S42-01

CHILDHOOD ABUSE AND THE GENETIC VARIANTS OF THE HPA AND SEROTONIN SYSTEMS

D. Wasserman
Swedish National prevention of Suicide and Mental Ill-Health (NASP), Karolinska Institutet, Stockholm, Sweden

According to a stress diathesis model, genes and environment, as well as possible interactions in-between (GxE), may result in vulnerability towards suicidal behaviors (SB), characterized by behavioral trigger endophenotypes such as increased depression-intensity and aggression/anger/impulsivity.

Excessive stress has the potential to induce unfavorable effects in a variety of higher brain-functions, incurred as side effects to maladaptive responses in the genetically controlled stress-responsive neurosystems eg. in the hypo- thalamic-pituitary-adrenal(HPA) axis. Childhood traumatic experiences are particularly relevant as stressful experiences are they frequently occur in the early childhood, when susceptibility to external stressors is higher. Childhood abuse has been found in several study as directly associated with suicidal behavior, independently from other psychological or psychopathological risk factors.

HPA-axis dysregulation is regarded as an endo- phenotype of depression-, anxiety- and alcohol abuse disorders, commonly found also in suicide behaviors. Various neurobiological alterations, suggesting abnormal HPA-axis activity and reactivity, have also been demonstrated to occur in suicidal behaviors including completed suicides.

The HPA axis is a major systemic stress-modulator being mainly controlled by the regulatory corticotropin releasing hormone receptor 1 (CRHR1) gene. An overview will be presented of the role of the HPA axis in suicidal behaviors with a focus on CRHR1 gene .and on interactions with childhood traumatic experiences.

https://doi.org/10.1016/S0924-9338(11)73864-0 Published online by Cambridge University Press