Shipping Emissions:

Summary of Relevant Health Effects of Sulfur Oxides, Nitrogen Oxides, and Particulate Matter

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Please note:

This document by U.S. EPA has been shortened from a longer document that focuses on shipping emissions in Puerto Rico and the Virgin Islands and the value of an Emission Control Area (ECA) to control emissions.

We thought that the Health Effects Section of the report would be of particular value to you, so we have made a copy of that section for your binder.

The full document is available at:


Please note that this is a U.S. EPA government document based on their analysis of research findings. Their conclusions do not always reflect the opinions of all USC/UCLA scientists at the Moving Forward Conference (e.g., their conclusions on UFPs (ultrafine particles)).

Andrea Hricko for THE Impact Project
Proposal to Designate an Emission Control Area for Nitrogen Oxides, Sulfur Oxides and Particulate Matter

Technical Support Document
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Executive Summary

Introduction

On June 25, 2010, the United States submitted a proposal (MEPC 61/7/3) to the International Maritime Organization (IMO) to designate an Emission Control Area (ECA) for specific portions of the coastal waters around Puerto Rico and the U.S. Virgin Islands. This action would control emissions of nitrogen oxides (NOX), sulfur oxides (SOX), and particulate matter (PM) from ships. Designation of the proposed ECA would significantly reduce emissions from ships and deliver substantial benefits to the local population, as well as to marine and terrestrial ecosystems.

Also submitted to the IMO is an Information Document (MEPC 61-INF.9), which provides a complete analysis of how the proposal addresses the IMO’s approval criteria. This Technical Support Document provides additional detail on the technical analyses supporting those submissions.

Description of Population and Areas

Chapter 1 provides a description of The Commonwealth of Puerto Rico and the U.S. Virgin Islands. This includes information about geography, population and population densities, special ecological areas, and the economies of these islands, and supplements information contained in the Information Document prepared for the proposal package. The combination of people and sensitive ecosystems being located in close proximity to ports and areas of ship activity with the high levels of ship activity in this area mean that emissions from ships are contributing to ambient concentrations of air pollution and to adverse environmental impacts in Puerto Rico and the U.S. Virgin Islands.

Emission Inventory

Chapter 2 describes how U.S. emission inventories were developed to describe air emissions from ships operating in waters within the proposed ECA. These inventories provide the foundation upon which all the subsequent analyses were built, and address Criterion 6 of Section 3, Appendix III to MARPOL Annex VI. Beyond the level of detail provided in MEPC 61/7/3, Chapter 2 explains how the inputs were developed and what assumptions were made in assessing what the emissions are from ships currently (2002 base year), what the emissions would look like in 2020 without the proposed ECA, and what reductions can be expected from the proposed ECA.

Chapter 2 describes the “bottom-up” methodology that was used, based on the latest state of the art models and inputs. This chapter describes which port-related emissions were included and why, and how emissions were obtained for ships while underway in U.S. waters. This chapter explains in great detail each parameter that went into the modeling and analyses, including which ships are included, which fuels are used by those ships, which other (non-ECA) emission controls are in place for each scenario, and what growth rates are expected, incorporating forecasts of the demand for marine transportation services in 2020.
Impacts of Emissions on Human Health and the Environment

Chapter 3 describes the impact of ships’ emissions on human health and ecosystems and supports Section 5.4 of the Information Document. Chapter 3 includes a description of the pollutants proposed for control in the U.S. Caribbean ECA. The proposed ECA would not only reduce direct emissions of NOX, SOX and PM, but also secondarily formed ambient PM and ground-level ozone. Section 3.1 describes the nature of these pollutants, formation processes, and relationship to ship emissions. Section 3.2 presents the health effects associated with exposure to NOX, SOX, PM and ground-level ozone, summarizing the key scientific literature. Section 3.3 describes the impacts of emissions from ships on terrestrial and aquatic ecosystems such as acidification, nutrient enrichment, ozone uptake and visibility degradation.

Cost Analyses

Chapter 4 describes our estimates of the costs associated with the reduction of NOX, SOX, and PM emissions from ships, not only to the shipping industry but also to marine fuel suppliers and companies who rely on the shipping industry. This chapter provides additional detail regarding the analyses conducted in support of Criteria 7 and 8 of Section 3, Appendix III to MARPOL Annex VI. This chapter describes the analyses used to evaluate the cost impact of Tier III NOX requirements combined with low sulfur fuel use on vessels operating within the proposed ECA, including estimates of low sulfur fuel production costs and operating costs. This chapter also presents cost per ton estimates for ECA-based NOX and fuel sulfur standards and compares these with the costs of established land-based control programs.

Economic Impact Analysis

Chapter 5 examines the economic impacts of the projected ECA costs on shipping engaged in international trade. This chapter provides additional detail in support of Criterion 8 of Section 3, Appendix III to MARPOL Annex VI. This chapter describes the econometric methodology that was used in estimating two aspects of the economic impacts: social costs and how they are shared across stakeholders, and market impacts for the new engine and new vessel markets.
CHAPTER 3: Impacts of Shipping Emissions on Air Quality, Health and the Environment

Designation of this Emission Control Area (ECA) would significantly reduce emissions of NOX, SOX and PM2.5 and thereby reduce ambient levels of particulate matter and ground-level ozone in Puerto Rico and the U.S. Virgin Islands. The improvement in ambient air quality would result in benefits to human health and the environment. This chapter describes the pollutants that would be reduced due to the ECA designation and their impacts on human health and the environment.

3.1 Pollutants Reduced by the ECA

3.1.1 Particulate Matter

Ships that operate in the proposed ECA generate emissions that increase on-land concentrations of harmful air pollutants such as particulate matter (PM). PM is a generic term for a broad class of chemically and physically diverse substances. It can be principally characterized as discrete particles that exist in the condensed (liquid or solid) phase spanning several orders of magnitude in size. Since 1987, EPA has delineated that subset of inhalable particles small enough to penetrate to the thoracic region (including the tracheobronchial and alveolar regions) of the respiratory tract (referred to as thoracic particles). Current national ambient air quality standards (NAAQS) use PM2.5 as the indicator for fine particles (with PM2.5 referring to particles with a nominal mean aerodynamic diameter less than or equal to 2.5 µm), and use PM10 as the indicator for purposes of regulating the coarse fraction of PM10 (referred to as thoracic coarse particles or coarse-fraction particles; generally including particles with a nominal mean aerodynamic diameter greater than 2.5 µm and less than or equal to 10 µm, or PM10-2.5). Ultrafine particles (UFPs) are a subset of fine particles, generally less than 100 nanometers (0.1 µm) in aerodynamic diameter.

Particles span many sizes and shapes and consist of numerous different chemicals. Particles originate from sources and are also formed through atmospheric chemical reactions; the former are often referred to as “primary” particles, and the latter as “secondary” particles. In addition, there are also physical, non-chemical reaction mechanisms that contribute to secondary particles. Particle pollution also varies by time of year and location and is affected by several weather-related factors, such as temperature, clouds, humidity, and wind. A further layer of complexity comes from a particle’s ability to shift between solid/liquid and gaseous phases, which is influenced by concentration, meteorology, and temperature.

Fine particles are produced primarily by combustion processes and by transformations of gaseous emissions (e.g., NOX, SOX and volatile organic compounds (VOC)) in the atmosphere. The chemical and physical properties of PM2.5 may vary greatly with time, region, meteorology and source category. Thus, PM2.5 may include a complex mixture of different chemicals including sulfates, nitrates, organic compounds, elemental carbon and metal compounds. These particles can remain in the atmosphere for days to weeks and travel through the atmosphere hundreds to thousands of kilometers.1
3.1.2 Ozone

Ground-level ozone pollution is typically formed by the reaction of VOC and NOX in the lower atmosphere in the presence of sunlight. These pollutants, often referred to as ozone precursors, are emitted by many types of pollution sources such as highway and nonroad motor vehicles and engines, including ships, power plants, chemical plants, refineries, makers of consumer and commercial products, industrial facilities, and smaller area sources.

The science of ozone formation, transport, and accumulation is complex. Ground-level ozone is produced and destroyed in a cyclical set of chemical reactions, many of which are sensitive to temperature and sunlight. When ambient temperatures and sunlight levels remain high for several days and the air is relatively stagnant, ozone and its precursors can build up and result in more ozone than typically occurs on a single high-temperature day. Ozone can be transported hundreds of miles downwind of precursor emissions, resulting in elevated ozone levels even in areas with low VOC or NOX emissions.

The highest levels of ozone are produced when both VOC and NOX emissions are present in significant quantities on clear summer days. Relatively small amounts of NOX enable ozone to form rapidly when VOC levels are relatively high, but ozone production is quickly limited by removal of the NOX. Under these conditions NOX reductions are highly effective in reducing ozone while VOC reductions have little effect. Such conditions are called “NOX-limited.” Because the contribution of VOC emissions from biogenic (natural) sources to local ambient ozone concentrations can be significant, even some areas where man-made VOC emissions are relatively low can be NOX-limited.

Ozone concentrations in an area also can be lowered by the reaction of nitric oxide (NO) with ozone, forming nitrogen dioxide (NO2); as the air moves downwind and the cycle continues, the NO2 forms additional ozone. The importance of this reaction depends, in part, on the relative concentrations of NOX, VOC, and ozone, all of which change with time and location. When NOX levels are relatively high and VOC levels relatively low, NOX forms inorganic nitrates (i.e., particles) but relatively little ozone. Such conditions are called “VOC-limited.” Under these conditions, VOC reductions are effective in reducing ozone, but NOX reductions can actually increase local ozone under certain circumstances. Even in VOC-limited urban areas, NOX reductions are not expected to increase ozone levels if the NOX reductions are sufficiently large. Rural areas are usually NOX-limited, due to the relatively large amounts of biogenic VOC emissions in such areas. Urban areas can be either VOC- or NOX-limited, or a mixture of both, in which ozone levels exhibit moderate sensitivity to changes in either pollutant.

3.1.3 NO2 and SO2

Sulfur dioxide (SO2), a member of the sulfur oxide (SOX) family of gases, is formed from burning fuels containing sulfur (e.g., coal or oil), extracting gasoline from oil, or extracting metals from ore. Nitrogen dioxide (NO2) is a member of the nitrogen oxide (NOX) family of gases. Most NO2 is formed in the air through the oxidation of nitric oxide (NO) emitted when fuel is burned at a high temperature. Ships emit both NO2 and SO2. SO2 and NO2 can dissolve in water vapor and further oxidize to form sulfuric and nitric acid which reacts with ammonia to form sulfates and nitrates, both of which are important components of ambient PM. The health
effects of ambient PM are discussed in Section 3.2.1. NOx along with non-methane hydrocarbons (NMHC) are the two major precursors of ozone. The health effects of ozone are covered in Section 3.2.2.

3.1.4 Diesel Exhaust PM

Ship emissions contribute to ambient levels of air toxics known or suspected as human or animal carcinogens, or that have noncancer health effects. The population experiences an elevated risk of cancer and other noncancer health effects from exposure to air toxics. These compounds include diesel PM.

Marine diesel engines emit diesel exhaust (DE), a complex mixture comprised of carbon dioxide, oxygen, nitrogen, water vapor, carbon monoxide, nitrogen compounds, sulfur compounds and numerous low molecular-weight hydrocarbons. A number of these gaseous hydrocarbon components are individually known to be toxic including aldehydes, benzene and 1,3-butadiene. The diesel particulate matter (DPM) present in diesel exhaust consists of fine particles (< 2.5µm), including a subgroup with a large number of ultrafine particles (< 0.1 µm). These particles have a large surface area, which makes them an excellent medium for adsorbing organics, and their small size makes them highly respirable. Many of the organic compounds present in the gases and on the particles, such as polycyclic organic matter (POM), are individually known to have mutagenic and carcinogenic properties. Marine diesel engine emissions consist of a higher fraction of hydrated sulfate (approximately 60-90%) due to the higher sulfur levels of the fuel, organic carbon (approximately 15-30%), and metallic ash (approximately 7-11%) than are typically found in land-based engines. In addition, while toxic trace metals emitted by marine diesel engines represent a very small portion of the national emissions of metals (less than one percent) and are a small portion of DPM (generally much less than one percent of DPM), we note that several trace metals of potential toxicological significance and persistence in the environment are emitted by diesel engines. These trace metals include chromium, manganese, mercury, and nickel. In addition, small amounts of dioxins have been measured in highway engine diesel exhaust, some of which may partition into the particulate phase. Dioxins are a major health concern but diesel engines are a minor contributor to overall dioxin emissions.

Diesel exhaust varies significantly in chemical composition and particle sizes between different engine types (heavy-duty, light-duty), engine operating conditions (idle, accelerate, decelerate), and fuel formulations (high/low sulfur fuel). Also, there are emissions differences between on-road and nonroad engines because the nonroad engines are generally of older technology. This is especially true for marine diesel engines. After being emitted in the engine exhaust, diesel exhaust undergoes dilution as well as chemical and physical changes in the atmosphere. The lifetime for some of the compounds present in diesel exhaust ranges from hours to days.
3.2 Health Effects Associated with Exposure to Pollutants Reduced by the ECA

3.2.1 PM Health Effects

This section provides a summary of the health effects associated with exposure to ambient concentrations of PM. The information in this section is based on the information and conclusions in the Integrated Science Assessment (ISA) for Particulate Matter (December 2009) prepared by EPA’s Office of Research and Development (ORD).

The ISA concludes that ambient concentrations of PM are associated with a number of adverse health effects. The ISA characterizes the weight of evidence for different health effects associated with three PM size ranges: PM$_{2.5}$, PM$_{10-2.5}$, and UFPs. The discussion below highlights the ISA’s conclusions pertaining to these three size fractions of PM, considering variations in both short-term and long-term exposure periods.

Information specifically related to health effects associated with exposure to diesel exhaust PM is included in Section 3.2.5 of this document.

3.2.1.1 Effects Associated with Short-term Exposure to PM$_{2.5}$

The ISA concludes that cardiovascular effects and all-cause cardiovascular- and respiratory-related mortality are causally associated with short-term exposure to PM$_{2.5}$. It also concludes that respiratory effects are likely to be causally associated with short-term exposure to PM$_{2.5}$, including respiratory emergency department (ED) visits and hospital admissions for chronic obstructive pulmonary disease (COPD), respiratory infections, and asthma; and exacerbation of respiratory symptoms in asthmatic children.

3.2.1.2 Effects Associated with Long-term Exposure to PM$_{2.5}$

The ISA concludes that there are causal associations between long-term exposure to PM$_{2.5}$ and cardiovascular effects, such as the development/progression of cardiovascular disease (CVD), and premature mortality, particularly from cardiopulmonary causes. It also concludes that long-term exposure to PM$_{2.5}$ is likely to be causally associated with respiratory effects, such as reduced lung function growth, increased respiratory symptoms, and asthma development. The ISA characterizes the evidence as suggestive of a causal relationship for associations between

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\[ \text{PM}_{10-2.5} \]

\[ \text{UFPs} \]

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long-term PM$_{2.5}$ exposure and reproductive and developmental outcomes, such as low birth weight and infant mortality. It also characterizes the evidence as suggestive of a causal relationship between PM$_{2.5}$ and cancer incidence, mutagenicity, and genotoxicity.

3.2.1.3 Effects Associated with PM$_{10-2.5}$

The ISA summarizes evidence related to short-term exposure to PM$_{10-2.5}$. PM$_{10-2.5}$ is the fraction of PM$_{10}$ particles that is larger than PM$_{2.5}$. The ISA concludes that available evidence is suggestive of a causal relationship between short-term exposures to PM$_{10-2.5}$ and cardiovascular effects, such as hospitalizations for ischemic heart disease. It also concludes that the available evidence is suggestive of a causal relationship between short-term exposures to PM$_{10-2.5}$ and respiratory effects, including respiratory-related ED visits and hospitalizations and pulmonary inflammation. The ISA also concludes that the available literature suggests a causal relationship between short-term exposures to PM$_{10-2.5}$ and mortality. Data are inadequate to draw conclusions regarding health effects associated with long-term exposure to PM$_{10-2.5}$.

3.2.1.4 Effects Associated with Ultrafine Particles

The ISA concludes that the evidence is suggestive of a causal relationship between short-term exposures to ultrafine particles (UFP) and cardiovascular effects, including changes in heart rhythm and vasomotor function (the ability of blood vessels to expand and contract). The ISA also concludes that there is suggestive evidence of a causal relationship between short-term UFP exposure and respiratory effects. The types of respiratory effects examined in epidemiologic studies include respiratory symptoms and asthma hospital admissions, the results of which are not entirely consistent. There is evidence from toxicological and controlled human exposure studies that exposure to UFPs may increase lung inflammation and produce small asymptomatic changes in lung function. Data are inadequate to draw conclusions regarding health effects associated with long-term exposure to UFPs.

3.2.2 Ozone Health Effects

Exposure to ambient ozone contributes to a wide range of adverse health effects. These health effects are well documented and are critically assessed in the EPA ozone air quality criteria document (ozone AQCD) and EPA staff paper. We are relying on the data and conclusions in the ozone AQCD and staff paper, regarding the health effects associated with ozone exposure.

Ozone-related health effects include lung function decrements, respiratory symptoms, aggravation of asthma, increased hospital and emergency room visits, increased asthma medication usage, and a variety of other respiratory effects. Cellular-level effects, such as inflammation of lungs, have been documented as well. In addition, there is suggestive evidence

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$^D$ Human exposure to ozone varies over time due to changes in ambient ozone concentration and because people move between locations which have notable different ozone concentrations. Also, the amount of ozone delivered to the lung is not only influenced by the ambient concentrations but also by the individuals breathing route and rate.
of a contribution of ozone to cardiovascular-related morbidity and highly suggestive evidence that short-term ozone exposure directly or indirectly contributes to non-accidental and cardiopulmonary-related mortality, but additional research is needed to clarify the underlying mechanisms causing these effects. In a recent report on the estimation of ozone-related premature mortality published by the National Research Council (NRC), a panel of experts and reviewers concluded that short-term exposure to ambient ozone is likely to contribute to premature deaths and that ozone-related mortality should be included in estimates of the health benefits of reducing ozone exposure.\textsuperscript{14} People who appear to be more susceptible to effects associated with exposure to ozone include children, asthmatics and the elderly. Those with greater exposures to ozone, for instance due to time spent outdoors (e.g., children and outdoor workers), are also of concern.

Based on a large number of scientific studies, EPA has identified several key health effects associated with exposure to levels of ozone found today in many areas of the country. Short-term (1 to 3 hours) and prolonged exposures (6 to 8 hours) to ambient ozone concentrations have been linked to lung function decrements, respiratory symptoms, increased hospital admissions and emergency room visits for respiratory problems.\textsuperscript{15, 16, 17, 18, 19, 20} Repeated exposure to ozone can increase susceptibility to respiratory infection and lung inflammation and can aggravate preexisting respiratory diseases, such as asthma.\textsuperscript{21, 22, 23, 24, 25} Repeated exposure to sufficient concentrations of ozone can also cause inflammation of the lung, impairment of lung defense mechanisms, and possibly irreversible changes in lung structure, which over time could affect premature aging of the lungs and/or the development of chronic respiratory illnesses, such as emphysema and chronic bronchitis.\textsuperscript{26, 27, 28, 29}

Children and adults who are outdoors and active during the summer months, such as construction workers, are among those most at risk of elevated ozone exposures.\textsuperscript{30} Children and outdoor workers tend to have higher ozone exposure because they typically are active outside, working, playing and exercising, during times of day and seasons (e.g., the summer) when ozone levels are highest.\textsuperscript{31} For example, summer camp studies in the Eastern United States and Southeastern Canada have reported statistically significant reductions in lung function in children who are active outdoors.\textsuperscript{32, 33, 34, 35, 36, 37, 38, 39} Further, children are more at risk of experiencing health effects from ozone exposure than adults because their respiratory systems are still developing. These individuals (as well as people with respiratory illnesses, such as asthma, especially asthmatic children) can experience reduced lung function and increased respiratory symptoms, such as chest pain and cough, when exposed to relatively low ozone levels during prolonged periods of moderate exertion.\textsuperscript{40, 41, 42, 43}

### 3.2.3 \(\text{SO}_2\) Health Effects

This section provides an overview of the health effects associated with \(\text{SO}_2\). Additional information on the health effects of \(\text{SO}_2\) can be found in the EPA Integrated Science Assessment for Sulfur Oxides.\textsuperscript{44} Following an extensive evaluation of health evidence from epidemiologic and laboratory studies, the U.S. EPA has concluded that there is a causal relationship between respiratory health effects and short-term exposure to \(\text{SO}_2\). The immediate effect of \(\text{SO}_2\) on the respiratory system in humans is bronchoconstriction. Asthmatics are more sensitive to the effects of \(\text{SO}_2\) likely resulting from preexisting inflammation associated with this disease. In laboratory studies involving controlled human exposures to \(\text{SO}_2\), respiratory effects have consistently been
observed following 5-10 min exposures at SO2 concentrations ≥ 0.4 ppm in asthmatics engaged in moderate to heavy levels of exercise, with more limited evidence of respiratory effects among exercising asthmatics exposed to concentrations as low as 0.2-0.3 ppm. A clear concentration-response relationship has been demonstrated in these studies following exposures to SO2 at concentrations between 0.2 and 1.0 ppm, both in terms of increasing severity of respiratory symptoms and decrements in lung function, as well as the percentage of asthmatics adversely affected.

In epidemiologic studies, respiratory effects have been observed in areas where the mean 24-hour SO2 levels range from 1 to 30 ppb, with maximum 1 to 24-hour average SO2 values ranging from 12 to 75 ppb. Important new multicity studies and several other studies have found an association between 24-hour average ambient SO2 concentrations and respiratory symptoms in children, particularly those with asthma. Generally consistent associations also have been observed between ambient SO2 concentrations and emergency department visits and hospitalizations for all respiratory causes, particularly among children and older adults (≥ 65 years), and for asthma. A limited subset of epidemiologic studies have examined potential confounding by copollutants using multipollutant regression models. These analyses indicate that although copollutant adjustment has varying degrees of influence on the SO2 effect estimates, the effect of SO2 on respiratory health outcomes appears to be generally robust and independent of the effects of gaseous and particulate copollutants, suggesting that the observed effects of SO2 on respiratory endpoints occur independent of the effects of other ambient air pollutants.

Consistent associations between short-term exposure to SO2 and mortality have been observed in epidemiologic studies, with larger effect estimates reported for respiratory mortality than for cardiovascular mortality. While this finding is consistent with the demonstrated effects of SO2 on respiratory morbidity, uncertainty remains with respect to the interpretation of these associations due to potential confounding by various copollutants. The U.S. EPA has therefore concluded that the overall evidence is suggestive of a causal relationship between short-term exposure to SO2 and mortality. Significant associations between short-term exposure to SO2 and emergency department visits and hospital admissions for cardiovascular diseases have also been reported. However, these findings have been inconsistent across studies and do not provide adequate evidence to infer a causal relationship between SO2 exposure and cardiovascular morbidity.

### 3.2.4 NO2 Health Effects

Information on the health effects of NO2 can be found in the EPA Integrated Science Assessment (ISA) for Nitrogen Oxides. The EPA has concluded that the findings of epidemiologic, controlled human exposure, and animal toxicological studies provide evidence that is sufficient to infer a likely causal relationship between respiratory effects and short-term NO2 exposure. The ISA concludes that the strongest evidence for such a relationship comes from epidemiologic studies of respiratory effects including symptoms, emergency department visits, and hospital admissions. The ISA also draws two broad conclusions regarding airway responsiveness following NO2 exposure. First, the ISA concludes that NO2 exposure may enhance the sensitivity to allergen-induced decrements in lung function and increase the allergen-induced airway inflammatory response following 30-minute exposures of asthmatics to...
NO\textsubscript{2} concentrations as low as 0.26 ppm. In addition, small but significant increases in non-specific airway hyperresponsiveness were reported following 1-hour exposures of asthmatics to 0.1 ppm NO\textsubscript{2}. Second, exposure to NO\textsubscript{2} has been found to enhance the inherent responsiveness of the airway to subsequent nonspecific challenges in controlled human exposure studies of asthmatic subjects. Enhanced airway responsiveness could have important clinical implications for asthmatics since transient increases in airway responsiveness following NO\textsubscript{2} exposure have the potential to increase symptoms and worsen asthma control. Together, the epidemiologic and experimental data sets form a plausible, consistent, and coherent description of a relationship between NO\textsubscript{2} exposures and an array of adverse health effects that range from the onset of respiratory symptoms to hospital admission.

Although the weight of evidence supporting a causal relationship is somewhat less certain than that associated with respiratory morbidity, NO\textsubscript{2} has also been linked to other health endpoints. These include all-cause (nonaccidental) mortality, hospital admissions or emergency department visits for cardiovascular disease, and decrements in lung function growth associated with chronic exposure.

3.2.5 Diesel Exhaust PM Health Effects

A large number of health studies have been conducted regarding diesel exhaust. These include epidemiologic studies of lung cancer in groups of workers and animal studies focusing on non-cancer effects. Diesel exhaust PM (including the associated organic compounds which are generally high molecular weight hydrocarbons but not the more volatile gaseous hydrocarbon compounds) is generally used as a surrogate exposure measure for whole diesel exhaust.

Diesel exhaust has been found to be of concern by several groups worldwide including the U.S. government. The IPCS (International Programme on Chemical Safety) has established environmental health criteria for diesel fuel and exhaust emissions. In these criteria, the IPCS recommends that for the protection of human health diesel exhaust emissions should be controlled. The IPCS explicitly states that urgent efforts should be made to reduce emissions, specifically of particulates, by changing exhaust train techniques, engine design and fuel composition.\textsuperscript{46}

3.2.5.1 Potential Cancer Effects of Exposure to Diesel Exhaust

The U.S. EPA’s 2002 final “Health Assessment Document for Diesel Engine Exhaust” (the EPA Diesel HAD) classified exposure to diesel exhaust as likely to be carcinogenic to humans by inhalation at environmental exposures, in accordance with the revised draft 1996/1999 U.S. EPA cancer guidelines.\textsuperscript{47,48} In accordance with earlier U.S. EPA guidelines, exposure to diesel exhaust would similarly be classified as probably carcinogenic to humans (Group B1).\textsuperscript{49,50} A number of other agencies (National Institute for Occupational Safety and Health, the International Agency for Research on Cancer, the World Health Organization, California EPA, and the U.S. Department of Health and Human Services) have made similar classifications.\textsuperscript{51,52,53,54,55} The Health Effects Institute has prepared numerous studies and reports on the potential carcinogenicity of exposure to diesel exhaust.\textsuperscript{56,57,58}
More specifically, the U.S. EPA Diesel HAD states that the conclusions of the document apply to diesel exhaust in use today including both onroad and nonroad engines including marine diesel engines present on ships. The U.S. EPA Diesel HAD acknowledges that the studies were done on engines with generally older technologies and that “there have been changes in the physical and chemical composition of some DE [diesel exhaust] emissions (onroad vehicle emissions) over time, though there is no definitive information to show that the emission changes portend significant toxicological changes.” In any case, the diesel technology used for marine diesel engines typically lags that used for onroad engines, which have been subject to PM standards since 1998. Thus, it is reasonable to assume that the hazards identified from older technologies may be largely applicable to marine engines.

For the Diesel HAD, the U.S. EPA reviewed 22 epidemiologic studies on the subject of the carcinogenicity of exposure to diesel exhaust in various occupations, finding increased lung cancer risk, although not always statistically significant, in 8 out of 10 cohort studies and 10 out of 12 case-control studies which covered several industries. Relative risk for lung cancer, associated with exposure, ranged from 1.2 to 1.5, although a few studies show relative risks as high as 2.6. Additionally, the Diesel HAD also relied on two independent meta-analyses, which examined 23 and 30 occupational studies respectively, and found statistically significant increases of 1.33 to 1.47 in smoking-adjusted relative lung cancer risk associated with diesel exhaust. These meta-analyses demonstrate the effect of pooling many studies and in this case show the positive relationship between diesel exhaust exposure and lung cancer across a variety of diesel exhaust-exposed occupations.59,60,61

The U.S. EPA recently assessed air toxic emissions and their associated risk (the National-Scale Air Toxics Assessment or NATA for 1996 and 1999), and concluded that diesel exhaust ranks with other emissions that the national-scale assessment suggests pose the greatest relative risk.62,63 This national assessment estimates average population inhalation exposures to DPM for nonroad and on-highway sources. These exposures are the sum of ambient levels weighted by the amount of time people spend in each of the locations.

In summary, the likely hazard to humans together with the potential for significant environmental risks leads us to conclude that diesel exhaust emissions from marine engines present public health issues of concern.

3.2.5.2 Other Health Effects of Exposure to Diesel Exhaust

Noncancer health effects of acute and chronic exposure to diesel exhaust emissions are also of concern. The Diesel HAD established an inhalation Reference Concentration (RfC) specifically based on animal studies of diesel exhaust exposure. An RfC is defined by the U.S. EPA as “an estimate of a continuous inhalation exposure to the human population, including sensitive subgroups, with uncertainty spanning perhaps an order of magnitude, which is likely to be without appreciable risks of deleterious noncancer effects during a lifetime.” The U.S. EPA derived the RfC from consideration of four well-conducted chronic rat inhalation studies showing adverse pulmonary effects.64,65,66,67 The diesel RfC is based on a “no observable adverse effect” level of 144 µg/m³ that is further reduced by applying uncertainty factors of 3 for interspecies extrapolation and 10 for human variations in sensitivity. The resulting RfC derived in the Diesel HAD is 5 µg/m³ for diesel exhaust, as measured by DPM. This RfC does
not consider allergenic effects such as those associated with asthma or immunologic effects. There is growing evidence that exposure to diesel exhaust can exacerbate these effects, but the exposure-response data is presently lacking to derive an RfC. The Diesel HAD states, “With DPM [diesel particulate matter] being a ubiquitous component of ambient PM, there is an uncertainty about the adequacy of the existing DE [diesel exhaust] noncancer database to identify all of the pertinent DE-caused noncancer health hazards” (p. 9-19).

While there have been relatively few human studies associated specifically with the noncancer impact of exposure to DPM alone, DPM is a component of the ambient particles studied in numerous epidemiologic studies. The conclusion that health effects associated with ambient PM in general are relevant to DPM is supported by studies that specifically associate observable human noncancer health effects with exposure to DPM. As described in the Diesel HAD, these studies identified some of the same health effects reported for ambient PM, such as respiratory symptoms (cough, labored breathing, chest tightness, wheezing), and chronic respiratory disease (cough, phlegm, chronic bronchitis and suggestive evidence for decreases in pulmonary function). Symptoms of immunological effects such as wheezing and increased allergenicity are also seen. Studies in rodents, especially rats, show the potential for human inflammatory effects in the lung and consequential lung tissue damage from chronic diesel exhaust inhalation exposure. The Diesel HAD concludes “that acute exposure to DE [diesel exhaust] has been associated with irritation of the eye, nose, and throat, respiratory symptoms (cough and phlegm), and neurophysiological symptoms such as headache, lightheadedness, nausea, vomiting, and numbness or tingling of the extremities.” There is also evidence for an immunologic effect such as the exacerbation of allergic responses to known allergens and asthma-like symptoms.

The Diesel HAD briefly summarizes health effects associated with ambient PM and discusses the PM2.5 NAAQS. There is a much more extensive body of human data, which is also mentioned earlier in the health effects discussion for PM2.5 (Section 3.2.1.1 of this document), showing a wide spectrum of adverse health effects associated with exposure to ambient PM, of which diesel exhaust is an important component. The PM2.5 NAAQS is designed to provide protection from the non-cancer and premature mortality effects of PM2.5 as a whole.

3.2.5.3 Exposure to Diesel Exhaust PM

Exposure of people to diesel exhaust depends on their various activities, the time spent in those activities, the locations where these activities occur, and the levels of diesel exhaust pollutants in those locations. The major difference between ambient levels of diesel particulate and exposure levels for diesel particulate is that exposure levels account for a person moving from location to location, the proximity to the emission source, and whether the exposure occurs in an enclosed environment.

Occupational exposures to diesel exhaust from mobile sources, including marine diesel engines, can be several orders of magnitude greater than typical exposures in the non-occupationally exposed population. Over the years, diesel particulate exposures have been measured for a number of occupational groups resulting in a wide range of exposures from 2 to 1280 µg/m³ for a variety of occupations. As discussed in the Diesel HAD, the National Institute of Occupational Safety and Health (NIOSH) has estimated a total of 1,400,000 workers are
3.2.5.3.1 Elevated Concentrations and Ambient Exposures in Mobile Source-Impacted Areas

While occupational studies indicate that those working in closest proximity to diesel exhaust experience the greatest health effects, recent studies are showing that human populations living near large diesel emission sources such as major roadways, rail yards, and marine ports are also likely to experience greater exposure to PM and other components of diesel exhaust than the overall population, putting them at a greater health risk. The percentage of total port emissions that come from ships varies by port. However, ships contribute to the DPM concentrations at ports, and elsewhere, which influence exposures.

Regions immediately downwind of marine ports may experience elevated ambient concentrations of directly-emitted PM$_{2.5}$ from diesel engines. Due to the nature of marine ports, emissions from a large number of diesel engines are concentrated in a small area. Recent studies conducted in the continental United States have looked at air quality impacts of diesel engine emissions from ports. Although this proposed ECA is for Puerto Rico and the U.S. Virgin Islands, the contribution from ports to elevated ambient concentrations of diesel exhaust in populated areas on the U.S. mainland is relevant since there are also ports near populated areas of Puerto Rico and the U.S. Virgin Islands.

A study from the California Air Resources Board (CARB) evaluated air quality impacts of diesel engine emissions within the Port of Long Beach and Los Angeles in California, one of the largest ports in the U.S. The port study employed the ISCST3 dispersion model. With local meteorological data used in the modeling, annual average concentrations of DPM were substantially elevated over an area exceeding 200,000 acres. Because the Ports are located near heavily-populated areas, the modeling indicated that over 700,000 people lived in areas with at least 0.3 µg/m$^3$ of port-related DPM in ambient air, about 360,000 people lived in areas with at least 0.6 µg/m$^3$ of DPM, and about 50,000 people lived in areas with at least 1.5 µg/m$^3$ of ambient DPM emitted directly from the port. This port study highlights the substantial contribution these facilities make to ambient concentrations of DPM in large, densely populated areas.

The U.S. EPA updated an initial screening-level analysis of selected marine port areas to better understand the populations that are exposed to diesel particulate matter (DPM) emissions from these facilities. The results of this study are summarized here and are also available in the public docket. In summary, the screening-level analysis found that for the 45 U.S. marine ports studied, at least 18 million people live in the vicinity of these facilities and are exposed to ambient DPM levels from all port emission sources that are at least 0.2 µg/m$^3$ above those found in areas further from these facilities. If only Category 3 engine DPM emissions are considered, then the number of people exposed is 6.5 million.

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This type of screening-level analysis is an inexact tool and not appropriate for regulatory decision-making; it is useful in beginning to understand potential impacts and for illustrative purposes.


U.S. EPA (2002). National-Scale Air Toxics Assessment for 1996. This material is available electronically at http://www.epa.gov/tnn/atw/nata/.


