Interestingly, particulate matter is the only criteria air pollutant in the United States that is not regulated based on its chemical composition. An important distinction because PM$_{2.5}$ chemistry varies by source and location.

PM$_{2.5}$ standards may be set lower than scientifically justifiable

Standards should offer well-defined public health benefits

Particulate matter (PM) is a “criteria” air pollutant for which the EPA establishes a National Ambient Air Quality Standard. PM is a complex mixture of liquids and small particles that may include acids (such as nitrates and sulfates), organic chemicals, metals, and dust particles. The EPA regulates PM on the basis of size, rather than composition, with particular emphasis on “fine particles” or PM$_{2.5}$—that is, particulates smaller than 2.5 microns in diameter.

Late last year the EPA revised the level that defines safe exposure to fine particulate matter, also known as PM$_{2.5}$. The previous standard was 15 µg/m$^2$ (micrograms per cubic meter) per year. In December of 2012, that level was lowered to 12 µg/m$^2$.

The TCEQ participated in the public comment period, and submitted comments to the EPA expressing concern that this new standard was unreasonably strict and unsupported by available scientific research. There have been many studies published that followed large groups of people over decades and these studies generally report small associations between illness and very high PM$_{2.5}$ concentrations in air. However, many questions remain regarding the nature of the risk posed by PM$_{2.5}$, at what concentration of PM$_{2.5}$ this risk may occur, and whether risk is the same for every area of the United States.

PM$_{2.5}$ occurs naturally (wind-generated dusts, sea sprays, fires, and pollen or spore production by plants) and as a result of human activities either from mobile sources (cars, trucks, ships, and planes) or stationary sources (electric power plants, factories, mines, and farms). Interestingly, particulate matter is the only criteria air pollutant in the United States that is not regulated based on its chemical composition. An important distinction because PM$_{2.5}$ chemistry varies by source and location. The health effects that have been linked to PM$_{2.5}$ exposure in human studies also vary by region. Multiple studies report potentially increased levels of disease possibly linked to PM$_{2.5}$ in the eastern United States, but the evidence is inconclusive for the central and western portions of the U.S.

Incidents involving very high levels of PM$_{2.5}$ have occurred in the past. For example, during the “London Smog” incident in 1952, levels of PM reached concentrations of 4500 µg/m$^2$. These levels of PM were linked with human disease and death. However, based on the scientific data, it is not clear that the risks identified as a result of these extraordinary circumstances also
extend to the very low levels of PM$_{2.5}$ present in ambient air today. In fact, such extrapolations can be contrary to the basic principles of toxicology where the biological threshold (a level below which no effect is apparent) is a key concept. For example, for the average adult a very high dosage of aspirin can be lethal; yet swallowing an 81-mg baby aspirin tablet is a widely recommended treatment regimen prescribed by Western medicine and is almost certainly not toxic. Indeed, the central tenet of toxicology as stated by Paracelsus, one of the pioneers of modern medicine, in the early 1500s, is that “the dose makes the poison.” In other words: health effects are expected to vary with dose and that a high enough dose of any substance may be harmful.

Levels of PM$_{2.5}$ have been declining in the United States. Since 2000, PM$_{2.5}$ emissions have declined 55 percent throughout the country.

As levels have declined, epidemiological investigations have become more sophisticated, incorporating complicated statistical techniques designed to find small associations where they exist. However, as with any model, the end result is highly dependent on the initial assumptions that are made.

For instance, most of the available studies assume the relationship between PM$_{2.5}$ levels in air (the dose) and observed health effects is linear—with no threshold. In other words, the researchers assumed that there was no dose below which health effects would not be expected to occur, and that doubling the level of exposure to PM$_{2.5}$ would double the risk. That is, there is no safe level of exposure to PM$_{2.5}$. However, in clinical studies exposing human volunteers to PM$_{2.5}$, much higher doses than are encountered in ambient air in real-world conditions are required to achieve even very subtle effects, such as changes in heart rate, much less more severe effects like respiratory distress, heart attacks, or death. This finding implies that there is in fact a threshold below which exposure to PM$_{2.5}$ is not expected to cause health effects.

While there have been many studies in human populations followed over decades, these studies generally report very small associations between adverse health outcomes and PM$_{2.5}$ concentrations in air. Nevertheless, many questions remain:

- **Who** (i.e., what subpopulation) is actually harmed (e.g., elderly, asthmatics, children)?
- **What** property of PM is harming them (size, concentration, composition, co-pollutants)?
- **How** are people harmed by low levels of PM$_{2.5}$ in ambient air, if they are harmed (i.e., what is the biological mechanism)?

In some studies, associations have been found between total PM$_{2.5}$ and health effects, but other studies have reported associations only for specific components of this complex mixture. Some associations are reported for specific areas of the country, but not others (e.g., in the eastern U.S., but not the western U.S.). Moreover, PM$_{2.5}$ is always accompanied by co-pollutant gases, such as ozone or sulphur dioxide. The roles of such co-pollutants in harming human health are not clear, although some studies indicate that these co-pollutants may actually be responsible for some or all of the risk attributed to PM$_{2.5}$. In such instances, the co-pollutant is said to be a confounding variable.

Due to the distinct possibility that there are confounding variables affecting the relationship between PM$_{2.5}$ and health effects, focusing on size-based PM$_{2.5}$ may not adequately capture potential risks to human health. As a result, continually lowering the National Ambient Air Quality Standards for PM$_{2.5}$ may not produce the intended improvements in human health.

Going forward, research in a number of areas could help clarify the current situation:

- Studies addressing the questions of who, what, and how (see above).
- Studies investigating which specific co-pollutants or other chemical components are responsible for the health effects associated with ambient PM$_{2.5}$.
- Studies examining how the risks posed by PM$_{2.5}$ in outdoor air compare to the risks posed by the (often much higher) concentrations indoors, where people spend most of their time.
- Studies considering how measurement error, confounding, and choices in statistical modeling impact the observed associations between PM$_{2.5}$ and human health.

In the absence of studies addressing the missing information discussed above, it is not clear that a lower national standard for particulate matter is necessary or that the proposed benefits will occur. Given the limited resources available to state agencies, approaches with well-defined benefits to public health are preferable. For more information, please see the TCEQ’s comments on the EPA’s proposed national standard.

The TCEQ is actively exploring the issues identified here. Stay tuned to Natural Outlook for additional information on these and other efforts to ensure that standards are designed to protect the citizens of Texas and also use the best available science. ☝️

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