Appendix E

Further Details on Health Benefit Modeling Methodology

Proposed Heavy-Duty Inspection and Maintenance Regulation

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Table of Contents

This appendix provides further details on the health benefit modeling for the Proposed Regulation.

I. Non-Cancer Health Impacts and Valuations

California Air Resources Board (CARB) staff evaluated a limited number of statewide non-cancer health impacts associated with exposure to particulate matter (PM) 2.5 and oxides of nitrogen (NOx) emissions from heavy-duty vehicles. NOx includes nitrogen dioxide, a potent lung irritant, which can aggravate lung diseases such as asthma when inhaled (US EPA, 2016). However, the most serious quantifiable impacts of NOx emissions occur through the conversion of NOx to fine particles of ammonium nitrate aerosol through chemical processes in the atmosphere. PM2.5 formed in this manner is termed secondary PM2.5. Both directly emitted PM2.5 and secondary PM2.5 from heavy-duty vehicles are associated with adverse health outcomes, such as cardiopulmonary mortality, hospitalizations for cardiovascular illness and respiratory illness, and emergency room (ER) visits for asthma. As a result, reductions in PM2.5 and NOx emissions are associated with reductions in these health outcomes.

II. Incidence-Per-Ton Methodology

CARB uses the incidence-per-ton (IPT) methodology to quantify the health benefits of emission reductions in cases where dispersion modeling results are not available. A description of this method is included on CARB's webpage (CARB, 2021j). CARB's IPT methodology is based on a methodology developed by U.S. EPA ((Fann et al., 2009), (Fann et al., 2012), & (Fann et al., 2018)).

Under the IPT methodology, changes in emissions are approximately proportional to changes in health outcomes. IPT factors are derived by calculating the number of health outcomes associated with exposure to PM2.5 for a baseline scenario using measured ambient concentrations and dividing by the emissions of PM2.5 or a precursor. The calculation is performed separately for each air basin using the following equation:

$$IPT = \frac{number\ of\ health\ outcomes\ in\ air\ basin}{annual\ emissions\ in\ air\ basin}$$

Multiplying the emission reductions from the Proposed Regulation in an air basin by the IPT factor then yields an estimate of the reduction in health outcomes achieved by the Proposed Regulation. For future years, the number of outcomes is adjusted to account for population growth. CARB's current IPT factors are based on a 2014-2016 baseline scenario, which represents the most recent data available at the time the current IPT factors were computed. IPT factors are computed for the two types of PM2.5: primary PM2.5 and secondary PM2.5 of ammonium nitrate aerosol formed from precursors.

III. Potential Future Evaluation of Additional Health Benefits

While CARB's PM2.5 mortality and illness valuation has been, and continues to be, a useful method for valuing the health benefits of regulations, it only represents a portion of those benefits. The basis for CARB's current methodology was documented in Appendix J of the Initial Statement of Reasons for CARB's 2010 Truck and Bus Regulation (CARB, 2010). Since that was released, additional scientific evidence and updated analysis methods have become available which show more ways of evaluating the health benefits of reducing air pollution. CARB staff is reviewing this new evidence and updated methods and will consider using them to evaluate the impacts of future rulemakings. Thus, the full health benefits of the Proposed Regulation are underestimated because not all the adverse health outcomes from PM2.5 and additional pollutants (e.g., toxic air contaminant (TAC)) are evaluated and monetized. Also, CARB's current evaluation methodology does not take into account all PM2.5 precursor emissions. An expansion of the emissions inputs and an assessment for other health outcomes, including, but not limited to, additional cardiovascular and respiratory illnesses, nonfatal/fatal cancers, nervous system diseases, and lost workdays would provide a more complete picture of the benefits from reduced exposure to air pollution. In fact, in 2021, United States Environmental Protection Agency (U.S. EPA) issued a Technical Support Document (TSD) for their Cross-State Air Pollution Rule that provided both health functions and health valuation for lung cancer incidence, Alzheimer's disease, and Parkinson's disease, among other health endpoints related to PM2.5 exposures (US EPA, 2021a).

While CARB's mortality and illness valuation is just for PM2.5, there are other pollutants that can cause health issues. For instance, NOx reacts with other compounds to form ozone, which can then cause respiratory problems. Updated health impact functions and valuation for ozone are also provided in the aforementioned Cross-State Air Pollution Rule TSD provided by the U.S. EPA. Additionally, TACs emitted from diesel engines can lead to cancers.

Expanding CARB's health evaluation and valuation methodology to include any of the above additional strategies would allow the public to reach a better understanding of the benefits from reducing air pollution by moving toward cleaner combustion and zero-emission technologies. Importantly, this understanding is valuable to the successful implementation of various emission reduction strategies, including moving toward cleaner heavy-duty vehicles to protect public health.

The scientific literature has demonstrated the broad impacts of exposures to pollution, which include but go beyond the outcomes CARB staff have quantified and are thus summarized in the next section.

IV. Diesel Pollution Impacts Human Health

Diesel-powered mobile sources, including heavy-duty vehicles, emit a complex mixture of air pollutants, including diesel particulate matters (DPM) and gases. The gaseous pollutants include volatile organic compounds (VOC) and NOx, which can lead to the formation of ozone (O_3) and the secondary formation of PM (CARB, 2021l).

A. Air Toxic Impacts

DPM is a TAC composed of over 40 known cancer-causing substances and PM (CARB, 2021l). Examples of these carcinogenic chemicals include polycyclic aromatic hydrocarbons (PAH), benzene, formaldehyde, acetaldehyde, acrolein, and 1,3-butadiene (CARB, 2021l). CARB listed DPM as a TAC in 1998, due largely to its association with lung cancer (CARB, 2021l). Since CARB's listing, additional studies on the cancer-causing potential of diesel exhaust were published, which led the International Agency for Research on Cancer (IARC, a division of the World Health Organization) to classify diesel engine exhaust as "carcinogenic to humans" in 2012 ((CARB, 2021l) & (IARC, 2012)). In California, about 70 percent of known cancer risks from TACs are from diesel engine emissions ((CARB, 2021l) & (Propper et al., 2015)).

B. Particle Pollution Impact

The majority of DPM particles are PM2.5 ((CARB, 2021m) & (US EPA, 2021b)). Due to their small size, PM2.5 in air can reach the lower respiratory tract and potentially pass into the bloodstream to affect other organs ((CARB, 2021m) & (US EPA, 2021c)). By this means, PM2.5 air pollution leads not only to increased cancer risk, but it also causes respiratory and cardiovascular diseases and even premature death; adverse health outcomes from PM2.5 include asthma, chronic heart disease, and heart attack ((CARB, 2021m), (US EPA, 2021c), (WHO, 2013), & (US EPA, 2019)). Moreover, PM2.5 air pollution can result in respiratory, cardiac, and mortality effects over short time periods of exposure such as hours, days, or weeks (US EPA, 2019). Exposures to PM2.5 may also lead to myriad other health outcomes, including metabolic, nervous system, reproductive, and developmental effects (US EPA, 2019). For example, adverse health conditions with possible links to airborne PM2.5 include high blood pressure, insulin resistance, and other risk factors for Type II Diabetes, as well as psychological/cognitive problems (US EPA, 2019). PM2.5 may especially impact women and children via health effects such as pre-term birth, reduced birth weight, and abnormal lung and cardiovascular development (US EPA, 2019).

In addition to its ability to increase risk for diseases, PM2.5 is also well known to exacerbate underlying illnesses such as asthma, bronchitis, and heart disease (US EPA, 2019). As a result, the health impacts of PM2.5 are typically studied not only using cancer diagnoses and the rates of onset for lung and cardiovascular diseases, but also via metrics on respiratory symptoms (e.g., cough, wheeze, asthma medication usage), measures of abnormal lung and

heart functioning (e.g., reduced lung volume, irregular heartbeat), plus rates of hospitalizations, ER visits, and restricted activity days associated with worsening of chronic lung and heart diseases.

C. Ozone Pollution Impacts

As a gaseous pollutant from diesel-powered heavy-duty vehicles, NOx can react with other compounds to form ozone, which is the main component of smog. Based on the extent of evidence from scientific studies, the U.S. EPA has determined that short-term exposure from ozone is causally linked to adverse respiratory effects (US EPA, 2020). Ozone can cause irritation and damage lung tissue, worsen asthma and chronic illnesses including chronic obstructive pulmonary disease (COPD) and reduced lung function. For instance, a study conducted in the San Joaquin Valley showed that increased ozone pollution led to increased risk for asthma ER visits, especially for children and Black residents (Gharibi et al., 2018). Metabolic functions are also likely to be affected by short-term ozone pollution, such as those leading to increased risk for complications and hospitalizations in diabetic individuals (US EPA, 2020). And, similar to PM2.5, other potential health effects from ozone exposure include impacts on the cardiovascular, nervous, and reproductive systems, and even increased risk of mortality (US EPA, 2020).