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# Contribution of pulmonary diseases to COVID-19 mortality in a diverse urban community of New York

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## Abstract

We examined the relative contribution of pulmonary diseases (chronic obstructive pulmonary disease, asthma and sleep apnea) to mortality risks associated with Coronavirus Disease (COVID-19) independent of other medical conditions, health risks, and sociodemographic factors. Data were derived from a large US-based case series of patients with COVID-19, captured from a quaternary academic health network covering New York City and Long Island. From March 2 to May 24, 2020, 11,512 patients who were hospitalized were tested for COVID-19, with 4,446 (38.62%) receiving a positive diagnosis for COVID-19. Among those who tested positive, 959 (21.57%) died of COVID-19-related complications at the hospital. Multivariate-adjusted Cox proportional hazards modeling showed mortality risks were strongly associated with greater age (HR = 1.05; 95% CI: 1.04–1.05), ethnic minority (Asians, Non-Hispanic blacks, and Hispanics) (HR = 1.26; 95% CI, 1.10–1.44), low household income (HR = 1.29; 95% CI: 1.11, 1.49), and male sex (HR = 0.85; 95% CI: 0.74, 0.97). Higher mortality risks were also associated with a history of COPD (HR = 1.27; 95% CI: 1.02–1.58), obesity (HR = 1.19; 95% CI: 1.04–1.37), and peripheral artery disease (HR = 1.33; 95% CI: 1.05–1.69). Findings indicate patients with COPD had the highest odds of COVID-19 mortality compared with patients with pre-existing metabolic conditions, such as obesity, diabetes and hypertension. Sociodemographic factors including increased age, male sex, low household income, ethnic minority status were also independently associated with greater mortality risks.

## Keywords

COPD, metabolic, sociodemographic, ethnic minority, mortality, Covid-19

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## Introduction

As the World Health Organization declared Coronavirus disease (COVID-19) a pandemic, attention quickly shifted to New York City as it became the epicenter of COVID-19.[1,2] Consternation arose rapidly, as the world watched a parade of patients seeking help to alleviate their severe breathing discomfort, some lying in the hallway of overcrowded hospitals, witnessing healthcare providers frantically attending to those needing urgent care. It became very clear then that the principal reason that the majority of patients sought urgent care was related to shortness of breath, some reporting a history of pulmonary conditions (e.g., chronic obstructive pulmonary disease [COPD], asthma and sleep apnea). In light of such evidence, public health officials began a media campaign advising that individuals should consider seeking care only if they had severe difficulty breathing, particularly if they had pre-existing metabolic conditions (e.g., obesity, hypertension, and/or diabetes).[3]

Early evidence from Wuhan (China) showed that individuals with cardio-metabolic conditions[3] had an elevated risk of severe COVID-19 diagnosis.[1] Moreover, such individuals also had a greater likelihood of death due to associated medical complications.[4–6] Published reports to date have focused on cardio-metabolic burden as the principal driver of COVID-19 morbidity and mortality, lessening the emphasis on potential effects of pre-existing pulmonary diseases. A focus on the effects of pulmonary diseases is warranted as patients with such conditions may be at greater risk for COVID-19 morbidity, over and above risks conferred by metabolic conditions alone. This is consistent with the evidence that patients with such conditions sustain remarkable damage to their lung tissue and/or over-reactive airways, impairing the natural immunity of the airways.[7]

In the first wave, New York City has been one of the largest epicenters of the COVID-19 pandemic. Therefore studying the characteristics of COVID-19 patients in a multi-ethnic city such as New York would yield a wealth of data to examine the contributions of cardio-metabolic burden and pulmonary conditions as potential "at-risk" conditions for COVID-19. In this report, we analyzed data derived from one of the largest existing US-based case series of patients with COVID-19, captured from a large quaternary academic health network covering a large catchment area from New York City and Long Island. Specifically, we assessed the relative contribution of common upper and lower airway pulmonary diseases (COPD, asthma and sleep apnea) in assessing likelihood of COVID-19 -related mortality independent of other medical conditions, health risks, and sociodemographic factors.

## Methods

### *Instrument and participants*

Data were retrieved from a validated electronic health record (EHR; Epic Systems, Verona, WI), which included all inpatient and outpatient visits, beginning on March 2, 2020 and ending on May 24, 2020. COVID-19 testing was performed using the SARS-CoV2 Xpert Xpress assay in the Cepheid GeneXpert instruments. The targets amplified by these assays are the ORF1/a and E in the Roche Cobas assay and N2 and E genes in the Cepheid XpertXpress. Patients were tested if they presented to the emergency department with COVID-19 complaints or as clinically indicated. In the event testing was repeated and was found discordant (i.e. negative test followed by a positive test), we used the positive result. Details regarding procedures for defining positive COVID-19 cases have been published elsewhere.[2]

EHR queries yielded age at time of testing, sex, self-reported ethnic minority aggregated as non-Hispanic black, Asian and Hispanic referenced to non-Hispanic white; cardio-metabolic conditions (e.g., hypertension, hyperlipidemia, diabetes, obesity, peripheral artery disease, and coronary artery disease); history of pulmonary disease (e.g., COPD, sleep apnea, or asthma); autoimmune disease; and cancer. This study was approved by the NYU Grossman School of Medicine Institutional Review Board.

*Determinants.* Factors in our analyses were selected on the basis of prior work on COVID-19 patients. Age at time of testing, sex, race and ethnicity as reported by the patients were included. Median household income quartiles based on patient's 5-digit ZIP code were included as a proxy of socioeconomic status (25th percentile $34,361; 50th percentile $37,580; 75th percentile $41,328).[8] History of preexisting chronic health conditions such as hypertension, diabetes, obesity, hyperlipidemia, coronary artery disease, peripheral artery disease, autoimmune disease and cancer were included. Since the focus of the present analysis was to explore the relative risk of preexisting pulmonary conditions (as captured in EHR), we included upper and lower airway respiratory conditions (i.e., COPD, asthma, and sleep apnea). We defined COPD as the

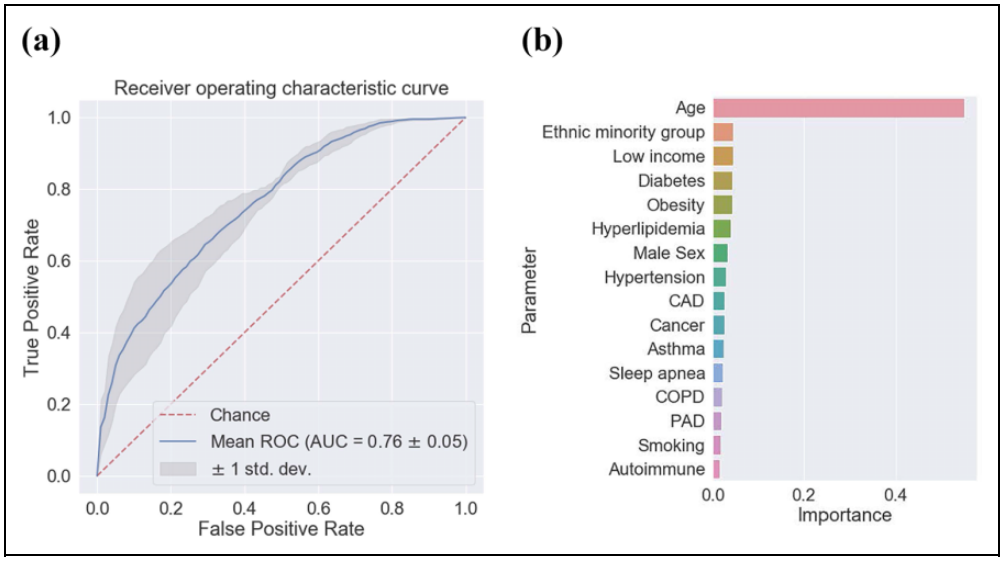


**Figure 1.** ROC curves for classifying patients with COVID-19 on based on status upon discharge. A random forest algorithm with 5-fold cross-validation was used. The model included demographic and clinical data: age, sex, and ethnic minority status, smoking, and preexisting conditions: obesity, hypertension, diabetes, hyperlipidemia, peripheral artery disease, coronary artery disease, autoimmune disease, cancer and pulmonary conditions (i.e., COPD, sleep apnea, and asthma).

presence of chronic bronchitis or emphysema.[9] For patients with COPD or asthma, an effort was made to cross-validate the history of these medical conditions with reported COPD or asthma treatment.

### *Data analysis*

Demographic characteristics of the sample were contrasted using t-tests for continuous variables and $\chi^2$ tests for nominal variables. When non-normal distributions were detected, Kruskal-Wallis test was used. Spearman correlations were used to test the association between continuous variables. Statistical significance was defined by $p < 0.05$. We used Cox proportional hazard modeling to explore the relative importance of each of the predictors in assessing mortality risk. Before constructing the models, we used Random-Forest modeling to select factors that should be entered in the Cox model based on their clinical importance and feature importance in building parsimonious prediction model (Figure 1).[4] In the model, we included all patients testing positive for COVID-19. A random forest classifier algorithm with 3-fold cross-validation was used on Python 3.7 for group prediction. Results of hyperparameter optimization showed that the optimal number of estimators (number of trees in the forest) was 10 and the maximum depth of the tree was 2. For Cox regression analysis, time-to-discharge was calculated using initial time stamp of the admission date and the time stamp on the discharge date. Event was defined based on the patient state (alive or deceased) at the moment of discharge. We included only patients who had been discharged alive or had died, omitting hospitalized patients with unknown state information. No information regarding post-discharge events was available during EHR queries. Anaconda Python 3.7 was used to perform all analysis and plots. Scikit-learn library was used for machine-learning and Lifelines for survival analysis.

## Results

As of May 24 2020, a total of 11,512 patients admitted to the hospital were tested for COVID-19, with 4,446 (38.62%) receiving a positive diagnosis. Among those who tested positive, 959 (21.57%) died of COVID-19-related complications at the hospital. Demographics and clinical characteristics of the sample contrasting patients who were alive or deceased upon discharge are provided in Table 1. Differences in mortality rates were observed across age, ethnic minority status, sex, COPD, hypertension, diabetes, hyperlipidemia, coronary artery disease, peripheral artery disease, cancer, and time-to-discharge. Results of our survival analysis are shown in Figure 1. The area under the curve (AUC) by the Random Forest classifier with 5-fold cross-validation was 0.76 (SD: 0.02) [Panel A]. From the random forest algorithm, age was the most important

**Table 1.** Patients are grouped by outcome upon discharge (alive or deceased). Demographic factors, preexisting conditions, and time to discharge are shown. Household income based on zip code was used to categorize patients by median income quartiles; 25th percentile $34,361; 50th percentile $37,580; 75th percentile $41,328); the lowest quartile was entered in all analyses. Group comparisons were made using ANOVA and Chi-square tests.

Demographic and Clinical Characteristics of Patients with COVID-19 Based on Outcome Upon Discharge (Alive vs Deceased)

| | Value | Alive (n = 3251) | Deceased (n = 959) | p |
|---|---|---|---|---|
| Age, Years (SD) | | 58.7 (18.9) | 72.7 (13.9) | 0.001 |
| Ethnic Minority (%) | No | 1384 (42.6) | 469 (48.9) | 0.001 |
| | Yes | 1867 (57.4) | 490 (51.1) | |
| Male Sex (%) | No | 1414 (43.5) | 352 (36.7) | 0.001 |
| | Yes | 1837 (56.5) | 607 (63.3) | |
| Low Income (%) | No | 2413 (74.2) | 717 (74.8) | 0.767 |
| | Yes | 838 (25.8) | 242 (25.2) | |
| Smoking (%) | No | 3060 (94.1) | 915 (95.4) | 0.148 |
| | Yes | 191 (5.9) | 44 (4.6) | |
| Obesity (%) | No | 1956 (60.2) | 594 (61.9) | 0.342 |
| | Yes | 1295 (39.8) | 365 (38.1) | |
| COPD (%) | No | 3029 (93.2) | 852 (88.8) | 0.001 |
| | Yes | 222 (6.8) | 107 (11.2) | |
| Asthma (%) | No | 2855 (87.8) | 862 (89.9) | 0.091 |
| | Yes | 396 (12.2) | 97 (10.1) | |
| Sleep apnea (%) | No | 3027 (93.1) | 893 (93.1) | 0.949 |
| | Yes | 224 (6.9) | 66 (6.9) | |
| Hypertension (%) | No | 1534 (47.2) | 286 (29.8) | 0.001 |
| | Yes | 1717 (52.8) | 673 (70.2) | |
| Hyperlipidemia (%) | No | 1979 (60.9) | 472 (49.2) | 0.001 |
| | Yes | 1272 (39.1) | 487 (50.8) | |
| Diabetes (%) | No | 2176 (66.9) | 561 (58.5) | 0.001 |
| | Yes | 1075 (33.1) | 398 (41.5) | |
| Peripheral Artery Disease (%) | No | 3119 (95.9) | 878 (91.6) | 0.001 |
| | Yes | 132 (4.1) | 81 (8.4) | |
| Coronary Artery Disease (%) | No | 2879 (88.6) | 751 (78.3) | 0.001 |
| | Yes | 372 (11.4) | 208 (21.7) | |
| Autoimmune Disease (%) | No | 3123 (96.1) | 921 (96.0) | 0.953 |
| | Yes | 128 (3.9) | 38 (4.0) | |
| Cancer (%) | No | 2940 (90.4) | 798 (83.2) | 0.001 |
| | Yes | 311 (9.6) | 161 (16.8) | |
| Time-To-Discharge, Days (SD) | | 8.4 (9.3) | 10.7 (9.4) | 0.001 |

factor, followed by low income, ethnic minority status, diabetes, obesity, hyperlipidemia, male sex, hypertension, cancer, asthma, coronary artery disease, sleep apnea, COPD, peripheral artery disease, smoking status, and autoimmune disease (all sequentially presented from most to least important) [Panel B].

In Table 2, we show the results of the multivariate-adjusted Cox proportional model. Of the demographic factors, age was strongly associated with higher mortality rates (HR = 1.05; 95% CI, 1.04–1.05). Mortality rate was greater for ethnic minority (Asian, Non-Hispanic black, and Hispanic populations combined) (HR = 1.26; 95% CI: 1.10–1.44) and for patients with low median household income (HR = 1.29; 95% CI: 1.11, 1.49), but was higher for those of the male sex (HR = 1.18; 95% CI: 1.03, 1.36). Of the clinical factors, higher mortality rate was associated with obesity (HR = 1.19; 95% CI, 1.04–1.37) and peripheral artery disease (HR = 1.33; 95% CI: 1.05–1.69). Of all the pulmonary conditions, COPD was independently associated with a higher mortality rate (HR = 1.27; 95% CI: 1.02–1.58). Multivariate-adjusted curves from the multivariate models are shown in Figure 2.

**Table 2.** Results of the multivariate-adjusted Cox proportional model predicting COVID-19-associated mortality. Household income based on zip code was used to categorize the patients by median income quartiles; 25th percentile $34,361; 50th percentile $37,580; 75th percentile $41,328); the lowest quartile was entered as a predictor in the model.

| | HR | 95% CI, Lower | 95% CI, Upper | p |
|---|---|---|---|---|
| Age | 1.05 | 1.04 | 1.05 | 0.01 |
| Ethnic Minority | 1.26 | 1.10 | 1.44 | 0.01 |
| Male Sex | 1.18 | 1.03 | 1.36 | 0.02 |
| Low income | 1.29 | 1.11 | 1.49 | 0.01 |
| Smoking | 1.07 | 0.79 | 1.47 | 0.66 |
| Obesity | 1.19 | 1.04 | 1.37 | 0.01 |
| COPD | 1.27 | 1.02 | 1.58 | 0.04 |
| Asthma | 0.83 | 0.67 | 1.04 | 0.10 |
| Sleep apnea | 0.92 | 0.70 | 1.20 | 0.52 |
| Hypertension | 0.91 | 0.77 | 1.07 | 0.24 |
| Hyperlipidemia | 0.92 | 0.79 | 1.06 | 0.25 |
| Diabetes | 0.98 | 0.85 | 1.13 | 0.79 |
| Peripheral Artery Disease | 1.33 | 1.05 | 1.69 | 0.02 |
| Coronary Artery Disease | 1.13 | 0.95 | 1.34 | 0.18 |
| Autoimmune Disease | 0.94 | 0.68 | 1.31 | 0.72 |
| Cancer | 1.10 | 0.92 | 1.31 | 0.29 |

## Discussion

Our observational study identifies determinants of COVID-19-associated mortality among patients residing in New York City. In univariate analyses, we found that advanced age, male sex, patients who were Asian, non-Hispanic black, or Hispanics, and history/diagnosis of COPD, hypertension, diabetes, hyperlipidemia, coronary artery disease, peripheral artery disease, or cancer were more likely to die of COVID-related complications. However, with appropriate adjustment for known confounders, mortality rate was independently associated with advancing age, individuals from ethnic minority groups (Asian, non-Hispanic black, and Hispanic populations), low median household income, male sex, obesity, peripheral artery disease, and COPD. The area under the curve of our machine-learned model was robust and could potentially serve as the basis for developing a risk-score for future mitigation strategies or for prioritization of limited primary and secondary prophylaxis measures including vaccines.

### *Clinical and sociodemographic determinants of COVID-19 mortality*

*Clinical factors.* Our finding that COPD is an independent factor associated with mortality among patients with COVID-19 is novel. Patients with COPD may suffer from greater risk for contracting more severe COVID due to their tenuous respiratory status, the de-implementation of noninvasive ventilation in many intensive care units due to concerns regarding aerosolization risk and greater risk for nosocomial spread to other patients and healthcare workers.[10] Noninvasive ventilation has been shown to reduce mortality in patients with COPD with acute respiratory failure[10] as well as likely over-expression of ACE2 receptors in epithelial cells which serve as targets for the SARS-CoV-2 virus.[11] Moreover, the antiviral immunity is impaired in patients with COPD and likely worsened by inhaled corticosteroids that are often prescribed to such patients.[12,13] A limitation of our study is that we do not have GOLD classification of the severity of COPD, although our findings of increased mortality risk among patients with COPD was independent of corticosteroid administration.

The COVID-19 mortality risks associated with obesity and heart disease are consistent with published findings. In two parallel studies in New York City, investigators observed that both obesity and heart failure were predictive of mortality (HR = 1.5; 95% CI: 1.0 to 2.2; HR = 1.9; 95% CI: 1.4 to 2.5).[2] Likewise, results of a multivariate Cox model revealed that in addition to increased age (aHR = 1·31; 95% CI: 1·09–1·57 per 10-year increase) and chronic pulmonary disease (aHR = 2·94; 95% CI: 1·48–5·84), chronic cardiac disease was strongly associated with mortality (aHR = 1·76; 95% CI: 1·08–2·86).[14] Several other investigations conducted in varying US cities and across the globe have documented these associations,

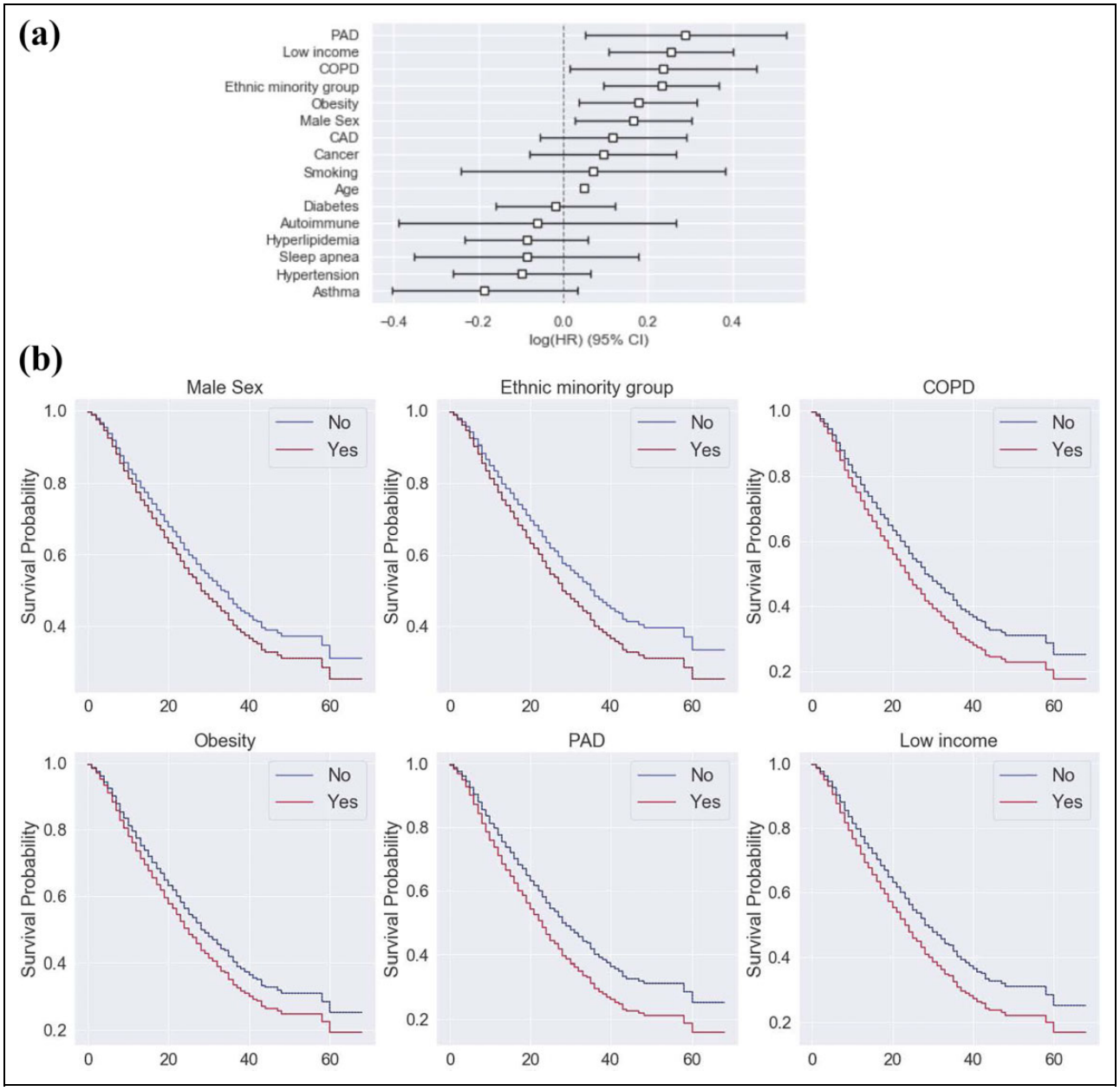


**Figure 2.** Results of the survival analysis. (Top panel) Hazard ratios and (Bottom panel) Kaplan-Meier estimated mortality curves for patients with covid-19 with contrast for low income, male sex, ethnic minority status (Black, Asian, and Hispanic vs non-Hispanic White, and a history of COPD, obesity and PAD.

rendering them ubiquitous features of COVID-19 manifesfation.[15–18] Our study makes a unique contribution to the literature in that it demonstrates that above and beyond the effects of other medical comorbidities, COPD seems the most important driver of mortality risks among the three pulmonary conditions we investigated.

*Sociodemographic factors.* Both older age and male sex were positively and independently associated with COVID-19 deaths in our sample. Older age was associated with a greater likelihood of COVID-19 mortality relative to younger individuals. The observed age-related mortality has been reported previously and may be attributable to the presence of various medical comorbidities (including hypertension, diabetes mellitus, and coronary artery disease) among older individuals or may be related to immune senescence.[13,15–18] Compared to females, males had a greater likelihood of COVID-19 deaths. Males infected with SARS-CoV-2

virus seem more likely to develop severe disease and die.[19] This is line with global data, showing that the case fatality ratio is greater among males, relative to females.[19] Such sex differences in mortality may be attributable to underlying biological (such as greater burden of cardiovascular disease) or high-risk behaviors (delayed medical attention, alcohol consumption or smoking). Genetic factors that relate to X-chromosome containing greater density of immune genes and females having more Angiotensin Converting Enzyme 2 (ACE2) genes by virtue of ACE-2 being on the X-chromosome may confer stronger innate immune response as protection against acute lung injury, respectively.[20] Lastly, greater estrogen receptor stimulation among females may reduce levels of systemic inflammation.[19]

We also observed a significant increase in mortality risk among ethnic minorities. While many other U.S. reports did not examine ethnic minority and socioeconomic status as determinants of COVID-19-related mortality in a comprehensive manner,[21–25] a few studies have examined effects of ethnicity, adjusting for variation in income, finding an increased risk for hospitalization among patients of black ethnicity.[26] Data from an integrated healthcare system in California revealed no mortality differences based on ethnic minority status, but that study might have been underpowered.[26] To our knowledge, ours is the largest study of COVID-19 that involved a machine-learning approach that revealed potential ethnic minority health disparities in COVID-19 related mortality. Such disparities in health and healthcare are not new; they are well documented and continue to persist especially in critical illnesses.[27] Indeed, in a large study of claims-based data from the Healthcare Cost and Utilization Project spanning from 2008 to 2012, blacks, Hispanics, and other ethnic minorities exhibited higher in-hospital mortality from sepsis-related respiratory failure compared with non-Hispanic whites.[27] Explanations for the observed ethnic minority differences in mortality rates could be that ethnic minorities typically exhibit greater comorbidities, may experience delayed or inadequate medical treatment due to limited health coverage, limited health literacy, and lack of access to quality medical care. Regarding access to care, it's notable that our finding of ethnic minority differences was independent of income although we did not determine other access to care issues such as insurance type or provider-availability.[15] Independent of healthcare system factors, there are genetic factors that confer race-based susceptibility to acute lung injury that have been well described.[16–18] Notwithstanding these observations, caution needs to be exercised as to how our findings are contextualized, which otherwise could create or potentiate harmful myths and perceptions that may unintentionally undermine the very same goal of dispelling health disparities.[14]

### Implications

Our findings provide a robust machine-learned model that can serve as the basis for developing a COVID-19 risk-stratification score, especially for patients who have a history of respiratory illness. The development and validation of a COVID-19 risk-stratification score that can predict mortality would be valuable for directing limited resources that include medications and vaccines aimed at improving outcomes in patients with COVID. Although such a score has been developed to predict ICU utilization[9] a population-based score that can predict mortality would be valuable for public and population health interventions aimed at limiting COVID-19 infections, transmissions and mortality rates.

### Limitations

Despite the significance of our findings, results should be interpreted cautiously given a few methodological limitations. First, our data were collected with the primary intent of clinical care. Therefore, a few critical variables were missing from our dataset that might be informative for future analyses. For example, mortality might be somewhat suppressed because it is likely that some patients who were discharged may have subsequently died; this was not available in EHR queries. Likewise, data regarding other respiratory conditions likely to play a role in Covid-19 morbidity were not available. We were also unable to ascertain whether mortality risks associated with COPD might have been partially influenced by clinicians' decision not to use ventilation in certain cases. Future studies should have a more robust follow-up and longitudinal data collection framework. Second, income was based on the median income of the patient's ZIP code data, which is a crude representation of patients' socioeconomic status. In the same vein, we were unable to investigate the association between other proxies of socioeconomic status and COVID-19 mortality, such as social vulnerability and social determinants of health, which have been linked to poor, differential health outcomes among racial/ethnic minorities.

## Conclusion

Our findings indicate that patients with COPD had the highest odds of COVID-19 mortality compared with patients who had pre-existing metabolic conditions. Indeed, even among all the pulmonary conditions, COPD was the strongest independent predictor of mortality, which represents a meaningful contribution to the extant literature. In a way, this seems to have overwhelmed the evidence of deleterious effects of obesity on COVID-19 mortality. Based on our findings, it appears that only COPD as a lower airway respiratory condition is significantly associated with COVID-19 mortality, as smoking or asthma history were not significantly associated with mortality. Indeed, our survival analysis demonstrated that the peak of cumulative COVID-19 mortality rates among patients with COPD occurs much sooner (within the first 20 days of receiving a positive diagnosis) compared with those without a COPD history (within the first 50 days of receiving a positive diagnosis), suggesting patients with COPD are more likely to die sooner. In addition to the clinical conditions, several sociodemographic factors—increasing age, low income, ethnic minority status (non-Hispanic Black, Asian and Hispanic populations), and being male—were independently associated with COVID-19 mortality. These findings add to our current understanding of factors that should be considered in developing COVID-19 risk-stratification strategies.

### Author contribution

GJL conceptualized, designed and led the group in drafting the manuscript. JC and GA processed and analyzed the data and prepared tables and figures. AS, RO, MR, and SP helped to develop the scientific arguments and contributed to data interpretation. All authors made substantial contributions to conception and design, acquisition of data, or analysis and interpretation of data; took part in drafting the article or revising it critically for important intellectual content; agreed to submit to the current journal; gave final approval of the version to be published; and agree to be accountable for all aspects of the work.

### Data availability statement

The dataset utilized for analyses of the current study are available from the corresponding author upon reasonable request.

### Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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### Ethical approval

All procedures performed in this study involving human participants were completed in accordance with the ethical standards of the NYU Langone Health Institutional Review Boards and with the Helsinki declaration and its amendments.

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