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ABOUT COVER

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LETTER TO THE EDITOR

Effects of vitamin family members on insulin resistance and diabetes complications

Hong-Jin Chen, Min Wang, Ding-Min Zou, Gui-You Liang, Si-Yuan Yang

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Abstract

The following letter to the editor highlights the article "Effects of vitamin D supplementation on glucose and lipid metabolism in patients with type 2 diabetes mellitus and risk factors for insulin resistance" in *World J Diabetes* 2023 Oct 15; 14 (10): 1514-1523. It is necessary to explore the role of vitamin family members in insulin resistance and diabetes complications.

Key Words: Vitamin; Insulin resistance; Diabetes complications; Letter

Core Tip: Vitamins are a class of small molecular organic compounds that cannot be synthesized or are synthesized in extremely small amounts by the body. Many recent studies have shown that vitamin supplementation plays an important role in inhibiting inflammation, controlling blood sugar, and promoting insulin secretion in diabetic patients. However, the function of vitamin for diabetes remains to be studied. It is clinically significant to explore the effects of other vitamin family members on insulin resistance and diabetes complications.

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TO THE EDITOR

We read the article by Sun *et al*[1] entitled "Effects of vitamin D supplementation on glucose and lipid metabolism in patients with type 2 diabetes mellitus and risk factors for insulin resistance". The authors found that 25 hydroxyvitamin D3 [25(OH)D3], a vitamin D deficiency marker, is an independent risk factor for insulin resistance (IR) and promotes glucose metabolism. Many recent studies have shown that vitamin D supplementation plays an important role in inhibiting inflammation, controlling blood sugar, and promoting insulin secretion in diabetic patients[2,3]. Therefore, it is clinically significant to explore the effects of other vitamin family members on IR and diabetes complications.

With societal and economic development, the prevalence of diabetes continues to increase. Approximately 1.6 million deaths per year are attributed directly to diabetes[4]. Currently, diabetes is classified primarily as type 1 diabetes (T1D) and T2D. It is estimated that by 2030, 578 million people worldwide will be diagnosed with diabetes[5]; T2D is the most common diagnosis, accounting for approximately 90%. An increasing number of studies show that most T2D patients have IR[6]. IR is a weakening of the body's response to insulin, resulting in increased blood sugar levels; physiologically, IR is defined as a state of reduced responsiveness of insulin-targeted tissues to high insulin levels[7]. When IR begins to occur, insulin levels increase to meet normal insulin requirements; however, over time, these increased levels lead to hyperglycemia-induced islet β cell failure, chronic hyperinsulinemia, and eventually T2D.

Vitamins are a class of small molecular organic compounds that cannot be synthesized or are synthesized in extremely small amounts by the body. Vitamins are necessary to maintain the normal physiological functions of the human body, and they must be obtained from food. Primarily, vitamins fall into two categories: fat-soluble vitamins and water-soluble vitamins. Many recent studies have shown that vitamins are involved in regulating T2D *via* IR. In fact, vitamin K4 supplementation can improve IR in patients with T2D[8]. Vitamin K deficiency under β cell stress may lead to β cell dysfunction by reducing endoplasmic reticulum Gla protein (ERGP) gamma carboxylation, thus increasing the risk of T2D, particularly in the case of overnutrition. Gamma carboxylated ERGP is needed to prevent uncontrolled insulin secretion by β cells and maintain normal insulin secretion[9]. A recent review reported that vitamin E, especially alpha tocopherol, has been shown to reduce lipid peroxidation, and the superoxide produced by vitamin E can damage the β cell structure as well as vital functional components for maintaining normal glucose concentrations[10]. Another study has shown that deficiencies in folic acid, vitamin B6, and vitamin B12 can lead to dyslipidemia, vascular endothelial dysfunction, abnormal glucose tolerance, and oxidative stress, leading to IR[11]. Pramono *et al*[12] have shown that obesity and IR are usually related to vitamin D deficiency. Vitamin D directly stimulates insulin secretion by reducing pancreatic β cells through vitamin D receptor (VDR) and improves peripheral IR. Moreover, VDR deficiency in cardiovascular tissue increases ventricular fibroblast mass dysregulation and accelerates the myocardial fibrosis process[13]. Studies have also shown that serum vitamin D deficiency aggravates inflammation due to circulating gamma-delta T cells in T2D patients. 1α,25(OH)2D3/fructose-1,6-bisphosphatase 1 (FBP1) signal transduction can inhibit glycolysis in γδ T cells by promoting targeted FBP1 expression, thereby driving Akt/p38 MAPK dephosphorylation. This can also reduce inflammatory cytokine production (TNF-α and IFN-γ) in γδ T cells to alleviate IR. The role of vitamin D in IR regulation to alleviate diabetes may be mediated by stimulating insulin receptor expression, improving insulin levels, and regulating cytokine expression and the calcium pool in various tissues[14]. Upon examining the mechanism through which vitamin D affects IR, supplementation with 1,25(OH)2D3 combined with insulin was shown to significantly improve the expression of glucose transporter 4, a key protein in regulating glucose metabolism and maintaining glucose homeostasis through glucose uptake[15].

Further studies have found that vitamin D is involved in the pathological process of multiple diabetes complications. In diabetic cardiomyopathy, the 1,25-dihydroxyvitamin D receptor inhibits autophagy by inhibiting nuclear FOXO1 translocation, thereby alleviating diabetic heart damage[16]. Vitamin D also improves blood glucose and insulin levels, reduces NF-kB activity in heart tissue, and down-regulates advanced glycosylation end products and the hexosamine pathway to alleviate diabetic cardiomyopathy[17]. In diabetic kidney disease (DKD), vitamin D deficiency up-regulates zinc finger e-box binding homeobox and down-regulates miR-200b, which promotes the epithelial-mesenchymal transformation process and changes the renal structure and function of diabetic rats to accelerate DKD development[18]. Vitamin D supplementation can effectively reduce albuminuria and creatinine, markers of kidney disease in diabetic nephropathy patients[19]. 1,25(OH)2D3 also significantly inhibits the expression of sirtuin 1 (SIRT1) during oxidative stress[20], thereby attenuating renal oxidative damage. Increasing evidence has indicated that the vitamin D-VDR-RXR complex inhibits macrophage infiltration and immune effects in diabetic nephropathy[21]. Moreover, vitamin D deficiency is a potential risk factor for diabetic retinopathy (DR)[22]. Vitamin D is a strong antioxidant that can significantly reduce free radical formation, exert anti-inflammatory effects, and regulate autophagy and apoptosis; consequently, vitamin supplementation can help reduce the destructive effects of free radicals on DR[23]. Neuroprotective effects are exerted through the SIRT1/nrf-2/NF-kB signaling pathway[24]. 25(OH)D3 may inhibit oxidative stress in human retinal microvascular endothelial cells induced by high glucose-mediated miR-93 down-regulation[25]. Supplementation with vitamin D also improves neuropathy[26], which might be related to its regulation of neurotrophic factor levels and neuronal calcium homeostasis. Additional studies have shown that vitamin D deficiency has a greater effect on

the long-term chronic complications of diabetes, especially in patients with painful diabetic neuropathy, where vitamin D supplementation can effectively improve pain symptoms and nerve function in patients[27].

In addition, IR-induced glucose abnormalities and lipotoxicity, resulting in unbalanced fatty acid intake, are key factors in diabetic cardiomyopathy[28]. Abnormal coronary microcirculation, mitochondrial dysfunction, subcellular component abnormalities, and myocardial insulin signaling and calcium homeostasis impairments were observed in IR states. These pathophysiological changes can lead to diastolic dysfunction, fibrosis[29], and hypertrophy, ultimately causing heart failure[30]. Impaired insulin signaling is an important pathophysiological abnormality in diabetic cardiomyopathy. However, supplementation with vitamin D can significantly improve glucose tolerance and insulin sensitivity[31]. Vitamin D plays a key role in insulin receptor expression and increases glucose transporter function in insulin reactivity [13].

In summary, Sun *et al*[1] investigated the protective effects of vitamin D supplementation on IR in diabetic patients, but they did not include a pathological exploration of diabetic complications. The mechanism through which vitamin D acts in the treatment of IR and multiple diabetes complications remains to be studied.

FOOTNOTES

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