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Tailored Molecular and Pathophysiological Approach to COVID-19: Ambition and Need

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Message from the Guest Editor

Dear Colleagues,

Common to all SARS-CoV-2 variants is the direct damage to the subtle mechanisms of the renin–angiotensin system that presides over a large part of systemic inflammation and hemodynamics, i.e., the damage to ACE2 (the cell membrane viral receptor) and ACE2-driven angiotensin 1–7 production. Attempts at replacing defective ACE2 or angiotensin 1–7 functions during the clinical course of COVID-19 have been common and pursued all over the world.

After two years of pandemic, a tailored and sensible approach to COVID-19 must include: a) a definition of the mechanisms of inflammatory disease that follow cellular infection; b) cognition of the most frequently found viral variants in terms of genomic and antigenic mutations; and c) identification of biomolecular viral and host targets, the latter being the non-classical renin-angiotensin system, that could be acted upon pharmacologically.

These aims warrant renewed international and multi-disciplinary efforts.