

Estimating Premature Deaths From Long-term Exposure to PM2.5

Summary of Major Evidence on PM2.5 and Premature Deaths in California



The February 26, 2010 California Air Resources Board (CARB) Symposium on “Estimating Premature Deaths from Long-term Exposures to PM2.5” included talks by Michael Jerrett, Ph.D., of UC Berkeley, James E. Enstrom, Ph.D., of UCLA, and many other experts on PM2.5 health effects. The Jerrett PPT presentation on “California-specific Studies on the PM2.5 Mortality Association” provides important new evidence. Jerrett slides 12 and 26 present relative risk (RR) results for the CA CPS II cohort showing RR ~ 1.00 (0.97-1.03) for all causes of death during 1982-2000. Jerrett slides 13 and 14 discuss these results. Note that RR = 1.00 means no increased risk due to PM2.5 and that 95% confidence limits including 1.00 mean no statistically significant effect. The Jerrett result is in exact agreement with the Enstrom 2005 result for the CA CPS I cohort RR = 1.00 (0.98-1.02) for all causes of death

during 1983-2002. The Enstrom PPT presentation on “Critique of CARB Diesel Science, 1998-2010” shows Enstrom 2005 results on Enstrom slide 22. Based on the CA CPS I and CA CPS II results, by far the two largest California-specific studies, the number of “premature deaths” associated with PM2.5 exposure is zero, not the thousands of deaths presented to the CARB members when it voted to approve the off-road and on-road diesel regulations.

Relevant Internet Websites:

Webcast of Entire Symposium

(<http://www.cal-span.org/cgi-bin/archive.php?owner=CARB&date=2010-02-26>)

Jerrett PPT Presentation “California-specific Studies on the PM2.5 Mortality Association”

(<http://www.arb.ca.gov/research/health/pm-mort/jerrett.pdf>)

Enstrom PPT Presentation “Critique of CARB Diesel Science, 1998-2010”

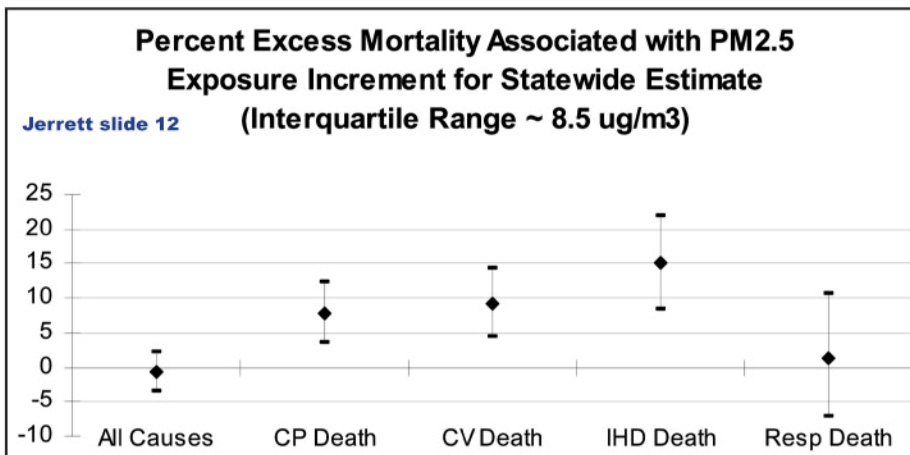
(<http://www.arb.ca.gov/research/health/pm-mort/enstrom.pdf>)



Transcript of Statements by Michael Jerrett, Ph.D. - UC Berkeley

California Results from 1982 ACS Cancer Prevention Study (CA CPS II):

Minutes 2:20:48 – 2:23:22 of Webcast



CP - Cardiopulmonary, CV - Cardiovascular, IHD - Ischemic Heart Disease, Resp - Respiratory

“This is from the statewide study and this is against the interquartile range of about 8.5 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$). [See Jerrett slide 12]. **These are percent increases in mortality and we don’t see in the statewide assessment an elevation in all cause mortality in relation to particulate matter [Jerrett slide 12 shows about -0.5% for ‘All Causes’].** But we do see this pattern that’s been observed in numerous other studies that Arden Pope brought up that cardiopulmonary mortality [CP Death], cardiovascular [CV Death] and ischemic heart disease [IHD Death] they order so as we move from less to even more plausible biological end points we see larger effects and we see an elevated effect for respiratory mortality [Resp Death], but we don’t have a lot of sample here so it’s not significantly elevated. So we tested for latitude, county clustering, and ozone as a co-pollutant and these results stand

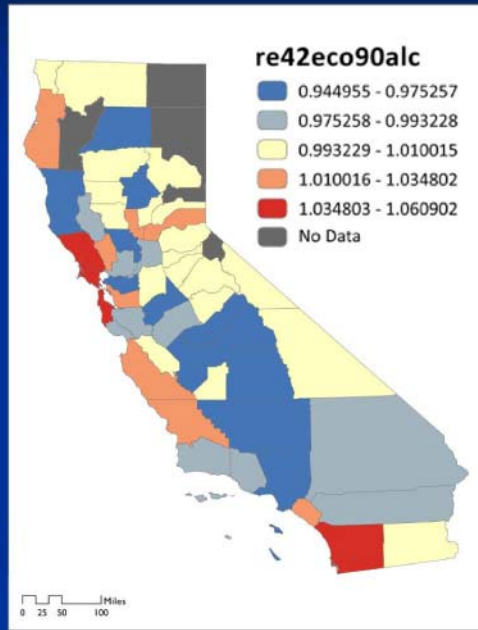
Results Continued Jerrett slide 13

- Results for PM2.5 not confounded by latitude, county clustering or by ozone as a co-pollutant
- All cause without cancer: RR = 1.04 (95% CI: 1.00,1.08)
- Why null results for PM2.5 on all causes?

All Cancer Death Negative

Jerrett slide 14

- Null PM2.5 effects on all cause mortality resulted from strong negative association with cancer risk
- Statistical models geared toward predicting for cardiopulmonary death
- Unexpected low cancer mortality residuals in LA and Central Valley where PM2.5 is high

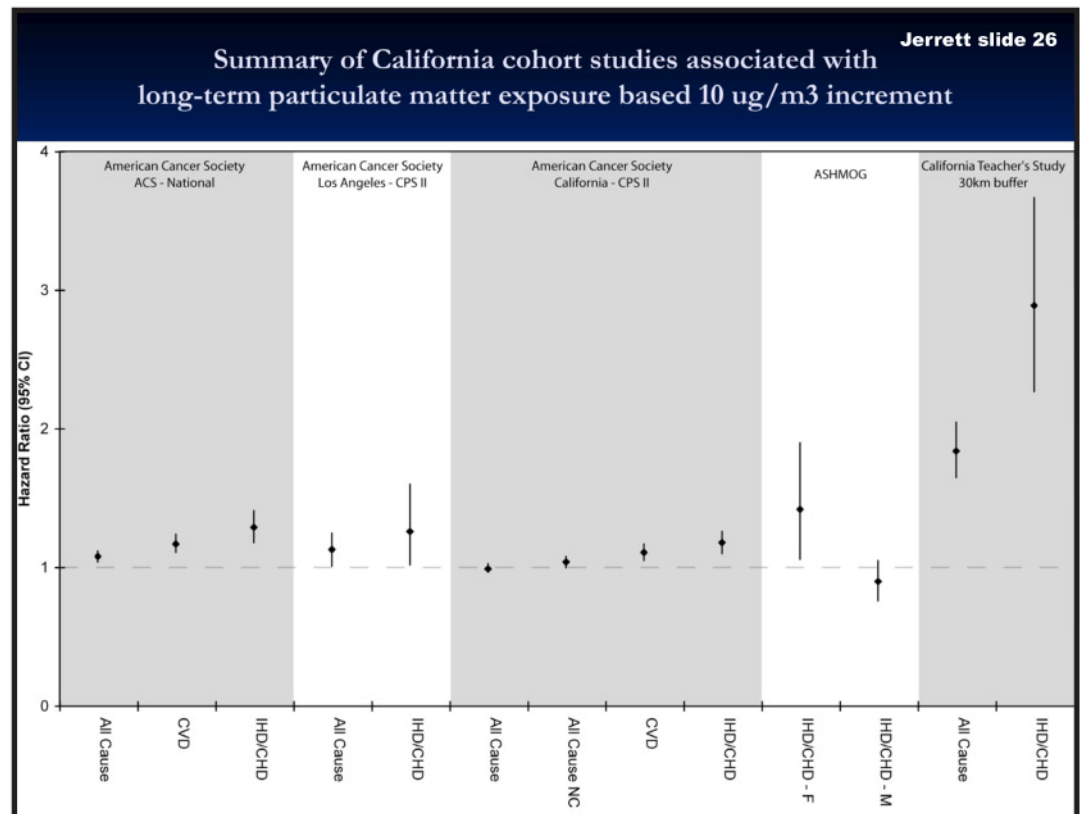


up [Jerrett slide 13, first point]. So that they are slightly lowered when we include ozone but significantly elevated. We started wondering why would we see such high elevation in cardiovascular disease but not high elevations in all cause mortality [Jerrett slide 13, third point]. Well if we look at our 22,000 deaths close to 10,000 of them are coming from cardiovascular disease but there's another 9,000 from other causes and the dominate cause in that other grouping is cancer. And what we see is when we take cancer out of the all cause, we see a risk estimate that is very similar to what Dr. Enstrom got about 4% increase [Jerrett slide 13, second point] and we have to ask "well, what does cancer have to do with it?" [Dr. Jerrett made an incorrect statement regarding Dr. Enstrom's 4%

increase. As shown below in slide 22 of Dr. Enstrom's presentation, the 4% increase involved the RR =1.04 for all cause mortality during 1973-1982, not the RR = 1.04 for non-cancer mortality during 1982-2000 shown in Jerrett slide 13.] And this is a map you can think of this as the mortality that we weren't able to predict with our individual level variables like smoking and alcohol consumption [Jerrett slide 14: map "re42eco90alc"]. And what we see is that after we apply all those individual variables there isn't much residual variation left in the cancer outcome where we have the most pollution [Jerrett slide 14, third point]. So our model is predicting these outcomes very well where we have a lot of pollution. We haven't honed our statistical models to look at cancer outcomes because we've been focused on cardiovascular mortality [Jerrett slide 14, second point]. I think we probably need these preliminary results. We need to go back and to include things like family history of cancer and other variables to get a better assessment of why we are seeing this negative association with cancer. But we do understand why we are getting a null result for all cause now and it's because we do see this negative association with all cancer [Jerrett slide 14, first point]."

Minutes 2:28:50 – 2:31:10 of Webcast

"Now, sometimes you need a picture to tell many words and I think this picture summarizes things quite nicely. I have the national level American Cancer Society Study risk estimates that are in the so called Krewski report [Jerrett slide 26, ACS CPS II National results]. So this shows them for all causes, cardiovascular disease, ischemic heart disease. This is from my Los Angeles study [Jerrett slide 26, ACS CPS II Los Angeles results]. Bigger error bars



CHD - Coronary Heart Disease, CVD - Cardiovascular Disease, IHD - Ischemic Heart Disease

because we have a smaller sample, but comparable risk estimates. This is the California-wide study [Jerrett slide 26, ACS CPS II California results]. They are slightly smaller overall than what we are seeing in some of the other studies, but significantly elevated for cardiovascular, ischemic heart disease, and cardio pulmonary, not shown here, and all causes minus cancer. The Adventist study we see this large increase for women but not for men [Jerrett slide 26, AHSMOG results]. And then the California Teachers Study we see a very large increase, nearly tripling of ischemic heart disease deaths and a near doubling of deaths for all causes [Jerrett slide 26, California Teachers Study results]. So, if we go back and we think about what leading epidemiologists like Rothman will say... they'll say don't worry about single studies, don't worry about particular confidence intervals. Look at the pattern in the risks. And the pattern we see here is that for every California-wide study, there is a significantly elevated risk of dying in relation to air pollution." [Dr. Jerrett made two incorrect statements in his last sentence. First, Jerrett slide 26 entirely omits the California-wide results from Enstrom 2005 that are shown in Enstrom slide 22 below. Second, the phrase "significantly elevated risk of dying" is misleading with respect to all causes of death, since only two points in Jerrett slide 26 pertain to California-wide deaths from all causes and the most significant of those two points (CA CPS II) is not elevated.]

Transcript of Statements by James E. Enstrom, Ph.D. - UCLA

California Results from 1959 California Cancer Prevention Study (CA CPS I)

Minutes 1:53:10 – 1:53:37 of Webcast

"My study came out at the end of 2005 using the original CPS I cohort for California subjects. And I found a small effect from 73 to 82, but no risk at all, 1.00, from 1983 to 2002 [Enstrom slide 22]. And so this again is shown no effect in California."

Minutes 2:32:23 – 2:32:41 of Webcast

"In terms of total deaths, which are what are used to calculate premature deaths by the Air Resources Board, if I didn't misinterpret what he [Dr. Jerrett] said, there was no effect—very consistent with my findings. And so that would make my study and his study by far the two largest studies in California."

Enstrom slide 22

December 15, 2005 *Inhalation Toxicology* Paper by James E Enstrom

49,975 elderly Californians in 11 counties followed during 1973-2002 in California Cancer Prevention Study (CA CPS I)

"For the initial period, 1973–1982, a small positive risk was found: RR was 1.04 (1.01–1.07) for a 10- $\mu\text{g}/\text{m}^3$ increase in PM_{2.5}. For the subsequent period, 1983–2002, this risk was no longer present: RR was 1.00 (0.98–1.02). For the entire follow-up period, RR was 1.01 (0.99–1.03)."

22

1 - Relative Risk or (RR): A relative risk of 1.0000 implies exactly no effect. In environmental studies there essentially is never a RR larger than 2 so there is no proof of cause and effect. Federal courts officially take the position that the RR must be greater than 2 to be admissible, i.e. start the argument for cause and effect.

A relative risk of 1.000 implies exactly no effect.

Randomness - Not very easy for human beings to understand. In the 1600s the nobles of France gambled a lot and they did not understand the odds of anything. In a game of dice, the total of the up faces range from 2 to 12 and they more or less thought them equally likely. So a RR can vary from one by randomness. 1.01 might be a random fluctuation from 1.00. The p-value helps you get a handle on random fluctuations. This is just a start. People can cheat with p-values.

Bias - RR can vary from one due to some other cause. For example, maybe the two groups are unbalanced with respect to some important factor. In the case of thinning bones, osteoporosis happens more often in women. So if the two groups ratios are 55:44 female:male, there is a problem. The unbalanced ratio can bias the results.

The problem here is that we don't actually measure or know all the potential sources of bias. So a conservative position is that a RR has to be larger than 2.00 to be considered admissible, i.e. start the discussion that the effect might be real. In real science, the burden of proof is on the person making the claim. Without a RR greater than 2 there is no proof worth paying attention to (legally).

Regulatory agencies such as CARB has no admissible evidence that air quality causes ill health. Essentially all the papers they use and cite add up to nothing from a legal point of view for cause and effect.