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COVID-19 is an emerging, rapidly evolving situation.

Get the latest public health information from CDC: https://www.coronavirus.gov.

Get the latest research from NIH: https://www.nih.gov/coronavirus.

Find NCBI SARS-CoV-2 literature, sequence, and clinical content: https://www.ncbi.nlm.nih.gov/sars-cov-2/.



Series GSE151161

Query DataSets for GSE151161

Status Public on Jul 01, 2020

Title Blocking of the CD80/86 axis as a therapeutic approach to prevent progression

to more severe forms of COVID-19

Organism Homo sapiens

Experiment type

Expression profiling by high throughput sequencing

Summary

In its more severe forms, COVID-19 progresses towards an excessive immune response, leading to the systemic overexpression of proinflammatory cytokines like IL6, mostly from the infected lungs. This cytokine storm can cause multiple organ damage and death. Consequently, there is a pressing need to identify therapies to treat and prevent severe symptoms during COVID-19. Based on previous clinical evidence, we hypothesized that inhibiting T cell costimulation by blocking CD80/86 could be an effective therapeutic strategy against progression to severe proinflammatory states. To support this hypothesis, we performed an analysis integrating blood transcriptional data we generated from rheumatoid arthritis patients treated with abatacept -a CD80/86 costimulation inhibitor- with the pathological features associated with COVID-19, particularly in its more severe forms. We have found that many of the biological processes that have been consistently associated with COVID-19 pathology are reversed by CD80/86 co-stimulation inhibition, including the downregulation of IL6 production. Also, analysis of previous transcriptional data from blood of SARS-CoVinfected patients showed that the response to abatacept has a very high level of antagonism to that elicited by COVID-19. Finally, analyzing a recent single cell RNA-seg dataset from bronchoalveolar lavage fluid cells from COVID-19 patients, we found a significant correlation along the main elements of the C80/86 axis: CD86+/80+ antigen presenting cells, activated CD4+ T cells and IL6 production. Our in-silico study provides additional support to the hypothesis that blocking of the CD80/CD86 signaling axis may be protective of the excessive proinflammatory state associated with COVID-19 in the lungs

Overall design Whole RNAseq Blood Samples of patients treated with abatacept at week 0 and

week 12

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Platforms (1) GPL24676 Illumina NovaSeq 6000 (Homo sapiens)

Samples (76) GSM4567526 Patient 1 week0

GSM4567527 Patient 1 week12

GSM4567528 Patient 2 week0

Relations

BioProject PRJNA634938 SRA SRP263603

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SRA Run Selector 🗵

Raw data are available in SRA

Processed data are available on Series record

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