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The physiology of vitamin K nutriture and vitamin K-dependent protein function in atherosclerosis.

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Abstract

Recent advances in the discovery of new functions for vitamin K-dependent (VKD) proteins and in defining vitamin K nutriture have led to a substantial revision in our understanding of vitamin K physiology. The only unequivocal function for vitamin K is as a cofactor for the carboxylation of VKD proteins which renders them active. While vitamin K was originally associated only with hepatic VKD proteins that participate in hemostasis, VKD proteins are now known to be present in virtually every tissue and to be important to bone mineralization, arterial calcification, apoptosis, phagocytosis, growth control, chemotaxis, and signal transduction. The development of improved methods for analyzing vitamin K has shed considerable insight into the relative importance of different vitamin K forms in the diet and their contribution to hepatic vs. non-hepatic tissue. New assays that measure the extent of carboxylation in VKD proteins have revealed that while the current recommended daily allowance for vitamin K is sufficient for maintaining functional hemostasis, the undercarboxylation of at least one non-hemostatic protein is frequently observed in the general population. The advances in defining VKD protein function and vitamin K nutriture are described, as is the potential impact of VKD proteins on atherosclerosis. Many of the VKD proteins contribute to atherogenesis. Recent studies suggest involvement in arterial calcification, which may be influenced by dietary levels of vitamin K and by anticoagulant drugs such as warfarin that antagonize vitamin K action.

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