Neuroplastic changes in male rats exposed to maternal deprivation: modulation by genetic alterations of the serotonin transporter

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Although the causes of psychiatric disorders are not fully understood, it is well established that mental illness originates from the interaction between genetic and environmental vulnerability factors. To this regard, compelling evidence demonstrates that depression can be the consequence of altered, and often maladaptive, response to adversities during pre- and early post-natal life. Indeed, adverse events occurring early in development may alter the correct program of brain maturation and render the organism more vulnerable to psychiatric disorders.
On these bases, in this study we investigated the impact of chronic maternal deprivation (MD) (from postnatal day 2 to postnatal day 14) on the expression of the neurotrophin brain-derived neurotrophic factor (BDNF) in male adult heterozygous as well as homozygous serotonin transporter (SERT) knockout rats. Specifically, we analyzed the gene expression levels of the total form of *Bdnf,* of the 3'UTR long transcripts and of *Bdnf* exon IV in key brain regions, namely the ventral and dorsal hippocampus as well as the ventromedial and dorsomedial prefrontal cortex, which have different anatomical connectivity and functional implications.
We found that in the ventral part of these regions both SERT deletion and MD significantly decreased *Bdnf* expression, whereas in the dorsal part the exposure to MD produced a significant effect on the neurotrophin's expression only in heterozygous SERT rats. In summary, our results demonstrate that deletion of the SERT gene as well exposure to early life stress may lead to similar changes in *Bdnf* expression with brain region specificity but limited gene * environment interaction.