Physiological vs. Pathological Changes of Nutritional Status over Life Time

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Introduction

Age-related changes in nutritional status include weight loss as well as weight gain. In the EURONUT-Seneca study on elderly subjects (age 75-80 years) who were investigated in different parts of Europe, the prevalence of a 5-year weight gain of >5 kg was highly variable and reached a highest prevalence of 9 and 17% in females and males, respectively [1]. By contrast the prevalence of age-related weight loss (>5 kg in 5 years) reached 27 or 37% in females and males, respectively. Concomitantly, underweight (i.e. a body mass index (BMI of <20 kg/m²) was seen in up to 24% of elderly subjects, whereas obesity was observed in up to 38% [1]. These data suggest that in the elderly there are two different clinically relevant manifestations of nutritional status. First, there is frequent weight loss and sarcopenia, which is explained by anorexia related to chronic diseases, depression or age-related changes in physiologic functions (e.g. impaired regulation of appetite). Weight loss and sarcopenia are associated with functional impairment, reduced muscle strength and immune function, and thus increased morbidity and mortality. Second, weight gain and an increase in fat mass (FM) are due to a sedentary life style. This manifestation is associated with a high prevalence of chronic metabolic diseases (e.g. non-insulin-dependent diabetes mellitus and metabolic syndrome).

Age-related changes in nutritional status include alterations in body composition. Thus, FM as well as fat-free mass (FFM) and thus body cell mass all
change with age. There are a number of determinants of body composition including dietary intake, physical activity and inactivity, heritability, age, chronic diseases and drugs (fig. 1). The contribution of the different determinants differs between individuals and also between different age groups resulting in a heterogeneity of physiological and pathophysiological changes in nutritional status over a life time.

**Methodological Issues**

Different methods can be used to characterize nutritional status. Faced with recent developments in body composition research, there is no gold standard in techniques used to assess different body compartments. In addition there is no unique parameter characterizing all aspects of body composition. Considering age-related changes in nutritional status, the different methods also differ with respect to accuracy and validity. Comparing BMI, bioelectrical impedance-derived FM and FM measured by densitometry (which is considered as one of the most suitable methods to assess fat mass) [2] in a group of 129 healthy women in Kiel (age range 18–84 years) the age-related increase in FM can only be seen by the use of densitometry (fig. 2). These data suggest that considering age-related changes in body composition, one has not only to consider the changes in the ‘true’ phenomena but also the methodological limitations of the methods used to assess body composition.

Using bioelectrical impedance analysis (BIA) in a greater group of subjects differing with respect to BMI and age, it becomes evident that BIA-derived FM increases with BMI. However, at a given BMI, FM decreases with age. The age-related differences in FM may reach 5%. BIA measures two variables,
i.e. resistance (the detected current is weaker than the source current) and reactance (i.e. the detected current lags behind the source current). Standard BIA algorithms only use resistance but ignore reactance. Considering the BMI- and age-related changes in resistance (R) and reactance (Xc), R to height and Xc to height both decrease with BMI. However, at a given BMI there is no effect of age on R/height. By contrast at a given BMI, Xc to height decreases with age. It becomes evident that the R/Xc vector downslopes with age. Thus standard BIA algorithms cannot be used to address age-related changes in body composition. Age-related changes in Xc reflect changes in body cell mass and cannot be used as measures of FM or other obesity-related components of body composition. Taken together this method is also not suitable to measure interrelations between energy intake, output and stores. Besides the high quality of BIA measurements (i.e. its high precision), the accuracy (i.e. the level of agreement between the measured value and the true value) is low and differs between age groups.

**Metabolic Impact of Age-Related Changes in Nutritional Status**

Age-related changes in BMI, FFM and FM are highly variable. Considering cross-sectional data, FFM and FM in kilograms both increase up to age 40–60 years and then decrease again. When compared to FM this decrease is more pronounced in FFM. Considering the age-related changes in FFM and FM as percent body weight, there is a continuous decrease in FFM with age. By contrast FM increases with age. Both changes are more pronounced in subjects >60 years.

FFM is the major determinant of resting energy expenditure (REE). Throughout the lifespan REE increases (up to age 40–60 years) and decreases

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**Fig. 2.** Age-related changes in body mass index (BMI), %fat mass\textsubscript{BIA} and %fat mass\textsubscript{ADP} in 129 women in Kiel, Germany. BIA = Bioelectrical impedance analysis; ADP = air-displacement plethysmography, densitometry.
Adjusting REE for FFM and/or FFM + FM, most of the age-related changes in REE disappear suggesting that age-related changes in metabolic rate are most likely explained by changes in body composition.

There is a close association between FFM and REE (R² between 0.50 and 0.80) [3–7]. Considering the association between REE and FFM in different age groups there is a progressive sloping of the REE–FFM relationship in adults of different ages [8]. These data also suggest that the ‘specific’ metabolic rate (i.e. REE/kg FFM) decreases with increasing age (fig. 3).

This finding can be explained by two different factors. First, there may be an age-related decrease in specific metabolic rate. Second, the composition of FFM may change with age thus affecting metabolic rate. Anatomically as well as metabolically FFM is heterogeneous. About 50% of FFM can be explained by metabolically active tissues and organs. By contrast bone, extracellular mass and plasma volume have a very low or no metabolic activity (i.e. <1 kcal/kg FFM x day but add to 50% of FFM. In a normal-weight man skeletal muscle mass adds to about 88% of metabolically active FFM (FFMm). Since skeletal muscle has a specific metabolic activity of about 15 kcal/kg FFMm x day⁻¹ but visceral organs reach a mean value of 468 kcal/kg FFMm x day⁻¹, the composition of FFM (i.e. the ratio between organs with a low and organs with a high metabolic rate) adds to the metabolic variance between subjects [4].

Fig. 3. Age dependency of the resting energy expenditure (REE) vs. fat-free mass (FFM) relationship. From Müller et al. [8].
Using modern body composition assessment technologies, one can directly address this question in young and elderly subjects [4]. Skeletal muscle and bone mass can be measured by DEXA. In addition magnetic resonance imaging (MRI) can be used to take transversal images at different distances at a slice thickness of 6–8 mm with a technical error of 0.7–1.1%. Determination of cross-sectional organ areas can be done by hand or using a computer program. Calculation of organ volumes is performed from the sum of cross-sectional areas

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\frac{\text{slice thickness}}{H} \]

Organ mass is then calculated from organ volumes

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\frac{\text{organ densities as taken from the literature (e.g. 1.036 g/cm}^3 \text{for brain and 1.06 g/cm}^3 \text{for heart muscle). Table 1 shows detailed body composition data in young and elderly subjects [6].}

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<thead>
<tr>
<th></th>
<th>Females</th>
<th>Males</th>
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<tr>
<td></td>
<td>young</td>
<td>elderly</td>
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<td>FM_{DXA}, kg</td>
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<td>27.6 ± 5.4**</td>
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<td>16.5 ± 2.1*</td>
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<td>Bone mass_{DXA}, kg</td>
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<td>Organ mass_{MRI}, kg</td>
<td>3.8 ± 0.3</td>
<td>2.9 ± 0.2*</td>
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<td>Brain, kg</td>
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<td>1.1 ± 0.1**</td>
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<tr>
<td>Heart, kg</td>
<td>0.28 ± 0.0</td>
<td>0.36 ± 0.1**</td>
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<tr>
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<td>Residuals, kg</td>
<td>10.3 ± 1.8</td>
<td>11.4 ± 3.4</td>
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According to Bosy-Westphal et al. [6].

*p < 0.05; **p < 0.01.

Physiological vs. Pathological Changes

Since REE is a function of body composition, it can be modeled from detailed body composition analysis: REEc (kJ/day) = 1,008 × brain + 840 × liver + 1,848 × heart + 1,848 × kidneys + 55 × muscle + 19 × FM + 1 × bone + 50 × residual mass. REEm (measured REE) was lower than REEc. When compared with young adults the difference was significantly greater in the elderly (0.60 vs. 0.11 MJ/day, p < 0.01) [6].

FFM showed a close association with the sum of organs, liver, kidney and spleen masses. Comparing the regression lines observed in young and elderly subjects, there were no differences in the slopes but organ masses at a given FFM were lower in the elderly (fig. 4). By contrast heart mass per FFM was higher in the elderly. In elderly subjects 60% of the variance in REEm–REEc was explained by heart mass. The difference between REEm and REEc between age groups disappeared after exclusion of elderly subjects with cardiac hypertrophy. Exclusion of 5 subjects with cardiac hypertrophy (i.e. a heart weight of >500 g) resulted in a reduced REE prediction error (i.e. in
the elderly the difference between REEm and REEc reached $-0.10\, \text{MJ/day}$ which is close to the data observed in young subjects) [6]. The study has certain limitations. It was assumed that specific organ metabolic rates are constant with increasing age and increasing organ mass. In addition organ composition and density was assumed to be constant too.

To summarize, the age-related decline in REE is mainly explained by a reduction in FFM as well as by alterations in FFM composition. Overestimation of REE in subjects with cardiac hypertrophy suggests a decrease in specific heart metabolic rate with increasing heart mass.

**Heritability of Nutritional Status and Metabolic Function**

There are genetic effects on nutritional status and metabolism. Heritability describes the additive genetic effect. It can be estimated from data on twins or parent–child relationships [9]. Heritability is a population parameter reflecting the extent of the contribution of genes to the individual phenotype. Heritability does not address genotype–environmental interactions (i.e. differences in the sensitivity of individuals to environmental or lifestyle changes). Heritability adds to physiological and pathophysiological changes in nutritional status and metabolism during the lifespan. Preliminary analysis of data from an ongoing family path study in Kiel which included data sets from 53 families (grandparents, parents, children) suggests heritability estimates of 0.42, 0.44 and 0.47.
for BMI, FFM and waist circumference, respectively. Thus heritability of nutritional status is between 40 and 50%. This is close to the estimates of the Quebec family path study [10]. By contrast 50–60% of the age-related changes in nutritional status are due to the effects of age itself, lifestyle and disease-related factors. These factors also include possible gene–environment interactions.

Regarding metabolism the heritability of REE was 0.53. After adjustment of REE for FFM, heritability decreased to 0.42. These results suggest that 42% of the individual differences in REE, after accounting for the influence of body composition are genotype-dependent. Since both FFM (i.e. the major determinant of REE) and REE adjusted for FFM have heritabilities of about 43 and 21%, respectively, there is a considerable genetic effect on metabolic rate. These
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data are again close to previous estimates [10]. By contrast there is only a minor effect of age on REE (about 4% of its variance is explained by age). However, age explained 21% of the variance in FFM. Taken together these data suggest that the age-related changes in REE are more likely due to age-related changes in nutritional status. Figure 5 shows the different estimates of our calculation.

These data also suggest that heritability estimates for nutritional status and metabolism differ. When compared with REE the heritability of nutritional status is higher (i.e. 40–50 vs. about 20%) suggesting that lifestyle- and disease-related factors add more to the variance in metabolism than to the variance in nutritional status. It is evident that heritability adds to physiological and pathophysiological changes of nutritional status and metabolism (fig. 6).

**Summary**

To summarize, body composition methods have to be selected carefully to assess age-related changes in nutritional status. Age-related changes in metabolism are likely explained by age-related changes in nutritional status and diseases. There is a certain heritability in nutritional status and metabolism (about 40–50 and 20%, respectively) which tracks throughout life.

**Acknowledgement**

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**References**

Discussion

Dr. Elia: Thank you very much for asking me to comment on the organ-specific constancy that you were using in your studies. These were largely derived from studies of arteriovenous oxygen differences in younger groups of people. If one is to use those and extrapolate to elderly subjects there is potential for error. For example, I actually don’t exactly know what a healthy old person is because disabilities increase with age, affecting large segments of the ‘normal’ population. You mentioned blood pressure and ischemic heart disease were present in one of your studies. It could be that the values obtained from younger healthy people may be different from those in the elderly population who had such problems. In addition there are some fluid changes in older people, which could confound some of the interpretations. But this age-specific effect also has relevance to other data that you showed, such as bioelectrical impedance measurements. These are largely empirically based relationships and it is important to know whether they apply to other age groups. Other studies using reference body composition techniques show an increase in percentage fat with age, even after adjusting for body mass index (BMI). The question I want to ask is about the heritability of BMI because if one looks at the literature the estimates of heritability have ranged from somewhere between 10 and 90%, with quite a few around the middle range like the one you reported. Could you comment as to the reasons for this large variability and the type of models used to establish these heritability factors?

Dr. Müller: Heritability estimates vary between the different types of studies. There are two types of study that have been used in the literature to calculate the heritability of parameters of nutritional status and also parameters of metabolic function [1]. The family path study is one possible model for calculations like this on the heritability of phenotype between generations. If DNA probes are available one can also estimate the heritability of the genotype of interest. When compared with heritability obtained in the family studies the highest heritability is reached using twins, monozygotic and dizygotic twins, and the highest numbers are reached when dizygotic twins are used to calculate the heritability between subjects. This is not a big surprise when you look at twins and you are totally convinced that there must be a high heritability in the parameters. With regard to family studies, I am only aware of 3 family studies which are cited in the area of body weight and body composition. These studies are also used as a basis for all the molecular studies which we are faced with now. I am not very sure that these 2 or 3 family studies can really be used as a reference standard since heritability varies for BMI for instance, so one has to be careful. In the case of the Quebec study by Bouchard [2], which is most frequently cited, he only calculated the parent–child relationship which is different to what I have shown where we have grandparents, parents and children. So we have one generation more, and it is no surprise that our data may differ to some extent from the data of the Quebec study [2]. On the one hand there is a clear need to bring these studies together and on the other hand to be careful with all these analyses. We have just ended heritability assessments with DNA probes on all these subjects which is part of the human genome on the metabolic syndrome. The data we found here are within the magnitudes which have been reported. Perhaps they can be used to differentiate
between nutritional status-dependent changes and nutritional status-independent changes in the variables of interest here; perhaps we can get an idea from these data.  

**Dr. Oltersdorf:** I know that there should be many more longitudinal nutritional studies. I know that there are still the data on the Framingham offspring [3]. So it is longitudinal. Has this generation study not been done? I am surprised.  

**Dr. Müller:** I have never seen heritability data or the longitudinal data of the Framingham study. I am not aware of this.  

**Dr. Oltersdorf:** Nobody knows about it because they select many things, and so it should be easy to analyze the generation.  

**Dr. Steinhagen-Thiessen:** I think the Baltimore longitudinal study [4] has those data.  

**Dr. Müller:** But I think they don't have body composition data, they only have BMI data.  

**Dr. Steinhagen-Thiessen:** I am not sure because there was a time where they were very much focused on water and fat consumption and so on.  

**Dr. Morley:** The Baltimore longitudinal study from Saint Louis does have data but it is based on skin-fold thickness, the data were published perhaps 10–15 years ago [5]. The problem always with the Baltimore longitudinal data is that very little of what they published is longitudinal so they often published cross-sectional and a little bit of longitudinal buried in the cross-sectional. It was in the *Journal of Gerontology* I think about 10–15 years ago, and as I said, it was based skin folds not on any of the more modern techniques.  

**Dr. Lochs:** When I look at your data on age, it seems that this is a rather uniform behavior and that there are not 2 groups which behave differently from one another. Do you think it is correct then to conclude that these are physiological changes because if there were a sick group and a healthy group then you would expect that these curves would grow apart with increasing age. Is that correct or not?  

**Dr. Müller:** Let me go back to Dr. Elia's question. These are self-reports of health, and if you assume something like a constant factor, e.g. heritability, for example the probability of a silent disease in all subjects increases with age. So then these are not really health-related changes. What we are seeing is a whole picture. I have no idea whether the data can be divided into subgroups. So what I see here is a more spontaneous situation in a heterogeneous group of subjects who are healthy with respect to their self-reported health, but there may be a contribution of certain diseases in some people. But you are right, there is a big variance in the data.  

**Dr. Lochs:** But still it looks quite uniform. It does not look as though it is divided up into groups. But of course you took only healthy people or subjectively healthy people, so it would be nice to see how nursing home subjects change or what their nutritional status is.  

**Dr. Müller:** The decrease in the second part of life is accelerated in this group; this is what we would expect in this group.  

**Dr. Oltersdorf:** Perhaps you can explain a little bit about how you got the marvelous database 1 as Precon data? What kind of people are they?  

**Dr. Müller:** Precon is a company involved in body weight reduction programs for overweight and obese people. This part of our study was in cooperation with this company which has a huge database in Germany of more than 100,000 impedance measurements. They gave us the data because we have the so-called German Reference Center of the Body Composition Research here. They didn't know what to do with all these data and asked us if we could find anything. We tried to systematically analyze the data, and found that there are true limits to this analysis because these are field measurements performed on numerous subjects.  

**Dr. Oltersdorf:** Do you only know the age of these people?  

**Dr. Müller:** We have age, weight, height, sex and the impedance data.
**Dr. Labadarios:** A very valid and important point you made regarding methodology and its limitations or advantages. Could I please have your thoughts on your approach to the criteria you use in accepting or ignoring data when you read publications on bioelectrical impedance?

**Dr. Müller:** There are numerous problems with impedance measurements, with the method itself, and the major problem is with the algorithm you use to calculate variables like fat-free mass or fat mass. Our personal decision in my group was to use raw data instead of the calculated data. When you start with impedance measurements, you need a reference standard and a reference population that fits your population of interest. There is some discussion about a suitable reference standard for impedance measurements. I think at present there is no real gold standard, but we are all sure that the four-compartment model, which uses different methods to combine these variables, is the most robust model we have for body composition research. For me, if you don’t have the four-compartment model, the best way is to speak about reference method-specific impedance numbers. So if you use impedance and your reference method is DEXA you have fat mass measured by impedance based on an algorithm generated by DEXA. You also have to say what your reference population is. In Germany when I work with children, the problem is that for instance in Jena, in the eastern part of Germany, I cannot use an algorithm that was generated in Kiel. So if we try to apply these algorithms or these formulas generated from measurements of densitometry, and we both (Kiel and Jena) use densitometry as a reference, we come up with different algorithms. So there is an absolute need to generate population-appropriate algorithms and to work with specific numbers. The results are not absolute numbers, i.e. this is not absolute fat mass, you don’t reach absolute fat mass. I think we have no gold standards, we have specific DEXA values, we have specific densitometry values, we have isotope dilution-specific values. They are close to each other, no question about that, but they are not really true numbers.

**Dr. Bozzetti:** As far as I know from all the clinical studies made by Roza and Shizgal [1] 20 years ago, the resting energy expenditure to body cell mass ratio measured by total exchangeable potassium usually remains constant during different intervals of age. Is this still correct or not?

**Dr. Müller:** It cannot be correct, when you look at the regression line between fat-free mass or body cell mass and resting energy expenditure, it doesn’t cross the zero point. It is slightly higher, at about 1, 2, 3 μL/day. So this association causes some doubts in simply dividing resting energy expenditure by body cell mass or fat-free mass. The problem can be addressed by regression procedures and Roza and Shizgal [1] did not do this at that time. So even in the case of healthy subjects and also in patients, the simple ratio of resting energy expenditure to body cell mass or resting energy expenditure to fat-free mass ratio does not allow a comparison of different groups with respect to energy expenditure. If you look at your own patients with cancer cachexia, you cannot compare this group with another group of normal weight subjects with respect to the ratio of resting energy expenditure to fat-free mass.

**Dr. Elia:** One of the potential variables here is the same issue that has been raised with fat-free mass. Body cell mass is a mixture of low energy expenditure muscle tissue and high energy expenditure organs; so if there is an alteration in the proportion of these such that the total body cell mass remains the same you will still get some differences in energy expenditure. Body cell mass is a summary measure of all the tissues with widely different metabolic rates and this can be disturbed in disease and possibly with aging as well.

**Dr. Ockenga:** Just another topic, this project is part of the worldwide genome project and the aim of this project is to get information about the genotype–phenotype
association especially in diseases. Do you think that a further analysis of your data would give us data which would identify patients at risk for obesity as well as patients at risk for malnutrition under special clinical circumstances?

**Dr. Müller:** This is what molecular biologists have been dreaming about for many years. Faced with the heterogeneity phenomena in this area, I am not very sure that they will come to a positive end with that kind of research. We are mainly interested in fatty acid binding proteins and fatty acid transport proteins. We look for genetic variances between subjects and between generations, and based on the data of these 53 families and these datasets and we are going to include 200 families. This is the upper level that we can reach in this study. So I am not very optimistic about this kind of study, we have to look further for the truth. I think that the influence of lifestyle and socioeconomic status is much greater. So if we are interested in the prevention of metabolic syndrome, overweight and obesity, we should look for socioeconomic factors and think about both education and improvement in the economic situation of the people, which is much more effective than selective prevention in subgroups characterized by different genetic makeup. This is not the way we can really solve the problem of overweight endemia in our society.

**Dr. Thomas:** You have commented on population variability and the software analysis regression equations. Can you comment on the reliability of repeated measures of bioelectrical impedance in the same individual? What is your standard of accuracy between two separate measurements with different time variance?

**Dr. Müller:** I consider the inter-individual variance as very low. It is about 2–3% within a subject if you measure him twice within let’s say 1 h. At our institute, in this setting, the intra-individual variance for resting energy expenditure is lower, about 2–5%. But if you ask the subject to come back a week later, his variance is higher. If you have him come back many times over a period of a year, you get something like that. It differs if he comes with a bicycle or on foot. There are a number of things which have an effect on the inter-individual variance of these data. So for the field situation there are problems with respect to the interpretation of difference between groups or within subjects over a long time period. We try to standardize the measurement condition by letting the subject lay down for at least 15 min to measure him and we try to take into account a number of factors which influence the measurement. But I am not sure whether we can really address all these factors which add to the variance.

**Dr. Morley:** First of all in answer to Dr. Lochs who wanted to know the difference between a nursing home population and a general population. This was studied many years ago by a methodology that I think is somewhat suspect, but the differences were enormous. The generally healthy elderly are very different from the population in a nursing home. I think that is what we can say as far as safety of the numbers. The question I really have, which has driven me crazy, is how should you measure body composition if you actually want to do it in a cross-sectional or longitudinal study? We know, for instance with DEXA, that there are real problems with measuring visceral fat as some studies show that if you put fat on the stomach you underestimate it dramatically in those circumstances. Even if you go to something like stable isotope studies there is sometimes a 10–15% variance in the methodology which makes it very difficult if you really want to look at smaller numbers which is necessary. For instance the genome projects have outgrown our ability to do something that we think is very simple, which is to measure body composition, which may be a lot harder than measuring parts of the genome.

**Dr. Müller:** If you are not a specialist in this field, I would advise you to start with one method, to try to standardize the method as well as you can in your hands and work with specific numbers which you can compare within your population. It is quite
correct to use anthropometric data, you can use impedance data, you can use DEXA data. The accuracy of DEXA is above body impedance analysis. But even if you have DEXA or densitometry data you have a lot of limitations with their interpretation which are due to the algorithms you use which for instance cannot be used in all age groups in the same way. So if you go on and on, you have to make a number of assumptions. If you use the Heymsfield [6] data for instance: in one of his studies on organ mass and the modeling of resting energy expenditure, he had at least 26 assumptions in his algorithms, which have not been proven at all. There is quite clearly some plausibility, but there are a lot of assumptions and this is something for the specialist, not the greater audience. So use one method which can be applied in your setting, try to standardize it and work with it over the years.

Dr. Morley: At present in the United States it is very difficult to get anything funded if you are not also to get CT or MRI data. How do you feel that interferes with each of the different methods? It is very clear that a 70-year-old has much more fat in his muscle than a 35-year-old and that is one of the problems we are now dealing with in addition to the rehydration status.

Dr. Müller: I cannot really imagine a context where you need CT or MRI measurements of body composition in a clinical setting or a field study. If you are interested in age-related changes, not specifically in changes in visceral fat mass or bone mass for instance, you can work with a standard method. I don't see a need for these imaging technologies in this context. If you have specific patients or specific questions (e.g. regarding visceral fat mass) there is some need for these imaging technologies, but I feel that even in this case DEXA is enough today. You don't need CT for visceral fat mass and you don't need MRI for the calculation or measurement of muscle and/or organ mass because these are really specific questions.

References
