Racial Disparities in COVID-19 Testing and Outcomes

Retrospective Cohort Study in an Integrated Health System

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Background: Racial disparities exist in outcomes after severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection.

Objective: To evaluate the contribution of race/ethnicity in SARS-CoV-2 testing, infection, and outcomes.

Design: Retrospective cohort study (1 February 2020 to 31 May 2020).

Setting: Integrated health care delivery system in Northern California.

Participants: Adult health plan members.

Measurements: Age, sex, neighborhood deprivation index, comorbid conditions, acute physiology indices, and race/ethnicity; SARS-CoV-2 testing and incidence of positive test results; and hospitalization, illness severity, and mortality.

Results: Among 3 481 716 eligible members, 42.0% were White, 6.4% African American, 19.9% Hispanic, and 18.6% Asian; 13.0% were of other or unknown race. Of eligible members, 91 212 (2.6%) were tested for SARS-CoV-2 infection and 3686 had positive results (overall incidence, 105.9 per 100 000 persons; by racial group, White, 55.1; African American, 123.1; Hispanic, 219.6; Asian, 111.7; other/unknown, 79.3). African American persons had the highest unadjusted testing and mortality rates, White persons had the lowest testing rates, and those with other or unknown race had the lowest mortality rates. Compared with White persons, adjusted testing rates among non-White persons were marginally higher, but infection rates were significantly higher; adjusted odds ratios [aORs] for African American persons, Hispanic persons, Asian persons, and persons of other/unknown race were 2.01 (95% CI, 1.75 to 2.31), 3.93 (CI, 3.59 to 4.30), 2.19 (CI, 1.98 to 2.42), and 1.57 (CI, 1.38 to 1.78), respectively. Geographic analyses showed that infections clustered in areas with higher proportions of non-White persons. Compared with White persons, adjusted hospitalization rates for African American persons, Hispanic persons, Asian persons, and persons of other/unknown race were 1.47 (CI, 1.03 to 2.09), 1.42 (CI, 1.11 to 1.82), 1.47 (CI, 1.13 to 1.92), and 1.03 (CI, 0.72 to 1.46), respectively. Adjusted analyses showed no racial differences in inpatient mortality or total mortality during the study period. For testing, comorbid conditions made the greatest relative contribution to model explanatory power (77.9%); race only accounted for 8.1%. Likelihood of infection was largely due to race (80.3%). For other outcomes, age was most important; race only contributed 4.5% for hospitalization, 12.8% for admission illness severity, 2.3% for in-hospital death, and 0.4% for any death.

Limitation: The study involved an insured population in a highly integrated health system.

Conclusion: Race was the most important predictor of SARS-CoV-2 infection. After infection, race was associated with increased hospitalization risk but not mortality.

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The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) pandemic has affected disadvantaged, Hispanic, and Black/African American populations disproportionately. Racial and ethnic disparities in coronavirus disease 2019 (COVID-19) incidence and outcomes have been widely reported and recently summarized (1). However, quantitative analyses showing how racial and ethnic disparities result in differential infection rates and adverse outcomes have not been reported. One important limitation of the current literature is that baseline population characteristics, testing rates, test positivity rates, incidence, hospitalization, and mortality have not been available within the same study. Integrated information systems that can track members across the care continuum provide an opportunity to gain insight into the role of race and ethnicity in the course of SARS-CoV-2 infection.

We performed a population-based study of SARS-CoV-2 testing, infection, hospitalization, and mortality during the initial phase of the pandemic in the United States. We sought to evaluate the contribution of race/ethnicity in COVID-19 testing, incidence, hospitalization, and death during the initial phase of the pandemic (1 February 2020 to 31 May 2020). A better understanding of the role of race/ethnicity could inform health system strategies aimed at mitigating disparities. Our study was done at Kaiser Permanente Northern California (KPNC), an integrated health care delivery system serving 4.4 million members. Integrated information systems permit quantifying predictors and outcomes across a continuum

See also:

Web-Only
Supplement

that includes patients' underlying risk, testing data, infection rates, and outcomes.

METHODS

Setting

Under a mutual exclusivity agreement, 9500 physicians of The Permanente Medical Group care for 4.4 million Kaiser Foundation Health Plan (KFHP) members at facilities owned by Kaiser Foundation Hospitals. Deployment of the Epic electronic health record (www.epicsystems.com) was completed in mid-2010.

Testing Protocols

Testing for suspected SARS-CoV-2 infection was initially performed by the Centers for Disease Control and Prevention and then by State of California and local health departments. Testing was internalized on 13 March 2020. All testing was performed by using SARS-CoV-2 assays with the Roche Cobas 8800, Hologic Panther Fusion, Hologic Panther Aptima, or Cepheid Infinity Testing systems. Testing was initially performed only for symptomatic patients. On 2 April 2020, testing was expanded to include asymptomatic surgical patients; on 21 April 2020, it was further expanded to asymptomatic patients, consistent with health agency recommendations prioritizing health care workers and first responders, close contacts, essential workers, those with high risk medical conditions, congregate setting residents, and employees.

Study Population

We identified all records of KFHP members who met these criteria: alive, 18 years of age or older, members on 1 February 2020, and with continuous membership (unless they died) during the study period (1 February 2020 to 31 May 2020). We then scanned KPNC databases, linking members' records to the following data elements: self-reported race and ethnicity (captured by medical assistants in the outpatient setting, admission clerks for hospitalized patients, and/or at the time of KFHP enrollment); individual Elixhauser comorbid conditions (2) and the aggregate Elixhauser score (3); Charlson Comorbidity Index score (4); and neighborhood deprivation index (NDI), a composite index ranging from –5 to 5, with more positive values indicating worsening neighborhood characteristics (such as poverty and unemployment) (5).

We also captured 3 composite indices that are assigned to adults in the KPNC system: a longitudinal comorbidity score (COmorbidity Point Score, version 2 [COPS2]), an outpatient physiology-based severity of illness score (abbreviated Laboratory-based Acute Physiology Score [abLAPS]), and a 72-hour severity of illness score (Laboratory-based Acute Physiology Score, version 2 [LAPS2]). Each month, all adults with a KPNC medical record number are assigned a COPS2, which is based on diagnoses accrued in the preceding 12 months, with higher scores associated with increasing mortality risk (6). They are also assigned a monthly abLAPS, which is based on 14 laboratory tests obtained in the preceding month; higher scores are associated with increased physiologic

abnormalities (7, 8) (Supplement 1, available at Annals.org). As described elsewhere (6, 9–11), we also scanned KPNC databases to identify hospitalizations, admission to the intensive care unit (ICU), assisted ventilation, and in-hospital and all-cause deaths. For hospitalized patients, we also captured their admission LAPS2, which is assigned at the time of first entry to a non–emergency department hospital unit (6). The LAPS2 is based on vital signs, neurologic status, pulse oximetry, and 16 laboratory tests in the 72 hours preceding hospitalization; higher scores indicate increasing physiologic derangement. In KPNC, the COPS2, abLAPS, and LAPS2 are used for multiple predictive models (for example, death, ICU admission, rehospitalization). They are used as scalar values permitting separate quantitative assessment of patients' acute physiology and longitudinal comorbidity burden; for example, 2 patients could have the same comorbidity burden but different acute physiology.

This data-only project was approved by the KPNC Institutional Review Board, which waived the requirement for informed consent from participants.

Outcomes

Using KPNC databases, we extracted information on whether SARS-CoV-2 testing occurred among all members in the cohort. For members with a positive test result, we used KPNC databases to extract information on hospitalizations and deaths (during the first hospitalization or ever during the entire study period). Follow-up for these 3 outcomes in KPNC data sources was complete during the study period. Among hospitalized patients, we also examined admission illness severity (LAPS2), ICU admission, occurrence of mechanical ventilation, and in-hospital death.

Statistical Analysis

Descriptive and nongeographic statistical analyses were conducted by using R, version 3.6.0.

The exposure of interest was race/ethnicity, which we grouped into 5 categories: White; Black/African American; Hispanic; Asian; and all other, including unknown race. For multivariate analyses, we used logistic regression for dichotomous outcomes (tested or not, infected, hospitalized, in-hospital death, death during study period) and linear regression for the continuous outcome (LAPS2). We adjusted all models for age, sex, NDI, and members' most recent abLAPS and COPS2. Because these latter 2 variables are correlated and because most members have very low scores, we adjusted for COPS2 and abLAPS by using a derived variable that divided patients into 3 mutually exclusive comorbidity groups: low risk (the reference category for all models), medium risk, and high risk. The low-risk group consisted of members with COPS2 of 10 or less and abLAPS of 0 (the lowest possible scores); after removing these healthy members, we defined the other 2 risk groups on the basis of COPS2 terciles and the upper and lower half of the abLAPS distribution in the remaining members (Supplement Table 1 and Table 2, available at Annals.org). We quantified the relative contribution of each predictor by first calculating the difference in log-likelihood comparing the full model with all predictors and the model that

omits the predictor of interest: that is, the likelihood ratio test statistic. This difference can be viewed as the contribution of information provided by the predictor. We then presented each difference as a percentage of the total of all differences corresponding to each predictor in the full model, which we refer to as the percent relative contribution of the predictor (12, 13).

As described elsewhere (14), we identified geographic SARS-CoV-2 infection clusters by using SaTScan software (www.satscan.org) (Supplement 2, available at Annals.org). We used a discrete Poisson-based model (15) to detect circular geographic clusters containing a higher-than-expected proportion of SARS-CoV-2 infection cases among the KPNC population. In addition to reporting the most likely cluster, secondary clusters reported were selected by using the Gini index (a measure of statistical dispersion [16]). Only clusters that were significant at the $P < 0.05$ level were reported. Members included in the study were assigned to census block groups based on their home address as of February 2020. The SARS-CoV-2 infection case and population counts were input to the SaTScan software at the census block group level. Owing to sparse membership outside the primary KPNC service area, the spatial analyses were restricted to 17 counties that comprise 96% of the study population. Spatial analyses for SARS-CoV-2 infection were adjusted for age and sex but not for NDI or race, and separate analyses were performed on the "low-risk" members (persons with COPS2 ≤10 and abLAPS of 0) and all other members. As a sensitivity analysis, we explored the possible confounding of the association between race/ethnicity and infection by location, using a multivariate model for infection that included an indicator variable for spatial cluster (in-cluster, not in-cluster) to represent location. Separate models were fit for low-risk members (persons with COPS2 ≤10 and abLAPS of 0) and all other members to parallel the spatial clustering analyses.

Role of the Funding Source

This study was funded by The Permanente Medical Group, Inc., and Kaiser Foundation Hospitals, Inc., which requested that the study be conducted and submitted for publication and provided comments on an initial draft. The analyses and final version of the paper were determined by the authors alone.

RESULTS

We identified a total of 3 481 716 members (48% male) who met our inclusion criteria (Table 1; Supplement Table 1). The study population was 42.0% White, 6.4% Black/African American, 19.9% Hispanic, 18.6% Asian, 3.7% other race, and 9.3% unknown race (these last 2 categories were pooled in analyses). Compared with the other groups, Black/African Americans had higher rates of diabetes, obesity, hypertension, chronic pulmonary disease, and congestive heart failure. White persons (2.5%) and Black/African American persons (2.3%) had higher rates of cancer (Supplement Table 3, available at Annals.org).

The low-risk COPS2/abLAPS comorbidity group included 2 765 338 members; the medium-risk group, 369 530; and the high-risk group, 346 848. The proportion of members who were in the low-risk comorbidity group was 74% of White persons, 74% of Black/African American persons, 84% of Hispanic persons, 83% of Asian persons, and 89% of those of other or unknown race (Supplement Table 2). White persons and Asian persons lived in more advantaged neighborhoods, as evidenced by lower NDI, than did Black/African American persons, Hispanic persons, and persons of other/unknown races.

A total of 91 212 (2.6%) members were tested for SARS-CoV-2 infection, of whom 3686 (4.0%) had positive results, for an incidence of 105.9 per 100 000 persons (range across racial groups, 55.1 to 219.6). Unadjusted testing rates were relatively similar across racial groups except for those with other/unknown race. Unadjusted rates of positive SARS-CoV-2 tests (incidence) were lower for White persons. Hospitalization, ICU admission, assisted ventilation, in-hospital death, and total deaths among infected patients during the study period were higher among African American persons, Hispanic persons, and Asian persons.

Unadjusted patient characteristics and outcomes among hospitalized patients are shown in Table 2, and comorbidities are shown in Supplement Table 4 (available at Annals.org). Compared with other racial/ethnic groups, White patients were older and had a higher comorbidity burden; they also came from more advantaged neighborhoods than did Black/African American persons, Hispanic persons, and Asian persons. Asian persons and Black/African American persons had somewhat higher unadjusted admission severity of illness scores (median LAPS2, 82 and 79, respectively, versus 70 for Hispanic persons and 75 for White persons). Unadjusted mortality rates were highest for White persons (17.0% during their first hospitalization and 27.7% during the study period), followed by Black/African American persons (12.7% and 19.0%, respectively), Asian persons (10.5% and 14.8%), persons of other/unknown race (8.1% and 11.3%), and Hispanic persons (9.7% and 10.3%).

In multivariate modeling (Table 3; Supplement Table 5, available at Annals.org), decreasing age, female sex, and increasing comorbidity burden were associated with testing, as were Black/African American and Hispanic race. Increasing NDI was associated with decreased likelihood of being tested, as was being of other/unknown race. Infection was much more likely among non-White persons, with the highest adjusted odds ratios (aORs) found among Hispanic persons (aOR, 3.93 [95% CI, 3.59 to 4.30]) and Asian persons (2.19 [CI, 1.98 to 2.42]). In sensitivity analysis, race remained a strong predictor of infection within geographic clusters even when NDI was included (Supplement 2). The risk for hospitalization was elevated for Black/African American persons, Hispanic persons, and Asian persons relative to White persons (aOR range, 1.42 to 1.47) but was not elevated for persons of other/unknown race. With respect to severity of illness on admission, LAPS2s were significantly elevated

Table 1. Study Cohort Characteristics and Unadjusted Outcomes

Characteristic	All Patients	White Patients	Black/African American Patients	Hispanic Patients	Asian Patients	Patients of Other or Unknown Race/Ethnicity
Participants, n (%)	3 481 716 (100)	1 463 870 (42.0)	221 681 (6.4)	693 103 (19.9)	649 095 (18.6)	453 967 (13.0)
Median age (IQR)	46.0 (32.0 to 61.0)	52.0 (36.0 to 66.0)	47.0 (32.0 to 61.0)	41.0 (29.0 to 55.0)	45.0 (33.0 to 58.0)	39.0 (28.0 to 54.0)
Male sex, n (%)	1 676 118 (48.1)	700 359 (47.8)	98 127 (44.3)	330 194 (47.6)	293 739 (45.3)	253 699 (55.9)
Selected comorbid conditions, *n (%)**						
Diabetes	309 893 (8.9)	113 079 (7.7)	28 803 (13.0)	73 239 (10.6)	68 679 (10.6)	26 093 (5.7)
Obesity	206 072 (5.9)	94 090 (6.4)	24 314 (11.0)	54 875 (7.9)	17 205 (2.7)	15 588 (3.4)
Hypertension (any)	559 466 (16.1)	270 517 (18.5)	55 434 (25.0)	93 712 (13.5)	98 305 (15.1)	41 498 (9.1)
Chronic pulmonary disease	241 335 (6.9)	122 316 (8.4)	22 649 (10.2)	44 806 (6.5)	31 952 (4.9)	19 612 (4.3)
Congestive heart failure	41 890 (1.2)	23 578 (1.6)	4572 (2.1)	5772 (0.8)	4790 (0.7)	3178 (0.7)
Cancer	64 023 (1.8)	37 053 (2.5)	5064 (2.3)	8835 (1.3)	9525 (1.5)	3546 (0.8)
Indices†						
Median NDI (IQR)	−0.370 (−0.866 to 0.224)	−0.548 (−0.959 to −0.0166)	0.0435 (−0.501 to 0.843)	0.0388 (−0.518 to 0.734)	−0.547 (−1.02 to 0.0244)	−0.341 (−0.860 to 0.321)
CCI score ≥ 4 (%)	137 132 (3.9)	69 087 (4.7)	13 940 (6.3)	22 428 (3.2)	21 346 (3.3)	10 331 (2.3)
Elixhauser score ≥ 6, %	247 164 (7.1)	126 090 (8.6)	19 428 (8.8)	39 946 (5.8)	44 125 (6.8)	17 575 (3.9)
COPS2 ≥ 65, %	85 043 (2.4)	49 642 (3.4)	8102 (3.7)	11 980 (1.7)	9477 (1.5)	5842 (1.3)
abLAPS ≥ 10, %	52 660 (1.5)	26 594 (1.8)	5094 (2.3)	9285 (1.3)	8036 (1.2)	3651 (0.8)
Population testing						
Tested, n (%)	91 212 (2.6)	40 110 (2.7)	7143 (3.2)	19 310 (2.8)	16 186 (2.5)	8463 (1.9)
Positive test rate, %	4.0	2.0	3.8	7.9	4.5	4.3
Outcome incidence per 100 000 persons						
SARS-CoV-2 infection	105.9	55.1	123.1	219.6	111.7	79.3
Hospitalized	22.9	14.1	35.6	41.8	25.0	13.7
Admitted to ICU	7.0	3.3	10.8	13.1	9.4	4.6
Mechanical ventilation	6.8	3.4	9.5	12.7	8.9	4.2
Died during first hospitalization	2.7	2.4	4.5	4.0	2.6	1.1
Died during study period	4.6	4.7	8.6	4.5	4.5	2.4

abLAPS = abbreviated Laboratory-based Acute Physiology Score; CCI = Charlson Comorbidity Index; COPS2 = COmorbidity Point Score, version 2; IQR = interquartile range; NDI = neighborhood deprivation index; SARS-CoV-2 = severe acute respiratory syndrome coronavirus 2.
* Comorbid conditions are based on the method of Elixhauser and colleagues (2). Supplement Table 3 (available at Annals.org) provides detail on the Elixhauser comorbid conditions in the overall study cohort.
† The NDI (5) ranges from −5 to 5; higher positive values indicate increasing neighborhood deprivation. The Charlson Comorbidity Index score (4) ranges from 0 to 40; increasing values indicating worsening comorbidity burden. The Elixhauser score was developed by van Walraven and colleagues (3) on the basis of the method of Elixhauser and colleagues (2); this score can range from −19 to 89, with higher scores associated with increasing in-hospital mortality risk. An Elixhauser score of 6 or more is roughly equivalent to a CCI score of 4 or more. The COPS2 (6) is assigned every month to all adults with a Kaiser Permanente Northern California medical record number. Range is from 0 to 1010; higher scores indicate worse mortality risk. The univariate relationship between the COPS2 and 1-year mortality is as follows: 0 to 39, 0.3%; 40 to 64, 5.3%; ≥65, 17.2%. The abLAPS is a monthly score using 14 laboratory tests that is based on the LAPS score (7). Range is from 0 to 256; higher scores indicate increasing physiologic abnormalities in the preceding month. In recent internal analyses, the univariate relationship between the abLAPS and 30-day mortality is as follows: 0 to 4, 0.06%; 4 to 9, 0.18%; ≥10, 1.32%. For all indices reported here other than NDI, most patients scored at the extreme low end, so reporting population medians or means is not informative.

among Asian persons (12.6 points compared with White persons), persons of other/unknown race (11.8), and Hispanic persons (6.7). However, none of the other racial/ethnic groups had statistically significant aORs for in-hospital death or death during the study period compared with White persons.

Table 4 shows the relative contributions of the key predictors in the multivariate models. With respect to testing, most of the model explanatory power was due to sex (12.8%) and comorbidity/acute physiology (77.9%), with race/ethnicity only accounting for 8.1%. In contrast,

the likelihood of infection was largely due to race, with 80.3% of the model explanatory power. For the remaining outcomes, the most important predictor was age, with race only contributing 4.5% for hospitalization, 12.8% for admission LAPS2, 2.3% for in-hospital death, and 0.4% for death during the study period.

Our geographic clustering analyses (Supplement 2) found that infections were clustered in areas with higher proportions of non-White members. This was evident among members with low risk as well as among members with a significant comorbidity burden.

DISCUSSION

This study reports and quantifies racial and ethnic differences across aspects of the care continuum, from testing to mortality, in the COVID-19 pandemic. Race/ethnicity was the most important factor in a patient's likelihood of becoming infected with SARS-CoV-2. Compared with White persons, the other racial groups had increased incidence of SARS-CoV-2 infection that persisted after controlling for age, sex, NDI, and preexisting comorbidity burden. Infection clusters were located in areas with higher proportions of non-White members. After infection, race/ethnicity made a smaller contribution than other factors with respect to severity of illness on admission, hospitalization, and death. Non-White persons had higher adjusted admission severity of illness scores, suggesting that future efforts to improve outcomes could focus on patients' initial contact with the health system when they first suspect SARS-CoV-2 infection.

Table 2. Hospitalized Cohort Characteristics and Unadjusted Outcomes

Characteristic	All Patients	White Patients	Black/African American Patients	Hispanic Patients	Asian Patients	Patients of Other or Unknown Race/Ethnicity
Participants, n (%)	799 (100)	206 (25.8)	79 (9.9)	290 (36.3)	162 (20.3)	62 (7.8)
Median age (IQR)	60.0 (49.0 to 74.0)	73.0 (59.0 to 85.0)	69.0 (53.5 to 79.0)	54.0 (44.0 to 64.0)	58.5 (51.0 to 68.0)	58.0 (46.0 to 70.8)
Male sex, n (%)	436 (54.6)	112 (54.4)	39 (49.4)	173 (59.7)	80 (49.4)	32 (51.6)
Selected comorbid conditions, n (%)*						
Diabetes	266 (33.3)	60 (29.1)	31 (39.2)	95 (32.8)	57 (35.2)	23 (37.1)
Obesity	123 (15.4)	32 (15.5)	17 (21.5)	54 (18.6)	10 (6.2)	10 (16.1)
Hypertension (any)	340 (42.6)	105 (51.0)	49 (62.0)	101 (34.8)	54 (33.3)	31 (50.0)
Chronic pulmonary disease	128 (16.0)	52 (25.2)	14 (17.7)	38 (13.1)	13 (8.0)	11 (17.7)
Congestive heart failure	69 (8.6)	32 (15.5)	9 (11.4)	11 (3.8)	7 (4.3)	10 (16.1)
Cancer	33 (4.1)	14 (6.8)	3 (3.8)	6 (2.1)	7 (4.3)	3 (4.8)
Indices†						
Median NDI (IQR)	–0.0833 (–0.686 to 0.600)	–0.538 (–0.881 to 0.162)	0.386 (–0.116 to 0.908)	0.246 (–0.433 to 0.861)	–0.426 (–0.749 to0.12)	–0.116 (–0.808 to 0.417)
CCI score ≥4 (%)	166 (20.8)	68 (33.0)	24 (30.4)	30 (10.3)	26 (16.0)	18 (29.0)
Elixhauser score ≥6, %	218 (27.3)	91 (44.2)	26 (32.9)	44 (15.2)	34 (21.0)	23 (37.1)
COPS2 ≥65, %	128 (16.0)	66 (32.0)	16 (20.3)	22 (7.6)	10 (6.2)	14 (22.6)
abLAPS ≥10, %	77 (9.6)	33 (16.0)	15 (19.0)	16 (5.5)	6 (3.7)	7 (11.3)
Severity of illness and outcomes among hospitalized patients						
Median LAPS2 (IQR)‡	75.0 (55.0 to 98.5)	75.0 (54.3 to 105.0)	79.0 (60.5 to 100.0)	70.0 (53.0 to 87.0)	82.5 (60.0 to 101.0)	75.0 (57.5 to 96.0)
LAPS2 ≥110, n (%)‡	128 (16.0)	45 (21.8)	14 (17.7)	29 (10.0)	28 (17.3)	12 (19.4)
Admitted to ICU, n (%)	245 (30.7)	48 (23.3)	24 (30.4)	91 (31.4)	61 (37.7)	21 (33.9)
Mechanical ventilation, n (%)	236 (29.5)	50 (24.3)	21 (26.6)	88 (30.3)	58 (35.8)	19 (30.6)
Died during first hospitalization, n (%)	95 (11.9)	35 (17.0)	10 (12.7)	28 (9.7)	17 (10.5)	5 (8.1)
Died during study period, n (%)	133 (16.6)	57 (27.7)	15 (19.0)	30 (10.3)	24 (14.8)	7 (11.3)

abLAPS = abbreviated Laboratory-based Acute Physiology Score; CCI = Charlson Comorbidity Index; COPS2 = COmorbidity Point Score, version 2; ICU = intensive care unit; IQR = interquartile range; LAPS2 = Laboratory-based Acute Physiology Score, version 2; NDI = neighborhood deprivation index.

* Comorbid conditions are based on the method of Elixhauser and colleagues (2). Supplement Table 4 (available at Annals.org) provides detail on the Elixhauser comorbid conditions in the hospitalized cohort.

† The NDI (5) ranges from –5 to 5; higher positive values indicate increasing neighborhood deprivation. The Charlson Comorbidity Index score (4) ranges from 0 to 40; increasing values indicating worsening comorbidity burden. The Elixhauser score was developed by van Walraven and colleagues (3) on the basis of the method of Elixhauser and colleagues (2); this score can range from –19 to 89, with higher scores associated with increasing in-hospital mortality risk. An Elixhauser score of 6 or more is roughly equivalent to a CCI score of 4 or more. The COPS2 (6) is assigned every month to all adults with a Kaiser Permanente Northern California medical record number. Range is from 0 to 1010; higher scores indicate worse mortality risk. The univariate relationship between the COPS2 and 1-year mortality is as follows: 0 to 39, 0.3%; 40 to 64, 5.3%; ≥65, 17.2%. The abLAPS is a monthly score using 14 laboratory tests that is based on the LAPS score (7). Range is from 0 to 256; higher scores indicate increasing physiologic abnormalities in the preceding month. In recent internal analyses, the univariate relationship between the abLAPS and 30-day mortality is as follows: 0 to 4, 0.06%; 4 to 9, 0.18%; ≥10, 1.32%. For all indices reported here other than NDI, most patients scored at the extreme low end, so reporting population medians or means is not informative.

‡ The admission LAPS2 (6) is assigned on the basis of a patient's worst vital signs, pulse oximetry, neurologic status, and 16 laboratory test results in the preceding 72 hours. The univariate relationship of an admission LAPS2 with 30-day mortality is as follows: 0 to 59, 1.0%; 60 to 109, 5.0%; ≥110, 13.7%. After age, sex, diagnosis, and comorbid conditions are controlled for, the adjusted odds ratio for inpatient mortality for an increase in LAPS2 of 5 points is 1.13.

Table 3. Multivariable Analyses to Risk Factors for SARS-CoV-2 Testing, Incidence, and Infection Outcomes

Characteristic	Outcome*					
	Testing	Infection	Hospitalization	Admission Severity of Illness (LAPS2)†	Death	
					In-hospital	Ever
Age‡	0.99 (0.99 to 1.00)	1.07 (1.05 to 1.10)	1.48 (1.39 to 1.58)	7.57 (6.11 to 9.02)	1.74 (1.45 to 2.09)	2.40 (2.06 to 2.80)
Male sex§	0.77 (0.76 to 0.78)	0.99 (0.93 to 1.05)	1.61 (1.35 to 1.92)	4.83 (0.63 to 9.02)	1.17 (0.74 to 1.84)	1.75 (1.21 to 2.52)
NDI	0.95 (0.95 to 0.96)	1.22 (1.18 to 1.26)	1.07 (0.97 to 1.18)	0.15 (−2.34 to 2.63)	1.16 (0.88 to 1.52)	1.08 (0.87 to 1.36)
Comorbidity group‖						
Medium risk	1.54 (1.51 to 1.57)	1.28 (1.16 to 1.43)	1.68 (1.31 to 2.16)	−2.75 (8.85 to 3.34)	0.90 (0.45 to 1.82)	1.23 (0.67 to 2.27)
High risk	2.71 (2.66 to 2.77)	1.62 (1.46 to 1.80)	3.02 (2.37 to 3.83)	5.77 (0.30 to 11.24)	0.94 (0.51 to 1.71)	2.63 (1.56 to 4.44)
Race/ethnicity§						
Black/African American	1.19 (1.16 to 1.23)	2.01 (1.75 to 2.31)	1.47 (1.03 to 2.09)	7.98 (0.00 to 15.96)	0.81 (0.36 to 1.82)	1.07 (0.58 to 1.98)
Hispanic	1.14 (1.12 to 1.16)	3.93 (3.59 to 4.30)	1.42 (1.11 to 1.82)	6.67 (0.72 to 12.63)	1.16 (0.62 to 2.16)	0.96 (0.57 to 1.63)
Asian	0.98 (0.97 to 1.00)	2.19 (1.98 to 2.42)	1.47 (1.13 to 1.92)	12.63 (6.25 to 19.01)	1.00 (0.52 to 1.96)	1.15 (0.69 to 1.92)
Other/unknown	0.79 (0.77 to 0.81)	1.57 (1.38 to 1.78)	1.03 (0.72 to 1.46)	11.78 (3.08 to 20.49)	0.81 (0.29 to 2.25)	0.86 (0.41 to 1.78)

abLAPS = abbreviated Laboratory-based Acute Physiology Score; LAPS2 = Laboratory-based Acute Physiology Score, version 2; NDI = neighborhood deprivation index; SARS-CoV-2 = severe acute respiratory syndrome coronavirus 2.
* For admission severity of illness, regression coefficients (i.e., severity of illness score points) are shown. For all other outcomes, values are the adjusted odds ratio (95% CI).
† The dependent variable is the admission LAPS2 (6), which is assigned only to hospitalized patients and is based on a patient's worst vital signs, pulse oximetry, neurologic status, and 16 laboratory test results in the 72 hours preceding admission. The LAPS2 ranges from 0 to 414; higher scores indicate increasing physiologic derangement. The univariate relationship of admission LAPS2 with 30-day mortality is as follows: 0 to 59, 1.0%; 60 to 109, 5.0%; ≥110, 13.7%. After age, sex, diagnosis, and comorbid conditions are controlled for, the adjusted odds ratio for inpatient mortality for an increase in LAPS2 of 5 points is 1.134 (95% CI, 1.133 to 1.135).
‡ Results shown are per 10-year increment in age.
§ Female sex is the reference group.
‖ Low risk is the reference group. On the basis of comorbidity scores and recent monthly acute physiology, we divided the cohort into low risk (COPS2 ≤10 and abLAPS of 0; 79% of all patients), medium risk (COPS2 of 0 to 31 and abLAPS of 0, or COPS2 of 0 to 14 and abLAPS of 1 to 164; 11% of all patients), and high risk (COPS2 ≥32, or COPS2 of 15 to 31 and abLAPS of 1 to 164; 10% of all patients). Supplement Tables 1 and 2 (available at Annals.org) provides more detail on these groupings and on outcomes.
§ White race is the reference group.

Several studies have reported that non-White persons are overrepresented among infected patients, hospitalized patients, and deaths after SARS-CoV-2 infection (17–21). It has also been observed that hospitalization rates for SARS-CoV-2 infection during the acute phase of the pandemic in New York City and Massachusetts were highest in areas with greater African American and Hispanic populations (22, 23). Our study has the unique advantage of being based on a defined population of patients at risk and having followed this cohort through the full range of outcomes from testing to death.

Studies that quantified risk-adjusted, race/ethnicity-specific hospitalization risk have reported similar results to ours. Using Sutter Health data in California, Azar and colleagues (17) found directionally similar results to ours with respect to hospitalization among African American persons (aOR, 2.7 [*P* = 0.007], compared with 1.47 [CI, 1.03 to 2.09] in our cohort), but, in contrast to our study,

Table 4. Percent Relative Contribution of Predictors for COVID-19 Outcomes*

Characteristic	Outcome					
	Testing	Infection	Hospitalization	Admission Severity of Illness (LAPS2)†	Death	
					In-hospital	Ever
Age	0.1	3.4	57.4	77.2	94.0	85.3
Sex	12.8	0.01	9.9	3.8	1.1	4.7
Race/ethnicity	8.1	80.3	4.5	12.8	2.3	0.4
COPS2/abLAPS‡	77.9	6.6	27.6	6.2	0.2	9.4
NDI	1.1	9.7	0.6	0.01	2.5	0.3

abLAPS = abbreviated Laboratory-based Acute Physiology Score; COPS2 = COmorbidity Point Score, version 2; COVID-19 = coronavirus 2019; LAPS2 = abbreviated Laboratory-based Acute Physiology Score, version 2; NDI = neighborhood deprivation index.
* For calculation of the relative contributions, see the Methods sections and references 12 and 13.
† The LAPS2 (Laboratory-based Acute Physiology Score, version 2) (6) is assigned on the basis of a patient's worst vital signs, pulse oximetry, neurologic status, and 16 laboratory test results in the preceding 72 hours. The univariate relationship of an admission LAPS2 with 30-day mortality is as follows: 0 to 59, 1.0%; 60 to 109, 5.0%; ≥110, 13.7%. After age, sex, diagnosis, and comorbid conditions are controlled for, the adjusted odds ratio for inpatient mortality for an increase in LAPS2 of 5 points is 1.134 (95% CI, 1.133–1.135).
‡ Based on the combined contribution of the COPS2 and abLAPS (see the text; Table 3; and Supplement Tables 1 and 2 [available at Annals.org] for details).

they did not find that infected Hispanic persons and Asian persons were at higher risk for hospitalization. Petrilli and coworkers (18) found an elevated hospitalization risk among Hispanic persons only. Like our study, articles providing risk-adjusted race- and ethnicity-specific mortality data in hospitalized patients with SARS-CoV-2 infection have reported that the effect of race/ethnicity is substantially decreased or even absent (20, 24). However, the OpenSAFELY study, which used an at-risk population of 17.3 million patients but did not provide hospitalization data, reported increased COVID-19–related mortality among non-White patients that persisted after risk adjustment (21). More recently, a report using data from the Epic system also showed results broadly similar to ours (25).

Our study expands on the recent literature in other ways. Among patients with SARS-CoV-2 infection, racial/ethnic variation is primarily driven by patients' comorbidity burden, because the risk attributable to race and ethnicity is substantially reduced when comorbidity is added to the models. In our cohort, non-White persons tended to be younger than White persons. However, non-White persons had elevated rates for other risk factors for infection (comorbid conditions and neighborhood deprivation). Many factors may contribute to comorbidity and intrinsic risk (26), including the totality of ways in which societies foster racial discrimination, through mutually reinforcing inequitable systems (structural racism) (27–29). Our data are consistent with the notion that one of the key proximate mediating mechanisms for variation in outcomes among non-White persons is having excess comorbidity (30, 31).

Our observation that admission severity of illness was 6 to 12 points higher in non-White persons points to possible loci for health system interventions. The severity of illness score we used, LAPS2, was based on variables reflecting the 72 hours preceding discharge from the emergency department (that is, when the patient physically entered a non–emergency department hospital unit). Thus, the most likely potential reason for higher scores is differential time to presentation for emergency department or outpatient care, although we cannot exclude other factors that could lead to delays in hospitalization. Understanding temporal factors in patients' decision to seek care, including quantification of possible racial disparities, could lead to changes in advice and referral protocols, particularly if stronger efforts were made to incorporate culturally competent care into such protocols.

Our study has limitations. First, our data derive from the early stage of the pandemic, a period characterized by rapidly changing understanding of the disease and diagnosis and treatment patterns. At present, we cannot quantify the potential sampling biases associated with the change in testing availability that occurred in the latter part (April and May) of our study period. In addition, KPNC, a health system with an unusually high degree of integration, cares for an insured population, so our findings may not be generalizable to other settings with worse access to or coordination of care, nor may they be applicable to other underserved, unemployed populations living in impoverished areas. However, this also permitted us to conduct a study in which the population's access to care was relatively equal. Given time pressure and limited resources, we could not measure other possible factors (such as trust in the health care system) that could affect patients' willingness to interact with the health system.

Nonetheless, our findings are robust and suggest that health systems that aim to decrease racial and ethnic disparities should expand their focus on community interventions to prevent the spread of infection to vulnerable populations, particularly through community education, contact tracing, and public health partnerships. Additional interventions in the initial phase of care before hospitalization, including standardized procedures for initial clinical contact through virtual outreach and culturally sensitive workflows, may also present a critical opportunity to mitigate the effects of racial and ethnic disparities among patients with SARS-CoV-2 infection.

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