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STRESS, SUICIDAL BEHAVIOUR AND GENES

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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 e.g. in the hypothalamic- pituitary-adrenal(HPA) axis. HPA-axis dysregulation is regarded as an endophenotype 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 incl. completed suicides. The HPA axis is a major systemic stress-modulator being mainly controlled by the regulatory corticotrophin 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.

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