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### Series GSE157103

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Status	Public on Aug 29, 2020
Title	Large-scale Multi-omic Analysis of COVID-19 Severity
Organism	<a href="#">Homo sapiens</a>
Experiment type	Expression profiling by high throughput sequencing
Summary	<p>Using RNA-seq and high-resolution mass spectrometry we performed a comprehensive systems analysis on 128 plasma and leukocyte samples from hospitalized patients with or without COVID-19 (n=102 and 26 respectively) and with differing degrees of disease severity. We generated abundance measurements for over 17,000 transcripts, proteins, metabolites, and lipids and compiled them with clinical data into a curated relational database. This resource offers the unique opportunity to perform systems analysis and cross-ome correlations to both molecules and patient outcomes. In total 219 molecular features were mapped with high significance to COVID-19 status and severity, including those involved in processes such as complement system activation, dysregulated lipid transport, and B cell activation. In one example, we detected a trio of covarying molecules – citrate, plasmenyl-phosphatidylcholines, and gelsolin (GSN) – that offer both pathophysiological insight and potential novel therapeutic targets. Further, our data revealed in some cases, and supported in others, that several biological processes were dysregulated in COVID-19 patients including vessel damage, platelet activation and degranulation, blood coagulation, and acute phase response. For example, we observed that the coagulation-related protein, cellular fibronectin (cFN), was highly increased within COVID-19 patients and provide new evidence that the upregulated proteoform stems from endothelial cells, consistent with endothelial injury as a major activator of the coagulation cascade. The abundance of prothrombin, which is cleaved to form thrombin during clotting, was significantly reduced and correlated with severity and might help to explain the hyper coagulative environment of SARS-CoV-2 infection. From transcriptomic analysis of leukocytes, we concluded that COVID-19 patients with acute respiratory distress syndrome (ARDS) demonstrated a phenotype that overlapped with, but was distinct from, that found in patients with non-COVID-19-ARDS. To aid in the global efforts toward elucidation of disease pathophysiology and therapeutic development, we created a web-based tool with interactive visualizations allowing for easy navigation of this systems-level compendium of biomolecule abundance in relation to COVID-19 status and severity. Finally, we leveraged these multi-omic data to predict COVID-19 patient outcomes with machine learning, which highlighted the predictive power of these expansive molecular measurements beyond the standardized clinical estimate of 10-year survival Charlson score.</p>
Overall design	126 samples were analyzed in total with 100 COVID-19 patients and 26 non-COVID-19
Contributor(s)	<a href="#">Jaitovich A</a> , <a href="#">Balnis J</a> , <a href="#">Swanson SA</a>
Citation(s)	Overmyer KA, Shishkova E, Miller IJ, Balnis J et al. Large-Scale Multi-omic Analysis of COVID-19 Severity. <i>Cell Syst</i> 2021 Jan 20;12(1):23-40.e7. PMID: <a href="#">33096026</a>

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Submission date Aug 28, 2020  
Last update date Nov 30, 2020  
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Platforms (1) [GPL24676](#) Illumina NovaSeq 6000 (Homo sapiens)

Samples (126) [GSM4753021](#) COVID\_01\_39y\_male\_NonICU

[More...](#) [GSM4753022](#) COVID\_02\_63y\_male\_NonICU

[GSM4753023](#) COVID\_03\_33y\_male\_NonICU

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Supplementary file	Size	Download	File type/resource
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GSE157103_genes.tpm.tsv.gz	3.8 Mb	(ftp)(http)	TSV

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Raw data are available in SRA

Processed data are available on Series record