The Role of Brain-Derived Neurotrophic Factor in Depression

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

Follow this and additional works at: https://digitalcommons.otterbein.edu/stu_msn

Part of the Medical Pathology Commons, Mental and Social Health Commons, and the Nursing Commons

Recommended Citation
Mendez, Katie, "The Role of Brain-Derived Neurotrophic Factor in Depression" (2014). Nursing Student Class Projects (Formerly MSN). 31. 
https://digitalcommons.otterbein.edu/stu_msn/31

This Project is brought to you for free and open access by the Student Research & Creative Work at Digital Commons @ Otterbein. It has been accepted for inclusion in Nursing Student Class Projects (Formerly MSN) by an authorized administrator of Digital Commons @ Otterbein. For more information, please contact jwu@otterbein.edu.
INTRODUCTION

According to The World Health Organization (2012), the leading cause of total disease burden and the leading cause of disability worldwide. This burden is due to the negative effects of so-called chronic diseases. The burden of chronic diseases is felt more acutely in ethnic and racial minority populations.

Current research shows that individuals are more likely to seek treatment for depression in a primary care setting as compared to a mental health specialty clinic, especially individuals of ethnic and racial minority populations. Approximately 50% of individuals suffering from depression are receiving no pharmacological or psychological/psychosocial treatment services (Shim, Roh, & Rust, 2011). Current research is investigating the role of brain-derived neurotrophic factor (BDNF) and the hypothalamic-pituitary-adrenal (HPA) axis, and its role in the diagnosis, progression, and treatment of depression (Komiya, Kitajima, & Takeda, 2010) (Komiya, Kitajima, & Takeda, 2010) (Komiya, Kitajima, & Takeda, 2010). This research provides insight into the mechanisms of action of antidepressant medications and expands the available knowledge to facilitate more thorough patient education regarding the benefits of treatment.

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 research, The Hypothesis of Depression has been accepted. The hypothesis is based on a deficit or imbalance of monoamine neurotransmitters, noradrenaline, dopamine, serotonin, and GABA. The enhanced effects of antidepressant treatments for depression and the specific system providers the primary support for this hypothesis, the use of antidepressants only occurs after weeks to months of treatment. Therefore, an early chronic use is ineffective. This phenomenon suggests that lasting changes in gene expression are required for antidepressant efficacy (Masi, 2011). Research within the last decade has expanded the earlier Monamine Hypothesis to include the gene transcription mechanism which has been found to play a critical role in the diagnosis and treatment of depression.

The Monamine Hypothesis of Gene Expression has pointed to the role of neurotrophic factors, particularly brain derived neurotrophic factor. The Neurotrophic Hypothesis of Neuronal Development further explores the role of BDNF in neuronal development, plasticity, and neurogenesis (Masi, 2012). Neurotrophic factors are synthesized as a precursor (pro-BDNF) then cleaved into BDNF and the mature form of BDNF (mBDNF). mBDNF then enters the synaptic vesicle at the target cell, TrkB and p75. mBDNF's trophic effects are mediated by TrkB receptor activation which is involved with promoting synaptic plasticity and regulating protein synthesis dependent long-term potentiation, while pro-BDNF binds to high affinity receptors which are linked to the induction of apoptosis (Hill, 2012).

BDNF is present in multiple tissues and organs, including brain tissues, kidney, liver, intestine, blood, and adipose tissue, and the adipose tumor necrosis, and the prostate, and the central nervous system. The highest concentration of BDNF is found in the dentate gyrus of the hippocampus, the cerebral cortex, and the basal forebrain. The majority of neurons are formed prenatally and continue to be born throughout life, particularly the hippocampus and the olfactory bulb. The ability to give birth to new neurons from neural stem cells, neurogenesis, is a hallmark of active neurotrophic factors involved in stimulating brain plasticity and neurogenesis. Patients with depression are found to have a reduction of hippocampal volume, decreased hippocampal neurogenesis, thus decreased neurogenesis (Masi & Breiding, 2012).

It is unclear whether reduced hippocampal volume is a result of depression or a cause or growing interest in gene expression research. This research supports the direct between decreased BDNF and depression. One of the hypothetical frameworks which links decreased BDNF with depression is the Hypothalamic-Pituitary-Adrenal Axis abnormalities and Brain-Derived Neurotrophic Factor Hypothesis. This hypothesis provides further insight into the environmental factors involved in depression. Acute and chronic stress can induce perturbation of the hypothalamic-pituitary-adrenal (HPA) axis with a resultant increase in the expression of BDNF and its interaction with the hippocampus to TrkB-receptor, thus decreasing BDNF signaling function (Masi, 2012). It is possible that a negative feedback loop, increased glucocorticoids in the bloodstream can cause negative effects on the hypothalamus and the pituitary gland which can lead to the development of corticosteroid releasing hormone and adrenocorticotropic hormone (ACTH). The increased ACTH will then cause the hippocampus also plays a role in this negative feedback loop by signaling the HPA axis to upregulate glucocorticoid production. The hippocampus has a high density of glial cells and increased cortical levels have been shown to decrease hippocampal volume, thus contributing to the hippocampus disease process. Lastly, the inclusion of factors which is involved in contributing to the other diseases, may include major depressive disorder, bipolar disorder, and schizophrenia.

REFERENCES


Babko, A., & Bubnov, Y., & Ye, & Rust, 2011). Current research is investigating the role of brain-derived neurotrophic factor (BDNF) and the hypothalamic-pituitary-adrenal (HPA) axis, and its role in the diagnosis, progression, and treatment of depression (Komiya, Kitajima, & Takeda, 2010) (Komiya, Kitajima, & Takeda, 2010). This research provides insight into the mechanisms of action of antidepressant medications and expands the available knowledge to facilitate more thorough patient education regarding the benefits of treatment.

IMPLICATIONS FOR 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 with essential nutrients in the treatment of depression.
• The role stress plays in depression and the subsequent need for 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.

CONCLUSION

The pathophysiology of depression plays an integral role in explaining the effects of treatment and antidepressant 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 functionality may also provide additional and internal tests to determine what role stress plays in the production of depression.

Shen, R., Babko, A., Ye, & Rust, 2011). Current research is investigating the role of brain-derived neurotrophic factor (BDNF) and the hypothalamic-pituitary-adrenal (HPA) axis, and its role in the diagnosis, progression, and treatment of depression (Komiya, Kitajima, & Takeda, 2010). This research provides insight into the mechanisms of action of antidepressant medications and expands the available knowledge to facilitate more thorough patient education regarding the benefits of treatment.