# Body Composition Variations in Ageing 

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#### Abstract

Age-related physiological variations of body composition concern both the fat-free mass (FFM) and the fat mass (FM). These variations expose the elderly person to the risk of malnutrition and could lead to conditions of disability. This paper aims to review the current state of knowledge on body composition in the aged population. The pattern of qualitative variations in body composition in old age is fairly well defined. In adulthood, the physiological variation of body mass involves a first increasing phase followed by a decreasing trend. The reduction is due mainly to the loss of fat-free mass, especially muscle mass. Total body water and bone mass also decrease. Fat mass tends to decrease and the reduction seems to be due mainly to the loss of subcutaneous fat. The quantitative aspects of the age of onset, rate and intensity of the physiological variations are still not completely clear. This poor quantitative definition is due to the variable and multifactorial phenomenology of ageing, the heterogeneity of assessment techniques and sampling models, and the limited number of empirical observations in oldest-old individuals.


Key words: sarcopenia, osteopenia, dehydration, malnutrition

## Introduction

Ageing is a continuous and gradual process. It is characterized by great variability among populations, among individuals and among organs of the same individual. This variability involves different rates and ways of ageing, and it depends on environmental, cultural and genetic characteristics as well as the presence or absence of pathological conditions.

Knowledge of body composition variations in ageing comes mainly from research on populations of industrialized countries (Table 1). The few cross-cultural studies show that, in spite of the significant quantitative differences in body composition among different ethnic groups ${ }^{1}$, inter-population age-related patterns of variation are very homogeneous ${ }^{2-12}$.

Age-related physiological variations of body composition concern both the fat-free mass (FFM), which includes the skeleton, muscles and body water, and the fat mass (FM). These variations expose the elderly person to the risk of malnutrition and could lead to conditions of disability ${ }^{13-16}$. In fact, reduced muscle mass and bone mass influence the nutritional, functional, endocrine and cognitive status, as well as the comorbidity. On the other hand, overweight is often associated with limitations of function and mobility ${ }^{17,18}$. Moreover, visceral obesity is
related to cardiovascular disease and diabetes. The classic U-shaped curve, showing the relationship between body mass index (BMI) and mortality, derives from the different and independent effects of the deficiency of FFM (left part of the curve) and the excess of FM (right part $)^{19}$. However, it has been observed that BMI values associated with the risk of mortality increase with age and that overweight is linked to better survival in advanced age ${ }^{20}$. This pattern of relations has been shown by the Baltimore Longitudinal Study on Aging ${ }^{21}$ and more recently by the National Longitudinal Study of Canadian Adults ${ }^{22}$.

The definitions proposed in the literature for ageing distinguish physiological decline from disease-related variations ${ }^{23}$. In the absence of disease, the ageing process is generally referred to as »normal« or »physiological«. »Primary« ageing is also used ${ }^{24}$. Successful ageing is used when deleterious effects are minimized ${ }^{25}$. In the presence of diseases, which occur with high frequency in elderly people, the terms »usual«, »pathological« and »secondary« are used.

A major goal of geriatrics and gerontology is the definition of body composition variability in ageing, with the aim of distinguishing the »physiological« from the »pa-

[^0]thological« condition. The last few decades have seen a marked increase in research on age-related changes in body composition, although the over 80 -year-old population is still relatively poorly studied. Moreover, the studies have been conducted with different techniques and different experimental sampling approaches. Data have been obtained mainly from cross sectional studies, or short term follow-up, which are relatively simple and economical. The age-related changes are inferred from the comparison of the mean values of the different cohorts. As discussed by Arking ${ }^{26}$, this procedure has several serious drawbacks: it does not distinguish the effects of ageing from the effects of the cohort (it may be that each cohort undergoes different and specific growth conditions, as exemplified by secular trend phenomena); it also suffers from the effects of selective mortality.

From the theoretical point of view, the data of a longitudinal study, which can also be reorganized in the form
of cross sectional data, are generally considered more reliable than the data of a cross sectional study. However, the longitudinal approach is also not immune from drawbacks: it does not distinguish the effects of ageing from the effects of the period; it often uses samples selected on the basis of socio-economic characteristics; it requires long-term economic and human resources ${ }^{26}$. Table 1 shows an annotated selection of longitudinal studies carried out in various parts of the world on anthropometric and body composition variations in the elderly population.

A good methodological approach is to integrate cross sectional and longitudinal studies. A recent interesting study ${ }^{27}$ simultaneously examined the effect of age and the effect of birth cohort due to the growing obesity epidemic. The authors found that, at the same age, later cohorts had greater fat mass than earlier cohorts (increase of $\%$ FM per birth year: $0.32 \%$ in men and $0.16 \%$ in women).

TABLE 1
ANTHROPOMETRIC AND BODY COMPOSITION LONGITUDINAL STUDIES

| Survey | Acronym | Country | Start year | Sample size | Age group* | Variables (methods) |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Australian Longitudinal Study of Ageing | ALSA | Australia | 1992 | 2,087 | 70+ | A |
| Baltimore Longitudinal Study on Aging | BLSA | USA | 1958 | 3,000 | 20-100 | A, BC <br> (dilution techniques, plethysmography) |
| Canadian Multicentre Osteoporosis Study | CaMos | Canada | 1996 | 9,423 | 25+ | A, BC (DXA) |
| Cardiovascular Health Study | CHS | USA | 1989 | 5,888 | $65+$ | BC (BIA, DXA) |
| EURONUT-SENECA Study | SENECA | Europe | 1988 | 2,586 | 75-80 | A |
| The Fels Longitudinal Study | FELS | USA | 1976 | 210 | 40+ | A, BC (underwater weighing) |
| Fredericton 80+ Study | Fredericton | Canada | 1998 | 238 | 80 | A |
| Göteborg Study | H70 | Sweden | 1971 | 1,148 | 70 | A, BC (DXA, BIA, neutron activation analysis, body count of potassium) |
| Health, Aging \& Body Composition Study | Health ABC | USA | 1997 | 3,075 | 70-79 | A, BC (CT, DXA) |
| Invecchiare in Chianti Study | InCHIANTI | Italy |  | 923 | $65+$ | A, BC (CT) |
| Italian Longitudinal Study on Aging | ILSA | Italy | 1992 | 5,493 | 65-84 | A |
| Longitudinal Aging Study Amsterdam | LASA | Netherlands | 1992 | 3,017 | 55-85 | A, BC (BIA) |
| Maastricht Aging Study | MAAS | Netherlands | 1992 | 2,043 | 24-81 | A |
| MacArthur Study of Successful Aging | MacArthur | USA | 1988 | 1,189 | 70-79 | A |
| National Institute for Longevity Sciences Longitudinal Study of Aging | NILS-LSA | Japan | 1997 | 2,300 | 40-79 | A, BC (DXA) |
| National Population Health Survey | NPHS | Canada | 1994 | 17,276 | 0+ | A |
| NHANES I Epidemiologic Follow-up Study | NHEFS | USA | 1971 | 14,407 | 25-74 | A |
| Normative Aging Study | NAS | USA | 1963 | 2,280 (M) | 21-81 | A |
| Rancho Bernardo Study | Bernardo | USA | 1972 | 1,000-6,000 | 20+ | A |
| Rotterdam Study | Rotterdam | Netherlands | 1990 | 7,983 | $55+$ | A, BC (DXA) |
| The New Mexico Aging Process Study | NMAPS | USA | 1978 | 780 | 65-98 | A |

[^1]In general, the incompleteness of the data and the heterogeneity of experimental designs and assessment techniques indicate the need of meta-analyses aimed at defining the state of knowledge and the major goals of research on variations of body composition in the aged population.

This paper aims to review the current state of knowledge on body composition in the aged population.

## Age-related Weight and BMI Variations

Most studies on ageing analyze weight and BMI variations (a selection of longitudinal studies in Table 1). Research conducted in industrialized countries indicates a tendency to an increase of body weight and BMI throughout life. The Fels longitudinal study estimated an age-related increasing trend from 40 to 66 years of age in both men (average annual rate: weight, $0.3 \mathrm{~kg} /$ year; BMI, 0.11 $\mathrm{kg} / \mathrm{m}^{2} /$ year) and women (average annual rate: weight, $0.55 \mathrm{~kg} /$ year; BMI, $0.22 \mathrm{~kg} / \mathrm{m}^{2} /$ year $)^{28}$. The increase remains virtually constant until the time at which the trend reverses.

The age at which the weight reduction begins has still not been defined, with estimates varying between 60 and 80 years. The weight loss is more precocious in men ${ }^{11}$. It has been determined that this weight decrease involves over $60 \%$ of individuals ${ }^{29}$. However, the reduction is not constant and there may be oscillations, as recently shown in the longitudinal Health, Aging, and Body Composition Study ${ }^{30}$.

The Göteborg H70 longitudinal study showed that BMI decreased significantly in both sexes after age 70, and there was a gender difference in the trend, as females decreased more than males ${ }^{31}$. The cross sectional survey from the InCHIANTI study showed that BMI increased with age up to 45-54 years in men and up to 65-74 years in women, after which it declined ${ }^{32}$. The Italian Longitudinal Study on Aging showed that BMI decreased in both sexes between 65 and 85 years; the trend was more regular in men and particularly after 75 years ${ }^{33}$.

The aetiology of weight loss in the elderly is complex. It may be voluntary or involuntary. The most important voluntary factors are the restriction of food intake and/or the increase of physical activity. Involuntary factors include sarcopenia, starvation and cachexia. Sarcopenia only affects FFM and can be considered, within limits, a physiological phenomenon. Starvation, related to the reduction of nutritional intake, is due to the anorexia of the elderly and results in a decrease of FFM and FM. Finally, cachexia is a condition of severe weight loss secondary to pathological conditions such as cancer and immunodeficiency syndrome; also in this case, the weight loss involves both FFM and FM.

## Age-related Fat-free Mass Variations

The FFM progressively increases in the first phases of the life-cycle and reaches a peak in the fourth decade, after which it begins to decrease. The reduction of FFM is
the most important part of the involuntary weight variation in the elderly ${ }^{34,35}$. The age-related FFM loss is smaller in active than sedentary individuals, as shown by the Longitudinal Research in Healthy Swiss Adults ${ }^{36}$ and by the Fels Longitudinal Study ${ }^{28}$. There is no general consensus on the magnitude and mean rate of the FFM decrease. As reviewed by Evans ${ }^{37}$, FFM decreases by around $15 \%$ between the third and eighth decade. The review by Young ${ }^{38}$ shows that the rate of decrease of FFM is $6.3 \%$ per decade and the percentage reduction can reach $30 \%$. According to the longitudinal study by Hughes et al. ${ }^{29}$, after 60 years of age FFM decreases in men $(2.0 \%$ per decade) but not in women. Strikingly similar estimates were found in the FELS Longitudinal Study ${ }^{39}$.

Studies of body composition at the molecular level show that the reduction of total body protein is particularly evident after 65 years and can be estimated at 5\% overall (see the review by Heymsfield et al. ${ }^{40}$ ). Studies at the atomic level show that total body potassium decreases at a rate of $7.20+/ 1.00 \mathrm{mg} / \mathrm{kg}$ per year in women and $9.16+/ 0.96 \mathrm{mg} / \mathrm{kg}$ per year in men ${ }^{41}$.

## Muscle mass

The depletion of FFM is largely attributable to the muscle component. Sarcopenia, i.e. the progressive and irreversible reduction of muscle mass and strength, is a widely documented process ${ }^{35}$, due to the numerical reduction of motoneurones and atrophy of muscle fibres, above all the IIa type. The process is more marked in $m^{42}$ and in the appendicular component ${ }^{43}$. With age, there is also adipose infiltration of the muscle mass, which helps reduce motor efficiency. The aetiology of sarcopenia is not completely clear. The most important proposed causal factors are the deterioration of neuromuscular functionality, the decline of muscle fibre contractility, variations in the levels of anabolic hormones, apoptosis and traumas. A sedentary lifestyle, smoking and an inadequate intake and/or reduced use of proteins contribute to the progression of sarcopenia.

Sarcopenia causes a significant decrease of motor performance and can become clinically important. It can activate a course of involution that causes the clinical syndrome of frailty, an invalidating condition associated with functional decline that exposes the elderly person to the risk of serious complication, loss of self-sufficiency and institutionalization. The risk is greater when elderly people are simultaneously sarcopenic and obese, as shown by The New Mexico Aging Process Study ${ }^{34}$.

The standard method for determining the level of sarcopenia is DXA (dual-energy x-ray absorptiometry). A diagnostic threshold can be identified using the skeletal muscle mass index (appendicular muscle mass divided by height squared, $\mathrm{kg} / \mathrm{m}^{2}$ ), i.e. the value 2 standard deviations below the mean of young adults. Baumgartner et al. ${ }^{44}$ calculated corresponding cut-off values of $7.26 \mathrm{~kg} / \mathrm{m}^{2}$ for men and $5.45 \mathrm{~kg} / \mathrm{m}^{2}$ for women in the cross sectional New Mexico Elder Health Survey. The prevalence of sarcopenia in the over- 80 population was $53 \%$ in men and $43 \%$ in women. On the basis fat-free mass estimates
by bioelectric impedance analysis, a lower prevalence of sarcopenia ( $16 \%$ in men, $13 \%$ in women) was found in the over-85 sample from the Rancho Bernardo study ${ }^{45}$.

However, DXA, like other imaging techniques (CT, NMR) and biochemical indicators, cannot easily be used in routine investigations and is thus unsuitable for screening and monitoring of the nutritional status of the elderly. In contrast, other techniques (anthropometry, bioelectrical impedance, dynamometric and motor tests) have the advantage of being simple, economical and non-invasive. The conventional bioimpedance method can be used to estimate the appendicular muscle mass. Nevertheless, the predictive efficacy of the BIA regression equations is significantly influenced by age, sex, population, health status and validation method. Even the use of age-specific equations can lead to substantial estimation errors. Indeed, aged subjects show great individual variability in the density of mineral mass, hydration and protein content of FFM. The alternative approach of bioelectrical impedance vector analysis (BIVA) is potentially more accurate, as it is not based on regression equations ${ }^{46}$. BIVA has been effectively used to describe physiological ageing ${ }^{47}$. However, the definition of cut-off values is required for validation of the procedure. Other indicators, such as knee extension isometric torque, handgrip and lower limb muscle power have been proposed to measure sarcopenia.

## Bone mass

The skeletal component of FFM shows important variations throughout life. Bone mass and density increase until the third decade, after which there is a progressive decrease, called »osteopenia ${ }^{111,48,49}$. According to Heymsfield et al. ${ }^{40}$, the mineral content of bones in people over 65 is $20 \%$ less than in 19-34-year-olds. The pattern of variation of the skeletal compartment is similar in men and women until 50 years ( $0.7-1 \%$ per year); following menopause, the decrease of the quantity and density of the bones becomes much faster in women $(2-3 \%$ per year $)^{11}$. The rate of bone loss increases in both sexes after 70 years of age ${ }^{11}$.

A major cause of osteopenia is estrogen deficiency, although calcium and vitamin D deficiencies and secondary hyperparathyroidism may also contribute to the pathogenesis. Family history, alcohol consumption, smoking habits, physical activity and nutritional factors have been shown to affect bone mineral density (BMD), directly or indirectly ${ }^{49}$. The body weight, particularly appendicular skeletal muscle mass, and the BMI are positively correlated with bone density ${ }^{50,51}$. A low BMI represents a risk factor for fracture, as recently confirmed by the Canadian Multicentre Osteoporosis Study ${ }^{52}$. This association seems to be directly related to mechanical load forces on bone ${ }^{53}$. The effect of fat mass on bone is less clear. However, the Longitudinal Aging Study Amsterdam showed that FM is associated with BMD ${ }^{54}$.

Osteopenia may turn into osteoporosis, a disease characterized by low bone mass and structural deterioration of bone tissue, which can lead to bone fragility, and an in-
creased susceptibility to fractures of the hip, spine and wrist. The NHANES I epidemiologic follow-up study ${ }^{55}$ and the NILS-LSA longitudinal study ${ }^{56}$ showed a significant relationship between BMD and mortality risk. The loss of height due to osteoporosis, associated with senile kyphosis, compression of the intervertebral discs and other pathologies, has been quantified as $0.5-5 \mathrm{~cm}$ per decade (see the discussion of this topic by Perissinotto et al. ${ }^{33}$ ). According to the $\mathrm{WHO}^{57}$, osteoporosis is defined as a BMD that lies 2.5 standard deviations or more below the mean value for young healthy women. The gold standard for the diagnosis of osteoporosis is DXA.

## Body water

Physiological ageing is associated with several changes that may affect water balance and expose older adults to the risk of dehydration. These changes include the decline in renal function and thirst perception, and the reduction of total body water (TBW). Body water decreases in parallel with the reduction of FFM, especially the intra-cellular compartment ${ }^{58}$. Heymsfield et al. ${ }^{40}$ estimated a TBW reduction of $12 \%$ in people over 65 with respect to 19-34-year-olds. As reviewed by Schoeller ${ }^{59}$, TBW is relatively constant until middle age and then begins to decline. In men, the loss is $0.3 \mathrm{~kg} /$ year until $70-80$ years, while in women it is more intense, especially after age $70(0.7 \mathrm{~kg} /$ year $)$. Literature data on the level of hydration of FFM (ratio between TBW and FFM) are not sufficient to provide a definitive picture. As reviewed by Wang et al. ${ }^{60}$, controversy exists as to whether FFM hydration is influenced by age. According to Heymsfield et al. ${ }^{40}$, the hydration of FFM passes from $72.1 \%$ in young adults to $71.2 \%$ in the elderly. In contrast to Heymsfield's data ${ }^{40}$, Baumgartner et al. ${ }^{61}$ and Bossingham et al. ${ }^{62}$ found that the TBW/FFM ratio was lower in the younger subjects than in the older ones.

Despite the strong influence of dehydration on health and disease, it is generally under-diagnosed ${ }^{63}$. While an increased volume of fluids is associated with oedema, a reduced volume does not result in evident and specific clinical manifestations, especially in mild and moderate cases. The difficulty of diagnosis is even greater in elderly subjects, since physical signs of dehydration (e.g. reduced skin turgor and orthostatic hypotension) are often present in normally hydrated older people. Biochemical parameters of dehydration (serum concentrations of sodium, urea and creatinine, urea/creatinine ratio, osmolality) are also of limited value in monitoring the fluid balance in aged individuals because of their variability and a lack of diagnostic standardization.

The gold standard method for the assessment of total body water (TBW) and extracellular and intracellular water is based on the dilution principle. Nevertheless, dilution methods are rather expensive, time-consuming and not readily available. Hence, they are not suitable for use in routine clinical practice. Bioelectrical impedance analysis, and its variants MFBIA (multi-frequency BIA) and BIS (bioelectrical impedance spectroscopy) can be valuable tools to assess changes in body hydration. How-
ever, the medical applications of the conventional BIA are limited by the already discussed problem of the specificity of the equations. Besides, the vector approach (BIVA) has proved particularly useful to detect hydration status in all pathological conditions with alterations of the water compartment ${ }^{46}$.

## Age - Related Fat Mass Variations

Fat mass increases progressively during adulthood because of the reduction of overall energy expenditure. According to the longitudinal study by Hughes et al. ${ }^{29}$, in the $7^{\text {th }}$ decade, FM increases similarly in both sexes (7.5\%). The mixed protocol adopted by Ding et al. ${ }^{27}$ shows that there is a decrease of fat accumulation at about 80 years of age, which is more accentuated in women than in men. This causes a reduction of the sexual dimorphism typical of adulthood (more fat mass in women).

Ageing is typically associated with an increase of the visceral fat component. In both sexes, marked fat redistribution occurs between 45 and 54 years ${ }^{32}$. Men show a centripetalization and internalization of fat ${ }^{64,65}$. Women are characterized by a peripheral distribution of fat (less visceral adiposity). However, the sex steroid hormone changes associated with menopause induce a more android fat distribution ${ }^{66}$. Few studies have looked at visceral fat variations in oldest-old individuals. According to the cross sectional study of Perissinotto et al. ${ }^{33}$, waist circumference decreases significantly in both sexes after 75 years of age. Herrera et al. ${ }^{7}$ found a reduction of sexual dimorphism.

The subcutaneous fat component, which increases until the 7th decade, tends to decrease thereafter ${ }^{67,68}$. The EURONUT SENECA longitudinal investigation showed that age-changes in skinfold thickness are small ${ }^{69}$. The variations in fat patterning between males and females and among different age groups are still not well defined. A cross sectional study by Buffa et al. ${ }^{70}$ showed that the typical dimorphic distribution of subcutaneous fat (peripheral in women, central in men) changes with age and becomes more homogeneous in the two sexes. Herrera el al. ${ }^{7}$ found that males and females appear to be more similar for triceps, subscapular and suprailiac skinfolds at advanced ages.

From the clinical point of view, the regional modifications of fat mass are most remarkable. A central pattern of fat distribution is associated with the most important cardiovascular risk factors, predisposes to metabolic syndrome and contributes to the worsening of respiratory functions. This relationship is confirmed by various epidemiological studies. The Normative aging study showed a significant relation between abdominal fat and cardiovascular and diabetes risk ${ }^{71}$. The Rotterdam study showed that waist circumference may have more potential as a predictor of all-cause mortality than the $\mathrm{BMI}^{72}$. The Health, Aging And Body Composition Study showed that abdominal obesity and hyperglycaemia are predictive of mobility limitations in the elderly ${ }^{73}$.

Variations of visceral fat are described by means of DXA, imaging techniques and anthropometric indicators (circumference and sagittal diameter of the waist, waist--to-hip ratio, conicity index). There are sex-specific cut--offs for estimating visceral obesity based on waist circumference ( 88 cm women; 102 cm men) ${ }^{74}$. However, age-related differences in body fat distribution may overestimate visceral obesity in elderly individuals. On the basis of waist circumference, over $75 \%$ of the elderly women examined in the Italian Longitudinal Study on Aging (ILSA) were obese ${ }^{33}$. The results of the Rotterdam Study indicated that cut-offs based on analyses of mid-dle-aged and younger adults are only useful to a limited degree in older populations ${ }^{75}$.

## Conclusion

The pattern of qualitative variations of body composition in old age is fairly well defined. During adulthood, the physiological variation of body mass involves an initial increasing phase followed by a decreasing trend. The reduction is due mainly to the loss of fat-free mass. Sarcopenia is the major causal factor of the FFM loss, particularly in men. These phenomena are associated with a decrease of total body water, especially the intra-cellular compartment. Bone mass also tends to decrease, especially in women. Fat mass, both the visceral and subcutaneous components, increases throughout adulthood, although it tends to decrease in advanced age, mainly due to a reduction of subcutaneous fat. All these modifications expose the elderly person to the risk of malnutrition.

The quantitative aspects of the age of onset, rate and intensity of the physiological variations are still not completely clear, and there are various reasons for this poor definition. One problem is the limited number of empirical observations in oldest-old individuals and the paucity of cross-cultural studies. However, the main difficulty is the variable and multifactorial phenomenology of ageing. Meta-analyses aimed at a detailed definition of the physiological variations of body composition are hindered by the intrinsic heterogeneity of the ageing process and by the need to consider numerous significant confounding factors. Moreover, there is a lack of methodological standardization of the assessment techniques, as well as difficulty in interpreting and integrating the information deriving from cross sectional and longitudinal studies.

The poor quantitative definition of the physiological variations makes it difficult to define cut-off values indicating the transition to pathological conditions, which are necessary to develop preventive strategies for body composition-related diseases in old age. Further research on healthy elderly men and women from different ethnic groups, particularly the over-80 population, and the application of mixed models for cross sectional-longitudinal studies will help to better define the vague boundary between physiology and pathology.

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## REFERENCES

1. ELLIS KJ, Biol Trace Elem Res, 26-27 (1990) 385. - 2. BISAI S, BOSE K, KHATUN A, BAURI H, J Life Sci, 1 (2009) 21. - 3. CAMPBELL B, GRAY PB, LESLIE P, Am J Hum Biol, 17 (2005) 601. - 4. COQUEIRO RDA S, BARBOSA AR, BORGATTO AF, Nutrition, 25 (2009) 33. - 5. GHOSH A, BOSE K, CHAUDHURI AB, Ann Hum Biol, 28 (2001) 616. - 6. GHOSH A, Coll Antropol, 28 (2004) 553. - 7. HERRERA H., REBATO E., ROCANDIO A.M., HERNANDEZ R., RODRIGUEZ N., BARBOSA J., HERNANDEZ-VALERA Y, Invest Clin, 46 (2005) 139. - 8. MENEZES TN, MARUCCI MDE F, Cad Saude Publica, 23 (2007) 2887. - 9. MOTT JW, WANG J, THORNTON JC, ALLISON DB, HEYMSFIELD SB, PIERSON RN Jr, Am J Clin Nutr, 69 (1999) 1007. - 10. SINGAL P, SIDHU LS. Anthropol Anz, 41 (1983) 179. - 11. SPIRDUSO WW, FRANCIS KL, MACRAE PG, Physical dimensions of aging (Human Kinetics, Champaign, 2005). - 12 . TYAGI R, KAPOOR S, KAPOOR AK, Coll Antropol, 29 (2005) 493. - 13. BUFFA R, FLORIS G, MARINI E, Nutrition, 25 (2009) 3. - 14. GILLETTE-GUYONNET S, VELLAS B, Mech Ageing Dev, 124 (2003) 247. - 15. HARRIS TB, Am J Epidemiol, 15 (2002) 122. - 16. SARKISIAN CA, GRUENEWALD TL, JOHN BOSCARDIN W, SEEMAN TE, J Am Geriatr Soc, 56 (2008) 2292. - 17. BANNERMAN E, MILLER MD, DANIELS LA, COBIAC L, GILES LC, WHITEHEAD C, ANDREWS GR, CROTTY M, Public Health Nutr, 5 (2002) 655. - 18 . VISSER M, LANGLOIS J, GURALNIK JM, CAULEY JA, KRONMAL RA, ROBBINS J, WILLIAMSON JD, HARRIS TB, Am J Clin Nutr, 68 (1998) 584. - 19. BIGAARD J, FREDERIKSEN K, TJØNNELAND A, THOMSEN BL, OVERVAD K, HEITMANN BL, SØRENSEN TI, Obes Res, 12 (2004) 1042. - 20. KALANTAR-ZADEH K, HORWICHB TB, OREOPOULOSC A, KOVESDYD CP, YOUNESSIE H, ANKERF SD, MORLEY JE, Curr Opin Clin Nutr Metab Care, 10 (2007) 433. - 21. BRANT L, FOZARD J, METTER E, Age differences in biological markers of mortality. In: BALIN A (Ed) Practical handbook of human biologic age determination (CRC Press, Boca Raton, 1994). - 22. ORPANA HM, BERTHELOT JM, KAPLAN MS, FEENY DH, MCFARLAND B, ROSS NA, Obesity, 18(1) (2010) 214. - 23. BEERS MH, BERKOW R (Eds) Merck Manual of Geriatrics, Merck \& Co., accessed 7/15/2009. Available from: URL: http://www.merck.com/pubs/. - 24. BUSSE EW, Theories of aging. In: BUSSE EW, PFEIEFFER E (Eds) Behaviour and adaptations in later life. (Little Brown, Boston, 1969). - 25. ROWE JW, KAHN RL, Science, 237 (1987) 143. - 26. ARKING R, Biology of aging (Sinauer Associates, Sunderland, 1998). - 27. DING J, KRITCHEVSKY SB, NEWMAN AB, TAAFFE DR, NICKLAS BJ, VISSER M, LEE JS, NEVITT M, TYLAVSKY FA, RUBIN SM, PAHOR M, HARRIS TB, THE HEALTH ABC STUDY, Am J Clin Nutr, 85 (2007) 405. - 28. GUO SS, ZELLER C, CHUMLEA WC, SIERVOGEL RM, Am J Clin Nutr, 70 (1999) 405. - 29. HUGHES VA, FRONTERA WR, ROUBENOFF R, EVANS WJ, FIATARO-NE-SINGH MA Am J Clin Nutr, 76 (2002) 473. - 30. LEE JS, KRITCHEVSKY SB, HARRIS TB, TYLAVSKY F, RUBIN SM, NEWMAN AB, Am J Clin Nutr, 82 (2005) 644. - 31. DEY DK, ROTHENBERG E, SUNDH V, BOSAEUS I, STEEN B, Eur J Clin Nutr, 53 (1999) 905. - 32. BARTALI B, BENVENUTI E, CORSI AM, BANDINELLI S, RUSSO CR, DI IORIO A, LAURETANI F, FERRUCCI L, Soz Praventivmed, 47 (2002) 336. - 33. PERISSINOTTO E, PISENT C, SERGI G, GRIGOLETTO F, Br J Nutr, 87 (2002) 177. - 34. BAUMGARTNER RN, Ann N Y Acad Sci, 904 (2000), 437. - 35. THOMAS DR, Clin Nutr, 26 (2007) 389. - 36. KYLE UG, MELZER K, KAYSER B, PICARD-KOSSOVSKY M, GREMION G, PICHARD C, J Am Coll Nutr, 25 (2006) 493. - 37. EVANS WJ, Clin Geriatr Med, 11 (1995) 725. - 38. YOUNG VR, Nutr Rev, 50 (1992) 454. - 39. SIERVOGEL RM, WISEMANDLE W, MAYNARD LM, GUO SS, ROCHE AF, CHUMLEA WC, TOWNE B, Arterioscler Thromb Vasc Biol, 18 (1998) 1759. - 40. HEYMSFIELD SB, WANG J, LICHTMAN S, KAMEN Y, KEHAYIAS J, PIERSON RN JR, Am J Clin Nutr, 50 (1989) 1167. - 41. KEHAYIAS JJ, FIATARONE MA, ZHUANG H, ROUBENOFF R, Am J Clin Nutr, 66 (1997) 904. - 42. GALLAGHER D, RUTS

E, VISSER M, HESHKA S, BAUMGARTNER RN, WANG J, PIERSON RN, PI SUNYER FX, HEYMSFIELD SB, Am J Physiol Endocrinol Metab, 279 (2000) E366. - 43. KYLE UG, GENTON L, HANS D, KARSEGARD VL, MICHEL JP, SLOSMAN DO, PICHARD C, J Am Geriatr Soc, 49 (2001) 1633. - 44. BAUMGARTNER RN, KOEHLER KM, GALLAGHER D, ROMERO L, HEYMSFIELD SB, ROSS RR, GARRY PJ, LINDEMAN RD, Am J Epidemiol, 147 (1998) 755. - 45. CASTILLO EM, GOODMAN-GRUEN D, KRITZ-SILVERSTEIN D, MORTON DJ, WINGARD DL, BARRETT-CONNOR E, Am J Prev Med, 25 (2003) 226. - 46. PICCOLI A, ROSSI B, PILLON L, BUCCIANTE G, Kidney Int, 46 (1994) 534. - 47. BUFFA R, FLORIS G, MARINI E, Nutrition, 19 (2003) 917. 48. CUMMINGS SR, NEVITT MC, BROWNER WS, STONE K, FOX KM, ENSRUD KE, CAULEY J, BLACK D, VOGT TM, N Engl J Med, 332 (1995) 767. - 49. LIM S, JOUNG H, SHIN CS, LEE HK, KIM KS, SHIN EK, KIM HY, LIM MK, CHO SI, Bone, 35 (2004) 792. - 50. BARRERA G, BUNOUT D, GATTÁS V, DE LA MAZA MP, LEIVA L, HIRSCH S, Nutrition, 20 (2004) 769. - 51. GILLETTE-GUYONNET S, NOURHASHEMI F, LAUQUE S, GRANDJEAN H, VELLAS B, Gerontology, 46 (2000) 189. - 52. DE LAET C, KANIS JA, ODÉN A, JOHANSON H, JOHNELL O, DELMAS P, EISMAN JA, KROGER H, FUJIWARA S, GARNERO P, MCCLOSKEY EV, MELLSTROM D, MELTON LJ 3RD, MEUNIER PJ, POLS HA, REEVE J, SILMAN A, TENENHOUSE A, Osteoporos Int, 16 (2005) 1330. - 53. EDELSTEIN SL, BARRETT-CONNOR E, Am J Epidemiol, 138 (1993) 160. - 54. PLUIJM SM, VISSER M, SMIT JH, POPP--SNIJDERS C, ROOS JC, LIPS P, J Bone Miner Res, 16 (2001) 2142. 55. MUSSOLINO ME, MADANS JH, GILLUM RF, Ann Epidemiol, 13 (2003) 692. - 56. SUZUKI T, YOSHIDA H, Osteoporos Int, 21 (1) (2010) 71. - 57. WHO, WORLD HEALTH ORGANIZATION, Assessment of fracture risk and its application to screening for postmenopausal osteoporosis. Report of a WHO Study Group. WHO Technical Report Series, No. 843 (World Health Organization, Geneva, 1994). - 58. STEEN B, J Nutr Health Aging, 1 (1997) 142. - 59. SCHOELLER DALE A, Am J Clin Nutr, 50 (1989) 1176. - 60. WANG Z, DEURENBERG P, WANG W, PIETROBELLI A, BAUMGARTNER RN, HEYMSFIELD SB, Am J Clin Nutr, 69 (1999) 833. - 61. BAUMGARTNER RN, HEYMSFIELD SB, LICHTMAN S, WANG J, PIERSON RN JR, Am J Clin Nutr, 53 (1991) 1345. - 62. BOSSINGHAM MJ, CARNELL NS, CAMPBELL WW, Am J Clin Nutr, 81 (2005) 1342. - 63. THOMAS DR, TARIQ SH, MAKHDOMM S, HADDAD R, MOINUDDIN A, J Am Med Dir Assoc, 4 (2003) 251. - 64. BORKAN GA, HULTS DE, GERZOF SG, ROBBINS AH, Am J Phys Anthropol, 66 (1985) 289. - 65. BOSE K, Coll Antropol, 26 (2002) 179. - 66. KUK JL, LEE S, HEYMSFIELD SB, ROSS R, Am J Clin Nutr, 81 (2005) 1330. - 67. HUGHES VA, ROUBENOFF R, WOOD M, FRONTERA WR, EVANS WJ, FIATARONE-SINGH MA, Am J Clin Nutr, 80 (2004) 475. 68. ZIVICNJAK M, SZIROVICZA L, PAVICIĆ L, SMOLEJ-NARANCIĆ N, JANIĆIJEVIĆ B, MILICIĆ J, RUDAN P, Coll Antropol, 21 (1997) 117. - 69. DE GROOT CP, PERDIGAO AL, DEURENBERG P, Eur J Clin Nutr, 50 (1996) S9. - 70. BUFFA R, FLORIS G, MARINI E, PORCU A, Caratteristiche antropometriche di un campione di popolazione senile della Sardegna. In: GUERCI A, CONSIGLIERE S (Eds) Vivere e curare la vecchiaia nel mondo - Curare la vecchiaia (Erga Edizioni, Genova, 2002). 71. GRINKER JA, TUCKER KL, VOKONAS PS, RUSH D, Int J Obes Relat Metab Disord, 24 (2000) 1369. - 72. VISSCHER TL, SEIDELL JC, MOLARIUS A, VAN DER KUIP D, HOFMAN A, WITTEMAN JC, Int J Obes Relat Metab Disord, 25 (2001) 1730. - 73. PENNINX BW, NICKLAS BJ, NEWMAN AB, HARRIS TB, GOODPASTER BH, SATTERFIELD S, DE REKENEIRE N, YAFFE K, PAHOR M, KRITCHEVSKY SB. J Gerontol A Biol Sci Med Sci, 64 (2009) 96. - 74. NIH, NATIONAL INSTITUTE OF HEALTH NIH Publication, 98 (1998) 4083. - 75. MOLARIUS A, SEIDELL JC, VISSCHER TL, HOFMAN A. J Am Geriatr Soc, 48 (2000) 1638.

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## VARIJACIJE U TJELESNOJ KOMPOZICIJI S OBZIROM NA STARENJE

## SAŽETAK

Fiziološke varijacije u tjelesnoj kompoziciji s obzirom na dob podrazumijevaju i nemasno i masno tkivo. One izlažu starije osobe riziku od pothranjenosti i mogu dovesti do invaliditeta. Cilj ovog rada je dati pregled trenutnih spoznaja o tjelesnoj kompoziciji u starijoj populaciji. Obrazac kvalitativnih varijacija u tjelesnoj kompoziciji kod starijih osoba je relativno dobro definiran. U odrasloj dobi, fiziološke varijacije tjelesne mase uključuju prvotnu fazu povećanja, koju slijedi trend smanjenja mase. Do smanjenja dolazi prvenstveno zbog gubitka nemasnog tkiva, pogotovo mišićne mase. Također se smanjuje udio vode i koštanog tkiva u tijelu. Masno tkivo se također reducira, za što se zaslužnim smatra prvenstveno gubitak potkožne masti. Kvantitativni aspekti koji uključuju dob početka fizioloških varijacija te njihovu učestalost i intenzitet još uvijek nisu posve razjašnjeni. Razlog tome je varijabilnost i multifaktorska fenomenologija starenja, heterogenost tehnika procjenjivanja i modela uzorkovanja te ograničen broj empiričkih opažanja kod pojedinaca starije dobi.


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[^1]:    * Age group represents the age at the baseline examination. A - anthropometric measurements, BC - body composition, CT - computed tomography, DXA - dual energy x-ray absorptiometry, BIA - bioelectrical impedance analysis, M - men, W - women

