



COVID-19 and Thyroid Dysfunction

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Message from the Guest Editors

Data following the SARS-CoV-2 isolation, the causative virus for the current pandemic, have shown that it is able to localize not only along the airways, but also in other organs and tissues. In particular, there is evidence of direct infection of the thyroid by SARS-CoV-2, as the receptor ACE2 by which the virus enters the cells, is highly expressed in thyroid follicular cells. Accordingly, data on the possible links between COVID-19 and thyroid dysfunction have been increasing in the past year. An important aspect in this relationship is the ability of the virus to trigger direct inflammatory damage to the thyroid through the occurrence of a "cytokine storm".

Moreover, the number of drugs proposed and tested for COVID-19 treatment raises questions about potential drug-drug interactions, as some of them may interfere with the hypothalamic-pituitary thyroid axis. In contrast, the use of thyroid hormones is currently being evaluated in critically ill COVID-19 infected patients requiring mechanical respiratory support or Extra Corporeal Membrane Oxygenation, for their ability to adapt injured tissue to hypoxic conditions (e.g., through p38 MAPK, and Akt signaling pathways).





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Message from the Editor-in-Chief

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