# Brain-derived neurotrophic factor

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Brain derived neutrotrophic factor (BDNF) is a molecule highly implicated in the development of bipolar disease because it is highly expressed in brain tissues that are pathological in such condition. Later on, neuroimaging, serum, epidemiological, and family studies have supported this theory as well. First, changes on BDNF gene have caused behavioral changes. In addition, association between BDNF and several treatment disorders support the molecule's role as a target for therapy. Moreover, BDNF concentration is positively correlated with the brain's structural integrity. Because of the multiple evidences implicating BDNF to BD, several studies have looked into BDNF in a genetic level to better understand the disease. Many studies have found that a change from val66val to val66met is highly critical in the development of BD. In particular, a study of Matsuo and Frey (2009), using free sulfur MRI, has compared 60 bipolar subjects with 99 healthy patients to determine the effect of val66val or val66met on hippocampus, cingulate, and amygdala. Based on these previous findings, this research hypothesizes that, upon neuroimaging through fMRI, there will be considerable decrease in the volumes of hippocampus, amygdala and cingulate among val66met patients as compared to healthy, val66val, individuals.

DISCUSSION

Brain derived neutrotrophic factor (BDNF) binding to TrkB is an important component of phospholipase Cg, mitogen-activated protein kinase/extracellular signal-regulated protein kinase (MAPK/ERK) and phosphoinositide 3-kinase pathways, all transducing signals into and out of cells. With roles in transmitting signals, changes in BDNF will thus be highly manifested as changes in memory, behavior and cognition.

Bipolar disease (BD) is a pathologic condition of the nervous system caused

by both environmental and biological factors. It affects structures in the brain that affect mood, cognition and behavior such as the limbic system, striatum, pallidum, thalamus and cortex. Particularly, highly implicated in the development of disease is the impairment of neuronal survival and synaptic plasticity, as mediated by BDNF, high quantities of which are found in cortex, limbic structures, olfactory bulb, and cerebellum. Briefly, these parts are important in attention, memory, smell perception, and emotions, respectively.

Because BDNF is found at high concentrations on brain areas highly affected during BD, many studies have looked into its particular characteristics with and without the disease. Modification of BDNF gene has an antidepressant activity, and stress was found to decreases BDNF concentration. In addition, clinically used treatments for mood disorders, such as valproate, lithium, electroconvulsive therapy and magnetic stimulation interfere with BDNF expressed in the frontal lobe or in the hippocampus. Serum BDNF levels are also positively associated with the concentration of N-acetyl aspartate, a marker of neuronal integrity, and choline, a marker of cell membrane turnover, in the anterior cingulate cortex (ACC), suggesting that peripheral BDNF content may be a potential marker of cerebral cortical integrity.

Because of the multiple evidences implicating BDNF to BD, several studies have looked into BDNF in a genetic level to better understand the disease. Many studies have found that a change from val66val to val66met is highly critical in the development of BD. In particular, val66met was found to cause lower cognitive task scores, partly because it results to lower volumes of the limbic system (hippocampus and parahippocampus), dorsolateral prefrontal cortex, and temporal lobe as compared to that of the val66val variety. As

well, low levels of creatinine and phosphocreatinine were found on the dorsolateral prefrontal cortex of val66met patients, thus suggesting an impairment of energy metabolism in the area.