# Supplementary Tables

**Table 1: List of Genes implicated in the entry and pathogenesis of Human coronaviruses**

| Genes | Reference (PMID/PMCID) | Evidence |
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| ACE2 | 32418199 | Angiotensin-converting enzyme 2 (ACE2) has been established as the functional host receptor for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) |
| CCL2 | 15919935 | SARS-CoV induced the expression of chemokines such as CXCL10/IFN-gamma-inducible protein 10 and CCL2/monocyte chemotactic protein 1 |
| CD40 | 24845462 | A poor virus-specific CD4 and CD8 T cell response is attributed to an inefficient immune activation by SARS-CoV-MA15, particularly of respiratory DC (rDCs), as shown by reduced expression of MHC-II, CD86 and CD40 on cells harvested from the lung |
| CLTC | 17522231 | These results suggest that when SARS-CoV binds ACE2 it is internalized and penetrates early endosomes in a clathrin-dependent manner and that the cytoplasmic tail of ACE2 is not required for the penetration of SARS-CoV. |
| CRP | 32243911 | In the early stage of COVID-19 CRP levels were positively correlated with lung lesions and could reflect disease severity. |
| CSF2 | 32513566 | In the short time since the emergence of COVID-19, numerous studies have described abnormal levels of the following cytokines and chemokines in the patients: IL-1, IL-2, IL-4, IL-6, IL-7, IL-10, IL-12, IL-13, IL-17, M-CSF, G-CSF, GM-CSF, IP-10, IFN-γ, MCP-1, MIP 1-α, hepatocyte growth factor (HGF), TNF-α, and vascular endothelial growth factor (VEGF) |
| CSF3 | 32513566 | In the short time since the emergence of COVID-19, numerous studies have described abnormal levels of the following cytokines and chemokines in the patients: IL-1, IL-2, IL-4, IL-6, IL-7, IL-10, IL-12, IL-13, IL-17, M-CSF, G-CSF, GM-CSF, IP-10, IFN-γ, MCP-1, MIP 1-α, hepatocyte growth factor (HGF), TNF-α, and vascular endothelial growth factor (VEGF) |
| CTSA | 32221306 | Depending on virus strains and cell types, CoV S proteins may be cleaved by one or several host proteases, including furin, trypsin, cathepsins, transmembrane protease serine protease-2 (TMPRSS-2), TMPRSS-4, or human airway trypsin-like protease (HAT) |
| CTSB | 32221306 | Depending on virus strains and cell types, CoV S proteins may be cleaved by one or several host proteases, including furin, trypsin, cathepsins, transmembrane protease serine |

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| | | protease-2 (TMPRSS-2), TMPRSS-4, or human airway trypsin-like protease (HAT) |
| CTSD | 32221306 | Depending on virus strains and cell types, CoV S proteins may be cleaved by one or several host proteases, including furin, trypsin, cathepsins, transmembrane protease serine protease-2 (TMPRSS-2), TMPRSS-4, or human airway trypsin-like protease (HAT) |
| CTSg | 32221306 | Depending on virus strains and cell types, CoV S proteins may be cleaved by one or several host proteases, including furin, trypsin, cathepsins, transmembrane protease serine protease-2 (TMPRSS-2), TMPRSS-4, or human airway trypsin-like protease (HAT) |
| CTSk | 32221306 | Depending on virus strains and cell types, CoV S proteins may be cleaved by one or several host proteases, including furin, trypsin, cathepsins, transmembrane protease serine protease-2 (TMPRSS-2), TMPRSS-4, or human airway trypsin-like protease (HAT) |
| CTSS | 32221306 | Depending on virus strains and cell types, CoV S proteins may be cleaved by one or several host proteases, including furin, trypsin, cathepsins, transmembrane protease serine protease-2 (TMPRSS-2), TMPRSS-4, or human airway trypsin-like protease (HAT) |
| CXCL10 | 28466096 | SARS-CoV-infected airway epithelial cells (AECs) also produce large amounts of CCL3, CCL5, CCL2, and CXCL10 |
| CXCL5 | 32446778 | SARS-CoV-2 upregulated only five of them, (namely, CXCL10, IL6, CCL2, CXCL1, CXCL5 |
| DPP4 | 32224164 | Antibodies directed against DPP4 inhibited human coronavirus-Erasmus Medical Center (hCoV-EMC) infection of primary human bronchial epithelial cells and Huh-7 cells. |
| FURIN | 32362314 | The host cell protease furin cleaves the SARS-CoV-2 spike protein at the S1/S2 site |
| IFNA10 | 26410416 | The nucleocapsid protein (N protein) of SARS-CoV plays an important role in inhibition of type I interferon (IFN) production via an unknown mechanism |
| IFNA14 | 26410416 | The nucleocapsid protein (N protein) of SARS-CoV plays an important role in inhibition of type I interferon (IFN) production via an unknown mechanism |
| IFNA16 | 26410416 | The nucleocapsid protein (N protein) of SARS-CoV plays an important role in inhibition of type I interferon (IFN) production via an unknown mechanism |
| IFNA2 | 26410416 | The nucleocapsid protein (N protein) of SARS-CoV plays an important role in inhibition of type I interferon (IFN) production via an unknown mechanism |
| IFNA21 | 26410416 | The nucleocapsid protein (N protein) of SARS-CoV plays |

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| | | an important role in inhibition of type I interferon (IFN) production via an unknown mechanism |
| IFNA8 | 26410416 | The nucleocapsid protein (N protein) of SARS-CoV plays an important role in inhibition of type I interferon (IFN) production via an unknown mechanism |
| IFNAR1 | 32275914 | IFN-I are thus among the first cytokines produced during a viral infection. They are recognized by the IFNAR receptor present at the plasma membrane in most cell types. |
| IFNAR2 | 32275914 | IFN-I are thus among the first cytokines produced during a viral infection. They are recognized by the IFNAR receptor present at the plasma membrane in most cell types. |
| IFNB1 | 15476870 | Interferon-beta and Interferon-Gamma Synergistically Inhibit the Replication of Severe Acute Respiratory Syndrome-Associated Coronavirus (SARS-CoV) |
| IFNG | 15476870 | Interferon-beta and Interferon-Gamma Synergistically Inhibit the Replication of Severe Acute Respiratory Syndrome-Associated Coronavirus (SARS-CoV) |
| IKBKB | 32194980 | melatonin indirectly targets several HCoV cellular targets, including ACE2, BCL2L1, JUN, and IKBKB |
| IKBKE | 32531085 | Along with interferon and innate immune signalling components, such as interferon regulatory factor 3 (IRF3), transmembrane protein 173 (TMEM173), TANK binding kinase 1 (TBK1), inhibitor of nuclear factor kappa B kinase subunit epsilon (IKBKE), tripartite motif containing 25 (TRIM25), mitochondrial antiviral signalling protein (MAVS) or DExD/H-box helicase 58 (DDX58), C1-INH is one of the proteins with the highest connectivity in the merged CoV-1 and CoV-2 interactomes |
| IKBKG | 28355270 | Compared to treatment with the prototypical inflammatory cytokine interleukin(IL)-1, HCoV-229E replication was found to attenuate the inducible activity of the transcription factor (TF) NF-κB and to restrict the nuclear concentration of NF-κB subunits by (i) an unusual mechanism involving partial degradation of IKKβ, NEMO and IκBα and (ii) upregulation of TNFAIP3 (A20), although constitutive IKK activity and basal TNFAIP3 expression levels were shown to be required for efficient virus replication. |
| IL10 | 32169119 | A few plasma cytokines and chemokines were observed ascended in COVID-19 patients, including IL-1, IL-2, IL-4, IL-7, IL-10, IL-12, IL-13, IL-17, GCSF, macrophage colony-stimulating factor (MCSF), IP-10, MCP-1, MIP-1α, hepatocyte growth factor (HGF), IFN-γ and TNF-α |
| IL10RB | 24751921 | Type III IFNs signal through a different receptor, which is composed of IFNLR1 and IL10RB. Upon binding to their cognate receptors, type I and type III IFNs induce the same Jak/STAT pathway: the transphosphorylation and activation |

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| | | of receptor-associated Jak1 and Tyk2 leads to the phosphorylation of STAT1 and STAT2 transcription factors. |
| IL1B | 32291137 | Evidence suggests that CRS might play a major role in severe COVID-19. Inflammatory cytokines and chemokines, including interleukin-6 (IL-6), interleukin-1β (IL-1β), induced protein 10 (IP10) and monocyte chemoattractant protein-1 (MCP-1) were significantly elevated in COVID-19 patients, and some were more commonly seen in severe patients than in nonsevere patients |
| IL6 | 32291137 | Elevated IL-6 levels were observed in patients with SARS and were correlated with disease severity |
| IL7 | 32169119 | A few plasma cytokines and chemokines were observed ascended in COVID-19 patients, including IL-1, IL-2, IL-4, IL-7, IL-10, IL-12, IL-13, IL-17, GCSF, macrophage colony-stimulating factor (MCSF), IP-10, MCP-1, MIP-1α, hepatocyte growth factor (HGF), IFN-γ and TNF-α |
| IL9 | 32561873 | Concentrations of IL-4, IL-9 and IL-13, which are associated with type 2 immunity, bronchial hypersensitivity and increased mucus production, are also high in patients with COVID-19 |
| IRF3 | 25093995 | The cellular pathways mediated by interferon regulatory factor (IRF)-3 and -7, activating transcription factor (ATF)-2/jun, activator protein (AP)-1, nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB), and nuclear factor of activated T cells (NF-AT), are the main drivers of the inflammatory response triggered after viral infections, with NF-κB pathway the most frequently activated |
| IRF5 | 32733388 | Researchers hypothesized that after COVID-19 infects human cells, the virus utilizes an excess of glucose for a fast viral replication from the hexosamine biosynthetic pathway (HBP) hijacking substrates from the metabolic environment. This process induces overexpression of interferon IRF5, leading to a massive inflammatory gene overexpression, endoplasmic reticulum (ER) stress, and cytokine dysregulation profile. |
| IRF7 | 25093995 | The cellular pathways mediated by interferon regulatory factor (IRF)-3 and -7, activating transcription factor (ATF)-2/jun, activator protein (AP)-1, nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB), and nuclear factor of activated T cells (NF-AT), are the main drivers of the inflammatory response triggered after viral infections, with NF-κB pathway the most frequently activated |
| IRF9 | 25093995 | Type I IFN signaling starts with its binding to IFNAR receptors at the cell surface, which leads to the activation of the JAK–STAT pathway . The members of the Janus Kinase |

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| | | (JAK) family JAK-1 and protein tyrosine kinase 2 (TYK-2) phosphorylate the signal transducer and activators of transcription (STATs) which become activated. Phosphorylated STAT1 and STAT2 recruit IRF-9, to form the IFN stimulated gene factor 3 (ISGF3) complex. The ISGF3 heterotrimer translocates to the nucleus and triggers the transcription of IFN-stimulated genes (ISGs) that will drive the antiviral response. |
| JAK1 | 25093995 | Type I IFN signaling starts with its binding to IFNAR receptors at the cell surface, which leads to the activation of the JAK–STAT pathway . The members of the Janus Kinase (JAK) family JAK-1 and protein tyrosine kinase 2 (TYK-2) phosphorylate the signal transducer and activators of transcription (STATs) which become activated. Phosphorylated STAT1 and STAT2 recruit IRF-9, to form the IFN stimulated gene factor 3 (ISGF3) complex. The ISGF3 heterotrimer translocates to the nucleus and triggers the transcription of IFN-stimulated genes (ISGs) that will drive the antiviral response. |
| MAP3K7 | 28933406 | Phosphorylation of all three MAPK members has been detected in cells infected with SARS-CoV |
| MAPK1 | 22936401 | Results point to SARS -C oV PL pro triggering TGF -β1 production via ubiquitin proteasome, p38 MAPK , and ERK 1/2-mediated signaling. |
| MAPK14 | 22936401 | Results point to SARS -C oV PL pro triggering TGF -β1 production via ubiquitin proteasome, p38 MAPK , and ERK 1/2-mediated signaling. |
| MAPK3 | 28933406 | Expression of SARS-CoV PLpro was shown to increase ERK1 ubiquitin-mediated degradation to suppress IFN-induced responses |
| MYD88 | 19079579 | MyD88 Is Required for Protection from Lethal Infection with a Mouse-Adapted SARS-CoV |
| NFKB1 | 24198408 | Inhibition of NF-κB-mediated Inflammation in Severe Acute Respiratory Syndrome Coronavirus-Infected Mice Increases Survival |
| NFKB2 | 24198408 | Inhibition of NF-κB-mediated Inflammation in Severe Acute Respiratory Syndrome Coronavirus-Infected Mice Increases Survival |
| NLRP3 | 32574259 | Due to this crucial role in triggering inflammatory response to infection, the NLRP3 inflammasome appears to be a potential drug target in the treatment of coronavirus disease 2019 (COVID-19), caused by SARS-CoV-2 |
| PCSK1 | 32099652 | Diabetes is associated with an increase in furin, which is a type-1 membrane-bound protease, belonging to the proprotein convertase subtilisin/kexin family (PCSK). It is involved in the entry of coronaviruses into the cell and |

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| | | increased Furin has been reported in diabetes, which might facilitate viral replication |
| PCSK2 | 32099652 | Diabetes is associated with an increase in furin, which is a type-1 membrane-bound protease, belonging to the proprotein convertase subtilisin/kexin family (PCSK). It is involved in the entry of coronaviruses into the cell and increased Furin has been reported in diabetes, which might facilitate viral replication |
| PCSK5 | PMC7122180 | the furin-like PCs PACE4, PC5, and PC7 may selectively compensate the activation of viral glycoproteins in furin-deficient cells and tissues with different efficiencies. |
| PCSK6 | 32838164 | furin is likely to be a functional complementarity or redundancy of PCSK6 |
| PCSK7 | PMC7122180 | the furin-like PCs PACE4, PC5, and PC7 may selectively compensate the activation of viral glycoproteins in furin-deficient cells and tissues with different efficiencies. |
| PIAS1 | PMC7129356 | The nucleocapsid protein of SARS CoV interacts with PIAS1 and affects the NFkappaB pathway |
| PIAS3 | 9724754 | Inhibition of Stat1-mediated Gene Activation by PIAS1 |
| RARG | 32459003 | RA is the biologically active retinoid metabolite that, acting through its cognate receptors RA receptors (RAR α, β and γ), regulates the expression of genes involved numerous biological pathways including both adaptive and innate immune responses |
| RARRES3 | 30988429 | Thus, while we cannot exclude additional antiviral actions, RARRES3 likely exerts an antiviral effect by downregulating mTOR |
| REL | 28933406 | Mammalian NF-κB family composes of five members, RelA (also named p65), RelB, c-Rel, NF-κB1 p50, and NF-κB2 p52, which form dimers in the cytoplasm,A recent study reported an enhanced nuclear NF-κB activity in PBMCs treated with a purified and recombinant SARS-CoV S protein |
| RELA | 28933406 | Mammalian NF-κB family composes of five members, RelA (also named p65), RelB, c-Rel, NF-κB1 p50, and NF-κB2 p52, which form dimers in the cytoplasm,A recent study reported an enhanced nuclear NF-κB activity in PBMCs treated with a purified and recombinant SARS-CoV S protein |
| RELB | 28933406 | Mammalian NF-κB family composes of five members, RelA (also named p65), RelB, c-Rel, NF-κB1 p50, and NF-κB2 p52, which form dimers in the cytoplasm,A recent study reported an enhanced nuclear NF-κB activity in PBMCs treated with a purified and recombinant SARS-CoV S protein |
| SOCS1 | 30185587 | The induction of SOCS1 and SOCS3 to counteract IFN |

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| | | antiviral responses is also observed in other viruses, such as SARS-CoV |
| SOCS3 | 16482545 | Induction level of suppressor of cytokine signaling-3 (SOCS3) by SARS-CoV was significantly lower than that by RSV in spite of the significant production of IL-6 |
| STAT1 | 25093995 | Type I IFN signaling starts with its binding to IFNAR receptors at the cell surface, which leads to the activation of the JAK–STAT pathway (Samuel, 2001) (Fig. 2 ). The members of the Janus Kinase (JAK) family JAK-1 and protein tyrosine kinase 2 (TYK-2) phosphorylate the signal transducer and activators of transcription (STATs) which become activated. Phosphorylated STAT1 and STAT2 recruit IRF-9, to form the IFN stimulated gene factor 3 (ISGF3) complex. The ISGF3 heterotrimer translocates to the nucleus and triggers the transcription of IFN-stimulated genes (ISGs) that will drive the antiviral response. |
| STAT2 | 25093995 | Type I IFN signaling starts with its binding to IFNAR receptors at the cell surface, which leads to the activation of the JAK–STAT pathway (Samuel, 2001) (Fig. 2 ). The members of the Janus Kinase (JAK) family JAK-1 and protein tyrosine kinase 2 (TYK-2) phosphorylate the signal transducer and activators of transcription (STATs) which become activated. Phosphorylated STAT1 and STAT2 recruit IRF-9, to form the IFN stimulated gene factor 3 (ISGF3) complex. The ISGF3 heterotrimer translocates to the nucleus and triggers the transcription of IFN-stimulated genes (ISGs) that will drive the antiviral response. |
| STAT3 | 26199948 | Severe acute respiratory syndrome coronavirus (SARS-CoV) infection results in STAT3 dephosphorylation. |
| TBK1 | 25093995 | ARS-CoV membrane (M) protein impairs the formation of TRAF3/TANK/TBK1/IKKε complex, inhibiting IFN-β production |
| TLR3 | 26015500 | Toll-Like Receptor 3 Signaling via TRIF Contributes to a Protective Innate Immune Response to Severe Acute Respiratory Syndrome Coronavirus Infection |
| TLR4 | 30571771 | Mice lacking TLR4 had either similar survival rates or even more severe disease than wild-type mice infected with DENV, EBOV, SARS-CoV, or RSV |
| TLR7 | 32342146 | The prominent ligand for TLR7 and 8 is viral ssRNA |
| TLR8 | 32342146 | The prominent ligand for TLR7 and 8 is viral ssRNA |
| TMPRSS11D | 32221306 | Expression of TMPRSS 2, 4, 11 A, 11D, and 11E on 293/hACE2 cells enhanced SARS-CoV-2 S protein-mediated cell–cell fusion similarly to SASR-CoV S |
| TMPRSS11E | 32221306 | Expression of TMPRSS 2, 4, 11 A, 11D, and 11E on 293/hACE2 cells enhanced SARS-CoV-2 S protein-mediated cell–cell fusion similarly to SASR-CoV S |

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| TMPRSS2 | 32221306 | Expression of TMPRSS 2, 4, 11 A, 11D, and 11E on 293/hACE2 cells enhanced SARS-CoV-2 S protein-mediated cell–cell fusion similarly to SASR-CoV S |
| TMPRSS4 | 32221306 | Expression of TMPRSS 2, 4, 11 A, 11D, and 11E on 293/hACE2 cells enhanced SARS-CoV-2 S protein-mediated cell–cell fusion similarly to SASR-CoV S |
| TRAF3 | 19380580 | Severe Acute Respiratory Syndrome Coronavirus M Protein Inhibits Type I Interferon Production by Impeding the Formation of TRAF3·TANK·TBK1/IKKϵ Complex* |
| TRAF6 | 27164085 | SARS Coronavirus Papain-Like Protease Inhibits the TLR7 Signaling Pathway Through Removing Lys63-Linked Polyubiquitination of TRAF3 and TRAF6 |
| TYK2 | 25093995 | Type I IFN signaling starts with its binding to IFNAR receptors at the cell surface, which leads to the activation of the JAK–STAT pathway (Samuel, 2001) (Fig. 2 ). The members of the Janus Kinase (JAK) family JAK-1 and protein tyrosine kinase 2 (TYK-2) phosphorylate the signal transducer and activators of transcription (STATs) which become activated. Phosphorylated STAT1 and STAT2 recruit IRF-9, to form the IFN stimulated gene factor 3 (ISGF3) complex. The ISGF3 heterotrimer translocates to the nucleus and triggers the transcription of IFN-stimulated genes (ISGs) that will drive the antiviral response. |

# Supplementary Figures

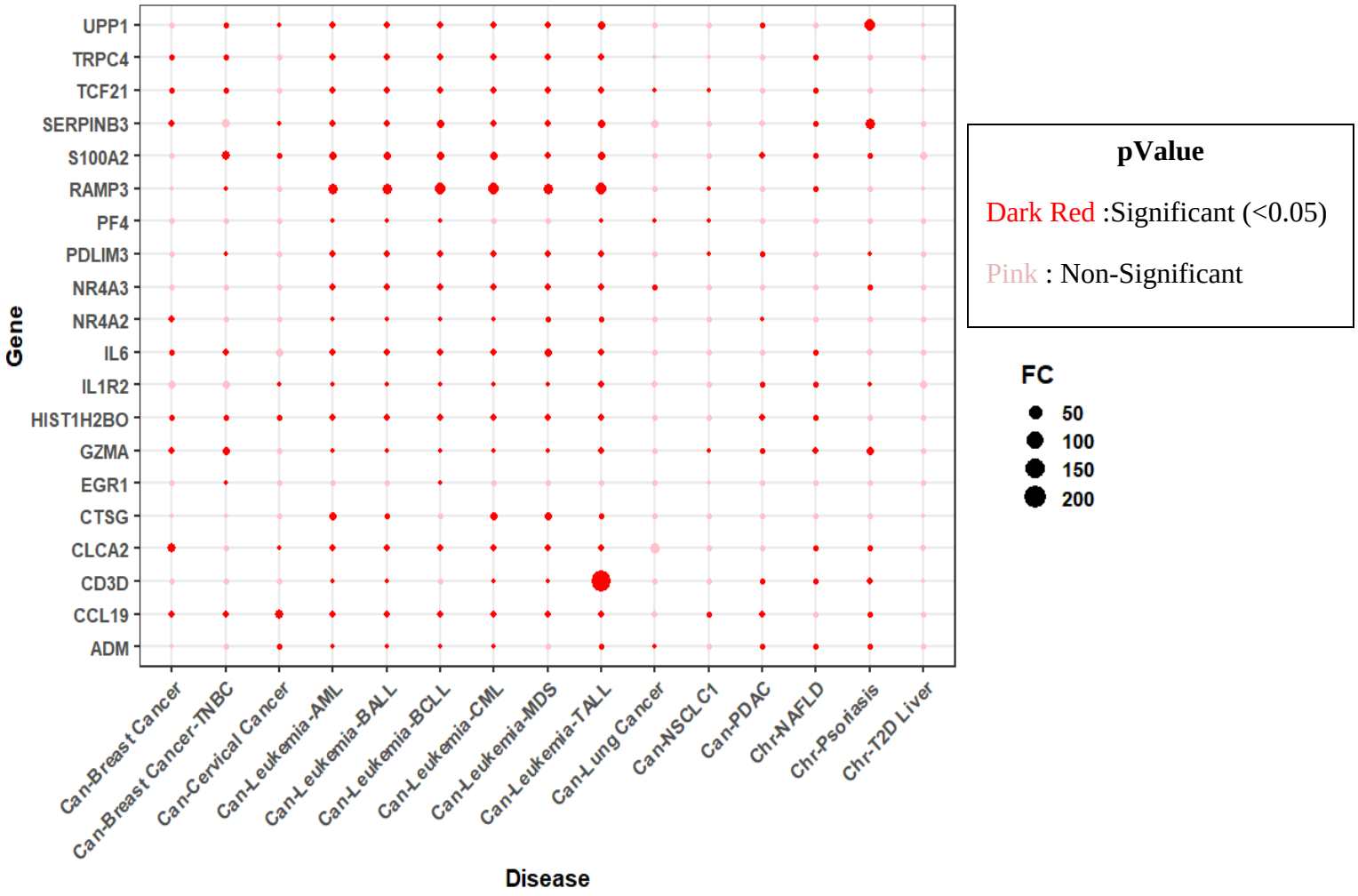


**Figure 1: Bubble plot of expression pattern, in select disease types, of top 20 genes that are differentially expressed in SARS-CoV-2 patients.**