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Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection

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Abstract:	Background: Acute kidney injury (AKI) is common in SARS-CoV-2 infection and COVID-19, often leading to long-term kidney dysfunction. However, the transcriptomic features of AKI severity and its long-term effects are underexplored. Methods: We performed bulk RNA sequencing on peripheral blood mononuclear cells (PBMCs) from hospitalized SARS-CoV-2 patients and complemented these findings with proteomic data from the same cohort. We compared the functional enrichment findings with historical sepsis-AKI data and subsequently examined the association between molecular signatures and long-term kidney function changes. Results: In 283 patients, 57 had mild AKI (stage 1) and 49 had severe AKI (stage 2 or 3). Following adjustments for age, sex, severity of infection, and pre-existing chronic kidney disease (CKD), we identified 6,432 differentially expressed genes (DEGs) in the severe AKI vs. control comparison, 840 in the mild AKI vs. control, and 1,213 in the severe vs. mild AKI comparison (FDR<0.05). Common pathways included unfolded protein response, cellular response to stress via eIF2, and IFN-g-mediated inflammatory response. Severe AKI was linked to pathways involved in mitochondrial dysfunction and endoplasmic reticulum stress. Proteomic analysis confirmed 40 established AKI and inflammation biomarkers, while gene-set enrichment of transcription regulators revealed additional biomarkers for severe AKI. Comparison with PBMC transcriptomics from sepsis-related AKI showed significant functional overlap (30%). Analysis of post-discharge eGFR data in 115 patients identified 177 DEGs for severe vs. control, 106 for mild vs. control, and 46 for severe vs. mild AKI. Key associations included kidney function decline related to carbohydrate and mitochondrial metabolism, inflammatory-response, and cardiovascular regulation. Conclusions: We demonstrate that severe AKI in SARS-CoV-2 infection is linked to mitochondrial dysfunction and ER stress. The functional overlap with sepsis-AKI suggests potential broader therapeutic applicability. Long-term kidney dysfunction is influenced by disruptions in cellular energy metabolism and immune response.
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Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection

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ABSTRACT

Background: Acute kidney injury (AKI) is common in SARS-CoV-2 infection and COVID-19, often leading to long-term kidney dysfunction. However, the transcriptomic features of AKI severity and its long-term effects are underexplored.

Methods: We performed bulk RNA sequencing on peripheral blood mononuclear cells (PBMCs) from hospitalized SARS-CoV-2 patients and complemented these findings with proteomic data from the same cohort. We compared the functional enrichment findings with historical sepsis-AKI data and subsequently examined the association between molecular signatures and long-term kidney function changes.

Results: In 283 patients, 57 had mild AKI (stage 1) and 49 had severe AKI (stage 2 or 3). Following adjustments for age, sex, severity of infection, and pre-existing chronic kidney disease (CKD), we identified 6,432 differentially expressed genes (DEGs) in the severe AKI vs. control comparison, 840 in the mild AKI vs. control, and 1,213 in the severe vs. mild AKI comparison (FDR<0.05). Common pathways included unfolded protein response, cellular response to stress via eIF2, and IFN-g-mediated inflammatory response. Severe AKI was linked to pathways involved in mitochondrial dysfunction and endoplasmic reticulum stress. Proteomic analysis confirmed 40 established AKI and inflammation biomarkers, while gene-set enrichment of transcription regulators revealed additional biomarkers for severe AKI. Comparison with PBMC transcriptomics from sepsis-related AKI showed significant functional overlap (30%). Analysis of post-discharge eGFR data in 115 patients identified 177 DEGs for severe vs. control, 106 for mild vs. control, and 46 for severe vs. mild AKI. Key associations included kidney function decline related to carbohydrate and mitochondrial metabolism, inflammatory-response, and cardiovascular regulation.

Conclusions: We demonstrate that severe AKI in SARS-CoV-2 infection is linked to mitochondrial dysfunction and ER stress. The functional overlap with sepsis-AKI suggests potential broader therapeutic applicability. Long-term kidney dysfunction is influenced by disruptions in cellular energy metabolism and immune response.

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INTRODUCTION

Acute kidney injury (AKI) is common in hospitalized patients with SARS-CoV2 infection and COVID-19.[1,2] During the pandemic in the United States, AKI incidence was variable.[1,3-5] While the rates of AKI have decreased, it is still a significant complication.[6,7] However, the molecular pathophysiology of AKI severity in COVID-19 is unclear.[2,8]

Previous studies limited by selection bias, used post-mortem histopathological samples to understand COVID-19 associated AKI pathophysiology.[2,9-11]. Regardless, they demonstrated comparable morphological, transcriptomic, and proteomic features between COVID-19 associated and sepsis associated AKI.[12] Few studies[13,14] have been done on peripheral transcriptomics in COVID-19 patients spanning the full spectrum of AKI severity. Kidney dysfunction is a major component of post-acute sequelae of SARS-CoV2 (PASC). [15-17,18] However, peripheral transcriptomics linked to long term kidney dysfunction and PASC are not fully understood.[18]

We utilized transcriptomic analysis of peripheral blood mononuclear cells (PBMCs) of patients. We sought to (1) identify canonical pathways and genes differentially expressed across AKI severity, and to understand how they differ from one another, (2) compare functional signatures of AKI in COVID to sepsis using a bulk-transcriptomics dataset, and (3) evaluate the implications of DEGs in COVID-AKI on long-term kidney dysfunction and PASC.

MATERIALS AND METHODS

Patient Cohort and Study Design

We extracted patient demographic and laboratory data from electronic health records (EHR) between March 24th – August 26th, 2020.[16,19] The detailed patient recruitment and specimen collection procedures are previously described.[20]. We defined the groups as 'mild AKI'(MIL) for patients who developed AKI stage 1 (n=57), and severe AKI (SEV) for those who developed AKI stages 2 or 3 during their admission (n=49) and controls (CTRL) as patients (n=177) without AKI through the course of the study **(Figure 1A)**. While specimen collection occurred at multiple time points during the study period, we used samples acquired at the last available timepoint **(Figure 1B)**. If a patient had multiple AKI events during their hospitalizations, we included the sample collected after their last AKI event to avoid repeat sampling/confounding.[21] We excluded patients who developed AKI after the last specimen collection. This research was reviewed and approved by the Icahn School of Medicine at Mount Sinai Program for the Protection of Human Subjects (PPHS) under study number **20-00341**. Data for the analysis including the clinical covariates are available in Synapse syn35874390.[16] All clinical experimentation methods pursued in this study are in adherence with the Declaration of Helsinki.

Definition of Acute Kidney Injury

We defined AKI per Kidney Disease Improving Global Outcomes (KDIGO)[22] criteria: increase in serum creatinine by an absolute value of 0.3 mg/dL in a period of 48 hours or by at least 1.5 times the baseline creatinine (historical measurement) within 7 days. For patients with previous serum creatinine measurements available in 365 days before admission, we considered the minimum value as the baseline creatinine. For patients without baseline creatinine in this period, a baseline reference value was used based on an estimated glomerular filtration rate (eGFR) of 75 ml/min per 1.73m^2 as per KDIGO AKI guidelines.[23,24]

Differential expression analysis

We carried out differential expression analysis between cases and controls using *limma* (R v4.3) and adjusted the model for sex, age, severity of infection, diabetes and chronic kidney disease (CKD) . We used cell-type deconvolution using CIBERSORTX[23,24] to estimate cell-type proportions in each sample using LM22 PBMCs as reference[16] and then iteratively adjusted the linear model for neutrophils, plasma cells, and CD4+ memory activated T-cells[25] (**Supplemental Information)**. We iteratively identified a series of non-redundant clinical, technical, and biological covariates whose effect was the strongest observed driver of and accounted for a substantial fraction of variation in gene expression. After multiple testing correction on the p-values of the genes (Bonferroni-adjusted *FDR* < 0.05), we plotted the statistically significant genes in volcano plots **(Figure 2A-C)** to depict the separation between the expression of genes

with increased and decreased transcription abundances through log-fold change (logFC) for each of the contrasts, 1) severe AKI vs no AKI (SEV-CTRL), 2) severe AKI vs mild AKI (SEV-MIL) and 3) mild AKI vs no AKI (MIL-CTRL).

Validation of molecular signatures across orthogonal proteomics data

To evaluate the link between our gene signatures and COVID-19-associated AKI, and to compare molecular subgroups, we applied the same transcriptomic analysis to a subset of COVID-19-AKI patients (n=283) using plasma proteome[21]. This allowed an independent comparison of RNA and protein data from the same patients. We used a linear model with the Somascan assay of 5,000 proteins, adjusting for the same clinical features. We then identified differentially expressed proteins (FDR < 0.05) and compared with transcriptomic DEGs.

Comparison with Sepsis associated AKI.

To account for differences between sepsis associated AKI and COVID-AKI, we compared statistically significant (FDR <0.05) DEGs and enriched pathways overrepresented in a previously published dataset (CTRL: n=58, SEV: n=39, MIL: n=36),.[25] We performed differential expression analysis using the same workflow adjusting the linear model for equivalent parameters gender (sex), sepsis severity groups (severity), age at enrollment (age), existing comorbidity parameter correlated with CKD and diabetes, equivalent scaled counts for cell-types to identify differentially expressed genes (FDR <0.05) across similarly designed contrasts as in the COVID-AKI

analysis (SEV-CTRL, SEV-MIL, MIL-CTRL). We then performed a one-sided Fisher's exact test to identify enrichment and analyze overlap between the DEGs from this study (sepsis-AKI) and COVID-AKI for each contrast group.

Functional association and statistical analysis

We performed pathway overrepresentation and gene-set enrichment analysis for the DEGs using annotations from the mSigDB[26] dataset to uncover functional associations and causal networks enriched (FDR < 0.05) for differentially expressed genes both in the COVID-AKI and sepsis-AKI. Annotations from hallmark pathways, c3 regulatory targets gene sets and Reactome pathways were investigated for both, increased, and decreased abundances of gene expression across all contrasts (**Figure 3A-C**). We then compared overrepresented pathways and gene set enrichment of regulatory targets to test (Fisher's test) for significance of overlap of common elements across COVID-AKI and sepsis-AKI using the GeneOverlap[27] v1.40.0 R package.

Characterization of long-term kidney function using markers of AKI

We calculated estimated glomerular filtration rate (eGFR) values using post-discharge creatinine measurements from returning patients, applying the CKD-EPI equation.[28] Clinical data, including protein measurements, were extracted from electronic health records starting the day after discharge until December 2, 2021. We included 115 surviving patients with at least one outpatient eGFR measurement. To explore the relationship between AKI gene signatures and long-term kidney function, we

used a mixed-effects linear regression model (lme4[29] package in R v4.0.3). This model considered factors such as comorbid CKD, ventilation, AKI severity (based on peak creatinine), and the interaction between RNA-seq sample extraction time and gene expressions, with patient ID as a random effect to account for repeated measures. We assessed gene expression significance using a t-test with Satterthwaite degrees of freedom from the *lmerTest* R package[29] and adjusted p-values using the Benjamini-Hochberg procedure (FDR < 0.05).[30] For each comparison group (SEV-CTRL, SEV-MIL, MIL-CTRL), we filtered the gene lists to exclude those with an absolute fold change less than 1.2 and absolute β-estimate values below 2. Remaining genes were then ranked by their fold change and β-estimate to identify the top genes for each comparison. For each comparison group (SEV-CTRL, SEV-MIL, MIL-CTRL), we filtered out genes with an absolute fold change below 1.2 and absolute β-estimates below 2, ranking the remaining genes by these metrics to identify the top candidates. We visualized average changes in eGFR over time by stratifying gene expression into tertiles (bottom, middle, top 33%) for each group (**Supplemental Figure 5. left panel**). Additionally, we estimated the monthly percentage change in eGFR by analyzing the interaction between gene expression levels and days since sample extraction (**Supplemental Figure 5. right panel**).

RESULTS

Description of the study population

Of 283 patients, 106 (37%) had AKI, with 49-severe AKI (stages 2/3) and 57-mild AKI (stage 1). Patients with AKI were (**Table 1.A.**) older (67 years vs. 61 years, p=0.0081). Race distribution was similar across groups. Severe AKI patients had higher SOFA scores[31] (7.27 vs. 4.53 for mild AKI and 1.2 for controls, p<0.001) and elevated baseline creatinine levels (1.22 mg/dL vs. 0.937 mg/dL for mild AKI and 0.929 mg/dL for controls, p<0.0001). AKI was also associated with higher rates of atrial fibrillation and type 2 diabetes, particularly in severe cases. Severe AKI patients had a higher prevalence of pre-existing CKD (31% vs. 11% for mild AKI and 5% for controls, p<0.001) and were more likely to receive vasopressors (57% vs. 47% for mild AKI and 7% for controls, p<0.001).

In the follow-up cohort of 115 patients (**Table 1B**), age was similar across groups (63 years for mild AKI, 58 years for severe AKI, and 59 years for controls). The cohort was 52% male and 24.3% White, with 11% having comorbid CKD (60% of whom had severe AKI) and 30% having type 2 diabetes. The median follow-up period was 162 days, with a median of four eGFR measurements per patient (**Table 1C and Supplemental Table 1A**).

Functional Analysis of Differential Expression

Differential expression analysis identified distinct gene expression patterns across three comparisons. Among severe AKI to controls (SEV-CTRL) comparison, we found 6,432 differentially expressed genes (DEGs), with 2,910 upregulated and 3,522 downregulated in the severe AKI group (FDR < 0.05). Comparing severe AKI to mild AKI (SEV-MIL) revealed 1,213 DEGs, including 514 upregulated and 699 downregulated genes in severe AKI. Comparison between mild AKI and controls (MIL-CTRL) identified 840 DEGs, with 369 genes upregulated and 471 downregulated in the mild AKI group. (**Figure 2.A-C, Supplemental Table 2A**).

Gene set enrichment analysis of differentially expressed genes using the mSigDB[32] C3 gene set collections identified 76 regulators involved in upstream regulation of genes across at least two groups (**Figure 3A**). Four genes—*HOXA1, TAF9B, ZFHX3*, and *ZNF318*—were common to all three groups. Seven regulators, including *KAT5, NKX2-2, FOXR2, SNRPN70, HOXA1*, and *SETD1A*, were enriched in both the SEV-CTRL and MIL-CTRL groups, while the *mTOR* regulator *ZZZ3* was common to both the MIL-CTRL and SEV-CTRL groups. The remaining 64 genes shared between the SEV-CTRL and SEV-MIL groups included known AKI regulators such as *TFAM* (OR 4.6, adj. p-value 0.02), *EGFR* (OR 3.7, FDR 0.01), and *PER1* (OR 3.7, FDR 0.01). In addition, enrichment analysis also uncovered potential promising biomarkers, *MIR492* (OR 4.6, FDR: 0.02), *MIR5571*_3P (OR 3.4, FDR: 0.01) and *MIR5591*_3P (OR 3.25, FDR: 0.05) for severe AKI. (**Supplemental Table 2B.a-c.**).

Pathway overrepresentation analysis using Hallmark H Pathways[32] and C2 KEGG and Reactome pathways identified several enriched canonical pathways via right-tailed Fisher's exact test ($p < 0.05$, Benjamini-Hochberg correction). The only pathway enriched in both the SEV-CTRL and MIL-CTRL groups within the Hallmark datasets was the 'HALLMARK_MYC_TARGETS_V1' pathway. (**Supplemental Table 3.A.**). Additionally, several pathways from C2 datasets related to SARS-CoV-2 infection and EIF1-driven eukaryotic translation initiation and elongation (**Supplemental Table 3.B.a-c**). Common pathways across all three groups included 'NONSENSE MEDIATED DECAY (NMD)', 'REGULATION OF EXPRESSION OF *SLITS* AND *ROBOS'* (regulated by *HOXA1* and *NKX2-2*), and 'RESPONSE OF *EIF2AK4 GCN2* TO AMINO ACID DEFICIENCY' (**Figure 3B**). Pathways shared between the SEV-CTRL and MIL-CTRL groups involved cytokine signaling, such as the 'CHEMOKINE SIGNALING PATHWAY', '*IL3* SIGNALING PATHWAY'. Enriched pathways unique to the severe AKI groups included 'OXIDATIVE PHOSPHORYLATION', the 'INSULIN SIGNALING PATHWAY', cell-adhesion and migration pathways (**Figure 3C1-3, Supplementary Information**).

Validation of molecular signatures across an orthogonal plasma proteomics dataset

Gene overlap across transcriptomic and proteomic analyses results uncovered 37 genes (**Supplemental Information**) between the 'SEV-CTRL' group in both COVID-AKI proteomic and transcriptomic analysis datasets. Top genes include *HAVCR1, CXCL16, IL17RC,* and *ITGB2*. MCL cluster analysis of these genes based on prior evidence from the STRING database uncovered clusters of known markers for

inflammation. Specifically, *HSPA1A, DNAJB12, CA3, CA4, TNFRSF1A and SUMO2* were significantly differentially expressed in both datasets. Comparatively, 2 genes overlapped the 'SEV-MIL' group in bath datasets, *CHP1* and *MAP2K2*. Overlap for enrichment of DEGs in the 'MIL-CTRL' group did not result in a significant overlap (**Supplemental Information**).

Comparison with sepsis associated AKI.

Comparison of the DEGs from the sepsis-AKI analysis with the COVID-AKI analysis only generated significant results (FDR<0.05) for the 'SEV-CTRL' group **(Supplemental Table 4.A.)**. A higher proportion of genes showed reduced expression (671 DEGs vs 9 DEGs upregulated). Of the 671 DEGs, 28 genes overlapped with the COVID-AKI DEGs with a positive fold change. Fisher's exact test for enrichment (performed via *GeneOverlap* R package) resulted (**Supplemental Information**) in non-significant overlap. Pathway overrepresentation analysis for the SEV-CTRL group in the sepsis-AKI dataset identified two significant Hallmark pathways related to apoptosis and ER-stress-response (**Supplemental Table 4.B.**).

Enrichment of regulatory targets in the SEV-CTRL group using C3 gene sets from the mSigDB database revealed 49 significant (FDR < 0.05) DEGs in the sepsis-AKI dataset (**Supplemental Table 4.C.**). Comparison with COVID-AKI cohort showed 13 common transcription regulators in the SEV-CTRL group, (p-value 2.2e-04, OR 3.8) determined by a Fisher's exact test for enrichment (**Supplemental Table 4.D., Supplemental Information**).

Characterization of post-acute kidney dysfunction

Mixed model analysis identified 2,490 significant differentially expressed genes (DEGs) for SEV-CTRL group, 411 for MIL-CTRL group, and 357 for SEV-MIL group, associated with changes in eGFR (Supplemental Table 5.A-C). After filtering out DEGs with absolute fold change below 1.2 and absolute β-estimates below 5, we retained 177 significant DEGs for SEV-CTRL, 106 for MIL-CTRL, and 46 for SEV-MIL. In the SEV-CTRL comparison, top genes (**Figure 4.A-C**) positively correlated with eGFR included *NECAB1, CD177, MYO10*, and *GPR84* (positive logFC), while *ADARB2* (*ADAR3*) and *GNG8* showed a negative logFC. Inversely correlated genes included *NR4A1* and *PCDH12* (positive logFC) and *TRDV1* and *CCN3* (negative logFC). For the MIL-CTRL group, *ITGA7, HPGD*, and *ROBO1* were positively correlated with eGFR, whereas *TRDJ2, CACNB4*, and *RPGRIP1* showed inverse correlations. In the SEV-MIL group, *MYO10, TRPM2, and NDRG1* were positively correlated, while *CDC42EP1, UBXN11, OCRL, and PDE4* had negative correlations.

Analysis of association of β-estimates for mean eGFR and gene expression revealed distinct signatures among severe AKI, mild AKI, and control patients over time. eGFR trend indicated that the interaction between ‘gene expression during hospitalization’ and ‘time’ since RNASeq sample extraction significantly influenced post-discharge eGFR measurements (**Supplemental Information, Supplemental Figure S5**). Additionally, significant percent changes in eGFR were observed across each gene expression tertile (**Supplemental Figure S6**).

DISCUSSION

This study is the largest peripheral transcriptomics analyses in SARS-CoV-2 patients with follow-up data, differentiating between mild (stage 1) and severe (stage 2/3) outcomes. Our bulk RNA-Seq analysis of PBMCs reveals that severe AKI is associated with disruptions in regulatory markers of ER stress and mitochondrial dysfunction in circulating immune cells. Additionally, we validate these findings using a proteomic dataset from the same cohort. Our results uncover functional similarities between transcriptomic regulation in COVID-19-associated AKI and sepsis-AKI. Finally, we show that some of these markers are associated with long-term kidney dysfunction, particularly involving autophagy, fibrosis, insulin regulation, and cardiac function.

The deliberately narrow time frame for patient selection was designed to minimize instances of reinfection or the application of FDA-approved emergency treatments, thereby enabling the identification of meaningful associations. By comparing the SEV-CTRL, SEV-MIL, and MIL-CTRL groups, we differentiated molecular changes associated with severe, mild and no AKI (controls). This analysis enabled us to investigate functional differences and identify promising biomarkers. Overlapping results between SEV-CTRL and SEV-MIL indicate signatures specific to severe AKI, while overlaps between SEV-CTRL and MIL-CTRL suggest signatures related to AKI development. Signatures common to all three groups point to associations with AKI across all stages. Our model accounted for known confounders, including diabetes, acuity (SOFA scores), infection severity, blood urea nitrogen (BUN), ventilatior dependence, and inotropic support, demonstrating that the inclusion of diabetes and severity effectively addressed the influence of these factors (Supplemental Information).

Our analysis revealed a widespread downregulation of mitochondrial genes in severe AKI. Mitochondrial dysfunction is a well-established contributor to the pathogenesis of tubular injury in acute kidney injury.[33,34] While our study primarily examined gene expression changes in PBMCs, it is important to note that systemic mitochondrial alterations can reflect and potentially influence organ-specific mitochondrial impairment, including in kidney tubular cells[35,36]. In the context of AKI, mitochondrial dysfunction can release mitochondrial DNA (mtDNA) and other components that act as damage-associated molecular patterns (DAMPs), capable of activating immune cells, including PBMCs, and triggering inflammatory cascades that may worsen kidney injury[37]. The observed downregulation of mitochondrial pathways in our study may contribute to maladaptive stress responses, potentially aggravating the injury process. Recent research has highlighted the critical role of mitochondrial dynamics[38] in proximal tubules during AKI pathogenesis and recovery, underscoring the complex interplay between mitochondrial function and kidney injury. This is particularly relevant in the context of COVID-19, as mitochondrial dysfunction and impaired oxidative phosphorylation have been implicated in both COVID-19-associated AKI and CKD. Given these findings, therapeutic strategies targeting mitochondrial dysfunction,[39,40] and cellular therapies that have shown promise in treating ischemia-reperfusion injury could potentially be repurposed for COVID-19-associated AKI[41] and CKD.[42] These approaches may offer novel avenues for intervention in this severe complication of COVID-19, though further research is needed to establish their efficacy in this specific context.

In addition to inflammation and cellular stress responses, several molecular biomarkers have been linked to worsening renal damage in patients with COVID-AKI. The differentially expressed genes (DEGs) include regulatory genes associated with renal recovery from ischemia[43], β-cell dysregulation[44], immune cell proliferation[45], and increased atherosclerosis risk[45]. Overall, AKI was linked to a known regulator of the mTOR pathway.[46] Certain microRNAs were found to regulate a significant number of DEGs, with MiR-492 (OR 4.62, p = 0.023) being particularly noteworthy; to our knowledge, it has not previously been associated with AKI[47]. However, it has been studied for its role in the progression of endometrial cancer[48] through NFE2L1-regulated cell proliferation and apoptosis inhibition. MicroRNAs may facilitate early diagnosis and prognosis of kidney injury due to their stability in biofluids.[49] Recent studies[50] have identified drugs targeting MiR-492 in endometrial cancer cells, which could be further investigated for AKI treatment.

Orthogonal validation of significant transcriptomic signatures using a plasma proteome dataset identified 37 known markers [51] of insulin metabolism[52], inflammation[53], cardiac[54], and kidney dysfunction. CHP1, which regulates pH through NHE1[55] and is involved in the MAPK2/ERK pathway[56] for cellular stress response, was overexpressed in AKI patients. Additionally, a cluster of heat-shock protein genes, including *HSP70 (HSPA1A)* and *HSP40 (DNAJB12)*, associated with cellular stress[57], tubular epithelial cells polyploidization[58], inflammation (*TNFRSF1A*), and post-translational sumoylation,[59] were significantly differentially expressed. Identifying these genes in another sample type from the same cohort validates our findings at both transcriptomic and proteomic levels.

Pathways enriched in persistent severe AKI are linked to molecular disruptions in stress markers,[60] insulin metabolism, cell adhesion and migration, mitochondrial dysfunction, and oxidative phosphorylation. Worsening AKI (SEV-MIL comparison) was associated with immune signaling along with apoptosis, cell proliferation and ER-stress response which may reflect the role of macrophages and cytokines in kidney injury in worsening AKI.[61,62] We show significant regulation (FDR<0.05) of the eIF2 and mTOR pathways, implicated in ischemia-reperfusion and sepsis-associated AKI[63,64]. The mTOR pathway is interconnected with ER stress through the eIF2/4 complex[63,65] and mTOR inhibitors (e.g. Rapamycin) have previously been shown to impair tubular cell regeneration[66] and delay the recovery of renal function[46] after AKI. However, mTOR inhibitors may provide protective effects in injured kidneys by increasing anti-inflammatory cytokines[67] and enhancing MDSC-mediated immunosuppression[68], potentially reducing acute kidney injury (AKI). This suggests that mTOR expression plays a role in AKI development and progression. However, further research is required to clarify these effects in kidney injury models and to determine the optimal conditions for their clinical use.

Previous studies using histopathology and gene expression highlight significant similarities in the morphological and molecular profiles of AKI in both COVID-19 and sepsis.[69,12] We find that 30% of the 49 reported pathways were common to both conditions. Key overlapping pathways included TNF-α signaling via the NFKB pathway, (immune dysregulation[70], and systemic inflammation), and unfolded protein response via the P53 pathway (autophagy[71] in AKI). The overlap of transcriptional regulators across both datasets (further supports a functional similarity between AKI in COVID-19

and sepsis. Thus, therapies targeting the immune system in sepsis-associated AKI offers potential therapeutic benefits.

CKD is a key component of PASC[15,18] with persistent inflammation contributing to fibrosis through cytokines. Our analysis identified a significant number of differentially expressed genes (DEGs) across the SEV-CTRL, MIL-CTRL, and SEV-MIL groups that are linked to post-discharge CKD progression. These DEGs could serve as biomarkers for risk stratification and potential therapeutic targets. Our model showed that during hospitalization, genes with fold changes that positively correlated with β-estimates (both positive or both negative) had a protective effect on long-term eGFR. Conversely, genes with fold changes inversely correlated with β-estimates were associated with worse long-term kidney function (**Table 1.D.**). Additionally, we found significant trends in the percentage of monthly eGFR change for each gene expression tertile (**Supplemental Information, Supplemental Figure 5-6**).

Major strengths of our study include (1) our relatively large and racially and ethnically diverse patient cohort[72] (2) the use of multiple modalities of 'omics' data from the same cohort. However, there are some limitations. First, samples were collected at inconsistent timepoints during hospitalization, with no uniform post-hospitalization collection. Our post-discharge cohort was also limited to 115 patients, as 168 either died (18%) or did not follow up. Additionally, while COVID-19 AKI samples were collected in 2020, the sepsis-AKI cohort came from a 2015 study with different processing methods, potentially affecting data comparison. However, this avoided confounding by SARS-CoV-2 infections. While AKI rates have declined with vaccination, cases in vaccinated individuals still suggest a need for further investigation[73-75] into causality and the

generalizability of our findings across vaccinated cohorts. While there is evidence suggesting that organ dysfunction or injury may be associated with mitochondrial stress in peripheral blood mononuclear cells (PBMCs)[76], our study does not directly evaluate tubular mitochondrial injury or endoplasmic reticulum stress in kidney tissue. The miRNA signatures detected may reflect early-stage miRNAs due to the TrueSeq protocol's filtering of small RNAs. Though statistically significant, it is unclear if these miRNAs reach functional maturity. Additionally, the lack of an external validation set for COVID-19-associated AKI limits the generalizability of our findings, though orthogonal validation with proteomic data supports their relevance. Finally, the study's cohort design is hypothesis-generating thus, does not establish causality. In addition, the absence of precise EHR timing for renal replacement therapy (RRT) limits conclusions. It is also important to note that these pathways were identified in PBMCs rather than kidney cells. While this provides insight into systemic responses to AKI, further research is needed to determine how closely these molecular changes in circulating immune cells reflect pathways activated in the kidney itself during injury.

In conclusion, we present transcriptomic analyses of acute and long-term kidney dysfunction in SARS-CoV-2 infection in a large clinical cohort. Mitochondrial dysregulation, oxidative phosphorylation, and ER stress were strongly associated with worsening AKI. Chronic inflammation, tissue damage, and extracellular matrix buildup contribute to CKD progression. Existing drugs targeting some of the identified pathways show potential for therapeutic repurposing. Future work will expand the cohort with single-cell RNASeq data and apply advanced stratification methods to better capture AKI progression, particularly in hemodialysis patients.

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FIGURES:

Figure 1a. Flowchart of patients included in the current study.

Figure 1b. Timeline of sample collection for current study

Figure 1 Legend. Figure 1a shows the patients and samples included in the current study, and Figure 1b shows a visualization of the sample collection timeline.

Fig 1.A.

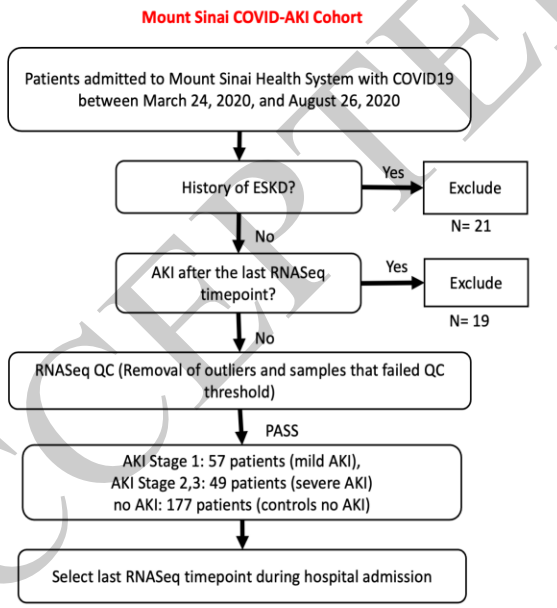


Fig 1.B.

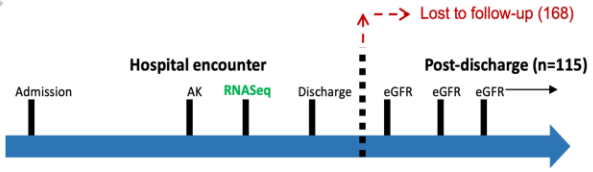


Figure 2a. Volcano plot of the differentially expressed genes for the severe AKI (AKI stage 2/3) vs control (no AKI) comparison group.

Figure 2b. Volcano plot of the differentially expressed genes for the severe AKI (AKI stage 2/3) vs mild AKI (AKI stage 1) comparison group.

Figure 2c. Volcano plot of the differentially expressed genes for the mild AKI (AKI stage 1) vs control (no AKI) comparison group

Figure 2 Legend. Volcano plots for each of the comparison groups show that a majority of the genes have fold changes greater than 1.5 across the comparison groups.

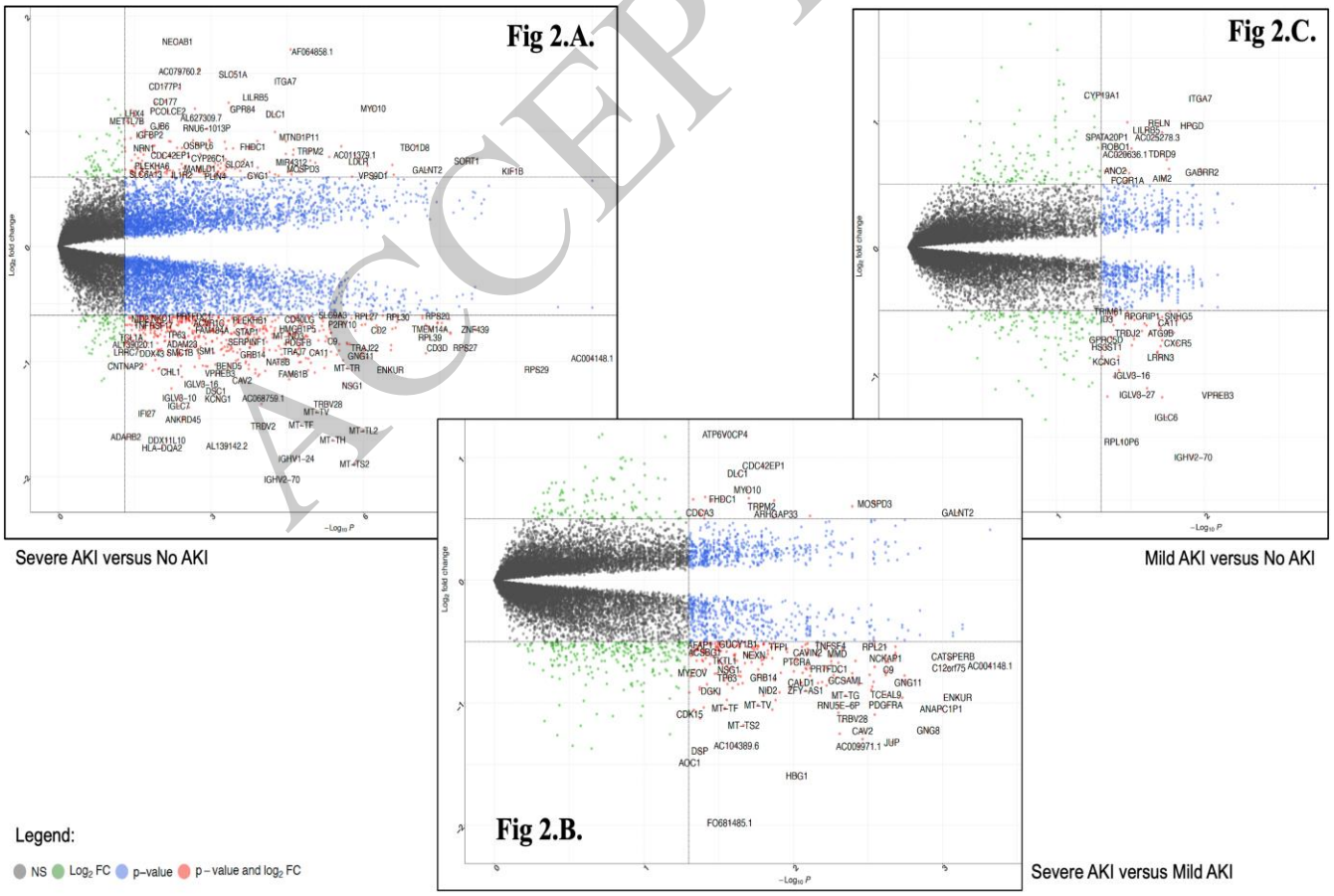
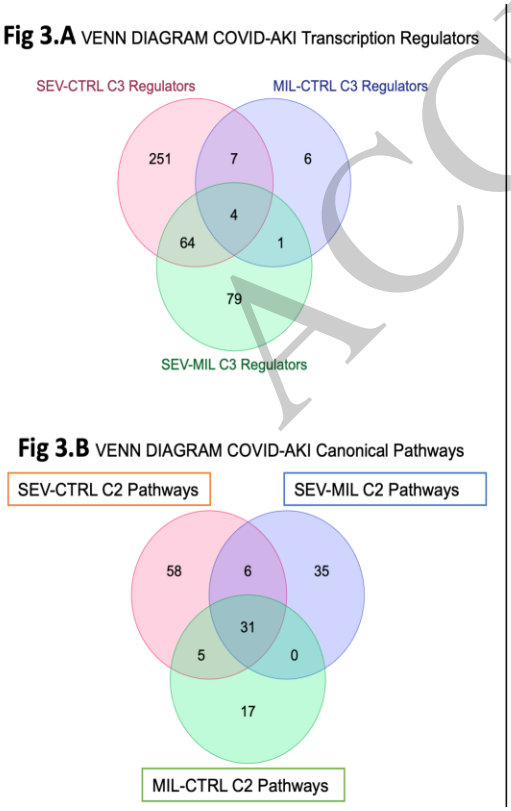


Figure 3.A. Legend. Venn Diagram of the common Transcription regulators across the three comparison groups uncovers 4 common regulators that are also known biomarkers for AKI.

Figure 3.B. Legend. Venn Diagram of the common Canonical Pathways across the three comparison groups uncovers 31 common pathways which also includes 6 pathways unique to severe or worsening AKI and 5 pathways that are common to all forms of AKI severity.

Figure 3.C1-3. Legend. Bar plots of significantly enriched (-log10 adjusted P-values) canonical pathways across the three comparison groups shows eukaryotic translation and elongation and EIF2AK4 GCN2 amino acid deficiency being common across all three comparison groups.



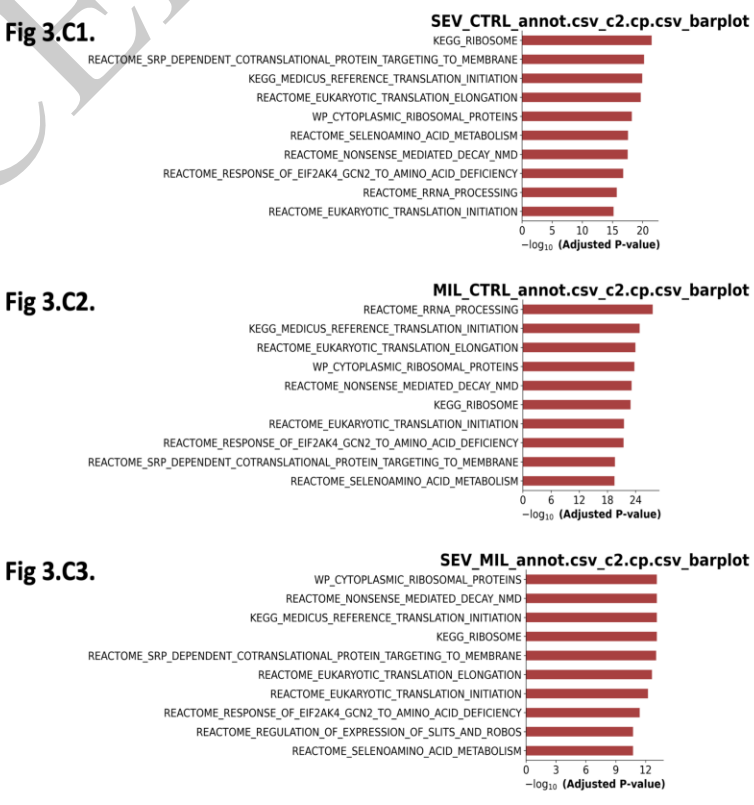
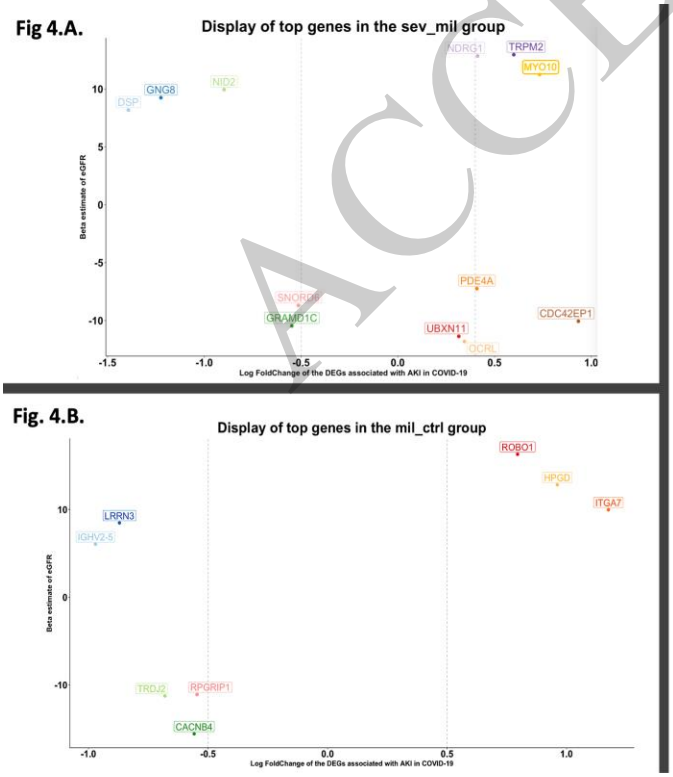


Figure 4.A-C. Association of top differentially expressed gene signatures in AKI in the COVID-AKI cohort and overall decline in eGFR in the long-term cohort 1-year post-discharge across all three comparison groups.

Figure 4 Legend. Figure 4 plots the molecular signatures of COVID-19-AKI (across all three comparison groups) from the COVID-AKI cohort against b-estimates of the linear mixed model analyzing the effect of these expressions on overall kidney function decline as measured by eGFR (over 1-year post-discharge). Gene expressions are plotted on the x-axis (as a function of their log2 fold change) and the y-axis is the beta estimate of the overall change in long-term eGFR. Genes with a significant fold-change that positively correlated β-estimate of association (both positive, or both negative) with mean eGFR conferred a protective effect on overall eGFR long-term. On the contrary, genes with a significant fold change that inversely correlated β-estimate of association (positive fold-change, negative β-estimate, or negative fold-change, positive β-estimate) worsened kidney function in the long-term.



LIST OF SUPPLEMENTAL MATERIALS:

Supplemental Methods

Sample Collection, Processing and RNA Sequencing

RNA-Seq Alignment and Quantification

RNA-Seq Count Data Processing and Normalization

Cell Type Deconvolution and Selection for The Linear Model.

Developing The Linear Model for RNA-Seq Analyses.

Differential Expression Analysis

Supplemental Results

Principal Component Analysis

Differential Expression Analysis

Validation using orthogonal proteomics cohort

Comparison With Sepsis Driven Aki (S-AKI)

Analysis Of Long-Term Kidney Dysfunction from Post-Discharge eGFR Measurements

Supplemental Tables

Data file Supplemental Table (Excel file) with sheets S1 - S5.C

SUPPLEMENTAL MATERIAL FIGURES:

Supplemental Information. Information on additional methods and descriptions used in the study.

Supplemental Figure 1A. Heatmap inspecting the collinearity of specific variables allowed the selection of “severity” as a parameter to also account for Bun and SOFA in the model.

Supplemental Figure 1.B. Principal Component Analysis (PCA) of the raw gene expression counts show possible separation along PC2 and PC3.

Supplemental Figure 1.C. The expression of a few select genes and their feature covariates have been plotted.

Supplemental Figure 2. Venn Diagram inspecting the common DEGs across the three original comparison groups and patients with RRT(n=10).

Supplemental Figure 2.B. Intersection of DEGs from the COVID-AKI transcriptomics cohort and orthogonal dataset from the proteomic analysis of AKI in COVID-19 patients shows common genes of interest within the SEV-CTRL cohorts across both studies.

Supplemental Figure 2.C. Gene-interaction network of the common genes from the proteomic analysis of AKI in COVID-19 patients using STRING DB identifies cluster of genes involved in complement dependent cytotoxicity, TNF-mediated adaptive immunity, unfolded protein response using heat-shock proteins, inflammatory mediation via cell chemotaxis, ER-Golgi transport using COPI and cardiomyopathy.

Supplemental Figure 3.A. Plots of distribution of key covariates across sepsis-AKI and COVID-AKI cohorts.

Supplemental Figure 3.B. Venn Diagram of the significant differentially expressed genes from a published sepsis-AKI manuscript and COVID-19-AKI analyses reveals 111 of the 630 sepsis-AKI genes are also involved in COVID-19-AKI as well.

Supplemental Figure 4A. Intersection of DEGs between sepsis-AKI cohort and COVID-19 AKI cohort.

Supplemental Figure 4.B. Intersection of key transcriptional regulators between the sepsis-AKI and COVID-19 AKI cohort showed 13 common regulators that were significant overlaps hinting at a functional similarity between sepsis-AKI and COVID-19 AKI.

Supplemental Figure 5.A. Trends in percent change in eGFR over time differ across gene expression tertiles. Percent change in eGFR are plotted across top genes from each of the comparison cohorts and show that the trends are different across the gene expression tertiles for hospitalized COVID-19 patients AKI.

Supplemental Figure 5.B. Significant DEGs from COVID-AKI analysis that are also associated with overall levels of eGFR and have a 50% change in their fold change. While the top genes are plotted in Figure 4 (Main manuscript), we provide the full figure with all genes below.

Supplemental Table 1. Summary of post-discharge eGFR measurements within the long-term eGFR decline cohort.

Supplemental Table 2.A. Result of the Differential Gene expression analysis for the COVID-AKI cohort detailing fold-change and adjusted pValues across all three comparison groups.

Supplemental Table 2.B.a-c Enriched targets of transcriptional regulation for COVID-AKI across three comparison groups (SEV-CTRL, SEV-MIL, MIL-CTRL) respectively.

Supplemental Table 3.A. Hallmark pathways for COVID-AKI enriched using the *mSigDB* datasets for each of the three comparison groups (SEV-CTRL, SEV-MIL, MIL-CTRL) respectively.

Supplemental Table 3.B.a-c. Canonical pathways for COVID-19 enriched using the *mSIGDB* datasets for each of the three comparison groups (SEV-CTRL, SEV-MIL, MIL-CTRL) respectively.

Supplemental Table 4.A. List of significant differentially expressed genes (DEGs) for the severe AKI comparison group in the sepsis associated AKI cohort.

Supplemental Table 4.B. Hallmark pathways enriched for the DEGs within the severe AKI comparison group for the sepsis-AKI study.

Supplemental Table 4.C. transcriptional regulatory targets enriched for the DEGs within the severe AKI comparison group for the sepsis-AKI study.

Supplemental Table 4.D. Common transcriptional regulatory targets between significantly DEGs from the sepsis-AKI study and the COVID-AKI study for the severe AKI comparison group.

Supplemental Table 5.A-C. DEGs associated significantly with post-discharge eGFR quantified by the b-estimate of overall eGFR across all three comparison groups.

TABLES:

Table 1.A. Summary statistics for the COVID-AKI cohort.

Cohort Characteristics	Developed AKI (Stage 1/2/3) during hospitalization		No AKI during hospitalization	p-value
	AKI Severity			
	AKI stage 1 "*mild* AKI"	AKI stage 2/3 "*severe* AKI"	No AKI "control"	
Count (n)	N=57	N=49	N=177	
Age, mean (SD)	69.1 (15.2)	63.4 (15.3)	61.4 (16.7)	0.0080951
Male, n (%)	31 (54%)	32 (65%)	99 (56%)	0.462174
Race, n (%)				0.343764
White	25 (44%)	13 (27%)	57 (32%)	
Black or African American	9 (16%)	13 (27%)	43 (24%)	
Other	23 (40%)	23 (47%)	77 (44%)	
Ethnicity, n (%)				0.141378
Hispanic or Latino	28 (49%)	21 (43%)	62 (35%)	
Other Ethnicity	29 (51%)	28 (57%)	115 (65%)	
Vitals & Lab Parameters during Hospitalization, n (SD)				
SOFA	4.53 (3.87)	7.27 (4.45)	1.2 (1.65)	<0.001
Baseline Creatinine (mg/dL)	0.937 (0.317)	1.22 (0.779)	0.929 (0.27)	<0.00008
Maximum Lactate (mmol/L)	1.72 (0.608)	2.03 (1.73)	1.3 (0.43)	0.5164

Minimum Systolic Blood Pressure (mmHg)	109 (18)	102 (15.5)	111 (13)	0.001088
Maximum Systolic Blood Pressure (mmHg)	145 (21.2)	139 (20.1)	137 (20.5)	0.03803
Maximum Pulse (bpm)	100 (17.5)	105 (19.5)	90.5 (17.9)	<0.001
Maximum Blood Urea Nitrogen (mmol/L)	35.8 (24.2)	69.8 (35.7)	16.9 (10.4)	<0.001
Maximum White Blood Cell Count (10^9/L)	11.7 (5.43)	14 (8.59)	7.8 (3.46)	<0.001
Minimum Platelet Count (10^9/L)	311 (162)	226 (108)	318 (140)	0.000396
Minimum Lymphocyte Count (10^9/L)	2.11 (3.39)	1.94 (2.95)	2.31 (3.71)	0.82351
Maximum Creatinine (mg/dL)	1.24 (0.707)	4.31 (2.96)	0.862 (0.365)	<0.0001
Maximum Ferritin (ug/L)	926 (982)	2420 (2720)	932 (1240)	<0.0001
Maximum IL1-b (pg/mL)	NAN (NA)	0.4(NA)	0.6 (0.216)	0.4683
Cell proportions of Neutrophils (n)	0.646 (0.126)	0.659 (0.132)	0.552 (0.133)	<0.001
Comorbidities, n (%)				
Atrial Fibrillation	13 (23%)	8 (16%)	17 (10%)	0.032968
Coronary Artery Disease	11 (19%)	8 (16%)	26 (15%)	0.656803
Arterial Hypertension	28 (49%)	20 (41%)	71 (40%)	0.47542
Diabetes	21 (37%)	18(37%)	32 (18%)	0.00215
Chronic Kidney Disease	6 (11%)	15 (31%)	8 (5%)	<0.0001
Highest Respiratory support n (%)				
Intubation	24 (42%)	25 (51%)	9 (5%)	<0.0001
Non-Invasive Ventilation	6 (11%)	3 (6%)	8 (5%)	0.22461
Nasal Cannula	21 (37%)	13 (27%)	91 (51%)	0.00353

No Ventilation	6 (11%)	8 (16%)	69 (39%)	<0.0001
Vasopressor Use during Hospitalization n (%)				
Any vasopressor	27 (47%)	28 (57%)	12 (7%)	<0.001
Dobutamine	2 (4%)	0 (0%0	3 (2%)	0.53919
Norepinephrine	23 (40%)	26 (53%)	10 (6%)	<0.0001
Vasopressin	5 (9%)	13 (27%)	2 (1%)	<0.001
Phenylephrine	5 (9%)	10 (20%)	1 (1%)	<0.001
Epinephrine	2 (4%)	1 (2%)	2 (1%)	0.346192
Milrinone	1 (2%)	2 (4%)	1 (1%)	0.0965
Dopamine	0 (0%)	1 (2%)	0 (0%)	0.173144

Table 1.B. Patient cohort summary statistics for the post-discharge follow-up cohort for long-term eGFR analysis.

Cohort Characteristics	Developed AKI (Stage 1/2/3) during hospitalization		No AKI during hospitalization	p-value	Total (Category %)
	AKI severity				
	AKI stage 1 "*mild* AKI"	AKI stage 2/3 "*severe* AKI"	No AKI "control"		
Count (n)	N=13	N=18	N=84		115
Age, mean (SD)	63.1 (15.6)	58.3 (16.2)	59 (15.76)	0.65556	
Male, n (%)	8 (62%)	8 (44%)	44 (52%)	0.6224	60 (52.17%)
Race, n (%)				0.536824	
White	4 (31%)	3(17%)	21 (25%)		28 (24.34%)
Black or African American	4 (31%)	3(17%)	25 (30%)		32 (27.82%)
Other	5 (38%)	12 (67%)	38 (45.23%)		55 (47.82%)
Ethnicity, n (%)				0.05034	
Hispanic or Latino	5 (38%)	12 (67%)	30 (36%)		47 (40.86%)
Other Ethnicity	8 (62%)	6 (33%)	54 (64%)		68 (59.13%)
Vitals & Lab Parameters During Hospitalization, n (SD)					
SOFA	2.85 (2.67)	4.56 (2.59)	1.17 (1.82)	$p < 0.0001$	115
Baseline Creatinine	1.03 (0.168)	1.27 (0.768)	0.968 (0.211)	0.00585	115

(mg/dL)					
Maximum Lactate (mmol/L)	1.15 (0.354)	2.15 (1.77)	1.7 (0.283)	0.67365	115
Minimum Systolic Blood Pressure (mmHg)	113 (18.6)	106 (14.7)	112 (12.9)	0.2682496	115
Maximum Systolic Blood Pressure (mmHg)	139 (17.8)	136 (17.2)	138 (20.8)	0.8972864	115
Maximum Pulse (bpm)	97.8 (16)	99.4 (17)	89.5 (14.2)	0.01412	115
Maximum Blood Urea Nitrogen (mmol/L)	25.5 (14.1)	52.8 (30.1)	16.3 (9.41)	$p < 0.0001$	115
Maximum White Blood Cell count (10^9/L)	8.48 (5.49)	10.1 (4.98)	7.62 (3.25)	0.052677	115
Minimum Platelet count (10^9/L)	280 (195)	234 (113)	328 (151)	0.05343	115
Minimum Lymphocyte count (10^9/L)	3.67 (4.26)	2.52 (3.98)	2.59 (4.45)	0.732634	115
Maximum Creatinine (mg/dL)	1.14 (0.529)	3.03 (2.43)	0.894 (0.408)	$p < 0.0001$	115
Maximum Ferritin (ug/L)	1020 (1500)	2620 (3450)	981 (1380)	0.03059	115
Maximum IL-1b (pg/mL)	NAN (NA)	0.4 (NA)	0.7 (0.283)	0.54563	115
Cell proportions of Neutrophils (n)	0.523 (0.134)	0.611 (0.164)	0.526 (0.121)	0.038769	115
Comorbidities, n (%)					
Atrial Fibrillation	3 (23%)	5 (28%)	11 (13%)	0.224773	19 (16.52%)
Coronary Artery Disease	5 (38%)	3 (17%)	13 (15%)	0.14281	21 (18.26%)
Arterial Hypertension	7 (54%)	8 (44%)	40 (48%)	0.90948	55 (47.82%)
Diabetes	6 (46%)	8 (44%)	21 (25%)	0.107785	35 (30.43%)

Chronic Kidney Disease	0 (0%)	8 (44%)	5 (6%)	0.000191	13 (11.3%)
Vasopressor use during hospitalization. n (%)					
Any Vasopressor	6 (46%)	9 (50%)	7 (8%)	p < 0.0001	22 (19.13%)
Dobutamine	1 (8%)	0 (0%)	2 (2%)	0.35995	3 (2.61%)
Norepinephrine	4 (31%)	8 (44%)	6 (7%)	0.0001392	18 (15.65%)
Vasopressin	2 (15%)	5 (28%)	1 (1%)	0.0002726	8 (7%)
Phenylephrine	0 (0%)	2 (11%)	1 (1%)	0.096795	3 (2.61%)
Epinephrine	0 (0%)	0 (0%)	2 (2%)	1.000	2 (1.7%)
Milrinone	1 (8%)	1 (6%)	1 (1%)	0.1764	3 (2.61%)

Table 1.C. Additional summary statistics for the long-term eGFR patient cohort in **Table 1B.**

Cohort features	Min	Max	Median	Mean	SD
Age	20	90	62	59	15.75
Number of eGFR Measurements	1	58	4	10	169
Mean eGFR Values	4.5	140.22	76.41	74.93	29.51
eGFR Measurement	0.0258	2014.68	46.62	157.7	
Least Number of Days between Discharge and Follow-Up eGFR Measurement	1	520	34	78.83	33
Highest Number of Days between Discharge and Follow-Up eGFR Measurement	13	536	375	326.1	30

Table 1.D. Summary of the top genes associated with mean eGFR post-discharge

LFC/b-estimate	positive b-estimate (direct correlation with gene expression)		negative b-estimate (inverse correlation with gene expression)	
	Genes	possible function	Gene	possible function
Severe AKI group				
positive LFC (upregulated in AKI)	NECAB1	calcium binding regulation of insulin secretion of pancreatic **β**-cells[77]	NR4A1	renal interstitial fibrosis via the MAPK pathway[78]
	CD177	trans-endothelial neutrophil migration and ERK-mediated attenuation of chemokine signaling[79]	PCDH12	regulation of vascular physiology[80]
	MYO10	regulation of Pi3K during phagocytosis leading to fibrosis[81]		
	GPR84	g-protein coupled regulation of kidney fibrosis in post-acute kidney injury[82]		
positive LFC (downregulated in AKI)	ADARB2	regulation of APOL1 via A-to-I editing[83] thus influencing risk for kidney disease	CCN3	regulation of vascular smooth cell proliferation-mediated CKD73[84]
	GNG8	part of the gamma subunit for G-protein couples regulators.[85] GPCR-Gβγ signaling has been suggested as a potential therapeutic target for treating cardiorenal syndrome[86] and acute kidney injury.	TRDV1	chronic inflammatory tubulointerstitial fibrosis[87]
Progressively worsening AKI group				
positive LFC (upregulated in AKI)	NDRG1	mitochondrial dysfunction in response to hypoxia[88]	CDC42EP1	part of the part of the Borg family of Cdc42 effector proteins involved in podocyte apoptosis[89] induced proteinuria
	TRPM2	kidney fibrosis[90]	PDE4A	
	MYO10	regulation of Pi3K during phagocytosis leading to fibrosis[81]	UBXN11	dysregulation of DNA methylation[91]
			OCRL	regulation of glomerular function[92]
positive LFC (downregulated in AKI)	NID2	worsening of migration and invasion[93] of cells via PI3K-Akt signaling	GRAMD1C	reduced GRAMD1C expression in kidney renal clear cell carcinoma (KIRC)

				is shown to be associated with poor prognosis[94] and significant immune cell infiltration, especially of regulatory T cells (Tregs)
	GNG8	part of the gamma subunit for G-protein couples regulators.[85] GPCR-Gβγ signaling has been suggested as a potential therapeutic target for treating cardiorenal syndrome[86] and acute kidney injury.		
	DSP	functional regulation of pathways[95] involved in cardiomyopathy[96]		
MILD-AKI group				
positive LFC (upregulated in AKI)	ITGA7	regulation of cardiac muscle[97]		
	ROBO1	inflammatory response and management of immune cell migration[98]		
	HPGD	degradation of prostaglandins responsible for exacerbating[99] CKD[100]		
positive LFC (downregulated in AKI)	LRRN3	downregulated LRRN3 was shown to worsen risk for DNA methylation[101] a strong risk factor[102] for CKD	TRDJ2	regulation of T-cell receptors immune response in apoptosis[103]
	IGHV2-5	associated with autoimmune and cardiovascular conditions, such as idiopathic pulmonary arterial hypertension[104] (IPAH), suggesting that possible contribution to immune dysregulation	CACNB4	modification of signaling pathways for acute myocardial infarction[102]

ASN American Society of Nephrology
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N. Beckmann has nothing to disclose.

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Noam D. Beckmann
Manuscript ID: K360-2024-000472R2
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: October 30, 2024
Disclosure Updated Date: September 27, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
KIDNEY360 Accessing Our World From Every Angle
CJASN Clinical Journal of the American Society of Nephrology

ASN Journal Disclosure Form

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L. Chan reports the following:
Employer: Icahn School of Medicine at Mount Sinai; Consultancy: CSL Vifor Pharma, INC; and Research Funding: NIH.

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Lili Chan
Manuscript ID: K360-2024-000472R3
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: November 15, 2024
Disclosure Updated Date: May 8, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
KIDNEY360 Accessing Our World From Every Angle
CJASN Clinical Journal of the American Society of Nephrology

ASN Journal Disclosure Form

As per ASN journal policy, I have disclosed any financial relationships or commitments I have held in the past 36 months as included below. I have listed my Current Employer below to indicate there is a relationship requiring disclosure. If no relationship exists, my Current Employer is not listed.

A. Charney reports the following:
Employer: Icahn School of Medicine at Mount Sinai

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Alexander W. Charney
Manuscript ID: K360-2024-000472R3
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: December 13, 2024
Disclosure Updated Date: December 13, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
KIDNEY360 Accessing Our World From Every Angle
CJASN Clinical Journal of the American Society of Nephrology

ASN Journal Disclosure Form

As per ASN journal policy, I have disclosed any financial relationships or commitments I have held in the past 36 months as included below. I have listed my Current Employer below to indicate there is a relationship requiring disclosure. If no relationship exists, my Current Employer is not listed.

S. Chen reports the following:
Employer: Icahn School of Medicine at Mount Sinai

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Steven Tiwen Chen
Manuscript ID: K360-2024-000472R3
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: November 27, 2024
Disclosure Updated Date: November 27, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
KIDNEY360 Accessing Our World From Every Angle
CJASN Clinical Journal of the American Society of Nephrology

ASN Journal Disclosure Form

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S. Coca reports the following:
Employer: Icahn School of Medicine at Mount Sinai; Mount Sinai owns part of Renalytix; Consultancy: Renalytix, Takeda, Bayer, Boehringer-Ingelheim, Alexion, Mission Therapeutics, Mylan Pharmaceuticals, SC Pharma, Whiteswell; Ownership Interest: Renalytix; Research Funding: Renalytix, ProKidney; Patents or Royalties: Renalytix; and Other Interests or Relationships: Associate Editor for Kidney360, Editorial Boards of JASN, CJASN, Kidney International.

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Steven G. Coca
Manuscript ID: K360-2024-000472R3
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: November 21, 2024
Disclosure Updated Date: October 15, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
KIDNEY360 Accessing Our World From Every Angle
CJASN Clinical Journal of the American Society of Nephrology

ASN Journal Disclosure Form

As per ASN journal policy, I have disclosed any financial relationships or commitments I have held in the past 36 months as included below. I have listed my Current Employer below to indicate there is a relationship requiring disclosure. If no relationship exists, my Current Employer is not listed.

D. Del Valle has nothing to disclose.

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Diane Marie Del Valle
Manuscript ID: K360-2024-000472R2
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: October 30, 2024
Disclosure Updated Date: October 30, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
KIDNEY360 Accessing Our World From Every Angle
CJASN Clinical Journal of the American Society of Nephrology

ASN Journal Disclosure Form

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S. Dellepiane reports the following:
Ownership Interest: Roche; and Other Interests or Relationships: I work for Visterra pharma.

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Sergio Dellepiane
Manuscript ID: K360-2024-000472R3
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: November 20, 2024
Disclosure Updated Date: November 20, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
KIDNEY360 Accessing Our World From Every Angle
CJASN Clinical Journal of the American Society of Nephrology

ASN Journal Disclosure Form

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B. Fox reports the following:
Employer: Icahn School of Medicine at Mt Sinai

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Benjamin Fox
Manuscript ID: K360-2024-000472R1
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: September 30, 2024
Disclosure Updated Date: September 30, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
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S. Gnjatic reports the following:
Employer: Icahn School of Medicine at Mount Snai; Consultancy: Taiho pharmaceuticals; and Research Funding: Takeda, Regeneron, Boehringer-Ingelheim, Bristol Myers Squibb, Celgene, Genentech.

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Sacha Gnjatic
Manuscript ID: K360-2024-000472R1
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: September 27, 2024
Disclosure Updated Date: September 27, 2024

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E. GONZALEZ-KOZLOVA reports the following:
Employer: Icahn School of Medicine

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Edgar GONZALEZ-KOZLOVA
Manuscript ID: K360-2024-000472R2
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: October 30, 2024
Disclosure Updated Date: October 30, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
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CJASN Clinical Journal of the American Society of Nephrology

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P. Gownivaripally reports the following:
Employer: Capital One(Spouse); and Ownership Interest: Amazon, Robinhood, Shopify, Meta, AIRBNB, NXPI, WALMART.

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Pooja Anand Gownivaripally
Manuscript ID: K360-2024-000472R3
Manuscript Title: "Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection"
Date of Completion: November 15, 2024
Disclosure Updated Date: November 15, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
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CJASN Clinical Journal of the American Society of Nephrology

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F. Gulamali reports the following:
Employer: Icahn School of Medicine

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Faris F. Gulamali
Manuscript ID: K360-2024-000472R3
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: November 15, 2024
Disclosure Updated Date: November 15, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
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CJASN Clinical Journal of the American Society of Nephrology

ASN Journal Disclosure Form

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J. He reports the following:
Employer: Icahn School of Medicine at Mount Sinai; Consultancy: Renalytix AI; Yingli Pharmaceutical, Ono Pharmaceutical Co, LTD.; Ownership Interest: Renalytix AI; Rila Therapeutics; Yingli Pharmaceutical.; Research Funding: Shangpharma Innovation; Honoraria: Renalytix AI; Yingli Pharmaceutical, Ono Pharmaceutical Co, LTD.; and Advisory or Leadership Role: Editorial Board for Kidney International; Journal of the American Society of Nephrology, Diabetes, American Journal of Physiology, Board member of Chinese American Society of nephrology and International Chinese Society of Nephrology, Associate Editor for Kidney Disease, Section Editor for Nephron.

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: John Cijiang He
Manuscript ID: K360-2024-000472R2
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: October 30, 2024
Disclosure Updated Date: January 10, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
KIDNEY360 Accessing Our World From Every Angle
CJASN Clinical Journal of the American Society of Nephrology

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P. Jayaraman reports the following:
Employer: Icahn School of Medicine Mt sinai

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Pushkala Jayaraman
Manuscript ID: K360-2024-000472R1
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: September 30, 2024
Disclosure Updated Date: September 30, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
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CJASN Clinical Journal of the American Society of Nephrology

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J. Jiang reports the following:
Employer: Icahn School of Medicine at Mount Sinai

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Joy Jiang
Manuscript ID: K360-2024-000472R2
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: October 30, 2024
Disclosure Updated Date: October 30, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
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CJASN Clinical Journal of the American Society of Nephrology

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J. Kauffman reports the following:
Employer: Icahn School of Medicine at Mt Sinai

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Justin Kauffman
Manuscript ID: K360-2024-000472R1
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: September 27, 2024
Disclosure Updated Date: May 21, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
KIDNEY360 Accessing Our World From Every Angle
CJASN Clinical Journal of the American Society of Nephrology

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E. Kenigsberg reports the following:
Employer: Genentech; and Ownership Interest: Roche.

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Ephraim Kenigsberg
Manuscript ID: K360-2024-000472R3
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: January 13, 2025
Disclosure Updated Date: January 13, 2025

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
KIDNEY360 Accessing Our World From Every Angle
CJASN Clinical Journal of the American Society of Nephrology

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S. Kim-Schulze reports the following:
Employer: Icahn School of Medicine

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Seunghee Kim-Schulze
Manuscript ID: K360-2024-000472R3
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: January 14, 2025
Disclosure Updated Date: January 14, 2025

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
KIDNEY360 Accessing Our World From Every Angle
CJASN Clinical Journal of the American Society of Nephrology

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R. Langley reports the following:
Employer: University of South Alabama College of Medicine

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Raymond J Langley
Manuscript ID: K360-2024-000472R2
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: October 31, 2024
Disclosure Updated Date: October 31, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
KIDNEY360 Accessing Our World From Every Angle
CJASN Clinical Journal of the American Society of Nephrology

ASN Journal Disclosure Form

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L. Liharska reports the following:
Employer: Valis Biosciences Inc.; and Consultancy: Valis Biosciences Inc.

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Lora E Liharska
Manuscript ID: 97c63241070d9377
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: November 7, 2024
Disclosure Updated Date: November 7, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
KIDNEY360 Accessing Our World From Every Angle
CJASN Clinical Journal of the American Society of Nephrology

ASN Journal Disclosure Form

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A. Lund has nothing to disclose.

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Anina Lund
Manuscript ID: K360-2024-000472R1
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: October 4, 2024
Disclosure Updated Date: October 4, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
KIDNEY360 Accessing Our World From Every Angle
CJASN Clinical Journal of the American Society of Nephrology

ASN Journal Disclosure Form

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M. Merad reports the following:
Employer: mount sinai school of medecine; Consultancy: MM serves on the scientific advisory board and hold stock from Compugen Inc., Myeloid Therapeutics Inc., Morphic Therapeutic Inc., Asher Bio Inc., Dren Bio Inc., Oncoresponse Inc., Owkin Inc., OSE Inc., and Larkspur Inc.; MM serves on the scientific advisory board Innate Pharma Inc., Genenta Inc. ;; Ownership Interest: Ownership Interest less than 5%: Compugen Inc., Morphic Therapeutic Inc., Myeloid Therapeutics Inc., Asher Bio Inc., Dren Bio Inc., Owkin, and Larkspur; Ownership interest greater than or equal to 5%: None; and Research Funding: MM receives funding for contracted research from Regeneron Inc. and Boerhinger Ingelheim Inc.

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Miriam Merad
Manuscript ID: K360-2024-000472R3
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: January 16, 2025
Disclosure Updated Date: January 16, 2025

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
KIDNEY360 Accessing Our World From Every Angle
CJASN Clinical Journal of the American Society of Nephrology

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As per ASN journal policy, I have disclosed any financial relationships or commitments I have held in the past 36 months as included below. I have listed my Current Employer below to indicate there is a relationship requiring disclosure. If no relationship exists, my Current Employer is not listed.

G. Nadkarni reports the following:
Employer: Icahn School of Medicine at Mount Sinai; Consultancy: Renalytix, Siemens Healthineers, Qiming Capital, GLG consulting, Daiichi Sankyo, Reata, Variant Bio; Ownership Interest: Renalytix, Verici, Doximity, Pensieve Health, Nexus iConnect, Data2Wisdom LLC; Research Funding: Renalytix; Honoraria: Daiichi Sankyo; Patents or Royalties: Renalytix; Advisory or Leadership Role: Renalytix; and Speakers Bureau: Daiichi Sankyo.

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Girish N. Nadkarni
Manuscript ID: K360-2024-000472R1
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: October 3, 2024
Disclosure Updated Date: March 27, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
KIDNEY360 Accessing Our World From Every Angle
CJASN Clinical Journal of the American Society of Nephrology

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W. Oh reports the following:
Employer: UnitedHealthcare

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Name: Wonsuk Oh
Manuscript ID: K360-2024-000472R2
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: October 30, 2024
Disclosure Updated Date: May 22, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
KIDNEY360 Accessing Our World From Every Angle
CJASN Clinical Journal of the American Society of Nephrology

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I. Paranjpe reports the following:
Employer: Character Biosciences; Consultancy: Magnetic Ventures; and Ownership Interest: Neurona Health.

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Ishan Paranjpe
Manuscript ID: K360-2024-000472R3
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: January 28, 2025
Disclosure Updated Date: January 28, 2025

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JASN Journal of the American Society of Nephrology
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M. Rajagopal reports the following:
Employer: Icahn School of Medicine at Mount Sinai

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Name: Madhumitha Rajagopal
Manuscript ID: K360-2024-000472R3
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: November 7, 2024
Disclosure Updated Date: November 7, 2024

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CJASN Clinical Journal of the American Society of Nephrology

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A. Sakhuja reports the following:
Ownership Interest: multiple technology, utility etc companies. No stock in health related companies.; Honoraria: SCCM, ASPEN; Advisory or Leadership Role: Carolinas/Virginia's chapter of SCCM - unpaid; and Other Interests or Relationships: Current Funding from NIH/NIDDK K08DK131286; PI: Ankit Sakhuja.

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Ankit Sakhuja
Manuscript ID: K360-2024-000472R2
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: October 30, 2024
Disclosure Updated Date: October 7, 2024

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CJASN Clinical Journal of the American Society of Nephrology

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M. suarez-farinas reports the following:
Employer: Icahn School of Medicine at Mount Sinai; and Consultancy: Symbrio.

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Mayte suarez-farinas
Manuscript ID: K360-2024-000472R3
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: January 6, 2025
Disclosure Updated Date: November 21, 2024

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R. Thompson reports the following:
Employer: Icahn School of Medicine at Mount Sinai

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Name: Ryan C. Thompson
Manuscript ID: K360-2024-000472R3
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: November 11, 2024
Disclosure Updated Date: September 27, 2024

ASN American Society of Nephrology
JASN Journal of the American Society of Nephrology
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E. Tsalik reports the following:
Employer: Danaher; Ownership Interest: Danaher; Research Funding: Danaher; Patents or Royalties: Duke University; Danaher; and Advisory or Leadership Role: Danaher.

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Ephraim L Tsalik
Manuscript ID: K360-2024-000472R3
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: November 7, 2024
Disclosure Updated Date: November 7, 2024

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JASN Journal of the American Society of Nephrology
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A. Vaid reports the following:
Employer: Mount Sinai; and Consultancy: Verily Life Sciences.

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: Akhil Vaid
Manuscript ID: K360-2024-000472R3
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: November 13, 2024
Disclosure Updated Date: November 13, 2024

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G. Vasquez-Rios reports the following:
Consultancy: Natera; Novartis; Vera;; and Honoraria: Natera; Vera; Novartis.

I understand that the information above will be published within the journal article, if accepted, and that failure to comply and/or to accurately and completely report the potential financial conflicts of interest could lead to the following: 1) Prior to publication, article rejection, or 2) Post-publication, sanctions ranging from, but not limited to, issuing a correction, reporting the inaccurate information to the authors' institution, banning authors from submitting work to ASN journals for varying lengths of time, and/or retraction of the published work.

Name: George Vasquez-Rios
Manuscript ID: K360-2024-000472R3
Manuscript Title: Peripheral Transcriptomics in Acute and Long-Term Kidney Dysfunction in SARS-CoV2 Infection
Date of Completion: January 12, 2025
Disclosure Updated Date: January 12, 2025