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Editorial

Vitamin D and Obesity: Which One Is Affected by the Other?

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Obesity is a major nutritional problem in both the developed and developing countries, and its prevalence is dramatically increasing (1). Obesity is physiologically defined as *"fat accumulation in an excessive pattern in adipose tissue"*. Individuals with BMI>= 30 kg/m^2 are considered obese (2).

The worldwide prevalence of obesity was reportedly increased nearly twice from 1980 to 2008 (3) and it has been approximated that in 2030, an estimation of 1.12 billion adults will be obese globally (4). Based on a systematic review, the range of obesity prevalence in the national studies in Iranian adults is 12.6-25.9 (5).

Vitamin D deficiency is another increasingly prevalent health concern in both the developed and developing countries (7), and there are evidences that vitamin D metabolism, storage, and action both influence and are influenced by adiposity (8). Neyestani et al. reported high prevalence of vitamin D deficiency among Iranian school children; they also suggested proper nutritional intervention to combat this problem (6).

In a clinic-based sample of Puerto Rican adults, Gonzalez et al showed that individuals with higher BMI and waist circumference (WC) had a significant lower vitamin D status (9). In another study, serum vitamin D3 was inversely related to weight and BMI but not to adipose mass (10). A research showed that each unit increase in BMI is associated with 1.15% lower concentration of 25(OH)D, after adjusting for sex, age, laboratory batch, and month of measurement (11). Observational studies have reported an increased risk of vitamin D deficiency in obese individuals; however, explanation of causality is unclear (8). In a descriptive study on 66 patients with migraine, no association was found between serum 25(OH)D and BMI or WC (12). In support of this finding, a national study on 1090 adolescents reported no association between serum 25(OH)D and anthropometric measures (13).

On the contrary, another study on women with polycystic ovarian syndrome (PCOS) found an inverse association between vitamin D status and WC and waist to hip ration (WHR) (14). This observation was endorsed by a clinical trial in which raised serum 25(OH)D following 12-week daily intake of 1000 IU vitamin D-fortified Persian yogurt drink (doogh) resulted in a significant decrease in WC and BMI in the subjects with type 2 diabetes (T2D). A significant correlation between the changes of 25(OH)D and body fat mass (FM) was also reported (15). These findings were further supported in a clinical trial using supplementation with 50'000 IU a week in T2D subjects (16). The reasons for the differences of findings coming from different studies are many but high prevalence of vitamin D deficiency among Iranian population is likely to veil any possible relationship between vitamin D and anthropometric measures, notably in descriptive studies (17).

Randomized control trials on the effects of vitamin D supplementation on weight loss in obese subjects have provided contradictory results (18-20). We found that supplementation with vitamin D 50 µg daily for 3 months resulted in a significant reduction in the anthropometric indices in obese and overweight women with normal primary 25(OH) D serum levels (data unpublished). These results may be explained due to an up-regulation of genes involved in fatty acid oxidation and mitochondrial metabolism, leading to increased energy expenditure in the liver and skeletal muscles (21).

There are probably several mechanisms for the basis of low vitamin D concentration in obesity:

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1) High content of body fat that acts as a reservoir for vitamin D and increases its trapping, thus determining its low bioavailability. Also obesity is associated with decreased sunlight exposure, limited outdoor activity, or clothing habits that limits cutaneous vitamin D synthesis.

2) Hepatic steatosis in obese subjects may result in low synthesis of 25 (OH) D by the liver.

3) Higher leptin and interlukin 6 circulating levels, mostly secreted in obese persons, may have inhibitory effects on 25(OH)D synthesis via their receptors (22, 23)

The complex biochemical interactions between adipose tissue and vitamin D *in vitro* raise the question as to whether hypovitaminose D *per se* may contribute to obesity or inhibit weight loss *in vivo*. A few studies have shown that vitamin D does not appear to have a definite effect on weight; rather it may affect fat mass and distribution. This effect was seen when 25(OH) D level was less than 50 nmol/l; it was not observed when 25(OH) D was over this threshold (18,24). So it may be giving supplemental vitamin D to those who were replete has no additional effect.

An unresolved problem is that what dose of vitamin D should be used in obese persons to replete its stores and how to maintain normal 25(OH) D levels after repletion. The Institute of Medicine (IOM) guidelines suggest that there is no evidence that increases in vitamin D intake beyond the requirements for non-obese subjects can affect health conditions among obese people (25), while Endocrine Society guidelines suggest two to three times more vitamin D in obese persons for their age group to satisfy their body vitamin D requirement (26). These suggestions are supported by a recent study showing how the response to vitamin D supplementations was dependent on body size (27). So it may be said that one size does not fit all: the dose depends on the threshold of vitamin D to be achieved and on the body size.

In conclusion, it is important to consider both obesity as a predisposing condition to hypovitaminosis D, and vitamin D as a cofactor in the pathogenesis of obesity. More RCTs studies are needed to warrant the role of vitamin D supplementation in weight loss.

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