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PLASMATIC DETERMINATION OF CYTOKINE INDUCTION TO ASSESS THE INFLAMMATORY RESPONSE AGAINST VIRAL ANTIGENS

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During the SARS-CoV-2 pandemic, the “cytokine storm” phenomenon has been widely described in severe cases of COVID-19, being established as the main biomarker of severity. This fact makes relevant to be able to predict those individuals who are going to develop an exaggerated innate response to viral antigens.

We developed a protocol to measure the intrinsic capacity of cytokine production against viral antigens in whole blood cultures. Thirty-nine patients who had overcome COVID-19 (mean time after infection= 5.5 months) without requiring hospitalization (mean age=67.9, N=14), requiring hospitalization on the ward (mean age=71.7, N=13) or requiring ICU admission (mean age=66.8, N=12) was studied to evaluate the test and to compare their ability to produce proinflammatory cytokines.

Peripheral blood samples anticoagulated with heparin were cultured for 18 hours both without stimulation and stimulated with R848, an agonist to TLR7 and TLR8. After incubation, plasma was obtained and the inflammatory cytokine profile was measured using the kit cytokine 9-Plex ProcartaPlex Panel (ThermoFisher) and LuminexxMAPtechnology. This kit includes the cytokines IL-1β, IL-2, IL-6, IL-8, IL-10, IFN-α, IFN-β, IFN-γ and TNF-α.

Cytokine levels were undetectable or very low in unstimulated cultures. Only IL-8 was detected in all patients, and TNF-α, IL-6 and IL-1β were at very low levels in more than half patients. R848 induced the production of all cytokines in all patients and a significant increase in those in which it was already detected without stimulation. IL-8, IL-1β, IL-6 and TNF-α reached the highest levels. When individuals who had different degrees of SARS-CoV-2 severity were compared, we found that those admitted to ICU produced higher levels of IL-2, IL-10 and TNF-α ($p<0.05$).

We conclude that whole blood cultures stimulated with R848 are a useful system to evaluate response to viral antigens and it may predict individuals with a higher risk of exaggerated inflammatory response.