Vitamin D

1- Vitamin D Receptor/Vitamin D Response Element Directly Modulate Nestin Transcription to Ameliorate PAN-Induced Podocyte Morphological Changes

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Abstract

Background: The change of podocyte morphology is a pathologic feature of chronic kidney disease. Several studies have suggested that vitamin D plays a role in the protection of podocytes, but the underlying mechanism remains unclear. Methods: The effects of paricalcitol on podocyte injury were tested in a puromycin aminonucleoside (PAN)-induced rat model and cultured mouse podocytes. Proteinuria, podocyte foot process (FP) effacement, and the expression of nestin and vitamin D receptor (VDR) were evaluated. VDR-siRNA or plasmids containing VDR-shRNA were transfected into podocytes to silence VDR expression. Chromatin immunoprecipitation (ChIP) and luciferase reporter assays were performed to verify the connection between VDR and nestin gene expression. Results: Paricalcitol significantly alleviated proteinuria and podocyte FP effacement in PAN-induced nephrosis, which was accompanied by increased VDR expression in the glomeruli. Paricalcitol also inhibited PAN-induced nestin overexpression in the glomeruli. In an in vivo study, PAN significantly inhibited VDR protein expression, stimulated nestin protein expression, and resulted in nestin filament derangement in mouse podocytes, while paricalcitol treatment abolished these effects. In contrast, downregulation of VDR resulted in derangement and overexpression of nestin. ChIP assays demonstrated the presence of a vitamin D response element (VDRE) in the nestin promoter, and paricalcitol enhanced the binding of VDR to VDRE. Furthermore, luciferase reporter assays of the nestin promoter fragment showed that paricalcitol effectively repressed nestin reporter gene expression after PAN treatment, and mutation of VDRE abolished this effect. Conclusions: Paricalcitol directly regulates nestin transcription through the interaction of VDR/VDRE, thereby preventing morphological changes of podocytes in PAN nephropathy.

Keywords
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2- Immunologic Effects of Vitamin D on Human Health and Disease

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Abstract

Vitamin D is responsible for regulation of calcium and phosphate metabolism and maintaining a healthy mineralized skeleton. It is also known as an immunomodulatory hormone. Experimental studies have shown that 1,25-dihydroxyvitamin D, the active form of vitamin D, exerts immunologic activities on multiple components of the innate and adaptive immune system as well as endothelial membrane stability. Association between low levels of serum 25-hydroxyvitamin D and increased risk of developing several immune-related diseases and disorders, including psoriasis, type 1 diabetes, multiple sclerosis, rheumatoid arthritis, tuberculosis, sepsis, respiratory infection, and COVID-19, has been observed. Accordingly, a number of clinical trials aiming to determine the efficacy of administration of vitamin D and its metabolites for treatment of these diseases have been conducted with variable outcomes. Interestingly, recent evidence suggests that some individuals might benefit from vitamin D more or less than others as high inter-individual difference in broad gene expression in human peripheral blood mononuclear cells in response to vitamin D supplementation has been observed. Although it is still debatable what level of serum 25-hydroxyvitamin D is optimal, it is advisable to increase vitamin D intake and have sensible sunlight exposure to maintain serum 25-hydroxyvitamin D at least 30 ng/mL (75 nmol/L), and preferably at 40-60 ng/mL (100-150 nmol/L) to achieve the optimal overall health benefits of vitamin D.

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3- Vitamin D Status and SARS-CoV-2 Infection and COVID-19 Clinical Outcomes

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Abstract

Background: Several studies suggest an association between serum 25-hydroxyvitamin D (25OHD) and the outcomes of Severe Acute Respiratory Syndrome Corona-Virus-2 (SARS-CoV-2) infection, in particular Coronavirus Disease-2019 (COVID-19) related severity and mortality. The aim of the present meta-analysis was to investigate whether vitamin D status is associated with the COVID-19 severity, defined as ARDS requiring admission to intensive care unit (ICU) or mortality (primary endpoints) and with the susceptibility to SARS-CoV-2 and COVID-19-related hospitalization (secondary endpoints).Methods: A search in PubMed, ScienceDirect, Web of Science, Google Scholar, Scopus, and preprints repositories was performed until March 31th 2021 to identify all original observational studies reporting association measures, or enough data to calculate them, between Vitamin D status (insufficiency < 75, deficiency < 50, or severe deficiency < 25 nmol/L) and risk of SARS-CoV-2 infection, COVID-19 hospitalization, ICU admission, or death during COVID-19 hospitalization.Findings: Fifty-four studies (49 as fully-printed and 5 as pre-print publications) were included for a total of 1,403,715 individuals. The association between vitamin D status and SARS-CoV2 infection, COVID-19 related hospitalization, COVID-19 related ICU admission, and COVID-19 related mortality was reported in 17, 9, 27, and 35 studies, respectively. Severe deficiency, deficiency and insufficiency of vitamin D were all associated with ICU admission (odds ratio [OR], 95% confidence intervals [95%CIs]: 2.63, 1.45-4.77; 2.16, 1.43-3.26; 2.83, 1.74-4.61, respectively), mortality (OR, 95%CIs: 2.60, 1.93-3.49; 1.84, 1.26-2.69; 4.15, 1.76-9.77, respectively), SARS-CoV-2

infection (OR, 95%CIs: 1.68, 1.32-2.13; 1.83, 1.43-2.33; 1.49, 1.16-1.91, respectively) and COVID-19 hospitalization (OR, 95%CIs 2.51, 1.63-3.85; 2.38, 1.56-3.63; 1.82, 1.43-2.33). Considering specific subgroups (i.e., Caucasian patients, high quality studies, and studies reporting adjusted association estimates) the results of primary endpoints did not change.Interpretations: Patients with low vitamin D levels present an increased risk of ARDS requiring admission to intensive care unit (ICU) or mortality due to SARS-CoV-2 infection and a higher susceptibility to SARS-CoV-2 infection and related hospitalization.

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