Sarcopenia in COPD

Dear Editor.

We read with great interest the paper by Jones *et al*¹ about the prevalence, clinical correlates and response to pulmonary rehabilitation of sarcopenia in COPD.

We believe that authors have meritoriously focused their attention on a highly relevant topic, which is the skeletal muscle decline in patients with COPD. Sarcopenia represents a condition exposing older persons to the risk of disability and negative health-related outcomes. Since respiratory conditions, including COPD, may directly (eg, through poor peripheral oxygenation, inflammation, hypercatabolic state) and indirectly (eg, inducing sedentary behaviours) affect the skeletal muscle, more attention should be given to sarcopenia. This implicitly suggests the need of a multidimensional approach to the older patient with respiratory conditions. A more comprehensive assessment beyond the standard measurement of the respiratory function is indeed necessary, potentially improving the diagnostic iter, clinical choices and therapeutic management.

In the present letter, we would like to point out a potential weakness of the article by Jones *et al.* In their work, the

authors chose to define sarcopenia according to the algorithm proposed by the European Working Group on Sarcopenia in Older People (EWGSOP).2 This is a legitimate choice, and these criteria likely represent the most commonly used operationalisation of sarcopenia. The EWGSOP algorithm was originally designed by a panel of international experts in the field who reached a formal consensus for standardising the objectivation of the sarcopenic condition. Nevertheless, several other operational definitions of sarcopenia exist today.³ Unfortunately, the agreement across them tends to be quite modest. The controversial identification of a clear/ unique phenotype has delayed research on the topic and limited the clinical implementation of sarcopenia.

In 2014, results from the Foundation for the National Institutes of Health Biomarkers Consortium (FNIH) Sarcopenia Project⁵ have been published. Differently from previous operational definitions largely based on experts' consensus, the FNIH criteria for defining the skeletal muscle decline were based on data analyses of multiple cohort studies. Interestingly, these recent criteria keep well separated the qualitative (ie. strength) and quantitative (ie. mass) domains of the skeletal muscle. This choice was indeed necessary: it is today clear that the skeletal muscle function and mass differently decline with aging and do not present the same clinical relevance and prognostic value. Furthermore, the use of usual gait speed (currently considered an aspecific 'vital sign' rather than a pure marker of mobility) does not help at isolating the sarcopenic phenomenon from the overall health status. Not surprisingly, the FNIH reports adopted the gait speed as endpoint (ie, marker of mobility disability) rather than as parameter of skeletal muscle function (as done in the EWGSOP definition).

In conclusion, while acknowledging the value of the Jones *et al* study, we respectfully propose the extension of the analyses using a (to us) more robust and reliable operational definition of sarcopenia as the FNIH one is. The development of this recent operationalisation of sarcopenia indeed represents a unique opportunity we have. In fact, a wider adoption of this data-generated definition may reduce the existing controversies and foster future research against the detrimental condition of sarcopenia.

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694 Thorax July 2015 Vol 70 No 7