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S070

Identification of a long lasting stress signatures associated with enhanced vulnerability for depression by using 'omics and cross species approaches

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Depression results from the interplay of vulnerability genes with environmental factors, a phenomenon named as 'gene-environment (GxE) interaction'. To date, GxE interaction studies have been limited to hypothesis-based candidate genes, since genome-wide (GWAS)-based GxE interaction studies would require enormous datasets with genetics, environmental and clinical variables. We used a novel, cross-species and cross-tissues "*omics*" approaches to identify genes predicting depression in response to stress in GxE interactions. We integrated the transcriptome and miRNome profiles from the hippocampus of adult rats exposed to prenatal stress (PNS) with transcriptome data obtained from blood mRNA of adult humans exposed to early life trauma, using a stringent statistical analyses pathway. Network analysis of the integrated gene lists identified the Forkhead box protein O1 (*FOXO1*), Alpha-2-Macroglobulin (*A2 M*) and Transforming Growth Factor Beta 1 (*TGFB1*) as candidates to be tested for GxE interactions, in two GWAS samples of adults either with a range of childhood traumatic experiences (Grady Study Project, Atlanta, USA) or with childhood emotional abuse only (Helsinki Birth Cohort Study, Finland). Six *FOXO1* SNPs showed significant GxE interactions with emotional abuse in the Grady Study that survived stringent permutation analyses and were all replicated in the Helsinki study. In addition, other SNPs in all the three genes showed significant GxE interactions with emotional, physical and sexual abuse in the Grady Study. We therefore provide a successful 'hypothesis-free' approach for the identification and prioritization of candidate genes for GxE interaction studies that can be investigated in GWAS datasets.

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S071

Epigenetic signatures of early life adversities in animal models: A role for psychopathology vulnerability

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Stressful experiences early in life (ELS) represent one of the most relevant factors for the vulnerability to psychopathologies. Epigenetic changes, such as DNA methylation, have emerged as a major mechanism through which ELS can alter adult behaviour leading to persistent changes of gene regulation.

We performed DNA methylation analyses in the hippocampus and prefrontal cortex of adult rats exposed to stress during gestation (PNS), a model that is associated with persistent behavioral alterations relevant for psychiatric disorders.

Using an epigenome-wide analysis, an overlap of 893 differentially methylated genes was observed between hippocampus and prefrontal cortex of adult male and female rats exposed to PNS. The list includes several genes previously associated with schizophrenia and other psychiatric conditions, such as calcium and potassium voltage operated channels as well as GABA and glutamate receptor subunits. By restricting the overlap to genes that were modulated in the same direction, we identified miR-30a as being less methylated in PNS rats. Interestingly one of the targets for this miRNA is the neurotrophin BDNF, whose expression was indeed reduced as a consequence of the prenatal manipulation. Interestingly chronic treatment of PNS rats with the multi-receptor modulator lurasidone during adolescence was able to prevent the changes in miR30a and BDNF expression.

These results highlight the importance for the identification of methylation signatures through which stress exposure early in life could engrave on the outcome of the adult phenotype, and may allow the identification of novel genes and pathways that are affected as a consequence of ELS.

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Symposium: Intergenerational transmission of parenting: Epigenetic, genetic, and psychological mechanisms

S072

Intergenerational transmission of well being–genetic and epigenetic mechanisms

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Introduction Maternal mental well being influences offspring development. Research suggests that an interplay between genetic and environmental factors underlies this familial transmission of mental disorders.

Objectives To explore an interaction between genetic and environmental factors to predict trajectories of maternal mental well being, and to examine whether these trajectories are associated with epigenetic modifications in mothers and their offspring.

Method We assessed maternal childhood trauma and rearing experiences, prenatal and postnatal symptoms of depression and stress experience from 6 to 72 months postpartum, and genetic and epigenetic variation in a longitudinal birth-cohort study ($n = 262$) (Maternal adversity, vulnerability and neurodevelopment project). We used latent class modeling to describe trajectories in maternal depressive symptoms, parenting stress, marital stress and general stress, taking polygenetic risk for major depressive disorder (MDD),