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Letter to the Editor

Measuring Sarcopenia Over Time Using DXA is Problematic, and Monitoring Mitochondrial Quality Should Include Antioxidative Capacity

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Letter to the Editor

We read with interest the article by Springer-Sapp *et al*. on markers of protein expression for mitochondrial fusion (Mfn2, Opa1), fission (Drp1, Fis1), mitophagy (Parkin), biogenesis (PGC-1α) and content (complex-IV) in sarcopenic and non-sarcopenic older adults and whether strength training affects the content of mitochondria in skeletal muscles and the expression of proteins involved in mitochondrial quality control in sarcopenic subjects [1].

It was found that the sarcopenia index (ratio of appendicular lean mass (ALM) to body mass index (BMI)) correlated negatively with the expression of complex-IV, that lean body mass and ALM correlated negatively with the expression of the fusion protein Opa1-SP, and that strength training increased strength in sarcopenia by 13% [1]. The study is interesting, but some uncertainties still need to be clarified.

The first point is that DXA may not be as accurate as claimed for measuring ALM [1]. One disadvantage of the DXA method is that it may overestimate intramuscular fat [2]. Another disadvantage is that repeated measurements increase the measurement error [2]. The least significant change (LSC) values (percentage change in appendicular lean soft tissue mass (LSTM) required to be accurately detected by DXA) range between 3.85 and 19.4% for individual limbs [3]. Therefore, tracking changes over time becomes problematic when at least a 4% increase/decrease in LSTM must occur for DXA to detect a change, which is exacerbated when other variables (e.g., hydration status, the possibility that changes in tissue properties may occur in response to an exercise intervention) increase measurement error [4]. Furthermore, there is evidence that the mass changes measured by DXA correlate poorly with the changes in mass/volume measured by MRI or CT [4].

The second point is that the exclusion criteria were very narrowly defined [1]. Surprisingly, several comorbidities associated with muscle wasting were not considered exclusion criteria. These include heart disease, immune disorders, metabolic disorders, endocrine disorders, and malignant tumors. Therefore, we should know whether any of the included subjects suffered from any of these conditions.

The third point is that concomitant medication was not included in the analysis [1]. Muscle strength can be affected by antidiabetic drugs, ciprofloxacin, levofloxacin, amiodarone, beta-blockers, diuretics, hydroxychloroquine, colchicine, immune checkpoint inhibitors, chemotherapeutic agents, ciclosporin, alendronate, zidovudine, phenytoin, lamotrigine, propofol, isotretinoin, and omeprazole [5]. It is therefore essential to record all these medications and exclude subjects who regularly took any of them.

The fourth point is that mitochondrial quality control did not include mechanisms to compensate for oxidative stress (antioxidant capacity), which is known to decline with age [6]. Enzymatic mitochondrial antioxidants that should have been included in the study include superoxide dismutase, glutathione peroxidase, and NADH-producing enzymes, while non-enzymatic antioxidants include mitochondrial glutathione, thioredoxin, and ubiquinol [6]. At least some of these parameters for oxidative stress should have been considered.

In summary, quantifying sarcopenia over time using DXA can be misleading. Before attributing sarcopenia exclusively to aging, numerous comorbidities and concomitant medications must be ruled out as influencing factors. Quality control of mitochondrial function should also include its antioxidant capacity.

Declarations

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