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'Unpopular' science plays a vital role in getting at the truth

LOIS HENRY
THE CALIFORNIAN

Is PM2.5 killing people, or not?

It seems like a basic scientific question.

Yet, as a recently published study shows, the science is anything but settled.

Despite significant questions in this new paper, which echo a growing number of other studies published in recent years, there is almost zero discussion in the scientific and regulatory communities of whether PM2.5 is truly a killer.

The silence is oddly deafening.

Yes, it involves complex scientific information. But you need to pay attention to the details because it affects you in very direct ways.

Regulations mandating reduction of PM2.5 (tiny particulate matter in soot, dust and exhaust) are what keep people from lighting fires on cold winter nights here in the San Joaquin Valley, which is nowhere near compliance with national standards and has some of the most restrictive rules on PM2.5.

More important than cold, empty fireplaces, PM2.5 regs cost us a lot of money, every single day.

That's because all long-haul trucks, tractors, bulldozers, etc., in California made before 2014 are required to be retrofitted with expensive filters and/or replaced, driving up costs for consumers on just about everything we use, eat or wear.

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HENRY

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Those regulations are justified on a public health basis by the claim that PM2.5 causes thousands of "premature deaths" every year in California and nationwide.

That idea was born in 1995 after a major study authored by C. Arden Pope found an association between exposure to PM2.5 and premature deaths.

INTO THE WEEDS

Here's what "association" means in this context.

This was a very large epidemiological study that looked at data on more than 500,000 Americans enrolled by the American Cancer Society in 1982 and followed for death through 1989.

Pope combined each subject's lifestyle data with their estimated PM2.5 exposure level based on their address in 1982.

He then did a statistical analysis to estimate the contribution of PM2.5 to their deaths through 1989.

Pope compared that data, which included the subjects' lifestyles, ages, address and deaths, to exposure levels of PM2.5 and created a statistical analysis of whether PM2.5 may have played a role in their deaths.

The role PM2.5 may have played is labeled as the "relative risk" in this and other studies.

In that seminal Pope study, the relative risk was found to be 1.07, meaning for every 10-unit increase in PM2.5 exposure, you have a 7 percent greater risk of dying early.

STUDY QUESTIONED

The 1995 American Cancer Society study by C. Arden Pope created a huge outcry at the time with many scientists questioning its conclusions.

That didn't stop the Environmental Protection Agency from creating a national ambient air quality standard for PM2.5 in 1997, based primarily on Pope's work.

OPPOSING VIEW

Particulate pollution is killing humans

DOUGLAS DOCKERY & JOHN EVANS

The hazard of air pollution was evident in the 4,000 excess deaths reported from the Great Smog of 1952 in London. However, measuring the cumulative health burden of living with chronically high air pollution is more difficult.

Findings from large prospective cohort studies in the U.S., Canada, and Europe have consistently shown that fine particle and, to a lesser extent, ozone air pollution are associated with increased mortality. Based on this evidence and air pollution monitoring in the United Kingdom, the Committee on the Medical Effects of Air Pollutants estimated that loss-of-life expectancy equivalent to 29,000 deaths was attributable to fine particles in 2008.

In the current issue of The Lancet, Aaron Cohen and colleagues used global population-weighted mean concentrations of particle mass with aerodynamic diameter less than 2.5 micrometers (PM2.5) and ozone and integrated exposure-response functions to estimate the relative risk of mortality from ischaemic heart disease, stroke, chronic obstructive pulmonary disease, lung cancer and lower respiratory infections.

Their article, "Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of Diseases Study 2015," reports that 4.2 million deaths globally were attributable to fine particles and an additional 254,000 to surface ozone in 2015.

Cohen and colleagues report that China and India, with the largest populations and commensurately high levels of pollution, had the largest estimated numbers of deaths attributable to air pollution: 1.11 million and 1.09 million, respectively, in 2015. Similar numbers have been estimated previously. Although the numbers of deaths are not as large as in China and India, the proportions of deaths attributable to air pollution were similarly high in neighboring countries on the Indian subcontinent.

Moreover, Cohen and colleagues estimate that the highest mortality rates attributable to fine particles were in countries in Central Asia, where air pollution has not been measured. Advances in remote sensing by satellites and air pollution modeling permit estimates of fine particle air pollution.

When coupled with the geographical distribution of the population, these methods can produce reliable estimates of exposure to fine particles. Therefore, estimation of the disease burden of air pollution is feasible in countries or even cities with few or no direct measurements of air pollution.

Such estimations also require extrapolation of epidemiological evidence from developed countries to the higher air pollution exposures in the developing world. Borrowing information from studies of analogous fine particle exposures to household air pollution, secondhand smoke, and active smoking provides a framework for extrapolation that is internally consistent across a range of inhaled doses.

The nonlinear shape of the integrated exposure-response function used by Cohen and colleagues means that the smallest benefits from incremental reductions in pollution would be expected in the most polluted countries. Because of the profound implications for air pollution control, this hypothesis needs to be tested in mortality studies and, wherever possible, by assessing the health benefits of interventions to reduce pollution in highly polluted communities. Until the results of such studies become available, the uncertainty in the shape and marginal slopes of the integrated exposure-response function (especially at high concentrations) must be fully acknowledged and, to the extent possible, quantified.

Cohen and colleagues also estimate high rates of deaths attributable to particulate air pollution in Egypt, Iraq, Libya, United Arab Emirates and Yemen. Fine particle pollution in these arid countries includes substantial fractions of resuspended crustal material. Assuming all fine particles have the same toxicity, the estimates of the effect of air pollution on mortality are quite large.

The evidence from developed countries has not yet identified a specific component of fine particles to be responsible for the reported mortality effects; therefore, this assumption needs to be tested in countries with particle characteristics that differ from those in developed countries. Again, until the results of such studies become available, the uncertainty in estimates due to the assumption of equal toxicity must be acknowledged and quantified.

Tallying counts of deaths attributable to air pollution on a national scale identifies the health problem, but does not pinpoint the sources of pollution. The chemical transformations that produce ozone and a major portion of the fine particle mix are complex, and the populations exposed are often far downwind of the pollution sources. Calling attention to the tens of thousands or hundreds of thousands of deaths attributable to air pollution each year in developing countries is not sufficient. We in the public health community must provide the methods to identify the pathways of exposure and the approaches to reduce those exposures. The challenge falls to risk assessors, atmospheric chemists and meteorologists to provide the methods to back-calculate the sources of fine particles and ozone air pollution, even in the parts of the world with few data.

Furthermore, we must be honest about the strengths and weaknesses of the estimates we make from even the best methods now available. Scientists will need time to resolve these uncertainties. However, decision-makers must resist the temptation to wait for perfect information before they act, because the costs in loss of life to be expected while waiting will be substantial. Providing the capacity to reduce air pollution in each part of the world will have immediate, local health benefits. To the extent that these actions reduce emissions of carbon dioxide and other greenhouse gases, the long-term health risks associated with global climate disruption will be ameliorated in the entire global population.

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The annual standard back then was set at 15 micrograms per cubic meter. The current annual standard is 12 micrograms per cubic meter.

The San Joaquin Valley has yet to meet the 1997 standard and has virtually no hope of hitting the new standard by the 2025 deadline, by the way.

Between then and now, a number of studies have come out showing no association between PM2.5 and premature deaths.

These other studies are not based on the "gold standard" American Cancer Society data, however.

And they are routinely discounted by the EPA and the California Air Resources Board, or CARB, when the latest scientific information is evaluated for proposed new regulations.

BLACK SHEEP

The latest study, published in the journal Dose-Response last month by James Enstrom, did use the American Cancer Society data.

For anyone who's followed air pollution science for any length of time, the name "Enstrom" will likely elicit a response.

Enstrom has been arguing the merits of Pope's and other researchers' PM2.5 findings for the better part of 15 years.

It was Enstrom who discovered a CARB researcher who helped propagate those truck rules I mentioned earlier had lied about his Ph.D. credentials and CARB staffers knew about the deception.

It wasn't long after that when UCLA attempted to fire Enstrom from his longtime research position with the Environmental Health Sciences Department.

In a termination letter, he was informed "your research is not aligned with the academic mission of the Department."

Enstrom sued.

As part of the 2012 settlement, UCLA rescinded Enstrom's termination, paid him $140,000 and allowed him access to school resources, which he still has.

What I've learned over the years of covering Enstrom and this topic is that while Enstrom's views may be unpopular, no one has been able to throw shade on his actual work.

I find that telling.

In his latest paper, Enstrom uses the American Cancer Society human data — which he won't say how he obtained (which is bound to be a big deal, see sidebox at right) — to recreate Pope's 1995 study.

He shows that you can get a relative risk similar to Pope's or you can get no relative risk.

It all depends on which PM2.5 exposure data you use and the best available PM2.5 data yields no relative risk.

Of particular interest, Enstrom found when he separated the Ohio Valley area from the rest of the country, there's no significant effect in either area no matter which PM2.5 data he used.

That kind of sensitivity brings the whole idea of PM2.5 as a killer into question.

SILENCE ISN'T AN ANSWER

Since Enstrom's paper directly calls out Pope and his findings, I called him up.

Pope would only say that he knows Enstrom to be a very bright scientist, that he takes the questions in Enstrom's paper seriously and would be reading it closely and writing up a response.

"Hey, this is what science is about," he said. "We need people to be skeptical and pick apart our work. It forces us to be more rigorous."

That's a refreshing attitude and one I hope is catching.

Unfortunately, up to now researchers who've been vocal in questioning PM2.5's effects have been essentially blackballed, as I've been told.

Regulators ignore their studies. They can't get funding from EPA, CARB or the Health Effects Institute, the main funding sources for air pollution research (itself a conflict of interest). And essentially all have been turned down by the American Cancer Society for access to that human data used by Pope.

Even so, studies have been coming out showing virtually no effect from PM2.5, particularly in the western United States.

Even one study in 2016 by George Thurston, which purported to show an effect, actually has a relative risk of dying early from PM2.5 exposure of 1.02 for California. When you add the margin of error to that figure, it's basically a nonrisk.

DON'T FORGET THE LIVING

"Air pollution does kill people," another longtime researcher, Fred Lipfert, told me, citing the terrible London smog that killed hundreds of people in 1952. "But we don't know who it will kill or why."

And these large epidemiological studies don't get at those questions because there's no way to say what an individual's actual PM2.5 exposure is over time.

Aside from that, he said, PM2.5 isn't a pollutant.

"It's a regulatory construct," he said. "It includes everything of a certain size, which includes all kinds of stuff. Some toxic, some not."

Science should focus on what people are actually exposed to instead of these mass studies where exposure levels are so variable.

"I think Enstrom's paper cracked the door on that issue," he said. "We need more science on this issue and perhaps a recognition that regulation is not the end all, be all."

Because, he said, while air pollution studies focus on deaths, "It's the living population that pays the bill for all these regulations."

As one of those people, I'd like to at least see an open, honest conversation about this among scientists and regulators.

Contact Californian columnist Lois Henry at 395-7373 or lhenry@bakersfield.com. Her work appears on Sundays and Wednesdays; the views expressed are her own.

SECRET DATA

Epidemiologist James Enstrom and other scientists who object to studies that have shown associations between PM2.5 exposure and early death have long complained that the data used in those studies has been kept under wraps.

Findings must be independently replicated, opposition researchers have said.

The Health Effects Institute did hire researchers to do a reanalysis in 2000 on the 1995 study that spawned the PM2.5 regulations.

But many have argued those researchers simply used the exact same data and methodologies so it wasn't a true replication.

If a finding is true, they have said, it should come up the same no matter how data is reviewed.

The argument rose to the level of Congress back in 2013.

The Committee on Science, Space and Technology actually issued a subpoena to the EPA trying to get the data that was used for the 1995 study.

But the EPA doesn't own that data.

It belongs to the American Cancer Society, a private nonprofit that did not have to turn over the information.

That prompted a bill called the Honest Act, which would require full data disclosure for any study used by government agencies to enact regulations.

That bill passed the House but has yet to be heard in the Senate.

Meanwhile, Enstrom was able to obtain the original American Cancer Society data that he used to show no association between PM2.5 exposure and premature death in a paper published in March.

But he is shielding the source of that data, which has brought criticism of his paper.

Enstrom said, to date, the American Cancer Society has not contacted him about his use of the data.