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Background: SARS-CoV-2 Virus can trigger severe pneumonia and lead to acute respiratory distress syndrome. Data from clinical, in vitro, and in vivo suggest that virus-induced cytokine dysregulation is a contributory factor to the pathogenesis. Drugs targeting the same are being tried.

Methods: To obtain a better understanding of the molecular events, we studied the transcriptome of infected macrophages and obtained a list of incriminated pathways using Gene Set Enrichment Analysis. Co-expression gene analysis was further used to predict drug targets.

Results: Immune system, hemostasis, RNA metabolism, cellular response to external stimuli, vesicle-mediated transport, cell cycle mechanisms, DNA replication, and repair are upregulated. Interferon alpha, beta, and gamma are upregulated. IL-1,6,10, 13, TNF NF-κB are upregulated. Signaling by non-receptor tyrosine kinase, NOTCH, Sonic Hedgehog, Leptin, MAP kinase-6, 4, and estrogen-mediated signaling are increased. Offactory sensation, transmission across chemical synapses, and sperm motility appear to be downregulated. Disease host signature has resemblance with cystic fibrosis, thombophobilia, leishmania, influenza, CMV, HIV and SARS infection, and diseases of programmed cell death like neurodegenerative diseases. We have predicted 24 precise drugs which can be explored in clinical trials of SARS-CoV-2 treatment.

Conclusions: Our study provides important mechanistic insights into the understanding of SARS-CoV-2 viral pathogenesis and the multi-faceted host immune responses.

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