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Subject: Examine Strong New Evidence Supporting EPA Transparency Rule

June 4, 2018

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Dear EPA Commenters,

I am writing because you made May 31, 2018 verbal public statements to the EPA Science Advisory Board that were critical of the proposed EPA Rule "Strengthening Transparency in Regulatory Science" (https://yosemite.epa.gov/sab/sabproduct.nsf/MeetingCalBOARD/7D239353BCECF85B852582600058B716?OpenDocument). Please read my May 30, 2018 written public comments in support of this Rule (https://yosemite.epa.gov/sab/sabproduct.nsf/D41456F68B9F91658525829D004DBD73/$File/88483770.pdf). My recent acquisition of and analysis of ACS CPS II data provide strong evidence that data access, transparency, and independent analysis must be essential aspects of EPA regulatory science.

My March 28, 2017 *Dose-Response* article "Fine Particulate Matter and Total Mortality in Cancer Prevention Study Cohort Reanalysis" (http://journals.sagepub.com/doi/full/10.1177/1559325817693345) found NO significant relationship between fine particulate matter (PM2.5) and total mortality during 1982-1988 in the ACS CPS II cohort, except for replication of the carefully selected results in the 1995 *AJRCCM* Pope article. Furthermore, none of my analyses or findings have violated the confidentiality of any CPS II subject. My null findings challenge the robustness and integrity of the positive relationship between PM2.5 and total mortality in the 1995 *AJRCCM* Pope article, the 2000 HEI Reanalysis Report, and the 2009 HEI Research Report 140. The 1995 *AJRCCM* Pope article, an example of 'pivotal regulatory science,' played the major role in the establishment of the 1997 EPA PM2.5 NAAQS and my Reanalysis shows that is severely flawed.

Dr. C. Arden Pope III and ACS have criticized my Reanalysis, but they have identified no errors in it. My May 29, 2018 *Dose-Response* "Response to Criticism" (http://journals.sagepub.com/doi/pdf/10.1177/1559325818769728) addresses their criticism and provides additional evidence of NO relationship between PM2.5 and total mortality in the CPS II cohort. My repeated requests to Dr. Pope and ACS (Dr. Susan M. Gapstur and Mr. W. Ryan Diver) for a full assessment of my findings and for collaboration have been rejected. Thus, I suggest that you ask them to confirm or refute my findings. My Reanalysis and Response, as they stand, indicate a clear need to reassess the EPA PM2.5 NAAQS. In any case, I have demonstrated the importance to EPA regulatory science of access to underlying data.

Please contact me if you would like to discuss my Reanalysis and/or the EPA Transparency Rule.

Thank you very much for your consideration.

Sincerely yours,

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Public Comments to
EPA Chartered Science Advisory Board Supporting
Proposed Rule "Strengthening Transparency in Regulatory Science"

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May 30, 2018

Below I have outlined strong justification for the EPA Proposed Rule "Strengthening Transparency in Regulatory Science" based on my recent access to ACS CPS II data.

1) My March 28, 2017 *Dose-Response* article "Fine Particulate Matter and Total Mortality in Cancer Prevention Study Cohort Reanalysis" (http://journals.sagepub.com/doi/full/10.1177/1559325817693345) found NO significant relationship between PM2.5 and total mortality during 1982-1988 in the ACS CPS II cohort, except for replication of 1995 *AJRCCM* Pope article results. My peer-reviewed results are based on my independent reanalysis of an old 1982-1988 version of the de-identified CPS II data that I recently obtained.

2) My null relationship findings challenge the robustness and integrity of the positive relationship between PM2.5 and total mortality in the 1995 *AJRCCM* Pope article, the 2000 HEI Reanalysis Report, and the 2009 HEI Research Report 140. In the 14 months since publication of my article, Pope and ACS have failed to assess the validity of my null findings, but have identified no errors. They have shown no willingness to cooperate on a matter that is very important to both air pollution epidemiology and EPA regulatory policy.

3) My attached May 29, 2018 *Dose-Response* "Response to Criticism" (http://journals.sagepub.com/doi/pdf/10.1177/1559325818769728) addresses the criticism by Pope and ACS of my March 28, 2017 Reanalysis, provides additional evidence of a null PM2.5-total mortality relationship, and includes more county-level CPS II data that does not violate subject confidentiality. Since my repeated requests to Pope, ACS, HEI, and other CPS II investigators have been rejected, the EPA SAB should ask ACS to cooperate with transparent analyses of the CPS II data, such as, the analyses I have requested. If ACS fully cooperates with SAB, then it might be useful to modify the EPA Transparency Rule to include a full cooperation option that does not require releasing actual data. If ACS fails to cooperate with SAB, then their CPS II research results should not be used for EPA regulations. I am certainly willing to cooperate with SAB on analyses using the 1982-1988 CPS II data that I possess.

4) My null CPS II findings basically agree with the null findings in the April 2016 *EHP* Thurston article (doi:10.1289/ehp.1509676), which analyzed the NIH-AARP Diet and Health Cohort and found NO significant relationship between PM2.5 and total mortality during 2000-2009. Since Thurston obtained these deidentified data from NIH, he should make his analytic data set available for additional analyses. Finally, SAB should request the publicly available Medicare data that was used by Schwartz for his recent *NEJM* and *JAMA* articles on PM2.5 deaths.

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Response to Criticism of "Fine Particulate Matter and Total Mortality in Cancer Prevention Study Cohort Reanalysis"

James E. Enstrom[1]

Keywords
epidemiology, PM2.5, deaths, CPS II, reanalysis

Response to Criticism by CPS II Investigators

Drs C. Arden Pope III (Pope), Daniel Krewski (Krewski), Susan M. Gapstur (Gapstur), Michelle C. Turner (Turner), Michael Jerrett (Jerrett), and Richard T. Burnett (Burnett),[1] as well as Gapstur and Otis W. Brawley (Brawley)[2] strongly criticized my *Dose-Response* article, Enstrom,[3] but they did not identify a single error, particularly regarding my findings of no relationship between fine particulate matter (PM2.5) and total (all-cause) mortality. Thus, my peer-reviewed findings showing no PM2.5-related deaths during 1982 to 1988 in the 1982 American Cancer Society (ACS) Cancer Prevention Study (CPS II) cohort stand unchallenged. In particular, my null findings indicate that the positive findings in 3 seminal publications by these investigators: Pope[4] and Health Effects Institute, HEI (2000)[5] and HEI (2009),[6] are not robust and not supportive of the claim that PM2.5 causes premature deaths. Instead of assessing the validity of my findings, these investigators focused on other aspects of their many analyses of CPS II data.

Their "Expanded Analyses of the ACS CPS-II Cohort" section inaccurately questions the validity of my findings: "The assertion regarding selective use of the CPS-II and PM2.5 data is false." I published prima facie evidence that their 1982 to 1989 PM2.5 mortality findings were indeed sensitive to selective use of PM2.5 and CPS II data. My evidence can be easily checked with minor modifications to the SAS programs that they used to calculate the findings in Table 34 of HEI (2009).[6] Instead of confirming or refuting my evidence, these investigators reiterated their various published analyses of PM2.5 deaths in CPS II, as summarized in their Table 1 and their Figure 1. All of their analyses could be just as sensitive to selective use of PM2.5 and CPS II data as the results in Pope,[4] HEI (2000),[5] and HEI (2009).[6]

Their "Deficiencies in Enstrom's Reanalysis" section does not identify a single error in my findings and suggests that they did not examine the data and findings in my article. For instance, they state, "In contrast, Enstrom[8] asserts that he estimates smaller PM2.5-mortality associations because he uses the 'best' PM2.5 data. He provides no evidence in support of this assertion nor does he provide any measures of the relative quality of models using alternative PM2.5 data." Strong evidence supporting my assertion is clearly presented in Tables 2 and 3 of my article and is described in the "Results" section on page 4. Then, they state, "It is not clear how or why his 'IPN' PM2.5 data differ from the 'HEI' PM2.5 data—especially given that these data come from the same monitoring network." The differences between the Inhalable Particulate Network (IPN) PM2.5 and HEI PM2.5 data are clearly shown in my Appendix Table A1 and discussed in the "Conclusion" section on page 6. To make sure that these differences are fully recognized and understood, an expanded version of Appendix Table A1 is shown in Table 1.

Their "Broader Evidence" section is not relevant to the validity of my findings and diverts attention away from my challenge to the PM2.5 death findings in Pope,[4] HEI (2000),[5] and HEI (2009).[6] Their last paragraph contains the following inaccurate statement: "But the study by Enstrom does not contribute to the larger body of evidence on the health effects of PM2.5 . . . " In conclusion, the authors have not assessed the validity of my peer-reviewed evidence of no relationship between PM2.5 and total mortality in the CPS II cohort and have not been willing to engage with me in addressing the substantive points of my findings.

Response to Criticism by ACS Officials

The ACS Vice President of Epidemiology Susan M. Gapstur and ACS Executive Vice President and Chief Medical Officer

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Table 1. List of the 85 Counties Containing the 50 Cities Used in Pope,[4] HEI (2000),[5] and HEI (2009),[6] As Well As the 35 Additional Counties Used in Enstrom (2017).[a]

State	ACS Division-Unit	FIPS Code	IPN/HEI County Containing IPN/HEI City	IPN/HEI City With PM2.5 Measurements	1979–1983 IPN PM2.5 $\mu g/m^3$ (Weighted Average)	1979–1983 HEIDC PM2.5 $\mu g/m^3$ (Weighted Average)	1979–1983 HEI PM2.5 $\mu g/m^3$ (Median)	1980 Age-Adjusted White Death Rate (DR)	HEI Figure 5 Mortality Risk (MR)
Alabama	01037	01073	Jefferson	Birmingham	25.6016	28.7	24.5	1025.3	0.760
Alabama	01049	01097	Mobile	Mobile	22.0296	22.0	20.9	1067.2	0.950
Arizona	03700	04013	Maricopa	Phoenix	15.7790	18.5	15.2	953.0	0.855
Arkansas	04071 + 2	05119	Pulaski	Little Rock	20.5773	20.6	17.8	1059.4	0.870
California	06001	06001	Alameda	Livermore	14.3882			1016.6	
California	06002	06007	Butte	Chico	15.4525			962.5	
California	06003	06013	Contra Costa	Richmond	13.9197			937.1	
California	06004	06019	Fresno	Fresno	18.3731	10.3	10.3	1001.4	0.680
California	06008	06029	Kern	Bakersfield	30.8628			1119.3	
California	06051 + 4	06037	Los Angeles	Los Angeles	28.2239	26.8	21.8	1035.1	0.760
California	06019	06065	Riverside	Rubidoux	42.0117			1013.9	
California	06020	06073	San Diego	San Diego	18.9189	18.9		943.7	
California	06021	06075	San Francisco	San Francisco	16.3522	16.4	12.2	1123.1	0.890
California	06025	06083	Santa Barbara	Lompoc	10.6277			892.8	
California	06026	06085	Santa Clara	San Jose	17.7884	17.8	12.4	921.9	0.885
Colorado	07004	08031	Denver	Denver	10.7675	10.8	16.1	967.3	0.925
Colorado	07047	08069	Larimer	Fort Collins	11.1226			810.5	
Colorado	07008	08101	Pueblo	Pueblo	10.9155	19.9		1024.1	
Connecticut	08001	09003	Hartford	Hartford	18.3949	18.4	14.8	952.0	0.845
Connecticut	08004	09005	Litchfield	Litchfield	11.6502			941.5	
Delaware	09002	10001	Kent	Dover	19.5280			959.4	
Delaware	09004 + 2	10003	New Castle	Wilmington	20.3743	20.4		1053.7	
District of Columbia	10001 + 2	11001	District of Columbia	Washington	25.9289	25.9	22.5	993.2	0.850
Florida	11044	12057	Hillsborough	Tampa	13.7337	13.7	11.4	1021.8	0.845
Georgia	12027 + 4	13051	Chatham	Savannah	17.8127	17.8		1029.6	
Georgia	12062	13121	Fulton	Atlanta	22.5688	22.6	20.3	1063.5	0.840
Idaho	13001	16001	Ada	Boise	18.0052	18.0	12.1	892.6	0.600
Illinois	14089 + 4	17031	Cook	Chicago	25.1019	23.0	21.0	1076.3	0.945
Illinois	14098	17197	Will	Braidwood	17.1851			1054.0	
Indiana	15045	18089	Lake	Gary	27.4759	27.5	25.2	1129.8	0.995
Indiana	15049	18097	Marion	Indianapolis	23.0925	23.1	21.1	1041.2	0.970
Kansas	17287	20173	Sedgwick	Wichita	15.0222	15.0	13.6	953.4	0.890
Kansas	17289	20177	Shawnee	Topeka	11.7518	11.8	10.3	933.7	0.830
Kentucky	18010	21019	Boyd	Ashland	37.7700			1184.6	
Kentucky	18055	21111	Jefferson	Louisville	24.2134			1095.7	
Maryland	21106 + 1	24510	Baltimore City	Baltimore	21.6922	21.7		1237.8	
Maryland	21101	24031	Montgomery	Rockville	20.2009			881.9	
Massachusetts	22105 + 1	25013	Hampden	Springfield	17.5682	17.6		1025.3	
Massachusetts	22136	25027	Worcester	Worcester	16.2641	16.3		1014.6	
Minnesota	25001 + 2	27053	Hennepin	Minneapolis	15.5172	15.5	13.7	905.3	0.815
Minnesota	25150 + 5	27123	Ramsey	St Paul	15.5823			935.7	
Mississippi	26086	28049	Hinds	Jackson	18.1339	18.1	15.7	1087.4	0.930
Missouri	27001 + 3	29095	Jackson	Kansas City	17.8488	17.8		1090.3	
Montana	28009	30063	Missoula	Missoula	17.6212			938.0	
Montana	28011	30093	Silver Bow	Butte	16.0405			1299.5	
Nebraska	30028	31055	Douglas	Omaha	15.2760	15.3	13.1	991.0	0.880
Nevada	31101	32031	Washoe	Reno	13.1184	13.1	11.8	1049.5	0.670
New Jersey	33004	34007	Camden	Camden	20.9523			1146.9	
New Jersey	33007	34013	Essex	Livingston	16.4775			1072.7	
New Jersey	33009	34017	Hudson	Jersey City	19.9121	19.9	17.3	1172.6	0.810
New Mexico	34201	35001	Bernalillo	Albuquerque	12.8865	12.9	9.0	1014.7	0.710
New York	36014	36029	Erie	Buffalo	25.1623	26.5	23.5	1085.6	0.960
New York	35001	36061	New York	New York City	23.9064	23.9		1090.4	
North Carolina	37033	37063	Durham	Durham	19.4092		16.8[b]	1039.2	1.000
North Carolina	37064	37119	Mecklenburg	Charlotte	24.1214	24.1	22.6	932.8	0.835
Ohio	39009	39017	Butler	Middletown	25.1789			1108.3	

(continued)

Table 1. (continued)

State	ACS Division-Unit	FIPS Code	IPN/HEI County Containing IPN/HEI City	IPN/HEI City With PM2.5 Measurements	1979–1983 IPN PM2.5 $\mu g/m^3$ (Weighted Average)	1979–1983 HEIDC PM2.5 $\mu g/m^3$ (Weighted Average)	1979–1983 HEI PM2.5 $\mu g/m^3$ (Median)	1980 Age-Adjusted White Death Rate (DR)	HEI Figure 5 Mortality Risk (MR)
Ohio	39018	39035	Cuyahoga	Cleveland	28.4120	27.9	24.6	1089.1	0.980
Ohio	39031	39061	Hamilton	Cincinnati	24.9979	25.0	23.1	1095.2	0.980
Ohio	39041	39081	Jefferson	Steubenville	29.6739	29.7	23.1	1058.6	1.145
Ohio	39050	39099	Mahoning	Youngstown	22.9404	22.9	20.2	1058.4	1.060
Ohio	39057	39113	Montgomery	Dayton	20.8120	20.8	18.8	1039.5	0.980
Ohio	39077	39153	Summit	Akron	25.9864	26.0	24.6	1064.0	1.060
Oklahoma	40055	40109	Oklahoma	Oklahoma City	14.9767	15.0	15.9	1050.4	0.985
Oregon	41019 + 1	41039	Lane	Eugene	17.1653	17.2		885.5	
Oregon	41026	41051	Multnomah	Portland	16.3537	19.8	14.7	1060.8	0.830
Pennsylvania	42101 + 1	42003	Allegheny	Pittsburgh	29.1043	30.0	17.9[b]	1115.6	1.005
Pennsylvania	42443	42095	Northampton	Bethlehem	19.5265			998.6	
Pennsylvania	43002 + 11	42101	Philadelphia	Philadelphia	24.0704	24.1	21.4	1211.0	0.910
Rhode Island	45001 + 6	44007	Providence	Providence	14.2341	14.2	12.9	1006.1	0.890
South Carolina	46016 + 1	45019	Charleston	Charleston	16.1635			1023.5	
Tennessee	51019 + 5	47037	Davidson	Nashville	21.8944	22.6	20.5	981.9	0.845
Tennessee	51088	47065	Hamilton	Chattanooga	18.2433	20.4	16.6	1087.9	0.840
Texas	52811 + 2	48113	Dallas	Dallas	18.7594	18.8	16.5	1024.9	0.850
Texas	52859 + 3	48141	El Paso	El Paso	16.9021	16.9	15.7	903.5	0.910
Texas	52882 + 2	48201	Harris	Houston	18.0421	18.0	13.4	1025.7	0.700
Utah	53024	49035	Salt Lake	Salt Lake City	16.6590	17.5	15.4	954.3	1.025
Virginia	55024	51059	Fairfax	Fairfax	19.5425			925.7	
Virginia	55002	51710	Norfolk City	Norfolk	19.5500	19.5	16.9	1139.3	0.910
Washington	56017	53033	King	Seattle	14.9121	14.9	11.9	943.6	0.780
Washington	56032	53063	Spokane	Spokane	13.5200	13.5	9.4	959.2	0.810
West Virginia	58130	54029	Hancock	Weirton	25.9181			1094.8	
West Virginia	58207	54039	Kanawha	Charleston	21.9511	21.7	20.1	1149.5	1.005
West Virginia	58117	54069	Ohio	Wheeling	23.9840		33.4[b]	1117.5	1.020
Wisconsin	59005	55009	Brown	Green Bay	20.5462			931.0	
Wisconsin	59052	55105	Rock	Beloit	19.8584			1019.4	

Abbreviations: ACS, American Cancer Society; HEI, Health Effects Institute; IPN, Inhalable Particulate Network; PM, particulate matter.

[a]Each location includes State, primary ACS Division-Unit number and an indication of additional numbers, Federal Information Processing Standards (FIPS) code, IPN/HEI county, IPN/HEI city with PM2.5 measurements, 1979-1983 IPN-weighted average PM2.5 level, 1979-1983 HEIDC [PM2.5 (DC)] weighted average PM2.5 level, 1979-1983 HEI [PM2.5 (OI, MD)] median PM2.5 level, 1980 age-adjusted white county total death rate (annual deaths per 100 000), and HEI (2000) Figure 5 Mortality risk for HEI city (metropolitan area). All 85 counties have IPN PM2.5 data, 58 counties have HEIDC PM2.5 data, and 50 counties have HEI PM2.5 data. However, 3 cities used in HEI, (2000)[5] (Raleigh, North Carolina; Allentown, Pennsylvania; and Huntington, West Virginia) were not part of IPN and origin of the HEI PM2.5 data in HEI (2000)[5] Appendix D for these 3 cities (indicated with superscript letter "b") is unknown. As an approximation, the Raleigh NC PM2.5 value has been assigned to Durham, North Carolina; the Allentown, Pennsylvania, PM2.5 value to Pittsburgh, Pennsylvania, and the Huntington, West Virginia, PM2.5 value to wheeling West Virginia.

Otis W. Brawley have not assessed the validity of my peer-reviewed findings that challenge the validity of 3 seminal CPS II-based publications: Pope,[4] HEI (2000),[5] and HEI (2009)[6]. They can easily check the accuracy of the results in Tables 1 to 3 of Enstrom[3] and they can determine whether I have correctly identified 85 counties using the ACS Division-Unit numbers shown in Appendix Table A1. Instead, they have made statements about my article like, "we cannot confirm the data are from the CPS-II cohort" and "we cannot substantiate the claim that we provided funding for the preparation of the computerized files and documentation for this research."

I want to address the statements that ACS officials Gapstur and Brawley made about my article. In my acknowledgments, I have never stated or implied that the current ACS endorsed or participated in my article or my use of CPS II data, because they did not endorse or participate. However, former ACS staff made it possible for me to obtain access to individual level data on both CPS I and CPS II participants, as I stated in my article. I received ACS external research support during the period 1973 to 1994. None of this ACS external research support was used for this article. However, ACS internal research support paid for all aspects of the 1982 to 1988 CPS II data that I possess: 1982 questionnaire data collection, 1982 to 1988 mortality follow-up, preparation of computer files, and preparation of detailed documentation.

The genuine version of the 1982 to 1988 CPS II data and detailed documentation that I possess did not come from the current ACS. My version was prepared by ACS many years ago, and I obtained it from a source with appropriate access to

Table 2. ACS CPS II Cohort Participants in Unit 41 (Jefferson County) of Division 39 (Ohio) Showing the Number of Researchers, Families, Participants, and Confirmed 1982 to 1988 Deaths for Each Group and for Each Researcher in Group 1.

Group Number	Researcher Number(s)	Number of Researchers	Family Codes	Number of Families	Number of Participants	Number of Confirmed 1982-1988 Deaths
1	5		1-15	15	29	2
1	6		1-17	14	20	3
1	7		1-15	15	30	1
1	8		1-10	9	19	3
1	9		1-16	15	26	1
1	10		1-14	14	27	2
1	5-10	6		82	151	12
2	1-8	7		41	78	1
3	1-4	3		25	36	1
4	1-9	8		91	168	7
5	1-9	8		82	105	16
6	4-10	4		36	37	9
Total		36		357	575	46

Abbreviations: ACS, American Cancer Society.; CPS, Cancer Prevention Study.

Table 3. Fully Adjusted Relative Risk (RR) of Death From All Causes (RR and 95% CI) From September 1, 1982, Through August 31, 1988, Associated With Change of 10 $\mu g/m^3$ Increase in PM2.5 for CPS II Participants Residing in 47 to 85 Counties in the Continental United States With 1979-1983 IPN PM2.5, HEIDC PM2.5, and HEI PM2.5 Measurements.[a,b]

PM2.5 Years and Source	Number of Counties	Number of Participants	Number of Deaths	RR	95% CI (Lower-Upper)	Average PM2.5
Fully adjusted RR for the Continental United States						
1979-1983 IPN	85	269 766	15 593	1.023	(0.997-1.049)	21.15
1979-1983 HEIDC	58	216 897	12 505	1.024	(0.987-1.061)	21.09
1979-1983 IPN	50	195 215	11 221	1.025	(0.990-1.061)	21.36
1979-1983 HEI	50	195 215	11 221	1.082	(1.039-1.128)	17.99
1979-1983 HEIDC, N = 47	47	189 676	10 836	1.023	(0.984-1.064)	20.95
1979-1983 IPN, N = 47	47	189 676	10 836	1.021	(0.984-1.058)	21.13
1979-1983 HEI, N = 47	47	189 676	10 836	1.081	(1.036-1.128)	18.01
Fully adjusted RR for the Ohio Valley Continental United States						
1979-1983 IPN	17	53 026	3293	1.096	(0.978-1.228)	25.51
1979-1983 HEIDC	10	43 945	2749	1.048	(0.922-1.191)	25.78
1979-1983 IPN	12	42 174	2652	1.050	(0.918-1.201)	25.75
1979-1983 HEI	12	42 174	2652	1.111	(0.983-1.256)	22.02
Fully adjusted RR for the non-Ohio Valley Continental United States						
1979-1983 IPN	68	216 740	12 300	0.994	(0.967-1.023)	20.09
1979-1983 HEIDC	48	172 952	9756	0.960	(0.919-1.003)	19.90
1979-1983 IPN	38	153 041	8569	0.975	(0.936-1.015)	20.15
1979-1983 HEI	38	153 041	8569	1.025	(0.975-1.078)	16.89

Abbreviations: CPS, Cancer Prevention Study; CI, confidence interval; HEI, Health Effects Institute; IPN, Inhalable Particulate Network; PM, particulate matter.
[a]Analysis includes continental United States, 5 Ohio Valley states, and remainder of the States. Table 1 lists up to 85 cities and counties with PM2.5 measurements
[b]1979-1983 PM2.5 data source: IPN = EPA Inhalable Particulate Network → yields insignificant RRs; HEIDC = HEI (2000)[5] Appendix D "PM2.5 (DC)" → yields insignificant RRs (apparently conducted but not reported in HEI 2000[5]); and HEI = HEI (2000)[5] Appendix D "PM2.5 (OI, MD)" → yields significant RRs, used in HEI (2000)[5].

an authorized copy of this version. I have confirmed the validity of this version by showing that (1) the numbers of participants by ACS Division agree almost exactly with the numbers shown in the Fall 1984 CPS II Newsletter (Volume 2, Number 2) Table "Final Numbers of Researchers and Participants by Division"; (2) Table 1 of Enstrom[3] has age at enrollment, sex, race, and education distributions of CPS II participants that agree almost precisely with the same distributions shown in Pope[4] and HEI (2000)[5]; and (3) the CPS II data file information on the participants that I personally enrolled in CPS II agrees with the data that I submitted to ACS in 1982. The ACS epidemiologists can confirm the version of the CPS II data used in my article by confirming my findings in Tables 1 to 3 and Appendix Table A1.[3]

They claim that "when classified using the Division and Unit numbers, the geographically-defined exposure measure

will be highly inaccurate for some participants." Actually, the Division-Unit number accurately identifies the county of residence for most CPS II participants. For instance, ACS Division 39 represents the state of Ohio, and its Unit 041 represents Jefferson County, which includes the city of Steubenville, where the PM2.5 measurements were made. Based on information I have obtained, at least 90% of the 575 CPS II participants in Unit 041 lived in Jefferson County as of September 1, 1982, and ACS can confirm this. In addition, ACS can confirm the detailed information that I have shown in Table 2, regarding the 575 CPS II participants in ACS Unit 041 of ACS Division 39. Table 2 shows the number of researchers, families, participants, and confirmed 1982 to 1988 deaths for the 6 ACS groups within ACS Unit 041. In addition, Table 2 shows these same numbers for each of the 6 researchers in ACS group 1. Thus, as of now, all of the findings in Enstrom[3] stand unchallenged. The ACS has not produced any evidence that invalidates my CPS II cohort findings.

Additional Evidence of No PM2.5 Deaths in CPS II

Since the above investigators criticized my article and did not assess my null findings, I searched their 3 seminal publications for more evidence that supports my null findings. I found evidence in HEI (2000)[5] that I had not previously recognized. Table 29 and Appendix D in HEI (2000)[5] describe 2 key sets of 1979 to 1983 PM2.5 measurements: (1) PM2.5 (OI MD), which is "median fine particle mass from Original Investigators" for 50 cities and designated by me as HEI PM2.5 and (2) PM2.5 (DC), which is "mean fine particle fraction from dichotomous sampler" values for 58 IPN cities and designated by me as HEIDC PM2.5. The PM2.5 (OI MD) values are the ones used in Pope.[4] I now realize that most of the HEIDC PM2.5 [PM2.5 (DC)] values are the same to 1 decimal point as the IPN PM2.5 values in Enstrom.[3]

Table 1 shows that the IPN PM2.5 and HEIDC PM2.5 are identical for 45 cities and somewhat different for 13 cities in HEI (2000)[5] Appendix D. Three cities with PM2.5 (OI MD) values (Raleigh, North Carolina; Allentown, Pennsylvania; and Huntington, West Virginia) were not part of IPN and it is not clear how the PM2.5 values for these 3 cities were measured. As an approximation, the Raleigh NC PM2.5 value has been assigned to Durham, North Carolina, and the Allentown, Pennsylvania, PM2.5 value has been assigned to Pittsburgh, Pennsylvania, and the Huntington, West Virginia, PM2.5 value has been assigned to Wheeling, West Virginia. Two cities in HEI (2000)[5] Appendix D (Boston, Massachusetts and St Louis, Missouri) were not used because of unclear ACS Division-Unit numbers. Table 1 is an expanded version of Appendix Table A1 in Enstrom.[3] Table 3 shows relative risks (RRs) based on IPN PM2.5, HEIDC PM2.5, and HEI PM2.5 values for 85, 58, 50, and 47 cities/counties. The RRs based on the HEIDC PM2.5 values are essentially identical to the null RRs based on the IPN PM2.5 values. Only the RRs based on HEI PM2.5 values are significantly positive, as shown in Enstrom.[3] I find it surprising that the null RRs based on the HEIDC PM2.5 values were not included in HEI (2000)[5] or HEI (2009).[6]

The HEI (2000)[5] Sensitivity Analysis "Risk Estimates Based on Alternative Air Quality Data" section states on page 170, "The means or medians of various indices of air pollution are summarized in Table 30." The data included in this section reveal that the investigators seemed to be aware of the differences in mortality risk associated with PM2.5 (OI MD) and PM2.5 (DC). Table 31 shows RR (all causes) = 1.18 (1.09-1.26) based on PM2.5 (OI MD) values for 50 cities. This value is reduced to RR (all causes) = 1.12 (1.06-1.19) based on PM2.5 (DC) values for 63 cities. Both of these RRs are based on a maximum change in PM2.5 of 24.5 $\mu g/m^3$. I did not previously recognize the similarity between the PM2.5 (DC) values and the IPN PM2.5 values because the only mention of IPN in HEI (2000)[5] occurs in the footnote at the end of Appendix D of Table D.1. Everywhere else in HEI (2000),[5] the term Inhalable Particulate Monitoring Network is used.

It appears that the investigators themselves found no relationship between PM2.5 and total mortality in CPS II in the 2007 *SERRA* article authored by Jerrett et al.[7] Although they cited 16 of their CPS II analyses in their Table 1, they did not cite Jerrett.[7] Figure 2 from Jerrett[7] shows no relationship between PM2.5 and total (all-cause) deaths during 1982 to 2000 in the CPS II cohort. The following quote accompanies Figure 2 "3.1 Health effects The RRs of mortality across the period of follow-up based on the subset of the 51 cities considered were smaller than in the full air pollution cohort considered in the previously full ACS cohort . . . . For example, all-cause mortality was significantly elevated by 6% in the larger cohort, but generally was not significantly elevated in these sub analyses." In addition, Figure 3 (A and B) from Jerrett[7] shows no relationship between PM2.5 and total (all-cause) deaths during 1982 to 1986, 1987 to 1990, 1991 to 1994, 1995 to 1998, and 1999 to 2000. Furthermore, they found low RRs outside the Ohio Valley, as they state in the Discussion section on page 518, "Overall estimated RRs in the 51 cities used in this study were lower than in previous national studies. The lower RR estimates probably resulted from the exclusion of cities in the Ohio River Valley, which tended to demonstrate larger RRs from air pollution than other geographic regions . . . ." Figures 2 and 3 (A and B) from Jerrett[7] are reprinted here.

On June 12, 2017, HEI President Daniel Greenbaum (Greenbaum) provided me with the July 25, 1997 HEI Reanalysis Project Request for Qualifications (RFQ) (http://www.scientificintegrityinstitute.org/Greenbaum061217.pdf). This RFQ specifies the background and requirements for the HEI Reanalysis Project: "HEI is seeking applications representing teams consisting of 2-4 epidemiologists, statisticians and air pollution exposure experts." According to Greenbaum, responses to the RFQ were received from 13 teams and HEI selected the 31-member Krewski team based at the University of Ottawa in Canada, apparently the only foreign-based team. The RFQ objectives and scope include this sentence: "(2) Conduct sensitivity analyses to test the robustness of the original

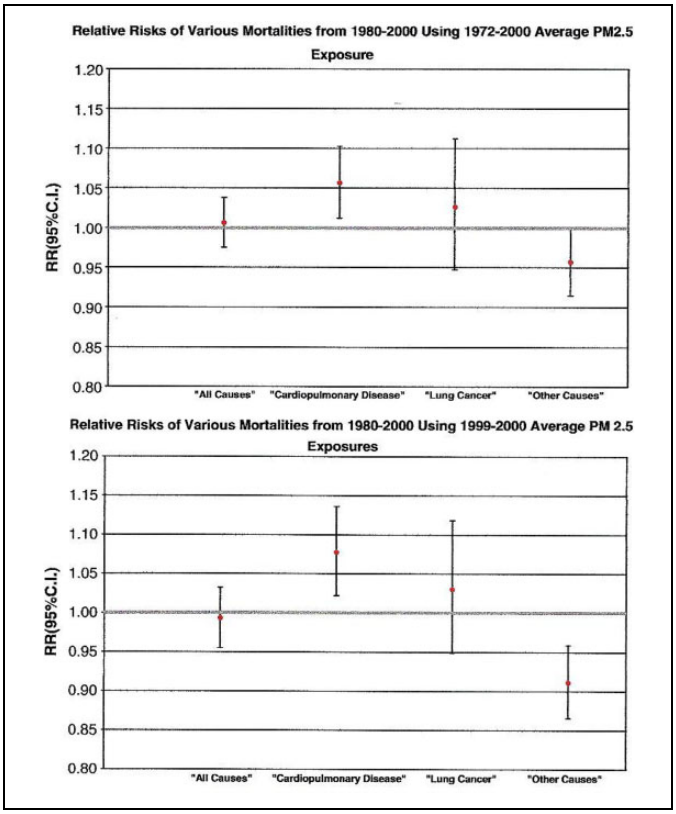

Figure 2 (Jerrett[7]). Summary of risks for different exposures over the entire follow-up.

findings and interpretations to alternative analytic approaches" (http://www.scientificintegrityinstitute.org/HEIRFQ072597.pdf). The Enstrom[3] findings challenge whether the robustness of the Pope[4] findings was properly tested with alternative PM2.5 data, such as IPN PM2.5 data, or alternative cities and counties and metropolitan areas within the CPS II cohort. I first published in 2005 the total mortality RRs for all 11 California counties in the CPS I cohort with IPN PM2.5 data.[8]

Cohen, Pope, and Burnett provided indirect support for my findings in their May 13, 2017, *Lancet* "Global Burden of Disease" article, which went online April 10, 2017.[9] Table 2 from this article shows that, based on their own PM2.5 deaths evidence, the United States had a very low 2015 annual PM2.5-related death rate (18.5 deaths per 100 000 persons) and very low average ambient PM2.5 exposure (8.4 $\mu g/m^3$). This table also shows that PM2.5 pollution is concentrated in other parts of the world, particularly China, India, and Africa, and not in the United States. In addition to the evidence of no PM2.5-related deaths in the CPS II cohort, there is null evidence in 2 other national cohorts: the NIH-AARP cohort[10] and the Veterans cohort.[11]

The null PM2.5 total mortality evidence is further described in my August 12, 2017, Doctors for Disaster Preparedness talk "Scientific Misconduct in PM2.5 Epidemiology" (https://www.youtube.com/watch?v=DaFUhJxMNco), my October 12, 2017, NEJM letter "Air pollution and mortality in the Medicare population,"[12] my November 9, 2017, America First Energy Conference talk "ACS Promotes Air Pollution

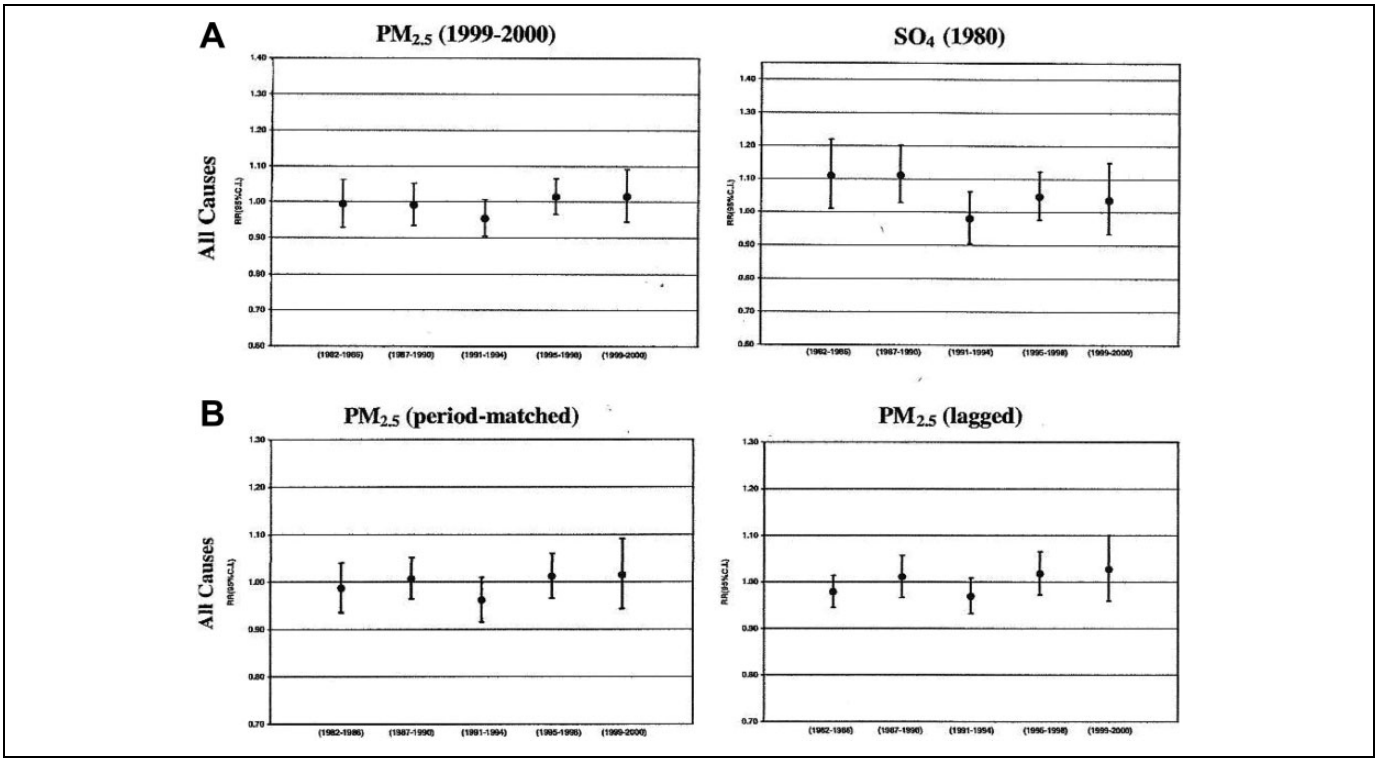

Figure 3 (Jerrett[7]). (A) Relative risks for all-cause, cardiopulmonary and lung cancer deaths estimated for five time periods of the follow-up (1982–1986, 1987–1990, 1991–1994, 1995–1998, and 1999–2000) with measured exposures. (B) Relative risks for all-cause, cardiopulmonary and lung cancer deaths estimated for five time periods of the follow-up (1982–1986, 1987–1990, 1991–1994, 1995–1998, and 1999–2000) with imputed exposures.

Pseudoscience" (http://americafirstenergy.org), and my key 2017 correspondence with the above investigators (http://www.scientificintegrityinstitute.org/DREmails101317.pdf).

Conclusions

My findings of no PM2.5-related deaths during 1982 to 1988 in the CPS II cohort, which are based on my peer-reviewed reanalysis of the CPS II data, stand unchallenged.[3] In addition, my null findings challenge the positive findings in 3 seminal publications by Pope,[4] HEI 2000,[5] and HEI 2009[6] as not robust and not supportive of the claim that PM2.5 causes premature deaths. The responses by Pope[1] and Gapstur[2] have failed to assess the validity or significance of my null findings,[3] but letters supporting the validity of my null findings have been published by Drs S. Stanley Young,[13] Frederick W. Lipfert,[14] and John D. Dunn.[15]

Every effort is being made to encourage ACS, HEI, and the CPS II investigators to cooperate in transparent and verifiable analyses of the CPS II cohort data. However, given the unchallenged null findings in Enstrom,[3] the Environmental Protection Agency (EPA) must reassess all CPS II evidence relating PM2.5 to mortality as part of the current integrated science assessment of the PM2.5 National Ambient Air Quality Standard (NAAQS).

Declaration of Conflicting Interests

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ORAL Statement of Lynn R. Goldman, MD, MS, MPH

May 31, 2018

Submitted to: the US Environmental Protection Agency Science Advisory Board

EPA Planned Action: NPRM "Strengthening Transparency in Regulatory Science"

Mr. Chairman and members of the EPA Science Advisory Board, it is my honor to testify to you in support the SAB workgroup's May 12 memo recommending that the SAB review the agency's April proposed rulemaking, "Strengthening Transparency in Regulatory Science."

I am Dean of the Milken Institute School of Public Health at the George Washington University. In the past, I served as Assistant Administrator for what's now called the Office of Chemical Safety and Pollution Prevention at the US Environmental Protection Agency (EPA).

I will summarize my comments which I have provided in writing.

- This NPRM suffers from lack of involvement of the scientific community.

- There is no clear justification is given for why the rule is needed.

- The proposed rule is a dramatic departure from how the EPA and other US regulatory agencies, and similar agencies internationally, develop dose response assessments in the context of regulatory decisions.

- The rule would have a number of adverse consequences:

    - EPA would have to ignore high quality research or attempt to compel submission of raw data for dose response assessment, which has never been deemed to be required by any expert body;
    - EPA would risk of disclosure of personal information of people volunteering for human subjects' research. With the Internet and "big data", this is increasingly a challenge;
    - EPA and researchers would require resources for preparation, curation and secure storage of such data;
    - EPA actions for some number of the more than 1,000 risk assessments performed annually would be delayed;

    - In cases where obtaining raw data is not feasible, best available science would be unavailable to the EPA for systematic review.

- By restricting access to data and causing delays in EPA processes this proposal threatens EPA's ability to protect public health and the environment.

- The NPRM includes a provision for the EPA to waive this requirement. No clear decision criteria are provided to allow EPA scientists and stakeholders to understand when and how such waivers might be granted. It thus appears that this requirement could be applied in an arbitrary and capricious manner that does not reflect science judgment.

- The NPRM would overturn years of regulatory science policy development and create an unfortunate precedent for EPA in the creation of science policy by rulemaking rather than guidance, thus freezing EPA's risk assessment processes in the future.

Conclusion

In conclusion, the proposed rule would make major changes and cause significant delays in how EPA uses science to make hundreds of regulatory decisions every year. It would overturn years of internal guidance and precedent, and advice from scientific experts outside of EPA. It would be burdensome, for the agency and researchers alike. I strongly urge the SAB to recommend the Administrator:

(1) Do not use the agency's regulatory authority to prescribe specific risk assessment processes. Period.
(2) Do not adopt any major changes to EPA's rules or policies related to the use of science in rule-making until EPA has received clear scientific advice from the SAB and other authorities.

Statement of Lynn R. Goldman, MD, MS, MPH

May 31, 2018

Submitted to: the US Environmental Protection Agency Science Advisory Board

Comments on EPA Planned Action: NPRM "Strengthening Transparency in Regulatory Science"

Mr. Chairman and members of the EPA Science Advisory Board, it is my honor to testify to you about the EPA regulatory agenda and specifically the Notice of Proposed Rule Making (NPRM" called "Strengthening Transparency in Regulatory Science". I am a pediatrician and an epidemiologist and have been Dean of the Milken Institute School of Public Health at the George Washington University since 2010. Prior to that time, I was a professor of environmental and occupational health at the Johns Hopkins Bloomberg School of Public Health. From 1993 through 1998, I served as Assistant Administrator for what now is called the Office of Chemical Safety and Pollution Prevention at the EPA. While serving in that position, I was responsible for the implementation of the nation's pesticide and chemicals laws. Prior to joining the EPA, I worked for eight years in public health with the California Department of Health Services. I am a member of the National Academy of Medicine. My testimony represents my expertise as an environmental health scientist, and a former EPA official, and not the views of any one organization.

I support the SAB workgroup's May 12 memo recommending that the SAB review the agency's April proposed rulemaking, "Strengthening Transparency in Regulatory Science." The workgroup points to a number of complex scientific issues for which the Agency should seek expert advice from the SAB. This NPRM suffers from lack of involvement of the scientific community, either within or outside of the EPA. No clear justification is given for why it is needed. The proposed rule is a dramatic departure from how the EPA and other US regulatory agencies, as well as similar agencies internationally, use regulatory science for the development of dose response assessments. It ignores a number of adverse downstream consequences including: rejecting high-quality academic research unless all raw data are made publicly available; generating risks of disclosure of personal information of people volunteering for human subjects' research; exacting unknown but probably considerable costs to the research community and to the EPA for preparation and curation of data; and making best available science unavailable to the EPA. It creates an unfortunate precedent for EPA in the creation of science policy by rulemaking rather than guidance, thus freezing EPA's risk assessment processes in the future. Finally, by restricting access to data and causing delays in EPA processes this proposal threatens EPA's ability to protect public health and the environment.

Lack of Justification for the Proposed Rule:

First, why does EPA think that this proposed rule is necessary? No justification is given in the preamble. There are no examples of dose response curves that have been proven "wrong"

because of lack of reanalysis of raw data. There is no evidence given demonstrating that stakeholders are requesting increased transparency of these data. In 2013, Ellen Silbergeld and I published a paper in *Environmental Health Perspectives* documenting the use of the Information Quality Act (IQA) of 2001 for requests for raw data.[1] Between 2002–2012 only two IQA requests to the U.S. EPA were for raw data. Both of these were fulfilled under FOIA, not the IQA. This is if anything evidence of little demand for more transparency in terms of access to raw data. If, during that ten year period, EPA had accumulated datasets for all raw data for all dose response assessments that had been conducted, it would have been a tremendous waste in terms of 1) *delays* in EPA conducting assessments until data were obtained; 2) *costs* to the academic community in preparing datasets and extensive meta data files for EPA for all of their studies; 3) *expenditure* of agency staff resources in EPA compelling the submission of the data from academics; and 4) *EPA staffing and funds* for establishing and maintaining systems to house, protect and make available the raw data.

The proposal ignores the many mechanisms that the scientific community have developed to review and assess a body of evidence about an individual substance or chemical. Such methods, known as "systematic review" of evidence, have been developed, refined and improved over a number of years, especially in the US in the context of EPA programs like IRIS, pesticides, toxics, and priority air pollutants. The application of such methods has been reviewed by the National Academy of Sciences and they have offered recommendations for their improvement over time.[2] Likewise, the National Toxicology Program has been engaged in developing and refining these methodologies[3]. Of note is that none of these processes, nor any recommendations from the National Academies, has ever required the availability of "raw data" in order to perform dose response assessments. Nor have they ever concluded that scientific findings should be disregarded if "raw data" for dose response assessments were not available.

Costly to EPA and the Research Community

During the years I worked at EPA I learned that risk assessment activities at EPA are extensive; while many are performed in the flagship EPA IRIS program, far more are produced for chemical and pesticide as well as other regulatory decisions. The NPRM does not provide any estimates, but in 1996 we estimated that the agency performed more than 1,000 risk assessments per year. Such assessments have been required under a number of EPA's statutes and range from premarket notification for chemicals, to periodic reviews of priority air standards, issuance of

[1] Goldman, L.R. and Silbergeld, E.K. Assuring access to data for chemical evaluations. Environ Health Perspect, 121(2):149-52, 2013.

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pesticide tolerances, development of drinking water MCLs and assessment of risks of existing chemicals. We did not estimate how many of these included dose response assessments.

In terms of downstream consequences, the 2013 EHP paper considered these. Any consideration of a requirement on EPA scientists to gather raw data must consider, across dozens if not hundreds of assessments performed annually, the costs to the U.S. EPA and researchers, the significant time and paperwork burdens for researchers, and major regulatory delays that will occur when EPA is waiting for data to be submitted. How would the EPA compel the submission of such data? The U.S. EPA regulatory authority in this area is weak, especially for research conducted in the past, studies not funded by the U.S. government, and/or research conducted abroad. In some cases, it simply would not be feasible to obtain the raw data, either because it is not forthcoming from industry or international sources or because the data no longer exist or are stored on media that is no longer accessible. The U.S. EPA is also constrained by industry confidential business information (CBI) claims for regulatory testing data under U.S. chemical and pesticide laws. For whatever data it could obtain, EPA would have to establish a data repository for this information that would securely house not only the data (especially personal health information and/or CBI) but also a number of unique meta data elements required to understand the data.

Risk of Disclosure of Personal Information for Human Subjects

For human studies, to manage potential risks of disclosure of sensitive human data, the EPA would not be able to rely solely on data submitters to have deidentified the data but, to avoid liability, would have to perform checks to assure that EPA would not inadvertently disclose any personal health information. The NPRM considers none of these challenges. What constitutes a personal identifier? At the beginning of my career this was fairly straightforward, with variable combinations or variables, such as, name+date of birth, name+address, social security number and/or medical record number being the only means of identifying individual persons. With more recent expansion of availability of massive quantities of "big data" on the web, this is now a rapidly moving target. Most recently, the renowned geneticist Craig Ventner and colleagues reported the ability to identify persons using their genetic code alone (without needing to do a DNA match).[4]

[4] "Here, we show that phenotypic prediction from WGS data can enable reidentification without any further information being shared. If conducted for unethical purposes, this approach could compromise the privacy of individuals who contributed their genomes into a database. In stratified analyses, we see that risk of reidentification correlates with variability of the cohort. Although sharing of genomic data is invaluable for research, our results suggest that genomes cannot be considered fully deidentifiable and should be shared by using appropriate levels of security and due diligence." From: Christoph Lippert, Riccardo Sabatini, M. Cyrus Maher, Eun Yong Kang, Seunghak Lee, et al. Genomics of physical traits, PNAS Sep 2017, 201711125; DOI: 10.1073/pnas.1711125114

Sound Science Will be Excluded from EPA Regulatory Decisions

The predictable result of this proposal is that EPA will be forced to exclude studies that should be included in a systematic review, based solely on failure to meet the proposed disclosure requirement. For years, both Congress and successive administrations have required the EPA to use the best science for its decisions. It is a major departure for this NPRM to direct EPA scientists to exclude key studies merely because they cannot meet the proposed disclosure requirement. This is not consistent with good scientific practice and is contrary to years of effort to improve the research base underpinning EPA's decisions as well as EPA's mission to protect the public's health.

Paradoxically, the NPRM includes a provision for the EPA to waive this requirement. No clear decision criteria are provided to allow EPA scientists and stakeholders to understand how, and under what set of decision criteria, such waivers could be predicted to be granted. As proposed, this appears to be a process that would allow arbitrary and capricious application of the "raw data" requirement and not as a process invoking science judgment.

Reversal of EPA Science Policy and Precedents

Finally, the proposal seems to attempt, via a single rule making, to overturn years of EPA science policy guidelines and precedents around the selection and application of dose-response models for toxicity assessment. In so doing it misrepresents the recommendations of prior expert reviews such as the NAS "Silver Book"[5] and the Bipartisan Commission review.[6] (I peer reviewed the first and was a member of the committee that produced the second report.) For example, the NPRM is oblivious to NAS conclusions that thresholds of chemical exposure for chemical effects are the exception rather than the rule when accounting for factors including background exposures, co-exposures, and differential biological susceptibilities across the population.

The NPRM also seems to naively assume that single studies are used to inform risk assessors of the possible shape of dose response curves. They are not. As described by the EPA, the first step of the dose-response modeling process is to evaluate all of the scientific information to gain a biological understanding of how each type of toxicity or response (adverse effect) occurs; the understanding of how the toxicity is caused is called the "mode of action". Via this evaluation (and not via modelling of raw data from a single study), EPA identifies a sequence of key events and processes, that result in the effect. Frequently the data do not conclusively prove mode of action. In those cases, EPA often applies default assumptions such as low dose linearity for carcinogens unless the carcinogens can be shown to have a mode of action for which a threshold would be expected. Such defaults have been developed to assure that, in the face of uncertainty, the EPA will protect the public's health. In recent years, it has been

[5] National Research Council. 2009. Science and Decisions: Advancing Risk Assessment. Washington, DC: The National Academies Press. https://doi.org/10.17226/12209.

[6] Bipartisan Commission. Improving the Use of Science in Regulatory Policy, Washington, DC. 2009

determined that often noncancer effects (e.g. lead and neurotoxicity) also have no threshold. Thus, dose response assessments require the review and analysis of many studies and endpoints.

This specific NPRM raises a general concern about opening the door to EPA enshrining its scientific practices in regulations. Issuing regulations on risk assessment methodology is a slippery slope that not only potentially subjects the process to at best, control by risk managers and attorneys, and at worst, politicization. Such rulemaking about risk assessment would freeze the science in procedures that may or may not make sense today, but will certainly not be scientifically defensible in the future. It would invite more such rulemaking and even legislating risk assessment methodologies and requirements in the years to come.

Conclusion

In conclusion, the proposed rule would make major changes and cause significant delays in how EPA uses science to make hundreds of regulatory decisions every year. It would overturn years of not only internal guidance and precedent, but also advice from scientific experts outside of EPA. It would be burdensome, for the agency and researchers alike. It would be contrary to EPA's mission to protect public health. I strongly urge the SAB to recommend the Administrator:

(1) Do not use the agency's regulatory authority to prescribe specific risk assessment processes;
(2) Do not adopt of any major changes to EPA's foundational policies on the use of science in rule-making without thorough advice and consultation with the SAB, and other authoritative scientific bodies.

Weaponizing Scientific Transparency

Comments on U.S. Environmental Protection Agency Proposed Rule

"Strengthening Transparency in Regulatory Science" (EPA-HQ-OA-2018-0259)

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My name is David Michaels. I am an epidemiologist and Professor of Environmental and Occupational Health at the Milken Institute School of Public Health of George Washington University. The views expressed in my testimony are my own and do not represent the views of George Washington University.

From 2009 until January 2017, I served as Assistant Secretary of Labor for Occupational Safety and Health, the longest serving Assistant Secretary in OSHA's history. From 1998 to 2001, I was Assistant Secretary for Environment, Safety and Health in the U.S. Department of Energy, charged with protecting the workers, community residents and environment in and around the nation's nuclear weapons complex.

I appreciate the opportunity to provide comments on the U.S. Environmental Protection Agency (EPA) Proposed Rule "Strengthening Transparency in Regulatory Science" (EPA-HQ-OA-2018-0259). As a scientist who has been deeply involved in promulgating regulations that protect the public's safety, health and environment, I recognize the importance of open science and providing access to best available science.

However, the proposed rule does not accomplish these goals. Instead it would make it more difficult for EPA to use the findings of scientific investigations to protect public health. I have no doubt it would result in more people made sick by pollution that would have been prevented in the absence of this new regulation. The cynical approach proposed by EPA can be best described as "**weaponized transparency**."

Decades ago, when studies started to show that smoking killed not only smokers but also the non-smoking spouses of smokers, the Tobacco industry recognized the government would use this evidence to reduce smoking. In response, the tobacco industry demanded access to the raw data of these studies.[1] Getting to the truth was never tobacco's objective. The cigarette manufacturers hired mercenary scientists to massage the numbers and manufacture doubt about the results.[2]

Big Tobacco turned transparency, an important scientific principle, into a weapon.

The strategy worked for tobacco for years, helping to delay regulation and increase the death toll from smoking-related illness. Since then, polluters and manufacturers of deadly products have followed Big Tobacco's playbook, first supporting legislation, and when that was unsuccessful, this proposed rule.

If promulgated, this regulation would permit the EPA Administrator to deny the agency the use of findings of any study unless the raw data, computer codes and virtually everything used by scientists to conduct the study are provided to the agency and posted on the agency's website "in a manner sufficient for independent validation." There are no constraints on the Administrator and she or he is not required to provide any rationale for rejecting a study because the underlying information is not publicly available.

The underlying justification for this "transparency" proposal is a caricature of how science really works. It is not "sound science." It is something that sounds like science, but is not. While in theory most studies could be reproduced, they rarely are because it is a waste of resources. The scientific enterprise involves approaching the same question in different ways to determine if the results support each other. Reanalyzing the same study over and over is little different from

checking on a surprising newspaper article by buying additional copies of the same paper to see if it says the same thing.

Polluting corporations, attempting to defend their products and emissions, will no doubt be able to provide whatever data and materials supporting their positions in any format the EPA might demand. But the requirements this could impose on independent researchers an onerous and burdensome requirement that much environmental science, particularly epidemiologic studies, would be excluded from the evidence base and become irrelevant to efforts by EPA to protect the public and the environment.

Depending on how it is implemented and interpreted by the Administrator, under this regulation, human studies could only be used by EPA if the investigators surrender confidential data (including personally identifiable information, trade secrets and commercial and financial information) that the investigators had promised the study subjects and institutional review boards would never be released. While the EPA might agree to redact these data before public posting, they could be given to anyone who signed a confidentiality agreement designed by the agency. Implications for the protections of human subjects and informed consent under the Common Rule (the Federal Policy for the Protection of Human Subjects)[3] have yet to be evaluated.

Investigators have other reasons to be concerned about sharing data with no preconditions. Too often, we have seen mercenary scientists taking raw data of others study, conduct *post hoc* analyses, and create uncertainty about the findings. That is what the tobacco industry tried to do. It is possible to conduct re-analyses with independence and integrity, as the Health Effects Institute jointly funded by EPA and industry, has done. But to ensure their validity and honesty, these should be performed under ground rules agreed upon in advance.[4]

Under the provisions of the Administrative Procedures Act, the EPA administrator does not have the authority to refuse to consider any comment submitted to the agency. If she or he thinks it is not valid, inaccurate or inapplicable, she or he must explain why. Under the APA, submissions, including scientific studies, cannot arbitrarily or capriciously be discarded because the underlying data are not provided. When I was OSHA Administrator, we wanted to protect the integrity of the science used in setting regulations, so we explored asking for conflict of interest disclosures, similar to those requested by every leading scientific journal. Our legal experts determined that we could request this disclosure, but we could not reject submissions that failed to include them. This is a comparable situation – rejecting submitted studies because the underlying data are not available is prohibited under the APA.

Furthermore, many of laws authorizing the activities of the EPA require the agency to use the best science in protecting the public's health and environment. For example, the Clean Air Act mandates that air quality criteria "accurately reflect" the "latest scientific knowledge." In the past, the EPA has considered all available studies in issuing these criteria without consideration of the availability of the underlying data. Promulgation of this proposed rule would be an arbitrary and capricious violation of the provisions of these laws.

These new strictures will be particularly burdensome to the environmental health research community, ironically imposing substantial new costs during a time of drastic proposed research funding cuts. Epidemiologic research will be particularly challenged. Epidemiology is the foundation of public health, providing essential evidence of the distribution of disease, risk

factors, and vulnerable populations. It has been the foundation of public health policy from infectious disease control to chronic disease prevention. The epidemiologic evidence base has provided the basis for our environmental policies, from safe drinking water to clean air to superfund site cleanup. While it remains to be determined how this historical evidence would be considered under this proposed rule, the implications are ominous for public health measures that have improved the health of millions of Americans.

Many in the scientific and regulatory communities recognize the importance of transparency and access to the data underlying important studies. Addressing these issues can be accomplished without injuring the scientific enterprise or discouraging scientists from pursuing pressing scientific questions. Mechanisms to accomplish this that do not require new regulation include development of public access databases, use of the Freedom of Information Act (FOIA), and reanalysis by third parties. Certain journals, for example require authors to agree to make their data available to editors and others upon request.

The current administration has made it clear that it hopes to use this regulation to radically transform the fundamental way the EPA uses science. When a law that is very similar to this NPRM was first considered by Congress some years ago, the EPA told the Congressional Budget Office (CBO) that it estimated the cost of gathering, redacting and posting the data on a public website at $250 million annually.[5]

The cost estimate made by the current administration for a substantially similar law dropped to $1 million annually because, in the candid, shocking words of the CBO:

> EPA officials have explained to CBO that the agency would implement the legislation with minimal funding and generally would not disseminate information for the scientific studies that it uses to support covered actions. That approach to implementing the legislation **would significantly reduce the number of studies that the agency relies on** when issuing or proposing covered actions for the first few years following enactment of the legislation.[6] (emphasis added)

There is every reason to believe this is the plan of the current leadership of the EPA. It is a recipe for privileging mercenary studies by industry commissioned to influence regulatory policy, because they will be structured and released in a way acceptable under this regulation. Moreover, years and years of valuable research findings from studies for which this extensive documentation is no longer available to EPA, would be ignored in its decision making to protect health and the environment.

Certainly, calls for transparency and reproducibility sound reasonable, but exposing poorly conducted studies is not what this effort is about. Instead, it is designed to impose onerous and burdensome requirements on independent researchers, and would result in much environmental health science, particularly epidemiologic studies, being excluded from the evidence base and becoming irrelevant to efforts by EPA to protect the public and the environment. Valuable research findings in areas like climate change, lead exposure and particulate pollution would be ignored. Some of the findings on which our public health protections are based are the result of studies done across the globe. It is unlikely that Canadian or European scientists would turn over their raw data to a US agency for "independent validation." And because important studies on

disasters like the Deepwater Horizon or Chernobyl are, fortunately, not reproducible – would they be discarded, too?[7]

In summary, by turning scientific transparency into a virtual weapon, the EPA will inflict severe damage to the nation's scientific enterprise. It will undermine the credibility and application of scientific evidence and impose costs and impediments that will discourage scientists from undertaking studies of great importance. Limiting the EPA's use of scientific evidence in the name of increased transparency will impede the EPA's ability to protect the health, safety and environment of the nation. This proposal must be withdrawn.

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EDITORIAL

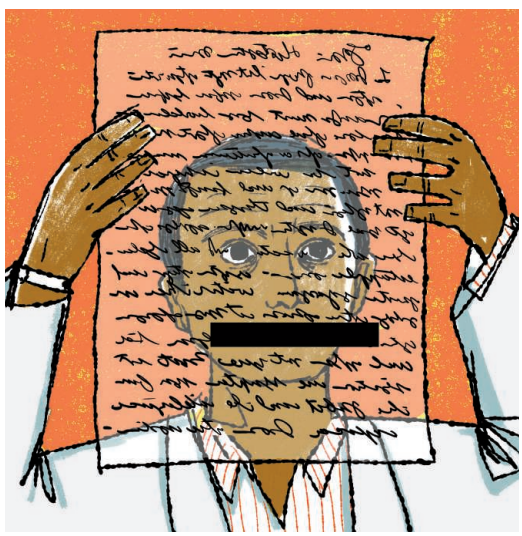
The dishonest HONEST Act

The Trump administration aims to eliminate many regulations and make it more difficult to adopt new ones. More subtle and dangerous are attempts in Congress to undermine public health and environmental protections by limiting the use of scientific evidence under the guise of increased transparency. This effort, which as envisioned by U.S. Environmental Protection Agency (EPA) leadership would greatly reduce the amount of science used in decision-making, undermines the credibility and application of scientific evidence, weakens the scientific enterprise, and imperils public and environmental health.

The Honest and Open New EPA Science Treatment (HONEST) Act, in the Senate after passing the House of Representatives in March, would prohibit the EPA from using studies for agency decision-making unless raw data, computer codes, and virtually everything used by scientists to conduct the study are provided to the agency and made publicly available online. Transparency and reproducibility are long-standing priorities in science, and we welcome good-faith efforts to evaluate scientific evidence for use in public policy. But on these issues, the Act is dishonest—an attempt by politicians to override scientific judgment and dictate narrow standards by which science is deemed valuable for policy. It imposes burdens that will detract from scientists' ability to do research and to have it influence decision-making, all aimed at bringing the process to a standstill, minimizing the role of science, and limiting regulations.

Federal agencies must already adhere to strict standards of transparency and quality while considering a broad body of scientific evidence, and uncertainties therein. Polluters and manufacturers of dangerous products have taken a page from the tobacco industry playbook, magnifying those uncertainties to prolong the review of scientific data, slow the regulatory process, and evade liability. By writing narrow data standards into law, the Act will provide another avenue for such challenges to regulations and to the underlying science.

The Act would not void prior EPA decisions, but future deliberations would be required to exclude peer-reviewed historical studies for which this extensive documentation is no longer available. To enable use of studies that include sensitive information, such as medical records, the Act permits such data to be redacted. But in practice, the limited budget allocated for potentially costly redaction leaves the role of such studies in doubt. For a similar unpassed bill, the 2015 Secret Science Reform Act, the Congressional Budget Office (CBO) estimated implementation costs at $250 million annually. Under President Trump, this dropped to $1 million because, according to the CBO, "EPA officials have explained...that the agency would implement [the Act] with minimal funding...[which] would significantly reduce the number of studies that the agency relies on." Costs of gathering, redacting, and posting data will erode the agency's effectiveness.



"...the Act is dishonest—an attempt by politicians to override scientific judgment..."

The scientific community continues to improve data access. Would the law adapt to allow the EPA to incorporate studies that take innovative approaches not foreseen by the Act? Improved transparency and reproducibility should ultimately expand the scientific foundation for public health and environmental protection. Unfortunately, the Act will erode the evidence base for regulatory decisions and burden investigators and agencies with threats of endless data reanalysis and challenges to defend findings.

If the HONEST Act becomes law, it will embolden attempts to dictate science and delay decisions at other federal, state, and local agencies. The community must make clear that the Act, a threat to health and the environment, is an unnecessary and burdensome political intrusion into the scientific enterprise.

–David Michaels and Thomas Burke*

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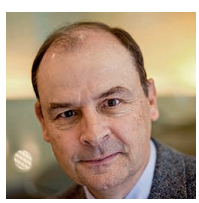

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World Health Organization Map

2015 Annual Mean Ambient $PM_{2.5}$ ($\mu g/m^3$)