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By Electronic Submission to www.regulations.gov

Administrator Andrew Wheeler
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, NW
Washington, D.C. 20460

Docket ID No. EPA-HQ-OAR-2015-0072

Re: COMMENTS ON REVIEW OF THE NATIONAL AMBIENT AIR QUALITY STANDARDS FOR PARTICULATE MATTER (PROPOSED RULE), 85 FED. REG. 24,094 (APR. 30, 2020)

On behalf of David C. Christiani, Francesca Dominici, Diane R. Gold, Francine Laden, Jonathan I. Levy, Murray A. Mittleman, Mary B. Rice, and Joel Schwartz, the Emmett Environmental Law & Policy Clinic at Harvard Law School submits these comments on the notice of proposed rulemaking "Review of the National Ambient Air Quality Standards for Particulate Matter," 85 Fed. Reg. 24,094 (Apr. 30, 2020) (the "Proposal"). Multiple other comment letters have identified a broad range of flaws with the Proposal. Most fundamentally, it disregards the substantial evidence that fine particulate matter ($PM_{2.5}$) causes serious, adverse health effects at levels below the current National Ambient Air Quality Standards ("NAAQS").[1]

We write separately here to emphasize a deeply ironic fact: even as the Environmental Protection Agency ("EPA") rushes through an erroneous decision resulting from a flawed process in the midst of the COVID-19 global pandemic, evidence is emerging that $PM_{2.5}$ pollution may be exacerbating the effects of that pandemic. We therefore urge EPA to heed the advice of the Independent Particulate Matter Review Panel and strengthen the $PM_{2.5}$ annual standard to 10

[1] A newly-published paper provides the strongest evidence yet for this link. Many previous studies have demonstrated a relationship between $PM_{2.5}$ exposure at levels below the current annual NAAQS and increased mortality, but critics have argued that the traditional statistical methods employed in those studies do not establish causality. In this new study, Wu et al. implement five statistical approaches, including both traditional and causal inference methods, to data from more than 68.5 million Medicare enrollees between 2000 and 2016. X. Wu et al., *Evaluating the Impact of Long-term Exposure to Fine Particulate Matter on Mortality Among the Elderly*, Science Advances (early release June 26, 2020), https://advances.sciencemag.org/content/early/2020/06/26/sciadv.aba5692. They found that a 10 $\mu g/m^3$ decrease in $PM_{2.5}$ results in a 6%-7% decrease in mortality risk and a shift to an annual $PM_{2.5}$ standard of 10 $\mu g/m^3$ would save between 115,581–170,645 lives over a ten year period. The authors conclude that the "study provides the most robust and reproducible evidence to date on the causal link between exposure to PM2.5, even at levels below 12 $\mu g/m^3$, and mortality among Medicare enrollees." *Id.* at 3. Two of the authors of this study, Francesca Dominici and Joel Schwartz, are signatories of this letter.

$\mu g/m^3$ or lower and the 24-hour standard to 30 $\mu g/m^3$ or lower. Failing that, EPA should at a minimum withdraw the Proposal to consider the new evidence of relationships between $PM_{2.5}$ exposure and COVID-19 susceptibility and mortality.

I. THE PROPOSAL IS THE RESULT OF A RUSHED AND FLAWED PROCESS AND IS INCONSISTENT WITH THE SCIENTIFIC RECORD

The Proposal is the result of a deeply flawed process. EPA's initial Integrated Review Plan for the National Ambient Air Quality Standards for Particulate Matter, issued in December 2016, envisioned a five-year process leading to the issuance of a proposal in 2021 and a final rule in 2022. This plan, consistently with EPA's historical practice, called for the staggered preparation of two rounds of public drafts of an Integrated Science Assessment ("ISA"), a Risk and Exposure Assessment ("REA"), and a Policy Assessment, with opportunities for both public comment and CASAC review during each round.

In 2018, however, EPA announced that it was streamlining the process. Under this accelerated schedule, EPA has now issued the Proposal in April 2020 and intends to issue the final rule by the end of the year.[2] To achieve this expedited schedule, EPA has provided only one round of public comment on the ISA and Policy Assessment and has eliminated the REA entirely.

EPA also undermined the ability of its scientific advisors to assist the agency in reviewing the NAAQS.[3] First, EPA removed scientists from the Clean Air Scientific Advisory Committee ("CASAC") based on the spurious claim that they lacked independence based on their receipt of EPA research grants. (The directive resulting in their removal has now been struck down as arbitrary and capricious by two federal courts.)[4] Next, EPA disbanded the 20-member Particulate Matter Review Panel, even though it had for decades relied on such specialized panels to assist CASAC with NAAQS reviews. Although CASAC requested that the review panel be reappointed because the "breadth and diversity of evidence to be considered exceeds the

[2] Memorandum from E. Scott Pruitt, Administrator, EPA, to Assistant Administrators (May 9, 2018), https://www.epa.gov/sites/production/files/2018-05/documents/image2018-05-09-173219.pdf.

[3] *See generally* Mary B. Rice et al., *Threats to Science Advising at the Environmental Protection Agency*, 17 Annals Am. Thoracic Soc. 267 (2020).

[4] *Physicians for Soc. Responsibility v. Wheeler*, 956 F.3d 634, 647 (D.C. Cir. 2020) (holding that the directive was arbitrary and capricious because, "in failing to grapple with how EPA's policy affected its statutory scientific mandates, the Directive 'failed to consider an important aspect of the problem'") (citation omitted); *Nat. Res. Def. Council, Inc. v. U.S. Envtl. Prot. Agency*, No. 19-CV-5174 (DLC), 2020 WL 2769491, at *1 (S.D.N.Y. Apr. 15, 2020) ("The provision of the Directive specifying that 'no member of an EPA federal advisory committee be currently in receipt of EPA grants, either as principal investigator or co-investigator, or in a position that would otherwise reap substantial direct benefits from an EPA grant,' is vacated and this matter is remanded.").

expertise of the statutory CASAC members,"[5] EPA declined to do so. At the end of the process, CASAC was unable to provide a consensus recommendation for EPA.[6]

The members of the Particulate Matter Review Panel, who reconvened outside of EPA's purview as the Independent Particulate Matter Review Panel, recommended that EPA strengthen the standards. In particular, they suggested that "[t]he annual standard should be revised to a range of 10 $\mu g/m^3$ to 8 $\mu g/m^3$" and "the 24-hour standard should be revised to a range of 30 $\mu g/m^3$ to 25 $\mu g/m^3$."[7] Nevertheless, the Proposal would retain the existing standards.

II. THE COVID-19 PANDEMIC IS AN ADDITIONAL REASON TO LOWER THE $PM_{2.5}$ STANDARD

The process EPA has followed would be fundamentally flawed under any circumstances. In the midst of a global pandemic, however, it is not merely insufficient but tragically misguided. On the broadest level, the outcome of the COVID-19 pandemic in the United States highlights the necessity for federal agencies to base their decisions on the best available scientific evidence, which the Proposal plainly fails to do. More specifically, there is a growing body of research tying the seriousness of COVID-19 symptoms to $PM_{2.5}$ exposure.

A. There is Evidence that Exposure to Elevated Levels of $PM_{2.5}$ Results in Greater COVID-19 Mortality

In a manuscript currently undergoing peer review, researchers at the Harvard T.H. Chan School of Public Health find a significant relationship between long-term $PM_{2.5}$ exposure and COVID-19 mortality in the United States.[8] The authors compared COVID-19 death counts through April 22, 2020, in counties representing 98% of the population in the United States, to 2000-2016 average $PM_{2.5}$ pollution levels in those counties. Based on this comparison, they found that "an increase of only 1 $\mu g/m^3$ in $PM_{2.5}$ is associated with an 8% increase in the COVID-19 death rate."[9] This result remained significant at a 95% confidence interval even after taking into

[5] Letter from Louis Anthony Cox, Jr., Chair, CASAC to Andrew R. Wheeler, Administrator, EPA (Apr. 11, 2019), https://yosemite.epa.gov/sab/sabproduct.nsf/LookupWebReportsLastMonthCASAC/6CBCBBC3025E13B4852583D90047B352/%24File/EPA-CASAC-19-002+.pdf.

[6] CASAC Review of the EPA's Policy Assessment for the Review of the National Ambient Air Quality Standards for Particulate Matter (External Review Draft – September 2019) (Dec. 16, 2019), https://yosemite.epa.gov/sab/sabproduct.nsf/LookupWebProjectsCurrentCASAC/E2F6C71737201612852584D20069DFB1/$File/EPA-CASAC-20-001.pdf.

[7] Final Report of the Independent Particulate Matter Review Panel 1 (Oct. 22, 2019), https://ucs-documents.s3.amazonaws.com/science-and-democracy/IPMRP-FINAL-LETTER-ON-DRAFT-PA-191022.pdf.

[8] Xiao Wu et al., *Exposure to Air Pollution and COVID-19 Mortality in the United States: A Nationwide Cross-sectional Study* (unpublished manuscript, Apr. 24, 2020), https://projects.iq.harvard.edu/files/covid-pm/files/pm_and_covid_mortality_med.pdf. A signatory of this letter, Francesca Domenici, is one of the authors of this manuscript.

[9] *Id.* at 2.

account a long list of potential confounding variables.[10] As the authors summarized their findings, their "results indicate that long-term exposure to air pollution increases vulnerability to the most severe COVID-19 outcomes."[11]

This finding is consistent with a number of other lines of research from around the world. For example, a manuscript by researchers at the University of Cambridge "identified $PM_{2.5}$ and PM_{10} as significant predictors of increased SARS-CoV-2 infectivity."[12] Similarly, a paper examining the outbreak in China found a significant positive association of $PM_{2.5}$ (as well as other pollutants) with confirmed cases of COVID-19.[13] A recently-published paper observed the correlation between the COVID-19 mortality rate and air pollution levels in different parts of Italy and raised the question whether "communities living in polluted area such as Lombardy and Emilia Romagna [are] more predisposed to die of Covid-19 due to their health status?"[14]

These findings for COVID-19 are consistent with a robust body of mechanistic and epidemiologic research on other respiratory infections, including influenza and SARS (a coronavirus) that have found that PM exposure weakens immune defenses and worsens the consequences of infection. For example, in animals infected with influenza, the lung immune system is altered and influenza viral loads are higher among those exposed to higher levels of PM pollution.[15] Air pollution is known to damage cilia in the upper respiratory tract, which provide the first line of defense against respiratory infections.[16] In addition, exposure to PM "may inhibit pulmonary antimicrobial responses, reducing clearance of the virus from the lungs and promoting infectivity."[17] PM exposure may result in "decreased ability of macrophages to phagocytize the virus and mount an effective immune response against the infection."[18]

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[10] These potential confounders were: "days since first COVID-19 case reported (a proxy for epidemic stage), population density, percent of population ≥65 years of age, percent of the population 45-64 years of age, percent of the population 15-44 years of age, percent living in poverty, median household income, percent black, percent Hispanic, percent of the adult population with less than a high school education, median house value, percent of owner-occupied housing, percent obese, percent current smokers, number of hospital beds per unit population, and average daily temperature and relative humidity for summer (June-September) and winter (December-February) for each county, and days since issuance of stay-at-home order for each state." *Id.* at 8.

[11] *Id.* at 14.

[12] Marco Travaglio et al., *Links between Air Pollution and COVID-19 in England*, at 13 (Unpublished Manuscript June 6, 2020), https://www.medrxiv.org/content/10.1101/2020.04.16.20067405v5.full.pdf+html.

[13] YongjianZhu et al., *Association between Short-term Exposure to Air Pollution and COVID-19 Infection: Evidence from China*, 727 Sci. Total Env't 138704 (2020).

[14] Edoardo Conticini et al., *Can Atmospheric Pollution be Considered a Co-factor in Extremely High Level of SARS-CoV-2 Lethality in Northern Italy?*, 261 Envtl. Pollution 114465 (2020).

[15] Kymberly M. Gowdy et al., *Role of Oxidative Stress on Diesel-enhanced Influenza Infection in Mice*, 7 Particle & Fibre Toxicology 34 (2010); Jonathan Ciencewicki & Ilona Jaspers, *Air Pollution and Respiratory Viral Infection*, 19 Inhalation Toxicology 1135 (2007).

[16] Yu Cao et al., *Environmental Pollutants Damage Airway Epithelial Cell Cilia: Implications for the Prevention of Obstructive Lung Diseases*, 11 Thoracic Cancer 505 (2020).

[17] Travaglio et al., *supra* note 12, at 17.

[18] Jonathan Ciencewicki & Ilona Jaspers, *Air Pollution and Respiratory Viral Infection*, 19 Inhalation Toxicology 1135, 1140 (2007).

Studies of viral and bacterial pneumonia in humans have found that short- and long-term exposure to PM increases hospitalization risk and mortality. A review article published in 2007 concluded, based on the published literature, that "chronic exposure to high PM levels and even short-term variations in PM levels can have significant consequences on infection-related respiratory health,"[19] and a more recent review article again concluded that "evidence supports the association between air pollution and respiratory infections."[20] A study in China after the 2003 SARS outbreak found that patients in areas with a moderate air pollution index (based on a combination of PM, sulfur dioxide, nitrogen dioxide, carbon monoxide and ground-level ozone) had an 84% greater chance of dying than SARS patients from areas with a low air pollution index.[21] The authors found relationships between the SARS fatality rate and both short-term and long-term pollution exposure. A 2019 paper found a significant relationship between $PM_{2.5}$ levels in China and the incidence of influenza-like illness.[22] Research has also demonstrated that PM levels were positively associated with increased disease incidence[23] and mortality[24] in the H1N1 influenza pandemic in 2009. Two recent papers, using coal-fired power capacity as a proxy for air pollution, have found that "cities with high levels of air pollution . . . experienced significant higher mortality rates" during the 1918-19 Spanish flu pandemic.[25]

The mechanisms underlying the excessive mortality from COVID-19 associated with exposure to air pollution are still uncertain. However, death from COVID-19 most commonly results from acute respiratory distress syndrome (ARDS), a severe inflammatory response of the lung to infection. "Air pollution represents one of the most well-known causes of prolonged inflammation, eventually leading to an innate immune system hyper-activation."[26] For example, Pope et al. found that high $PM_{2.5}$ episodes in Utah were associated with endothelial injury and inflammation in young, healthy non-smokers.[27] A subsequent paper by Tsai et al. examining a cohort in Switzerland found that the degree of impact varied with the length of exposure:

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[19] *Id.* at 1139.

[20] José L. Domingo & Joaquim Rovira, *Effects of Air Pollutants on the Transmission and Severity of Respiratory Viral Infections*, 187 Envtl. Res. 109650 (2020), https://doi.org/10.1016/j.envres.2020.109650.

[21] Yan Cui et al., *Air Pollution and Case Fatality of SARS in the People's Republic of China: An Ecologic Study*, 2 Envtl. Health 15 (2003).

[22] Wei Su et al., *The Short-term Effects of Air Pollutants on Influenza-like Illness in Jinan, China*, 19 BMC Public Health 1319 (2019).

[23] Zhiwei Xu et al., *Air Pollution, Temperature and Pediatric Influenza in Brisbane, Australia*, 59 Env't Int'l 384 (2013).

[24] Kathleen F. Morales et al., *Possible Explanations for Why Some Countries Were Harder Hit by the Pandemic Influenza Virus in 2009—A Global Mortality Impact Modeling Study*, 17 BMC Infectious Diseases 642 (2017).

[25] Karen Clay et al., *What Explains Cross-city Variation in Mortality during the 1918 Influenza Pandemic? Evidence from 438 U.S. Cities*, 35 Econ. & Human Biology 42, 49 (2019); *see* Karen Clay et al., *Pollution, Infectious Disease, and Mortality: Evidence from the 1918 Spanish Influenza Pandemic*, 78 J. Econ. History 1179 (2018).

[26] Conticini et al., *supra* note 14, at 2; *see also* Travaglio et al., *supra* note 12, at 16 ("Although the molecular mechanisms underlying the relationship between pollutant exposure and COVID-19 remain to be determined experimentally, they are hypothesised to include the stimulation of chronic, background pulmonary inflammation.").

[27] C. Arden Pope III et al., *Exposure to Fine Particulate Air Pollution Is Associated with Endothelial Injury and Systemic Inflammation*, 119 Circulation Res. 1204 (2016).

"[l]onger duration of elevated exposure was associated with an exacerbated inflammatory response."[28] In the United States, PM exposure has been associated with the risk of ARDS among adults on Medicare: each 1 $\mu g/m^3$ higher annual $PM_{2.5}$ associated with 0.72% higher annual hospital admission rate for ARDS.[29]

Another recent study from the Bergamo region in northern Italy raises the possibility that PM might serve as a means of transmission of the virus, thereby increasing infectivity in polluted areas. Specifically, this study detected SARS-CoV-2 RNA in air samples taken in the Bergamo region in February and March 2020.[30] Based on this observation, the authors suggest that "in conditions of atmospheric stability and high concentrations of PM, SARS-CoV-2 could create clusters with outdoor PM_{10} and—by reducing their diffusion coefficient—*enhance the persistence of the virus in the atmosphere*."[31] The authors caution that there are still uncertainties; for example, they "still do not know if the virus remains vital on PM" or "the average concentrations of PM_{10} eventually required for a potential 'boost effect' of the contagion."[32] Nevertheless, this finding suggests yet another reason to step back and reconsider the Proposal, especially because these findings are consistent with prior research suggesting that air pollution can play a role in the transmission of infectious diseases.[33]

B. These Adverse Impacts Fall Disproportionately on Communities of Color and Other Environmental Justice Communities

The impacts of COVID-19 in the United States have fallen disproportionately on minority and low income communities that are also disproportionately exposed to PM pollution. A manuscript reviewing data from the 28 states that reported race and ethnicity-stratified COVID-19 mortality through April 21, 2020, found that Black Americans were 3.5 times as likely to die from COVID-19 as white Americans, and that LatinX people were nearly 2 times as likely to die.[34] A more recent analysis of data from 45 states and the District of Columbia found that the

[28] Dai-Hua Tsai et al., *Effects of Short- And Long-Term Exposures to Particulate Matter on Inflammatory Marker Levels in the General Population*, 26 Envtl. Sci. & Pollution Res. 19,697, 19,697 (2019).

[29] Jongeun Rhee et al., *Impact of Long-Term Exposures to Ambient $PM_{2.5}$ and Ozone on ARDS Risk for Older Adults in the United States*, 156 Chest 71 (2019).

[30] Leonardo Setti, et al., *SARS-Cov-2 RNA Found on Particulate Matter of Bergamo in Northern Italy: First Evidence*, Envtl. Res. 188 (2020), https://doi.org/10.1016/j.envres.2020.109754.

[31] *Id.* at 3 (emphasis added).

[32] *Id.*

[33] Lu Peng et al., *The Effects of Air Pollution and Meteorological Factors on Measles Cases in Lanzhou, China*, 27 Envtl. Sci. Pollution Research Int'l 13,524 (2020); Qing Ye Q et al., *Haze Is a Risk Factor Contributing to the Rapid Spread of Respiratory Syncytial Virus in Children*, 23 Envtl. Sci. Pollution Res. Int'l 20,178 (2016); Pei-Shih Chen et al. *Ambient Influenza and Avian Influenza Virus during Dust Storm Days and Background Days*, 118 Envtl. Health Persp. 1211 (2010).

[34] Cary P. Gross et al., *Racial and Ethnic Disparities in Population Level Covid-19 Mortality* (Unpublished Manuscript May 11, 2020), https://www.medrxiv.org/content/10.1101/2020.05.07.20094250v1.full.pdf.

"overall COVID-19 mortality rate for Black Americans is about 2.3 times as high as the rate for Whites and Asians."[35]

In Massachusetts, there are several sources of evidence demonstrating racial and ethnic disparities in the impacts of the pandemic. A mapping tool and analysis by the Boston University School of Public Health show that cities and towns with larger percentages of minority populations have suffered the greatest rate of COVID-19 mortalities.[36] Similarly, an analysis by the University of Massachusetts Boston Donahue Institute shows "clear relationships between race, housing, and socioeconomic status and known cases of COVID-19 around Massachusetts."[37] On June 19, 2020, the Massachusetts Department of Public Health's COVID-19 Health Equity Advisory Group released data showing that Black and Hispanic residents have higher rates of infection, hospitalization, and age-adjusted mortality than White and Asian residents.[38]

While a variety of factors likely underlie these differences, air pollution—including $PM_{2.5}$ pollution—may play a role. Previous research from Boston University has shown that in Massachusetts, Black and LatinX residents experience higher exposure to $PM_{2.5}$ and nitrogen dioxide than white residents.[39] The study found that these relative inequalities have grown worse even as overall pollution levels have declined. The mapping tool mentioned above shows that the parts of the state with the highest COVID-19 mortality rates match up closely with the areas with the greatest $PM_{2.5}$ pollution. Studies have also found that the same groups who are disproportionately dying of COVID-19 are also disproportionately dying of PM exposure. A 2017 paper involving data from more than 60 million Medicare beneficiaries in the United States found that "each increase of 10 μg per cubic meter in annual exposure to $PM_{2.5}$ . . . was associated with an increase in all-cause mortality of 7.3%," with the strongest associations at levels *below* the current annual $PM_{2.5}$ NAAQS.[40] This study also found that "that black men and persons eligible to receive Medicaid had a much higher risk of death associated with exposure to air pollution than other subgroups."[41]

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[35] APM Research Lab Staff, *The Color of Coronavirus: COVID-19 Deaths by Race and Ethnicity in the U.S.*, APM Research Lab, https://www.apmresearchlab.org/covid/deaths-by-race (last updated June 24, 2020).

[36] *Vulnerability in Massachusetts During COVID-19 Epidemic*, Boston University School of Public Health, https://bucas.maps.arcgis.com/apps/MapSeries/index.html?appid=e820a92d6bbc4c9099c59494a4e9367a.

[37] *Donahue Data Dash: Linking COVID-19 Cases with Race, Housing and Socioeconomic Status in Massachusetts Spotlights Inequalities*, University of Massachusetts Boston Donahue Institute, http://www.donahue.umassp.edu/our-publications/donahue-data-dash-inequalities.

[38] Press Release, Mass. Dep't Public Health, Department of Public Health Releases Recommendations of COVID-19 Health Equity Advisory Group to Address Pandemic's Impact on Communities of Color (June 19, 2020), https://www.mass.gov/news/department-of-public-health-releases-recommendations-of-covid-19-health-equity-advisory-group.

[39] Anna Rosofsky et al., *Temporal Trends in Air Pollution Exposure Inequality in Massachusetts*, 161 Envtl. Res. 76 (2018).

[40] Qian Di et al., *Air Pollution and Mortality in the Medicare Population*, 376 New England J. Med. 2513, 2517, 2520 (2017).

[41] *Id.* at 2518.

C. The Proposal Does Not Address the Effects of $PM_{2.5}$ Exposure on Susceptibility to, and Severity of, Respiratory Illness

The Proposal says virtually nothing about the potential relationships between $PM_{2.5}$ exposure and an individual's susceptibility to respiratory infections or the severity of respiratory illness. The Federal Register notice contains only the passing reference that "[e]pidemiologic evidence for associations between long-term $PM_{2.5}$ exposure and . . . respiratory infection and the severity of disease was limited, both in the number of studies available and the consistency of the results." 85 Fed. Reg. at 24,110. It also provides brief summaries of the evidence discussed in the Integrated Science Assessment ("ISA"), which we describe below. *Id.* at 24,110-11. These statements, however, were not explicitly connected to the decision to retain the existing $PM_{2.5}$ standards rather than strengthen them.

The ISA includes longer discussions of these topics, but does not rely on them when concluding that there is likely to be a causal relationship between both short-term and long-term $PM_{2.5}$ exposure and respiratory effects. For example, the ISA presents the results of a number of studies showing a relationship between short-term $PM_{2.5}$ exposure and hospital admissions and emergency room visits. The ISA observes that the "overall evidence base" examining the relationship between short-term $PM_{2.5}$ exposure and respiratory infections "has expanded considerably since the 2009 PM ISA" and that "[t]hese recent studies report generally positive associations between $PM_{2.5}$ and hospital admissions and ED visits for pneumonia . . . and all respiratory infections grouped together."[42]

The ISA also discusses research on the relationship between long-term $PM_{2.5}$ exposure and susceptibility to respiratory infections and severity of respiratory disease. It generally finds the evidence here to be weaker than for short-term $PM_{2.5}$ exposure. As of the time of the previous review in 2009, "only one study examined the relationship between long-term exposure to $PM_{2.5}$ and respiratory infection."[43] While there have been additional studies in the intervening years, the ISA concludes that these "studies do not indicate a clear relationship between long-term $PM_{2.5}$ exposures and respiratory infection in infants or adults."[44] As for severity of respiratory disease, the ISA notes that only "[a] limited number of recent epidemiologic studies showed an association between long-term exposure to $PM_{2.5}$ and severity."[45]

The ISA concludes that there is "likely to be a causal relationship" between both short-term and long-term $PM_{2.5}$ exposures and respiratory effects.[46] These conclusions, however, do not rely primarily on evidence about susceptibility to or severity of respiratory infections. Instead, the short-term exposure conclusion focused on asthma exacerbation, exacerbation of chronic obstructive pulmonary disease, and total respiratory mortality,[47] while the long-term exposure

[42] EPA Office of Res. & Dev't, *Integrated Science Assessment for Particulate Matter*, at 5-64 (2019).

[43] *Id.* at 5-193.

[44] *Id.* at 5-195.

[45] *Id.*

[46] *Id.* at 5-148, 5-215.

[47] *Id.* at 5-148 to 1-152.

conclusion emphasizes "epidemiologic evidence demonstrating associations between long-term $PM_{2.5}$ exposure and changes in lung function or lung function growth rate in children" as well as "the consistency of findings across different locations."[48]

III. CONCLUSION

Given the existing and emerging evidence, described in Part II.A above, linking both short-term and long-term $PM_{2.5}$ exposure with severity of COVID-19 and with other viral respiratory illnesses, it is irrational for EPA to rush forward with its flawed decision not to strengthen the $PM_{2.5}$ standards. Instead, EPA should heed the advice of the Independent Particulate Matter Review Panel and strengthen both the 24-hour and annual $PM_{2.5}$ NAAQS. At a minimum, EPA should abandon the Proposal and reconsider the scientific evidence (including the new COVID-19 related evidence) through a sound process.

Thank you for your attention to these comments.

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[48] *Id.* at 5-215.

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