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**New Ways of Predicting Efficacy of Antidepressants**

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**Abstract**

Major depressive disorder (MDD) is a neuropsychiatric condition that is becoming increasingly prevalent in society. According to the National Institute of Mental Health, MDD is “common and devastating” (p. 61) and has a very complex pathophysiology. Currently, a definitive strategy that can be used to treat MDD has not been identified. One key indicator of mitochondrial dysfunction (BDNF) and cognitive-emotional biomarkers may be a key to the mechanism of this disorder.

A literature review of articles found in PubMed, CINAHL, Cochrane Library, and PsycINFO has shown that in the last decade, there has been an abundance of research showing that levels of BDNF are lower in depressed patients versus healthy controls and in depressed patients before treatment versus after treatment. Measuring BDNF levels before and during the course of treatment may help to better treat depression and in the future, provide new ways to predict treatment options that will be effective for patients who will work.

**Introduction**

In the last decade, there has been an abundance of research showing that levels of BDNF are lower in depressed patients versus healthy controls and in depressed patients before treatment versus after treatment. Measuring BDNF levels before and during the course of treatment may help to better treatment and in the future, provide new ways to predict treatment options that will be effective for patients who will work.

**Literature Review**

**Pathophysiology of Major Depressive Disorder**

- Monotone-deficiency hypothesis is partly explained through the pathophysiology of MDD.
- Other hypotheses include inflammation cytokines, hypothyroid-pituitary-adrenal axis, glutamate receptors, BDNF dysfunction, increased apoptosis, and vitamin D dysregulation.
- Rif (2009) stated that polymorphisms of BDNF affects the intracellular transport and secretion of BDNF and may increase depression vulnerability.

**Brain-Derived Neurotrophic Factor**

- CASTANEC et al. (2018) found that both SNRIs and SNRIs reduced the effective antidepressant in MDD and significantly improved the global cognitive function. Both SNRIs and SNRIs improved executive function and verbal memory, however, this improvement was independent from the efficacy of affective symptoms and brain derived neurotrophic factor (BDNF) levels in MDD and may accurately predict the efficacy of antidepressant medications and the remission of depressive symptoms.

**Statement of the Problem**

- According to Cai et al. (2015), “MDD can reduce the capacity of a patient to study, work, and engage in social activities” (p. 61). It also increases the risk of suicide and disability rate and has a very significant impact on quality of life, reducing the quality of life and the patient's ability to function in daily life (Cai et al., 2015).

- The Centers for Disease Control and Prevention (2016) states that almost 10% of adults age 18-24 are not receiving current treatment.

**Research Questions**

- In adults with MDD, does BDNF play a role in the pathophysiology of MDD?
- In treatment of adults with MDD, does cognitive biomarkers predict the efficacy and outcome of treatment and remission?
- In treatment of adults with MDD, does BDNF predict the efficacy and outcome of treatment and remission?

**Discussion**

- Both Cao et al. (2015) and Roz et al. (2009) stated that the pathophysiology of MDD has been mainly based on the monoamine-deficiency hypothesis. This hypothesis focuses on decreased levels of serotonin, norepinephrine, and dopamine being the cause of MDD. However, there are antidepressant medications to alleviate all of these.

- Rinke et al. (2015) stated that neurotrophic, such as BDNF, regulate growth in order to support neuronal plasticity in the CNS and PNS. BDNF levels are increased, an improvement in the brain, and in only a small number of patients that actually reach remission. This suggests that there is more to the pathophysiology of depression.

- Rinke et al. (2015) stated that neurotrophic and neuropeptides are involved in memory and learning, and its demand as with decreased BDNF levels, MDD may occur.

- Eke et al. (2015) stated that “response with antidepressant medication can be reliably predicted for outpatients with MDD by treatment performance on a self-rating scale of cognitive and emotional function”.

- Glyn et al. (2016) showed that activation in the frontoparietal region of the brain was positively related with antidepressant response, particularly SNRIs.

- Overall, the review of literature showed that BDNF levels were decreased in patients with MDD. It also showed that these BDNF levels increase over time with antidepressant treatment with certain medications. These results may offer a tremendous gain in the treatment of adults with MDD and may finally be able to help find treatment options for patients that will work.

**Applicability to Clinical Practice**

- In clinical practice, MDD is a disease process that will be encountered many times in a provider’s career. It can be treated with antidepressant medications that are available on the market.

- Based on cognitive and emotional tests performed prior to antidepressant treatment, studies have been able to predict treatment response to certain SSRIs and SNRIs.

- BDNF is decreased in patients with MDD and certain antidepressants are able to increase this level. Some studies showed that a higher BDNF level showed a larger antidepressant effect.

- If providers are able to predict whether certain medications will work before starting them, it may help to provide more effective treatment.

- Also, measuring BDNF levels before and during the course of treatment may help providers to predict if antidepressant treatment will work much sooner than has previously been possible.

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**References**


Rotkova-Dziubanowska, G., Szulc, P., Grajek, P., Wrobel, A., Potocki, K., Brzezinska, M., ... Dunaj, T. (2015). Neurological and psychiatric disorders: new possibilities of major depression treatment and BDNF mRNA levels are normalized by antidepressant treatment, while BDNF showed partial hypofunction. Thus, SSRI and SNRI responders showed opposing patterns of activation in the anterior cingulate.

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