

A second update on mapping the human genetic architecture of COVID-19

<https://doi.org/10.1038/s41586-023-06355-3>

The COVID-19 Host Genetics Initiative*

Received: 23 December 2022

Accepted: 21 June 2023

Published online: 6 September 2023

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ARISING FROM: COVID-19 Host Genetics Initiative. *Nature* <https://doi.org/10.1038/s41586-021-03767-x> (2021)

Investigating the role of host genetic factors in COVID-19 severity and susceptibility can inform our understanding of the underlying biological mechanisms that influence adverse outcomes and drug development^{1,2}. Here we present a second updated genome-wide association study (GWAS) on COVID-19 severity and infection susceptibility to SARS-CoV-2 from the COVID-19 Host Genetic Initiative (data release 7). We performed a meta-analysis of up to 219,692 cases and over 3 million controls, identifying 51 distinct genome-wide significant loci—adding 28 loci from the previous data release². The increased number of candidate genes at the identified loci helped to map three major biological pathways that are involved in susceptibility and severity: viral entry, airway defence in mucus and type I interferon.

We conducted a meta-analysis for 3 phenotypes across 82 studies from 35 countries, including 36 studies of individuals with non-European ancestry (Fig. 1, Supplementary Figs. 1 and 2 and Supplementary Table 1): critical illness (respiratory support or death; 21,194 cases), hospitalization (49,033 cases) and SARS-CoV-2 infection (219,692 cases). Most of the studies were collected before the widespread introduction of COVID-19 vaccination. We found 30, 40 and 21 loci that are associated with critical illness, hospitalization and infection due to SARS-CoV-2, respectively, for a total of 51 distinct genome-wide significant loci across all three phenotypes ($P < 5 \times 10^{-8}$; Fig. 2, Supplementary Fig. 3 and Supplementary Table 2), adding 28 genome-wide significant loci to the 23 previously identified by the COVID-19 Host Genomics Initiative (HGI; data release 6)^{1,2}. We observed a median increase of 2.9-fold in statistical power across lead variants owing to a median increase of 1.6-fold in effective sample sizes from the previous release (Supplementary Table 3). After correcting for the number of phenotypes examined, 46 loci remained significant ($P < 1.67 \times 10^{-8}$). Of the 28 additional loci, 6 loci were originally reported by the GenOMICC study³, which also contributed to the current meta-analysis, and 9 other loci were identified by the new GenOMICC meta-analysis⁴ during the preparation of this paper. We found nine more loci that reached genome-wide significance, but we excluded them as they were probably false positives, as determined using a leave-most-significant-biobank-out analysis (Supplementary Table 4 and Supplementary Note). Comparing the effect sizes and statistical significance between the previous² and current analysis indicated that all of the previously identified loci were replicated and showed an increase in statistical significance (Supplementary Fig. 4). Using our previously developed two-class Bayesian model for classifying loci as being more likely involved in infection susceptibility or severity², we determined that 36 loci are substantially more likely (higher than 99% posterior probability) to affect disease severity (hospitalization) and 9 loci are substantially more likely to

influence susceptibility to SARS-CoV-2 infection, while the remaining 6 loci could not be classified (Supplementary Fig. 5, Supplementary Table 5 and Supplementary Note). We observed that the 1q22 locus (lead variant: rs12752585:G>A) showed significant effect-size heterogeneity across ancestries ($P_{\text{het}} < 9.80 \times 10^{-4} = 0.05/51$), whereas the previously reported heterogenous locus (*FOXP4*) remained at the same level of significance as before², despite an increase in sample size ($P_{\text{het}} = 2.01 \times 10^{-3}$; Supplementary Fig. 6 and Supplementary Table 6). We found significant observed-scale single-nucleotide polymorphism heritabilities of all the three phenotypes (1.2–8.2%, $P < 0.0001$). We also estimated liability-scale heritabilities for a range of population prevalences (Supplementary Fig. 7, Supplementary Table 7 and Supplementary Note).

To better understand the biological mechanisms underlying COVID-19 susceptibility and severity, we further characterized candidate causal genes by mapping them onto biological pathways and performing a phenome-wide association analysis (Extended Data Fig. 1, Supplementary Fig. 8 and Supplementary Tables 2, 8 and 9). In total, 15 out of 51 loci could be linked to three major pathways involved in susceptibility and severity defined by expert-driven classification (Supplementary Note): (1) viral entry; (2) entry defence in airway mucus; and (3) type I interferon response. Moreover, the phenome-wide association analysis identified nine loci involved in the upkeep of healthy lung tissue.

First, five loci include candidate causal genes involved in the viral entry pathway (Extended Data Fig. 1a), such as previously reported *SLC6A20* (3p21.31), *ABO* (9q34.2), *SFTPD* (10q22.3) and *ACE2* (Xp22.2), as well as *TMPRSS2* (21q22.3), which was also identified in the data release 7. We found that the lead variant rs9305744:G>A, an intronic variant of *TMPRSS2*, is protective against critical illness (odds ratio (OR) = 0.92, 95% CI = 0.89–0.95, $P = 1.4 \times 10^{-8}$) and is in linkage disequilibrium with the missense variant rs12329760:C>T (p.Val197Met; $r^2 = 0.68$). SARS-CoV-2 uses the serine protease *TMPRSS2* for viral spike protein priming, as well as the previously reported *ACE2* for host cell entry which functionally interacts with *SLC6A20* (refs. 5,6). Notably, the previously reported association between ABO blood groups and susceptibility could be attributed to the interference of anti-A and anti-B antibodies with the spike protein, potentially interfering with viral entry⁷. Furthermore, the previously reported *SFTPD* encodes pulmonary surfactant protein D (SP-D), which contributes dually to the lung's innate immune molecules and viral-entry response in pulmonary epithelia^{8,9} along with other genes for airway defence.

Second, four loci contain candidate causal genes for entry defence in the airway mucus (Extended Data Fig. 1b), such as previously reported *MUC1/THBS3* (1q22) and *MUC5B* (11p15.5) as well as novel *MUC4* (3q29) and *MUC16* (19p13.2). We found that the novel lead variants

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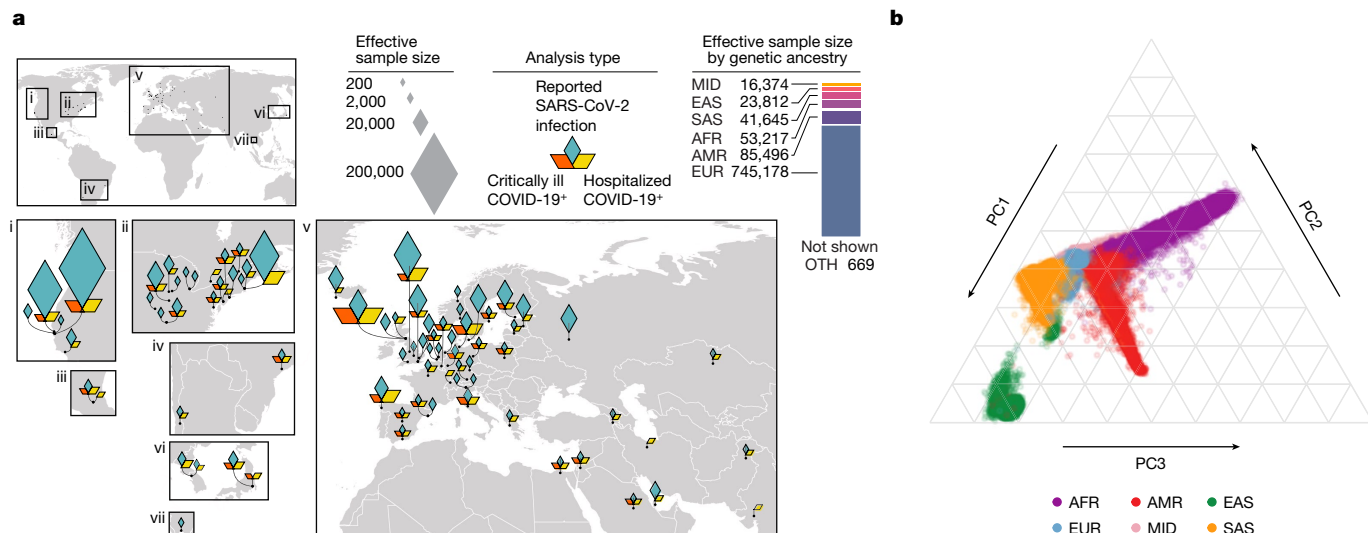


Fig. 1 | Overview of the contributing studies in the HGI data release 7.

a, Geographical overview of the studies contributing to the COVID-19 HGI and the composition by major ancestry groups. Populations are defined as Middle Eastern (MID), South Asian (SAS), East Asian (EAS), African (AFR), admixed American (AMR), European (EUR) and other (OTH). **b**, A principal component

analysis (PCA) highlights the population structure and the sample ancestry of the individuals participating in the COVID-19 HGI. Per-cohort PCA results are provided in Supplementary Fig. 2. This figure was reproduced from the original publication by the COVID-19 HGI¹ with modifications reflecting the updated analysis from data release 7.

rs2260685:T>C in *MUC4* (intronic variant; in linkage disequilibrium ($r^2 = 0.65$) with a missense variant rs2259292:C>T (p.Gly4324Asp)) and rs73005873:G>A in *MUC16* (intronic variant) increase the risk of SARS-CoV-2 infection (OR = 1.03 and 1.03, 95% CI = 1.02–1.04 and 1.02–1.04, $P = 4.1 \times 10^{-8}$ and 6.4×10^{-10} , respectively). Moreover, the previously reported locus 1q22 contains an intergenic lead variant rs12752585:G>A that decreases the risk of infection (OR = 0.98, 95% CI = 0.97–0.98, $P = 1.5 \times 10^{-11}$) and increases *MUC1* expression in the oesophagus mucosa in GTEx v8 ($P = 5.2 \times 10^{-9}$). Notably, the 1q22 locus also contains an independent lead variant, rs35154152:T>C, a missense variant (p.Ser279Gly) of *THBS3*, that decreases the risk of hospitalization (OR = 0.88, 95% CI = 0.86–0.90, $P = 5.6 \times 10^{-22}$) but not infection ($P = 5.7 \times 10^{-4}$), suggesting potential distinct mechanisms in the locus. Consistent with these association patterns, *MUC1*, *MUC4* and *MUC16* are three known major transmembrane mucins of the respiratory tracts that prevent microbial invasion, whereas previously reported *MUC5B*, together with nearby *MUC5AC*, are primary structural components of airways mucus that enable mucociliary clearance of pathogens¹⁰.

Third, six loci contain candidate causal genes that are linked to the type I interferon pathway (Extended Data Fig. 1c), such as previously reported *IFNAR2* (21q22.11), *OAS1* (12q24.13) and *TYK2* (19p13.2), as well as additionally identified *JAK1* (1p31.3), *IRF1* (5q31.1) and *IFNA*-coding genes (9p21.3). Previous studies have reported additional genes in this pathway: *TLR7* (refs. 11,12) and *DOCK2* (ref. 13). Here we found that the lead variant rs28368148:C>G, a missense variant (p.Trp164Cys) of *IFNA10* located within the *IFNA* gene cluster, increases the risk of critical illness (OR = 1.56, 95% CI = 1.38–1.77, $P = 3.7 \times 10^{-12}$). *IFNA* is one of the type I interferons that binds specifically to the *IFNA* receptor consisting of *IFNAR1*–*IFNAR2* chains, in which mutations are also known to increase the risk of hospitalization and critical illness. In the genes that enable signalling downstream of *IFNAR*, we identified that the lead variant rs11208552:G>T, an intronic variant of *JAK1*, is protective against critical illness and hospitalization (OR = 0.92 and 0.95, 95% CI = 0.89–0.94 and 0.93–0.96, $P = 5.5 \times 10^{-10}$ and 2.2×10^{-9} , respectively). This variant was previously reported to decrease lymphocyte counts¹⁴ ($\beta = -0.016$, $P = 5.5 \times 10^{-15}$) and increase the *JAK1* expression in the thyroid in GTEx¹⁵ ($P = 6.1 \times 10^{-23}$). *JAK1* and previously reported *TYK2* are Janus kinases (JAKs) that are required for type I interferon-induced JAK–STAT signalling. JAK inhibitors are used to treat patients with severe

COVID-19 (ref. 16). Furthermore, downstream of JAK–STAT signalling, we found that the lead variant rs10066378:T>C, located 67 kb upstream of *IRF1*, increases the risk of critical illness and hospitalization (OR = 1.09 and 1.07, 95% CI = 1.06–1.13 and 1.05–1.09, $P = 2.7 \times 10^{-9}$ and 3.74×10^{-10} , respectively).

Furthermore, the phenome-wide association analysis identified nine loci previously associated with lung function and respiratory diseases. These loci contain genes involved in the upkeep of healthy lung tissue such as previously reported *FOXP4* (6p21.1), *SFTPD* (10q22.3), *MUC5B* (11p15.5) and *DPP9* (19p13.3), as well as additionally identified *CIB4* (2p23.3), *NPNT* (4q24), *ZKSCAN1* (7q22.1), *ATP11A* (13q34) and *PSMD3* (17q21.1). For example, we found that three lead variants, rs1662979:G>T (intronic variant of *CIB4*), rs34712979:G>A (splice region variant of *NPNT*) and rs2897075:C>T (intronic variant of *ZKSCAN1*), are significantly associated with hospitalization (OR = 1.05, 0.94 and 1.05, 95% CI = 1.03–1.07, 0.92–0.96 and 1.03–1.07, $P = 5.6 \times 10^{-9}$, 3.8×10^{-8} and 8.9×10^{-9} , respectively) and lung function (FEV1/FVC)¹⁷, similar to the previously reported lead variant rs3934643:G>A (intronic variant of *SFTPD*). Notably, whereas the alleles associated with increased risk of COVID-19 severity of rs1662979 and rs3934643 decrease lung function ($\beta = -0.013$ and -0.025 , $P = 5.3 \times 10^{-8}$ and 6.3×10^{-10}), those of rs34712979 and rs2897075 increase lung function ($\beta = 0.068$ and 0.023 , $P = 4.2 \times 10^{-134}$ and 1.6×10^{-20} , respectively). Likewise, we found lead variants that were significantly associated with hospitalization and idiopathic pulmonary fibrosis^{18,19}, such as the aforementioned rs2897075 and rs12585036:C>T (intronic variant of *ATP11A*; OR = 1.10, 95% CI = 1.08–1.12, $P = 3.2 \times 10^{-21}$), in addition to the previously reported rs35705950:G>T (promoter variant of *MUC5B*). Whereas the COVID-19 severity risk-increasing alleles of rs2897075 and rs12585036 increase the risk of idiopathic pulmonary fibrosis (OR = 1.12 and 1.27, $P = 3.0 \times 10^{-14}$ and 7.0×10^{-9} , respectively), those of rs35705950 decreases the risk (OR = 0.50, $P = 3.9 \times 10^{-80}$). These results highlight the complex pleiotropic relationships between COVID-19 severity, lung function and respiratory diseases.

We used genetic correlations and Mendelian randomization analyses to identify potential causal effects of risk factors on COVID-19 phenotypes (Supplementary Fig. 9 and Supplementary Tables 10 and 11). In total, 14 novel genetic correlations and 10 novel robust exposure-COVID-19 trait pairs showed evidence of causal associations

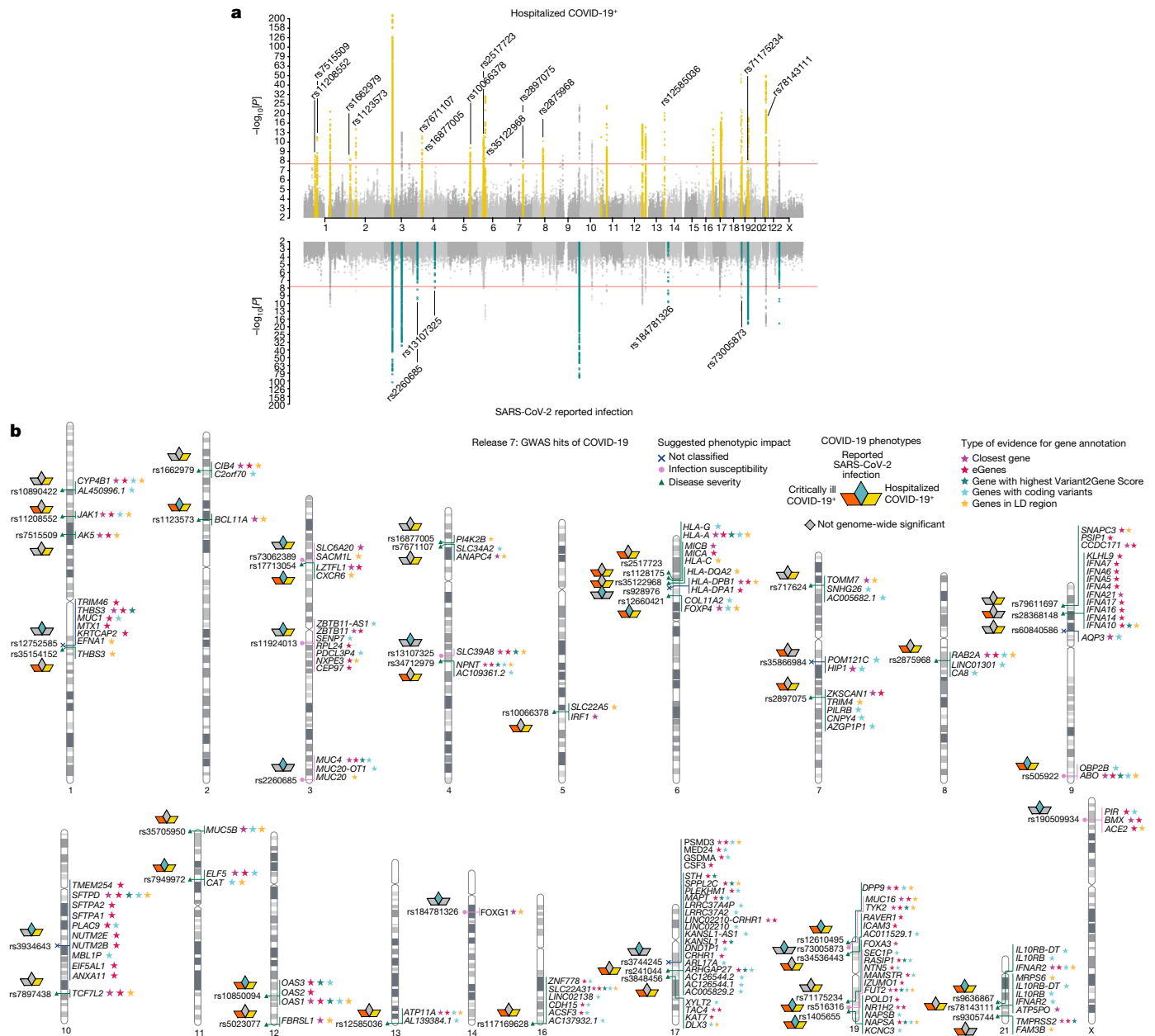


Fig. 2 | GWAS results for COVID-19. a, The results of a GWAS analysis of hospitalized individuals with COVID-19 ($n = 49,033$ cases and $n = 3,393,109$ controls) (top), and the results for individuals with reported SARS-CoV-2 infection ($n = 219,692$ cases and $n = 3,001,905$ controls) (bottom). The loci highlighted in yellow (top) represent regions that are associated with severity of COVID-19. The loci highlighted in green (bottom) are regions associated with susceptibility to SARS-CoV-2 infection. Lead variants for the loci that were identified in this data release are annotated with their respective rsID. The y axis is on the $-\log_{10}(P)$ scale up to 10, after which it switches to a $10 \times \log_{10}[-\log_{10}(P)]$ scale to aid presentation. **b**, Results of gene prioritization

using different evidence measures of gene annotation. For the genes in a linkage-disequilibrium (LD) region, genes with coding variants and eGenes (fine-mapped *cis*-expression quantitative trait locus (*cis*-eQTL) variant with posterior inclusion probability (PIP) > 0.1 in GTEx Lung) are annotated as such if they are in linkage disequilibrium with a COVID-19 lead variant ($r^2 > 0.6$). V2G, the highest gene prioritized by OpenTargetGenetics V2G score. The pink circle indicates SARS-CoV-2 infection susceptibility, the green triangle indicates COVID-19 severity and the blue cross indicates unclassified. This figure was reproduced from the original publication by the COVID-19 HGI¹ with modifications reflecting the updated analysis from data release 7.

(Supplementary Note). In particular, smoking initiation and the number of cigarettes per day were positively correlated with severity and susceptibility phenotypes; Mendelian randomization indicated that smoking was causally associated with increased risk of COVID-19, further highlighting the role of the healthy lung tissue in COVID-19 severity. Moreover, genetically instrumented higher glomerular filtration rate (indicative of better kidney function) was associated with a lower risk of COVID-19 critical illness, whereas genetically predicted chronic kidney disease was associated with an increased risk of

COVID-19 critical illness, suggesting that better kidney function would be beneficial for a lower risk of COVID-19 severity.

In summary, we have substantially expanded the current knowledge of host genetics for COVID-19 susceptibility and severity by further doubling the case numbers from the previous data release² and identifying 28 additional loci. The increased number of loci enables us to map genes to pathways that are involved in viral entry, airway defence and immune system response. Notably, we observed severity loci mapped to type I interferon pathway, while susceptibility loci

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mapped to viral entry and airway defence pathways, with notable exceptions for severity-classified *TMPRSS2* and *MUC5B* loci. Further investigation of how such susceptibility and severity loci map to different pathways would provide mechanistic insights into the human genetic architecture of COVID-19.

Online content

Any methods, additional references, Nature Portfolio reporting summaries, source data, extended data, supplementary information, acknowledgements, peer review information; details of author contributions and competing interests; and statements of data and code availability are available at <https://doi.org/10.1038/s41586-023-06355-3>.

Reporting summary

Further information on research design is available in the Nature Portfolio Reporting Summary linked to this article.

Data availability

Summary statistics generated by the COVID-19 HGI are available online, including per-ancestry summary statistics for African, admixed American, East Asian, European and South Asian ancestries (<https://www.covid19hg.org/results/r7/>). The analyses described here used the data release 7. If available, individual-level data can be requested directly from contributing studies, listed in Supplementary Table 1. We used publicly available data from GTEx (<https://gtexportal.org/home/>), the Neale laboratory (<http://www.nealelab.is/uk-biobank/>), the Finucane laboratory (<https://www.finucanelab.org>), the FinnGen Freeze 4 cohort (https://www.finnngen.fi/en/access_results) and the eQTL catalogue release 3 (<http://www.ebi.ac.uk/eQTL/>).

Code availability

The code for summary statistics lift-over, the projection PCA pipeline including precomputed loadings and meta-analyses (<https://github.com/covid19-hg/>); for heritability estimation (https://github.com/AndrewsLabUCSF/COVID19_heritability); for Mendelian randomization and genetic correlation (<https://github.com/marcoralab/MRcovid>); and subtype analyses (https://github.com/mjpirinen/covid19-hgi_subtypes) are available at GitHub.

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Acknowledgements We acknowledge M. O'Reilly and B. Cooley from the Pattern team at the Broad Institute of MIT and Harvard for designing the geographical map in Fig. 1. A full list of the members of the Genes & Health Research Team is available online (<https://www.genesandhealth.org/research/scientific-publications-authorship-and-acknowledgments>). A full list of acknowledgements is provided in Supplementary Table 12.

Author contributions Detailed author contributions are integrated in the authorship list.

Competing interests A full list of competing interests is provided in Supplementary Table 12.

Additional information

Supplementary information The online version contains supplementary material available at <https://doi.org/10.1038/s41586-023-06355-3>.

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Matters arising

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The Danish Blood Donor Study

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The Genetic Predisposition to severe COVID-19

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Variability in Immune Response Genes and Severity of SARS-CoV-2 Infection (INMUNGEN-CoV2 study)

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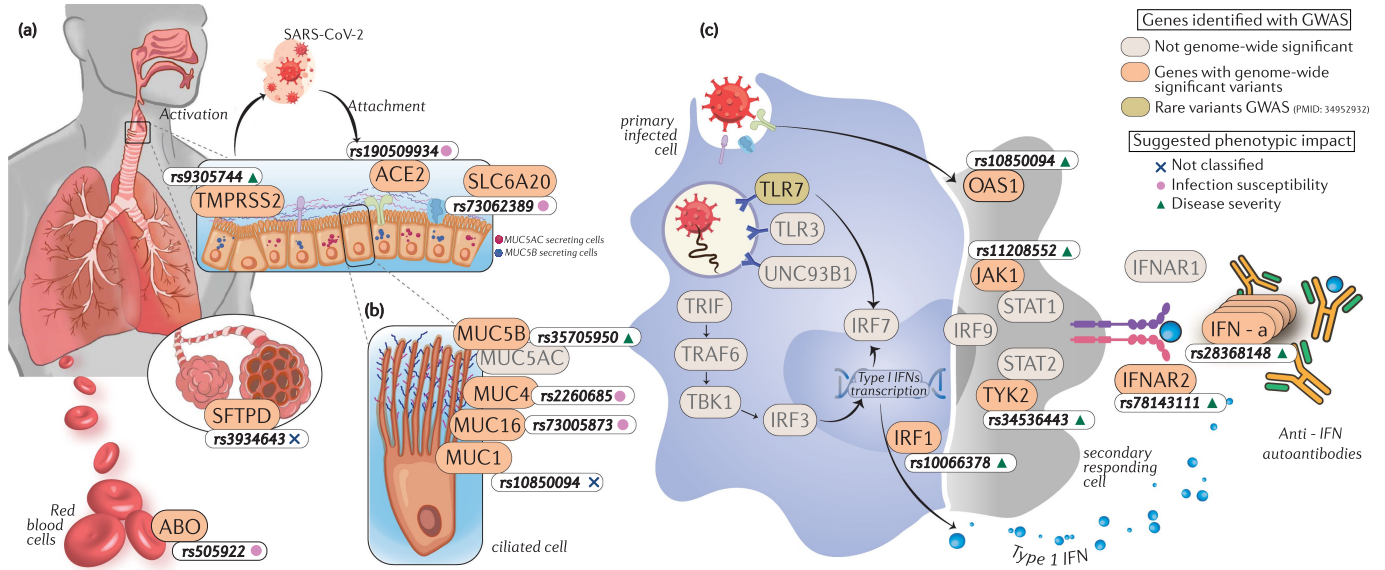
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Matters arising



Extended Data Fig. 1 | Major COVID-19 biological pathways mapped with susceptibility and severity GWAS loci. Genome-wide significant variants associated with COVID-19 (white boxes) and the annotated genes (peach boxes) are mapped on to pathways known to be involved in (a) viral entry and innate immunity, (b) entry defence in airway mucus, and (c) type I interferon. The suggested phenotypic impact of the significant variants using the Bayesian

approach² are denoted with shapes; COVID-19 susceptibility (pink circles), COVID-19 disease severity (green triangles), and unclassified variants (blue cross). Other genes known to be involved in the aforementioned pathways are shown using grey boxes. Detailed list of references to studies used to design this pathway can be found in Supplementary Note.

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Software and code

Policy information about [availability of computer code](#)

- | | |
|-----------------|--|
| Data collection | No code was used to collect data in the study. |
| Data analysis | <p>Each individual study that contributed genetic-phenotype association summary statistics to the consortium carried out their association analyses independently of the consortium (study-specific information outlined in Supplementary Table 1). However, the consortium released phenotyping and analysis guidelines as a recommendation (https://www.covid19hg.org/). For quality control of genotype data we recommended using the Ricopili pipeline (PMID: 31393554). For genotype phasing and imputation we recommended the TopMed Imputation Server (PMID: 27571263) or Michigan Imputation Server (PMID: 27571263). For genome-wide association study (GWAS), we recommended SAIGE (PMID: 30104761), but some studies used PLINK (PMID: 17701901). Each study then submitted their GWAS summary statistics to the consortium for meta-analysis. No version was specified in the recommendation—each study used the best available version for them. LD score regression v 1.0.1 (PMID: 25642630) was used for heritability and partitioned heritability analyses. Variants for Mendelian randomization instruments were selected using PLINK version 1.90b6.18 (PMID: 17701901). Exposure and outcome datasets were harmonized, and MR statistical analysis conducted using R version 4.0.3. with the R-package TwoSampleMR version 0.5.5 (PMID: 29846171) (which included a fixed-effect IVW analysis [PMID: 24114802], weighted median estimator [WME] [PMID: 27061298], weighted mode based estimator [WMBE] and MR Egger regression [PMID: 26050253]) and additionally MR-PRESSO version 1.0 (PMID: 29686387). Code availability statement: The code for summary statistics lift-over, the projection PCA pipeline including precomputed loadings and meta-analyses are available at https://github.com/covid19-hg/; heritability estimation at https://github.com/AndrewsLabUCSF/ COVID19_heritability; Mendelian randomization and genetic correlation at https://github.com/marcoralab/MRcovid; and subtype analyses at https://github.com/mjpirinen/covid19-hgi_subtypes.</p> |

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- Accession codes, unique identifiers, or web links for publicly available datasets
- A description of any restrictions on data availability
- For clinical datasets or third party data, please ensure that the statement adheres to our [policy](#)

Summary statistics generated by COVID-19 HGI are available online, including per-ancestry summary statistics for African, Admixed American, East Asian, European, and South Asian ancestries (<https://www.covid19hg.org/results/r7/>). The analyses described here utilize the data release 7. If available, individual-level data can be requested directly from contributing studies, listed in Supplementary Table 1. We used publicly available data from GTEx v8 (<https://gtexportal.org/home/>; dbGaP Accession phs000424.v8.p2), the Neale lab (<http://www.nealelab.is/uk-biobank/>), Finucane lab (<https://www.finucanelab.org>), FinnGen Freeze 4 cohort (https://www.finnngen.fi/en/access_results), and eQTL catalogue release 3 (<http://www.ebi.ac.uk/eqtl/>).

Human research participants

Policy information about [studies involving human research participants and Sex and Gender in Research](#).

Reporting on sex and gender

Biological sex was collected by each study. The consortium recommended using biological sex as one of the covariates to correct for potential confounding effects (full recommended covariates are: age + age² + sex + age*sex + 20 principal components obtained using genetic data). No sex or gender specific analysis was reported in this study. The consortium did not collect gender information.

Population characteristics

Summary statistics from 82 independent studies were included in consortium meta-analyses. The sample size for genetic ancestry populations for Sars-CoV2 infection was: n=17,898 Middle Eastern; n=53,116 South Asian; 40,580 East Asian; 148,891 African; 129,629 Ad-mixed American; 2,824,391 European. Population characteristics regarding age, sex and exact case and control sample numbers for each contributing study are given in Supplementary Table 1.

Recruitment

The consortium pre-defined phenotype criteria for cases and controls, but the specific recruitment was carried out independently by each contributing study. COVID-19 disease status (critical illness, hospitalization status) was assessed following the Diagnosis and Treatment Protocol for Novel Coronavirus Pneumonia (PMID: 32358325). The critically ill COVID-19 group included patients who were hospitalized due to symptoms associated with laboratory-confirmed SARS-CoV-2 infection and who required respiratory support or whose cause of death was associated with COVID-19. The hospitalized COVID-19 group included patients who were hospitalized due to symptoms associated with laboratory confirmed SARS-CoV-2 infection. The reported infection cases group included individuals with laboratory-confirmed SARS-CoV-2 infection or electronic health record, ICD coding or clinically confirmed COVID-19, or self-reported COVID-19 (e.g. by questionnaire), with or without symptoms of any severity. Genetic ancestry-matched controls for the three case definitions were sourced from population-based cohorts, including individuals whose exposure status to SARS-CoV-2 was either unknown or infection-negative for questionnaire/electronic health record based cohorts.

Ethics oversight

Ethical statements for each contributing study are given in Supplementary Table 1.

Note that full information on the approval of the study protocol must also be provided in the manuscript.

Field-specific reporting

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Life sciences study design

All studies must disclose on these points even when the disclosure is negative.

Sample size

The consortium meta-analysed genome-wide association study (GWAS) summary statistics from any individual study that had included a minimum of 100 cases and 100 controls in their analysis. The cutoff was aimed at reducing noise for the meta-analysis, but also to be inclusive of studies that had not yet accumulated large numbers of COVID-19 patient data. The consortium aimed to reach the largest possible sample size for the study and meta-analyzed all the submitted summary statistics. No statistical calculation for adequate sample size was performed, but the results identifying multiple genomic regions at genome-wide significance threshold indicates adequate power for genetic discovery.

Data exclusions

Individual level phenotype and genotype data exclusions were performed by each individual study, following the consortium analysis plan recommendations (www.covid19hg.org). Possible reasons for sample exclusion included removing genetic ancestry outliers within a study

(using principal components analysis), poor quality of genetic data or lack of phenotypic data for a sample. The consortium manually examined GWAS summary statistics data submitted by each study (for each submitted analysis separately), including sample size used for analysis, allele frequency check against Gnomad reference panel, and distribution of test statistics. After meta-analysis, the results were checked for heterogeneity variant effects between contributing studies in Supplementary Table 2

Replication

No replication was performed. The consortium meta-analysed GWAS summary statistics, bringing together as many studies as possible to achieve the largest possible sample size and statistical power for association. This meant that the consortium included most large studies of COVID-19 host genetics that have been performed to date, so it was not possible to perform replication analyses in external cohorts. Therefore we performed manual checks on each study contributing summary statistics before entering them into the meta-analysis. In addition, after meta-analysis, we performed a check for heterogeneity between variant association estimates across studies contributing data. This allowed us to better understand whether the variant effects differed much between individual studies.

Randomization

No randomization was performed because there was no allocation of samples to experimental groups

Blinding

Blinding was not relevant to the study because no intervention was implemented in the study. The case status and severity of symptoms was evaluated for each sample by investigators from each study respectively. The consortium recommended using covariates to control for confounding: age + age² + sex + age*sex + 20 principal components (obtained using genetic data) + study specific covariates (if any). The consortium meta-analysed summary statistics from these case/control studies, not individual level data. Details of which variables each study used and how the calculated PCs for their analysis are available in Supplementary Table 1.

Reporting for specific materials, systems and methods

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