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The Role of Brain-Derived Neurotrophic Factor in Depression

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INTRODUCTION

According to The World Health Organization, depression is the fourth leading cause of global burden of disease and the second leading cause of disability worldwide. Major Depressive Disorder (MDD) is the most prevalent mood disorder, results from the National Health and Nutrition Examination Survey reflect a 12.6% lifetime prevalence. Depression can range from mild to severe and can be episodic or chronic. Depression has a high rate of comorbidity with multiple chronic diseases and other mental health conditions. Research has shown that individuals are more likely to seek treatment for depression in a primary care setting than a mental health specialty clinic, especially individuals of ethnic and racial minority populations. Approximately 50% of individuals suffering from depression are not receiving pharmacological or psychological treatment (Shim, Bulbun, Ye, & Raitt, 2011). Current research is investigating the role of brain-derived neurotrophic factor (BDNF) and the hypothalamic-pituitary-adrenal axis (HPA), and its role in the diagnosis, progression, and treatment of depression (Kunugi et al., 2010; H. Hill, 2011). The hypothalamic-pituitary-adrenal axis plays an integral role in explaining the patients disease process. Neurotrophic factor Hypothesis has provided further insight into the environmental factors involved in depression. As a result of this research, particularly brain derived neurotrophic factor. The Neurotrophic Hypothesis of Neurodevelopment further explores the role of BDNF in neurodevelopment, psychiatric disorders, and mental illness (Brovedani, 2011). BDNF is synthesized in a precursor (pro-BDNF) which then is cleaved to the mature form of BDNF (mBDNF). mBDNF then binds to its receptors which are linked to the induction of apoptosis (Hill, 2011).

UNDERRLYING PATHOLOGY

The neurobiology mechanisms of depression are not well known but are hypothesized to be a combination of genetic and environmental factors. For the past fifty years the Monoamine Hypothesis of Gene Expression has pointed to the role of antidepressants efficacy changes in gene expression are required for antidepressants efficacy (Kunugi, 2011). Research has shown that increased BDNF in the hippocampus is linked to decreases in depression (Kunugi, Y., & Sato, T., 2012). It is unclear whether reduced hippocampal volume is a result of depression or a cause but growing research supports a direct link between decreased hippocampal volume and depression. One of the hypothetical frameworks which links decreased BDNF with depression is the Hypothalamic-Pituitary-Adrenal Axis hypothesis and Brain-Derived Neurotrophic Factor Hypothesis. This hypothesis provides further insight into the environmental factors involved in depression. Acute and chronic stress can induce perturbations of the hypothalamic-pituitary-adrenal (HPA) axis with a resultant increase in glucocorticoids in the bloodstream. This increase in glucocorticoids leads to the release of BDNF and interactions with its binding to the TrkB receptor, thus decreasing BDNF signaling and decreasing the negative feedback loop, increased glucocorticoids in the bloodstream cause an increase in glucocorticoid levels which perpetuates a vicious cycle of continual production of glucocorticoids in the bloodstream. The resultant increase in glucocorticoid levels is the hypothalamus and the pituitary gland are signaling the HPA axis to down regulate cortisol production. This leads to decreased BDNF and depression. One of the major implications of this hypothesis is that antidepressants only occurs after weeks of treatment and the acute use is effective. This phenomenon suggests that lasting changes in gene expression are required for antidepressant efficacy (Masi, 2010). BDNF levels are decreased in patients with depression and also correlate with the severity of the depression. BDNF levels after chronic antidepressant treatment increase to the levels found in healthy, non-depressed individuals. Due to the central role of the HPA axis in depression treatment, antidepressant therapy can only be given to baseline BDNF to target a neural network toward depression treatment to determine efficacy (Kunugi, Kitoh, Kotera, & Numata, 2012). Gender specific considerations also warrant further investigation, estrogen receptors co-localize with BDNF synthesizing neurons and induce BDNF expression in the hippocampus, while estrogen deficiencies or of post-menopausal age may present uniquely (Bridges, 2010). Additional therapies that have been reported to increase BDNF include diet modification, exercise, and corticosteroid supplementation, omegas fatty acids, regular exercise, and caloric restrictions (Rainhart, 2014). The HPA-BDNF link is also associated with the benefits of stress reduction in the treatment of depression, thus regular exercise may be used to increase BDNF and decreasing stress. Providers should consider the use of exercise regimen can also be very useful as adjunct therapies.

SIGNIFICANCE OF NURSING CARE

Educate Patients Regarding:

• The importance of antidepressant medication compliance and continual treatment.
• The dual benefits of regular exercise.
• The benefits of a healthy diet within their specific diagnosis of depression.
• The role stress plays in depression and the beneficial mechanisms of regular relaxation therapy to their daily routine.
• The basic pathological process involved in depression to assist the patient with better understanding of the disease and appropriate coping mechanisms.

REFERENCES


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CONCLUSION

The pathophysiology of depression plays an integral role in explaining the affective, cognitive and somatic symptoms and implications 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 functionality may also provide additional testing tools to determine what role stress, weight, or dietary changes plays in the contributing to the patients disease process. Lastly, the inclusion of education on symptoms, contributing factors, additional therapies, and lifestyle modifications may lead to better disease management.

ADDITIONAL SOURCES

