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Gene Expression and COVID-19 Immune Cell Signatures Analysis

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Abstract

The respiratory syndrome that results from the coronavirus disease 2019 is accompanied by a cytokine storm, the release of numerous proinflammatory factors, and other symptoms. The Delta variant, which has been associated with a high mortality risk, has taken over in many nations. Therefore, comprehension of the immune responses linked to COVID-19 lineages may help in the creation of therapeutic and diagnostic approaches. The severity of COVID-19 was correlated with innate and adaptive immunological factors and pathways, according to numerous single-cell gene expression studies. Additional research on the features of the host-pathogen response to infection caused by various lineages is needed. Here, we used single-cell transcriptome profiling to identify variant-specific molecular immune factors in venous mononuclear cells from people with various COVID-19 and virus lineage severity. Our research shows that the Delta lineage of SARS-CoV-2 is associated with a large population of monocytes with distinct gene expression signatures, which may suggest immune components for targeted therapy.

Keywords: COVID-19 • Gene expression • Molecular mechanism • T-cells • Immunological changes

Introduction

The COVID-19 pandemic, which broke out in 2020, profoundly altered many facets of social life and made immunological research urgently necessary to understand the molecular mechanism underlying this same viral infection. One of the best ways to gain a thorough understanding of the crucial molecular pathways regulating immune response is to use single-cell sequencing techniques. Recent research on transcriptomes and chromatin accessibility showed that the immune response varied greatly depending on the severity of the disease. Changes in the density of dendritic cells, monocytes, and T-cells are part of the landscape of immune changes. Notably, disease severity and decreased lymphocyte interferon IFN- expression were correlated with a decline in T-cells and plasmacytoid dendritic cells. Additionally, in severe cases, monocytes exhibit elevated levels of a large number of cytokines, a decrease in IFN levels, and an increase in the type I IFN inflammatory genes. Additionally, the activation of adaptive T- and B-cells, the release of cytokines, and the recruitment of monocytes and macrophages all contribute to the immune response and the destruction of lung cells. Monocytes with proinflammatory or immunosuppressive gene signatures are involved in the immunological mechanisms that control severe COVID-19.

Additionally, analyses of gene expression revealed a number of transcription factors that function as drivers of cytokine production, potential drug targets for COVID-19 treatment, and indicators of disease severity. Numerous transcription factors have been identified as driver regulators of cytokine production in extensive studies of gene expression. They are potential drug targets for COVID-19 treatment and disease severity markers. There are currently a number of SARS-CoV-2 variants with distinct virus traits, such as transmissibility, pathogenicity, and antigenicity, which call for further research into the immunological changes in COVID-19 at the cellular and gene expression levels. The main goal is to provide a thorough description of the cell state repertoire that goes along with COVID-19 severity. Approach is a great tool for analyzing changes in gene expression and transcription factor regulatory networks to find out how the immune response is triggered. Uncover the molecular and regulatory mechanisms underlying the anti-COVID-19 response. Depending on the virus version and its severity, we found changes in the levels of the significant types of immune cells.

Literature Review

Our findings identified fundamental alterations in monocytes that result in deliberately offensive gene signatures in COVID-19 lawsuits filed on by the Delta variant.

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We looked into gene regulatory networks and identified key transcriptional regulators and the genes they control as contributors to cytokine storms, which may indicate potential therapeutic targets. This is the first high-resolution transcriptomic analysis of blood mononuclear immune cells, to our knowledge. Every patient with COVID-19 was enrolled in the Federal Clinical Center for High Medical Technologies of the Federal Medical Biological Agency. According to the World Health Organization's diagnosis recommendations, patients were divided into mild/moderate and severe/critical groups. Healthy volunteers who had negative COVID-19 PCR outcomes were sought out.

All participants in the study provided their written consent. Ancillary blood was drawn from healthy volunteers and specimens which tested positive for COVID-19. The research material consisted of PBMC cell suspension samples. A Countess II FL cell viability counter and analyzer was used for biomaterial quality control. To create an emulsion, the mixture was applied to a chip and automatically stirred ten times. The processor was then covered in oil and gel particles. The Metal Controller station filtered the loaded chip. Following incubation, the emulsion was cleaned as follows: Each sample received 125 mL of Recovery Agent, the tubes containing the mixture were allowed to sit for 2 minutes while being carefully turned over, and then 130 mL of the lower pink phase were quickly centrifuged and chosen. Then, each sample was treated with the prepared Dynabeads Cleanup Mix and left at room temperature for 10 minutes. Following this, the magnetic particles were cleaned with 80% alcohol, and cDNA fragments were eluted using an EB buffer. A full-sized cDNA was produced through hen amplification. Following cDNA amplification, magnetic particle purification was done, and the purified cDNA's quality was checked with a Qubit 4 fluorimeter and an automated Tape Station.

Discussion

A prepared Fragmentation Mix and 10 L of the sample were used for cDNA fragmentation. The samples were washed bilaterally on magnetic particles after fragmentation. The adapters were then ligated by adding 50 L of the ready-made Adapter Ligation Mix to 50 L of the sample, which was then incubated for 15 min at 20 °C. After ligation, magnetic particles underwent purification. Indexing PCR was the last step in the library preparation process. After indexing, the samples underwent quality control and double-sided cleaning on magnetic particles. The nuclei with 200–2500 genes and less than 15% mitochondrial reads were kept by filters. Maintaining biological diversity. First, we determined the Jaccard similarity between each cluster found using either integration method, and we discovered a strong correlation between the two methods. For further analysis that offers a more biological separation of cell groups, we used integration based on Seurat CCA. The top 30 selected features were used as the basis for PCA and UMAP feature reduction.

The top 30 PCA reducing dimensions were used to calculate nearest-neighbor graphs, and clustering was used. With patient ID serving as the batch key and fallback parameters, Harmony was run on the PCA matrix. Two cell clusters that were thought to be doublets and had poor quality metrics were eliminated. Further ligand-receptor interactions discovered by immune cells' primary difference pathways in the Delta samples were looked into. Between Mon IFI30 cells, lymphocytes, and monocytes, we saw a wide range of communication. However, only macrophages have high fidelity complement pathways. The interactions created by SPP1 that were strongly overactivated in Delta but not Wuhan-like samples were then looked into. The SPP1-CD44 pair, which has been found to have the highest communication probability between monocyte subgroups, including autocrine loops, mediates the majority of SPP1 interactions. A robust immunological shift caused by cytokine storms that emerge

during COVID-19 results in a switch to a proinflammatory curriculum of myeloid cells in both venous and lung tissue, as well as compositional dysregulation in the lymphoid compartment. To determine the efficacy of treatments, identify disease prognostic markers, and comprehend the heterogeneity of disease severities, researchers are primarily interested in looking into immune responses in COVID-19 patients.

Depending on the severity of the symptoms and the SARS-CoV-2 virus variant, we combined clinical observations and single-cell transcriptome data using computational methods to generate a thorough, integrated view of the COVID-19 immune response. Here, using blood mononuclear cells from 40 newly sequenced individuals and 36 previously published samples, we created and examined a sizable single-cell transcriptomic atlas. We looked into PBMC samples with Wuhan-like or Delta virus variants and compared them to severe Flu, giving way different immunological mechanisms, in order to gain insight in to the immune response differences that SARS-CoV-2 variations bring to the immune response[1-5]. Here, using blood mononuclear cells from 40 newly sequenced individuals and 36 previously published samples, we created and examined a sizable single-cell transcriptomic atlas. and PBMC samples with Wuhan-like or Delta virus variants and compared them to severe Flu, giving way different immunological mechanisms, in order to gain insight in to the immune response differences that SARS-CoV-2 variations bring to the immune response.

Conclusion

The scRNA-seq profiling to look into the molecular processes connected to a severity of COVID-19 and the SARS-CoV-2 variant. The increased expression inflammatory signatures in the PBMCs from COVID-19 and HIV patients showed significant overlap in prior studies. The infection agent may cause different gene expression responses, but different immune cell subsets may share signatures with other conditions while also bringing their own characteristics. According to other studies, the sepsis cytokine storm that developed in COVID-19 patients had a milder effect but was associated with higher levels of immunoglobulin and complement protein. The severe Delta variant COVID-19 leads to specific immune shifts, according to the Mon IFI30 subtype's unique gene expression profile. A significant effect may activate extracellular vesicles and lead to future virus spreading mechanisms.

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