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Vitamin D's Therapeutic Potential in the Treatment of Depression and Anxiety: Molecular Basis

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Introduction

In this study, depression and anxiety will collectively be referred to as major depressive disorder (MDD) and anxiety disorders, which are devastating and highly prevalent clinical entities that rank among the world's top causes of disability. Patients with these two comorbidities usually report with symptoms that are more severe and last longer than those who do not have them. These illnesses regularly co-occur and their symptoms frequently overlap in persons. Therefore, it is still difficult to diagnose and treat these illnesses in a clinical environment. Despite being distinct disorders, the causes of depression and anxiety have many of the same traits, including biological mechanisms, contextual influences, and genetic predispositions [1]. Among the primary molecular processes thought to be involved in the pathogenesis of these illnesses, strong evidence has suggested that neuroinflammation plays a key role a critical element in the start and development of many illnesses. Notably, a neuroinflammatory process may be the cause of other biological processes that have been linked to depression and anxiety, including gut dysbiosis, impaired neurogenesis, and monoaminergic dysfunction. This raises new questions about potential molecular targets and neuroprotective treatments for these mood disorders. Due to its antioxidant, anti-inflammatory, pro-neurogenic, and neuromodulatory qualities, vitamin D has been more well-known in recent years due to its impact on depression and anxiety [2]. Given this context, we highlight in this review the key pathways that may underlie vitamin D's possible depressive and anxiolytic effects. Additionally, we go over preclinical and clinical research that back up this drug's therapeutic potential vitamin to treat these mental health issues.

Description

The steroid hormone vitamin D is ingested in two forms: vitamin D2 (ergocalciferol), which is present in yeast, mushrooms, and plants, and vitamin D3 (cholecalciferol), which is present in foods of animal origin such cod liver oil and oily fish. However, cholecalciferol, which is created internally by exposure to solar ultraviolet B (UVB) light at wavelengths of 290-315 nm, is the principal source of vitamin D. UVB photons enter the dermis through this mechanism, where they are absorbed by 7-dehydrocholesterol and transformed into previtamin D3. Vitamin D-binding protein (DBP) then transports vitamin D3 in the blood when previtamin D3 isomerizes into it [3]. Vitamin D3 is mostly hydroxylated at C-25 in the liver by the enzyme 25-hydroxylase To create 25(OH)D3, cytochrome P450 Family 2 Subfamily R Member 1 (CYP2R1) is required. Following this, 25(OH)D3 can either be stored or put through a second hydroxylation by 25-hydroxyvitamin D-1-hydroxylase in the kidneys, resulting in the biologically active form of 25(OH)D3 known as 1,25(OH)2D3. Humans are thought to be able to produce enough of this vitamin to take in 20,000 IU orally.

It is well known that vitamin D contributes significantly to calcium-phosphorus metabolism and helps to maintain bone homeostasis. Additionally, vitamin D also has pleiotropic effects that have attracted more attention recently. Vitamin D serves as a conduit for calcitriol's biological effects. VDR receptors. Other transcription factors, such as the retinoid X receptor (RXR), can interact with this complex and create a heterodimer as a result of the interaction between calcitriol and VDR. By attracting complexes of either co-activators or co-repressors, this heterodimer can bind to vitamin D-responsive elements (VDREs) and influence gene expression [4]. 200–2000 genes are thought to be directly or indirectly regulated by vitamin D. Vitamin D mediates a wide range of biological processes, making blood vitamin D concentration measurements crucial. In this regard, vitamin D deficiency is defined as values of 20 ng/mL, insufficiency as values of between 21 and 29 ng/mL, and adequacy as values of between 30 and 100 ng/mL by the Endocrine Society's Clinical Practice Guideline. Reference values for vitamin D, however, are still an issue of debate The symptoms of sadness and anxiety linked to a number of medical illnesses, such as type II diabetes, Crohn's disease, ulcerative colitis, and obesity, have been found to be improved by vitamin D supplementation in several trials.

Vitamin D's potential therapeutic benefits for patients with major depression or anxiety, however, continue to be debatable. For instance, supplementing patients with vitamin D (1600 IU daily for 6 months) significantly reduced anxiety symptoms but not depressed symptoms. Similarly, supplementing with 2800 IU of vitamin D did not significantly lower Hamilton D-17 scores in patients with depression. However, treatment with 50,000 IU of vitamin D for two weeks reduced the severity of depression as measured by the Beck Depression Inventory BDI-II), even though no variations in serotonin levels were found. In contrast, cholecalciferol therapy dramatically reduced BDI scores in women with moderate, severe, and intense depression while considerably raising serum serotonin levels [5]. It's interesting to note that only men with severe depression were found to benefit from vitamin D treatment in terms of less severe depressed symptoms. Elderly persons (over 60 years of age) with depression have also seen positive results from vitamin D treatment.

Conclusion

In this review, we covered the key research highlighting vitamin D's therapeutic potential in the treatment of anxiety and depressive disorders. Of special importance, strong evidence points to vitamin D's potential to work similarly to traditional antidepressants by having antioxidant, anti-inflammatory, pro-neurogenic, and neuromodulatory qualities. Numerous preclinical studies that corroborate this claim have demonstrated the advantages of vitamin D supplementation in animal models of various mood disorders.

However, there are very few clinical trials that evaluate the effectiveness of vitamin D supplementation in the treatment of depression and anxiety, and their findings can be inconsistent controversial. Future research is required in order to develop a vitamin D supplementation technique that is helpful in preventing or lessening depression and anxiety symptoms in light of the inconsistent outcomes from clinical trials. Therefore, to determine the genuine therapeutic efficacy of this vitamin in the setting of depression and anxiety,

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adequate randomised clinical trials testing the potential of vitamin D as a co-adjuvant for the treatment of these mood disorders are necessary.

Conflict of Interest

None.

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