Response to the Letter to the Editor: "Understanding the Feasibility and Validity of Muscle Strength Measurements in Aging Adults"

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It is difficult to disentangle what measures of muscle strength should be used for accurately assessing dynapenia in aging adults. The ages and functional capacities of individuals undergoing muscle strength assessments and setting (clinical or epidemiological) should be deliberated before testing. For example, older adults with poor function may have difficulty performing KES measurements; however, younger and middle-aged adult populations could more easily provide measures of both HGS and KES. Similarly, large epidemiological studies that are examining muscle strength should use HGS for feasibility purposes, whereas imaging in clinical settings may provide detailed insights into the age-related changes of the musculoskeletal system (eg, bone, muscle quality, articular cartilage).[10] Therefore, it is important to consider the balance between feasibility and validity in assessments of muscle strength. Figure 1 provides a depiction for how measures of muscle strength fit on a smoothed feasibility and validity slope. Although researchers and health care providers should consider feasibility and validity of muscle strength measurements, the demographic factors, setting, and health outcomes being evaluated should also be contemplated when selecting muscle strength assessment modes.

The debate on how to appropriately measure muscle strength in aging adults will continue. More research is needed to develop a process for evaluating muscle strength during aging that acknowledges age, standardization, setting, functional capacity, feasibility, validity, and what clinically relevant health outcomes are being examined. Doing so will help to develop consistency in methods for researchers, health care providers, and their patients. Additionally, more research that identifies how much accuracy is gained by performing dynamometry at multiple sites on the body, while also accounting for demographic factors, functional capacity, setting, and health outcomes is needed to determine if the information gained from both KES and HGS measurements outweighs the feasibility and robust information that HGS alone already provides.

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Response to the Letter to the Editor: "Understanding the Feasibility and Validity of Muscle Strength Measurements in Aging Adults"

Thank you for the response to our recent article "Handgrip Strength Cannot Be Assumed a Proxy for Overall Muscle Strength".[1]

There are ample epidemiologic studies underlining handgrip strength (HGS) as a powerful biomarker for health; lower HGS is associated with negative health outcomes in older adults.[2] However, in our previous study among community-dwelling older adults visiting an outpatient clinic due to mobility impairments, HGS showed weaker associations with health characteristics compared to knee extension strength (KES) in the same individuals.[3] The difference in the association between HGS and KES with health outcomes might be partly dependent on population characteristics. In a large cohort of community-dwelling individuals, both HGS and KES were equally predictive for functional performance, whereas KES was a better predictor of functional performance than HGS in older adults in assisted living facilities.[4]

It should also be noted that HGS is not as sensitive as KES to physical interventions. Tieland et al showed that substantial improvement in KES as high as 40% was observed after a whole-body resistance-type exercise program in frail older individuals, but no change in HGS was detected.[5] Furthermore, a recent meta-analysis revealed that KES significantly improved, but HGS was not, after exercise interventions targeting older adults with sarcopenia.[6] Therefore, HGS does not represent an appropriate measure to evaluate the efficacy of interventions to increase muscle strength or mass in older adults.

There is no doubt that HGS is easier to measure than KES. However, the use of HGS alone is likely to misclassify individuals for dynapenia as HGS only explains about 40% of the variance of lower extremity strength.[7] We agree that there are feasibility issues measuring KES, such as the availability of standardized equipment and the need for special training. However, KES isokinetic dynamometry has been validated and found to have high interrater and intrarater reliability in older adults.[8] For a portable, easy-to-use, and less costly option, handheld dynamometer has been shown to have high test-retest reliability in older adults.[9] Based on our experience in epidemiologic studies and in geriatric outpatient clinics, where the measurement of KES is part of usual care, it is feasible to measure KES even in frail patients. It is understood that equipment for measuring KES is not readily available in most outpatient clinics; therefore, Manini and Clark suggested that referral to a separate assessment venue, such as physiotherapy clinic or specialized clinic where equipment is available, would be an approach for measuring KES.[10] However, because patients attending geriatric outpatient clinics are likely to suffer from sarcopenia and/or dynapenia, investment in equipment is strongly recommended.

Overall, the use of KES measurement in clinical practice is feasible and should be encouraged because of its additional value compared to HGS. We agree with the authors that further studies are required to reach a standardized procedure in muscle strength

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measurement and define the cut points for dynapenia for use in research and clinical settings.

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Preventing Alzheimer's Disease: Why Not Targeting the Muscle First?

Alzheimer's disease (AD) is the commonest cause of dementia and in fact the most frequent neurologic disorder that accompanies the ageing process, currently affecting more than 30 million persons around the world.[1] Although research is rapidly progressing, AD prevalence continues to rise worldwide, and currently available treatments produce minimal improvements, prompting the need for new strategies, including nonpharmacologic approaches. In this regard, a key lifestyle intervention, regular physical activity (PA), might play a major role in AD prevention,[2] with the pandemic of physical inactivity being at odds with our biological makeup. Our species is indeed unique compared to the rest of hominoids in that the increase in aerobic fitness that occurred as we evolved as persistent hunters or "endurers" paralleled a remarkable increase in cognition.[3]

There is evidence that PA is needed to maintain brain health over life. Regular PA has positive effects on hippocampal volume, preventing the decreases that occur as we age.[4] Because the hippocampus is one of the major brain sites of neuroplasticity, this promising finding suggests the need for implementing PA interventions to attenuate age-related neurologic decline. In fact, a meta-analysis of 15 prospective studies (including 33,816 nondemented subjects followed for 1-12 years) found that individuals with high-to-moderate levels of PA reduced their risk of cognitive decline by 35% to 38% compared to their sedentary peers.[5] There is also meta-analytic evidence in support of PA implementation to prevent AD: our group showed that adherence to the World Health Organization recommendations (ie, ≥150 minutes/wk of moderate-vigorous PA such as brisk walking) at late life (70-80 years) is associated with a remarkable (40%) reduction in the risk for AD.[6] PA can also attenuate the decline in cognitive function in individuals who already have dementia, including those with AD.[7] On the other hand, a crucial problem of patients with AD that can be reverted, at least in part, with PA interventions is physical deterioration and reduced muscle mass, which in turn are linked to higher risks of falls and fractures, decline in mobility, poorer quality of life, and further loss of independence.[8]

More research is needed for a better and more balanced understanding of the pathogenesis of AD. In this regard, exercise models might provide interesting insights. A recent preclinical study showed that inducing hippocampal neurogenesis per se (pharmacologically and genetically) did not confer any benefits over AD markers.[9] By contrast, increases in hippocampal neurogenesis along with exercise-induced increases in the levels of brain-derived neurotrophic factor and reductions in β-amyloid load led to improvements in memory. These findings suggest that hippocampal neurogenesis could ameliorate AD, but only in the presence of an optimal brain environment such as that induced by physical exercise.[9]

A holistic view of AD development (vs the original "amyloid-only" hypothesis) is needed, especially during the early phases of this condition. In this context, an exercise milieu helps to combat systemic inflammation and protect vascular health, both of which might participate in the genesis of AD. There is a cross-talk between the muscle tissue and several organs including the brain, with contracting muscles producing myriad myokines, that is, cytokines or peptides (eg, interleukin-6, cathepsin B, irisin, among many others), able to induce numerous health benefits at the multisystemic level, such as an improved neurotrophism.[10]

More research is needed to establish new pharmacologic treatments against AD, but this should not overshadow the importance of lifestyle interventions, notably those focused on regular PA, a "natural pill" that comes at a low price and largely free of adverse effects.

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