationship between NES and brain structure were different between the diagnostic groups. Error control was achieved with a false discovery rate of 5%.

Results: NES was significantly higher in schizophrenia patients than relatives (p<0.0001), who were in turn significantly higher than controls (p=0.034). With the groups combined, lower GMV (p<0.0001), as well as lower gyration (p=0.004), predicted higher NES above a covariate-only model. GMV predicted greater variance in NSS in comparison to gyration, explaining an additional 20.3% of the variance in NES, in comparison to the additional 5.5% of variance in NES explained by gyration. Diagnostic group moderated the association between GMV and NES (p=0.019), but not between gyration and NES (p=0.245). Follow-up tests revealed that lower GMV was associated with higher NES in schizophrenia (t=4.5, p<0.0001) and relatives (t=2.5, p=0.015) but not controls (t=1.9, p=0.05).

Discussion: Our findings indicate that NSS is heritable, being present in patients with established schizophrenia, and to a lesser extent, in unaffected relatives. Consistent with previous research, we revealed that GMV predicted NSS severity, suggesting that abnormalities in volume may underlie these signs. We additionally found that gyration predicted, although to a lesser extent than volume, NSS severity, providing some support for schizophrenia being of possible neurodevelopmental origin. Evidence for an association between volume and NSS in relatives, whom are not confounded by illness-related factors such as medication and symptom severity, indicates a familial contribution to the neural underpinnings of NSS. Together, our study suggests that there may be various aetiological pathways underlying soft signs across the schizophrenia diathesis, some that may be of familial or neurodevelopmental origin.

S188. VISUAL ATTENTION IN EARLY-ONSET SCHIZOPHRENIA AND OTHER NEURODEVELOPMENTAL DISORDERS: THE IMPACT OF SPEAKING WHILE LOOKING AT PICTURES OF SOCIAL INTERACTIONS – EVIDENCE FROM EYE TRACKING DATA

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