Levels of initial hormones of the hypothalamic-pituitary-adrenal (HPA) axis, corticotropin-releasing hormone (CRH) and arginin-vasopressin, can be altered in adulthood with a context of early-life stress. The effect can be stronger after an acute stress. Some authors suggest that higher levels can be caused by hypomethylation of that genes promotors. High level of default hormones often results in high concentration of corticosterone. Higher concentration of corticosterone in blood can also be supported by a lower level of transcortine, which has been observed in adulthood after a prenatal malnutrition. Locally in tisues is the concentration of corticosterone regulated by 2 types of 11β-hydroxysteroid dehydrogenase. Hyperreactivity of HPA axis can cause an anxiety-like behavior. Anxiety-like behavior is regulated by binding CRH to its receptors. Mice with knockouted gene of the first type receptor (CRHR1) shows generally less anxiety-like behavior. Conversely, some author say that binding CRH to its second type receptor (CRHR2) can inhibit an anxiety-like behavior. Prenatally stressed males show increased expression of CRHR1, prenatally stressed females show reduced expression of CRHR2. Early-life stressed animals also shows a depression-like behavior. It can be related to the presence of some pro-inflammatory cytokines and its receptors in brain. Higher levels of pro-inflammatory cytokines are observed also on the periphery. Conversely, levels of anti-inflammatory cytokines are reduced or coincide with control levels. Early-life stressed animals were also observed to have an abnormal percentage and higher reactivity of immune cells.