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Intestinal Ca²⁺ absorption revisited: A molecular and clinical approach

Areco VA *et al.* Ca²⁺ absorption/molecular and clinical approach

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Abstract

Ca²⁺ has an important role in the maintenance of the skeleton and is involved in the main physiological processes. Its homeostasis is controlled by the intestine, kidney, bone and parathyroid glands. The intestinal Ca²⁺ absorption occurs mainly *via* the paracellular and the transcellular pathways. The proteins involved in both ways are regulated by calcitriol and other hormones as well as dietary factors. Fibroblast growth factor is a strong antagonist of vitamin D action. Part of the intestinal Ca²⁺ movement seems to be vitamin D independent. Intestinal Ca²⁺ absorption changes according to different physiological conditions. It is promoted under high Ca²⁺ demands such as growth, pregnancy, lactation, dietary Ca²⁺ deficiency and high physical activity. In contrast, the intestinal Ca²⁺ transport decreases with aging. Oxidative stress inhibits the intestinal Ca²⁺ absorption whereas the antioxidants counteract the effects of prooxidants leading to the

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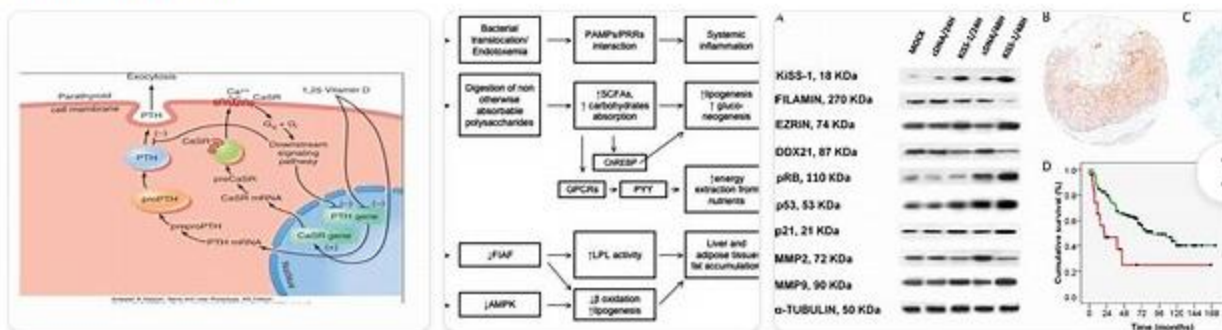
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THE absorption of calcium from the gastro-intestinal tract is one of the chief regulators of calcium metabolism and is known to be disturbed in a variety of clinical disorders.

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Plasma calcium (Ca²⁺) is maintained by amending the release of parathyroid hormone and through direct effects of the Ca²⁺-sensing receptor (CaSR) in the renal tubule. Combined, these mechanisms alter **intestinal Ca²⁺ absorption** by **modulating** 1,25-dihydroxyvitamin D₃ production, bone resorption, and **renal Ca²⁺ excretion**.

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We have previously demonstrated that melatonin (MEL) blocks the inhibition of the **intestinal Ca²⁺ + absorption** caused by menadione (MEN). The purpose of this study were to determine whether MEL not only restores but also prevents the **intestinal Ca²⁺ + absorption** inhibited either by MEN or BSO, two **drugs that** deplete glutathione (GSH) in different ways, and to analyze the mechanisms by which ...