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A brief review of vitamin D as a potential target for the regulation of blood glucose and inflammation in diabetes-associated periodontitis

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Abstract

Diabetes is a metabolic disorder associated with various complications, including periodontitis. The risk of periodontitis is increased in patients with diabetes, while vitamin D deficiency is associated with both diabetes and periodontitis. Thus, there is a need to identify the molecular effects of vitamin D on the regulation of inflammation and glucose in diabetes-associated periodontitis. The Web of Science, Scopus, and PubMed databases were searched for studies of the molecular effects of vitamin D. Molecular effects were reportedly mediated by salivary secretions, interactions of advanced glycation end products (AGEs) with receptors of AGEs (RAGEs), cytokines, and oxidative stress pathways linking diabetes with periodontitis. Vitamin D supplementation attenuates inflammation in diabetesassociated periodontitis by reducing the levels of inflammatory cytokines and numbers of immune cells; it also has antibacterial effects. Vitamin D reduces cytokine levels through regulation of the extracellular signal-related kinase 1/2 and Toll-like receptor 1/2 pathways, along with the suppression of interleukin expression. Glucose homeostasis is altered in diabetes either because of reduced insulin production or decreased insulin sensitivity. These vitamin D-related alterations of glucoregulatory factors may contribute to hyperglycaemia; hyperglycaemia may also lead to alterations of glucoregulatory factors. This review discusses the pathways involved in glucose regulation and effects of vitamin D supplementation on glucose regulation. Further studies are needed to characterise the effects of vitamin D on diabetes-associated periodontitis.

Keywords: Diabetes; Inflammation; Insulin; Periodontitis; Vitamin D.

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