Sport and Fitness Journal
E-ISSN: 2654-9182 Volume 11, No.3, Sept 2023: 206-215

THE POTENTIAL EFFECT OF L-CARNITINE AND EXERCISE ON MUSCLE STRENGTH IN AGING: A LITERATURE REVIEW

Kusuma Sri Whisnu Puteri[1*], Kristina Puspadewi[1], Indira Vidiari Juhanna[2], I Putu Adiartha Griadhi[2]
[1]Biomedical Anti Aging Medicine Magister Program, Faculty of Medicine, Universitas Udayana, Denpasar, Bali, Indonesia.
[2]Department of Physiology, Faculty of Medicine, Universitas Udayana, Denpasar, Bali Indonesia.
Email: kusumaswput@gmail.com

ABSTRACT

Aging is an inevitable process, and it can affect various aspects of the human body, including the musculoskeletal system. L-carnitine (LC) has the potential to be used in conjunction with exercise to mitigate the loss of muscle strength associated with aging. This review aims to investigate the impact of LC and exercise on muscle strength in the context of aging. We conducted a comprehensive literature search using various online databases such as PubMed, EMBASE, CENTRAL, and Google Scholar. Our search involved keywords such as "L-carnitine," "exercise," "muscle strength," and "aging" to identify relevant literature. A total of 8 studies were included in this review and 7 of them were conducted in elderly. We present a narrative review to examine the role of exercise and L-carnitine in relation to muscle strength during the aging process. The combination of LC and exercise holds promise as a potential intervention. Both LC and exercise have shown benefits in mitigating the loss of muscle mass, strength, and function. However, when used together, they may yield even better results, particularly because LC can help improve functions that are typically impaired by aging. The decline in muscle mass, function, and subsequently strength associated with aging can significantly impact one's quality of life. LC and exercise both have impact to improve muscle strength in aging but further studies have to be made to assess precisely how the combination of both may benefit for the elderly.

Keywords: aging; exercise; L-carnitine; muscle strength

INTRODUCTION

The aging process is unavoidable and will affect all levels of human body. It has been gaining attraction with life expectancy has been increasing in the past few decades, which means that there will be a significant increase of people over the age of 60 years old by 2050. The musculoskeletal system is not immune to this process and the effect of aging on it can be seen in the increased risk of degenerative joint disease, muscle mass, and strength loss, along with the disabilities that will follow often found among the elderly. This can cause major impairments in the quality of life. Researchers thus far had suggested that cellular senescence is the link between aging and deficits in structure and function and is induced by high levels of reactive oxygen species (ROS) and telomere damage (1,2).

Many have suggested the use of exercise as a strategy to counter aging in the musculoskeletal system, as it prevents the accumulation of senescent cells and even stimulates muscle protein synthesis. However, geriatric patients are often with comorbidities that would

make it difficult to do certain exercises. Problems with anabolic resistance often found in geriatric patients can lead to muscle protein net imbalance (2–6).

L-carnitine (LC) has been researched for many years and is considered a useful endogenous component that helps with energy production and fatty acid metabolism (4,7). Its role has been numerously studied in affecting muscle strength. It also plays a role in suppressing RING-finger protein-1 (MuRF-1) and ubiquitin-protein conjugates, protein catabolism, and increasing levels of IGF-1 and Akt1 that play an important role in protein anabolism. It also has antioxidant and anti-inflammatory properties. Hence, making it potentially beneficial when combined with exercise. Studies have also highlighted that the lower level of L-carnitine is seen in aging condition. However, there is still very little research on this (4,8). In addition, there are very limited studies on how L-carnitine may help to affect the muscle condition in the aging process, whether it is being combined with the exercise or not. Therefore, this review aims to explore the effects of LC and exercise on muscle strength in aging and the potential research gap to better understand the role of both in aging.

METHODS

This was a narrative literature review study. Eligibility criteria were generated based on the PICO framework. The population was adult males and females; the interest was L-carnitine and exercise; the comparator was any comparator; and the outcome was muscle strength. Based on the PICO framework, the keywords in this review were (L-carnitine) OR (exercise) AND (muscle strength) AND (aging) to perform literature searching. We found 994 articles and after the screening and abstract reading, we included 8 studies. The online databases were PubMed, EMBASE, CENTRAL, and Google Scholar. We included studies from 2013-2023 comparing L-carnitine and exercise and reported the assessment of muscle strength which were published in English. Studies that were not original articles and were conducted in otherwise healthy subjects (e.g. congenital abnormalities) were excluded. The findings were narratively elaborated.

Authors	Subjects	Intervention	Results	Conclusion
Evans et al., 2017(8)	42 healthy older adults (55-70 years old)	L-carnitine combination (L-carnitine 1500 mg + L-leucine 2000 mg, creatine 3000 mg, vitamin D3 10 μg), L-carnitine 1500 mg, or placebo	Average leg strength L-carnitine showed higher leg strength than placebo (p = 0,007) and its effect was maintained until the end of the study (average 2.8 kg ~ p =0,061)	L-carnitine improved muscle strength in healthy older adults
Sawicka et al., 2018(9)	28 healthy older women divided into two groups: intervention and placebo	Intervention group: 1500 mg L-carnitine L-tartate for 24 weeks Placebo group: isonitrogenous placebo for 24 weeks	Average Power Extension (W/kg) Baseline I: 8.6 ± 1.4 P: 8.3 ± 2.0 After 24 weeks: I: 5.4 ± 11% P: 3.9 ± 6.2%	No significant changes after L-creatinine supplements were observed in muscle strength

			Average Power Flexion (W/kg) Baseline I: 3.9 ± 1.4 P: 4.4 ± 1.7 After 24 weeks I: 5.8 ± 17% P: 14 ± 12%	
Bardasawi et al., 2016 (10)	50 elderly (age 60 years and above) divided into two group: control vs intervention	Placebo: Corn starch filling for 5 weeks Intervention: L-carnitine 500 mg/cap for 5 weeks	Frailty Index Score Intervention Base: 0.112 ± 0.065 5-Wks: 0.097 ± 0.063 10-Wks: 0.076 ± 0.0578 P = 0.001 Placebo Base: 0.118 ± 0.045 5-Wks: 0.116 ± 0.056 10-Wks: 0.111 ± 0.062 P = 0.764	L-carnitine significantly improved frailty indicator which included assessment of muscle strength
Kennis et al., 2013 (11)	83 elderly age 60-80 years old divided into control (C) and strength-training intervention group (I)	C: participate in no training program I: exercised 3 times weekly for a year combining resistance and aerobic training or whole-body vibration training	Control Change after 1 year Static: +0.22±3.28 Dynamic 60°/s: +0.35±3.09 Dynamic 240°/s: +2.80±2.66 Intervention Change after 1 year Static: +11.46%±1.86% Dynamic 60°/s: +6.96%±1.65% Dynamic 240°/s: +9.25%±1.68% P < 0,001	Intervention after 1 year improves muscle strength in older adults
Hamed et al., 2018 (12)	47 older adults (65-80 years old) divided into 3 groups: (i) muscle strength, (ii)	Muscle strength group: performed resistance training for leg and trunk muscles	Maximum ankle joint moment (Nm/kg) Baseline Strength: 1.48 ± 0.42 Perturbation: 1.42 ± 0.46	Perturbation-based program showed promising muscle strength

	perturbation-based, (iii) control	Perturbation-based: performed exercise mechanisms of dynamic stability under unstable conditions All conducted two times a week, 90 minutes, for 14 weeks	Control: 1.73 ± 0.44 After 14 weeks Strength: 1.77 ± 0.56 Perturbation: 1.74 ± 0.40 Control: 1.73 ± 0.52 Maximum knee joint moment (Nm/kg) Baseline Strength: 2.05 ± 0.53 Perturbation: 1.81 ± 0.46 Control: 2.24 ± 0.53 After 14 weeks Strength: 2.22 ± 0.57 Perturbation: 1.86 ± 0.46 Control: 2.19 ± 0.44	enhancement outcome
Abrahin et al., 2022 (13)	30 elderly women divided into two groups: 1-set and 3-set	1-set of resistance training program 3-set of resistance training program Both were conducted two times a week for 12 weeks, duration of minimum 48 hours between session	Calf Raise 1-set Baseline: 21.8±3.5 Post: 38.6±2.3 3-set Baseline: 25.0±2.8 Post: 40.6±8.6 Bench press: 1-set Baseline: 13.8±1.7 Post: 21.6±3.2 3-set Baseline: 14.3±3.5 Post: 22.0±4.0	Both single- or multiple-set resistance training improved muscle strength in elderly women
Sadeghi et al., 2021 (14)	58 elders aged above 65 years old divided into three groups: mix, balance training, virtual reality, and control	Mix: performed a combination set of warm-up, balance training, and virtual reality Balance: performed exercise such as single-leg stance with eyes open	Quadriceps muscle (change) Balance: 5.5% Virtual: 24.7% Mix: 27,6% Control: -4,2% Hamstring muscle (change) Balance: 9.1% Virtual: -12.4%	Mix group revealed more significant results in muscle strength, followed by virtual and balance training

		and closed, tandem walking, and weight shifting Virtual reality: performed exercise-focusing games that enabled participants to move Intervention was given for 8 weeks	Mix: 28.0% Control: 17.3%	compared to control.
Ahmad et al., 2023 (6)	68 overweight and obese adults divided into four groups: control, L-carnitine only, exercise only, and combination of both	L-carnitine: 1000 mg of L-carnitine taken every day for 12 weeks Exercise: brisk walking exercise for 30 minutes at 50% heart rate maximum, followed by Tabata exercise for 10-20 minutes, 3 times a week	IL-6 Control Pre: 7.2 ± 0.2 Post: 7.4 ± 0.2 L-carnitine Pre: 6.4±0.2 Post:5.9±0.1 Exercise Pre: 6.6±0.2 Post:7.0±0.2 Combination Pre: 6.3±0.2 Post: 5.6±0.2	Intervention focusing on both L-creatinine supplement and exercise may improve inflammatory markers.

AGING AND THE SKELETAL MUSCLE

The skeletal muscle is made out of fibers, which are differentiated skeletal muscle cells, satellite cells, endothelial cells, pericytes, fibroadipogenic progenitor cells, macrophages, neurons, tenocytes, and neutrophils (2). One of the effects of aging is the decreasing size of muscle fibers led to a decline and an increase in total fat mass that is simultaneously joined by a decrease in lean body mass (1,2). Adipose tissues are capable of releasing inflammatory cytokines, such as interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α), which activated molecular pathways that play a role in muscle wasting which leads to protein imbalance according to some literature. Increased IL-6 and TNF-α have also been linked to lower muscle strength according to one study linking C-reactive protein (CRP) and IL-6 with lower muscle strength in geriatric subjects. It was also reported that insulin-like growth factor 1 (IGF-1) has a role in regulating anabolic and catabolic processes via its involvement in the skeletal muscle signalling pathways (9,15). IGF-1, Akt/Protein Kinase B-mTOR pathway helps in stimulating the production of new myofibril proteins which allows the process of remodelling for skeletal muscle. As IGF-1 binds with its receptor, it will trigger the phosphorylation of the intracellular

adaptor protein insulin receptor substrate (IRS-1), which will lead to the phosphorylation of phosphoinositide 3-kinase (PI3K) followed by the phosphorylation of the Akt. The activation of the Akt signalling pathway led to the inhibition of proteins that were involved in the ubiquitin-proteasome system (UPS), such as MuRF-1 and atrogin-1, which are responsible for protein degradation (15,16). Aging is also highly linked to mitochondrial dysfunction, which occurs due to a prolonged imbalance between the produced and scavenged ROS by endogenous antioxidants and a decrease in ATP availability. A declining metabolic function worsens oxidative stress via the accumulation of lipotoxic intermediates which then lead to protein damage. Together with ROS and a decline in protein turnover, it serves as the primary component of skeletal muscle dyshomeostasis (9,17). Atrogin and MuRF-1, which happens to be nuclear factor-kB (NF-kB), in skeletal muscle are often increased by cytokines or ROS, leading to muscle loss and atrophy. Collectively these aspects led to the loss of muscle strength (18).

THE EFFECTS OF EXERCISE ON AGING MUSCLE

Exercise is reported to be beneficial since it can significantly attenuate or even prevent declines in muscle metabolism and function. It can also mitigate problems that have risen because of both the physiological and structural changes in the muscle along with the changes that come due to inactivity. Exercise has been shown to be associated with significant declines in IL-6 and TNF-α after aging. Lower CRP levels were also reported after a 16-week aerobic exercise intervention. There were also studies documenting how exercise training can stimulate mitochondrial biogenesis by increasing the peroxisome proliferator-activated receptor γ coactivator 1α (PGC-1α). It was also suggested that exercise can help improve the function of the mitochondria by remodelling the mitochondrial network. This is supported by findings that show how exercise has been found to be associated with better mitochondria organization and lower expression of genes related to autophagy and ROS. One study also reported how physical activity has been shown to positively affect muscle regeneration as shown by how both resistance and endurance exercise training increased the number of satellite cells (1,19).

Some would argue, however, that reduced net protein balance will still happen in an aging muscle due to anabolic resistance. The phosphorylation of both serine-threonine residues is considered important for Akt activity. In a healthy phenotype, this mechanism occurs immediately as a response to exercise, but due to aging this response is blunted. This causes a disruption in downstream signalling and metabolism. The Tuberous sclerosis complex (TSC) will activate the mTOR complex 1. TSC, phosphorylated by Akt, will allow the interaction between mTORC1 and Ras homolog enriched in the brain (Rheb) and lead to the phosphorylation of mTORC1. mTOR itself is similar to Akt and plays an important role in mTORc signalling that will result in muscle protein synthesis. After exercising and ingesting dietary protein, mTORC1 activity is upregulated which will then result in an increase in muscle protein synthesis. Hence, through this acute activation of the Akt-mTORC1 pathway, exercise actively promotes skeletal muscle remodelling, which prevents muscle protein breakdown pathways such as autophagy. Therefore, in order to prevent the negative protein balance, regular exercise would be more effective when paired with the correct dietary intake. An increase in ROS due to strenuous exercise should also be taken into account, because it may pose a risk considering the physiological changes that occur in older populations (16,20,21).

THE EFFECTS OF L-CARNITINE AND ITS POTENTIAL USE

LC is an amino-acid-like molecule found in skeletal muscle that is essential for energy metabolism due to its role in the oxidation of fatty acids. It plays a role in muscle atrophy, which is supported by some investigations reporting improved nitrogen balance via the enhancement of protein synthesis and decreased protein breakdown, apoptosis prevention, anti-inflammatory properties, improvement in muscle strength, or the reduction of muscle weakness in subjects. Albeit, these studies are done with healthy adults and children instead of older adults (8,22–24). One study, however, uses the combination of LC with creatine and L-leucine for older adults and it showed significant improvements in muscle mass and strength compared to placebo (8).

LC affected protein synthesis and degradation by increasing the plasma IGF-1 concentration that plays a major role in the Akt/Protein kinase-B-mTOR pathway. Until now, the certain mechanism on how LC affect increased expression of IGF remained unclear, however the process itself might involve the role of high-energy phosphate metabolism, which therefore allowing the individuals to perform greater resistance intensity. Therefore, the IGF-1 itself increases (25). Higher levels of IGF-1 led to the activation of the signalling pathway, which causes mTOR phosphorylation (7,18). The activation of mTOR would then result in accelerated protein synthesis. Simultaneously, Akt will phosphorylate and inactivate forkhead box O (FoxO) thus inhibiting proteins responsible for proteolysis ubiquitin ligases, which are MuRF-1 and atrogin-1 (18). The use of LC for mitochondrial dysfunction has also shown some promise, as LC deficiency can cause mitochondrial dysfunction. This is because LC plays an important role in fatty acid metabolism, production of ATP and mitochondrial processes. LC also played a role in cellular detoxification, control of ketogenesis and glucogenesis, and stabilization of cell membranes (26). It is reported as well that LC can reduce stress induced by strenuous exercise and in vitro studies stated that it is an efficient superoxide anion radical and hydrogen peroxide scavenger (4). Decreased inflammatory markers have also been reported with the supplementation of LC. Long-term supplementation has been associated with lower IL-6 and TNF-α levels. This will be beneficial since inflammation also produces ROS, which controls the production of inflammatory cytokines and activates the NF-kB pathway. Some have speculated that LC obstructs the NF-kB pathway by limiting ROS production. LC's role in preventing lipid peroxidation is also one of the mechanisms that are suggested to be linked to its anti-inflammatory effects and its anti-inflammatory effects would also be beneficial due to the build-up of adipocytes that comes with aging (6).

THE COMBINATION OF L-CARNITINE AND EXERCISE

The reasoning for combining LC and exercise comes from the hypothesis that it will be beneficial for post-exercise recovery and exercise-induced muscle damage due to its effect against oxidative stress. One study documented that daily supplementation of 2g LC for at least 2 weeks significantly increased total antioxidant capacity and lower lipid peroxidation and another reported an increase in muscle carnitine is linked with a 20% increase in the rate of whole-body total fat oxidation during exercise at 50% VO2 max. This was supplemented to participants who underwent moderately-intense exercise (brisk walking and Tabata exercise) for 12 weeks. LC's abilities to decrease MuRF-1 and ubiquitin-protein conjugates, protein catabolism, and increase levels of IGF-1 and Akt1 should theoretically be able to help this process that is blunted with aging, thus making exercise more effective in older adults. All of this combined, should be able to optimize both individual benefits, thus delaying the effect of aging on the skeletal muscle which will help in preserving the muscle mass, function, and in effect, muscle strength as well (6,8,18). In this review, the mechanism of aging in skeletal

muscles, the effect of both exercises, LC, and the combination of both has been reported. To the author's knowledge, it's the only review thus far reporting the combination of LC and exercise on muscle strength. However, this review has limitations, which include the involvement of very little literature speaking about the combination of LC and exercise, and the side effects of LC supplementation that were not explored.

CONCLUSION

In conclusion, the effects of aging on the skeletal muscle which resulted in the loss of muscle mass, function, and in effect, strength, can cause damage to the quality of life. Either L-creatinine supplementation (minimum of 500 mg for 5 weeks) and exercise (either resistance, aerobic, or combination of both done routinely) can help to improve muscle strength in aging populations. The combination of LC and exercise has the potential for therapeutic use but further research involving geriatric patients is still needed to confirm this hypothesis.

CONFLICT OF INTEREST

There is no conflict of interest related to the materials or methods used in this study.

FUNDING

This article received no specific funding from any funding agency in the public, commercial, or not-for-profit sectors.

AUTHOR CONTRIBUTION

The authors took part in the manuscript, contribute to data collection, and participated in writing the manuscript and all agree to accept equal responsibility for the accuracy of the content of this article.

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