Vitamin D deficiency, especially seen in Western populations, can lead to serious health problems. Vitamin D is a group of steroids that are hormones and hormone precursors because they can be synthesized in the appropriate biological conditions which is one of the vitamins that are soluble in fat. The main task of Vitamin D is to provide the mineral balance in our bodies. It regulates the metabolism and absorption of minerals and determines serum calcium and phosphorus levels. The target organs that vitamin D affects are the kidneys, small intestines, and bone. In these organs, the effect is caused by the active form of calcitriol. Vitamin D deficiency causes rickets disease in children and osteoporosis in adults.

Vitamin D Resources and Metabolism

Vitamin D has two sources, cholecalciferol (Vitamin D₃) and ergocalciferol (Vitamin D₂). Cholecalciferol is derived from 7-dehydrocholesterol by the action of sun (UV) rays and is the main source of Vitamin D. Ergocalciferol, which we call Vitamin D₂, is derived from foods. Since they have similar metabolisms, they are both called D vitamins. Dietary uptake of vitamin D₂ is limited. Vitamin D in foods is mostly found in fatty fish species such as salmon (salmon fish), mackerel, tuna fish, sardine and also in nutrients such as egg yolk, milk, broccoli, green onion, parsley water terrace. But no food contains vitamin D until it supplies the daily vitamin D requirement. In the breast milk there is vitamin D of 10-60 IU/L. In America and some European countries, milk, yogurt, orange juice, and cereals are enriched with Vitamin D. Dietary or endogenously synthesized Vitamin D₂ or D₃ is stored in fat cells and circulated if necessary.

Vitamin D is converted to 25-hydroxyvitamin D₂ (25(OH)D) by the 25-hydroxylase enzyme in the liver. Parathyroid hormone is involved in the mechanism of the conversion of 25 (OH) D to 1.25(OH)2D. In the absence of this hormone, 1.25(OH)2D cannot occur. Vitamin D is an important vitamin in our lives in terms of our health cause many health problems.

Keywords: Chronic diseases, Vitamin D deficiency, Vitamin D toxicity, Vitamin D
Recently, scientific research shows that vitamin D has different effects besides the known classical effects. The active form of Vitamin D, the cholecalciferol (Vitamin D$_3$) receptor, has been found in many tissues and cells of the human body. Many important diseases such as cancer, autoimmune diseases, and diabetes mellitus (DM) have been suggested to be related to vitamin D.

Inadequate sunlight intake, such as long-term stay in indoor environments such as home and office, clothing style, use of sun protection creams with high protection factor, and seasonal changes cause vitamin D insufficiency to occur more frequently. Studies conducted in different countries show that vitamin D deficiency is a worldwide problem and this is a major health problem. Mortality due to fragile fractures due to insufficient 25(OH)D, especially hip fracture, is common.

Vitamin D is derived skin and diet and is carried through the circulation of vitamin D by binding to the vitamin D binding protein (DBP). It is converted to 25(OH)D by 25-hydroxylase enzyme in the liver. Vitamin D deficiency occurs when 25 (OH) D level is <20 ng/mL and vitamin D is 25 (OH) D level >30 ng/mL. 25(OH)D level vitamin D intoxication occurs when 25 (OH) D level >150 ng/mL. Active Vitamin D plays a role in the maintenance of normal bone mineral balance and insulin secretion and, at the same time, inhibits parathyroid hormone (PTH) synthesis and secretion, leading to the proliferation of parathyroid glands.

Cardiovascular diseases are one of the most important causes of death in patients with end-stage renal disease (ESRD). Vitamin D deficiency is an independent factor for the development of ESRD. In hemodialysis patients, it has been shown that there is an association between insufficient Vitamin D status and endothelial dysfunction.

Vitamin D is suggested to be a potential regulator in the prevention of diabetes. Although Vitamin D is shown to be effective in the prevention of type 1 DM, the mechanism is not fully explained. The hypotheses put forward in this regard are that active Vitamin D improves beta-cell function, increases insulin sensitivity in target cells, and protects beta-cells from harmful immunological damage. It is thought that Vitamin D has a direct or indirect protective effect on the immune response. As is known, type 2 DM develops in the form of complete insulin deficiency due to damage of pancreatic beta cells. Unlike type 1 DM, the function of pancreatic beta-cells is impaired, insulin resistance and inflammation are common. Vitamin D is associated with type 1 DM, as is the effect on type 2 DM. Some studies show that there is a relationship between vitamin D and obesity, but it is being debated whether if vitamin deficiencies are caused by obesity or obesity causes vitamin D deficiency. The causes of vitamin D deficiency in obese patients have been investigated and realized that they have been avoiding open-air activities due to limited mobility and socialization. They have been feeding with missing foods due to Vitamin D, having more closed clothing choices than weaker ones, lack of basic dietary habits, (OH)2D$_3$ levels increased due to increased PTH level, suppression of 25(OH)D$_3$ synthesis in the liver, and increased levels of PTH in the body, resulting in decreased bioavailability due to adherence to adipose tissue, reduction of liver synthesis 25(OH)D$_3$ synthesis due to hepatic steatosis as been reported as the major factors. Prospective studies investigating the relationship between vitamin D and obesity and determination of the relationship by reference methods is required.

It has also been reported in a study that there is a negative correlation between antenatal Vitamin D supplementation and bilirubin levels in newborns during pregnancy. As the need for Vitamin D increases during pregnancy, the risk of Vitamin D deficiency in pregnancies is also increasing. D vitamins can pass placentally through the mother to the fetus. In the absence of Vitamin D, both mother and fetus can be affected. These effects include low birth weight infant, growth retardation, retardation (congenital rickets, craniotabes, wide fontanelle, decrease in bone mineral density, and ossification), neonatal hypocalcemic seizure, enamel hypoplasia in the teeth, and calcium balance disorders in the development of bone tissue. The lack of vitamin D deficiency during pregnancy increases the risk for infants fed breastfeeding. The World Health Organization states that in pre-pregnancy, pregnancy, and lactation periods, women are exposed to iron, zinc, folic acid, iodine, and Vitamin A supplementation, and it is emphasized that the evaluation of pregnancies with respect to Vitamins D in terms of maternal and infant health is important in the tests performed.

Clinical data show that Vitamin D is associated with neuronal differentiation, axonal attachment, dopaminogenosis, brain structure, and function. The lack of Vitamin D in fetal life and childhood is associated with autistic spectrum disorder and schizophrenia also stresses the importance of Vitamin D deficiency. However, there is a need to study more so that these mechanisms can be fully explained.

Recent epidemiological studies have reported that some psychiatric disorders (such as depression and Alzheimer’s) that are not associated with brain anomalies are associated with Vitamin D levels. Detection and treatment of Vitamin D deficiency with tests to be performed in depression.
and other mental disorders increase the quality of life of patients as well as being a cheap, easy, and effective treatment for patients.\cite{47}

**Vitamin D Toxicity**

Vitamin D deficiency can lead to many health problems, and excessive intake also causes toxicity. Vitamin D deficiency treatment is performed in two phases. In the first stage, the acute deficiency is treated in the patient, and the second phase is the filling of the body reserves. The upper limit for children younger than 1 year has been reported to be 1000 IU/day for long-term Vitamin D therapy, and 2000 IU/day for children over 1 year.\cite{48,49} Pediatric toxic threshold doses are not clearly defined and serum 25(OH) D concentration is up to 140 ng/it is considered harmful if it is low. Depending on the variations in the calcic response to Vitamin D in the treatments administered, there may be individual responses at specific doses. Failure to determine appropriate doses in long-term treatments, age inappropriate doses, and variable response to contraindication may result in toxicity.\cite{51,52}

Elevated levels of Vitamin D emphasize hypercalcemic response. In vivo studies suggest that 25(OH)2D is responsible for 1.25(OH)2D rather than toxic by binding competitively to Vitamin D receptors.\cite{53}

Vitamin D increases calcium absorption from the gastrointestinal tract. Vitamin intoxication is secondary to high doses of Vitamin D intake. Reported toxicity is associated with overdosage of prescribed Vitamin D in cases of exposure, or consumption of over-the-counter supplements containing Vitamin D.\cite{54,55} In our country, an excess of Vitamin D is generally reported to be associated with unnecessary or inappropriate doses of Vitamin D and long-term outpatient prescribing.

Vitamin D toxicity may indicate symptoms such as hypercalcemia, hypercalciuria, renal calculus, hyperphosphatemia, polyuria, polydipsia, ectopic calcification of soft tissues (kidney and lung), nausea, and vomiting.\cite{56,57} Loss of appetite, vomiting, constipation, growth retardation, polyuria, dehydration and the indication of vitamin D poisoning such as fever develops as hypercarbia secondary. Vitamin D is characterized by hypercalciuria and hypocalcemia-associated nephrocalcinosis and 25(OH)D vitamin (>150 ng/mL) parathyroid hormone suppression.\cite{58}

In the treatment of Vitamin D poisoning, external Vitamin D intake is discontinued and intravenous fluid therapy, loop diuretics, glucocorticoids, and calcium-restricted diet are administered. In addition to treatment, calcitonin and bisphosphonates (pamidronate) are used in severe cases.

In the United States, it has been reported that Vitamin D supplementation in foods and beverages from 1930 to 1950 led to Vitamin D toxicity.\cite{59}

In Turkey since 2005, according to the recommendations of the Ministry of Health, free Vitamin D supplements (400 IU/day) are regularly given to infants to reduce the prevalence of nutritional risk.\cite{60}

**Conclusion**

The figures for Vitamin D used in food supplements may be misleading and should not be used unless credibility is proved. Again multivitamins should not be preferred for Vitamin D treatment. It has been reported that unnecessary treated patients should be evaluated for signs of hypervitaminosis. The use of Vitamin D in the form of prescription and unconsciousness can cause health problems. Vitamin D levels should be controlled at regular intervals and the deficiency or excess of Vitamin D to be detected by standard tests should be resolved by appropriate treatments. Clinical trials evaluating the metabolism of Vitamin D and its relationship to chronic diseases are needed.

**Disclosures**

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