Methods: Clinical high-risk symptoms, i.e. attenuated and transient psychotic symptoms (APS, BIPS) as well as agnosic and perceptive basic symptoms (BS), were assessed by well-trained psychologists performing assessments of risk symptoms, using established interviews. Differentiating between perceptive and non-perceptive/cognitive phenomena, impact of age groups on risk symptoms and their clinical significance (current psychosocial functioning deficits or non-psychotic DSM-IV axis-I disorder) was assessed by logistic regression analyses.

Results: Altogether, 9.9% of interviewees (N=689) reported APS, and 18.1% BS: 1.3% met APS, 3.3% COPER and 1.2% COGDIS criteria. For APS, an age effect was detected around age 16: compared to 16-40-year-olds, 8-15-year-olds reported more perceptive APS and lesser clinical significance of non-perceptive APS. Similar age effects of BS on prevalence and clinical significance that differed between perceptive and cognitive BS and followed brain maturation patterns were also detected: around age 18 for perceptive and in the early twenties for cognitive BS.

Discussion: These findings strongly suggest differential developmental factors affecting prevalence and clinical significance of APS and BS: While neurocognitive maturation might influence the presence of APS, brain maturation seems to influence the presence of BS. These findings emphasize the need to address the differential effects of perceptive and non-perceptive risk phenomena, and their interaction with age, also in terms of conversion to psychosis, in future studies.

F27. LATENT PROFILES OF DEVELOPMENTAL SCHIZOTYPY IN THE GENERAL POPULATION: ASSOCIATIONS WITH CHILDHOOD TRAUMA AND FAMILIAL MENTAL ILLNESS

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Background: Latent liability for schizophrenia (schizotypy) is expressed in various combinations of cognitive, psychological, and behavioural characteristics evident in the general population. Historical models propose that distinct classes of individuals expressing different forms of schizotypy may represent manifestations of differential levels of genetic and environmental risk for schizophrenia (or related illness). However, there has been little investigation of developmental models of schizotypy in childhood. Here, we sought to delineate latent profiles of schizotypy among children aged 11–12 years, and to examine associations between emerging schizotypal profiles and parental history of mental illness (as a proxy for genetic risk), early life trauma, and childhood contact with health services for mental illness up to age 13 years.

Methods: Latent profiles of schizotypy were distinguished among 22,137 children (mean age=11.9 years) for whom intergenerational records of health service contact for mental illness and child protection reports were linked to the Middle Childhood Survey (MCS) within the NSW Child Development Study. Selected MCS items were used to index schizotypy across six domains (Unusual Experiences, Cognitive Disorganisation, Impulsive Non-conformity, Introversion, Dysphoria and Self-Other disturbance). Using Latent Profile Analyses (LPA), four groups emerged according to patterns of expression across these domains; membership of three putative schizotypy groups were examined in relation to the likelihood of being exposed to childhood maltreatment and parental mental illness, and the child’s own mental illness up to age 13 years, relative to the no-risk group.

Results: Four classes emerged from the LPA: (1) ‘schizotypy’ (n=1323; 6%); (2) ‘dysphoric pseudo-schizotypy’ (n=4261, 19%); (3) ‘introverted pseudo-schizotypy’ (n=4473; 20%) and; (4) ‘no psychopathology’ (no-risk, n=12,080; 55%). Children in the schizotypy group had the greatest odds of being the subject of a child protection report (OR=2.9, 95% CI 2.6–3.3) and in contact with health services for mental illness by age 13 years (OR=2.7, 95% CI 2.2–3.3), relative to the no-risk group. The odds of child protection reports and childhood mental disorders were smaller, yet significantly increased, among dysphoric pseudo-schizotypy (OR=1.9 and 1.8, respectively) and introverted pseudo-schizotypy (ORs=1.7 and 1.4, respectively), relative to the no-risk group. Parental mental illness exposure was greatest among the schizotypy (OR=2.3, 95% CI 2.0–2.6) subgroup, and was also increased in dysphoric pseudo-schizotypy (OR=1.6, 95% CI 1.5–1.8) and introverted pseudo-schizotypy (OR=1.4, 95% CI 1.3–1.5), relative to the no-risk group.

Discussion: We provide evidence for distinct subtypes of children expressing different forms of schizotypy among a large Australian sample from the general population. The subgroup of children labeled ‘schizotypy’ (6%) characterized by high levels of cognitive disorganisation, impulsive non-conformity, introversion, and self-other disturbance may be at highest risk for developing schizophrenia or other mental illness in adulthood, and had a greater likelihood of childhood maltreatment and parental mental illness history, than other ‘pseudo-schizotypy’ groups.

Reference:

F28. PROGRESSIVE POST-ONSET REORGANISATION OF MRI-DERIVED CORTICAL THICKNESS IN ADOLESCENTS WITH SCHIZOPHRENIA

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Background: Cortical thickness changes continuously throughout healthy adolescent reflecting ongoing maturation. In schizophrenia, distributed abnormalities in cortical maturation are suspected. To study if these distributed changes are a result of a co-ordinated process, we investigated the structural covariance among the longitudinal post-onset thickness changes that occur across various brain regions in adolescent-onset schizophrenia.

Methods: 19 healthy adolescents and 18 age-matched patients with early-onset schizophrenia were scanned twice (~2 years’ interval). The rate of change in cortical thickness was estimated both at lobar and sulcalgyral level. Group level structural covariance was studied using a graph theoretical framework.

Results: At baseline, patients had distributed reduction in cortical thickness compared to controls, though this deviation was abolished over the next 2 years. Occipital cortex had a significantly deviant rate of change in patients (0.8% increase per year) compared to controls (2.5% thinning/ year). Patients had a significant increase in covariance of right anterior insula and calcarine sulcus with rest of the brain.

Discussion: Post-onset structural changes in EoS are not a result of random, mutually independent processes. A spatially interconnected reorganization process, distinct from normal maturational events may underlie these distributed changes.

F29. HIGH-RISK SYMPTOMS FOR PSYCHOSIS IN ADOLESCENTS AND ITS RELATIONSHIP WITH FAMILY BURDEN

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