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# *Summary and Conclusion*

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The products of vitamin D hydroxylation lead to 25-hydroxyvitamin D<sub>2</sub> [25(OH)D<sub>2</sub>] and 25 hydroxyvitamin D<sub>3</sub> [25(OH)D<sub>3</sub>], ercalcidiol and calcidiol, respectively .

The second hydroxylation of these compounds leads to the production of 1,25-dihydroxyvitamin D<sub>2</sub> [1,25(OH)<sub>2</sub>D<sub>2</sub>] and 1,25-dihydroxyvitamin D<sub>3</sub> [1,25(OH)<sub>2</sub>D<sub>3</sub>], ercalcitriol and calcitriol respectively .

25-Hydroxyvitamin D has a long half-life (approximately 3 wk) and is the best measure of vitamin D status.

In humans, the primary source of vitamin D is UV B induced conversion of 7dehydrocholesterol to vitamin D in the skin. Just 10–20% of our vitamin D comes from dietary sources, such as fish, eggs, or vitamin D fortified milk .

25(oH)D is transformed by renal or extrarenal 1 $\alpha$ hydroxylase into 1,25dihydroxyvitamin D (1,25[oH]<sub>2</sub>D), which circulates at much lower serum concentrations than 25(oH)D, but has a much higher affinity to the vitamin D receptor (vDr).

It present not only in classical target tissues such as bone, kidney, and intestine, but also in many other non-classical tissues, for example, in the immune system (T and B cells, macrophages, and monocytes), in the reproductive system(uterus, testis, ovary, prostate, placenta, and mammary

glands), in the endocrine system (pancreas, pituitary, thyroid, and adrenal cortex), in muscles and in brain, skin, and liver.

In a much smaller randomized control trial 1100 IU per day of vitamin D3 along with calcium decreased the overall cancer risk.

There is an immunoregulatory role for VDR and its agonists. Vitamin D deficiency has been implicated in a range of autoimmune diseases as RA, SLE, ATIDS, and IBDS.

Recent studies confirm vitamin D's role in autoimmunity and neuroprotection from diseases such as Multiple sclerosis (MS), Narcolepsy with cataplexy (NC) and Parkinson's disease (PD) .

Vitamin D exerts various effects on the heart and blood vessels, which may be important for maintenance of normal CV system function.

Impaired vitamin D metabolism may have deleterious consequences on renal function, proteinuria, and outcome in CKD patients.

A poor vitamin D status is associated with a higher prevalence and incidence of diabetes mellitus.

Native vitamin D supplementation is an easy and cost-effective therapeutic measure. Recently, some groups have decided to suggest a 25(OH)D target level of > 30 ng/mL .

A daily intake of 1,000 IU raises 25(OH)D levels about 10 ng/mL .

Adverse effects, mainly related to hypercalcemia, are not observed at daily doses of up to 10,000 IU vitamin D or at 25(OH)D levels up to 150 ng/mL. These data were mainly derived from general populations, but studies among CKD patients showed similar results on the safety of vitamin D.

Vitamin D deficiency, defined as a 25(OH)D level <30 ng/ml, was highly prevalent in adults with coronary heart disease (79%), heart failure (83%), and stroke (74%).