A2-10-11 weeks of gestation (n=12). The Control group (K) includ control samples K1-8–9, K2-10-11 weeks (n=14). The analysis of changes in morphometric parameters was used to identify quantitative changes among glioblasts, correlation between the degree of differentiation components and the degree of influence of alcohol. For this, the program AxioVision 4.8 was used Parameters of GABAA/benzodiazepine receptors were studied by the radioreceptor assay of [3H]-flunitrazepam with synaptoneurosomes.
Results: Changes in glioblasts tof human brain embryos and fetuses were revealed under conditions of chronic prenatal alcoholization with an increase in gestational age compared to the control subgroups: a significant increase in the average number of glioblasts, the length of the perimeters of the presynaptic terminal, postsynaptic density, presynaptic terminal areas were significantly less (p<0, 01) in the study group than in the control. Exposure to ethanol reduces the affinity of GABAA/benzodiazepine receptors, which affects neuronal plasticity associated with the development of glioblasts and neuroblasts during embryogenesis.
Conclusions: Changes in microglial cause disruption of the neuronal activity

Disclosure: No significant relationships.
Keywords: embryogenesis; neuroimmune system; brain; alcohol; glioblast; GABAA receptor; synapse

EPV0897

Identifying prodromal biomarkers for schizophrenia and bipolar disorder using magnetoencephalography

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Introduction: Schizophrenia (SZ) and bipolar disorder (BD) are severe mental illnesses with large overlapping heritability. Both disorders are associated with altered neurophysiological responses, as measured with magnetoencephalography (MEG) or electroencephalography (EEG), particularly reduced mismatch negativity (MMN) and 40 Hz auditory steady-state responses (ASSR). These deficits could potentially both serve as early markers of illness and provide insight into the underlying pathophysiology as endophenotypes. First-degree relatives to patients with SZ and BD also show some neurophysiological deficits, however whether these deficits can be detected in adolescent offspring of patients is undetermined.
Objectives: We aim to investigate whether adolescents at familial high risk of schizophrenia and bipolar disorder show aberrant MMN and ASSR compared to population-based controls.
Methods: We will investigate MMN and 40 Hz ASSR in 15 year old adolescents (n ≈ 175) born to parents diagnosed with either SZ (FHR-SZ), BD (FHR-BD), or neither SZ or BD (population-based controls, PBC) using MEG. We will first perform sensor-level analyses and subsequently apply dynamic causal modeling (DCM) to investigate effective connectivity and make inferences about the underlying neurobiological mechanisms. We will investigate whether current psychopathology, cognitive status, and genetic risk for SZ and BD predict neurophysiological responses in the adolescents. Investigations are part of The Danish High Risk and Resilience Study - VIA (VIA15), a population-based longitudinal cohort study integrating social, psychological and biological risk factors and outcomes for SZ and BD.
Results: Final results are expected in 2024
Conclusions: The VIA15 study will allow for unprecedented insight into the neurobiological development of schizophrenia and bipolar disorder.

Disclosure: No significant relationships.
Keywords: bipolar disorder; Magnetoencephalography; familial high-risk; schizophrénia

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Exploring the selective gray matter profile of autism spectrum disorder through Bayes Factor Modeling

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Introduction: Despite decades of brain MRI research demonstrating atypical neuroanatomical substrate in patients with autism spectrum disorder (ASD), it remains unclear whether and to what extent disorder-selective neuroanatomical abnormalities occur in this spectrum. This, and the fact that multiple brain disorders report a common neuroanatomical substrate, makes transference and the application of neuroimaging findings into the clinical setting an open challenge.
Objectives: To investigate the selective neuroanatomical alteration profile of the ASD brain, we employed a meta-analytic, data-driven, and *reverse inference*-based approach (i.e.; Bayes fACtor mOdeliNg).
Methods: Eligible voxel-based morphometry data were extracted by a standardized search on BrainMap and MEDLINE databases (849 published experiments, 131 brain disorders, 22747 clinical subjects, 16572 x-y-z coordinates). Two distinct datasets were generated: the ASD dataset, composed of ASD-related data; and the non-ASD dataset, composed of all other clinical conditions data. Starting from the two unthresholded activation likelihood estimation (ALE) maps, the calculus of the Bayes fACtor mOdeliNg was performed. This allowed us to obtain posterior probability distributions on the evidence of brain alteration specificity in ASD.
Results: We revealed both cortical and cerebellar areas of neuroanatomical alteration selectivity in ASD. Eight clusters showed a selectivity value 90%, namely the bilateral precuneus, the right inferior occipital gyrus, left lobule IX, left Crus II, right Crus I, and the right lobule VIIIA (Fig. 1).
Conclusions: The identification of this neuroanatomical pattern provides new insights into the complex pathophysiology of ASD, opening attractive prospects for future neuroimaging-based interventions.

Disclosure: No significant relationships.
Keywords: reverse inference; cerebellum; default-mode network; structural MRI