# Association of Vitamin D and Obesity: An Overview

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***ABSTRACT:*** *Vitamin D, a fat soluble vitamin, is an essential nutrient whose deficiency (causes rickets in children) in the modern world is a major problem to be addressed. This vitamin deficiency has been associated with different clinical and pathological conditions. As per the World Health Organisation,Obesity, defined as a body mass index (BMI) of 30 kg/m² or more..In today's world Vitamin D deficiency is most common. All of the body compartments are increased in volume in obesity, so the lower vitamin D is probably a result of dilution in larger body volume while the general stores are adequate.Decreased concentration of vitamin D has been corroboratedin obese subjects when compared to their non-obese counterparts. Obese persons are seen to have decreased calcium and Vitamin D intake in their food. Lower vitamin D in obese people is a well-documented amongst people of different age, ethnicity, and geographic variation. Hypovitaminosis D has been observed in subjects who have undergone bariatric or gastric bypass procedures, in which a malabsorptive state is knowinglyprompted, but there is no evidence in the favour of the fact that obesity is often caused due to reduced absorption of dietary vitamin D.The association between reduced 25-D concentrations and obesity has been well established.The initial doserequired for vitamin D in obese subjects is much higher to achieve the same serum 25-hydroxyvitamin D as normal weight. Hence this review will try to correlate the association of vitamin D with obesity.*

***Key Words:*** *Obesity, Vitamin D, Obese, Body mass index, 25-hydroxyvitamin D*

## Introduction

In today's modern world, obesity is a major problem which is often correlated with the quality of life. In accordance with the proverb "all work and no play makes Jack a dull boy" – the life of every individual is devoid of physical activity and burdened with unhealthy junk food which aggravates obesity. As per WHO guidelines, a body mass index (BMI) of 30 kg/m² or more is categorize as Obese (1). According to reports obesity is a pandemic in Australians and substantial numbers in most developed nations.The increase in obesity rates demonstrates a chief public health concern. The incidence in Europe is 10–25% in men and 10–30% in women (2). The condition is further worsened as Obesity is associated with a number of co-morbidities such as cardiovasculardisease, hypertension, stroke, type 2 diabetes mellitus (T2DM), dyslipidemia, osteoarthritis and some cancers (3).

Vitamin D (VD) is the key molecule involved in the maintenance of bone tissue, as well as for homeostasis of the minerals calcium and phosphorus. It has its receptors have all over the human body. As vitamin D is a result of endogenous synthesis, it is often referred to as a hormone.1,25-dihydroxyVD (1,25(OH)2D), the active form, not only stimulates calcium absorption, osteoclastic bone resorption and osteoblast function and decreases PTH (parathyroid hormone) secretion but also has extraskeletal functions leading to the stimulation of the immune system (4). Reports have established a consistent association between increasing BMI and lower serum 25-hydroxyVD (25D) concentrations but the presence of a causal relationship is still unclear (5). Reports have corroborated an inverse relationship of 25D concentration in serum with fat content of the body (6). There is an evidence demonstrating a decrease of 0.74 nmol/l of serum 25- hydroxyVD for every kg/m² increase in BMI (7).There are several probable explanations about the inverse relationship between increased fat deposition (particularly abdominal) and lower VD concentration but none of these elucidate the entire mechanism. Hence, apart from studies directed towards finding the causal relationship between VD and obesity, more research should be focussed at finding the role of VD in the development and progression of obesity and other morbid and chronic health conditions. This review endeavours to summarise the existing state of documented information regarding the causes of these reduced 25D concentrations, as well as the probable effects of VD supplementation on obesity.

## Why do Obese Individuals have Lower 25D Concentrations?

VD intake has been reported as being lower in obese men, but not women, when compared to their non-obese counterparts (8). Obese persons are seen to have decreased calcium and Vitamin D intake in their

food.

Even though obese and non-obese subjects have similar amounts of VD, in overweight people, VD is distributed into a larger volume, making serum concentrations lower. The form of vitamin D, i.e. 25(OH) D is dispersedin the different compartments of the body of obese subjects (9).

As perthe hypothesis of Wortsman et al., VD, being a fat soluble vitamin remains sequestered in the adipose tissue of the obese subjects leading to a lower concentration of VD in the plasma even if the dermal synthesis of VD does not differ in both obese and non-obese subjects (10).

Concentrations of cutaneous 7-dehydrocholesterol (the substrate converted by ultraviolet light into previtamin D) appear not to vary between obese and non-obese individuals (11). Obese persons doing outdoor exercise have reduced risk of Vitamin D deficiency (12).Patients having Crohn's Disease, celiac disease and other malabsorption syndromes or persons undergoing bariatric surgeries are seen to have decreased absorption of VD.

Another possible mechanism for lower 25(OH) D is decreased hepatic 25-hydroxylation. Studies have documented the loss of 25-hydroxylation in Non-alcoholic fatty liver disease NAFLD.Since NAFLD is often associated with obesity, further studies are required to investigate the condition after VD supplementation (13).

Studies have documented that there is a difference in gene expression in VD metabolizing enzymes between normal weight and obese people. The expression of cytochrome P450 2J2 gene which codes for the enzyme 25-hydroxylase is decreased by 71% and a 49% ($p < 0.05$) decrease in the expression of cytochrome P450 27B1, which codes for the enzyme 1a-hydroxylase in the subcutaneous AT of the obese group compared to lean subjects has been reported (14). The gene (cytochrome P450 24A1) which codes the enzyme required for inactivation of 1, 25(OH) 2D is not found to be differently expressed in obese and persons with normal weight. However, there is 79% increase in the expression of this particular gene after weight loss.

An association between vitamin D status and insulin resistance has been well established. The vitamin D receptors present of the β-cells of the pancreas bind with VD and indirectly cause insulin resistance in obese persons (15). VD have been associated with obesity but the exact mechanism leading towards the causal relationship of obesity, increase in abdominal adiposity and BMI is still to be unravelled. Further clinical study should focus on the integration of VD with different pathological conditions associated with obesity.

**What are the effects of VD supplementation?**

There are a wide range of reports which document the role of VD and calcium in the regulation of adipose tissue especially abdominal mas and visceral mass. VD is one of the major precipitating factors for abdominal obesity in women. Recently it has been implicated that VD and calcium lead to apoptosis of adipocytes and increase lipid metabolism leading to loss of weight. The effects of VD and calcium are integrated with each other and there are several conflicting views regarding the use of VD in management of anti-obesity. Reports demonstrated that VD supplementation does not adversely affect bodyweight, but it could significantly improve several cardiovascular risk markers. Other studies also showed no effect of VD treatment on bodyweight reduction and body composition (16). A decrease in the visceral adiposity was documented in a double blind, randomized study where calcium (1050 mg) was given daily along with VD (300 IU) to obese subjects (17). Insulin resistance is observed in obese subjects, so supplementation of VD aided with a weight loss management reduced insulin resistance and increases serum 25(OH) D in obese subjects when compared with healthy individuals (18). This effect was more pronounced when VD was given in large doses for a short time period in VD deficient and baseline glycemic control patients. Contrastingly, there have been studies which showed a negative association of BMI and fat mass with the change in VD after supplementation (19). Interestingly, improvement of VD status decreased plasma proinflammatory cytokines in patients with congestive heart failure (20). Hence, the scientific literature has documented diverse effects of VD in obese subjects, which indicate that more research should be directed towards finding the most probable mechanism.

**Conclusion**

Vitamin D or the "sunshine vitamin" as we call it is well associated with obesity and other detrimental health hazards like cardiovascular diseases, liver diseases and several degenerating bone diseases. Studies have documented the decreased levels of 25D concentrations in obese subjects as compared to the lean controls. It has been proposed that this reduction is due to the dilution of VD in the increased compartments of the body such as adipose tissue and visceral mass in obese subjects.

Furthermore obese subjects required higher doses of VD supplementation as compared to the general population. The fact that VD supplementation is a treatment of choice have been reported but simultaneously reports have also been inconsistent. Clinical trials have provided inadequate conclusions and more research should be directed in elucidating the probable mechanism of the role of VD in causal obesity. Since the effect of VD is integrated with the role calcium, hence in vitro and therapeutic studies should be addressed separately for VD and calcium. In the present scenario, it has been witnessed that weight loss aided with loss of adipose tissue and visceral mass content is beneficial outcomes in different health conditions like arterial hypertension, cardiovascular risk, Polycystic ovarian disease, increase in the density of bones, osteoarthritis, osteoporosis and liver diseases. Hence, changes in lifestyle towards an active and vibrant one would address both issues of obesity and VD deficiency simultaneously which would also open up avenues for the treatment of choice in obesity.

**Conflict of Interest**

The author declares no conflict of interest.

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