Table S1: Phenotypic and function alterations of NKT cells in SARS-CoV-2 infection.								
	Type of sample	**Activated mediators**	**Produced Cytokines**	**In situ recruitment**	**Chemokine receptors**	**Expression Markers**	**Differential expression markers in subsets**	
Jouan et al (Ref 39)	Serum, Blood, ETAs. Tool: Flow cytometry.	↑ level of IL-1β, IL-6, IL-1RA, IFNα2, IFNγ, IL17 in supernatants than serum. ↑IL18 in COVID-19 pts.	Upon PMA/ Iono: ↑ % IL17 NKT cells than IFNγ NKT in COVID-19 pts compared to HCs.	Airway NKT cells undetectable	In ETAs: ↑CXCL10, CXCL12	PD-1; CD69	Plasmatic IL18 Corr.+ with CD69 NKT in COVID-19 pts. ↑CD69 in COVID-19 pts compared to HCs and Non-COVID-19. CD69 NKT Corr.+ with decreasing hypoxemia.	↑ PD1 NKT in COVID-19 pts compared to HCs.
Parrot et al (Ref 40)	Serum, Blood. Tool: Flow cytometry; Sc-RNA-seq.	↑CXCL10, CX3CL1 in serum of COVID-19 pts compared to HDs.	ND	ND	ND	ND	ND	ND
Odak et al (Ref 41)	Blood analyzed by Flow cytometry	ND	ND	ND	ND	ND	ND	ND
Tomi et al (Ref 42)	Blood, Serum; Tool: Flow cytometry	In severe: ↑ IL-6, IL-8, MCP-1, IL-18, TGFβ, IL-10. ↓IL-12p70.	ND	ND	ND	ND	ND	ND
		In mild: ↓IL-10 , IL4. ↑TGFβ. ↓ IL-17A, IL-17F.						
Vigon et al (Ref 45)	Blood, Serum; Tool: Flow	Plasma of Critical: ↑IL-8/ CXCL8, IL-6, TNFα; IFNγ;	ND	↓%CD56+CD16+GZB+ in response to Hsp70 peptide.	ND	Degranulation and	↑% CD3+CD56+CD16+ in C.C.	↑% CD3+CD56+CD16+ CD107a+ in C.C. in

	Cytometry, Luminex.	Plasma Severe and Critical: ↑CD25/IL-2Ra; ↓ IL2, compared to mild disease.		↓cytotoxic capacity against target cells without MHC class I molecules in comparison with M.C.		cytotoxic markers.		comparison with mild (data not significant).

Surnatant of endotracheal aspirates: *ETAs;* Positively correlated: *Corr.+;* critical COVID-19: *C.C.;* mild COVID-19: *M.C;* ↑ increases frequency or number compared to Controls; ↓ decreases frequency or number compared to Controls. Patients: *pts*.

Table S2: Phenotypic and function alterations of γδT cells in SARS-CoV-2 infection.

	Type of sample	**Activation marker in blood**	**Activation marker in situ**	**Expression marker**	**Differential expression markers in subsets**			
Odak et al (Ref 41)	Blood; Tool: Flow cytometry	ND	↓% γδ eff cells in blood	CD45RA, CD62L	↑CD45RA+CD62+ (γδ naïve).	↓CD45RA-CD62- (γδ eff).	↓CD45RA-CD62L+ memory- in Severe patients compared to HCs.	↓CD45RA+CD62L- in Severe patients compared to HCs.
Zhang et al (Ref 46)	Blood; Tool: Sc-RNA-seq	ND	ND	Genes: GZMK GZMA PRF1 TRAV1-2 TYROBP	ND			
Jouan et al (Ref 39)	Serum, blood and surnatant of ETAs. Tool: Flow cytometry.	↑%CD69+ γδ T cells, % PD1+ γδ T cells compared to HDs.	↑%CD69+ γδ T cells, PD-1+ γδ T cells in ETA compared to blood of matched pts	ND	Vδ2+: ↑ CD69+ compared to HDs. ↑ PD1 compared to HDs.	CD69+ Vδ1: from COVID-19 pts were not significantly changed compared with non–COVID-19 ill controls. ↑ PD1+ Vδ1 compared to HDs.	Vγ1Vδ2-: from COVID-19 pts were not significantly changed compared with non–COVID-19 ill controls. PD1+ Vγ1Vδ2-: did not change compared to HDs.	

Stephenson et al (Ref 44)	Blood, Serum; Tool: gene expression.	IL1B, IL1A and TNFα.	ND	↓ CD28, CTLA4, CD40GL	ND

Effector-like γδ cells; ↑ increases frequency or number compared to Controls; ↓ decreases frequency or number compared to Controls.

Table S3: Phenotypic and function alterations of ILCs in SARS-CoV-2 infection.

	Type of sample	Activation marker in blood		Differential expression markers in subsets	
Garcia et al (Ref 55)	Blood analyzed by Flow cytometry	Levels of the cytokine IL- 10 positively correlated with the levels of CD69+ ILCs in the COVID-19 patients.	ND	↓ CXCR3. CCR4−/lo ILC1 clusters were overrepresented in severe COVID-19 patients.	↓CD56+ ILC1 in COVID-19 patients.
				ILC2 negatively correlated with CCL20 levels.	↑CD69+ ↓ CD62L+
		Activated ILCp positively correlated with serum IL-6 levels in the COVID-19 patients.		ILCp positively correlated with CXCL10 levels in the COVID-19 patients. ↓CXCR3+ activated ILCp in both moderate and severe COVID-19.	↑CD69+ ; (CD45RA+/hi/CD62L+/hi) were reduced in COVID-19 pts as compared with controls. IL18R1 and PD-L1 negatively correlated with the level of CD45RA+ ILCp.
Segundo et al (Ref 58)	Blood, serum; Tool: Flow cytometry.	IL6	ND	ND	ND
Silverstein et al (Ref 56)	Blood analyzed by Flow cytometry.	ND	ND	ND	ND
	RNA-seq profiles of ILC from available dataset online	ND	ND	ND	ND
	Blood analyzed by Flow cytometry.	Males had a lower median fraction of AREG+ ILCs than females. AREG expression in males was lower than females.	IL13 and amphiregulin (upon in vitro stimulation with Iono/PMA).	They hypothesis that ILC2 were recruited in lung tissue. They compared RNA-seq on blood ILCs from 9 HCs to previously	ND

				published RNA-seq profiles of ILCs sorted from lung, spleen and Intestine.	
	RNA-seq profiles of ILC from available dataset online	ND	ND	ND	ND
Gomez-Cadena et al (Ref 57)	Mouse model and Blood from patients; serum analysis.	Elevated levels of the type 2 cytokines IL-5 , IL-13 and significant increase in IL-33 in COVID-19 patients compared to that in healthy donors; in particular serum IL-18 and IL6 levels were significantly higher in severe COVID-19.	ND	ND	ILC2s in severe pts ↑ NKG2D+ population compared to those in mild pts and controls, CD25 and KLRG1 ↓. No differences in NKG2D, KLRG1, or CD25 expression were observed in ILC1s or ILCPs. PD-1, NKG2A, and NKp46, were similar on ILC2s from HDs and pts.
	ND	ND	ND	ND	ILC2 NKG2D expression: Low 31.2%, High 68.18% of severe pts.

↑ increases frequency or number compared to Controls; ↓ decreases frequency or number compared to Controls. Patients: *pts*.

Table S4: Phenotypic and function alterations of MAIT cells in SARS-CoV-2 infection.

	Type of sample	**Activated mediators**	**Produced Cytokines**	**In situ recruitment**	**Chemokine receptors**	**Expression Markers**	**Differential expression markers in subsets**	
Chen et al (Ref 47)	Blood; Tool: sc-RNA seq and TCR sequencing	↑*IFNGR1, STAT1, TNFRSF1B, TNFAIP3,* and *TNFSF8* in Pcov (Severe, moderate and healthy) compared to Non-Pregnant (Severe, moderate and healthy) significantly.	ND	GO terms of "leukocyte cell–cell adhesion" was pronouncedly enriched in PCov groups than in the NCov groups compared to their corresponding HCs.	↑CXCR4 in PCov groups compared to NHC.	ND	ND	ND

	Type of sample	Activated mediators	Produced Cytokines	Expression Markers
Deschler et al (Ref 43)	Blood; Tool: Flow Cytometry	↓ IL-12R and IL-18R from COVID-19 patients compared to healthy controls.	Upon in vitro E.coli stimulation, significantly ↑ IFNγ expression; MAIT cells failed to upregulate expression of IL-17A and TNFα. Upon in vitro stimulation with IL-12 and IL-18, ↓IL-17A and TNFα from COVID-19 pts compared to healthy controls; ↑ IFNγ in response to IL-12/IL-18 ↓ in response to E. coli stimulation. Any differences in the expression of TNFα and IL-17A among mild and severe COVID-19 irrespective of the way of stimulation.	Expression of CD38, CD69 and HLA-DR, CTLA-4 and PD-1 in COVID-19 patients compared to healthy controls. Precisely, ↑CD38 and CD69 significantly in acute COVID-19 than Convalescent. ND
			↓ granzyme B expression and perforin expression upon IL-12/IL-18 stimulation. MAIT from severe COVID-19 were able to significantly ↑ granzyme B expression upon IL-12/IL-18 stimulation and significantly higher levels from patients with mild COVID-19.	↑CTLA-4 and PD- 1 in severe COVID-19 but not significantly. ↓CTLA-4 in convalescent patients with severe COVID-19 and ↓↓ CTLA-4 in mild COVID-19.
			Ex vivo analysis: ↑ TNFα and IL-17A, GRZ B in Covid-19 compared to healthy controls while IFNγ and perforin were unchanged. ↑Perforin in patients with severe COVID-19 compared to mild COVID-19 and healthy controls; IFNγ, TNFα, IL-17A and GRZ B did not differ between MAIT cells isolated from patients with mild or severe COVID-19.	
Hubrack et al (Ref 48)	Blood; Tool: Flow Cytometry.	ND	Upon in vitro long stimulation with IL12/IL18: ↓ GRZ B, TNF, IFNγ, Perforin compared to controls.	↑CD69 (% and MFI) in Severe patients compared to HC.
			Upon in vitro stimulation with IL7: ↓ GRZ B, IFNγ, Perforine but not significantly. ↓ TNFα significantly.	Slight decrease level of CD69 (% and MFI) in mild patients compared to severe. Data did not significant.
			Upon in vitro IL12/IL18 or IL7: ↑ MFI of Perforin compared to Unstimulated condition.	
Notarbartolo et al (Ref 49)	Blood. Tool: Sc-RNAseq and TCR-seq;	↑CD69, FOS, DUSP1	↑ IFN- responsive genes in patients with severe disease.	*KLRB1 (CD161), SLC4A10, RORC, and CCR6* *TCR* gene usage *(TRAV1-2, TRAJ33/12/20, and TRBV20/6)* *CCL5, NKG7, PRF1, CST7, and GZMK*

	Flow cytometry.					
	Type of sample	Chemokines	**Expression markers**	**Differential expression markers in subsets**		
Shi et al (Ref 50)	scRNA-seq raw data from GSA	↑ genes associated with chemotaxis and apoptosis in severe cases.	↑*TRBV9*, *TRAV8-2*, *S100A8*, *GZMH*, *S100A9*, *KLF6*, *CD8B*, *KLRD1*, *IGLV3-19*, and *JCHAIN* ↓ *SLC4A10* and *TRAV1-2* The number of high frequencies of clonotypes of MAIT cells was relatively high. There were more large clonal expansions (clonal size >10) in the severe cases than in the other conditions. ↓The expression levels of genes associated with cell activation in MAIT of severe cases.	↑% TRAV1−2 or TRBV33 cells. ↓ TRAV1-2 or TRBV33 compared with moderate and convalescent cases.	↓TRAV1-2 in severe cases compared with other conditions.	↓The usage level of gene pair TRAV1-2 and TRAJ12 in severe cases.

	Type of sample	**Cytokine production by Flow Cytometry analysis**	**Cytokine production by Sc-gene expression**			
Flament et al (Ref 53)	Blood analysed by flow cytometry. Serum to quantify proteins.	In the blood of surviving patients with COVID-19 hospitalized in an IDU: ↓IL-6, IL-8, IL-10, ↑IFNα2, ↓IL15, IL18, IL1β compared to ICU o fatal Covid-19.	Upon Iono/ (PMA) ionomycin in vitro stimulation and withou stimulation:↑ IFNγ, IL2 and GZB and IL17 of circulating MAIT in severe patients.			
		In the blood of surviving patients with COVID-19 hospitalized in an ICU: ↓IL-6, IL-8, IL-10, IFNα2, ↓IL15, IL18, IL1β compared to fatal Covid-19.	↑IFNγ and TNFα and GzB in deceased patients compared to surviving patients.			
	Blood analysed by Facs Sorting and RT-qPCR. Sc-gene expression.	In the blood of surviving patients with COVID-19 hospitalized in fatal COVID-19: ↑IL-6, IL-8, IL-10, IFNα2, IL15, IL18, IL1beta compared to ICU and IDU except for IFNγ. Note: differences in IL1b are not significant.	Gene expression in ICU: ↓ *IFNA* ↑ *IL18* in opposite compared to IDU. Gene expression in ICU: ↓ *STATA1*, *IRF1*, *IRF9* compared to IDU. Gene Expression in ICU: ↑*NLRP3*, *TXNIP* compared to IDU. Gene Expression in ICU: ↑*PRDM1*, *HOBIT*, *TBX21*.			
Yang et al (Ref 52)	Blood analyzed by Mass cytometry and Flow Cytometry.	Upon E. coli stimulation or IL12/IL18: severe COVID-19 ↓IFNγ, GzmB, and CD107a than those from HC; Upon coculture with CD14+ cells from patients with severe COVID-19 ↓IFNγ and GzmB production.	TEM (CD45RA + CD197-).	naive (CD45RA + CD1971+)	central memory T (CD45RA	TEM (CD45RA-CD197 +) like cells.

		Upon E.coli stimulation or IL12/IL18: mild COVID-19 ↓ GzmB and CD107a than those from HC. In asymptomatic carriers with COVID-19 function of MAIT remain unchanged. Upon E.coli stimulation or IL12/IL18: convalescent patients restored their functions.		. Significantly decreased in patients with severe COVID-19	-CD1971-). Increase in both Mild and Severe COVID-19 patients.	Significantly increased in patients with mild and severe COVID-19.
	Type of sample	**In situ recruitment**	**Expression markers**	**Differential expression markers in subsets**		
Yu et al (Ref 51)	Blood, BALF, NPS. Tool: Flow cytometry; sc-RNA seq.	↑MAIT in airway tissue of Covid-19 Females more than males.	IL-4, IL-13, IL-10, TGF-b, NGF- TRKA signaling axis, RAF-independent mitogen-activated protein kinase (MAPK), estrogen-dependent pathway.	MAIT alpha in healthy individuals but after SARS-CoV2 infection females pts lost this phenotype.	MAIT beta in exposed and infected groups; females in late disease increased percentage of phenotype beta.	
		airway tissue of Covid-19 Females: *IL7R, CISH, SOCS1.* ↑ *BCL2, FOXP1, CDKN1B,BTG2, KLF2, MYC,* and *CEBPD.* ↓*BAX* and *CASP3*	↑CD69 in hospitalized group without any correlation with sex or phenotype.			
		airway tissue of COVID-19 Males: ↑CCL2.				
Parrot et al (Ref 40)	Serum, blood. Tool: Flow cytometry and Sc-RNA-seq.	The transcriptional profile indicated that MAIT cells were the main subset of airway T cells expressing IL17A. This profile was paired with expression of TNF and an apparent lack of IFNG and GZMB transcripts.	Inverse correlation between CCL28, CXCL11, CCL20 , IL17C and absolute number of MAIT.	↑CD69 in AS patients together with GrzB and Ki67 than AM patients . PD-1, IL-7R, CXCR6, granzyme A (GrzA), and CD56 were similar between HD, AM, and AS groups.↑ CD69 on MAIT cells of died patients (analyzed 4 patients) than patients		

				who survived. In convalescent patients CD69 repristinated its normal values.	
	ND	↓CXCR3 in both AM and AS COVID-19 patients. In convalescent patients, CXCR3 levels were still suppressed.	ND	ND	ND
Zhang et al (Ref 46)	Blood; Tool: Sc-RNA-seq	ND	ND	GO analysis demonstrated that MAIT from moderate and severe patients responded more to IFN than the acute inflammation.	
	Type of sample	**Cytokine expression in situ**	**Cytokine expression in blood**	**Differential expression markers**	
Jouan et al (Ref 39)	Serum, blood and surnatant of ETAs. Tool: Flow cytometry.	↑% MAIT in the airways.↑ IL-1β, IL-6, IL-1RA, IFN-α2, IFNg and IL17 in supernatants of ETAs. The plasma level of IL18 ↑ in Covid-19 pts.	Circulating MAIT produced less IFNγ compared to HCs and increase capacity to produce IL17A.	CD69+ MAIT positively correlated with the level of plasmatic IL18. ↑CD69, PD1 MAIT in ETA compared to blood of the same patients.	

Non-Pregnant healthy controls: *NHC*; Genome Sequence Archive: *GSA;* Bronchoalveolar lavage fluid: *BALF*; nasopharyngeal swabs: *NPSs*;