Alliance assessment criteria (<https://rheum-covid.org/>). Information about Disease Modifying Anti-Rheumatic Drug (DMARD) drug use was also recorded. Participants' serum samples were collected for quantitative serological assessment of antibodies to SARS-CoV-2 using the Mologic Enzyme-Linked Immuno-Sorbent Assay (ELISA) IgG kit. The optical density values were plotted against time using a normalisation as previously described (1). A cut-off above 0 indicated positive serology.

Results: A total of 114 subjects were recruited into the study. Subjects were recruited if they had suspected Covid symptoms. Of the population recruited, the total number subsequently testing positive for SARS-CoV-2 was $n=59$ (either on serology or PCR). The number of subjects with autoimmune conditions diagnosed with Covid-19 was 32, with 30 subjects being symptomatic (the asymptomatic 2 subjects were excluded from further analyses). Of the 30 symptomatic subjects, the average age was 56, with a female to male ratio of 19:11. The most prevalent diagnoses were RA (30%), Psoriatic Arthritis (16.7%), SLE (10%), sarcoidosis (10%), Ulcerative Colitis (10%), Crohn's disease (10%), seronegative inflammatory arthritis (6.7%), Sjogren's (3.3%) and Juvenile Idiopathic Arthritis (3.3%). The majority were on csDMARDs (60%), biologic DMARDs (20%), tacrolimus (3.3%) and the remaining 16.7% were not on DMARDs. Most subjects required treatment in hospital (56.7%), a smaller number required high dependency care (6.7%) and the rest were treated at home (36.6%). The majority required no oxygen (63.3%), with a further 30% requiring oxygen and 6.7% needed more supportive care i.e. CPAP/ventilation. We also had a matched control group ($n=29$) of subjects who developed SARS-CoV-2 but had no underlying autoimmune conditions. These subjects had a mean age of 55, female: male ratio 18: 11. Of these 31% had home management, 55.2% had ward level treatments and 13.8% had intensive care treatment. Of the controls, 48.3% did not require oxygen, 37.9% needed simple oxygen and 13.8% were on CPAP. A total of 35 subjects from the original study have attended for follow-up visits. Antibody titres for IgG using the Mologic ELISA detection assay were compared at two visits for the control and autoimmune group. Results showed that subjects with autoimmune conditions and those without who developed SARS-CoV-2 showed very similar antibody titres by optical density (OD) (Figure 1) and maintained antibody responses beyond 6 months in most cases.

Conclusion: Serological assays can assist in the understanding of disease severity of SAR-CoV-2 infection. They can be a useful tool for patient surveillance, especially in people who are on immunomodulatory drugs and are being seen in Rheumatology services. Future work is needed to assess the duration and potential protective nature of humoral antibody responses to SARS-CoV-2.

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Figure 1.

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POS1196

COVID-19 VACCINE WILLINGNESS IN RA? RESULTS FROM THE RA LOUVAIN BRUSSELS COHORT

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Background: Over 2 million deaths from the COVID-19 disease have been reported in the world. Since no antiviral treatment is available, the vaccination is the most important option to fight the SARS-CoV-2 infection. Patients living with rheumatoid arthritis (RA) affecting the immune system or under immunosuppressive agent will be considered as a high risk population and will start

the vaccination shortly. But many patients could decline the vaccine for several reasons including safety concerns.

Objectives: The aim of this study is to evaluate the proportion of patients with RA who are favourable to COVID-19 vaccination, to know the reasons to decline and to analyse the characteristics of these patients.

Methods: We included patients with RA from the UCLouvain Brussels cohort who met the ACR/EULAR 2010 classification criteria. A simple and standard questionnaire was distributed to patients who attended rheumatology out patient and day care clinic from 14 december 20 to 14 januari 21. All patient and RA characteristics (Age, gender, education, smoking habits, disease duration, ACPA, RF, DAS28-CRP, HAQ and therapies) were collected at the same time.

Results: Data from 460 eligible RA patients were analyzed. The average age of the population is 58.21 years. 72% of the patients are women. 21% are smokers and 65% are positive for anti-citrullinated protein antibody (ACPA) with a mean DAS28-CRP of 2.39 and a mean HAQ of 0.821.

281 patients (61%) indicated they would receive the vaccine as soon as it is available. For the 179 patients (39%) who decline, the reasons for not having vaccine were no trust in the vaccine at this time (53%), fear of side effects (28%), opposition to vaccine (4%), previous SARS-CoV-2 infection (2%) and unknown (5%).

Interestingly, there were differences among RA patients not willing to receive the vaccine. Patient under the age of 50, women, low education grade, smokers, presence of RF/ACPA and treatment with a bioDMARD were less willing to receive the vaccine. No differences were observed for RA disease duration, HAQ and DAS28-CRP (see Table 1).

Table 1. Characteristics of RA population and correlation between groups.

	TOTAL n=460	VACCIN-YES n=281 (61%)	VACCIN-NO n=179 (39%)	
Age, mn	58,21 +/-0,71	60,37 +/- 0,9004	54,82 +/- 1,090	p=0.0001*
Group Age <30 yrs	25 (5%)	15 (5%)	10 (6%)	
Group Age =31-50 yrs	112 (24%)	53 (19%)	59 (33%)	
Group Age =51-70 yrs	209 (45%)	127 (45%)	82 (46%)	
Group Age > 70 yrs	114 (25%)	86 (31%)	28 (16%)	p=0.0003**
Gender, F/M, %-F	332/128 (F-72%)	193/88 (F-69%)	139/40 (F-78%)	p=0.043**
Educ0-1	63 (15%)	27 (11%)	36 (22%)	
Educ2-3	349 (85%)	222 (89%)	127 (78%)	p=0.003**
RA Disease duration yrs, mn	14,77 +/-0,51	15,24 +/-0,69	14,04 +/-0,73	
Smokers: YES/NO/	85/248/79 (21%)	42/156/52 (17%)	43/92/27 (27%)	p=0.018**
Ex-Smokers, %-YES				
RF YES/NO, %	243/137 (64%)	133/94 (59%)	110/43 (72%)	p=0.009**
ACPA YES/NO, %	244/132 (65%)	127/93 (58%)	117/39 (75%)	p=0.001**
HAQ, mn	0,814 +/-0,039	0,797 +/-0,051	0,838 +/-0,061	
DAS28-CRP, mn	2,39 +/-0,05	2,35 +/-0,07	2,44 +/-0,09	
GC, YES/NO, %	360/86 (19%)	217/55 (20%)	143/31 (18%)	
csDMARDs (MTX), YES/NO, %	124/322 (72%)	72/200 (74%)	52/122 (70%)	
BioDMARDs, NO/YES, %	151/295 (66%)	107/165 (61%)	44/130 (75%)	p=0.003**

(*) - Unpaired t Test; (**) - Fisher's exact test

Conclusion: In our RA cohort, the rates of willingness to receive the vaccine are promising. Dedicated education and outreach efforts should be developed, especially in some RA subpopulation.

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POS1197

IN DEPTH IDENTIFICATION OF RISK FACTORS FOR SEVERE COVID-19, REQUIRING HOSPITALIZATION, IN PATIENTS WITH INFLAMMATORY RHEUMATIC DISEASES: RESULTS OF A DUTCH NESTED CASE CONTROL STUDY (PRELIMINARY RESULTS)

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Background: Several risk factors for severe COVID-19 have been identified¹. An important question is whether in addition to these generic risk factors, patients with a potentially altered immune response, either by disease (inflammatory rheumatic diseases (IRDs)) or use of immunomodulatory agents (IA), carry a higher or lower risk of severe COVID-19. In addition, several other potential risk factors for severe COVID-19 have been suggested, such as vitamin D status and specific medication (NSAIDs, ACE-inhibitors)².

Objectives: To identify risk factors for severe COVID-19, requiring hospitalization, in patients with IRDs.

Methods: Multicenter, unmatched nested case control study in four rheumatology centers in the Netherlands. Cases are IRD patients requiring hospitalization for COVID-19 between March 1st 2020 and May 31st 2020. Control patients are IRD patients not hospitalized for COVID-19 within this period and were included in a 1:4 ratio. Patient-, disease- and treatment characteristics were extracted (still ongoing) from electronic patient files, and a questionnaire was used to collect additional data (e.g. on behavioral aspects). Potential risk factors for severe COVID-19 were analyzed using unconditional logistic regression, corrected for confounders.

Results: 77 cases and 198 controls were included. 26 case patients died as a result of COVID-19, 14 were admitted to the ICU. Crude ORs for female sex, age, BMI and presence of one or more comorbidities were 0.46 (95% CI 0.27-0.79), 1.1 (95%CI 1.0-1.1), 1.0 (95%CI 0.98-1.1) and 3.0 (95%CI 1.7-5.2) respectively. Table 1 displays corrected OR, highlighted when OR <0.5/>2.0. Corrected ORs were not significantly increased for any IRD, but a trend for gout was present. Use of hydroxychloroquine (HCQ) shows a protective effect, while use of other csDMARDs than methotrexate or HCQ leads to a higher risk of severe COVID-19. Trends were present for increased risk for rituximab (RTX), glucocorticoids and ACE inhibitors, and protective effect for IL-6R blockers.

Conclusion: The outcomes of this study are in agreement with other research regarding risk of severe COVID-19 related to IRDs and use of IA¹. The suggested increased risk of severe COVID-19 in RTX use is of concern and should be monitored closely. Strengths of this study include low bias due to behavioral effects as only cases early in the pandemic were included, completeness of the determinants of interest and ultimately, it provides answers to a relevant and urgent question.

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Table 1. Corrected ORs for several potential risk factors

Potential risk factor	Corrected OR (95% CI)
IRD	
RA*	1.38 (0.68 – 2.8)
Spondylarthritis	0.60 (0.25-1.5)
Gout	2.8 (0.80-9.8)
Polymyalgia	0.54 (0.18-1.6)
Systemic diseases	1.6 (0.49-5.0)
DMARDs	
csDMARDs (any)	0.39 (0.17-0.88)
Methotrexate	0.90 (0.46-1.7)
HCQ	0.40 (0.16-0.98)
Other	2.0 (0.99-4.1)
bDMARDs (any)	0.96 (0.43-2.2)
TNF-inhibitors	1.0 (0.43-2.6)
IL6R-blockers	0.43 (0.044 – 4.2)
RTX	4.5 (0.25-79)
tsDMARDs	0.89 (0.08-9.8)
Other	
Glucocorticoids > 10 mg/day	2.5 (0.70-9.0)
NSAIDs	0.82 (0.37-1.8)
ACE-inhibitors	3.4 (1.6-7.1)
Vitamin D sufficient	0.77 (0.40-1.5)

*incl. unspecified arthritis

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POS1198 THE ROLE OF SOCS3 IN COLCHICINE ANTI-INFLAMMATORY EFFECT ON COVID-19 INFECTION

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Background: In the fight against COVID-19, the use of an old anti-gout agent which is inexpensive and easily administered (1). A meta-analysis suggests a definite benefit with the addition of colchicine in patients with COVID-19(2). It was suggested that colchicine can have efficacy for hospitalized patients with a severe course of COVID-19 and concurrent inflammatory rheumatic diseases who have a higher risk of venous thromboembolism (3). On the other hand, some reports suggested that colchicine is associated with an increased risk of infections (4). Currently, there are nine clinical trials using colchicine for COVID-19(5), nevertheless, the exact mechanism of action is not well understood.

Objectives: Using publicly available transcriptomic data profiling human cells' response to colchicine to identify core differentially expressed genes (DEGs) then examine the dynamics of the identified DEGs in different datasets of COVID-19 transcriptomics databases to suggest the possible mechanisms of action on the molecular level.

Methods: Gene Expression Omnibus was searched for human samples treated with colchicine compared to controls. GSE4090 dataset was identified where global gene expression of HUVEC (human umbilical vein endothelial cell line) cells exposed to various concentrations of colchicine (n=9) compared to controls (n=7). We reanalyzed this dataset using GEO2R online tool to identify DEGs. The identified DEGs were searched in SARS-CoV-2 transcriptome datasets using metascape online tool to identify gene sets where the DEGs identified showed significant similarity.

Results: Fifty DEGs were identified in response to colchicine no matter what the concentration is used, (Figure 1A). Two gene sets (COVID323, RNA_Wilk_CD4+T-cells_patient-C2_Up [peripheral immune cells] and COVID008, RNA_Blanco-Melo_A549_Up[lung epithelial cells]) showed significant enrichment of the identified DEGs, (Figure 1B). SOCS3, ZC3H12C, CD55, GLIPR1, BACH1, PHLD1, JUNB, NFKB1, and MYLIP were the shared genes between the identified DEGs, and the two gene sets with top enriched terms related to important immune-related pathways like IL-6, chemotaxis, leukocytes migration, cytokine release, and response to glucocorticoid, (Figure 1C). Only Suppressor of cytokine signaling 3 (SOCS3) was shared in the three datasets. SOCS3 expression was significantly downregulated in the HUVEC cells exposed to colchicine, (Figure 1D). Next, the normalized gene expression of SOCS3 from whole blood of 62 COVID-19 patients was compared to that of 24 healthy controls using (GSE15264) showed significant upregulation of SOCS3 in the blood of COVID-19 patients indicating its role in COVID-19, (Figure 1E).

Figure 1-4: Volcano plot showing DEGs between GSE4090 dataset and GSE15264 dataset. Panel 1-A shows a volcano plot of DEGs. Panel 1-B shows a Venn diagram of gene sets. Panel 1-C shows a heatmap of enriched terms. Panel 1-D shows a bar chart of SOCS3 normalized gene expression. Panel 1-E shows a scatter plot of SOCS3 normalized gene expression in COVID-19 patients vs controls.

Conclusion: Our reanalysis showed that the possible beneficial effect of colchicine in COVID-19 patients might be through its direct effect on SOCS3 which is specifically upregulated in COVID-19 by IL6 and interferon (IFN)-gamma.

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