

#### Cohort

Blood samples were collected from 10 patients in the ADAPT\* study that had long-COVID symptoms two years after their original infection with SARS-COV2.

An additional 10 blood samples were collected from patients that were also infected but had fully recovered.

In each of these groups, we have 5 females and 5 males, age matched. The severity of the original illness was classified based on hospital admission.

> \*Darley et al. (2021) Persistent symptoms up to four months after community and hospital-managed SARS-CoV-2 infection The Medical Journal of Australia.

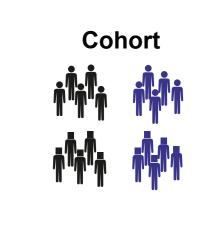
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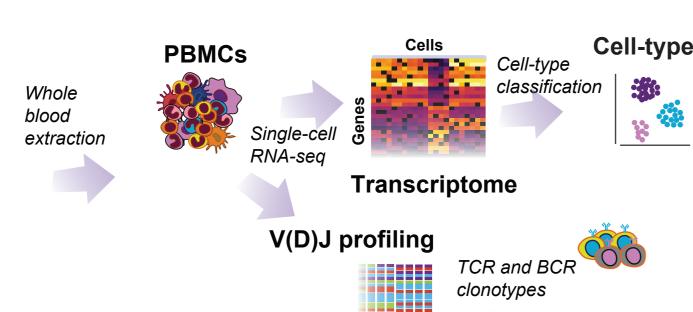
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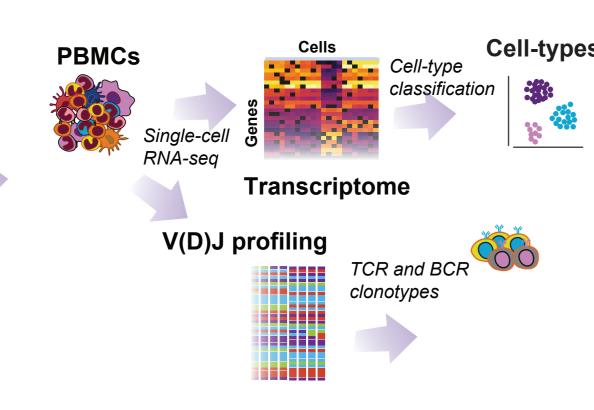
Samples are currently being sequenced on the 10X genomics platform. We are using the 5' gene expression and V(D)J profiling of B-cell and T-cell receptors.

This will provide transcriptional profiles for each cell, and the antigen receptor profiles for B and T lymphocytes. T cells and B cells express receptors that recognize antigens and receptor repertoire analysis is useful for evaluating the diversity of an immune system.





# Immune profiling of peripheral blood mononuclear cells (PBMCs)

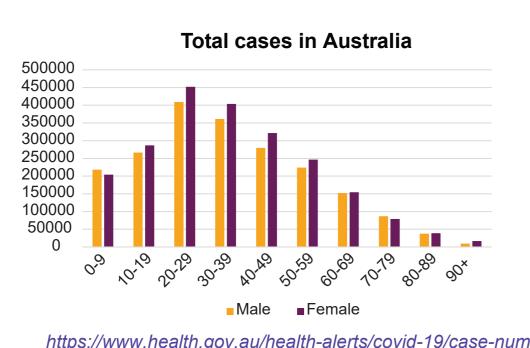


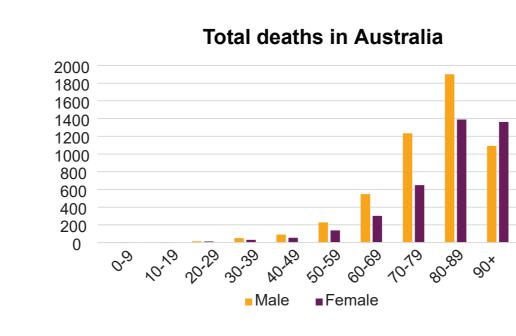
#### **Garvan Institute** of Medical Research



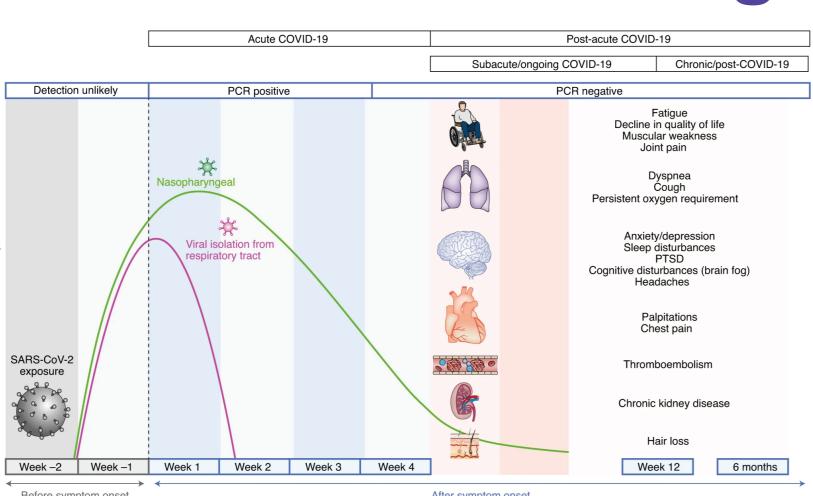
GARVAN-WEIZMANN

Death rates are different across sex and age





More males die from COVID-19 complications while females suffer more from long-COVID

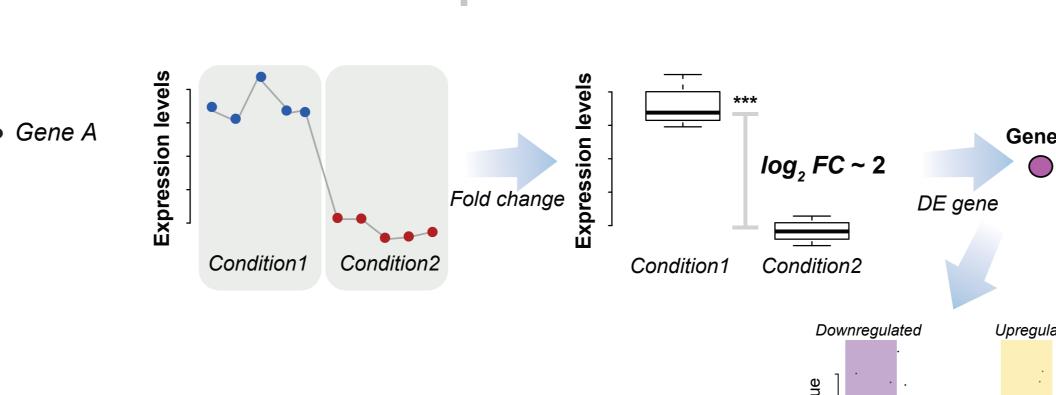


Long-COVID refers to persistent symptoms beyond 3 months of infection

Nalbandian et al. (2021). Post-acute COVID-19 syndrome. Nature Medicine.

#### Expression analysis

Differential expression



Pathway enrichment analysis

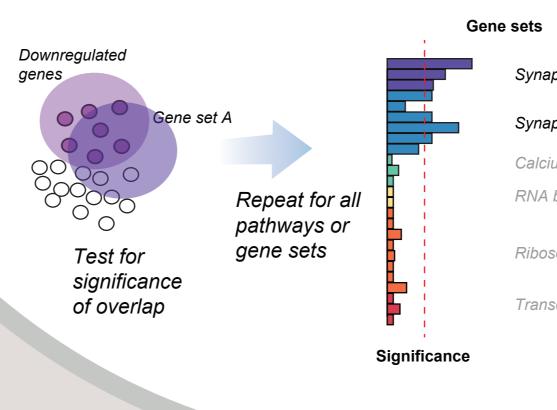
Exploring sex differences of

long-COVID-19 at

single-cell resolution

Single-cell UMAP of 1.2M PBMCs

from the OneK1K\* cohort

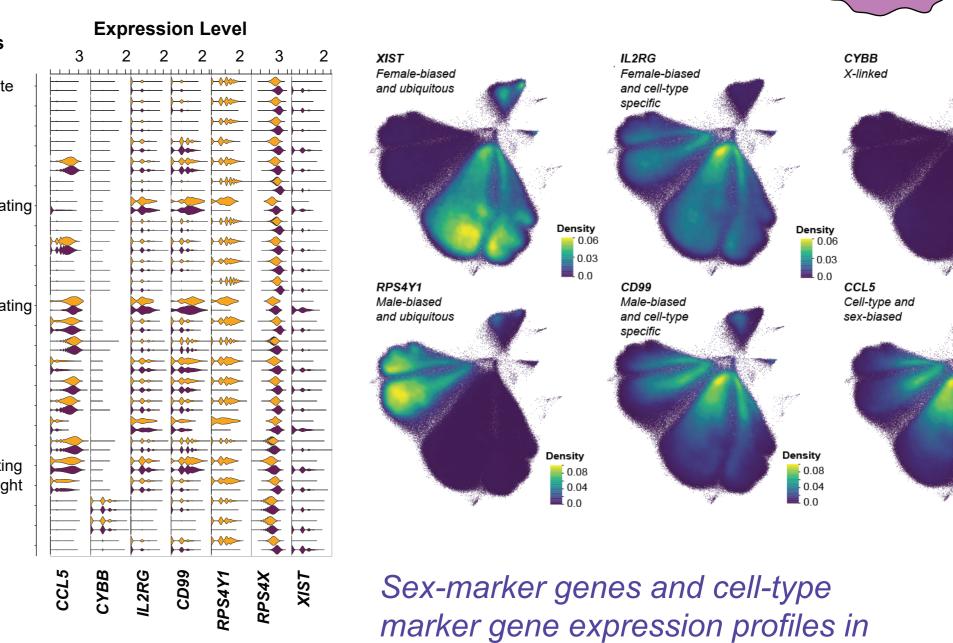


Ribosome

#### website



Transcriptome is an endophenotype linking genotype to phenotypes Single-cell RNA-seq for cellular resolution of gene expression



the OneK1K cohort.



### Questions to explore:

What genes and pathways are differentially expressed in long-COVID?

Inflammatory cytokines Lymphocte activation and dysregulation Chronic myeloid cell activation

Are these the same when we look at sex-specific differential expression?

Are there cell-type differences

between cases and those who

**Autoimmunity** 

viral remnants

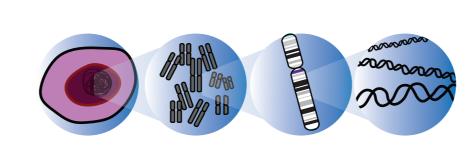
Long-COVID hypotheses Dysbiosis

Tissue damage

Viral reservoirs or

recover?

What other cellular or gene profiles are associated with long-COVID symptoms?



## Interested? Reach out!



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