The interconnection between Covid-19, sarcopenia and lifestyle

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Editorial

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In March 2020, Covid-19 was declared a global pandemic by the World Health Organization. Infection with Sars-Cov-2, the virus responsible for Covid-19, can have a wide range of consequences, the severity of which depends on the individual [1]. Moreover, this vulnerability is increased by the presence of comorbidities such as chronic respiratory disease, diabetes, hypertension and other pathologies affecting immunity [2]. Also, musculoskeletal disorders, and particularly sarcopenia, have recently been suggested to be either a consequence of [3] or a risk factor for [4] Covid-19. For example, studies have reported muscle damage in patients hospitalized for Covid-19 and greater loss of muscle function in older women living independently during the pandemic [5,6]. Some studies, but not all, have also highlighted that low skeletal muscle mass and reduced muscle strength are associated with higher severity of Covid-19 [7–9]. Very recently, histopathological changes in muscle, such as muscle atrophy, have also been identified in patients complaining of long-term Covid-19 symptoms, including fatigue and weakness [10]. In addition, results from a meta-analysis highlighted a twofold greater risk of getting severe complications and of mortality due to Covid-19 for sarcopenic people compared with non-sarcopenic ones [4].

Three potentially modifiable factors appear to be associated with both sarcopenia and Covid-19 and can be considered a target for prevention: high level of systemic inflammation, low levels of physical activity and poor nutrition:

- *High level of systemic inflammation:* Sarcopenia can potentially induce a higher level of inflammation, which can be observed with trough biomarkers such as interleukin-15 (IL-15), interleukin-6 (IL-6) and C-reactive protein (CRP) [11,12]. In parallel, in severe forms of Covid-19 disease, a high level of biomarkers of inflammation (i.e. IL-6 and IL-10) have been found [13] and can potentially increase the risk of acute sarcopenia [14].
- *Low physical activity:* The model of "catabolic crisis", which proposes that sarcopenia is not only like a progressive process but also aggravated by acute periods of inactivity, was meaningful during the Covid-19 crisis [15]. Indeed, quarantine, lockdown restriction and hospitalization were among the many conditions that affected lifestyle behaviours and consequently might have increased the risk of developing sarcopenia. In this context, a systematic review showed an overall global trend of decreased physical activity in older adults due to lockdown restrictions [16]. The authors of this review noted that in addition to induced lower muscle mass and strength, which is directly associated with sarcopenia, lower levels of physical activity can lead to other non-communicable diseases that increase vulnerability to Covid-19 [16].
- *Poor nutrition*: Some harmful eating behaviours have been reported during Covid-19 quarantine, such as skipping warm meals, eating less than normal and having less appetite [6]. The impact of these changes was partially highlighted in a study in which 25 % of 1407 patients hospitalized for Covid-19 had significant weight loss before hospitalization [17]. In that study, this malnourishment could be partly explained by Covid-19-related symptoms like ageusia, change of taste, nausea, vomiting, and anorexia [17]. Those changes in eating habits may cause a decrease in protein intake. Indeed, insufficient intake can lead to a reduction in muscle protein synthesis which is an aggravating factor for sarcopenia [3]. In addition, the decrease in physical activity observed during the pandemic could indirectly lead to changes in other lifestyle behaviours, including those related to nutrition. Indeed, during the Covid-19 pandemic, people with lower physical activity levels increased snacking habits and alcohol consumption [6]. These eating habits may lead to overnutrition, as discussed in a Brazilian study conducted in older women during the pandemic that revealed an increase of body mass index and body weight but also an increased risk of sarcopenia [18].

The good news, at least for preventive actions for both Covid-19 and sarcopenia, is that inflammation, physical activity and nutrition are all potentially modifiable and interconnected factors. As specified by the International Clinical Practice Guidelines for Sarcopenia, physical activity and adapted diet are crucial in the management of sarcopenia [19]. For physical activity, to date, resistance-based training is still preferred for its benefits for muscle strength, physical performance and

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muscle hypertrophy [19]. Regarding nutrition, it is recommended that older adults are monitored to ensure energy balance as well as adequate intake of protein and vitamin D. Acting on these two together can generate a decrease in inflammation and further potentially reduce the risk of sarcopenia. Indeed, in their systematic review, Bautmans et al. have highlighted a substantial lower level of inflammatory markers such as IL-6 and CRP in older adults following a physical exercise program in comparison with those who have not [20]. In addition, a recent meta-analysis highlighted that healthy eating behaviours such as high intake of fresh fruits are correlated with lower level of inflammatory biomarkers [21].

In conclusion, even though most restrictive rules have been lifted in almost all countries, Covid-19 is still active. On 15 August 2022, for one year 375 million new cases and over 2 million deaths were reported on a global scale [22]. At present, older populations are still considered vulnerable to Covid-19. Sarcopenia appears to be a risk factor for as well as a consequence of Covid-19 and has repercussions in terms of severity and mortality. In line with what some authors have called "a wake-up call for lifestyle-related preventable conditions in older adults" [23], public health authorities and health professionals [24] should be aware of the connection between sarcopenia and Covid-19 and the factors aiming at reducing their burden.

Contributors

Céline Demonceau participated in data collection and analysis, and in drafting and editing of the paper.

Charlotte Beaudart participated in data collection and analysis, and in drafting and editing of the paper.

Jean-Yves Reginster participated in data collection and analysis.

Nicola Veronese participated in data collection and analysis.

Oliver Bruyère participated in data collection and analysis, and in drafting and editing of the paper.

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