**Int. j. adv. multidisc. res. stud.** 2025; 5(5):954-955

**Received:** 20-08-2025
**Accepted:** 30-09-2025

International Journal of Advanced Multidisciplinary Research and Studies

**ISSN: 2583-049X**

**Letter to the Editor**

# Comment on "Phase Angle Is a Potential Novel Early Marker for Sarcopenia and Cognitive Impairment in the General Population" by Ikeue *et al.*

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**Letter to the Editor**

We read with interest the article by Ikeue *et al*. on the relationship between sarcopenia as measured by appendicular skeletal muscle mass (ASM)/height, ASM/body weight index (BMI), handgrip strength (HGS), HGS/upper limb muscle mass and phase angle [1]. It was found that high phase angle was associated with a lower risk of mild cognitive impairment (MCI) in women, while other indices of sarcopenia showed no association with cognitive impairment [1]. The study is noteworthy, but several points should be discussed.

First, we disagree with the statement that sarcopenia is associated with an increased risk of dementia [1]. We also disagree that sarcopenia is a health problem [1]. Sarcopenia in the sense of the index study is a physiological process that affects almost every aging individual, depending on the age they reach, and is characterized by the inability to increase muscle mass through exercise [1]. Since not everyone experiences cognitive decline with aging, but almost everyone experiences sarcopenia, a causal relationship between the two is unlikely.

The second point is that the parameters used to assess sarcopenia are not ideal. Sarcopenia is primarily characterized by muscle atrophy (reduction in muscle mass) and not necessarily by muscle weakness. Weakness only occurs when a certain threshold of muscle atrophy is exceeded. Therefore, measuring handgrip strength (HGS) may not be an optimal parameter for assessing sarcopenia and may lead to false positive or negative results. Better than measuring muscle strength is the assessment of muscle volume using magnetic resonance imaging (MRI).

The third point is that HGS depends not only on muscle volume, but on several other influencing factors. In particular, HGS depends on whether the right or left hand is examined, whether the subject exercises regularly, the type of occupation, daily activities, hobbies, medication and comorbidities. As long as these determinants of HGS are not included in the analysis, the results may be misleading.

The fourth point is that the phase angle is a weak parameter for assessing muscle mass. The phase angle is calculated by measuring impedance, resistance and reactance using bioelectrical impedance analysis (BIA) [2]. As these parameters are dependent on several influencing factors, they must be included in the analysis before final conclusions can be drawn. BIA results are highly dependent on skin thickness and texture, subcutaneous fat thickness and texture, muscle fascia, vascularization, tissue hydration and excitability of muscle and nerve fibers. The excitability of muscle and nerve fibers depends on the intracellular and extracellular electrolyte concentrations, the function of the receptors, pores and signalling proteins, the function of the motor endplates, the intracellular metabolism and the electrical and physical stress to which the muscle or nerve fibers are exposed.

The fifth point is that cognitive status was only assessed using the Japanese version of the Montreal Cognitive Assessment (MOCA) [1]. The MOCA is a simple and quick test that only superficially assesses a limited number of cognitive domains. Therefore, the results and conclusions on cognitive dysfunction in the included patients are of limited relevance. To adequately investigate cognitive function, a sophisticated battery of neuropsychological tests that adequately covers all domains of cognitive function is desirable. Alzheimer's dementia should not be diagnosed with MOCA alone.

The sixth point is that physical muscle performance is influenced not only by the muscular system but also by its control and regulation by the central nervous system (CNS). However, CNS abnormalities are not usually associated with muscle wasting, but weakness, coordination and muscle tone are mainly controlled by CNS functions, so the CNS must be excluded as a cause of reduced muscular performance in all included patients before attributing reduced muscle performance to sarcopenia.

The seventh point is that sarcopenia in patients with dementia may also be highly dependent on the type of dementia and its

impact on motor function. Patients with hyperkinesia may have less muscle atrophy than patients with hypokinesia or concomitant depression. Therefore, the type of dementia should have been clearly determined and the included patients should also have been assessed for the presence or absence of depressive symptoms.

In conclusion, there is no causal relationship between sarcopenia and cognitive function and loss of muscle mass with age should not be used as a biomarker for dementia.

**Declarations**

**Ethical approval:** Not applicable.

**Consent to participation:** Not applicable.

**Consent for publication:** Not applicable.

**Funding:** None received.

**Availability of data and material:** All data are available from the corresponding author.

**Completing interests:** The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

**Author contribution:** JF was responsible for the design and conception, discussed available data with coauthors, wrote the first draft, and gave final approval. SM: contributed to literature search, discussion, correction, and final approval.

**Acknowledgements:** None.

**Keywords:** Sarcopenia, Cognitive Function, Dementia, Phase Angle, Muscle Mass

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