Pre-COVID-19 lung function and other risk factors for severe COVID-19 in first responders

To the Editor:

Starting 1 March 2020, New York City (NYC) suffered an intense coronavirus disease 2019 (COVID-19) outbreak caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). By 31 May 2020, there were 203,248 diagnosed patients, 52,301 hospitalisations and 17,756 confirmed deaths in NYC [1]. Cardiac arrests with resuscitation increased three-fold compared with the same time period in 2019 [2]. Despite proper personal protective equipment (PPE) and compliance with Centers for Disease Control guidelines [3], medical leave for suspected/confirmed COVID-19 in Fire Department of the City of New York (FDNY) responders was substantial, affecting 40.7% of emergency medical service providers (EMS) and 34.5% of firefighters [4]. We examined risk factors for COVID-19 diagnosis and severe COVID-19 disease (hospitalisation or death) in FDNY responders.

Our study population included FDNY firefighters and EMS who were actively employed on 1 March 2020 and had received ≥2 pulmonary function tests (PFTs) between their hire date and 29 February 2020. COVID-19 diagnoses, hospitalisations and deaths between 1 March 2020 and 31 May 2020 were obtained from the FDNY electronic medical record. Responders who had a positive nucleic-acid COVID-19 test had confirmed COVID-19, and those with reported symptoms consistent with COVID-19 but not tested were classified as suspected COVID-19. Additionally, those who had both a confirmed diagnosis and a COVID-19-related hospitalisation or death were classified as severe COVID-19. Multivariate logistic regression assessed whether class of employment (firefighter or EMS), longitudinal rate of forced expiratory volume in 1 s (FEV1) decline/year between participants’ first PFT and 1 March 2020, smoking history, age, sex, race, body mass index (BMI), blood glucose and high-density lipoprotein cholesterol levels were associated with either severe COVID-19. Analyses were performed using SAS, version 9.4 (SAS Institute, Cary, NC, USA). We followed STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) reporting guidelines. The Montefiore Medical Center/Albert Einstein College of Medicine Institutional Review Board approved this study (#2020-11983).

Of 15,670 FDNY responders employed on 1 March 2020, the study population (n=14,290) comprised 3,501 EMS and 10,789 firefighters. Participants had a mean age of 40.4±9.2 years, a mean BMI of 29.8±4.5 kg·m−2, and were 8.0% female, 67.8% white, 11.2% black, 16.6% Hispanic and 4.4% other races/ethnicities. Sixty-eight per cent were never-smokers, 25.1% were former-smokers, 4.1% current-smokers and 2.9% had missing smoking status. The mean±SD number of FEV1 measurements used in calculating FEV1 decline was 10.9±4.9, and the mean duration was 11.9±6.1 years. From 1 March 2020 to 31 May 2020, 9,115 responders had no COVID-19 diagnosis, 5,175 (362.1 out of 1000) were confirmed or suspected COVID-19 cases, and 62 (4.4 out of 1000) were hospitalised. Three participants died in hospital and one died at home (0.3 out of 1000). Responders aged 18–44 had a lower rate of COVID-19-related hospitalisations (3.0 out of 1000) and experienced no COVID-19-related deaths, despite having an elevated infection rate (405.3 out of 1000). EMS had more cases of severe COVID-19 than firefighters (42 out of 3,501 (1.2%) versus 21 out of 10,789 (0.2%);
Fisher's exact p-value <0.001). EMS were slightly younger than firefighters (mean±SD: 38.7±10.2 versus 41.0±8.8 years) and had a larger proportion of females (29.3% versus 1.1%) and non-white individuals (60.7% versus 22.9%). After adjustment for confounders, multivariate logistic regression showed that EMS had a 4.23-fold increased odds of severe disease (95% CI 2.20–8.15) when compared with firefighters (table 1). EMS also had a modest increase in odds of confirmed and a non-significant increase in odds of suspected COVID-19 (table 1).

A greater rate of FEV₁ decline prior to 1 March 2020 was associated with severe COVID-19 but was not associated with confirmed or suspected COVID-19 infection. Compared with never-smokers, ever-smokers did not have increased odds of severe or confirmed COVID-19. Higher age, BMI, blood glucose level and non-white race were independent risk factors for severe COVID-19.

Overall, between 1 March 2020 and 31 May 2020 we observed that the COVID-19 infection rate among FDNY responders was 362 out of 1000 – 15 times the NYC rate (24 out of 1000) [1]. EMS had over a four-fold increased risk of severe COVID-19 and a 26% increased risk of confirmed COVID-19 disease when compared with firefighters. It is likely that their higher-intensity COVID-19 exposures contributed to the increased risk and severity of COVID-19 illness. Although both FDNY firefighters and EMS responded to the pandemic-related surge in 9-1-1 emergency medical calls and followed the same PPE protocols [4], EMS had greater COVID-19 exposure than firefighters due to greater COVID-19-related call volume and being solely responsible for patient transport, nebulisation of bronchodilators and intubation.

Despite having a higher COVID-19 case rate than NYC, FDNY responders had a lower rate of severe COVID-19 than the city as a whole (4.1 out of 1000 versus 6.2 out of 1000 hospitalisations, and 0.3 out of 1000 versus 2.1 out of 1000 deaths) [1]. This may be due to the healthy worker effect, i.e. lower prevalence of comorbidities in FDNY responders than the general NYC population. The lower hospitalisation and death rates observed in this study may be a result of differences in the age distributions of the FDNY cohort and the general NYC population. The COVID-19-related hospitalisation rate in FDNY responders aged 18–44 was similar to that of NYC residents in the same age group (3.0 out of 1000 versus 2.8 out of 1000), though the infection rate was over 15-fold higher (405.3 out of 1000 versus 25.5 out of 1000) [1].

As seen in other healthcare worker cohorts, higher age was associated with lower risk of COVID-19 infection [5]. This may be due to age-related changes in behaviour. Alternatively, since immunity to coronaviruses increases with age [6], cross-reactive cellular immunity to other coronaviruses may provide protection from contracting SARS-Cov-2 [7].

The FDNY longitudinal medical monitoring programme enabled assessment of FEV₁ decline rate prior to the COVID-19 pandemic as a potential risk factor. We previously found that greater FEV₁ decline rates were associated with asthma and COPD in World Trade Center-exposed FDNY responders [8]. In our current study, we observed greater FEV₁ decline rates to be associated with severe COVID-19 disease. Further investigation of parameters of lung health associated with severe COVID-19 are needed in other longitudinal cohorts. Interestingly, we observed confirmed COVID-19 diagnosis was not associated with smoking and that ever-smoking was less prevalent in severe COVID-19 disease, a surprising finding as

### TABLE 1 Multivariate logistic regression model predicting coronavirus disease 2019 (COVID-19)-related outcomes

| Variables                  | Hospitalisation/death versus no COVID-19 diagnosis | Confirmed COVID-19 versus no COVID-19 diagnosis | Suspected COVID-19 versus no COVID-19 diagnosis |
|----------------------------|---------------------------------------------------|------------------------------------------------|-----------------------------------------------|
|                           | OR (95% CI) p-value                               | OR (95% CI) p-value                               | OR (95% CI) p-value                             |
| Subjects n                 | 63                                                | 1569                                             | 3543                                           |
| EMS versus firefighter     | 4.23 (2.20–8.15) <0.001                           | 1.28 (1.10–1.49) 0.001                           | 1.07 (0.96–1.20) 0.228                         |
| FEV₁ decline per 100 mL·year⁻¹ | 1.70 (1.12–2.59) 0.012                          | 0.93 (0.81–1.06) 0.265                           | 1.01 (0.92–1.11) 0.803                         |
| Ever-smoker                | 0.46 (0.25–0.86) 0.014                            | 0.99 (0.88–1.12) 0.920                           | 1.14 (1.04–1.24) 0.005                         |
| Age per 10 years           | 1.59 (1.20–2.10) 0.001                            | 0.79 (0.74–0.84) <0.001                          | 0.68 (0.65–0.71) <0.001                        |
| Male sex                   | 1.55 (0.60–4.02) 0.365                            | 1.12 (0.88–1.44) 0.355                           | 1.07 (0.90–1.29) 0.438                         |
| Non-white race             | 2.46 (1.34–4.51) 0.004                            | 1.21 (1.06–1.38) 0.004                           | 1.24 (1.13–1.37) <0.001                        |
| BMI                        | 1.09 (1.05–1.13) <0.001                           | 1.02 (1.00–1.03) 0.029                           | 1.00 (0.99–1.01) 0.665                         |
| Blood glucose mmol·L⁻¹     | 1.13 (1.02–1.25) 0.015                            | 0.95 (0.89–1.01) 0.093                           | 1.01 (0.97–1.05) 0.626                         |
| HDL cholesterol mmol·L⁻¹   | 0.38 (0.15–0.97) 0.042                            | 1.11 (0.94–1.30) 0.222                           | 0.95 (0.84–1.07) 0.359                         |

n=13879 due to missing covariates. Model adjusted for first forced expiratory volume in 1 s (FEV₁) measurement after hire date. Between 1 March 2020 and 31 May 2020. OR: odds ratio; EMS: emergency medical service providers; BMI: body mass index; HDL: high-density lipoprotein.
smoking increases expression of the SARS-CoV-2 receptor in human lung epithelium [9, 10]. However, the impact of smoking on COVID-19 is complex, and while our findings are consistent with ecological epidemiology studies where smoking was less prevalent in hospitalised patients, further studies are needed before reaching any conclusion [11–14]. Lastly, similar to other reports, we found that non-white race was strongly associated with COVID-19-related hospitalisation [15].

This study has limitations. The data are derived from a single occupational cohort, limiting its generalizability. As with all observational studies, there may be residual confounding. Since molecular testing for COVID-19 was limited early on in the NYC outbreak, a majority of FDNY responders who had symptoms of COVID-19 did not have a confirmed COVID-19 diagnosis via a positive nucleic acid test. The similarity in risk factors for suspected and confirmed COVID-19 in this cohort lends support to the accuracy of symptom screening for COVID-19 diagnosis in high prevalence settings; however, respiratory symptoms related to smoking or seasonal influenza may reduce the specificity of suspected COVID-19 diagnosis. These limitations are balanced by the strength of a closed cohort with extensive information from prior monitoring exams and with paid medical leave, reducing the likelihood of selection bias as well as ascertainment biases.

In summary, our study emphasises the elevated risk of disease present in FDNY responders during the COVID-19 pandemic, particularly EMS and those with greater pre-pandemic lung function decline. To protect healthcare workers, including pre-hospital responders, requirements of proper PPE use, environmental controls, influenza vaccination (to minimise additional medical leave, mischaracterisation and potential additional comorbidity) and COVID-19 vaccination, when available, are all critical. Without these measures, healthcare systems will be unable to maintain surge capacity during this pandemic.

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