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Nutrition and protein energy homeostasis in elderly

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ABSTRACT

Protein-energy homeostasis is a major determinant of healthy aging. Inadequate nutritional intakes and physical activity, together with endocrine disturbances are associated with of sarcopenia and frailty. Guidelines from scientific societies mainly address the quantitative aspects of protein and energy nutrition in elderly. Besides these quantitative aspects of protein load, perspective strategies to promote muscle protein synthesis and prevent sarcopenia include pulse feeding, the use of fast proteins and the addition of leucine or citrulline to dietary protein. An integrated management of sarcopenia, taking into account the determinants of muscle wasting, i.e. nutrition, physical activity, anabolic factors such as androgens, vitamin D and n-3 polyunsaturated fatty acids status, needs to be tested in the prevention and treatment of sarcopenia. The importance of physical activity, specifically resistance training, is emphasized, not only in order to facilitate muscle protein anabolism but also to increase appetite and food intake in elderly people at risk of malnutrition. According to present data, healthy nutrition in elderly should respect the guidelines for protein and energy requirement, privilege a Mediterranean way of alimentation, and be associated with a regular physical activity. Further issues relate to the identification of the genetics determinants of protein energy wasting in elderly.

1. Introduction

In 2009, life expectancy without disability stood at 61.3 years for men in the European Union (EU27), which represents nearly 80% of their life expectancy at birth (76.7 years). Life expectancy without disability reached 62 years for women, representing three-quarters of their life expectancy at birth (82.6 years). Thus, a major challenge of current public health is to fight against the factors of disability in order to get an extension of life expectancy without disability. Disability is often associated with a situation known as "frailty", a state of vulnerability that is responsible for an inability to maintain a normal physiological balance and respond to stress. Frailty may be the result of dysfunction related to age or chronic disease. It affects the quality of life, undermines the autonomy, can compromise life at home without assistance and lead to institutionalization.

Thus, it is crucial to identify frailty at an early stage and take charge before it alters the autonomy of old people. The main signs that announce frailty are related to nutrition and activity: unintentional weight loss greater than 5% in one year, decreased muscle strength, appearance of unusual fatigue, reduced walking speed and physical activity. Protein-energy homeostasis is a major determinant of healthy aging. In community-dwelling people older than 70 years, 5–10% are undernourished, and for institutionalized elderly, this is up to 30–65% (van Staveren and de Groot, 2010; Cereda et al., 2011). It was shown that loss of lean tissues occurs exponentially with aging (Genton et al., 2011). A low fat-free mass index appeared as an independent factor of survival in still healthy old subjects (Genton et al., 2013). Sarcopenia was reported to be associated with physical disability in old men (Chien et al., 2010). Reduced nutritional intakes and physical activity, together with endocrine disturbances such as of low serum levels of dihydroepiandrosterone and, in men, bio available testosterone are associated with the risk of sarcopenia and frailty (Koutsari et al., 2009). Restrictive diets in patients over 75 increase the risk of protein energy wasting and, at an individual level, need to be reassessed (Zeanandin et al., 2011). Finally, vitamin D deficiency and associated parathormone changes were shown to be associated with frailty in men (Shardell et al., 2009).

Guidelines from scientific societies mainly address the quantitative aspects of protein and energy requirements in elderly and aim to prevent sarcopenia and associated osteoporosis

Abbreviations: AA, amino acids; AEE, activity energy expenditure; BEE, basal energy expenditure; DEE, daily energy expenditure; FFM, fat-free mass; FM, fat mass; REE, resting energy expenditure; WPIL, whey proteins providing the same amount of leucine; WPIN, isonitrogenous whey proteins.

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(Verschueren et al., 2013). However, recent data have shown body composition abnormalities such as fat overload, and age-specific changes in protein, energy and micronutrient metabolism that could intervene in the risk of malnutrition and deserve qualitative recommendations for nutrition in elderly aiming at decreasing the incidence of nutrition-connected chronic diseases (van Staveren and de Groot, 2010; Luhrmann et al., 2009).

2. Characteristics of energy metabolism in elderly

In old people, energy expenditure and insulin sensitivity can be altered.

2.1. Energy expenditure in elderly

In a cross-sectional study conducted in 529 adults aged 18–96 years, the associations between body composition and daily (DEE), basal (BEE), and activity energy expenditure (AEE) were examined throughout the adult life span (Speakman and Westerterp, 2010). DEE was measured by using doubly labelled water, BEE by using respirometry, and body composition by isotope dilution. AEE was calculated as DEE–BEE, and physical activity level as DEE/BEE. Up to age 52 years, fat-free mass (FFM) and fat mass (FM) were positively associated with age in men, but no significant effect was observed in women. Subjects aged more than 52 years were characterized by lower values of FFM, FM, DEE, BEE and AEE. BEE similarly decreased in women (−22 kJ/day/year) and men (−45 kJ/day/year). It was noticeable that the decrease in AEE was much greater than the decrease in BEE, showing that physical activity was strongly dependent on age for those subjects. No relation was found between age-adjusted physical activity level and FFM suggesting that, in elderly, greater physical activity was not associated with higher FFM. Similar longitudinal changes in energy expenditure, remarkable by the decrease in physical activity, were observed during a 12-year follow-up of an elderly German population (Luhrmann et al., 2009). An association between AEE, appetite and survival was noted among well-functioning, community-dwelling older adults underlining that appetite assessment could provide important information regarding the risk for health deterioration and mortality in elderly as in chronic organ diseases (Carrero et al., 2007; Shahar et al., 2009). In some healthy aging subjects, the simultaneous decline in physical activity and calorie intake can result in the lack of variation in body weight (Sarti et al., 2012).

The decrease in physical activity in elderly is associated with the occurrence of falls in community-dwelling adults over 50 years old (Pereira et al., 2013). Consistently, studies of energy expenditure and activity levels using the multisensor SenseWear Pro Armband® showed that older men with lower energy expenditure, lower activity, or greater sedentary time were more likely to develop a functional limitation (Cawthon et al., 2013). In both community-dwelling and institutionalized older adults, the beneficial effect of physical activity was demonstrated. As a matter of fact, physical activity improved different domains of both the physical and mental components of quality of life questionnaires and decreased depressive symptoms (Salguero et al., 2010). Resistance exercise, which can increase REE and serum adiponectin in overweight elderly individuals, may represent an effective approach for weight management and metabolic control in overweight elderly individuals (Fatouros et al., 2009). The determinants of AEE in elderly are poorly understood. Recently, mitochondrial DNA variation was shown to be associated with free-living AEE in older persons suggesting that specific mitochondrial DNA complexes, genes, and variants may contribute to the maintenance of activity levels in late life (Tranah et al., 2012).

2.2. Energy metabolism and predisposition to insulin resistance

The size and distribution of fat depots dramatically change throughout life. Increased body fatness can be largely explained by changes in energy metabolism favouring the occurrence of positive energy balance. First, hormonal changes, mainly in women during menopause (Ferraro et al., 1992; Morio et al., 1997), and age-related muscle loss (Tzankoff and Norris, 1978) are responsible for diminished basal metabolism rate. Furthermore, and partially as the consequence of the first point, a lower level of physical activity is an important factor associated to the reduced energy expenditure during aging (Paivi et al., 2010). Increased body fatness is strongly involved in the development of metabolic disorders in the elderly. In that context, prevalence of insulin resistance, i.e. impaired insulin sensitivity, increases in elderly human.

During the past decade, numerous studies have explored the potential association between the reduction in insulin sensitivity and the age-related diminished energy-metabolism, with a specific focus on muscle mitochondrial functioning. Indeed at the whole body level, insulin resistance is associated to low aerobic fitness and high body fatness, which have been linked to the decrease in muscle mitochondrial oxidative capacity (review in Phielix et al., 2011). At the cellular level, insulin resistance has been correlated to lipid accumulation in myocytes, which could be the result of deterioration in mitochondrial oxidative capacity (Johannsen et al., 2012). Debates still exist with regards to the origin of mitochondrial alterations and how they relate to the development of insulin resistance. This is probably due to the fact that mitochondria are complex organelles not limited to substrate oxidation and energy production. However, increasing evidence suggests that changes in insulin sensitivity and mitochondrial function may not be causally related, but mutually amplify each other during aging (Karakelides et al., 2010; review in Phielix et al., 2011). We here briefly develop arguments supporting this conclusion.

Impact of aging on muscle mitochondria functioning. In the elderly, alterations in energy metabolism may relate to low mitochondrial oxidative capacity. Most studies in animals evidenced that mitochondrial oxidative capacity decreases with aging. By contrast, adverse effects of aging on muscle energy metabolism in humans are not unanimously accepted (review in Chanseaume et al., 2010). Several key pathways altered with aging have been identified. They include decreased mitochondrial density, mitochondrial oxidative capacity and ATP production rate. In addition, several studies have shown that a decrease in the fractional synthesis rate of mitochondrial proteins may participate to the age-related decline in mitochondrial oxidative capacity (review in Chanseaume et al., 2010). It remains controversial, however, whether the reduction in mitochondrial oxidative capacity is due to a consequence of aging or to unhealthy lifestyle. Indeed, physical inactivity and insulin resistance are characteristics of the aging process and those two situations can contribute to the muscle mitochondrial dysfunction related to aging.

Impact of insulin resistance on muscle mitochondria functioning. It was shown that acute insulin infusion stimulates muscle mitochondrial protein synthesis (Halvatsiotis et al., 2002; Stump et al., 2003) as well as mitochondrial biogenesis, oxidative capacity and ATP production (Stump et al., 2003). Furthermore, insulin resistance has been related to decreased stimulation by insulin of muscle mitochondrial protein fractional synthesis rate (Guillet et al., 2009) as well as mitochondrial ATP production (Stump et al., 2003; Petersen et al., 2005). An impaired insulin-stimulated phosphate transport was also reported in muscle, which may contribute to the defects in insulin-stimulated rates of mitochondrial ATP synthesis (Petersen et al., 2005). Finally, withdrawal of insulin in type 1 diabetic patients decreased muscle mitochondrial ATP production rate and expression of oxidative phosphorylation