

**Public Comments by Dr. Dan Costa on EPA's Proposed Action on National Ambient Air Quality Standards for Particulate Matter**

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My name is Dr. Dan Costa, and I served as the EPA National Program Director in Air, Climate, and Energy Research for the last 10 years of my 34-year science career at EPA.

Ever since the EPA was established in 1970, the foundation of its policies to clean and protect the air we breathe has been science. Tension has always existed between those who translate this science into policy and those who are regulated, but this vigorous discourse on its interpretation has been the best means to achieve balanced, fiscally-sound progress to clean the air and to protect public health.

Most of the testimonies in this public hearing will cry foul about the Administrator's disregard for the science that underlies arguments to strengthen the PM NAAQS. Hundreds of studies from the past 30 years have shown that PM poses a real and imminent threat to public health, and the current ISA delineates science that has led legitimate experts to conclude that PM impacts can be discerned at levels significantly below the current NAAQS level of 12 ug/m<sup>3</sup>. Indeed, the EPA science and policy staff who compiled and dissected the new science formally recommended a strengthening of the NAAQS to protect public health and reduce lives lost. This recommendation included all relevant uncertainties within established ISA processes, as well as uncertainties interjected by the political appointees along the way. The need for a tightened NAAQS was also the conclusion of the Independent Particulate Matter Review Panel convened by the Union of Concerned Scientists, acting in the role of the aborted expert PM panel that typically advises CASAC on specific science questions.

However, the strength of this collective conclusion was not mirrored by CASAC, which could not reach a consensus recommendation – perhaps due to its skewed reconstitution. Those members notable for their pertinent expertise in the science objected to the unfounded position of the CASAC Chair, whose opinion was clearly taken to heart by the Administrator – underscoring EPA leadership's disregard for quality science within its deregulatory agenda. Perhaps the Administrator should adopt the advice of Sir Patrick Lawther who, while on duty in St. Bartholomew's Hospital during the 1952 London smog, quipped to a friend – just breathe through your nose...that'll keep particles from getting deep into your lungs.

As I watch this debacle of air pollution science and its follow-on policy development, I reflect on the co-evolution of my science and personal storylines embedded in my 34-year career in EPA.

One storyline is grounded in my study of the toxicological effects and mechanisms whereby PM impacts the lungs and heart in rodent models. In the early 1990s, when the air pollution community was jolted by epidemiological evidence linking ambient levels of PM previously thought to be “safe” to adverse health outcomes, including death, there was a outcry to ascertain “biologic plausibility” as

these findings must clearly be a statistical fluke. In our research group at EPA, studies in animal models of compromised heart and lung function showed that fugitive oil fly ash particles could cause arrhythmias and cardiac arrest when introduced to the lungs of these models. Other studies in animals and humans have since confirmed that the heart is likely a prime target of PM-related adversities even at the low concentrations of the new epidemiological science. The CAAA mandates the newest science be considered in the NAAQS-setting process and that science now points to the risk of premature mortality of thousands of people exposed to PM below the current NAAQS.

A second storyline is more personal. I grew up in the coastal town of Somerset, MA, a town that housed two power plants – including one at Brayton Point, which was for decades the dirtiest power plant in New England. In the late 1970s, my father repainted his house and noted shortly thereafter that the paint turned bluish-green. Most people would assume a problem with the paint, but my Dad thought the discoloration was caused by acidic air pollution drifting from the Brayton plant a mile or so away. My Dad was a quiet, gentle man, a disabled World War II veteran with only an eighth-grade education, yet he gathered himself to trek to the plant and give his thoughts to the plant's public relations people. They asked him to return in a few days and handed him a check for about \$275.... Is that acknowledgement of responsibility?!

My father stayed active for years thereafter working in his garden, despite congestive heart issues. During the night of August 22, 1998, he experienced a fatal arrhythmia. Earlier that year, I had published that seminal study with the late Dr. Penn Watkinson that I noted above: *Cardiac arrhythmia induction after exposure to residual oil fly ash particles in a rodent model of pulmonary hypertension*. Why did I not connect the dots earlier and perhaps intervene for my father in some way? I don't know. Now the evidence lies before us that the lives of a few thousand people can be saved with a stronger NAAQS – how close to home need the evidence lie?