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## The association of race and COVID-19 mortality

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### ABSTRACT

**Background:** COVID-19 mortality disproportionately affects the Black population in the United States (US). To explore this association a cohort study was undertaken.

**Methods:** We assembled a cohort of 505,992 patients receiving ambulatory care at Bronx Montefiore Health System (BMHS) between 1/1/18 and 1/1/20 to evaluate the relative risk of hospitalization and death in two time-periods, the pre-COVID time-period (1/1/20–2/15/20) and COVID time-period (3/1/20–4/15/20). COVID testing, hospitalization and mortality were determined with the Black and Hispanic patient population compared separately to the White population using logistic modeling. Evaluation of the interaction of pre-COVID and COVID time periods and race, with respect to mortality was completed.

**Findings:** A total of 9,286/505,992 (1.8%) patients were hospitalized during either or both pre-COVID or COVID periods. Compared to Whites the relative risk of hospitalization of Black patients did not increase in the COVID period ( $p$  for interaction=0.12). In the pre-COVID period, compared to Whites, the odds of death for Blacks and Hispanics adjusted for comorbidity was statistically equivalent. In the COVID period compared to Whites the adjusted odds of death for Blacks was 1.6 (95% CI 1.2–2.0,  $p = 0.001$ ). There was a significant increase in Black mortality risk from pre-COVID to COVID periods ( $p$  for interaction=0.02). Adjustment for relevant clinical and social indices attenuated but did not fully explain the observed difference in Black mortality.

**Interpretation:** The BMHS COVID experience demonstrates that Blacks do have a higher mortality with COVID incompletely explained by age, multiple reported comorbidities and available metrics of sociodemographic disparity.

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### 1. Introduction

The SARS-CoV2 viral infection first emerged in China in December of 2019 [1–3]. As of April 24 there were over 270,000 cases in New York City alone, with close to 17,000 Coronavirus Disease 2019 (COVID-19) related deaths reported [4].

Black individuals have been disproportionately affected. US counties that are majority-Black have three times the rate of infection and almost six times the rate of death as counties where White residents are in the majority [5]. Reporting on a cohort of persons in 99

counties in 14 states, the Centers for Disease Control noted that while Blacks constitute 18% of the population they represent 33% of the COVID-19 hospitalized population [6]. In New York City Black persons represent 22% of the population but accounted for 28% of the deaths [7]. Age and population adjusted Black mortality was reportedly more than twice that for Whites [4]. And boroughs with the highest proportion of Black and Hispanic residents reported the worst outcomes [8]. These reports, however, do not fully adjust for individual comorbidity nor relevant socio-demographic factors that may shed light on the drivers of worse outcomes [9–11].

In the US, Black persons suffer from higher rates of diabetes, hypertension, asthma, HIV and obesity than White persons [12]. Thus the disproportionate rates of COVID related morbidity and mortality

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## Research in context

### Evidence before this study

Prior to this study an ecologic study by Wadhera et al. describing borough level distribution of age and race, reported that the borough with the highest number of minority residents (the Bronx) also had the highest prevalence of COVID related hospitalization and death. A large case series of hospitalized COVID + patients in Louisiana by Price-Haywood et al. alluded to a higher Black prevalence among admitted COVID+ patients than expected from the racial distribution in their hospital's patient population. No study to date evaluated the association of race and COVID outcomes at the individual patient level from a defined cohort of patients who were clinically and socio-economically well characterized prior to the entry of the pandemic. Similarly, no study used patient level data to establish expected mortality from a historic non-COVID period to the mortality in a COVID period.

### Added value of this study

Because of the limited access to reliable testing the ability to examine hospitalization and mortality without the need for laboratory tests in a catchment area heavily impacted by COVID is critical to establish credibility of deduction, without the fear of sampling bias. We were able to establish clinical comorbidity prior to COVID and to adjust for comorbidities and granular socioeconomic variables in our observations of mortality. This paper explicitly and individually assessed the question of how much of this mortality disparity is explained by the differential detected comorbidities in the different racial groups. By identifying a large cohort of patients (>505,000) prior to COVID who served as the denominator population we directly addressed the important question of contribution of race, age, and comorbidity (prior to hospitalization) and socioeconomic factors to COVID related mortality.

### Implications of all the available evidence

The COVID pandemic has unmasked a disparity in healthcare affecting Black patients. The path forward is to scrutinize how our health system leaves unaccounted those comorbidities and social forces that disproportionately affect our Black population.

in the Black population are consistent with these established trends [8,9,13]. To evaluate the root cause of COVID-associated inequities it is important to evaluate the contribution of baseline comorbid conditions and socioeconomic factors to worse outcomes [11]. The Bronx has one the highest COVID case rate, hospitalization rate and death rate by county in the US [4,7,8]. We undertook a study of the impact of the COVID pandemic on a well-defined cohort of patients in the Bronx to explore the role of race, comorbidity and socioeconomic factors on COVID-related hospitalization and mortality.

## 2. Methods

### 2.1. Analytic framework

After obtaining Institutional Review Board approval by the Albert Einstein College of Medicine, we conducted a retrospective cohort study of patients with any outpatient encounter between 1/1/18–1/1/20 within BMHS to attempt to answer the following:

- 1) Are Black patients being tested for COVID at the same rate as White patients?

- 2) Are Black patients testing positive for COVID more frequently than White patients?
- 3) Are Blacks hospitalized more frequently than Whites in the COVID time-period (COVID: 3/1/20–4/15/20) compared to the pre-COVID time-period (1/1/20–2/15/20)?
- 4) Is the risk of Black mortality higher in the COVID period than in the pre-COVID period?
- 5) Among COVID + patients, are Blacks hospitalized more frequently than Whites?
- 6) Once hospitalized, are more COVID+ Blacks dying than COVID+ Whites?

### 2.2. Setting

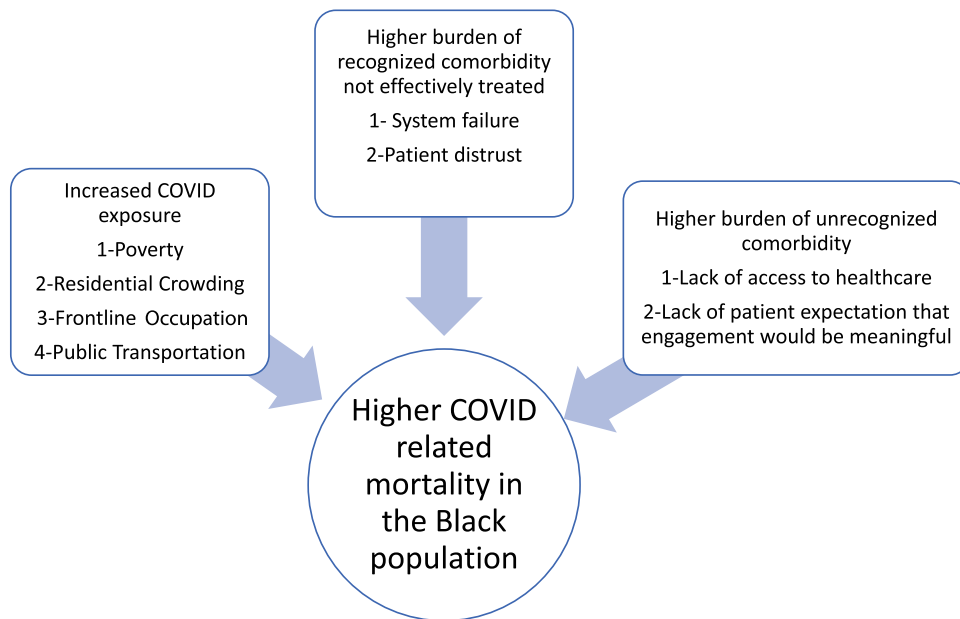
The Montefiore Health System is composed of 15-member hospitals and more than 200 outpatient ambulatory sites providing comprehensive care to patients across the Bronx, Westchester and Hudson Valley. With a core historical service area in Bronx, NY (BMHS) composed of three hospitals, an extensive Bronx based ambulatory care network with 2.8 million visits annually, BMHS is a major Bronx healthcare provider.

The patient population was any individual with a Bronx address receiving outpatient care in BMHS at least once between January 1, 2018 and January 1, 2020. We matched these patients to admissions and deaths in the pre-COVID and COVID periods (1/1/20–2/15/20 and 3/1/20 – 4/15/20, respectively) and to their available clinical data to establish a rate of hospitalization and mortality which is then accounted for by race, age, time-period, and comorbidity. A sub-cohort of patients who had been hospitalized with confirmed COVID + tests between 3/11/20–4/11/20 ( $n = 1755$ ) was also studied.

We devised a conceptual model (Fig. 1) to inform our strategy of selection of adjustment variables. Age, sex and relevant comorbidities used in multivariate adjustment included history of asthma (any asthma related ICD code recorded), smoking (as recorded during clinical encounter), body mass index (BMI) and hypertension (as defined by any related ICD code used during a clinical encounter or a recorded systolic blood pressure > 150 mmHg), all sought between 1/1/2018–3/1/2020 [14]. Morbid obesity was defined as BMI > 35 kg/m<sup>2</sup>. Diabetes was established by Hba1c >= 6.5 within 10 years before cohort enrollment. Charlson comorbidity scores were derived from "Clinical Looking Glass"(see below) and were calculated based on comorbidity data gathered within 6 months prior to the last time they were seen in the outpatient setting between 1/1/2018–1/1/2020 [15]. Age was categorically defined by decile with the lower limit defined as those younger than 40 years and upper limit as those older than 80 years. Poverty (% households living under the poverty line), education (% completed high school), community racial composition (% Black residents), internet access (% with an active internet subscription), access to insurance (% without health insurance), use of public transportation (% using public transportation as a primary means of commuting to work dichotomized at 60%) and crowding (average size of household), were obtained by matching patients addresses to census tract ("Mapitude Caliper Corporation 2020") and using data from the American Community Survey(ACS) [16]. Socioeconomic status(SES) was also provided by "Clinical Looking Glass" (see below under "Cohort Creation and Outcome") and was calculated with the method of Diez Roux and reported (census tract linked to community of residence) as standard deviations from New York State mean [17]. These variables were also included as adjustments in multivariate models.

### 2.3. Cohort creation and outcome

All BMHS based care is served by a single electronic medical record, Epic© (Verona, WI). To create the study cohort "Clinical



**Fig. 1.** Conceptual Model of Potential Drivers of Disproportionate COVID-Mortality in the Black Population.

Looking Glass”, a sophisticated cohort building and outcome analysis tool that uses all clinical encounters, laboratory tests, outpatient and inpatient visits to BMHS, with their diagnoses, to create cohorts and analyze outcomes, was utilized [18-21]. (Supplementary index) COVID testing became available in early March 2020. Using Clinical Looking Glass, the following outcomes were assessed: (1) presence of a COVID swab test, as ascertained through Montefiore laboratory tests and included in EPIC; (2) presence of a **positive** COVID swab test, (3) all-cause hospitalizations and 4) all-cause mortality events, during pre-COVID and COVID time periods and 5) hospitalization and 6) mortality, of a COVID+ hospitalized sub-cohort.

#### 2.4. Exposure variables: race/ethnicity

Race/ethnicity data is patient self-defined at time of initial registration for BMHS care. It was registered as “non-Hispanic White”, “non-Hispanic Black”, “Hispanic,” and “Other” which includes patients not belonging to preceding categories (patients who define themselves multiracially, or those patients that are unwilling or unable to provide information).

#### 2.5. Statistical methods

Stata 14.0 (StataCorp, LLC, College Station TX)) was used for all analyses. Simple bivariate analyses used one-way ANOVA and Kruskal-Wallis tests for continuous variables, and chi-square tests for comparisons of categorical variables. For analyses 1 and 2, logistic regression was used to determine the association of obtaining COVID testing(yes/no), and testing positive(yes/no), with race/ethnicity while adjusting for age. For analyses 5 and 6 logistic regression was used to determine binary outcomes of hospitalization and in-hospital death in the subset of patients who were hospitalized with COVID+ tests. Relevant adjustment variables included age, sex and clinical comorbidity, (Model 2) and socioeconomic variables (Model 3).

For analyses 3 and 4, patients were assigned an admission outcome variable based upon the admission occurrence in the pre-COVID period and/or in the COVID period. Comparisons of hospitalization rate and in-hospital death rate for all patients were made between the pre-COVID and COVID time periods in unadjusted (Model 1), Model 1 adjusted for clinical comorbidity (Model 2) and Model 2 adjusted for socioeconomic variables (Model 3) analyses,

with White as the baseline population. The denominator cohort was the Bronx Montefiore Health System (BMHS) ambulatory care population. The representations of the baseline population in the two time-periods were then combined for a total dataset that was twice the size of the original to enable interaction testing of time-period and race/ethnicity with respect to outcome. Logistic regression with a random effect term for patient identification (ID)(to account for double representation of each patient) was used to test for interaction effect [22]. The combined dataset could examine if the time-period changed the odds of Black vs White hospitalization or mortality. Because for rare events the odds ratios (OR) and relative risk (RR) are numerically equivalent, relative risk was used interchangeably for clarity. Results were divided into separate short sections to address the posed questions in the analytic framework. The funding sources of the manuscript authors had no role in the preparation of this manuscript.

### 3. Results

A total of 505,992 patients were seen at least once in the BMHS ambulatory centers between 1/1/2018–1/1/2020, 6.4% were White, 26.9% Black and 37.9% Hispanic. Black patients compared to White patients were younger, had more asthma, more morbid obesity but had less hypertension, and smoking (Table 1). Black patients more frequently lived in communities with a higher percentage of Black residents and of households living under the poverty line but lower percentage of households reporting use of public transportation to commute to work (Table 1).

#### 3.1. Are Black patients being tested for COVID-19 at the same rate as White patients?

Between 3/11/2020 - 4/11 2020, 4312 members of the cohort were COVID tested with a test rate per 1000 patients of 9.7 for Whites, 11.7 for Blacks, 8.3 for Hispanics and 5.6 for Other ( $p < 0.001$ ).

#### 3.2. Are Black patients testing positive for COVID more frequently than White patients?

The proportion tested positive per 1000 patients (from BMHS cohort) was 6.0 for Whites, 8.3 for Blacks, 5.5 for Hispanic and 3.8 for

**Table 1**  
Clinical and Socio-Demographic Characteristics of the BMHS Ambulatory Population.

N = 505,991	White N = 32,398 (6.4%)	Black 136,297 (26.9%)	Hispanic 191,686 (37.9%)	Other 145,611 (28.8%)	p value
Mean Age (+/- SD)	53.2 (24.4)	40.6 (24.0)	37.6 (23.9)	34.5 (24.8)	<0.001
Male Sex (n(%))	14,241 (44.0)	52,629 (38.6)	75,501 (39.4)	62,811 (43.1)	<0.001
DM Yes (n(%))	3594 (11.1)	20,842 (15.3)	24,231 (12.6)	11,740 (8.1)	<0.001
Hypertension Yes n (%)	14,275 (44.1)	53,279 (39.8)	62,451 (32.6)	34,593 (23.8)	<0.001
Asthma Yes n (%)	2720 (8.4)	19,674 (14.4)	31,086 (16.2)	12,449 (8.6)	<0.001
Smoking Yes n (%)	11,276 (34.8)	30,625 (22.5)	41,912 (21.9)	21,019 (14.4)	<0.001
Charlson Comorbidity Score (n (%))					
0–1	16,588 (51.2)	98,990 (72.6)	146,688 (76.6)	115,472 (79.3)	<0.001
2–3	10,132 (31.3)	26,572 (19.5)	32,602 (17.0)	23,790 (16.3)	
>3	5678 (17.5)	19,735 (7.9)	12,396 (6.5)	6348 (4.4)	
Morbidly Obese Yes (BMI>35 kg/m <sup>2</sup> ) n (%)	4165 (14.6)	22,250 (18.2)	24,316 (14.2)	12,630 (10.4)	<0.001
Average household size Mean (+/- SD)	2.6 (0.4)	2.8 (0.4)	2.8 (0.3)	2.8 (0.4)	<0.001
% of households living under the poverty line Mean (SD)	14.5 (11.8)	23.1 (13.7)	27.6 (12.2)	24.3 (13.0)	<0.001
% completed high school Mean (+/- SD)	26.9 (6.4)	28.5 (5.0)	28.2 (5.0)	27.9 (5.3)	<0.001
% with active internet subscription Mean (+/- SD)	78.2 (7.2)	72.2 (7.7)	71.8 (7.7)	73.2 (7.9)	<0.001
% using public transportation to commute to work Mean (+/-SD)	48.2 (15.7)	59.5 (11.2)	62.4 (11.6)	60.0 (12.8)	<0.001
% Black residents Mean (+/- SD)	19.9 (18.6)	48.3 (21.2)	32.1 (17.0)	32.8 (19.8)	<0.001

BMHS - Bronx Montefiore Health System; DM-Diabetes Mellitus

Other ( $p < 0.001$ ). (Table 2) Logistic modeling of the odds of positive tests demonstrated that compared to baseline Whites, Blacks were more likely to be positive: OR 1.7 (95% CI: 1.5–2.0;  $p < 0.001$ ) as were Hispanics: OR 1.3 (95% CI 1.1–1.5). These findings persisted even with adjustment for clinical and sociodemographic factors (ETable 3; Supplementary Index). The presence of diabetes was related with an OR of 1.5 (95% CI: 1.4–1.7;  $P < 0.001$ ), hypertension with an odds of 1.9 (95% CI 1.7–2.2), asthma with an odds of 1.6 (95% CI 1.4–1.7) and morbid obesity with an odds of 1.3 (95% CI 1.2–1.5) to positive testing (ETable 3, Supplementary Index).

### 3.3. Are Blacks hospitalized more frequently than Whites in the COVID time-period compared to the pre-COVID time-period?

Of the original 505,992 BMHS cohort, 9286 (1.8%) were hospitalized in either the pre-COVID (1/1/20 – 2/15/20), or the COVID (3/1/2020–4/15/2020) time periods, with 1.3% in the pre-COVID periods and 1.0% in the COVID period. In the pre-COVID period Black patients were hospitalized more frequently than Whites (Table 3). Age category, diabetes, asthma history, hypertension and morbid obesity, were all significantly associated with hospitalization and attenuated, but did not eliminate, the association of Black race and increased hospitalization (adjusted OR 1.1 (95% CI 1.0–1.2);  $p = < 0.01$ ) (Table 3).

**Table 2**  
COVID Tested Index by Race, Ethnicity.

N = 505,991	Proportion tested (n/1000)	p value	Proportion who tested positive (n/1000)	p value
Race/Ethnicity				
Non-Hispanic White (n = 32,398)	9.7	<0.001	6.0	<0.001
Non-Hispanic Black (n = 136,297)	8.3		5.5	
Hispanic (n = 191,686)	5.6		3.8	
Other (n = 145,611)				

Similarly, in the COVID period Black patients were more frequently hospitalized than White patients, (adjusted OR 1.6 (95% CI 1.5–1.8;  $p < 0.01$ ). Adjustment for comorbidities attenuated but did not eliminate this relationship (adjusted OR 1.3 (95% CI 1.2–1.5)) (Table 3). The addition of ACS variables (poverty, % Black residents, % with high school education, % using public transportation and household size) to the models attenuated the hospitalization risk of pre-COVID Blacks towards the null: OR 1.0 (95% CI: 0.9–1.1). While it also attenuated the relative risk in the COVID period, Black vs White race remained significantly associated with hospitalization risk in the COVID period OR 1.2 (95% CI 1.0–1.3) for the COVID period). Of note, the addition of clinical and socioeconomic variables also dramatically attenuated the OR of hospitalization in Hispanics such that the relative risk was no longer significant during either time-period. There was no increased risk of Black hospitalization in the COVID period compared with Black hospitalization in the pre-COVID period. (Supplementary ETable 1) and addition of an interaction term of race and time-period with respect to hospitalization was non-significant ( $p$  for interaction = 0.14).

### 3.4. Is the risk of Black mortality higher in the COVID period than the pre-COVID period?

The number of in-hospital deaths in the BMHS population increased from 259 to 748 between the pre-COVID and COVID periods, demonstrating the impact of the pandemic in the Bronx. In the pre-COVID period the DOH view (with adjustment for race, age and sex only) of the relative risk of Black and Hispanic mortality showed no relative risk increase compared to White (OR 1.2 (95% CI 0.8–1.8);  $p = 0.3$ ) and OR 1.0 (95% CI 0.7–1.5,  $p = 0.8$ ; respectively). In the COVID period the department of health (DOH) view of Black mortality with adjustment for race, age and sex only, showed a persistent and significantly higher relative risk of Black and Hispanic death compared to White (OR 2.1 (95% CI 1.6–2.7),  $p = 0.001$  and OR 1.5 (95% CI: 1.1–1.9),  $p = 0.004$ ; respectively).

**Table 3**

Separate logistic regression models for Odds Ratio of hospitalization by race/ethnicity pre-COVID and COVID adjusted for age and race and further adjustment for comorbidity and Charlson comorbidity score\* (N = 505,992).

	Pre-COVID period hospitalized n = 6403 (1.3%) Model 1: Adjusted for age and race/ethnicity	pre-COVID period Model 2*	COVID period hospitalized N = 5061 (1.0%) Model 1: Adjusted for age and race/ethnicity	COVID period Model 2*
Non-Hispanic White	1	1	1	1
Non-Hispanic Black	<b>1.4 (1.3–1.6)</b>	1.1 (1.0–1.2)	<b>1.6 (1.5–1.8)</b>	1.3 (1.2–1.5)
Hispanic	1.5 (1.3–1.5)	1.1 (1.0–1.2)	1.4 (1.2–1.5)	1.1 (1.0–1.2)
Other	0.8 (0.7–0.9)	0.9 (0.8–1.0)	0.8 (0.7–0.9)	0.9 (0.8–1.0)

\* With further adjustment for Sex, DM, asthma, obesity, hypertension and smoking and Charlson comorbidity score. Adjustments for SES variables not shown.

Adjustment for additional clinical risk variables (diabetes, hypertension, asthma, obesity, and Charlson comorbidity score) attenuated the relative risk of death during COVID in Black patients to 1.6 (95% CI: 1.2–2.1; from 2.1(95% CI 1.6–2.7)) but it remained significant (p = 0.001). In contrast, such adjustment in Hispanic patients reduced the relative risk to an insignificant value (adjusted OR 1.1(95% CI 0.9–1.5; from 1.5(95% CI:1.1–1.9)) Compared to White mortality, the relative risk of Black mortality increased from 1.2(95% CI 0.7–1.9) in the pre-COVID period to 1.6(95% CI 1.2–2.0) in the COVID period (Table 4). The interaction effect of Black race and time-period with respect to mortality was significant with p for interaction=0.02. (Fig. 2)

**3.5. Are COVID positive Blacks hospitalized at a higher rate than COVID positive Whites?**

Of those testing COVID positive (n = 2934), Blacks and Hispanics were hospitalized at a higher rate than Whites (OR 1.7 (95% CI: 1.2–2.4) and OR 1.5 (95% CI: 1.1–2.2); respectively).

**3.6. Once hospitalized, are more COVID diagnosed Blacks dying than COVID diagnosed Whites?**

Of the 5061 hospitalized patients in the COVID period (3/1/2020–4/15/2020), we studied the 1755 COVID positive patients hospitalized between March 11th, 2020 and April 11th<sup>th</sup>, 2020 at BMHS. Compared to Whites, Blacks were younger (65.4 years vs 70.3 years), more frequently male (55.0% vs 37.5%), with a higher proportion with diabetes (47.2% vs 25.8%), asthma (21.9% vs 15.8%) and a lower proportion with smoking history (38.8% vs 44.1%). (ETable 2) Hispanics were significantly younger and poorer than Whites and had lower comorbidity scores. (ETable 2, Supplementary Index) Of the 1755 hospitalized COVID+ patients, 390(22.2%) died, 343(19.5%)

required mechanical ventilation, and 232(13.2%) required renal replacement therapy (RRT) (Etable 2). Compared to Whites, Blacks were more likely to require ventilatory support (RR 1.9 (95% 1.1–2.9, p = 0.03)) and RRT (RR 3.0 (95% CI 1.8–8.4)), but there was no increased in-hospital mortality(RR: 1.2(95% CI 0.7–2.0), p = 0.5). (ETable 4) Compared to Whites, Hispanics were also more likely to require ventilators and RRT (RR for mechanical ventilation 1.5(95% CI 0.9–2.6) and relative risk for RRT 2.9 (1.2–7.0)) but exhibited no increase in mortality (RR 1.0(0.6–1.7) (ETable 2 and 4, Supplementary Index).

**4. Discussion**

In the BMHS ambulatory population, compared to White patients in the COVID time-period, Black patients had a relative risk (RR) of death of 1.6 (95% CI: 1.2–2.1);p = 0.001) while in the pre-COVID period the RR was 1.1(95% CI 0.7–1.9);p = 0.8) suggesting that something about the COVID period increased Black mortality disproportionately (p for interaction=0.02). Compared to Whites, Blacks in the BMHS ambulatory care population suffer from more diabetes, morbid obesity and adverse socioeconomic conditions. Although these factors attenuate the risk of hospitalization of Black patients towards the null, they do not eliminate the association of Black race with increased mortality in the COVID period.

The failure of comorbidity adjusters to explain the mortality difference may be due to insensitivity of our comorbidity metrics to the true Black comorbidity burden. The determination of comorbidity is dependent upon access to the health care delivery system to make diagnoses and measure relevant laboratory tests like the HgA1c. (Fig. 1) Decreased healthcare access from whatever cause could thereby reduce the detection of comorbidities and weaken our comorbidity construct to fully explain the observed mortality. In

**Table 4**

Logistic regression models for Odds Ratio of Death by race COVID and non-COVID periods with adjustment (N = 505,992).

OR for Mortality	pre-COVID period(95% CI)n = 382,954	p-value	COVID period(95% CI)n = 382,954	p-value
Non-Hispanic White	1	0.8	1	0.004
Non-Hispanic Black	1.2 (0.7–1.9)	0.6	1.6 (1.2–2.0)	0.2
Hispanic	1.0 (0.7–1.7)	0.002	1.1 (0.9–1.5)	0.4
Other	0.5 (0.3–0.9)	0.6	1.2 (0.8–1.6)	<0.001
Age (years; baseline <=40)	1	0.3	1	<0.001
41–50	1.2 (0.5–3.1)	0.5	4.8 (2.5–9.2)	<0.001
51–60	1.6 (0.7–3.3)	0.1	5.2 (2.9–9.4)	<0.001
61–70	1.4 (0.7–3.0)	0.006	12.1 (6.8–21.3)	<0.001
71–80	1.8 (0.9–3.9)	0.05	17.7 (10.0–31.5))	<0.001
>80	2.9 (1.4–6.2)	0.1	24.5 (13.6–44.2)	<0.001
Male Sex	1.3 (1.0–1.8)	0.6	2.0 (1.7–2.3)	<0.001
Diabetes Yes	1.3 (1.0–1.6)	<0.001	1.7 (1.5–2.0)	<0.001
Asthma Yes	0.9 (0.6–1.2)	0.003	1.5 (1.3–1.8)	0.02
Hypertension Yes	9.6 (4.6–19.8)	0.8	3.0 (2.2–4.1)	<0.001
Smoking Yes	1.2 (1.1–1.4)	<0.001	1.1 (1.0–1.2)	<0.001
Morbidly Obese Yes	0.8 (0.5–1.5)		1.5 (1.3–1.9)	
Charlson Comorbidity Score	1.3 (1.2–1.3)		1.1(1.1–1.2)	
For every 1 point increase				

ACS variables in model Household size (for every 1 person);% completed high school;% with active internet;% uninsured;% living under the poverty line;% Black residents;% using public transportation; n = 382,954 because of the missing data points for ACS and obesity variables.

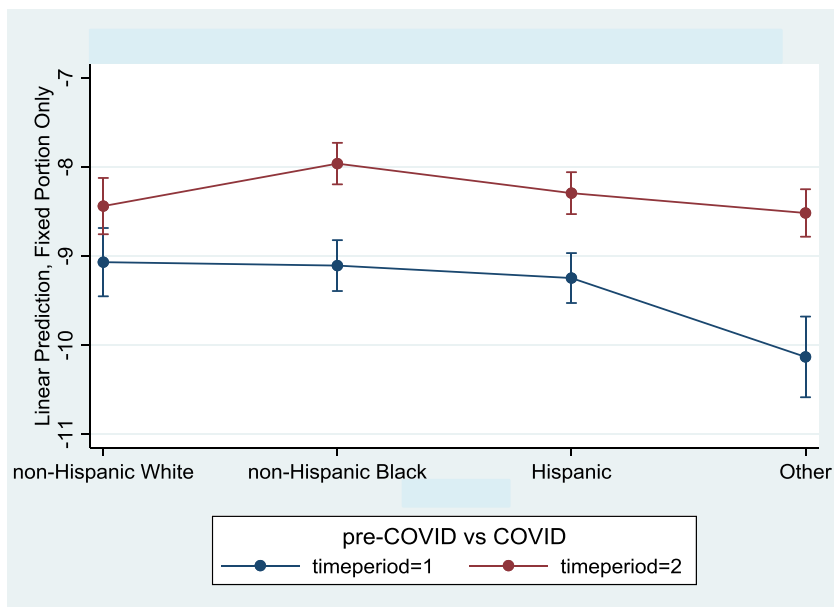


Fig. 2. Mortality Interaction Effect by Race ( $p$  for interaction for Blacks=0.02); (timeperiod 1=pre-COVID, timeperiod2-COVID).

addition, more subtly, the meaning of comorbidity in minority populations might be different. If the healthcare delivery system's response to discovered comorbidity in the minority population is disproportionately inadequate, then the recording of the comorbidity has more pernicious implications. (Fig. 1) A comorbid diagnosis in Blacks might be more severe than in Whites because it might be functionally disproportionately unaddressed. Thus, while comorbidity might present as a billing entity with an ICD10 diagnosis its failure to be adequately remediated might be more of a persistent biologic threat to the Black patient than to his/her White counterpart. Our findings are consistent with department of health and media reports of disproportionate Black mortality with respect to COVID-19 infection [4–6,23,24].

In the hospitalized COVID positive cohort ( $n = 1755$ ), we did not find any difference in mortality for Blacks compared to Whites. This is similar to other reports of hospitalized COVID + cohorts [25,26]. However, we did find that the parent population of all ambulatory Black patients engaged in care did suffer disproportionate higher mortality in the COVID period which we deductively attributed to the entry of COVID into our population. This paradox is confusing as the source of death information came from the hospital admission itself, but it is a paradox that is also observed in the greater literature [4,6,8,13,25–27]. The simplest explanation could be that although COVID infected Blacks once admitted did not die at a higher rate than Whites, proportionately more of them were admitted to the hospital in the COVID period so that the net effect on mortality for the parent population (BMHS population engaged in care) was higher for Black patients. Compared to Whites, we observed in Blacks a higher prevalence of COVID testing, a higher proportion of those tests with positive results, and a higher proportion of the tested positive hospitalized. The fact that COVID+ Blacks and Whites equally survived their hospitalization could possibly be explained by equality of admission severity criteria by race. However, while in both the pre-COVID and COVID periods Blacks were admitted more frequently than Whites, we did not find a statistically significant interaction effect to support the hypothesis of increasing Black vs White hospitalization in the COVID period. Another possible explanation would concede that there was no increase in relative hospitalization but invoke the “replacement hypothesis”. “Gray zone” Black admissions in the pre-COVID period, those admissions for which clinical severity was not exclusively responsible but required consideration of social determinants, are replaced in the COVID period with

clinically indicated admissions with a higher risk for in-hospital death. The actual relative admission rate of Blacks vs Whites does not change, but the nature of those admitted has changed (the “replacement hypothesis”). In this scenario, admitted COVID-positive Blacks are not sicker than COVID positive Whites, but COVID period Blacks are displacing those Blacks who in the pre-COVID period would have been admitted to the hospital and discharged alive. Hence, although proportionate disparity between Black and White hospitalizations has not increased, the replacement hypothesis creates a bulk effect showing proportionately higher mortality in Blacks from the parent population.

Distracted by the COVID onslaught, it would not be unreasonable to also hypothesize that hospitalized patients who in normal times might have been salvaged, now due to clinician distraction were ignored with resultant increased mortality [28]. Thus, even if COVID admitted Blacks did not die in the hospital at a disproportionate rate from their White COVID + counterparts, the non-COVID + Blacks in that period were sicker than their pre-COVID period Black counterparts. Because of this and structural racist conditioning rendering Black patients with less agency to self-advocate than Whites [29], in order to compete for scarce resources they were more vulnerable and would suffer more from this distraction creating a larger number of “non-biologic” COVID related deaths. By this reasoning “non-biologic” deaths are those deaths that occurred as a result of changes in care delivery in response to the COVID surge.

Our results are consistent with the department of health (DOH) reports that adjust for race, age and sex. Our DOH style adjustment for race, age and sex only, demonstrated the reported increase in Black and Hispanic mortality [4,6,30]. However we were able to show an attenuation of this risk with the addition of relevant clinical data/socioeconomic data demonstrating the explanatory roles of each.

Limitations of this study include studying mortality as manifested in only one health system's population which might be insensitive to deaths seen at neighboring hospitals. While true, our study population was 505,992 in a borough of 1.5 million and was a good representation of the Bronx population. Further, one would have to posit differential hospital care follow up in Whites and Blacks to attenuate the observed interaction. If Black patients differentially died at home because of COVID without hospitalization, this lack of access would only mean that our estimate of relative risk is a lower limit. In addition, individual level socioeconomic information was not available, the authors relied instead on Census Tract based socioeconomic

information provided by the Census Bureau. This may have misrepresented the contribution of contextual factors to outcomes. The major strength of this study is that it builds its observations on a clinically and socioeconomically well-defined and well characterized baseline population of 505,992 that serves as a valid indicator group for the population at large which is used to draw inferences about the impact of a new disease by race/ethnicity.

The COVID pandemic has unmasked a disparity in health outcomes. We cannot be sure of its biologic significance, but it seems to be real and present in the Black community beyond the usual explanations of clinical comorbidities and easily available socioeconomic factors. The possibility that unrecognized severity of comorbidity or social vulnerability because of pre-pandemic unequal access to care, or differential failure to remediate those recognized comorbidities/social vulnerabilities, cannot be excluded.

### Author contribution

Drs Golestaneh and Bellin participated in data collection, design, statistical analysis, and write-up of the manuscript. Drs. Coco, Fisher, Yunes, Mokrzycki, Perez, Scott, Billett, Reyes-Gil and Norris participated in data-analysis, data interpretation and writing of the manuscript. Dr. Johns and Dr. Kim assisted in data interpretation and writing of the manuscript.

### Declaration of Competing Interest

Dr. Golestaneh, Dr. Perez, Dr. Norris and Dr. Johns report funding by the NIH outside of the work. The other authors declare no conflict of interest with respect to the content of this manuscript.

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### Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:[10.1016/j.eclinm.2020.100455](https://doi.org/10.1016/j.eclinm.2020.100455).

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