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SARCOPENIA – BRIEF CHARACTERISTICS, ETIOLOGY AND POSSIBLE THERAPY

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SUMMARY

Sarcopenia is considered to be a geriatric syndrome with multifactorial etiology: mitochondrial dysfunction, replication defects in mitochondrial DNA that lead deficit in energy production and muscle fibre atrophy, changes in protein synthesis, imbalance between protein degradation and the ability of muscle fibre to new proteins synthesis, changes in secretion and plasmatic levels of hormones (growth hormone, androgens, insulin etc.), metabolic syndrome or imbalance in antioxidant system and many more (Buford, et al., 2010). We are still far from being able to fully understand the causes and characteristics of sarcopenia as well as to solve this problem. However, an appropriate lifestyle (movement activity) and appropriate diets (energy restriction) seem to be the most effective therapeutic approaches (Morley, et al., 2010).

Key words: sarcopenia, geriatric syndrome, physical activity, energetic restriction

INTRODUCTION

One of the numerous aging symptoms is gradual atrophy of skeletal muscles. As a consequence of this atrophy there occurs a significant reduction in muscular strength during lifetime which can negatively influence mobility in the elderly, especially the ones who evince signs of lower muscular strength in their productive age (Rantanen, et al., 1999). Progressive reduction in muscular strength and muscle mass significantly contribute to reduction of physical performance, to falls and fractures regarding seniors. Insufficient muscular strength also means a significant risk factor for mortality and a requirement for assistance during essential daily activities (Laukkanen, Heikkinen, & Kauppinen, 1995; Rantanen, Avlund, Suominen, Schroll, Frändin, & Pertti, 2002). Finding effective means how to prevent or at least temper muscular atrophy caused by aging is very important. In relation to aging of the population in developed countries the problems of immobility in the elderly come into great significance mostly because the means spent on senior care institutionalisation extend to amounts of many millions (Cruz-Jentoft, Landi, Topinková, & Michel, 2010).

Therefore it is understandable that according to seriousness of this problem there were considerable means invested to a research engaged in revealing mechanisms having a dominant influence on developing muscular atrophy caused by aging aimed at creating its effective therapy. In connection with that a new clinical unit called sarcopenia was defined in the end of 1990's. This multifactorial disease is currently classified as the so called geriatric syndrome (Kalvach, et al. 2008). In this mini review we are going to discuss the factors influencing system and cellular mechanisms which contribute to skeletal muscle atrophy and we are going to state several current overviews of medical treatments related to sarcopenia.

CAUSES OF SARCOPENIA

Sarcopenia can be simply defined as reduction of muscular matter related to aging. It is a natural process, but its development can be accelerated by many circumstances more or less genetically conditioned. Comorbid diseases common for older people such as ischemic heart disease, atherosclerosis, metabolic syndrome, cancer, kidney disease, diabetes mellitus and others (Buford, et al., 2010) including iatrogenic effects accompanying such diseases very often (Kalvach, et al., 2008) can be involved in the progress of sarcopenia. Furthermore, bad nutrition and insufficient physical activity can be involved too (DiTano, Fulle, Pietrangelo, Bellomo, & Fanò, 2005). A characteristic feature of sarcopenia is a reduced ability of muscular cells to produce energy necessary for performing their physiological functions. Identifying the biological mechanisms responsible for pathophysiological changes of sarcopenia is currently the main aim not only for geriatrists but also for other medical or nonmedical disciplines. In relation to sarcopenia as a geriatric syndrome with multifactorial etiology there occurred many experimental and clinical studies aimed at revealing dominant causes of this illness. In spite of the fact that it is not possible to accurately determine the main cause of sarcopenia, as one of the major factors of muscular matter reduction can be considered insufficient movement activity which is along with genetic predispositions mentioned in numerous studies (Buford, et al., 2010; Hedayati, 2010; Kim, et al., 2009; Marzetti, Lees, Wohlgemuth, & Leeuwenburgh, 2009; Muscaritoli, et al., 2010; Siervo, Stephan, Nasti, & Colantuoni, 2011; The, Suchindran, North, Popkin, & Gordon-Larsen, 2010). Other mechanisms contributing to the etiology of sarcopenia are oxidative stress and muscle damage by free radicals (Meng & Yu, 2010). Another frequently discussed topic is mitochondrial dysfunction. Long-term inflammations, oxidant stress and insulin resistance may significantly contribute to mitochondrial changes which can be modulated by changes in the nutrients intake and the nutrition state (Barazzoni, 2011). Mitochondria – as a cell energy generator have an important role among other cell organelles. There exists epidemiologic evidence that the mitochondrial dysfunction can occur at many chronic diseases connected with reduction in muscular matter (Paul, et al., 2009). It was exactly this fact that lead to contemplations that the mitochondrial dysfunction in skeletal muscles can significantly contribute to the loss of muscular matter and play an important role in the etiology of sarcopenia. Moreover, the results of experimental studies support the hypothesis that mutations of mitochondrial DNA influence the effectiveness of respiratory

chain enzymes which can cause a decrease in oxidative phosphorylation and also contribute to the development of sarcopenia (Hiona, et al., 2010). Aging is among all characterized as a loss of spinal motor neurons as a consequence of apoptosis. In relation to this loss a part of muscular fibres is paralysed which leads to reduction of muscular function and subsequent atrophy (Aagaard, Suetta, Caserotti, Magnusson, & Kjær, 2010; Deschenes, Mackenzie, Eason, & Brennan, 2010; Sacheck, et al., 2007). Sarcopenia is also connected with a low level of androgenic hormones and most importantly correlates with a low level of testosterone. Several clinical studies have already implied a causal connection between low levels of testosterone and reduced muscular strength regarding in older men (Schmidt, et al., 2010). Moreover, a positive influence of androgens on women after the menopause was experimentally proved (Saad, 2011). It is also connected with a reduced amount of androgenic receptors which is related to age (Schmidt, et al., 2010). Another contributing factor is predominance of myostatin determined by a testosterone deficit which plays a major role in skeletal muscle mass regulation (Ratkeviciu, et al., 2011). Also insulin, as a major humoral cause regulating the level of blood glucoses, influences by its anabolic effect the metabolism of skeletal muscles. A reduction in susceptibility to insulin connected with age can therefore negatively affect the metabolism of muscular proteins and participate in the etiology of sarcopenia (Jung, et al., 2008). However, the results of experimental studies imply that the effect of insulin does not play any major role in the development of sarcopenia regarding healthy, non-obese women after the menopause (Goulet, Lord, Chaput, Aubertin-Leheudre, Brochu, & Dionne, 2007). There is also a chronic inflammation connected with sarcopenia which produces anti-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor alpha (TNF-α) which are considered to negatively influence the appendicular muscles (Aagaard, Suetta, Caserotti, Magnusson, & Kjær, 2010; Witham, Sumukadas, & McMurdo, 2008). Cytokines play major role during immunopathological reactions. It is being considered that the genetically determined ability to produce a certain kind of cytokines is the base of developing the immunopathological states (Klener, et al., 1997). And the anorectic effects of anti-inflammatory cytokines can through their effects play an important role in the etiology of sarcopenia (Meng & Yu, 2010). A long-term inflammation can negatively influence skeletal muscles by a direct catabolic effect or through indirect mechanisms (i.e. by lowering the growth hormone concentration and insulin-like growth factor 1 (IGF-1)). The decreased level of IGF-1 is connected with sarcopenia, fragility and mortality in the elderly (Renganathan, Messi, & Delbono, 1998). Growing age brings muscles preferential aging with predominance of fibres type II, therefore the reduction of muscle mass during sarcopenia is disproportional. The loss of mobility of fast muscular fibres significantly limits primarily the function of phasic muscles, by which the locomotion quality is somehow influenced. Limited ability to perform fast movements as a reaction to external impulses emerging from selective reduction of muscular fibres type II then contributes, regarding seniors, to several risks of injuries connected with falls. The following connection with sarcopenia is to some extent mediated by the limited cardiopulmonale capability emerging from the worsened mobility of seniors or from a long-term stay in bed (Chien, Huang, & Wu, 2008; Chien, Kuo, & Wu, 2010). The reduction of muscle mass is then retrospectively in causal connection with cardiopulmonale capability because pure weight without fat (so called FFM = fat free mass) and muscular

strength significantly correlate with aerobic capacity (Jacó de Oliveira, et al., 2009). Coexistence of these conditions leads to creating a vicious cycle in which the decline in physical activity in connection with reduction of lowers basal metabolism and the lowered energetic demands of organism lead to increased energetic substrates storage in the adipose tissue. As a consequence there occurs the adipose tissue hypertrophy of which adipocytes secret an increased amount of anti-inflammatory catabolic cytokines and therefore fasten the muscular matter loss (Marzetti, Lees, Wohlgemuth, & Leeuwenburgh, 2009; Li & Reid, 2000; Tsujinaka, et al., 1996). An increased prevalence of sarcopenia regarding obese people is often connected with metabolic complications which increase the risk of cardiovascular diseases (Barbat-Artigas, Fex, Karen's, Dionne, & Aubertin-Leheudre, 2010; Kim, et al., 2009). However, a slight overweight has a protective effect against muscle mass loss and sarcopenia regarding healthy women (Kirchengast & Huber, 2010). Another potential factor of sarcopenia can be malnutrition. A dysfunction of mechanisms regulating food intake occurs during the old age and leads to increasing the feeling of hunger and limiting the food intake. The lowered food intake in the organism also leads to a deficit of some essential substances such as vitamins and trace elements which natural supply to organism is limited by the food reduction. Subsequent hypovitaminosis can also contribute to muscular atrophy. For instance, there exists an epidemiologic evidence which connects the vitamin D deficit with sarcopenia (Marzetti, Lees, Wohlgemuth, & Leeuwenburgh, 2009; Morley, et al., 2010). To conclude this humble and by far not so comprehensive enumeration it is necessary to name chronic alcoholism and cigarettes as potential circumstances of the sarcopenia etiology (Lee, Auyeung, Kwok, Lau, Leung, & Woo, 2007).

POSSIBLE PREVENTION AND TREATMENT OF SARCOPENIA

Recently, several studies focused on research of biological mechanisms directly dependent on chronological aging or comorbid diseases in the background of sarcopenia. Based on the results achieved it is currently possible to suggest various medical strategies. However, it is probable that the therapy of sarcopenia in context with a treatment of life threatening diseases where muscular atrophy is only a symptom does not play a key role, even though an effective support of muscular trophy as a means to increase the resistance of organism is very useful in this field. However, it is possible to say that research in this field is still required because revealing physiologic effects of various forms of interventions in relation to sarcopenia has not been comprehensively clarified yet. According to the experimental studies results there are several possibilities of interventions for an effective therapy such as a movement programme based on exercising of various intensity, diet precautions, pharmacologic treatment, which will not be discussed in this context, and various combination of the above mentioned solutions. According to the movement programme it is necessary to ask which form of exercising is the most effective to increase the muscular strength, more precisely to enlarge the cross-section of muscular fibres. There are two basic possibilities – aerobic exercise or resistant training. Both can improve the function of appendicular muscles and at the same time increase the cross-section of muscle fibres also in very old people (Witham, Sumukadas, & McMurdo, 2008). It has

been proven that the resistance training increase muscular hypertrophy along with important changes in neuromuscular function regarding also very high-aged people (> 80 years) (Aagaard, Suetta, Caserotti, Magnusson, & Kjær, 2010). Moreover, an intensive neuromuscular activity can postpone the myofibrillar denervation related to age (Deschenes, Mackenzie, Eason, & Brennan, 2010). On the contrary, aerobic exercising increases mitochondrial biogenesis and the overall oxygen consumption (Barazzoni, 2011) and is also connected with better insulin sensivity (Petersen & Pedersen, 2005). The alternative to previous solutions can be a passive load of atrophied muscles through vibration methods on special apparatuses (Jelen, Zeman, Kubový, Drahorádová, & Holub, 2007). However, the knowledge of correct dosage and timing of exercising must be supported by another research. Nevertheless, it seems that one set of a certain exercise done to a complete muscular exhaustion once a week is regarding old people as effective as exercising twice a week (DiFrancisco-Donoghue, Werner, & Douris, 2007). Another highly discussed topic is the influence of various diet precautions on the development of sarcopenia. As the results of some studies imply that the energy restriction (reducing the amount of food while keeping its biological quality) regarding adult men and women causes beneficial metabolic, hormonal and functional changes (Fontana & Klein, 2007). Some recent studies have shown that the energy restriction can support muscular mitochondrial biogenesis regarding middle-aged and high-aged people (Marzetti, Lees, Wohlgemuth, & Leeuwenburgh, 2009). However, it is essential that the following research examine whether the energy restriction is a safe and effective strategy to postpone the beginning of sarcopenia and reduce its development. To keep the volume of muscle mass a balanced intake of all essential nutrients is preferred – proteins, fats and carbohydrates. According to some studies the protein intake should, regarding older people, be higher than recommended (0.8 g/kg/day) for regular population in order to keep the muscle function and strategic amino acid reserves in organism. An adequate intake is app. 1.5 g/kg of proteins a day, however, special situations to which sarcopenia belongs can require a higher amount of proteins (Wolfe, Miller, & Miller, 2008). An adequate intake of proteins itself only retards the muscle mass loss but in combination with exercise (resistant or aerobic), with an adequate intake of energy and balanced intake of vitamins and trace elements is a key part of sarcopenia prevention and treatment (Morley, et al., 2010).

CONCLUSION

Finding appropriate methods for sarcopenia treatment (also as a part of other diseases) based on the knowledge of causal relation with pathophysiological influences in the background of this disease has become an aim for many specialists. Research in the area of effective prevention and therapy should consider all factors which manipulation can stop or at least slower the development of sarcopenia. One of these factors is the lifestyle of afflicted people. Behavioural influences, sedentary lifestyle, redundant energy intake or insufficient vitamins and trace elements intake are factors which can be to some extent influenced. As a key factor of sarcopenia prevention is individual physical activity. It plays more important role than nutrition. However, it seems that the best precaution is to

motivate the afflicted to accept the positive lifestyle and better eating habits. Nevertheless, a lot of old people are not willing or able to do the exercises, therefore it is necessary to examine appropriate ways how to motivate them to change their lifestyle.

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SARKOPENIE – STRUČNÁ CHARAKTERISTIKA, ETIOLOGIE A MOŽNOSTI TERAPIE

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SOUHRN

Sarkopenie je považována za geriatrický syndrom s multifaktoriální etiologií: mitochondriální dysfunkce, chyby replikace v mitochondriální DNA, které vedou k energetickému deficitu a atrofii svalových vláken, změny syntézy proteinů, nerovnováha mezi degradací proteinů a schopností svalového vlákna k syntéze nových proteinů, změny v sekreci a plazmatických hladinách hormonů (růstový hormon, androgeny, inzulín aj.), metabolický syndrom nebo nerovnováha v antioxidačním systému a mnoho dalších (Buford, a další, 2010). Jsme ještě daleko od úplného pochopení příčin a charakteristik sarkopenie i od řešení tohoto problému. Nicméně vhodný životní styl (pohybová aktivita) a vhodné diety (energetická restrikce) se zdají být nejúčinnější terapeutické přístupy (Morley, a další, 2010).

Klíčová slova: sarkopenie, geriatrický syndrom, fyzická aktivita, energetická restrikce

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