Effects of Vitamin D on Satellite Cells

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Vitamin D is a micronutrient that plays a role in the homeostasis of various body organs, including skeletal muscle. Skeletal muscle growth and regeneration are critically affected by satellite cells, skeletal muscle stem cells. The discovery of vitamin D receptors on satellite cells supports the role of vitamin D in regulating satellite cell function. In vivo studies have shown the effect of vitamin D on skeletal muscle growth in early life, muscle homeostasis in aging, and skeletal muscle regeneration in conditions of muscle injury or chronic disease.

vitamin D | satellite cells | skeletal muscle | in vivo

1. Introduction

Vitamin D is a prohormone that has two main inactive isoforms, namely vitamin D2 (ergocalciferol) and vitamin D3 (cholecalciferol) [1]. Vitamin D2 is obtained from ultraviolet (UV) irradiation of ergosterol, a steroid that is found in some plants and fungi. Meanwhile, vitamin D3 is obtained mainly from UV irradiation of 7-dehydrocholesterol in the skin [2][3]. In addition, vitamin D3 is also obtained to a small extent from dietary intake such as oily fish, meat, or egg [1]

Vitamin D either obtained from UV exposure or food is then hydroxylated in the liver by vitamin D-25-hydroxylase to become 25-hydroxyvitamin D (25(OH)D) or also called calcidiol. 25(OH)D is a stable metabolite in the blood and best reflects exposure and absorption of Vitamin D. Therefore, 25(OH)D is used as an indicator of vitamin D status [1][4]. Furthermore, 25(OH)D needs to be converted by the enzyme 25(OH)D-1-OHase (CYP27B1) in the kidneys to become 1.25-dihydroxyvitamin D ($1.25(OH)_2D$), the active form of vitamin D or also called calcitriol [5].

The classical function of vitamin D that has long been recognized is its role in the regulation of calcium and phosphate homeostasis and bone metabolism [6][7]. In the last few decades, some studies have indicated the non-classical function of vitamin D in various organs including skeletal muscle [8][9].

Satellite cells are skeletal muscle stem cells located between the basal lamina and sarcolemma which play a pivotal role in skeletal muscle growth and regeneration [10][11]. In homeostatic condition, satellite cells typically are in a quiescent state. The satellite cells will be activated when there are stimuli for skeletal muscle regeneration or hypertrophy, such as during an injury or exercise. Activated satellite cells will become myoblasts which proliferate and differentiate further to form new muscle fibers or fuse with preexisting muscle fibers [11][12]. This process is regulated by several myogenic regulatory factors (such as Myf5, MyoD, MRF4, and myogenin) [13][14]. In addition,

some activated satellite cells can also undergo self-renewal and return to a quiescent state to replenish the satellite cell population (**Figure 1**) [15].

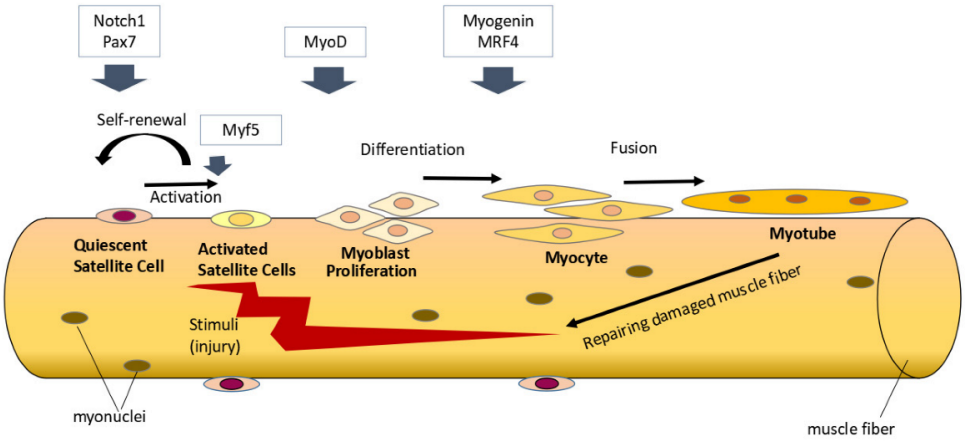

Figure 1. Satellite cells' activity in skeletal muscle regeneration. Under homeostatic conditions, satellite cells are typically in a quiescent state and express Pax7. Notch signaling plays a role in maintaining the quiescent state of satellite cells. When there are stimuli for skeletal muscle regeneration, various myogenic regulatory factors (Myf5, MyoD, myogenin, and MRF4) regulate satellite cells' activation, proliferation, and differentiation to form new muscle fibers. Some satellite cells will undergo self-renewal and return to a quiescent state to replenish the satellite cell population. Notch signaling through its regulation of Pax7 plays a role in promoting satellite cell self-renewal.

Several studies have shown that satellite cells express vitamin D receptor (VDR) [16][17][18]. Therefore, vitamin D may play a role in regulating satellite cells' activity and function. A previous systematic review has discussed the effects of active vitamin D on myogenesis in vitro [19]. However, in vitro models cannot fully mimic the microenvironment of in vivo models [20][21]. Satellite cells' fate is strongly influenced by their niche. Signals and properties of muscle fibers, basal lamina, as well as microvasculature, and surrounding interstitial cells influence the regulation of satellite cells' function [22]. When satellite cells are removed from their in vivo niche and cultured in vitro, the satellite cells are activated and committed to proliferation and differentiation, thereby losing their stem cell properties [23]. This research discusses the latest evidence from in vivo studies regarding the role of vitamin D on satellite cells.

2. Effects of Vitamin D on Satellite Cells

In the neonatal period, skeletal muscle undergoes a high growth rate that involves high protein synthesis accompanied by a rapid increase in myonuclei. Satellite cells contribute to the addition of myonuclei to growing muscle fibers in the postnatal period [24]. This process of skeletal muscle growth depends on the proliferation of satellite cells. In 4-day-old rats with a chronically unloaded hindlimb, there was impaired growth of the soleus muscle associated with a decrease in mitotically active satellite cells [25]. The rapid growth of skeletal muscle during early life requires adequate nutrition. Nutrition has a critical influence in providing components for muscle mass synthesis and various signaling involved in the muscle fiber anabolism [24].

Vitamin D is a micronutrient that plays a role in maintaining various body functions throughout life [26]. Srikuea et al. demonstrated that satellite cells are the target cells of vitamin D action and the response of satellite cells to vitamin D varies depending on age. There is a decrease in satellite cell response to vitamin D in aged muscles compared to muscles in the developmental age [18]. These findings support the importance of vitamin D signaling in early life when satellite cell activity is high. Improved vitamin D status is associated with increased proliferation and myogenic capacity of satellite cells in the early weeks of life[27][28]. However, studies do not support the effect of vitamin D signaling on satellite cell number during muscle growth in the early life period [27][29]. A possible explanation is that the high rate of satellite cell proliferation in the early life period plays a role in providing new myonuclei to growing muscles and not increasing the number of satellite cell reserves.

Acute injury involves sudden changes in the form of damage to muscle fibers, infiltration of inflammatory cells, edema, and damage to surrounding tissues. All of these lead to a change in the niche and trigger the activation of satellite cells [30]. Vitamin D signaling appears to influence satellite cells' function in skeletal muscle regeneration. In mice with vitamin D deficiency, there was a decrease in markers of activation and differentiation of satellite cells [31]. However, high-dose vitamin D supplementation without considering baseline vitamin D status leads to impaired satellite cell differentiation, delayed muscle fiber formation, and fibrosis formation in regenerating muscle [32]. Dosing appears to be a crucial issue when administering vitamin D during muscle regeneration. In vitro studies suggest that the administration of vitamin D supports muscle regeneration in a dose-dependent manner. However, at very high doses, it inhibits muscle formation [33][34][35]. The exact mechanism of the effect of various doses of vitamin D on the satellite cells' function in skeletal muscle regeneration needs to be explored further.

In aging, there are some changes in the satellite cell niche, which causes the satellite cells to lose their quiescence and tend to differentiate prematurely [36]. Exposure of satellite cells in aging mice with serum from young mice can restore the regenerative function of satellite cells [[37]. Aging is associated with decreased Notch signaling, a master regulator in maintaining the quiescent state of satellite cells[36]. Decreased Notch signaling in aged rats with vitamin D deficiency suggests that vitamin D may play a role in the regulation of Notch signaling in aging [38]. In in vitro study, Olsson et al. showed that the administration of vitamin D to human-derived myoblasts increased Hes1 mRNA expression, the gene target of Notch signaling [39]. One possible mechanism for vitamin D to regulate Notch signaling is its role in increasing Forkhead box O3 (FOXO3) expression [39]. A previous study demonstrated that FOXO3 is expressed in quiescent satellite cells. FOXO3 modulates Notch signaling by directly increasing Notch receptor expression. The FOXO3-Notch axis is required for satellite cell self-renewal by restoring satellite cell quiescence in regenerating muscle[40].

Vitamin D deficiency in aged rats was also associated with decreased Bmp4 and Fgf2 mRNA expression [38]. BMP signaling plays a role in increasing the satellite cell pool by promoting satellite cell proliferation and preventing precocious differentiation [41]. Stantzou et al. showed that inhibition of BMP signaling decreases the satellite cell pool [42]. Meanwhile, Fgf2 enhances satellite cell proliferation without suppressing differentiation [43]. Faria et al. assumed that the aged rats in their study experienced discrete regeneration episodes due to daily damage, so satellite cell proliferation was needed [38]. In another study, Fgf2 expression increased in aging muscle and triggered satellite cell proliferation and myogenic differentiation in homeostatic conditions. This causes satellite cell

depletion and reduced muscle regeneration capacity [44]. Further studies are needed to confirm the role of vitamin D supplementation on Fgf2 in aged rats.

In chronic illness, there may be a chronic injury to the muscle depending on the severity and type of disease. Alterations in energy metabolism, inflammation, or restriction of movement can be factors that cause changes in satellite cells' activity in diseased states [45][46]. Han et al. reported that an increase in extracellular adenosine (eADO) in diabetic mice decreased the regenerative function of satellite cells [47]. Satellite cells cultured on a high-glucose medium showed decreased proliferation and expression of Pax7, MyoD, and myogenin proteins [48]. In diseased experimental animal models, vitamin D deficiency aggravates the impaired function of satellite cells. Meanwhile, vitamin D supplementation ameliorates the impaired function of these satellite cells. Thus, it is important to pay attention to vitamin D status in various chronic diseases.

3. Conclusion and Future Perspectives

In vivo studies support a direct role of vitamin D on satellite cells' function during muscle growth, injury, aging, or chronic disease. Vitamin D appears to increase satellite cell proliferation in the early life period during rapid muscle growth. Adequate vitamin D status is required to support the satellite cells' function in skeletal muscle regeneration during acute injury. However, the administration of high doses of vitamin D decreases satellite cell differentiation and delays new muscle fiber formation. Vitamin D deficiency in aging was associated with the decrease in Notch signaling resulting in satellite cells losing their quiescent and differentiating prematurely. Vitamin D supplementation ameliorates the impairment of satellite cell function in chronic disease. Thus, to provide optimal effects on satellite cells' function, it is necessary to administer vitamin D at a dose according to the physiological needs of each individual. Further research is needed to determine the most appropriate dose and duration of vitamin D supplementation in the various age groups and specific conditions such as in early life, injury, aging, or chronic disease.

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