P02-226

BDNF VAL66MET MODULATES 5-HTTLPR FOR DEVELOP NEUROTICISM IN BORDERLINE PERSONALITY DISORDER

J. Villarroel Garrido[1], H. Silva Ibarra[1], P. Iturra Constant[2], S. Jerez Concha[1], C. Montes Aguirre[1], M. Fuentes Salgado[1], M.L. Bustamante Calderon[1]
[1]Mental Health and Psychiatry, [2]Human Genetics Program, Universidad de Chile, Santiago, Chile

Brain derived neurotrophic factor (BDNF) is the most widely distributed and highly expressed neurotrophin in the CNS. BDNF gene have been associated with increased risk psychiatric disorders. It has been described interaction between BDNF and serotonin system at a neural and genetic level. Neuroticism as a personality trait relevant in borderline personality disorder (BPD) has genetic inheritance and is associated with serotonergic dysfunction. Has been reported that BDNF Val66Met variant is associated with neuroticism in general population. The aim of this study is to test the association between Val66Met and neuroticism and evaluate if the presence of Val66Met allele interacts with polymorphism in promoter region of serotonin transporter gene (5-HTTLPR) for develop neuroticism in BPD. We evaluate personality with NEO PI R in 104 BPD subjects that did not meet criteria for axis I diagnoses and other personality disorders. Genetic analysis of BDNF was performed determining the presence of Val/Val Val/Met and Met/Met BDNF variants. 5-HTTLPR was performed determining the presence of L and S 5-HTTLPR alleles. Statistical analysis were tested with parametric and correlation method with Stata10. We did not found differences in neuroticism between BDNF variants, but when controlled by BDNF alleles we found that Met/Met modulate the expression of 5-HTTLPR, with S-carriers (LS+SS) having higher neuroticism than LL ($F=6.36$, $p=0.0031$). We found no differences in expression of 5-HTTLPR in other BDNF variants. We conclude that BDNF have a differential modulating effect of 5-HTTLPR in neuroticism in BPD.

https://doi.org/10.1016/S0924-9338(11)72527-5 Published online by Cambridge University Press