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Vitamin D and Physical Performance: What Is the Ergogenic Actions of Vitamin D?

Rodrigo Nolasco and Marise Lazaretti-Castro

Abstract

Vitamin D produced in the skin by the action of the sun's rays turns into calcitriol, a powerful hormone, recognized as important for health. Although its most known effects are on mineral homeostasis and bone metabolism, its receptors (VDRs) have been identified in almost all tissues, suggesting that it should have other actions. Vitamin D acts directly on the skeletal muscle system maintaining muscle mass, strength levels and speed of muscle contraction. Thereby, allied to that, vitamin D is among the potential factors that are related to maintaining bone, and cannot be dissociated from the prevention of osteoporosis and sarcopenia. However, in the physical performance aspect, there are still uncertainty in the literature about the use of vitamin D as an ergogenic resource aimed at improving the physical performance of amateur and professional athletes. Therefore, due to the biological actions of vitamin D and high prevalence of low levels in sedentary and physically active individuals, this chapter will discuss the facts pointed out in the literature about the action of vitamin D as an ergogenic resource aiming at the preservation or improvement of the physical, including strength muscular, aerobic capacity and balance.

Keywords: vitamin D, athletic performance, lung function, sarcopenia

1. Introduction

The increase in human life expectancy during the previous century has raised new health issues, especially the control of aging-related deterioration. Important efforts have been made to gain new insights that may lead to modalities to delay the functional impairment and progression of chronic degenerative diseases, as well as sarcopenia and osteoporosis. Undoubtedly, the physical exercises are directly inter-related with the improvement or even rehabilitation of the physical performance besides increasing the life expectancy [1]. However, the effect of hormonal action, especially vitamin D, has lately been part of this prospect.

In this context, vitamin D has attracted considerable interest among health researchers, professional organizations and the lay public in recent times. Although it is called vitamin, conceptually vitamin D is a hormone. This is due to its hormonal nature, such as the ability to be integrally produced by the organism and to have specific receptors in several tissues [2].

Vitamin D has emerged for more than 500 million years. Even though its function in plants and invertebrates is unknown, the close association between vitamin D and

sunlight has become essential in the evolution of terrestrial vertebrates. The main physiological function of vitamin D is to maintain the supply of calcium and phosphorus for complete mineralization of bone tissue [2].

Sunlight is the main source for producing the right amount of vitamin D for most humans. In food, it is found in small amounts, and there are few food sources. When it comes from sunlight, it is estimated that around 80–100% of the human needs for vitamin D come from exposure to sunlight [2, 3].

Although it is recognized as important for health, it is estimated that there are approximately 1 billion people in the world with inadequate concentrations of vitamin D. Furthermore, individuals with insufficient vitamin D levels have an increased risk of bone disease, such as: rickets, low bone mass and fractures due to increased bone resorption as a consequence of an overproduction of parathyroid hormone (PTH) [4].

Regarding functional capacity, the inadequacy of vitamin D stocks has catabolic effects on the musculoskeletal system, causing muscle weakness, lags in balance and impairs the formation of cross-bridges, which could impair physical performance. However, there remains divergences. Reports from recognized institutes do not corroborate the adequate levels for non-skeletal outcomes, or even the existence of evidence of a non-linear association for some results on physical performance [5].

Finally, recent studies have suggested a possible action of vitamin D in the lung function of individuals without lung disease. Similarly, results were observed on aerobic capacity being influenced by a possible action of vitamin D [6].

2. Vitamin D actions

Vitamin D is a steroid hormone. Its precursor found in animal tissues is 7-dehydrocholesterol, which is synthesized in the skin and is also the immediate precursor of cholesterol. Cholecalciferol has as its main source cutaneous synthesis catalyzed by ultraviolet light B (UVB). Its synthesis is initiated in the skin, under the action of UVB rays, which transform its precursor, 7-dehydrocholesterol, into cholecalciferol or vitamin D₃. In smaller amounts, vitamin D can also be obtained through the diet of fortified foods such as dairy products and cereals, fatty fish and cod liver oil. After food intake or synthesis in the skin, vitamin D is transported into the liver where it is converted into 25-hydroxyvitamin D [25(OH) D] or calcidiol, which is the main form of circulating vitamin D and also used for serum dosage. In the kidney, 25(OH) D is converted to its biologically active form, 1,25 dihydroxyvitamin D (1,25 (OH) ₂ D) [7, 8]. The biological actions of 1,25 (OH) ₂ D are mediated by the nuclear transcription factor, called the vitamin D receptor (VDR) located in the cell nucleus [9].

VDR is part of the nuclear receptors of transcription factors regulating steroid hormones, retinoic acid, thyroid hormone, and vitamin D. Following the connection with VDR, there is the formation of the heterodimeric complex (Vitamin D Receptor—Retinoic Acid Receptor). This, in turn, binds to specific DNA sequences, also called the vitamin D responsive element (VDRE), promoting conformational changes that lead to the recruitment of several other transcriptional coactivators, resulting in the transcription of target genes [10].

After conversion to its biologically active form, 1,25-dihydroxyvitamin D regulates the expression of more than 900 gene variants [11]. Considering this aspect, it has been observed that these gene expressions have a significant impact on a range of variables related to health and performance, such as exercise-induced inflammation, pulmonary function, tumor suppressor genes, neurological function, cardiovascular health, glucose metabolism, health bone and skeletal muscle performance [12].

Vitamin D plays a key role in maintaining basal serum calcium and phosphorus levels for a variety of metabolic functions, regulation of transcription and bone metabolism. 1,25 (OH) 2D interacts with VDR in the small intestine to increase intestinal calcium absorption from 10–40% and phosphorus from approximately 60–80% [13]. Its action also extends to the cells responsible for bone remodeling, from the connection to the pre-osteoblasts, acting as a stimulus for precursor cells in osteoclasts from the RANK/RANK ligand system. Active osteoclasts remove calcium and phosphorus from bone to maintain serum levels of these elements. In the kidneys, 1,25 (OH) 2D stimulates calcium reabsorption of glomerular filtrate [3, 13]. Vitamin D and calcium are among the potential factors related to maintaining bone and muscle health, and cannot be separated from osteoporosis prevention in postmenopausal women [14].

2.1 Vitamin D and muscle

Moreover to the effects on bone metabolism, studies over 80 suggest improvements in physical performance in individuals exposed to UV radiation. Although these studies do not directly describe the action of vitamin D, induced changes in vitamin D levels may have played a role in muscle function [15].

Especially in older women with low vitamin D status, several intervention studies have reported that vitamin D supplementation increases appendicular muscle strength and improves physical function. In the musculoskeletal system, vitamin D exerts specific receptor-mediated functions (VDR) in processes ranging from protein synthesis to kinetics of muscle contraction, directly affecting the functional capacity of postmenopausal women [16]. Apart from this, the important mechanisms by which vitamin D can exert on human skeletal muscle can be classified as genomic or non-genomic [15].

Considering the genomic theory, it describes that 1,25 (OH) 2 D exerts a direct effect on the human muscular VDR, sparking progressive epigenetic changes that may have an impact on the morpho functionality of skeletal muscle. Within this context, in a study of women with limited mobility and with a relatively low level of vitamin D, vitamin D3 supplementation resulted in a 30% increase in intramuscular VDR protein concentration and a 10% increase in total muscle fiber size I and II. These findings corroborate the hypothesis that vitamin D contributes to the muscle mass of individuals with a tendency towards functional disability [17].

Muscle tissues have specific nuclear receptors for 1,25-[OH] 2D. In patients with strokes with atrophied type II fibers, improvement after vitamin D supplementation was observed for 25-OHD serum deficient patients before therapy. Also, improvements in muscle strength on the intact side of these patients with vitamin D-supplemented strokes were observed. In addition, in cross-sectional analysis, there was a correlation between 25-OHD and fiber diameter and type II [18].

Conversely, the non-genomic hypothesis credits a rapid and indirect mechanism by which 1,25 (OH) 2 D activates a series of secondary messenger processes that promote increased calcium kinetics. Researchers have extensively searched the so-called 1,25 (OH) 2 D non-genomic activities through the investigation of membrane proteins and intracellular signaling [19]. They have demonstrated that 1,25 (OH) 2 D can be mediated by a different membrane-associated rapid response steroid binding protein (MARRS) to facilitate rapid responses. It has been found that this protein is similar to the multifunctional isomerase disulfide protein of family A, member 3 (PDIA3), an endoplasmic reticulum enzyme. Interestingly, the antibody that blocks this protein prevented the transport of calcium and phosphate through the membranes of the intestinal epithelial cells. Therefore, skeletal muscle functionality can be influenced by intracellular effects on calcium handling through the action of vitamin D [20].

In addition, the presence of the VDR in vascular tissue and cardiac muscle seems to support the hypothesis that the vitamin D may impact the cardiovascular system's ability to transport oxygenated blood and the ability of skeletal muscles to use oxygen [21]. In a randomized double-blind placebo-controlled study with postmenopausal women, was observed a 23.5% increase in muscle strength and a reduction in the number of falls by 76% after 9 months of vitamin D supplementation [22]. In another study with older adults, significant improvements in physical performance, specifically in the up-and-go test, were reported with 2000 IU of vitamin D per day in 300 elderly women with a baseline level of 25 OHD below 24 ng/mL (60 nmol/L), [23].

In relation to the adequacy of vitamin D levels for individuals with low bone mass, this population seems to benefit in the physical performance, by serum concentrations of 25 (OH) D from 30 ng/mL (75 nmol/L) concentrations close to 36 ng/mL (90 nmol/L) as the most advantageous. Likewise, these 25 (OH) D values seem to benefit lower limb muscle strength, which was assessed by the walking test. Individuals with 25 (OH) D concentrations between 36 and 40 ng/mL (90 and 100 nmol/L) appear to perform the test faster [24].

Considering the adequacy of vitamin D levels, researchers in prospective, double-blind, placebo-controlled, randomized trial included Brazilian people institutionalized that received a 6-month supplementation of vitamin D, had as result, the increase in 16.4% in their maximum isometric strength of hip flexors and 24.6% in knee extensors measured by a portable mechanical dynamometer at 6 months, nevertheless the calcium/placebo group showed no improvement at all [25]. The same way, in other study, researchers have found an increase in neuromuscular parameters such as the balance (4.5%), functional mobility (10.1%) and muscle strength (5.7%) after elderly supplementation with vitamin D (6 month), without the regular practice of physical activity, considering that after the study most subjects reached the sufficiency level [26].

Vitamin D supplementation in youngsters has also been shown to be effective on the muscular strength of dancers who received oral supplementation of 2000 IU/day of vitamin D3 for 4 months during the winter. At the end of the study, the supplemented group presented increased isometric strength (18.7%, $p < 0.01$), plyometry (7.1%, $p < 0.01$), and reduction of lesions when compared to control ($p < 0.01$) [27]. On the other hand, in a study with 179 vitamin D-deficient Lebanese adolescents, vitamin D supplementation did not show improvement in manual grip strength [28].

Even with some intriguing results, studies are still conflicting about the action of vitamin D on muscle performance. Some meta-analyses have found antagonistic outcomes. The meta-analysis who analyzed 17 randomized controlled trials in individuals of all ages including younger subjects just showed benefit in muscle strength in subjects with vitamin D serum levels below 25 nmol/L at baseline. In another hand, the meta-analysis which reviewed results from 13 randomized controlled trials in individuals older than 60 years old, observed a small benefit of daily vitamin D supplementation (800 IU–1000 IU per day) for balance and muscle strength [29].

2.2 Vitamin D actions on the respiratory system

The interrelationships between vitamin D metabolism and respiratory function have been studied in the literature, mainly in diseases of the respiratory tract, however the results are still not conclusive. Accordingly, the mechanisms by which life D could affect lung function have not yet been fully elucidated. However, some explanations have been pointed out. It is believed that vitamin D influences approximately 3% of the human genome and directly or indirectly, vitamin D controls

many genes that are involved in the regulation of cell proliferation, differentiation and apoptosis of healthy and pathological cells [30].

1,25 (OH) 2D is mostly derived from the kidneys, however, other tissues, including the breast and prostate, may hydroxylate vitamin D in its active form. Activated, this metabolite is transported throughout the circulation by additional vitamin D binding proteins and lipoproteins; a function that allows vitamin D to actuate a wide range of skeletal and extra-skeletal functions [31].

Studies on the action of vitamin D on different cell types suggest that 1,25 (OH) 2D modulates smooth muscle excitation-contraction via intracellular Ca^{2+} release and Ca^{2+} sensitization. Airway resistance is dictated largely by the diameter of these pathways, and minor changes in this structure can significantly increase airway resistance. The absence of Vitamin D could affect the diameter of the airways, impairing pulmonary function [31, 32].

Most nucleated cells express the VDR, however the expression varies according to cell specificity. In the lungs, VDR was found in smooth muscle cells of the airway and in alveolar cells, also known as pneumocytes [32–34]. The enzyme 1α -hydroxylase, responsible for the conversion of 25 (OH) D into its active form, is also expressed by tracheal and bronchial cells [35].

Nguyen and cols, demonstrated through a series of studies with rat fetuses that type II alveolar cells underwent 1,25 (OH) 2D 3 action and suggested that vitamin D is important for maturation and the production of surfactants [36, 37]. Although in humans the mechanisms are more complex than in rats, the effect of vitamin D on the production of surfactants has been confirmed [33].

In relation to the growth and pulmonary maturation, studies with rats born to mothers with dietary vitamin D showed a loss of lung compliance compared to those born to mothers whose vitamin D supplementation [38]. In a similar study, vitamin D-deficient mice decreased lung volumes compared to the offspring of vitamin D-rich mice [39].

In human studies, the authors used the presence of calbindin, a vitamin D-dependent calcium binding protein, as a molecular marker of the action of 1,25 (OH) 2D 3 on tissues. The authors found high levels of calbindin in fetal lung tissue between the 14th and 32nd weeks of gestation, suggesting an action of vitamin D on lung development [40].

In a cross-sectional study from the Third National Health, Nutrition and Examination Survey (NHANESIII) with a sample of 14,000 Americans, researchers found a positive correlation between FEV 1 and FVC with serum 25 (OH) D levels. The authors also observed that adults with low serum 25 (OH) D levels had lower than predicted FEV 1 of pulmonary function [41].

Although vitamin D intervention studies in postmenopausal women aiming for respiratory capacity are still scarce, in our study center, we developed a research project to evaluate the effects of a differential aquatic exercise program (HYDROS) on the musculoskeletal system in postmenopausal women compared to sedentary controls [26]. The large volume of parameters obtained during the six-month study was presented as an opportunity to evaluate, in a post-hoc analysis, the effects of vitamin D supplementation, combined or not with high intensity aquatic exercises, on the pulmonary function of postmenopausal women. We observed an improvement in the spirometric parameters of women submitted to vitamin D supplementation, even without regular physical exercise. The supplemented group obtained a 7% improvement in peak expiratory flow, similarly forced vital capacity, according to the data presented in **Figure 1** [42].

In a prospective, double-blind, randomized study, vitamin D supplementation in asthmatic children reduced the risk of exacerbation triggered by acute respiratory tract infection and significantly improved FEV1, [43]. Another prospective study in

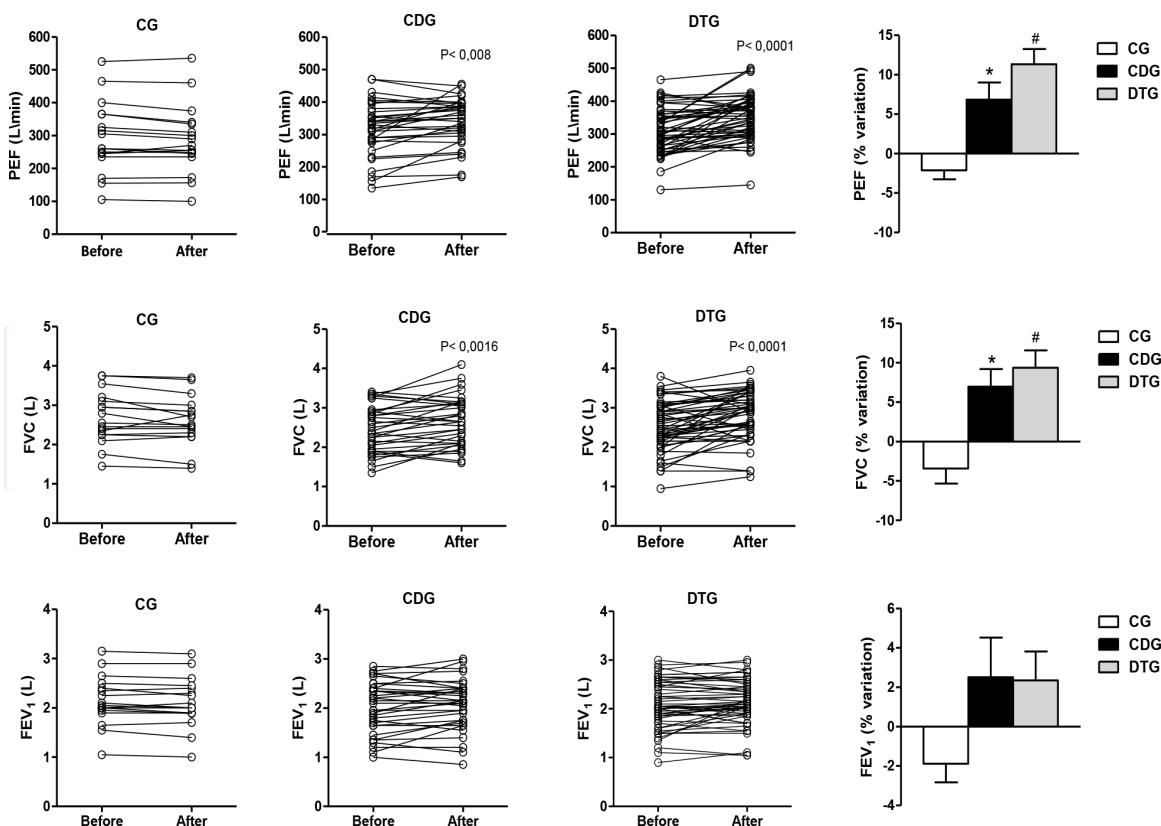


Figure 1.

Follow-up of the spirometric parameters throughout the study of the three groups. Mean \pm SD and percentage of change for peak expiratory flow (PEF), forced vital capacity (FVC) and forced expiratory volume in 1 s (FEV₁) in the control (CG), control with supplementation of vitamin D and calcium (CDG), and training groups (DTG) before and after the intervention. * $p < 0.0001$, # $p < 0.01$ and $p < 0.05$ indicate differences detected using Student's *t*-test versus before intervention. Different letters indicate statistically significant differences ($p < 0.05$) on one-way analysis of variance (Tukey's post hoc test) in relation to the percentage variation between the groups. Ref. [42]: <http://dx.doi.org/10.1590/2359-3997000000211>.

patients with chronic obstructive pulmonary disease (COPD) and vitamin D deficiency demonstrated increased respiratory muscle strength, improved dyspnea scale, and superior physical performance after 1 year of vitamin D supplementation [44].

In other components of respiratory system, vitamin D status has been associated with cardiorespiratory fitness in cross-sectional investigations in the general population. Regarding the maximum volume of oxygen (VO₂max), in an observational study, no correlation was observed between 25 (OH) D levels and VO₂max in 53 junior and collegiate ice hockey players. However, analysis of data soccer players exposure at ultraviolet radiation, revealed a linear association between vitamin D and VO₂max in both experimental sessions [45]. Corroborating with these results, across from vitamin D supplementation of soccer players during an eight-week high-intensity training program, significant results were observed in aerobic capacity. As upshot, a significant improvement in VO₂max in the supplemented group was observed compared to non-supplemented subjects [46].

A recent study found that more than 60% of athletes had vitamin D insufficiency even when the data collected near hot and sunny summer. Studies with high intensity athletes have been suggesting that athletes with vitamin D3 deficiency should be supplemented with this vitamin to improve physical performance, especially VO₂max [47]. The explanation would be to athletes to achieve the best results in the summer, while exposure to solar radiation. The authors suggest that the replenished 25 (OH) D3 level may protect athletes against acute and chronic diseases. Considering the above, vitamin D3 supplementation along with the training load may induce adaptive changes of aerobic and anaerobic metabolism in athletes of different sports [47].

In rowers, significantly higher energy and oxygen consumption scores were observed during a continuous exercise test in the vitamin D3 supplemented group over the 8-week training period. They demonstrated a significantly increase in VO₂max (12.1 and 10.3%, respectively) [21]. At the same time, the blood parameters of the supplemented athletes, such as IL-1b, CRP, LDH, reached lower values. These results suggest that vitamin D3, whose blood concentration increased by 400% in the supplemented group after supplementation, could be the justification for improving aerobic metabolism in rowers and reducing their inflammatory reactions in response to high intensity training [21].

The possible assumption by which vitamin D to affect VO₂max would be caused the influence of the enzymes Cytochrome P450 (CYP) [48]. These enzymes activate vitamin D3, which has no hormonal action, converting it to an active hormonal form (1 α , 25 (OH) 2D3) by the action of CYP enzymes. Reactions catalyzed by the CYP enzymes (mitochondrial CYP27A1, microsomal CYP2R1 in liver and the latter reaction by mitochondrial CYP27B1 in kidney) have proteins containing heme and could potentially affect the binding affinity of oxygen to hemoglobin [29, 49]. The existence of this compound is important for the transport of oxygen, since it is present mainly in hemoglobin, myoglobin and enzymes. Beside this, vitamin D could also influence VO₂max through iron metabolism and erythropoietin. Complementing, the vitamin D deficiency results in dysregulation of innate immunity and inflammation which is affecting iron metabolism and contributes to erythropoietin resistance, and this well documented that is linearly associated with changes in red blood cells levels [47].

2.3 Vitamin D levels in athletes and physical performance

Even after 100 years its discovery, when early researchers suggested sunbathing to prevent and cure rickets, vitamin D remains in the spotlight. The actions of the vitamin D extend beyond bone health, becoming recognized at appropriate levels, beneficial to various non-skeletal health outcomes [49, 50]. Vitamin D is a multiactive hormone acting in different spheres of the body. Research over time has shown that vitamin D3 biological action is much broader than researchers originally thought, as shown by the tissue distribution of the VDR, from mediating only calcium homeostasis. Along with this, even after this recognition, it is visible from epidemiological data; that vitamin D deficiency excessively prevalent globally [50, 51].

Based on population data, the values of 25 (OH) D discussed in the literature with emphasis on bone outcomes range from 20 to 32 ng/mL (50–80 nmol/L). Several authors have confirmed that the best cut point of is 30 ng/mL (75 nmol/L) for the correction of secondary hyperparathyroidism, reduced risk of falls and fractures, and maximum absorption of calcium. Thus, serum concentrations below 20 ng/mL (50 nmol/L) are classified as deficiency, between 20 and 29 ng/mL (50 and 74 nmol/L) as insufficiency and between 30 and 100 ng/mL (75 and 250 ng/mL) as sufficiency [14, 52].

Surprisingly, it is estimated that around the world, 1 billion people fall into these categories. In addition, both vitamin D insufficiency and deficiency are increasing in prevalence [53]. Among athletes of different categories, deficiencies or deficiencies were observed in most dancers, taekwondo fighters, jockeys, elite wheelchair athletes, handball players, athletics athletes, weightlifters, swimmers and volleyball players. In relation to other professional sports, the athletes are affected in the same way. National Football League players, 26% were found to have deficient levels of vitamin D, and 42–80% of athletes had levels defined as insufficient. Similarly, professional basketball players, 32% of athletes are vitamin D deficient and 47% are insufficient [53].

The detection of vitamin D levels in athletes, in addition to the main function of vitamin D, acting in the interrelationship between the bone and muscular systems, also had as objective the sporting performance. Athletes need to potentiate the training stimulus, so it is a fundamental principle of the training program. The great scope of high performance training is to provide a stimulus to bring an adaptive response to the entire structure of the body that improves the performance of the competition. Thus, considering these aspects, the ergogenic resources to complement the adaptive response to a physical/metabolic challenge are intensively researched [53].

In the last decade, in vitro and animal studies have provided information on a beneficial role derived from vitamin D in skeletal muscle repair and remodeling. Although in humans the action of vitamin D within muscle tissue, still raise questions, in rodent were observed possible effects. The cytochrome precursor (CYP27B1) responsible for rendering vitamin D of 25 (OH) D3 inactive, in metabolically active (1,25 (OH) 2 D3), was found in cells at different stages of differentiation; expression in rodent muscle fibers [29, 54]. This finding suggests the action of vitamin D on the regulation of muscle tissue. In addition, recent studies suggest that VDR is expressed in myoblasts and C2C12 myotubes in murine skeletal muscle [54].

In human skeletal, in situ detection of VDR points towards a role of vitamin-D on muscle function. In addition, VDR has been localized to skeletal muscle cells that promote *de novo* protein synthesis. Considering the genomic effects, the activation of VDR induces heterodimerization between the active VDR and the retinoic receptor (RXR). In this way, this induces the activation of the vitamin D response element (VDRE), a complex of genes coding for the “genomic effects” of vitamin D [29, 54].

Among the genomic repercussions of VDR (**Figure 2**) [29], the increase in calcium handling by enhancing the activities of the calcium binding protein (calbindin-D9K) in cell sarcoplasm, muscle cell differentiation and proliferation through effects on insulin growth factor (IGF) expression which in turn induces skeletal muscle hypertrophy [29].

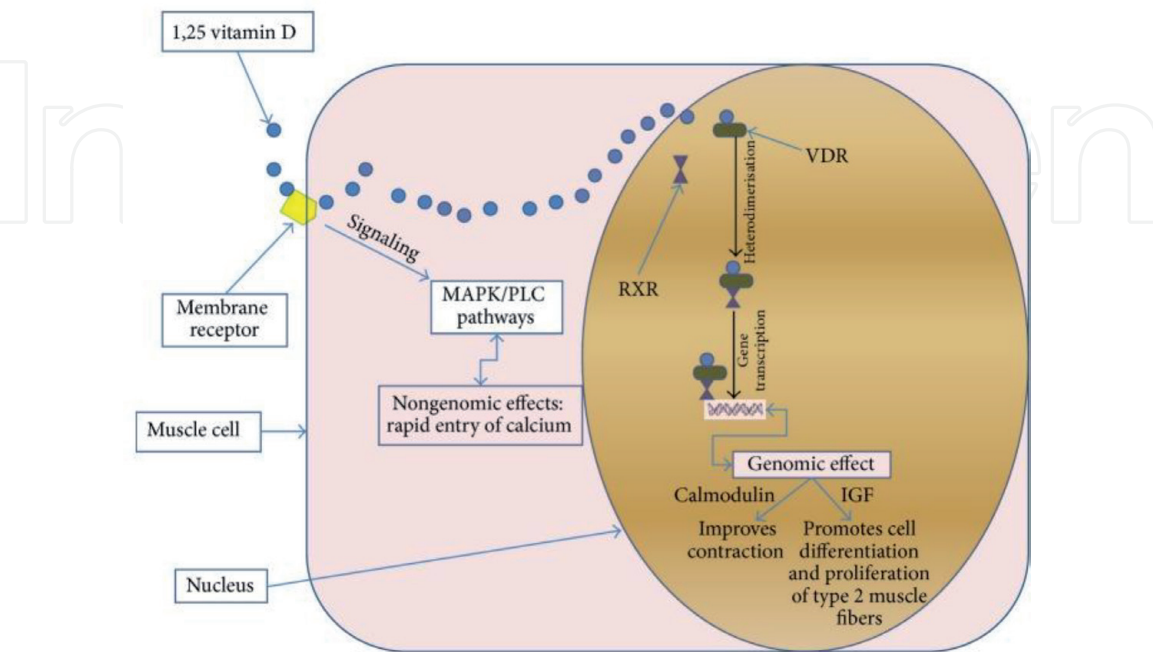


Figure 2. Repercussions of the 1.25 vitamin D on skeletal muscle system: Molecular and nuclear pathways. Genomic and non-genomic effects. Ref. [29]: <http://dx.doi.org/10.1155/2015/953241>.

In an intervention study with 61 male athletes, it was observed that 62% had a serum concentration of 25 (OH) D of 20 ng/L at baseline and after supplementation with (5000 IU) of vitamin D3 per day for 8 weeks increased significantly 25 (OH) D concentrations and reflected on the velocity, verified through the 10-meter sprint time and the explosive force, through the vertical jump when compared to a placebo group [55].

Also in relation to physical performance, in a randomized, double-blind study, judo athletes supplemented with vitamin D3 achieved a 13% increase in muscle strength compared to a placebo group ($p = 0.01$) [56]. In a British study, through jump mechanography, was observed a positive association between serum vitamin D levels and jump height, velocity and power ($p = 0.005$, 0.002 and 0.003 , respectively) in postmenarche adolescent girls [57].

Although some studies have demonstrated a possible outcome on physical performance [58], on the other hand, researchers investigated whether weekly supplementation with vitamin D3 at doses of 20,000 IU (500 IU) or 40,000 IU (1000 IU) for 12 weeks improved 25 (OH) D levels or performance measures in 30 club-level athletes. No correlations were observed between 25 (OH) D concentration and performance measures, indicating either vitamin D3 supplementation does not influence skeletal muscle function to induce measurable effect. These results were obtained even after 25 (OH) D levels were increased at the end of the study [59].

3. Conclusion

Vitamin D deficiency results in poor muscle function and weakness that are reversible upon reaching a complete vitamin D state. However, prospective studies examining the effects of vitamin D on muscle function have shown conflicting results, measures of physical performance of the distinct population (non-athletes, athletes, young and old). In this way, to make emphatic conclusions on the ergogenic use of vitamin D, we still have reflections on the potential mechanisms of interaction, or even, what would be the appropriate levels for the different age groups.

The direct mechanisms by which vitamin D could affect lung function have not yet been fully elucidated. Perhaps in future well-controlled studies, the parameters of cause and effect may be better supported. However, studies have shown that treatment with Vitamin D3 was effective in postmenopausal women to produce a significant increase in plasma concentrations of 25 (OH) D [42]. This adequacy of vitamin D status was associated with improved pulmonary function parameters, independent of the performance of the aquatic exercise program. In addition, the correction of its deficiency could also be a supporting measure for the strategies used to prevent and treat diseases with impaired pulmonary function or incapacitated individuals.

Furthermore, new questions are raised as to whether athletes amateurs and professional susceptible to muscle damage and/or Vitamin D inadequacy, such as have been described in the elderly to exhibit low serum 25 [OH] D, experience aggravated declines in regenerative capacity and remodeling when serum 25 [OH] D is low. In addition to the health benefits, future studies may establish new views on the action of vitamin D as a possible legal ergogenic resource contributing to better athletic performance and record breaking.

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Author details

Rodrigo Nolasco* and Marise Lazaretti-Castro
Division of Endocrinology, School of Medicine, Federal University of São Paulo/
UNIFESP, São Paulo, Brazil

*Address all correspondence to: rodrigonolasco@yahoo.com.br

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