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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.

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

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