

THE RELATIONSHIP BETWEEN VITAMIN-D AND OBESITY

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ABSTRACT

Overweight and obesity are a growing health problem in both adults and children worldwide. Obesity, which has become a health threat today in many countries in the world as well as in Turkey, is defined by the World Health Organization (WHO) as "excessive fat accumulation in the adipose tissue that presents a risk to health". According to the World Health Organization, obesity is an important public health problem with its social and psychological aspects and increasing prevalence that involves all age groups. Vitamin D, a member of the fat-soluble vitamin groups, is also called calciferol. Pro-vitamin D transforms into vitamin D due to the effect of solar rays on the skin. Vitamin D is naturally available in very few foods. The best way to meet the need for vitamin D is to make enough contact with the sun's rays. Vitamin D is most commonly found in liver, milk and eggs, but the amount is not sufficient. In recent epidemiological and scientific studies, it has been observed that there is a relationship between vitamin D and obesity. This literature review aims to study the effect of vitamin D on obesity.

Keywords: Obesity, Vitamin D, Vitamin D deficiency, Obesity and Vitamin D

INTRODUCTION

The World Health Organization (WHO) defines obesity as "excessive fat accumulation that presents a risk to health". This process, in which the body weight based on height increases above the desired level due to an increase in the ratio of body fat mass to fat-free mass, is closely related to fat tissue. The Body Mass Index (BMI) value >25 is considered overweight and >30 is considered obese (1). Obesity is one of the most important causes of preventable premature deaths. Obesity leads to many chronic

illnesses and increases liability to type 2 diabetes mellitus, hypertension, cerebrovascular disease, ischemic heart disease, gallstones, impaired pulmonary function and cancer. Obesity, which is increasing day by day all over the world, is one of the most important health problems of our time. Obesity should be considered as an illness that has reached the epidemic level all over the world. Obesity poses a public health problem in both developed and developing countries. According to the data of WHO,

in 2014 1.9 billion adults over 18 years of age were considered overweight and approximately 600 million adults were considered obese, and in 2013 42 million children under 5 years of age were considered overweight or obese (2). Obesity is the second most common cause of preventable deaths in the United States after smoking (3).

Observations and statistics show that obesity has been increasing in child and adolescent age groups, similar to adults (2). In the United States, where obesity in children and adults increases the fastest in the world, the number of overweight adolescents has tripled since 1980 (4). Likewise, the rate of obesity has been increasing in European countries and Turkey. According to the study conducted in Kayseri, Turkey on children in the age group of 7-17; 15.4% was considered overweight and 3.9% was obese (5). In the study conducted in the Eastern Anatolian region, 10.9% of the boys and 11.4% of the girls were overweight while 2.1% of the boys and 2.3% of the girls were obese (6). In the Western Black Sea region, among the 6-17 age group, 10.3% was considered overweight and 6.1% was considered obese (7). In Konya, the obesity rate at 9-14 years was 5.3% (8). In the Kocaeli region, obesity was found to be 7.3% while overweight was found to be 11.8% (9). Ultimately, it is a great public health problem as well as an economic issue. Obesity is considered to be a chronic disease that needs treatment due to the fact that it shortens life span, arises complications, and deteriorates life standards (10).

Aim

In recent epidemiological and scientific studies, it has been observed that there is a relationship between vitamin D and obesity. This literature review aims to study the effect of vitamin D on obesity.

Materials and methods

This study was conducted in January, 2018 by scanning various sources. Scanned resources are presented under different headings. It is desirable to keep a light on whether there is an association between vitamin D and obesity.

Treatment Strategies for Obesity

Today, the treatment options accepted in obesity treatment are Diet, Exercise, Behavioral therapy, Medical treatment, and Surgical treatment. The aim of obesity treatment is to spend more energy than the energy intake. The most important component of weight loss is the reduction of dietary energy. Low-calorie diets are 1000-1200 kcal/day for women and 1200-1600 kcal/day for men. Diets below 800 kcal/day are defined as very low-calorie diets. There is no significant difference in weight loss regarding very low-calorie diets in the long term (15). Low calorie diets are recommended for obesity treatment (16). The primary aim of obesity treatment is to adjust eating and exercise behaviors according to body weight.

Vitamin D

Vitamin D, having a similar structure to a steroid, is a prohormone synthesized from 7-dehydrocholesterol primarily by exposure of skin to ultraviolet light (sunlight). Approximately 95% of vitamin D required by the body is synthesized by the sun's rays (17). Apart from its endogenous synthesis by the body, vitamin D can be taken through a diet (18). However, vitamin D intake through a diet is limited. Vitamin D in foods is mostly found in oily fish such as salmon, mackerel, sardines and egg yolks (19). Vitamin D is taken in two forms as the ergocalciferol (vitamin D2) found in plants and the cholecalciferol (vitamin D3) found in animal tissues (17). Dietary vitamin D2 and vitamin D3 forms are absorbed by the small intestines and combine with the chylomicrons and enter the lymphatic system. Here they join the venous circulation. Dietary or endogenously synthesized vitamin D2 (ergocalciferol) or vitamin D3 (cholecalciferol) is stored in fat cells and is used by being released into the circulation when necessary (17, 20).

Vitamin D Metabolism

As vitamin D2 and vitamin D3 synthesized in the skin or taken with diet are not biologically active, they are carried to the liver with Vitamin D-binding protein and converted to 25-Hydroxy Vitamin D (25(OH)D) with 25-hydroxylase enzyme in the liver.

However, in order for vitamin D to become active, it must be converted to 1,25(OH)₂D by the enzyme 1- α hydroxylase in the kidney. 25(OH) D, is the actual form of vitamin D in the circulation (21).

Vitamin D Deficiency

Dark complexion, limited skin synthesis caused by inadequate sunlight exposure, and poor dietary intake are the main causes of low levels of 25(OH)D (22). The synthesis of vitamin D through the skin decreases during winter due to the change of the angle of the sun's rays and reduced exposure to sunlight.

Skin pigmentation also plays an important role on vitamin D deficiency. Melanin pigment, which is produced at high levels in people with a darker complexion, blocks the absorption of ultraviolet (UVB) rays (23,24).

Many gastrointestinal disorders can also cause vitamin D deficiency due to the fact that vitamin D is absorbed through the ileum in the small intestine. Malabsorptions such as Crohn's and Celiac Disease cause vitamin D deficiency. Obesity can also cause vitamin D deficiency because fat-soluble vitamin D is stored in the adipose tissue instead of joining the circulation (25).

The primary risk factors for vitamin D deficiency include advanced age, genetic factors, living in a society with covered clothing habits due to traditional and beliefs, frequently being in a closed environment, excessive use of protective sun cream, low physical activity, smoking, air pollution, kidney disease, liver disease, and the use of drugs adversely affecting vitamin D metabolism such as anticonvulsants and glucocorticoids (26).

Vitamin D Deficiency and Diseases

Vitamin D deficiency is associated with high plasma triglycerides, high Very Low-Density Lipoprotein (VLDL) and impaired insulin metabolism (27). Vitamin D deficiency is thought to be a significant risk factor for cardiovascular diseases. Vitamin D deficiency causes impaired calcium balance and secondary hyperparathyroidism. The supplementation of inactive and active vitamin D analogs reduces the

increase of the Parathyroid hormone (PTH) level. This reduces the adverse effects of PTH on the myocardium and vascular wall, thus provides a protective effect on the heart (28). It has been suggested that low levels of 25(OH)D among diabetic hemodialysis patients are associated with sudden cardiac death (29).

An observational study has suggested that low levels of serum 25(OH)D are associated with obesity and diabetes mellitus. The study points out that vitamin D is soluble and stored in fat tissue, the demonstration of inverse relationship between increased body fat percentage and body mass index and vitamin D levels in large cohort studies, and the demonstration of vitamin D modulating insulin synthesis and secretion in animal studies (30).

In a prospective cohort study of Pittas et al. titled 'Nurses' Health Study', levels of 25(OH)D and glucose intolerance in women were observed for 20 years and vitamin D and calcium intake was found to be inversely related to the risk of developing type 2 diabetes. Moreover, those who consumed 3 or more portions of milk a day were found to have a lower risk of diabetes compared to the ones only consuming 1 portion a day (31). In a recent study by Devaraj et al., a relation between vitamin D levels and prediabetes (32) was identified. Low levels of 25(OH)D in obese African American adolescents were found to be correlated with low levels of adiponectin, increase of fat, obesity, and insulin resistance (33).

Causes of Vitamin D Deficiency in Obese People

- Lack of physical activity and less participation in outdoor activities
- Not enough exposure to sun's rays, using excessive and continuous sun cream and hats
- Wearing covered clothing for traditional reasons
- Inadequate vitamin D intake in basic dietary habits (inadequate consumption of fish, milk, yogurt, and cheese)
- High consumption of vitamin D-deficient, unhealthy and empty calories

- Dietary differences secondary to lactose intolerance among blacks and Asians
- Since vitamin D is a fat-soluble molecule, its bioavailability is reduced by being stored in the fat tissue (adipose tissue) in obese people. Thus, the level of serum can be measured low even though it is sufficient in the body.
- Increased levels of 1,25(OH)2D3 due to high PTH, suppressing 25(OH)D3 synthesis in KC
- Decrease in 25(OH) D3 synthesis in KC due to hepatic steatosis (34,35).

RESULTS AND DISCUSSION

Studies on the Relationship between Obesity and Vitamin D

Some studies have shown that obesity occurs more frequently in adults with low levels of vitamin D (36, 37).

Another study conducted on 42,024 people revealed that each unit increase in the body mass index caused a 1.15% decrease of 25(OH) D (38). It was shown that body fat had decreased in obese women with vitamin supplements of 1000 IU per day for 12 weeks (39). Carrillo et al. have shown that vitamin D supplementation of 4000 IU per day reduces waist-to-hip ratio in obese people (40).

It has been demonstrated that in cases where passage of calcium to the fat cells increases, lipolysis is suppressed while lipogenesis is induced. Increased calcium inhibits the phosphodiesterase enzyme, suppresses the catecholamine-induced lipolysis and causes a triglyceride accumulation in the adipose tissue while stimulating lipogenesis by activating the fatty acid synthase that plays a role in the synthesis. In another study, Shi et al. have emphasized that with the genomic effect of 1,25 (OH) 2D3 administrations and the non-genomic effect of VDR, the passage of calcium to the adipose tissue had increased significantly and played a role in obesity (41).

Vitamin D is an important determinant of serum PTH level and increases 1,25(OH) 2D3 synthesis and passage of calcium to the cell by increasing the

1-alpha hydroxylase activity in high PTH adipocytes, leading to lipoidosis and obesity. It also contributes to the mechanism by suppressing lipid oxidation in PTH muscle cells. Increased adipose tissue, decreased dyslipidemia and insulin secretion due to vitamin D deficiency suggests that deficiency is a potential cause of obesity, but it would be an optimistic view to expect that increasing the level of vitamin D would completely prevent obesity. It is suggested that compared to normal weight individuals, PTH levels in obese people can be suppressed with a lower dose of vitamin D. Accordingly, there may be a different modulation in the calcium-PTH relationship in obese people. Studies have shown that vitamin D supplementation at doses sufficient to overcome the deficiency and suppress the PTH level may be beneficial in vitamin D deficiency (42).

In another study, vitamin D deficiency was associated with fatigue, daytime sleepiness and the level of physical activity. Serum vitamin D concentrations and physical activity levels were shown to be favorably associated (43). It is known that vitamin D levels below 20 ng/mL are associated with low physical activity (44).

CONCLUSION

vitamin D deficiency alone has been known to play a role in the pathogenesis of diseases such as rickets and osteomalacia but it is also considered to be a contributing factor in diseases such as DM, metabolic syndrome, multiple sclerosis, rheumatoid arthritis, systemic lupus erythematosus, epilepsy, polycystic ovary syndrome and cancer. The studies carried out in recent years have raised concerns regarding the effects of vitamin D on obesity, however since the studies are not randomized and are cross-sectional, a definitive conclusion could not be made.

REFERENCES

1. World Health Organization, About the BMI database, BMI classification. http://apps.who.int/bmi/index.jsp?introPage=intro_3.html Date of access: 03.01.2018.

2. World Health Organization. Obesity and overweight. <http://www.who.int/mediacentre/factsheets/fs311/en/> Date of access: 05.01.2018.
3. McGinnis JM, Foege WH. Actual causes of death in the United States. *JAMA*. 1993;270:2207-12.
4. Skelton JA, Cook SR, Auinger P, Klein JD, Barlow SE. Prevalence and trends of severe obesity among US children and adolescents. *Acad Pediatr*. 2009;9:322-29.
5. Ozturk A, Mazicioglu MM, Hatipoglu N, Budak N, Keskin G, Yazlak Z, Balci N, Yildiz H, Yildiz K, Ustunbas HB, Kurtoglu S. Reference body mass index curves for Turkish children 6 to 18 years of age. *J Pediatr Endocrinol Metab*. 2008;21:827-36.
6. Yuca SA, Yılmaz C, Cesur Y, Doğan M, Kaya A, Başaranoğlu M. Prevalence of overweight and obesity in children and adolescents in Eastern Turkey. *J Clin Res Pediatr Endocrinol*. 2010;2:159-63.
7. Simsek E, Akpınar S, Bahcebasi T, Sense DA, Kocabay K. The prevalence of overweight and obese children aged 6-17 years in the West Black Sea region of Turkey. *Int J Clin Pract*. 2008;62:1033-38.
8. Kutlu R, Çivi S, Karaoğlu O. Can waist circumference clinically be useful as a predictor of obesity/underweight in children? *Gülhane Tıp Dergisi*. 2011;53:170-76.
9. Etiler N, Cizmecioglu FM, Hatun S, Hamzaoglu O. Nutritional status of students in Kocaeli, Turkey: a population-based study. *Pediatr Int*. 2011;53: 231-35.
10. Hill J. Dealing with obesity as a chronic disease. *Obesity Research*. 1998; 6:34-8.
11. Mustajoki P, Pekkarinen T. Very low energy diets in treatment of obesity. *Obesity Review*. 2001; 2:61-72.
12. Muszkat P, Camargo MB, Griz LH, Lazaretti-Castro M. Evidence-based non-skeletal actions of vitamin D. *Arq Bras Endocrinol Metabol*. 2010;54:110-17.
13. Holick MF. High Prevalence of Vitamin D Inadequacy and Implications for Health. *Mayo ClinProc*. 2006;81:353-73.
14. Muszkat P, Camargo MB, Griz LH, Lazaretti-Castro M. Evidence-based non-skeletal actions of vitamin D. *Arq Bras Endocrinol Metabol*. 2010;54:110-17.
15. Holick MF. Vitamin D Deficiency Medical Progress. *N Engl J Med*. 2007;357:266-81.
16. Wacker M, Holick MF. Vitamin D-Effects on Skeletal and Extraskelatal Health and the Need for Supplementation. *Nutrients*. 2013;5:111-48.
17. Holick MF, Binkley NC, Bischoff-Ferrari HA, Gordon MC, Hanley DA, Heaney RP et al. Evaluation, Treatment and Prevention of Vitamin D Deficiency: an Endocrine Society Clinical Practice Guideline. *J Clin Endocrinol Metab*. 2011;96:1911-30.
18. Moy FM, Bulgiba A. High prevalence of vitamin D insufficiency and its association with obesity and metabolic syndrome among Malay adults in Kuala Lumpur, Malaysia. *Public Health*. 2011;11:735-42.
19. Kaehler ST, Baumgartner H, Jeske M, et al. Prevalence of hypovitaminosis D and folate deficiency in healthy young female Austrian students in a health care profession. *Eur J Nutr*. 2011;11:281-85.
20. Whayne TF. Vitamin D: Popular Cardiovascular Supplement But Benefit Must Be Evaluated. *International Journal of Angiology*. 2011;20:63-71.
21. Thomas GN, Hartaigh BO, Bosch JA, Pilz S et al. Vitamin D Levels Predict All-Cause and Cardiovascular Disease Mortality in Subjects With the Metabolic Syndrome. *Diabetes Care*. 2012;35:1158-64.
22. Wang TJ, Pencina MJ, Booth SL, et al. Vitamin D Deficiency and Risk of Cardiovascular Disease. *Circulation*. 2008;117:503-11.
23. Harris SS, Dawson-Hughes B. Seasonal Changes in Plasma 25-hydroxy Vitamin D Concentrations of Young American Black and White Women. *Am J ClinNutr*. 1998; 67:1232-36.
24. Goswami R, Gupta N, Goswan D, Marwaha RK, Tandon N, Kochupillai N. Prevalence and Significance of low 25- hydroxy vitamin D Concentrations in Healthy Subjects in Delhi. *Am J ClinNutr*. 2000;72:472-75.
25. Heath KM, Elovic EP. Vitamin D Deficiency: Implications in the Rehabilitation Setting. *Am J PhysMedRehabil*. 2006; 85:916-23.
26. Lavie CJ, Lee JH, Milani RV. Vitamin D and Cardiovascular Disease Will It Live Up to its Hype? *Journal of American College of Cardiology*. 2011; 58:1547-56.
27. Whayne TF. Vitamin D: Popular Cardiovascular Supplement But Benefit Must Be Evaluated. *International Journal of Angiology*. 2011; 20:63-71.
28. Pilz S, Tomaschitz A, Drechsler C, de Boer RA. Vitamin D deficiency and heart disease. *Kidney International Supplements*. 2011; 1:111-15.
29. Drechsler C, Pilz S, Obermayer-Pietsch B, et al. Vitamin D deficiency is associated with sudden cardiac death, combined cardiovascular events, and mortality

- in haemodialysis patients. *Eur Heart J.* 2010;31:2253-61.
30. Rosen CJ, Adams JS, Bikle DD, et al. The Nonskeletal effects of Vitamin D: an Endocrine Society Scientific Statement. *Endocrine Reviews.* 2012; 33:456-92.
 31. Pittas AG, Chung M, Trikalinos T, et al. Systematic review: Vitamin D and cardiometabolic outcomes. *Ann Intern Med.* 2010; 152:307-14.
 32. Devaraj S, Jialal G, Cook T, et al. Low vitamin D levels in Northern American adults with the metabolic syndrome. *Horm Metab Res.* 2011;43:72-74.
 33. Nunlee-Bland G, Gambhir K, Abrams C, et al. Vitamin D deficiency and insulin resistance in obese AfricanAmerican adolescents. *J Pediatr Endocrinol Metab.* 2011;24:29-33.
 34. Zenari L, Falezza G, Arcaro G. "Associations between serum 25-hydroxyvitamin D3 concentrations and liver histology in patients with non-alcoholic fatty liver disease". *Nutr Metab Cardiovasc Dis.* 2007;17(7):517-24.
 35. Deepu D, Hardigan P, Bray N, Penzell D, Savu C. The incidence of vitamin D deficiency in the obese: a retrospective chart review. *J Comm Hosp Int Med Pers.* 2015;5:260-69.
 36. Nagpal S, Na S, Rathnachalam R. Noncalcemic actions of vitamin D receptor ligands. *Endocrine Rev.* 2004;26(5):662-87.
 37. Mai XM, Chen Y, Camargo CA Jr, Langhammer A. Cross-sectional and prospective cohort study of serum 25hydroxyvitamin D level and obesity in adults: the HUNT study. *Am J Epidemiol.* 2012; 15;175(10):1029-36.
 38. Vimalleswaran KS, Berry DJ, Lu C, Tikkanen E, Pilz S, Hiraki LT, et al. Causal relationship between obesity and vitamin D status: Bi-directional mendelian randomization analysis of multiple cohorts. *PLoS Med.* 2013; 10:e1001383.
 39. Salehpour A, Hosseinpanah F, Shidfar F, Vafa M, Razaghi M, Dehghani S, et al. A 12-week double-blind randomized clinical trial of vitamin D3 supplementation on body fat mass in healthy overweight and obese women. *Nutr J.* 2012;11:78.
 40. Carrillo A, Flynn M, Pinkston C, Markofski M, Jiang Y, Donkin S, et al. Impact of vitamin D supplementation during a resistance training intervention on body composition, muscle function, and glucose tolerance in overweight and obese adults. *Clin Nutr.* 2013;32:375-81.
 41. Shi H, Norman AW, Okamura WH, Sen A, Zemel MB. 1 Alpha,25-Dihydroxyvitamin D3 modulates human adipocyte metabolism via nongenomic action. *FASEB J.* 2001;15(14):2751-53.
 42. Aydın M. Vitamin D ve obezite. *Türkiye Klinikleri J Pediatr Sci.* 2012;8(2):88-90.
 43. Kluczynski MA, Lamonte MJ, Mares JA, WactawskiWende J, Smith AW, Engelman CD, et al. Duration of physical activity and serum 25-hydroxyvitamin D status of postmenopausal women. *Ann Epidemiol.* 2011; 21:440-49.
 44. Gerdhem P, Ringsberg KA, Obrant KJ, Akesson K. Association between 25-hydroxyvitamin D levels, physical activity, muscle strength and fractures in the prospective population-based OPRA Study of Elderly Women. *Osteoporos Int.* 2005; 16:1425-31.

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