

O0091

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

M.-L. Paillere-Martinot^{1*}, A. Briffod², P.-A. Beaudoin²,
O. Hassani², J.-L. Martinot¹ and E. Artiges¹

¹INSERM U1299 “Developmental trajectories & psychiatry”, Université Paris-Saclay, Ecole Normale Supérieure Paris-Saclay - CNRS - Centre Borelli and ²Ecole CentraleSupélec, Université Paris-Saclay, CentraleSupélec, Gif-sur-Yvette, France

*Corresponding author.

doi: 10.1192/j.eurpsy.2023.293

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

C. Musillo^{1*}, A. Berry¹, K. C. Creutzberg², B. Collacchi¹, M. Samà¹,
L. Giona¹, M. A. Riva² and F. Cirulli¹

¹Istituto Superiore di Sanità, ROMA and ²Università degli Studi di Milano, Milan, Italy

*Corresponding author.

doi: 10.1192/j.eurpsy.2023.294

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

E. Nosková^{1*}, I. Fajnerová², D. Pajuelo^{2,3}, P. Stopková⁴ and
J. Horáček⁵

¹anxiety department; ²NIMH: National institute of mental health, Klecany; ³MR Unit, Department of Diagnostic and Interventional Radiology, Institute for Clinical and Experimental Medicine, Prague; ⁴anxiety department and ⁵Center for advanced studies of brain and consciousness, Institute for Clinical and Experimental Medicine, Klecany, Czech Republic

*Corresponding author.

doi: 10.1192/j.eurpsy.2023.295

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