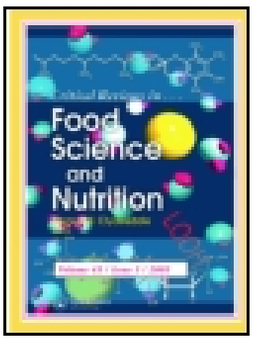

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REVIEW

Dysphagia, texture modification, the elderly and micronutrient deficiency: a review

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ABSTRACT

Dysphagia is an underlying symptom of many health issues affecting a person's ability to swallow. Being unable to swallow correctly may limit food intake and subsequently micronutrient status. The elderly may be the most at risk group of suffering dysphagia as well as most likely to be deficient in micronutrients. The use of texture-modified meals is a common approach to increasing dysphagia sufferer's food intake. The modification of food may affect the micronutrient content and currently there is a limited number of studies focusing on micronutrient content of texture-modified meals. This review considers the prevalence of dysphagia within the elderly UK community whilst assessing selected micronutrients. Vitamin B_{12}, C, D, folate, zinc and iron, which are suggested to be most likely deficient in the general elderly UK population, were reviewed. Each micronutrient is considered in terms of prevalence of deficiency, metabolic function, food source and processing stability to provide an overview with respect to elderly dysphagia sufferers.

KEYWORDS

Deficiency; dysphagia; elderly; malnutrition; micronutrients; texture modification

Introduction

Dysphagia is a medical term used to describe swallowing difficulties. Swallowing is a complex process involving voluntary and involuntary reflexes across four stages. The first two stages 'preparatory' and 'oral' are under voluntary control, concerning food entering the mouth, mastication and forming a bolus before moving to the 'pharyngeal' and 'oesophageal' stages, which are involuntary. Dysphagia occurs from disruption, neurologically or structurally, to one or more of these stages, interfering with adequate intake of food and fluids (Copeman and Hyland 2014; Martino, Beaton, and Diamant 2010; Ney et al. 2009; Ramsey, Smithard, and Kalra 2003). Dysphagia is considered a severe complication in those elderly suffering from it, as it can cause dehydration and malnutrition that will complicate recovery and outcomes of disease (Baijens et al. 2016).

Estimating the prevalence of dysphagia in the UK population is difficult as it is usually a symptom of another health concern such as certain cancers, Parkinson's or Motor Neurone disease, or physical injury affecting the head and neck (Martino, Beaton, and Diamant 2010; Copeman and Hyland 2014). The Royal College of Speech and Language Specialists (2020) estimated that between 50-60% of head and neck cancer patients, 10% of acutely hospitalized adults and 33% of multiple sclerosis patients suffer some form of dysphagia, rising to between 50-75% of people in residential care, which is supported by some UK guidelines (NICE 2006; SIGN 2010).

Studies agree that the onset of dysphagia impedes feeding and is often linked to discomfort or injury, such as aspirating food previously swallowed. This can cause an aversion to eating so ensuring correct nutritional intake becomes challenging (Martino, Beaton, and Diamant 2010; Ramsey, Smithard, and Kalra 2003; Ney et al. 2009; Gordon, Hewer, and Wade 1987).

Studies considering malnutrition, particular in the elderly or populations with dysphagia, do so based on macronutrients and primarily that of calorific energy intake or protein intake (Saito et al. 2018; Namasivayam 2017; Namasivayam-MacDonald et al. 2017). This review focuses on micronutrients, as there is limited research that has reviewed the impact of dysphagia and micronutrient intake. The limiting factor is that the criteria for malnutrition does not directly consider micronutrient status. There is limited research available specifically assessing micronutrient deficiency, as a form of malnutrition, in respect of dysphagic elderly. This study aims to provide an initial review of micronutrient information available when considering this specific population, texture modified meals as a feeding solution, and how processing may alter micronutrient content of food suitable for dysphagia sufferers.

Dysphagia and malnutrition

Malnutrition can be defined as an imbalance of energy, protein and other essential nutrients that causes measurable effects on the body's function, including structure and neurology (BAPEN 2019).

Malnutrition screening is based on the 'Malnutrition Universal Screening Tool' (MUST) in the UK (BAPEN

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2019). The tool assesses three factors: Body mass index (BMI), unplanned weight loss and if a patient is acutely ill or will not eat for more than 5 days. The final score is either low, medium or high. Medium or high scores recommend enhanced nutritional support such as dietician referral and increased monitoring (BAPEN 2019; Tagliaferri et al. 2019). The tool does not take into account specific dietary intake or current nutritional status and is based mainly on weight. A link is therefore perceived between weight changes and nutritional status that require further clinical intervention to assess for acute nutritional deficiency.

Assessing the prevalence of malnutrition within the population suffering from dysphagia has a number of variables. Prevalence rates differ by subpopulation, health condition and if institutionalized or free living. One study, focused on hospitalized patients only, found 6.6% of the population (N = 17,580) had dysphagia and the risk of malnutrition was 18.9% although the population included anyone aged 18 years or older and focused on American patients only (Blanař et al. 2019). However, other recent studies have suggested a malnutrition prevalence linked to dysphagia of between 3% and 29% depending on population (Namasivayam 2017; Namasivayam-MacDonald et al. 2017).

Madhavan et al. (2016) reviewed studies for free living elderly and found assessment criteria for dysphagia ranged widely from 'minor swallowing difficulties' to 'ease of swallowing water' giving prevalence of 5% and 72% respectively (N = 9,947). This complicates ascertaining true prevalence based on available research. Blanař et al. (2019) suggested that the risk of malnutrition increased threefold (95% CI [2.96, 3.46]) for dysphagia sufferers. This does not take into account the underlying health conditions that may exacerbate malnutrition, such as gastrointestinal carcinoma. Research supports the co-occurrence of malnutrition risk and dysphagia, particularly where there is an underlying disease such as stroke or cancer (Carrión et al. 2015; Govender et al. 2017; Huppertz et al. 2018; Madhavan et al. 2016; Rofes et al. 2018; Blanař et al. 2019). Due to the apparent, higher rate of malnutrition in line with disease and dysphagia, there is evidence to suggest that aiming to maintain correct nutritional status in sufferers is of importance.

Prevalence of dysphagia in elderly (residential and healthcare settings)

In the UK, anyone aged over 65 years old can be classified as elderly (NHS 2020) with around 4% of this population (65 to 84-year olds) living in institutional care. This increases to 16% of those aged over 85 years old (LaingBuisson 2016). The rate of disease increases with age and therefore the risk of suffering dysphagia as a symptom may also increase. The World Health Organization (WHO 2019) estimate 75.6 million people worldwide will suffer from dementia for example, and a 2020 study (Espinosa-Val et al. 2020) found the prevalence of dysphagia was up to 93.6% in a population of dementia sufferers (N = 255). This is higher than other studies that estimate the prevalence at 15% of the general elderly population, rising to 68% of those in institutional care (Abu-Ghanem, Chen, and Amin 2020), but the link of dysphagia to disease, appears to be more prominent in studies which consider elderly with underlying health conditions.

Abu-Ghanem, Chen, and Amin (2020) reviewed 15 studies into the elderly and dysphagia and found prevalence ranging from 5% to 72% (N = 9,947). This was similar to Madhavan et al. (2016) who concluded that the range found may be due to the different dysphagia assessment methods used in different studies, further supporting the issue of estimating dysphagia in the elderly accurately. One European study proposed that dysphagia in the elderly is underdiagnosed but provided detail on the use of two verified clinical methods for diagnosis. These methods do not correspond to some of the methods used in other studies such as ability to swallow a glass of water. The study concluded with a recommendation that dysphagia is added to a list of 'geriatric syndromes' (Baijens et al. 2016). These syndromes are criteria used to assess elderly for enhanced geriatric care.

Dysphagia and malnutrition in elderly (residential and healthcare settings)

Aging is associated with an overall reduced appetite (Saito et al. 2018) without the compounding factors of dysphagia. Reviewed research agrees that there is a higher risk of general malnutrition in the elderly, which is becoming a novel issue in developed countries with aging populations (Saito et al. 2018; Namasivayam 2017; Namasivayam-MacDonald et al. 2017). Several studies have suggested higher malnutrition in dysphagia suffering elderly, including 12.9% (N = 117) using supplements to reach daily calorific goals (Roy et al. 2007). This is supported by Wright, Cotter, and Hickson (2008) who found 54% of elderly on texture modified diets (N = 16) required additional nutritional supplementation, compared to 24% (N = 30) on normal texture diets.

A further study proposed that malnutrition prevalence in dysphagia suffering elderly was 18.6% compared to 12.3% of elderly without dysphagia (N = 254) (Serra-Prat et al. 2012). Contrarily, a study supported the notion that texture modified diets for the elderly with a wide variety of choices increased food intake (N = 8) compared to a restricted menu, leading to weight gain and improved nutrition status (Germain, Dufresne, and Gray-Donald 2006). The sample sizes in these studies are minor but the outcomes show similar results in that malnutrition is more common in elderly with dysphagia.

In the general elderly population in the UK, a recent study has identified deficiencies in vitamin B_{12} at 12% and folate at 15% (N = 5,290) (Laird et al. 2018). In comparison, an older study identified deficiency prevalence at 5% for folate and 16% for vitamin C (N = 128) (Bailey et al. 1997). Vitamin D deficiency is relatively common in the UK population with one study estimating 50% of the population is deficient overall, rising to 17% being severely deficient during winter months (Pearce and Cheetham 2010). Vitamin C has been found to be deficient in 44.7% of people

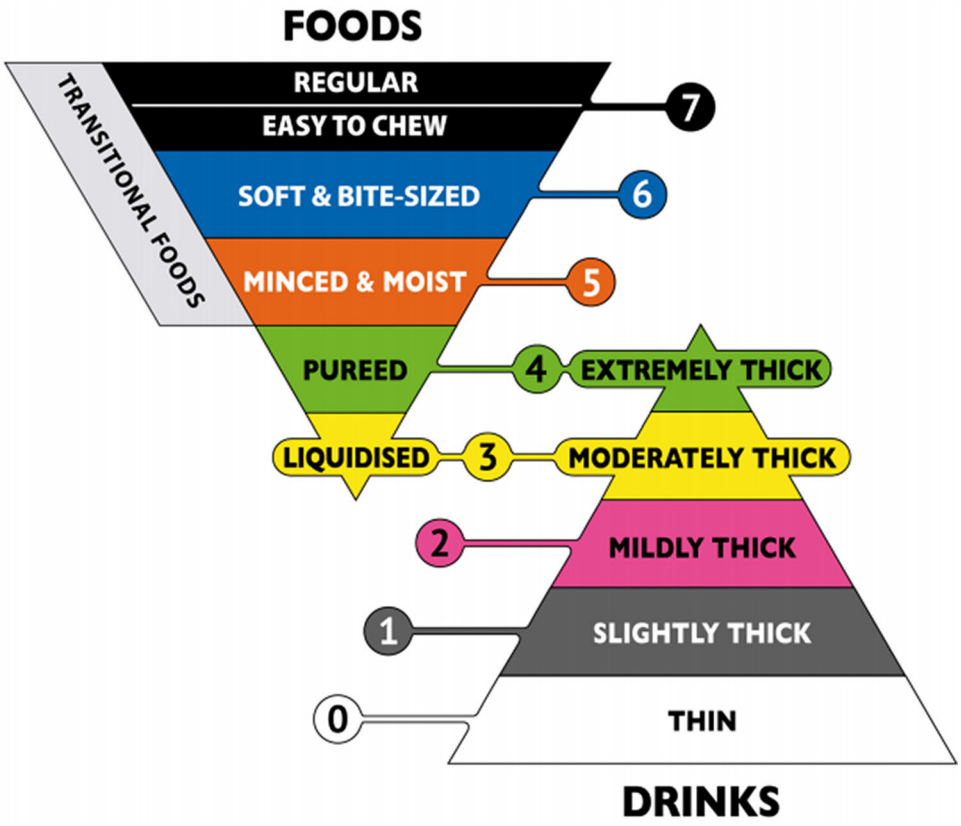

Figure 1. Levels of texture modification as defined in the IDDSI Framework (IDDSI 2019). © The International Dysphagia Diet Standardization Initiative 2019 @ https://iddsi.org/framework. Licensed under the CreativeCommons Attribution Sharealike 4.0 License https://creativecommons.org/licenses/by-sa/4.0/legalcode. Derivative works extending beyond language translation are NOT PERMITTED.

(N = 1,276) by one study of UK adults (Wrieden et al. 2000). Another UK study found 67.8% of over 65-year olds were deficient to some degree (N = 851) (Gale, Martyn, and Cooper 1996). The UK National Diet and Nutrition Survey (published in 2014) found intakes of vitamin D, vitamin K, magnesium, selenium and zinc to be consistently below the RNI for people aged 65 years and over throughout the 4 years of the survey (Bates, Lennox, and Prentice 2014).

This review will consider the institutionalized elderly with dysphagia due to the population having less choice in meals and ability to live fully autonomously. The reduced variety of food and movement is a compounding factor that needs to be considered as this may reduce micronutrient intake, particularly if meals on offer are not sufficient in vitamin and minerals to begin with.

Texture modification of ready meals

The quality of texture-modified meals is of importance for those suffering with dysphagia and good quality has been shown to increase food consumption (Germain, Dufresne, and Gray-Donald 2006).

There is no legal definition of texture modification. The International Dysphagia Diet Standardization Initiative (IDDSI) created an internationally recognized framework the 'Complete IDDSI Framework 2.0′ (IDDSI 2019) to define various levels of texture modification. In the UK, the NHS has adopted this framework to ensure conformity across all hospitals. The framework sets standards and testing mechanisms for each of the 7 levels to confirm if a food is of suitable consistency, depending on the severity of dysphagia (IDDSI 2019; NHS 2018) (Figure 1).

Texture modification involves altering the structural properties of foods in order to produce a different texture to what is usually expected. Food can be increased or decreased in thickness, made into a puree or soup consistency, or have reduced particle size to aid chewing and swallowing. Modification can be achieved industrially or in a commercial kitchen with various techniques. The simplest is cooking food until soft and blending it to a smooth consistency. Depending on the level of modification, this puree can be thickened with starches or thinned with further fluids to achieve the desired consistency.

To aid visual acceptance of texture-modified meals, the puree can be mixed with starches and placed in molds (Figure 2).

The molds make the foods appear representative of the non-modified version they were pre-processing. There are numerous temperature changes throughout the production of meals, usually from various ingredients of chilled, ambient or frozen state. These ingredients need to be heated to ensure food safety. Heating is also used to ensure the ingredients reach a tenderness suitable for smooth pureeing. The products, once produced, may then require chilling or freezing prior to being sold. Once ready to consume, they may require another round of heating. These changes in temperature, from −18 °C to >72 °C, may cause vitamin and

Figure 2. Example of a texture-modified meal, level 4, that has been molded to represent the ingredients it was produced from. From the top clockwise: carrots, gravy, broccoli, mashed potato, chicken with sage and onion.

mineral loss, as well as the initial boiling and cooking of the product before puree.

Food safety, quality and legality considerations of texture modification

All foods produced in the UK must be compliant with The Food Safety Act 1990, The Food Safety and Hygiene Regulations 2013 and The General Food Regulations 2004. These regulations enforce that products must be safe to consume and not mislead the consumer. Texture modified meals will differ in appearance and consistency and must be named appropriately to inform consumers of the difference. Specific dietary requirements such as allergens must also be correctly defined for the food product to ensure safety and compliance with *The Food Information Regulations 2014*, even in institutional care. A person suffering with suspected dysphagia may be referred to a specialist dietician who will be able to prescribe the correct level of texture modification to ensure safety when eating (NHS 2018).

Impact of texture modification on macronutrients and micronutrients

Changes in texture have been found to change the macronutrient content of meals. One study reported losses of total energy, protein, carbohydrates and fat in pureed meals compared to the normal variants. It concluded that the difference was not statistically significant ($P < 0.05$). The results suggested this was because of losses during processing such as protein in water and fats and carbohydrates within cooking vessels (Vigano et al. 2011). Further studies on the macronutrient loss in texture-modified meals do not appear to be available at the time of writing. This may be because modification involves the comminution of foods but does not add or remove macronutrients already present. Texture modification for dysphagia meals involves macro comminution of foods and may not be severe enough to alter the molecular structure of macronutrients, rendering them unavailable for uptake.

Micronutrients may be impacted by texture modification. Often, texture modified foods require longer cooking to ensure they are tender enough to produce a smooth consistency once blended. Many micronutrients including vitamin B_{12}, vitamin C, vitamin D and folate may be degraded by excess exposure to heat and possibly air during processing, although heat processing does not appear to affect minerals as much (Sungsinchai et al. 2019). Depending on the processing, many micronutrients are also enhancers or inhibitors of each other. If texture modification is less controlled and batch pureed as a whole meal, this may influence the biochemical activity of the vitamins and minerals. This may expose them to their enhancers or inhibitors that will then have time to further interact before the food is consumed. For example, a level 3 product of soup consistency may have reduced zinc bioavailability if it includes cereals that contain phytate. Level 4 may reduce this exposure between ingredients due to the individual components remaining separate prior to consumption.

Micronutrients

Dietary recommendations for general population and elderly

The Dietary Reference Values (DRVs) for vitamins and minerals are given as Reference Nutrient Intake values (RNIs). Over the age of 65, energy requirements drop from 2000 kcal/day to 1912 kcal/day based on women. The macronutrient requirements also change such as protein increasing for women by 1.5 g/day but decreasing by 2.2 g/day for men. Fat and carbohydrate levels drop for both sexes. Further, the levels of macronutrients reduce for those aged 75 years and older where energy, for females as an example, falls to 1840 kcal/day, although protein intakes remain unchanged, fat and carbohydrates decrease (Public Health England 2016).

Micronutrient intakes differ by vitamin and mineral, for vitamins women are given slightly decreased values compared to men. In most cases however, the requirements do not change from 19 years old to 75 years and older except for thiamin and niacin, reducing by a minor amount e.g. 1.1 mg/day niacin 19-64 years old compared to 75 years old. Vitamin D figures for the UK are 5 µg/day less than that of the European Union recommended intake of 15 µg/day due

to the reduced level of sunlight exposure in some northern EU countries (Public Health England 2016; European Food Safety Authority 2016).

Mineral RNIs do not change depending on the age category of the population except iron requirements for females. Iron requirements for menstruating woman are 14.8 mg/day but once menopause occurs, recommendations reduce to 8.7 mg/day, same as males. Most minerals are the same for both sexes except for magnesium, zinc and selenium, which remain higher for men at all age categories (Public Health England 2016).

Important micronutrients in elderly nutrition

Vitamin B_{12}

Vitamin B_{12} is a term for all corrinoids, compounds containing a corrin ring shaped nucleus with a cobalt center, similar to those of hemoglobin and chlorophyll. Vitamin B_{12} is synthesized by bacteria and found in the tissues of animals, being able to be stored for up to a year (Combs 2008). It is not found in plant tissues due to its synthesis by bacteria in nature or from gut microflora, which is a possible reason those following strict vegetarian or vegan diets may be deficient (Combs 2008; Berdanier and Berdanier 2015).

Vitamin B_{12} absorption, bioavailability and food sources. Absorption has been estimated at 25-65% when considering eggs, meat and fish, but studies agree that estimation is difficult. This may be due to the similarity of the vitamin to folic acid as they both interact in some pathways concurrently (Heyssel et al. 1966; Doscherholmen, McMahon, and Ripley 1975; Doscherholmen, McMahon, and Economon 1981). Absorption of the vitamin through supplements was suggested to be lower at ± 1% (Berlin, Berlin, and Brante 1968). The vitamin usually bound in coenzyme form to proteins and supplements do not contain the vitamin in such a format. This limits their ingestion from the missing proteolytic activity of pepsin and subsequent binding to the Intrinsic Factor (IF) in the small intestine (Combs 2008). Vitamin B_{12} intake is also dependent on adequate intake of ascorbic acid, thiamin, carnitine and fiber. Propionate production increases if these are low, increasing vitamin B_{12} requirements (Berdanier and Berdanier 2015; Combs 2008).

Vitamin B_{12} is classified as a water-soluble vitamin, literature suggests the normal turnover rate of the vitamin is 2.5 µg/day, slightly higher than the UK RNI of 1.5 µg/day (Berdanier and Berdanier 2015; Public Health England 2016). This may be due to some studies theorizing that the vitamin is one of the most potent; little being needed to prevent deficiency (Allen et al. 1993; Carmel 2011; Nexo and Hoffman-Lucke 2011; Yetley et al. 2011). Due to its exclusivity to animal tissues and bacterial synthesis, certain tissues such as the liver accumulate the vitamin well and are good sources when consumed. Beef liver contains 69-122µg/100g compared to beef muscle at 1.94-3.64 µg/100g (Combs 2008). Most sources of animal tissue humans consume contain some level of the vitamin, but it varies significantly between species and tissue type (Combs 2008).

Vitamin B_{12} metabolic function and deficiency (including prevalence and physiological presentation). Vitamin B_{12} functions in two coenzyme forms: adenosylcobalamin (ACB) and methylcobalamin (MCB), which catalyzes the conversion of odd chain fatty acids. Deficiency in the vitamin, and therefore these coenzymes, has been linked to accumulation of irregular fatty acids in neural tissues. This can be compounded by reduction in methionine synthase, a catalyst for regeneration of methionine, leading to impaired creatine, phospholipid and acetylcholine synthesis that may reduce a range of physiological functions. Neurological symptoms present in a quarter of deficient individuals as peripheral neuropathy with numbness in limbs, memory loss, depression and possibly dementia (Combs 2008; Levitt, Wesson, and Joffe 1998). This process is irreversible as the myelin sheaths on nerves are degenerated, although preventing further damage with correcting vitamin B_{12} status is possible (Berdanier and Berdanier 2015). Therefore, with the increased risk of dysphagia associated with dementia sufferers and the link of vitamin B_{12} to correct neurological function, status of this vitamin should be considered important in the elderly suffering dysphagia.

Studies have found increased homocysteine levels, a marker of inadequate vitamin B_{12} intake, correlated with lower neuropsychological test scores. There were also supporting findings that suggested increased vitamin levels did not improve cognition, although minor improvements were found in the frontal lobes (Moretti et al. 2004; Combs 2008).

Prevalence of deficiency has been suggested at 10-15% (N = 725) (Carmel et al. 1999) of 'healthy' American elderly, compared to another study in which up to 50% of elderly Americans had inadequate vitamin B_{12} status (N = 809) (Norman and Morrison 1993). The UK National Diet and Nutrition Survey (published in 2014) found low vitamin B_{12} status in women decreased by 1% year on year (Bates, Lennox, and Prentice 2014) in contrast to the American study . Using homocysteine levels to determine vitamin B_{12} status should be used with caution as it is also linked to folate status which may affect the accuracy of any results (Berdanier and Berdanier 2015; Combs 2008).

Vitamin B_{12} processing stability. Vitamin B_{12} is susceptible to photodecomposition, which is enhanced in aerobic conditions. Anaerobic conditions have been shown to reduce the rate of photodecomposition (Demerre and Wilson 1956; Ahmad et al. 1992; Wang et al. 2019) suggesting that vitamin B_{12} is also susceptible to oxidation. The oxidation occurs due to the Co^{3+} reacting with available O_2 forming cob(II)alamin and a methyl free radical (Vaid et al. 2018).

Contrary evidence on the vitamin's heat stability have been found stating the vitamin is very stable in heat in its crystalline form (Combs 2008), but that it is not stable during food production as it is sensitive to heat (Ahmad et al. 1992). Combs (2008) is supported by a study which showed aqueous B_{12} lost 10% of mass up to 200 °C, compared with vitamin C which lost up to 30% of its residual mass (de Britto et al. 2016). Food processing temperatures aim to

achieve a heat of 70 °C for 2 minutes in the UK (Food Standards Agency 2018) providing sufficient safety. Therefore, it could be suggested that vitamin B_{12} may be capable of withstanding general food processing techniques.

The vitamin has been found to degrade rapidly in the presence of ascorbic acid, which catalyzes oxidation. It has also been shown to perform poorly in the presence of iron, which appears to limit its absorption availability (Combs 2008). The presence of iron in animal tissue however is usually bound in hemoglobin complexes and may therefore reduce risk of reaction. There is limited ascorbic acid in foods, which are sources of vitamin B_{12} too, reducing the risk of this cross-reactivity occurring.

Vitamin C

Vitamin C is the common name for ascorbic (AA) and dehydroascorbic (oxidized) acid (DAA) that can exist in either form due to readily accepting or donating H^+. The compound is closely related to glucose, which is similar in structure however, vitamin activity is only seen in the L-form whereas glucose is chemically active in D-form (Berdanier and Berdanier 2015). The RNI for vitamin C in the UK is 40 mg/day for both genders over 18 years old (Public Health England 2016; British Nutrition Foundation 2019).

Vitamin C absorption, bioavailability and food sources. AA is a water-soluble vitamin and absorbed primarily in the ileum by active transport. It requires sufficient Na^+, which bind to carrier proteins with AA. The compound travels through the mucosal membrane to cells where the sodium is removed through the sodium-potassium pump. The vitamin is then disassociated from the carrier protein and remains free within the cell for metabolic functions. Excess AA is excreted through urine primarily as AA, although metabolites such as oxalic acid also contribute to AA loss (Berdanier and Berdanier 2015).

It is suggested that 3% of AA stores are used daily and that equates to a turnover rate of 60 mg/day according to one study, slightly higher than the UK RNI (Kallner, Hartmann, and Hornig 1979). The UK RNI is based on research that is more recent and suggests 40 mg/day is sufficient for the population to show AA markers in blood serum even though intakes of 10 mg/day were shown to be able to prevent deficiency symptoms (COMA. 1991; Jacob et al. 1987). Adults will maintain a body store of around 900 mg AA, anything below 300 mg may present with deficiency symptoms (Olson and Hodges 1987).

Humans lack L-gluconolactone oxidase that is required for the final step in the biosynthesis of AA from glucose. AA therefore needs to be gained from the diet. It is found in citrus fruits, tomatoes and strawberries as well as some vegetables such as potatoes, capsicums and members the *Brassica* genus i.e. broccoli and cabbage (Dominguez-Perles et al. 2014; Nishikimi et al. 1994).

Vitamin C metabolic function and deficiency (including prevalence and physiological presentation). Once absorbed, AA is one of few vitamins able to move throughout the body freely and can cross the blood-brain barrier, similar to glucose. Due to its redox capabilities, AA performs as an antioxidant and has been shown to potentially support other antioxidants, including those that are fat soluble e.g. vitamin E (Padh 1991; Carr and Frei 1999; Berdanier and Berdanier 2015). It has been shown to protect proteins from oxidative stressors and maintain correct levels of saturation in fatty acids (Kirkwood and Mathers 2009, 33).

Literature suggests an important consideration of AA is its role in wound healing. AA acts as a coenzyme in a number of reactions, of which one pathway is the hydroxylation of proline to hydroxyproline, an amino acid of collagen (Berdanier and Berdanier 2015; Jenkins, Wainwright, and Green 2009, 120). Collagen is an important component of wound healing in the initial stages, supporting the wound to seal, preventing microbiological and physical contamination and blood loss (Jenkins, Wainwright, and Green 2009, 120). Some research considers the use of high dose vitamin C to be ineffective at enhancing wound healing however, as it has been found that tissue saturation of AA occurs at 200 mg/day, so becomes its own limiting factor (Levine et al. 1999; Jenkins, Wainwright, and Green 2009, 120).

On the other hand, studies have agreed that those suffering severe illness, such as sepsis and the elderly in hospital settings have shown improved wound healing with supplementation of between 300 mg and 500 mg/day. Elderly hospital patients are described in one study as the most at risk group for vitamin C deficiency (ter Riet, Kessels, and Knipschild 1995; Williams 2002; Selvaag, Bohmer, and Benkestock 2002). It should also be considered that slower wound healing reduces speed of recovery as it increases the metabolic needs of patients, thus increasing nutrient needs (Jenkins, Wainwright, and Green 2009, 120).

Vitamin C deficiency has been found to be between 6.9% and 40% of free living elderly, rising to 32 to 68% of hospitalized elderly (N = 18,292) (Raynaud-Simon et al. 2010; Mosdol, Erens, and Brunner 2008; Hampl, Taylor, and Johnston 2004; Fletcher, Breeze, and Shetty 2003), in studies on UK and USA populations including those aged 18 years or older.

Clinical presentation of deficiency occurs when the body's pool of AA drop to around 300 mg, which may take up to 6 weeks on a vitamin C free diet (Raynaud-Simon et al. 2010). Initial symptoms include malaise and weight loss that are nonspecific to vitamin C deficiency and therefore deficiency in vitamin C may not be suspected (Raynaud-Simon et al. 2010; NHS 2017). Further symptoms develop after 2-3 months such as skin lesions, bleeding gums and changes to hair. This advanced stage of deficiency can be classified as scurvy. Scurvy is easily treated with the addition of sufficient vitamin C to a patient's diet, with symptoms improving within 48 hours of treatment (NHS 2017).

Vitamin C processing stability. AA, due to its redox potential, is highly susceptible to oxidation. One study noted an AA loss of 8-25% of peeled apples exposed to air for up to 4 hours (Oyetade et al. 2012). Further losses are seen with

increasing storage time, temperature and pH as Jeney-Nagymate and Fodor (2008) found different beverages started at ±35mg/L AA and dropped to less than 5 mg/L AA after 5 days without temperature control. Orange juice appeared to retain most of its AA content, due to the maintenance of a pH <4. This study also suggested that the use of added vitamin E decreased the loss of AA, potentially due to the symbiotic protective potential of vitamin E and AA as described previously.

The vitamin is also lost in heat-processed foods, with green vegetables being shown to lose 65% of their AA content when boiled for less than 40 minutes, from leaching into the cooking water. Microwaving was found to retain the most AA (Allen and Burgess 1950; Lee et al. 2018). Further loss is seen during chilling at up to 10% (Charlton et al. 2004). These losses are most likely due to oxidation as well as leaching during cooking (Charlton et al. 2004). It should also be considered for foods that require reheating, further losses in the vitamin would be seen, as agreed by Williams (1996). One study found the highest loss of AA was during reheating of vegetables for 40 minutes to 75 °C. The total loss was suggested at 94% (McErlain et al. 2001). Studies have suggested that blanching and freezing is able to slow AA loss in fruits and vegetables (Favell 1998; Giannakourou and Taoukis 2003; Armstrong, Jamieson, and Porter 2019) however, the loss may still occur as these products will not generally be consumed frozen and will require a form of heating and exposure to air.

Vitamin D

Vitamin D is a term used to describe a family of sterol-based compounds that differ based on their side chains. Two main variants are considered in respect of nutrition, vitamin D_2 ergocalciferol and vitamin D_3 cholecalciferol. Vitamin D_2 is primarily synthesized in plant tissues with exposure to UV and vitamin D_3 is synthesized in animals and in the skin of humans when exposed to UV radiation (Loveridge and Lanham-New 2009; Berdanier and Berdanier 2015).

Vitamin D absorption, bioavailability and food sources. There are limited food sources that contain naturally occurring vitamin D in either form with oily fish and eggs being the main categories. Many foods are voluntarily fortified with vitamin D such as fats, spreads and breakfast cereals to increase intake. It is estimated that fortified cereals contribute 13% of the daily RNI for men and 20% for women in the UK (British Nutrition Foundation 2018). The majority of vitamin D is synthesized by the body from UV radiation in sunlight (Berdanier and Berdanier 2015).

The vitamin is fat-soluble and requires the presence of lipids and bile salts during digestion to aid absorption. The absorption takes place in the ileum with long chain fatty acids and there is no active transport system for the vitamin. This may be due to the limited levels required which does not necessitate such an absorption method, as well as the body being able to synthesize it (Berdanier and Berdanier 2015).

The synthesis of vitamin D occurs on the skin where UV light converts 7-dehydrocholesterol to a pre-vitamin D form. This is then metabolized through a number of pathways in the liver and kidneys to create the active vitamin D_3. People suffering with liver or kidney disease have been found to have reduced or no ability to fully synthesis the vitamin and therefore food becomes the only source (Loveridge and Lanham-New 2009, 62).

Combs (2008) suggests that there are links between vitamin D and zinc. Low zinc intake may reduce certain enzyme's activity linked to the absorption of vitamin D. Further, Combs (2008) also suggests that iron deficiency has been shown to impair vitamin A absorption and suggests the same may occur with vitamin D. Therefore, ensuring correct intake of these minerals may support a person's vitamin D status.

Vitamin D metabolic function and deficiency (including prevalence and physiological presentation). In the UK, the RNI is 10 μg/day irrespective of age or gender. Supplementation is suggested in winter months when exposure to sunlight is reduced (Public Health England 2016). Vitamin D plays a role in the formation and maturation of bone, regulating the metabolism of calcium and phosphorous absorption in the digestive system. It stimulates the production of specific calcium binding proteins in gut cells (Berdanier and Berdanier 2015; Loveridge and Lanham-New 2009, 62). During ageing, the natural amount of this protein drops and humans become less efficient at absorbing calcium. In elderly patients with vitamin D_3 supplementation, calcium absorption was increased (Berdanier and Berdanier 2015; Bell 1995; Christakos et al. 2011). Vitamin D intake has also been linked to a delay in cognitive decline and improvements in mood (Butriss 2009, 276) suggesting importance in elderly dysphagia suffers who may suffer low mood and depression. Deficiency presents as bone deformities in children, known as rickets, whereas in the elderly, deficiency is indicated by osteoporosis (McLaren 2006).

It is estimated that in free-living elderly deficiency prevalence was 26.4% to 58.7% dependent on assessment method (N = 6,004) (Aspell et al. 2019). This rate of deficiency has been linked to seasonal changes in sunlight exposure. This is compounded by increased use of sunscreen application blocking UV on the skin and reduced rate of exposure to sunlight due to limited movement as people age (Berdanier and Berdanier 2015; Laird et al. 2010; O'Neill et al. 2016). In one European study, deficiency in hospitalized elderly patients was higher at 87.4% (N = 217) (Boettger et al. 2018). This suggests those in institutional care may be subject to less exposure to UV light compared with free-living elderly. This supports the notion of the importance of micronutrient content of foods for the institutionalized elderly. Further, vitamin D deficiency may be higher risk for ethnicities with darker skin tones, due to the reduced synthesis by UV light. It has been suggested that those with darker skin tones require up to 5 times the exposure to sunlight to create the same level of the vitamin as Caucasians (Hintzpeter et al. 2008; Nair and Maseeh 2012).

Studies have linked vitamin D intake to bone health and frailty. It has been suggested sufficient intake of vitamin D can reduce the prevalence of falling. One meta-analysis study found the risk of fractures from falling was reduced by at least 20% in a population aged 65 years and older, supplemented with 10 μg/day vitamin D (N = 42,279) (Bischoff-Ferrari et al. 2009). This is somewhat supported by another study which found supplementation of the vitamin at 20 μg/day, reduced the risk of falling (Kalyani et al. 2010). Larger doses of the vitamin have however, been shown to increase the risk of falls and fractures (Sanders et al. 2010) as hypervitaminosis may occur. This results in increased risk of excessive calcification of bones and soft tissues (Berdanier and Berdanier 2015). Hathcock et al. (2007) proposed the upper intake limit of vitamin D should not exceed 250 μg/day, as the vitamin is easily stored in the liver.

Vitamin D processing stability. Vitamin D_2 has been found to be less stable than vitamin D_3 but both are susceptible to oxidation when stored in air for 24-72 hours (Berdanier and Berdanier 2015). The vitamin is unstable in acidic conditions and is photosensitive but stable in alkaline conditions (Berdanier and Berdanier 2015). This is somewhat counter intuitive considering the synthesis of the vitamins requires UV light on skin. Vitamin D can be made miscible in water with the use of surfactants; however, these solutions are very unstable and vulnerable to oxidation (Berdanier and Berdanier 2015). The addition of antioxidants, such as carotene, has been shown to improve vitamin D retention in solution. The use of refrigeration, protection from air and water have been found to reduce vitamin loss (Berdanier and Berdanier 2015).

Vitamin D is somewhat heat stable, a study found heat treatment of eggs, bread and margarine showed retention of 39-45% when oven cooked for 40 minutes at 175 °C (Jakobsen and Knuthsen 2014). It further suggested that cooking method significantly changed the rate of retention as pan-frying the same products for 3 minutes gave retention of 82-84% (Jakobsen and Knuthsen 2014). This suggests that the length of exposure to heat affects the loss of the vitamin.

The results for heat loss are supported by Jakobsen and Knuthsen (2014) and suggest that the vitamin is more stable in heat for shorter periods, than when exposed to air. Therefore, attention should be paid to reducing oxidation and length of time in heats.

Folate

Folate is a common term used to describe folic acid and its derivatives. There are six derivatives of the compound, all of which show vitamin activity if the structure is correctly hydrogenated. Folate is found in every cell of animals and humans. It is a recently discovered compound having only been isolated once the DNA process was better understood, due to its role in DNA synthesis (Berdanier and Berdanier 2015; Combs 2008).

Folate absorption, bioavailability and food sources. Folates are found in a variety of plant and animal tissues such as the liver, meat and leafy green vegetables. Plants of *Brassica* genus can have up to 190 μg/100g folate and chicken up to 1080 μg/100g (Combs 2008). It is suggested that folates from plant origin are less bioavailable than those from animal origin and an estimated 50% of ingested folate is not absorbed although this can vary between 10-90% (Combs 2008). Bioavailability differs significantly between foods and Combs (2008) suggests three main variables that affect absorption. These are foods containing 'antifolates', which act as inhibitors preventing absorption; the bio-potency of the different derivatives and the nutritional status of the consumer. Deficiencies in vitamin C and iron are linked to reduced absorption of folates.

Absorption is carrier-mediated and pH dependent with the highest affinity to its carrier at pH 5.5-6.0 (Berdanier and Berdanier 2015). It then circulates in plasma and is taken in by cells using a highly specific folate binding protein (Combs 2008; Berdanier and Berdanier 2015). Reduced absorption is seen in those suffering diseases of the digestive system or on certain drugs such as sulfasalazine, used to treat rheumatoid arthritis (RhA) (Berdanier and Berdanier 2015 Combs 2008,). These drug interactions may be of importance when considering the elderly. An estimated 520,000 people suffer with RhA in the UK, with the peak onset at 45-75 years old (NICE 2016).

Folate metabolic function and deficiency (including prevalence and physiological presentation). The UK RNI for folate is 200 μg/day regardless of age or gender, although pregnancy requires double the RNI (Public Health England 2016). Folate functions as a coenzyme in a number of important biochemical pathways. One of these is the concurrent metabolism of methionine with vitamin B_{12} (Berdanier and Berdanier 2015). This process is linked to the maintaining correct levels of homocysteine, which if elevated has been linked to increased odd chain fatty acid accumulation and changes in cognitive performance as well as dementia (Berdanier and Berdanier 2015; Combs 2008; Levitt, Wesson, and Joffe 1998; Moretti et al. 2004). It also complicates ascertaining folate and vitamin B_{12} status if homocysteine levels are used as a marker, due to both participating in this pathway.

Another notable interaction of folate is in the pathway for the production of purine and pyrimidine bases. These are used for the creation of mRNA in cell reproduction and maintenance (Berdanier and Berdanier 2015). This pathway explains why folate is found in all cells within humans. Folate turnover has been suggested to be dependent on intake. 200 μg/day intake equates to 28.5 μg/day catabolism, whereas 400 μg/day intake is catabolised at 32.2 μg/day. Folate excretion is seen at less than 1% of total body stores per day (Combs 2008).

Folate deficiency presents as megaloblastic anemia, dermatitis and decreased growth (Berdanier and Berdanier 2015). Deficiency can be linked to depression and cognitive decline, similar to vitamin B_{12}. Improved folate status has

Table 1. Summary of vitamins based on reviewed literature.

Vitamin	Sources	Function	Symptoms of deficiency	Processing stability
Vitamin B_{12}	Animal proteins, dairy, eggs	Coenzyme in the catalyzing of odd chain fatty acids and correct neurological function	Peripheral neuropathy. Numbness, memory loss and possibly dementia	Sensitive to irradiation. Heat stable to 200°C. Sensitive to oxidation, light, ascorbic acid and metal ions
Vitamin C	Citrus fruits, tomatoes, *Brassica* genus vegetables	Antioxidant protects from oxidative stress. Role in wound healing and collagen synthesis	Malaise, weight loss, scurvy. Changes to hair and wound healing	Easily oxidized, More stable in acidic solutions. Liable to leaching from food during cooking
Vitamin D	Oily fish, eggs and fortified fats and spreads. Synthesized by skin in UV light	Supports calcium uptake and bone strength. May reduce risk of falling. Delay in cognitive decline	Osteoporosis, brittle bones	Liable to oxidation. Addition of antioxidants reduces oxidative loss. Heat sensitive if exposure is prolonged
Folate	Animal proteins, fruits and vegetables	Linked to methionine pathway with vitamin B_{12}. Odd chain fatty acid catalysis, and interaction in the purine and pyrimidine bases production	Anemia, dermatitis, reduced growth. Also linked to depression and cognitive decline	Highly liable to oxidation. Accelerated by heat, light and metal ions. Liable to leaching during cooking

been shown to help slow cognitive decline linked with vitamin B_{12} deficiency, but not reverse or stop it (Combs 2008). Clarke et al. (2004) suggested that folate deficiency in the elderly did not differ significantly between UK free-living and institutionalized elderly populations. The study suggested the prevalence of folate deficiency increased with age ($P < 0.05$) with 5% clinically deficient at 65 years old, rising to 10% of those aged over 75 years old (N = 3,511). These findings are supported by Aytekin, Mileva, and Cunliffe (2018) who suggest deficiency in the UK elderly population ranges from 8.5% in men to 12.4% in women aged 65 years and over. It was also suggested that the UK folate deficiency rate falls within the 0-23.5% of free living and 0-68% of institutionalized elderly across Europe (Aytekin, Mileva, and Cunliffe 2018) concluding that European intake levels of folate are generally below RNIs.

At present, there is no legal requirement for the mandatory fortification of foods in the UK with folates. The UK Government has launched a public consultation into whether folate fortification should become mandatory, as per similar fortification schemes of wheat flour (The Bread and Flour Regulations 1998). However, this consultation is due to proposed reduction in neural tube defects of newborn children and not linked to general population deficiency (Department of Health and Social Care 2019), although could be a potential benefit.

Folate processing stability. The folates in food are highly susceptible to oxidation during storage and preparation, which is accelerated in the presence of heat, acid and metal ions, particularly copper (Nisha, Singhal, and Pandit 2005), somewhat similar to that of vitamin B_{12} (Combs 2008). The monoglutamate form of folic acid is most stable in heat but decomposes when in acid, whilst higher pH increases stability (Nisha, Singhal, and Pandit 2005). Nisha, Singhal, and Pandit (2005) found the degradation of folic acid in legumes to be limited up to 70 °C. A loss of 35.8% was seen at 120 °C after 60 minutes. The study suggested there was no significant difference in folic acid loss when considering cooking method as a variable.

Silveira et al. (2017) reviewed folic acid retention in fortified rice using different cooking methods. The lowest retention was found at 75.69% when cooked in a 300 L food service cauldron for 40 minutes. This is comparable to the results found by Nisha, Singhal, and Pandit (2005) although Silveira et al. (2017) suggested retention of 96.11% was possible using a stir-fry method. In contrast, Shrestha, Arcot, and Paterson (2003) found retentions of only 7-39% when boiling rice. This may be due to an increased use of water and time by Shrestha, Arcot, and Paterson (2003) as it is agreed that folic acid is liable to leaching during cooking similar to other water-soluble vitamins (Combs 2008; Shrestha, Arcot, and Paterson 2003; Silveira et al. 2017). Porasuphatana et al. (2008) provided support for this suggestion when a 92% retention of folic acid was found using a 5-minute boiling time.

One UK study reviewed the retention of folate in fresh vegetables and meat and found retention of 49% in boiled spinach and 44% in boiled broccoli (McKillop et al. 2002). Steaming was found to result in no significant difference compared to raw vegetables. The study further noted that grilling beef did not result in any significant loss (54.3 μg/100g raw compared to 51.5 μg/100g cooked) ($P < 0.05$). This further supports the evidence that cooking method and time contribute the most to retention, particular when cooked in water.

The summary of vitamins is given in Table 1.

Zinc

Zinc is a good reducing agent and is able to form stable complexes with other ions. It is also able to form salts, such as halides, carbonates, phytates and phosphates (Berdanier and Berdanier 2015). Zinc is present in some form in all body tissues and fluids and the total body content is estimated at 1.5-2.5g with most of this in tissue stores. Zinc is present as only one valence Zn^{++} unlike iron, which can be in a number of valences (Berdanier and Berdanier 2015).

Zinc absorption, bioavailability and food sources. UK RNI for zinc is 9.5 mg/day for men and 7.0 mg/day for women after the age of 18 years old. The requirement remains the same for all proceeding age groups (Public Health England 2016). Zinc absorption is agreed in literature as relatively

poor at between 10-40% of that which is ingested due to interactions with other chemicals such as phytate, reducing its bioavailability (Berdanier and Berdanier 2015; Maares and Haase 2020).

Zinc is found in numerous foods ranging from meat and poultry to seafood, legumes, nuts, seeds, eggs, fruits and vegetables. Meat and poultry provide up to 6.1 mg/100g zinc whilst some seeds can provide up to 7.8 mg/100g (Hotz and Brown 2004). Meat and poultry are high in protein and have been suggested to increase zinc absorption in the small intestine due to the proteins being hydrolyzed to peptides and amino acids. These smaller molecules are able to complex with zinc and increase its bioavailability (Lonnerdal 2000; Wapnir 2000; Maares and Haase 2020). Some reduction in bioavailability has been recognized in meat which has undergone the Maillard reaction, as the zinc is bound to ligands involved in the process (O'Brien and Morrissey 1997; Furniss et al. 1989).

On the contrary, although some nuts and seeds may have higher levels of zinc than meat, these products also contain phytate. Phytate is found in many cereal-based foods. Zinc can be bound to phytate and prevented from being absorbed, being described as the main nutritional inhibitor of zinc (Maares and Haase 2020). Foods can be described with a phytate: zinc molar ratio. Meat and dairy have a ratio of 0, whereas seeds can be up to a ratio of 88. It suggests that the higher the diet in animal protein, the more likely an adequate intake of zinc, compared with a seed and legume-based diet. Calcium can also decrease the bioavailability of zinc, particularly in the presence of phytate due to the formation of an insoluble calcium-phytate-zinc complex that is excreted in its entirety (Hotz and Brown 2004; Maares and Haase 2020). Fermentation and germination on the other hand, have both been shown to reduce phytate levels in susceptible products, supporting increased zinc availability (Sandberg and Andlid 2002; Kumar et al. 2010).

Zinc metabolic function and deficiency (including prevalence and physiological presentation). Zinc performs as a cofactor for over 70 enzymes binding to the histidine and cysteine. This complex exposes the active sites of the enzyme catalyzing reactions. Further, zinc has been found to have over 500 binding proteins linked to specific DNA sequences (Berdanier and Berdanier 2015; Vural et al. 2020).

Clinical features of zinc deficiency may include altered taste and smell, alopecia, increased infections and changes in skin condition (Berdanier and Berdanier 2015; Vural et al. 2020; Jenkins, Wainwright, and Green 2009). Zinc is active in inflammation responses due to its reducing capabilities and deficiency is linked to increased levels of cytokines and reduced antioxidant protection of cells (Vural et al. 2020). Jenkins, Wainwright, and Green (2009, 120) noted that zinc is also a cofactor in the production of collagen and therefore deficiency may impact wound healing due to the reduced inflammatory response as well as the delayed closure of sores. Vural et al. (2020) further suggested that zinc deficiency is linked to cardiovascular disease from increased oxidative stress and is important in the elderly that are deficient. Meunier et al. (2005) suggested a link in deficiency to osteoporosis as zinc is also linked to bone matrix mediation. These impacts of deficiency have also been identified in vitamin C and vitamin D.

The latest National Diet and Nutrition Survey (published in 2018) in the UK found prevalence of zinc deficiency was highest for women over 75 years old (Public Health England, 2018). 12% had intakes below the Lower Reference Nutrient Intake (LRNI), compared to 3% for those between 65 to 74 years old (N = 194). 5% men aged 65 to 74 years old had zinc below the LNRI, increasing to 8% for those older than 75 years (N = 141). The study does note that data for DRVs of zinc are limited and therefore the LRNI should be used with caution. A further observational study on institutionalized elderly in the UK measured intake by recording leftovers after meals. The observed rate of zinc deficiency against RNI was 34.8% for men and 18.6% for women over 3 days (N = 34) (Leslie et al. 2006). This study used a recipe analysis tool, which combines published standardized nutritional data and does not use analyzed samples. Vural et al. (2020) meta-analysis study of 11 published research papers across Europe, Brazil, USA and Australia concluded that of the pooled sample 31% of women (N = 4,038) and 49% men (N = 2,282) were consuming below the RNI for zinc. Further, 23% of this total population was considered institutionalized and deficiency rose to 50% for women and 66% for men.

Zinc processing stability. As zinc is a metal and present in Zn^{++} valence, decomposition of the mineral is not possible during cooking. Cooking losses may be seen due to leaching during cooking, as with other vitamins and minerals. One study found the lowest retention of zinc during boiling was 86.17% for cowpeas. The highest retention was 99.05% using pressure-cooking and the same soaking water (Pereira, Carvalho, and Dellamora-Ortiz 2014). A study on various cereals found that soaking for 24 hours prior to cooking equated to a significant reduction in zinc ($P < 0.05$) for millet, maize and rice (Lestienne et al. 2005). This study also found a significant reduction in phytate during soaking ($P < 0.05$) but the phytate: zinc molar ratio did not change significantly. A final study concluded that the cooking method does not appear to significantly affect the zinc content in common beans (*Phaseolus vulgaris* L.). A plausible theory is drawn in relation to soaking and the increased extraction of zinc from the beans. This is useful to consider when preparing products that may require long cook times in a lot of liquid. It was found that the cooking liquid contained an amount of zinc, so retaining the cooking liquid if technically viable, may be a way to reduce loss during processing (Carvalho et al. 2012).

Iron

Iron is the most abundant mineral found in the human body with up to 4 g in a 70 kg male and 2.6 g in a 60 kg female (Berdanier and Berdanier 2015). Iron is present mainly in blood in the form of hemoglobin, accounting for up to 65% of the body stores. It can be saturated with

oxygen for transport around the body and therefore essential to all aerobic processes (Berdanier and Berdanier 2015; World Health Organisation 2004).

Iron absorption, bioavailability and food sources. The body mediocrely absorbs iron, like zinc. It can be found in foods in its heme form in animal proteins and non-heme form in some plants such as spinach, quinoa and dried apricots. It is estimated that absorption from heme containing meals is 25% (World Health Organisation 2004). It is suggested that up to 85% of iron intake is in the non-heme form but this can vary depending on the composition of the meal as animal proteins and vitamin C will increase non-heme absorption (Berdanier and Berdanier 2015). In the UK, fortification of wheat flour with iron is mandatory (The Bread and Flour Regulations 1998) at not less than 1.65 mg/100g. This aims to increase dietary iron of the UK population in general as flour is used in a wide variety of products.

Iron uptake is also affected by the presence of phytate, certain fibers and zinc, which can limit absorption of the mineral. Many plant-based foods containing non-heme iron also contain phytate and fiber, but literature suggests iron absorption is dependent on the whole meal composition. This is due to the availability of inhibitors as well as enhancers, which may balance the availability of iron (Lopez and Martos 2004; World Health Organisation 2004). Heme iron, although more easily absorbed, is not compatible with enhancers so a balance of heme and non-heme sources is suggested as most beneficial (Berdanier and Berdanier 2015). In order to be absorbed the available iron must be in its ferrous state (Fe^{2+}) as the initial uptake of iron involves its conversion to ferritin and this may be another factor in the limited bioavailability of iron in food (Berdanier and Berdanier 2015).

Iron is absorbed in the intestines although this process is inefficient. The body's iron absorption has been suggested as almost a closed system due to the use and re-use of the mineral. The main cause of iron loss is through blood loss, which is why the UK RNI for females is higher pre-menopause and supplementation is suggested (Berdanier and Berdanier 2015; Public Health England 2016). The UK RNI for iron is 8.7 mg/day for men and women over 65 years old and does not change for later life stages (Public Health England 2016). It is estimated that only 10% of iron stores are lost in men per year equivalent to 1 mg/day, but slightly higher in females at 2 mg/day (Berdanier and Berdanier 2015). Since there is no specific excretion mechanism for iron, the control of iron homeostasis is controlled by the hepcidin hormone, formed in the liver. Damage to the liver may cause absorption issues (Abbaspour, Hurrell, and Kelishadi 2014).

Iron metabolic function and deficiency including prevalence and physiological presentation. Iron forms a key part of the production and role of hemoglobin in the body's blood system. Hemoglobin is a crucial component of red blood cells as it is able to bond with oxygen in the lungs and transport it to cells for use. It is then able to carry carbon dioxide from cells back to the lungs for removal. Hemoglobin has a high affinity for both oxygen and carbon dioxide with iron acting as the central atom and maintaining its structure and function (Berdanier and Berdanier 2015; Marengo-Rowe 2006; Abbaspour, Hurrell, and Kelishadi 2014). The life of a red blood cell is estimated at 120 days and therefore this may account for the minor losses of iron in the body per day although some will be recovered during the death of cells (Berdanier and Berdanier 2015). Other functions of iron include a role in wound healing, as could be expected with its role in blood (Jenkins, Wainwright, and Green 2009, 120: Landsdown 2001).

Clinical features of iron deficiency do not occur until there is a decrease in hemoglobin activity. Once this stage has been reached and body stores are not replenished, anemia becomes the main symptom (Berdanier and Berdanier 2015). Signs of anemia include a severe lack of energy, heart palpitations, changes in the skin and a shortness of breath (NHS 2018). One study suggests however, that deficiency can be severe without presentation with anemia (Abbaspour, Hurrell, and Kelishadi 2014). Other symptoms include 'restless leg syndrome' due to reduced brain iron levels and changes to nail formation, which can become spoon shaped. The most severe impact of iron deficiency is a marked increase in risk of morbidity (Berdanier and Berdanier 2015; Miller 2013).

Iron deficiency in the elderly has been found at 5.2% in one UK study (N = 3,816) in those aged over 65 years old, based on confirmation of anemia (Fairweather-Tait et al. 2014). Another UK cohort study over 14 years reviewed data and found prevalence of non-anemia iron deficiency (NAID) at 8.8% (N = 4,451). It further reported that prevalence was slightly lower in men but overall increased the risk of mortality by 1.58 times (95% CI [1.29, 1.93]) (Philip et al. 2020). No discrimination was made in either study between free-living and institutionalized elderly. It may be suggested however, that these studies used relatively large sample sizes and therefore the risk of anemia or NAID is of importance in the UK population. No studies were found which specifically focused on the iron status of institutionalized elderly.

Iron processing stability. Iron is similar to zinc in that it cannot be decomposed during processing. Studies that looked at zinc loss during cooking also considered iron. Pereira, Carvalho, and Dellamora-Ortiz (2014) found no significant difference in the amounts of iron that were lost when cooking beans in water ($P < 0.05$). Ferreira et al. (2014) also reported similar findings on beans of no significant difference. This suggested losses that were seen should be attributed to leaching during cooking. Another study reported similar findings in fortified rice where extra cooking water decreased iron retention, with pan-frying showing the highest retention at more than 90% (Losso et al. 2017).

One study reviewed cooking utensil material and found green vegetables cooked in an iron-based pot had an increased bioavailable iron content of up to four times their raw bioavailable content (Kumari et al. 2004). A further

Table 2. Summary of minerals based on reviewed literature.

Mineral	Sources	Function	Symptoms of deficiency	Processing stability
Zinc	Meat, nuts, seeds, fruit and vegetables	Cofactor enzyme to many reactions. Oxidative stress protection, bone health	Osteoporosis, cardiovascular disease, increased infection and reduced wound healing	Stable in heat but susceptible to leaching
Iron	Meat, some vegetables, fortified flour	Structural component of hemoglobin and red blood cell function	Anemia, skin changes, nail changes	Stable in heat but susceptible to leaching

study reported no significant difference in the iron content of pumpkin when steamed or boiled, compared to raw ($P < 0.05$) (Davidson, Ojukwu, and Anyaogu 2017). One study on beef found dry heat caused a reduction in heme iron of up to 43% at 200 °C for up to 8 minutes. Contrarily, the same study also found a significant increase ($P < 0.01$) in non-heme iron during the same cooking process. The study suggests that the overall bioavailable iron would decrease, as heme iron is more easily absorbed (Purchas et al. 2004).

The findings suggest that iron overall is relatively heat stable in vegetables and meat although the specific iron type may change in quantity and bioavailability. The largest effect on iron content seen were reported in studies where water was used as a cooking medium and therefore suggests leaching as a main contributor to the retention of iron in food.

The summary of minerals is provided in Table 2.

Summary and future remarks

This review has provided a summary of a selection of micronutrients and their associated deficiencies in the specific population of the elderly with dysphagia. It also considered the impact of processing on the retention of these micronutrients within foods, in the context of texture modified meal solutions. These meals may be more processed than non-texture modified versions, although resources and studies in this area are lacking. The limited research available on this specific population and subject area warrants further investigation, particularly as micronutrient status supports the correct physiological function, which is of growing importance as people age. Deficiency becomes more prevalent in respect of many micronutrients within the general elderly population, whilst those with underlying conditions (including dysphagia as a symptom) are more at risk of deficiency and the various health risks associated to decreased micronutrient status.

Texture modified meals have been improved and refined to increase consumption for those that require them, with much attention paid to the macronutrient status however, it could be suggested that further research is required in respect of micronutrients. Further research may include assessing processing parameters to ensure selected micronutrients are maintained or their bioavailability even enhanced through the production process. The ability to provide consistent, increased or even targeted micronutrient rich texture modified meals would particularly benefit the elderly by reducing or removing deficiency risks, lessening further health complications and supporting individuals maintaining better overall health.

Contributions

BGR, AAT and KDAT conceived the topic. BGR critically analyzed the existing literature and drafted the manuscript. AAT provided research support, guidance and critical review of the subject area and publication format. KDAT provided critical review and appraisal of work prior to submission.

Conflict of interest

None.

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