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Vitamin D in Obesity

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Abstract

Purpose of review: Vitamin D is essential for bone health, and may also have important functions in immunity and other systems. Vitamin D deficiency is common, and testing and supplementation is increasing. Serum vitamin D is lower in obese people; it is important to understand the mechanism of this effect and whether it indicates clinically significant deficiency.

Recent findings: Vitamin D is fat soluble, and distributed into fat, muscle, liver and serum. All of these compartments are increased in volume in obesity, so the lower vitamin D likely reflects a volumetric dilution effect and whole body stores of vitamin D may be adequate. Despite lower serum vitamin D, obese adults do not have higher bone turnover or lower bone mineral density (BMD). Patients undergoing bariatric surgery do have bone loss, and ensuring vitamin D sufficiency in these patients may help to attenuate bone loss.

Summary: Lower vitamin D in obese people is a consistent finding across age, ethnicity and geography. This may not always reflect a clinical problem. Obese people need higher loading doses of vitamin D to achieve the same serum 25OHD as normal weight.

Keywords vitamin D, obesity, bone,

Introduction

The main source of vitamin D in humans is production from the action of UVB light on cholesterol in skin. There is a small contribution from dietary sources such as oily fish and fortified foods. There are some geographical variations, for example in Scandinavia, dietary fish is a major source, and in the USA there is more food fortification. Vitamin D deficiency is very prevalent in the northern hemisphere; most of the UK population are vitamin D deficient in winter [1].

There is ongoing debate about the fundamental principles of defining vitamin D deficiency. The most commonly used measure of vitamin D status is total 25-hydroxyvitamin D (25OHD) which captures $25OHD_3$ and $25OHD_2$. 25OHD has a long serum half-life and is a technically easy measurement, but it has been proposed that free (non-protein-bound) 25OHD is a better measurement because it more closely reflects biologically available 25OHD status. There are variations in the sufficiency threshold recommended by different advisory bodies, with recommendations generally between 50 and 75 nmol/l [2,3].

Vitamin D is a pre- hormone. It is converted to the active hormone 1,25 dihyroxyvitamin D $(1,25(OH)_2D)$ by 25-hydroxylation in the liver and 1-hydroxylation in the kidney. The final step of 1-hydroxylation is tightly regulated to prevent hypercalcaemia.

The main form circulating form of vitamin D is 25OHD. It is protein-bound in circulation (about 85% to vitamin D bonding protein (VDBP), and about 15% to albumin). 25OHD is fat soluble, and distributed into fat, muscle and liver, with smaller amounts into other tissues. 25OHD is cleared by 24-hydroxylation to $24,25(OH)_2D$ or other inactive metabolites.

Active vitamin D, $1,25(OH)_2D$ is required for gut absorption of dietary calcium. Severe vitamin D deficiency causes osteomalacia in adults and in children. Less severe insufficiency causes increased bone turnover and is associated with increased fracture risk. Vitamin D deficiency has been associated with a wide range of other disorders, particularly auto-immune disorders and cancers, but a causative role in these conditions is not yet well established.

Obesity is inversely correlated with serum 25OHD. This is a consistent finding in adults and children of different ethnicities in a range of geographic locations. This observation raises several questions: Is the low serum 25OHD a consequence or cause of obesity? What is the cause of low serum 25OHD in obesity? Does the low serum 25OHD in obesity have clinical consequences for bone or other systems? Do obese people need higher doses of vitamin D supplements? Does vitamin D supplementation in obesity ameliorate any of the metabolic consequences of obesity? What happens to vitamin D status when weight changes rapidly after weight loss surgery?

Relationship between body weight and vitamin D

Obese people have lower serum 25OHD than normal weight people, and serum 25OHD is inversely correlated with body weight, BMI and fat mass. This has been shown in adults and children in northern and southern Europe, Australia and New Zealand, Saudi Arabia, Latin America and in White, Black and Hispanic groups in the USA [4-12]. Serum 25OHD is about 20% lower in obese people than normal weight [4,5,13], and the prevalence of 25OHD is greater in obese people, reported at between 40-80% [4,9,12].

Other measures of vitamin D status (free 25OHD and $1,25(OH)_2D$) are also lower in obesity [4,14].

It is likely that low serum 25OHD is a consequence of obesity, rather than the cause of obesity. A large genetic study found that high BMI and genes that predispose to obesity decrease serum 25OHD, while low 25OHD and genes associated with low 25OHD have very little effect on obesity[15]. In meta-analysis, vitamin D supplementation has no effect on body weight or fat mass[16].

Causes of low 25OHD in obesity

There are a number of possible mechanisms that could cause low 25OHD in obesity.
There could be lower input due to lower dietary intake, lower sunlight exposure or impaired skin synthesis of vitamin D. Alterations in protein binding or faster metabolic clearance in obesity could lead to lower serum 25OHD. Or the lower serum 25OHD could be due to distribution of 25OHD into a larger whole body tissue volume, particularly if 25OHD were actively sequestered in other tissues.

Dietary vitamin D intake did not differ between obese and normal weight adults in a UK population [4]. Obese people may have less sunlight exposure than normal weight people [17] in some geographic regions, although in two UK studies, sunlight exposure did not vary with BMI [4,18]. When exposed to UVB, normal-weight and obese people have a similar cutaneous synthesis of vitamin D [19]. Clearly, diet and sunlight behaviours will vary between different geographic and cultural groups, and may be a contributory factor to lower vitamin D in some groups.

Serum VDBP and albumin concentrations affect the total 25OHD measurement, and there are genetic variations in VDBP which cause variation in the binding affinity of DBP. However, DBP and albumin do not differ between obese and normal weight people, and the distribution of DBP genotypes is similar in obese and normal weight groups [4,20].

If metabolic clearance rate of 25OHD were faster in obesity, the half-life of 25OHD would be shorter and circulating 25OHD would be lower. Measurements using a stable isotope 25OHD tracer show that metabolic clearance is similar in obese and normal weight adults [4], so this is unlikely to be the explanation for lower serum 25OHD.

The most important mechanism is probably volumetric dilution of 25OHD into greater tissue volume in obese people. 25OHD is distributed into serum, fat, muscle, liver and a small amount into other tissues [21], and all of these compartments are increased in obesity. The difference in serum 25OHD between obese and normal weight groups is greater in summer than in winter because the summer rise in serum 25OHD is less in obese people [4,22,23]. Obese people get similar sunlight exposure to normal weight people, and produce the same amount of vitamin D in response to sunlight exposure, but the synthesised vitamin is distributed into a larger volume, so the amount distributed into serum is less. There is a similar difference between obese and normal weight people in response to oral vitamin D dosing, with a smaller serum 25OHD rise in obesity [24]. The dose-response relationship of vitamin D supplementation in obese adults is BMI-dependent and curvilinear, consistent with volumetric dilution [25] **(Figure 1)**. It has been proposed that vitamin D is actively retained (sequestered) in fat but the modelling of the dose-response doesn't seem consistent with that hypothesis. There is experimental evidence that vitamin D concentration in abdominal subcutaneous fat increases with vitamin D supplementation [26,27], and one in vitro study suggests that adipocytes from insulin-resistant obese people might have impaired release of vitamin D [28]. However the distribution and concentration of vitamin D and 25OHD in subcutaneous and omental fat does not differ between obese and normal weight people, and the correlation between 25OHD in subcutaneous fat and serum is similar in obese and normal weight [29].

Does lower 25OHD have clinical consequences in obesity?

Usually, low total 25OHD, free 25OHD and 1,25(OH)$_2$D would lead to lower dietary calcium absorption, and increased bone turnover with lower bone mineral density (BMD). However, obese adults have lower bone turnover than normal weight, and higher BMD with thicker, denser cortices and greater trabecular number [30]. It is important to note that in contrast, obesity in children has adverse effects on bone strength [31].

This lack of adverse effects on bone may indicate that obese people are not truly vitamin D deficient; although serum 25OHD is lower due to volumetric dilution, their whole body vitamin D stores are greater due to the reservoir in their fat tissue, which maintains an equilibrium with serum 25OHD and a sufficient supply.

An alternative explanation is that obese people are vitamin D deficient but that other effects of obesity compensate for the effects of vitamin D deficiency. For example, greater skeletal loading or the action of hormones such as leptin, adiponectin or oestrogen are known to have positive effects on bone mass.

If obese people are truly vitamin D deficient, there may be implications for systems other than bone. Vitamin D deficiency has been associated with a large number of disorders, such as auto-immunity, cancer, neurodegenerative disease and metabolic syndrome. However, there is not yet clear evidence for a causative role of vitamin D deficiency in many of these conditions [32]. Obesity does increase the risk for several of these disorders but there are other possible mechanisms than low vitamin D for these associations, and the interaction of vitamin D and obesity in causation has not yet been clearly characterised [33].

In the US NHANES population study, low serum 25OHD was associated with higher all-cause mortality in postmenopausal women with normal waist circumference; the hazard ratio for the lowest versus the highest serum vitamin D quartile (< 36.5 nmol/l vs > 65.4 nmol/l) was 1.85 (95% CI 1.00 to 3.44). In women with abdominal obesity there was no association between serum 25OHD quartile and all-cause mortality (HR 0.96, 95% CI 0.52 to 1.76) [34]. This result suggests that obesity does modulate the relationship between serum vitamin D and health outcomes.

Does vitamin D supplementation improve the adverse metabolic profile in obesity?

A recent meta-analysis found only four or five good quality intervention studies for each of the metabolic parameters they considered (fat mass, blood pressure, lipids and glucose tolerance). There were some positive effects of vitamin D supplementation on fat mass, triglycerides, HDL cholesterol and oral glucose tolerance, but adverse effects on LDL cholesterol and blood pressure. However, due to the small numbers of studies and significant heterogeneity of methodology, the current evidence is insufficient to draw conclusions [35].

Inflammatory markers do not seem to be higher in vitamin D deficient obese people than in vitamin D replete obese people [36] and vitamin D supplementation has little effect on circulating inflammatory markers in overweight or obese people [37-39].

Vitamin D with weight loss surgery

Obesity surgery leads to substantial weight loss in most patients, often about 30% of their body weight. The two main types of surgery are gastric volume reduction (eg sleeve gastrectomy) or malabsorptive (most commonly Roux-en-Y gastric bypass (RYGB), and less commonly biliopancreatic diversion with gastric switch). As expected, obese people have low serum 25OHD pre-operatively. Most studies find that serum 25OHD increases in the first month after surgery [40], which may reflect the smaller volume of dilution with weight loss. One study tried to determine whether the vitamin D stored in fat was mobilised into serum during weight loss, but found no association between vitamin D concentration in fat at time of surgery and subsequent change in serum 25OHD [41]. With biliopancreatic diversion, serum 25OHD tends to decline immediately after surgery, which is probably due to the greater bile and fat malabsorption with this procedure [42].

In the longer term, vitamin D decreases. Five years after RYGB, with calcium and vitamin D supplementation in most patients, they are just vitamin D sufficient at about 50 nmol/l, but have parathyroid hormone above the reference range [43]. Bone density and microarchitecture deteriorates for at least two years after RYGB, beyond stabilisation of body weight, despite vitamin D supplementation which maintained serum 25OHD well into the replete range [44]. The bone loss is likely to be multifactorial, with decreased loading, changes in adipokines, gut hormones, insulin and sex hormones as well as other nutritional factors. Maintaining vitamin D deficiency after bariatric surgery may require higher doses than standard supplementation, but in combination with calcium and protein supplements and physical activity may attenuate bone loss [45].

Optimum vitamin D dosing for BMI

As explained above, the observed serum 25OHD of about 20% below the normal weight population in obese people may not indicate true deficiency or a clinical problem. However, despite higher total body stores, obese people can become vitamin D deficient for the same reasons as any other group. If obese people are treated to the same serum 25OHD repletion threshold as normal weight, higher loading doses will be required due the greater volume of distribution. Obese people may need repeated loading courses to achieve repletion. (Conversely, people with below-normal BMI may need lower doses, but more frequently). Drincic [25] describes the formula to calculate the required vitamin D dose based in BMI and the required increment in serum 25OHD. Because there are no alterations in protein binding or metabolic clearance rate in obesity, maintenance dose requirements should be similar to normal weight (although as above higher doses may be need after bariatric surgery).

Summary

Obesity is associated with lower serum vitamin D, assessed as total 25OHD, free 25OHD or $1.25(OH)_2D$. This is likely due to volumetric dilution into the greater volumes of serum, fat, muscle and liver. The observed lower vitamin D in obesity doesn't seem to have adverse consequences for bone health. There is less available information about consequences for other systems, but no clear evidence of adverse effects. Vitamin D supplementation has not yet been clearly shown to benefit the adverse metabolic profile in obesity. Vitamin D does decrease after bariatric surgery, and careful attention to vitamin D repletion might help to prevent bone loss in these patients. Obese people will

need greater loading doses of vitamin D to achieve the same serum 25OHD threshold as normal weight.

References

1.   Hypponen E, Power C. Hypovitaminosis D in British adults at age 45 y: nationwide cohort study of dietary and lifestyle predictors. AmJClinNutr 2007;85:860-8.
2.   Institute of Medicine. Dietary reference intakes for calcium and vitamin D. The National Academies Press; Washington DC. 2011.
3.   Holick MF, Binkley NC, Bischoff-Ferrari HA, et al. Evaluation, treatment, and prevention of vitamin D deficiency: an Endocrine Society clinical practice guideline. The Journal of clinical endocrinology and metabolism 2011;96:1911-30.
**4.   Walsh JS, Evans AL, Bowles S, et al. Free 25-hydroxyvitamin D is low in obesity, but there are no adverse associations with bone health. The American journal of clinical nutrition 2016;103:1465-71.
**Serum free and total 25OHD are low in obesity, and this is likely to be due to volumetric dilution rather than lower dietary intake, less sunlight exposure, altered protein binding or increased metabolic clearance. First study of 25OHD metabolic clearance in obesity using recently-described stable isotope tracer method.
5.   Macdonald HM, Mavroeidi A, Barr RJ, Black AJ, Fraser WD, Reid DM. Vitamin D status in postmenopausal women living at higher latitudes in the UK in relation to bone health, overweight, sunlight exposure and dietary vitamin D. Bone 2008;42:996-1003.
6.   Ardawi MS, Sibiany AM, Bakhsh TM, Qari MH, Maimani AA. High prevalence of vitamin D deficiency among healthy Saudi Arabian men: relationship to bone mineral density, parathyroid hormone, bone turnover markers, and lifestyle factors. Osteoporosis international : a journal established as result of cooperation between the European Foundation for Osteoporosis and the National Osteoporosis Foundation of the USA 2012;23:675-86.
7.   Shea MK, Houston DK, Tooze JA, et al. Correlates and prevalence of insufficient 25-hydroxyvitamin D status in black and white older adults: the health, aging and body composition study. JAmGeriatrSoc 2011;59:1165-74.
8.   Rajakumar K, de las HJ, Chen TC, Lee S, Holick MF, Arslanian SA. Vitamin D status, adiposity, and lipids in black American and Caucasian children. JClinEndocrinolMetab 2011;96:1560-7.
9.   Lagunova Z, Porojnicu AC, Lindberg F, Hexeberg S, Moan J. The dependency of vitamin D status on body mass index, gender, age and season. Anticancer Res 2009;29:3713-20.
10.   McKinney K, Breitkopf CR, Berenson AB. Association of race, body fat and season with vitamin D status among young women: a cross-sectional study. ClinEndocrinol(Oxf) 2008;69:535-41.
11.   Palacios C, Gil K, Perez CM, Joshipura K. Determinants of vitamin D status among overweight and obese Puerto Rican adults. Annals of nutrition & metabolism 2012;60:35-43.
12.   Samuel L, Borrell LN. The effect of body mass index on optimal vitamin D status in U.S. adults: the National Health and Nutrition Examination Survey 2001-2006. Annals of epidemiology 2013;23:409-14.
13.   Ardawi MS, Qari MH, Rouzi AA, Maimani AA, Raddadi RM. Vitamin D status in relation to obesity, bone mineral density, bone turnover markers and vitamin D receptor genotypes in healthy Saudi pre- and postmenopausal women. Osteoporosis international : a journal established as result of cooperation between the European Foundation for Osteoporosis and the National Osteoporosis Foundation of the USA 2011;22:463-75.
14.   Lagunova Z, Porojnicu AC, Vieth R, Lindberg FA, Hexeberg S, Moan J. Serum 25-hydroxyvitamin D is a predictor of serum 1,25-dihydroxyvitamin D in overweight and obese patients. JNutr 2011;141:112-7.
15.   Vimaleswaran KS, Berry DJ, Lu C, et al. Causal relationship between obesity and vitamin D status: bi-directional Mendelian randomization analysis of multiple cohorts. PLoS medicine 2013;10:e1001383.
16.   Chandler PD, Wang L, Zhang X, et al. Effect of vitamin D supplementation alone or with calcium on adiposity measures: a systematic review and meta-analysis of randomized controlled trials. Nutrition reviews 2015;73:577-93.

17.   Kull M, Kallikorm R, Lember M. Body mass index determines sunbathing habits: implications on vitamin D levels. InternMedJ 2009;39:256-8.

18.   Macdonald HM, Mavroeidi A, Aucott LA, et al. Skin color change in Caucasian postmenopausal women predicts summer-winter change in 25-hydroxyvitamin D: findings from the ANSAViD cohort study. JClinEndocrinolMetab 2011;96:1677-86.

19.   Wortsman J, Matsuoka LY, Chen TC, Lu Z, Holick MF. Decreased bioavailability of vitamin D in obesity. AmJClinNutr 2000;72:690-3.

20.   Winters SJ, Chennubhatla R, Wang C, Miller JJ. Influence of obesity on vitamin D-binding protein and 25-hydroxy vitamin D levels in African American and white women. Metabolism 2009;58:438-42.

21.   Heaney RP, Horst RL, Cullen DM, Armas LA. Vitamin D3 distribution and status in the body. Journal of the American College of Nutrition 2009;28:252-6.

22.   Bolland MJ, Grey AB, Ames RW, et al. The effects of seasonal variation of 25-hydroxyvitamin D and fat mass on a diagnosis of vitamin D sufficiency. AmJClinNutr 2007;86:959-64.

23.   Blum M, Dolnikowski G, Seyoum E, et al. Vitamin D(3) in fat tissue. Endocrine 2008;33:90-4.

24.   Gallagher JC, Yalamanchili V, Smith LM. The effect of vitamin D supplementation on serum 25(OH)D in thin and obese women. The Journal of steroid biochemistry and molecular biology 2013;136:195-200.

25.   Drincic A, Fuller E, Heaney RP, Armas LA. 25-hydroxyvitamin d response to graded vitamin d3 supplementation among obese adults. The Journal of clinical endocrinology and metabolism 2013;98:4845-51.

26.   Didriksen A, Burild A, Jakobsen J, Fuskevag OM, Jorde R. Vitamin D3 increases in abdominal subcutaneous fat tissue after supplementation with vitamin D3. European journal of endocrinology / European Federation of Endocrine Societies 2015;172:235-41.

27.   Heaney RP, Recker RR, Grote J, Horst RL, Armas LA. Vitamin D(3) is more potent than vitamin D(2) in humans. JClinEndocrinolMetab 2011;96:E447-E52.

*28.   Di Nisio A, De Toni L, Sabovic I, et al. Impaired release of vitamin D in dysfunctional adipose tissue: new cues on vitamin D supplementation in obesity. The Journal of clinical endocrinology and metabolism 2017.
*In vitro study of vitamin D uptake and release in adipocytes from healthy and insulin-resistant people. Suggests that insulin-resistant adipocytes might sequester vitamin D

**29.   Carrelli A, Bucovsky M, Horst R, et al. Vitamin D Storage in Adipose Tissue of Obese and Normal Weight Women. Journal of bone and mineral research : the official journal of the American Society for Bone and Mineral Research 2017;32:237-42.
**Described distrubution of vitamin D across fat compartments in normal weight and obese women. Distribution and concentration of vitamin D in subcutaneous and omental adipose tissue, and correlation of adipose tissue concentration with serum concentration was similar in both groups.

30.   Evans AL, Paggiosi MA, Eastell R, Walsh JS. Bone density, microstructure and strength in obese and normal weight men and women in younger and older adulthood. Journal of bone and mineral research : the official journal of the American Society for Bone and Mineral Research 2015;30:920-8.

31.   Dimitri P, Bishop N, Walsh JS, Eastell R. Obesity is a risk factor for fracture in children but is protective against fracture in adults: A paradox. Bone 2012;50:457-66.

32.   Autier PB, M.; Pizot, C.; Mullie, P. Vitamin D status and ill health: a systematic review. Lancet Diabetes Endocrinol 2014;2:76-89.

33.   Shanmugalingam T, Crawley D, Bosco C, et al. Obesity and cancer: the role of vitamin D. BMC cancer 2014;14:712.

34.   Eaton CB, Young A, Allison MA, et al. Prospective association of vitamin D concentrations with mortality in postmenopausal women: results from the Women's Health Initiative (WHI). The American journal of clinical nutrition 2011;94:1471-8.

35.   Manousopoulou A, Al-Daghri NM, Garbis SD, Chrousos GP. Vitamin D and cardiovascular risk among adults with obesity: a systematic review and meta-analysis. European Journal of Clinical Investigation 2015;45:1113-26.

36.   Vilarrasa N, Vendrell J, Maravall J, et al. Is plasma 25(OH) D related to adipokines, inflammatory cytokines and insulin resistance in both a healthy and morbidly obese population? Endocrine 2010;38:235-42.

37.   Beilfuss J, Berg V, Sneve M, Jorde R, Kamycheva E. Effects of a 1-year supplementation with cholecalciferol on interleukin-6, tumor necrosis factor-alpha and insulin resistance in overweight and obese subjects. Cytokine 2012;60:870-4.

38.   Wamberg L, Kampmann U, Stodkilde-Jorgensen H, Rejnmark L, Pedersen SB, Richelsen B. Effects of vitamin D supplementation on body fat accumulation, inflammation, and metabolic risk factors in obese adults with low vitamin D levels - results from a randomized trial. European journal of internal medicine 2013;24:644-9.

39.   Jorde R, Sneve M, Torjesen PA, Figenschau Y, Goransson LG, Omdal R. No effect of supplementation with cholecalciferol on cytokines and markers of inflammation in overweight and obese subjects. Cytokine 2010;50:175-80.

*40.   Himbert C, Ose J, Delphan M, Ulrich CM. A systematic review of the interrelation between diet- and surgery-induced weight loss and vitamin D status. Nutr Res 2017;38:13-26.
*Systematic review of changes in serum vitamin D after diet-induced and surgical weight loss. Also the response of serum vitamin D to supplementation in both situations.

41.   Pramyothin P, Biancuzzo RM, Lu Z, Hess DT, Apovian CM, Holick MF. Vitamin D in Adipose Tissue and Serum 25-Hydroxyvitamin D After Roux-en-Y Gastric Bypass. Obesity(SilverSpring) 2011;19:2228-34.

42.   Aasheim ET, Bjorkman S, Sovik TT, et al. Vitamin status after bariatric surgery: a randomized study of gastric bypass and duodenal switch. The American journal of clinical nutrition 2009;90:15-22.

*43.   Switzer NJ, Marcil G, Prasad S, et al. Long-term hypovitaminosis D and secondary hyperparathyroidism outcomes of the Roux-en-Y gastric bypass: a systematic review. Obesity reviews : an official journal of the International Association for the Study of Obesity 2017;18:560-6.
* Describes change in serum vitamin D and PTH for more than five years after RYGB

**44.   Shanbhogue VV, Stoving RK, Frederiksen KH, et al. Bone structural changes after gastric bypass surgery evaluated by HR-pQCT: a two-year longitudinal study. European journal of endocrinology / European Federation of Endocrine Societies 2017;176:685-93.
**Important observation that bone density and microstructure deteriorate for two years after RYGB, despite vitamin D repletion and stabilisation of weight and metabolic parameters

**45.   Muschitz C, Kocijan R, Haschka J, et al. The Impact of Vitamin D, Calcium, Protein Supplementation, and Physical Exercise on Bone Metabolism After Bariatric Surgery: The BABS Study. Journal of bone and mineral research : the official journal of the American Society for Bone and Mineral Research 2016;31:672-82.
** Vitamin D, calcium and protein supplements with physical activity may attenuate bone loss assessed by DXA after RYGB.

Key points

Serum vitamin D is low in obese people across age, ethnicity and geography.

The low vitamin D is due to greater volumetric dilution, and may not necessarily indicate vitamin D deficiency.

Despite lower serum vitamin D, obese people have lower bone turnover and higher bone density than normal weight people.

Bariatric surgery does cause bone loss, and vitamin D supplementation may be important in this group of patients.

Obese people need higher loading doses of vitamin D than normal weight people to achieve a similar increment in serum vitamin D.

Figure legend

Figure 1 Serum response to oral vitamin D supplementation is BMI- and dose-dependent.

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