

**Preliminary Comments on the Policy Assessment from Dr. Michael Jerrett  
(revised 11-09-16)**

**Comments on Chapter 3**

*1. To what extent does Chapter 3 capture and appropriately characterize the key aspects of the evidence assessed and integrated in the ISA?*

For the short-term effects on respiratory, cardiovascular and mortality outcomes, Chapter 3 accurately conveys the science and key aspects of the evidence base used in the ISA. The chapter also does a good job of presenting the human chamber studies and the in vitro and in vivo toxicological evidence. The upgrading of the relationship between short-term effects and respiratory outcomes to “causal relationship” is well supported by the science and the EPA has correctly characterized the science in reaching this conclusion. For the most part, with the exception of children’s respiratory health and long-term exposures, all other conclusions about the causal determinations for other outcomes appear well-supported by the science and appear to have been correctly interpreted by the EPA.

On the longer-term effects, particularly with respect to children’s respiratory health, especially asthma incidence, the document does not always accurately capture the science as presented in the original articles. There are issues with the interpretation of the McConnell study. In particular, the EPA notes that there is not adequate control for confounding by PM2.5 or other pollutants related to traffic. The McConnell study did control for confounding by PM2.5 and other pollutants through the central site monitor, and the relationships remained largely the same after control for co-pollutants. Much of the PM2.5 in Southern California forms as a secondary pollutant, with the direct contribution from traffic being relatively small. The authors were unable to control for other constituents of traffic because they were using a dispersion model that resulted in extraordinarily high correlations among the estimated pollutants, which were likely not indicative of the actual correlations that would be observed between NO2 and some of the other pollutants.

*2. To what extent is staff’s consideration of the evidence from epidemiologic and controlled human exposure studies, including important uncertainties, technically sound and clearly communicated? What are the Panel’s views on staff’s interpretation of the health evidence for short-term (section 3.2) and long-term (section 3.3) NO2 exposures for the purpose of evaluating the adequacy of the current standards?*

Overall the interpretations of the evidence from controlled human studies and epidemiological findings are sound and well presented, with, as mentioned, the exception of childhood respiratory health.

My major concern is with the presentation and conclusions regarding the long-term effects of NO2 on children’s respiratory health, particularly asthma. The primary argument against taking any further action appears to hinge on the difficulty of separating out the effects of NO2 from other pollutants in the complex mixture and lack of control for confounding pollutants.

The argument about co-pollutants is potentially an argument that could be used against making regulatory changes for any criteria pollutant, as there are other pollutants, which depending on the spatiotemporal scale and means of assessing exposure, have moderate to high correlations with other criteria pollutants. Regionally, PM<sub>2.5</sub> and ozone, for example, often have moderate to high correlations ( $r \sim 0.7$ ).

I would like the EPA to clarify whether they have even taken action to tighten (meaning lower the standard) for any pollutant based on a “likely to be causal” determination. Have they ever recommended against adopting more stringent standards primarily on the basis of co-pollutants have moderate to high correlations with the pollutant in question?

Correlations with BC would be less problematic– this pollutant is at least on it’s own – has weak toxicological plausibility – but it too could be a marker for diesel exhaust. The correlations noted between NO<sub>2</sub> and other traffic pollutants appear to be partly artifacts of the similarities in model structure (e.g., dispersion or land use models), which have likely increased the correlations observed beyond what would be expected if multiple measurements had been made in the field.

On the second question in Charge Question 2, it is concerning that so many of the long-term studies have concentrations well below the annual average of the current standard of 53 ppb. Many of the communities in the Southern California studies, which likely had influence on the results, were well below the design values for the annual average.