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ASSOCIATION OF VITAMIN D DEFICIENCY WITH METABOLIC DISORDERS IN PATIENTS WITH OBESITY

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Abstract. Vitamin D deficiency is frequently observed in individuals with obesity and has attracted increasing attention because of its potential association with metabolic disturbances. This article examines the relationship between vitamin D deficiency and metabolic disorders in patients with obesity, with particular emphasis on insulin resistance, impaired glucose metabolism, dyslipidemia, hypertension, and chronic low-grade inflammation. The possible mechanisms underlying this association include sequestration of vitamin D in adipose tissue, reduced bioavailability, limited outdoor activity, altered vitamin D metabolism, and interactions between vitamin D signaling and metabolic pathways. Current evidence suggests that low serum 25-hydroxyvitamin D concentrations are common among people with obesity and may be associated with an unfavorable metabolic profile. However, the direction and clinical significance of this relationship remain incompletely established because obesity, lifestyle, diet, physical activity, and other factors may act as confounders.

Keywords: vitamin D deficiency, obesity, metabolic disorders, insulin resistance, glucose metabolism, dyslipidemia, hypertension, inflammation, 25-hydroxyvitamin D, metabolic syndrome.

Introduction. Obesity is a major public health problem and is associated with an increased risk of type 2 diabetes mellitus, cardiovascular disease, hypertension, dyslipidemia, metabolic dysfunction-associated steatotic liver disease, and other chronic conditions. The increasing prevalence of obesity has stimulated research into nutritional, hormonal, and metabolic factors that may contribute to obesity-related complications.

Vitamin D is a fat-soluble vitamin and steroid hormone precursor that plays an important role in calcium and phosphate homeostasis and bone metabolism. Vitamin D signaling is also involved in several extra-skeletal processes, including immune regulation, cellular differentiation, and metabolic pathways.

Low serum concentrations of 25-hydroxyvitamin D [25(OH)D], the major circulating indicator of vitamin D status, are commonly reported in people with obesity. Several mechanisms may contribute to this phenomenon. Because vitamin D is fat soluble, increased adipose tissue may influence its distribution and bioavailability. In addition, people with obesity may have lower exposure to sunlight, reduced physical activity, and dietary factors that contribute to inadequate vitamin D status.

At the same time, obesity is frequently accompanied by insulin resistance, impaired glucose tolerance, dyslipidemia, elevated blood pressure, and chronic low-grade inflammation. These observations have led researchers to investigate whether vitamin D deficiency is associated with the metabolic abnormalities observed in obesity.

The **aim of this article** is to analyze the available evidence concerning the association between vitamin D deficiency and metabolic disorders in patients with obesity and to discuss possible biological mechanisms and clinical implications.

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Literature review. A growing body of epidemiological research has demonstrated an inverse association between adiposity and circulating 25(OH)D concentrations. Meta-analyses and observational studies generally report lower vitamin D levels among individuals with obesity compared with individuals of normal weight.

Several explanations have been proposed. First, vitamin D may be distributed into adipose tissue, reducing its concentration in the circulation. Second, reduced mobility and outdoor physical activity may decrease cutaneous vitamin D synthesis. Third, dietary patterns associated with obesity may provide insufficient amounts of vitamin D.

Vitamin D has also been investigated in relation to insulin sensitivity. Vitamin D receptors are expressed in pancreatic beta cells and various tissues involved in glucose metabolism. Experimental studies suggest that vitamin D signaling may influence insulin secretion and inflammatory pathways. Observational studies have frequently reported an association between low vitamin D status and insulin resistance.

However, the evidence does not establish a simple causal relationship. Obesity itself may contribute to vitamin D deficiency, while lifestyle factors may simultaneously influence both vitamin D status and metabolic health. Therefore, the association may be bidirectional or partly explained by shared risk factors.

Research concerning dyslipidemia has produced mixed findings. Some studies have found that lower vitamin D concentrations are associated with higher triglyceride levels and less favorable lipid profiles, whereas other studies have not demonstrated consistent independent associations.

Vitamin D deficiency has also been investigated in relation to hypertension. Possible mechanisms include effects on the renin-angiotensin-aldosterone system, vascular function, inflammation, and endothelial activity. Nevertheless, randomized clinical trials of vitamin D supplementation have generally not demonstrated a consistent blood-pressure-lowering effect in the general population.

The relationship between vitamin D and inflammation is another area of interest. Obesity is characterized by chronic low-grade inflammation, with increased production of inflammatory mediators. Vitamin D has immunomodulatory properties, and laboratory studies suggest that vitamin D signaling may influence inflammatory pathways. However, clinical evidence remains heterogeneous.

Overall, the literature supports a frequent coexistence of vitamin D deficiency and metabolic abnormalities in obesity, but whether vitamin D deficiency independently causes these disorders remains uncertain.

Research methodology. This article is based on a narrative analysis of scientific literature concerning vitamin D status and metabolic disorders in individuals with obesity.

The following methodological approaches were used:

- analysis of peer-reviewed scientific publications;
- comparison of observational and interventional studies;
- synthesis of evidence concerning vitamin D status and metabolic indicators;
- analysis of possible physiological mechanisms;
- comparative evaluation of findings concerning insulin resistance, glucose metabolism, lipid abnormalities, hypertension, and inflammation.

The principal biochemical marker considered for vitamin D status is serum 25-hydroxyvitamin D [25(OH)D].

Metabolic health can be evaluated using several indicators, including:

- fasting plasma glucose;
- glycated hemoglobin (HbA1c);

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- fasting insulin;
- indices of insulin resistance;
- triglycerides;
- total cholesterol;
- LDL cholesterol;
- HDL cholesterol;
- blood pressure;
- markers of systemic inflammation.

Obesity is associated with chronic low-grade systemic inflammation. Adipose tissue can produce inflammatory mediators that contribute to insulin resistance and metabolic dysfunction.

Vitamin D has immunomodulatory properties and may interact with inflammatory signaling pathways. Low vitamin D status may therefore coexist with increased inflammatory activity.

However, whether vitamin D deficiency directly drives inflammation in obesity remains uncertain. More controlled clinical research is required to clarify this relationship.

Metabolic parameter	**Potential association with low vitamin D**	**Proposed mechanism**	**Strength of evidence**
Insulin resistance	Frequently reported	Insulin signaling, inflammation, calcium metabolism	Moderate
Glucose intolerance	Frequently reported in observational studies	Beta-cell and insulin signaling	Moderate
Type 2 diabetes	Association reported in observational studies	Multiple metabolic pathways	Inconsistent causality
Triglycerides	Possible association	Lipid metabolism and insulin resistance	Inconsistent
LDL cholesterol	Possible association	Lipid metabolism	Inconsistent
HDL cholesterol	Possible inverse association	Metabolic and inflammatory pathways	Inconsistent
Hypertension	Possible association	Renin-angiotensin and vascular pathways	Inconclusive
Inflammation	Biological plausibility	Immunomodulatory effects	Inconclusive

The frequent coexistence of obesity and vitamin D deficiency suggests that vitamin D status may be relevant when evaluating patients with obesity. Assessment of vitamin D status can be considered when clinically indicated, particularly in individuals with risk factors for deficiency.

However, vitamin D supplementation should not be regarded as a substitute for established obesity-management strategies. Weight management, dietary improvement, physical activity, sleep optimization, and appropriate treatment of metabolic disorders remain fundamental. When vitamin D deficiency is confirmed, correction should follow evidence-based clinical recommendations and individualized medical assessment.

An important distinction should be made between association and causation. A low vitamin D concentration may reflect obesity-related biological and lifestyle factors without necessarily being the primary cause of metabolic disorders.

Conclusion. Vitamin D deficiency is common among individuals with obesity and frequently coexists with metabolic abnormalities such as insulin resistance, impaired glucose regulation,

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dyslipidemia, hypertension, and chronic low-grade inflammation. Several biological mechanisms may explain the relationship, including adipose tissue sequestration of vitamin D, reduced sunlight exposure, altered vitamin D metabolism, and interactions between vitamin D signaling and metabolic pathways.

Nevertheless, current evidence does not establish that vitamin D deficiency independently causes metabolic disorders in patients with obesity. Many observational associations may be influenced by adiposity, physical activity, diet, age, socioeconomic factors, and other confounding variables. Moreover, clinical trials of vitamin D supplementation have not consistently demonstrated improvements in major metabolic outcomes.

References

1. Holick, M.F. Vitamin D deficiency. New England Journal of Medicine, 2007, 357(3), 266–281.
2. Holick, M.F. Vitamin D: Photobiology, metabolism, mechanism of action, and clinical applications. In: Endocrinology. Williams Textbook of Endocrinology.
3. Wortsman, J., Matsuoka, L.Y., Chen, T.C., Lu, Z., Holick, M.F. Decreased bioavailability of vitamin D in obesity. American Journal of Clinical Nutrition, 2000, 72(3), 690–693.
4. Pereira-Santos, M., Costa, P.R.F., Assis, A.M.O., Santos, C.A.S.T., Santos, D.B. Obesity and vitamin D deficiency: A systematic review and meta-analysis. Obesity Reviews, 2015, 16(4), 341–349.
5. Vimaleswaran, K.S., Berry, D.J., Lu, C., et al. Causal relationship between obesity and vitamin D status: bi-directional Mendelian randomization analysis of multiple cohorts. PLoS Medicine, 2013, 10(2), e1001383.
6. Pittas, A.G., Lau, J., Hu, F.B., Dawson-Hughes, B. The role of vitamin D and calcium in type 2 diabetes. A systematic review and meta-analysis. Journal of Clinical Endocrinology & Metabolism, 2007, 92(6), 2017–2029.
7. Muscogiuri, G., Sorice, G.P., Prioletta, A., et al. 25-Hydroxyvitamin D concentration correlates with insulin-sensitivity and BMI in obesity. Obesity, 2010, 18(10), 1906–1910.
8. Mathieu, C., Gysemans, C., Giulietti, A., Bouillon, R. Vitamin D and diabetes. Diabetologia, 2005, 48, 1247–1257.
9. Bouillon, R., Marcocci, C., Carmeliet, G., et al. Skeletal and extraskeletal actions of vitamin D: current evidence and outstanding questions. Endocrine Reviews, 2019, 40(4), 1109–1151.
10. Pilz, S., Kienreich, K., Rutters, F., et al. Role of vitamin D in the development of cardiovascular disease and metabolic disorders. Endocrine Reviews, relevant literature.
11. Rosen, C.J., Abrams, S.A., Aloia, J.F., et al. IOM committee members respond to Endocrine Society vitamin D guideline. Journal of Clinical Endocrinology & Metabolism, 2012.
12. LeFevre, M.L. Screening for vitamin D deficiency: U.S. Preventive Services Task Force recommendation statement. Annals of Internal Medicine, 2015, 162(2), 133–140.