

Review Article

Childhood Obesity and Vitamin D Deficiency

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Abstract

The global escalation of childhood obesity and vitamin D deficiency are major public health issues. Both have been attributed to lifestyle changes. Obesity and vitamin D status are known to be associated, but the degree of linkage and whether it has any significance is unknown. Another important issue is the potential role of vitamin D in the development of the complications of obesity, because the health consequences of vitamin D deficiency in obese children and adolescents are not clear. This paper discussed the relationship between obesity and low vitamin D concentrations and its consequences, with reference to the possible underlying mechanisms.

The global escalation of childhood obesity is a major public health issue. Despite a known genetic contribution, the increase in pediatric obesity has been attributed mainly to diet and a sedentary lifestyle. Another increasingly prevalent public health concern, probably caused mainly by the same lifestyle factors, is vitamin D deficiency. Both conditions are often associated, and many studies confirmed a high prevalence of the vitamin D deficiency in obese patients. In children and adolescents that are overweight and obese it affects approximately 60% in comparison to 20% of non obese subjects [1-4]. Moreover, an inverse correlation between body mass index, fat tissue content and vitamin D levels had been proven in many studies [1-5]. Obesity and vitamin D status are known to be associated, but the degree of linkage and whether it has any significance is unknown. A recently published metaanalysis of 21 adult cohorts (up to 42,024 participants) revealed that higher BMI lead to lower vitamin D level, while any effects of lower increasing BMI are likely to be small [5]. These results confirm, that vitamin D deficiency is a consequence, not a cause of obesity, and that it is rather impossible, that vitamin D supplementation may assist in preventing or treating obesity. The possible explanations of a high prevalence of vitamin D deficiency in obese children and adolescents might be lower, compared to non obese children, dietary intake, reduced cutaneous synthesis due to less exposure to the sun, altered metabolism and sequestration in the adipose tissue. Despite these hypotheses, it had been shown in an elegant study of 686 community-dwelling individuals that a volumetric dilutional model accounted essentially for all the variability in serum vitamin D concentrations attributable to obesity. Even though the factors described above may be operative, they are effectively –captured by body weight. Once serum 25D concentrations in obese individuals are adjusted for body size, there is no longer a difference between obese and non-obese individuals [6,7]. Although the association between obesity and

in consequence low vitamin D concentrations is therefore well-established, it remains uncertain what the health consequences of this might be. It has been suggested, that low vitamin D levels may contribute to the development of insulin resistance and subsequent metabolic alterations. A strong association between insulin resistance and an excess of fat tissue has been recognized for decades, but there is still confusion on what the risk factors are. One of the candidates is a low vitamin D level. Some authors have pointed to low vitamin D status as an important risk factor of insulin resistance and impaired glucose metabolism independent of body adiposity [1]. One of the possible explanations might be a potential anti-inflammatory role of vitamin D. Since the immunomodulatory functions of vitamin D are incontestable, its deficiency in obese patients may coincide with enhanced systemic inflammation. It has also been shown that low-grade inflammation is associated with reduced insulin sensitivity. This theory has been supported by the results of a recently published pediatric study. The results showed an association between low vitamin D level, and activation of a pro-inflammatory, pro-diabetic and atherogenic pathways in obese children. It might justify an attempt to inhibit this process and prevent or treat metabolic syndrome by vitamin D supplementation [4]. Despite these promising results, another study failed to demonstrate any reduction in systemic inflammatory markers in adult participants receiving 7000 IU oral vitamin D daily [8]. There is a clear need for adequately-powered, prospective interventions in pediatric populations. To date the role of vitamin D supplementation in obesity prevention has been uncertain. Novel insights into the potential contribution of vitamin D in the development of insulin resistance were possible after the discovery of FGF, especially FGF23 and its endocrine action [9]. It has been shown that FGF23 may play a key role in the early phase of the decrease in insulin sensitivity in obese adolescents [10,11]. It has the potential to act as a hormone, and is pivotal in the control of phosphate and

vitamin D metabolism. However the exact mechanisms of the contribution of FGF23 to alterations in insulin metabolism in obese patients remains unclear, recent studies conducted on mice indicate that insulin signaling, or fat metabolism disturbances in the genetically ablated FGF23 subjects are mediated by vitamin D [12,13]. Some studies have pointed to the presence of low FGF23 levels in vitamin D deficient individuals, however it was not confirmed in larger groups [13]. Moreover, recent studies explicitly negate a simple correlation between vitamin D levels and insulin resistance in obese adolescents. They have shown that low vitamin D levels are one of the possible consequences of obesity rather than an independent risk factor of insulin resistance [2,3].

CONCLUSIONS

Obesity, and perhaps vitamin D deficiency, are among the most important modifiable risk factors for in number of chronic diseases. Nevertheless it remains controversial whether their association is significant, or if is a coincidental occurrence of two conditions with similar origin. However interventions to reduce BMI are expected to decrease the prevalence of vitamin D deficiency, the results of the vitamin D supplementation in obese patients remain unknown.

REFERENCES

1. Alemzadeh R, Kichler J, Babar G, Calhoun M. Hypovitaminosis D in obese children and adolescents: relationship with adiposity, insulin sensitivity, ethnicity, and season. *Metabolism*. 2008; 57: 183-91.
2. Creo AL, Rosen JS, Ariza AJ, Hidaka KM, Binns HJ. Vitamin D levels, insulin resistance, and cardiovascular risks in very young obese children. *J Pediatr Endocrinol Metab*. 2013; 26: 97-104.
3. Reyman M, Verrijn Stuart AA, van Summeren M, Rakhshandehroo M, Nuboer R, de Boer FK, et al. Vitamin D deficiency in childhood obesity is associated with high levels of circulating inflammatory mediators, and low insulin sensitivity. *Int J Obes (Lond)*. 2013; 10.1038/ijo.2013.75.
4. Vimaleswaran 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: 1549-1676.
5. Vanlint S. Vitamin D and obesity. *Nutrients*. 2013; 5: 949-56.
6. Drincic AT, Armas LA, Van Diest EE, Heaney RP. Volumetric dilution, rather than sequestration best explains the low vitamin D status of obesity. *Obesity (Silver Spring)*. 2012; 20: 1444-8.
7. Wamberg L, Cullberg KB, Rejnmark L, Richelsen B, Pedersen SB. Investigations of the anti-inflammatory effects of vitamin D in adipose tissue: results from an in vitro study and a randomized controlled trial. *Horm Metab Res*. 2013; 45: 456-62.
8. Kharitonov A. FGFs and metabolism. *Curr Opin Pharmacol*. 2009; 9: 805-10.
9. Wojcik M, Janus D, Dolezal-Oltarzewska K, Drozd D, Sztefko K, Starzyk JB, et al. The association of FGF23 levels in obese adolescents with insulin sensitivity. *J Pediatr Endocrinol Metab*. 2012; 25: 687-90.
10. Wojcik M, Dolezal-Oltarzewska K, Janus D, Drozd D, Sztefko K, Starzyk JB, et al. FGF23 contributes to insulin sensitivity in obese adolescents - preliminary results. *Clin Endocrinol (Oxf)*. 2012; 77: 537-40.
11. Razzaque MS, Sitara D, Taguchi T, St-Arnaud R, Lanske B. Premature aging-like phenotype in fibroblast growth factor 23 null mice is a vitamin D-mediated process. *FASEB J*. 2006; 20: 720-2.
12. Streicher C, Zeitz U, Andrukhova O, Rupprecht A, Pohl E, Larsson TE, et al. Long-term Fgf23 deficiency does not influence aging, glucose homeostasis, or fat metabolism in mice with a nonfunctioning vitamin D receptor. *Endocrinology*. 2012; 153: 1795-805.
13. Dai B, David V, Martin A, Huang J, Li H, Jiao Y, et al. A comparative transcriptome analysis identifying FGF23 regulated genes in the kidney of a mouse CKD model. *PLoS One*. 2012; 7: e44161.

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