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The perils of premature phenotyping in COVID-19: a call for caution

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By prematurely phenotyping patients with COVID-19, we and our patients are exposed to considerable and preventable risk. If data-driven phenotypes are not insisted upon, our cognitive biases guarantee that we'll end up with phenotype-driven data. https://bit.ly/2ZM8wZV

Cite this article as: Bos LDJ, Sinha P, Dickson RP. The perils of premature phenotyping in COVID-19: a call for caution. *Eur Respir J* 2020; 56: 2001768 [https://doi.org/10.1183/13993003.01768-2020].

The severe acute respiratory syndrome coronavirus 2 poses an unprecedented global healthcare challenge. Severe coronavirus disease 2019 (COVID-19) pneumonia frequently causes hypoxaemic respiratory failure, manifesting in the acute respiratory distress syndrome (ARDS). Recently, authors have proposed distinct clinical phenotypes of COVID-19 pneumonia in several influential, high-profile essays [1–3]. For example, in a recent perspective in this journal [3], the authors speculated that COVID-19 has five phenotypic presentations: three phenotypes based on severity of hypoxaemia and need for supportive care (no hypoxaemia, mild hypoxaemia, and moderate hypoxaemia), and two phenotypes of severely hypoxaemic patients based on additional physiological and clinical features. Aligned with other recent efforts to phenotype COVID-19 patients [1, 2], the authors subtyped patients into a supposedly prevalent phenotype with normal compliance, low lung weight, and predominant perfusion abnormalities ("L" phenotype), and a less-prevalent phenotype with more typical features of ARDS, such as profound consolidation and low compliance ("H" phenotype). The authors advocate for distinct management strategies for these purported phenotypes, include permitting increased tidal volumes and restricted positive end-expiratory pressure in the "L" phenotype patients.