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Review Article: Vitamin's D Physiologic Properties and Functions

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ABSTRACT

When 7-dehydrocholesterol is exposed to ultraviolet light, a prohormone called In the skin D3 is created. Being biologically inert, it must first be changed into 25-hydroxyvitamin D. D3 must first be converted to 1,25-dihydroxyvitamin D3 in the kidney formerly usage. The 1,25-dihydroxyvitamin D3 hormone, which has an array of functions, interacts with a nuclear receptor, includes calcium concentration, intestinal phosphate concentration, and bone calcium mobilization, and kidney calcium reabsorption. There are numerous additional noncalcemic uses for it in the body. In this review, physiological, endocrinologic, and molecular biologic properties of vitamin D are briefly discussed.

Keywords- Bone, calcium homeostasis, vitamin D endocrine system, and vitamin D metabolism.

I. INTRODUCTION

One of medicine's greatest accomplishments must be the encounter ending the usage of rickets of vitamin D as a serious health issue. (1,2) beginning in 1913 with the discovery of the first vitamin and ending in 1940 the identification of the last vitamin. Since Sir Edward Mellanby discovered that feeding dogs a Scottish diet improved their health, his work was very notableSir Edward Mellanby was not aware that he was also depriving those dogs of sunshine, even though he was aware that some foods, such oats, may cause rickets. (3). By heating it and bubbling oxygen through it, McCollum was able to reduce the vitamin A motion of cod liver oil, but the preparation's effectiveness in treating rickets was unaffected. McCollum was right when he said that vitamin D was a brand-new vitamin. (4). UV radiation, which can be created naturally or artificially, has been demonstrated to be effective in treating rachitic youngsters, according to studies by Huldshinsky (5) and Chick et al (6). When Steenbock and Black collaborated, they were able to finally solve the conundru.. (7) Both the animals and the food they consumed exhibited antirachitic action after being exposed to radiation. Goldblatt and Soames are both (8) found that radiotherapy may be used to cure rickets in rats. As a result, two significant advances were made. Steenbock and Black follow after that. (7) If nutrients might be treated to provide vitamin D, rickets as a serious medical issue might be eradicated. Second, Irradiating fat-soluble compounds taken from tissues could provide significant levels of vitamin D for further investigation. Askew and colleagues first identified the structure of vitamin D2 in 1931. Windaus and other. (9), for instance, developed artificial methods to ascertain the erection of vitamin D3. (10). Since its discovery, vitamin D—along with a number of other vitamins—has been classified as a vitamin.

II. METABOLISM AND VITAMIN D PROCESSING

Previtamin D is formed by a powerful photolytic progression from a cholesterol derivative, such as 7-dehydrocholesterol, and is progressively isomerized for skin-bound vitamin D3 (11). Ergosterol is irradiated to generate vitamin D2, which is made from the fungus ergot. and follows to some amount in plankton in natural surroundings (which contains as much as 2 percent ergosterol).

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Vitamin D is necessary for everyone and should be taken into consideration at all times. It is essential for the development of bone as well as other physiological systems to function properly. In addition to acting as an anticancer agent, its use may help prevent a number of degenerative disorders.

We now understand that vitamin D3 is physiologically inert since genetic abnormalities that cause rickets while receiving adequate vitamin D doses have been demonstrated. (12 ,13) and (14) By 1969, scientists had figured out how to produce, chemically define, and identify the circulating form of vitamin D (15, 16). on the other hand, needs to be changed before use because it is not metabolically active. In 1971, the structure 1,25-dihydroxyvitamin D3 was used to identify the last hormone active formed from vitamin D. [1,25(OH)2D3] (17) manufactured and validated in advance (18)

III. THE PHYSIOLOGICAL FUNCTIONS OF VITAMIN D

The turn by the vitamin D hormone in skeletal mineralization and the inhibition of hypocalcemic tetanus. (20). Insufficient calcium and phosphate in the plasma prevents mineralization, which sources rickets in kids and osteomalacia in grownups. (24). Three distinct mechanisms are used by calcium blood levels are raised by vitamin D hormone. For starters, Only this hormone is known to exist. to activate the proteins necessary for colonic absorption of calcium actively. Additionally, it facilitates the gut's ability to absorb active phosphate Second, normal blood calcium levels are maintained even when a pet is fed a no-calcium diet. Since the environment lacks calcium, an animal's capacity to mobilize calcium through enterocytes is essential. There are two ways to raise blood calcium concentrations when intestinal calcium absorption is absent. The nuclear factor-B ligand (RANKL) receptor activator is produced by osteoblasts as a result of the vitamin D hormone (25). RANKL then activates resting osteoclasts and increases osteoclastogenesis for bone degeneration (25). As a way to mobilize calcium from the bones when the diet is lacking in calcium, the vitamin D hormone is necessary. (27). kidney's distal tubule is accountable due to the third for reabsorbing the remaining filtered out data, 1% calcium shipment, besides the two hormones collaborate to increase recapture of this remaining 1% of the filtered calcium load. (28). The calcium pool is greatly augmented by the 7 g of calcium that individuals filter daily. Once more, we require hormones for vitamin D and parathyroid. Taking just one small dosage of the vitamin D hormone, in accordance with calcium physiologic processes, encourages use of enterocytes for the consumption of calcium and phosphate.

IV. VITAMIN D ACTIONS APART FROM CALCIUM

An outstanding one significant discoveries following the receptor's finding was that it was present not just in osteoblasts, distal renal tubule cells, and enterocytes, which are their target targets, but also in ovarian cells, skin keratinocytes, promyelocytes, lymphocytes, colon cells, pituitary gland cells, and cells of the parathyroid gland (20). VDRs are expressed within these cells don't however from skeletal, cardiac, or hepatic muscle, indicating that these tissues are where they are used (20).

It is essential that VDR is discovered in the parathyroid glands. We now understand that the parathyroid glands contain the VDR is a significant target for this therapy due to Renal osteodystrophy treatment using vitamin D hormone and its analogs (20). Preproparathyroid gene regulation and repression are two of the vitamin D hormone's most important roles. (20, 45). Additionally, the receptor regarding the vitamin D hormone somehow prevents the multiplication of parathyroid gland cells. As a result, in healthy individuals, the role that vitamin D hormone has a significant part in sustaining appropriate parathyroid status. those who have kidney failure experience the destruction of the vitamin D hormone manufacturing site resulting in a lack of vitamin D in the parathyroid gland, which then overproliferates It causes secondary hyperparathyroidism by secreting too much parathyroid hormone (31). The management of the parathyroid glands in dialysis patients by the use of the vitamin D hormone and its analogs is a significant therapy option.

Yet another crucial region of research having been immunological system. While vitamin D excess inhibits several immunological processes, vitamin D Deficiency has an impact on immunity, particularly T cell-mediated immunity. (32). Because of this, scientists are investigating utilizing vitamin D molecules to treat autoimmune illnesses. Utilizing an animal model of experimental autoimmune encephalomyelitis to study multiple sclerosis, which is the first autoimmune disease to be researched. This condition can be treated or cured in any developmental stage with enough quantities of vitamin D hormone given orally every day. (33). An further crucial component of study having been immunological system. the defense mechanism, particularly T cell-mediated immunity, is obviously impacted by vitamin D shortage, while too much vitamin D suppresses some immunological processes (32). In order to cure autoimmune illnesses, scientists are now investigating the use of vitamin D molecules (30), stops the cell death in the islet organ. Similar findings were acquired utilizing simulations for systemic lupus (30), gut inflammation illness (31,25) together with rheumatoid arthritis (32,33) The interaction between the hormone that produces vitamin D and T helper lymphocytes reduces the T helper type 1 cells'

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inflammatory reactions, which suppresses a number of autoimmune diseases. Alternative ideas include inhibiting Dendritic cells are the cells convey antigens to T lymphocyte. ()29

V. REPORTING ON THE STATUS OF VITAMIN D

One of vitamin D's crucial functions properties is the variety of physiologic processes it performs. The necessity of having vitamin D on hand at all times is obvious. A lack of vitamin D impacts several biological processes, including bone mineralization. Rickets is growing more prevalent worldwide, even in the most industrialized nations. Almost no foods in Europe are with added vitamin D, and people's skin in the northern and southern hemisphere produce very little vitamin D throughout the season of to prevent bone illnesses, as well as other types of illness that is autoimmune and degenerative, vitamin D concentrations must be appropriate. The recommended dietary need for vitamin D, in the opinion of many experts in the area, is too low. It is completely safe to take vitamin D3 supplements an amount of 2000 IU per day. Safety evaluations require knowledge of vitamin D levels. In general, 25(OH)D3 levels are regarded as to be most effective predictor of level of vitamin D. Unfortunately, results from 25(OH)D3 tests that were accessible commercially were very inconsistent. (31). Everyone is aware that a noteworthy portion of vitamin D administered to a patient is kept in fat tissues. Following saturation of such websites, vitamin D stays in the bloodstream and is changed into 25(OH)D3, poisonous equivalent of 1,25(OH)2D3 (33). Vitamin D3 should be assessed to make sure that it is not being stored to the point that vitamin D intoxication develops when estimating the dietary amounts of vitamin D3 necessary to get back to regular plasma meditations of 25(OH)D3. (32)

VI. CONCLUSIONS

A series of compounds that are derived from vitamin D can be used to treat a number of illnesses. In a two-step process, vitamin D3 is transformed to 1,25(OH)2D3. Target genes are activated or inhibited by this hormone's interaction using just one nuclear type 2 receptor. The traditional and novel vitamin D's functions are performed by the proteins that are generated in response to the hormone. There are signs that vitamin D may affects the growth and generation of parathyroid hormones, as well as that the skeleton has been mineralized and the improvement phosphorus and calcium levels in serum levels. It functions in pancreatic islet cells, significantly affects the immune system and can be beneficial to guard against autoimmune illnesses and cancer.

REFERENCES

[1] Steenbock H. The induction of growth promoting and calcifying properties in a ration by exposure to light. Science 1924;60:224 –5.

[2] McCollum EV, DavisM. The necessity of certain lipins in the diet during growth. J Biol Chem 1913;25:167–231.

[3] Mellanby E. An experimental investigation on rickets. Lancet 1919;1: 407–12.

[4] McCollum EV, Simmonds N, Becker JE, Shipley PG. An experimental demonstration of the existence of a vitamin which promotes calcium deposition. J Biol Chem 1922;53:293– 8.

[5] Huldshinsky K. Heilung von Rachitis durch kunstalich Hohen-sonne. (The healing of rickets with artificial high altitude sun.) Dtsch Med Wochenschr 1919;45:712–3 (in German).

[6] Chick H, Dolyell EJ, Hume EM. Studies of rickets in Vienna 1919 – 1922. Med Res Counc (GB) Spec Rep Ser 1923;77.

[7] Steenbock H, Black A. Fat-soluble vitamins. XVII. The induction of growth-promoting and calcifying properties in a ration by exposure to ultraviolet light. J Biol Chem 1924;61:405–22.

[8] Goldblatt H, Soames KM. Studies on the fat-soluble growth-promoting factor. Biochem J 1923;17:446 –53.

[9] Askew FA, Bourdillon RB, Bruce HM, Jenkins RGC, Webster TA. The distillation of vitamin D. Proc R Soc Lond 1931;8107:76 –90.

[10] Windaus A, Schenck F, von Werder F. Uber das antirachitisch wirksame Bestrahlungs-produkt aus 7-Dehydrocholesterin. (Concerning the antirachitic activity of the irradiation product of 7-dehydrocholesterol.) Hoppe-Seyler's Z Physiol Chem 1936;241:100 –3 (in German).

[11] Velluz L, Amiard G. Chimie organique-equilibre de réaction entre précalciférol et calciférol. (The organic chemical equilibrium of the reaction between precalciferol and calciferol.) C R Assoc Anat 1949;228:853–5 (in French).

[12] Prader A, Illig R, Heierli E. Eine besondere Form der prima¨ren Vitamin D-resistenten Rachitis mit Hypocalca¨mie und autosomaldominantem Erbgang: Die heredita¨re Pseudo-mangelrachitis. (A special form of primary vitamin D-resistant rickets with hypocalcemia and autosomal receiver inheritance: The hereditary pseudodeficiency rickets.) Helv Paediatr Acta 1961;16:452– 68 (in German).

[13] Lund J, DeLuca HF. Biologically active metabolite of vitamin D3 from bone, liver, and blood serum. J Lipid Res 1966;7:739 – 44.

[14] Morri H, Lund J, Neville PF, DeLuca HF. Biological activity of a vitamin D metabolite. Arch Biochem Biophys 1967;120:508 –12.

ISSN: 2583-4053

Volume-2 Issue-4 || August 2023|| PP. 151-154

https://doi.org/10.55544/jrasb.2.4.21

[15] Blunt JW, DeLuca HF, Schnoes HK. 25-Hydroxycholecalciferol: a biologically active metabolite of vitamin D3. Biochemistry 1968;7:3317–22.

[16] Blunt JW, DeLuca HF. The synthesis of 25-hydroxycholecalciferol: a biologically active metabolite of vitamin D3. Biochemistry 1969;8: 671–5.

[17] Holick MF, Schnoes HK, DeLuca HF, Suda T, Cousins RJ. Isolation and identification of 1,25-dihydroxycholecalciferol: a metabolite of vitamin D active in intestine. Biochemistry 1971;10:2799 – 804.

[18] Semmler EJ, Holick MF, Schnoes HK, DeLuca HF. The synthesis of 1,25-dihydroxycholecalciferol: a metabolically active form of vitamin D3. Tetrahedron Lett 1972;40:4147–50.

[19] DeLuca HF, Schnoes HK. Vitamin D: recent advances. Annu Rev Biochem 1983;52:411–39.

[20] Jones G, Strugnell SA, DeLuca HF. Current understanding of the molecular actions of vitamin D. Physiol Rev 1998;78:1193–231.

[21] RosenH,ReshefA,MaedaN,etal.Markedly reduced bileacid synthesis but maintained levels of cholesterol and vitamin D metabolites in mice with disrupted sterol 25-hydroxylase gene. J Biol Chem 1998;273:14805–12.

[22] Cheng JB, Motola DL, Mangelsdorf DJ, Russell DW. De-orphanization of cytochrome P450 2R1: a microsomal vitamin D 25-hydroxylase. J Biol Chem 2003;278:38084 –93.

[23] Ohyama Y, Okuda K. Isolation and characterization of a cytochrome P-450 from rat kidney mitochondria that catalyzes the 24-hydroxylation of 25-hydroxyvitamin D3. J Biol Chem 1991;266:8690 –5.

[24] Underwood JL, DeLuca HF. Vitamin D is not directly necessary for bone growth and mineralization. Am J Physiol 1984;246:E493– 8.

[25] Suda T, Ueno Y, Fujii K, Shinki T. Vitamin D and bone. J Cell Biochem 2002;88:259 – 66. 26. Garabedian M, Holick MF, DeLuca HF, Boyle IT. Control of 25-hydroxycholecalciferol metabolism by the parathyroid glands. Proc Natl Acad Sci USA 1972;69:1673– 6.

[26] Garabedian M, Tanaka Y, Holick MF, DeLuca HF. Response of intestinal calcium transport and bone calcium mobilization to 1,25- dihydroxyvitamin D3 in thyroparathyroidectomized rats. Endocrinology 1974;94:1022–7.

[27] Yamamoto M, Kawanobe Y, Takahashi H, Shimazawa E, Kimura S, Ogata E. Vitamin D deficiency and renal calcium transport in the rat. J Clin Invest 1984;74:507–13.

[28] Brown EM, Gamba G, Riccardi R, et al. Cloning and characterization of an extracellular Ca2-sensing receptor from bovine parathyroid. Nature 1993;366:575–80.

[29] Tanaka Y, DeLuca HF. Rat renal 25-hydroxyvitamin D3-1- and 24-hydroxylases: their in vivo regulation. Am J Physiol 1984;246:E168 –73.

[30] Brenza HL, DeLuca HF. Regulation of 25-hydroxyvitamin D3 1- hydroxylase gene expression by parathyroid hormone and 1,25- dihydroxyvitamin D3. Arch Biochem Biophys 2000;381:143–52.

[31] Chambers TJ, Magnus CJ. Calcitonin alters behaviour of isolated osteoclasts. J Pathol 1982;136:27–39.

[32] Shinki T, Ueno Y, DeLuca HF, Suda T. Calcitonin is a major regulator for the expression of renal 25-hydroxyvitamin D3-1-hydroxylase gene in normocalcemic rats. Proc Natl Acad Sci USA 1999;96:8253– 8.

[33] Sutton AL, MacDonald PN. Vitamin D: more than a “bone-a-fide” hormone. Mol Endocrinol 2003;17:777–9