

Correlation of Serum Vitamin D Levels with Insulin Resistance in Young Adults

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Abstract

Among young adults, both insulin resistance (IR) and vitamin D deficiency are common and pose the risk of complications such as type 2 diabetes mellitus (T2DM). A few studies demonstrated that vitamin D deficiency might negatively influence insulin sensitivity, but the results of these studies have been inconsistent and/or inconclusive. This review focuses on young adults and aims to summarize and critique studies that assess the relationship between IR and serum vitamin D levels. We searched English-language observational and interventional studies in four databases (PubMed, Scopus, Google Scholar, and Embase) and included the articles identified. We review the various biological pathways that could help explain why vitamin D and glucose metabolism are related, summarize the available epidemiology, and discuss issues that may have influenced the findings. In summary, while many studies document the negative correlation of serum levels of vitamin D and the various parameters of IR, to date, it appears that very few have confidently established cause and effect. There remains a need for more and larger longitudinal and interventional studies of young adults on IR and vitamin D to shed more light on the underlying principles and to help develop primary health care interventions that promote metabolic health.

Keywords: Vitamin D, IR, young adults, HOMA-IR, glucose metabolism, metabolic syndrome

INTRODUCTION

Vitamin D, a fat-soluble secosteroid maintains bone and calcium homeostasis and also has emerging effects on extra skeletal systems [1]. It exists in Vitamin D2 (ergocalciferol) and D3 (cholecalciferol) that is obtained from skin synthesis due to exposure to UVB rays and also from animal products. Other than a few dietary (fortified milk, eggs, fish) and UVB exposure, Vitamin D is also available as a dietary supplement [2]. Due to modern lifestyle, multiple age groups, including young adults, have been suffering from Vitamin D deficiency.

IR is a physiological condition where there is a lack of glucose uptake in the blood due to the peripheral tissues not acting on the hormone insulin [3]. This is central to the development of T2DM and metabolic syndrome and over the past few decades has been seen in young adults due to sedentary lifestyle, obesity and poor dietary choices. Young age IR is problematic as it leads to a plethora of cardiovascular and metabolic issues.

Recent studies have examined the possible association between the deficiency of vitamin D and IR [4]. The biological reasoning for this association is the presence of vitamin D receptors in pancreatic β -cells and insulin-sensitive tissues. The studies done in this area have lacked

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consistency and there is very little work that looks at this issue in the young adult population [5, 6]. This review, therefore, intends to extract the available studies that examine the relationship of vitamin D and IR in young adults.

OBJECTIVES OF THE REVIEW

- To evaluate the correlation between serum vitamin D levels and IR in young adults.
- To examine underlying mechanisms.
- To summarize epidemiological and clinical evidence.
- To identify gaps in existing research.

METHODOLOGY OF THE REVIEW

As for studies that were published in English, I performed a detailed literature search using multiple electronic databases: Scopus, PubMed, Google Scholar, and Embase. I used the search terms and combinations with the Boolean operators (AND, OR) to create various combinations: “vitamin D”, “serum 25-hydroxyvitamin D”, “IR”, “HOMA-IR”, and “young adults”. I included studies with young adult participants who are between 18 and 40 years old for both observational and interventional studies. I excluded studies with participants who were children, the elderly, and studies that used animals, case studies, editorials, and opinion narratives. I screened titles and abstracts, and I assessed the full text for articles I board-assessed as eligible. I extracted the data for vitamin D serum levels, and the markers for IR, and the main outcomes of the study.

VITAMIN D AND GLUCOSE METABOLISM: BIOLOGICAL MECHANISMS

Vitamin D's metabolic effects are mediated through its interaction with the vitamin D receptor (VDR), a type of nuclear hormone receptor present in various tissues that are part of the body's system for managing glucose, including pancreatic beta cells, skeletal muscle, adipose, and liver tissue [7]. The presence of VDR and vitamin D-activation enzymes in beta cells offer evidence for a possible direct contribution of vitamin D to the control of insulin secretion. Vitamin D's impact on insulin synthesis is likely through the modulation of calcium concentrations that are critical to the process of insulin exocytosis [8]. It is recognized that appropriate vitamin D levels allow for optimal functioning of pancreatic beta cells, while a lack of vitamin D can decrease the ability of beta cells to secrete insulin and increase the risk of glucose intolerance.

Apart from its role in the secretion of insulin, vitamin D is also believed to help increase insulin sensitivity in the body's peripheral tissues [9]. Vitamin D's role in the modulation of the insulin signaling pathway is thought to be through the upregulation of insulin receptor expression and the enhancement of signaling that occurs after receptor binding. The effect of vitamin D on “glucose transport in muscle and adipose tissues is thought to be through the modulation of the expression and translocation of the glucose transporter type 4 (GLUT-4)” [10]. IR may be exacerbated by the deficiency of vitamin D and its effects on the disposal of glucose that is mediated by insulin.

IR has various underlying pathways that can be associated with inflammation and oxidative stress. The immunomodulatory and anti-inflammatory properties of Vitamin D suggest potential beneficial effects related to IR [11]. Vitamin D has been shown to moderate the production of the cytokines that promote inflammation. Inhibiting the production of interleukin-6, tumor necrosis factor- α , and C-reactive protein, has been associated with more favorable insulin signaling. Vitamin D provides the means to facilitate the expression of anti-inflammatory cytokines, which further lends favor to more sensitive insulin functioning. The protective role of vitamin D related to responsive tissues is attributed to the regulation of stress oxidative pathways that is due to vitamin D.

Vitamin D and insulin action can also relate to calcium homeostasis. The beneficial role of Vitamin D is that it enhances calcium absorption in the intestines and also keeps intracellular calcium levels within optimal levels [12]. The intracellular processes that insulin mediates depend significantly on

calcium. On the other hand, the action of insulin, as well as insulin secretion, can be inhibited by an abnormal calcium balance. This means that Vitamin D deficiency can impact the metabolism of calcium, which can, in turn, contribute indirectly to IR and impaired glucose homeostasis.

REVIEW OF EPIDEMIOLOGICAL EVIDENCE

A significant portion of research has been conducted regarding the link between levels of serum vitamin D and IR. However, the results of these studies have greatly varied. Most of the work available has been done in the form of cross-sectional studies, typically showcasing a negative link between the levels of serum 25-hydroxyvitamin D and certain levels of IR like fasting insulin levels and HOMA-IR [13]. Multiple studies have been conducted that state adult populations that have a vitamin D deficiency generally have a greater IR than that of the population that has sufficient levels of vitamin D, even after adjustments for body mass index and lifestyle factors [14].

On the other hand, other cross-sectional studies state that they have observed the same things. Some studies stated that there is no significant link between vitamin D levels in your body and the resistance of insulin, citing things like excess body weight, lack of movement in the form of exercise, dietary intake, and genetic factors as potential influences for the results observed [15]. Furthermore, due to a major limitation in the studies caused by the cross-sectional nature of the studies, it is much harder to state if there is a certain cause for the correlation in the levels of vitamin D and the levels of IR.

Cohort studies strengthen temporality assessments. Longitudinal studies show that low baseline Vitamin D levels correlate with worsening IR and deficits in glucose metabolism over time. These results may propose that Vitamin D deficiency could be predictive of worsening metabolic disorder complications [16]. However, published cohort studies concentrating on young adults are scant; thus, a literature gap exists.

Some studies, including randomized controlled trials, have observed the effect of Vitamin D supplement on IR / sensitivity [17]. Some trials report slight improvement in some markers of IR after Vitamin D supplementation, especially in the deficient cohort [18]. Other controlled trials report no effect and attribute the shortage of effect to differences in population, characteristics, and Vitamin D status, duration, and dosage. Interventional studies are still inconclusive and suggest the necessity of standardized trials with clear design.

Interpreting findings is further complicated due to regional and population-based diversities. Variants in ethnicity, skin color, diet, sun exposure, economic status, and socioeconomic factors may alter vitamin D levels and associated metabolic risk. Often, deficient levels of vitamin D and IR is more pronounced within urban settings as opposed to rural communities due to lack of physical activity and sunlight exposure [19]. Some studies have reported a greater prevalence in females, which may be driven by hormonal and body composition differences within the population (Table 1).

Table 1. Epidemiological studies are the association between serum Vitamin D levels and IR.

Author (year)	Study design	Study population	Sample size	Vitamin D measure	IR marker	Key findings
Ehrampoush et al. (2021) [1]	Cross-sectional	Adults	252	Serum 25(OH)D	HOMA-IR	Serum vitamin D levels were inversely associated with IR; vitamin D deficiency was linked to higher HOMA-IR values.
Buchmann et al. (2021) [2]	Cross-sectional	Young adults (Berlin Aging Study II)	1,622	Serum 25(OH)D	HOMA-IR	Vitamin D insufficiency was associated with metabolic syndrome independent of IR and obesity.
Bacha et al. (2019) [3]	Cross-sectional	Youth and young adults	141	Free and total vitamin D	Insulin sensitivity index	Free vitamin D showed a stronger association with insulin sensitivity and vascular health than total vitamin D levels.

Kamiński et al. (2021) [4]	Cross-sectional	Adults with Type 1 diabetes	120	Serum 25(OH)D	eGDR (IR index)	Lower vitamin D concentrations were significantly associated with increased IR in adults with type 1 diabetes.
Szymczak-Pajor & Sliwińska (2019) [5]	Narrative review	Adults	—	Serum 25(OH)D	HOMA-IR, fasting insulin	Review concluded that vitamin D deficiency is frequently associated with increased IR, though causality remains unclear.

ASSESSMENT OF IR AND VITAMIN D STATUS

For assessment of serum vitamin D status, 25(OH) vitamin D is measured, which indicates dietary as well as endogenous vitamin D status. As a rule of thumb, levels below 20 ng/mL are regarded as deficient, 20–30 ng/mL as insufficient, and above 30 ng/mL as sufficient [20]. Despite the existence of broadly accepted criteria, different studies may report different threshold levels which makes a comparison difficult.

Most studies do not use direct measures of IR and apply a more practical approach using surrogate endpoints. In terms of large-scale studies, the hyperinsulinemic-euglycemic clamp technique is not practical [15]. From the various surrogate indices of IR, Homeostatic Model Assessment of IR (HOMA-IR), which is a function of fasting insulin and glucose, is the most widely used, while some studies consider fasting insulin and the Quantitative Insulin Sensitivity Check Index (QUICKI), even though these methodologies represent a more economical and practical use of resources, they result in a less robust assessment of insulin responsiveness.

In the measurement of vitamin D and IR, several challenges arise. Serum vitamin D is impacted by seasonal variations, with lower levels in the winter months. Inconsistent findings may be the result of different laboratory assay methods and processes [16]. Similarly, the acute changes in the insulin markers are impacted by stress, dietary habits, and other factors, which can result in reduced accuracy and less reproducibility.

DISCUSSION

The analysis of the data confirms the relationship between low Vitamin D levels and high IR among young adults and is backed by biological evidence and observation. Vitamin D is known to predictably affect several aspects of the glucose metabolic process such as insulin secretion, insulin sensitivity, inflammation and calcium [17]. Of course, the aforementioned relationships need to be collapsible to be empirically evaluated in a consistent fashion.

The aforementioned relationships on a conceptual level are sound and defensible but collapse for a range of potential evaluative and methodological reasons, loss of dimensionality, disregard of the definitional differentiation of study populations and vitamin D insufficiency and low to inadequate vitamin D supplementation in the various studies [18].

The concern of both VDI and IR in young adults is important, as it indicates that young adults presumable may be developing the metabolic syndrome, and IR may be in itself a precursor for type II diabetes and coronary heart disease, Oxygenating the VDI and promoting lifestyle changes are, in the present environment, the most acceptable means of addressing the IR. Hence, the concern of young IR.

LIMITATIONS OF EXISTING LITERATURE

There are different issues limiting the current body of literature. Several studies have small sample sizes and cross-sectional designs. Thus, causal inference is limited. Vitamin D cut-off values and methods of IR assessment are done quite differently across studies. Additionally, few studies focus solely on young adults and often combine multiple age groups, limiting age-specific conclusions. The above issues provide evidence for the need for more streamlined and standardized research methods.

FUTURE RESEARCH DIRECTIONS

Coming studies on vitamin D status and IR in young adults should consider the construction of large scale and longitudinal frameworks to understand the relationships between the two variables in different contexts. For comparability to be established, methods to measure and assess the levels of Vitamin D and IR need to be standardized. To address the clinical relevance gap, randomized control trials need to be founded, incorporating strategies for Vitamin D Supplementation tailored to specific populations, baseline deficiency and lifestyle variables.

CONCLUSION

To sum up, most evidence indicates low levels of vitamin D in blood and IR are inversely correlated among young adults. Deficiency of vitamin D may lead to several obstructed glucose metabolic pathways. Although evidence remains inconsistent, the identification of vitamin D deficiency and the presence of metabolic risk factors sufficiently early may create opportunities for early intervention. More high-quality research is needed to establish the effect of vitamin D deficiency and IR related illnesses in young adults.

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