ISSN 1075-7007, Studies on Russian Economic Development, 2021, Vol. 32, No. 4, pp. 357–363. © Pleiades Publishing, Ltd., 2021.
Russian Text © The Author(s), 2021.

ECONOMY AND ECOLOGY

The COVID-19 Pandemic: New Knowledge on the Impact of Air Quality on the Spread of Coronavirus Infection in Cities

B. A. Revich[a] and D. A. Shaposhnikov[a, *]

[a] *Institute of Economic Forecasting, Russian Academy of Sciences, Moscow, Russia*

**e-mail: dshap2014@gmail.com*

Received March 1, 2021; revised March 4, 2021; accepted March 15, 2021

Abstract—Suspended substances in the air form clusters with coronavirus particles and spread up to 10 m from the source of infection. The importance of taking air pollution into account when simulating the spread of the COVID-19 pandemic is due to the similarity in the health impacts made by the coronavirus and the respirable fraction of suspended solids. In Europe, up to 6.6% of deaths and in China up to 11% of deaths from COVID-19 are due to ambient air pollution. In epidemiological models, this factor must be considered along with other reasons for the development of coronavirus infection. In Russian cities, increased air pollution can be one of the risk factors for the development of an epidemic.

Keywords: Coronavirus infection, risk factors, atmospheric air pollution, fine particles

DOI: 10.1134/S1075700721040134

Specifics of the COVID-19 pandemic, the high virulence of this virus, i.e., the number of viral particles required to infect the body, and contagiousness caused infection of more than 118 million people and excess mortality of three million people worldwide, and in Russia, 4.3 million people and about 400 thousand cases, respectively (as of March 15, 2021). The pandemic of the new coronavirus SARS-CoV-2 has presented the scientific community with many unexplored aspects of this situation. This called for its in-depth study not only by physicians and biologists but also by economists, urbanists, geographers, meteorologists, climatologists, educators, sociologists, psychologists, cultural scientists, etc. For the first time in the history of mankind, this pandemic has created such an epidemiological situation, which during its course is studied from completely different positions: not only that of natural science but also political, humanitarian and others. Understanding the historical significance of previous pandemics became possible only centuries later. Some historians believe that the plague of the 14th century not only led to a demographic catastrophe, but also caused profound changes in the foundations of medieval society. The epidemic resulted from the interaction between the civilizations of the West and the East with all their political, social, religious, and spiritual contradictions after the short-term resumption of the "Great Silk Road" [1]. It is difficult to say what the consequences of today's pandemic will be, but they can significantly change attitudes towards those additional health risk factors that can be identified, corrected, and managed. First of all, these are the factors affecting the mobility and quality of air masses in urban development, i.e., ecological and geographical features of the area, compactness of buildings, population density, intensity of traffic flows, emissions from stationary sources, including power plants. These factors affect the quality of atmospheric air, the pollution of which contributes to the development of pathological processes in the human body.

It is extremely important that there is a certain similarity between the effects of the SARS-CoV-2 virus and fine suspended particles less than 10 μm in size (PM_{10}): in addition to solid microparticles, these aerosols also include tiny droplets of liquids. The content of PM_{10} in atmospheric air is the main indicator of its quality; this indicator is used to assess the consequences of the impact of polluted urban air on the health of the population. The toxicological properties of PM, its content in the air of various cities, and the effects of exposure on various health indicators are considered in our review [2]. Prolonged exposure to urban air with an increased content of such particles leads to excess mortality of the population, which, according to our estimates, in Russia reaches 68–88 thousand cases per year, and the highest level of air pollution is characteristic for the cities located in the Asian part of the country that use coal [2]. PM_{10} levels in Russian cities are roughly the same as in industrialized European countries such as Germany. The groups

at increased risk of the exposure to polluted atmospheric air, as well as with exposure to the virus, are the elderly, children, and pregnant women. Fine suspended particles are risk factors for the development of atherosclerosis, myocardial infarction, stroke, coronary heart disease (CHD), other diseases of the circulatory system, as well as respiratory diseases: lung cancer, chronic obstructive pulmonary disease (COPD), etc.

One of the main effects of an infectious and physicochemical factor is a cytokine storm, as a result of which a severe form of a systemic inflammatory reaction is formed, leading to damage to the respiratory system and other systems of the human body. Fine particles are deposited in the lungs and blood vessels, causing inflammation. Both during the immune response to the ingress of the virus and when the respirable fraction of $PM_{2.5}$ enters the blood, the number of special signaling proteins—cytokines—increases. They trigger a chain of biochemical reactions that ultimately lead to thrombosis, CHD, heart attack and other complications [3]. At the regular, eighth meeting of the working group of the WHO European Office on Health in a Changing Climate (December 8–9, 2020) almost every report addressed not only the climatic aspects of health but also the impact of air pollution during a pandemic.

Impact of quarantine measures on air quality and public health. The impact of reduced emissions due to declining economic activity on air quality is most evident in the megalopolises of Southeast Asia. In New Delhi, $PM_{2.5}$ emissions fell by 70% in the first week of quarantine, but levels averaged over two weeks of quarantine already showed a 30% decrease. In other large cities (Ahmedabad, Pune), the decrease in $PM_{2.5}$ was 15% [4]. During the period of the first lockdown, the aerosol optical density (AOD) over India decreased on average by 24% [5]. Reduction in nitrogen dioxide (NO_2) emissions in Central China by 30% made it possible to estimate the number of averted deaths [6]. In the four largest cities in China (Beijing, Shanghai, Chengdu, Guangzhou), in the first two months of 2020, daily $PM_{2.5}$ concentrations decreased by 15–17 $\mu g/m^3$ compared to the average levels for these months in 2016–2019. Extrapolating this reduction to the entire urban population of China and using the following age-specific mortality rates (for children 0–5 years old, the increase in monthly mortality is 2.9% and for the elderly over 70 years old it is 1.4% per 1 $\mu g/m^3$ of $PM_{2.5}$), the number of averted deaths in the country was estimated as 51700 cases [7]. An even greater number of excess cases due to air pollution (about 100000 cases) was prevented in the spring of 2020 after two months of quarantine. In this context, the authors used the concentration of nitrogen dioxide as a marker of air quality. This assumption is based on the fact that China completely closed a third of its cities [8]. Finally, the most conservative estimate of averted mortality was based on the fact that in China, the national average air pollution index (API) and $PM_{2.5}$ levels decreased by 25%, which could have led to 36000 cases averted per month [9]. Similar emission reductions have been reported in other countries. For example, the quarantine in the northeastern United States in March 2020 reduced NO_2 emissions by 30% from the 2015–2019 average, while in Barcelona, two weeks after the quarantine, soot and NO_2 levels dropped by 45–51%, mainly as a result of reduced traffic, and PM_{10} levels decreased less, by only 28–31% [10].

Role of Aerosols in COVID-19 Virus Transmission. The ingress of the infection mostly occurs through the transfer of the virus by droplets of saliva and other biological fluids from person to person during close contact, i.e., at a distance of about a meter. Such droplets have an aerodynamic diameter of more than 20 μm and upon inhalation are deposited in the upper respiratory tract. Observations and laboratory experiments show that the virus is also carried by fine aerosols in indoor air but the relative contribution of this infection route in comparison with the main one has not yet been quantified. The SARS-CoV-2 airborne virus can be transmitted by aerosols with an aerodynamic diameter of less than 10 μm, and particles with a diameter of less than 5 μm enter the lower respiratory tract.

Infected droplets of saliva released during coughing, sneezing, and lively speech, which have an aerodynamic diameter of more than 20 μm, i.e., are relatively heavy, quickly settle on the ground and other surfaces and therefore can serve as a source of secondary infection through objects that have been in contact with the virus. If the virus is attached to aerosols in the air, especially with a diameter less than 10 μm, then it can be transported over considerable distances, which is especially important in urban areas with high housing density. The possibility of transporting the viable SARS-CoV-2 virus by aerosols over distances of more than two meters indoors was demonstrated in laboratory experiments in [11, 12]. The transport of virus particles by aerosols significantly increases the airborne survival time of the virus before deposition, facilitating long-range transmission of the virus and subsequent deposition in the respiratory tract. In the open air, until now, only the adsorption of RNA fragments of the SARS-CoV-2 virus by suspended matter, but not the viable virus itself, has been observed (in Bergamo, Italy) [13]. Presumably, in conditions of heavy air pollution and slow dispersion of atmospheric pollutants, the virus formed clusters with PM_{10} particles in the atmosphere and was able to spread in this way up to 10 m from the source of infection. The study of the previous SARS-CoV-1 virus in 2002–2003 also showed the possibility of its transport by aerosols over dis-

tances of several meters both in laboratory experiments [14, 15] and in a hospital with infected patients [16].

Therefore, in order to model the transport of virus-infected aerosols, it is necessary to know the typical size of these particles. Measurements in Wuhan in the open air near hospitals showed that most (by number, not by weight) aerosol particles containing the virus had an aerodynamic diameter of 0.2–0.5 μm. Modeling the behavior of such particles in air showed that only particles with a diameter of less than 0.1 μm coagulate, reaching this (or smaller) size in about 30 minutes. At the same time, larger particles retain their size and do not stick together. Such differences in the behavior of particles sticking together were established in a numerical experiment simulating the dynamics in the behavior of aerosols in air over time, which made it possible to test the hypothesis about the relationship between polluted air and human infection [17]. The difference between the aerodynamic diameter and the geometric size is insignificant and is mainly explained by the shape of the particles, and the aerodynamic diameter is approximately one and a half times smaller than the geometric size. The adhesion of particles <0.1 μm in size only reduces the likelihood of their retention in the lungs, since in this size range the curve representing the relation between the particle size and efficiency of their retention in the lungs goes down, tending to its minimum, and larger particles are not subject to coagulation. The point is that the relative fraction of fine particles trapped in the lungs, depending on their geometric size, is nonmonotonic and reaches a minimum at a particle size of about 0.1 μm [18]. Therefore, air pollution cannot contribute to virus infection by increasing the efficiency of viral particles deposition in the lungs. This conclusion was originally obtained for outdoor air, but in the absence of data on ventilation and air filtration in rooms, Dobricic et al. [17] propose to extend it to indoor air. However, this is inconsistent with the results of a number of epidemiological studies on the relationship between fine particles with a diameter of less than 10 μm and the number of infected discussed below.

Statistical relationship between air pollution and infection. In 120 cities in China, a generalized linear regression modeling the daily number of new cases of COVID-19 infection was applied for the period from January 23 to February 29, 2020, in which the explanatory variables were the daily pollution levels averaged over the previous 14 days, by priority, i.e., the most common, substances (fine particles, sulfur dioxide, carbon monoxide, nitrogen dioxide and ozone). This impact is interpreted as "short-term." During this period, a positive correlation was observed between the incidence of the disease and the exposure levels of these substances, except for sulfur dioxide and carbon monoxide. The increase in the impacting concentrations of these substances for every 10 μg/m^3 was significantly associated with the rise in the number of confirmed cases of COVID-19 infection: when exposed to $PM_{2.5}$ by 2.24% (95% CI: 1.02–3.46); to PM_{10} by 1.76% (95% CI: 0.89–2.63); to NO_2 by 6.94% (95% CI: 2.38–11.51) and when exposed to O_3 by 4.76% (95% CI: 1.99–7.52) [19].

A more detailed study of the impact made by polluted air on the number of infected people was carried out in Italy, where two samples were compared: especially polluted cities, in which the standard for the content of PM_{10} or ozone in the air was exceeded for more than 100 days a year, and cleaner cities [20]. The European Union Air Quality Directive 2008/50/EU has established binding standards for these substances in the air throughout the EU. According to this legislative act, the daily PM_{10} standard is 50 μg/m^3, and this standard can be exceeded for no more than 35 days a year, and the concentration of ground-level ozone averaged over 8-hour periods should not exceed 120 μg/m^3; this standard can be exceeded for no more than 25 days in three years of observations [21]. In the abovementioned study [20], the regression reflecting the dependence of the logarithm of coronavirus incidence at a certain date on the logarithm of population density for a sample of 55 centers of administrative territories was calculated, since the rate of virus transmission from person to person can depend on this indicator. The change in the slope of the regression line for the sample of "dirty" cities compared to "clean" cities indicates a possible role of air pollution in the transmission of the virus. The results of this analysis showed that for the group of clean cities, where the content of fine particulate matter with a size of less than 10 μm (PM_{10}) or ozone exceeded the standard level, less than 100 days a year, a 1% increase in population density led to an increase in the number of infected equal to 0.25%; for a sample of dirty cities, where standards were exceeded for more than 100 days a year, a 1% increase in population density led to an increase in the number of infected people already by 0.85%.

Moreover, findings on the dependence of the number of infected on air pollution and population density made it possible to compare the relative contributions of these variables to the development of the coronavirus epidemic. The dimensionless two-factor log-log model is described by the regression equation (1)

$$\log[E(y_t)] = \alpha + \beta_1 \log x_{1;t-1} + \beta_2 \log x_{2;t-1}, \qquad (1)$$

where $\log[E(y_t)]$ is the natural logarithm of the expected number of infections on a given date in a year t, and the indices "1" and "2" stand for the air pollution and population density, respectively. The index $t-1$ for independent variables means a one-year lag

between cause and effect. More precisely, the quantitative measure of pollution x_1 was the number of days in 2018 with an excess of the daily standard for the content of PM_{10} or ozone in the air. The population density x_2 in the studied cities varied from 20 to 8 thousand people/sq. km. The estimated values of the regression coefficients differed both in magnitude and in statistical significance: $\beta_1 = 1.27$, $p < 0.001$; $\beta_2 = 0.31$, $p < 0.05$. This indicates that atmospheric air pollution has a stronger effect on the transmission of the virus than population density. Another important conclusion from this study is that by varying the calendar date, on which the number of infected y_t is taken, it is possible to study the indirect effect of quarantine on the increase in the number of infected residents.

The second, nonlinear model (2) simulating the dependence of the number of infected people on air pollution with a quadratic term made it possible to estimate at what level of pollution x_t a break-point in this dependence can be expected

$$E(y_t) = \alpha + \beta_1 x_{t-1} + \beta_2 (x_{t-1})^2. \quad (2)$$

Having solved the optimization problem on the position of the minimum of the regression parabola describing the change in the number of infected y_t in different cities depending on the air pollution, the authors of the study came to the following conclusion: for cities in northern Italy of comparable population with similar weather conditions (cities are located in mountain basins with weak wind and poor conditions for dispersing pollution), the influence of pollutants on the spread of infection will be significantly less if the PM_{10} or ozone standard is exceeded less than 48 days a year. This work shows how, given an array of publicly available baseline data on the number of people infected in different cities, one can obtain significant conclusions about the underlying causes of the spread of the epidemic using regression analysis. The difficulty, however, lies in choosing the "correct" sample of cities for such an analysis, based on the similarities or differences in local meteorological conditions. Certainly, this approach would be very interesting for analyzing the situation in Russia with its greater variety of landscape and meteorological conditions.

The authors of another study, based on the same data from 71 capitals of the Italian provinces, drew attention not only to the level of air pollution but also to the factor reflecting the duration of exposure to pollution. In order to characterize the long-term exposure to air pollution, a variable was investigated equal to the number of years in the last decade (2010–2019) when the daily PM_{10} standard (50 $\mu g/m^3$) was exceeded for more than 35 days a year, i.e., more than 10% of days. This figure is specified in the European Union Directive 2008/50/EU [21]. This variable proved to be positively correlated with the number of confirmed cases of COVID-19 in various cities. This means that in cities where the population has been exposed to polluted atmospheric air for a long time (more than 35 days a year for several years), the number of people infected with coronavirus is higher [22]. This study also developed two more models for the incidence of COVID-19. The first assessed the impact made by annual average concentrations of nitrogen dioxide and fine particulate matter over the last four years before the pandemic; and in the second, the impact was expressed in the number of days exceeding the standards for the content of PM_{10} and ozone (which is a precursor for photochemical synthesis of NO_2 in the ambient air) for the three years preceding the epidemic. These two models also showed a positive correlation between the concentrations of pollutants and the number of people infected. The reason for this is the combined cytokine effect of two powerful health risk factors. The authors explain the correlations by the overexpression of proinflammatory cytokines and chemokines (i.e., an excessive immune response to the inflammation focus) in the presence of atmospheric air pollution.

Air pollution and mortality from coronavirus. The study of the association between air pollution and mortality began in China as early as in 2003 during the last SARS-CoV-1 epidemic, and it also proved the relationship between the level of air pollution and mortality [23]. The first spatial study of the link between air pollution and the mortality rate from COVID-19 was carried out in the United States [24]. The geographical unit of study was taken to be 3080 counties providing residence to 98% of the entire American population. The dependent variable was the number of deaths from COVID-19 accumulated by April 4, 2020, per 100 thousand people; the independent variable was the average long-term concentration of $PM_{2.5}$ for 2000–2016. The spatial distribution for the concentration of this substance is calculated based on satellite observations of the optical density of the atmosphere on a coordinate grid $0.01° \times 0.01°$. The work uses a negative binomial mortality model, which is more general than the commonly used Poisson model, since it takes into account the excess variance of a random variable. This model allows us to abandon the assumption of the independence of outcomes (the number of deaths). The choice of the model is due to the fact that the authors use not the number of deaths per unit of time but the cumulative (accumulated) mortality. Importantly, the mortality model took into account potential confounding factors: county population density, number of hospital beds, number of people tested, winter and summer temperatures and humidity, socioeconomic and behavioral factors including obesity and smoking, and potential correlation of effects between counties within the same state.

The results of this study also show that previously documented effects of $PM_{2.5}$ coincide with factors that increase the risk of death from COVID-19: heart and lung disease, nonfatal heart attacks, heart rhythm disturbances, asthma, decreased lung function, respiratory symptoms like pneumonia and respiratory tract inflammations, cough, and respiratory stress [25]. This confirms that increased atmospheric air pollution can aggravate the severity of coronavirus disease outcomes for those infected.

In addition to fundamental research, links between the level of air pollution in cities and mortality from coronavirus have been proven in Italy, India, and China. Thus, in Italy, 33% of deaths from COVID-19 were accompanied by at least one concomitant disease statistically significantly associated with air pollution [26]. Multiplying this 33% by the attributive share of air pollution in the development of such diseases, equal to 20%—this is the result obtained in Europe for the incidence of chronic obstructive pulmonary disease, coronary heart disease, diabetes, and lung cancer [27]—we find that at least 6.6% of deaths from COVID-19 are caused by ambient air pollution. Using the same approach, it is determined that in the countries of Southeast Asia with a high level of air pollution, for example, in China, up to 11% of deaths from the described viral infection can be associated with air pollution [17].

A study of the nine most polluted Asian megacities (Delhi, Islamabad, Jakarta, and others) also showed a statistically significant relationship between the average long-term (for 2007–2016) $PM_{2.5}$ concentration and the COVID-19 mortality rate [28]. Mortality was calculated as the ratio of the number of deaths from COVID-19 to the number of infected in each city. The average mortality rate for these cities as of July 2, 2020, was 2.3%. Average $PM_{2.5}$ concentrations in the ambient air of these cities varied from 45 $\mu g/m^3$ in Jakarta to 143 $\mu g/m^3$ in Delhi and 173 $\mu g/m^3$ in Kanpur, with an average of 85 $\mu g/m^3$, which is much higher than in European and Russian cities. For comparison: in Moscow, the average annual concentration of $PM_{2.5}$ in 2019, according to Mosecomonitoring, was 16 $\mu g/m^3$, but in Krasnoyarsk, Novokuznetsk, Chelyabinsk and other cities with large metallurgical enterprises, the level of pollution is higher.

A. Gupta et al. (2020) applied a linear regression of the mortality rate by the decimal logarithm of the $PM_{2.5}$ concentration and obtained a high statistical significance of the regression coefficient: with each doubling of the concentration, the lethality increases by $(1.7 \pm 0.3)\%$; $p < 0.01$. The authors explain the dependence by the weakening of the immune system under systematic exposure to atmospheric air pollution. At the same time, the authors discommend using the obtained dependence to predict the mortality rate outside the specified concentration range. A similar regression for PM_{10} levels in the same cities did not show a statistically significant relationship, which confirms the predominant effect of the finest $PM_{2.5}$ fraction on mortality.

Mortality from COVID-19 depends on the level of air pollution not only with fine particles, but also with nitrogen dioxide (NO_2). This substance causes death of epithelial cells in the lungs, inflammation of the airways; it is a significant risk factor for the development of COPD, CHD, hypertension, diabetes, and other diseases. The presence of such diseases in infected individuals is confirmed by the fact that 74% of those who died from COVID-19 had hypertension, 34% had diabetes, and 30% had coronary heart disease. The relationship between mortality from COVID-19 and NO_2 concentrations not only in the surface air but also in the troposphere has been confirmed. In Italy, Spain, France, and Germany, using spatial correlation methods, the effect of tropospheric NO_2 levels on mortality from COVID-19 has been estimated [29]. Four provinces of Northern Italy and Madrid in Spain together account for 78% of COVID-19 deaths in these four countries, and the same five regions have the highest tropospheric NO_2 levels combined with poor dispersion conditions (downdrafts). The author explains this result by the fact that long-term exposure to nitrogen dioxide increases the risks of the most common comorbidities in deaths from COVID-19. The ranking of the number of deaths from COVID-19 (as of March 19, 2020) in all regions of Italy, Spain, France and Germany by tropospheric NO_2 levels shows that 83% of all deaths occur in regions with levels of more than 100 μmol/sq. m, 15.5% with levels from 50 to 100 μmol/sq. m, and only 1.5% with levels below 50 μmol/sq. m.

Conclusions. Studies of the relationships between the level of air pollution, tropospheric nitrogen dioxide content, and mortality from COVID-19 in 2020 have shown that these factors lead to a statistically significant increase in morbidity and mortality from coronavirus but many aspects of these relationships have not yet been clarified. Modern epidemiological models of mortality should take into account, among other things, such significant risk factors at the regional level as the capacity of the bedspace, the number of infectious disease specialists, the availability of primary medical services, emergency medical care, pharmacies and other resources of the health care system, which for Russia is partially shown in the article by V. Stepanov [30]. The use of statistical models for cross-country comparisons between the development processes of the COVID-19 epidemic is impossible without taking into account socioeco-

nomic indicators, including per capita income, differences in the collection and processing of statistical information on morbidity and mortality; features of the epidemiological and economic policies of different countries during a pandemic at various stages of its development. New intercenter studies are needed based on agreed protocols and the development of modern statistical models that allow assessing the contributions of various risk factors, including atmospheric air pollution in areas of population with different socioeconomic status, to the dynamics of infection and disease outcomes, taking into account the specifics of different regions and countries of the world.

FUNDING

The research was carried out within the framework of scientific topic 168.5. "Compilation and refinement of short, medium and long-term forecasts for the development of the social sector of the economy," IEF RAS.

REFERENCES

1. A. G. Emanov, "The great pandemic of the middle of the XIV century as the finale of medieval history," Vestn. Tyumen. Gos. Univ., Gumanit. Issled., No. 2, 49–55 (2013).
2. B. A. Revich, "Fine suspended particles in the atmospheric air and their impact on the health of residents of megalopolises," Probl. Ekol. Monit. Model. Ekosist., No. 3, 53–78 (2018).
3. Gouda M.M., Shaikh S.B., and Bhandary, Y.P., Inflammatory and fibrinolytic system in acute respiratory distress syndrome, Lung **196** (5), 609–616 (2018). https://doi.org/10.1007/s00408-018-0150-6
4. N. A. Anjum, "Good in the worst: COVID-19 restrictions and ease in global air pollution", Preprints, 2020040069 (2020). https://doi.org/10.20944/preprints202004.0069.v1
5. M. Pathakoti, A. Muppalla, S. Hazra, M. Dangeti, R. Shekhar, S. Jella, S. S. Mullapudi, P. Andugulapati, and U. Vijayasundaram, "An assessment of the impact of a nation-wide lockdown on air pollution-a remote sensing perspective over India," Atmos. Chem. Phys. Discuss. (2020). https://doi.org/10.5194/acp-2020-621
6. NASA Earth Observatory. 2020. Airborne Nitrogen Dioxide Plummets over China. https://earthobservatory.nasa.gov/images/146362/airborne-nitrogen-dioxide-plummets-over-china.
7. M. Burke, COVID-19 Reduces Economic Activity, Which Reduces Pollution, Which Saves Lives. G-FEED (Global Food, Environment and Economic Dynamics) Website, March 8, 2020. http://www.g-feed.com/2020/03/COVID-19-reduces-economic-activity.html.
8. F. Dutheil, J. S. Baker, and V. Navel, "COVID-19 as a factor influencing air pollution?," Environ. Pollut. **263** (A), 114466 (2020). https://www.sciencedirect.com/science/article/abs/pii/S0269749120316468.
9. G. He, Y. Pan, and T. Tanaka, "The short-term impacts of COVID-19 lockdown on urban air pollution in China," Nat. Sustainability **3**, 1005–1011 (2020). https://doi.org/10.1038/s41893-020-0581-y
10. A. Tobías et al., "Changes in air quality during the lockdown in Barcelona (Spain) one month into the SARS-CoV-2 epidemic," Sci. Total Environ. **726**, 138540 (2020). https://doi.org/10.1016/j.scitotenv.2020.138540
11. J. L. Santarpia et al., "Aerosol and surface contamination of SARS-CoV-2 observed in quarantine and isolation care," Sci. Rep. **10**, 12732 (2020). https://doi.org/10.1101/2020.03.23.20039446
12. N. van Doremalen et al., "Aerosol and surface stability of SARS-CoV-2 as compared with SARS-CoV-1," N. Engl. J. Med. **382**, 1564–1567 (2020). https://doi.org/10.1056/NEJMc2004973
13. L. Setti et al., "SARS-Cov-2 RNA found on particulate matter of Bergamo in Northern Italy: First preliminary evidence," Environ. Res. **188**, 109754 (2020). https://doi.org/10.1016/j.envres.2020.109754
14. S. Xiao, Y. Li, and T.-W. Wong, "Role of fomites in SARS transmission during the largest hospital outbreak in Hong Kong," PLoS ONE **12** (7), e0181558 (2017). https://doi.org/10.1371/journal.pone.0181558
15. I. T.-S. Yu, H. Qiu, L. A. Tse, and T. W. Wong, "Severe acute respiratory syndrome beyond Amoy Gardens: Completing the incomplete legacy," Clin. Infect. Dis. **58** (5), 683–686 (2014). https://doi.org/10.1093/cid/cit797
16. T. F. Booth et al., "Detection of airborne severe acute respiratory syndrome (SARS) coronavirus and environmental contamination in SARS outbreak units," J. Infect. Dis. **191** (9), 1472–1477 (2005). https://doi.org/10.1086/429634
17. S. Dobricic, E. Pisoni, L. Pozzoli, R. Van Dingenen, T. Lettieri, J. Wilson, and E. Vignati, *Do Environmental Factors Such as Weather Conditions and Air Pollution Influence COVID-19 Outbreaks? EUR 30376 EN* (Publications Office of the European Union, Luxembourg, 2020).
18. J. Rissler, H. Nicklasson, A. Gudmundsson, P. Wollmer, E. Swietlicki, and J. Löndahl, "A set-up for respiratory tract deposition efficiency measurements (15–5000 Nm) and first results for a group of children and adults," Aerosol Air Qual. Res. **17**, 1244–1255 (2017). https://doi.org/10.4209/aaqr.2016.09.0425
19. Y. Zhu, J. Xie, F. Huang, and L. Cao, "Association between short-term exposure to air pollution and COVID-19 infection: Evidence from China," Sci. Total Environ. **727**, 138704 (2020). https://doi.org/10.1016/j.scitotenv.2020.138704
20. M. Coccia, "Factors determining the diffusion of COVID-19 and suggested strategy to prevent future accelerated viral infectivity similar to COVID," Sci. Total Environ. **729**, 138704 (2020). https://doi.org/10.1016/j.scitotenv.2020.138704
21. "Directive 2008/50/EC of the European Parliament and of the Council of 21 May 2008 on Ambient Air Quality and Cleaner Air for Europe," Official J. Eur. Union **51** (2008).

22. D. Fattorini and F. Regoli, "Role of the chronic air pollution levels in the COVID-19 outbreak risk in Italy," Environ. Pollut. **264**, 114732 (2020).

23. Y. Cui, Z. F. Zhang, J. Froines, et al., "Air pollution and case fatality of SARS in the People's Republic of China: An ecologic study," Environ. Health **2**, 15 (2003). https://doi.org/10.1186/1476-069X-2-15

24. X. Wu, R. C. Nethery, M. B. Sabath, D. Braun, and F. Dominici, "Air pollution and COVID-19 mortality in the United States: Strengths and limitations of an ecological regression analysis," Sci. Adv. **6** (45), eabd4049 (2020). https://doi.org/10.1126/sciadv.abd4049

25. R. Burnett and A. Cohen, "Relative risk functions for estimating excess mortality attributable to outdoor PM2.5 air pollution: Evolution and state-of-the-art," Atmosphere **11** (6), 589 (2020).

26. Istituto Superiore di Sanità, Characteristics of SARS-CoV-2 Patients Dying in Italy (2020). https://www.epicentro.iss.it/en/coronavirus/bollettino/Report-COVID-2019_9_july_2020.pdf.

27. *GBD Compare* (IHME, Seattle, WA, 2018). http://vizhub.healthdata.org/gbd-compare.

28. A. Gupta, H. Bherwani, S. Gautam, et al., "Air pollution aggravating COVID-19 lethality? Exploration in Asian cities using statistical models," Environ. Dev. Sustainability **23**, 6408–6417 (2020). https://doi.org/10.1007/s10668-020-00878-9

29. Y. Ogen, "Assessing nitrogen dioxide (NO_2) levels as a contributing factor to the coronavirus (COVID-19) fatality rate," Sci. Total Environ. **726**, 138605 (2020). https://doi.org/10.1016/j.scitotenv.2020.138605

30. V. S. Stepanov, "Dependence of the mortality rate in the regions on the prevalence of active carriers of SARS-CoV-2 and the resources of healthcare organizations," Anal. Riska Zdorov'yu, No. **4**, 12–22 (2020). https://doi.org/10.21668/health.risk/2020.4.02

Translated by I. Pertsovskaya