Scientific Distortions in Fine Particulate Matter Epidemiology

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ABSTRACT

The theoretical prevention of premature deaths from the inhalation of fine particulate matter is being used by the U.S. Environmental Protection Agency (EPA) to justify the National Ambient Air Quality Standard (NAAQS) and multibillion dollar regulations across the U.S., including the EPA Clean Power Plan and the California Air Resources Board (CARB) Truck and Bus Regulation. The epidemiology is severely flawed. Fine particulates probably make no significant contribution to premature mortality in the U.S. The publication of null findings has been blocked or marginalized and studies claiming excess mortality need to be reassessed.

Basics of Fine Particulate Matter

Fine particulate matter ($PM_{2.5}$) is defined by its size (≤2.5 μm diameter), not its composition. Major sources in the U.S. are forest fires, commercial and residential burning, and diesel engines. In California, a major source is China; on some days up to 30% of fine particulates had crossed the Pacific Ocean.

Of these invisible particles, the average adult in the U.S., based on actual 2015 exposure levels, would inhale about 1 gram in an 80-year lifespan, assuming that he breathes about 10,000 liters of air a day at rest. For comparison, the amount inhaled while smoking 100 cigarettes is about 4 grams.[1]

In 1997, the EPA established the NAAQS for $PM_{2.5}$ as 15 μg/m³. This was lowered to 12 μg/m³ in 2012. This standard has been largely justified on the basis of secret science epidemiology. These regulations are very powerful and impose huge costs on American businesses. The $PM_{2.5}$ NAAQS, has been used to justify several multi-billion-dollar rules, such as the EPA Clean Power Plan and the CARB Truck and Bus Regulation.

Although a significant effect from such extremely low levels is on its face highly implausible, the stringent EPA regulations are justified primarily by a claim of preventing premature deaths, assuming a value of $10 million per statistical life saved. The controversy over the issue was brought to general attention in 2002 by Professor Robert Phalen.[2]

Epidemiology of Fine Particulate Matter

The EPA claim that $PM_{2.5}$ causes "premature deaths" is based on epidemiologic cohort studies purporting to show that the relative risk (RR) for total mortality is slightly greater than 1.0 in U.S. populations exposed to higher levels of $PM_{2.5}$. No etiologic mechanism has been established, and there is no experimental evidence that inhalation of 1 g or 5 g of $PM_{2.5}$ can cause death. Weakly positive RRs do not prove causality. Major difficulties include: (1) geographic and temporal variation in $PM_{2.5}$ mortality risk; (2) exaggeration of actual human exposure by $PM_{2.5}$ monitors, which measure ambient outdoor levels far from the subjects; and (3) confounding variables such as co-pollutants. Moreover, the key study relied on by EPA, the American Cancer Society (ACS) 1982 Cancer Prevention Study (CPS II)[3] is seriously flawed.

Reanalysis of the American Cancer Society Cancer Prevention Study II (ACS CPS II)

CPS II began in 1982 and is similar to the original CPS I, which began in 1959. The seminal paper published by Pope et al. in 1995[3] was so controversial that the Health Effects Institute (HEI) sought applications from teams consisting of two to four epidemiologists, statisticians , and air pollution exposure experts to conduct a reanalysis, including "sensitivity analyses to test the robustness of the original findings and interpretations to alternative analytic approaches."[4] The HEI Reanalysis published in 2000 did not complete the mandated sensitivity analysis to assess the effect of alternate data.[5] HEI published a report in 2009,[6] which extended the mortality follow-up of the study from 1989 to 2000, but it did not incorporate the EPA Inhalable Particulate Network (IPN) $PM_{2.5}$ data[7,8] that I had called to the authors' attention in my 2005 paper.[9]

In 2016 I was able to obtain access to data in an original 1982-1988 version of CPS II. The data had been previously inaccessible since 1995 despite a congressional subpoena and repeated requests by different agencies. I am the only independent scientist who has gained access to the individual level data in both CPS I and CPS II. I was able to reproduce the same key results as Pope et al. by doing exactly what the authors did in 1995.[3] However, their results were sensitive to the $PM_{2.5}$ data that they used and to their particular analysis.

HEI did not follow its own mandate to conduct a comprehensive reanalysis. In particular, their sensitivity analysis was not done properly. Of the 13 teams that submitted reanalysis applications, HEI selected a 31-member team based in Canada, headed by statistician Daniel Krewski. It included a geographer, Michael Jerrett, and another statistician, Richard Burnett, but only had one epidemiologist, Yue Chen. Chen's degree was from Shanghai Medical University, and he was not a coauthor on either the 2000 HEI report[5] or the 2009 HEI report.[6] Thus, to reanalyze a major U.S. epidemiological study, HEI used a Canadian team that had essentially no epidemiologist.

An early clue to the existence of problems is seen in Figure 21 in the 2000 HEI Reanalysis Report.[5] (Figure 1 in this article.) This map shows that in 50 cities across the U.S. the level of $PM_{2.5}$ mortality risk varies. Higher risks were found mainly in the Rust Belt or the Ohio Valley, and levels were actually reasonably low in California and throughout most of the western part of the U.S. Beginning in 2002, I asked the head of HEI, Daniel Greenbaum, and its principal scientist, Aaron Cohen, to send me the underlying data for that map. For 16 years, they have consistently refused to reveal this data to me.

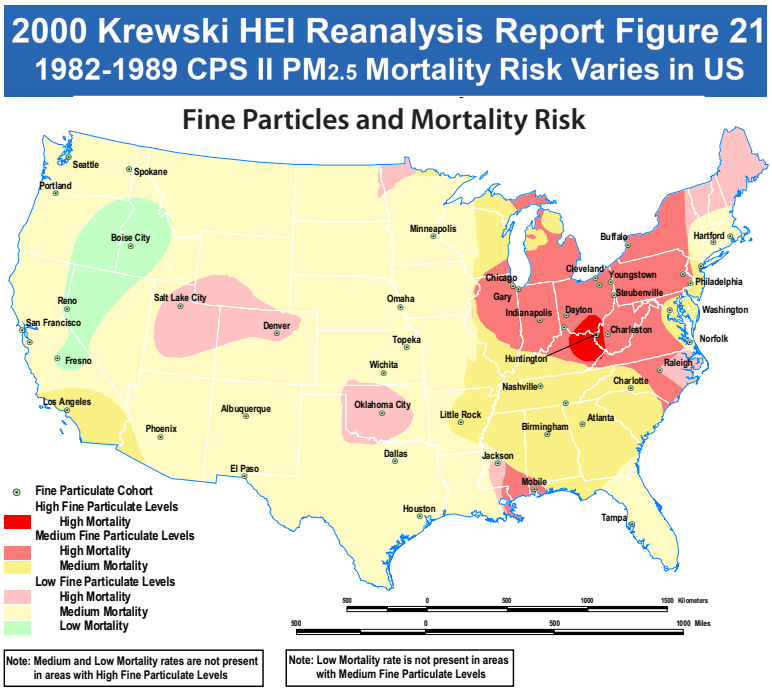

Figure 1. $PM_{2.5}$ Levels and Mortality Risk in the U.S. [Reprinted from 2000 HEI Reanalysis Report,[5] with permission.]

My analysis of the CPS II data revealed that the county of residence of subjects could be approximated based on the ACS Division and Unit numbers. The CPS II data were collected by about 70,000 researchers, including myself, who enrolled 1.2 million subjects in Fall 1982. I performed an analysis comparable to the HEI Reanalysis, as shown in Table 1. The $PM_{2.5}$ data labeled IPN in the table was published in EPA reports from the Inhalable Particulate Network (IPN) by David Hinton et al. in 1984[7] and 1986.[8] Because of the evasions that I have experienced in attempting to obtain information from HEI, I took a closer look at the 2000 HEI Reanalysis Report and found it actually contains the data that I used, although in a mislabeled and somewhat altered form. I have designated that data as HEIDC, which is labeled $PM_{2.5}$ DC in the 2000 Report. This data was indirectly referred to in a couple of places in the 2000 HEI report, although it was not analyzed.

Enstrom 2017 Analysis of $PM_{2.5}$ and Total Mortality During 1982-1988 in ACS CPS II Cohort: HEIDC

1979-83 $PM_{2.5}$	Subjects	Relative Risk (95% CI)
Fully Adjusted for 50 Counties in Continental US		
IPN [Hinton]	195,215	1.025 (0.990-1.061)
HEIDC [PM2.5 DC]	216,897	1.024 (0.987-1.061)
HEI [PM2.5 OI MD]	195,215	1.082 (1.039-1.128)
Fully Adjusted for Ohio Valley (IN,KY,OH,PA,WV)		
IPN	42,174	1.050 (0.918-1.201)
HEIDC	43,945	1.048 (0.922-1.191)
HEI	42,174	1.111 (0.983-1.256)
Fully Adjusted for States Other Than Ohio Valley		
IPN	153,041	0.975 (0.936-1.051)
HEIDC	172,952	0.960 (0.919-1.003)
HEI	153,041	1.025 (0.975-1.078)

Table 1. Enstrom Analyses of ACS CPS II Data Using Three Sources of $PM_{2.5}$ Data

Thus, using the HEI $PM_{2.5}$ data of Pope et al.,[3] there is a statistically significant slight increase in RR of 1.082. That means that if the $PM_{2.5}$ level increases by 10 µg/m³, the risk of dying goes up by about 8%. But, using the IPN $PM_{2.5}$ data, the effect is nonsignificant, RR = 1.025 (95% CI, 0.990-1.061). Note that if one divides the U.S. into the Ohio Valley (Indiana, Kentucky, Ohio, Pennsylvania, and West Virginia) and the rest of the country, the RR is indistinguishable from 1.0, no matter what $PM_{2.5}$ data is used. Only by combining the Ohio Valley, which has both a higher mortality risk and a higher level of $PM_{2.5}$, with the rest of the country can HEI show a statistically significant effect.

My reanalysis[10] has been published online since Mar 28, 2017, and so far its validity has not been challenged.

The selection of data by HEI was also very interesting, as seen in Table 2. There were actually 11 counties in California that were part of the IPN network, and the HEI analyses omitted 7 of the 11 counties for reasons the authors have not explained. HEI had data from 50 different cities, and the only ones they included from California were Fresno, Los Angeles, San Francisco, and San Jose (in Santa Clara County). Two other counties that represent the extremes in $PM_{2.5}$ levels are highlighted in the table. The Pope 1995 paper[3] was based primarily on these extremes. HEI had Albuquerque, N.M., at 9 µg/m³, as the lowest value, and Huntington, W.V., at 34.4 µg/m³, as the highest value. This is curious because the data that comes from the IPN network actually shows different high and low values. In fact, there is no measurement in the IPN for Huntington, W.V., but rather for Wheeling, W.V., listed in the IPN column. From the table, both the low and the high values are in California, both of which omitted from the HEI analysis. The low value is 10.6 µg/m³ in Santa Barbara County, and the high value is 42.0 µg/m³ in Riverside County. The $PM_{2.5}$ DC data that I found in the 2000 HEI Report appendix table, labeled HEIDC by me, had more than 50 cities, but only five of the 63 total cities were from California. The IPN network as a whole has about 85 cities. These major inconsistencies need to be addressed by these investigators. And so far, there is nothing but silence. This is only one of the issues that must be addressed if the investigators want to maintain any credibility.

CA NM WV Counties with $PM_{2.5}$ Values Used in Pope 1995, HEI 2000, HEI 2009, Enstrom 2017

State	ACS Div-Unit	County	1979-83 $PM_{2.5}$ (µg/m³) IPN (N=85)	HEIDC (N=63)	HEI (N=50)
CA	06001	Alameda	14.3882		
CA	06002	Butte	15.4525		
CA	06003	Contra Costa	13.9197		
CA	06004	Fresno	18.3731	10.3	10.3
CA	06008	Kern	30.8628		
CA	06051	Los Angeles	28.2239	26.8	21.8
CA	06019	Riverside	42.0117		
CA	06020	San Diego	18.9189	18.9	
CA	06021	San Francisco	16.3522	16.4	12.2
CA	06025	Santa Barbara	10.6277		
CA	06026	Santa Clara	17.7884	17.8	12.4
NM	34201	Bernalillo	12.8865	12.9	9.0
WV	58117	Ohio	23.9840		[33.4]

Table 2. Comparison of Data on $PM_{2.5}$ and Mortality from Enstrom and HEI[9]

Relationship between $PM_{2.5}$ and Mortality in California

Because of the Feb 26, 2010, conference in Sacramento, which I attended along with Professor Robert Phalen, other prominent scientists, and impacted business groups, we were able to get an analysis done by HEI that dealt with the California portion of the national CPS II results. The California data was partitioned out from the national analysis in the 2009 HEI Report.[6] Based on the four HEI California counties shown in Table 2, the RR is about 0.9, significantly below 1.0, as shown in Table 3. This inverse relationship was reproduced using either the HEI data or the IPN data. Of course, this relationship cannot be etiologically correct, but it shows what can result from data omission and manipulation.

Enstrom 2017 Analysis of $PM_{2.5}$ and Total Mortality During 1982-1988 in California ACS CPS II Cohort Compared with Krewski 2010 HEI Special Analysis

1979-83 $PM_{2.5}$	Subjects	Relative Risk (95% CI)
Enstrom 2017 Fully Adjusted For 1982-1988 Deaths		
IPN (4 Counties)	36,201	0.879 (0.805-0.960)
HEI (4 Counties)	36,201	0.870 (0.788-0.960)
Krewski 2010 Fully Adjusted For 1982-1989 Deaths		
"Same" Standard Cox Model		
HEI (4 Counties)	40,408	0.872 (0.805-0.944)
"Different" Standard Cox Model		
HEI (4 Counties)	38,925	0.893 (0.823-0.969)

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Table 3. Relative Risk for $PM_{2.5}$ and Mortality in California Based on Four Counties

There are actually six California cohorts that have been used to analyze the relationship between $PM_{2.5}$ and total mortality, as shown in Table 4. The cohort that I initially used is labeled CA CPS I;[9] the cohort used by Jerrett et al.[11] is labeled CA CPS II. The Adventist Health Study of Smog (AHSMOG) was the original cohort study in California.[12] There are also the California Teachers Cohort,[10] the "West" portion of the Medicare Cohort Air Pollution Study (MCAPS),[13] and the National Institutes of Health-American Association of Retired Persons (NIH AARP) cohort, which was published in 2016 by Thurston et al.[14] The NIH AARP cohort is supposed to be an open access database, but is apparently currently controlled by Thurston. I have been able to get access to only the California portion of the data, and my analysis shows no effect in California. Averaging all six cohorts gives an RR of exactly 1.00, which means no relationship between $PM_{2.5}$ and total mortality.

The lack of an effect in California might explain why Pope et al.[3] omitted seven California cities from the national analysis. As Figure 1 shows, there is tremendous variation across the country. Yet the most severe regulations are in California, despite the clear absence of mortality risk there!

$PM_{2.5}$ and Total Mortality in California: Six Cohorts

Author & Year	CA Cohort	Relative Risk (95% CI)
McDonnell 2000	AHSMOG	RR ~ 1.03 (0.95-1.12)
Enstrom 2005	CA CPS I	RR = 1.00 (0.98-1.02)
Zeger 2008	MCAPS "West"	RR = 0.99 (0.97-1.01)
Jerrett 2011 9 RRs	CA CPS II	RR = 1.00 (0.99-1.01)
Ostro 2015	CA Teachers	RR = 1.01 (0.98-1.05)
Thurston 2016	CA NIH AARP	RR = 1.02 (0.99-1.04)
Weighted Average (Six Cohorts)		**RR = 1.00 (0.99-1.01)**

(http://scientificintegrityinstitute.org/ORI111116.pdf) 6

Table 4. $PM_{2.5}$ and Total Mortality in Six California Cohorts

Both my analysis and that by Thurston et al. on the NIH AARP cohort,[14] summarized in Table 5, show no effect nationwide or in California.

$PM_{2.5}$ and Total Mortality in US and California: Enstrom 2017 re 1982-1988 ACS CPS II Cohort Thurston 2016 re 2000-2009 NIH AARP Cohort

Geographic Area	Subjects	Relative Risk (95% CI)
United States		
Enstrom Analysis Fully Adjusted for 1982-1988 Deaths		
85 Counties	269,766	1.023 (0.997-1.049)
Thurston Analysis Fully Adjusted for 2000-2009 Deaths		
6 States & 2 Cities	517,041	~1.025 (1.000-1.049)
California		
Enstrom Analysis Fully Adjusted for 1982-1988 Deaths		
11 Counties	60,521	0.992 (0.954-1.032)
Thurston Analysis Fully Adjusted for 2000-2009 Deaths		
58 Counties	160,209	~1.017 (0.990-1.040)

Table 5. Comparison of Enstrom and Thurston Analyses for U.S. and California

An International Perspective on $PM_{2.5}$

Despite the null effect shown by their own data and analyses, prominent advocates of drastic measures to reduce $PM_{2.5}$ levels state in a major paper in the May 13, 2017, *Lancet* that ambient $PM_{2.5}$ was the fifth-ranking mortality risk factor worldwide in 2015. Aaron J. Cohen, until recently HEI Principal Scientist, is the lead author, and Pope is a coauthor. The study is part of the World Health Organization (WHO) Global Burden of Disease (GBD) Project and was largely funded by HEI. The article claims that $PM_{2.5}$ causes 4.2 million deaths annually worldwide, with 88,000 deaths in the U.S. (see Table 6). The mean $PM_{2.5}$ level is 8.4 μg/m³ in the U.S. and 58.4 μg/m³ in China. Clearly, the $PM_{2.5}$ level and premature deaths are low in the U.S. and high in China, India, and Africa.

May 13, 2017 (on line April 10, 2017) *Lancet*
'Global Burden of Disease' by Cohen & Pope

2015 Deaths Attributed to $PM_{2.5}$

Table 2

Country	Deaths	Death Rate (per 100,000)	Mean $PM_{2.5}$ ($\mu g/m^3$)
USA	88,400	18.5	8.4
China	1,108,100	84.3	58.4
India	1,090,400	133.5	74.3
Pakistan	135,100	136.3	65.0
Bangladesh	122,400	133.2	89.4
World	4,200,000		

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Table 6. Global Deaths Attributed to $PM_{2.5}$[15]

Agenda-driven Science

Since publishing my 2005 critique of the relationship between $PM_{2.5}$ and total mortality[9] and my 2017 critique,[10] I have sent numerous requests to Pope, ACS, HEI, and others, inviting a rebuttal. I have received no response that confirms or refutes any of my analyses. It has, however, been incorrectly asserted that, "The study by Enstrom does not contribute to the larger body of evidence on the health effects of $PM_{2.5}$." ACS has criticized me for having CPS II data that they have deliberately tried to keep secret. My invitations to authors and ACS officials to attend meetings, teleconferences, and symposia have simply been ignored. They even ignored an August 1, 2013, subpoena from the U.S. House Science, Space, and Technology Committee.

The control over air pollution research and assessments that is recognized by EPA is not based on special expertise in epidemiology. Pope, the self-proclaimed "world's leading expert on the effects of air pollution on health," is a professor of economics at Brigham Young University and holds a 1981 Ph.D. in agricultural economics from Iowa State University, where he studied the dynamics of crop yields. Michael Jerrett, who is one of the most prolific publishers and a member of the HEI reanalysis team, has a 1996 Ph.D. in geography from the University of Toronto, and no formal training in epidemiology. Aaron J. Cohen, until recently HEI's Principal Scientist, does hold a 1991 D.Sc. degree in epidemiology from Boston University, but he has badly misused the principles and standards of epidemiology. Although he supervised the 1998-2000 HEI Reanalysis Project, he has refused to clarify findings from this project and has refused to confirm or refute the findings in my 2017 CPS II reanalysis. It is very disturbing that ACS has allowed CPS II data to be used for more than 20 years for research that misuses the principles and standards of epidemiology and that has nothing significant to do with cancer.

The principal qualification for admission to the elite circle of influence appears to be dedication to the agenda of global controls on economic activity via air pollution regulations. The conclusion reached by researchers is apparently predetermined, as stated in the last paragraph of the GBD study on ambient air pollution: "As the experience in the U.S. suggests, changes in ambient $PM_{2.5}$ associated with aggressive air quality management programmes, focused on major sources of air pollution including coal combustion, household burning of solid fuels, and road transport, can lead to increased life expectancy over short timeframes."[15]

What is the state of scientific integrity? It is very dangerous to one's career to criticize views backed by powerful interests, and I do it only because I believe current trends are anti-science and dangerous to our country. Simply being a passive observer is no longer acceptable.

To disclose my own background, I obtained a Ph.D. in physics in 1970, but I became an epidemiologist starting in 1973 in order to apply the rigorous principles of physics to observational epidemiology. I had a long career as a research professor and researcher at the UCLA School of Public Health. My research has examined the influence of environmental and lifestyle factors on mortality, and has on occasion reached politically incorrect conclusions. My research in air pollution epidemiology has been strongly influenced by Dr. Frederick Lipfert and Professor Robert Phalen. In February 2010 I was terminated from UCLA without warning and told that my "research is not aligned with the academic mission of the Department." In February 2015 I settled a three-year federal whistleblower retaliation lawsuit against UCLA and my termination was reversed. My case and some of the issues related to my air pollution epidemiology research have been discussed in this journal.[16]

My background and publications, including rejections of my research, often without peer review, are documented on my website, www.scientificintegrityinstitute.org. I believe that major journals simply will not accept articles that challenge the established view. Moreover, authors of the papers promoting $PM_{2.5}$ premature deaths omit null results, even their own. For example, Jerrett is the lead author of a 2007 study that shows no increased mortality associated with $PM_{2.5}$ in the CPS II cohort if the results are divided into five time periods.[17] Although researchers are paid millions of dollars, they're not under any obligation to address any of the concerns about their work. Those who disagree with the agenda are denied research funding.

We must prevent American science from following historical examples like that of Trofim Denisovich Lysenko. He was a phony plant geneticist, who gained the favor of Joseph Stalin because he didn't believe in Mendelian genetics. Lysenko's views controlled much of Soviet agriculture in the 1930s, 1940s, and 1950s, with devastating effect. False crop statistics were published, and dissenting scientists were purged. Nikolai Vavilov, a renowned plant geneticist, was imprisoned by Stalin and died of malnutrition.

Concerns about integrity in Western science are being raised. Richard Horton, editor of *The Lancet*, writes: "The case against science is straightforward: much of the scientific literature, perhaps half, may simply be untrue. Afflicted by studies with small sample sizes, tiny effects, invalid exploratory analyses, and flagrant conflicts of interest, together with an obsession for pursuing fashionable trends of dubious importance, science has taken a turn towards darkness."[18]

A U.S. House of Representatives bill called the Secret Science Reform Act was passed in 2014 and 2015 in order "to prohibit the Environmental Protection Agency from proposing, finalizing, and disseminating regulations or assessments based upon science that is not transparent or reproducible." The bill was revived in 2017 as the Honest and Open New EPA Science Treatment (HONEST) Act, labeled H.R. 1430, and was passed by the U.S. House of Representatives.

American science needs to guard against the heirs of Sinclair Lewis's protagonist in his 1927 novel *Elmer Gantry*, an itinerant preacher who is able to sell false religion to gullible people. We have prominent scientists who have successfully sold the notion that inhaling 1 g of invisible particles over an 80-year lifetime can cause premature death.

Conclusions

There is strong evidence from two large national cohorts that $PM_{2.5}$ does not cause premature deaths in the US. There is strong evidence that this relationship has been falsified by EPA, the Health Effects Institute, and leading researchers for more than 20 years. Better oversight to assure scientific integrity, such as access to data, transparency, and consideration of opposing views, is imperative.

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