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Background. African Americans (AA) and Latinos, compared with Whites, experience disproportionately higher rates of morbidity and mortality in COVID-19. Exuberant inflammatory responses may explain, in part, the differences in disease severity in COVID-19 observed among different demographic groups.

Methods. In a retrospective cohort study, we analyzed data from patients aged ≥18 years hospitalized for COVID-19 (confirmed by positive SARS-CoV-2 PCR) from 3/1/2020 – 12/31/2020 at Emory Healthcare hospitals. Patient demographics, clinical characteristics, and peak levels of high-sensitivity C-reactive protein (hs-CRP) during hospitalization were abstracted from electronic medical record. Comorbidity burden was defined as the number of six total comorbidities assessed per patient. Multivariable logistic regression (adjusted for age, sex, body mass index [BMI], smoking status) assessed the effects of race and comorbidity burden on peak hs-CRP level.

Results. 3,860 patients, median age 60 [18-108] years, 51% female, 57% AA, 28% White, 6% Latino and 9% other races were enrolled. Median comorbidity burden per patient was 2 (Q1-Q3, 1-3), with prevalent comorbidities distributed as follows: 68% had hypertension, 43% renal disease, 42% diabetes, 16% cardiovascular disease, 12% lung disease, and 5% cancer. Unadjusted peak hs-CRP (mg/L) levels were highest among Latino patients (144.9) followed by other races (137), AA (130.3), and Whites (122.2). In adjusted models (including race), the mean difference in peak hs-CRP (mg/L) compared with patients who had no comorbidities was 18.7 (p=0.108), 56.7 (p< 0.001), and 78.2 mg/L (p< 0.001) for 1, 2, and ≥3 comorbidities, respectively. In adjusted models (including comorbidity burden), the mean level of peak hs-CRP, compared with Whites, was 34.2 (p< 0.001), 38.4 (p=0.003), and 36.0 mg/L (p=0.06) higher in AA, Latinos, and other races, respectively.

Conclusion. Among patients hospitalized with COVID-19, non-White race and comorbidity burden were associated with significantly higher levels of inflammation. These findings suggest that exuberant inflammatory responses may be driving, in part, the differences in COVID-19 disease severity observed across different demographic groups.

Disclosures. **Lauren F. Collins, MD, MSc**, Nothing to disclose

442. Sex-Related Differences in Mortality from COVID-19: Survival Analysis of Patients from an Urban Hospital

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Background. Mortality from COVID-19 is associated with male sex, older age, black race, and comorbidities including obesity. Our study identified risk factors for in-hospital mortality from COVID-19 using survival analysis at an urban center in Detroit, MI.

Methods. This was a single-center historical cohort study. We reviewed the electronic medical records of patients positive for severe acute respiratory syndrome coronavirus 2 (the COVID-19 virus) on qualitative polymerase-chain-reaction assay, who were admitted between 3/8-6/14/20. We assessed risk factors for mortality using Kaplan-Meier analysis and Cox proportional hazards models.

Results. We included 565 patients with mean age (standard deviation) 64.4 (16.2) years, 52.0% male (294) and 77.2% (436) black/African American. The overall mean body mass index (BMI) was 32.0 (9.02) kg/m^2. At least one comorbidity was present in 95.2% (538) of patients. The overall case-fatality rate was 30.4% (172/565). The unadjusted mortality rate among males was 33.7% compared to 26.9% in females (p=0.08); the median time to death (range) for males was 16.8 (0.3, 33.9) compared to 14.2 (0.32, 47.7) days for females (p=0.04). Univariable survival analysis with Cox proportional hazards models revealed that age (p=< 0.0001), admission from a facility (p=0.002), public insurance (p< 0.0001), respiratory rate ≥ 22 bpm (p=0.02), lymphocytopenia (p=0.07) and serum albumin (p=0.007) were additional risk factors for mortality (Table 1). From multivariable Cox proportional hazards modeling (Table 2), after controlling for age, Charlson score and qSofa, males were 40% more likely to die than females (p=0.03).

Table 1. Univariate analysis with Cox proportional hazards model on factors associated with mortality in patients with COVID-19

Variables	HR (95% CI)	*p* value
Age ≥ 60 yrs.	2.4 (1.6, 3.4)	<0.0001
Male Sex	1.4 (1.02, 1.9)	0.04
Race	0.74 (0.5, 1.01)	0.06
Nursing facility	1.6 (1.6, 4.5)	0.003
Public Insurance	2.7 (1.6, 4.5)	<0.0001
Obesity	0.75 (0.6, 1.01)	0.06
Respiratory Rate ≥ 22 breaths per minute	1.5 (1.07, 2.0)	0.02
Lymphocytopenia on hospital admission	1.3 (1.0, 1.8)	0.07
Serum albumin (<3.5 gm/dl)	1.6 (1.12, 2.2)	0.008

Abbreviations: HR: Hazard ratio, CI: Confidence interval

Table 2. Multivariable analysis with Cox proportional hazards model on factors associated with mortality in patients with COVID-19

Variables	HR (95% CI)	*p* value
Age	1.03 (1.02, 1.04)	<0.0001
Male Sex	1.4 (1.03, 1.9)	0.03
CWIC at hospital admission	1.3 (1.04, 1.18)	0.002
qSOFA at hospital admission	1.3 (1.1, 1.6)	0.006

Abbreviations: HR: Hazard ratio, CI: Confidence interval, CWIC: Charlson weighted index of comorbidity, qSOFA: Quick sepsis related organ failure assessment

Conclusion. After controlling for risk factors for mortality including age, comorbidity and sepsis-related organ failure assessment, males continued to have a higher hazard of death. These demographic and clinical factors may help healthcare providers identify risk factors from COVID-19.

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443. Pre-vaccination Antibody Titers Against Seasonal Coronaviruses And Antibody Responses to the Pfizer-BioNTech BNT162b2 COVID-19 mRNA Vaccine in Healthcare Workers

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Background. The Prospective Assessment of SARS-CoV-2 Seroconversion (PASS) study is following over 200 healthcare workers who have received the Pfizer-BioNTech BNT162b2 COVID-19 mRNA vaccine. A major aim of the study is to determine whether baseline antibody titers against the seasonal human coronaviruses are associated with altered levels of vaccine-induced antibody responses to SARS-CoV-2.

Methods. Serial serum samples obtained pre-vaccination and 1 month after the second dose were tested for IgG antibodies against the full pre-fusion spike protein and the receptor binding domain (RBD) of SARS-CoV-2, as well as the full pre-fusion spike proteins of OC43, HKU1, 229E, and NL63. Antibodies were measured using highly sensitive and specific multiplex assays based on Luminex-xMAP technology.

Results. Preliminary analyses of the first 103 subjects in whom we have 1 month post-vaccination serum demonstrate development of high IgG geometric mean titers (GMT) to both the full spike protein (GMT: 13,685, 12,014-15,589, 95% CI) and the RBD (GMT: 19,448, 17,264-21,908, 95% CI) of SARS-CoV-2 after the 2nd vaccine dose. Preliminary analysis demonstrates no association between baseline antibody titers against spike protein of OC43 and antibody titers against SARS-CoV-2 spike protein (Pearson's r-value= 0.13, *P*-value= 0.21) or RBD (Pearson's r-value= 0.09, *P*-value= 0.36) one month after vaccination. Future analyses will evaluate whether there is an association with baseline seasonal coronavirus antibody titers and either SARS-CoV-2 neutralization titers or anti-SARS-CoV-2 spike protein titers at 6 months after vaccination.

Conclusion. These preliminary results suggest that baseline antibody responses to seasonal coronaviruses neither boost nor impede SARS-CoV-2 vaccine-induced antibody responses. Longitudinal sampling will enable assessment of vaccine durability and determination of whether baseline seasonal coronavirus antibody levels are associated with altered duration of detectable COVID-19 vaccine-induced antibody responses.

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444. County-level COVID-19 Case Fatality Rate in Medicaid Expansion States Compared to Non-Expansion States

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Background. Medicaid expansion has been adopted by 38 states and the District of Columbia,[1,2] contributing to lower rates of uninsured individuals in the US.[3] During

the COVID-19 pandemic, Medicaid enrollment offset employer-based insurance losses precipitated by the recession.[4] The aim of this study was to evaluate whether Medicaid expansion may have impacted COVID-19 mortality.

Methods. We conducted an ecologic study that included all US counties in the 50 states and District of Columbia. County-specific Medicaid expansion status was based on whether expansion was adopted within the state. COVID-19 cases and deaths for each county were obtained from the Centers of Disease Control (CDC). Unadjusted and multivariable negative binomial regression with robust standard errors to account for clustering of counties within each state were used to evaluate the association of COVID-19 case fatality rate and Medicaid expansion status. Adjusted models included the addition of four sets of county-level covariates thought to influence the association of Medicaid status and COVID-19 fatality rate: demographics, comorbidities, economic indicators, and physician density. These analyses were then performed in subgroups of counties defined by urbanicity (metro, suburban or rural) and quartiles of poverty rates. Incidence Rate Ratios (IRR) and 95% confidence intervals (CI) are reported.

Results. A total of 1,814 Medicaid expansion and 1,328 non-expansion counties were included in the analysis. Crude case fatality rates were 2.1% (non-expansion) and 1.8% (expansion). Medicaid expansion was not associated with a significantly lower COVID-19 case fatality rate in either the unadjusted (IRR: 0.86; 95% CI: 0.74, 1.01) or fully adjusted (IRR: 1.02; 95% CI: 0.90, 1.16) models. In adjusted models, Medicaid expansion status was also not associated with differences in COVID-19 case fatality rate when counties were stratified by either urbanicity or percent of individuals living below the poverty line.

Table 1 Incidence Rate Ratios of Expansion Counties Compared to Non-Expansion Counties in Unadjusted and Adjusted Models

Analysis	Number of Counties	Unadjusted Model	Adjusted for Demographics[a]	Adjusted for Demograhics & Comorbidities[b]	Adjusted for Demographics, Comorbidities, & Economic Indicators[c]	Adjusted for Demographics, Comorbidities, Economic Indicators & Physician Density[d]
All Counties[e]	3139	0.86 (0.74, 1.01)	0.97 (0.85, 1.10)	1.02 (0.90, 1.15)	1.02 (0.90, 1.16)	1.02 (0.90, 1.16)
Metropolitan Counties	436	1.09 (0.94, 1.27)	1.09 (0.95, 1.25)	1.17 (1.04, 1.33)	1.08 (0.95, 1.23)	1.08 (0.95, 1.23)
Suburban Counties	729	0.91 (0.77, 1.06)	1.04 (0.92, 1.19)	1.08 (0.96, 1.22)	1.06 (0.93, 1.20)	1.06 (0.93, 1.20)
Rural Counties	1974	0.82 (0.69, 0.98)	0.92 (0.78, 1.07)	0.95 (0.82, 1.10)	0.97 (0.83, 1.14)	0.98 (0.84, 1.14)
Poverty 4th Quartile[f]	786	0.82 (0.68, 1.00)	0.94 (0.80, 1.10)	0.95 (0.81, 1.11)	N/A	0.95 (0.81, 1.11)
Poverty 3rd Quartile[f]	785	0.94 (0.78, 1.13)	0.98 (0.85, 1.14)	1.02 (0.89, 1.17)	N/A	1.03 (0.89, 1.18)
Poverty 2nd Quartile[f]	785	1.01 (0.85, 1.20)	1.03 (0.86, 1.22)	1.10 (0.96, 1.26)	N/A	1.10 (0.96, 1.26)
Poverty 1st Quartile[f]	783	0.90 (0.72, 1.14)	0.97 (0.82, 1.15)	1.04 (0.90, 1.21)	N/A	1.04 (0.90, 1.21)

[a] Adjusted for county demographics: population, age distribution, percent of male, Black, and Hispanic residents.
[b] Adjusted for county demographics and comorbidities: prevalence of diabetes, obesity, chronic kidney disease, coronary artery disease, and chronic obstructive disease.
[c] Adjusted for county demographics, comorbidities, and economic indicators: median household income, mean percent unemployed in 2020, and percent living in poverty. The economic indicators were not included in this model for the sub-analyses stratified by poverty quartiles.
[d] Adjusted for county demographics, comorbidities, economic indicators, and number of physicians per 100,000 residents. The economic indicators were not included in this model for the sub-analyses stratified by poverty quartiles.
[e] Three small counties with 0 COVID-19 cases were excluded from all analyses
[f] Poverty Quartiles: Percent of county residents living under the federal poverty level:
1st Quartile: <10.99%; 2nd Quartile: 10.99%-14.77%; 3rd Quartile: 14.78%-19.08%; 4th Quartile: >19.08%

Conclusion. In this county-level analysis, Medicaid expansion status was not associated with a significant difference in county-level COVID-19-related case fatality rates among people of all ages. Future individual-level studies are needed to better characterize the effect of Medicaid on COVID-19 mortality.

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445. COVID-19 Pharmacotherapy Was Not Associated with Mortality in a Community Teaching System

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Background. During the COVID-19 pandemic, a task force was assembled to collect data on patient characteristics and treatment exposures to assess what factors may contribute to patient outcomes, and to help develop institutional treatment guidelines.

Methods. A retrospective study was performed on COVID-19 inpatient admissions within a four-hospital community health system over a six-month period from April-October 2020. Positive COVID-19 immunology results and/in conjunction with an inpatient admission was criteria for inclusion. Covariates for age, gender, race were added *apriori*. Covariates of interest included baseline comorbidities, admission level-of-care, vital signs, mortality outcomes, need for intubation, and specific pharmacological treatment exposures. Logistic regression was performed on our final model and reported as OR +/- 95% CI.

Results. A total of 349 patients met inclusion criteria. Pharmacotherapies were not associated with a difference in mortality in a four-hospital system. Corticosteroids (p = 0.99); Remdesivir (p = 0.79); hyrdroxychloroquine (p = 0.32); tocilizumab (p = 0.91); were not associated with mortality. ACE-inhibitor or angiotensin II receptor blockers OR 0.29 (0.09-0.93) (p = 0.03); convalescent plasma OR 7.85 (1.47-42.1) (p = 0.02); neuromuscular blocking agents (NMBA) OR 5.51 (1.28-23.8) (p = 0.02); vasopressors OR 17.6 (5.62-54.9) (p = 0.00) were associated with in-hospital mortality. Covariates that were associated with a difference in mortality were: age > 60 years OR 2.73 (1.04-7.14) (p = 0.04); structural lung disease OR 3.02 (1.28-7.10) (p = 0.01). Covariates not associated with mortality included African American race (p = 0.30); critical care admission (p = 0.19); obesity (p = 0.06); cardiovascular disease (p = 0.89); diabetes (p = 0.28).

Conclusion. The use of corticosteroids, remdesivir, tocilizumab, and hydroxychloroquine, and admission to a critical care bed was not associated with a difference of in-hospital mortality. Patients who required vasopressors or NMBA were associated with in-hospital mortality. Despite national trends reporting increased mortality in patients with obesity, diabetes, cardiovascular disease, and of African American race, this was not observed in our health system safety net hospitals.

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446. Prognostic Value of Absolute Lymphocyte Count for Disease Severity and Clinical Outcomes in Adult COVID-19 Inpatients

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Background. Lymphopenia has been reported as a relatively frequent finding in patients with coronavirus disease 2019 (COVID-19). This study aimed to assess the use of absolute lymphocyte count (ALC) as a prognostic biomarker for disease severity and clinical outcomes.

Methods. A cohort of adult patients with COVID-19 admitted to Memorial Healthcare System, Hollywood, Florida from March 7, 2020 to January 18, 2021 was retrospectively analyzed. An absolute lymphocyte count (ALC) < 1.1×10^9/L was used as cutoff point to define lymphopenia. Correlations of ALC upon admission with age and serum levels of C-reactive protein, interleukin-6, lactate dehydrogenase, and creatinine were analyzed. Univariate and multivariate regression models were developed to assess the association of lymphopenia with the risk of ICU admission and clinical outcomes.

Results. 4,485 hospitalized patients were included in the final analyses. Median age was 61 (interquartile range, 47-73) years and 2,311 (51.5%) were men. Lymphopenia was more frequent in patients admitted to the ICU compared to those that were not admitted to the ICU, with an odds ratio of 2.14 (95% confidence interval [CI], 1.78-2.56, p < .0001) (Figure 1). The actual value of the ALC was negatively correlated with age and serum levels of C-reactive protein, interleukin-6, lactate dehydrogenase, and creatinine (all p < 0.005). Patients with lymphopenia (n=2,409) compared to those without lymphopenia (n=2,076) had multivariable-adjusted odds ratios of 1.85 (95% CI, 1.53-2.24) for ICU admission, 2.08 (95% CI, 1.67-2.58) for intubation, 1.98 (95% CI, 1.31-3.00) for development of acute kidney failure, and 2.23 (95% CI, 1.79-2.79) for in-hospital mortality (Table 1). Analyses were adjusted for age, gender, race, hypertension, diabetes, chronic obstructive pulmonary disease, chronic kidney disease, coronary artery disease, malignancy, obesity, and smoking.

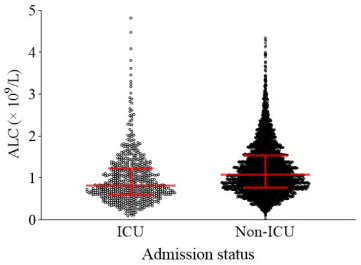

Figure 1. Scatter plots for distribution of absolute lymphocyte counts (ALC) among patients with different admission status. The ALC significantly decreased in patients with ICU admission versus Non-ICU admission (p < 0.000). Red solid lines represent median with interquartile range. ALC < 1.1×10^9/L was more frequent in patients admitted to the intensive care unit (ICU) compared to those who were not, with an odds ratio of 2.14 (95% CI, 1.78-2.56, p < .0001).

Table 1. Multivariable-adjusted risk association of absolute lymphocyte count and outcomes

	Absolute Lymphocyte Count			
Outcomes	< 1.1×10^9/L	≥ 1.1×10^9/L	OR (95% CI)	P value
Patients, n (%)	2409 (53.7)	2076 (46.3)		
ICU admission, n (%)	431 (17.9)	197 (9.5)	1.85 (1.53-2.24)	0.000
Required intubation, n (%)	347 (14.4)	135 (6.5)	2.08 (1.67-2.58)	0.000
Developed AKI, n (%)	82 (3.4)	37 (1.8)	1.98 (1.31-3.00)	0.001
In-hospital mortality, n(%)	401 (16.6)	137 (6.6)	2.23 1.79-2.79)	0.000

ICU, intensive care unit; AKI, acute kidney injury; OR, odds ratio; CI, confidence interval.

Conclusion. Lymphopenia in adult COVID -19 hospitalized patients was associated with increased risk of disease severity (as evidenced by need for ICU admission) and poor clinical outcomes. Absolute lymphocyte count may help with prognostication in individuals hospitalized with COVID-19.

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447. An Ordinal Scale Assessing SARS-CoV-2 Infected Patient Outcomes Using Electronic Health Records

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