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Mini Review

Vitamin D and Insulin deficiency

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INTORDUCTION

Renewed interest in vitamin D, the so-called “sunshine vitamin,” has occurred currently because it has been linked to the entirety from cancer and heart disease to diabetes. Studies have continued to pour into the literature mentioning that vitamin D is a celeb when it comes to health. However, most of the research is based on observational, epidemiological studies, which are important for producing hypotheses, however, do no longer show causality.

VITAMIN D PHYSIOLOGY

Vitamin D exists in 2 forms: cholecalciferol (vitamin D3) and ergocalciferol (vitamin D2). Vitamin D3 is synthesized in the skin on exposure to solar ultraviolet B (UVB) radiation. During exposure to solar UVB radiation, 7-dehydrocholesterol in the skin is converted to pre-vitamin D3, which is immediately converted to vitamin D3 in a heat-dependent non-enzymatic process. The major circulating form of vitamin D (25OHD) is synthesized in the skin as cholecalciferol (vitamin D3) with very few food sources containing either ergocalciferol (vitamin D2) or cholecalciferol. Endogenous skin synthesis requires the skin to be exposed to ultraviolet B (UVB) light (290–315 nm wavelengths). Apart from fortified foods, dietary intake of vitamin D is limited (1).Vitamin D status is also influenced by non-modifiable genetic factors implicated in vitamin D metabolism. These can include inter-individual differences in vitamin D/calcium absorption and transport, or genetic polymorphisms of proteins and receptors involved with vitamin D. Certain polymorphisms of the vitamin D receptor, for example, have been found to lead to inter-individual differences in bone mineral density [2].

The challenge for health care providers and nutrition researchers is to decide whether vitamin D deficiency certainly causes or increases the incidence of certain diseases or whether, instead, low degrees of vitamin D are certainly coincidental given that the majority of the widely wide-spread population, regardless of disease, is likely to have insufficient levels of vitamin D. In other words, do people who develop disorder states just happen to be deficient in vitamin D, or do low levels of vitamin D cause the disease? Will supplementation with vitamin D prevent diseases, and can it be used to treat diseases such as diabetes?

Low 25-hydroxyvitamin D (25OHD) levels are highly prevalent among type 2 diabetic patients [3, 4]. The association between vitamin D and type 2 diabetes 5,6) may be explained by the effects of vitamin D on the regulation of insulin secretion or sensitivity or the attenuation of systemic inflammation [7].

Vitamin D and Insulin Secretion.

Vitamin D can promote pancreatic beta cell function in several ways. The active form of vitamin D (1,25(OH)2D), enters the beta cell from the circulation and interacts with the vitamin D receptor-retinoic acid x-receptor complex (VDRRXR),which binds to the vitamin D response element (VDRE) found in the human insulin gene promoter, to enhance the transcriptional activation of the insulin gene and increase the synthesis of insulin. Vitamin D may promote beta cell survival by modulating the generation (through inactivation of NF-kB) and effects of cytokines. The anti-apoptotic effect of vitamin D may also be mediated by down-regulating the Fas-related pathways (Fas/Fas-L).Activation of vitamin D also occurs intracellularly by 1-alpha-hydroxylase, which is expressed

in pancreatic beta cells. Vitamin D also regulates calbind in, a cytosolic calcium-binding protein found in beta cells, which acts as a modulator of depolarization-stimulated insulin release via regulation of intracellular calcium. Calbind in may also protect against apoptotic cell death via its ability to buffer intracellular calcium. The effects of vitamin D may be mediated indirectly via its important and well-recognized role in regulating extracellular calcium (Ca21), calcium flux through the beta cell and intracellular calcium ([Ca21] i). Alterations in calcium flux can directly influence insulin secretion, which is a calcium-dependent process. insulin target tissues may contribute to peripheral insulin resistance (6,7) via an impaired insulin signal transduction,(8,9) leading to decreased glucose transporter activity (10).

Vitamin D is supposed to improve the body's sensitivity to insulin and so reduces the insulin resistance risk, that often a primer to type 2 diabetes. Some scientists consider that this vitamin helps in regulation of the production of insulin in the pancreas. Finally, vitamin D insufficiency has been associated with increased fat infiltration in skeletal muscle, which appears independent of body mass and is thought to contribute to a decreased insulin action (11).

THE INFLUENCE OF VITAMIN D SUPPLEMENTATION ON DIABETES

The potential effect of vitamin D supplementation appears to be more prominent among persons who are at high risk for diabetes (e.g. prediabetes). In a post hoc subgroup analysis conducted using data from a completed trial designed for fractures, combined vitamin D3 (700 IU/d) and calcium carbonate (500 mg/d) supplementation prevented the rise in insulin resistance (HOMA-IR) and fasting plasma glucose (FPG) in people with impaired fasting glucose, but not in individuals with normal fasting glucose at baseline,(12)suggesting that vitamin D may benefit only individuals at high risk for diabetes. In this study, the reduction in FPG over 3 years was similar to the reduction in FPG achieved with metformin or lifestyle, in the Diabetes Prevention Program, which was associated with a 31% to 58% decrease in incident diabetes(13).In the Calcium and Vitamin D for type 2 Diabetes Mellitus study, vitamin D supplementation(2000 IU/d) in adults at risk for type 2 diabetes improved beta cell function and had anearly statistically significant effect on the rise in A1c values(14).Most studies investigating the effect of Vitamin D supplementation on patients with type 2 diabetes have not reported glycemic control to be significantly impacted (15). A recent meta-analysis (16) reported that there is insufficient evidence of a beneficial effect to recommend vitamin D supplementation as a way of improving glycemic control in type 2 diabetic patient.

However, the role of vitamin D in regulating blood glucose is remained poorly understood, the status of vitamin D plays a role in the incidence and treatment of diabetes. Optimal vitamin D levels in the serum may be different for people at risk of diabetes, and diabetics. According to Danesco *et al.,* "Both animal and human studies revealed that adequate vitamin D supplementation may decrease type 1 diabetes and possibly type 2 diabetes and enhance metabolic control in the case of diabetes." Mechanisms are uncleared and need more investigation'.

CONCLUSION

Oral vitamin D supplementation has shown better effects in enhancing serum 25(OH)D levels and reducing insulin resistance compared with placebos among type 2 diabetes patients. However, it did not appear to influence FBG, HbA1c and fasting insulin levels. Large dosage, short-term vitamin D supplementation was most likely to yield preferred changes in vitamin D deficient, non-obese groups, Asians, especially Middle Easterners, and patients with optimal glycemic control at baseline. Additional large well-designed studies with longer duration are required to further clarify the impacts of BMI and baseline HbA1c. D There is currently insufficient evidence of beneficial effect to recommend vitamin D supplementation as a means of improving glycaemia or insulin resistance in patients with diabetes, normal fasting glucose or impaired glucose tolerance.

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