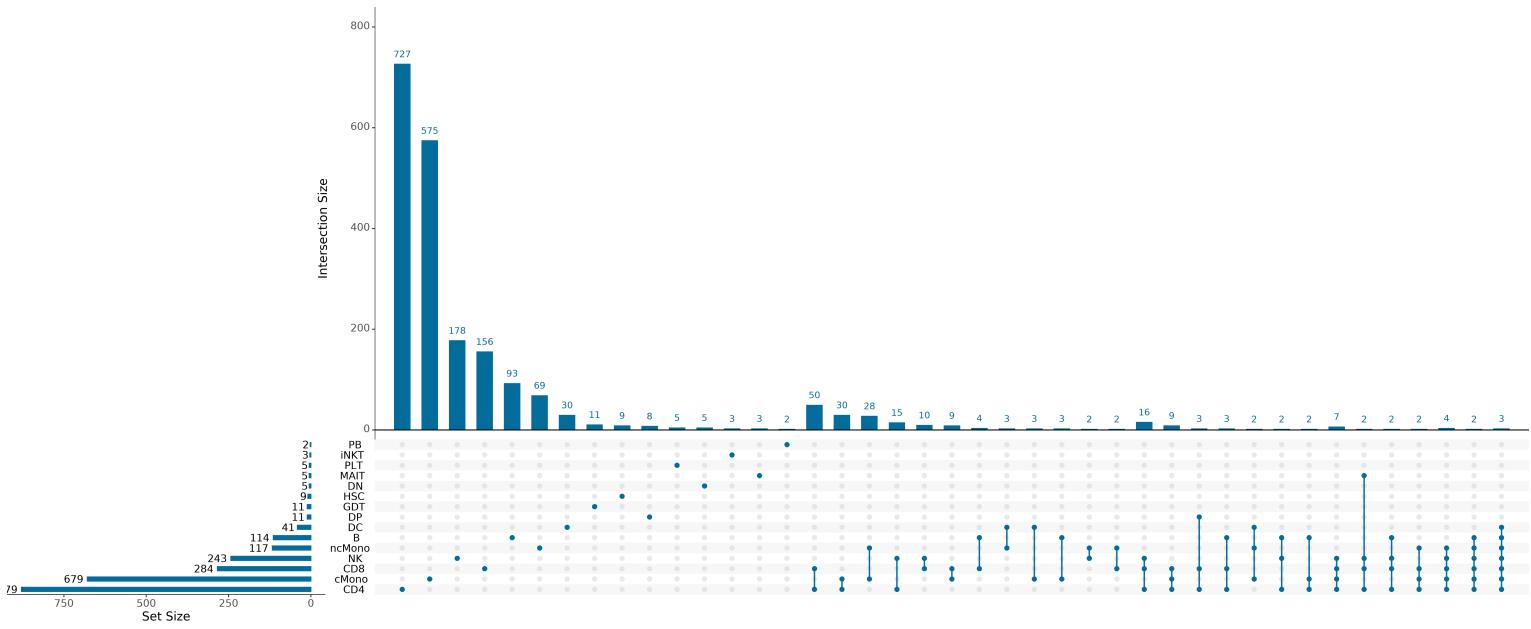


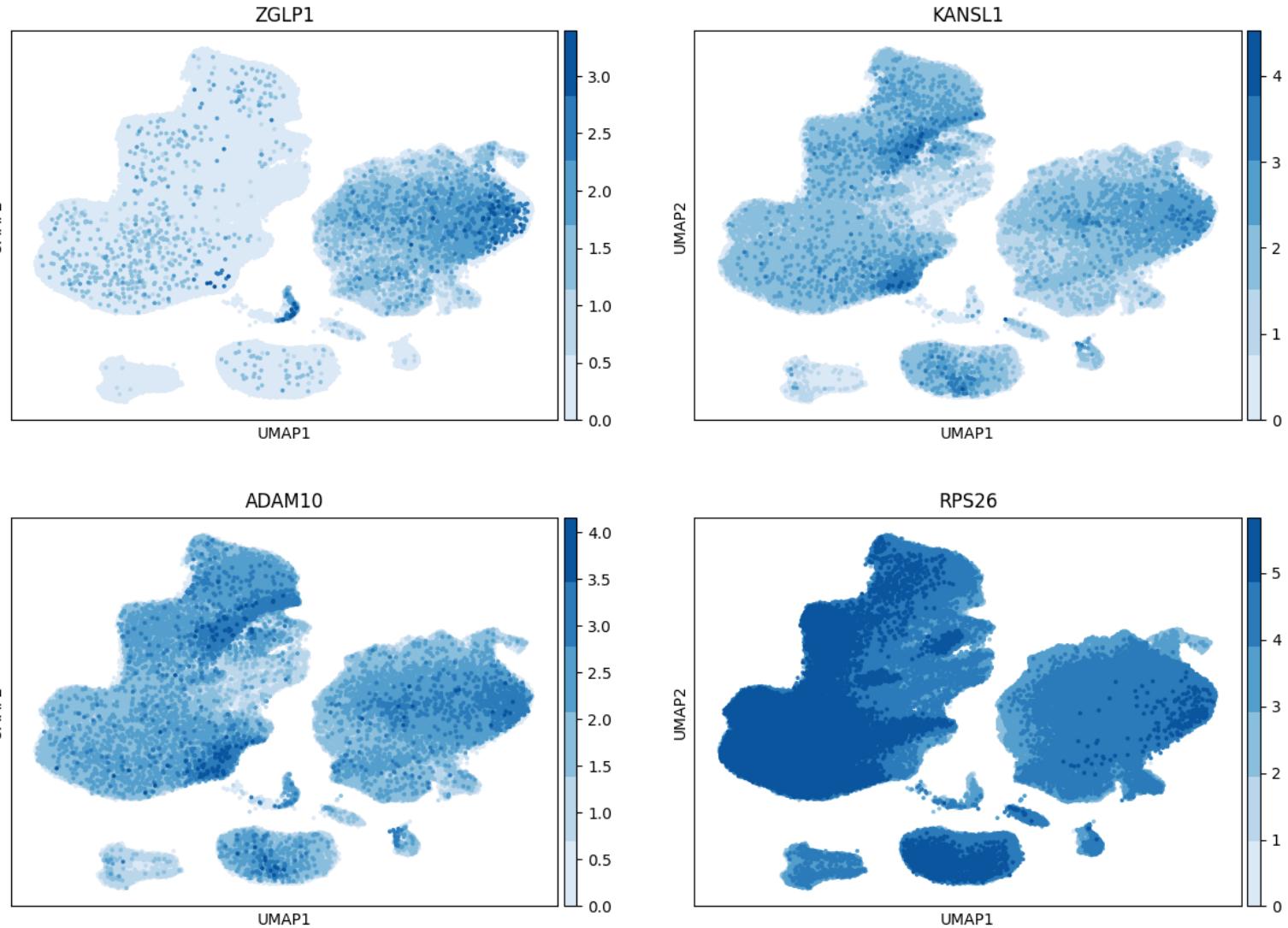
**Supplementary Figure 1:**

Heatmap showing overlapping eGenes for each cell type. Colour: Jaccard index percentage (100 \* size of intersection over size of union), label: raw number of eGenes.



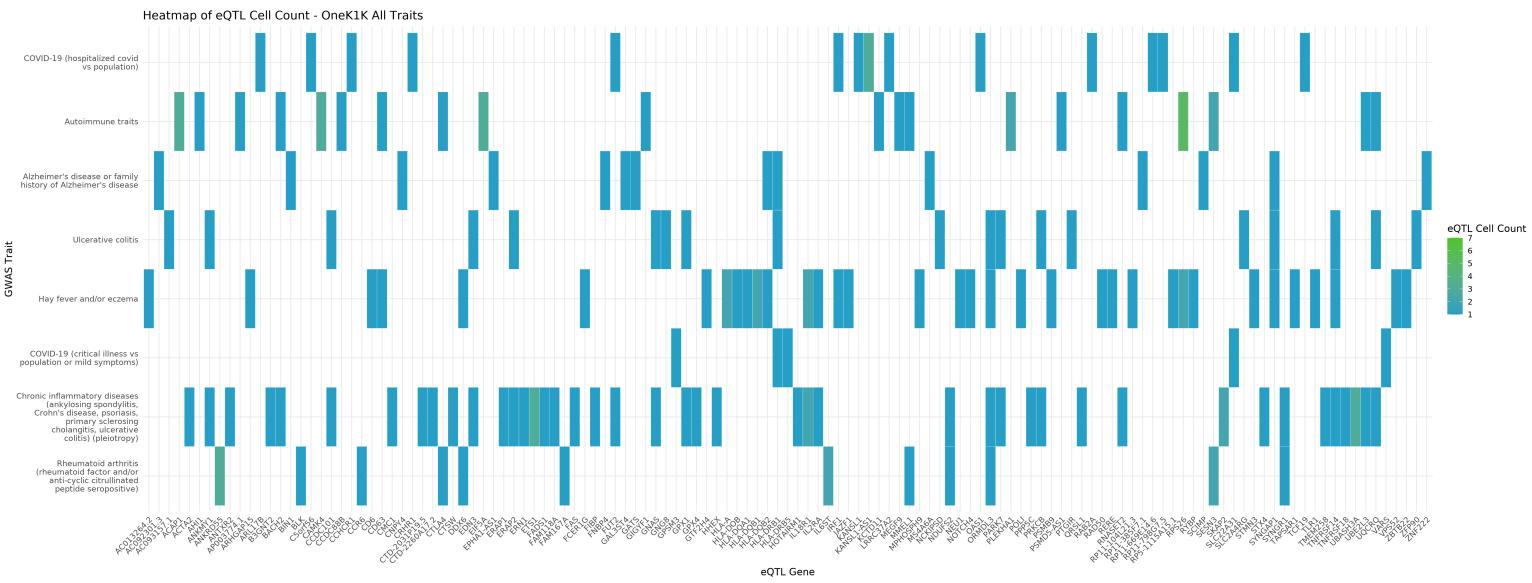
## Supplementary Figure 2:

Upset plot showing all overlapping eGene results between cell types. Horizontal bars show how many Gene...SNP pairs are in each individual cell type. Vertical bars show how many Gene...SNP pairs are shared across the cell types indicated by the connected dots below each bar. Vertical bars of only one Gene–SNP pair have been removed for visual clarity.



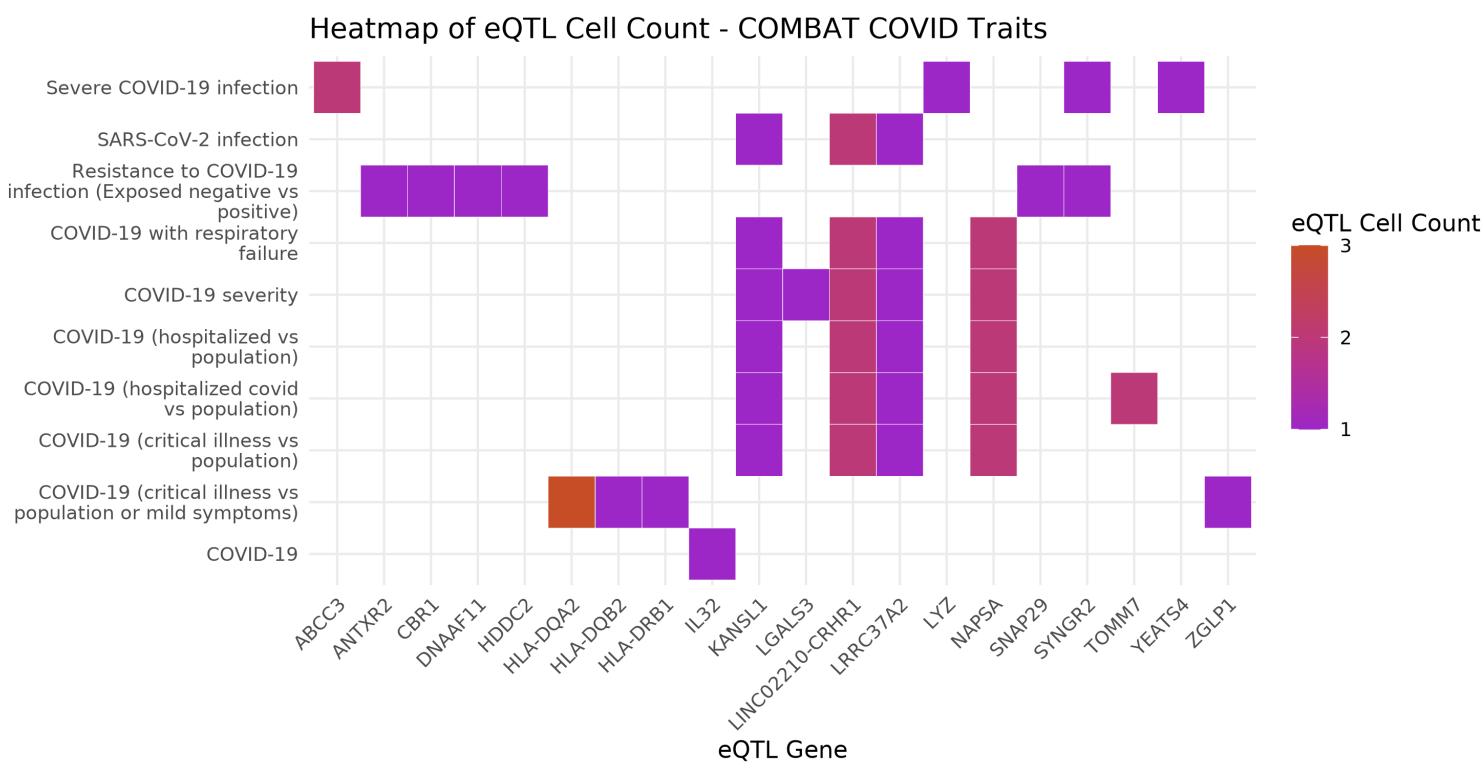
### Supplementary Figure 3:

UMAP plot with differential expression of ZGLP1, KANSL1, ADAM10, RPS26. The left cluster contains CD4 and CD8 cells, the right classical and non-classical monocytes, and the bottom middle B cells. UMAP of all cell types shown in Figure 2.



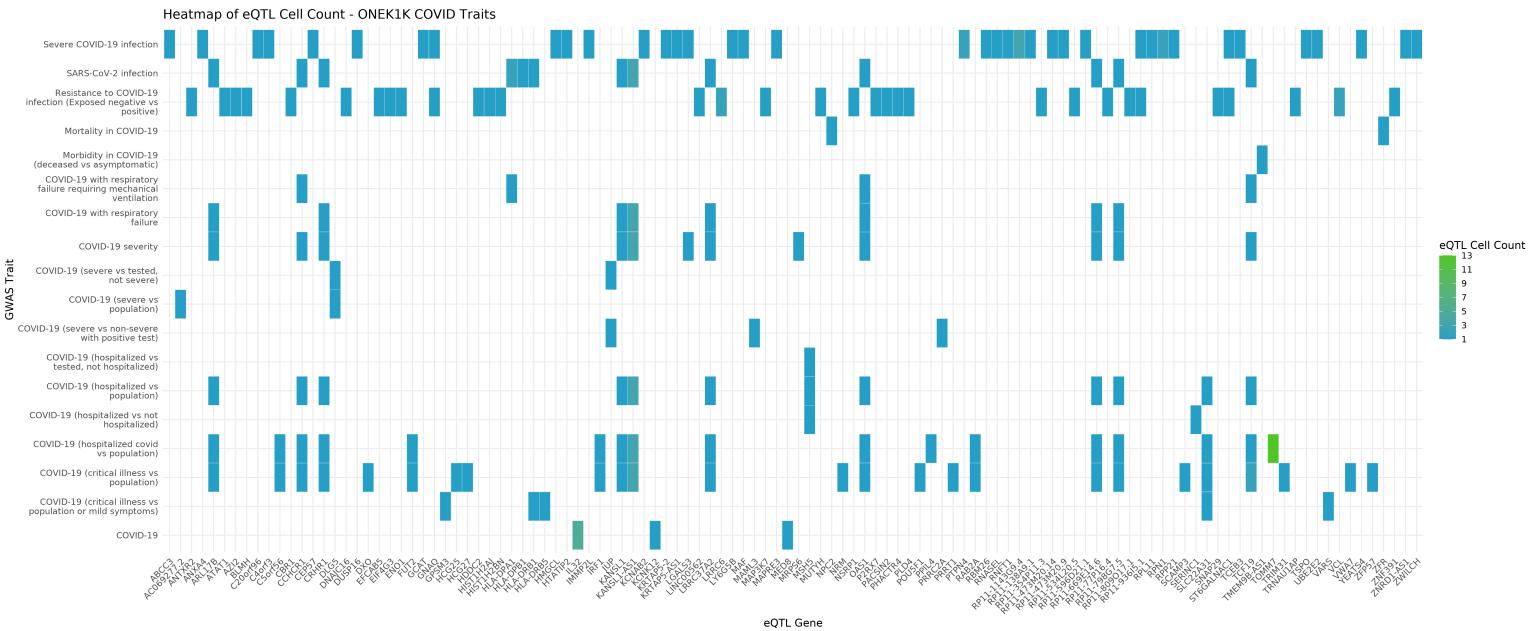
#### Supplementary Figure 4:

OneK1K eQTL SNPs colocalising with GWAS risk variants for infectious and inflammatory traits. The x-axis shows the eGene associated with the eQTL variant and the y-axis shows the trait associated with the variant (or a high-LD variant) in GWAS. The colour of the tile shows the number of cell types in which the eQTL was found.



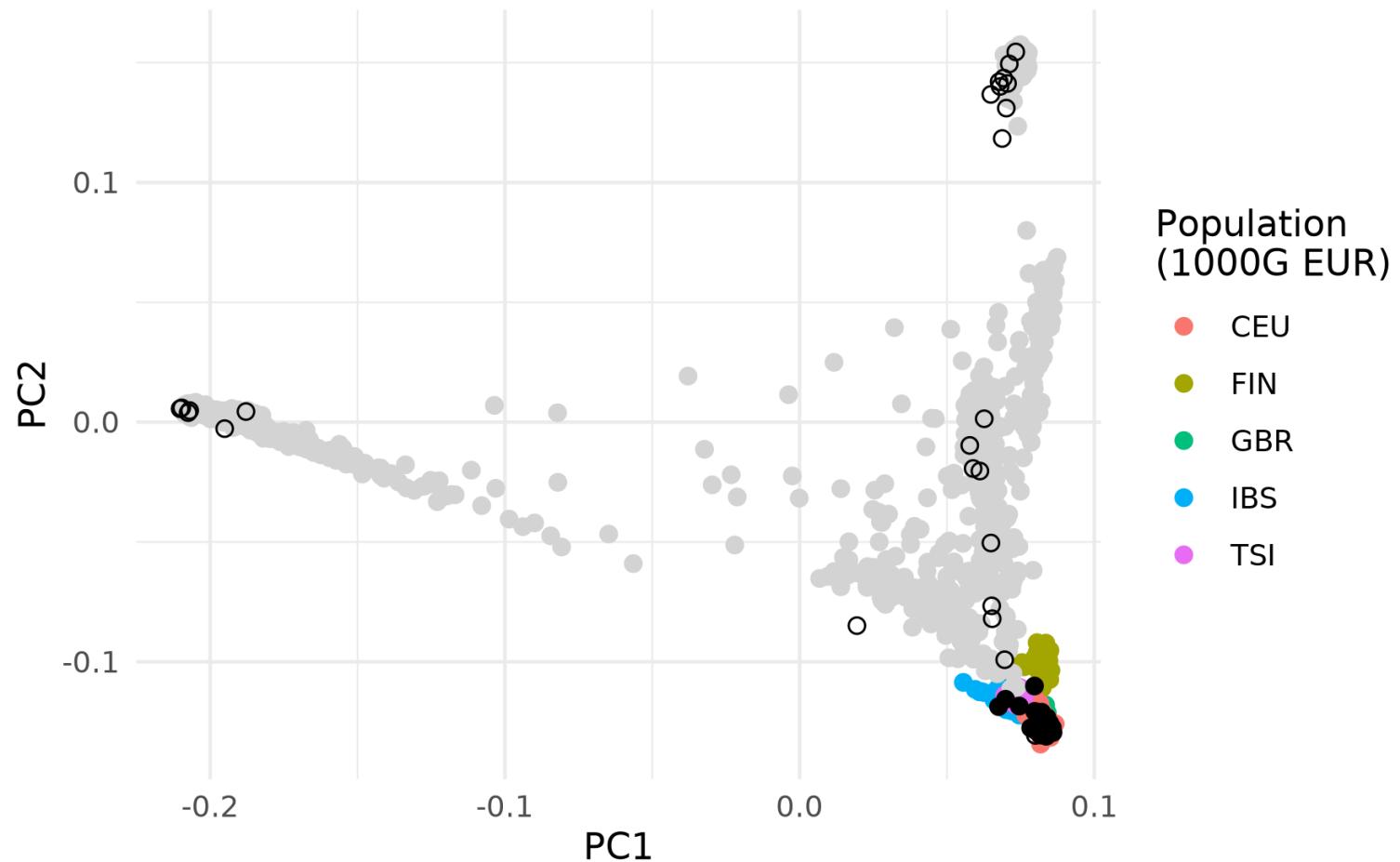
### Supplementary Figure 5:

COMBAT eQTL SNPs colocalising with GWAS risk variants for COVID-19 traits (severity, hospitalisation and resistance of disease as well as SARS-CoV-2 infection). The x-axis shows the eGene associated with the eQTL variant and the y-axis shows the trait associated with the variant (or a high-LD variant) in GWAS. The colour of the tile shows the number of cell types in which the eQTL was found.



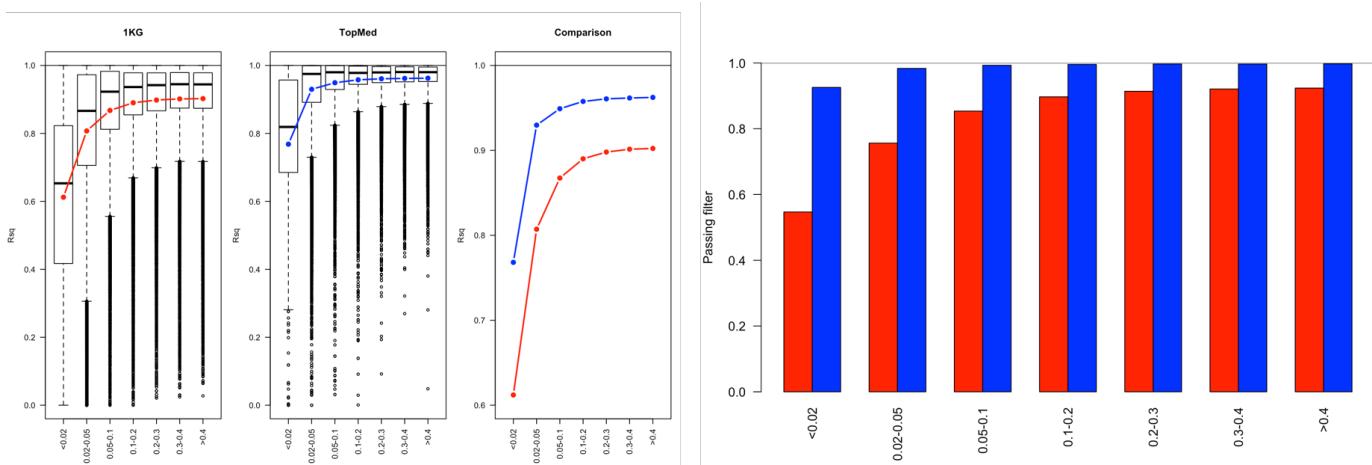
## Supplementary Figure 6:

OneK1K eQTL SNPs colocalising with GWAS risk variants for COVID-19 traits (severity, hospitalisation and resistance of disease as well as SARS-CoV-2 infection). The x-axis shows the eGene associated with the eQTL variant and the y-axis shows the trait associated with the variant (or a high-LD variant) in GWAS. The colour of the tile shows the number of cell types in which the eQTL was found.



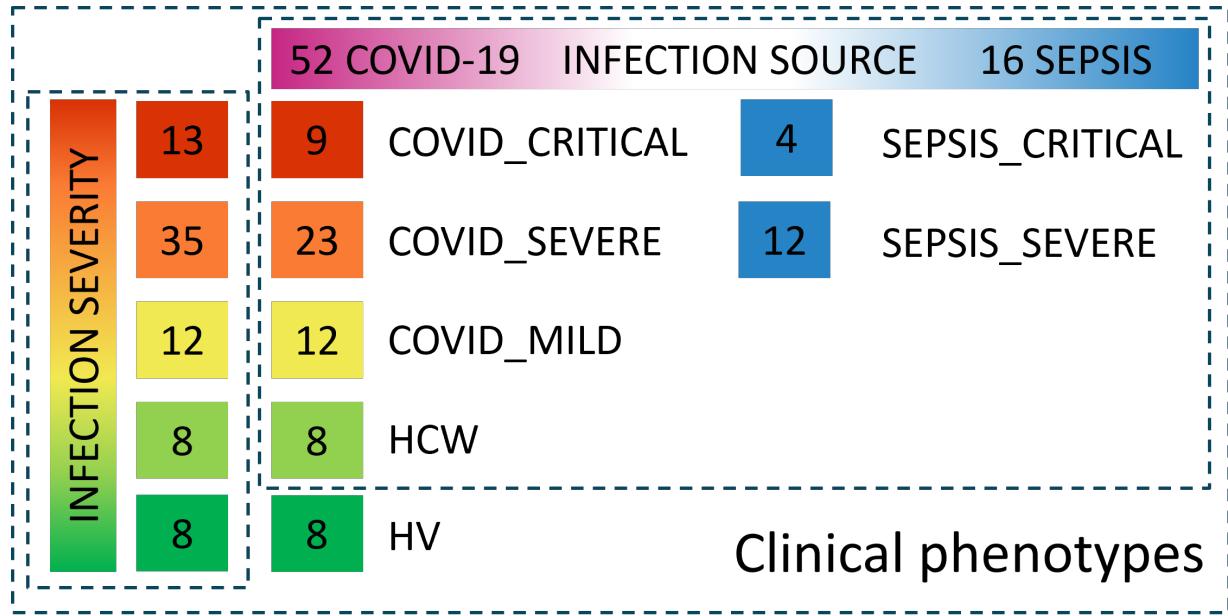
### Supplementary Figure 7:

Principal component analysis of the COMBAT individuals combined with 1000 Genomes (Auton et al., 2015). Individuals from 1000 Genomes European (EUR) populations were highlighted in colour, European-assigned COMBAT individuals showed as filled black circles and non-European samples as non-filled black circles.



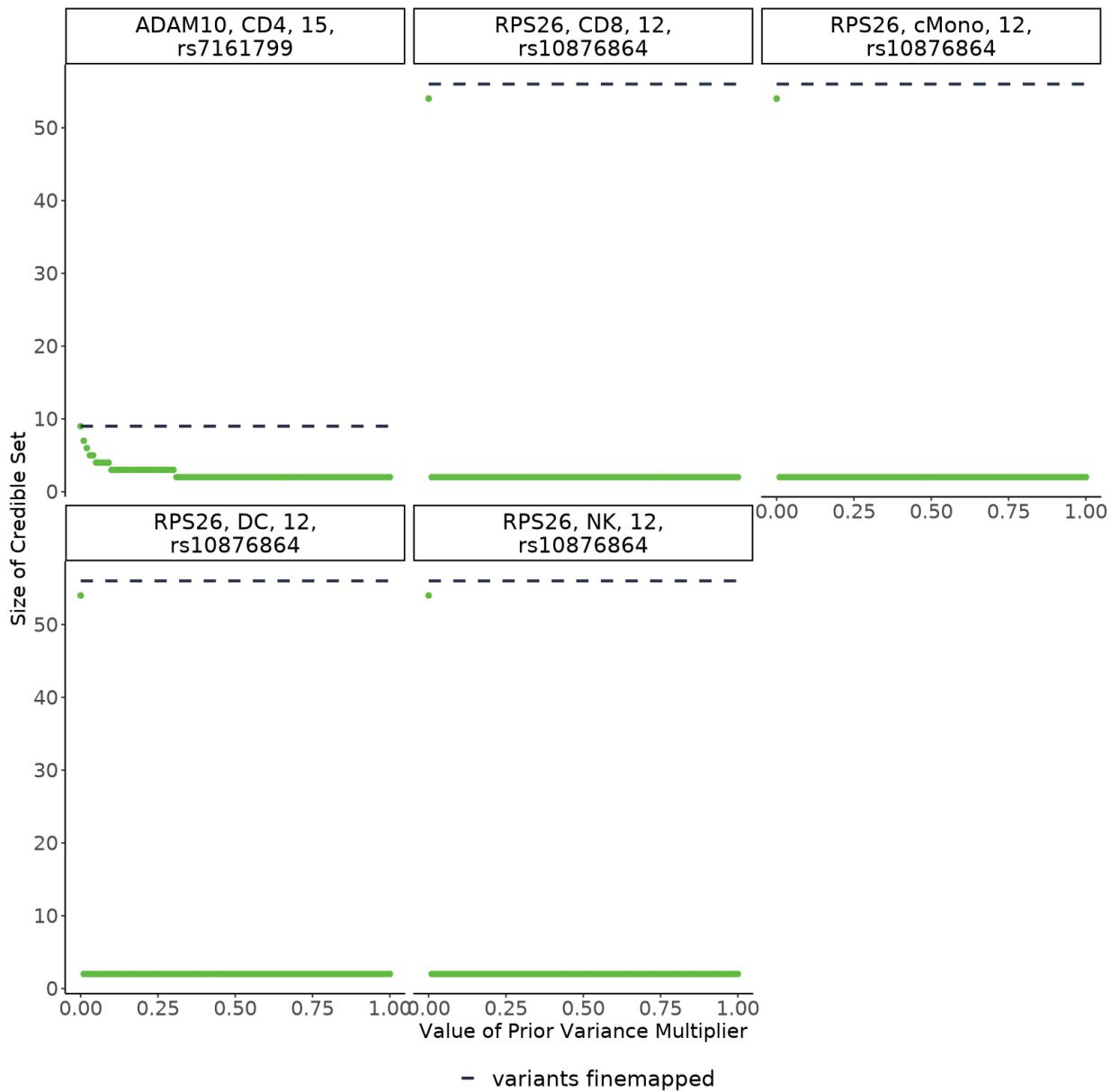
### Supplementary Figure 8:

Imputation quality and proportion of variants passing filters across minor allele frequency bins using 1000 Genomes (1KG) and TOPMed imputation reference sets.



### Supplementary Figure 9:

Categorisation of phenotype groups for disease term in the interaction model. Infection severity comparison includes all individuals, infection source (COVID–19 vs. sepsis) comparison does not include healthy individuals. Both are linear models.



### Supplementary Figure 10:

Sensitivity analysis of the effect of prior variance multiplier on the size of the resultant credible set. The x-axis shows the different values of prior variance multiplier used in the sensitivity analysis. The y-axis shows the size of the credible set after finemapping. In most cases, only values very close 0 affect the result. A value of 0.2 was used in accordance with susieR.