

Brain Derived Neurotrophic Factor as a predictor of the cognition and prognosis in first episode psychosis after antipsychotic treatment.

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DISCLOSURE

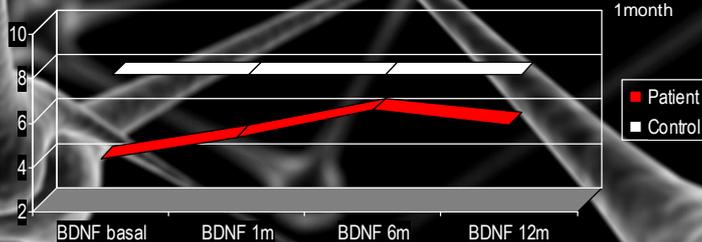
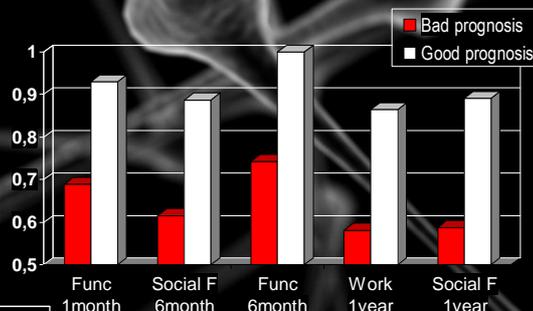
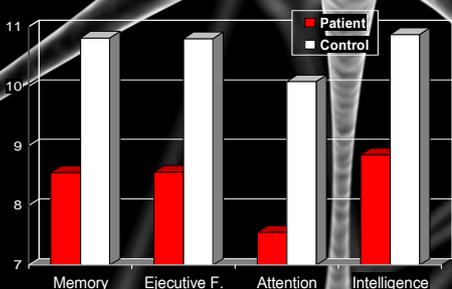
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Purpose of the study:

Brain-derived neurotrophic factor (BDNF), the most abundant neurotrophin in the brain, promotes growth and maintenance of connections, serves as a neurotransmitter modulator, and participates in plasticity mechanisms such as long-term potentiation and learning. The cognition of the patients who suffer a first episode of psychotic (FEP) is altered and there is a reduction in the functioning of the patients. Usually, under this cognitive performance there are neurobiological underpinnings that might underlie these changes. In this study, we analyze the relation between the brain derived neurotrophic factor (BDNF) and the cognitive performance and prognosis in patients with recent psychosis.

Summary of the results:

We observed a positive correlation between BDNF levels after six months of treatment and five cognitive domains: abstract verbal reasoning ($r=0.468$), motor and processing speed ($r=0.397$), learning capacity ($r=0.559$), immediate memory ($r=0.409$) and delayed memory ($r=0.382$). Also, we found that the patients with lower BDNF plasma levels at baseline at 6 months follow-up had worse social activity (0.61 vs. 0.89; $t=-2.137$; $p=0.041$) and functioning (0.69 vs. 0.93; $t= 2.109$; $p=0.044$) and at a year follow-up are related with worse functioning at 6 months (0.54 vs. 0.84; $t=-3.734$; $p=0.001$) and an a year follow-up (0.61 vs. 0.93; $t=-3.043$; $p=0.010$). The BDNF levels increased along the follow up, after the pharmacological treatment (basal-1 month: $Z= -2.88$; $p\leq 0.004$ and 1-6 months: $Z= -2.23$; $p\leq 0.05$).



Methods used:

45 patients with a FEP were selected from the Basque Country. The diagnoses were made using the SCID-I and met the DSM-IV criteria for psychotic disorder. Plasma BDNF levels were measured using the BDNF Sandwich ELISA Kit. All patients were assessed clinically three times over a year using the following scales: PANSS, GAF and Strauss Carpenter. Also, a battery of cognitive tests (Wechsler Memory Scale and Wechsler Adult Intelligence Scale, WAIS-III) was applied six months after the acute episode when the patients were clinically stabilized.

Conclusions:

Our results suggest that BDNF is associated with cognitive impairment seen after a FPE and with the prognosis of the illness. When the patients begin the pharmacological treatment, the BDNF levels increase significantly but it's necessary, at list, 6 moths of treatment to obtain normal levels. Further investigations of the role of this neurotrophin in the symptoms associated with onset of psychosis are warranted. These neurotransmitters could be offer us a relevant key for the pharmacological treatment in the psychotic diseases.