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Commentaries on Viewpoint: The interaction between SARS-CoV-2 and ACE2 may have consequences for skeletal muscle viral susceptibility and myopathies

Ai Lyn Tan[1, 2], Matthew Farrow[1, 2, 3], John Biglands[1, 4]

[1]NIHR Leeds Biomedical Research Centre, Chapel Allerton Hospital, Leeds Teaching Hospitals NHS Trust, Leeds, UK

[2]Leeds Institute of Rheumatic and Musculoskeletal Medicine, University of Leeds, Leeds, UK.

[3] School of Pharmacy and Medical Sciences, University of Bradford, UK.

[4] Medical Physics and Engineering, Leeds Teaching Hospitals NHS Trust, Leeds, UK.

A. L. Tan, ORCiD: 0000-0002-9158-7243

J. Biglands, ORCiD: 0000-0002-1161-5022

Email: a.l.tan@leeds.ac.uk

Address for correspondence: Leeds Institute of Rheumatic and Musculoskeletal Medicine

Chapel Allerton Hospital

Chapeltown Road

Leeds LS4 7SA

UK

With emerging knowledge regarding the pathogenesis involving SARS-CoV-2, it is becoming apparent that no tissue is spared in the body. Ferrandi et al (5) suggested a cause for myopathy in COVID-19 via the ACE2 expression that leads to myocytes becoming susceptible to SARS-CoV-2. We would like to add that, the muscle involvement in COVID-19 is likely to be early in the infection. We have recently explored the muscles in rheumatoid arthritis (RA) (4) using quantitative MRI measures, which reflect physiological changes regarded as an indirect measure of muscle inflammation (3). These abnormal muscle changes which are present in established RA patients, are also noticeable in newly diagnosed untreated RA patients. This suggests that muscle is likely to be sensitive to the inflammatory process and is involved early. COVID-19 also involves multiple pro-inflammatory cytokines; the muscles may already be affected in susceptible patients at the time of diagnosis, which may then lead to various muscle related pathologies including affecting the respiratory muscles as eluded to by Ferrandi et al. Further, Ferrandi et al highlighted that the aging population are at increased risk of COVID-19; we showed that there are age-related muscle changes on MRI that correlates with frailty and muscle function (2). Our RA patients, when treated to arthritis remission, continued to have persistent abnormal MRI changes and weaker muscles. This is analogous to patients who 'recovered' from COVID-19, but continue to manifest various muscular symptoms (1). Therefore therapeutic strategies targeting the muscle may be important throughout the course of COVID-19.

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