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Will the Trump Fuel Economy Reform Proposal Create Deadly Air Pollution?

By Steve Milloy[*]

The Environmental Protection Agency (EPA) and National Highway Traffic Safety Administration (NHTSA) have proposed to partially roll back the corporate average fuel economy (CAFE) standards issued in 2012.[1] The proposal, called the Safe Affordable Fuel Efficient (SAFE) Vehicles Rule, is partly justified on the basis that it will reduce traffic fatalities by about 1,000 deaths per year or about 12,000 deaths in total by model year 2029.

Opponents of SAFE are claiming that the proposal's lives-saved claim should be offset by deaths resulting from the increased emissions of allegedly deadly air pollutants associated with the rollback of mileage standards.

One such critic, William Schlesinger, a member of the EPA's Science Advisory Board and the former dean of Duke University's Nicholas School of the Environment, said any rollback should account for premature deaths from air pollution, along with traffic fatality numbers. "The science is clear that air pollution kills people, particularly particulates," said Schlesinger. "You would have to estimate what a fleet of nationwide heavier vehicles would mean in terms of mileage or heavy pollution, and do the same for lighter vehicles." [2]

The basic claim put forward by SAFE critics is that lower fuel economy standards will result in more tailpipe emissions of particulate matter (PM) and that these PM emissions will kill many more people than the number of lives saved by SAFE's estimated reduction in traffic fatalities. Is this claim valid?

What is PM? PM is soot and dust in outdoor air. There are many sources of PM, both natural and man-made. Natural sources include volcanic eruptions, forest fires, dry or desert areas, plants and trees, and molds. Man-made sources include smokestacks, tailpipes, chimneys, barbeques, smoking—basically any activity that produces smoke and soot.

PM comes in different sizes and different chemical compositions. That is, pollen is different than tobacco smoke, which is different than tail pipe emissions. This variation, or "speciation," in PM found in the environment defies easy discernment and classification. As a result, various types of particles are all lumped together as under the general term, PM.

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Under the Clean Air Act, the EPA is tasked with setting, monitoring and enforcing a national standard for ambient PM (i.e., PM in outdoor air). As ambient PM levels have declined over the decades, the agency has turned to regulating smaller and smaller sizes of PM.

When the EPA began regulating PM in 1971, the agency's focus was on relatively large PM, 25 to 45 microns (millionths of a meter) in diameter. Having substantially cleaned large particulate matter from the air by the mid-1980s, the agency then turned to reducing PM on the order of 10 microns in diameter (called "PM_{10}"). Having gotten that problem in hand by the early 1990s, the EPA then turned its focus to reducing PM on the order of 2.5 microns in diameter, about one-twentieth the width of a human hair (called "fine particulate matter" or "$PM_{2.5}$").

As a result, over the past 20 years, the EPA has turned $PM_{2.5}$ into its main regulatory hammer for promulgating air quality and smokestack/tailpipe-related emissions rules.

The EPA's aggressive campaign against PM is premised on the notion that it is an unusually dangerous pollutant in need of the strictest regulation. However, as shown below, that notion lacks any basis in science.

Claim: PM kills. A typical cost-benefit analyst charged with weighing this claim would accept at face value the notion that PM kills, estimate by how much SAFE would increase emissions of PM and then estimate how many people would die from causes related to that increased level of PM in outdoor air. Then estimated PM deaths would be directly compared with estimated traffic fatality deaths avoided. While that seems like a reasonable analysis to undertake, it has a built-in assumption that is without a basis in science.

We know that traffic fatalities actually occur in the real world and we know that, on a population level, more driving increases the number of fatalities. On October 6, 2017, the National Highway Traffic Safety Administration released fatal traffic crash data for calendar year 2016. The data, was collected from all 50 states and the District of Columbia, indicated that 37,461 lives were lost on U.S. roads in 2016, an increase of 5.6 percent from 2015. The NHTSA release also noted: "The number of vehicle miles traveled on U.S. roads in 2016 increased by 2.2 percent, and resulted in a fatality rate of 1.18 deaths per 100 million VMT – a 2.6-percent increase from the previous year."[3]

These traffic deaths are real. No one disputes that they happen. But can the same be said for the claim that PM in outdoor air kills people? What follows is an examination of that question.[4]

Deaths related to air pollution are associated with three major pollution incidents during the 20th century, discussed in detail below. These incidents prompted scientific research into the precise agent or agents in the ambient air responsible for the deaths. Particulate matter was one of the agents under consideration. But as late as the 1980s, PM had not been singled out as a culprit.[5] Some researchers had hypothesized that perhaps some combination of acidic gases or aerosols and PM could be lethal under some circumstances to some people. But

because of data shortcomings and methodological weaknesses, the EPA could not draw definitive conclusions into the early 1990s. Then the dynamic at the agency changed.

In December 1993, Harvard researchers published an EPA-funded study in the *New England Journal of Medicine* reporting that $PM_{2.5}$ was statistically correlated with premature deaths. Prepared for the release of what is now known as the "Six City" study, the EPA was able to juice media coverage upon the study's publication by estimating that ambient PM from tailpipe and smokestack emissions kill 50,000 to 60,000 people per year.[6]

Fifteen months later, in March 1995, the principal authors of the Six City study published a much larger EPA-funded study that also reported that $PM_{2.5}$ was statistically correlated with premature death.[7] It also received considerable media coverage. For instance, a front-page *Arizona Republic* headline for what is now known as the "Pope" study, after lead author Brigham Young University economics professor C. Arden Pope III, emphasized the mutually confirmatory nature of the two studies—"Particulate pollution's lethal risk: Study affirms link to early deaths."[8]

Armed with these two studies, in July 1997 the EPA proceeded to issue its first-ever air quality standards for $PM_{2.5}$. The agency estimated that this rule would prevent an estimated 15,000 premature deaths per year.[9] Over the next 14 years, EPA staff and EPA-funded researchers would work to bolster their notion that $PM_{2.5}$ was not only a killer, but a killer demanding the severest regulation.

By 2004, the EPA had concluded that inhaling $PM_{2.5}$ in outdoor air could cause death either within hours or after decades of inhalation and that the elderly and sick were most or particularly vulnerable to the effect of $PM_{2.5}$.[10] By 2009, the EPA concluded that any inhalation of $PM_{2.5}$ could cause death.[11] Taken together, these two conclusions suggested that any level of $PM_{2.5}$ can kill within hours of inhalation—which essentially declared $PM_{2.5}$ the most toxic substance known to man.

These points were emphasized during the September 2011 congressional testimony of then-EPA Administrator Lisa Jackson. When asked about the nature of the health effects caused by $PM_{2.5}$ by Rep. Ed Markey (D-Mass.), Jackson replied:

> Particulate matter causes premature deaths. It's doesn't make you sick. It is directly causal to dying sooner than you should.

Further queried by Rep. Markey about the scope of the risk to public health, she replied:

> If we could reduce particulate matter to levels that are healthy, we would have an identical impact to finding a cure for cancer.[12]

At the time of Jackson's testimony, the American Cancer Society had estimated that cancer caused about 570,000 deaths per year.[13] So she was pegging the death toll from $PM_{2.5}$ at a similar level to that of cancer, about one in five deaths in the U.S. annually.

Does PM kill Anyone? Advocates of the notion that ambient PM causes deaths, including the EPA, claim that "thousands of studies" support their position.[14] Yet, the claim of "thousands of studies" is merely a rhetorical device to deter lay readers from questioning the alleged link between PM and excess deaths. Undeterred, we will examine the purported link between PM and death by the available lines of evidence:

1. Studies of human populations (epidemiology);
2. Clinical studies of humans (human experiments);
3. Animal studies; and
4. Real-world experiences.[15]

Epidemiologic Studies of Human Populations. Epidemiology is the statistical study of disease patterns in human populations. The aforementioned Six City and Pope studies are both epidemiologic studies. Those two studies and their ongoing progeny are the two lines of epidemiologic studies on which the EPA relies to this day as the main support for the claim that PM kills. Nevertheless, these studies are highly controversial, to say the least.

Both studies purport to statistically correlate exposure to $PM_{2.5}$ with premature death, defined as dying sooner than one otherwise would have without inhalation of $PM_{2.5}$. There are two major problems with this assumption. First, statistical correlation does not establish causation. Second, the studies' statistical correlations are very weak and not substantially different from correlations of zero.[16] The Six City and Pope studies' results are not meaningfully different from those of previous epidemiologic studies that had failed to lead the EPA to the conclusion that PM caused death.

Skeptical of the claims of the Six City study, in 1994 the EPA's board of independent science advisors, the Clean Air Scientific Advisory Committee (CASAC), asked the EPA, which funded the study, to provide to CASAC the study's raw data for the purposes of attempting to replicate the results. This is a standard procedure in science, but the EPA never even responded to CASAC's request.

Then in 1996, about six months before the EPA proposed to regulate $PM_{2.5}$ for the first time, CASAC completed its review of the agency's summary of the PM science. CASAC concluded there was insufficient evidence to support the claim that $PM_{2.5}$ was associated with death.[17] But the EPA ignored CASAC and moved on to propose $PM_{2.5}$ rules. After the EPA proposed its $PM_{2.5}$ rules, Congress asked the EPA to provide it the data underlying the Six City and Pope studies for purposes of independent replication of study results.[18] The agency refused to provide the data and, in July 1997, finalized its rules for $PM_{2.5}$.

This "secret science" controversy went dormant until about 2011, when Congress again began asking the EPA for the raw data underlying the Six City and Pope studies. An unresponsive EPA drove Congress to subpoena it for the data in 2013.[19] The EPA ignored the subpoena, leaving that Congress, and the next two Congresses, to pass bills barring the agency from relying on secret science—like that underlying the Six City and Pope studies—as justification for taking regulatory action. The full Senate never took up any of the secret science bills. The failure of House efforts to ban secret science led to the EPA science

transparency proposal in April 2018, which, if finalized as proposed, would ensure that, over time, more of the data and models underlying EPA's regulatory science are available for independent validation.

Unsurprisingly, the epidemiology in the Six City and Pope studies remains controversial. The "secret science" controversy cannot be resolved as long as the EPA continues to refuse to make the data at issue available to independent scientists who could attempt to replicate the claims made in the Six City and Pope studies.

This is especially important given that there are a number of epidemiology studies that report or indicate no association between PM and death. Some recent studies include:

- **Enstrom reanalysis of the Pope study.** Former University of California, Los Angeles epidemiologist James E. Enstrom reanalyzed the Pope study with improved exposure data and reported no association between PM2.5 and death.[20]

- **California study.** A team including University of North Carolina statistician Richard Smith and S. Stanley Young of the National Institute of Statistical Sciences, who are both now members of the EPA's Science Advisory Board, reported no association between $PM_{2.5}$ and death in an analysis of virtually every death—more than 2 million—that occurred in California between 2000-2012.[21]

- **Cox "natural experiment" study.** Reduction in $PM_{2.5}$ levels should be associated with reduced death rates. But Anthony Cox, now chairman of the EPA's CASAC, reported that, although $PM_{2.5}$ levels declined 30 percent in the U.S., no associated decline in death rates was observed.[22]

Reasons for the large number of published research linking $PM_{2.5}$ with death include (1) publication bias and (2) immense government funding, in excess of $580 million from EPA alone, for $PM_{2.5}$ researchers.[23] The number of studies on one side of a scientific debate is not an indication of the validity of that point of view.

Moreover, in litigation with this author over its $PM_{2.5}$ clinical research program involving humans, the EPA admitted to the federal court that the $PM_{2.5}$ epidemiology studies, because of their exclusively statistical nature, prove nothing by themselves. The EPA told the court it was conducting the human experiments because:

> Epidemiologic studies do not generally provide evidence of direct causation.[24]

The purpose of the human experiments, according to the EPA, was to develop a medical or biological explanation to support the merely statistical, and controversial results of the $PM_{2.5}$ epidemiology studies.

EPA Clinical Studies of Humans. For more than 20 years, the federal government has conducted clinical studies in which humans are exposed to $PM_{2.5}$ to see the effect of exposures on human subjects. The EPA has a facility at the University of North Carolina

(UNC) School of Medicine, where it conducts such research. Universities around the country have also received EPA grants to conduct similar research. In these studies, humans—who are often elderly or afflicted with heart disease, asthma, diabetes, or a combination of these—are exposed in a controlled chamber to very high levels of $PM_{2.5}$, as much as 20 times the national $PM_{2.5}$ standard, for up to two hours at a time.[25]

Keeping in mind the EPA's claims that any inhalation of $PM_{2.5}$ can cause death within hours, and that the elderly and sick are particularly vulnerable to the dangers of inhaling $PM_{2.5}$, these experiments raise obvious ethical and legal difficulties, which will not be discussed here. However, it is worth noting that in the legally mandated disclosure forms submitted to the UNC institutional review board responsible for reviewing the experiments, the EPA never disclosed that it had already taken the position that any exposure to $PM_{2.5}$ could cause death within hours and that the elderly and sick were particularly vulnerable.[26]

Over the years, the EPA has experimented on over 6,000 human subjects with a variety of air pollutants and mixtures thereof. *Not a single human has been harmed, much less killed, by exposure to very high levels of $PM_{2.5}$.*[27] The only fatality associated with federally funded air pollution experiments occurred in 1996, when a University of Rochester student was accidentally fatally overdosed with an anesthetic during a procedure known as a bronchoscopy.[28]

EPA Laboratory Animal Experiments. In addition to laboratory experiments on humans, the EPA has conducted and funded in university laboratory experiments in which various animals, such as rodents and dogs, were exposed to $PM_{2.5}$ at levels hundreds of times greater than occur in outdoor air. Despite the high exposures, no laboratory animal has ever been killed by $PM_{2.5}$ in these experiments.[29]

Real-World $PM_{2.5}$ Experiences. There are myriad real-world experiences with ambient $PM_{2.5}$. Advocates of the PM-kills claims routinely distort or ignore them for several reasons.

Past and Current Episodes of Fatal Air Pollution. The 20th century witnessed three episodes of extreme air pollution associated with fatalities:

- Meuse Valley, Belgium, December 1930;
- Donora, Pennsylvania, October 1948; and
- London, UK, winter 1952.

All three incidents occurred because of unusual weather inversions that trapped and concentrated the emissions of a variety of air pollutants in the air.

Advocates of the PM-kills hypothesis cite these episodes as evidence that PM kills and justification for EPA $PM_{2.5}$ regulation. But published reports written by experts in the near-term aftermath of these incidents tell a different story.[30]

- **Meuse Valley.** Researchers deemed the carbon PM or soot by itself to be "innocuous." Deaths were blamed on unidentified "irritant gases" that might have had been adsorbed onto particles.[31]

- **Donora.** Autopsies indicated that deaths were caused by acidic gases destroying respiratory tract tissue. PM by itself was not implicated.[32]

- **London.** The famous London Smog occurred simultaneously with a deadly influenza epidemic. To the extent that increased deaths could possibly be attributed to the poor air quality, it was the concentration of acidic gases, not PM by itself that was blamed.[33]

The hypothesis of acidic gases causing the deaths is supported by the high air pollution levels experienced today in China that occur without a contemporaneous spike in deaths. Quite simply, it is difficult to blame $PM_{2.5}$ for deaths that have not occurred.

$PM_{2.5}$ levels in highly polluted Chinese cities can exceed levels 100 times higher than the average $PM_{2.5}$ levels found in air in the United States. If $PM_{2.5}$ were as lethal as claimed, contemporaneous spikes in death rates in Chinese cities would be evident, but none have been reported.[34] The only actual deaths reported associated with air quality involve visibility problems, such as in vehicular accidents. A possible explanation for the absence of deaths in Chinese cities is that, despite the obviously highly polluted air, sulfur dioxide levels (a source of the acidic, irritant gases present in the 20th century incidents) in Chinese cities is well within safe levels.

$PM_{2.5}$ from Smoking. Smokers are exposed to relatively immense amounts of $PM_{2.5}$ as compared to levels in outdoor air. Someone breathing typical U.S. outdoor air, may inhale 100 micrograms (millionths of a gram) *per day* of $PM_{2.5}$. Smoking a single cigarette, however, exposes a smoker to *10,000 to 40,000 micrograms in just a few minutes*.[35] Someone smoking an unfiltered marijuana joint may inhale as much as 160,000 micrograms in just a few minutes.[36]

Given these immense $PM_{2.5}$ exposures and the absence of reports of anyone ever dying in the immediate aftermath of smoking anything, the claim that inhaling any amount of $PM_{2.5}$ from outdoor air can result in death within hours rings hollow.

What does the epidemiology of smoking tells us about long-term exposures to $PM_{2.5}$? Someone living to age 80 or so breathing average U.S. air will inhale an ounce or so in total of $PM_{2.5}$[37]—an amount that can be visualized as two sugar packets' worth of $PM_{2.5}$. A recent study in the *New England Journal of Medicine* reported that people who stop smoking by age 35 have normal life expectancy, which translates to about 80 years for white women.[38] Assuming such an individual had smoked half a pack of cigarettes per day, she would have inhaled over four pounds of $PM_{2.5}$. What does it say about the lethality of $PM_{2.5}$ on a long-term basis if a non-smoker and smoker can have the same life expectancy despite the vast differences in $PM_{2.5}$ inhaled—a sugar packet versus more than a sugar bag's worth, respectively?

Recall that the EPA says the elderly and sick are most vulnerable to the effects of $PM_{2.5}$. Yet physicians now prescribe medical marijuana to patients that include the elderly and sick. Presumably the physicians are not violating the "first, do no harm" part of the Hippocratic Oath. The absence of deaths among medical marijuana users indicates they are not.

Occupational Exposures to $PM_{2.5}$. Keeping in mind that someone inhaling average U.S. outdoor air will inhale about 100 micrograms of $PM_{2.5}$ per day, federal regulations tightened in 2016 permit coal miners to inhale as much as 12,000 micrograms of $PM_{2.5}$ per day. For the 40 years prior to the 2016 change, coal miners could be exposed to 16,000 micrograms per day. So coal miners may inhale more than 100 times more $PM_{2.5}$ than people who do not work in coal mines. But guess what? On average coal miners live longer than non-coal miners.[39]

Another relevant example is that of workers' exposure to high levels of diesel exhaust, which is 95 percent $PM_{2.5}$. In 2012, the U.S. National Cancer Institute reported longer life expectancy among a population of 12,315 operators of forklifts, locomotives and other heavy equipment compared to all other workers.[40]

Conclusion. Summarizing the scientific evidence on $PM_{2.5}$ and death:

1. The $PM_{2.5}$ epidemiology is conflicted and controversial to say the least. But even if it were not, the EPA has admitted to a federal court that because of its statistical nature, the $PM_{2.5}$ epidemiology is an insufficient basis for concluding that $PM_{2.5}$ causes death.
2. Because the $PM_{2.5}$ epidemiology is insufficient for determining whether $PM_{2.5}$ causes death, the EPA and others have conducted numerous clinical experiments in which humans were exposed to very high levels of $PM_{2.5}$. No deaths or harm were reported in any of these experiments.
3. The EPA has conducted or sponsored numerous laboratory experiments in which various types of animals were exposed to very high levels of $PM_{2.5}$. No deaths have been reported.
4. The deaths that occurred in historical air pollution incidents were attributed by contemporaneous researchers to acidic or irritant gases in the atmosphere, not to $PM_{2.5}$ by itself. Because emissions of these gases are now tightly controlled, such temperature inversions no longer present a lethal threat — even in Chinese cities.
5. The most common and acute exposure to $PM_{2.5}$ is tobacco and marijuana smoke. The epidemiology of smoking debunks the notions that either short-term or long-term exposure to ambient $PM_{2.5}$ is lethal.
6. Workers heavily exposed to $PM_{2.5}$ live longer than average workers.

It is clear that the available evidence fails to link $PM_{2.5}$ in outdoor air with death. Therefore, a benefit-cost analysis for the SAFE rule need not concern itself with $PM_{2.5}$ and death. Whatever minor changes in $PM_{2.5}$ levels that might be brought about by the proposed SAFE rule—$PM_{2.5}$ levels could slightly increase or even decrease because of the rule—will not cause or prevent deaths or change death rates.

Notes

[1] The Safer Affordable Fuel-Efficient (SAFE) Vehicles Rule for Model Years 2021-2026 Passenger Cars and Light Trucks, *Federal Register*, Vol. 83, No. 165 (August 24, 2018), p. 42986, https://www.federalregister.gov/documents/2018/08/24/2018-16820/the-safer-affordable-fuel-efficient-safe-vehicles-rule-for-model-years-2021-2026-passenger-cars-and.
[2] Zack Colman and Scott Waldman, "What's deadlier—car crashes or pollution? Trump picks one," Climatewire, August 1, 2018, https://www.eenews.net/climatewire/stories/1060091777.
[3] National Highway Traffic Safety Administration, "USDOT Releases 2016 Fatal Traffic Crash Data. October 6, 2017," news release, https://www.nhtsa.gov/press-releases/usdot-releases-2016-fatal-traffic-crash-data.
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[6] The six communities analyzed in the study were Watertown, Massachusetts; Harriman, Tennessee; specific census tracts of St. Louis; Steubenville, Ohio; Portage, Wisconsin; and Topeka, Kansas. Douglas W. Dockery et al, "An Association between Air Pollution and Mortality in Six U.S. Cities," *New England Journal of Medicine*, Vol. 329, No. 4 (December 9, 1993), pp. 1753-1759, https://www.nejm.org/doi/full/10.1056/NEJM199312093292401#article_references.
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[12] Lisa Jackson, Testimony before the House of Representatives Energy and Commerce Subcommittee on Oversight and Investigations, September 22, 2011, https://www.c-span.org/video/?c4492073/clip-environmental-protection-agency-oversight. Testimony beginning at 1:50:30 of the hearing.
[13] American Cancer Society, "Cancer Facts and Figures 2011," p. 6-182, https://www.cancer.org/research/cancer-facts-statistics/all-cancer-facts-figures/cancer-facts-figures-2011.html.
[14] EPA, Fact Sheet for the 2012 PM NAAQS, https://www.epa.gov/sites/production/files/2016-04/documents/health_2012_factsheet.pdf.
[15] Milloy, *Scare Pollution*.
[16] Epidemiologic correlations are statistical associations often calculated as "relative risks" (RRs) or "odds ratios" (ORs)—terms that are interchangeable for the present discussion. An RR of 1.0 is a zero correlation, meaning no statistical relationship between the variables at issue, in this case $PM_{2.5}$ and death. The Six City and Pope study correlations are on the order of 1.1. Although their reported values are greater than 1.0, these RRs are so close to zero that they effectively amount to zero correlations. For example, in his famous 1965 address to the Royal Society, British epidemiologist Sir Austin Bradford Hill said that RRs on the order of 2.0 or less are unreliable and may very well be the result of poor data quality or chance. Austin Bradrofd Hill, "The Environment and Disease: Association or Causation," *Proceedings of the Royal Society of Medicine*, Vol. 58, No. 5 (1965), pp. 295-300, https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1898525/pdf/procrsmed00196-0010.pdf.
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[19] House Committee on Science, Space and Technology. U.S. House of Representatives, Committee on Science, Space, and Technology, "Smith Subpoena's EPA's Secret Science," news release, August 1, 2013, https://science.house.gov/news/press-releases/smith-subpoenas-epa-s-secret-science.
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[27] Ibid.
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[29] Milloy, *Scare Pollution*, pp. 183-184.
[30] Ibid., pp 199-208.
[31] Ibid., pp. 199-201.
[32] Ibid., pp. 201-206.
[33] Ibid., pp. 207-208.
[34] Ibid., pp 209-210.
[35] Andrew J. Ghio et al., "Particulate Matter in Cigarette Smoke Alters Iron Homeostasis to Produce a Biological Effect," *American Journal of Respiratory and Critical Care Medicine*, Vol. 178, No. 11 (December 1, 2008), pp. 1130-1138, https://www.atsjournals.org/doi/abs/10.1164/rccm.200802-334OC.
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