The Role of Brain-Derived Neurotrophic Factor in Depression

Katie Mendez
Otterbein University, katie.mendez@otterbein.edu

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Katie Mendez, RN
Otterbein University, Westerville, Ohio

INTRODUCTION


PATHOLOGICAL PROCESS


UNDERLYING PATHOLOGY

The neurobiology mechanisms of depression are not well known but are hypothesized to be a combination of genetic and environmental factors. For the purpose of this paper, the Hypothesis of Hypothyroidism has been the most widely accepted model. This hypothesis is based on a deficit or imbalance of monounsaturated fatty acids, neurotrophic factor, and dopamine. The enhancing effects of anti-antidepressant medications on this system provides the primary support for this relationship. Antidepressants have been found to prevent antideprenants only occurs after weeks to months, therefore, early chronic use is ineffective. This phenomenon suggests that lasting changes in gene expression are required for antidepressant efficacy (Mark and Brodvin, 2011). Research within the last decade has expanded the earlier Monounsaturated Hypothesis to integrate the gene transcription element which has been found to play a critical role in the diagnosis and treatment of depression. The Monounsaturated Hypothesis of Gene Expression has pointed to the role of neurotrophic factors, particularly brain-derived neurotrophic factor. The Neurotrophic Hypothesis of Neurodevelopment further explores the role of BDNF in neurodevelopment, physiological adaptation, and depression (Mark and Aragon, 2012).

BDNF is normally synthesized as a precursor (pro-BDNF) which then is cleaved by the non-esterified form of BDNF (mBDNF). mBDNF then binds to two different receptors at the target cell, TrkB and p75. mBDNF's trophic effects are mediated by TrkB receptors which are involved in promoting synaptic plasticity and regulating protein synthesis dependent long-term potentiation, while pro-BDNF binds to high affinity coreceptors which are linked to the induction of apoptosis (Hill, 2012).

SIGNIFICANCE OF NEUROPROTECTION

Research has found that BDNF expression in the hippocampus is increased in response to acute antidepressant treatment (Kumag, et al., 2011). BDNF levels have been found to increase in patients with depression and also correlate with the severity of the depression. BDNF levels after chronic antidepressant treatment increase to a level found in healthy, non-depressed individuals. Due to the central role of BDNF in the antidepressant therapy, this coreceptor may provide the basis to new strategies for antidepressant treatment to determine efficacy (Kurita, Nishino, Kato, & Sato, 2012). Gender specific consideration also warrants further investigation, estrogen receptors co-localize with BDNF, synthesizing neurons and induce BDNF expression in hippocampal regions with estrogen deficiencies or of premenopausal age may present uniquely (Kurita, et al., 2010). Additional therapeutics that have been reported to increase BDNF expression include astrocyte-derived supplementation, omega fatty acids, regular exercise, and caloric restrictions (Ranaeth, et al., 2014). The BDNF-Link has been hypothesized to improve the beneficial effects of stress reduction in the treatment of depression, thus regular exercise may serve as a dual purpose in improving BDNF levels as well as providing additional benefits of stress reduction in the treatment of depression. 

CONCLUSION

The pathophysiology of depression plays an integral role in explaining the effects of exercise and antidepressant treatments. The underlying pathological mechanisms that may be involved. The effects of BDNF levels in depressed patients may provide resources for additional screening tests, diagnostic tests, and tests for monitoring treatment efficacy. The HPA axis functionally may also provide additional tests to determine what roles, if any, it plays in contributing to the pathogenesis of depression. First, the inclusion of education on contributing factors, symptomatic treatment, additional antidepressants, and lifestyle modifications may lead to better disease management.

REFERENCES


Otterbein, University, Westerville, Ohio