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- Review -

Elderly Sarcopenia and Vitamin B Deficiency: A Relationship?

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Sarcopenia is a leading cause of increased medical and nursing care costs among the elderly. In Korea, preventive measures for sarcopenia are mostly targeted toward the general elderly population without specific diseases. However, it is also necessary to implement measures for elderly individuals living in nursing homes and hospitals, where the prevalence of sarcopenia is high. Currently, computed tomography and/or magnetic resonance imaging are considered standard diagnostic tools. However, their complexity and time-consuming nature make them unsuitable for clinical use. The exact pathophysiological mechanisms of sarcopenia are unclear, as they involve various molecular biological pathways, including decreased exercise, protein and nutrient intake, changes in testosterone and growth hormone, and inflammation. Sarcopenia symptoms can lead to several diseases, such as osteoporosis, fractures, dementia, diabetes, and cardiovascular disease. Vitamin B deficiency is a significant factor in sarcopenia induction, with B vitamins being directly involved in energy and protein metabolism and nerve function. Vitamin B deficiency can lead to neuromuscular and neurogenic disorders, which often overlap with sarcopenia. Suboptimal intake of B vitamins, malabsorption, and anorexia are common among the elderly. This study aims to provide information on the role of water-soluble B vitamins in preventing and controlling muscle mass loss and deterioration among the elderly with sarcopenia. In addition, we discuss the potential of myokines from the B vitamin family in modulating sarcopenia.

Key words : Elderly, muscle, sarcopenia, vitamin B family

Introduction

Korea is entering a super-aged society. Statistics Korea predicts that the number of elderly people aged 65 and over will be 9.1% in 2005, 16.5% in 2021 and 24.1% in 2030, the fastest rate of aging among OECD countries. A major challenge of ageing is to extend the 'health lifetime', the period of time during which elderly people can live independently and without assistance in their daily lives. As humans age, skeletal muscle mass decreases each year. The rate of decline is 0.1-0.5% from the age of 30 and accelerates after the age of 65. This loss of muscle mass is accompanied by a decline in muscle function. This age-related loss of muscle mass and muscle function is called sarcopenia (from Greek *sarx*: flesh and *penia*: deficiency). The definition of sarcopenia was introduced by Irwin Rosenberg in 1989 [110]. More recently, a substantial clinical definition has been established, defining it as "a syndrome characterized by a progressive and generalized loss of skeletal muscle mass and strength with a risk of adverse outcomes such as physical disability, reduced quality of life, and death." Studies have been conducted worldwide to determine the exact definition and diagnostic criteria of sarcopenia. Due to ethnic differences, Asian, European, and American criteria have been established. These include the European Working Group on Sarcopenia in Older People (EWGSOP) in 2010 and 2019 [24, 26], the International Working Group on Sarcopenia (IWGS) in 2011 [36], and the Asian Working Group for Sarcopenia (AWGS) in 2014 and 2020 [17, 18]. As a result, various biomarkers of sarcopenia have been reported [74].

Sarcopenia is a condition characterized by a loss of muscle mass and negative muscle processes that occurs with aging [25]. Sarcopenia is caused by a combination of factors, including altered regulation of muscle formation, chronic in-

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flammation, hormonal changes, and lifestyle factors such as physical inactivity and poor nutrition, and can lead to a number of negative health outcomes, including falls, fractures, disability, and reduced quality of life [100]. Management of sarcopenia typically involves a combination of resistance exercise, dietary interventions, and potential pharmacological interventions such as hormone replacement therapy or nutritional supplements. Early detection and intervention are important to prevent or delay the progression of sarcopenia. Sarcopenia is associated with falls, impaired performance of activities of daily living, reduced social engagement, and decreased basal metabolic rate in the elderly, which promotes the development of type 2 diabetes and increases the risk of cardiovascular disease by 3-5 times. It is distinguished from cachexia, the decreased muscle function caused by anorexia and muscle wasting in cancer patients [31, 92]. In 2016, the WHO assigned a disease code (ICD-10-CM M62.84) to senile sarcopenia for the first time. In 2018, Japan recognized sarcopenia as a disease, and in 2021, sarcopenia was listed as a disease code (M62.5) in the Korean Standard Classification of Diseases (KCD). This means that senile sarcopenia is considered a target for active treatment, even though there are no strict clinical judgement criteria.

In the era of super old age, the study of sarcopenia is crucial for promoting healthy living among the elderly and mitigating the escalating social and economic expenses. This review aims to examine the role of sarcopenia and explore how deficiencies in the vitamin B family can interact and have additive effects. The review further highlights the importance of maintaining optimal vitamin B levels to impede the deterioration of muscle mass and function that occurs with age, as well as the role of vitamin B in supporting normal skeletal muscle physiological and metabolic processes in older adults. The potential of myokine intervention in preventing sarcopenia is also discussed.

Sarcopenia

There are three stages of sarcopenia: presarcopenia, sarcopenia, and severe sarcopenia. Presarcopenia is defined as a decrease in muscle mass, but not in strength or physical performance; sarcopenia is defined as a decrease in muscle mass and decreased strength or decreased physical performance; and severe sarcopenia is defined as a decrease in muscle mass, decreased strength, and decreased physical performance [115]. In general, hospitals use a combination of age, muscle mass, handgrip strength, walking speed, body mass index (BMI), waist circumference (WC), skeletal muscle index (SMI), fasting glucose (FG), triglyceride, and systolic blood pressure (SBP) to determine sarcopenia. In the United States, five criteria are generally considered loss of more than 5% of body weight in the past year, severe fatigue 3-4 days/week, significant reduction in physical activity, walking more than 6-7 sec/5 m, and grip strength of 29-32 kg (M) 17-21 kg (F) or less. According to the results of the Korean National Health and Nutrition Examination Survey (IV), appendicular skeletal muscle mass (ASM) began to decline around the age of 40 in men and 55 in women. When sarcopenia was judged by ASM, the prevalence of sarcopenia in people aged 65 years and older was 31.2% in men and 8.8% in women [47, 135].

The well-known symptoms associated with sarcopenia are as follows; skeletal muscle has slow-twitch type I and fast-twitch type II. Sarcopenia is associated with a decrease and atrophy of type II muscle fibers [127]; patients with vitamin D deficiency have muscle weakness and atrophy of type II muscle fibers [134]. Differences in muscle anabolism to hyperinsulinemia in the elderly, as opposed to the young, are a major cause of sarcopenia [21]. Decreased growth hormone/insulin-like growth factor-1 (IGF-I) in the elderly is associated with increased visceral fat, decreased muscle mass, and decreased bone mineral density [86]. Increased blood cortisol concentrations in the elderly are associated with decreased muscle mass [93]. Testosterone secretion decreases by 1% per year after age 30, which is associated with decreased muscle mass and strength [46]. Proinflammatory cytokines promote the degradation of myofibrillar proteins, decreasing protein synthesis and inducing apoptosis, leading to muscle atrophy [37]. The coexistence of chronic metabolic disease, non-alcoholic fatty liver and sarcopenia increases the risk of death by 2.2 times [85, 87].

However, the physiological phenomenon specific to the loss of muscle mass with aging has received little medical attention. This is due to a lack of scientific understanding of sarcopenia and the absence of standardized diagnostic criteria and treatments. There are no globally approved treatments, including those approved by the Food and Drug Administration (FDA) and the European Medicine Agency (EMA), and the treatment and prevention of sarcopenia is limited to adequate protein intake and appropriate exercise regimens [33]. In sarcopenia, muscle cells are characterized by 'myonuclear apoptosis', a process of nuclear condensation and decreased number of cells without death [32]. Therefore, global pharmaceutical companies are focusing on two main

areas to treat senile sarcopenia [66]. 1. Promotion of stem cell differentiation; drugs that favor the environment of stem cells and promote the production of myofibrillar proteins are being developed. 2. Inhibition of muscle fiber degeneration and apoptosis; blockers of myogenic signaling receptors and inhibitors of myogenic enzymes have been developed. It is reported that the market for sarcopenia drugs will exceed the market size of osteoporosis drugs (18 billion $/yr-US) in the future. There is an urgent need to fully understand the social costs of the elderly and the rapid increase in medical expenses (more than 40% of total national health costs) [56]. New concept of 'Comprehensive Management of Elderly Muscle Aging', which is a departure from fragmented elderly management, is expected to maintain a healthy and vibrant aging society [136].

Sarcopenia factors

Sarcopenia is recognized as the degenerative loss and atrophy of muscles that accompanies the aging process. Although aging is the primary contributor to sarcopenia, there are numerous factors that can significantly contribute to its development. While factors such as normal aging, hormonal changes, and genetics are beyond our control, other factors such as nutrition, medication usage, and physical inactivity are critical in mitigating sarcopenia. Individuals with a lifestyle that lacks physical activity are at a greater risk of developing sarcopenia compared to those who engage in regular exercise. This is because physical activity promotes muscle growth and maintenance, thereby inhibiting the age-related decline in muscle mass and strength. A sedentary lifestyle can increase the susceptibility to chronic diseases such as obesity, cardiovascular disease, and diabetes, leading to muscle loss and weakness. Therefore, regular physical activity and exercise are essential in preserving muscle mass and strength [114].

Hormonal changes are deeply implicated in the development of sarcopenia because hormones play an important role in maintaining muscle mass and strength. The main hormones involved in sarcopenia are testosterone, estrogen, growth hormone, and IGF-1. Testosterone declines as men age, and in women, estrogen declines after menopause, which can lead to muscle loss and weakness, which can contribute to sarcopenia. Growth hormone and IGF-1 are also important hormones for muscle growth and maintenance. As we age, muscle mass and strength decrease, and the decline in these hormones contributes to sarcopenia [105].

Certain medications can have an impact on muscle mass and strength. Corticosteroids are commonly used to treat inflammation, asthma, and autoimmune disorders, but their prolonged use can result in muscle loss and weakness. Antipsychotics are often prescribed to manage schizophrenia and bipolar disorder, but they may also lead to weight gain and metabolic changes, contributing to sarcopenia. Statins, which are used to lower cholesterol levels, can have side effects such as muscle pain and weakness, potentially leading to sarcopenia. Diuretics are used to treat high blood pressure and fluid buildup, but their use may result in potassium loss, which can cause muscle weakness and contribute to sarcopenia [14].

Nutrition plays an important role in maintaining muscle mass and strength. Sarcopenia is caused by not getting enough energy and nutrients for muscle protein synthesis, including protein, essential amino acids, vitamins, and minerals. In particular, protein is pivotal for muscle growth and maintenance, and insufficient protein intake leads to muscle loss and weakness. Therefore, elderly people who do not get enough protein are at a higher risk of developing sarcopenia. Vitamins and minerals are important for maintaining bone health, and not getting enough of these nutrients can lead to abnormal decreases in bone density, such as osteoporosis, which can lead to muscle loss and weakness. Poor nutrition contributes to obesity, diabetes and cardiovascular disease, which eventually leads to sarcopenia. To prevent sarcopenia and slow its progression, it is essential to maintain a balanced diet with adequate amounts of protein, essential amino acids, vitamins, and minerals [109].

Several gene variants have been identified as being associated with muscle mass and strength IGF-1 and insulin-like growth factor binding protein-3 (IGFBP-3), tumor necrosis factor alpha (TNFα), apolipoprotein E (APOE), ciliary neurotrophic factor receptor subunit alpha (CNTFRα), actinin alpha 3 (ACTN3), angiotensin-converting enzyme (ACE), vitamin D receptor (VDR) and uncoupling proteins 2/3 (UCP2/3) genes were found to be significantly associated with the muscle phenotype. Ten DNA polymorphisms (rs154410, rs2228570, rs1800169, rs3093059, rs1800629, rs1815739, rs1799752, rs7412, rs429358 and 192bp allele) were reported to be significantly associated with muscle phenotype [104]. Recently, genetic variants in Koreans have been reported. They are ribosomal protein S10 (RPS10), nudix hydrolase 3 (NUDT3) and glycerol-3-phosphate dehydrogenase 1 like (GPD1L) genes and three DNA polymorphisms (rs1187118; rs3768582 and rs6772958) [57].

Recently, a growing body of research has shown that chronic inflammation plays an important role in the develop-

ment of sarcopenia. Inflammation destroys muscle tissue and actively negatively affects muscle physiology (muscle growth, maintenance, regeneration and repair). In particular, elderly people with high levels of C-reactive protein (CRP) and interleukin-6 (IL-6) are at higher risk of developing sarcopenia. Proper exercise, diet and anti-inflammatory medications can reduce inflammation and improve muscle mass and function in the elderly. Reducing inflammation levels is an effective strategy for preventing and treating sarcopenia, as inflammation and sarcopenia are closely related, with inflammation due to aging being referred to as inflammation [28].

Metabolic and physiological factors involved in the development of sarcopenia have been reported to include decreased anabolic hormones, decreased motor units, decreased satellite cell number/function, mitochondrial dysfunction, altered proteostasis, increased oxidative stress, decreased membrane fluidity in muscle cells, and inhibition of mammalian target of rapamycin complex 1 (mTORC1) expression [34, 35, 41, 70, 82, 141].

Vitamin B family and Sarcopenia

A vitamin B family deficiency is only one of many factors in the development of sarcopenia. Maintaining a balanced diet that includes a variety of nutrient-dense foods, along with regular exercise and lifestyle modifications, is a good way to prevent sarcopenia [12, 30].

Vitamin B1 (thiamin) is an essential nutrient that plays an important role in energy metabolism and neurological function and is an important enzymatic cofactor in oxidative metabolism. It is directly involved in 24 enzymatic reactions [19]: pyruvate dehydrogenase, transketolase (biosynthesis and maintenance of the myelin sheath) 2-oxoglutarate dehydrogenase (biosynthesis of acetylcholine and 4-aminobutanoic acid) [96]. The UK National Diet and Nutrition Survey (NDNS) reported that approximately 1-2% of elderly people have problems with transketolase activity due to vitamin B1 deficiency [94]. Although no clear interrelationship between vitamin B1 and sarcopenia has been reported, some studies suggest that low vitamin B1 levels may increase the risk of muscle loss in the elderly [5], and that vitamin B1 deficiency may affect skeletal muscle exercise-related glycogen metabolism and adenosine monophosphate-activated kinase (AMPK) activation levels [114, 116]. Age-related neurodegenerative disorders caused by decreased muscle mass, such as sarcopenia, occur in the lower limbs of the elderly [55]. Other studies have shown that vitamin B1 supplementation can improve muscle function in elderly people with sarcopenia [91]. Vitamin B1 is found in a variety of foods, including cereals, nuts, seeds, and legumes, but some older adults become vitamin B1 deficient due to poor dietary habits or gastrointestinal disorders. Vitamin B1 may play an important role in muscle health.

Vitamin B2 (riboflavin) is an essential component of the flavin mononucleotide and flavin adenine dinucleotide and plays a role in energy metabolism and antioxidant mechanisms [48]. *In vivo*, it works in concert with other B vitamins to play an important role in fetal development, body growth, red blood cell production, and energy production in the heart muscle [6]. Vitamin B2 deficiency induces endocrine disruption, leading to hypothyroidism and impaired macrophage activity in the immune system [3, 79, 81], and hyperemia, throat edema, cheilosis, and reproductive disorders have been reported [39, 44, 79]. At the cellular level, it induces endoplasmic reticulum stress and the unfolded protein response, which impairs protein secretion [78]. Although specific evidence for vitamin B2 and sarcopenia is lacking, some studies have demonstrated an interaction; vitamin B2 supplementation prior to prolonged running reduced muscle soreness and enhanced early recovery of muscle function [45]. Vitamin B2 also improved muscle weakness in adolescent patients with multiple acyl-CoA dehydrogenase deficiency (MADD) [53], and in cachexia, vitamin B2 inhibited skeletal muscle atrophy in cancer-related sarcopenia [88].

Vitamin B3, niacin (nicotinic acid), is a coenzyme of NAD^+ & $NADP^+$ and is involved in calcium homoeostasis [40], gene expression [52], maintenance of mitochondrial function [68], antioxidant [54], and immune function [3]. In particular, age-dependent dysfunction of mitochondria leads to sarcopenia by lowering NAD^+ levels, making cellular energy production difficult [113]. However, normal mitochondrial oxidative capacity and NAD^+ biosynthesis can reduce sarcopenia [83]. The most well-known vitamin B3 deficiency-related disorder is pellagra, which causes dementia, delirium, and extreme anxiety [43]. Associated symptoms include gait ataxia, truncal ataxia, limb areflexia, and myoclonus due to muscle weakness [13, 112, 128]. Recently, a close relationship between vitamin B3 and sarcopenia has been reported; vitamin B3 induces muscle fiber transition from type II to type I [63], induces glycolytic skeletal muscle fibers to oxidative skeletal muscle fibers in obesity [108], and vitamin B3 improves muscle function in mitochondrial myopathy [103] and enhances endurance by inducing changes in skeletal muscle composition [107]. These results suggest that avoiding

vitamin B3 deficiency holds promise for the prevention and treatment of sarcopenia in the elderly.

Vitamin B5 (pantothenic acid) is essential for the biosynthesis of CoA and phosphopantetheine, which are important for collagen biosynthesis, promotes the synthesis of glucocorticoids, and cooperates with vitamin B6 to improve immune function [140]. There are reports of dystonia, spasticity, and pigmentary retinopathy associated with vitamin B5 deficiency [42, 144]. Muscle-related conditions include cardiac automatism, in which cardiac muscle cells excite themselves without stimulation, and rheumatoid arthritis, but no association with sarcopenia in the elderly has yet been reported [9, 60]. On the other hand, skeletal muscle CoASH, acetyl-CoA content, and exercise performance were not affected by vitamin B5 administration [137]. The induction of vitamin B5 deficiency-related sarcopenia has been reported in fewer studies compared to other members of the vitamin B family.

Vitamin B6 (pyridoxine) is an essential nutrient that is deeply involved in amino acid metabolism (biosynthesis of steroid hormones, hemoglobin, serotonin, and purines) and plays an important role in neurotransmitter synthesis, immune function, bone metabolism, and osteoporosis [27, 29, 140]. The active form of vitamin B6, pyridoxal 5′-phosphate, is also required for the enzymes that govern glucose release [72]. Vitamin B6 deficiency results in neurological symptoms such as loss of peripheral nerve sensation, decreased motor skills, and weakness and loss of tendon reflexes, leading to sarcopenia and osteoporosis [120, 122, 142]. Vitamin B6 is further required to prevent hyperhomocysteinaemia, a high prevalence in the elderly, from reducing bone density [89]. Recent studies have reported a direct and positive association with muscle regeneration and physical function as a novel function of vitamin B6 [7, 71]. In particular, vitamin B6 increases myokines, Nrf2-related factors, and myogenin gene expression in skeletal muscle, and vitamin B6 deficiency causes muscle spasms in diabetic patients [125, 146].

Vitamin B7 (biotin or vitamin H) is required for the biosynthesis of fatty acids, isoleucine and valine, and as a coenzyme of carboxylase for gluconeogenesis. Vitamin B7 is also important for maintaining healthy pregnancies, lowering blood sugar, controlling neuropathy, and controlling epilepsy [73, 85, 111]. Vitamin B7 deficiency-related conditions include telogen effluvium, infantile seborrhoeic dermatitis, and developmental regression [16, 77, 132]. There are reports of hyperthyroidism and progressive multiple sclerosis caused by excess vitamin B7 [10, 11]. Vitamin B7 reduces muscle cramps in hemodialysis patients and increases plasma biotin metabolites in haemodialysis patients with cramps [38, 98]. At this time, there are no reports of vitamin B7 as direct evidence of sarcopenia.

Vitamin B9 (folic acid) is important for fetal brain nerve and blood vessel development, preventing cleft lip and cleft palate, congenital heart disease, and perm chromosomal abnormalities in early pregnancy [131]. It is involved in the making and repairing of DNA and vitamin B12 activity [23, 76]. The best known symptoms of vitamin B9 deficiency are megaloblastic anemia, which results in thrombocytopenia, fetal neural tube defects, spina bifida and anencephaly [119]. High homocysteine is an important factor in age-related neurodegenerative diseases, which can be treated with vitamin B9 [118]. Vitamin B9 promotes myogenic differentiation and is essential for the proliferation and differentiation of myoblasts [50, 51]. Vitamin B9 deficiency in the elderly (>65 years of age) has been shown to cause sarcopenia, and serum vitamin B9 is strongly correlated with walking speed and muscle mass [49, 61, 65, 95, 138].

Vitamin B12, (cyanocobalamin), acts as a cofactor in DNA biosynthesis, fatty acid and amino acid metabolism, and is involved in myelin biosynthesis, which is important for the maintenance of the nervous system [101, 129], and is also involved in the development of red blood cells in bone marrow [62]. Vitamin B12 deficiency causes limb neuropathy and pernicious anemia, and plays an indirect role in sarcopenia by increasing homocysteine, methylmalonic acid and holotranscobalamin levels in muscle tissue, which can reduce muscle strength and gait speed, or increase the risk of fractures and postural instability [84, 117]. A number of studies have directly linked vitamin B12 deficiency to the development of sarcopenia [4, 15, 20, 75, 99, 106, 121]; muscle mass is significantly reduced in elderly people with type 2 diabetes [126]; and vitamin B12 deficiency is associated with gait speed and balance and affects muscle quantity rather than muscle strength or physical performance [97, 139]. Other sarcopenia-related findings include; vitamin B12 is associated with methylmalonic acid concentrations and muscle strength and is directly and positively related to physical functioning [2, 143]. Homocysteine concentrations are inversely related to physical performance (e.g., walking speed) in the elderly, and elevated serum homocysteine is associated with a decline in physical functioning [59, 133].

Vitamin B family and Myokines

Although skeletal muscles are important tissues that keep

the body functioning, recent studies have reported that they secrete bioactive substances similar to hormones secreted by the endocrine system. The word myokine was first used by Swedish scientist Dr. Bengt Saltin in 2003 [102]. Myokine is a combination of myocyte, meaning muscle cell, and cytokine, a class of small hormones in the body, and refers to the cytokines secreted by contracting skeletal muscles. In particular, myokines are secreted by skeletal muscles after exercise and can be released autocrine, paracrine, or endocrine, which means that they are transmitted not only to the muscle but also to other tissues in the surrounding area. To date, approximately 600 myokines are known [69]. Age-related sarcopenia has a variety of negative consequences, including decreased muscle mass and strength and increased risk of falls, disability, and death. Therefore, myokine biosynthesis and secretion in the elderly is important for maintaining muscle function and interactions with other tissues. Typical myokines include IL-6, which regulates glucose and fat metabolism [90], and irisin, which converts white adipose tissue to brown adipose tissue to reduce energy expenditure and improve insulin sensitivity [22]. Brain-derived neurotrophic factor (BDNF) is involved in neuroplasticity and cognitive function [58], and secreted protein acidic and rich in cysteine (SPARC) inhibits colon tumorigenesis [67]. Myostatin regulates muscle growth and development [8]. Vascular endothelial growth factor (VEGF) is involved in the growth and maintenance of new blood vessels [145]. Adequate exercise, good nutrition, and sleep play an important role in regulating myokine production and release in the elderly.

Vitamin B6 upregulates the expression of nine myokine genes (interleukin-6, interleukin-7, interleukin-8, secreted protein acidic and rich in cysteine, growth differentiation factor 11, myonectin, leukemia inhibitory factor, apelin and retinoic acid receptor responder 1) involved in promoting skeletal muscle growth and repair [123]. Although, to date, vitamin B6 is the only member of the vitamin B family that has been reported to function in myokine gene regulation. Vitamin B12, on the other hand, has been associated with loss of muscle mass in elderly type 2 diabetics [1]. N1-methylnicotinamide, as a vitamin B3 metabolite, improves the exercise capacity of skeletal muscle [124]. It is difficult to expect an adequate amount of exercise in the elderly, which means that the production and secretion of various myokines by the normal activity of skeletal muscles becomes difficult. As a result, the prevalence of various metabolic diseases with sarcopenia and degenerative brain diseases can easily increase in the elderly. In this regard, more studies of myokine gene regulation by the vitamin B family are expected, and eight myogenesis-related factors (myogenin, muscle RING-finger protein-1, myogenic factor 5, myogenic factor 6, myogenic differentiation 1, Atrogin-1, and specificity protein) that show differential gene expression by vitamin B6 have already been reported [64]. Their involvement in sarcopenia by the B vitamin family is also expected.

Conclusion

As we age, a decline in muscle mass and strength occurs, which is defined as sarcopenia. This can lead to weakness in limb muscles, resulting in falls, difficulty performing daily activities, social isolation, and reduced exercise. Unfortunately, this also increases the likelihood of developing conditions such as hyperlipidemia, obesity, hypertension, diabetes, and myocardial infarction, which can result in higher mortality rates. Research has suggested that the B vitamin family may play a role in preventing sarcopenia, although the molecular mechanisms are not yet fully understood. Maintaining a "health lifetime" where elderly individuals can live independently is crucial in a society that is aging at an unprecedented rate. Adequate intake of B vitamins through food is essential in preventing and treating sarcopenia. This review summarizes the latest research on sarcopenia related to B vitamins, including B1-3, B5-7, B9, and B12. The contents described in this review are summarized and shown in a schematic diagram (Fig. 1).

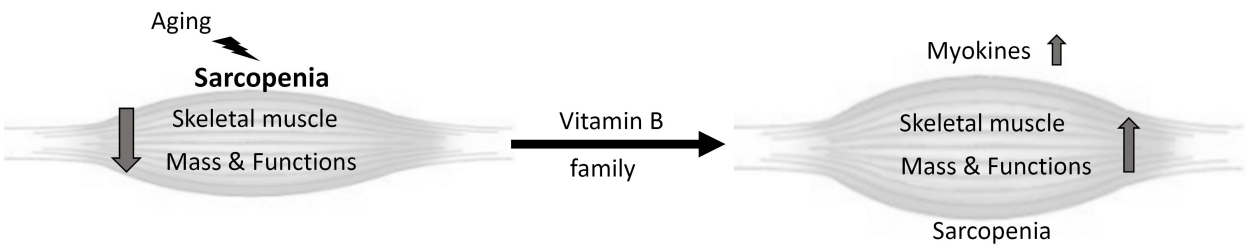

Fig. 1. Effect of vitamin B family on sarcopenia.

The Conflict of Interest Statement

The authors declare that they have no conflicts of interest with the contents of this article.

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초록 : 비타민 B 결핍에 의한 노인성 근감소증

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노인들에서 근감소증은 의료-간호비용 증가의 주요 원인 중 하나가 되고 있다. 한국에서는 근감소증 예방 대책이 일반적으로 특정 질병이 없는 노인들을 대상으로 하지만, 요양원-요양병원에서 집단 거주하는 노인들의 근감소증 대책도 필요하다. 근감소증은 운동량 감소, 단백질 및 영양분(미네랄, 비타민 포함) 섭취 감소, 테스토스테론 및 성장호르몬 변화, 염증 등의 원인으로 발생한다. 분자 생물학적인 정확한 병태생리 기전의 이해가 요구된다. 근감소증은 골다공증, 낙상으로 인한 골절, 치매, 당뇨병, 심혈관 질환 등의 증상을 연결될 수 있다. 비타민 B 패밀리(B1-3, B5-7, B9 및 B12) 결핍을 근감소증 유발의 연구 대상으로 선택한 이유는 다음과 같다. 이는 비타민 B가 에너지 및 단백질 대사에 직접 관여하여 정상적인 신경 기능 유지에 필수적이다. 비타민 B 결핍은 신경-근육 질환, 신경성 질환으로 나타날 수 있으며, 노인성 근감소증과 병행하는 경우가 많다. 노인들은 적정치 이하의 비타민 B 패밀리 섭취, 흡수 장애 및 무식증 문제 등을 겪을 가능성이 높다. 초고령화 사회에서 elderly가 자립적으로 일상생활을 할 수 있는 'health lifetime'을 유지하는 것은 개인의 행복추구와 사회경제적 부담을 줄일수 있는 최고의 목표이다. 본 연구는 근감소증과 관련하여 노인들의 근육량 감소 및 근육 기능 저하를 조절하는 수용성 비타민 B 패밀리의 최신 정보를 제공한다. 또한, 비타민 B 패밀리를 통한 마이오카인에 의한 근감소증의 조절 가능성도 소개한다.