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REVIEW

The role of exercise, milk, dairy foods and constituent proteins on the prevention and management of sarcopenia

LYDIA AM COOPER, SARAH L BROWN, ERICA HOCKING and ANNE C MULLEN*

The Dairy Council, 210 High Holborn, WC1V 7EP, London, UK

Sarcopenia, an age-related decline in muscle mass, is a burgeoning public health concern in the UK, with the number of people over the age of 65 expected to double by 2050. Resistance exercise is an effective intervention in its prevention and management. Increasing quantity and improving quality of dietary protein, by inclusion of high-availability leucine, are also purportedly beneficial. Leucine is a key anabolic amino acid, found in dairy foods. A number of studies have investigated dairy foods in prevention of sarcopenia. This paper reviews interventions of exercise, amino acids including leucine, dairy protein and foods for prevention of sarcopenia.

Keywords Nutrition, Age, Amino Acids, Health.

INTRODUCTION

While obesity remains to the fore of public health nutrition in the United Kingdom, malnutrition is of increasing concern. It currently affects over 10% of people aged 65 and over 16% of those aged 85 in the community, costing the economy £7.3bn per year (ENHA, 2006). The number of people aged 65 and over is expected to rise by half in the next two decades to over 16 million and age-related malnutrition is a burgeoning public health concern. This review summarises the aetiology and prevalence of age-related muscle degeneration, known as 'sarcopenia'.

The main aim of this review paper was to summarise current evidence on lifestyle interventions in the mitigation of sarcopenia. Studies on the role of exercise in preventing sarcopenia are presented. In terms of nutrition, we specifically reviewed studies on the role of animal and dairy amino acids, proteins and food products, which may have a specific role to play in mitigating sarcopenia (Norton and Jakeman 2013;).

SARCOPENIA: DEFINITION, AETIOLOGY AND PREVALENCE

The term sarcopenia was coined by Rosenberg (1989; from Greek *sarx* (flesh) and *penia* (loss)) to describe the decline in muscle mass associated with ageing. In 2009, the International Sarcopenia Consensus Conference Working Group described sarcopenia as an age-related loss of skeletal muscle mass, with or without an increase in fat mass (Fielding *et al.* 2011). Key mechanisms in the aetiology of sarcopenia are presented in Figure 1 (Batsis and Buscemi 2011). Muscle mass declines due to a reduction in the quality and size of muscle fibres (Lexell *et al.* 1983), by approximately 30% to 50% between the ages of 20 and 80 years; yearly deterioration of muscle mass is estimated at around 1% to 2% from the ages of 50 to 80 years and as high as 3% thereafter (Frontera *et al.* 2000; Faulkner *et al.* 2007). Reduced physical activity is a major aetiological driver and occurs for a variety of reasons including illness, disability, environment and infrastructure. Lack of regular muscle use increases muscle loss in older people (Suetta *et al.* 2009). Furthermore, poor nutritional intake, particularly of protein and energy, plays a role in the risk of developing sarcopenia (Robinson *et al.* 2012; Rom *et al.* 2012). Low protein consumption has been associated with a reduction in lean muscle mass and a decline in muscle function, and increasing protein intake in older people to reduce their risk of developing sarcopenia is

*Author for correspondence.
E-mail: a.mullen@dairycouncil.org.uk

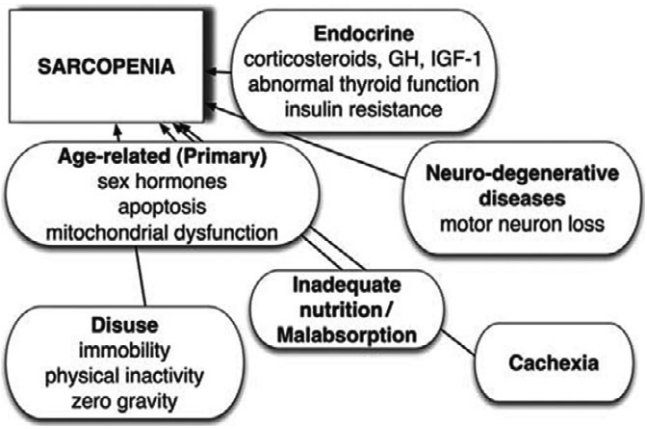

Figure 1 Mechanisms of sarcopenia; reproduced from Cruz-Jentoft *et al.* (2010) and Batsis and Buscemi (2011).

advocated (Wolfe *et al.* 2008). In general, as people get older, they eat less – around 25% less from the age of 40 to 70 years. This is attributable to a variety of factors: as people age, they eat more slowly, have smaller appetites, eat smaller meals and have fewer snacks between meals (Nieuwenhuizen *et al.* 2010). Therefore, it is important that older people consume high-quality, energy- and nutrient-dense food. A combination of immediate (e.g. nutrient intake and physical health), underlying (e.g. food security, mobility, society, technology and environment) and basic (e.g. infrastructure, economy, welfare) factors mean it can become easy for older people to fall into a vicious cycle of poor nutrition and declining muscle mass and strength (Bartali *et al.* 2003). In addition to reduced physical activity and poor nutritional intake, hormones, inflammatory mediators, neuronal integrity, cachexia, increased body fatness, mitochondrial dysfunction, appetite regulation, chronic disease, enforced inactivity, hospitalisation and depression feature in aetiological models of sarcopenia (Cruz-Jentoft *et al.* 2010; Batsis and Buscemi 2011; Fielding *et al.* 2011).

Data relating to the prevalence of sarcopenia varies, but the condition is reported to affect 5%–13% of those aged 60–70 years old and up to 50% of adults over the age of 80 from European and American cohorts (von Haehling *et al.* 2010). The Hertfordshire Cohort Study reported that sarcopenia prevalence in men and women from a cohort with an average age of 67 years was about 5% and 8%, respectively (Patel *et al.* 2013), in line with international data cited above. The lack of precision in prevalence data is partly due to a lack of consensus on diagnosis and also suboptimal clinical tools for diagnosis. The most accurate way to assess body composition is the DEXA scan; this method is very costly and not widely available to practitioners. Sarcopenia is more than body compositional changes; it becomes problematic when functionality of muscle, and not just mass, is lost. The European Working Group on Sarcopenia in Older People, therefore, developed practical guidelines for the diagnosis of sarcopenia on the existence of low muscle mass combined with either low muscle strength or low physical performance (Cruz-Jentoft *et al.* 2010). The group also recommends the method be applied to identify cases of hypothetical 'presarcopenia', sarcopenia and 'severe sarcopenia'.

SARCOPENIA AND OBESITY

Sarcopenia may occur with or without increased fat mass (Prado *et al.* 2012). The co-existence, and in fact potential for shared aetiology, of sarcopenia and obesity is of significant molecular, clinical and public health interest. Key features of the relationship are outlined in Figure 2 (Zamboni *et al.* 2008). A low-grade, chronic systemic inflammation is believed to underpin the metabolic syndrome, with

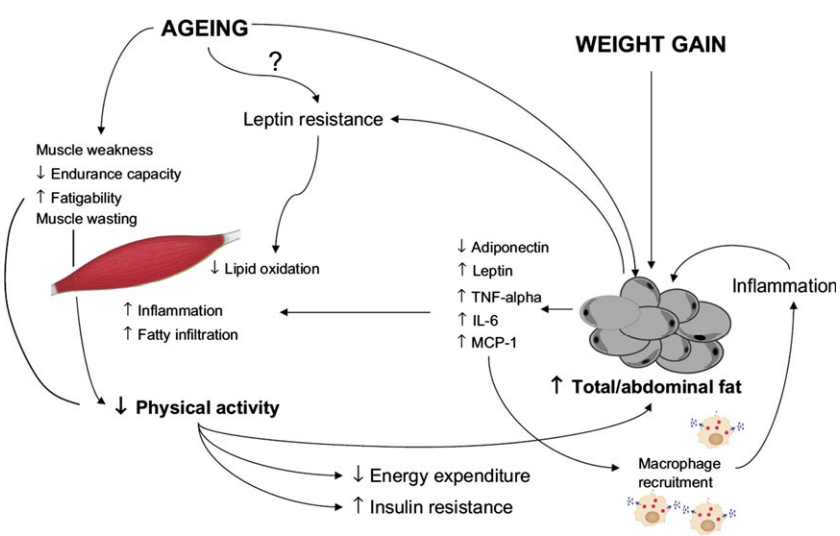

Figure 2 Mechanisms of sarcopenic obesity; reproduced from Zamboni *et al.* (2008).

inflammatory infiltration of expanding adipose tissue mediating systemic inflammation, insulin resistance and ectopic fat accumulation in muscle and visceral tissue (Wellen and Hotamisligil 2003; Roche *et al.* 2005). Sarcopenic obesity or 'sarcobesity' carries even greater health risks than either condition alone (Rolland *et al.* 2009; Stenholm *et al.* 2009). An ageing population combined with the pervasive problem of obesity has caused an inevitable increase in sarcopenic obesity prevalence (Roubenoff 2004). A review by Stenholm *et al.* (2008) presented prevalence of sarcopenic obesity at just over 4% of men and 3% of women among 831 participates aged 60 and over in the New Mexico Aging Process Study, at almost 10% of men (of 1391 subjects with mean age of 76 years) and over 7% of women (of 1591 subjects with mean age of 77 years) in the USA, and at just over 12% among 167 Italian women with an average age of 72 years. The criteria used for the definition of sarcopenia (skeletal muscle mass less than 2SD from mean of young population in the New Mexico cohort; within two lower quintiles of muscle mass in both the US and Italian cohorts) and the definition of obesity (body fat percentage greater than median in the New Mexico cohort; within two highest quintiles of fat mass in both the US and Italian cohorts) varied between studies.

PREVENTION AND MANAGEMENT OF SARCOPENIA

Resistance exercise & muscle maintenance

It is well established that resistance exercise augments muscle protein synthesis (Koopman and van Loon 2009). Several studies have reported that even a single bout of resistance exercise increases muscle protein synthesis in both young and older individuals (Sheffield-Moore *et al.* 2004; Kumar *et al.* 2009; Symons *et al.* 2011), if perhaps delayed in older individuals (Dionne *et al.* 2004; Drummond *et al.* 2008), and with no difference between genders (Leenders *et al.* 2013).

Holviala *et al.* (2014) remarked that the majority of studies reporting positive effects of resistance training on muscle gain in the elderly were 6 months in duration or shorter. They investigated whether one session of strength training per week for 21 weeks would be sufficient to maintain muscle gains achieved from a prior 21-week intervention of twice weekly strength training. A group of 72 women and 63 men with a mean age of 58 years were randomised to strength training or control. Those in the strength-training groups were re-randomised in the second 21-week period to carry out either one or two sessions of strength training per week. While the strength-training groups increased their strength during the first 21-week period, only the women training at a frequency of twice weekly maintained their strength through to week 52. Men training twice weekly also maintained strength gained; however, this was only up until 32 weeks. A similar study was carried out by Bickel *et al.* (2011) testing two muscle and strength maintenance programmes in young (20 to 35 years) and older adults (60 to 75 years). Phase 1 involved knee extensor training 3 days per week for 16 weeks. Following this, phase 2 saw the participants randomised into three groups; group 1 performed no further training, group 2 performed the equivalent of one-third of the original training load, and group 3 performed the equivalent of one-ninth. All participants experienced gains in muscle mass and strength after phase 1. Both maintenance programmes either preserved or improved muscle mass and strength in the young adults, but not in their older counterparts. Authors concluded that older adults require a higher weekly training load to maintain muscle and strength gains from a previous study. A study randomised older women (aged 60 to 80 years) with previous experience of resistance training, but not within the previous 3 months, to perform 24 sessions of either single or three sets of 8 to 12 repeat sit-to-stand exercises (Abrahin *et al.* 2014). Both groups significantly improved skeletal muscle strength with no difference observed between groups. It was concluded that older women who struggle to find the time or interest in physical activity should be advised to begin with low frequency resistance exercise.

Age-associated muscle loss is largely due to atrophy of Type II muscle fibres (Lexell *et al.* 1988; Dreyer *et al.* 2006). Furthermore, satellite cells exist in similar quantities in Type I and II muscle fibres in younger adults, but this is not the case in older people (Kadi *et al.* 2006; Verdijk *et al.* 2007). A small study investigating the effects of a 12-week resistance exercise programme on muscle fibres in 13 healthy elderly men (aged 65 to 85 years old) noted that at baseline, the total number of Type II muscle fibres was much less than Type I muscle fibres (Verdijk *et al.* 2009). Results of the intervention showed a significant increase in muscle strength, lean leg mass, reduction in whole-body fat mass and fat percentage. Whole-body lean mass increased but not significantly. Also, interestingly, a 28% increase in Type II muscle fibres was observed. Researchers concluded that a 12-week resistance exercise intervention is a successful method of improving strength and muscle mass, and subsequently reversing the effects of muscle atrophy and sarcopenia in older men.

Both ageing and reduced physical activity are factors in the aetiology of sarcopenia, but their relative contribution and relationship are unclear. Studies have shown myofibres in older men respond similarly to the same resistance exercise training as younger men (Häkkinen *et al.* 1998). A 'use it or lose it' hypothesis exists. Kumar *et al.* (2009) and Hunter *et al.* (2004) recommend that older adults should train twice a week, as well as once-a-week low-velocity exercise for strength building and maintenance, but for individuals already suffering from sarcopenia, a specific plan should be devised.

Exercise in sarcopenic obesity

Obesity brings with it a plethora of adverse health risks including diabetes and coronary heart disease, and thus is associated with increased mortality and morbidity. However, weight reduction in the obese elderly has shown clinically important effects on these diseases (Bales and Buhr 2008). Here, we consider exercise in the sarcopenic obese adult. While resistance exercise is known to be effective for muscle gain and physical function in the sarcopenic elderly, sarcopenic obese individuals have the issue of excess body fat in addition to poor muscle mass. A widely accepted and utilised strategy for weight reduction in obesity is aerobic exercises; however, this does not address the factor of low muscle mass and can in fact cause muscle loss, further contributing to sarcopenia (Benton *et al.* 2011).

There is evidence that a combination of weight loss and exercise improves physical function and mobility in older obese individuals without affecting lean body mass (Villareal *et al.* 2006). A year-long randomised control trial examined the effects of exercise, weight loss and combined interventions on physical function in 93 obese elderly (Villareal *et al.* 2011). The authors found that a combination of weight loss and exercise produced the greatest improvements in strength, balance and gait in the elderly than either intervention alone. A recent randomised control trial investigated the possibility that high-speed circuit training may be more beneficial than resistance training in obese sarcopenic individuals, in which 21 participants were randomly assigned to complete a 15-week trial programme of either strength or circuit training (Balachandran *et al.* 2014). Results showed that those who completed the circuit training had improved physical function with no significant effect observed in the strength-training group. There were significant improvements in strength as a result of both strength and circuit training; however, overall circuit training exerted the biggest strength and functional improvements in an elderly cohort. Although this is a small study, it is one of the first to examine the best way of tackling sarcopenic obesity and warrants further research into this growing area of public health concern.

Nutrition: protein, meat and dairy foods

The amount, essential amino acid composition and intake distribution of protein may be involved in mitigation of sarcopenia (Norton and Jakeman 2013). Paddon-Jones and Rasmussen (2009) found that consumption of 25 g to 30 g of protein per meal encourages protein synthesis to a similar extent in both young and older adults and that the innate response to a high-protein meal does not decline with age. This is consistent with recommendations of 20 g to 30 g protein/meal from Breen and Phillips (2011), while Wall *et al.* (2014) advocated higher intakes of 30 g to 40 g/meal. Others have reported that factors such as the 'quality' of the protein, in terms of their composition of essential amino acids such as leucine, the time-course of daily protein consumption, and the digestion rate ('slow' vs 'fast') of protein should also be considered; modulating dietary protein pattern rather than quantity alone may optimise protein turnover and retention in older people (Gryson *et al.* 2014). With growing interest in sarcopenia research, in recent years, a number of small randomised controlled trials have investigated the role of amino acids, protein supplements, meat and dairy foods on muscle function.

Evidence suggests that the metabolic response to the essential amino acid leucine is poorer in older individuals and that increasing the leucine content of the diet could have a positive role to play in improving anabolic responses and increasing protein synthesis (Katsanos *et al.* 2006; Rieu *et al.* 2006). Leucine is a major amino acid component of milk protein. It is a key regulator of protein metabolism (Lynch 2001; Garlick 2005) by activating the anabolic protein mammalian target of rapamycin (mTOR) signalling pathway to S6K1 and 4E-BP1 (Fujita *et al.* 2007). A small study of 8 sedentary individuals (mean age 68 years), who met protein intake recommendations and were supplemented with 4 g leucine/meal for 3 meals/day for 2 weeks, showed significant increases in muscle protein synthesis and phosphorylation of mTOR, p70S6K1 and 4E-BP1 following the leucine supplementation (Casperson *et al.* 2012). The authors concluded that for older adults consuming almost adequate protein, supplementing with a small amount of leucine is sufficient to improve protein synthesis as well as anabolic signalling. Similarly, Gryson *et al.* (2014) showed that postprandial protein retention was improved in healthy elderly men when a high-protein supplement was provided as 'fast-digesting' soluble milk proteins with high leucine availability compared to casein. From the available literature, leucine supplementation has been advocated as a potential prevention and treatment for sarcopenia (van Loon 2012), particularly a mixed amino acid supplement with an increased content of leucine (Fujita and Volpi 2006). Solerte *et al.* (2008) randomised 41 subjects aged 66 to 83 years with sarcopenia in a crossover trial to receive an orally administered 16 g/day mixture of essential amino acids. After 6 and 18 months, significant increases in whole-body lean mass were observed. Fasting blood glucose, insulin and insulin resistance significantly decreased by the amino acid treatment. Serum TNFα decreased significantly and insulin-like growth factor-1 increased. This indicates that amino acid treatments can increase lean body mass and reduce inflammation in older people with sarcopenia, targeting two key features on the aetiological schematic for sarcopenia and sarcopenic obesity (Zamboni *et al.* 2008; Batsis and Buscemi 2011).

Tieland *et al.* (2012) randomised 65 frail elderly subjects to 30 g/day protein supplement or placebo for 24 weeks. There was no change in skeletal muscle mass or Type I and

II muscle fibre size in the subjects over the trial. Muscle strength increased significantly in both groups, and physical performance improved significantly in the protein group over time, but not in the placebo group. The authors concluded that dietary protein supplementation improves physical performance but does not increase skeletal muscle mass in frail elderly people. Dirks *et al.* (2014) randomised 23 healthy older men to either a dietary protein supplement (21 g protein, with 9 g carbohydrate and 3 g fat) twice daily or control, and all underwent 5 days of one-legged knee immobilisation. Muscle cross-sectional area and leg muscle strength were measured, as well as gene expression from muscle fibre biopsies. Immobilisation significantly decreased muscle cross-sectional areas and muscle strength in both groups, without difference between the supplement groups. Skeletal muscle myostatin, myogenin and muscle RING-finger protein 1 mRNA increased following immobilisation in both groups, indicating increases in both myogenesis and muscle protein degradation, and muscle atrophy F-box/atrogen-1 mRNA expression significantly increased in the protein supplement group only. Overall, there was no attenuation of muscle loss during short-term disuse in health older men following the protein supplement. Kim *et al.* (2015) randomised 22 healthy subjects aged 52 to 75 years to receive 0.8 g or 1.5 g/kg/day protein with an uneven or even distribution of intake over the day. Subjects were infused continuously with labelled phenylalanine and tyrosine following 3 days of intervention. Whole-body protein kinetics (synthesis, breakdown and net balance) were expressed as changes from the fasted to the fed state. The group found that whole-body net protein balance was greater with higher protein intake, but without any demonstrable effects of protein intake pattern.

Pennings *et al.* (2011) randomised 48 older men to ingest 20 g of phenylalanine-labelled whey, casein or casein hydrolysate, and all received a continuous intravenous-labelled phenylalanine infusion to assess *in vivo* digestion and absorption kinetics of dietary protein. They found that there was greater appearance of dietary-derived phenylalanine from whey and casein hydrolysate in circulation than from whole casein. They found that whey protein stimulates postprandial muscle protein accretion more effectively than casein or casein hydrolysate. This was purportedly due to whey's faster digestion and absorption kinetics but also due to its higher leucine content. Chalé *et al.* (2013) randomised 80 mobility-limited adults aged 70–85 years to receive a whey protein concentrate of 40 g/day or control for 6 months, as well as high intensity resistance training. Although there were no significant differences between the groups at the end of the trial, the intervention group had a greater increase in lean mass and muscle cross-sectional area. The authors concluded that, at this dose, there was no additional effect of the whey protein concentrate on resistance training.

Symons *et al.* (2009) found that protein synthesis in muscle increased by about 50% during a 3- to 5-h postprandial period among 17 young and 17 elderly volunteers following consumption of a single moderate serving of lean beef (113 g, 220 kcals, 30 g protein), with no further increase following consumption of a large serving (340 g, 660 kcals, 90 g protein). Daly *et al.* (2014) randomised 100 women aged 60 to 90 years to receive progressive resistance training for 2 days/week with about 160 g red lean meat for 6 days/week or progressive resistance training with control diet (pasta or rice). All women also received 1000 IU Vitamin D_3/day. The red meat group experienced greater gains in total body lean tissue mass, leg lean tissue mass and muscle strength, an increase in serum insulin-like growth factor-1 and a reduction in pro-inflammatory IL-6 after 4 months compared to the control group. The authors concluded that a protein-enriched diet, equivalent to 1.3 g/kg/day, enhances the effects of progressive resistance training in elderly women.

Despite a large literature on the effects of dairy amino acids and proteins, such as leucine and whey, there is a relative dearth of interventions using milk and dairy foods specifically for sarcopenia prevention or treatment in the literature. A few intervention studies have shown that dairy can have a positive effect on muscle mass in older adults. Specifically, a randomised controlled trial over a 12-week period in nonsarcopenic men and women over 60 years of age found that a daily supplement of dairy improved total appendicular skeletal muscle mass and balance test scores (Alemán-Mateo *et al.* 2014). The dairy intervention was in the form of ricotta cheese, providing an additional 18 g of protein to the habitual diet. Furthermore, this study found that dairy attenuated the loss of muscle mass that was seen in the control group. Importantly, these findings were in the absence of any fat mass gain or side effects on kidney function. Additionally, a 12-month randomised controlled trial in healthy postmenopausal women found that daily consumption of 3 portions of dairy products (in the form of 250 ml milk and 200 g Yogurt) fortified with calcium and vitamin D_3 led to favourable changes in mid-arm muscle circumference (an index of total body skeletal muscle mass) and leg lean mass (Manios *et al.* 2009). However, it has been suggested that dairy may only provide benefit before overt sarcopenia develops. In a similar study to the one above, using a ricotta cheese supplement, there were no improvements in skeletal muscle mass or strength compared to a control group in subjects who were already sarcopenic. This suggests that those already suffering with pronounced loss of muscle mass will be less responsive to dairy supplementation compared to those who are free from sarcopenia (Alemán-Mateo *et al.* 2012). Observational studies have also shown a positive association between dairy intake and muscle mass in elderly women (Radavelli-Bagatini *et al.* 2013, 2014). Most recently, Radavelli-Bagatini *et al.* (2014)

found that elderly women (n = 564, mean age 84.7 years) who consumed 2.2 or more servings of dairy daily had a 3.3% higher appendicular muscle mass than those who consumed just 1.5 or fewer servings of dairy daily (P = 0.041). This provides further support to suggest that dairy could have a role to play in preventing sarcopenia. The same group previously found that those with the greatest dairy intake (mean intake 3 servings/day) had better hand-grip strength and performed better in other physical performance tests than those with the lowest intake (mean intake of 0.9 servings/day). Additionally, they found that self-reported falls decreased over a 3-month period with increased dairy intake, suggesting a potential benefit of dairy intake on falls risk reduction (Radavelli-Bagatini *et al.* 2013). The results of these studies are consistent with those from randomised controlled trials in younger adults which have shown better maintenance of lean body mass in those reporting high dairy intake (3 servings/day) compared with those reporting low intake (<1 serving/day), during caloric restriction (Zemel 2005).

CONCLUSION

Sarcopenia was defined in 2009 by the International Sarcopenia Consensus Conference Working Group as loss of muscle mass with or without concomitant fat accumulation, but loss of muscle functionality is recognised widely as a discriminating factor between malnutrition and sarcopenia. Sarcopenic obesity poses additional problems for management, as it requires a different approach to diet and exercise than sarcopenia without fat accumulation. The underlying molecular mechanisms of sarcopenia draw a striking resemblance to those of inflammation in metabolic syndrome.

Recommendations for physical activity caution to 'use it or lose it', with older adults recommended to exercise for strength building and maintenance. Here, we have reviewed evidence for physical activity in both sarcopenia and sarcopenic obesity. In terms of nutrition interventions reviewed here, studies have shown that leucine and essential amino acid supplements (in the region of 4 g/meal or 16 g/day, respectively), protein supplements (in the region of 20–40 g/meal) and whey protein supplements (in the region of 20–40 g/day) have positive effects on muscle strength, muscle protein synthesis and the attenuation of muscle loss. Some interventions have also been investigated for effects on pro-inflammatory cytokines, which feature in the aetiological models of both sarcopenia and metabolic syndrome. A number of studies have investigated the effects of dietary proteins from dairy and meat on the prevention and management of sarcopenia.

Current dietary reference values (DRVs) for adult protein intake in the UK are 46.5 g/day for women and 53.3 g/day for men (Department of Health, 1991). Some studies suggest increasing protein intake throughout the day to between 25 g and 40 g per meal to mitigate sarcopenia, profoundly higher than the DRVs, and others promote the use of 'fast-digesting' proteins with high leucine availability, such as soluble milk proteins for protein retention. Compared to amino acid and protein interventions, there is a relative dearth of studies using food-based interventions, such as milk and dairy products, in sarcopenia. A number of studies reviewed here support the benefits of consuming 3 portions per day of milk and dairy products. However, it should be noted that one portion of semi-skimmed milk (200 mLs) contains 6.8 g protein and, by our estimation, 1.6 g of whey and 0.14 g of leucine, substantially lower quantities of protein, whey and leucine recommended from many intervention studies. This limits the translation between dietary intervention without supplementation and the results of the amino acid and protein studies reviewed here, and obfuscates interpretation of the literature as whole. There is a need for further research into the definition of amino acid and protein requirements for sarcopenia, and whether these can be best achieved through supplementation or specially adapted food-based dietary guidelines for those at risk of age-related muscle loss.

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CONFLICT OF INTEREST

This review has been written by registered nutritionists and registered dietitians working at The Dairy Council in the United Kingdom. The Dairy Council is a non-profit-making organisation with a remit to translate scientific findings on milk, dairy, nutrition and health for consumers, healthcare professionals, the media, the dairy industry, national and international public health bodies. Information about the work of The Dairy Council can be obtained by consulting the website www.milk.co.uk or contacting the corresponding author of this review.

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