Novel Approaches in Drug Designing & Development
ISSN: 2575-8942

Juniper Publishers
key to the Researchers

Research Article
Volume 5 Issue 5 - March 2021
DOI: 10.19080/NAPDD.2021.05.555672

Nov Appro Drug Des Dev

The Effect of Vitamin D Deficiency on The Nervous System and Cardiovascular System

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Submission: January 22, 2021; **Published:** March 05, 2021

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Abstract

Vitamin D is one of the most effective vitamins in the body on the nervous system and cardiovascular system. Its deficiency can be life threatening. Vitamin D deficiency is associated with Adiponectin, LDL, TG, VLDL, HDL. Vitamin D deficiency in the elderly can lead to forgetfulness, depression and mental health problems. In this article, we have a brief and practical overview of the effects of vitamin D on the nervous system and cardiovascular system.

Keywords: Vitamin D deficiency; Cardiovascular system; Nervous system; Blood pressure, Lipoprotein

Abbreviations: CVD: Cardiovascular Disease; CKD: Chronic Kidney Disease; IHD: Ischemic Heart Disease; BP: Blood Pressure; AF: Atrial Fibrillation; TG: Triglyceride; LDL: Low density lipoprotein; VLDL: Very Low-Density Lipoprotein; HDL: High Density Lipoprotein; HCY: Homocysteine; HbA1c: Hemoglobin A1c (glycated hemoglobin); VDR: Vitamin D Receptor; SNP: Single Nucleotide Polymorphism; CCA-IMT: Common Carotid Intimal Medial Thickness; hsCRP: High Sensitivity C-Reactive Protein; PTH: Parathyroid Hormone; MI: Myocardial Infarction; HF: Heart Failure; CRP: C-Reactive Protein; IL: Interleukin; CHD: Coronary Heart Disease; SCD: Sudden Cardiac Death; CAC: Coronary Artery Calcification; SBP: Systolic Blood Pressure; LV: Left Ventricular; UVB: Ultraviolet-B; TC: Total Cholesterol; MPV: Mean Platelet Volume; ERI: Erythropoietin Resistance Index; FMD: Flow Mediated Dilation; AIX: Augmentation index; SEVR: Sub Endocardial Viability Ratio; PWV: Pulse Wave Velocity; RHI: Reactive Hyperemia Index; DRDD2 : Dopamine Receptor D2; MS : Multiple Sclerosis; MS NAWM : MS Normal-Appearing White Matter; ADHD: Attention Deficit Hyperactivity Disorder; MMSE: Mini-Mental State Examination; NAD: Non-Alzheimer Dementia

Introduction

To date, much research has been done on the mechanism and tissues of the effect of vitamin D, and many experts emphasize on maintaining the natural level of this vitamin in the body.

The heart can be severely affected by vitamin D deficiency, which causes CVD. People with CKD are more likely to develop cardiovascular disease, which researchers believe can help reduce vitamin D intake [1]. Epidemiological findings indicate an increase in mortality due to IHD and BP in patients who are further away from the equator and less exposed to sunlight, resulting in less vitamin D synthesis in their body [2]. Vitamin D deficiency can be treated with Non-Valvular AF [3] and correlation of flow in the coronary arteries [4].

Normal levels of vitamin D are important for the nervous system. Vitamin D deficiency can cause various cancers, including glioma [5]. Vitamin D deficiency has been reported in pregnant women taking anticonvulsant drugs. Also, the percentage of autism in these mothers' infants is higher than others [6]. People with migraines should pay attention to their poor vitamin D levels because its deficiency can be one of the causes of migraines [7].

The skin as a tissue is very important in direct contact with vitamin D. Vitamin D can prevent psoriasis [8]. Extensive research has been conducted on women and men in the United States. The study showed that there was no significant relationship between vitamin D levels and skin cancer as expected [9]. It should be noted that obese people receive less vitamin D [10], which is more exposed to skin risks due to deficiency of this vitamin.

In this article, we review the effects and effects of vitamin D deficiency on the cardiovascular system and nervous system.

Association of Vitamin D Deficiency with Cardiovascular System

Some studies on the effect of vitamin D deficiency on CVD have linked this to race. [11,12] For example, an increase in BP in vitamin D deficiency in blacks is half that of whites. [13] Studies have also been performed on gender. Vitamin D intake in men was associated with a reduced risk of CVD, although this was not observed in women [14].

Vitamin D deficiency is associated with obesity [15] and increased TG [16,17] LDL [18], VLDL [19]. However, other studies show these results to be random [10,20-24]. Therefore, vitamin D is associated with the regulation of atherogenic fats and primary markers of CVD [25].

VDR is expressed throughout the vascular system. The calcitriol-VDR complex prevents the proliferation of vascular smooth muscle cells, reduces coagulation, and has anti-inflammatory properties. [26] Organic changes in VDR are associated with vitamin D deficiency and are more common in bb genotypes than genotypes. Bb and BB are more deficient in vitamin D in Bsml SNP gene and aa genotype than Aa and AA genotypes in Apal SNP gene [27].

Vitamin D deficiency in patients with CKD is associated with inflammation [28] and albuminuria [29]. Patients with CKD have higher levels of CCA-IMT, hsCRP, and CD4 + CD28 + null cells, and there is a strong inverse association between low levels of vitamin D in these patients and increased factors. It has been reported to be the cause of atherosclerosis in these patients [30].

Vitamin D levels are inversely proportional to PTH levels [31]. Patients with higher PTH levels are at higher risk for CVD, and vitamin D deficiency with secondary hyperparathyroidism can cause CVD [32]. But it was not related to HF but was the opposite in PTH [33]. In some other studies, unlike PTH, vitamin D was not associated with CVD [34].

Severe vitamin D deficiency with high CRP causes CVD, but this effect is not seen for vitamin D alone. Therefore, the association between vitamin D and CVD depends on the inflammatory status [35]. Vitamin D itself plays a role in inflammation with a slight decrease in IL-6 [36]. Vitamins affect the activity and expression of macrophages and lymphocytes in atherosclerotic plaques and cause chronic inflammation of the arterial wall [37].

Vitamin D deficiency is an independent predictor of cardiovascular mortality in ACS patients [38]. Vitamin D deficiency is associated with an increased risk of CAD (not its prevalence) [39] and individuals with vitamin D deficiency are more likely to have coronary artery disease [40]. Vitamin D deficiency is associated with increased risk of HF [41-43], CHD [44], MI [45,46], aortic calcification [47] SCD [48], CAC [49] as well as increased SBP [50]. The effect of vitamin D on IHD [51,52] and AF [53,54] is contradictory. No association was found between vitamin D deficiency and LV diastolic dysfunction and only a slight association was found between vitamin deficiency and interventricular septal thickness [55]. Vitamin D CHD had no effect on secondary cardiovascular events [56].

In patients with metabolic syndrome, the risk of cardiovascular factors increases [57] and vitamin D deficiency is associated with metabolic syndrome [58]. UVB radiation has also been shown to reduce type 2 diabetes by increasing vitamin D levels [59]. However, no other study has shown an association between vitamin D and diabetes [60]. Studies in diabetic patients with vitamin D deficiency have shown weight gain, TC and TG [61] Vitamin D deficiency in these patients was also associated with increased BP and HbA1c. [62] The higher risk of CVD due to vitamin D deficiency in diabetic patients may be due to inadequate heart regeneration.

Vitamin D has an inverse relationship with MPV [63] and ERI [64] and a direct relationship with hemoglobin [65]. Also, vitamin D deficiency causes less FMD and thus causes Vascular endothelial dysfunction [66,67]. Vitamin D deficiency with arterial stiffness indices (AIX, SEVR, PWV) and arterial function branches (FMD, RHI). Vitamin D deficiency was associated with less SEVR, FMD, and RHI, and more AIX and PWV [68,69]. And an abdomen [70].

The real relationship between the effect of vitamin D and CVD is difficult. One of the reasons for the effect of CVD risk factors on serum vitamin D concentration. For example, weight loss reduces cardiovascular risk factors and at the same time increases the concentration of vitamin D due to reduced fat mass [71].

Some studies have not found vitamin D to be associated with CVD [72]. Other studies have found that vitamin D supplementation has no effect on CVD [73-75]. Other studies have found that vitamin D supplementation has an effect on CVD only in people with vitamin D deficiency. It is considered ineffective in people with normal serum levels of vitamin D [76].

Vitamin D Deficiency Can Cause Severe Disorders of The Nervous System

Serum levels of vitamin D are generally measured at serum levels of 25 (OH) D. The borderline level for vitamin D is 56 nmol / L in women but 50 nmol / L in men. Serum levels of Caucasians are higher than serum levels of vitamin D in other breeds. Latitude has no significant effect on serum levels of vitamin D; But the culture of the people of each region is effective based on the way of covering and feeding on their serum vitamin D level [77]. Vitamin D levels in summer compared to winter, younger ages (50-70 years) than older ages (70-87 years), women are different from men, but serum vitamin D levels do not depend on BMI [78]. The use of vitamin D supplements at different ages has different effects on the psychological level of people. The use of vitamin D supplements has no effect on the mental and emotional functioning of adolescents [79]. Taking vitamin D supplements has no effect on mental function and memory in middle-aged people; However, the use of vitamin D supplements and high serum levels of vitamin 25 (OH) D, in aging, has a significant effect

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on reducing the risk of Alzheimer's disease and dementia. In older people with higher serum levels of 25 (OH) D, they perform better at remembering words than older people with lower levels of 25 (OH) D, but higher levels of 25 (OH) D in speaking fluently have mental performance. And the depressive state of the elderly has no effect. Elderly people with lower serum levels of 25 (OH) D are more likely to develop amnesia [80]. Vitamin D deficiency affects the genes of mitochondrial, cytoskeletal and synaptic proteins by affecting the genes of intracellular processes and inter-cell synapses in the brains of adult mice [81]. Taking vitamin D supplements in middle age improves short-term memory but has no effect on the memory factor associated with semantic memory [82]. Among older men and women, people with lower serum levels of vitamin D have slower reaction time, lower endurance, slower gait, and poorer performance-related performance and visual-spatial performance tests. There was no difference in the number of falls between the elderly in the two groups with sufficient serum vitamin D levels and the group with vitamin D deficiency [83]. Elderly people who do not have dementia but have MCI have lower serum concentrations of vitamin D than a group of mentally healthy elderly people. People with higher levels of serum vitamin D are less likely to develop MCI (MCI: Mild Cognitive Impairment) [84]. In the elderly, vitamin D deficiency is seen, as a result of which the brain activities of these people, especially in the field of spatial memory, are less efficient than their peers with sufficient levels of vitamin D [85]. In a group of mice deficient in vitamin D, they had a lower pain threshold than in a control group that received a sufficient vitamin D diet. Vitamin D receptor expression increased at the time of spinal cord injury in both groups, while, as expected, serum 25 (OH) D did not change [86]. Taking vitamin D supplements if the serum concentration of vitamin D is between 50-80 ng / ml, improves sleep; If the serum concentration of vitamin D is more than 80 or less than 50 ng per ml, it can cause sleep disorders [87].

The effect of taking vitamin D supplements is that with a 10-fold increase in the dose of vitamin D, the serum concentration of 25 (OH) D doubles. In the hippocampus of mice with high serum vitamin D levels, the expression of genes involved in cellular communication, synaptic translocation, and G protein-coupled receptor activity is increased [88].

Vitamin D has improved the symptoms of patients with irritable bowel syndrome by affecting the peripheral nervous system, inflammatory processes, and on the other hand, by affecting the central nervous system, the level of anxiety in individuals [89].

Increased expression of vitamin D receptors increases dopamine synthesis by affecting the gene responsible for dopamine packaging and protection; Vitamin D increases dopamine by reducing the expression of DRD2 gene [90].

Vitamin D does not affect the severity of stroke. Vitamin D has an effect on IGF-I, which acts as a neuroprotectant in the stroke ward and improves the post-stroke process [91].

The use of vitamin D supplements in people with migraines reduces the frequency of migraine headaches and the effects that migraine headaches have on the course of life. In people treated with vitamin D supplements, CGRP levels (an important peptide that is higher in people with migraine headaches than in others) were lower than in controls [92].

Numerous results have been obtained in the study of the effect of vitamin D deficiency in the fetal period, from which the opposite can be mentioned. In mice deficient in vitamin D, the interval between bregma and lambda and tyrosine hydroxylase levels, which are involved in the synthesis of dopamine by dopaminergic and adrenergic neurons, is lower than normal due to low tyrosine hydroxylase levels. The number of dopaminergic neurons in the substantia nigra and part of the tegmentum decreases [93]. Mice that were deficient in vitamin D during pregnancy had the same learning as mice that received normal levels of vitamin D during pregnancy, but between 30 and 70 weeks after birth, the hippocampus of the group that received vitamin D during pregnancy They were deficient in vitamin D, had a greater reduction in volume than the other group, and at 30 weeks after birth had a smaller volume of cerebral ventricles than the other group, which disappeared at 70 weeks [94]. Vitamin D deficiency in the fetus leads to changes in the amount of neurotransmitters in different parts of the brain such as increased dopamine, increased noradrenaline in the hippocampus and thalamus and hypothalamus and midbrain, decreased serotonin in the basal ganglia and caudate and putamen, decreased glutamine and glutamate in Different parts of the brain and the increase in serine, glycine and taurine occur only in limited parts of the brain [95].

In female mice deficient in vitamin D, an increase in dopamine transporters is seen in the Putamen, Caudate, and Nucleus accumbens parts of the brain; While there is no difference in other receptors and neurotransmitters in other parts of the brain between mice with normal vitamin D levels and vitamin D deficiency [96].

1-alpha hydroxylase is one of the enzymes that converts vitamins into active forms in the brain. Expression of this enzyme is found only in the cytoplasm of nerve cells and glia. Vitamin D receptors are present almost exclusively in the nucleus of nerve cells and glia and are not present in the cytoplasm of these cells. Most of the vitamin D receptors are found in the superficial granular layer of the prefrontal part of the cerebral cortex, but in the molecular layer of the prefrontal part of the cerebral cortex. The enzyme 1-alpha hydroxylase is found in greater amounts in the molecular layer of the cerebral cortex and in the superficial granular layer. In the molecular part of the cingulate gingiva, there is a relatively large amount of vitamin D receptors. None of the amygdala parts have a receptor for vitamin D, and the enzyme 1-alpha hydroxylase is moderately expressed in the amygdala. All thalamic nuclei have small or moderate amounts of vitamin D and

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1-alpha hydroxylase receptors, but are abundant in the supraoptic and paraventricular nuclei of the hypothalamus, especially in large cells. Vitamin D receptors are not present in the molecular and porcine layers of the cerebellum, but are abundant in the granular layer. The enzyme 1-alpha hydroxylase is moderately found in the molecular layers and cerebellar Purkinje but is either absent or present in small amounts in many cells of the granular layer [97].

Mutations in the vitamin D receptor gene in mice cause many changes in behavior, mental function, balance, etc., which are explained below. Vitamin D can affect balance processes by affecting muscles. By causing a mutation in the vitamin D receptor gene and altering the expression of the vitamin D receptor gene in the balance sections, disturbances in balance at altitude, swimming in a pool that, like other mice, were unable to swim vertically [98]. By mutating the vitamin D receptor gene on one of the mouse chromosomes, the mice did not change their behavior and acted like mice with two healthy vitamin D receptor genes [99].

By deleting the vitamin D receptor gene in mice, apoptosis in various parts of the brain is dramatically reduced; Including gingival cingulate, dentate, hypothalamic and basal nuclei. Due to the decrease in apoptosis, the number of mitotic cells in these areas increases [100]. vidra and vdrb are two paralogs of vitamin D receptors. In zebrafish, vdrb was removed in larvae by MO injection, which resulted in the removal of Meckel cartilage and palatoquadrate, as well as cartilage hypoplasia. By removing both vdra and vdrb by MO injection, removal of cartilaginous structures of the cranial-series was seen. These findings suggest that VDR expression and signaling are involved in the formation of cranial-serial muscles [101]. Mice that were heterozygous for the lack of the vitamin D receptor gene on one of their chromosomes (heterozygous) showed the same results in depression tests as mice that were homozygous for the vitamin D receptor gene (wild type). Whereas mice homozygously lacked the vitamin D receptor gene (null mutant) showed more depressive behaviors than the other two groups in the same tests [102].

Most people with mental health problems are deficient in vitamin D; Most of these mental problems are in the realm of timing and semantic memory. Older people who are deficient in vitamin D are more likely to develop mental illness [103]. The effect of vitamin D deficiency on mental processes in older mice is greater than that of vitamin D deficiency in young mice; But older mice generally had slower reactions than younger mice. Older mice with normal levels of vitamin D had higher levels of anti-inflammatory cytokines and lower levels of proinflammatory cytokines than older mice deficient in vitamin D; However, both anti-inflammatory and pro-inflammatory cytokines were higher in older mice than in young mice [104], which have a significant effect on the course of the disease in MS [105].

24-OHase is one of the genes affecting the expression of vitamin D receptor, which is most expressed in cerebral cortex cells and parts of the hypothalamus such as periventricular and supraoptic nuclei. In glia cells in the brain, vitamin D receptor expression was seen in both control and MS patients; But in MS NAWM patients, cytoplasmic vitamin D receptor expression was seen, which is not present in the control group. Expression of vitamin D receptors in active affected parts is higher than NAWM. In fact, it appears that the vitamin D receptor mRNA in the affected areas was higher than the vitamin D receptor mRNA in the NAWM MS, which probably indicates the formation of the active 1,25 (OH) 2D form in the affected area and increased tissue response in Has been equivalent to this metabolite [106].

In people with MS who have been on vitamin D supplementation for 6 or 12 months and whose serum levels have increased by 50 nmol / L, 57% less than other people have seen an increase in the number of nerve lesions and a recurrence rate of 27% The reduction [107] and the volume of T2 lesions [108] were less during the period and had 0.27% less brain tissue damage [109]. Vitamin D supplementation outside the human body indicates a decrease in IL-17, but in vivo and in the form of high-dose vitamin D supplementation in patients with MS, leads to an increase in IL-17 by 60%. Decreases in IL-17 occur in 40% of individuals, none of whom showed signs of exacerbation of symptoms [110]. Vitamin D supplementation increased TGF-Beta in patients with MS, while there was very little increase in TGF-Beta in the control group. Use of vitamin D supplements in other cytokines including IL-2, IL-4, IL-5, IL-6, IL-9, IL-10, IL-13, IL-17A, INF-gamma and TNF -alfa did not change [111]. Taking high-dose vitamin D supplements over 3 months leads to an increase in serum vitamin D levels, which is positively correlated between vitamin D supplementation and IL-10 logarithm [112]. In patients with MS, patients taking vitamin D supplementation did not experience neurological damage to the CNS, whereas in the group taking vitamin D supplementation, neurological damage was observed [113]. The risk of developing MS depends on the mother's serum level of vitamin D during pregnancy; Thus, mothers who have insufficient serum vitamin D levels during pregnancy and are deficient in vitamin D are more likely to develop MS in their children. [114] Serum levels of vitamin D at birth have no effect on the risk of developing MS [115].

The percentage of people with vitamin D deficiency is highest in winter and lowest in summer; This is while in winter, people are more prone to depression than other seasons. Among patients with depression, the percentage of people who are deficient in vitamin D or inadequate levels of vitamin D is higher than people with normal levels of vitamin D. Post-stroke depression is more common in people with vitamin D deficiency [116]. By placing patients with depression treated with vitamin D, the rate of fatigue, insomnia, physical weakness and feelings of depression in these patients is reduced [117]. A group of people who have been or are suffering from depression are more likely to be deficient in vitamin D than the control group, and people with lower serum levels of vitamin D have experienced longer periods of depression, which is evidence of the effect of concentration and level. Serum

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of vitamin D is on the periods and symptoms of depression [118].

During the 8-week treatment of children with ADHD with vitamin D supplementation in combination with methylphenidate, in the experimental group and methylphenidate supplementation without vitamin D supplementation in the control group, children who took vitamin D supplementation had fewer symptoms in the afternoon than They were in the control group, while the symptoms of morning ADHD were not different between the two groups [119].

Among Swedes, people with autism have lower levels of vitamin D than their siblings who do not have autism. People with autism were more likely to be born in the summer or spring, which had nothing to do with vitamin D levels [120]. In general, the serum level of 25 (OH) D in children with autism is lower than normal, due to which they also have lower serum calcium levels [121]. The use of vitamin D supplements has had a positive effect on improving the symptoms of autism in children; Especially if this supplement is used at a younger age [122]. 2 months after stopping taking vitamin D supplements, the symptoms of the disease in these children worsened, and with continued vitamin D supplementation, their symptoms improved again. In patients with autism, serum levels of 25 (OH) vitamin D are lower than normal serum levels. One way to improve the symptoms of autism is to take vitamin D so that its serum level reaches at least 40 ng / ml; If the serum level of vitamin D is lower, the therapeutic effects of vitamin D will not be observed [123].

Taking a daily supplement of 2000 IU in the first year of life reduces the risk of schizophrenia in men by 77% in adulthood, but such an effect has not been observed in women and other mental illnesses [124]. Schizophrenia, treatment with vitamin D supplementation for 8 weeks, did not change the psychological symptoms and metabolic parameters compared to the control group [125] Among people with schizophrenia, patients with lower levels of vitamin D, more severe negative symptoms and neurological function - Show a weaker cognition [126]. There is an inverse relationship between the risk of schizophrenia and fetal vitamin D levels; Vitamin D deficiency in the fetus increases the risk of schizophrenia. Surprisingly, people with maximal serum vitamin D levels during pregnancy are more likely to develop schizophrenia than fetuses with normal vitamin D serum levels [127].

People with inadequate vitamin D levels or vitamin D deficiency are more likely to have dementia, cognitive decline, and Alzheimer's disease [128,129]. There is no difference in spatial learning between mice with Alzheimer's with a normal diet and with vitamin D supplementation; However, spatial learning was poorer in the group of mice with Alzheimer's disease and on a diet deficient in vitamin D than in the other groups [130]. Taking mematine with vitamin D supplements in Alzheimer's patients improves their mental abilities in MMSE, while taking mematine or vitamin D supplement alone does not affect people's mental abilities [131].

Older people with a serum level of 25 (OH) D borderline or lower lip are more likely to develop NAD. The onset of NAD is not related to the serum level of vitamin D in individuals. (NAD is actually a mental disorder associated with the destruction of the subcortical area, of which Parkinson's is a type of NAD [132].

Serum levels of 25 (OH) D in patients with Parkinson's and Alzheimer's are lower than normal. Serum levels of vitamin D in patients with Parkinson's are even lower than serum levels of vitamin D in patients with Alzheimer's disease [133].

People with serum levels of 25 (OH) D are lower than normal levels are more likely to develop Parkinson's, which is associated with a higher level of serum (OH) D; Thus, people with a serum level of vitamin D of 50 nmol / L are 65% less likely to develop Parkinson's disease than people with a serum level of vitamin D of 25 nmol / L [134]. There are differences between the genotypes of people with Parkinson's disease and healthy people, so that in patients with Parkinson's disease, the frequency of CC + CT replication in the vitamin D receptor gene in Fok1 polymorphism is higher than in healthy people in Hungary; Due to the change in the vitamin D receptor gene, it becomes 3 amino acids shorter than normal and thus affects the structure of the vitamin D receptor and its function. But there was no difference in vitamin D receptor gene in Apal, Taql and Bsml [135].

There was no difference in vitamin D receptor gene in Apal and Taql between healthy people with MS [136].

In patients with epilepsy, vitamin D receptors on cells around blood vessels were lower than in healthy individuals, which was accompanied by a deficiency of vitamin D in the serum of patients with epilepsy [137]. In a group homozygously lacking the vitamin D receptor gene. Seizures and epileptic seizures are more frequent than groups that are homozygous for vitamin D receptor gene or heterozygous for vitamin D receptor gene, which indicates the effect of vitamin D receptor on epileptic brain-related processes [138]. Taking vitamin D supplements in people with epilepsy leads to a 40% reduction in seizures [139].

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