O0091

Brain structure changes associated with depression outcome in adolescents bullied throughout adolescence

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Introduction: Being bullied in adolescence has been associated with developing depressive symptoms in adulthood.
Objectives: We sought to describe the trajectories of peer victimization across adolescence and their relationships with grey matter volumes and depression outcomes in young adulthood.
Methods: Community adolescents from the IMAGEN database (n = 724) with both peer victimization and neuroimaging data were included. A longitudinal clusterization method (normal mixture model) was used to analyze the bullying scores at baseline (age 14), and at follow-ups at age 16, 18 and 22. Relations between clusters and brain volumes or depression diagnosis were examined using logistic and linear multivariate regression models.
Results: Three victimization trajectories were observed. A first trajectory included participants who were never bullied and had no depression outcome, a second trajectory identified participants who were bullied at age 14 and 16 only, and had no depression outcome, and finally, a third trajectory of continuous bullying throughout adolescence to young adulthood (age 22) that was significantly associated with depression outcomes (r=0.87, p=0.0004). In addition, the continuously bullied participants displayed larger volumes of bilateral hippocampus, posterior cingulate cortex and right putamen at age 22.
Conclusions: These data confirm that chronic peer victimization throughout adolescence is associated with brain structure changes and might increase vulnerability to depressive disorders. They highlight the need for preventive school interventions in early adolescents.

Disclosure of Interest: None Declared

O0090

Oxidative stress as a shared mechanisms for different prenatal stressors: long-term effects on adolescent male and female mouse offspring

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Introduction: Stressful experiences *in utero* can produce physiological changes which become embedded biological traces affecting fetal brain development and ultimately leading to increased vulnerability for psychiatric disorders.
Objectives: We hypothesized that stressors as diverse as maternal obesity and maternal psychophysical stress might disrupt fetal programming resulting in long-lasting effects on offspring brain development by acting through shared oxidative stress (OS)-mediated mechanisms.
Methods: We compared a mouse model (C57Bl/6N) of maternal high-fat diet (HFD) consumption (13 weeks, until delivery) to prenatal restraint stress (PNS) repeatedly administered during the last week of pregnancy. To counteract the negative effects of both stressors, the antioxidant N-acetyl-cysteine (NAC, 1 g/kg) was administered to female breeders for 8 weeks until delivery. Emotionality was assessed in adolescent male and female offspring through the elevated-plus-maze (EPM). Moreover, hippocampal gene expression levels of Brain-Derived-Neurotrophic-Factor (*Bdnf*), Nuclear factor erythroid 2–related factor 2 (*Nrf-2*) and Kelch-like ECH-associated protein 1 (*Keap-1*) were measured, by qPCR, as markers of brain plasticity and antioxidant capacity.
Results: Prenatal exposure to both HFD and PNS enhanced behavioral disinhibition, increasing time spent in the open arms of the EPM and decreasing the frequency of risk-assessment behaviors, especially in female offspring. Moreover, both prenatal stressors led to decreased *Bdnf* (in females) and *Nrf-2* levels, and disrupted *Keap-1* levels. Prenatal NAC was able to counteract these effects on the brain.
Conclusions: Our data support the hypothesis of a "funnel effect" model explaining how different prenatal stressors result in long-term negative effects on the adolescent offspring, increasing risk assessment behaviors and affecting brain plasticity and antioxidant defenses. The beneficial preventive effects of NAC suggest that OS may be a common mechanism, playing a pivotal role in fetal programming of mental disorders. *ERANET-NEURON-JTC-2018-Mental Disorders-"EMBED" and Bando Ricerca Indipendente ISS 2021-2023; MOMINFLAM. Unique signatures underlying placental-fetal brain crosstalk in maternal obesity to F Cirulli.*

Disclosure of Interest: None Declared

O0091

1H-MRS of the anterior cingulate cortex in obsessive-compulsive disorder: metabolic abnormalities in pgACC - controlled study

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Introduction: Obsessive-compulsive disorder (OCD) is connected with increased activity in cortico-striatal-thalamic-cortical (CSTC) loop. The anterior cingulate cortex (ACC) is a part of this loop and

plays a role in error detection and monitoring and processing of conflicting information, core OCD clinical signs. This area also contains a high density of Von Economo neurons. Biochemistry in this area is closely connected with the pathophysiology of OCD.
Objectives: Decreased concentrations of total N-acetylaspartate (tNAA) have been reported in ACC in patients with OCD compared to healthy controls (HC), with increase after successful treatment. Findings by other metabolites: choline-containing compounds (tCho), total creatine (tCr) and myo-inositol are not consistent. Differences in levels of tNAA, tCho, tCr would correlate with the severity of symptoms measured by Y-BOCS. In the comparison in the subgroup of patients with/ without medication, there will be differences in levels of metabolites.
Methods: 54 patients diagnosed with OCD according to ICD-10 and DSM-IV criteria, and 54 HC matched for age and sex were included in the study. They underwent MRI and MR Spectroscopy on a 3T Magnetom Prisma scanner (Siemens, Germany) equipped with a 64-channel volume head coil (Fig. 2). After spectral quality control, 28 OCD and 28 HC subjects were included in the statistical analysis. OCD subjects were interviewed using the Y-BOCS to evaluate the severity of the symptoms. Patients enrolled in the study were without medication at least 5 days before MRI or on a stable dosage of SSRI antidepressants. To assess the intergroup differences Wilcoxon Rank Sum test or Kruskal-Wallis test was used as appropriate. The correlation between metabolite levels and clinical characteristics was assessed by Spearman's rank correlation coefficient. The statistics were calculated using R, version 4.1.1.
Results: We found no differences in levels of tNAA in ACC in OCD vs. HC (p=0,21; see Tab.1, Fig.1). We found significantly increased level of tCho, tCr and Ins in OCD vs. HC (p=0,03; p=0,004 resp.; p=0,017 resp.). tCr levels corelated negatively with YBOCS compulsions subscale (p=0,046; cor=-0,38). tCho levels showed a trend to negative correlation with Y-BOCS compulsions subscale (p=0,067; cor=-0,35). Analysis on the subgroup with (13 subjects, 46,43 %) and without (13 subjects, 46,43 %) stable SSRI medication did not reveal significant differences.
Image:

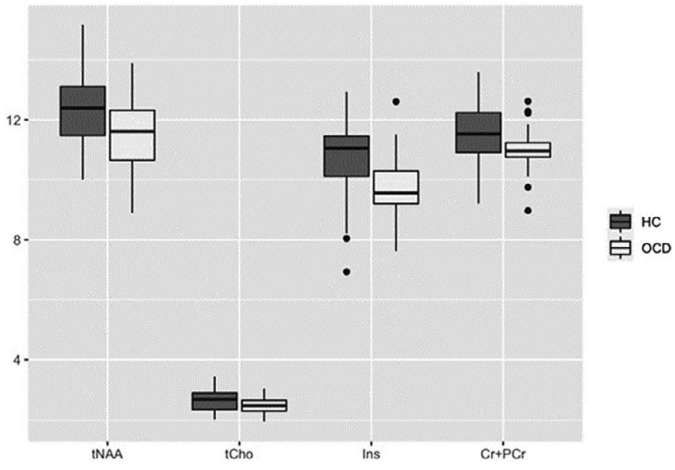
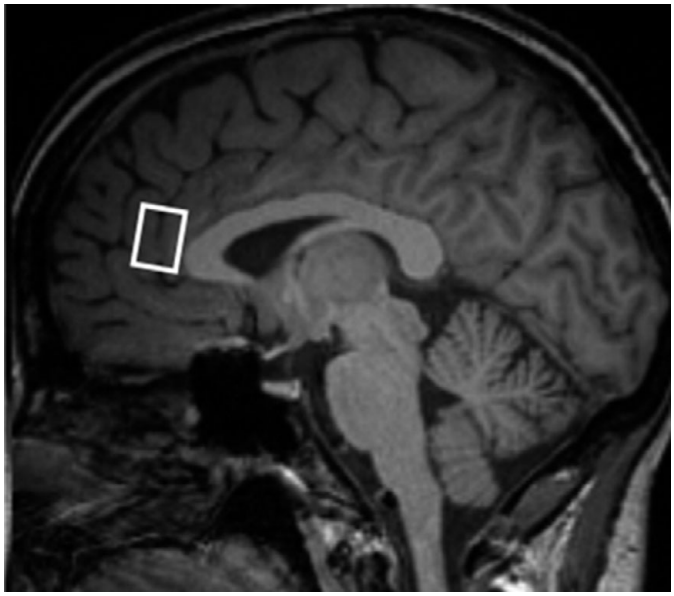


Image 2:



Conclusions: Our study found difference in ACC by OCD patients compared to HC, mainly increased tCho, tCr and Ins. Also, the study shows a significant correlation between the severity of compulsions and tCr levels. We can see this trend also in the correlation of the tCho and Y-BOCS compulsions subscale. Similar tNAA level by OCD and HC groups could indicates correctly adjusted medication or stable state by enrolled patients.

Disclosure of Interest: None Declared

O0092

Exploring Decision-Making Strategies in the IOWA Gambling Task and Rat Gambling Task

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Introduction: Impairments in decision-making processes are believed to play an important role in both substance use disorders and behavioral addictions. Clinical and pre-clinical experimental testing provide complimentary insights on the psychobiological mechanisms of decision-making. The IOWA Gambling Task