

Article: 0078

Topic: S27 - Symposium 29: Childhood trauma in severe mental disorders: clinical expression and mechanisms

Biomarkers and Childhood Trauma

A. Cattaneo¹, G. Plazzotta², M.A. Riva³, C.M. Pariante⁴

¹Dept of Psychological Medicine, King's College London/IRCCS Fatebenefratelli.it, London/Brescia, United Kingdom ; ²IRCCS Fatebenefratelli, IRCCS Fatebenefratelli.it, Brescia, Italy ; ³Dept of Pharmacological and Biomolecular Sciences, University of Milan, Milan, Italy ; ⁴Dept of Psychological Medicine, King's College London, London, United Kingdom

It is well known that a history of early life stressful events increases the vulnerability in the adulthood to develop psychiatric disorders, however, the underlying mechanisms require further investigation. This talk will focus on the role of childhood trauma in causing changes in specific molecular pathways, which persist over time, and thus, are responsible of increasing the vulnerability to develop psychopathologies in the adulthood.

By using a transcriptomic approach we found that control subjects, which were exposed to childhood trauma events, have higher blood mRNA levels of several inflammatory biomarkers as well as alterations in cytokines- related pathways. Moreover, we have found alterations in the mRNA levels of genes involved in the stress response, including the serum glucocorticoid kinase (SGK1), a kinase specifically activated by glucocorticoids. SGK1 mRNA levels are higher in controls with a history of childhood trauma events as compared with subjects without such experiences; higher SGK1 mRNA levels are also observed in depressed patients without early life stressful events and a further increase can be observed in depressed patients with childhood trauma. This suggests an additive effect of the two components, illness and trauma, in the modulation of SGK1. Similarly, SGK1 mRNA levels are increased also in the hippocampus of adult rats, which have been exposed to prenatal stress, whereas no alterations can be observed during the previous ages. This supports a long lasting effect of the prenatal stress on SGK1 levels. In order to explain the effect of an early stress on molecular alterations observed later in life, putative mechanisms involving miRNAs and methylation changes will therefore be discussed.