with PP when compared with healthy postpartum women. However, no study has ever evaluated women at risk who do not develop the disorder.
We conducted an exploratory analysis of a gene expression profile that could distinguish women with PP episode (PPE) from women at risk who do not develop PP (NPPE) after delivery.
Methods The sample was characterised by 24 women at risk of PP of which $n = 12$ with PPE and $n = 12$ with NPPE and 21 healthy women in the same postpartum period. Following Microarray analysis, we assessed gene expression signature across the 3 groups using ANOVA. We then studied Pathway analysis of genes differently expressed in PPE and PPE exploring canonical pathways and upstream regulators using Ingenuity Pathway Analysis software.
Results Following an exploratory gene expression analysis we identified 719 genes that are differently expressed across PPE and NPPE. The PPE presented upregulation of several genes involved in the inflammatory pathway and increased gene expression levels of *GRIA4*, *AKT3*, *SP4* and *NRG1* genes, which have been previously described in psychotic disorders. Moreover, 5 differently expressed canonical pathways were identified including ones relevant to development, mitochondrial formation and immune system.
Conclusion These preliminary results reveal the presence of an immuno-neuro-endocrine dysregulation in postpartum psychosis, with an upregulation of the immune system specific to those women at risk who actually develop postpartum psychosis episodes.
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A meta-analysis of gene (5-HTT) × environment interactions in eating pathology using secondary data analyses

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Background Gene × environment (G × E) interactions in eating pathology have been increasingly investigated, however studies have been limited by sample size due to the difficulty of obtaining genetic data.
Objective To synthesize existing G × E research in the eating disorders (ED) field and provide a clear picture of the current state of knowledge with analyses of larger samples.
Method Complete data from seven studies investigating community ($n = 1750$, 64.5% female) and clinical ($n = 426$, 100% female) populations, identified via systematic review, were included. Data were combined to perform five analyses: 5-HTTLPR × Traumatic Life Events (0–17 events) to predict ED status ($n = 909$), 5-HTTLPR × Sexual and Physical Abuse ($n = 1097$) to predict bulimic symptoms, 5-HTLPR × Depression to predict bulimic symptoms ($n = 1256$), and 5-HTTLPR × Impulsivity to predict disordered eating ($n = 1149$).
Results The low function (s) allele of 5-HTTLPR interacted with number of traumatic life events ($P < .01$) and sexual and physical abuse ($P < .05$) to predict increased likelihood of an ED in females but not males (Fig. 1). No other G × E interactions were significant, possibly due to the medium to low compatibility between datasets (Fig. 1).
Conclusion Early promising results suggest that increased knowledge of G × E interactions could be achieved if studies increased uniformity of measuring ED and environmental variables, allowing for continued collaboration to overcome the restrictions of obtaining genetic samples.

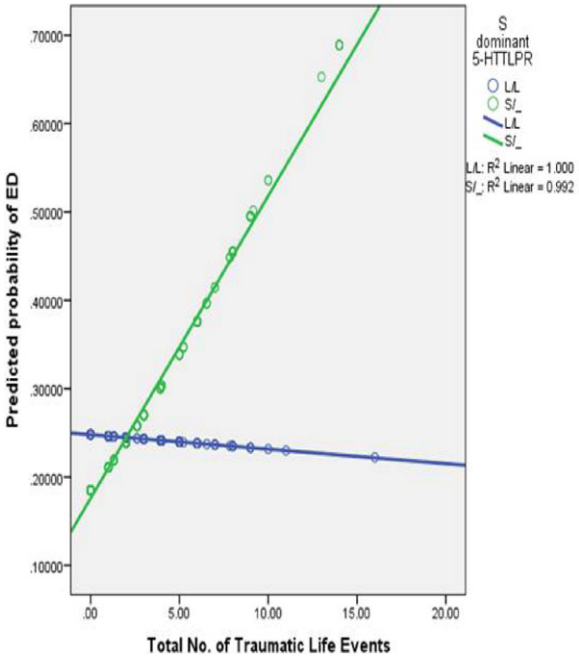

Fig. 1 The interaction between 5-HTTLPR (s-allele present versus s-allele absent) and number of traumatic life events to predict likelihood of an eating disorder in females.

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