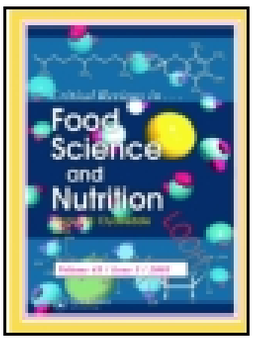

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Nutrition and sarcopenia: A review of the evidence of nutritional influences

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ABSTRACT

Prevention of age related decline in muscle mass and strength is a key strategy to keeping physical capacity in older age and allowing independent living. To emerge preventive strategies, a better understanding is required of life style factors that impacts on sarcopenia. However, since muscle mass and strength in later life depend on both the rate of muscle loss and the peak achieved in early life, attempts to prevent sarcopenia also require considering diet through the life course and the potential benefits of early interventions. Optimizing diet and nutrition status during the life may be an important strategy to preventing sarcopenia and enhancing physical ability in older age.

KEYWORDS

Nutrition; Diet; Sarcopenia; Muscle strength; Muscle function; Body composition

Introduction

Sarcopenia is the progressive loss of muscle mass and function, muscle strength and performance (Tanimoto et al. 2012), that occurs after the fifth decade of life (Kim, Wilson, and Lee 2010). The functional limitation and deficiencies due to sarcopenia reduce quality of life (Kim, Wilson, and Lee 2010), and resulting to significant impairments in physical performance, metabolic disorders, and disability (Roth et al. 2004). Losses in muscle strength have also been related to mortality in aged adults (Rantanen et al. 2000).

Decreasing muscle mass and strength are anticipated to be parts of ageing. While the rate of decrease varies across the population (Cruz-Jentoft et al. 2010; Syddall et al. 2009), proposing that modifiable factors such as diet and lifestyle may have major impacts on muscle performance in older age. This paper reviews the evidence that associates diet and nutrition with muscle mass and strength, and implications for strategies to prevent or retard sarcopenia in elderly population.

Methods

The data was obtained according to the result of original and review articles associating with the nutrition intake, muscle mass, muscle strength and function, and risk of frailty. For this purpose, we mostly used the online database PubMed and google scholar search engines, with the use of following keywords: "nutrition intake", "ageing", "dietary acid-base load", "sarcopenia", "muscle strength", "muscle function", "muscle mass". Then, we chose the relevant free- access full texts and reviewed the appropriate articles. We considered suitable published articles in the English language with no restrictions for the dates of articles. Our review was incorporated animal and human studies. In addition, we searched some of the references of the selected articles to make clear the associated topics better. We summarized the information about some of the most important papers in Tables 1 and 2.

Nutrition and ageing

Food intake reduces around 25% between 40 and 70 years of age (Nieuwenhuizen et al. 2010). Compared with young ages, older adults eat more slowly, they become less hungry and thirsty, ingest smaller meals, and they consume snack, rarely (Nieuwenhuizen et al. 2010). The mechanisms for the "anorexia in ageing" don't have completely understood, but there may be different causes such as physiological, psychological, and social factors that affect appetite and food intake, comprising loss of taste and sense of smell, chewing troubles, and damaged gut function (Nieuwenhuizen et al. 2010; Murphy 2008). The negative results of these alterations combine with the impacts of functional disorders that affect the ability to access food, psychological impairments like depression and dementia, and also social impacts of living and eating solely. Low food intakes and repetitive diets put aged people at risk of insufficient nutrient intakes (Bartali et al. 2003). Hence, in a vicious cycle, decreasing muscle strength and physical capacity in old ages may enhance the risk of poor nutrition, while poor nutrition may lead to further decreases in physical capacity.

The exact evaluations of prevalence of poor nutrition may vary according to the definitions utilized, but studies in community- dwelling adults constantly propose that it is common in elderly population. For example in the National Diet and Nutrition Survey in the UK, 14% of older population living in the community, and 21% of those living in institutions were at risk of undernutrition (Margetts et al. 2003). These findings

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Table 1. Results from studies of relationships between macronutrient intakes and sarcopenia outcomes.

First Author (Reference No)	Nutrient(s) Investigated	Intervention or Exposure	Time	Result
Dillon (Dillon, 2013)	Protein	Randomized to daily dose of 15 g essential amino acid (EAA) (n = 7) or placebo (n = 7) in older women (68 ± 2 y).	For 3 months	EAA ameliorated lean body mass (LMB) and basal protein synthesis in older individuals. The acute anabolic response to EAA supplementation is kept over time and can ameliorate LBM.
Vafa (Vafa et al., 2017)	Protein	Among 190 women aged 40–60 yr, data including of muscle strength and function, anthropometric indices, dietary intakes, and physical activity were assessed.	Aged 40–60 y Throughout postmenopause period	There was a positive association between protein intake and fat free mass percentage, but there was not a significant association between protein intake and other variables.
Houston (Houston et al., 2008)	Protein	Dietary protein intake and changes in lean mass (LM) and appendicular lean mass (aLM) were evaluated in 2066 men and women aged 70–79 y who were participating in the Health, Aging, and Body Composition study.	Between 1997–1988	Protein intake was related to 3-y alterations in LM and aLM. Participants in the highest quintile of protein intake lost 40% less LM and aLM than did those in the lowest quintile of protein intake.
Børsheim (Børsheim et al., 2008)	Protein	Twelve glucose intolerant men (n = 5) and women (n = 7) (67 ± 5.6 y) consumed 11 g essential amino acid+arginine two times a day, between meals.	For 16 weeks	Lean body mass and the lower extremity strength score elevated during the study. Advances were also observed in usual gait speed, timed 5-step test, and timed floor-transfer test.
Stinick (Sitnick, Bodine, and Rutledge, 2009)	Fat	Male C57BL/6 mice (n = 120) were randomized into two diet groups: a low fat, high carbohydrate diet and a high fat, low carbohydrate diet. Each diet group were divided into two groups: sedentary or functional overload of the plantaris muscle.	At 5 weeks old For 14 weeks	High fat diet damages the ability of skeletal muscle to hypertrophy in response to increased mechanical load.
Welch (Welch, MacGregor, and Minnihane, 2014)	Fat	Body composition (fat free mass (FFM; in kg) and the fat free mass index (FFMI; kg FFM/m^2) was assessed in 2689 women aged 18–79 y and then figured according to quintile of dietary fat.	Between 1996–2000	Positive relationships were shown between the polyunsaturated-to-saturated fatty acid ratio and indices of FFM, and inverse relationships were shown with percentage of fat energy, saturated fatty acids, monounsaturated fatty acids, and trans fatty acids.
Smith (Smith et al., 2011)	Fat	Sixteen older adults (≥65 y of age) randomized to daily omega-3 fatty acids or corn oil intake.	8 weeks	Omega-3 fatty acid supplementation enhanced the hyperaminoacidemia-hyperinsulinemia-induced elevate in the rate of muscle protein synthesis, which was coexist with greater enhances in muscle mTOR and p70s6k phosphorylation, but had not impact on the basal rate of muscle protein synthesis. Corn oil had not impact on muscle protein synthesis and anabolic pathways.
Fiaccavento (Fiaccavento et al., 2010)	Fat	δ-sarcoglycan-null dystrophic hamsters were received a diet enriched in omega-3 α-linolenic fatty acid.	From weaning until death	Omega-3 α-linolenic fatty acid modulated the sarcolemma lipid composition and protein pattern, and conserved appropriate morpho-functional quality in dystrophic skeletal muscles.

demonstrate that there are significant numbers of aged adults have less than optimal nutrition.

Is diet an effective factor on sarcopenia?

Some nutrients and bioactive components have been most constantly related to sarcopenia and weakness. These nutrients with their specific mechanisms by which they can affect the muscle, are discussed in the following sections.

Protein

Protein turnover is important for muscle and the balance between anabolism and catabolism is essential for keeping of skeletal muscle mass. The considerable metabolic pathway which essential amino acids act to make an anabolic response is the mammalian target of rapamycin (mTOR) pathway and present belief is that this is controlled by the branched chain amino acid leucine (Dillon 2013). Recently, some evidence

Table 2. Results from studies of relationships between sarcopenia with micronutrient intakes and dietary acid-base load.

First Author (Reference No)	Nutrient(s) Investigated	Intervention or Exposure	Time	Result
Abiri (Abiri et al., 2016)	Vitamin D	Seventy one women randomized to daily intake of vitamin D (1000 IU) (n = 37) or placebo (n = 34).	Aged 40–55 y For 3 months	Vitamin D supplementation led to improvement of muscle functions and body composition, but had no significant impact on muscle strength.
Marantes (Marantes et al., 2011)	Vitamin D	Examined the relationship of skeletal muscle mass and strength with each of 25(OH)D, 1,25$(OH)_2$D, and PTH quartiles, in 311 men and 356 women.	Men: mean age, 56 y; range, 23–91 y Women: mean age, 57 y; range, 21–97 y	There was no consistent relationship between 25(OH)D or PTH and any of measurements of muscle mass or strength, in both men and women. But, in those who are younger than 65, there was a positive significant relationship between 1,25$(OH)_2$D levels and skeletal mass in both men and women.
Scott (Scott et al., 2010)	Vitamin D	Appendicular lean mass percentage (%ALM), body fat, leg strength, leg muscle quality (LMQ), physical activity (PA), and serum 25(OH)D were measured in 686 older adults.	62 ± 7 years old Mean follow up of 2.6 ± 0.4 years	Subjects with 25(OH)D ≤ 50 nM had lower mean %ALM, leg strength, LMQ and PA.
Visser (Visser, Deeg, and Lips, 2003)	Vitamin D	In men and women of the Longitudinal Aging Study Amsterdam, grip strength (n = 1080) and appendicular skeletal muscle mass (n = 331) were meseared.	Aged 65 y In 1995–1996 after a 3-yr follow-up	Lower 25(OH)D and higher PTH concentrations increased the risk of sarcopenia in older men and women.
Bartali (Bartali et al., 2008)	Antioxidant micronutrients	Longitudinal survey of 698 community-dwelling persons. Serum micronutrient concentrations and physical function were assessed.	65 y or older Follow up from 2001–2003	Low serum level of vitamin E is related to subsequent decrease in physical function.
Lauretani (Lauretani et al., 2007)	Antioxidant micronutrient (selenium)	Plasma selenium and hip, grip, and knee strength in a cross-sectional survey of 891 men and women from InCHIANTI Study were measured.	65 y or older	Low plasma selenium was related to poor skeletal muscle strength.
Kelaiditi (Kelaiditi et al., 2012)	Antioxidant micronutrients	Nutrient intake and body composition were measured among 2570 women.	Aged 18–79 y	Differences for fat free mass index were significant for vitamin C, magnesium, potassium, and total carotene intakes, but not significant for vitamin E and selenium.
Bobeuf (Bobeuf et al., 2011)	Antioxidant micronutrients (vitamins E/C)	Fifty-seven men and women randomized into four groups: control-placebo; resistance training (RT); vitamins E/C supplementation (AS); AS+RT.	Mean age 65 ± 3.8 y For 6 months	A significant impact on body composition was shown, but no difference was shown on strength gain, in AS+RT.
Dominguez (Dominguez et al., 2006)	Mineral (magnesium)	Muscle performance and serum magnesium were measured in 1138 men and women of InCHIANTI study.	Aged 66.7 ± 15.2 y Between 1998–2000	Serum magnesium levels were significantly related to muscle performance.
Moslehi (Moslehi et al., 2013)	Mineral (magnesium)	Randomized into daily intake of magnesium supplement (250 mg) or placebo.	Aged 40–55 y For 8 weeks	There was significant increase in lean body mass only in the intervention group. Handgrip strength and muscle function ameliorated in the intervention group. There were not significant differences in elevating knee extension strength between two groups at the end of study.
Dawson-Hughes (Dawson-Hughes, Harris, and Ceglia, 2008)	Dietary acid-base load	Potassium in 24-h urine collections, lean body mass percentage, and physical activity were assessed in 384 men and women.	Aged ≥ 65 y 3-y trial	Higher consumption of foods rich in potassium may conserve muscle mass.

suggests that the threshold concentration of circulating amino acids are needed to make an anabolic response in skeletal muscle, and also elevating concentrations of leucine are needed to maintain strong anabolic responses (Dillon 2013).

Cross-sectional studies relating protein intake and muscle mass have found conflicting associations (Vafa et al. 2017; Baumgartner et al. 1999; Scott et al. 2010; Meng et al. 2009; Mitchell et al. 2002; Houston et al. 2008; Stookey, Adair, and Popkin 2005). Protein intake was not related to muscle mass in older adults in two studies (Baumgartner et al. 1999; Mitchell et al. 2002), but positively associated in another study (Scott et al. 2010); also at follow-up it was related to alteration in

appendicular lean mass during 2.5-year period (Scott et al. 2010). Lower percentage protein intake was related to greater loss of mid-arm muscle area during 4 years, in Chinese population aged 50–69 years (Stookey, Adair, and Popkin 2005). Moreover, in men and women aged 70–79 years, energy adjusted protein intake was related to 3-year alterations in lean mass (Houston et al. 2008). In compared with the lowest percentage protein intake, those with a higher had a lower rate of loss of lean mass and appendicular lean mass, by ~ 40% (Houston et al. 2008). In Australian women aged 72–78 years lean mass, appendicular lean mass and upper arm muscle area were all significantly and positively related to protein intake (Meng et al. 2009). In our recent study, among women aged 40–60 years old, protein intake, adjusted for physical activity and weight, was positively and significantly associated with fat free mass percentage (Vafa et al. 2017).

A substantial number of intervention trials with protein, whey, casein, and mixed or individual amino acids have been carried out. Of the studies of amino acid or protein supplementation alone, in interventions with essential amino acids, two studies demonstrate elevations in lean body mass over the intervention period (Dillon 2013; Fukagawa 2013; Malafarina et al. 2013; Dillon et al. 2009; Børsheim et al. 2008). In general, the response to protein and amino acid supplementation (with or without exercise) has not been constant and this may be resulted from differences in nutritional status at baseline, varying methods of evaluating outcomes, and geographical and racial variations (Malafarina et al. 2013; Casperson et al. 2012). Supplementation with protein have not always increased the impacts of resistance training (Candow et al. 2012; Kukuljan et al. 2009). In a meta-analysis of twenty-two randomized controlled trials of protein supplementation studies (in the form of protein, whey, casein, and mixed essential or single amino acids) coexist with resistance exercise, protein supplementation had an overall positive influence on fat free mass and leg strength (Cermak et al. 2012).

The research to date from both cross-sectional and intervention trial studies demonstrates that adequate protein intake with high quality is essential for muscle mass, and leucine is the most effective amino acid. While, another nutritional factors may also affect muscle, similar to protein, have been less investigated.

Vitamin D

The mechanisms that describe the association of vitamin D with muscle mass and strength may be direct or indirect through calcium controlling and signaling and accumulation in the sarcoplasmic reticulum or through the activation of vitamin D receptors in muscle (Ceglia and Harris 2013). Vitamin D deficiency results to atrophy of type II muscle fibers (Ceglia and Harris 2013). However, one study has not found vitamin D receptors in human skeletal muscle, showing the influence of vitamin D on muscle may be indirect (Wang and DeLuca 2010). Another study indicated that vitamin D receptor polymorphisms in muscle were related to lower fat free mass in older adults (Scott et al. 2011). In a number of studies, vitamin D status has also been related to muscle strength, in fact better vitamin D status related to greater muscle strength (Ceglia and Harris 2013). Some intervention studies demonstrated a positive impact of vitamin D supplementation on type IIa or II muscle fibers (Ceglia and Harris 2013). In our study vitamin D supplementation (1000 IU, daily, for 3 months) in vitamin D-deficient middle-aged women (40–55 yr) resulted to improvement in muscle function in intervention group compared to the placebo group, and also fat mass percentage significantly reduced in vitamin D group at the end of intervention, but alterations did not reach significant compared with the placebo group. In both groups muscle strength did not differ significantly at the end of the intervention (Abiri et al. 2016), that maybe explained by some reasons: it is possible that lack of significant improvement in muscle strength and mass in the vitamin D group in compared to the placebo may be due to insufficient dose of vitamin D supplementation in vitamin D-deficient women, short period of vitamin D supplementation, or the combination of both factors. In addition, baseline vitamin D status or baseline muscle strength or mass might have impacts on the response to vitamin D supplementation. Moreover, some cross-sectional studies found a positive relationship between vitamin D status and muscle mass in men and women (Szulc et al. 2004; Marantes et al. 2011; Scott et al. 2010; Visser, Deeg, and Lips 2003), although in one of these studies the relationship was shown only in women below the age of 65 years; not in men or older women (Marantes et al. 2011). Another cross-sectional study did not show a relationship between vitamin D status and muscle area, but this study found that infiltration of fat into muscle was more considerable in those with lower vitamin D status (plasma 25(OH)-D $\leq$ 29 ng/ml) (Gilsanz et al. 2010).

In spite of the fact that vitamin D probably affect muscle, further research is required to establish the extent to which vitamin D can impact on muscle.

Dietary fat

The fatty acids obtained from dietary fat are the important source of energy for muscle and fats are vital elements of myocellular membranes (Corcoran, Lamon-Fava, and Fielding 2007). Dietary fat composition also impacts on inflammation and insulin resistance, because it is associating with loss of fat free mass content (Corcoran, Lamon-Fava, and Fielding 2007).

In vitro studies have shown eicosapentaenoic acid (EPA) elevates fatty acid oxidation in myotubules (Kalupahana, Claycombe, and Moustaid-Moussa 2011; Wensaas et al. 2009). Fatty acid could also influence membrane fluidity and the situation of proteins, and levels of muscle ceramide, diacylglycerol, Triacylglycerol (TAG) and acylcarnitines (Corcoran, Lamon-Fava, and Fielding 2007).

Because inflammation is related to loss of muscle mass in older adults and since dietary fat composition can impact on inflammation, so dietary fat may impact on muscle loss (saturated and trans-fatty acids are pro-inflammatory, and the n-3 and n-6 fatty acids anti-inflammatory) (Kalupahana, Claycombe, and Moustaid-Moussa 2011; Wensaas et al. 2009).

Moreover, total dietary fat have an impact on muscle mass via some mechanisms, such as reduced hepatic and skeletal muscle oxidation ability and also by elevating the availability of fatty acids via skeletal muscle for oxidation (Corcoran, Lamon-

Fava, and Fielding 2007). In animal models, a high fat diet resulted to damaged protein turnover and muscle hypertrophy, by decreased activation of Akt and S6K1 (kinases in mTOR pathway) (Sitnick, Bodine, and Rutledge 2009).

Some human studies have demonstrated that supplementation with n-3 fatty acid enhances rates of protein synthesis and elevates the muscle protein anabolic response, and also decrease muscle loss (Kumar et al. 2010; Magee, Pearson, and Allen 2008; Smith et al. 2011; Smith et al. 2011; Khal and Tisdale 2008; Fiaccavento et al. 2010). One observational study demonstrated a negative relationship between appendicular lean mass and saturated fat. Another study of dietary fat composition and muscle mass showed positive relationships between muscle mass with PUFA:SAFA ratio and negative relationships with saturated and trans- fatty acids (Scott et al. 2010; Welch, MacGregor, and Minnihane 2014).

Antioxidant micronutrients

Oxidative stress and the accumulation of reactive oxygen species (ROS) lead to age- related muscle loss; hence antioxidant nutrient intakes may decrease oxidation in muscle (Liochev 2013). While, the utilization of dietary antioxidants with the aim of decreasing oxidative harm in muscle may not be fully beneficial, because although antioxidants may decrease oxidative harms in mitochondria they also decrease the redox signaling, started by ROS, needed for muscle contraction (Jackson 2009).

Antioxidant nutrients include of vitamins C, E, and carotenoids and the trace elements: Cu, Mn, Se, and Zn. In this regard few intervention and observational studies are available that have mainly assessed the association between skeletal muscle mass and function with vitamins C and E. Skeletal muscle is an important body supply of vitamin C, suggested to preserve up to 67% of whole body vitamin C. Thus, vitamin C is probably to be vital for muscle construction and function because of its role as an antioxidant and also enzyme cofactor in collagen and carnitine synthesis (Carr et al. 2013). An intervention study demonstrated that skeletal muscle responds to elevated vitamin C intake and its uptake in muscle is greater than leucocytes (Carr et al. 2013).

Some studies have linked decrease in physical function or weakness to serum micronutrient levels of vitamin E or carotenoids or Se. Also, low consumption of fruit and vegetables has been related to functional limitations in middle-life and aged men and women (Bartali et al. 2008; Semba et al. 2006; Tomey et al. 2008; Myint et al. 2007; Houston et al. 2005; Lauretani et al. 2007). One study among women aged 18–79 years indicated a positive association between total carotene and vitamin C intake and fat free mass index but no relationship with vitamin E and Se (Kelaiditi et al. 2012). Another cross-sectional study demonstrated that in aged women higher antioxidant consumption was related to specific features of physical function such as walking and chair rise time (Martin et al. 2011). One longitudinal study indicated a preservation association between vitamin C intake and loss of muscle mass in aged men and women during 2.6 years (Scott et al. 2010). Intervention studies in human have differed in methodology. One study found daily supplementation with vitamin E (600 mg) and vitamin C (1000 mg) combined with resistance training ameliorated fat free mass content more than resistance training alone (Bobeuf et al. 2011; Labonté et al. 2008). In rats supplementation with vitamins A and E, Zn and Se ameliorated the anabolic response to the amino acid leucine, and also decreased inflammation, but the beneficial impacts of antioxidants on the leucine response are mainly resulted from a systemic reduction in oxidative stress and inflammation (Mosoni et al. 2010; Marzani et al. 2008).

The limited investigations to date propose that antioxidant nutrients would be useful, and in association with prevention of muscle loss, further studies are required.

Minerals

Magnesium condition has impacts on muscle function and performance, due to its roles in energy metabolism and membrane transport and 27% of total body magnesium is reserved in skeletal muscle (Corcoran, Lamon-Fava, and Fielding 2007). Also, magnesium is contributed in modulation of muscle contraction and relaxation, oxygen uptake, anabolic hormones secretion (Maggio et al. 2011; Cinar et al. 2011) and inflammation (Moslehi et al. 2012); the known causes of decreasing muscle strength (Borst 2004; Schaap et al. 2009). Thus, it is probable that magnesium may have indirect effects on muscle strength and muscle mass via its impacts on inflammation and anabolic hormone. Low levels of serum magnesium have been related to lower muscle strength but the role of magnesium has been found to be positively associated with muscle mass in some studies (Scott et al. 2010; Kelaiditi et al. 2012; Dominguez et al. 2006). One intervention study demonstrated that magnesium supplementation (250 mg, daily, for 8 weeks) in middle-aged overweight women, resulted to significant increase in lean body mass coexist with a significant decrease in fat mass only in the intervention group at the end of week 8 compared to the baseline values, but the alterations were not significant in compared to placebo group. Handgrip strength and muscle function ameliorated in the intervention group compared to baseline but they were not significant compared to the placebo group. There were not significant differences in elevating knee extension strength between two groups at the end of study (Moslehi et al. 2013). Lack of a significant improvement in muscle strength and function in the magnesium group in compared to the placebo may be due to insufficient dose of magnesium, poor bioavailability of magnesium oxide, or short duration of magnesium supplementation (Moslehi et al. 2013). Also, differences in baseline magnesium conditions or baseline muscle strength might have impacts on the response to magnesium supplementation (Moslehi et al. 2013). Over a 2.6-year follow up study, magnesium was a positive predictor of alteration in appendicular lean mass, as was iron, phosphorus and zinc, demonstrating a role of minerals in preservation of lean mass, and better mineral intake was related to less muscle loss over time (Scott et al. 2010). However, more research is required in this area.

Bioactive components

There is evidence demonstrating certain bioactive components may affect age-related skeletal muscle loss. In old

rodents, olive oil derived antioxidant blend was beneficial in restoring some sarcolemma ion channels in muscle (Pierno et al. 2014). Circumin (a phenol component found in turmeric) has been investigated as a supplement, in association with contractile function in, *in vitro* studies, although the results have been unclear (Corcoran, Lamon-Fava, and Fielding 2007). Bioactive components that affect muscle function and strength may also be useful for prevention of age-related muscle loss and so further investigation is required in this field.

Dietary acid-base load

Metabolic acidosis is one of the important causes of muscle loss in chronic kidney disease, and the mild metabolic acidosis may also be connected to skeletal muscle loss (Mitch 1995; Workeneh et al. 2006; Welch et al. 2013).

Metabolic acidosis is related to a more acidic blood acid-base makeup and, while the acid-base balance is conserved within narrow limits, the pH in blood becomes more acidic with advancing age (Frassetto, Morris, and Sebastian 1996), because keeping of blood acid-base system is relying on excretion of H^+ ions via the urine, and since renal function progressively decreases with ageing there is potential for mild metabolic acidosis to elevate (Frassetto, Morris, and Sebastian 1996).

Diet has the capacity to create mild metabolic acidosis in population by consumption of potentially acidogenic and alkalinogenic foods. Acidogenic foods are protein containing foods that contain amino acids cycteine and methionine which produce hydrogen ions that reduce blood pH (Welch et al. 2008; Remer and Manz 1995; Frassetto et al. 1998; Buclin et al. 2001; Wynn et al. 2009). The important acidogenic foods are meats, fish, eggs, cereals and dairy foods and these foods have been positively related to a more acidic urine pH in population investigations (Welch et al. 2008; Remer and Manz 1995; Frassetto et al. 1998; Buclin et al. 2001; Wynn et al. 2009). The alkalinogenic foods are fruits and vegetables. These foods balance the H^+ ions produced by the metabolism of acidogenic foods, because of the carbonate compounds of them (Welch et al. 2008; Remer and Manz 1995; Frassetto et al. 1998; Buclin et al. 2001; Wynn et al. 2009).

Metabolic acidosis promotes proteolysis and amino acid catabolism, likely via activation of caspase-3 and the ubiquitin proteasome system or by the effects on the growth hormone/insulin-like growth factor-1 (GH/IGF-1) axis (Corcoran, Lamon-Fava, and Fielding 2007).

The association between supplementation with bicarbonate components, to decrease metabolic acidosis, has been evaluated in some studies of middle- and older aged populations and two of these studies demonstrated decrease in nitrogen excretion, proposing this might be occurred by IGF-1 (Frassetto, Morris Jr, and Sebastian 1997; Dawson-Hughes, Harris, and Ceglia 2008; Dawson-Hughes et al. 2010; Ceglia et al. 2009; Dawson-Hughes et al. 2009). A decrease in nitrogen excretion shows more nitrogen has been kept for the anabolic processes in muscle. Two intervention trials were planned to understand the impact of decreasing the metabolic acidosis of renal disease, by use of sodium bicarbonate, demonstrated enhancements in nitrogen equilibrium and serum albumin as well as mid-arm muscle circumference after 2 years supplementation (Papadoyannakis, Stefanidis, and McGeown 1984; de Brito-Ashurst I, Raftery, and Yaqoob 2009).

In one study an alkaline diet was positively associated with muscle mass and alteration in lean body mass during 3 years (Dawson-Hughes, Harris, and Ceglia 2008). In another study, an alkalinogenic diet was positively associated with muscle mass and the proportion of fruits and vegetables to alkaline foods such as meat, fish, eggs and dairy (Welch et al. 2013).

These results show that fruits and vegetables as well as protein are essential for muscle preservation; however, more intervention studies in populations are needed in this field.

A rodent study planned to understand the interaction between vitamin D and alkaline condition on the loss of muscle mass, demonstrated an interaction between vitamin D and alkaline condition (Ceglia et al. 2013). Vitamin D condition alters the impact of potassium bicarbonate supplementation on muscle mass after 12 weeks (Ceglia et al. 2013). It has been proposed that alkali supplementation results to enhancing muscle but this impact was increased when vitamin D condition was sufficient (Ceglia et al. 2013). For better understanding future research in this area is required.

Nutrients and dietary patterns

One complication in the existing evidence is that dietary compounds are often related to each other. This may explain why the impacts of supplementation with single nutrients may be less than that anticipated by the observational evidence. It also suggests that in observational studies it may be difficult to know the relative significance of the impacts of different nutrients on sarcopenia. Therefore, because diets are patterned high fruit and vegetable intake may be markers of another dietary impact which may be significant for muscle function, similar to greater intake of oily fish, vitamin D and n-3 long chain fatty acids (Robinson et al. 2009).

In compared with the data that connects differences in nutrient intake and condition to physical function, less is known about the impacts of dietary patterns and diet quality in old ages. Healthy diets, described by the greater fruit and vegetable intake, whole cereals, and oily fish, have been demonstrated to be related to higher muscle strength in older adults (Robinson et al. 2008). Also, among women aged 42–52 years, unhealthy diets, described by higher saturated fat intakes and low fruit and vegetable consumption, were related to higher functional impairments during 4-year follow up (Tomey et al. 2008). Advantages of healthier diets and higher fruit and vegetable consumption on physical function in mid-life have also been demonstrated in women in the Whitehall study (Stafford et al. 1998), and in men and women in the Atherosclerosis Risk in Communities study (Houston et al. 2005). Intervention studies with "whole diet" approach are likely to alter intakes of a range of nutrients and, hence, have the potential in preventing age-related declines in muscle mass and strength.

Lifelong nutrition

A major problem to the current data that connects nutrition to sarcopenia is that much of them are from cross-sectional

studies, this may limit our knowledge of the importance of the role of nutrition in the muscle mass and function decline with advancing age.

Firstly, the health of elderly population is affected by events during their lives (Kaiser, Bandinelli, and Lunenfeld 2010), and hence, attainment to optimal performance relies on lifelong exposure to a healthy diet and lifestyle. The impact of lifelong nutrition on age-related alterations in muscle mass and function has been little investigated, and the effects of interventions to retard or prevent sarcopenia in older adults are needed to further investigation.

Second, muscle mass and strength attained in later life are not only estimated by the rate of muscle loss, but also depends on the peak achieved in early life (Sayer et al. 2008). Therefore, factors that impact growth, such as differences in early nutrition, may have effect on muscle mass and strength in older ages.

A key notice, that highlights the importance of life cycle impacts, is that low weight at birth forecasts lower muscle mass and strength in adult life (Sayer 2010).

Few studies have investigated the effects of diet in early childhood in the gain of muscle mass and impacts on later function, although there is some data that shows its importance. For example, the risk of weakness has been indicated to be higher in older adults who grew up in poverty conditions, and who were undergone hunger in childhood (Alvarado et al. 2008). While, animal studies demonstrate that nutrition in early life may be key, because muscle growth in neonatal time is sensitive to differences in nutrient intake (Davis and Fiorotto 2009).

At the present time we know little about the role of nutrition during the life course in muscle mass and function in adult life, and further research is required to get how early nutrition impacts on the attain of peak muscle mass, and its role in the age-related losses in muscle performance. Considering a lifespan approach to understanding the connections between nutrition and muscle mass and function in old ages could alter dietary strategies to prevent sarcopania in later life.

Another candidate: Calorie restriction

Calorie restriction (CR), which typically is consuming 20–40% fewer calories than normal diet, conserve mitochondrial health and reduces sarcopenia. CR is accepted as the strongest intervention that delay both primary ageing (natural age-related impairments) and secondary ageing (early ageing resulted from disease and negative lifestyle behaviors), and promoting lifespan in many species. Studies in rodent have constantly indicated that CR expands maximum lifespan up to 50% and decreases the occurrence of many age-related diseases. These protective impacts are likely resulted from the ability of CR to decrease the mitochondrial disorders (mitochondrial proton leakage) and reduce oxidative stress (Hancock et al. 2011). One study found that CR conserves mitochondrial function via keeping the integrity and function of existing cellular compounds. Moreover, CR appears to prevent the age-associated increases in pro-apoptotic signaling in skeletal muscle (Dirks and Leeuwenburgh 2004). Considerably, CR has been demonstrated to modulate the pro-apoptotic pathways included in age-related skeletal muscle decline, such as mitochondrion-, cytokine/receptor- mediated signaling (Dirks and Leeuwenburgh 2004). In addition, CR combined with exercise is suggested to counteract the apoptosis related to sarcopenia more effectively.

Many studies have shown that PGC-1α is elevated with CR in different organs such as brain, liver, heart, and brown and visceral adipose tissue (Gouspillou and Hepple 2013). Barker *et al.* indicated a significant promote in PGC-1α in gastrocnemius muscle of rats after a 40% CR starting at 16 weeks of age (Baker et al. 2006). On the other hand, PGC-1α has different roles, such as in fatty acid oxidation, myokine secretion, autophagy activation, and up-regulation of mitochondrial genesis (Chan and Arany 2014). Valdez *et al.* (Valdez et al. 2010) indicated lifelong CR significantly reduced the occurrence of pre- and postsynaptic impairments in 24 month old mice and the age-associated loss of motor neurons probability due to PGC-1α production. Because the level of basal autophagy in muscle has been demonstrated to be decreased with ageing (Wohlgemuth et al. 2010; Sakuma, Aoi, and Yamaguchi 2014), normal performance of autophagy due to CR may decrease the muscle fiber atrophy with advancing age.

One study demonstrated that CR has no useful impact on health and longevity in rhesus monkeys (Mattison et al. 2012), opposite to many findings from studies applying the same species (McKiernan et al. 2011; McKiernan et al. 2012). Further investigations are required to estimate whether CR is beneficial in decreasing the age-associated muscle loss in human subjects and to what extent of dietary intervention can be used in human populations. Because excessive CR (over 50%) may have many side effects, such as frailty, osteoporosis, depression, and anorexia nervosa. Mild CR should be used in the elderly population.

Diet and exercise training

Resistance exercise has been shown to be beneficial in promoting muscle strength and physical function in older adults (Liu and Latham 2009). The synergic impacts of diet and exercise on physical function have been investigated mainly in relation to protein/amino acid supplementation. For example, while the intake of a high protein meal has led to promote muscle synthesis in older adults about 50%, combination of a high protein meal with resistance exercise promote synthesis more than 100% (Symonsi et al. 2011). Although, some studies in older adults have not shown further advantages of protein/amino acid supplementation on the skeletal muscle response to resistance exercise training (Koopman 2011; Kim, Wilson, and Lee 2010), and thus, the implications for long-term impacts of combination of exercise and high protein intakes are not understood clearly (Paddon-Jones and Rasmussen 2009). As yet, little is known about the combined impacts of vitamin D supplementation and resistance exercise on muscle strength and performance (Bunout et al. 2006). Further investigation is required in this area, especially to show the influences of different quantity and timing of supplementation and exercise.

Conclusion

To emerge strategies to counteract or retard sarcopenia, a better understanding of the lifestyle factors that impact on the rate of muscle mass and function loss in older age, and also their

mechanisms is required. Current data demonstrates the importance of sufficient quality and quantity of diets. The high prevalence of low nutrient intakes among elderly population made this a present concern. While, muscle mass and strength attained in old ages are not only estimated by the rate of muscle decline, but also depend on the peak achieved in early life, so attempts to prevent and delay sarcopenia also require to understand the potential effectiveness of interventions in the lifespan. It means optimizing diet during the life may be a key strategy to prevent sarcopenia in older age. Because the elderly population has had an elevating trend during the last century hence, future work in this area should be design in younger as well as older populations.

Conflict of interest

None conflict of interest

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