Editorial

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Biochemistry & Molecular Biology Journal
ISSN 2471-8084

2021
Vol.7 No.7:31

Vitamin D and its Receptor Have Tissue-Specific Regulatory Effects on Calbindin-D28K and Calbindin-D9K

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Citation: Thirunahari A (2021) Vitamin D and its Receptor Have Tissue-Specific Regulatory Effects on Calbindin-D28K and Calbindin-D9K. Biochem Mol Biol Vol.7 No.7:31

Received: July 05, 2021; **Accepted:** July 10, 2021; **Published:** July 15, 2021

Editorial

In mammalian growth, a healthy balance of calcium and vitamin D is critical. Calbindin-D28K (CaBP-28K) and calbindin-D9K (CaBP-9K) are calcium-binding proteins in the cytosol that mediate the dynamic balance of vitamin D and calcium, particularly in intestinal calcium absorption, urine calcium excretion, and bone formation. However, the precise functions of CaBP-28K and CaBP-9K remain unknown. Under regular calcium food intake, CaBP-9K/CaBP-28K double-knockout (KO) animals have a normal phenotype.

These double-KO mice, on the other hand, had higher declines in serum calcium levels and bone length when fed a calcium-deficient diet than wild-type mice. We summarise and analyse the literature on the connection of vitamin D and its receptor with CaBP-28K and CaBP-9K in mammals in this review. The human body requires calcium to function properly. Cell membrane integrity, musculoskeletal excitability, blood coagulation, neurotransmitter and hormone production, and cardiac contraction all require calcium ion homeostasis.

Many calcium transport proteins, such as transient receptor potential vanilloid type 5 (TRPV5), transient receptor potential vanilloid type 6 (TRPV6), plasma membrane Ca^{2+} ATPase (PMCA), parvalbumins (PVs), calbindin-D9K (CaBP-9K), calbindin-D28K (CaBP-28K), calretinin (CR), and sodium-calcium exchanger 1 (NCX1), CaBP-9K and CaBP-28K are the two calcium-binding proteins that are vitamin D dependent. Vitamin D, CaBP-9K, and CaBP-28K all play essential roles in calcium homeostasis modulation, including intestinal calcium absorption, urine calcium excretion, and bone production. The vitamin D endocrine system and its modes of action have been the subject of numerous reviews.

Vitamin D is obtained by the human body either through photosynthesis in the skin or by dietary consumption. 1,25-dihydroxyvitamin D_3 ($1,25[OH]2D_3$), the active form of vitamin D hormone, interacts to and activates its receptor (VDR), a nuclear transcription factor Vitamin D deficiency or a VDR mutation can cause rickets and a variety of extra-skeletal biologic responses, including inhibition of colon, breast, and prostate cancer cell progression, cardiovascular effects, and protection against autoimmune diseases like inflammatory bowel disease and multiple sclerosis.

The dosage of vitamin D supplementation, on the other hand, is still up for debate, in part because the role of vitamin D signalling in calcium handling systems, particularly in bone, is not entirely understood. CaBP-9K and CaBP-28K are cytoplasmic proteins that bind Ca^{2+} and are controlled by 1,25(OH)2D]. CaBP-28K was first found in the chicken duodenal mucosa and rat intestinal mucosa as a 28-kDa protein; the protein isolated from the rat intestinal mucosa was later identified as the 9-kDa CaBP-9K. These proteins are from separate subfamilies and have little in common in terms of sequence, however they both use the EF-hand structural motif. Bone, kidney, brain, pancreas, gut, and teeth have all been discovered to have CaBP-28K. Vitamin D has a tissue-specific effect on CaBP-9K and CaBP-28K. CaBP-28K and CaBP-9K are both dependent on $1,25(OH)2D_3$ in the gut and kidney.

CaBP-28K has been found in the bone, kidney, brain, pancreas, gut, and teeth. Vitamin D affects CaBP-9K and CaBP-28K in tissue-specific ways. In the gut and kidney, both CaBP-28K and CaBP-9K are reliant on $1,25(OH)2D_3$. Herein, we review data on the correlation of $1,25(OH)_2D_3$ and VDR with CaBP-9K and CaBP-28K in the intestine, kidney, and bone to summarize our understanding of the processes of calcium absorption, excretion, and incorporation. Dietary calcium absorption is the principal source of calcium absorption in mammals.

Three routes are thought to transport calcium in the gut. The transcellular route is the primary mode of transport in the duodenum and upper jejunum. It requires calcium entry via an apical calcium channel (TRPV6 or TRPV5), calcium translocation across the inside of an enterocyte assisted by CaBP-9K, and calcium extrusion by an intestinal plasma membrane pump (PMCA1b or NCX1). Vesicular calcium transport is another shuttling process in which calcium is sequestered and transported predominantly by lysosomes. Paracellular transport is the third mechanism, which is a type of fast, energy-independent, concentration-dependent diffusion that occurs throughout the intestine. The regulation of calcium absorption in the transcellular and vesicular routes is dependent on $1,25(OH)2D_3$ and needs the presence of VDR. When the calcium content in the intestinal lumen exceeds around 2 to 6 mmol/L, the predominant method of absorption is paracellular transport.