pregnancy is a causal risk factor for psychotic symptoms in the offspring, or whether this relationship is explained by shared etiological factors, such as genetic and environmental vulnerabilities. More innovative study designs are needed to address this question. Here, we examined the adverse effects of cannabis exposure during pregnancy on psychotic symptoms in pre-adolescent offspring. Such a method would help causal inference as comparisons can be made between the observed associations of maternal versus paternal cannabis use during pregnancy and the risk of psychotic symptoms in the offspring. If the association between cannabis use and psychotic symptoms is causal, early intra-uterine exposure to cannabis could potentially affect neurodevelopment and, hence, contribute to the pathogenesis of psychotic phenomena in children who have not yet used cannabis themselves.

Methods: This study used data from the Generation R Study, a prospective population-based birth cohort from Rotterdam, the Netherlands. Participants were included if data on maternal cannabis use during pregnancy of offspring psychotic-like symptoms at age ten years were available (N = 3692). To determine cannabis exposure, we used prospective maternal self-reports during pregnancy and cannabis metabolite levels from urine. Paternal cannabis use during pregnancy was obtained through maternal report. At age ten years, children were queried regarding psychotic symptoms. Ordinal logistic regression was conducted to investigate whether maternal and paternal cannabis use were associated with offspring psychotic symptoms. In a secondary analysis, a distinction was made between maternal cannabis use exclusively before versus continued maternal cannabis use during pregnancy. All models were adjusted for covariates that were previously associated with cannabis use in this cohort.

Results: Maternal cannabis use was associated with an increased risk for psychotic symptoms in their offspring (n = 183, ORadjusted=1.38 [95% CI 1.03–1.85]). Estimates were comparable for cannabis use exclusively before pregnancy versus continued cannabis during pregnancy (cannabis use before pregnancy: n = 98, ORadjusted=1.39 [95% CI 0.94–2.06]; continued cannabis use during pregnancy: n = 85, ORadjusted=1.37 [95% CI 0.90–2.08]). Paternal cannabis use was significantly associated with offspring psychotic symptoms (n = 297, ORadjusted=1.44 [95% CI 1.14–1.82]).

Discussion: Using data from a large population-based birth cohort, we demonstrated that maternal and paternal cannabis use were each associated with offspring psychotic symptoms at age ten years, well before the risk period of adolescent cannabis use initiation. Notably, estimates were similar for maternal cannabis use exclusively before pregnancy versus continued cannabis use during pregnancy. Moreover, estimates were comparable for maternal versus paternal cannabis use during pregnancy. This suggests that common etiologies, rather than solely causal intra-uterine mechanisms, underlie the association between parental cannabis use and offspring psychotic symptoms, shedding potential new light on the debated causal path from cannabis use to psychosis. Our findings indicate that diagnostic screening and preventative measures need to be adapted for young people at risk for severe mental illness, and that these programs need to offer a family-focused approach.

F34. AUDITORY SENSORY GATING IN YOUNG ADOLESCENTS WITH EARLY-ONSET PSYCHOSIS: A COMPARISON WITH ADHD

Cecilie Lemvigh*[,1], Jens Richardt Møllegaard Jepsen[1], Birgitte Fagerlund[1], Anne Katrine Pagsberg[2], Birte Glenthoj[1], Jacob Rydkjaer[1], Bob Oranje[3]

[1]Center for Neuropsychiatric Schizophrenia Research, Center for Clinical Intervention and Neuropsychiatric Schizophrenia Research, University of Copenhagen; [2]Child and Adolescent Mental Health Center; [3]University Medical Center Utrecht, Holland/Center for Neuropsychiatric Schizophrenia Research, and Center for Clinical Intervention and Neuropsychiatric Schizophrenia Research, University of Copenhagen

Background: Numerous studies have demonstrated impaired sensory gating in schizophrenia and this phenomenon has been proposed as a candidate biomarker for the disorder. Sensory gating is typically assessed during an auditory paired-click test commonly referred to as a P50 suppression paradigm. When two identical stimuli are presented, healthy subjects show a decrease in their neural response to the second stimulus, reflected in a decreased P50 amplitude, whereas schizophrenia patients on average show a much smaller decrease. So far, sensory gating has primarily been investigated in adult patients with schizophrenia, but gating disturbances have also been demonstrated in other illnesses, e.g. in schizotypal personality disorder, albeit less marked. Although the typical age of onset for schizophrenia is late adolescence to early adulthood, a sizable group of patients presents with psychotic symptoms during childhood or early adolescence. Manifestation of psychotic symptoms before the age of 18 is commonly referred to as early-onset psychosis (EOP). Various studies have reported a more severe course of illness and a poorer outcome in EOP compared to the adult-onset form of the disorder. In parallel, we expect more pronounced sensory gating deficits in EOP.

Impaired sensory gating may not be specific to psychosis, but rather a shared disturbance of neuropsychiatric disorders. Although symptoms of attention deficit hyperactivity disorder (ADHD) differ in many ways from those found in schizophrenia, there are common characteristics. Compared to schizophrenia, relatively few studies have investigated sensory gating in ADHD, and some report P50 gating deficits similar to those frequently found in patients with schizophrenia.

Methods: We investigated P50 suppression in a large cohort of adolescents (12–17 years old) consisting of patients with either EOP (N=56) or ADHD (N=28) as well as age and gender matched healthy controls (N=72). In our paradigm two identical sounds (clicks) were presented separated by a 500ms interval. The amount of suppression was expressed as the ratio between the P50 amplitude of a subject's response to the first click and his/her amplitude in response to the second click.

Results: The EOP patients scored significantly higher on PANSS (positive, negative, general, and total PANSS scores) compared to both ADHD patients and healthy controls. However, there were neither significant group differences in raw P50 amplitude, nor in the gating ratios between young adolescents with EOP, ADHD and healthy controls.

Discussion: This is the first study to investigate sensory gating in young adolescents with EOP. We found no P50 suppression deficits in these patients which, given the relatively large sample size in our study, cannot merely be ascribed to power issues. The results are in contrast with the majority of studies investigating sensory gating in schizophrenia and ADHD. However, the results are in agreement with earlier studies from our lab showing evidence of inconsistent P50 suppression deficits in two separate cohorts of adult, antipsychotic naïve, first-episode patients with schizophrenia. Based on our findings, P50 sensory gating cannot differentiate between young adolescents with EOP or ADHD, and deficient P50 suppression does not seem to be a valid biomarker for EOP.

F35. PRESCRIPTION PATTERN OF ANTIPSYCHOTICS FOR CHILDREN AND ADOLESCENTS WITH SCHIZOPHRENIA IN KOREA BASED ON NATIONWIDE DATA

Kee Jeong Park*[,1], Jung Sun Lee[1], Hyo-Won Kim[1]

[1]Asan Medical Center, University of Ulsan College of Medicine

Background: This study aimed to analyze the extent and pattern of antipsychotic prescription for Korean children and adolescents with schizophrenia using population-based data.

Methods: Our data was retrieved from the Korean National Health Insurance Review & Assessment Service-National Sample for 2013, which was a stratified sampling from the entire population under the Korean national health insurance program. Among 0.2 million children and adolescents aged 6–18 years from data, subjects who had received any