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Immunochemical detection of trkB receptor in the brain of a rat model of depression

Maria Pina Serra[1], Laura Poddighe[1], Marianna Boi[1], Maria Antonietta Piludu[2], Osvaldo Giorgi[2], Maria Giuseppa Corda[2], Marina Quartu[1]

[1] Dipartimento di Scienze Biomediche, Sezione di Citomorfologia, Università degli studi di Cagliari, Monserrato, Italy - [2] Dipartimento di Scienze della Vita e dell'Ambiente, Sezione di Neuroscienze, Università degli studi di Cagliari, Cagliari, Italy

The outbread Roman High- (RHA) and Roman Low-Avoidance (RLA) rat lines were psychogenetically selected for rapid versus poor acquisition of active avoidance, respectively, and differ in many behavioural traits that closely resemble the cardinal symptoms of depression (1). Beyond the monoamine hypothesis of depression, compelling evidence suggests that mood disorders are characterized by reduced neuronal plasticity. Consistently, it has been shown that exposure to stress and antidepressant treatment modulate the expression of neurotrophic molecules and their relevant receptors, and that these changes show an anatomical specificity (2). With the aim to characterize the molecular and neuronal systems involved in the pathogenesis of depression and in the mechanism of action of the antidepressant treatments, here we investigate on the immunochemical occurrence of trkB, the high affinity tyrosine-kinase receptor for brain-derived neurotrophic factor (BDNF), in selected areas of the RHA and RLA rat brain by means of western blot (WB) and immunohistochemistry. WB analysis indicates that the relative levels of trkB patently and markedly differed in the prefrontal cortex and the hippocampus, where they were lower in RLA vs RHA rats, and in the caudate-putamen complex proper where, by contrast, they were higher in RLA vs RHA rats. No statistically significant differences were seen in nucleus accumbens and ventral tegmental area. In tissue sections, trkB-like immunoreactive (LI) labelling was mainly localized to neuronal cell bodies and proximal processes, unevenly distributed in the telencephalic cerebral cortex, the hippocampus, and the ventral tegmentum of the midbrain. Densitometric analysis of immunostained brain sections revealed that differences among the two groups are consistent to a good extent with WB data. As a whole, the finding of a different expression of trkB receptor in the RLA vs RHA rat brains implies the occurrence of an altered neuronal responsiveness to BDNF in specific brain regions and may contribute to outline the molecular and morphological basis for the distinct vulnerability to depression in the two rat lines.

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Keywords

Depression; BDNF; trkB; hippocampus; nucleus accumbens; VTA; western blot; immunohistochemistry.