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October 6, 2017

Gregory Aker, Esq.
Burke Williams & Sorensen LLP
1901 Harrison Street #900
Oakland, CA 94612

RE: Expert Report of H. Nadia Moore, Ph.D., DABT, ERT in the matter of Oakland Bulk & Oversized Terminal, LLC v. City of Oakland, Sierra Club and San Francisco Baykeeper. Case No. 3.16-cv-07014-VC

Dear Mr. Aker:

I was asked to provide expert opinions regarding potential adverse health effects that would be anticipated in the West Oakland community upon installation and operation of the proposed Oakland Bulk and Oversized Terminal (OBOT) facility for rail-based transport, transfer, and shiploading of coal.

Summary of primary opinions

- **Opinion 1: The transport, storage, and handling of coal through the City and Port of Oakland poses an increased risk of adverse health effects to West Oakland residents, including premature mortality, increased hospital admissions and emergency department visits, and development of chronic respiratory disease** (page 19).

    - Additional health risks associated with ambient air pollution from coal activities include nonfatal heart attacks and strokes, irregular heartbeat, increased risk of heart disease and lung cancer, aggravated asthma, decreased lung function, reduced lung development and the development of chronic respiratory diseases (e.g., asthma) in children, and increased respiratory symptoms such as irritation of the airways, coughing, or difficulty breathing.

- West Oakland has been specifically identified as an at-risk community for the adverse health effects from particulate matter (PM) air pollution for over a decade. The community includes many daycare centers, preschools, and an increased elderly population, which is important because the most vulnerable populations to the effects of PM air pollution are children, the elderly, and asthmatics.

- The San Francisco Bay Area, which includes West Oakland, has historically poor air quality and employs active measures to improve air quality and reduce health risks posed by airborne $PM_{2.5}$ (particulate matter whose median diameter is 2.5 μm or smaller). Introduction of a significant *new* source of $PM_{2.5}$ would be directly contrary to Oakland's and the Port's goals of *reducing* airborne pollutants in the West Oakland area.

- Coal transport by rail releases $PM_{2.5}$ into the ambient air and adversely effects ambient air quality.

- It is generally accepted within the scientific community that increased concentrations of $PM_{2.5}$ pollution are associated with adverse health outcomes among exposed populations, including premature mortality, increased hospital admissions and emergency department visits, and development of chronic respiratory disease.

- **Opinion 2: Modeling results provide added support that transport, storage, and handling of coal through the City and Port of Oakland poses an increased risk of adverse health effects to West Oakland residents, including premature mortality, increased hospital admissions and emergency department visits, and development of chronic respiratory disease** (page 34).

    - Modeled coal-associated $PM_{2.5}$ concentrations cause West Oakland's ambient air quality to exceed National (NAAQS) and State (CAAQS) air quality standards.

- **Opinion 3: Smoke constituents from a coal fire will expose the West Oakland community to adverse health risks, including increased premature mortality and hospital admissions for cardiovascular and respiratory diseases** (page 38).

    - Coal fires generate and release hazardous air pollutants and other pollutants, including several known carcinogens.

- Exposure to constituents released into the West Oakland community air during a coal fire will introduce health hazards into the community, including increased risk of hospital admissions and emergency department visits, cancer, and premature mortality; adverse cardiovascular, dermal (skin), developmental, kidney, liver and respiratory tract effects; and adverse effects to the hematological (blood), immune, nervous, and reproductive systems.

- In the event of a coal fire, West Oakland's $PM_{2.5}$ levels will sharply rise, far exceeding National (NAAQS) standards.

Report organization

My report is organized into several sections including a background section (describing adverse health effects from air pollution, air quality regulations and standards, and concepts regarding particulate matter) followed by my opinions regarding rail-based coal transport operations through Oakland. My opinions are held to a reasonable degree of scientific certainty. A table of contents for the report is provided as Table 1.

Table 1. Contents

Qualifications

My background, training, and professional experience make me well qualified to discuss the evolution of the state of knowledge as it pertains to setting exposure limit values and evaluating whether harm would be likely from exposures.

I am certified in toxicology as a Diplomate of the American Board of Toxicology and am admitted to both the United Kingdom and EUROTOX registries as a European Registered Toxicologist. I am a member of the Society of Toxicology, American College of Toxicology, British Toxicology Society, American College of Occupational and Environmental Medicine, American Association for the Advancement of Science, American Conference of Governmental Industrial Hygienists, American Chemical Society, American Industrial Hygiene Association, and Society for Experimental Biology and Medicine. I currently serve as Councilor for the Women in Toxicology Specialty Interest Group for the Society of Toxicology and as Vice-President for the Pacific Northwest Association of Toxicologists

(PANWAT). Previously I served as Councilor for the Pacific Northwest Association of Toxicologists. I received a Bachelor of Science degree in Chemistry with a biochemistry emphasis from Pacific Lutheran University in 1992 and a Ph.D. in Environmental Toxicology from the University of Washington, School of Public Health and Community Medicine, Department of Environmental Health and Occupational Health Sciences in 2008.

Following completion of my undergraduate degree, I joined an analytical chemistry group at Battelle Memorial Institute ("Battelle") primarily supporting laboratory animal inhalation studies. During my nine-year tenure, I was responsible for method development, validation, and implementation of methods to determine test article purity and test article concentration within exposure atmospheres. I was also responsible for developing methods to quantitate test articles and/or metabolite concentrations in biological samples for governmental (National Toxicology Program) and industrial (e.g., pharmaceutical) clients. In 2001, I transferred to a health protection group and prepared risk-based toxicology reports for single compounds, chemical classes, and complex mixtures until 2003, when I successfully applied for and was granted an educational leave to pursue graduate study in toxicology. My dissertation at the University of Washington focused on mechanisms underlying the developmental neurotoxicity of ethanol.

I returned to Battelle's inhalation laboratory after obtaining my Ph.D. as a toxicologist and study director. In this role my responsibilities included study design; scientific, technical, and procedural oversight of all study phases; and the overall conduct, interpretation, and reporting of studies, including review and evaluation of all scientific literature and data available for inclusion in study design or result interpretation. In addition, I consulted with Battelle's industrial hygienist to derive and/or establish acceptable exposure levels for staff working with or near studied compounds, which were primarily selected for testing due to a lack of available inhalation toxicity data.

In January of 2011, the Chief Executive Officer of Battelle Memorial Institute, Jeffrey Wadsworth, appointed me to the Institutional Animal Care and Use Committee (IACUC) serving Pacific Northwest National Laboratory, Sequim Laboratory, and the Columbus-Based Toxicology Laboratory (ToxNW) as a practicing scientist member. I worked closely with other committee members (a veterinarian, other practicing scientists involved in animal research, scientists not involved in animal research, and a community member not affiliated with Battelle) to review all animal-use protocols prior to implementation. We were responsible for oversight of animal care and use as required by law and ensured study design protocols met all criteria prior to issuance of IACUC approval. In addition, we inspected, monitored, and evaluated ongoing animal care and use and addressed animal welfare concerns.

My responsibilities grew again in November 2011 when I was approved by the National Toxicology Program (NTP) as the Toxicology Discipline Leader for inhalation studies performed at Battelle. In this role, I provided leadership for all of NTP's research and testing efforts in the characterization of toxicological and carcinogenic potential of chemicals, mixtures, and agents.

In the fall of 2013, I transitioned from Battelle to Veritox, Inc., a health-based consulting company focusing on toxicology and industrial hygiene. Currently I am a Senior Toxicologist at Veritox, Inc.

I have attached a true and correct copy of my *curriculum vitae* to this report as Attachment 1. I have not testified as an expert in connection with any court proceeding during the past four years.

Veritox, Inc. charges $260 per hour for my time to consult in this matter and charges $500 per hour (with a one-hour minimum) for deposition and trial testimony.

Basis of opinions

The basis for my opinions in this case includes my education; training in basic science; experience in toxicology; experience in inhalation toxicology; experience in complex risk-based toxicology evaluations; general knowledge of the adverse effects of chemicals on mammalian species including humans; and review of case-specific materials provided to me. My training, experience, and study of the published literature provide me with an in-depth knowledge of occupational and public health.

Records received

I received the following records:

- First Amended Complaint, 6/14/17
- Complaint Under Title VI of the Civil Rights Act of 1964, 42 U.S.C. § 2000d
- Stipulated Protective Order with Exhibit A, 7/5/17
- Paul B. English, Ph.D., MPH, Report on Public Health Impacts of Coal Exports at the Former Oakland Army Base, 9/14/15 [OAK 0006-7]

- Phyllis Fox, Ph.D., PE, Report on Environmental, Health and Safety Impacts of the Proposed Oakland Bulk and Oversized Terminal, Prepared for Sierra Club, 9/21/15 [OAK 1702-24]

- Sustainable Systems Research, LLC, Report on Technical Memorandum Air Quality, Climate Change, and Environmental Justice Issues from Oakland Trade and Global Logistics Center, Prepared for EarthJustice, with Appendix A-B, 9/18/15 [OAK 1726-50]

- Golder Associates, Peer Review of HDR Report for California Capital & Investment Group, 10/4/15 [OAK 1905-11]

- HDR, Report on Oakland Bulk and Oversized Terminal Air Quality & Human Health and Safety Assessment of Potential Coal Dust Emissions, Prepared for California Capital and Investment Group, with HDR Engineering Credentials and Attachment 1, 9/15

- ESA, Report on the Health and/or Safety Impacts Associated with the Transport, Storage, and/or Handling of Coal and/or Coke in Oakland, including at the Proposed Oakland Bulk and Oversized Terminal in the West Gateway Area of the Former Oakland Army Base, Prepared for City of Oakland, 6/23/16

- Cardno, Peer Review Report, Preliminary Engineering – Oakland Bulk and Oversized Terminal, Prepared for California Capital Investment Group, 10/6/15 [OB075973-96]

- Northgate Environmental Management, Inc., Memo re Special Air Quality Study, Oakland Army Base Redevelopment Project, Oakland, California, 9/8/15 [OB077916-78108]

- Andrew Gray of Gray Sky Solutions, $PM_{2.5}$ modeling for North and South rails, annual and 24 hr average-98th percentile, $PM_{2.5}$ modeling for fire scenarios

- Carlos Fernandez-Pello, PhD of REAX Engineering Expert Report, Fire and Explosion Safety at the Proposed OBOT Facility, 10/6/17, with Appendices 1-4 and CV

- Environmental Defense Fund (EDF), Article on Pollution and Health Concerns in West Oakland, 2017

- Moving Forward Network, Article on West Oakland Environmental Indicators Project research reveals dangerous pollution hotspots, 6/10/17

- California EPA Air Resources Board Stationary Source Division, Draft Health Risk Assessment for the Union Pacific Railroad Oakland Railyard, 3/19/08
- California EPA Air Resources Board on West Oakland Study, 7/8/09
- EDF, Mapping Air Pollution with New Mobile Sensors, 2017
- KALW, Mapping West Oakland Pollution, Block by Block, 8/14/17
- Bowie Resource Partners LLC, Oakland Terminal Presentation to The Union Pacific, 7/17/14 [OB057454-91]
- Email from Mark McClure of California Capital & Investment Group to Megan Morodomi, re UP Presentation, 7/15/14
- A. Jha and N.Z. Muller, Handle with Care: The Local Air Pollution Costs of Coal Storage, a NBER Working Paper Series, , National Bureau of Economic Research, 5/17
- TLS Operating Plan Framework, 6/19/15 (V2 CCIG Edits 7/14/15) [OB082060-83]
- Alt Daily, Article on Op-ed: Is Norfolk Southern Corporation Poisoning Us for Profits?, 5/6/15
- Sightline Institute, Article on What Coal Dust Looks Like in Alaska – Photos taken nears a coal terminal in Seward., 1/25/12
- OPB, Article on What Coal-Train Dust Means for Human Health, 3/10/13
- Cliff Mass Weather and Climate Blog, Article on Strong Winds, Coal Dust, and the Proposed Gateway Pacific Coal Terminal, 2/26/15
- Hawaii News Now, Article on Power Plant's Ash Dust Triggers Environmental Concerns, 1/12/17

Background: Adverse health effects from exposure to particulate matter in ambient air

Toxicology is the study of adverse effects of chemical, biological, or physical agents on living organisms or ecosystems, including the prevention and amelioration of such adverse effects.[1] Toxicologists use scientific studies and data to determine types of hazard(s) (e.g., adverse effects including exacerbation of asthma, cancer, birth defects, and death) for chemical, biological, or physical agent exposure and exposure scenarios (concentration, route, and duration) associated with risk of adverse effects. The sections below explain the adverse health effects associated with ambient exposure to particulate matter pollution.

Adverse health effects associated with poor air quality

Adverse health effects associated with poor air quality have been known for centuries. Although effects were noted prior to the industrial revolution (e.g., Evelyn 1661),[2] most legislation limiting air pollution did not occur until extended, sustained periods of extremely poor air quality were associated with extra human deaths (e.g., Meuse Valley of Belgium in 1930; Donora, Pennsylvania, in 1948; and London's Great Smog of 1952).[3] These reports were the first in the modern era to clearly link hazards associated with air pollution exposure to adverse human health effects. In these instances, the extremely poor air quality was attributed to the combination of the industrial coal combustion, automobile emissions, and stagnant air due to weather inversions in each region.

Poor ambient air quality continues to adversely impact health through increasing the burden of disease from stroke, heart disease, lung cancer, and both chronic and acute respiratory diseases, including asthma. Worldwide, approximately 3 million premature deaths were attributed to ambient air pollution in 2012.[4]

[1] Hayes, A.W. Principles and Methods of Toxicology. 6th ed. Boca Raton: CRC Press: Taylor & Francis. 2014., p.597.
[2] Evelyn, J. (1661). Fumifugium: or the inconvenience of the air and smoke of London dissipated. In: Ballantyne, B., *et al.* General and Applied Toxicology. 3rd ed.: John Wiley and Sons, Ltd. 2009. p.2058.
[3] Klaassen, C.D. Casarett and Doull's Toxicology: The Basic Science of Poisons. 8th ed. New York: McGraw-Hill; 2013., p.1233.
[4] WHO. Ambient air pollution: A global assessment of exposure and burden of disease. Geneva, Switzerland. 2016. p.40.

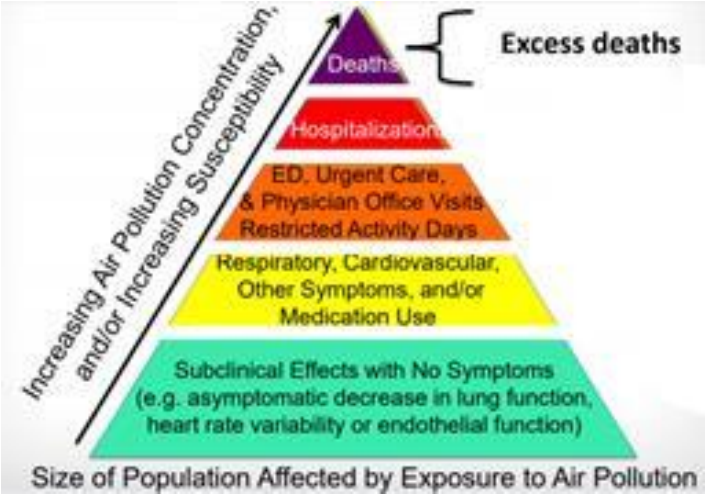

Figure 1. US EPA's adverse health effects pyramid for air pollution exposure (adapted from Cascio, 2016)[5]

As depicted in Figure 1, the range of adverse health effects among the population associated with air pollution is frequently presented as a pyramid, with the least severe effect (lung function decrements, inflammation, and cardiac effects), affecting the largest population, comprising its base. As each severity category increases, the affected population decreases until death, at the apex, represents the smallest number affected.[5]

Air quality regulations and standards

In the United States, the first regulations of air pollutants arose in the early 1970s, when the United States Environmental Protection Agency (US EPA) was charged with protecting the public from the hazards of outdoor air pollution through regulation of six criteria air pollutants, which were identified as significant public health hazards (carbon monoxide, nitrogen dioxide, sulfur dioxide, total suspended particulate matter, hydrocarbons, and photochemical oxidants).[6] To protect public health, including the health of at-risk (sensitive) populations, the US EPA established National Ambient Air Quality Standards (NAAQS) as the maximum permissible ambient air concentration for each criteria pollutant that will protect the health of any [sensitive] group of the population.[7] Periodic reviews of

[5] Cascio, W. Healthy Heart: a healthier environment for healthier hearts. ORD Tools and Resources Webinar. US EPA National Health and Environmental Effects Research Laboratory, February 17, 2016.

[6] US EPA. Clean Air Act Requirements and History. Last updated January 10, 2017. Available from: https://www.epa.gov/clean-air-act-overview/clean-air-act-requirements-and-history. Accessed: 9/12/2017. ; US EPA. National primary and secondary ambient air quality standards. 42 CFR Part 10. Federal Register. 1971; 36(84):8186-201.

[7] US EPA. National ambient air quality standards for particulate matter. Final Rule. 40 CFR Parts 50, 51, 52, 53, and 58. Federal Register. 2013; 78(10):3086-287.

standards combined with advances in scientific knowledge of health effects and air pollution composition during the next four decades resulted in revisions to the list of criteria air pollutants. Photochemical oxidants were later regulated as ozone; total suspended particulate matter was later regulated by PM_{10} (≤10 μm particulate matter) and $PM_{2.5}$ (≤2.5 μm particulate matter); total hydrocarbons were removed; and lead was added. Today, the criteria air pollutants are PM (PM_{10}, $PM_{2.5}$), lead, carbon monoxide, nitrogen dioxide, sulfur dioxide, and ozone.[8]

The NAAQS include two types of standards: primary and secondary. Primary standards are intended to provide protection to the most sensitive subpopulations whereas secondary standards provide public welfare protection (e.g., protection against decreased visibility and damage to animals, crops, vegetation, and buildings).[9] Because the focus of my report is potential human health impacts, no further discussion is included regarding secondary standards; my report focuses on primary standards.

The State of California also promulgates its own ambient air quality standards, which are established by the California Air Resources Board (CARB) as "outdoor pollutant levels considered safe for the public."[10]

Both the US EPA and CARB utilize the terms "attainment" and "nonattainment" to denote geographical areas whose pollutant concentrations are within or in excess of ambient air quality standards, respectively.[11]

Particulate matter (PM)

Particulate matter (PM) in the air is a heterogeneous mixture of suspended solid particles, homogeneous or heterogeneous agglomerates, biological materials (fungi, mold spores, pollen), and liquid aerosols. Airborne PM arises from both natural and manmade sources, although many of the historical examples of extremely poor air situations referenced above were associated with coal combustion and automobile emissions.

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[8] US EPA. Natitional Ambient Air Quality Standards - NAAQS Table. Last updated December 20, 2016. Available from: https://www.epa.gov/criteria-air-pollutants/naaqs-table. Accessed: 8/17/17.

[9] US EPA. Natitional Ambient Air Quality Standards - NAAQS Table. Last updated December 20, 2016. Available from: https://www.epa.gov/criteria-air-pollutants/naaqs-table. Accessed: 8/17/17.

[10] California Air Resources Board. Air Quality Standards and Area Designations. California Environmental Protection Agency; Air Resources Board; Last updated May 5, 2016. Available from: https://www.arb.ca.gov/desig/desig.htm. Accessed: 9/12/2017.

[11] California Air Resources Board. Air Quality Standards and Area Designations. California Environmental Protection Agency; Air Resources Board; Last updated May 5, 2016. Available from: https://www.arb.ca.gov/desig/desig.htm. Accessed: 9/12/2017. ; US EPA. National ambient air quality standards for particulate matter. Final Rule. 40 CFR Parts 50, 51, 52, 53, and 58. Federal Register. 2013; 78(10):3086-287.

Airborne PM consists of a vast array of differently sized particles. PM size categories correlate to the mass median aerodynamic diameter (MMAD) of the particles: PM_{10} corresponds to particles with MMADs less than or equal to 10 micrometers (µm; equivalent to 0.00001 meters, 0.001 cm, or 0.010 mm) and $PM_{2.5}$ corresponds to particles with MMADs less than or equal to 2.5 µm. Thus by definition, $PM_{2.5}$ is a subgroup of PM_{10}. Diesel PM is a subset of PM particles emitted from diesel engines. More than 90% of diesel PM is less than 1 µm in diameter and thus diesel PM is a subgroup of $PM_{2.5}$.[12]

PM particle size is biologically important because size influences where each particle is deposited within the respiratory tract. As humans breathe, inhaled air travels in through the nasal passages, then through progressively smaller conducting airways (trachea, bronchi, smaller bronchi, and bronchioles), and finally into the gas exchange region of the lungs (alveoli). As the air travels deeper into the lungs, airflow decreases as the airways narrow, split, and increase in number.[13] Airway architecture combines with particle deposition mechanisms to generate consistent deposition patterns for differently sized particles. The largest PM_{10} particles are deposited in the upper airways (nasal cavity, trachea); progressively smaller particles penetrate deeper in the respiratory tract such that only particles with diameters <5µm reach the alveolar (gas exchange) region.[14]

Therefore, PM_{10} represents particles that may deposit in the lung and $PM_{2.5}$ represents particles that are likely deposited in the alveolar zone (where they may hinder gas-exchange function and/or may be directly absorbed into the bloodstream to exert potential systemic effects). $PM_{2.5}$ was added as a criteria air pollutant to specifically represent the concentration of particles that penetrate more deeply into the lung.[15]

My opinions focus specifically on $PM_{2.5}$ because:

- Transport of coal by rail increases ambient $PM_{2.5}$ levels in the surrounding community;[16] and

[12] California Air Resources Board. Overview: Diesel exhaust and health. Last updated April 12, 2016. Available from: https://www.arb.ca.gov/research/diesel/diesel-health.htm. Accessed: October 3, 2017.

[13] Silverthorn, D.U., *et al.* Mechanics of Breathing. In: Human Physiology: An Integrated Approach. 3rd ed. San Francisco: Pearson; 2004. p. 546-73.

[14] Maynard, R.L. Air Pollution. In: General and Applied Toxicology. 3rd ed. Ballantyne, B., Marrs, T.C., Syversen, T., editors. West Sussex, UK: John Wiley & Sons; 2009. p. 2057-99.

[15] US EPA. Integrated Risk Information System (IRIS) Chemical Assessment Summary: Formaldehyde; CASRN 50-00-0. October 1, 1989., p.702; Maynard, R.L. Air Pollution. In: General and Applied Toxicology. 3rd ed. Ballantyne, B., Marrs, T.C., Syversen, T., editors. West Sussex, UK: John Wiley & Sons; 2009. p. 2057-99.

[16] Jaffe, D., *et al.* Diesel particulate matter and coal dust from trains in the Columbia River Gorge, Washington State, USA. Atmospheric Pollution Research. 2015; 6(6):946-52.

- As detailed in the next section, increased $PM_{2.5}$ pollution levels are associated with adverse health outcomes among exposed populations.

Health effects of $PM_{2.5}$

It is generally accepted within the scientific community that increased concentrations of $PM_{2.5}$ pollution are associated with adverse health outcomes among exposed populations, including premature mortality, increased hospital admissions and emergency department visits, and development of chronic respiratory disease.

- The World Health Organization (WHO) concluded the evidence of airborne PM exposure on public health consistently shows adverse respiratory and cardiovascular health effects whose risk increases with exposure, with little evidence to suggest a threshold below which no adverse health effects are anticipated. Therefore, they concluded current scientific evidence does not support a guideline value to offer complete protection against adverse health effects of PM. Instead, the standard-setting process is consistent with the lowest concentrations possible in the context of local constraints, capabilities, and public health priorities.[17]
- US EPA concluded that health effects associated with long- and short-term exposure to $PM_{2.5}$ include premature mortality, increased hospital admissions and emergency department visits, and development of chronic respiratory disease.[18]
- The State of California concluded long-term exposure to $PM_{2.5}$ caused excess deaths and illnesses including respiratory symptoms, asthma exacerbation, and hospital admissions for cardiac and respiratory diseases.[19]

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[17] Samet, J.M., *et al.* Particulate Matter. In: Air Quality Guidelines Global Update 2005: Particulate matter, ozone, nitrogen dioxide and sulfur dioxide World Health Organization. Germany: World Health Organization; Druckpartner Moser; 2006. p. 217-91.

[18] US EPA. National ambient air quality standards for particulate matter. Final Rule. 40 CFR Parts 50, 51, 52, 53, and 58. Federal Register. 2013; 78(10):3086-287.; US EPA. Integrated Science Assessment for Particulate Matter. Research Triangle Park, NC. Report No.: EPA/600/R-08/139F December 2009. Includes errata sheet created on 2/10/2010.; US EPA. Policy Assessment for the Review of the Particulate Matter National Ambient Air Quality Standards. Report No.: EPA 452/R-11-003 April 2011.; US EPA. Provisional assessment of recent studies on health effects of particulate matter exposure. Research Triangle Park, NC. National Center for Enviromental Assessment-RTP Division; Office of Research and Development; US EPA, Report No.: EPA/600/R-12/056F December 2012.; US EPA. Quantitative Health Risk Assessment for Particulate Matter. Research Triangle Park, NC. Report No.: EPA-452/R-10-005 June 2010.

[19] 17 CCR § 70200. Ambient Air Quality Standards: Table of Standards. September 2017.

- $PM_{2.5}$ exposure contributes to cardiovascular morbidity and mortality.[20]

Exposure to particle pollution can increase illness incidence, hospitalization admissions, emergency room visits, rates of school absenteeism, and premature death.[21] $PM_{2.5}$ exposure has been associated with:

- Premature death,
- Nonfatal heart attacks and strokes,
- Irregular heartbeat,
- Increased risk of heart disease and lung cancer,
- Aggravated asthma,
- Decreased lung function,
- Reduced lung development and the development of chronic respiratory diseases (e.g., asthma) in children, and
- Increased respiratory symptoms such as irritation of the airways, coughing, or difficulty breathing.[22]

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[20] Brook, R.D., *et al.* Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association. Circulation. 2010; 121(21):2331-78.

[21] American Lung Association. State of the Air 2017. Chicago, IL., p.36-37.

[22] American Lung Association. State of the Air 2017. Chicago, IL., p.35; Bell, M.L., *et al.* Seasonal and regional short-term effects of fine particles on hospital admissions in 202 US counties, 1999-2005. Am J Epidemiol. 2008; 168(11):1301-10.; Delfino, R.J., *et al.* Asthma morbidity and ambient air pollution: effect modification by residential traffic-related air pollution. Epidemiology. 2014; 25(1):48-57.; Dominici, F., *et al.* Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. Jama. 2006; 295(10):1127-34.; Krewski, D., *et al.* Reanalysis of the Harvard six cities study and the American Cancer Society study of particulate air pollution and mortality. Cambridge, MA. Contract No.: A Special Report of the Institute's Particle Epidemiology Reanalysis Project July 2000.; Krewski, D., *et al.* Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. Boston, MA. Contract No.: HEI Research Report 140 May 2009.; Laden, F., *et al.* Reduction in fine particulate air pollution and mortality: extended follow-up of the Harvard six cities study. Am J Respir Crit Care Med. 2006; 173(6):667-72.; Moolgavkar, S.H. Air pollution and daily deaths and hospital admissions in Los Angeles and Cook Counties. In: HEI Special Report Revised Analyses of Time-Series Studies of Air Pollution and Health Revised Analyses of the National Morbidity, Mortality, and Air Pollution Study, Part II Revised Analyses of Selected Time-Series Studies. Boston, MA: Health Effects Institute; 2003. p. 183-98.; Metzger, K.B., *et al.* Ambient air pollution and cardiovascular emergency department visits. Epidemiology. 2004; 15(1):46-56.; Ostro, B., *et al.* The effects of fine particle components on respiratory hospital admissions in children. Environ Health Perspect. 2009; 117(3):475-80.;Pope, C.A., III, *et al.* Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA. 2002; 287(9):1132-41.; Pope, C.A., III and Dockery, D.W. Health effects of fine particulate air pollution: lines that connect. J Air Waste Manag Assoc. 2006; 56(6):709-42.; Shi, L., *et al.* Low-Concentration PM2.5 and Mortality: Estimating Acute and Chronic Effects in a Population-Based Study. Environ Health Perspect. 2016; 124(1):46-52.; US EPA. Particulate Matter (PM) Pollution: Health and Environmental Effects of Particulate Matter (PM). Last updated July 1, 2016. Available from: https://www.epa.gov/pm-pollution/health-and-environmental-effects-particulate-matter-pm. Accessed: 9/21/2017. ;

$PM_{2.5}$ air quality standards

The US EPA established the NAAQS for $PM_{2.5}$ to provide increased protection against health effects associated with short- and long-term exposures. Specifically, the US EPA's action provides increased protection for at-risk (sensitive) populations including children, older adults, and persons with pre-existing heart and lung disease against an array of $PM_{2.5}$-related adverse health effects including premature mortality, increased hospital admissions and emergency department visits, and development of chronic respiratory disease.[23]

Because $PM_{2.5}$ exposure is ubiquitous to our environment, the World Health Organization indicated the impracticality of identification of either non-exposed groups or a true threshold concentration for adverse effects. Instead, they establish air quality guidelines to protect the public against adverse health effects based on the collective scientific dataset for the increased risk(s) associated with increased $PM_{2.5}$ concentrations.[24]

California's ambient air quality standards (CAAQS) for PM are designed to protect the most sensitive groups of people, including infants and children, the elderly, and persons with heart or lung disease against $PM_{2.5}$-related adverse health effects, including

- Premature mortality,
- Increased hospitalizations and emergency room visits
- Worsening of asthma symptoms and acute and chronic bronchitis, and
- Doctor's visits for respiratory illnesses or heart disease.[25]

Current air quality regulatory standards for $PM_{2.5}$ set by CARB and US EPA and voluntary guidelines set by the WHO are included in Table 2.

Zanobetti, A. and Schwartz, J. The effect of fine and coarse particulate air pollution on mortality: a national analysis. Environ Health Perspect. 2009; 117(6):898-903.

[23] US EPA. National ambient air quality standards for particulate matter. Final Rule. 40 CFR Parts 50, 51, 52, 53, and 58. Federal Register. 2013; 78(10):3086-287.

[24] Ostro, B. Health Impact Assessment. In: Air Quality Guidelines Global Update 2005: Particulate matter, ozone, nitrogen dioxide and sulfur dioxide. Germany: World Health Organization; Druckpartner Moser; 2006. p. 153-71.; Samet, J.M., *et al.* Particulate Matter. In: Air Quality Guidelines Global Update 2005: Particulate matter, ozone, nitrogen dioxide and sulfur dioxide World Health Organization. Germany: World Health Organization; Druckpartner Moser; 2006. p. 217-91.

[25] California Air Resources Board. Ambient Air Quality Standards (AAQS) for Particulate Matter. California Environmental Protection Agency; Last updated October 25, 2015. Available from: https://www.arb.ca.gov/research/aaqs/pm/pm.htm. Accessed: 9/13/2017.

Table 2. $PM_{2.5}$ Ambient Air Quality Standards and Guidelines

Agency	$PM_{2.5}$ Annual average ($\mu g/m^3$)	$PM_{2.5}$ 24-hr 98th percentile ($\mu g/m^3$)[A]	$PM_{2.5}$ 24-hr 99th percentile ($\mu g/m^3$)[A]
US EPA (NAAQS)[27]	12	35	---
California Ambient Air Quality Standard (CAAQS)[25]	12	---	---
WHO Air Quality Guideline[26]	10	---	25

KEY: --- No established value;
[A]Daily (24-hr) values refer to the distribution of all yearly daily measurements; the NAAQS 24-hr standard refers to the 98th percentile, which is the sample whose concentration bounds (is greater or equal to) 98% of all yearly samples; the WHO 24-hr standard refers to the 99th percentile, which is the sample whose concentration bounds (is greater or equal to) 99% of all yearly samples

Based on the ambient air quality standards listed in Table 2, regions with adequate monitoring data are assigned compliance rankings of either "attainment" or "nonattainment." US EPA and CARB have different criteria for compliance.

US EPA designates NAAQS compliance ("attainment" or "nonattainment") for each air quality monitoring area by comparing the area-specific "design value" (DV) to the NAAQS level.[27] Attainment occurs when DVs are less than NAAQS values. DVs are area-specific values based on 3-year average metrics. Specifically:

- For annual $PM_{2.5}$ NAAQS:
  - The annual $PM_{2.5}$ NAAQS DV is the 3-yr average of annual mean mass concentrations for each eligible monitoring site.[28]
  - "Attainment" is designated when the DV is less than the NAAQS.
- For 24-hr $PM_{2.5}$ NAAQS:
  - The 24-hr $PM_{2.5}$ NAAQS DV is the 3-yr average of the 98th percentile values for all credible 24-hr $PM_{2.5}$ samples collected per year (where

[26] Samet, J.M., *et al.* Particulate Matter. In: Air Quality Guidelines Global Update 2005: Particulate matter, ozone, nitrogen dioxide and sulfur dioxide World Health Organization. Germany: World Health Organization; Druckpartner Moser; 2006. p. 217-91.

[27] US EPA. 40 CFR part 50 National primary and secondary ambient air quality standards. Code of Federal Regulations. 2016., Appendix N, Interpretation of the National Ambient Air Quality Standards for PM2.5.

[28] The annual PM2.5 NAAQS DV is calculated by first averaging daily values by each quarter of each year; annual mean concentrations by year are calculated by averaging the quarterly means; and the annual PM2.5 NAAQS DV is calculated by averaging the three yearly means.

98th percentile values represent the concentration that bounds [i.e., is greater than or equal to] 98% of all 24-hr measurements).[29]

- "Attainment" is designated when the DV is less than the NAAQS.

California determines CAAQS compliance ("attainment" or "nonattainment") for each air quality monitoring area as follows:

- "Attainment" is designated if data demonstrate no area samples exceeded the CAAQS value.[30]
- "Nonattainment" is designated if data demonstrate at least one area sample, which met representativeness criteria, was in violation (excess) of the CAAQS value.[31]

The US EPA established the Air Quality Index (AQI) to communicate daily air quality for criteria air pollutants with their relationships to public health.[32] Table 3 summarizes $PM_{2.5}$ AQI values with recommended actions to protect public health.

In the California San Francisco Bay Area, the "Spare the Air" program of the Bay Area Air Quality Management District (BAAQMD) uses weather patterns and air monitoring measurements to issue daily air pollution forecasts to predict air pollution levels for the next 5 days. BAAQMD uses the AQI metric (Table 3) to communicate forecasts in terms of human health risks and issues community alerts on days when the air quality forecast is unhealthy (or worse).[33]

[29] The 24-hr PM2.5 NAAQS DV is calculated by first ordering all daily values for each year based on value (largest to smallest); the value that represents the 98th percentile of the yearly data (i.e., the value that is greater than 98% of the collected data) is selected; and the 24-hr PM2.5 NAAQS DV is calculated by averaging the three yearly 98th values.

[30] 17 CCR § 70304. Criteria for Designating an Area as Attainment. September 2017.

[31] 17 CCR § 70303. Criteria for Designating an Area as Nonattainment. September 2017.; 17 CCR § ADC Appendix 1. Criteria for Determining Data Representativeness. September 2017.; 17 CCR § ADC Appendix 2. Air Resources Board Procedure for Reviewing Air Quality Data Possibly Affected by a Highly Irregular or Infrequent Event. September 2017.

[32] US EPA. Air Quality Index: A guide to air quality and your health. EPA-456/F-14-002. Research Triangle Park, NC. Office of Air Quality Planning and Standards, February 2014.

[33] BAAQMD. Spare the Air: Today's Air Quality. http://www.sparetheair.org/stay-informed/todays-air-quality. Accessed: 9/13/2017. ; BAAQMD. Spare the Air: Five Day Forecast. http://www.sparetheair.org/stay-informed/todays-air-quality/five-day-forecast. Accessed: 9/13/2017.

Table 3. $PM_{2.5}$ Ambient Air Quality Standards and Guidelines

Air Quality Index (AQI) [color]	$PM_{2.5}$ Breakpoints (24-hr average $\mu g/m^3$)[34]	AQI Category	Air Quality Category Meaning	Public Actions Recommended to Protect Health from Particle Pollution*
0 to 50 [green]	0 to 12.0	Good	Satisfactory (air pollution poses little or no risk)	None
51 to 100 [yellow]	12.1 to 35.4	Moderate	Acceptable (there may be a moderate health concern for a very small number of people)	Unusually sensitive people should CONSIDER REDUCING prolonged or heavy exertion
101 to 150 [orange]	35.5 to 55.4	Unhealthy for Sensitive Groups	Unhealthy for sensitive groups (people with heart or lung disease, older adults, and children are at greater risk from the presence of particles in the air; the general public is not likely to be affected)	Children, older adults, and people with heart or lung disease should REDUCE PROLONGED or HEAVY EXERTION.
151 to 200 [red]	55.5 to 150.4	Unhealthy	Unhealthy (everyone may begin to experience some adverse health effects; members of sensitive groups may experience more serious effects)	Children, older adults, and people with heart or lung disease should **AVOID** PROLONGED or HEAVY EXERTION. Others should REDUCE PROLONGED or HEAVY EXERTION
201 to 300 [purple]	150.5 to 250.4	Very Unhealthy	Very unhealthy (triggers a health alert; everyone may experience more serious health effects)	Children, older adults, and people with heart or lung disease should **AVOID ALL PHYSICAL ACTIVITY OUTDOORS.** Others should **AVOID** PROLONGED or HEAVY EXERTION
301 to 500 [maroon]	250.5 to 500.4	Hazardous	Hazardous (triggers a health warning of emergency conditions; the entire population is more likely to be affected)	(no guidance given)

*Emphasis added

Opinion 1: The transport, storage, and handling of coal through the City and Port of Oakland poses an increased risk of adverse health effects to West Oakland residents, including premature mortality, increased hospital admissions and emergency department visits, and development of chronic respiratory disease

Oakland City Council prohibited the transport, storage, and handling of coal and coke at the bulk material facilities or terminals throughout the City of Oakland

The City of Oakland City Council passed a resolution to prohibit the transport, storage, and handling of coal and coke at the bulk material facilities or terminals throughout the City of Oakland on June 27, 2016.[35]

The remainder of this section summarizes the scientific dataset that demonstrated that transport, storage, and handling of coal through the City and Port of Oakland posed a health risk to its residents. Specifically, I will review (1) the historically poor air quality data for the San Francisco Bay Area; (2) the implications for health risks from $PM_{2.5}$ air pollution in the San Francisco Bay Area and the West Oakland community, which was identified as one of the most at-risk communities within the Bay Area Air District; (3) the historical and on-going initiatives targeted to improve air quality, lessen $PM_{2.5}$ levels, and reduce health risks posed by ambient air pollution emission associated with the Port of Oakland; (4) the scientific evidence regarding the impact of the proposed coal-operations on ambient $PM_{2.5}$ levels in the West Oakland community; and (5) whether the proposed coal-operations increase the risk of adverse health effects to West Oakland residents.

[34] US EPA. 2017. AQI Breakpoints. AQI Reference Table. Accessed August 24, 2017. Available at: https://aqs.epa.gov/aqsweb/documents/codetables/aqi_breakpoints.html; US EPA. National ambient air quality standards for particulate matter. Final Rule. 40 CFR Parts 50, 51, 52, 53, and 58. Federal Register. 2013; 78(10):3086-287.

[35] Oakland Bulk & Oversized Terminal, LLC vs. City of Oakland, First Amended Complaint, p. 15.

The San Francisco Bay Area, which includes West Oakland, has historically poor air quality and employs active measures to improve air quality and reduce health risks posed by ambient air $PM_{2.5}$ pollution

The Bay Area Air Quality Management District (BAAQMD) consists of Alameda, Contra Costa, Marin, Napa, San Francisco, San Mateo, Santa Clara, southwestern Solano, and southern Sonoma counties.[36]

Historically, the highest PM concentrations in the BAAQMD occur in the winter when consecutive days of stagnant and clear winter days facilitate a lesser sunlight-driven atmospheric temperature inversion layer.[37] Inversions are atmospheric conditions caused by increasing temperature with elevation, and result in a layer of warm air that blankets the air underneath; an inversion prevents the rise of cooler air (and air pollutants) trapped beneath it.[38] Inversions are associated with larger air pollution concentrations because they trap pollutants by preventing atmospheric dispersion. High $PM_{2.5}$ episodes can be regional in scale, affecting multiple Bay Area locations. Exceedances of the 24-hour national $PM_{2.5}$ standard most frequently occur between November and February. During other seasons, Bay Area $PM_{2.5}$ tends to be less due to the area's natural ventilation system.[36]

BAAQMD design values (i.e., 3-year average values, as described on page 16) are depicted for the years 2001 – 2014 in Figure 2. The BAAQMD is in attainment for the annual $PM_{2.5}$ NAAQS (established as 15 $\mu g/m^3$ in 1997[39] until 2012, when it was reduced to 12 $\mu g/m^3$).[40] Specifically, Alameda County, which includes West Oakland, was established as an attainment area in US EPA's first $PM_{2.5}$ compliance classification and remained in attainment upon implementation of the 12 $\mu g/m^3$ standard.[40]

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[36] BAAQMD. About the Air District. San Francisco, CAhttp://www.baaqmd.gov/home/about-the-air-district. Accessed: 8/18/2017.

[37] BAAQMD. Spare the Air: Cool the climate. A blueprint for clean air and climate protection in the Bay Area. Final 2017 Clean Air Plan. April 19, 2017., 2/16.

[38] US EPA. Inversion. From Environmental Issues Terms & Acronyms. Terminology Services - Terminology and Acronyms Report Date Accessed: 9/29/2017. Available from: https://iaspub.epa.gov/sor_internet/registry/termreg/searchandretrieve/termsandacronyms/search.do. 2017.

[39] US EPA. Air quality designations and classifications for the fine particles (PM2.5) national ambient air quality standards. Final Rule. 40 CFR Part 81. Federal Register. 2005; 70(3):944-1019.

[40] US EPA. Air quality designations for the 2012 primary annual fine particle (PM2.5) national ambient air quality standards (NAAQS). Final Rule. 40 CFR Part 81. Federal Register. 2015; 80(10):2206-84.

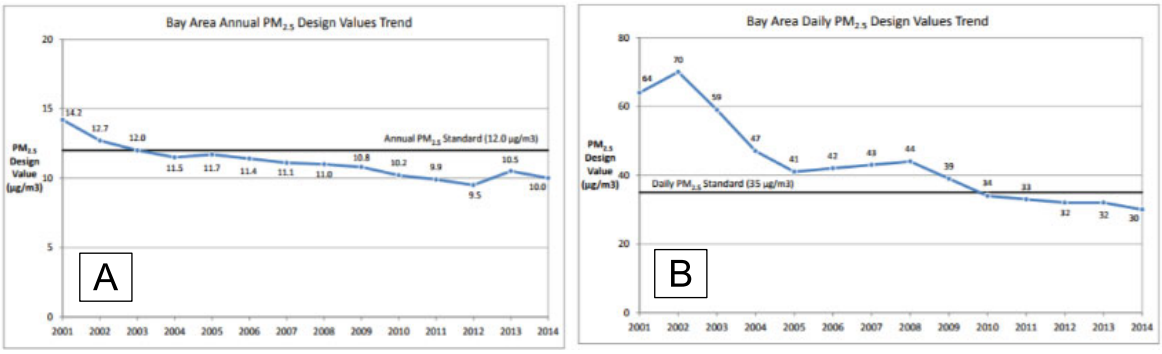

Figure 2. Bay Area trends for $PM_{2.5}$ design values (2001 through 2014)[41]

In contrast, the BAAQMD has been historically a non-attainment area for the 24-hr $PM_{2.5}$ NAAQS.[42] The trend toward lesser ambient air $PM_{2.5}$ concentrations of the BAAQMD (Figure 2B) led its recent (and first) classification as an attainment area by the US EPA (effective June 9, 2017). The new attainment status was based on BAAQMD ambient air quality that resulted in a 24-hr design value (DV) below the NAAQS. Specifically, the 98th percentile 24-hr $PM_{2.5}$ values averaged over the three years from 2013 to 2015 resulted in a DV of 30 µg/m^3, which was less than the NAAQS of 35 µg/m^3.[43]

Addressing health risks posed by exposure to PM in ambient air pollution together with implementation of active strategies to reduce exposures and risks have a long history in the State of California and the BAAQMD. The main sources of $PM_{2.5}$ in the BAAQMD are combustion of fossil fuels and combustion of wood (primarily residential wood burning).[44] The reductions in 24-hr $PM_{2.5}$ that have occurred over time have been attributed to targeted efforts by the State of California and BAAQMD to decrease emissions from key sources (i.e., motor vehicles and wood burning).[45] **Specific and targeted initiatives to reduce PM (and $PM_{2.5}$) air pollution in the Bay Area, include**:

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[41] BAAQMD. Air monitoring five-year assessment. July 1, 2015.

[42] The NAAQS for $PM_{2.5}$ was established as 65 µg/m^3 in 1997 and reduced to 35 µg/m^3 in 2006; US EPA. Table of Historical Particulate Matter (PM) National Ambient Air Quality Standards (NAAQS). Last updated March 16, 2017. Available from: https://www.epa.gov/pm-pollution/table-historical-particulate-matter-pm-national-ambient-air-quality-standards-naaqs. Accessed: 9/30/2017.

[43] US EPA. Determinations of attainment by the attainment date, determinations of faiure to attain by the attainment date and reclassification for certain nonattainment areas for the 2006 24-hour fine particulate matter national ambient air quality standards. Final Rule. 40 CFR Parts 52 and 81. Federal Register. 2017; 82(89):21711.

[44] BAAQMD. Spare the Air: Cool the climate. A blueprint for clean air and climate protection in the Bay Area. Final 2017 Clean Air Plan. April 19, 2017., ES-4.

[45] BAAQMD. Spare the Air: Cool the climate. A blueprint for clean air and climate protection in the Bay Area. Final 2017 Clean Air Plan. April 19, 2017.

- Winter 1991: Launch of the voluntary Don't Light Tonight (later Spare the Air Tonight) program to target wintertime PM pollution from wood burning stoves and fireplaces.[46]
- 1998: Approval of the Model Wood Smoke Ordinance to provide cities and counties guidance in regulating new installations of wood stoves and fireplaces.[46] This ordinance combined with the Spare the Air program reduced wood smoke emissions through:
  (1) Communication of options to switch to cleaner ways to heat homes (besides burning wood; i.e., replace old woodstoves/fireplaces with new certified models),
  (2) Recommendations to weatherize homes in order to use less wood to heat, and
  (3) Ordinances to make general use of fireplaces and woodstoves illegal when a Winter Spare the Air Alert is in effect.[47]
- 2000: Adoption of a statewide Diesel Risk Reduction Plan to address the health risks associated with ambient diesel PM emissions; the Plan projected reductions in diesel PM emissions and associated cancer risks by 85% by 2020.[48]
- 2013: BAAQMD passed resolutions to reduce fugitive PM emissions from foundry and forging operations and from metal recycling and shredding operations.[49]

Adverse health effects due to $PM_{2.5}$ exposure are anticipated despite the attainment status classification of the Bay Area. Figure 3 provides the estimated annual incidence for health effects from exposure to air pollution for Bay Area residents. A comparison of incidences for Bay Area air pollution levels from "then" (late 1980s) to "now" (2015) shows that while health effects due to air pollution, particularly $PM_{2.5}$, have declined, $PM_{2.5}$ continues to impose health impacts for residents.[50]

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[46] BAAQMD. History of the Air District. http://www.baaqmd.gov/home/about-the-air-district/history-of-air-district. Accessed: 9/14/2017.

[47] BAAQMD. Particulate Matter: Wood Smoke. http://sparetheair.org/stay-informed/particulate-matter/wood-smoke.aspx. Accessed: 9/14/2017.

[48] California Air Resources Board. Risk Reduction Plan to Reduce Particulate Matter Emissions from Diesel-Fueled Engines and Vehicles. October 2000.

[49] BAAQMD. A Resolution of the Board of Directors of the Bay Area Air Quality Management District Adopting District Regulation 6, Rule 4: Metal Recycling and Shredding Operations; Adopting District Regulation 12, Rule 13: Foundry and Forging Operations; and Adoptiug a CEQA Negative Declaration for the Project. Contract No.: Resolution No. 2013-04.

[50] BAAQMD. Spare the Air: Cool the climate. A blueprint for clean air and climate protection in the Bay Area. Final 2017 Clean Air Plan. April 19, 2017., 2/26-7.

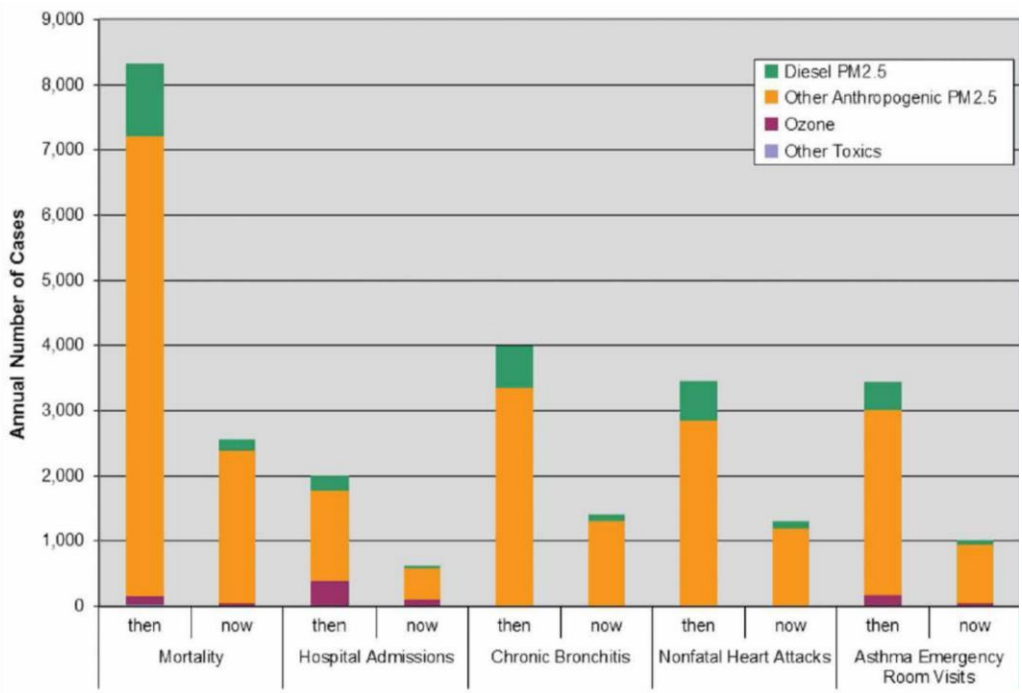

Figure 3. Estimated incidence of health effects from exposure to air pollution for the Bay Area, "then" represents air pollution levels from the late 1980s and "now" represents 2015 levels[51]

In 2017, the American Lung Association ranked the San Jose-San Francisco-Oakland as the 4th worst metropolitan area in the United States for year-round particle pollution (annual $PM_{2.5}$) and the 6th worst for short-term particle pollution (24-hr $PM_{2.5}$), indicating risk attributable to children, elderly, asthmatics, individuals suffering from chronic obstructive pulmonary disease, lung cancer, cardiovascular disease, diabetes, and poverty.[52]

West Oakland has been specifically identified as an at-risk community for the adverse health effects from PM air pollution for over a decade

The West Oakland community located in Oakland, California, in this report is the same community defined by the CARB: specifically, the geographical community bounded by the Maritime Port of Oakland (the Port), the Union Pacific (UP) Oakland Railyard, and the I-

[51] BAAQMD. Spare the Air: Cool the climate. A blueprint for clean air and climate protection in the Bay Area. Final 2017 Clean Air Plan. April 19, 2017., p. 2/26-7, Figure 2-15.

[52] American Lung Association. State of the Air 2017. Chicago, IL.

480, I-880, and I-980 freeways.[53] Neighborhoods in West Oakland communities are diverse containing intermixed light industrial, commercial, and residential areas. Sources of diesel PM within the community include activities at the Maritime Port of Oakland, the Union Pacific Railyard, and other sources such as local freeways and marine vessels in the San Francisco Bay.[54]

The scientific community agrees people with heart or lung disease, older adults, and children are considered populations of greatest risk from the adverse effects of PM.[55]

The community of West Oakland has increased ratios of the population subgroups most vulnerable to the health impacts of pollution (children, older adults, and people with asthma).[56]

Concerns regarding potential health risks from PM among West Oakland community members are well established and have existed for over a decade. A timeline of documents detailing the health risks of ambient PM exposure to residents of the community of West Oakland include:

- 2004: Launch of BAAQMD's Community Air Risk Evaluation (CARE) program. As described below, the CARE program demonstrated in its initial and subsequent analyses that the regional community of West Oakland was one of the most adversely impacted by air pollution (e.g., relatively high emissions or exposures with increased ratios of populations most vulnerable to the health impacts of pollution).[57]
- 2006 (CARE Phase I findings): Identification of Western Alameda county, which included West Oakland as a community with (1) increased diesel PM emissions and (2) increased young (<18) and aged (>64) populations per geographical area; and (3) and increased age-adjusted asthma hospitalization rates for children <14 years of age compared to most of the surrounding BAAQMD community.[58]

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[53] California Air Resources Board. Diesel Particulate Matter Health Risk Assessment for the West Oakland Community. December 2008.

[54] California Air Resources Board. Diesel Particulate Matter Health Risk Assessment for the West Oakland Community. December 2008.

[55] US EPA. National ambient air quality standards for particulate matter. Final Rule. 40 CFR Parts 50, 51, 52, 53, and 58. Federal Register. 2013; 78(10):3086-287.

[56] BAAQMD. Community Air Risk Evaluation Program: Phase I Findings and Policy Recommendations Related to Toxic Air Contaminants in the San Francisco Bay Area. September 2006.

[57] BAAQMD. Identifying Areas with Cumulative Impacts from Air Pollution in the San Francisco Bay Area - Version 2. March 2014.

[58] BAAQMD. Community Air Risk Evaluation Program: Phase I Findings and Policy Recommendations Related to Toxic Air Contaminants in the San Francisco Bay Area. September 2006.

Characterization of vulnerable, at-risk (sensitive) subpopulations is important because the overall disease burden among geographical populations containing increased ratios of vulnerable subpopulations will be increased (and be disproportionately affected) compared to geographical populations with lesser ratios of vulnerable subpopulations.

Within the BAAQMD, the West Oakland community (and the Port of Oakland) were the only geographical areas identified for follow-up (Phase II) CARE Phase activities, which included conduction of Health Risk Assessments for both the Port of Oakland and the West Oakland community.[58]

- 2008 (West Oakland Health Risk Assessment): Concluded West Oakland community residents were exposed to unhealthful levels of diesel PM [$PM_{2.5}$] emissions. The estimated increase in excess cancers from lifetime residence (70 years) in the West Oakland community breathing 2005 diesel PM levels (i.e., emissions from all sources) was about 1,200 excess cancers per million residents.[59]
- 2009: West Oakland identified as a community within the BAAQMD likely to face the highest health risks from air pollution due to its relatively high emissions of toxic air contaminants, relatively high exposures of youth and seniors to toxic air contaminants, and relatively high levels of poverty.[60]
- 2014: West Oakland continued to be identified as an "impacted community," or a BAAQMD area where air pollution's health impacts are relatively high and associated with a greater chance of individual risks from air pollutants.[61]

Initiatives targeted to improve air quality, lessen $PM_{2.5}$ levels, and reduce health risks posed by ambient air pollution associated with the Port of Oakland's seaport emissions

The State of California has developed specific air pollution control strategies that were enacted by the Port of Oakland to attempt to reduce PM emission to surrounding communities (i.e., West Oakland) and thereby decrease ambient air pollution, lessen $PM_{2.5}$ levels, and consequently reduce the health risks associated with those pollutants to community residents.

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[59] California Air Resources Board. Diesel Particulate Matter Health Risk Assessment for the West Oakland Community. December 2008.

[60] Bay Area Air Quality Management District (BAAQMD). Applied Method for Developing Polygon Boundaries for CARE Impacted Communities. Technical Memorandum. December 2009.

[61] BAAQMD. Identifying Areas with Cumulative Impacts from Air Pollution in the San Francisco Bay Area - Version 2. March 2014.

As explained below, the Port of Oakland's dramatic decreases in air emissions between the calendar years 2005 and 2015[62] were attributed to a targeted series of initiatives, including:

- CARB's Drayage Truck Regulation (initially approved in 2007 and revised in 2011): targeted reduction of emissions from drayage trucks at California's ports and intermodal rail yards by requiring truck compliance with statewide emissions requirements. It applies to all trucks transporting cargo going to or coming from California's ports and intermodal rail yards. [63]
- CARB's At-Berth Regulation (approved in 2007): targeted reduction of emissions from diesel auxiliary engines on container ships, passenger ships, and refrigerated-cargo ships while berthing at a California Ports. The regulation allowed operators two options to reduce at-berth emissions: (1) turn off auxiliary engines and connect to an alternate source of power, most likely grid-based shore power; or (2) use alternative control technology to achieve equivalent emission reductions.[64]
- CARB's Ocean-Going Vessels Fuel Rule (approved in 2008): targeted reduction of emissions through mandating shipping lines switch to cleaner burning, low sulfur fuel.[65]
- The Maritime Air Quality Improvement Plan (MAQIP approved in 2009): targeted reduction of diesel PM to reduce the associated risk of cancer among West Oakland community residents.[66]

  In addition to compliance with regulations aimed to reduce emissions, the MAQIP indicated:

[62] Lindhjem, C., *et al.* Port of Oakland: 2015 Seaport Air Emissions Inventory. Final Report. Prepared for Port of Oakland by Ramboll Environ, Novato, CA. October 2016.

[63] California Air Resources Board. Drayage Truck Registry Help Information. Last updated March 16, 2012. Available from: https://www.arb.ca.gov/arber/dtrregistration/dtrregistration.htm. Accessed: 9/23/2017. ; 13 CCR § 2027. In-Use On-Road Diesel-Fueled Heavy-Duty Drayage Trucks. September 2017.

[64] California Air Resources Board. Shore Power for Ocean-going Vessels. Last updated August 22, 2017. Available from: https://www.arb.ca.gov/ports/shorepower/shorepower.htm. Accessed: 9/23/2017.

[65] California Air Resources Board. Ocean-Going Vessels - Fuel Rule. Last updated August 1, 2017. Available from: https://www.arb.ca.gov/ports/marinevess/ogv.htm. Accessed: 9/23/2017.

[66] Port of Oakland. Maritime Air Quality Improvement Plan (MAQIP). Final. Approved by Board of Port Commissioners. April 2009.

"…the Port [of Oakland] would implement emissions reduction measures in advance of regulatory deadlines in order to reduce the duration of people's exposure to emissions that may cause health risks."[66]

These early actions included incentives for replacement or retrofit of polluting drayage trucks, mechanisms for enforcing prohibition of Port truck parking or operation on neighborhood streets, and feasible and cost-effective means of reducing ship idling emissions. The goal for the MAQIP was an 85% reduction (from 2005 to 2020) in neighboring-community cancer risk related to exposure to diesel PM emissions from the Port's maritime operations.[67]

- The Maritime Comprehensive Truck Management Program (CTMP, a MAQIP program adopted by the Port of Oakland in 2009): reduction of emissions through early action retrofit and/or replacement of port drayage trucks (as well as general support of CARB's Drayage Truck Regulation). Specifically, the CTMP enacted a ban on all trucks with older (pre-1994) engines; a progressive emission compliance schedule for 1994-2006 engines (for full emission compliance by 2014); and required compliance for 2007 and newer engines.[68]

Overall, the Port of Oakland estimated air emissions for diesel PM decreased from 261 tons in 2005 to 63 tons in 2015. Health impacts associated with trucks, which may drive in close proximity to sensitive populations, decreased as truck diesel PM decreased from 16 tons in 2005 to 0.4 tons in 2015.[69]

Allowing a new operation known to significantly *increase* PM concentrations (such as the transport, storage, and handling of coal) is in direct opposition to the efforts spent to *reduce* airborne PM emissions and health impacts in the West Oakland community. Thus, introducing a major coal-handling facility in the West Oakland area (i.e., significantly *elevating* local PM concentrations) would be at odds with the City's and the Port's goals of *lowering* airborne pollution levels within the neighborhoods of West Oakland.

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[67] Port of Oakland. Maritime Air Quality Improvement Plan (MAQIP). Final. Approved by Board of Port Commissioners. April 2009.

[68] Port of Oakland. Maritime Comprehensive Truck Management Program: A MAQIP Program. Adopted June 16, 2009. June 16, 2009.

[69] Lindhjem, C., *et al.* Port of Oakland: 2015 Seaport Air Emissions Inventory. Final Report. Prepared for Port of Oakland by Ramboll Environ, Novato, CA. October 2016.

West Oakland ambient $PM_{2.5}$ concentrations

BAAQMD monitors ambient $PM_{2.5}$ concentrations from a single monitoring station in West Oakland. The "West Oakland" air monitoring station is approximately one mile (generally downwind) from the Port of Oakland, located in a shelter in a paved parking lot at 1100 21st St, Oakland, CA 94607; $PM_{2.5}$ monitoring began December 18, 2012.[70]

The West Oakland monitor was established as a "population exposure" and "source oriented" site, downwind of major sources of pollutants (i.e., the Port of Oakland, which was considered a major area source of diesel particulate matter emissions).in an area of high population density.[70]

A study investigating the spatial variations of $PM_{2.5}$ in the West Oakland area demonstrated $PM_{2.5}$ concentrations were relatively consistent between the West Oakland BAAQMD monitor and other West Oakland locations (although the $PM_{2.5}$ fraction associated with diesel PM varied).[71]

Annual $PM_{2.5}$ concentrations recorded at the BAAQMD West Oakland monitor are included in Table 4.

Table 4. $PM_{2.5}$ Ambient Air $PM_{2.5}$ Concentrations at the West Oakland BAAQMD Monitor[72]

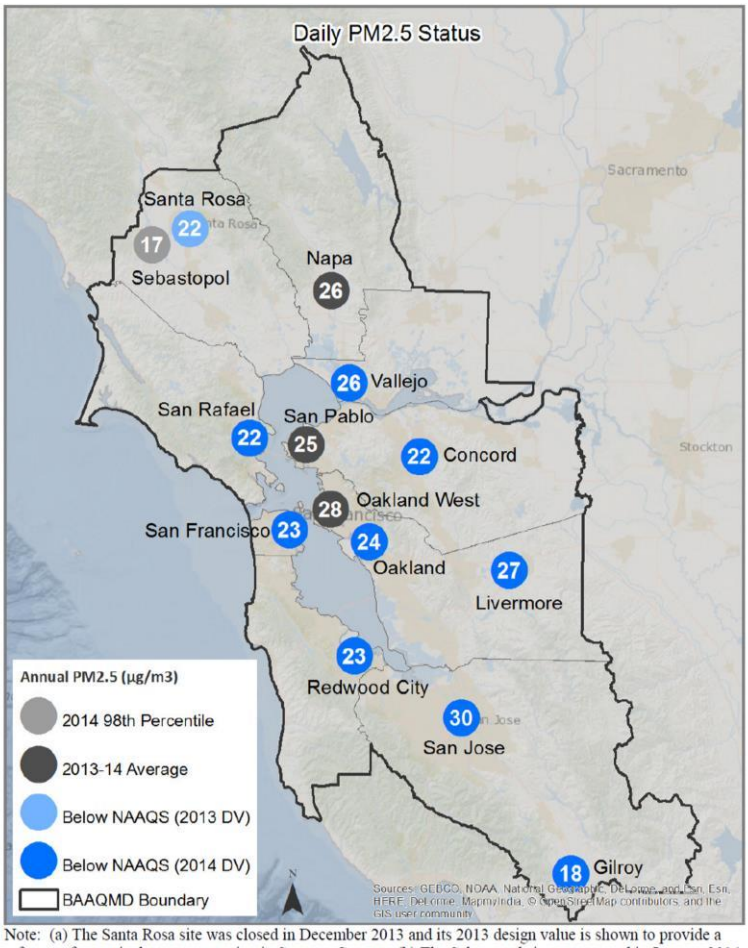
Year	$PM_{2.5}$ Annual average ($\mu g/m^3$)	$PM_{2.5}$ 24-hr 98th percentile ($\mu g/m^3$)
2013	12.8	30.0
2014	9.5	25.7
2015	10.1	29.9

[70] BAAQMD. 2015 Air Monitoring Network Plan. July 1, 2016.

[71] Fujita, E.M., *et al.* Spatial variations of particulate matter and air toxics in communities adjacent to the Port of Oakland. Journal of the Air & Waste Management Association. 2013; 63(12):1399-411.

[72] US EPA. Air Quality System Data Mart [internet database]. Last updated 05/26/2017. Available from: http://aqsdr1.epa.gov/aqsweb/aqstmp/airdata/download_files.html. Accessed: 9/26/2017.

As depicted in Figure 4, daily (24-hr) $PM_{2.5}$ concentrations in West Oakland (averaged over the years 2013 to 2014) are among the highest concentrations within the BAAQMD when compared to design values (i.e., 3-year averages) from other geographical locations.



Note: (a) The Santa Rosa site was closed in December 2013 and its 2013 design value is shown to provide a reference for particulate concentration in Sonoma County. (b) The Sebastopol site was opened in January 2014 and replaced the Santa Rosa site. Therefore, only the 2014 98th percentile can be provided. (c) FEM $PM_{2.5}$ monitoring began at Napa, Oakland West and San Pablo in December 2012. Therefore, only 2013-2014 average of the 98th percentiles is provided.

Figure 4. Spatial variation for 2014 daily $PM_{2.5}$ concentrations among the BAAQMD[73]

These levels of ambient air quality are associated with adverse health effects among the exposed communities. A 2011 BAAQMD Health Impact Analysis evaluating the incremental benefits of increasing air quality (i.e., through analyzing impacts of adverse health effects in the community for each incremental 1 $\mu g/m^3$ $PM_{2.5}$ reduction in Bay Area ambient air from a design value of about 31 $\mu g/m^3$), estimated the benefits for a variety of

[73] BAAQMD. Air monitoring five-year assessment. July 1, 2015.

health endpoint incidents. Each 1 μg/m^3 reduction in ambient $PM_{2.5}$ was associated with a reduction in the numbers of asthma exacerbations by 1,362, nonfatal acute myocardial infarctions (heart attacks) by 71, and all-cause mortality by 66.[74]

Studies demonstrate ambient PM concentration rises in locations surrounding areas of coal storage, handling, and shipment

California's South Coast Air Quality Management District (SCAQMD) Rule 1158, which affected storage, handling, and shipment of petroleum coke, coal, and sulfur, was amended in June 1999 to further reduce particle emissions from those sources (full compliance was mandated for June 2004).[75] A series of subsequent studies monitored the efficacy of the Rule and provided supporting data for the Legislative Report. These studies evaluated PM_{10} concentrations during spring/summer and fall/winter seasons. Monitoring site selection criteria included locations relative to coal and coke facilities with respect to the local prevailing wind patterns and their importance as locations at or near student populations. In the fall/winter analyses spanning 2005-6 and 2006-7, three monitored sites exhibited higher concentrations of airborne elemental carbon PM, which arises in part from coke and coal storage and transportation (including diesel emissions from trucks, trains, and ships) compared to community monitoring sites in either Los Angeles or Long Beach. In addition, the measured average ambient PM_{10} levels at all study sites were higher than those at the Long Beach and Central Los Angeles network stations for the duration of these studies, indicating coal/coke operations contribute to ambient air pollution.[75]

Coal transport by rail releases $PM_{2.5}$ into the ambient air

Scientific literature has shown train (rail) transport of coal releases respirable coal dust (as $PM_{2.5}$) into the ambient air of traversed regions, even if surfactant was applied (this was in addition to $PM_{2.5}$ released as diesel particulate matter). Specifically, Jaffe et al. (2015) measured $PM_{2.5}$ levels along trains and observed coal trains approximately doubled $PM_{2.5}$ levels compared to the $PM_{2.5}$ levels associated with trains' diesel engine emissions. Overall, 5.4% of coal trains passing the monitoring station were annotated as "super-duster" trains (i.e., those with visible coal dust emissions; these trains also had the highest observed $PM_{2.5}$ concentrations).[76]

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[74] BAAQMD. Health Impact Analysis of Fine Particulate Matter in the San Francisco Bay Area. September 2011.

[75] SCAQMD. Rule 1158 Follow-Up Study #12/13. Sampling conducted November 12, 2005 - February 1, 2006 and December 1, 2006 - March 1, 2007. Report# MA 2006-02.

[76] Jaffe, D., *et al.* Diesel particulate matter and coal dust from trains in the Columbia River Gorge, Washington State, USA. Atmospheric Pollution Research. 2015; 6(6):946-52.

HDR Engineering Report

The HDR report prepared for the California Capital and Investment Group concluded the amount of coal dust emissions to the City of Oakland from transport of coal to the Oakland Bulk and Oversized Terminal facility and related terminal operations will be negligible and that impacts from coal dust emissions and deposition will not harm health or the environment.[77] HDR's conclusion was based on a draft (i.e., not finalized) version of an environmental impact statement for the Tongue River Rail Project (a 42-mile rail line in Montana) that assumed application of a topping agent with open rail cars. According to HDR:

> *"[the draft assessment] modeled concentrations of airborne coal dust from train cars (including PM_{10} and $PM_{2.5}$) and determined that they are expected to be below the standards set in the NAAQS and the Montana Ambient Air Quality Standards (Montana AAQS) to protect human health."*[77]

However, exceeding air quality standards will depend on ambient (background) concentrations as well as emission concentrations. Furthermore, emission concentrations calculated for the Tongue River project are dependent on meteorology, topology, and rail characteristics and may change with the Final version of the report. HDR did not indicate values for the $PM_{2.5}$ emission concentrations. From the Draft Environmental Impact Statement Tongue River Railroad issued in April 2015 (HDR's report cited a draft from 2014), the contribution of coal dust (depending on number of trains per day and routes) ranged from 1.5 to 5.3 $\mu g/m^3$ for 24-hr average $PM_{2.5}$ and 0.3 to 1.2 $\mu g/m^3$ for annual average $PM_{2.5}$ concentrations.[78]

Therefore, the Tongue River Railroad Draft Environmental Impact Statement (on which the HDR report relied), demonstrated rail transport of coal is expected to increase ambient $PM_{2.5}$ concentrations. The precise concentrations expected within Oakland due to rail transport, storage, and related activities will depend on Oakland-specific meteorology, topology, and rail characteristics.

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[77] Leibsch, E. J. and Musso, M. HDR Engineering. Oakland Bulk and Oversized Terminal Air Quality and Human Health and Safety Assessment of Potential Coal Dust Emissions. Prepared for California Capital and Investment Group. September 2015.

[78] Surface Transportation Board, Office of Environmental Analysis. Draft Environmental Impact Statement, Tongue River Railroad Company Construction and Operation of a New Rail Line in Southeastern Montana, Chapter 6: Coal Dust. Last updated April 17, 2015. Available from: http://www.tonguerivereis.com/documents/draft_eis/chapters/Ch06_Coal%20Dust.pdf. Accessed: 8/4/17.

Environmental Science Associates (ESA) Report

The ESA report prepared for the City of Oakland indicated transport, storage, and handling of coal in Oakland, including at the proposed OBOT in the west gateway area of the former Oakland Army Base, was associated with fugitive dust emissions from rail cars during transport. Dust emissions included ambient dispersion of PM_{10} and $PM_{2.5}$ in the BAAQMD and in West Oakland specifically.[79] ESA qualified (flagged) their presented emission rates several ways, including:

- Estimated coal dust emission characteristics were deemed conservative (i.e., likely lower than would occur) because resuspension of previously deposited coal dust from earlier trains was not included in their estimates.
- Coal-generated $PM_{2.5}$ emissions were associated with both (1) open, uncovered coal filled rail cars and (2) coal filled rail cars treated with dust suppressants or surfactants (although, as expected, estimated emission characteristics were higher for uncovered rail cars than rail cars using dust suppressants or surfactants).
- The lack of data regarding the unknown effectiveness of surfactants to reduce coal dust emissions from open rail cars during a complete long distance (over 700 miles) rail trip over mountainous terrain before reaching Oakland.

Conclusion

The transport, storage, and handling of coal through the City and Port of Oakland poses an increased risk of adverse health effects to West Oakland residents, including premature mortality, increased hospital admissions and emergency department visits, and development of chronic respiratory disease. Therefore, the City of Oakland had adequate knowledge to conclude transport, storage, and handling of coal through the City and Port of Oakland posed a health risk to its residents. Specifically, the following were known:

- It was generally accepted within the scientific community that increased concentrations of $PM_{2.5}$ pollution are associated with adverse health outcomes among exposed populations including premature mortality, increased hospital admissions and emergency department visits, and development of chronic respiratory disease.[80]

[79] ESA. Report on the Health and/or Safety Impacts Associated with the Transport, Storage, and/or Handling of Coal and/or Coke in Oakland, including at the Proposed Oakland Bulk and Oversized Terminal in the West Gateway Area of the former Oakland Army Base. Prepared for the City of Oakland. June 23, 2016.

[80] US EPA. National ambient air quality standards for particulate matter. Final Rule. 40 CFR Parts 50, 51, 52, 53, and 58. Federal Register. 2013; 78(10):3086-287.

- It was generally accepted within the scientific community that at-risk (sensitive) populations for the adverse effects associated with $PM_{2.5}$ exposure were children, older adults, and persons with pre-existing heart and lung disease (asthmatics).[81]
- The West Oakland community had increased ratios of the population subgroups most vulnerable to the health impacts of pollution (children, older adults, and people with asthma).[82]
- The regional community of West Oakland was identified as the community within the BAAQMD likely to face the highest health risks from air pollution due to its relatively high emissions of toxic air contaminants, relatively high exposures of youth and seniors to toxic air contaminants, and relatively high levels of poverty.[83] It was also identified as one of the most adversely impacted communities by air pollution (e.g., relatively high emissions or exposures with increased ratios of populations most vulnerable to the health impacts of pollution).[84]
- The community of West Oakland has employed active measures to improve their historically poor air quality and reduce health risks posed by ambient air $PM_{2.5}$ pollution emission.
- In 2016 (at the time the Oakland City Council prohibited coal transport), West Oakland had nonattainment status for the 24-hr $PM_{2.5}$ NAAQS.
- Scientific literature established coal transported by rail, even in the presence of surfactants, released and contributed to ambient $PM_{2.5}$ pollution levels.[85]
- The ESA report estimated that transport, storage, and handling of coal in Oakland would result in fugitive dust emissions from rail cars during transport that would add additional burden to ambient $PM_{2.5}$ pollution levels in West Oakland specifically.[86]

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[81] US EPA. National ambient air quality standards for particulate matter. Final Rule. 40 CFR Parts 50, 51, 52, 53, and 58. Federal Register. 2013; 78(10):3086-287.

[82] BAAQMD. Community Air Risk Evaluation Program: Phase I Findings and Policy Recommendations Related to Toxic Air Contaminants in the San Francisco Bay Area. September 2006.

[83] Bay Area Air Quality Management District (BAAQMD). Applied Method for Developing Polygon Boundaries for CARE Impacted Communities. Technical Memorandum. December 2009.

[84] BAAQMD. Identifying Areas with Cumulative Impacts from Air Pollution in the San Francisco Bay Area - Version 2. March 2014.

[85] Jaffe, D., *et al.* Diesel particulate matter and coal dust from trains in the Columbia River Gorge, Washington State, USA. Atmospheric Pollution Research. 2015; 6(6):946-52.

[86] ESA. Report on the Health and/or Safety Impacts Associated with the Transport, Storage, and/or Handling of Coal and/or Coke in Oakland, including at the Proposed Oakland Bulk and Oversized Terminal in the West Gateway Area of the former Oakland Army Base. Prepared for the City of Oakland. June 23, 2016.

The City of Oakland had adequate knowledge to conclude transport, storage, and handling of coal through the City and Port of Oakland posed a health risk to its residents given the known adverse effects of $PM_{2.5}$ air pollution, the nonattainment status of the West Oakland community, the increased ratios of sensitive individuals within the West Oakland community, and the propensity for coal handling and transport to release and contribute to $PM_{2.5}$ levels. Adverse health effects associated with coal-associated $PM_{2.5}$ in the community of West Oakland include increased risk of premature mortality, increased hospital admissions and emergency department visits, and development of chronic respiratory disease.

Opinion 2: Modeling results provide added support that transport, storage, and handling of coal through the City and Port of Oakland poses an increased risk of adverse health effects to West Oakland residents, including premature mortality, increased hospital admissions and emergency department visits, and development of chronic respiratory disease

Background

Air dispersion modeling uses mathematical formulations to model atmospheric processes that disperse a pollutant emitted by a source. The modeling uses emission data (how much and how fast a pollutant is released) with local meteorological data (i.e., winds, air temperature, atmospheric stability, mixed layer heights, etc.) to simulate the transport and dispersion of emitted pollutants and estimate concentrations in the surrounding community.[87] For this matter, Dr. Gray used West Oakland meteorology data with specific emission rates involving coal transport (through West Oakland) and coal storage and handling (at the Port of Oakland) to model dispersion of coal-associated $PM_{2.5}$ in the community of West Oakland.[88]

I understand Dr. Gray's review of the report by the City's rail expert may, if appropriate, result in a revision of Dr. Gray's dispersion modeling. In that event, I will revise my opinions based on Dr. Gray's revisions for the dispersion of coal-associated $PM_{2.5}$ in the community of West Oakland.

[87] US EPA. Air quality dispersion modeling. Support Center for Regulatory Atmospheric Modeling (SCRAM). Last updated June 23, 2017. Available from: https://www.epa.gov/scram/air-quality-dispersion-modeling. Accessed: September 28, 2017.

[88] H. Andrew Gray. Expert Report. Modeling PM Air Quality Impacts of the Proposed OBOT Facility. October 6, 2017.

The Bay area historically records the highest concentrations of ambient $PM_{2.5}$ in winter, when consecutive days of stagnant and clear winter days facilitate a lesser sunlight-driven atmospheric temperature inversion layer.[89] The same pattern (i.e., highest concentrations observed in winter during atmospheric inversions) is expected in dispersion modeling, which incorporates site-specific meteorological data.

Modeled coal-associated $PM_{2.5}$ concentrations cause West Oakland's ambient air quality to exceed National (NAAQS) and State (CAAQS) air quality standards

Ambient air monitoring stations measure air concentrations of $PM_{2.5}$ to indicate communitywide exposure concentrations. $PM_{2.5}$ concentrations recorded at BAAQMD's "West Oakland" air monitoring station, located approximately one mile (generally downwind) from the Port of Oakland,[90] were previously shown to be representative and consistent with $PM_{2.5}$ concentrations throughout the West Oakland community[91] and therefore approximate "background" ambient air conditions for the West Oakland community (i.e., the background air quality without any input from coal dust).

The West Oakland monitor began recording ambient air concentrations in December 2012[92] and yearly data are available beginning for calendar year 2013. Because $PM_{2.5}$ can fluctuate from year to year due to natural conditions (e.g., weather, wildfires) as well as anthropogenic activities, I compared the estimated (modeled) coal-attributed $PM_{2.5}$ burden to ambient air quality at the BAAQMD West Oakland Monitor recorded for the three years prior to the City of Oakland's coal decision. Addition of the coal-attributed $PM_{2.5}$ to West Oakland's background air quality will estimate air quality conditions that may exist if coal operations were to occur in the community.

Dr. Gray provided a table of modeled ambient air concentrations for different locations where West Oakland community receptors (residents) would breathe.[93]

In estimating the total $PM_{2.5}$ concentrations among the West Oakland community expected if coal operations were to occur, I added West Oakland background concentrations to the maximum concentrations predicted in Dr. Gray's modeling. Using maximum values ensures all community locations comply with NAAQS and CAAQS. This approach (using

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[89] BAAQMD. Spare the Air: Cool the climate. A blueprint for clean air and climate protection in the Bay Area. Final 2017 Clean Air Plan. April 19, 2017., 2/17.

[90] BAAQMD. 2015 Air Monitoring Network Plan. July 1, 2016.

[91] Fujita, E.M., *et al.* Spatial variations of particulate matter and air toxics in communities adjacent to the Port of Oakland. Journal of the Air & Waste Management Association. 2013; 63(12):1399-411.

[92] BAAQMD. 2015 Air Monitoring Network Plan. July 1, 2016.

[93] H. Andrew Gray. Expert Report. Modeling PM Air Quality Impacts of the Proposed OBOT Facility. October 6, 2017.

maximum concentrations) also agrees with BAAQMD's requirements for selecting location of air monitors, which state that:

> *"Network design requirements for $PM_{2.5}$ monitors require sites…in areas of expected maximum concentrations….The primary objective of these maximum concentration SLAMS [state or local air monitoring station] is to determine compliance with the $PM_{2.5}$ NAAQS. Because the forms of the NAAQS are based on annual averages or the 98th percentile daily $PM_{2.5}$ concentrations, these sites should be located where the annual average or 98th percentile concentration are expected to be highest most years, even though another location may experience higher concentrations on a specific day."*[70]

As demonstrated in Table 5, addition of coal-attributed $PM_{2.5}$ concentrations to annual average background levels of $PM_{2.5}$ causes the West Oakland community to exceed both the national (NAAQS) and state (CAAQS) air quality standards for all evaluated years..

Table 5. Estimated Annual Average $PM_{2.5}$ Concentration in West Oakland from Coal Dust from Transport, Storage, and Handling of Coal through the City and Port of Oakland*

Background[94] West Oakland Annual average, µg/m^3 (year)	Maximum Estimated North Rail[95] Annual average, µg/m^3	Maximum Estimated South Rail[95] Annual average, µg/m^3	Total Annual average, North Rail plus Background (µg/m^3)	Total Annual average, South Rail plus Background (µg/m^3)	Comparison of Total to NAAQS and CAAQS	Ambient Air Quality Standards (NAAQS and CAAQS)
12.8 (2013)	3.2	3.1	16.0	15.9	Exceeds	12.0
9.5 (2014)			12.7	12.6	Exceeds	
10.1 (2015)			13.3	13.2	Exceeds	

*A graphical presentation of the data is included in Attachment 2.

As stated previously, daily $PM_{2.5}$ concentrations typically reach their highest values in winter due winter's characteristic meteorological conditions (i.e., atmospheric inversions). Thus, the same meteorological conditions (i.e., inversions trapping air near ground level and preventing dispersion) that contribute to the highest daily $PM_{2.5}$ concentrations in the West

[94] Annual average PM2.5 concentrations from the West Oakland Monitor as reported in: US EPA. Air Quality System Data Mart [internet database]. Last updated 05/26/2017. Available from: http://aqsdr1.epa.gov/aqsweb/aqstmp/airdata/download_files.html. Accessed: 9/26/2017.

[95] H. Andrew Gray. Expert Report. Modeling PM Air Quality Impacts of the Proposed OBOT Facility. October 6, 2017.

Oakland community will also drive the highest daily $PM_{2.5}$ concentrations in modeling coal operations-associated $PM_{2.5}$ concentrations in the West Oakland community.

In considering the 98th percentile for daily $PM_{2.5}$ concentrations, addition of airborne coal dust levels to community background levels causes the West Oakland community to exceed the national air quality standard for two of the three evaluated years (see Table 6). Furthermore, the estimated 98th percentile for the single year in compliance (e.g., 34.1 $\mu g/m^3$) was only barely below the NAAQS value (35 $\mu g/m^3$).

Table 6. Estimated Daily 98th Percentile $PM_{2.5}$ Concentration in West Oakland from Coal Dust from Transport, Storage, and Handling of Coal through the City and Port of Oakland*

Background[96] West Oakland Daily 98th percentile, $\mu g/m^3$ (year)	Maximum Estimated North Rail[97] Daily 98th percentile, $\mu g/m^3$	Maximum Estimated South Rail[97] Daily 98th percentile, $\mu g/m^3$	Total Daily 98th percentile, North Rail plus Background ($\mu g/m^3$)	Total Daily 98th percentile, South Rail plus Background ($\mu g/m^3$)	Comparison of Total to NAAQS	Ambient Air Quality Standard (NAAQS)
30.0 (2013)	8.4	8.4	38.4	38.4	Exceeds	35
25.7 (2014)			34.1	34.1	Does not exceed	
29.9 (2015)			38.3	38.3	Exceeds	

*A graphical presentation of the data is included in Attachment 2.

Conclusion

As detailed in the previous sections of my report:

- The BAAQMD (including the community of West Oakland) has had historically poor air quality.
- The community of West Oakland's $PM_{2.5}$ concentrations are among the highest concentrations within the BAAQMD.
- Dispersion modeling of coal operations in West Oakland indicate they will contribute to the relatively high ambient $PM_{2.5}$ air pollution levels in West Oakland to result in airborne concentrations that exceed annual and daily air quality standards.

[96] Daily 98th Percentile PM2.5 concentrations from the West Oakland Monitor as reported in: US EPA. Air Quality System Data Mart [internet database]. Last updated 05/26/2017. Available from: http://aqsdr1.epa.gov/aqsweb/aqstmp/airdata/download_files.html. Accessed: 9/26/2017.

[97] H. Andrew Gray. Expert Report. Modeling PM Air Quality Impacts of the Proposed OBOT Facility. October 6, 2017.

- Increased ambient airborne $PM_{2.5}$ concentrations are associated with an array of $PM_{2.5}$-related adverse health effects including premature mortality, increased hospital admissions and emergency department visits, worsening of asthma symptoms, and development of chronic respiratory disease, especially in sensitive populations (i.e., children, older adults, and persons with pre-existing heart or lung disease).[98]
- The West Oakland community has increased ratios of the population subgroups most vulnerable to the health impacts of pollution (children, older adults, and people with asthma).[99]
- $PM_{2.5}$ from coal-associated activities will cause the West Oakland community to have increased health risks for premature mortality, increased hospital admissions and emergency department visits, worsening of asthma symptoms, and development of chronic respiratory disease, especially in the sensitive populations.

Opinion 3: Smoke constituents from a coal fire will expose the West Oakland community to adverse health risks, including increased premature mortality and hospital admissions for cardiovascular and respiratory diseases

Coal

Coal is a combination of organic matter and inorganic mineral matter formed over eons from successive layers of fallen vegetation. Coal classification as anthracite, bituminous, subbituminous, or lignite occurs based upon its heating value and composition, specifically the relative amounts of fixed carbon, volatile matter, ash, sulfur, and moisture.[100] The

[98] California Air Resources Board. Ambient Air Quality Standards (AAQS) for Particulate Matter. California Environmental Protection Agency; Last updated October 25, 2015. Available from: https://www.arb.ca.gov/research/aaqs/pm/pm.htm. Accessed: 9/13/2017. ; Samet, J.M., *et al.* Particulate Matter. In: Air Quality Guidelines Global Update 2005: Particulate matter, ozone, nitrogen dioxide and sulfur dioxide World Health Organization. Germany: World Health Organization; Druckpartner Moser; 2006. p. 217-91.; US EPA. National ambient air quality standards for particulate matter. Final Rule. 40 CFR Parts 50, 51, 52, 53, and 58. Federal Register. 2013; 78(10):3086-287.

[99] BAAQMD. Community Air Risk Evaluation Program: Phase I Findings and Policy Recommendations Related to Toxic Air Contaminants in the San Francisco Bay Area. September 2006.

[100] Acurex Environmental Corporation for US EPA. Emission Factor Documentation For AP-42 Section 1.1 Bituminous and subbituminous coal combustion. Report No.: 68-DO-00120 April 1993.

chemical composition of coal also includes toxic metals, such as mercury, arsenic, lead, and cadmium.[101]

Coal burning in power plants

The availability and inexpensive cost has made coal burning a convenient source of energy. US coal-burning power plants are optimized for overall power plant operation, including lowest possible emissions, as well as maximum thermal efficiency, lowest possible cost, readily marketable by-products, and maximum system availability for power generation.[102]

Coal combustion, which generates primarily heat and carbon dioxide, is optimized within the furnace through burner flame temperature, mixing turbulence, and contact time. Organic compounds in combustion exhaust are due to incomplete combustion; their formation is reduced in power plant emissions through burner maintenance and maintaining a proper air/fuel mixture.[103] Despite optimization (e.g., combustion conditions) and emission mitigation (e.g., filtration) techniques, coal-fired power plants in the U.S. emit 84 of the 187 hazardous air pollutants identified by US EPA as posing a threat to human health and the environment.[104]

Constituents released into West Oakland community air from a coal fire will generate health hazards associated with respiration and exposure to those compounds

The risk from fire exists anywhere significant amounts of coal are in use or storage. Coal is a combustible material, making it susceptible to a variety of ignition scenarios, one of the most frequent and serious being spontaneous combustion.[105] Because coal can react with the oxygen in the air to initiate self-heating, spontaneous combustion may occur during any

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[101] Panel on the Trace Element Geochemistry of Coal Resource Development Related to Health*, et al.* Trace-Element Geochemistry of Coal Resource Development Related to Environmental Quality and Health. Washington, DC.p.19-20; Klaassen, C.D. Casarett and Doull's Toxicology: The Basic Science of Poisons. 8th ed. New York: McGraw-Hill; 2013. p.981-1001.

[102] Clean Coal Technology. Power Plant Optimization Demonstration Projects. Topical Report Number 25, September 2007, p.5.

[103] TSI Incorporated. Combustion analysis basics: An overview of measurements, methods and calculations used in combustion analysis, p. 1-8.

[104] Environmental Health & Engineering, Inc. Emissions of hazardous air pollutants from coal-fired power plants - Prepared for American Lung Association. Report No.: 17505 March 7, 2011.

[105] US DOE. Environmental Safety and Health Bulletin: The Fire Below: Spontaneous Combustion in Coal - How coal self-ignites. Report No.: EH 93-4 May 1993.

stage of storage, handling and transport between the mine and the site of ultimate coal usage (utility or industry boiler).[106]

Knowledge that coal burning generates large amounts of pollutants (carbon dioxide, PM_{10}, $PM_{2.5}$, organic compounds, sulfur and nitrogen oxides, and toxic metals) has led to active efforts by modern coal-fired power plants to minimize pollutant release into the atmosphere. These efforts include both controlling combustion conditions and employing post-combustion pollution control systems.[107]

Smoke emissions from a fire that may occur during transport, storage, or handling bulk coal in the bulk and oversized terminal facility will be different from coal-fired power plant exhaust: the burn will not occur under optimal conditions and the smoke will not be subject to emission control technologies. Furthermore, coal burning in non-optimal (i.e., less efficient) conditions results in production of a wide range of partially oxidized by-products including benzene, toluene, and xylene.[108] In addition, lack of mitigation measures will result in airborne release of other potentially toxic compounds generated during coal combustion, including PM and carcinogenic polycyclic aromatic hydrocarbons (PAHs; a group of organic chemicals containing only carbon and hydrogen with two or more fused aromatic [benzene] rings).[109] Therefore, when compared to power plants, which utilize both optimized combustion parameters and mitigation techniques to diminish emissions,[110] emissions from a coal fire are anticipated to release more chemical compounds and, depending on the volume of the fire, potentially increased concentrations of constituents characterized in power plant emissions. As depicted in Table 7, constituents of coal-fired power plant emissions[111] include compounds classified as carcinogens by the state of California[112] and compounds classified by the US EPA as hazardous air pollutants (HAPs, or "pollutants that are known or suspected to cause

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[106] Sloss, L.L. and IEA Clean Coal Centre. Assessing and managing spontaneous combustion of coal. Report No.: CCC/259 October 2015.

[107] Finkelman, R.B. Potential health impacts of burning coal beds and waste banks. International Journal of Coal Geology. 2004; 59(1):19-24.

[108] Melody, S.M. and Johnston, F.H. Coal mine fires and human health: What do we know? International Journal of Coal Geology. 2015; 152:1-14.

[109] ATSDR. Toxicological Profile for Polycyclic Aromatic Hydrocarbons. Atlanta, GA. August 1995.; World Health Organization. WHO Guidelines for Indoor Air Quality: Selected Pollutants. Copenhagen. 2010., p. 289; Reisen, F.*, et al.* Characteristics of an open-cut coal mine fire pollution event. Atmospheric Environment. 2017; 151:140-51.

[110] Finkelman, R.B. Potential health impacts of burning coal beds and waste banks. International Journal of Coal Geology. 2004; 59(1):19-24.

[111] American Lung Association. Toxic Air - The Case for Cleaning Up Coal-fired Power Plants. March 2011.

[112] California Office of Environmental Health Hazard Assessment (OEHHA). Chemicals Known to the State to Cause Cancer or Reproductive Toxicity. Proposition 65 List of Chemicals. July 7, 2017.

cancer or other serious health effects, such as reproductive effects or birth defects, or adverse environmental effects").[113]

Extensive searching of the literature found no literature regarding emission constituents or concentrations from coal fires that occurred during transport or at storage or transfer facilities. In contrast, scientific literature was found regarding characterization of constituents emitted during coal mine fires. Emissions characterized during an open-pit coal mine fire included elevated $PM_{2.5}$, benzene, toluene, ethylbenzene, xylenes, formaldehyde, and benzo[a]pyrene.[114] Another study demonstrated toxic metals are also released during coal fires as they quantitated airborne mercury in emissions from an underground coal mine fire.[115]

Fires can volatilize heavy metals from the coal (such as mercury, arsenic, lead) and release organic pollutants (such as HAPs and PAHs), each representing different health hazards for the surrounding population.

Overall, coal fires generate and release many toxic compounds, including several known carcinogens, into the air. As summarized in Table 7, constituents released into the West Oakland community air during a coal fire will introduce health hazards into the community, including increased risk of:

- Hospital admissions and emergency department visits, cancer, and premature mortality;
- Adverse cardiovascular, dermal (skin), developmental, kidney, liver and respiratory tract effects; and
- Adverse effects to the hematological (blood), immune, nervous, and reproductive systems.

[113] US EPA. What are Hazardous Air Pollutants? Last updated February 9, 2017 Date Accessed: 8/31/2017. Available from: https://www.epa.gov/haps/what-are-hazardous-air-pollutants. 2017.

[114] Reisen, F., *et al.* Characteristics of an open-cut coal mine fire pollution event. Atmospheric Environment. 2017; 151:140-51.

[115] Hower, J.C., *et al.* Gaseous emissions and sublimates from the Truman Shepherd coal fire, Floyd County, Kentucky: A re-investigation following attempted mitigation of the fire. International Journal of Coal Geology. 2013; 116:63-74.

Table 7. Selected pollutants released from burning of coal in coal-fired power plants (adapted from American Lung Association, 2011)[116]

Class	Notable Pollutants	Human Health Hazard(s)[A]	US EPA HAP[B]	CA Prop-65 Carcinogen[C]
Volatile organic compounds (VOCs)	Benzene	Adversely effects hematological (blood) and neurological systems. May adversely affect immune system.[117]	Yes	Yes
	Toluene	Adversely effects nervous system. May adversely affect immune, kidney, liver, reproductive systems and fetal development[118]	Yes	No
	Ethylbenzene	Eye and respiratory tract irritation and ototoxicity (inner ear damage). May adversely affect kidney[119]	Yes	Yes
	Xylene	Adversely effects nervous system; eye, skin, and respiratory tract irritation. May impair respiratory function or adversely affect kidney or liver[120]	Yes	-
	Formaldehyde	Eye and respiratory tract irritation[121]	Yes	Yes
Polycyclic aromatic hydrocarbons (PAHs)	Benz[a]anthracene	Site of tumor induction by PAHs influenced by route of exposure (stomach, ingestion; lung; inhalation; skin, dermal). May effect proliferating tissues such as bone marrow, lymphoid organs, gonads, and intestinal epithelium[122]	-	Yes
	Benzo[b]fluoranthene		-	Yes
	Chrysene		-	Yes
	Dibenz[a,h]anthracene		-	Yes
	Benzo[a]pyrene	May cause developmental effects, reproductive effects, and immunotoxicity; carcinogenic at multiple tumor cites including alimentary tract, liver, kidney, respiratory tract, and skin[123]	-	Yes
	Naphthalene	Hematological effects (hemolytic anemia)[124]	Yes	-

[116] American Lung Association. State of the Air 2017. Chicago, IL.

[117] US EPA. Toxicological Review of Benzene (Noncancer Effects) (CAS No. 71-43-2). Washington DC. Report No.: EPA/635/R-02/001F October 2002.

[118] ATSDR. Toxicological profile for Toluene. Atlanta, GA. June 2017.

[119] ATSDR. Toxicological profile for Ethylbenzene. Atlanta, GA. November 2010.

[120] ATSDR. Toxicological profile for Xylene. Atlanta, GA. August 2007.

[121] US EPA. Integrated Risk Information System (IRIS) Chemical Assessment Summary: Formaldehyde; CASRN 50-00-0. October 1, 1989.; ATSDR. Toxicological Profile for Formaldehyde. Atlanta, GA. July 1999.

[122] ATSDR. Toxicological Profile for Polycyclic Aromatic Hydrocarbons. Atlanta, GA. August 1995.

[123] US EPA. Integrated Risk Information System (IRIS) Executive Summary: Toxicological review of benzo[a]pyrene; CASRN 50-32-8. Washington, DC. January 2017.

[124] ATSDR. Toxicological Profile for Naphthalene, 1-Methylnaphthalene, and 2-Methylnaphthalene. Atlanta, GA. August 2005.

Table 7 (continued). Selected pollutants released from burning of coal in coal-fired power plants (adapted from American Lung Association, 2011)[116]

Class	Notable Pollutants	Human Health Hazard(s)[A]	US EPA HAP[B]	CA Prop-65 Carcinogen[C]
Metals & Metalloids	Mercury	Respiratory, renal, neurological, and cardiovascular system effects[125]	Yes	No
	Lead	Developmental neurotoxin; adversely effects hematological, renal, and cardiovascular systems[126]	Yes	Yes
	Arsenic	Respiratory tract irritation; Dermal, neurological, and circulatory system effects[127]	Yes	Yes
	Cadmium	Respiratory and renal effects[128]	Yes	Yes
	Chromium	Respiratory and hematological effects. May cause reproductive effects[129]	Yes	Yes (chromiumVI)
	Manganese	Respiratory and neurological effects[130]	Yes	-
	Nickel	Respiratory and immune effects; contact dermatitis[131]	Yes	Yes
	Selenium	Respiratory effects. May effect hepatic, nervous, cardiovascular, and renal systems[132]	Yes	Yes (as selenium sulfide)
	Antimony	Eye, skin, and respiratory tract irritation; May effect cardiovascular and respiratory systems[133]	Yes	Yes (antimony trioxide)
	Beryllium	Respiratory effects[134]	Yes	Yes
	Cobalt	Respiratory, thyroid, and cardiac effects; allergic dermatitis[135]	Yes	Yes

[125] ATSDR. Toxicological Profile for Mercury. Atlanta, GA. March 1999.
[126] ATSDR. Toxicological Profile for Lead. Atlanta, GA. August 2007.
[127] ATSDR. Toxicological Profile for Arsenic. Atlanta, GA. August 2007.
[128] ATSDR. Toxicological Profile for Cadmium. Atlanta, GA. September 2012.
[129] ATSDR. Toxicological Profile for Chromium. Atlanta, Georgia. September 2012.
[130] ATSDR. Toxicological Profile for Manganese. Atlanta, GA. September 2012.
[131] ATSDR. Toxicological Profile for Nickel. Atlanta, GA. August 2005.
[132] ATSDR. Toxicological Profile for Selenium. Atlanta, GA. September 2003.
[133] ATSDR. Toxicological profile for antimony and compounds. Atlanta, GA. 1992.
[134] ATSDR. Toxicological Profile for Beryllium. Atlanta, GA. September 2002.
[135] ATSDR. Toxicological profile for Cobalt. Atlanta, GA. April 2004.

Table 7 (continued). Selected pollutants released from burning of coal in coal-fired power plants (adapted from American Lung Association, 2011)[116]

Class	Notable Pollutants	Human Health Hazard(s)[A]	US EPA HAP[B]	CA Prop-65 Carcinogen[C]
Particulate Matter	$PM_{2.5}$	Premature mortality, increased hospital admissions and emergency department visits, and development of chronic respiratory disease.[136]	- [Criteria Air Pollutant]	-
Dioxin	2,3,7,8-Tetrachlorodioxin	Dermal effects; May effect immune, endocrine, and developmental systems[137]	Yes	Yes
Acid gases	Hydrogen chloride	Eye, skin, and respiratory tract irritation. May cause corrosive injuries to exposed tissues[138]	-	-
	Hydrogen fluoride	Skin, eyes, and respiratory tract irritation. May harm lungs and heart[139]	Yes	-
	Hydrogen cyanide	Respiratory tract irritation; neurological, respiratory, cardiovascular, and thyroid effects[140]	Yes	-

[A] Effects on human health depend on concentration, route of exposure, and the length of the exposure; [B] Hazardous Air Pollutant (HAP) for which emissions are regulated under the Clean Air Act by the US EPA;[141] [C] Listed as a carcinogen by the State of California[142]

In the event of a coal fire, West Oakland's $PM_{2.5}$ levels will sharply rise, far exceeding National (NAAQS) standards

Dr. Gray modeled ambient $PM_{2.5}$ air concentrations that could result from a medium- or large-size fire (assumed to burn across the top surface area of a rail car and across ten percent of a ship's coal surface, respectively) and provided a summary table of the resulting daily 98th percentile values for different locations where West Oakland community receptors (residents) would be exposed.[143] The same comparison described earlier was performed (see

[136] US EPA. National ambient air quality standards for particulate matter. Final Rule. 40 CFR Parts 50, 51, 52, 53, and 58. Federal Register. 2013; 78(10):3086-287.
[137] ATSDR. Toxicological Profile for Chlorinated Dibenzo-p-Dioxins. Atlanta, GA: 1998. US DHHS. December 1998.
[138] ATSDR. ToxFAQs: Hydrogen chloride. Atlanta, GA. April 2002.
[139] ATSDR. Toxicological Profile for Fluorides, Hydrogen Fluoride, and Fluorine. Atlanta, GA. September 2003.
[140] ATSDR. Toxicological Profile for Cyanide. Atlanta, GA. July 2006.
[141] US EPA. Initial List of Hazardous Air Pollutants with Modifications. Last updated March 16, 2017 Date Accessed: 8/29/2017. Available from: https://www.epa.gov/haps/initial-list-hazardous-air-pollutants-modifications. 2017.
[142] California Office of Environmental Health Hazard Assessment (OEHHA). Chemicals Known to the State to Cause Cancer or Reproductive Toxicity. Proposition 65 List of Chemicals. July 7, 2017.
[143] H. Andrew Gray. Expert Report. Modeling PM Air Quality Impacts of the Proposed OBOT Facility. October 6, 2017.

pages 35-37) to consider the effects of smoke from a coal fire on ambient $PM_{2.5}$ concentrations in the West Oakland community. This analysis included estimations for historical background concentrations as well as total ambient concentrations estimated to occur during transport, storage, and handling of coal in West Oakland (see Table 6 and associated discussion). As summarized in Table 8, addition of airborne coal dust levels to community background levels dramatically elevates $PM_{2.5}$ concentrations and causes the West Oakland community to far exceed the national air quality standard for all considered scenarios. Increased short-term exposures to $PM_{2.5}$ increase community risk for premature mortality and increased hospital admissions for an array of health effects, including aggravation of respiratory and cardiovascular disease (e.g., irregular heartbeat, heart attack/myocardial infarction, stroke, and exacerbation of asthma and COPD).[144]

[144] Bell, M.L., *et al.* Seasonal and regional short-term effects of fine particles on hospital admissions in 202 US counties, 1999-2005. Am J Epidemiol. 2008; 168(11):1301-10.; Dominici, F., *et al.* Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. Jama. 2006; 295(10):1127-34.; Moolgavkar, S.H. Air pollution and daily deaths and hospital admissions in Los Angeles and Cook Counties. In: HEI Special Report Revised Analyses of Time-Series Studies of Air Pollution and Health Revised Analyses of the National Morbidity, Mortality, and Air Pollution Study, Part II Revised Analyses of Selected Time-Series Studies. Boston, MA: Health Effects Institute; 2003. p. 183-98.; Ostro, B., *et al.* The effects of fine particle components on respiratory hospital admissions in children. Environ Health Perspect. 2009; 117(3):475-80.; US EPA. Air quality designations for the 2012 primary annual fine particle (PM2.5) national ambient air quality standards (NAAQS). Final Rule. 40 CFR Part 81. Federal Register. 2015; 80(10):2206-84.

Table 8. Estimated Daily 98th Percentile $PM_{2.5}$ Concentration in West Oakland from $PM_{2.5}$ Emissions from Coal Fire Scenarios[B]

Background[145] West Oakland Daily 98th percentile, µg/m³ (year)	Maximum Estimated Hopper A Medium Fire[146] Daily 98th percentile, µg/m³	Maximum Estimated Transloading Large Fire[146] Daily 98th percentile, µg/m³	Total Daily 98th percentile, Medium Fire plus Background (µg/m³)	Total Daily 98th percentile, Large Fire plus Background (µg/m³)	Comparison of Total to NAAQS	Ambient Air Quality Standard (NAAQS)
30.0 (2013)	20.5	11.7	50.5	41.7	Exceeds	35
25.7 (2014)			46.2	37.4	Exceeds	
29.9 (2015)			50.4	41.6	Exceeds	
38.4 (2013)[A]			58.9[A]	50.1[A]	Exceeds	
34.1 (2014)[A]			54.6[A]	45.8[A]	Exceeds	
38.3 (2015)[A]			58.8[A]	50.0[A]	Exceeds	

[A]Historical background with additional contribution from transport, storage, and handling of coal (see Table 6)

[B]A graphical presentation of the data is included in Attachment 2

Conclusion

As detailed in the previous sections of my report:

- A coal fire will release hazardous air constituents into the community of West Oakland, including hazardous air pollutants and several known carcinogens.
- Exposure to constituents released into the West Oakland community air during a coal fire will introduce health hazards into the community, including increased risk of hospital admissions and emergency department visits, cancer, and premature mortality; adverse cardiovascular, dermal (skin), developmental, kidney, liver and respiratory tract effects; and adverse effects to the hematological (blood), immune, nervous, and reproductive systems
- A coal fire will release many compounds listed as carcinogens by the State of California, including volatile organic compounds, polycyclic aromatic hydrocarbons, dioxin, and heavy metals and metalloids.

[145] Daily 98th Percentile PM2.5 concentrations from the West Oakland Monitor as reported in: US EPA. Air Quality System Data Mart [internet database]. Last updated 05/26/2017. Available from: http://aqsdr1.epa.gov/aqsweb/aqstmp/airdata/download_files.html. Accessed: 9/26/2017.

[146] H. Andrew Gray. Expert Report. Modeling PM Air Quality Impacts of the Proposed OBOT Facility. October 6, 2017.

- Dispersion modeling of coal fire scenarios in West Oakland indicate they will contribute to elevated ambient $PM_{2.5}$ air pollution levels in West Oakland to result in airborne concentrations that far exceed daily air quality standards.
- Short-term increases in ambient $PM_{2.5}$ from a coal fire will cause the West Oakland community to have increased health risks for premature mortality and increased hospital admissions for an array of health effects, including aggravation of respiratory and cardiovascular disease (e.g., irregular heartbeats, heart attack/myocardial infarction, stroke, and exacerbation of asthma and COPD).

Overall Conclusions

Increased levels of $PM_{2.5}$ air pollution are associated with adverse health outcomes among exposed populations including premature mortality, increased hospital admissions and emergency department visits, and development of chronic respiratory disease, especially in vulnerable populations.

The West Oakland community has historically suffered from increased risks to the adverse health effects of air pollution due to its poor air quality and increased ratios of subpopulations most vulnerable to the health impacts of pollution. Impacted subpopulations include infants and young children, the elderly, and people with asthma.

The following criteria were available to the Oakland City Council in 2016, when it concluded transport, storage, and handling of coal through the City and Port posed a health risk to its residents:

- West Oakland was a non-attainment area for the national ambient air quality standard for $PM_{2.5}$, a level established by the US EPA to provide increased protection for at-risk (sensitive) populations against $PM_{2.5}$-related adverse health effects.
- Extensive studies and mitigation strategies by the BAAQMD, the CARB, and the Port of Oakland aimed to characterize and reduce the health risks posed to West Oakland residents by ambient $PM_{2.5}$ levels were on-going (and continue through the date of my report). Introduction of a major coal-handling facility in the West Oakland area will significantly increase ambient $PM_{2.5}$ levels, undermining the Port's and City's goals of reducing airborne pollution levels.

- Scientific literature established coal transport by rail, even in the presence of surfactants, releases and contributes to ambient $PM_{2.5}$ pollution levels.

Given the adverse effects of $PM_{2.5}$ air pollution, the nonattainment status of the West Oakland community, the increased ratios of sensitive individuals within the West Oakland community, and the propensity for coal handling and transport to release and contribute to $PM_{2.5}$ levels, the City of Oakland had adequate knowledge to conclude transport, storage, and handling of coal through the City and Port of Oakland posed health risks to its residents. Health risks due to the coal-associated contribution to ambient $PM_{2.5}$ pollution levels include:

- Premature mortality,
- Increased hospital admissions and emergency department visits, and
- Development of chronic respiratory disease.

The conclusion that OBOT's proposed rail-based coal transport, transfer, and shiploading facility posed health risks to West Oakland residents was reaffirmed though analysis of dispersion modeling results. The magnitude of coal dust contributions to $PM_{2.5}$ pollution levels in the West Oakland community (estimated through addition of the dispersion modeling estimates to background levels) demonstrated that resulting concentrations would exceed national and state ambient air quality standards and pose increased health risks for the community.

If a coal fire were to occur, it would be associated with release of carcinogens and hazardous air constituents into the community of West Oakland. Dispersion modeling of different fire scenarios indicates they will each result in $PM_{2.5}$ air pollution levels in excess of national air quality standards, which will pose increased health risks for the community.

To the extent that additional information becomes available, I may modify or add to my current opinions.

Hope (Nadia) Moore, PhD, DABT, ERT
Senior Toxicologist

06 October 2017
Date

Attachment 1: CV, Hope (Nadia) Moore, PhD, DABT, ERT
Attachment 2: $PM_{2.5}$ Graphs

Attachment 1: CV, Hope (Nadia) Moore, PhD, DABT, ATS

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Hope (Nadia) Moore, PhD, DABT, ERT

Education

Ph.D., Toxicology, University of Washington, Seattle WA, 2008

B.S., Chemistry, Pacific Lutheran University, Tacoma, WA, 1992

Certifications

Diplomate of the American Board of Toxicology, 2012-present

Registered Toxicologist (United Kingdom and EUROTOX registries), 2015-present

NIOSH Spirometry Testing Certification, 2015-present

Project Management Professional, 2011-2014

Professional Affiliations and Associated Appointments

American Board of Toxicology

Society of Toxicology (SOT), Full member
- Inhalation and Respiratory Specialty Section member
- Nanotoxicology Specialty Section member
- Women in Toxicology Interest Group member
  - WIT Councilor (2016-2018 term)
- Pacific Northwest Association of Toxicologists (PANWAT) member
  - PANWAT Councilor (2014-2016 term)
  - PANWAT Vice President Elect (2016-2017 term)
  - PANWAT Vice President (2017-2018 term)

American College of Toxicology (ACT), Full member

British Toxicology Society (BTS), Member

American College of Occupational and Environmental Medicine (ACOEM), Associate member

American Association for the Advancement of Science (AAAS), Member

American Conference of Governmental Industrial Hygienists (ACGIH), Voting member

American Chemical Society (ACS), Member

American Industrial Hygiene Association (AIHA), Full member

Society for Experimental Biology and Medicine (SEBM), Associate member

Hope (Nadia) Moore, PhD, DABT, ERT

Other Professional Activities

Practicing Scientist Member, Institutional Animal Care and Use Committee (IACUC) Pacific Northwest National Laboratory, Sequim Laboratory, and the Columbus-based Toxicology Laboratory (ToxNW), 2011 - 2013

Invited lecturer, Fundamentals of Toxicology Graduate Course ENVH514, University of Washington (Lung: Structure, Function, Absorption, & Inhalation)

Invited reviewer, Food and Chemical Toxicology (Elsevier Sciences), Human and Experimental Toxicology (Sage Journals), NeuroToxicology (Elsevier Sciences), Toxicology Letters (Elsevier Sciences), and Toxicological Sciences (Oxford Journals)

Experience

2013 - Present **Veritox®, Inc.** **Redmond, Washington**
Senior Toxicologist

2008-2013 **Battelle Toxicology Northwest** **Richland, Washington**
Pharmacologist / Safety Toxicologist

2003-2008 **University of Washington** **Seattle, Washington**
Toxicology Doctoral Student / Teaching Assistant / Research Assistant

2001-2003 **Battelle Pacific NW National Laboratory** **Richland, Washington**
Senior Research Scientist

2000-2001 **Battelle Toxicology Northwest** **Richland, Washington**
Principal Research Scientist

1992-2000 **Battelle Toxicology Northwest** **Richland, Washington**
Research Scientist / Technical Specialist /Technician

Professional Honors

National Toxicology Program Toxicology Discipline Leader Battelle Toxicology Northwest, Richland, WA (2011-2013)

Outstanding Performance Award in recognition of Outstanding Efforts as Study Director. Battelle Toxicology Northwest, Richland, WA (2009)

Pre-Doctoral Fellow National Institute of Environmental Health Sciences Environmental Pathology and Toxicology Training Grant.
University of Washington, Seattle, WA (2005-2008)

Outstanding Student Poster Award Recipient from the PANWAT (Pacific Northwest Association of Toxicologists, Regional Chapter of the Society of Toxicology) Annual Meeting (2007)

Hope (Nadia) Moore, PhD, DABT, ERT

Student Merit Meeting Award Recipient from the Research Society on Alcoholism, 30th Annual Meeting of the Research Society on Alcoholism, Chicago, IL (2007)

Selected Publications

TJ Mast, F Adeshina, N Moore, H Choudhury, A Protzel, and A Mahfouz. 2002. Identification of common toxic effects with common mechanisms of toxicity for pesticides selected from the drinking water contaminant list (CCL). *The Toxicologist,* Supplement to Toxicological Sciences, 66(1):494.

F Adeshina, T Mast, N Moore, A Mahouz, A Protzel, and H Choudhury. 2003. Identifying triazine herbicides on EPA drinking water contaminant candidate list (CCL) for common mechanism of toxicity and cumulative risk assessment. *The Toxicologist,* Supplement to Toxicological Sciences, 72(1):436.

N Moore, M Guizzetti, B Gallis, S Shaffer, DR Goodlett, and LG Costa. 2006. Use of proteomic approaches for the identification of changes in astrocyte secretion following ethanol exposure. *The Toxicologist,* Supplement to Toxicological Sciences, 90(1):1437.

M Guizzetti, G Giordano, N Moore, and LG Costa. 2008. Ethanol inhibits hippocampal neuron differentiation induced by carbachol-treated astrocytes. *The Toxicologist,* Supplement to Toxicological Sciences, 102(1):1963.

M Guizzetti, NH Moore, G Giordano, and LG Costa. 2008. Modulation of neuritogenesis by astrocyte muscarinic receptors. *J. Biol. Chem.* 283(46): 31884-31897.

N Moore, M Guizzetti, G Giordano, and LG Costa. 2008. Ethanol inhibits muscarinic receptor-induced release by astrocytes of extracellular proteins involved in neuronal development. *The Toxicologist,* Supplement to Toxicological Sciences, 102(1):1964.

NH Moore, LG Costa, SA Shaffer, DR Goodlett, and M Guizzetti. 2009. Shotgun proteomics implicates extracellular matrix proteins and protease systems in neuronal development induced by astrocyte cholinergic stimulation. *J. Neurochem.* 108(4):891-908.

M Guizzetti, NH Moore, G Giordano, KL VanDeMark, and LG Costa. 2010. Ethanol inhibits neuritogenesis induced by astrocyte muscarinic receptors. *Glia.* 58(12):1395-406.

M Guizzetti, NH Moore, KL VanDeMark, G Giordano, and LG Costa. 2011. Muscarinic receptor-activated signal transduction pathways involved in the neuritogenic effect of astrocytes in hippocampal neurons. *Eur. J. Pharmacol*. 659(2-3):102-7.

CY Chan, LJ Swenson, J Hobden, N Moore, and BJ Kelman. 2014. Risk from exposure to triorthocresyl phosphate (TOCP) in aircraft cabins and flight decks. *The Toxicologist,* Supplement to Toxicological Sciences, 138(1):2247.

BC Sayers, MD Stout, MF Cesta, N Moore, GL Baker, KM Patton, BK Hayden, JA Dill, and NJ Walker. 2014. Thirty-day whole-body inhalation toxicity and tissue burden study of multiwalled carbon nanotubes in Harlan Sprague-Dawley rats and B6C3F1 mice. *The Toxicologist,* Supplement to Toxicological Sciences, 138(1):2004A.

N Moore, CY Chan, M Krause, and BJ Kelman. 2015. Risk from traffic-related air pollution in schools: beyond distance to roadway. 2015. *The Toxicologist,* Supplement to Toxicological Sciences, 149(1):577.

CY Chan, M Krause, B Kelman, and N Moore. 2016. Risk from breaches in portable, consumer sized lithium batteries. *The Toxicologist,* Supplement to Toxicological Sciences, 150(1):2671.

B Kelman, R Evoy, C Chan and N Moore. 2016. Fluoride: friend or foe. *The Toxicologist,* Supplement to Toxicological Sciences, 150(1):2277.

N Moore, B Hardin, C Robbins, and B Kelman. 2016. Smoker's Risk of Lung Cancer from Asbestos Exposure. *The Toxicologist,* Supplement to Toxicological Sciences, 150(1):2696.

BA Magnuson, MC Carakostas, NH Moore, SP Poulos, and AG Renwick. 2016. Biological fate of low calorie sweeteners. *Nutr Rev*. 74(11):670-689.

JA Deyo, KA Tucker, CY Chan, NH Moore, and BJ Kelman. 2017. Marijuana: the smoke hasn't cleared. *The Toxicologist,* Supplement to Toxicological Sciences, 151(1):1282.

BJ Kelman, CY Chan, NH Moore, and LC Diener. 2017. Weight-of-evidence assessment for polyhexamethylene guanidine and interstitial lung disease. *The Toxicologist,* Supplement to Toxicological Sciences, 151(1):1287.

Selected Continuing Education

Analytical Validation for the Pharmaceutical Industry, American Association of Pharmaceutical Scientists (AAPS). AAPS Workshop on Current Issues, Arlington, VA, 1998.

Bioanalytical Methods Validation – A Revisit with a Decade of Progress. Co-sponsored by AAPS and FDA, Arlington, VA, 2000.

GLP Essentials for Technical Staff. Debi Garvin, Instructor, West Coast Quality Training Institute, Richland, WA, 2001.

A Practical Approach to Blood and Lymphoid Tissue (BLT) in Toxicology Assessments. JCL Schuh and L Lanning, Chairpersons. Society of Toxicology Continuing Education Course, Nashville, TN, 2002.

Good Laboratory Practices for Study Directors and Monitors. D Garvin, Instructor and Director, West Coast Quality Training Institute, Hood River, OR, 2008.

Introduction to Good Laboratory Practice Regulations. D Garvin, Instructor and Director, West Coast Quality Training Institute, Hood River, OR, 2008.

Primer in Pathology: Interpreting and Integrating Nonclinical Study Results. Continuing Education Course. Pacific Northwest Chapter of the Society of Toxicology, Pacific Northwest Association of Toxicologists Annual Meeting, Seattle, WA, 2009.

Stress as a Confounding Factor in Toxicology Studies. K Sprugel and N Everds, Chairpersons. Society of Toxicology Continuing Education Course, Baltimore, MD, 2009.

Immunology for Toxicologists. R Pieters and I Kimber, Chairpersons. Society of Toxicology Continuing Education Course, Baltimore, MD, 2009.

Integrative Toxicity Test Methods to Improve Hazard Identification. Society of Toxicology Pacific Northwest Chapter, Pacific Northwest Association of Toxicologists Annual Meeting, Corvallis, OR, 2010.

Segment-Specific Renal Pathology for the Non-Pathologist. D Hoivik and SG Emeigh Hart, Chairpersons. Society of Toxicology Continuing Education Course, Salt Lake City, UT, 2010.

Evaluating Toxicity of Engineered Nanomaterials: Issues with Conventional Toxicology Approaches. SS Nadadur and FA Witzmann, Chairpersons. Society of Toxicology Continuing Education Course, Washington DC, 2011.

Current Nonclinical Strategies and Methods for Evaluating Drug-Induced Cardiovascular Toxicity. H Wang and DJ. Murphy, Chairpersons. Society of Toxicology Continuing Education Course, Washington DC, 2011.

Northwest Association for Biomedical Research IACUC Education Conference, Seattle, WA, 2011.

Art and Science of Research Translation in Toxicology. Society of Toxicology Pacific Northwest Chapter, Pacific Northwest Association of Toxicologists Annual Meeting. North Bonneville, WA, 2011.

Mid America Toxicology Course, CD Klaassen, Course Director, Kansas City, Missouri, April 2012.

The What, When, and How of Nonclinical Support for an IND Submission. P Nugent and D Colagiovanni, Chairpersons. Society of Toxicology Continuing Education Course, San Antonio, TX, 2013.

Understanding Toxic Neuropathy in Drug Development: Both Clinical and Nonclinical Perspectives. MJ Kallman and J Benitez, Chairpersons. Society of Toxicology Continuing Education Course, San Antonio, TX, 2013.

Innovations in Methodologies for Inhalation Exposures and Interpretations of In Vivo Toxicity, Urmila Kodavanti and Juergen Pauluhn, Chairpersons. Society of Toxicology Continuing Education Course, Phoenix, AZ, 2014.

Methodologies in Human Health Risk Assessment, Qiyu (Jay) Zhao and M.E. (Bette) Meek, Chairpersons. Society of Toxicology Continuing Education Course, Phoenix, AZ, 2014.

Advances in Safety Assessment of Medical Devices, Niranjan S Goud and Ron Brown, Chairpersons. Society of Toxicology Continuing Education Course, San Diego, CA, 2015.

Hope (Nadia) Moore, PhD, DABT, ERT

New Horizons in Chemical Carcinogenesis: Advances in Mode of Action and Mechanism of Cancer Pathogenesis, James E Klaunig and Udayan M Apte, Chairpersons. Society of Toxicology Continuing Education Course, San Diego, CA, 2015.

The New World of Cancer Immunotherapy: Challenges in Bench to Bedside Translation, Rodney Prell and Rafael A Ponce, Chairpersons. Society of Toxicology Continuing Education Course, San Diego, CA, 2015.

OECD GLP and Documentation Training Course, Robin Guy and Dave Hobson, Instructors. Robin Guy Consulting, Morristown, NJ, Aug. 31 – Sept. 1, 2015.

Nanomaterials, Chemical Exposures and Control Banding: What Does It Mean for Workplace Safety? University of Washington Continuing Education Programs, hosted by the Pacific Northwest Section – American Industrial Hygiene Association. Oct. 14, 2015

NIOSH-Approved 2-Day Initial Spirometry Training Course, Martha Horike-Pyne, Instructor. University of Washington Continuing Education Programs, Seattle WA. Nov. 7-8, 2015.

Advancing the Detection, Imaging, and Pitfalls in Monitoring Oxidative Stress in Health and Disease. Maria B. Kadiiska and Ronald P. Mason, Chairpersons. Society of Toxicology Continuing Education Course, New Orleans, LA, 2016.

Basic Principles and Practices for Applying Epigenetics in Mechanistic Toxicology. Shaun D. McCullough and Ronald N. Hines, Chairpersons. Society of Toxicology Continuing Education Course, New Orleans, LA, 2016.

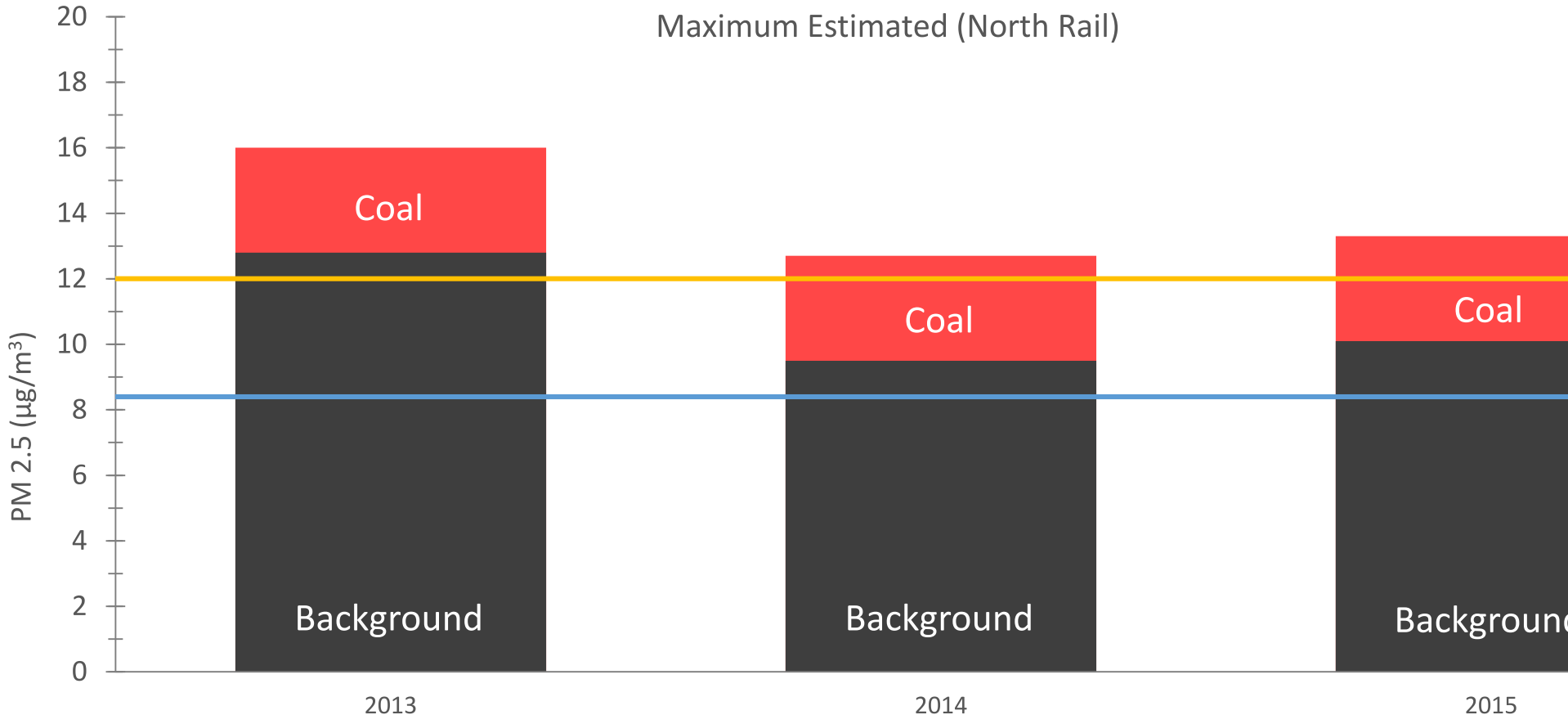
Human Health Risk Assessment: A Case Study Application of Principles. John C. Lipscomb and M.E. (Bette) Meek, Chairpersons. Society of Toxicology Continuing Education Course, New Orleans, LA, 2016.

Adding Up Chemicals: Component-Based Risk Assessment of Chemical Mixtures. Jane Ellen Simmons and Richard C. Hertzberg, Chairpersons. Society of Toxicology Continuing Education Course, Baltimore, MD, 2017.

Extrapolation in the Airways: Strategies to Incorporate *In Vivo* and *In Vitro* Data to Better Protect Human Health. Marie C. Fortin and Madhuri Singal, Chairpersons. Society of Toxicology Continuing Education Course, Baltimore, MD, 2017.

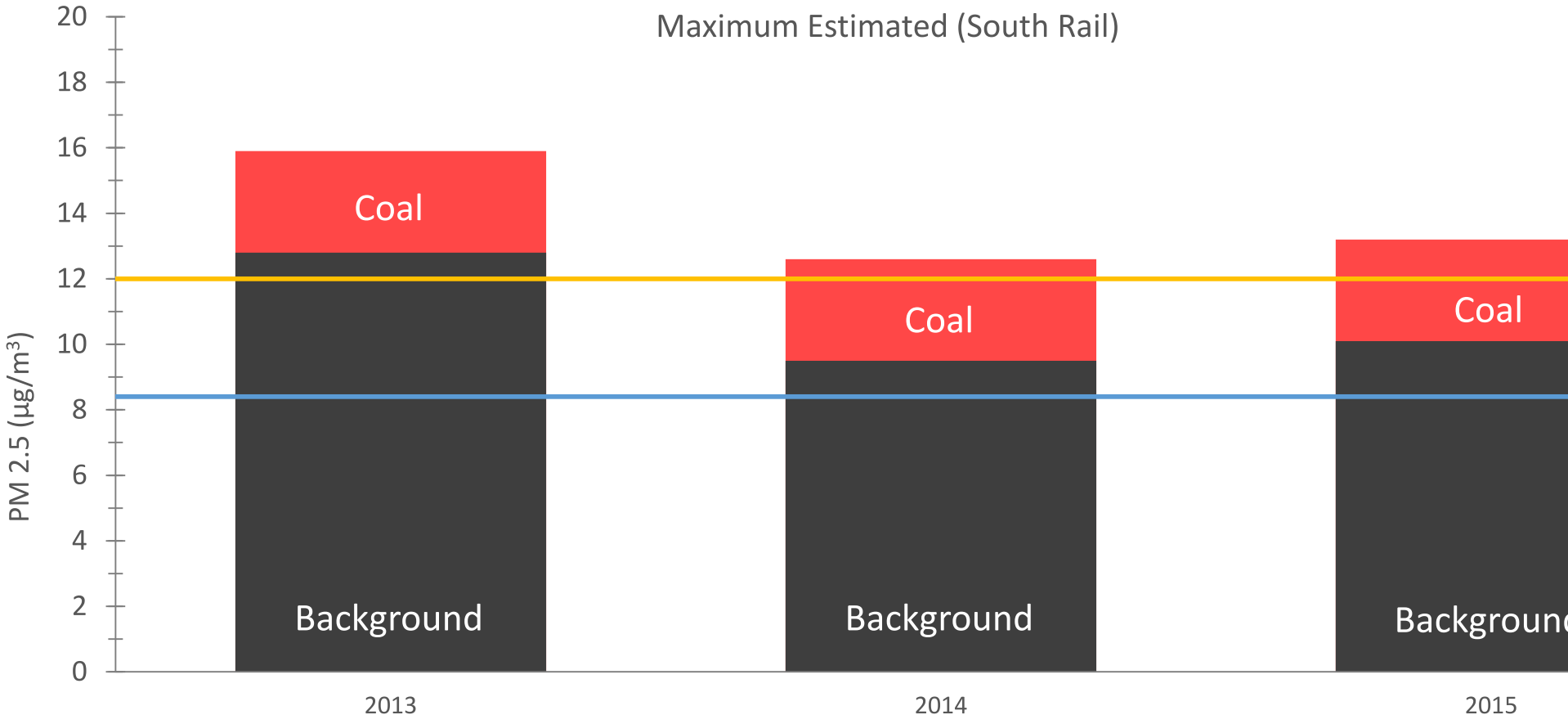
Attachment 2: $PM_{2.5}$ Graphs

[Table 5] Estimated **Annual** Average $PM_{2.5}$ Concentration in West Oakland from Coal Du from Transport, Storage, and Handling of Coa



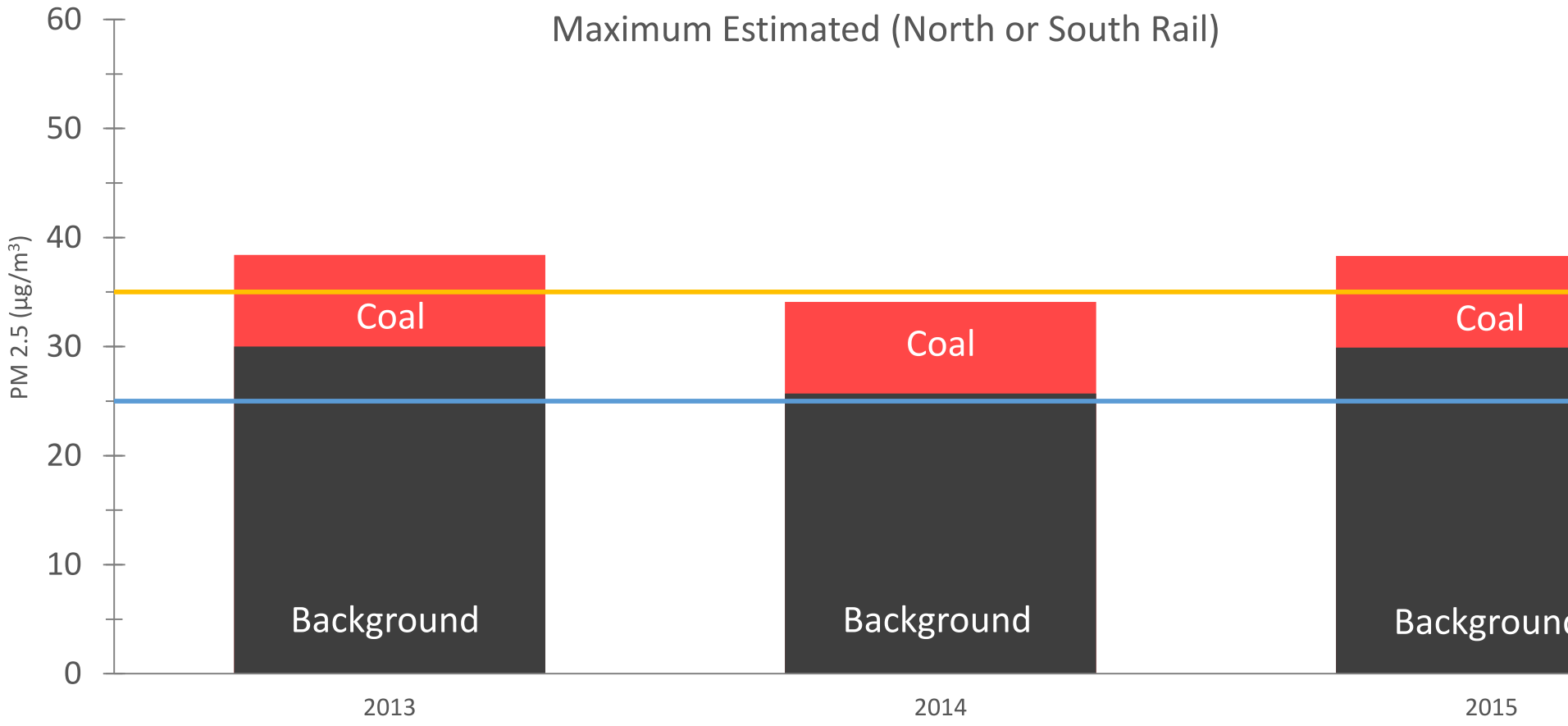
*DV for San Fra

[Table 5] Estimated **Annual** Average $PM_{2.5}$ Concentration in West Oakland from Coal Du from Transport, Storage, and Handling of Coa



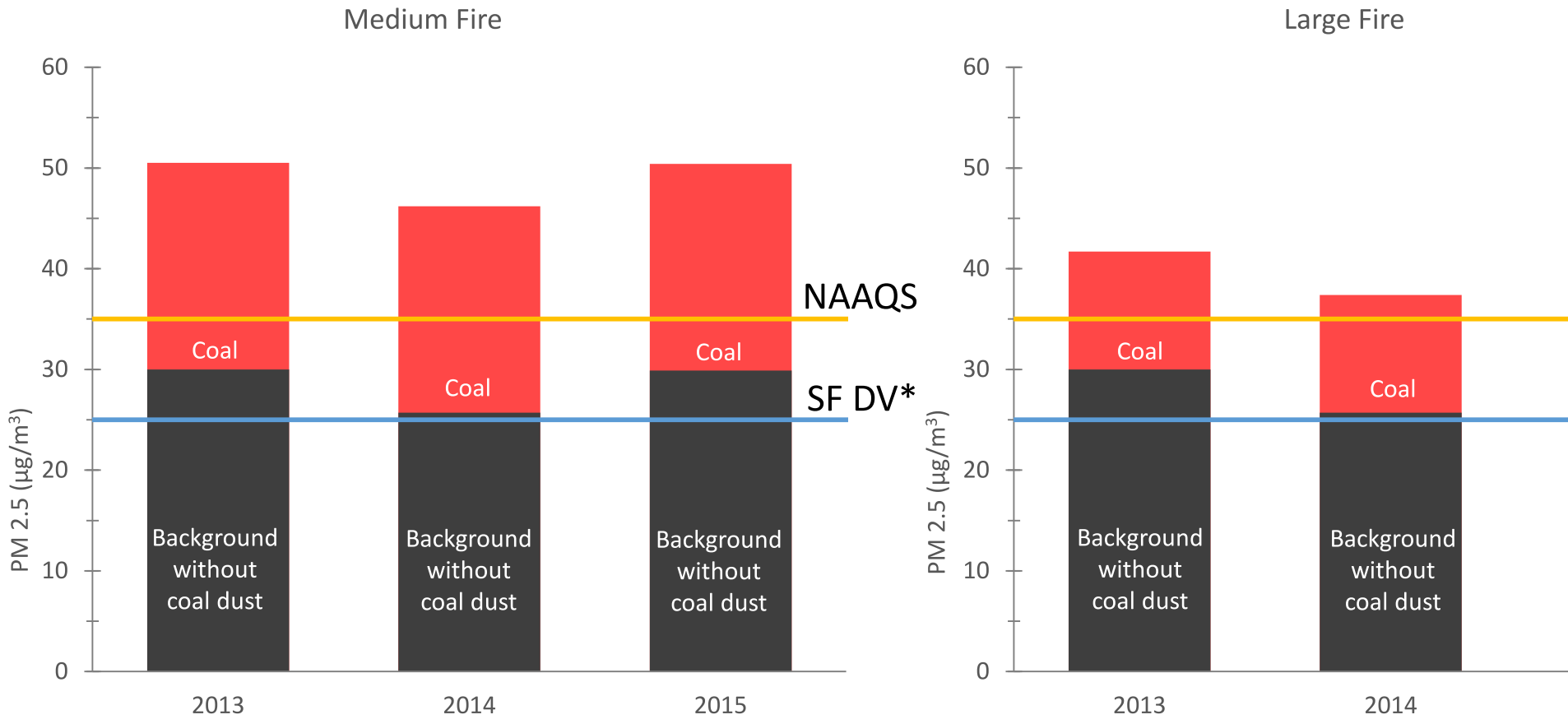
*DV for San Fra

[Table 6] Estimated **Daily** 98th Percentile PM_2 Concentration in West Oakland from Coal Du from Transport, Storage, and Handling of Coa



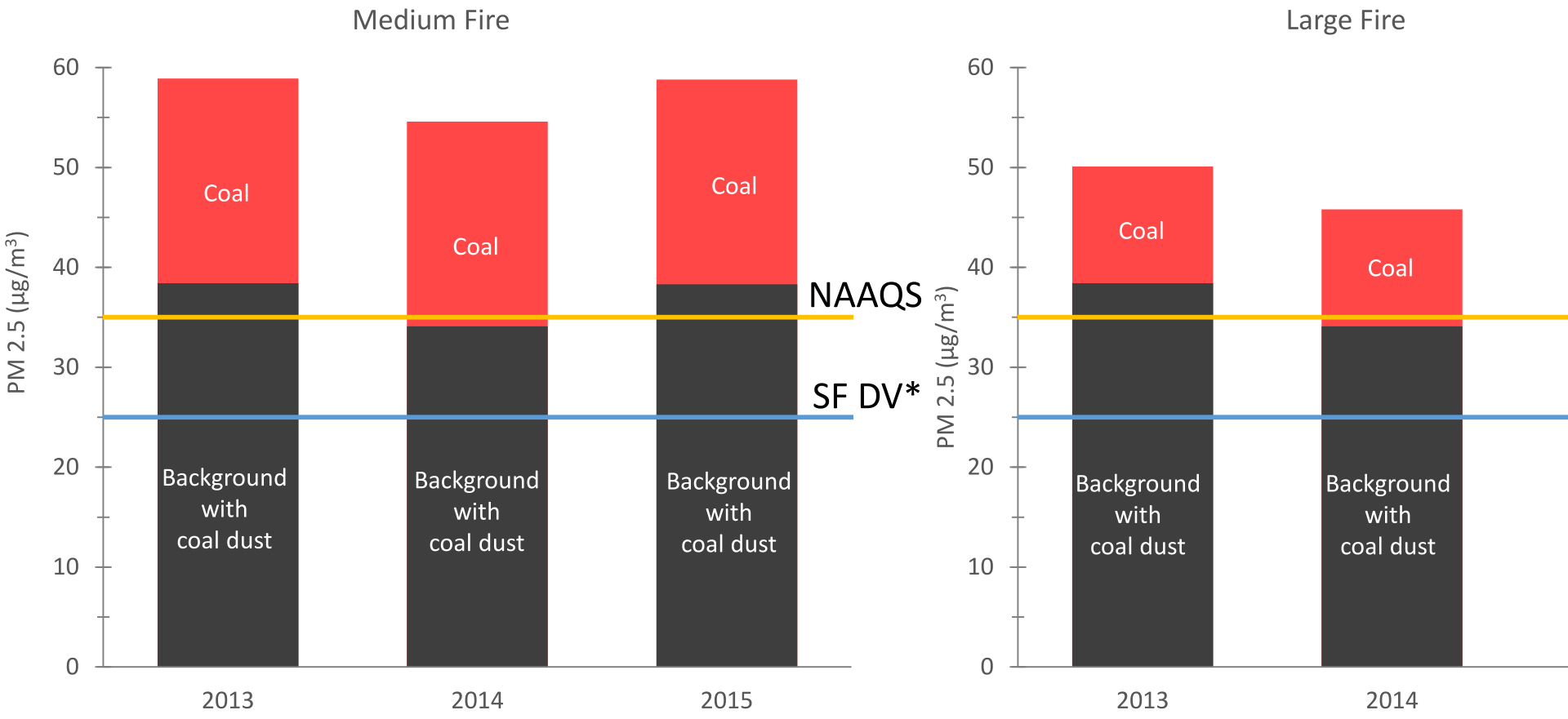
*DV for San Fra

[Table 8] Estimated **Daily** 98th Percentile PM_2 Concentration in West Oakland from a Coal F Scenario: Coal-Dust Free Background



*DV for San Fra

[Table 8] Estimated **Daily** 98th Percentile PM_2 Concentration in West Oakland from a Coal F Scenario: Background with Coal-Dust Contribu



*DV for San Fra