Graph showing disease severity on admission by Race/Ethnicity (upper). Notice the predominance of severe disease (orange) in Hispanic patients. Graph showing Race/Ethnicity Distribution by Week (lower). Notice the gradual increase and predominance of Hispanic patients (orange) in the later weeks of the study period compared to Black (blue) and White (green) patients.

Disclosures. **All Authors**: No reported disclosures

289. Post COVID Syndrome Cohort Characterization

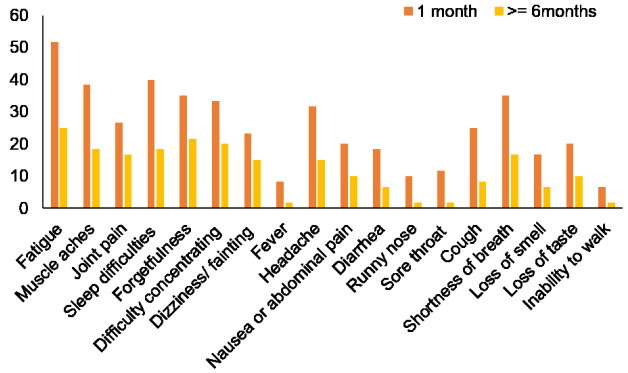
Bhoomija Chatwani, MPH, BDS[1]; Shelby Flaherty, BS[1]; Sharon Liu, MS, BS[2]; Marc Theberge, BS[2]; Mark Zeller, PhD[3]; Kristian Anderson, PhD[3]; Matt Boisen, PhD[4]; Luis Branco, PhD[4]; Robert Garry, PhD[2]; Arnaud Drouin, MD, PhD[2]; Dahlene Fusco, MD, PhD[2]; [1]Tulane School of Public Health and Tropical Medicine, New Orleans, Louisiana; [2]Tulane School of Medicine, New Orleans, Louisiana; [3]The Scripps Research Institute, San Diago, California; [4]Zalgen Labs, Germantown, Maryland

Session: P-14. COVID-19 Complications, Co-infections, and Clinical Outcomes

Background. Post COVID Syndrome (PCS) is significant morbidity following COVID-19. This study aims to identify biomarkers that predict PCS in a Gulf Coast cohort known for poor health outcomes.

Methods. Since March 2020 the study Collection of Serum and Secretions for SARS CoV-2 Countermeasure Development (aka ClinSeqSer) has been enrolling subjects with confirmed acute COVID-19, with initial visit at 1 month and follow up every three months from symptom onset. At follow-up, subjects complete symptom questionnaire, physical examination, nasopharyngeal swab/saliva collection, blood draw. Subjects with >= one symptom new since COVID are PCS, remainder are Non-PCS experienced at initial one month visit and six months or longer. Univariate and bivariate analysis was carried out to study significant associations of currently available dataset (N=60).

Figure 1. Post-COVID Symptoms



Included if "new since covid". For 60 subjects consented post-covid with completed questionnaire, results were analyzed. Most common symptoms reported were fatigue/tiredness or exhaustion (52%), muscle aches (38%), difficulty concentrating (33%) and headache (32%) as the most common symptoms during one month prior to their initial follow-up visit. The persistent symptoms experienced for six months or longer were fatigue/tiredness or exhaustion (25%), forgetfulness (22%), muscle aches (18%), and sleep difficulties (18%).

Results. Cohort is 36 (60%) female, 24 (40%) male, age group of 49 (82%) 18-64 years, 11 (18%) 65+ years, 33 (55%) African American, 27 (45%) Caucasian. Median follow-up time after symptom onset: 290 days. Study cohort reported fatigue (52%), myalgias (38%), difficulty concentrating (33%), headache (32%) as most common symptoms during first month from initial symptom onset. Persistent symptoms (>=6 months) are fatigue (25%), forgetfulness (22%), myalgias (18%), sleep difficulties (18%). Bivariate analysis shows that gender (female, P=0.04), past stroke/transient ischemic attack (P=0.04), deep venous thrombosis (P=0.02), abnormal kidney function (P=0.01) associate with PCS. Convalescent antibodies (ReSARS N IgG, S-RBD IgG) were measured and percentage inhibition of ACE2 spike interaction was recorded. Plasma inflammatory protein levels were measured using multiplex ELISA and Proximity Extension Assay technology during follow-up visit. Increased antibody ReSARS N IgG (2.91, 0.74-10.93; P=0.02) response and higher convalescent IL-10 (P=0.04) was associated with PCS. Percent inhibition of ACE2: spike interaction was not associated (P=0.79) with PCS. Nasal swab/saliva SARS-COV-2 sequencing has not identified a specific SARS-CoV-2 virus mutation predictive of PCS.

Table 1. Demographic and Clinical Characteristics

Variables	PCS N (%)	Non-PCS N (%)	OR (95% CI)	p-value
Age groups (years), 18-64	39 (65.00)	10 (16.67)	0.45 (0.11, 1.84)	0.2638
65+	7 (11.67)	4 (6.67)		
Gender, female	31 (51.67)	5 (8.33)	0.27 (0.08, 0.94)	0.0354
male	15 (25.00)	9 (15.00)		
Race, black	24 (40.00)	9 (15.00)	1.65 (0.48, 5.68)	0.4219
white	22 (36.67)	5 (8.33)		
Ethnicity, non-Hispanic	44 (73.33)	14 (23.33)	1.05 (0.98, 1.11)	1
Hispanic	2 (3.33)	0 (0)		
Clinical severity during hospitalization,				
Severe	5 (8.33)	1 (1.69)	1.53 (0.16, 14.51)	1
Non-severe	36 (67.92)	11 (20.75)		
Stroke or Transient ischemic attack	1 (1.69)	3 (5.08)	0.08 (0.01, 0.88)	0.0382
Heart attack	1 (1.69)	1 (1.69)	0.29 (0.02, 5.06)	0.4212
Chest pain from narrow heart vessels	3 (5.08)	4 (6.67)	0.18 (0.03, 0.93)	0.0479
Blood clot in lung (pulmonary embolism)	4 (6.67)	2 (3.33)	0.59 (0.1, 3.6)	0.6204
Blood clot in leg (deep venous thrombosis)	2 (3.33)	4 (6.67)	0.12 (0.02, 0.73)	0.0241
Other blood clots	1 (1.69)	1 (1.69)	0.3 (0.02, 5.06)	0.4214
Abnormal kidney function	3 (5.08)	5 (8.33)	0.13 (0.03, 0.64)	0.0142
Abnormal lung function	9 (15.25)	1 (1.69)	0.63 (0.16, 2.46)	0.4849
Abnormal liver function	0	0	.	.
Abnormal heart function	12 (20.69)	4 (6.67)	0.94 (0.25, 3.57)	1
High blood pressure (hypertension)	20 (34.48)	9 (15.52)	0.46 (0.13, 1.6)	0.2172
Diabetes	9 (15.52)	6 (10.34)	0.34 (0.09, 1.24)	0.1582

The bivariate analysis results showed that the gender (female, P=0.0354), history of stroke or transient ischemic attack (P=0.0382), chest pain from narrow heart vessels (P=0.0479), deep venous thrombosis (P=0.0241) and abnormal kidney function (P=0.0142) were associated with Post-COVID syndrome.

Table 2. Antibodies and ACE2 spike inhibition.

Variables	Mean	Median	Upper Quartile	Lower Quartile	Std Dev	P-value
N IgG (U/mL)	8.68	2.91	10.93	0.74	12.31	0.0159
S-RBD (U/mL)	9.72	7.1	15.74	3.48	8.05	0.3076
ACE2 spike %inhibition	42.26	21	100	5	46.31	0.7932

The convalescent antibodies, ReSARS N IgG and S-RBD IgG were measured in U/mL and percentage inhibition of ACE2 spike interaction was recorded during follow-up visit for PCS vs Non-PCS subjects. The increased antibody ReSARS N IgG (2.91, 0.74-10.93; P=0.0159) response was associated with Post-COVID syndrome. Percent inhibition of ACE2: spike interaction was not associated (P=0.7932) with PCS.

Table 3. Plasma inflammatory protein levels.

Variables	Mean	Median	Upper Quartile	Lower Quartile	Std Dev	P-value
GM-CSF	0.08	0.08	0.1	0.06	0.03	0.974
IFN	3.38	3.25	4.35	2.19	1.7	0.3622
IL-10	0.23	0.15	0.2	0.1	0.32	0.0379
IL-1ß	0.11	0.1	0.17	0.05	0.07	0.9277
IL-5	2.53	0.33	0.8	0.19	7.6	0.8993
IL-6	5.9	1.62	2.66	0.9	18.58	0.4183
IL-8	5.93	3.9	5.66	2.89	6.49	0.4442
TNFa	0.62	0.54	0.64	0.43	0.35	0.6283
G-CSF	10.06	8.77	11.75	5.85	5.5	0.5738
IFN-a2a	0.7	0.68	0.94	0.39	0.44	0.814
IL-1RA	209.28	128.43	231.03	102.68	169.83	0.6171
IL-7	4.01	2.63	7.3	1.55	2.73	0.5483
IL-9	0.32	0.14	0.3	0.1	0.49	0.2338
IP-10	236.28	192.47	267.4	159.25	110.79	0.2222
MCP-1	141.33	93.23	147.51	80.02	163.67	0.9306
MIP-1a	13.5	12.91	15.12	10.28	6.25	0.7727
VEGF-A	24.01	21.16	31.45	10.69	16.7	0.643
IL8	5.52	5.47	5.76	5.24	0.42	0.7019
VEGFA	11.06	11.08	11.39	10.25	0.79	0.2842
CD8A	9.49	9.99	10.03	8.95	0.87	0.5618
MCP-3	2.56	2.68	3.16	1.63	0.83	0.2542
GDNF	2.42	2.39	2.73	2.29	0.45	0.7895
CDCP1	3.01	3.18	3.7	2.07	0.82	0.2463
CD244	7.74	7.11	8.38	6.83	1.35	0.5745
IL7	2.65	2.2	3.01	1.98	1.02	0.5083
OPG	10.01	10.14	10.62	9.31	0.7	0.3195
LAP TGF-beta-1	6.68	6.23	7.01	5.9	1.17	0.484
uPA	9.5	9.53	9.95	9.12	0.47	0.8069
IL6	3.06	3.12	4.33	1.68	1.22	0.2531
IL-17C	2.82	2.86	3.57	2.29	0.81	0.5938
MCP-12	11.08	11.16	11.27	10.58	0.5	0.941
IL-17A	2.03	2	2.16	1.54	0.57	0.8425
CXCL11	9.23	9.59	10.51	7.71	1.47	0.307
AXIN1	6.47	5.89	7.31	5.7	1.25	0.566
TRAIL	7.73	7.71	7.81	7.58	0.23	0.696
IL-20RA	1.45	1.42	1.98	0.97	0.51	0.9893
CXCL9	6.26	6.06	7.18	5.59	0.85	0.5752
CST5	6.31	6.27	6.83	5.71	0.64	0.129
IL-2RB	1.26	1.29	1.36	1.09	0.18	0.1623
IL-1 alpha	-0.38	-0.41	-0.17	-0.56	0.22	0.9527
OSM	4.1	4.13	5.38	2.66	1.5	0.3151
IL2	1.4	1.6	1.65	1.09	0.43	0.6494
CXCL1	10.82	10.53	10.84	10.14	1.12	0.5701
TSLP	1.6	1.6	1.71	1.56	0.16	0.8456
CCL4	5.48	5.33	6.14	5	0.67	0.2239
CD6	5.87	5.57	5.99	5.12	1.16	0.6369
SCF	9.05	8.97	9.47	8.84	0.37	0.5889
IL18	8.56	8.52	8.95	8.25	0.46	0.6481
SLAMF1	2.1	2.15	2.32	1.93	0.38	0.6889
TGF-alpha	2.28	2.39	2.73	1.87	0.67	0.5637
MCP-4	14.46	13.97	14.96	13.93	0.98	0.4464
CCL11	7.45	7.6	7.88	6.84	0.52	0.426
TNFSF14	4.96	4.52	5.58	4.2	1.25	0.5634
FGF-23	2.74	2.19	3.45	2.09	1.33	0.7107
IL-10RA	1.3	1.09	1.88	0.81	0.64	0.9469
FGF-5	0.95	0.98	1.13	0.76	0.26	0.5194
MMP-1	14.22	14.31	15.14	12.83	1.34	0.3021
LIF-R	3.5	3.41	3.9	3.16	0.38	0.3917
FGF-21	5.43	5.89	6.51	4.78	1.41	0.4628
CCL19	9.44	9.73	10.21	8.53	0.97	0.1236
IL-15RA	1.12	1.08	1.27	1.03	0.37	0.7038
IL-10RB	5.23	5.2	5.58	4.93	0.39	0.9675
IL-22 RA1	1.97	1.92	2.17	1.83	0.39	0.7964
IL-18R1	8.13	8.12	8.98	7.24	0.84	0.2926
PD-L1	7.37	7.17	8	6.51	1.12	0.2833
Beta-NGF	0.06	0.06	0.11	-0.03	0.09	0.358
CXCL5	12.44	12.17	12.82	12.03	0.76	0.3167
TRANCE	4.3	4.2	4.6	4.08	0.33	0.2192
HGF	8.37	8.35	8.99	7.77	0.77	0.4553
IL-12B	5.58	5.53	6.47	5.02	1.07	0.6191
IL-24	1.23	1.25	1.75	0.81	0.55	0.4593
IL13	1.3	1.29	1.49	1.18	0.18	0.5084
ARTN	1.63	1.38	1.61	1.22	0.81	0.6334
MMP-10	8.58	8.59	9.01	8.14	0.5	0.8144
IL10	1.72	1.71	1.85	1.51	0.28	0.4737
TNF	2.33	2.44	2.71	1.75	0.54	0.2815
CCL23	9.72	9.77	9.98	9.04	0.72	0.9641
CD5	6.27	5.78	6.36	5.39	1.47	0.5764
CCL3	6.08	6.03	7.53	4.42	1.57	0.2924
Flt3L	8.02	7.89	8.21	7.71	0.42	0.7278
CXCL6	9.23	9.07	9.66	8.58	0.96	0.2568
CXCL10	9.45	9.4	10.64	7.87	1.72	0.3737
4E-BP1	8.15	8.02	9.56	6.71	1.73	0.2906
IL-20	0.63	0.66	0.87	0.59	0.3	0.9965
SIRT2	5.81	5.12	7.26	4.36	1.8	0.4367
CCL28	2.78	2.55	2.74	2.42	0.64	0.9569
DNER	8.28	8.31	8.52	7.99	0.44	0.6116
EN-RAGE	4.17	4.15	5.55	2.73	1.52	0.353
CD40	13.68	13.55	14.03	13.51	0.4	0.2
IL33	1.1	1.26	1.34	0.71	0.42	0.7321
IFN-gamma	7.03	6.94	8.83	5.78	1.69	0.4806
FGF-19	8.12	7.98	8.67	7.61	0.95	0.0783
IL4	0.25	0.24	0.44	0.02	0.27	0.579
LIF	0.57	0.63	0.71	0.19	0.49	0.0685
NRTN	0.82	0.82	0.98	0.69	0.24	0.9964
MCP-2	9.28	8.99	10.28	8.52	1.12	0.5541
CASP-8	5.5	5.27	5.51	5.07	0.9	0.8294
CCL25	5.34	5.21	6	4.97	0.73	0.9661
CX3CL1	3.32	3.52	3.77	2.55	0.68	0.6254
TNFRSF9	6.11	6.21	6.58	5.63	0.5	0.7228
NT-3	2.19	2.21	2.42	1.97	0.28	0.4088
TWEAK	8.3	8.26	8.68	7.95	0.43	0.3873
CCL20	7.99	8.13	8.59	7.61	0.74	0.6326
ST1A1	6.5	6.49	6.81	6.33	0.48	0.067
STAMBP	6.84	6.36	7.96	5.63	1.53	0.4506
IL5	1.6	1.69	1.89	1.27	0.43	0.6713
ADA	5.52	5.42	5.94	4.81	0.75	0.3548
TNFB	4.45	4.34	4.91	4.15	0.4	0.9042
CSF-1	9.61	9.6	9.87	9.38	0.48	0.6572

Plasma inflammatory protein levels were measured using multiplex ELISA (MSD) and Proximity Extension Assay technology (Olink) recorded during follow-up visit for PCS vs Non-PCS subjects, revealing IL-10 (P=0.0379) was associated with development of PCS.

Conclusion. This study identifies initial clinical and biomarker predictors of PCS in a cohort that is 55% African American.

Figure 2. Antibody ReSARS N IgG

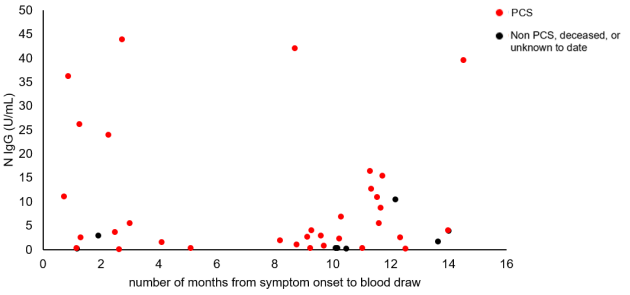
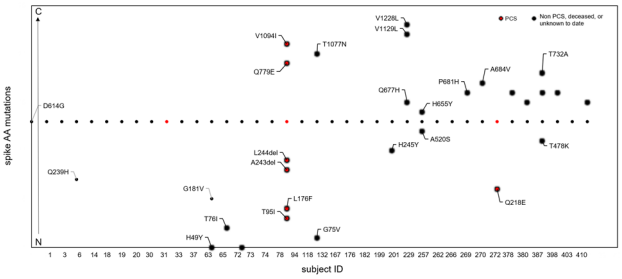


Figure 2. ReSARS N IgG measured in post-covid patients is significantly associated with post-COVID syndrome(P=0.0159). X axis: number of months from symptom onset to blood draw. Y axis: N IgG U/mL.

ReSARS N IgG measured in post-covid patients is significantly associated with post-COVID syndrome(P=0.0159). X axis: number of months from symptom onset to blood draw. Y axis: N IgG U/mL.

Figure 3. Spike amino acid mutations



Spike amino acid mutations detected in SARS-CoV-2 from acute-phase respiratory isolates. Nasal swab/saliva samples were collected from subjects with acute COVID-19 at time of enrollment into ClinSeqSer, stored at -80°C followed by RNA isolation and SARS-CoV-2 qRT-PCR. Samples with Ct value of ≤30 were then sequenced using NextSeq (Illumina). All sequences are deposited on GISAID and under BioProject (ID PRJNA681020). X axis: subject ID, with ID number increasing chronologically. Y axis: amino acid position of each mutation moving from N- to C-terminus.

Disclosures. **Robert Garry, PhD**, **Zalgen Labs** (Shareholder)

290. Persistence of Long COVID in SARS-CoV-2 Confirmed Cases One-Year Post Infection

Harrison L. Howe, BS[1]; Danielle A. Rankin, MPH, CIC[2]; Sean M. Bloos, MPH[1]; Kailee N. Fernandez, BS[2]; Seifein Salib, MD[1]; Rana Talj, MD[1]; Danya Waqfi, MD[1]; Jessica Villarreal, BS[1]; Ahmad Yanis, MD[1]; James Chappell, MD, PhD[1]; Leigh Howard, MD, MPH[1]; Natasha B. Halasa, MD, MPH[1]; Natasha B. Halasa, MD, MPH[1]; [1]Vanderbilt University Medical Center, Goodlettsville, Tennessee; [2]Vanderbilt University Medical Center; Division of Pediatric Infectious Diseases, Nashville, TN

Session: P-14. COVID-19 Complications, Co-infections, and Clinical Outcomes

Background. Regardless of severity of acute SARS-CoV-2 illness, adults infected with SARS-CoV-2 are at risk for post-acute sequelae of COVID-19. Long COVID is typically classified as symptoms lasting greater than four weeks post-infection. We aimed to evaluate the frequency of resolved and unresolved long COVID symptoms in adults residing in greater Nashville, TN.

Methods. We conducted a longitudinal cohort study of SARS-CoV-2-positive and exposed individuals from March 20 to May 15, 2020. Participants for this analysis were included if: 1) ≥18 years; 2) SARS-CoV-2 positive by molecular or antibody testing; and 3) completed a one-year visit. Demographic and illness information were collected at enrollment, and long COVID symptoms were systematically collected at the one-year survey. Long COVID symptoms are defined as an adult experiencing at least one of the following symptoms four weeks post-infection: fatigue, confusion, loss of smell or taste, shortness of breath, chest pain, cough, muscle aches, inability to exercise, or heart palpitations. Unresolved symptoms are defined as an individual with long COVID still experiencing symptoms at the one-year visit.

Results. A total of 115 adults enrolled and completed the one-year survey, of which 63 (54.8%) were SARS-CoV-2-positive, with one asymptomatic individual. Of SARS-CoV-2-positive symptomatic adults, 32 (51%) were female, 5 (88%) were of Hispanic ethnicity, and 58 (92%) were white. At the one-year visit, 33 (52%)