

Abstract

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which exploded in Wuhan (Hebei Region, China) in late 2019, has later spread around the world, causing pandemic effects on humans. During the first wave of the pandemic, Italy, and especially its Northern regions around the Po Valley, faced severe consequences in terms of infected individuals and casualties (more than 31,000 deaths and 255,000 infected people by mid-May 2020). While the spread and effective impact of the virus is primarily related to the lifestyles and social habits of the different human communities, environmental and meteorological factors also play a role. Among these, particulate pollution may directly impact the human respiratory system or act as virus carrier, thus behaving as potential amplifying factor in the pandemic spread of SARS-CoV-2. Enhanced levels of PM2.5 and PM10 particles in Northern Italy were observed over the 2-month period preceding the virus pandemic spread. Threshold levels for PM10 (< 50 µg/m³) were exceeded on 20–35 days over the period January–February 2020 in many areas in the Po Valley, where major effects in terms of infections and casualties occurred, with levels in excess of 80 µg/m³ occasionally observed in the 1–3 weeks preceding the contagious activation around February 25, 2020. Threshold values for PM2.5 indicated in WHO air quality guidelines (< 25 µg/m³) were exceeded on more than 40 days over the period January–February 2020 in large portions of the Po Valley, with levels up to 70 µg/m³ observed in the weeks preceding the contagious activation. In this paper, PM10 particle measurements are compared with epidemiologic parameters' data. Specifically, a statistical analysis is carried out to correlate the infection rate, or incidence of the pathology, the mortality rate, and the case fatality rate with PM concentrations. The study considers epidemiologic data for all 110 Italian provinces, as reported by the Italian Statistics Institute, over the period 20 February–31 March 2020. Corresponding PM10 concentrations covering the period 15–26 February 2020 were collected from the network of air quality monitoring stations run by different regional and provincial environment agencies. The case fatality rate is found to be highly correlated to the average PM10 concentration, with a correlation coefficient of 0.89 and a slope of the regression line of  $(6.7 \pm 0.3) \times 10^{-3} \text{ m}^3/\mu\text{g}$ , which implies a doubling (from 3 to 6%) of the mortality rate of infected patients for an average PM10 concentration increase from 22 to 27 µg/m³. Infection and mortality rates are also found to be correlated with PM10 concentrations, with correlation coefficients being 0.82 and 0.80, respectively, and the slopes of the regression lines indicating a doubling (from 1 to 2%) of the infection rate and a tripling (from 0.1 to 0.3%) of the mortality rate for an average PM10 concentration increase from 25 to 29 µg/m³. Considerations on the exhaled particles' sizes, their concentrations and residence times, the transported viral dose and the minimum infective dose, in combination with PM2.5 and PM10 pollution measurements and an analytical microphysical model, allowed assessing the potential role of airborne transmission through virus-laden PM particles, in addition to droplet and the traditional airborne transmission, in conveying SARS-CoV-2 in the human respiratory system. In specific circumstances which can be found in indoor environments, the number of small potentially infectious particles coalescing on PM2.5 and PM10 particles is estimated to exceed the number of infectious particles needed to activate COVID-19 infection in humans.

Assessment of the potential role of airborne transmission

An analytical model has been developed in order to get a rough quantitative assessment of the potential role of airborne transmission through virus-laden PM particles in conveying SARS-CoV-2 in the human respiratory system and trigger COVID-19. The purpose of the model is to assess this additional transmission vehicle with a specific attention to indoor conditions. A proper simulation of this process requires specific information on exhaled particles' sizes and concentrations, their residence time, transported viral dose, and minimum infective dose. The model simulates particle collection efficiency, accounting for the combined effect of collision and coagulation efficiencies in the formation of virus-transmitting PM particles. The model provides a quantitative assessment of the role of Brownian diffusion, laminar shear, turbulent fluctuations, and gravitational and drag forces. Concerning the transported viral dose and the minimum infective dose, only limited virological information is available in the open international literature specifically for SARS-CoV-2. However, for the purpose of getting a rough estimate of the importance of airborne transmission through virus-laden PM2.5 and PM10 particles, missing specific information on the above quantities for SARS-CoV-2 can be replaced with analogous information from other viral pathogens. This obviously leads to results affected by a large degree of uncertainty. The developed analytical model relies on the following assumptions/hypotheses.

- Outdoor PM2.5 and PM10 concentrations are assumed to be 60 and 70 µg/m³, respectively; these values are representative of the high particulate pollution conditions observed in Northern Italy in the weeks preceding the pandemic outbreak.
- Outdoor PM2.5 and PM10 concentrations are assumed to be also present indoor. In this regard, it is to be specified that PM2.5 and PM10 pollution concentrations in outdoor environments are frequently found to be highly correlated with indoor concentrations (Mohammed et al. 2015; Pallarés et al. 2019; Saramak 2019), and are often characterized by comparable concentrations (Massey et al. 2009). Thus, the presence of high outdoor PM2.5 and PM10 pollution levels is likely to translate into high indoor PM2.5 and PM10 concentrations, which is what we assume.
- SARS-CoV-2 is assumed to remain viable in aerosols for ~ 3 h in indoor conditions, with a half-life of 1.1 h and a reduction in infectious titer from 10<sup>3.5</sup> to 10<sup>2.7</sup> TCID50 per liter of air (van Doremalen et al. 2020).
- The considered indoor space has a volume of 50 m³ and includes 5 individuals, all of them but one asymptomatic infected patients.
- 80% of the particles exhaled during breathing or speaking are assumed to have diameters smaller than 1 µm (see above in Section 3.1 in more detail: Wan et al. 2014; Fairchild and Stampfer 1987; Papineni and Rosenthal 1997), while 40% of the particles expelled during coughing are assumed to have diameters smaller than 1 µm (Lindsley et al. 2010).
- The number of particles exhaled on tidal breathing by subjects infected by a respiratory virus is assumed to be 5000 per liter (7200 particles per liter were reported by Fabian et al. (2011) for human rhinovirus (HRV), while 4644 particles per liter were reported by Wan et al. (2014) for mechanically ventilated patients affected by pneumonia); the number of particles produced on coughing by influenza-infected patients was estimated to be 75,400 particles per cough (Lindsley et al. 2012), which is the number assumed in the model.
- Tidal volume of human breath and respiratory rate are assumed to be 500 ml and 15 breaths/min, respectively (Carroll 2007; Riediker and Tsai 2020); thus, 3-h pulmonary ventilation corresponds to a volume of 1350 l; coughing volume and rate are assumed to be 250 ml and 2 coughs/min, respectively (Hsu et al. 1994); thus, air expelled during 3-h coughing corresponds to a volume of 90 l.
- The velocity of particles produced on breathing and coughing is assumed to be 1 and 10 m s<sup>-1</sup>, respectively (breathing particle velocities in the range 1–7 m s<sup>-1</sup> were estimated by Tsuda et al. (2013), while coughing particle velocities in the range 10–30 m s<sup>-1</sup> and in the range 20–90 m s<sup>-1</sup> were estimated by Bourouiba (2020) and La Rosa et al. (2013), respectively).

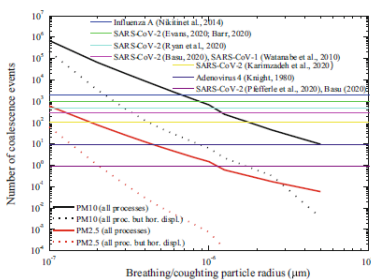


Fig. 1 Overall number of coalescing events of breathing and coughing particles on PM2.5 (red lines) and PM10 (black lines) particles as a function of the size of the breathing and coughing particles. Two lines are present for each particle size: one (solid line) representing the overall number of coalescing events associated with all considered coagulation processes, the other one (dotted line) representing the number of coalescing events when excluding the coagulation process associated with the primarily horizontal component of the initial motion of exhaled particles. The horizontal lines in the figure represent the minimum infectious doses of SARS-CoV-2 and other respiratory viruses

$K_a = \frac{2\lambda}{d}$  the motion of the suspended particles considered in the present study is governed by the continuum regime equations

The continuum regime characterizes particles which are large compared to the mean free path of the suspending gas ( $Kn \ll 1$ ).

**Number of colliding aerosol particles in continuum regime**

$$N_{ab} = \alpha \times \beta(a, b) \times n_a \times n_b$$

$$\alpha = \left( \frac{x}{r_a + r_b} \right)^2$$

**Brownian motion term**

$$\beta_B(a, b) = 4\pi(r_a + r_b)(D_a + D_b)$$

$$D_{a/b} = \frac{kT C_c}{6\pi\mu r_{a/b}}$$

**Laminar fluid shear term**

$$\beta_{LS}(a, b) = 1.33G(r_a + r_b)^3$$

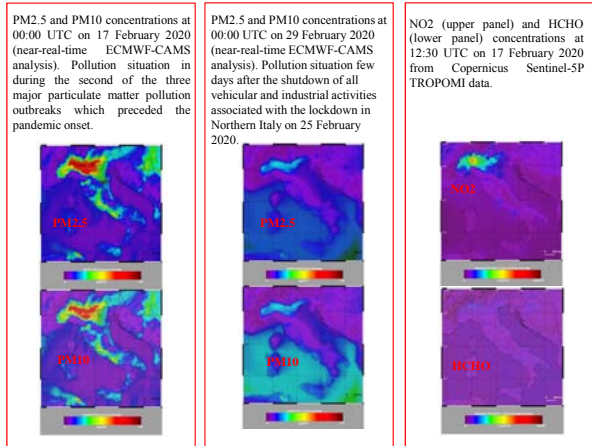
**Turbulent fluid shear term**

$$\beta_{TS}(a, b) = 1.3\mu(r_a + r_b)^3$$

**Differential sedimentation term**

$$\beta_S(a, b) = \pi(r_a + r_b)^2 |v_a - v_b|$$

**Terminal settling velocity**

$$v_{a/b} = \frac{r^2(\rho - \rho_0)g C_c}{9\mu}$$


Near-real-time ECMWF-CAMS analysis of PM2.5 (upper panel) and PM10 (lower panel) concentrations over the month of February 2020 for several metropolitan cities in Lombardia (Bergamo, Brescia, Cremona, Milano, Monza, and Pavia). One to three locations are considered for each city

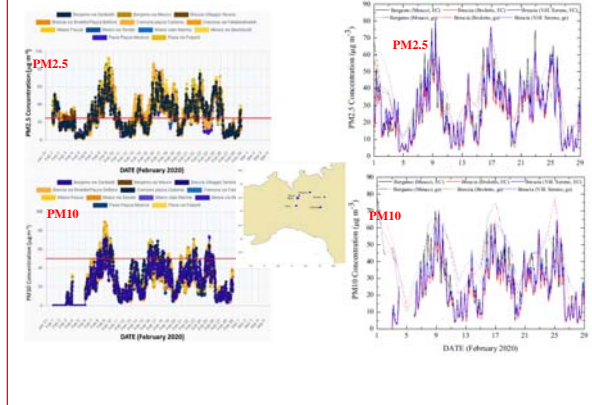


Table 1 List of all 110 Italian provinces, their positions, and their population density. The province is identified by its name, its location (Region, Province, and Municipality), and its population density (inhabitants/km²).

Province	Region	Province	Province	Province	Province	Province	Province	Province	Province
Aosta Valley	Abruzzo	Basilicata	Basilicata	Basilicata	Basilicata	Basilicata	Basilicata	Basilicata	Basilicata

Comparison of epidemiologic parameters in the period 20 February–31 March 2020 with the corresponding average PM10 concentration values in the period 15–26 February 2020 for all 110 Italian provinces. A linear fit is applied to the data, using a linear regression function with the form  $Y = A + B \times X$ , with X being the average PM10 concentration values and Y the corresponding epidemiologic parameter values. Values of PM10 concentration variability within each province territory, i.e., its standard deviation, are included as error bars AX and are used as a weighting factor in the statistical analysis.

**Case fatality rate vs. PM10 concentration**

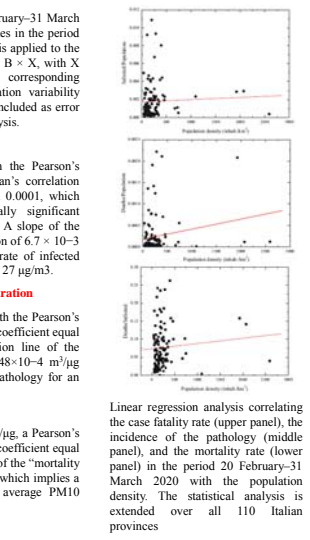
$A = (-0.1233 \pm 0.002)$ ,  $B = (6.7 \pm 0.3) \times 10^{-3} \text{ m}^3/\mu\text{g}$ , with the Pearson's correlation coefficient being equal to 0.89 and the Spearman's correlation coefficient being equal to 0.87. The p value is smaller than 0.0001, which indicates less than 0.01% probability that no statistically significant relationship is present between the two compared quantities. A slope of the regression line of the "case fatality rate" vs. PM10 concentration of  $6.7 \times 10^{-3} \text{ m}^3/\mu\text{g}$  implies a doubling (from 3 to 6%) of the mortality rate of infected patients for an average PM10 concentration increase from 25 to 27 µg/m³.

**Incidence of the pathology vs. PM10 concentration**

$A = (-5.6 \pm 0.1) \times 10^{-3}$  and  $B = (2.48 \pm 0.06) \times 10^{-4} \text{ m}^3/\mu\text{g}$ , with the Pearson's correlation coefficient equal to 0.82, a Spearman's correlation coefficient equal to 0.79, and p value < 0.0001. The slope of the regression line of the "incidence of the pathology" vs. PM10 concentration of  $2.48 \times 10^{-4} \text{ m}^3/\mu\text{g}$  implies a doubling (from 1 to 2%) of the incidence of the pathology for an average PM10 concentration increase from 25 to 29 µg/m³.

**Mortality rate vs. PM10 concentration**

$A = (-1.25 \pm 0.03) \times 10^{-3}$  and  $B = (5.46 \pm 0.14) \times 10^{-5} \text{ m}^3/\mu\text{g}$ , a Pearson's correlation coefficient equal to 0.80, a Spearman's correlation coefficient equal to 0.79, and p value < 0.0001. The slope of the regression line of the "mortality rate" vs. PM10 concentration is  $(5.46 \pm 0.14) \times 10^{-5} \text{ m}^3/\mu\text{g}$ , which implies a tripling (from 0.1 to 0.3%) of the mortality rate for an average PM10 concentration increase from 25 to 29 µg/m³.



Linear regression analysis correlating the case fatality rate (upper panel), the incidence of the pathology (middle panel), and the mortality rate (lower panel) in the period 20 February–31 March 2020 with the population density. The statistical analysis is extended over all 110 Italian provinces

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Assessment of the potential role of atmospheric particulate pollution and airborne transmission in intensifying the first wave pandemic impact of SARS-CoV-2/COVID-19 in Northern Italy

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