Does Air Pollution Cause Diabetes?

By Chuck Dinerstein, MD, MBA — August 24, 2020

There are any number of papers supporting the idea that higher levels of air pollution are inversely correlated with poorer health outcomes. These studies all suffer from the fellow travelers of air pollution, traffic density, poverty, and lesser education which confound a clear linkage between air pollution and health. A new study offers a possibility.

There are no autopsy studies except for occupational exposure to dust that link polluted air with poor health. Some studies have demonstrated how small pollution particles and let's limit ourselves now to PM$_{2.5}$, might enter the bloodstream and be engulfed by tissue. But no direct evidence of causation. A laboratory study looked at the genomics and metabolomics of diabetes mellitus in mice.

Although the mechanisms by which inhalation of PM$_{2.5}$ induces [insulin resistance and Type 2 diabetes mellitus] remain unclear, a constellation of responses including inflammation, and redox stress, have been implicated.
A control group of mice was fed a high-fat diet that would result in the development of insulin resistance and diabetes and served as a control; the treatment group had "mice chow," and were exposed to 10 times the concentration of ambient PM$_{2.5}$ for several hours a day over about three months. [1]

"PM$_{2.5}$ induced abnormalities in glucose clearance and insulin responses that were comparable to HFD [high-fat diet] but were seen only in males."

- The mice with chronic air pollution exposure had alterations of their oxygen consumption and energy expenditure, inflammation of the liver, the elevation of triglycerides, and depletion of stored glucose, glycogen. More importantly, none of these findings were present in female mice, only the males.
- The mice demonstrated differences in genetic expression, again the diet caused the most significant perturbations primarily in pathways involving lipid regulation and synthesis, inflammation, and glucose creation. Mice with chronic air pollution showed alterations in pathways involving inflammation, anti-oxidation, and the transport of metals and glucose metabolism. Most interesting is that the greatest change in genetic expression was for genes involved in circadian rhythms. Following the associative chain, circadian rhythm alterations are associated with "the development of cancer, metabolic, and cardiovascular disease."
- The differences in gene expression were due to epigenetic "reprogramming" in the face of the stress of air pollution or diet; changes that partially reverse for those mice exposed to PM$_{2.5}$ when the exposure ended.

As with all good studies, it raises more questions than it answers; and of course, it has limitations. It was a study in mice, employing significantly higher concentrations of PM$_{2.5}$ than our exposure. The dosage that the mice received (exposure time multiplied by PM$_{2.5}$ density) was also considerably higher since most human activity takes place indoors where ambient PM$_{2.5}$ is significantly lower. Finally, the differences were not seen in female mice, only the males.

So what can we take-away? At least based upon this data, the linkage of air pollution to health outcomes may be found in alterations in multiple gene expressions; there is no single pathway making the metabolic outcome a significant computational problem. There is no recipe for factors, only their relative relationship to one another. More importantly, the effects of air pollution are much less than the impact of the high-fat diet, which translates into our dietary choices. Would I like cleaner air? Absolutely. But in the tradeoff between cleaner air and a healthier diet, we can get much more bang for our buck by the steady decline in air pollutants already underway and a greater emphasis on discovering and sharing a more healthful, nutritious diet. Let me put this another way, our individual health outcomes are much more strongly related to the food than the air we inhale.

[1] Ambient PM$_{2.5}$ was obtained using a collection system and presumably was obtained in Cleveland, the site of the laboratory. What constituted the PM$_{2.5}$ was not addressed.