Airborne Fine Particulate Matter and Short-Term Mortality:

Exploring the
California Experience,
2007-2010

JunkScience.com
December 2013

Executive Summary

The U.S. Environmental Protection Agency regulates ambient airborne fine particulate matter ($PM_{2.5}$) on the basis that it is causally associated with short-term mortality — i.e., daily increases in $PM_{2.5}$ cause increases in daily deaths. This is the first epidemiologic study to test that hypothesis on a systematic basis, i.e., using all the relevant and available data from a large contiguous geographic area. Based on a comparison of air quality data from the California Air Resources Board and death certificate data for 854,109 deaths from the California Department of Public Health for the years 2007-2010, no correlation was identified between changes in ambient $PM_{2.5}$ and daily deaths, including when the analysis was limited to the deaths among the elderly, heart and/or lung deaths only, and heart and/or lung deaths among the elderly. Although this is only an epidemiologic or statistical study that cannot absolutely exclude the possibility that $PM_{2.5}$ actually affects mortality in some small and as yet unknown way, these results also illustrate that it would be virtually impossible to demonstrate through epidemiologic study that such an effect actually exists. Notwithstanding the limits of the epidemiologic method, if a significant causal relationship between $PM_{2.5}$ and mortality existed, that relationship should have been visible in this study. But it was not.

Introduction

The U.S. Environmental Protection Agency (EPA) has regulated airborne fine particulate matter ($PM_{2.5}$) since 1997 on the basis that inhalation of such particles causes death.[1] In a 2004 scientific assessment, the EPA asserted that any inhalation of $PM_{2.5}$ can cause death on a short-term basis, meaning within hours or days of exposure, particularly among vulnerable populations, e.g., the elderly. This view was reasserted by the EPA in its 2009 scientific assessment,[2] and has been reasserted since in testimony before, and communications with Congress, and public statements.[3]

Despite the EPA's repeated claims that $PM_{2.5}$ is causally associated with increased mortality, the alleged association remains controversial for a variety of reasons.

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[1] 62 *FR* 38652 (July 18, 2007).

[2] EPA, Integrated Science Assessment for Particulate Matter (December 2009), Available at http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546.

[33] *See e.g.*: (1) Commentary of Clean Air Scientific Advisory Council chairman Jonathan Samet, "The Clean Air Act and Health — A Clearer View from 2011," NEJM 365;3, pp.198-201 (July 21, 2011); (2) Testimony of EPA Administrator Lisa P. Jackson before the Oversight and Investigations Subcommittee of the House Energy and Commerce Committee (September 22, 2011); and (3) Letter from Gina McCarthy, EPA Assistant Administrator Office of Air and Radiation to Rep. Fred Upton., Chairman of the House Energy and Commerce Committee (February 3, 2012).

These include the inherent limitations of the epidemiologic method,[4] no demonstrated biological plausibility,[5] absence of systematic study, barriers to independently replicating published epidemiologic studies, and a lack of transparency regarding results. This study represents an effort to tackle the latter three deficiencies.

Systematic Study

Epidemiologic studies of $PM_{2.5}$ typically involve an arbitrarily selected number of cities, ranging from the so-called "Harvard Six-City Study" to a 112-city study.[6] Despite the apparent authority of names and number of cities, there is in reality little justification for why the particular number or particular non-contiguous population centers were selected for study. Study timeframes can also appear to be somewhat arbitrary. Although the 112-study was published in 2009, for example, the timeframe of study was only 1999 to 2005. At least one study arbitrarily discarded two-thirds of its data without thorough discussion and consideration of the available options and implications.[7] Given the inherent integrity and statistical frailties of these studies, the selection of cities, timeframes and data raise questions as to how their results may change if different data were analyzed.

Independent Replication

It is a long-established principle of science that study results be capable of replication. But this has largely not been possible in the context of $PM_{2.5}$. Although air quality monitoring data are available to the public, mortality and other relevant health-related data used in published studies have not been made available to the

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[4] *See e.g.,* EPA has admitted in recent litigation: "[E]pidemiological studies do not generally provide direct evidence of causation; instead they indicate the existence or absence of a statistical relationship. Large population studies cannot assess the biological mechanisms that could explain how inhaling ambient air pollution particles can cause illness or death in susceptible populations." *See* American Tradition Institute Environmental Law Center v. U.S. Environmental Protection Agency, Memorandum in Opposition to Plaintiff's motion for Temporary Restraining Order, Case 1:12-cv-01066-AJT-TCB (October 4, 2012).

[5] *Ibid*.

[6] *See* Dockery D *et al.* An Association between Air Pollution and Mortality in Six U.S. Cities. *NEJM* 1993; 329:1753-1759 (December 9, 1993); and Zanobetti A and Schwartz J. The effect of fine and coarse particulate air pollution on mortality: a national analysis. Environ Health Perspect. 2009 Jun;117(6):898-903.

[7] *See* Franklin M, Zeka A, and Schwartz J. Association between $PM_{2.5}$ and all-cause and specific-cause mortality in 27 U.S. communities. *Journal of Exposure Science and Environmental Epidemiology* (2007) 17, 279-287.

public or to independent researchers. This is despite repeated efforts by Congress.[8] Without the mortality data, it has been impossible to replicate and confirm claimed results.

Study Transparency

While the hypothesis behind the claimed link between $PM_{2.5}$ and mortality is easy to understand — i.e., increases in $PM_{2.5}$ levels increase the number of daily deaths — few, if any, studies go to the effort of presenting their data and results in a manner that facilitates reader understanding. Merely describing the inputs to, and outputs of a "black box" statistical analysis is insufficient for facilitating reader comprehension. This is especially important in the case of non-experts who need to understand the data and results for the purposes of formulating and evaluating public policy.

This study, then, is an effort to independently, systematically and transparently test the hypothesis that inhalation of $PM_{2.5}$ is statistically associated with death.

Data and Methods

The state of California provides a unique opportunity to examine the purported $PM_{2.5}$-mortality relationship because it has made necessary data readily available.

Mortality Data

The California Department of Public Health makes available to researchers statewide death certificate data as far back as 1999. These so-called "public use death files" are electronic files of data from individual death certificates.[9] Relevant data available from each death certificate include, among other data, zip code of residence at time of death, age at death, date of death, and cause of death by International Classification of Disease (ICD) code.

Air Quality Data

The state of California is divided into 15 regions or "air basins" on the basis of similarity in meteorological and geographical conditions.[10] The California Air Resources Board makes available to the public, through its Air Quality and

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[8] *See e.g.,* Letter from Rep. Andy Harris, Chairman, Subcommittee on Energy and Environment of the Committee on Science, Space and Technology, to Gina McCarthy, Assistant Administrator, Office of Air and Radiation, EPA (September 22, 2011), available at http://junksciencecom.files.wordpress.com/2011/09/harris-to-mccarthy-092211.pdf.

[9] *See* http://www.cdph.ca.gov/data/dataresources/requests/Pages/DeathDataFiles.aspx.

[10] *See* http://www.arb.ca.gov/ei/maps/statemap/abmap.htm.

Meteorological Information System, daily air quality measurements for each air basin for a variety of substances, including $PM_{2.5}$.[11] Two types of summary statistics are presented by CARB to the public, apparently as plausible indicators of air quality: (1) daily average $PM_{2.5}$ at the highest site; and (2) daily maximum one-hours average at the highest site. The former statistics were selected for this study because the daily average $PM_{2.5}$ statistic seemed more representative of daily air quality than the daily 1-hour maximum measurement.

Zip Code-Air Basin Data

Upon request, CARB provided a data file linking zip codes with air basins, thereby facilitating accurate classification of individual death certificate data with the air basins of decedent residence.

Each annual death certificate file was converted in an Excel file and then sorted by zip code. Using the zip code-air basin conversion data, death certificates were assigned to air basins, within which correlations were developed between daily death tolls by cause and daily average $PM_{2.5}$ at the highest site.

To see whether various causes of death increase with $PM_{2.5}$ levels (presumably a linear relationship), Pearson correlation coefficients were developed for each air basin and 0-day, 1-day, 2-day and 3-day lag bases for the following cause-of-death categories:

- All-cause deaths; non-violent and non-accidental deaths (exclude ICD Codes; deaths from heart or lung causes; deaths from heart causes;
- Deaths from lung causes; all-cause deaths, 65 years of age and older;
- Non-violent and non-accidental deaths, 65 years of age and older;
- Deaths from heart or lung causes, 65 years of age and older; and
- Deaths from heart causes, 65 years of age and older;
- Deaths from lung causes, 65 years of age and older.

Fisher's transformation was then used on the correlation coefficients in order to combine air basin correlations, weighted by deaths, into a meta-analysis representing statewide correlations.

Results

For the years 2007-2010, 941,888 total death certificates were available. This analysis includes 854,109 (90.6%) of those records. Some death certificate data were excluded from the analysis because they could not readily be assigned an air

[11] *See* http://www.arb.ca.gov/aqmis2/aqmis2.php.

basin.[12] The majority of the excluded death certificates were due to the exclusion of seven air basins from this analysis because of the unavailability of daily $PM_{2.5}$ monitoring data.[13] Although excluding 7 of 15 air basins sounds like a significant amount of data, the basins only comprise 6 percent of the California population.

Apparently the air quality in the excluded seven air basins is considered by regulators to be of such good quality that daily monitoring of $PM_{2.5}$ is not conducted. While the exclusion of these air basin data may at first seem to weaken the systematic intent and nature of this analysis, the fact that people die in these air basins despite the comparatively low levels of ambient $PM_{2.5}$ would only tend to attenuate correlations between $PM_{2.5}$ and mortality. Table 1 presents the deaths counts by air basin and cause-of-death.

TABLE 1. DEATH TOTALS BY AIR BASIN AND CAUSE-OF-DEATH

	Cause-of-Death									
Air Basin	All-Cause	Non-Violent/ Non-Accident	Heart & Lung	Heart Only	Lung Only	All-Cause, 65+	Non-Violent/ Non-Accident, 65+	Heart & Lung, 65+	Heart Only, 65+	Lung Only, 65+
Mountain Counties	17128	15713	7331	5473	1858	12812	12419	6151	4548	1603
Sacramento Valley	78556	72407	34156	25505	8651	56186	54808	27993	20655	7338
Salton Sea	17068	15734	7250	5763	1487	12558	12224	6068	4759	1309
San Diego County	74291	68877	30707	24147	6560	54542	53122	25687	20240	5447
San Fran. Bay	172232	159940	71890	55360	16530	126322	123141	59862	45557	14305
San Joaquin Valley	94773	84467	42563	32793	9770	63584	62119	33957	26099	7858
South Cen. Coast	36924	34323	16259	12809	3450	27550	26887	13741	10754	2987
South Coast	363137	339666	167043	132480	34563	258318	253644	136588	106510	30079
Total	854109	793127	377199	294330	82869	611872	598364	310047	239122	70926

The results of the correlation analysis are presented in Table 2 (0-Day Lag) and Table 3 (1-Day Lag). While there are absolute rules for interpreting correlations other than the absolutes of -1.0 (perfect inverse correlation), 0 (absolutely no correlation) and 1.0 (perfect correlation), we may turn to the EPA's own Causal Analysis/Diagnosis Decision Information System (CADDIS) for guidance.[14]

In its section on "Exploratory Data Analysis," the EPA presents the graphic below as a guide for interpreting correlations:

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[12] Excluded death certificates had zip codes that were either missing, erroneous or not included in the zip code-air basin filed provided by CARB.
[13] The seven excluded air basins are: Great Basin Valleys, Lake County, Lake Tahoe, Mojave Desert, North Central Coast, North Coast, and Northeast Plateau.
[14] *See* http://www.epa/gov/caddis.

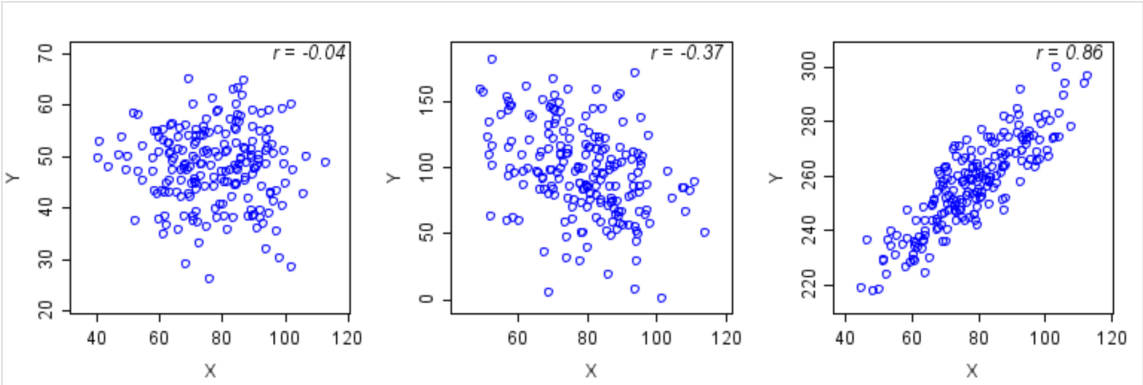


Figure 1. Examples of different correlations between two variables, *X* and *Y*.
Left: *r* = -0.04. The points are diffusely scattered, indicating no association of *X* and *Y*.
Center: *r* = -0.37. The plot indicates a weak negative association in which *Y* decreases as *X* increases.
Right: *r* = 0.86. The scatterplot indicates a linear increase in *Y* with increasing values of *X*.

Ignoring the negative sign of the example correlations, the EPA considers a correlation on the order of 0.04 to indicate "no association" between the compared variables, and a correlation on the order of 0.37 to be only a "weak" association.

Relying on this EPA-endorsed standard, this analysis found no associations between ambient $PM_{2.5}$ levels in California and mortality for the period 2007-2010 for any cause-of-death. Even putting aside the systematic nature of this analysis and focusing on the two air basins with the greatest correlation values — i.e., San Francisco Bay Air Basin and San Joaquin Valley Air Basin — the reported correlations on the order of 0.14-0.17, are far below the level that EPA considers even to be "weak", i.e., 0.37.

TABLE 2. SUMMARY CORRELATIONS FOR $PM_{2.5}$ AND MORTALITY IN CALIFORNIA, 2007-2010, 0-DAY LAG

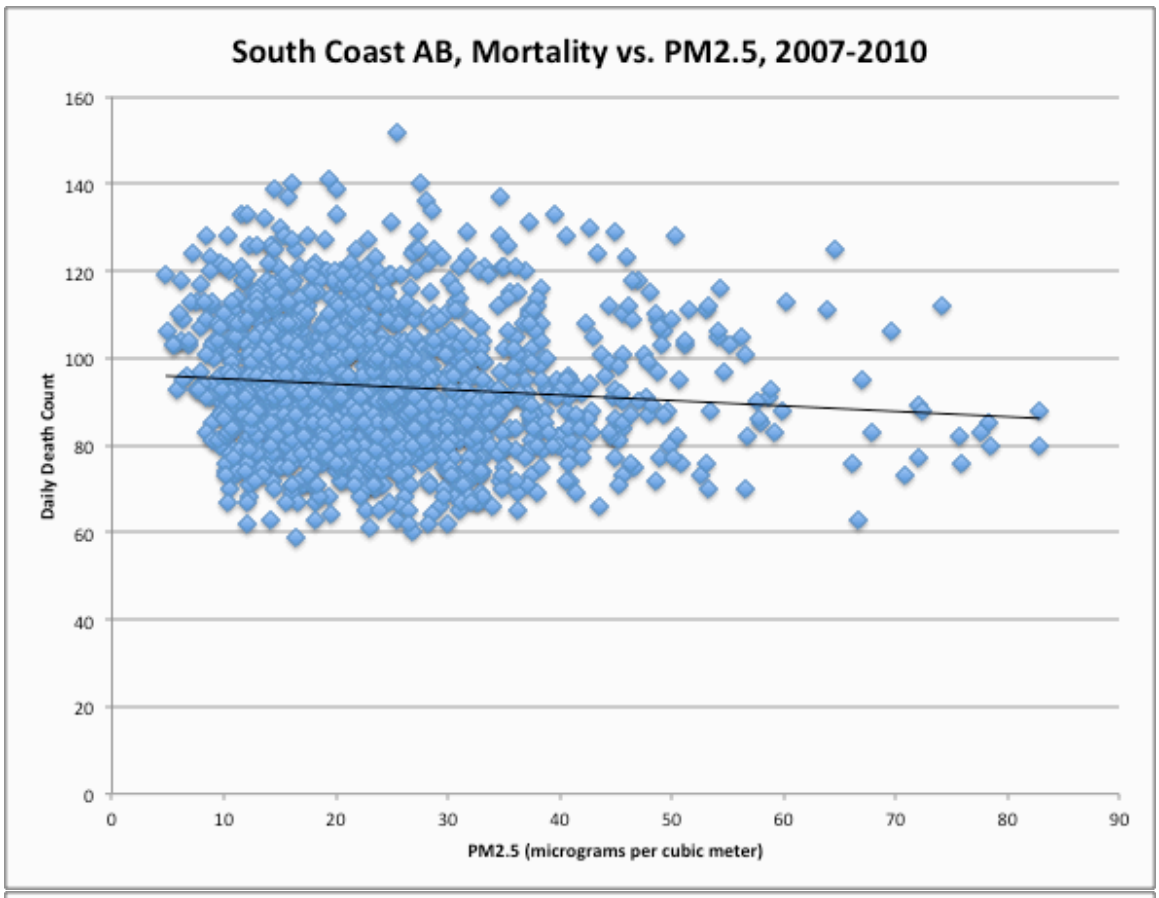
	Cause-of-Death									
Air Basin	All-Cause	Non-Violent/ Non-Accident	Heart & Lung	Heart Only	Lung Only	All-Cause, 65+	Non-Violent/ Non-Accident, 65+	Heart & Lung, 65+	Heart Only, 65+	Lung Only, 65+
Mountain Counties	0.02	0.03	0.03	0.04	0	0.04	0.04	0.04	0.04	0
Sacramento Valley	0.04	0.06	0.07	0.08	0.01	0.03	0.04	0.04	0.06	0
Salton Sea	0	-0.01	0	0.02	-0.04	-0.01	-0.02	0	0.02	-0.05
San Diego County	-0.03	-0.05	-0.03	0	-0.07	-0.05	-0.05	-0.03	0	-0.07
San Fran. Bay	0.17	0.16	0.15	0.15	0.07	0.16	0.16	0.14	0.14	0.06
San Joaquin Valley	0.14	0.16	0.12	0.14	0.01	0.12	0.12	0.09	0.11	0
South Cen. Coast	-0.08	-0.08	-0.07	-0.03	-0.09	-0.08	-0.08	-0.06	-0.03	-0.07
South Coast	-0.05	-0.06	-0.10	-0.09	-0.06	-0.06	-0.06	-0.10	-0.08	-0.03
Cumulative	**0.03**	**0.03**	**0**	**0.01**	**-0.02**	**0.01**	**0.01**	**0**	**0.01**	**-0.03**

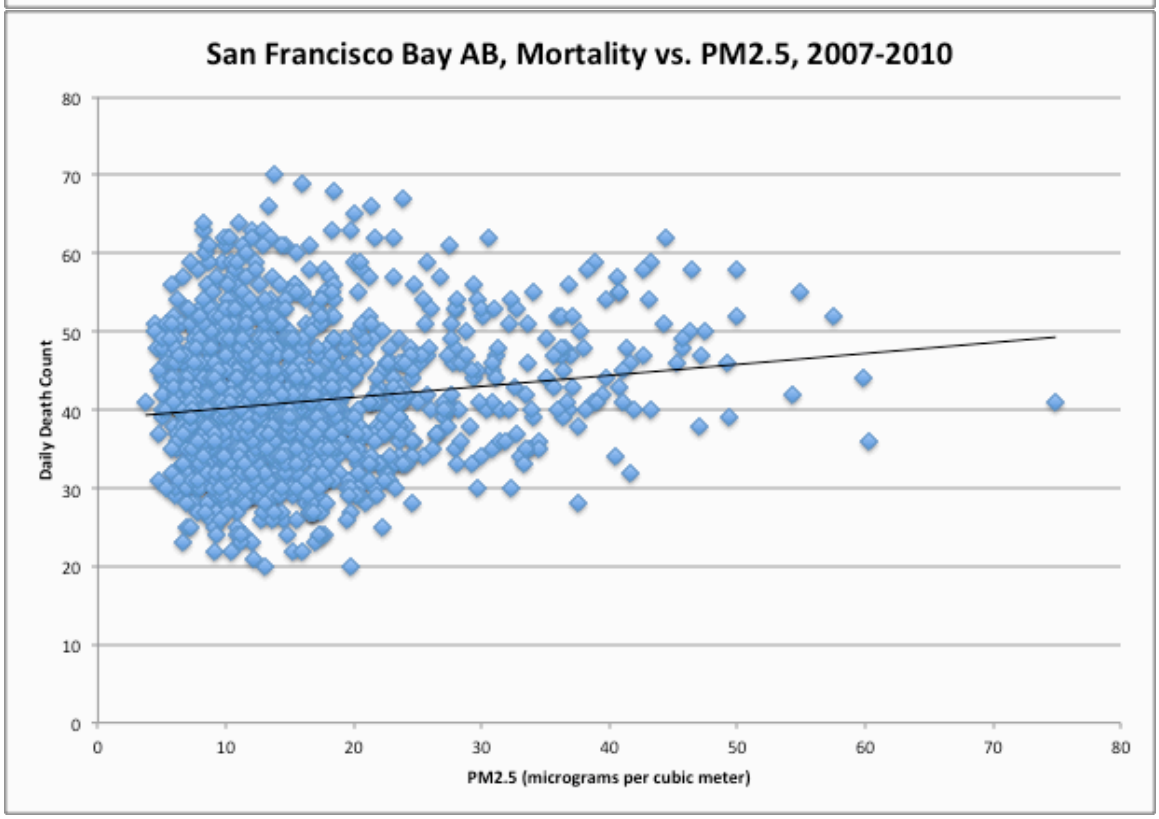
TABLE 3. SUMMARY CORRELATIONS FOR $PM_{2.5}$ AND MORTALITY IN CALIFORNIA, 2007-2010, 1-DAY LAG										
	Cause-of-Death									
Air Basin	All-Cause	Non-Violent/ Non-Accident	Heart & Lung	Heart Only	Lung Only	All-Cause, 65+	Non-Violent/ Non-Accident, 65+	Heart & Lung, 65+	Heart Only, 65+	Lung Only, 65+
Mountain Counties	0.04	0.05	0.04	0.06	-0.03	0.04	0.05	0.05	0.07	-0.02
Sacramento Valley	0.06	0.07	0.08	0.09	0.01	0.05	0.06	0.06	0.07	0.01
Salton Sea	0	-0.01	0	0	-0.02	0.01	0.01	0	0.01	-0.02
San Diego County	-0.04	-0.05	-0.03	-0.01	-0.06	-0.05	-0.04	-0.03	0	-0.01
San Fran. Bay	0.15	0.15	0.13	0.13	0.07	0.14	0.14	0.12	0.13	0.05
San Joaquin Valley	0.14	0.15	0.12	0.13	0.03	0.11	0.11	0.11	0.12	0.02
South Cen. Coast	-0.10	-0.10	-0.10	-0.07	-0.10	-0.11	-0.11	-0.10	-0.07	-0.10
South Coast	-0.08	-0.08	-0.10	-0.10	-0.06	-0.09	-0.09	-0.10	-0.09	-0.07
Cumulative	**0.01**	**0.01**	**0**	**0.01**	**-0.02**	**0**	**0.**	**-0.01**	**0**	**-0.03**

Though EPA has asserted that $PM_{2.5}$ is causally associated with short-term death from heart and lung causes and that the elderly are more vulnerable than the general population to the effects of $PM_{2.5}$, these results provide no evidence to support those claims. Similar results were produced for the 2-Day Lag and 3-Day Lag analyses.

The detailed analyses, as well as the 2007-2010 daily $PM_{2.5}$ measurements and death counts for the South Coast Air Basin, are included in the Appendix.

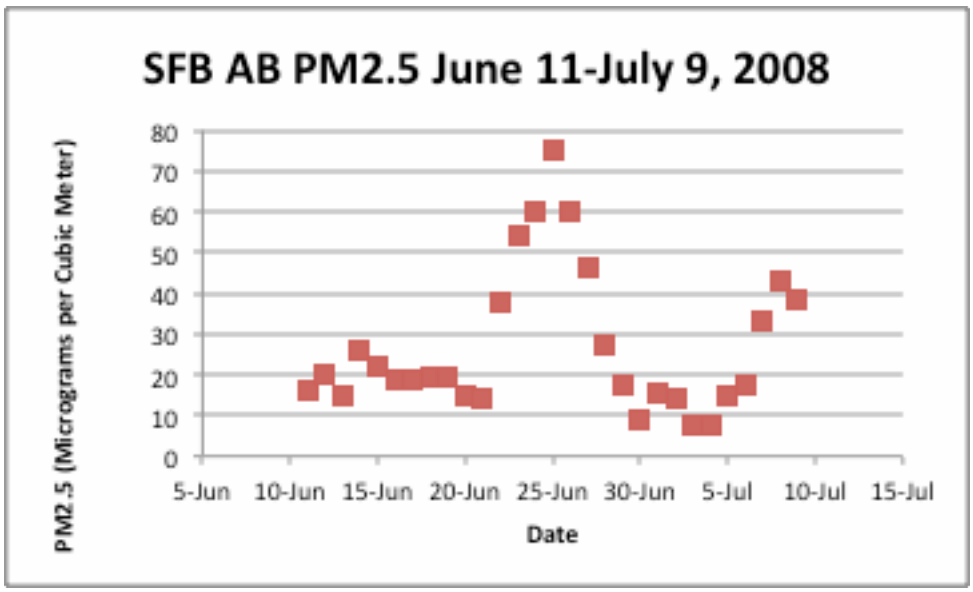
Sample scatter plots of daily death counts vs. $PM_{2.5}$ measurements for the two largest air basins (San Francisco Bay and South Coast) are presented below. Both charts illustrate the lack of meaningful correlation between daily death counts (n=1,461) and ambient $PM_{2.5}$ levels.

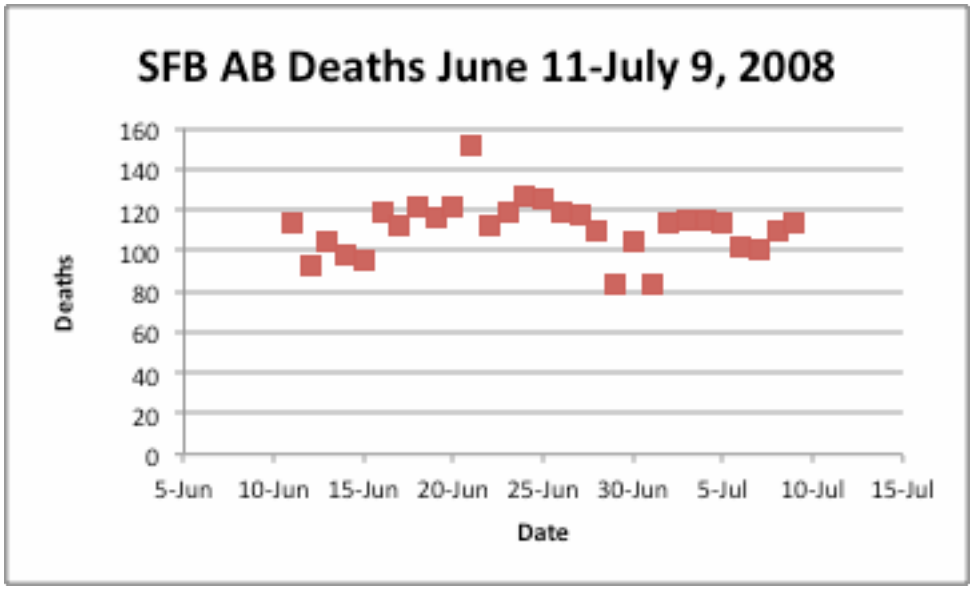

South Coast AB, Mortality vs. PM2.5, 2007-2010
Daily Death Count
0
20
40
60
80
100
120
140
160
0
10
20
30
40
50
60
70
80
90
PM2.5 (micrograms per cubic meter)


San Francisco Bay AB, Mortality vs. PM2.5, 2007-2010
Daily Death Count
0
10
20
30
40
50
60
70
80
0
10
20
30
40
50
60
70
80
PM2.5 (micrograms per cubic meter)

The absence of response in deaths to increases in $PM_{2.5}$ levels can also be seen during spikes in $PM_{2.5}$ levels. Consider the pairs of charts below for the San Francisco Bay Air Basin showing spikes in $PM_{2.5}$ levels and the corresponding death counts.

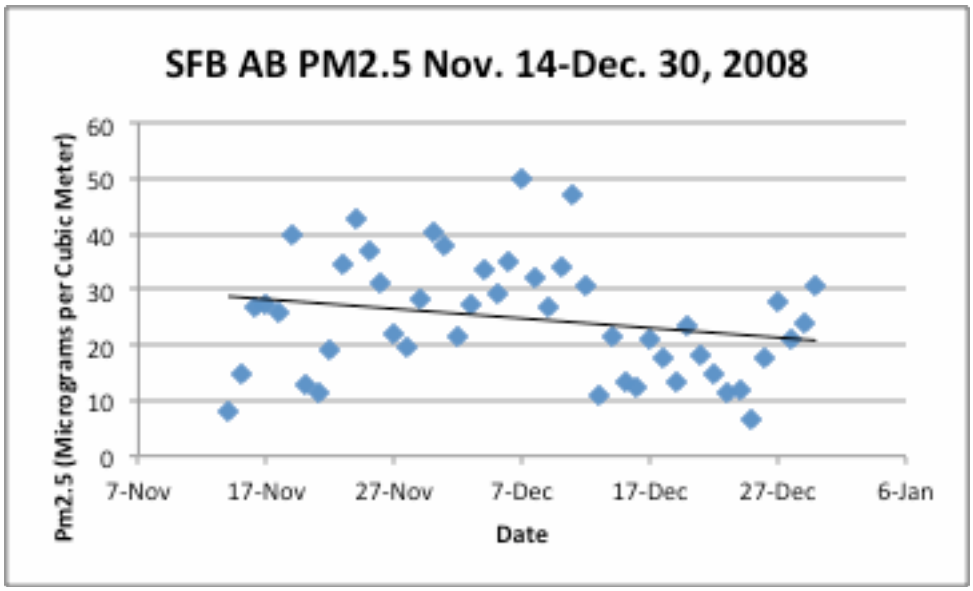
In the June 11-July 9 charts, the $PM_{2.5}$ spike *follows* by 5 days a spike in daily deaths.

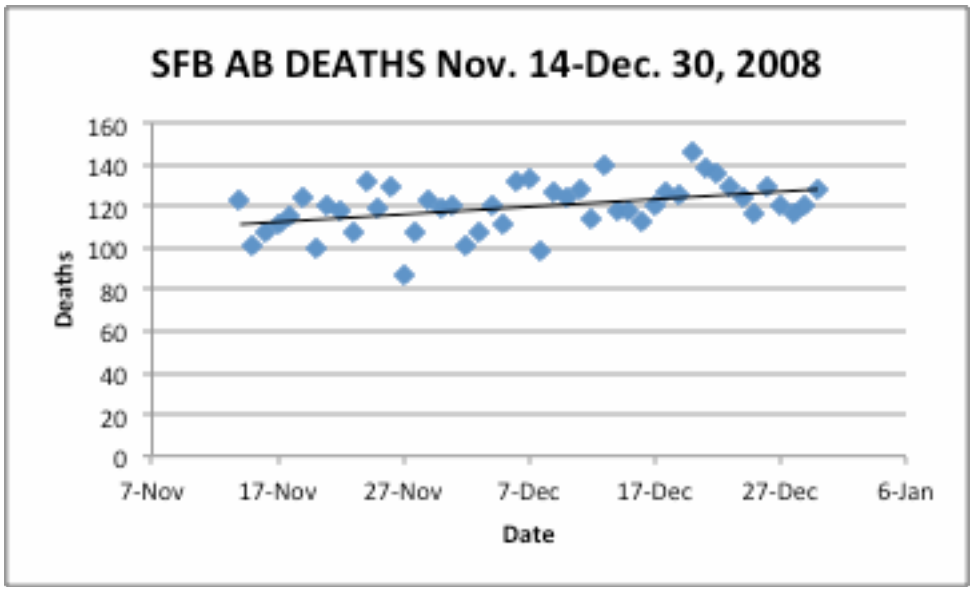


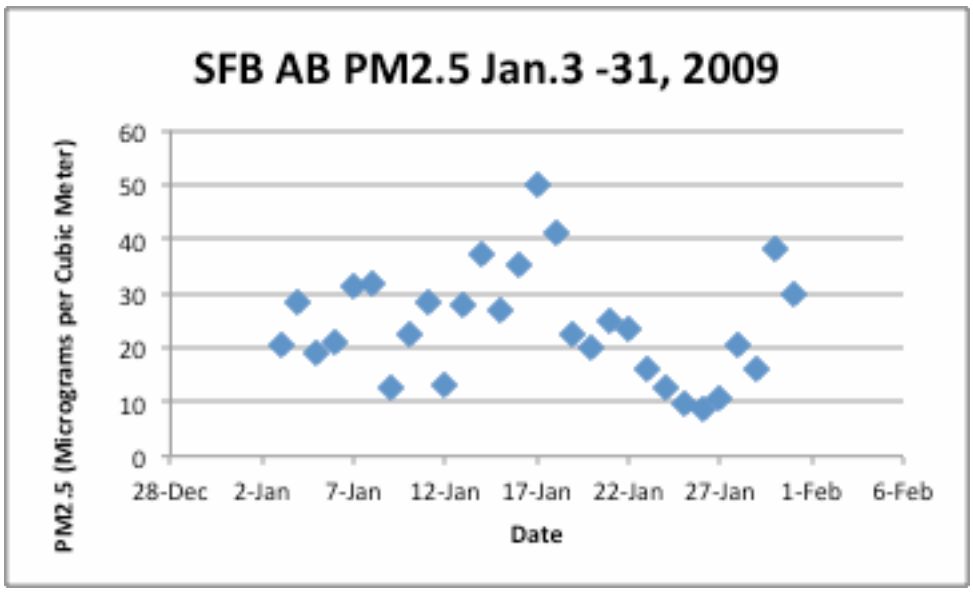


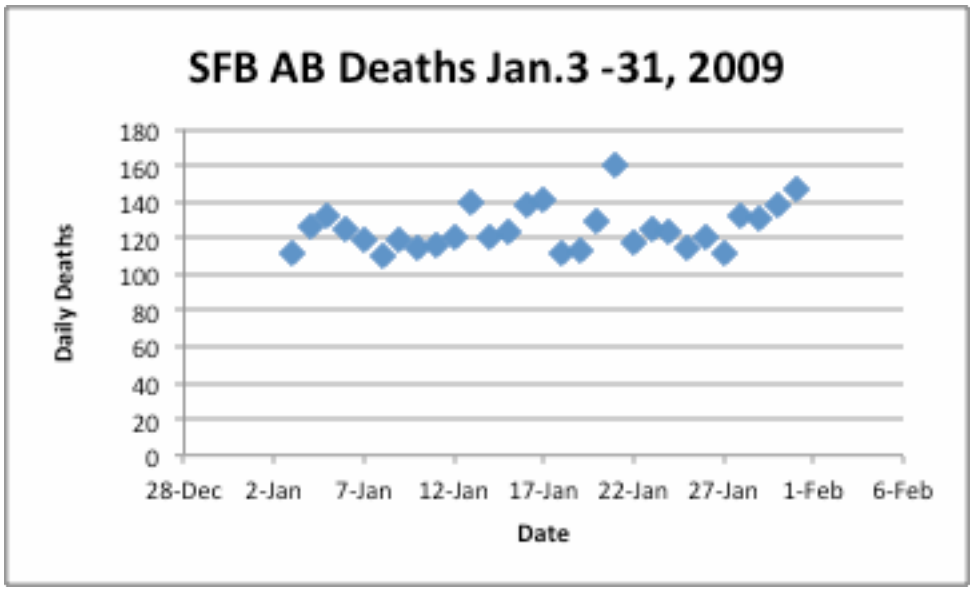
Not only is there no corresponding spike in deaths to match the $PM_{2.5}$ spike in the Nov. 14-Dec. 30, 2008 comparison, but during the period in question, as $PM_{2.5}$ levels are trending downward, deaths are trending in the opposite direction.

In the Jan.3-31 charts, below, once again there is no appreciable relationship between the peak in $PM_{2.5}$ and daily death count. Although daily deaths to spike up, following a spike in $PM_{2.5}$, the death spike occurs four days after the particle spike, beyond any alleged timeframe of causality.

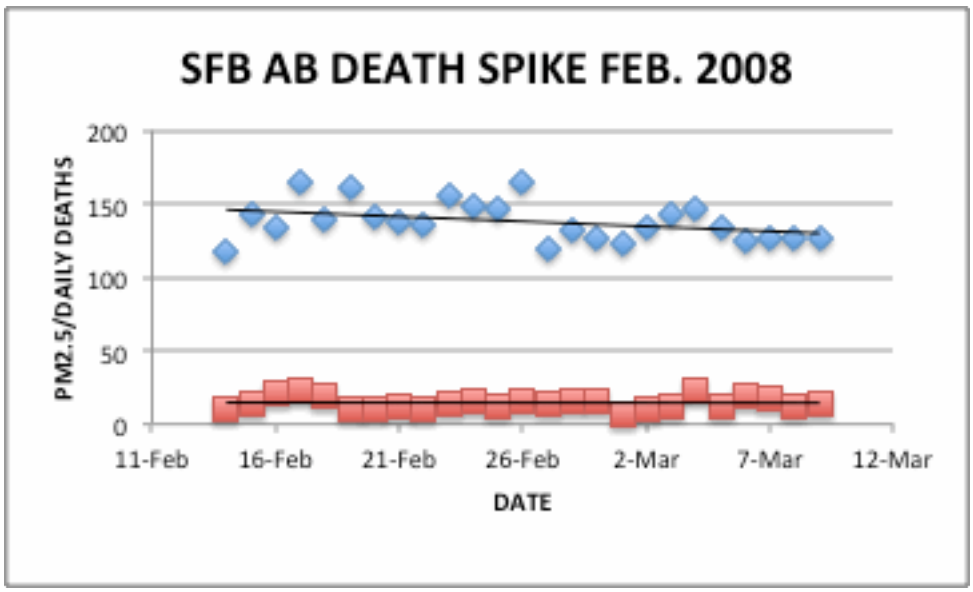


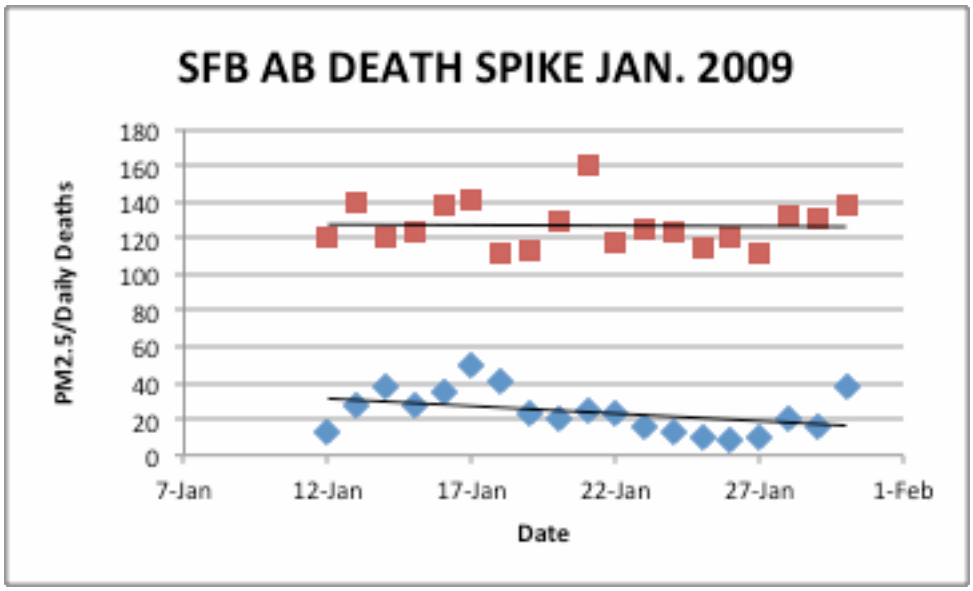






This data can also be looked at from the perspective of spikes in daily deaths. Again using examples from the San Francisco Bay Air Basin, there seems to be no corresponding relationship between death spikes and changes in daily ambient $PM_{2.5}$ on the days with the highest daily death tolls.





Discussion

The EPA hypothesizes that: (1) there is a linear relationship between ambient $PM_{2.5}$ and mortality; (2) the primary cause of mortality is cardio-respiratory in nature; and that (3) the elderly are particularly vulnerable. For the general population, according to the EPA, deaths should increase, by approximately one percent for every 10 micrograms per cubic meter of $PM_{2.5}$. This study does not confirm these hypotheses.

No correlation is reported between changes in ambient $PM_{2.5}$ levels and mortality on a daily basis in California, 2007-2010, including when limiting cause-of-death to

cardio-respiratory deaths among the general population, the all-cause deaths among the elderly and cardio-respiratory deaths among the elderly — i.e., the groups that the EPA alleges are most vulnerable to increases in ambient $PM_{2.5}$.

This is the largest and most recent systematic search for a statistical association between changes in ambient $PM_{2.5}$ and mortality. The study covers the entire state of California except where the air is so clean that daily $PM_{2.5}$ monitoring is not conducted. These excluded areas represent only about 6% of the state's population. The years 2007-2010 represent the most recent years for which CARB air quality data and California death certificate are available. So this study uses all the California data available.

This study's 854,109 deaths over four years (2007-2010) is comparable, for example, to the 1.3 millions deaths over six years (1997-2002) analyzed in the 27-community study (Franklin).[15] While the current study is smaller than the 5.6 million deaths (1999-2005) analyzed in the 112-city study (Zanobetti),[16] neither Zanobetti nor Franklin, were systematic in nature.

The deaths included in this analysis represent the deaths occurring among approximately 94 percent of the California population, as compared to Zanobetti's 36% of the U.S. population. Moreover, the data used in this study are more recent than any other study of $PM_{2.5}$ and mortality.

This study also empirically demonstrates that, even if $PM_{2.5}$ were associated with mortality as claimed by the EPA, it would be statistically impossible to verify the claim.

As shown in Table 4, the margin of error for daily deaths, selected to be two standard deviations from the mean (i.e., equivalent to a 95% confidence interval), is never mathematically exceeded even assuming the highest $PM_{2.5}$ measurement in conjunction with the EPA-asserted dose-response rate of a 1% increase in deaths for every 10 microgram per cubic meter increase in $PM_{2.5}$.

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[15] Franklin M, Zeka A, and Schwartz J. Association between $PM_{2.5}$ and all-cause and specific-cause mortality in 27 U.S. communities. *Journal of Exposure Science and Environmental Epidemiology* (2007) 17, 279-287.

[16] Zanobetti A and Schwartz J. The Effect of Fine and Coarse Particulate Air Pollution on Mortality: A National Analysis. *Environmental Health Perspectives* (2009) 117:6, 898-903.

Table 4. Maximum Expected Deaths Under EPA $PM_{2.5}$-Mortality Hypothesis							
Air Basin	Average Daily Deaths (Nearest 1)	Margin of Error (2σ)	Maximum Daily Death Count	Maximum $PM_{2.5}$ Measurement (Nearest 10s)	Maximum Expected Increment In $PM_{2.5}$ Deaths	Upper MoE for Daily Deaths	Avg. Deaths + $PM_{2.5}$ increment
Mountain Counties	12	8	23	140	2	20	14
Sacramento Valley	54	16	92	200	11	70	65
Salton Sea	12	8	25	100	1	20	13
San Diego County	51	16	79	150	8	67	59
San Fran. Bay	118	28	165	70	8	146	126
San Joaquin Valley	65	20	96	200	13	85	78
S. Central Coast	25	10	46	110	3	35	28
South Coast	249	48	344	80	20	297	269

In common with all epidemiologic studies of $PM_{2.5}$ and potential health effects, the study has several major deficiencies that prevent any definitive conclusions from being drawn.

Lack of Actual Individual Exposure Data

First, there is no information on actual exposures to $PM_{2.5}$. The $PM_{2.5}$ data is from monitoring stations that, while possibly providing a reasonably accurate idea of the air quality in the immediately vicinity of the various monitors, but provide no information about how much $PM_{2.5}$ anyone is actually respiring.

Indoor $PM_{2.5}$ levels can exceed outdoor levels. The time spent indoors versus outdoors varies between individuals. People may commute between areas of different outdoor $PM_{2.5}$ levels. Work $PM_{2.5}$ may be different from home $PM_{2.5}$. Some people spend more time in traffic. Some smoke or are exposed to secondhand smoke, both of which dramatically increase exposure to $PM_{2.5}$.

As a surrogate for $PM_{2.5}$ exposure, this study relied on the daily average reading from the highest monitor in the air basin, as measured and spotlighted by the California Air Resources Board. Other surrogate measures could have been devised (e.g., average daily reading from the closest monitoring station to the decedent's residence as determined by zip code or a daily median of all monitors in an air basin). But in the end, all surrogate measures are arbitrary and no surrogate measure will likely very accurately substitute for actual individual exposures.

It should further be noted that virtually any other surrogate exposure selection would like result in a flatter daily $PM_{2.5}$ trend line, thereby further reducing the

possibility of finding any correlations. The selected surrogate, daily average $PM_{2.5}$ at the highest site is a virtual best-case scenario for identifying a correlation between daily $PM_{2.5}$ and mortality, as it allows for almost the greatest possible variation in the daily $PM_{2.5}$ trend line.

The exposure surrogate selected for this study is how CARB has opted to describe and represent to the public $PM_{2.5}$ levels in California air basins. It can therefore be recognized as an acceptable surrogate exposure measurement.

Lack of Information on Actual Cause-of Death

Although death certificates include a physician-determined cause-of-death in the form of an ICD code, there is no ICD code for cause of death due specifically to air quality. Relatively few deaths have ever been attributed to ambient air quality and non have ever been medically attributed to any form of particulate matter. Moreover, although the EPA has postulated that $PM_{2.5}$ can trigger adverse cardio-respiratory events that lead to death, to date, no recognized mechanism for $PM_{2.5}$-induced death has been identified, much less verified.

Mortality, perhaps especially from cardio-respiratory causes, may be multifactorial in nature. Death certificates typically do not contain information on confounding or competing risk factors, and there have been few, if any, scientifically credible efforts to collect such information.

Limited to short-term mortality

This analysis only provides information on the relationship between exposure to ambient $PM_{2.5}$ and mortality on a short-term basis, i.e., hours and days. Although the EPA asserts that there is a long-term statistical association between long-term exposure (i.e., years) to ambient $PM_{2.5}$ and mortality, EPA's hypothesis that ambient $PM_{2.5}$ causes short-term mortality is central to its $PM_{2.5}$ regulatory program. Moreover, if $PM_{2.5}$ is actually associated with mortality, that association would be more credibly identified on a short-term basis rather than a long-term basis since long-term exposure estimates would be much more uncertain than short-term exposure estimates and long-term mortality is a far more complex phenomenon than short-term mortality.

Despite the above-mentioned limitations, which apply to all existing epidemiologic study of air quality, these results are consistent with what is known about $PM_{2.5}$. First, this study is essentially consistent with all other $PM_{2.5}$ studies in terms of reporting a zero correlation.

While many prior studies have claimed to report statistically significant associations between ambient $PM_{2.5}$ levels and mortality, without exception all those results are arguably zero correlations as the magnitude of the associations all fall within the

"noise" range of epidemiology.[17] They are the results of non-systematic (read "cherry-picked") data collection and arbitrary (read "biased") model selection. Not much confidence ought be placed in them.

This study, in contrast, is a simple test of EPA's hypothesis using all the recent data from a large contiguous geographic area in which an association between $PM_{2.5}$ and mortality should be readily identifiable if one exists at all.

Conclusion

This is the first systematic epidemiologic study to examine the potential relationship between population exposures to $PM_{2.5}$ and mortality. It is comparable in size to other large epidemiologic studies, but is far more objective in terms of data selection and involves more recent data. Looking at essentially the entire state of California for the years 2007-2010 and using data provided by the state of California, there no correlation was found between changes in ambient $PM_{2.5}$ and mortality.

The lack of correlation was confirmed by examination of unusual spikes in $PM_{2.5}$ and mortality. No spike examined indicated any sort of relationship to the other variable. If a causal relationship existed between $PM_{2.5}$ and mortality, it could reasonably be expected to have been found by this study, particularly as the state of California has some of the "worst" air quality in the U.S.

This study also demonstrates that even if the EPA-claimed association between $PM_{2.5}$ and mortality existed, even among the claimed most vulnerable groups, it is likely not possible to verify the association through epidemiologic or other statistical study. This fact, of course, would affect all $PM_{2.5}$-related cost-benefit analyses as it would be necessary to reduce the lower range of deaths to zero.

[17] *See e.g.,* Hill AB. The environment and disease: Association or causation? *Proceedings of the Royal Society of Medicine*. 1965;58:295-300.