

inhibit unwanted behaviours. Children's tendency to attribute negative emotions to daily events, which could lead to increased anxiety, was associated with two main neonatal brain features. These were: 1) weaker structural connectivity in a long-range white matter projection tract called the uncinate fasciculus which connects the frontal lobe with the anterior temporal lobe and 2) altered fronto-limbic functional connectivity, both of which play a critical role in several aspects of social and emotional development. These findings show that early brain changes can be used to predict children's social and emotional outcomes, hence could be used to inform preventative interventions aimed at averting and targeting emerging emotional disorders.

**Disclosure:** No significant relationships.

**Keywords:** preterm birth; brain development; emotion regulation; Psychopathology

## S0082

### Emotional Dysregulation: Epidemiology and Genetic Features from Childhood towards Adulthood

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Emotional dysregulation (ED) is a dimensional, transdiagnostic domain that is associated with multiple categorical psychiatric diagnoses from childhood to adulthood, representing a risk for increased problems in affect, behavior, and cognition [1]. Traditionally, the nature of ED trait has been studied with top down approaches: quantitative evaluation of ED is possible through "Dysregulation Profile" scoring, which is measured through composite scales of the "Achenbach System of Empirically Based Assessment" (ASEBA) [2] questionnaires. Dysregulation profile is characterized by severe anxiety and affective symptoms, impulsive and/or aggressive behaviours and metacognitive difficulties. More recently, different researchers also applied bottom up approaches to evaluate the presence of ED in both general population and clinically referred samples [3]. Also in these cases, the results showed that ED is a trait, stable through time and across different cultures and societies, associated with higher presence of psychiatric diagnosis. It is important to note that these non-traditional statistical approaches highlighted that, in adulthood, ED is characterized by elevated scores in both externalizing and internalizing areas. In this contribution, the research aimed at disentangling the etiology of ED, which is crucial to treat and prevent worst evolution associated with this trait, will be revised. Many efforts have been done to understand the complex interaction between genetic and environmental risk factors which predispose patients to develop and maintain ED. [1] Aitken, et al. (2019). *JAD*, 253, 87-95. [2] Achenbach & Rescorla (2001). *Manual for the ASEBA school-age forms and profiles*. [3] Bianchi, et al (2017). *ECAP*, 26(5), 549-557.

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**Keywords:** gene-environment interaction; emotional dysregulation; Developmental trajectories; Methylation

## Challenges and Advances in Pharmacogenomics

### S0083

#### Pharmacogenomics of MDD as a Developing Field: Challenges and Opportunities

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While first gene-drug pairs have emerged to be clinically actionable in the treatment of major depressive disorders (MDD) (e.g., CYP2D6 and TCAs/SSRIs), genomic studies have not yet been successful in identifying replicable and valid biomarkers of pharmacological treatment outcome. While some trials suggest that candidates such as CYP2D6, CYP2C19, CYP1A2, SLC6A4 and HTR2A polymorphisms may improve the prediction of response/remission, these results should be interpreted cautiously and required confirmation in larger samples. This presentation will cover state of the art of pharmacogenomics for MDD as well as the emerging field of pharmacotranscriptomics and functional genomics analyses in MDD. Specifically, pharmacotranscriptomics in combination with genomics may be a promising avenue in overcoming some of the current limitations in treatment response prediction research. More recently, the combined genetic effect of polygenic risk scores has shown promising results in predicting treatment response. Importantly, adequately large and well phenotyped clinical trials are required to be conducted with pharmacogenomics/-transcriptomics prospectively in mind.

**Disclosure:** No significant relationships.

**Keywords:** MDD; pharmacogenomics; Transcriptomics; polygenic risk scores

### S0084

#### Clinical Phenotypes Characterization in Pharmacogenetics Testing Trials for Major Depressive Disorder Treatment

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Several data indicate that the success of pharmacological treatment in major depressive disorder (MDD) is still unsatisfactory. The determination of the optimal treatment generally requires multiple trials with different treatments, with the sobering observation that the more treatments tried without success, the less likely a successful outcome, with the result of a long unremitted disease, worse long term prognosis, increased rates of side effects, and important medical, social and economic burden. The reasons for the low response and remission rates are multiple and depend on environmental and biological factors intrinsic to the disease and drug treatments. Pharmacogenetic (PG) tests have the potential to increase efficacy predicting outcome and to reduce antidepressant