

## EPA Response to Dr. Mark Frampton's Question on the PM PA and Ozone ISA

### Question from Dr. Frampton:

*I have a specific question that...would help me in formulating my comments and considerations on both the PM PA and ozone ISA.*

*In considering the epidemiology studies, for health effects where the causality determinations are causal or likely to be causal, it is the practice in the ISA to exclude studies from outside of North America. I understand this has something to do with policy relevance, but I would like to better understand the justification for this. Where in the documents is this discussed/justified?*

*In the case of the Ozone ISA, short-term cardiovascular effects were downgraded to suggestive. However, it appears the restriction on studies outside of NA remained in the current ISA. What is the rationale for this?*

### EPA Response:

#### PM ISA

For the PM ISA, the scope that was used to define the epidemiologic studies that should be considered and ultimately included in the ISA was not limited to studies conducted in North America. For those health effect categories where we concluded a “causal relationship” in the 2009 PM ISA we further refined our scope to ensure we were focusing on the most policy relevant science. As detailed in the Preface of the PM ISA we stated the following:

“For epidemiologic studies, the scope is further refined when evaluating the evidence for those health outcomes where the 2009 PM ISA concluded that a “causal relationship exists” (i.e., short- and long term PM<sub>2.5</sub> exposure and mortality and cardiovascular effects) to ensure the evaluation of the evidence focuses on the studies that are the most policy relevant. As such, the focus is on those studies conducted in areas where mean PM<sub>2.5</sub> concentrations are <20 µg/m<sup>3</sup> or in the case of a multicity study where more than half of the cities have concentrations <20 µg/m<sup>3</sup>. However, studies where mean PM<sub>2.5</sub> concentrations exceed 20 µg/m<sup>3</sup> are included if the studies address specific areas where the evidence was limited, as identified in the 2009 PM ISA, such as copollutant confounding.” (P. P-14)

With this scope in place it did result in the exclusion of a large number of international studies from the discussion of short- and long-term PM<sub>2.5</sub> exposure and mortality and cardiovascular effects discussions, but it did not result in the exclusion of all studies conducted outside North America.

#### PM PA

As was the case in the last review of the PM NAAQS and in other recent NAAQS reviews (e.g., NO<sub>2</sub>, ozone), the PA's consideration of epidemiology studies focuses on multicity studies conducted in the U.S. or Canada. These studies are particularly useful for informing conclusions on the NAAQS because they examine associations over large geographic areas with diverse atmospheric conditions and population demographics. While studies conducted outside the U.S. and Canada are informative for conclusions on the overall strength of evidence linking exposures to health effects, such studies reflect

air quality and exposure patterns that may be less typical of the U.S., and thus less likely to be informative for purposes of setting standards in the U.S. This approach to considering what the epidemiologic evidence may indicate for standards has been reviewed by previous CASAC's and has been found to be appropriate.

#### Ozone ISA (Health)

Section 10.3.1.4 “The evaluation of epidemiologic studies focused on the associations between short- and long-term exposure to ozone and a range of health effects, including respiratory, cardiovascular, reproductive and developmental, metabolic, and nervous system outcomes (Table 10-2). In instances when a “causal” or “likely to be a causal” relationship was concluded in the 2013 Ozone ISA (e.g., short-term ozone exposure and respiratory and cardiovascular effects and total mortality, and long-term ozone exposure and respiratory effects), the epidemiologic studies evaluated for those outcomes were more limited in scope and targeted towards study locations that include U.S. airsheds or airsheds that are similar to those found in the U.S., as reflected in the PECOS tool. For outcomes for which the 2013 Ozone ISA concluded that evidence was “suggestive of” or “inadequate to infer” a causal relationship, the epidemiologic studies evaluated were not limited geographically or by airshed characteristics, as reflected in the PECOS tool.”

#### Ozone ISA (Welfare)

Section 10.3.1.5 “Similar to health effects, this ISA builds on information available during the last review (i.e., effects of ozone exposure on vegetation and ecosystems). For research evaluating ecological effects, emphasis was placed on recent studies that: (1) evaluated effects at realistic ozone concentrations occurring in North American airsheds and (2) investigated effects on any individual, population (in the sense of a group of individuals of the same species), community, or ecosystem in North America (Table 10-3). In instances when a “causal relationship” was concluded in the 2013 Ozone ISA (i.e., visible foliar injury, vegetation growth, reduced yield/quality of agricultural crops, reduced productivity, alteration of belowground biogeochemical cycles) the current review only evaluated studies conducted in North America. For all other ecological endpoints in Table 10-3 (terrestrial water cycling, carbon sequestration, terrestrial community composition, plant reproduction, phenology, or mortality, insects, other wildlife, plant animal signaling) there are no geographic constraints and all available evidence was considered.”