



June 13, 2016

RE: Oregon and Washington Physicians for Social Responsibility Comments on Draft Environmental Impact Statement for Millennium Bulk Terminals Longview

To Whom It May Concern:

On April 29, 2016, Co-leads Cowlitz County and Washington Department of Ecology issued the draft Environmental Impact Statement (“DEIS”) prepared under the State Environmental Policy Act (“SEPA”) for the proposed Millennium Bulk Terminals Longview (“MBT”) coal export project. Oregon Physicians for Social Responsibility (OPSR) and Washington Physicians for Social Responsibility (WPSR) have reviewed the document and supporting materials and submit the following comments. We incorporate by reference the comments of Columbia Riverkeeper *et al.*, Earthjustice, Friends of the Columbia Gorge, Greenpeace, Sierra Club, Stand, and The Lands Council.

The MBT project must be denied due to major and unavoidable consequences (identified in the DEIS) that could harm terminal workers, the people of Longview, the state, and the region, drinking water supplies, the Columbia River, and sovereign tribal nations. Despite some significant shortcomings, including the failure to incorporate a Health Impact Assessment (HIA), the DEIS confirms that MBT’s operation would threaten public safety, public health, and salmon. Significantly, although the DEIS significantly understates the project’s potential impact on greenhouse gas (“GHG”) emissions, it confirms that MBT would be among the state’s worst sources of carbon pollution.

SEPA regulations do not require a full cost-benefit analysis of projects. However, to the extent that economic information is included in the Final EIS (FEIS), it must include some independent review so as to be balanced, inclusive, accurate and fair. This DEIS provides a one-sided picture of economic benefits, without any countervailing assessment of economic harm. It fails to examine the cumulative costs of MBT, including costs of emergency department visits, hospitalizations, medications; lost days of school and work for patients and caregivers; and the stress associated with a significant drop in home values, for example.

However, the DEIS reveals many significant impacts and risks that, individually and collectively, provide a basis for the Co-lead to deny the project. Section S.7 summarizes the areas of impacts that cannot be mitigated. While we believe an accurate list of significant and unavoidable impacts would be both broader and deeper, this list alone provides a more than sufficient basis to deny this project under SEPA.

I. THE DEIS UNDERSTATES THE IMPACTS AND RISKS TO HUMAN HEALTH AND SAFETY

“The co-lead agencies received over 217,500 comments on the Proposed Action during the scoping period. Many of these comments expressed concerns about the Proposed Action.” (DEIS Summary at S-9.) One of the three top concerns was health.

A direct impact of the proposed coal shipping terminal at Longview would be sixteen 1.3-mile-long trains traveling across the region and through Washington state each day, and adding 1680 deep-draft vessel transits to the Columbia River each year. This will result in increased airborne pollutants from diesel engines and coal dust. The increased train traffic will also cause significant delays at rail crossings, increased risk of vehicle and pedestrian injuries along the tracks, and increased noise pollution.

Although the DEIS describes risks to communities, it minimizes them and does not examine or predict with data the potential health risks resulting from its proposed actions. Risks to human health from massive coal shipments are numerous and complex. They can be immediate, synergistic, cumulative and/or long-term in nature. Overall, the DEIS lacks detail and overall substance in regards to the human health impacts of MBT. A comprehensive Health Impact Assessment should be performed in order to give proper consideration to human health in the FEIS.

A. The DEIS Fails to Disclose the Health Risks and Costs Associated with Climate Change to Residents of Washington State and the Region

1,700 national and international health associations representing 13 million doctors, nurses, and public health professionals call for an end to dirty energy and a rapid transition to a healthier world.¹ They include the American Medical Association, American Nurses Association, American Public Health Association, American Academy of Pediatrics, American Academy of Family Practitioners, American Academy of Allergy, Asthma, and Immunology, American College of Physicians, American College of Preventive Medicine, American College of Chest Physicians, American College of Sports Medicine, American Psychological Association, American Thoracic Society, American Lung Association, National Association of County and City Health Officials, Association of State and Territorial

¹ www.climate911.org

Health Officials, National Academy of Sciences, National Medical Association, and U.S. Centers for Disease Control and Prevention.

By facilitating the mining, transport, and burning of coal, the MBT project will contribute to climate change-induced injury and disease, including:

- Increased heat related illness and health care costs; ^{54, 57, 68} (Jackson et al., 2010; Knowlton et al., 2011; McCoy & Hoskins, 2014)
- Increased extreme weather events with associated injuries and deaths; 5, 52, 54, 72 (Ashley & Ashley, 2008; IPCC, 2012; Jackson et al., 2010; NOAA, 2012)
- Food supply disruption; 64 (Luber et al., 2014)
- Spread of infectious diseases; 64 (Luber et al., 2014) and
- Disproportionate adverse effects on low income and communities of color. 52 (IPCC, 2012)

Beyond the more obvious hazards associated with coal mining, transport, storage, and burning, there is an undeniable connection between increased fossil fuel usage and its impact on climate change and health. The science is clear that the earth is warming and that people, through the burning of massive quantities of fossil fuels, are the main cause of this rapid increase in global temperatures. Heat trapping and warming temperatures are a result of increases in atmospheric greenhouse gases, which efficiently absorb heat from the earth's surface and prevent outgoing thermal energy from radiating back into space. The coal carried by trains into Longview, when eventually burned, will significantly add to an already dangerous burden of greenhouse gases being emitted into the atmosphere.

Numerous studies, reported in leading scientific and medical journals, show that ongoing changes to our climate are correlated with: changes in rainfall patterns; worsening heat waves; an increased frequency and magnitude of extreme weather events, droughts, and fires; a rise in sea level; increased potency of allergens; and the spread of infectious diseases – all of which pose a real and serious threat to human health. Unless global carbon emissions start to fall within the next decade, we can expect to see further and more drastic changes in our climate, and related adverse health impacts all over the world.

Populations that could be most vulnerable to health impacts of climate change include those with:

- Demographic vulnerability: People with existing illnesses, people with disabilities, older adults, mothers, infants, children, people with low socioeconomic status, linguistically or socially-isolated populations, immigrants and refugees, communities of color, and American Indians
- Occupational vulnerability: Wildland firefighters, outdoor workers, growers, ranchers and farmworkers, emergency responders and health care workers
- Geographic vulnerability: Urban and suburban areas, coasts, steep slopes, and private water systems (Haggerty et al., 2014)

MBT's emissions will contribute to increased ground level ozone.

- Ground level ozone increases with hot weather, vehicle and diesel exhaust, gasoline vapors, and other outdoor air pollutants. Ground level ozone is known to irritate the respiratory tract, cause premature aging of the lungs, and has been linked to the development of asthma and exacerbation of existing asthma cases. In fact, people who spend more time being active in the outdoors working or playing are at greater risk for adverse health effects from ozone exposure than those who spend more time inside or are sedentary (McConnell et al., 2002; Gent et al., 2003).
- Asthma currently affects over 9% of Washington adults (ages 18 and older), and over 110,000 youth in Washington suffer from asthma. The Centers for Disease Control ranks asthma prevalence in Washington State residents as higher than the national average. In 2010, \$73 million was spent on hospitalization costs for asthma-related illness in Washington. Asthma is the primary cause of school-age absenteeism nationally and is associated with reduced quality of life, depression, and suicidal ideation (WA DOH, 2013).
- In Oregon, an estimated 10.8% of adults and 7.8% of children have asthma. Oregon has a higher burden of asthma than the overall US and was among the top six states with the highest percentage of adults with asthma in 2011. Children 0-4 years and females have the highest rates of asthma hospitalizations. In 2011, the total cost of asthma hospitalizations was more than \$28 million, with an average of over \$14,000 per hospitalization (Garland-Forshee & Gedman, 2013).
- The University of Washington's Climate Impacts Group has estimated that ozone levels will rise due to climate change and increases in train, auto, bus, and truck transportation in the state. Ozone levels are expected to increase by 16% in Spokane County and 28% in King County by midcentury (2045-2054) from 1997-2006, increasing the risk for deaths from cardiovascular disease, asthma, and lung cancer. They also estimate an increase in ozone-related deaths by 17% in Spokane County and 27% in King County during the same time period (Jackson et al., 2010).
- Health related costs of current ozone air pollution nationally were an estimated \$6.5 billion in 2008 and will continue to rise without change in regulatory controls (Knowlton et al., 2011).

MBT will contribute to negative health impacts of increased extreme weather events and wildfires.

- Extreme weather events with associated injuries are already being witnessed globally. Precipitation extremes including heavy rainfall, flooding, and droughts are projected to increase in all regions of the US (IPCC, 2012).
- Floods account for approximately 98 deaths per year in the US and are the second deadliest of all weather-related hazards (Ashley & Ashley, 2008; NOAA, 2012).
- Steep slopes and intense rainfall can trigger landslides that result in injury and death.

- Smoke from wildfires is associated with cardiopulmonary disease, ischemic heart disease, asthma, bronchitis, pneumonia, cancer and motor vehicle crash injury (Haggerty et al., 2014).

MBT will contribute to negative health impacts of shifting disease ranges.

- Climate change is associated with the spread of vector- and water-borne disease and illness. Vectors such as fleas, ticks, and mosquitoes transmit pathogens that cause diseases including Lyme, dengue fever, West Nile virus, and Rocky Mountain spotted fever.
- Large-scale weather shifts in temperature, precipitation, and humidity can result in vector adaptation or geographic expansion, increasing the number of people at risk for acquiring vector-borne diseases.
- Water-borne illnesses such as pediatric gastrointestinal infections have also been associated with extreme weather events, large-scale flooding, and water source contamination (Luber et al., 2014).

MBT will contribute to loss of food security and increase risk to vulnerable populations.

- An anticipated decline in crop yields, livestock, and fish production from extreme weather, changes in rainfall patterns, and ocean acidification is predicted to raise food prices and result in food shortages.
- Elevated atmospheric carbon dioxide is also associated with decreased plant nitrogen concentration, resulting in decreased protein content of existing plants.
- Mental health disorders and anxiety around climate-related disease and illnesses are an additional concern for health care providers (Luber et al., 2014).
- Air pollution and climate change will continue to disproportionately affect minorities and lower socio-economic populations in Washington, the US and worldwide. Those least responsible for the atmospheric content of carbon and other pollutants are positioned to bear the most significant brunt of their ill effects, including increased respiratory and infectious illness, extreme weather events and food shortages.

The FEIS should include and count the GHG emissions generated by coal extraction. While the technical analysis for GHGs properly includes transportation to and operations at the terminal, as well as some of the impacts of coal combustion, it does not include the GHGs of extraction of coal. It is difficult to see how the extraction of coal for the terminal should be treated any differently than the transportation of that coal to the terminal site. Both are proximately caused by the terminal—the 44 million tons of coal that would be shipped out of the terminal would not be mined but for the terminal, as it would be supplementary to any coal mined used for other purposes.

The FEIS should estimate GHG emissions on the basis of an estimated 50-year lifetime of the project. Specifically, the analysis looks at a time scale of 2018 to 2038, with full operations not occurring (due to a multi-year ramp up) until 2028. (Technical report at 2-13.) In other words, the analysis only assumes that this project will be operating at full capacity for 11 years.

Medical Approach to Climate Change: The Precautionary Principle

The Intergovernmental Panel on Climate Change unequivocally states that a substantial and ongoing reduction in greenhouse gas emissions is necessary to prevent further imbalances in earth's climate and subsequent climate-related disease and illnesses (McCoy & Hoskins, 2014). Many medical professionals and public health advocates, including our organizations, firmly invoke The Precautionary Principle in consideration of proposed coal export projects and this specific proposal by MBT.

The Precautionary Principle – a substantial component of public and environmental health practice – states: “should an activity raise threats of harm to human health or the environment, precautionary measures should be taken even if some cause and effect relations are not fully established scientifically” (Wingspread Conference, 1998).

The proposed increase in mining, transport, storage, export, and burning of coal externalizes massive long-term threats to human, environmental, and economic health in favor of short-term financial incentives. MBT and other fossil fuel export projects in their totality pose significant risks to the health and livelihood of future generations and the viability of our planet.

B. The DEIS Fails to Disclose the Full Health Risks and Costs to Residents of Washington State and the Region associated with Air Pollution and Diesel Particulate Matter (DPM)

Diesel particulate matter is associated with:

- increased cardiopulmonary mortality and all-cause mortality;
- impaired pulmonary development in adolescents;
- measurable pulmonary inflammation;
- increased severity and frequency of asthma attacks, ER visits, and hospital admissions in children;
- increased rates of myocardial infarction (heart attack) in adults;
- increased risk of ischemic stroke;
- neurodevelopmental and reproductive disorders; and
- cancer.

One of the largest potential health impacts of the Millennium Bulk Terminals Longview lies in the increase in air pollution resulting from diesel locomotive emissions all along the transportation corridor, from the Powder River Basin and the Uinta Basin to Longview and back, plus the diesel emissions from the vessels transporting the coal through Columbia River to the Pacific Ocean and back.

Anticipating coal train traffic moving through the Multnomah County to Longview and other locations, the county produced a report in 2013 entitled “The Human Health Effects of Rail Transport of Coal Through Multnomah County, Oregon *A Health Analysis and Recommendations for Further Action.*” It states:

Diesel particulate matter is one of the air toxins that contributes the most to air pollution-related health risks in the Portland region. According to an estimate by the Oregon Department of Environmental Quality, in 2017 the region’s airshed will have on average more than ten times the level of diesel particulate that is considered safe.

(See Exhibit: Multnomah County, Oregon Coal Report, 2013)

The effects of air pollution are not hypothetical, but real and measurable. Many studies show significant health effects of exposure to everyday airborne pollutant levels that are below national U.S. Environment Protection Agency (EPA) guidelines. The data show a linear effect with no specific “safe threshold.” Recognizing this, the EPA has recently taken steps to enact more stringent standards.

The conclusion that airborne pollutants pose a significant and measurable health risk was also found by the American Lung Association, in their review, “State of the Air 2012”, and by the American Heart Association, in their 2011 review, “Particulate Matter Air Pollution and Cardiovascular Disease.”

Diesel particulate emissions are of special concern, particularly the size fraction up to 2.5 microns, known as PM2.5. This size of particle can be respired deep into the lungs. PM2.5 from all sources has been implicated in numerous diseases ranging from cardiopulmonary disease to cognitive decline to cancer. The deleterious impact on human health is incontrovertible (WA DOE 2008, California Air Resources Board 1998, and many other studies). Diesel engines are huge sources of particulate matter, as they typically produce PM2.5 at a rate about 20-times greater than gasoline engines.

The DEIS discloses significant information about air pollution and diesel particulate matter (DPM):

- Diesel particulate matter was identified as the most likely contributor to cancer risk in Washington State.
- In Longview, all rail traffic in the study area is projected to increase emissions for all air pollutants by about 11%,

- Locomotive emissions in Cowlitz County are estimated to increase by about 6% overall with the proposed action. The largest emissions increase for a single pollutant would be for PM10, which would increase by approximately 15%.
- Vessel emissions in Cowlitz County with the proposed action are estimated to increase by about 12%.
- Cumulative vessel traffic in 2038 is projected to increase air emissions by about 24%.
- Table 5.6-10. Estimated Maximum Annual Emissions *in Washington State* for Locomotive and Commercial Marine Vessels for the Proposed Action in Comparison with the 2011 Statewide Emissions Inventory:

Locomotives will emit 47 tons/year DPM
(46 tons/year PM2.5 and 47 tons/year 10 DPM)

Marine vessels will emit 10 tons/year DPM
(11tons/year PM 2.5 and 13 tons/year of PM 10)

- Table 6-25. Estimated Maximum PM10 and PM2.5 Concentrations—BNSF Main Line in *Eastern Washington*
Will exceed the 24-hour PM10 and annual PM2.5 ambient air quality standard at 100 feet from the rail line.
- With respect to hazardous air pollutants, the 2005 EPA National-Scale Air Toxics Assessment was used by Ecology to estimate cancer risk (Washington State Department of Ecology 2011). Inhalation cancer risks were highest in the major population centers along the rail route (Vancouver and Spokane), with a cancer risk of up to 500 cancers per million. For the smaller communities (Kelso-Longview, Spokane, Yakima, and Pasco), cancer risks were up to 300 cancers per million.

However, the air quality impact summary in 5.7.5 of the DEIS states “Overall the impacts of PM10 and PM2.5 emissions from proposed-action related rail transport of coal would not be significant because emissions would be below applicable federal standards.” This is a misleading statement. While it is true that PM10 and PM2.5 emissions would fall below federal standards, this does not mean that there would be no negative health impacts. In fact, according to the World Health Organization (WHO) “Small particulate pollution have health impacts even at very low concentrations – indeed no threshold has been identified below which no damage to health is observed.”²

² WHO Fact sheet N°313 - Ambient (outdoor) Air Quality and Health including links and references to: WHO Air Quality Guidelines, Air Pollution and Cancer

Again, human health impacts of particulate matter include cancer, cardiovascular, cerebrovascular and respiratory disease. These health consequences accumulate with increasing exposure. There is a close quantitative correlation between exposure and negative health impacts (morbidity and mortality). Comparing the guidelines used in the DEIS (which are from the U.S. National Ambient Air Quality Standards and Washington State Air Quality standards) against the WHO guidelines, we find that the WHO guidelines are lower and more restrictive. In some cases (particularly PM₁₀), they are considerably lower. The following table compares WHO guidelines with NAAQS:

WHO Particulate Matter Exposure Guideline values³ with NAAQS/Washington State Standards shown in parentheses for comparison

PM_{2.5}

Annual mean - 10 µg/m³ -- (NAAQS 12 µg/m³)
 24-hour mean - 25 µg/m³ -- (NAAQS 35 µg/m³)

PM₁₀

Annual mean - 20 µg/m³ (Not included in the DEIS)
 24-hour mean - 50 µg/m³ -- (NAAQS 150 µg/m³)

Below are examples of expected emissions from project operations with comparison to WHO Air Quality Guidelines:

Table 5.6-6. Maximum Modeled Concentrations from the Operation of the Coal Export Terminal shows total predicted concentrations of PM₁₀ (24 hour average) of 80mcg/m³. This exceeds the WHO guideline of 50mcg/m³.

Table 5.6-7. Project Area Concentration from Operations (All Sources) shows total predicted concentrations of PM_{2.5} (24 hour average) of 29.8mcg/m³. While under the NAAQS 35mcg/m³ threshold is over the WHO standard of 25mcg/m³. Total predicted concentrations of PM₁₀ (24-hour average) would be 108mcg/m³, which is over twice the WHO threshold of 50mcg/m³.

Table 5.7-6. Estimated Maximum PM₁₀ and PM_{2.5} Concentrations—BNSF Main Line, Cowlitz County shows the total concentration of PM₁₀ at 50ft and 100ft to be 58mcg/m³ and 51mcg/m³ respectively, both of which exceed the WHO guideline of 50mcg/m³.

IARC's 2013 Assessment, Review of Evidence on the Health Aspects of Air Pollution (REVIHAAP) - <http://www.who.int/mediacentre/factsheets/fs313/en/>

³ WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide Global update 2005 - http://apps.who.int/iris/bitstream/10665/69477/1/WHO_SDE_PHE_OEH_06.02_eng.pdf

The 24-hour average of PM_{2.5} at 50 feet is 25.5mcg/m³ which is above the WHO guideline of 25mcg/m³, while at 100 feet it is 24.8, just below the WHO standard.

Table 5.7-9. Estimated Maximum PM₁₀ and PM_{2.5} Concentrations 100 Feet From Rail Line— BNSF Main Line, Washington State (Outside Cowlitz County) shows the total concentration of PM_{2.5} (annual average) to be 9.8mcg/m³, which is just under the WHO guideline of 10mcg/m³.

The 24-hour average of PM_{2.5} is 27mcg/m³, which exceeds the WHO guideline of 25mcg/m³.

The PM₁₀ (24-hour average) is 125mcg/m³, which is two and a half times the WHO guideline of 50mcg/m³.

Of particular interest is Table 5.7-9, which demonstrates that the current baseline PM₁₀ (24-hour average) is 101mcg/m³ is already twice the level established by the WHO. This is important in light of data summarized in the WHO Air Quality Guidelines and statements that “reducing *annual* average particulate matter (PM₁₀) concentrations from levels of 70 µg/m³, common in many developing cities, to the WHO guideline level of 20 µg/m³, could reduce air pollution-related deaths by around 15%. However, even in the European Union, where PM concentrations in many cities do comply with Guideline levels, it is estimated that average life expectancy is 8.6 months lower than it would otherwise be, due to PM exposures from human sources.”⁴
^{5 6 7} (Note that the above numbers refer to *annual* PM₁₀ concentrations which were not measured/modeled/included in this DEIS.)

Though particulate matter and coal dust emissions from the Millennium Bulk Terminal Project are expected to fall under NAAQS and Washington State Standards, they will have negative health impacts. The DEIS identified places in Washington State, especially near the railroad tracks, where current air quality is already unacceptably poor, exceeding WHO guidelines by two times in at least one case.

Improvements in ambient air quality in these locations can be expected to have considerable positive health impacts, while the effect of MBT would be, in all instances, to increase exposure to particulate matter, which has negative health impacts even at very low doses.

⁴ Pope CA et al. (1995). Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. *American Journal of Respiratory and Critical Care Medicine*, 151:669–674.

⁵ Pope CA et al. (2002). Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *Journal of the American Medical Association*, 287:1132– 1141.

⁶ Cohen A et al. (2004). Mortality impacts of urban air pollution. In: Ezzati M et al., eds. *Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors*. Geneva, World Health Organization:1353–1434

⁷ Dockery DW et al. (1993). An association between air pollution and mortality in six U.S. cities. *New England Journal of Medicine*, 329:1753–1759.

We object to DEIS air quality assessments based on modeling with insufficient actual monitoring. In Section 5.6.4.2, the following statement appears:

The only available local (Cowlitz county near project site) air pollutant monitoring is for PM_{2.5}, at a station approximately 1.5 miles east of the project area. The monitoring data show that PM_{2.5} levels are well within the PM_{2.5} air quality standards. Although no other monitoring data are available, concentrations of other criteria air pollutants in the study area also are expected to be well within air quality standards.

The City of Portland and Oregon Department of Environmental Quality recently discovered the risk of underestimating air pollution when air quality modeling is based on a small number of actual monitoring stations. The U.S. Forest Service used moss bio-indicators as a novel air quality monitoring strategy and found very high levels of cadmium (49 times higher than Oregon air quality standards) near several stained glass manufacturers.⁸ These very high toxic emissions were not predicted based on prior, inadequate air quality monitoring. The data from only a few stations was available and assumptions in modeling led to significant errors. The DEIS assumption that “concentration of other criteria air pollutants in the study area also are expected to be well within air quality standards” does not rise to the level of rigor one must have when working to protect public health and the health of workers at the terminal.

1. The DEIS Understates the Health Impacts and Risks of DPM on Cancer

Studies show an association between exposure to diesel exhaust and lung cancer (Bhatia, 1998), as well as cancers of the bladder and soft tissues (Guo et al., 2004). Several extensive and detailed reviews have been conducted on the body of literature relating long-term exposure to diesel exhaust particles and lung cancer (California EPA, 1998; USEPA, 2002; Cohen and Nikula, 1999). In addition, over 40 studies conducted among those populations exposed to diesel exhaust have found increased rates of lung cancer associated with diesel exhaust particles exposure (as cited in Cohen and Nikula, 1999). Occupational studies conducted in railroad workers and truck drivers have consistently found increased lung cancer risk, even after adjusting for co-morbidities such as smoking (Bofetta, 2001). The impact of DPM on cancer risk must be considered in the decision making process for the MBTL.

2. The DEIS Understates the Health Impacts and Risks of DPM on Cardiac and Pulmonary Disease

Although cancer risk is understandably of great concern to the public, cardiac and respiratory effects of diesel exposure have an even larger public health impact

⁸ Donovan, Geoffrey H, et al. 2016. Using an epiphytic moss to identify previously unknown sources of atmospheric cadmium pollution. *Science of The Total Environment*. 559: 84-93

because they cause death and illness for a greater number of people. DPM can exacerbate asthma and emphysema, induce heart attacks and strokes, and has been associated with congenital heart abnormalities. According to a landmark study by Pope et al. (2002), each 10 ug/m³ increase in DPM was associated with a 6% increase in cardiopulmonary mortality. In a follow-up to this study, Pope et al. (2004) demonstrated that their previously observed increase in cardiopulmonary mortality was largely driven by increases in cardiovascular, as opposed to pulmonary, mortality. In this follow-up study, a 10 ug/m³ increase in PM_{2.5} was associated with a 12% increase in mortality due to ‘all cardiovascular disease plus diabetes’ and an 18% increase in mortality due to ‘ischemic heart disease’. Further epidemiological investigations have revealed that these estimates are likely largely underestimating the effect of PM_{2.5} due to inadequate exposure characterization. Published in the *New England Journal of Medicine*, Miller et al. (2007) utilized a novel exposure characterization method and reported from the Women’s Health Study that a 10 ug/m³ increase in PM_{2.5} was associated with a 76% increase in death due to cardiovascular disease. To further highlight the impact of PM_{2.5} on public health, the ‘Global Burden of Disease’ report recently published in *Lancet* reported ambient PM_{2.5} as the #9 cause of disease worldwide, and the #14 cause of disease in North America (Lim et al. 2013) in the year 2010.

It is well understood that ambient air pollution and fine ambient particulate matter strongly contribute to disease burden and death, but it has been less clear as to how much an individual’s living proximity to a major roadway or direct PM_{2.5} source influences health risks. Due to research led by those at the University of Washington, it is becoming clearer that an individual’s exposure to PM_{2.5} is dependent on where he/she lives and works and that this strongly influences health outcomes. Van Hee et al. (2009) demonstrated that living close to a major roadway was strongly associated with left ventricular hypertrophy, an important marker of cardiovascular disease and a strong predictor of heart failure and mortality. Additional work by this group has demonstrated an individual’s exposure to PM_{2.5} impairs how well blood vessels dilate and how well the heart functions, providing a basis for our understanding of previously observed increases in mortality (Van Hee et al., 2011, Krishnan et al., 2012).

There are very specific physiological effects with DPM exposure. A very recent study by Cosselman et al. (2012) showed that diesel exhaust exposure, to healthy human volunteers, rapidly increases systolic blood pressure (SBP). In their study, SBP increased within 15 minutes of being exposed to dilute diesel exhaust and reached a maximum increase in SBP within one hour. Additional work utilizing controlled diesel exhaust exposures to human volunteers has revealed that these acute exposures result in an impairment in blood vessel function and alter blood coagulability, both of which are extremely deleterious effects and increase the risk of acute cardiovascular events such as heart attack and stroke (Mills et al., 2005, 2007, and Törnqvist et al., 2007). Fitting with these findings, epidemiological investigations have consistently demonstrated that acute increases in PM_{2.5} result in an increased risk of heart attack (Peters et al., 2001).

In addition to cardiovascular risk, cerebrovascular effects and risk of stroke associated with PM_{2.5} exposure have been investigated. Research published in the

Archives of Internal Medicine (2012) (See References: DPM) examines, for the first time, the risk of acute, short term exposures to PM2.5 as a key factor in triggering stroke, often within hours of exposure. The study found a linear relationship between PM2.5 level and stroke risk even when the exposure was well below the EPA daily exposure limit. Overall, the risk of ischemic stroke was 34 % higher on days when the PM2.5 level was on the higher range of “moderate” exposures (15-40 ug/m³), as opposed to days when pollutants are lower than 15 ug/m³. This is an unprecedented finding, and points to the acute danger of even short term exposures to levels of particulate pollution previously thought “safe.”

Studies conducted at Seattle Children’s Hospital show that air pollution leads to asthma exacerbations, increased ER visits, and increased hospitalization, at levels that currently exist in Seattle. (See References: DPM at end of this document.) A study in California shows that about half of the economic costs of asthma can be attributed to air pollution, costing society millions of dollars per year. Thus, it is emphasized that additional DPM exposure adds to an existing problem.

3. The DEIS Fails to Disclose the Impacts of DPM on Reproductive and Neurodevelopmental Disorders

A review of peer-reviewed journal articles makes evident concerns about impacts of DPM on reproductive and neurodevelopmental disorders:

- Reduced sperm quality in men exposed to air pollution, particularly diesel exhaust (De Rosa et al., 2003)
- Disruption of normal sexual differentiation during fetal development, including 2.42% higher odds of male cryptorchidism (undescended testes) amongst babies of fathers exposed to diesel exhaust before conception (Kurahashi et al., 2005)
- Increased congenital heart, lung, and immune system anomalies in children (Gauderman et al., 2004; Vrijheid et al., 2011)
- A 10 microgram increase in DPM (2.5) is associated with a 3.4% increase risk in daily mortality (Laden et al., 2000)
- In 2005 the World Health Organization published a summary of the health risks of air pollution on childhood health and concluded that “sound evidence already exists for a causal link between air pollution and children’s health” (WHO, 2005, p.7)
- In the same document the WHO recommended that policy makers take measures to reduce childhood exposure to air pollution (WHO, 2005)

We have witnessed a profound increase in the number and severity of children (per capita) with neurodevelopmental disorders such as autism, ADHD, and learning impairments. The Centers for Disease Control and Prevention corroborates this increase in their recent counts of pediatric disorders. This is likely due in part to increased exposures to neurotoxic chemicals in the environment. Recent studies have correlated prenatal and early life exposure to diesel particulate exhaust with autism, ADHD,

lowered IQ and cognitive function, and increased behavioral symptoms of anxiety, depression, and aggressive behavior.

Diesel components, and heavy metals found in coal dust, can cause permanent damage to the developing nervous systems of embryonic and young children, even at low levels. The proposed terminal, which would increase the number of mile-and-a-third-long trains (8 trains full and 8 returning mostly empty) passing through the region daily, each carrying 125 uncovered coal hopper cars, pulled by three to four diesel engines, would add cumulative impacts of further diesel emissions, as well as coal dust.

Exposure to toxins in airborne particulate matter from diesel engines and coal dust will predictably increase neurodevelopmental impairments in our children and other adverse health effects in adults and children, such as asthma, cancer, heart attacks and strokes. Over time, this is likely to have a major health impact and cost to our population. Unlike other potential disaster scenarios, added air toxins from increased trains transporting coal would be a certainty, with well-studied human health effects.

4. The DEIS Understates the Health Impacts and Risks of DPM and Associated Toxins

While hundreds of different airborne toxins may be present in the gas phase of diesel exhaust, some of the most commonly identified are acrolein, acetaldehyde, formaldehyde, benzene, 1,3-butadiene, and polycyclic aromatic hydrocarbons (PAHs). The human health impact of all of these associated toxins should be studied in detail.

Formaldehyde is carcinogenic to humans. It is also a highly reactive substance that can be irritating to the nose, eyes, skin, throat and lungs at fairly low levels of chronic exposure.

Benzene is carcinogenic to humans. Chronic exposure to benzene leads primarily to disorders of the blood.

1,3-Butadiene is linked to cancers of the blood and lymph systems, including leukemia. It has also been linked to disorders of the heart, blood and lungs, and to reproductive and developmental effects.

Some Polycyclic Aromatic Hydrocarbons (PAH) are carcinogenic to humans. Because this group of compounds covers a wide range of physical-chemical properties, some PAH are found in air on particles while others are gaseous. PAH of both forms may be deposited in the lung.

C. The DEIS Fails to Disclose the Health Risks and Costs to Residents of Washington State and the Region of Additional Toxic Air Pollutants

The DEIS understates the toxic impacts of coal dust. The DEIS states, “One review of the chemical composition of coal dust (U.S. Geological Survey 2007) suggests that the risk of exposure to concentrations in toxic materials (e.g., PAHs and trace metals) from coal are low because the concentrations are low and the chemicals bound to coal and not easily leached.” The DEIS fails to address other studies identifying risks from toxic materials in coal dust. Co-leads should evaluate the expert report prepared by Leyda Consulting, Inc., on the proposed Morrow Pacific coal export project (hereafter “Leyda EXHIBIT”).⁹ The Leyda EXHIBIT includes an in-depth toxicology report on coal dust.

It should be emphasized that children are not "little adults" and are thus more vulnerable to the health effects of environmental contaminants. Children eat more, breathe more, and drink more per body weight than adults, and therefore receive a greater exposure and dose of any material. In addition, children have unique behaviors such as hand-to-mouth actions that increase exposure to contaminants. Developing organ systems are more vulnerable to adverse effects.

Toxic Air Pollutants (TAPS) of concern that may be emitted by this project include arsenic, cadmium and mercury. We request that the Washington Department of Ecology conduct independent health risk assessments for all TAPS that may be emitted by this project.

1. *The DEIS fails to fully disclose health impacts of exposure to arsenic.*

The DEIS states that arsenic is one of the pollutants that would continue to be introduced as a result of the proposed action in Longview and along the tracks. While the DEIS states that maximum concentrations of arsenic will be lower than acceptable source impact levels, recent studies published in journals such as Environmental Health Perspectives suggest that arsenic is harmful to human health at lower levels than previously thought (Carlin et al., 2016; Naujokas et al., 2013), including increased risk for skin and lung cancer. (Bailey et al., 2016)

“Inhalation exposures to arsenic-bearing dusts and aerosol, in both occupational and environmental settings, have been definitively linked to increased systemic uptake, as well as carcinogenic and non-carcinogenic health outcomes.” (Martin et al., 2014)

Low levels of exposure to arsenic are particularly worrisome for pregnant women and children. Recent studies show that exposure to arsenic in the womb and in early childhood may cause decreased fetal growth and adverse epigenetic effects. (Naujokas et al., 2013; Flora, 2011; Bailey et al., 2016)

⁹ Leyda Consulting, Inc., *Ecological Impacts of Proposed Coal Shipping on the Columbia River Port of Morrow and Port Westward, OR* (2012).

The risk of exposure to arsenic and its toxicity is further supported by the CDC's Agency for Toxic Substances and Disease Registry: arsenic has been ranked Number 1 on that agency's Substance Priority List since 1997, and before that it was second only to lead. (ATSDR 2007)

Government standards for allowable levels of arsenic in water have already been lowered by 80% in the last 16 years as evidence has grown demonstrating toxicity at lower levels of exposure. (USEPA Chemical Contaminant Rules) This evidence will likely continue to grow. Changing standards remind us that, though certain current standards may be met, these standards may not and, at times, do not adequately protect human health.

2. *The DEIS fails to adequately disclose health impacts of exposure to cadmium.*

Cadmium is a highly toxic metal with a very long half-life of 20-30 years in humans and accumulates in soft tissues, kidneys, and the liver. Specific mechanisms of cadmium toxicity are not well understood; however, evidence suggests that cadmium affects DNA repair, and cell signaling and control. These effects lead to kidney damage, cancer, mutations, damage to hormone regulating mechanisms, reproductive disorders, and problems with cellular differentiation (Rani et al., 2014). Some evidence also points to harmful long-term and heritable effects of cadmium (Ray et al., 2014). In humans, cadmium disrupts biologic pathways involving calcium, leading to bone and muscle issues (Choong et al., 2014).

The International Agency for Research on Cancer (IARC) classifies cadmium and cadmium compounds as carcinogenic to humans (Group 1) (IARC, 2012). Group 1 classification is the strongest assertion of carcinogenicity.

Recent studies have linked cadmium with bladder cancer (Feki-Tounsi et al., 2014). Evidence also exists for associations with breast cancer (Dematteo et al., 2013) and pancreatic cancer (Wei Qu et al., 2012; Garcia-Esquinas et al., 2014). A recent review found that "exposure to low concentrations of Cd is associated with effects on bone, including increased risk of osteoporosis and fractures..." (Akesson et al., 2014).

Cadmium was implicated in Itai Itai disease due to industrially contaminated water in people exposed (especially women). They suffered osteomalacia and osteopenia, decreased bone mineral content, and decreased bone density (Kobayashi, 1971; Kasuya, 2000; Inaba et al., 2005).

In a retrospective study of over 2,000 children, the authors concluded that children who have higher urinary cadmium concentrations may have increased risk of both [acquiring] LD [learning disability] and [being more likely to receive] special education. These associations were found at exposure levels that were previously

considered to be without adverse effects, and these levels are common among U.S. children (Ciesielski et al., 2012).

In a prospective study of 270 children, the authors “noted in boys a 1.53 times higher risk for emotional problems with a twofold increase in cord blood cadmium” (Sioen et al., 2013).

In a prospective study of over 1000 children, the authors concluded: “Early-life low-level cadmium exposure was associated with lower child intelligence scores in our study cohort” (Kippler et al., 2012).

Cadmium mimics estrogen (Johnson, MD, 2003) so it is an endocrine disrupting chemical. It also affects male reproduction in animal studies, and has recently been implicated in human epidemiological studies as causing decreased birth weight. (Johnston et al., 2014; Kippler et al., 2012).

A recent study showed that cadmium exposure was related to leukocyte telomere length (a marker of cellular aging). The authors concluded: “These findings provide further evidence of physiological impacts of cadmium at environmental levels and might provide insight into biological pathways underlying cadmium toxicity and chronic disease risk” (Zota et al., 2014).

3. *The DEIS fails to adequately disclose health impacts of exposure to lead.*

Stanford University produced a fact sheet on lead that demonstrates that the inhalation or ingestion of lead-containing particles can result in “lead poisoning” which has been associated with a number of short term (acute) and long term (chronic) adverse health effects. Depending on the amount of exposure (dose) immediate symptoms may not always be apparent or may resemble other illnesses and result in misdiagnoses.¹⁰

Acute, short term health effects may include: cramps (lead colic), irritability and moodiness, headaches, insomnia, tiredness, nausea, loss of libido, birth defects, miscarriage, stillbirth, constipation, and, in children, hyperactivity, lower IQ, slowed growth, and hearing loss. Chronic, long term health effects may include: muscle and joint soreness, fine tremors, numbness, hypertension, anemia, infertility, and kidney damage.

Lead can stay in the body for years and is stored in bone or soft tissue including the liver and kidneys. During periods of high calcium demand such as pregnancy, menopause and aging, lead stored in bone tissue can be released back into the

¹⁰ <https://web.stanford.edu/dept/EHS/prod/general/asbestoslead/leadfactsheet.html>

bloodstream. Lead is also able to cross the placenta and blood/brain barrier.

Exposure to lead can occur almost anywhere. Studies have shown that lead dust can be carried on coveralls or other work clothing resulting in contamination of worker's cars, homes and family.

There is lead in coal dust. There is no safe level of lead. Lead dust is 100% absorbed when inhaled by infants. Lead causes neurodevelopmental disorders. It can cause severe toxic effects in children in multiple organs and widespread disruption of cellular functioning. It damages the bone marrow and nervous system with direct nerve cell damage harming brain development, which in turn causes seizures, schizophrenia, cognitive loss, and many serious sequelae, including academic failure.

There is no good treatment for lead poisoning. Chelation therapy is difficult and does not reverse cognitive impairment. As in Flint, Michigan, lead poisoning is usually identified after the fact, when the harm has already been done.

What will be the cumulative levels of lead deposited in air and in soil in Longview and along coal train tracks? We must answer questions like this now, before 44 million tons of coal, and its associated burden of lead, is brought in uncovered rail cars through Washington and the region each year and is stored, in uncovered piles, in Longview.

4. *The DEIS fails to adequately disclose health impacts of exposure to mercury.*

According to the City of Portland's June 9, 2016 MBT DEIS comments: "...In the Columbia River Basin more than 80 percent of the mercury pollution is from overseas sources." A peer-reviewed 2008 study¹¹ found that coal-fired power plants in Asia contribute 18% of springtime mercury concentrations at Mount Bachelor.

Snowpack melts into our rivers and lakes where mercury contaminates the fish we eat. Pregnant women and children are particularly vulnerable to the toxic effects of mercury. Mercury is a potent neurotoxin that can damage developing brains in fetuses and children.

Dr. Martha Neuringer, a renowned biomedical researcher at Oregon Health Sciences University, stated in testimony she presented to the Portland City Council in September 2012,

The effects of coal-derived mercury on infant brain development are well known. Coal-derived mercury has significant negative impacts on the visual system, on motor development, and on cognitive development. It insidiously limits human potential. A massive increase in coal traffic through our region would greatly increase the mercury burden in our

¹¹ http://www.atmos.washington.edu/jaffegroup/publications/Pacific_Transport_Hg.pdf

environment and therefore the damage to our children. This is a moral issue, but can also be reduced to its economic impacts.

The effects of mercury from coal on reduced intellectual development - on this one health effect - are estimated to cost \$3 billion per year in the U.S.¹¹ This is just one part of the overall health costs of \$10-30 billion, which in turn is just part of the estimated total externalities – environmental, economic and health effects of coal -- which total half a trillion dollars per year.¹²

Coal export projects would have a reverberating impact in our region, as coal dust increases mercury and many other toxins in our air and our water; and then, when it is burned in China, as the prevailing winds bring air-borne toxins back to us...

To preserve the health and human potential of our children, I urge you to oppose Northwest coal export projects in any way possible.

What does the DEIS disclose about mercury?

- Mercury is one of four primary contaminants found in the broader Columbia River basin.
- Trace elements of environmental concern (TEEC) in Powder River and Uinta Basin coal include mercury.
- All scenarios show an increase in mercury deposition by 2040.

In Appendix I, the DEIS estimates mercury deposition resulting from coal burning in Asia. It estimates the maximum contribution from the coal exported from the MBT terminal would be less than 0.3%. We believe that this estimate is too low, because Asian mercury emissions were estimated to contribute between 5-36% of total mercury deposition on the US (p. I-6). Mt. Bachelor mercury levels from Asian emissions were 18% and 14% (p. I-7) and the Asian Hg^{II} is largest at low latitudes (where most people in Washington live). We must not overlook or minimize the fact that mercury would keep blowing back to Washington residents and others as long as the coal is burned, replenishing the supply of dangerous forms of mercury.

The DEIS should disclose by what percentage Asian Hg emissions will increase under the proposal. These numbers are not provided in Appendix I, which instead gives estimates of Hg deposition here. Again, we believe these estimates are low.

A direct result of the MBT will be a substantial increase in airborne pollutant emissions from train and marine traffic from the Powder River Basin and the Uinta Basin, all through the rail transportation corridor, at the terminal site, and on and near

¹² Epstein PR et al. Full cost accounting for the life cycle of coal. Annals of the New York Academy of Sciences, vol 1219, pp 73-98, 2011.

the Columbia River. If MBT is not built, these significant increases in negative impacts will not occur.

D. The DEIS understates the health risks and costs to human health from coal dust.

The amount of coal dust that escapes from Powder River Basin coal trains has been estimated by Burlington Northern Santa Fe (BNSF) railroad to be from 500 pounds to 1 ton per car, or up to 3% of transported coal (BNSF, 2011). A study on a West Virginia rail line, transporting bituminous coal similar to the coal from the Powder River Basin, showed a similar loss of coal dust of up to a pound of coal per mile per car. (Simpson Weather Associates, 1993). BNSF reports that escaped coal dust on the tracks can increase risk for derailments. Visible coal dust can be a costly pollutant requiring frequent cleaning for businesses and residences along a rail line or near a coal terminal as documented in a study from British Columbia (Cope et al., 1994).

In addition, recent data from Australia underscores our concern that “real world” measurements do a substantially better job predicting what will happen than the models used in the DEIS. In a recent study in Australia, monitors showed dramatic spikes—including spikes that exceed levels set to protect human health and safety—when uncovered coal cars passed by. One particularly startling finding of this study was that empty coal trains had higher particulate pollution than loaded ones. (See MBT DEIS comments and exhibit from Columbia Riverkeeper *et al.*) However, the DEIS dismisses pollution concerns from empty cars, an omission that must be rectified in the FEIS.

A significant article from Dr. Dan Jaffe et al., from University of Washington in 2015 quantifies emissions from diesel engines and coal dust from unit trains that travelled through the Columbia River Gorge.¹³

Furthermore, Dr. Jaffe, Professor of Atmospheric Chemistry at University of Washington, submitted comments in June on the DEIS after reviewing Chapter 5.7: Coal Dust. He states:

- The DEIS describes an “acceptable level of dust deposition” in terms of g/m²/month. However, nowhere does the document describe an acceptable level of human health impacts. Our data demonstrates short-term PM_{2.5}

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http://www.atmos.washington.edu/jaffegroup/modules/APOLLO/Jaffe_DPM_coal_dust_trains_ColumbiaRivGorge_2015.pdf

concentrations of up to 232 ug/m³ due to coal trains. These exposures were documented on private property adjacent to rail lines in the Columbia River Gorge (Jaffe et al 2015). This was due to a large, clearly visible cloud of coal dust. While the health effects of such short-term exposure have not been extensively studied, some peer-reviewed published scientific papers have documented significant health effects from short-term exposure to PM_{2.5} (Salvi et al., 1999; Pope et al., 2015; Li et al., 2016).

- The DEIS seems to imply that coal dust does not contribute to inhalable particulate matter that contributes to human health problems (eg PM_{2.5}). This is clearly not the case. To quote from the DEIS Coal Dust fact sheet: “The study found that coal dust particles from rail cars are typically large and fall close to the rail tracks.” Our data, and the Cowlitz County data, clearly indicate that coal dust constitutes a range of particle sizes including particles smaller than 2.5 micrometers in diameter. The EIS needs to clearly indicate that coal dust includes inhalable PM_{2.5}.
- The DEIS reports on one study down in Cowlitz County (pg 5.7-5 main document and 2.2-4 in technical supplement). There are a number of problems associated with this study. First, this study was likely influenced by an inherent bias due to the fact that the shipper knew the date, time and location of the tests. They can then utilize their best operating conditions to minimize diesel, coal dust and other PM impacts. This is not the same as a “blind” test, whereby the shipper is not notified of the date and location of the testing. In particular, a “blind” test will identify the true failure rate for the surfactant and true coal dust emissions, whereas the biased test will not identify the true failure rate. This study apparently never saw a coal train with a PM_{2.5} concentration greater than 22 ug/m³ (Figure 4), whereas we identified coal trains with large dust plumes and much higher PM_{2.5} concentrations of up to 232 ug/m³. This is likely due to the fact that the shipper was aware of the testing that was taking place.

Despite this point,

the data from the Cowlitz study clearly show that the dust suppression methods are much less than 100% effective. This is because the observed concentrations are 4 times higher than the modeled concentrations, as shown in Figure 4 of the SEPA Coal Technical Report. Models are useful to estimate many environmental situations, but they must be constrained and confirmed by observations. In this case, the model is clearly not reproducing the observed concentrations. This can only be explained if the modeled coal dust emissions are much larger than what is being put into this model. The Cowlitz County data, shown in Figure 4, indicate that the model is under-estimating the coal dust emissions by about a factor of four. This would imply that the actual coal dust emissions are four times greater than the modeled emissions. The DEIS cites a dust suppression effectiveness of 61%, whereas the data indicate an effectiveness of only 25%. Section 2 in the technical document ends with Figure 4 and does not discuss the implications of this large discrepancy between the model and observations. It is absolutely essential that the modeling be redone with the significantly higher, and correct, coal dust emissions. Based on both the UW and Cowlitz County studies, the data show much higher dust emissions than are accounted for by the modeling.

- Based on these results, the modeling should be redone using two different approaches: First, the model should assume that 5% of the coal trains had no surfactant (equivalent to saying that the surfactant was incorrectly applied or otherwise ineffective). The model results should then be re-evaluated based on this assumed failure rate. Second, the modeling should be redone with a coal dust emission factor that is four times larger. This would be consistent with the Cowlitz County data presented in Figure 4 of the SEPA Coal Technical Report. This is particularly important given that the trigger level for impacts (2 g/m²/day) are already exceeded for some receptor location (Table 5.7-7) or very close to the currently model estimates (1.88 g/m²/day, Table 5.7-3) and that human health impacts from short term exposure to high concentrations have been documented.
- Our peer-reviewed and published scientific analysis (Jaffe et al., 2015) clearly indicates that the surfactant coating does not always work. At present there is no information on the cause of these failures. As such, it is impossible to know if additional coating facility (e.g. requiring a facility in Pasco) will

significantly reduce coal dust emissions. The EIS needs to address what are the causes for failure in the surfactant coating.

- The coal dust modeling (Table 5.7.2) fails to incorporate any failure rate into the calculations. Our data show that the failure rate for moving trains is at least 5%. The contribution from these coal trains where the surfactant has been misapplied (or not applied at all) likely dwarf the emissions from all other trains.

1. Health Impacts of Coal Dust and Environmental Contamination

Deposition of coal from transport spills and dust may lead to contamination of soil, fresh water sources and the marine environment. Coal contains arsenic and heavy metals such as lead, boron, chromium, cadmium, and mercury (see summary contaminants in coal in Gottlieb et al., 2010). Contamination of farmland, animal pasture, and especially fisheries can impact human health.

Arsenic from coal dust can persist in soil for years and has been shown to be a pollutant originating from a coal shipping terminal (Bounds and Johannesson, 2007). Arsenic concentrates in food crops such as apples and rice and is associated with increased rates of skin, bladder and lung cancers, cardiovascular, and lung disease.

Because of the negative effects of mercury on neurologic development, pregnant women and young children are advised to limit their consumption of certain kinds of fish with increased mercury content (FDA/EPA Consumer Advisory, 2004). While mercury in coal dust is less biologically active before it is burned, mercury from coal burned in China is carried in the air across the Pacific Ocean to the west coast of the United States and across the country. Fourteen percent of the mercury in the Great Lakes originates in China (National Oceanic and Atmospheric Administration, 2011).

2. Health Impacts of Airborne Coal Dust

The DEIS acknowledges that so much dust is produced by coal trains that it creates a safety hazard by destabilizing rail road ballast (DEIS 5.7-15). The point is well taken, as coal dust accumulation in railroad ballast has been documented as a factor in derailments, and BNSF has recently undertaken significant efforts to remove coal dust near the the Columbia River and elsewhere. However, the DEIS does not acknowledge the huge inconsistency between its modeled conclusions of “insignificant” dust deposition with the known experience that so much coal dust is escaping that it is destabilizing rail infrastructure.

Another point that demonstrates that the DEIS model-based approach is inconsistent with known experience is hidden in the technical report itself. Figure 4 of

the coal dust technical report compares the “modeled” emissions of coal dust with the actual emissions as measured during the October 2014 test. As Dr. Dan Jaffe has pointed out in his independent comments, actual emissions are four times higher than the modeled emissions. Even so, the DEIS conclusions are all based on the modeled emissions, likely understating the dust impacts by a considerable degree. This must be corrected in the FEIS.

Coal dust is associated with:

- Chronic bronchitis;
- Emphysema;
- Pulmonary fibrosis (pneumoconiosis); and
- Environmental contamination through the leaching of toxic heavy metals.

Airborne coal particles pose a potential health risk to workers and to people in communities near railroad tracks, as well as near the mines and the proposed export terminal. Health risks of airborne coal dust to coal miners have been well documented to cause lung disease, ranging from severe pneumoconiosis to chronic bronchitis and exacerbations of asthma (Hathaway, et al., 1991).

While pneumoconiosis has only been conclusively associated with intense exposure, there is evidence that lower levels of respirable coal dust may also cause lung disease. A recent study (Wade et al., 2010) examined miners who developed lung disease even while exposed to currently legal and well-regulated levels of coal dust. Animal studies (Vincent et al., 1987) have examined the pulmonary effects throughout a wide range of coal dust exposures. They show that pulmonary clearance mechanisms tend to sequester the dust in lymphatic tissue and the interstitial space between alveoli. This inhibits further clearance mechanisms and facilitates the inflammatory cascade in the lung tissue. In addition, the synergistic effects of respirable coal dust with other pollutants such as diesel particulate matter may accelerate lung damage beyond that which might be predicted by the coal mine epidemiologic data (Karagianes et al., 1981).

What does the MBT DEIS disclose about air pollution and coal dust?

- Table 6-21 shows violations of the National Ambient Air Quality Standards (NAAQS) for particulate matter (PM_{2.5}) from coal dust in Cowlitz County.
- “The estimated maximum monthly coal dust deposition along the BNSF main line in Cowlitz County would exceed the trigger level for certain residential receptors (Table 5.7-7).” (Chap. 5 at 5.7-21.)
- “[R]esidents who live along the main line could experience nuisance levels [of coal dust] which may visible soiling on window sills, outdoor

furniture, and other property.” (Chap. 5 at 5.7-21.)

- “The average and maximum deposition of coal dust on the BNSF main line in Cowlitz County was estimated to be above the nuisance thresholds at 50 and 100 feet, and because no state or federal standards apply, this an unavoidable but not significant impact.” (Chap. 5 at 5.7-25.)
- Table 6-22. Estimated Average Maximum and Maximum Monthly Coal Dust Deposition—BNSF Main Line *in Cowlitz County* PM 2.5 and PM 10 are expected to exceed NAAQS.
- Table 6-24. Estimated Average Maximum and Maximum Monthly Coal Dust Deposition—BNSF Main Line *in Columbia River Gorge* Average maximum and maximum monthly coal dust deposition for Columbia Gorge would be exceeded. (Chap. 6 at 6-69.)
- Table 6-26. Estimated Average Maximum and Maximum Monthly Coal Dust Deposition—BNSF Main Line *in Eastern Washington*. The estimated average maximum monthly coal dust deposition is above the trigger level at 100 feet from the rail line.
- Toxic constituents of coal include polycyclic aromatic hydrocarbons (PAHs) and trace metals, which are present in coal in variable amounts and combinations dependent on the type of coal. (DEIS p. 4.8-23)
- The concentration of PAHs in Powder River Basin coal was not investigated. (DEIS. 4.5-26)
- Seventy percent of the Cowlitz County air toxic cancer risk is due to just three air toxics: PAHs, formaldehyde, and benzene. (DEIS p.5.7-16)

Note that Table 5.7-3 (Estimated Maximum Annual and Monthly Coal Dust Deposition) utilizes a trigger level for sensitive areas based on a New Zealand Ministry of the Environment level for nuisance dust. This is not a health based measure. This is a misleading comparison in that it serves to minimize impact of the coal dust deposition for the study area by comparison with a non-health based number.

The Health Department in Multnomah County, Oregon analyzed potential health impacts to County residents from coal train transportation only. The analysis

drew upon the available literature to estimate that coal dust may travel approximately 500 m to 2 km (1/3 to 1 1/4 miles) from the train tracks, depending on weather conditions and train speed.^{57,58} Census tracts—relatively small geographic areas used for census-taking—offer a rough

proxy for the 2 km distance from the rail line. Using this approximation allowed the Health Department to utilize Census Bureau data to describe potentially affected populations. Almost one-third of Multnomah County's population lives in census tracts that either border or cross rail lines that may carry coal... Many of these people live near major roadways and industrial areas and probably already experience a high burden of air pollution and noise disturbance. Accordingly, the potential burdens of the coal export projects would fall on the same populations who are already exposed to the highest levels of air toxins and industrial noise. (See EXHIBIT: Multnomah County Coal Report, 2013.)

The FEIS should also consider evidence from Australia, which has had a long history of large coal-export terminals with open coal stockpiles and extensive experience with the pollution that they cause. One analysis for a new terminal in Newcastle, performed as part of the project's license, shows that it would discharge over 300,000 kg/year of coal dust at operations of 66 million ton/year. The analysis breaks down the emissions rate for each stage of the process. The largest source of emissions is from wind erosion of stockpiles. Another analysis, based on data from Australia's National Pollutant Inventory¹⁴--that nation's most authoritative data source for pollutant information—shows that coal terminals were the primary sources of particulate air pollution in two areas where major coal terminals operated.

The Hay Point coal terminal in MacKay self-reported a release of 160,000 kg of PM10 and 17,000 kg of PM2.5 in 2014-15. A news report from April of this year reported that the three coal export terminals in Newcastle were responsible for 62% of that city's PM10 air pollution.¹⁵ These authoritative figures collide sharply with the DEIS's modeled emissions—which anticipated releases an order of magnitude lower, using the exact same approaches to reduce dust from open stockpiles. Clearly, the real-world experience in Australia has more to offer than the flawed models of the DEIS.

The MBT DEIS contains recommended mitigation measures, many of which are unenforceable and speculative. For example, to address the impacts of coal dust from trains, the DEIS states: "BNSF should conduct a dust monitoring study along BNSF main line in Cowlitz County to evaluate coal dust emissions from coal trains, and if necessary, take further actions to reduce such emissions." However, BNSF is not applying for any permits for the Millennium project.

No meaningful attempt is made on the part of the applicant to prevent exposures resulting from its projected activities. For example, MBT could enclose the predicted 85-foot high piles of coal at the terminal. Ambre Energy proposed such a plan for the Morrow Pacific project in Boardman, Oregon. But MBT has no intention to do so. It can therefore be predicted that adverse health impacts will result from the MBT project.

¹⁴ <http://www.npi.gov.au/>

¹⁵ <http://www.smh.com.au/environment/air-pollution-increases-69-per-cent-as-coal-named-top-polluter-20160417-go8b82.html>

Mitigation measures to address coal dust near the terminal include creating a system for people to report coal dust complaints. (DEIS Coal Dust Fact Sheet at 4.) This is unacceptable. Reporting will not reverse the negative health impacts already experienced and the health care costs already incurred.

The only mitigation for coal particulate pollution - a health hazard, an environmental issue, and a nuisance - is to eliminate the pollution.

E. The DEIS fails to disclose potential health risks and costs to human health from exposure to surfactants.

The 2013 Multnomah County coal study states:

The company states that the proper application of certain topper agents along with the use of a modified loading chute can potentially reduce coal dust levels by at least 85 percent. However, there is no evidence of independent verification of these findings. In a series of cases before the federal Surface Transportation Board, utility companies that are required to follow BNSF Railway's rules for shipping coal have argued that there is insufficient evidence for the effectiveness of these substances and that shippers should not be responsible for the costs of applying them.³⁸⁻⁴¹

BNSF requires the use of these chemical agents by companies shipping coal from mines. Given that they are used at the mine and it has been found that they need to be reapplied along the route (e.g., at Pasco), then what is the fate of these surfactants or dust suppressants in Longview and along the transportation route (from the Powder River Basin and the Uinta Basin to Longview, and back)?

Six topper agents have been approved for use on rail cars shipping coal on BNSF railroads. (See BNSF Rules and Other Governing Provisions, Appendix B, BNSF 6041-B, Page 19, September 2011) These topper agents have been tested for effectiveness in dust suppression. But information is not given in the DEIS on their fate and transport or toxicity in the environment. If they are washed off by rain or through fire suppression efforts or lost with blowing coal dust (while losing efficacy in time along the route), what is their impact on ground and surface water, on plants and animals that may be exposed to them, or to humans who may be exposed by ingestion, inhalation, or dermal absorption of these chemical compounds?

While dust suppressants are available for use, little or no information is available on their chemical make-up or toxicity to human health or the environment. The DEIS must identify all components of dust suppressants or "topper agents" in order to determine whether there is risk associated with their use.

F. The DEIS understates the health risks and costs to human health from noise pollution.

Noise pollution is a growing health concern in this country and around the world. The World Health Organization has recognized it as a major threat to human health and well-being. Some of the well-documented adverse health effects include sleep disturbance, cognitive impairment, cardiovascular disease, including increased blood pressure, arrhythmia; stroke and ischemic heart disease, increased rate of accidents and injuries, hearing loss, and exacerbation of mental health disorders.

Coal trains produce significantly greater noise and vibration than other trains. Longer trains mean more prolonged noise, greater weight means increased vibrations and more wheel squeak noise, and more locomotives per train are required, resulting in more engine noise. Indeed, people can tell whether it is a coal train or not without looking at it, simply based on the noise and vibration they experience.

Noise and vibrational effects from rail traffic vary from idling, rumbling, screeching, and horn sounding. Horn sounding poses the most significant risk to human health. The U.S. Department of Transportation's "Train Horn Rule" (49 CFR Part 222) requires engineers to sound train horns at 96 to 110 decibels (dB) fifteen to twenty seconds in advance of all public grade crossings (US DOT, 2005). The World Health Organization's "Guidelines on Community Noise" (1999) state that sleep disturbances and risk of adverse health effects of noise have been observed at 45 dB or less, recommending that noise events exceeding 45 dB be "limited if possible" (p. 8).

Like many of the previously listed health concerns, the noise of coal trains could represent an increase to an existing health problem. A person awakened from sleep every hour -- as would be expected when the MBT is at full operation -- would experience a different order of magnitude of adverse health impacts than a person awakened or otherwise disturbed once or twice a night from existing train traffic. The train traffic associated with MBT will directly impact multiple dense residential areas along the entire rail line in several states.

According to Washington noise standards, the maximum permissible levels for Class A (residential) are 55 dBA in the nighttime and 45 dBA at night. These are not averages. The DEIS relies too heavily upon averages. Even so, it discloses that approximately 60 residences in Longview would be exposed to severe noise impacts with coal trains. It also states that "absent the creation of a Quiet Zone, the potential adverse indirect noise impacts during rail operations along the Reynolds Lead would result in a disproportionately high and adverse effect on minority and low income populations. This potential disproportionately high and adverse effect would affect a total of approximately 289 residences located in Census Tract 3 Block Group 1, Census Tract 5.02 Block Group 1, and Census Tract 5.02 Block Group 2, all of which have been identified as minority communities (see Table 18)." (DEIS at 3-13)

Hundreds of thousands of other people along multiple transportation routes will likely experience severe noise impacts and sleep disruption multiple times through the night as a direct result of MBT. The FEIS should disclose these impacts to other communities, including environmental justice communities away from the project site.

1. The DEIS Fails to Adequately Disclose Impacts of Noise on Sleep Disturbance

Noise can have both auditory and non-auditory deleterious effects on human health. Auditory effects include delay in falling asleep, frequent night time awakenings, alteration in sleep stages with reduction of REM sleep, and decreased depth of sleep. Non-auditory effects, including increased blood pressure, increased heart rate, vasoconstriction, changes in respiration, and arrhythmia, continue to have deleterious effects on human health even after the subject has acclimated to the noise. Decreased alertness from sleep disturbance is associated with an increased rate of accidents, injuries and premature death.

Studies have shown that noise >55 dB (night, outside level) is associated with sleep disturbance, that railway noise has greater impacts than road noise, and that even a single railway noise event significantly decreases REM sleep (Aasvang et al., 2011; Brink et al., 2011; Carter NL 1996; Chang et al., 2012; Clark C. et al., 2012; Halonen JI et al., 2012; Hong J et al., 2010; Hume KI 2011).

The health implications of chronic and nocturnal noise from increased rail traffic are highly likely and will impact human health in multiple areas and all age ranges (Goines & Hagler, 2007; Babisch, 2005).

A case-control experimental study found that train transportation noise and subsequent vibrations led to a significant acceleration of heart rate of at least 3 beats per minute in 79% of sleeping participants after experiencing high-vibration periods produced from trains passing. These nocturnal heart rate accelerations are believed to potentially affect long-term cardiovascular health for populations living in close proximity to railroads with frequent rail traffic (Croy et al., 2013).

Despite public safety mitigation in some urban areas where quiet zones have been established (at great expense to taxpayers), a growing body of research demonstrates that continuous noise, as well as sudden intermittent nocturnal noise and vibrations, result in sleep disturbances and can cause impaired cognitive function and cardiovascular effects.

2. The DEIS Fails to Adequately Disclose Impacts of Noise on Cardiovascular Disease

In adults, both short-term and long-term adverse health effects have been documented, including increased blood pressure, increased heart rate, vasoconstriction, elevated stress hormones such as epinephrine and cortisol, arrhythmias, ischemic heart disease, and strokes.

Specifically, the elevation of stress hormones such as epinephrine and cortisol resulting from high decibel noise exposure increases endogenous risk factors of heart disease from both short-term and chronic exposures (Ising & Kruppa, 2004; Selander et al., 2009; Sørensen et al., 2011; Sørensen et al., 2012).

Sleep disturbance resulting in fatigue increases risk of hypertension, arrhythmia, and risk of accidental injury. Recent epidemiological studies support previous evidence that night-time noise and noise in excess of 90 decibels are associated with cardiovascular disease including risk of acute coronary events, myocardial infarction, arrhythmia, accelerated hypertension, and stroke (Hume et al., 2012),

3. The DEIS Fails to Adequately Disclose Impacts of Noise on Cognitive Impairment in Children

Children exposed to increased noise have shown lower academic achievement in various forms including long term memory, reading comprehension, learning, problem solving, concentration, social and emotional development, and motivation (Clark, C et al., 2012; Cohen, S. et al., 1980; Evans GW 2003; Evans GW and SJ Lepore, 1993; Evans GW and L Maxwell, 1997; Haines MM et al., 2001; Haines MM et al. #2, 2001; Hygge S et al., 2002; Stansfeld SA et al., 2005).

Increased stress-related hormones and elevated blood pressures have especially been seen in children with lower academic achievement (Selander J 2009; Sorensen M et al., 2012; Sorensen M et al. #2, 2012; Sorensen M et al., 2011; Willich SN et al., 2006).

The health implications of chronic and nocturnal noise from increased rail traffic are highly likely and will impact human health in multiple areas and all age ranges (Goines & Hagler, 2007; Babisch, 2005).

4. The DEIS Fails to Adequately Disclose Impacts of Noise on Mental Health

Another less well appreciated risk of high decibel rail noise and vibration exposure is the impact on mental health. Increased noise is known to accelerate and intensify development of latent mental health disorders including depression, mental instability, neurosis, hysteria, and psychosis. It is also a major environmental cause of annoyance leading to diminished quality of life (Evans GW et al., 1995; Fidell S et al., 1991; Haines MM et al., 2001; Haines MM et al. #2, 2001).

Elevated noise is associated with cognitive impairment in children, as well as exacerbation of mental health disorders including depression and anxiety (Lercher, et al., 2003; Haines et al., 2001; Hygge et al., 2002).

G. The DEIS understates the risks and costs of delayed emergency response times from increased frequency of long trains and delays at at-grade crossings.

Increased frequency of very long trains at rail crossings will lead to delayed emergency medical service response times and to increased accidents, traumatic injury

and death. We request a comprehensive HIA that discloses related risks and costs along the entire rail corridor as part of the FEIS.

For many of our most common acute health issues, such as stroke, heart attack, massive hemorrhage, and trauma, every second counts. A delay of just a few minutes can mean the difference between life and death or permanent impairment and disability. Hospitals routinely measure parameters such as “door to balloon time,” the length of time it takes from the arrival in the Emergency Department until the moment the artery is successfully opened, in the case of a heart attack, to measure the quality of the care delivered and improve outcomes. The same is true for stroke, where thrombolytic medications given to break down clots and to open occluded arteries to the brain can be given only if administered within three hours of the onset of symptoms. Failure to promptly re-establish arterial blood flow to the heart and brain leads to cell death and permanent injury very quickly.

There are many locations in Cowlitz County and other counties along the rail corridor where residents may be cut off from emergency medical services by rail lines and access to timely healthcare impaired by increased rail traffic. We are also aware of communities in the state where rail lines separate the major population densities from the hospital or EMS facilities. In many cases, an ambulance must cross a set of tracks twice to bring a patient to a hospital. Emergent procedures may also be delayed when critical personnel (such as physicians, nurses, anesthesia techs, or people transporting blood for transfusion) are delayed en route to meet a patient at a hospital.

The DEIS discloses many of these challenging rail traffic impacts that could delay emergency response times:

- “Trains related to the Proposed Action could affect accessibility to community resources and public services during peak travel times because of increasing wait times at grade crossings along the Reynolds Lead, BNSF Spur, and BNSF main rail line.” (Summary at S-12)
- “Trains related to the Proposed Action would also increase emergency vehicle delay at rail crossings. The total gate downtime would increase over 130 minutes a day at crossings along the Reynolds Lead and BNSF Spur, and up to 20 minutes a day at the study crossings along the BNSF main line.” (Summary at S-32)
- Table 6-6 shows every segment on the rail system greatly over capacity if all proposed projects, including coal and oil, are built.
- Table 6-7 shows a significant increase in train accidents (this

analysis includes oil trains).

- Table 6-9 shows unacceptable level of service at multiple rail crossings due to delays from cumulative projects.
- The probability of an increase in emergency response time at all at-grade crossings would also increase because at-grade crossings would be blocked more frequently.
- 16 coal trains each day in Longview will result in over two hours of additional rail gate downtime at one at least one location.
- Spokane County will experience 13-22% increase in trains/day.
- The 5 study crossings with the largest increase in vehicles delays are all in Spokane County.
- Franklin Co. (Pasco) will experience 22% increase in trains/day.
- Yakima Co. (Yakima) will experience 42% increase in trains/day.
- Assuming coal trains travel at the same freight train speeds identified in Table 5.3-13, the five study crossings (of 44 study crossings located near or across state highways in Washington only) with the largest increase in daily vehicle delay compared to baseline 2028 conditions would be the following:
 - Big Hanaford Road, Lewis County (8 coal trains daily, 10 mph)
 - Pine Street, Spokane County (16 trains coal daily, 35 mph)
 - F Street/Cheney-Spangle, Spokane County (16 coal trains daily, 35 mph)
 - Cheney-Plaza Road, Spokane County (16 coal trains daily, 35 mph)
 - Russel Avenue, Skamania County (8 coal trains daily, 20 mph)
(Chap. 5 at 5.3-39)
- When factoring in existing annual average daily traffic, the five study crossings with the largest increase in vehicle delay compared to the baseline 2028 conditions would be the following:

- Pines Road-SR 27, Spokane County (16 coal trains daily)
- Park Road, Spokane County (16 coal trains daily)
- Barker Road, Spokane County (16 coal trains daily)
- Harvard Road, Spokane County (16 coal trains daily)
- Flora Road, Spokane County (16 coal trains daily)

The DEIS fails to disclose likely impacts on all at-grade crossings in transportation corridors in Washington and fails to disclose similar impacts in any communities in Utah, Colorado, Wyoming, Montana, Idaho, and Oregon.

The DEIS shows that coal trains will travel through the city of Portland in Multnomah County. The 2013 report “The Human Health Effects of Rail Transport of Coal Through Multnomah County, Oregon” states:

Because of their length (up to 1 1/4 miles long) and low speed, coal trains could block roadways for relatively long periods of time. In densely settled areas, such as the city of Portland, this could result in a cumulative delay of up to two hours per day at each crossing.⁵⁴ It could also disrupt routes and increase response times for emergency vehicles called to fires, medical incidents, and other public safety crises.

H. The DEIS fails to disclose the risks to human health and safety from rail accidents.

Among the more startling admissions of the DEIS is that the project will proximately cause a substantial increase in the number of rail accidents—a 22% increase statewide. What is not disclosed is any meaningful analysis of the potential safety, human health and environmental risks of such accidents. Increased rail traffic of the magnitude that is proposed has significant potential for increased traumatic injury and death at rail crossings or by derailments. Many crossings on the rail corridor in several states have no barriers or other warning signals, and local city, county, and state governments are struggling financially with limited funds for providing this basic safety service. Data from the Federal Railroad Administration Office of Safety inform us that there were 739 fatalities and 8,167 injuries at railroad crossings nationally in 2010. There were at least 19 coal train derailments in North America in 2012, including fatalities.

The DEIS also discloses:

- Unavoidable and Significant Adverse Environmental Impacts: “Proposed Action-related trains could increase the number of potential train accidents along in the rail routes in Cowlitz County and Washington State.” (DEIS at 5.2-10)
- Unavoidable and Significant Adverse Environmental Impacts: Without improvements to increase capacity, the Reynolds Lead; BNSF Spur; and three

segments on the BNSF main line routes in Washington State (Idaho/Washington State Line–Spokane, Spokane–Pasco, and Pasco–Vancouver) are not projected to have the capacity to handle the projected baseline rail traffic and Proposed Action-related rail traffic in 2028. (DEIS at 5.1-24)

- Over 11 additional rail-related accidents are predicted every year statewide, with an additional accident every year in Longview. When looked at in terms of cumulative risk including other proposed projects, the DEIS predicts 19 coal train accidents per year.
- Coal trains would increase the accident probability at all at-grade crossings because 8 or 16 coal trains would pass at each crossing depending on location, and the Proposed Action would not change crossing protection at the study crossings.
- BNSF and UP could address safety issues as they emerge using capital improvements or operational changes, but it is unknown when those actions would be taken or permitted.

But the DEIS fails to disclose likely injuries and death from accidents at all at-grade crossings and along transportation corridors in Washington and fails to disclose similar impacts, including potential public health emergencies, to rail line communities in Utah, Colorado, Wyoming, Montana, Idaho, and Oregon.

I. The DEIS understates the number of increased derailments leading to injury, death, and public health emergencies.

The DEIS fails to disclose likely injuries and death from derailments in all transportation corridors in Washington and fails to disclose similar impacts to any communities in Utah, Colorado, Wyoming, Montana, Idaho, and Oregon.

Just this month, a unit train carrying Bakken crude oil derailed in the Columbia River Gorge near Mosier, Oregon, creating a massive fire, depleting the city's drinking water source, destroying the city's sewer system, closing an interstate highway, leaking oil into the Columbia River and threatening tribal fisheries. Initial reports blamed the incident on track failure.

The FEIS must disclose how frequent operations of coal unit trains—among the longest and heaviest trains on the rail system—contribute to higher-than-normal degradation of rail infrastructure, increasing the risk of rail accidents, injury, public health emergencies, and death. Given the desire to substantially increase the number of crude oil trains on the regional rail system, the FEIS needs to look closely at the extent to which the project will contribute not just to accidents generally but to crude oil

accidents specifically. Any increase in the risk of a crude oil accident it totally unacceptable.

J. The DEIS fails to adequately analyze the risk of fires and related dislocation, injury and death.

The DEIS inadequately addresses fires and how these may cause burn injuries and respiratory problems for individuals in and near the terminal, as well as people living in communities along the rail route, including low-income individuals and communities, minority populations, and individuals with pre-existing respiratory disease.

MBT will be dealing with a hazardous type of coal. Powder River Basin (PRB) coal is notorious for the hazard it presents regarding fires and explosions^{1, 2, 3, 4, 5} (de Place, E, 2016; Khambekar & Barnum, 2013; Doubery, 2013; Smoker & Albinger; Hossfeld & Hatt). This was a conclusion by NIOSH following an investigation into the deaths of two firefighters. They were killed in an explosion when trying to put out a fire of PRB coal^{6, 7} (Ellis, B, 2013; NIOSH, 2012).

Self-combustion of coal presents a fire risk⁸ (USDOE, 1993) and this is an even greater problem with PRB coal which is twice as likely to self-combust than other types of coal^{6, 9} (Ellis, B, 2013; Merritt & Rahm, 2000). It will not only smolder and catch fire while in storage piles at power plants and coal terminals, but has been known to be delivered to a power plant with the rail car or barge partially on fire^{1, 10, 11} (de Place, 2016; groundtruthtrekking, 2014; Fox23, 2014).

PRB coal dust is also a fire hazard^{9, 1e} (Merritt & Rahm, 2000; Block, S.). Sparks from machinery and heat from conveyor belts have caused major coal dust fires and explosions^{4, 13, 14} (Smoker & Albinger; VandenHeuvel & de Place, 2011; Casper Star Tribune, 2013). The potential for fires along the entire rail route is also a concern. BNSF has stated that coal dust deposits have caused fires in areas where coal dust has accumulated¹⁵ (BNSF Railway). A fire department in Wyoming has found that coal fires along railroad tracks account for at least 50% of the department's summer call volume¹⁶ (West Antelope II Coal Lease Application).

Operators familiar with the unique requirements of burning PRB coal say that it's not a case of "if" there will be a PRB coal fire, it's "when."¹⁷ (dePlace, E, 2014)

The 2013 report "The Human Health Effects of Rail Transport of Coal Through Multnomah County, Oregon" states:

...Powder River Basin coal may be particularly susceptible to spontaneous combustion as a result of its chemical composition. According to discussions between mining and energy companies

that handle Powder River Basin coal, there have been reports of fires in railcars and barges transporting this type of coal.

Given coal's combustibility, fires and attendant injuries and property damage could also occur as a result of a train collision.

The FEIS must identify and analyze the risk of fire along with associated risks of injury, dislocation and death at the terminal in Longview and throughout the transportation corridor, including in forested areas like the Columbia Gorge, Stampede Pass, and communities east and west of the Cascades.

K. The DEIS fails to adequately analyze the risk to human safety from wildfires.

The DEIS discloses that Cowlitz County is considered a high-risk area:

Cowlitz County is considered a high-risk area (Washington State Emergency Planning Division 2012c). A wildfire could affect the project area from the undeveloped areas adjacent to the project area or a Proposed Action-related train in the study area. Wildfires in Cowlitz County numbered more than 350 from 2004 to 2013, burning more than 561 acres. In late summer and early fall, dry easterly winds can produce extreme fire conditions. This threat has increased over time because of four climate-related factors: earlier snowmelt, higher summer temperatures, longer fire season, and an expanded vulnerable area of high-elevation forests (Washington State Emergency Planning Division 2012c) (DEIS at 5.8-32)

At Stage 2 (Full Build-out Operations), there could be 1.5 million metric tons of highly combustible PRB coal stored at the project site (DEIS 2-24). A wildfire leading to a terminal fire would have serious health impacts to workers and neighbors near and far and so must be fully analyzed in a Health Impact Assessment.

L. The DEIS fails to adequately analyze the risk to human health and safety from a vessel oil spill.

The DEIS demonstrates significant impacts from MBT's unprecedented proposal to increase vessel traffic by 44 percent in the Columbia estuary. The DEIS does not include a qualitative or quantitative risk analysis of bunkering (*i.e.*, refueling) associated with 840 vessels per year calling on MBT. The DEIS, however, acknowledges oil spill risks associated with bunkering, stating:

Increased vessel traffic associated with the Proposed Action also has the potential to result in an increased risk of oil spills during bunkering activities. Causes of oil spills during bunkering transfers include

overflow of the tank, parting the hose due to mooring fault, operator error in connecting the hose, failure of the hose or pipework, and failure of bunker tanks (HSE 2012). Experience from insurance claims (Gard 2002) is that most bunker spills result from an overflow of the bunker tank due to carelessness or negligence, either on the part of those supplying the bunkers, or those on board the vessel receiving them.

If an incident occurred that resulted in an impact, a fuel tank could be damaged and fuel spilled.¹⁶

Bunker fuel is a combustible liquid associated with acute health hazards and chronic health hazards. A Material Data Safety document shows potential health impacts of acute exposure:

- *Inhalation*: May cause irritation to the nose, throat and upper respiratory tract. Symptoms may include pain, headache, nausea, vomiting, dizziness, drowsiness and other central nervous system effects. Irritating or noxious gases may be released during thermal decomposition.
- *Releases*: Hydrogen sulfide. Severe respiratory irritation (from vapors or mists) is possible. Could also cause convulsions, coma, respiratory arrest and death.
- *Skin*: May cause mild to moderate skin irritation. Prolonged contact, such as when trapped against the skin under clothing or jewelry, may be more irritating. Can be absorbed through skin. Exposure to hot material may cause thermal burns.
- *Eyes*: May cause moderate eye irritation.
- *Ingestion*: May cause irritation of mouth, throat, and stomach. Symptoms may include pain, headache, nausea, vomiting, dizziness, drowsiness and other central nervous system effects.

Potential impacts of long-term (chronic) exposure:

- Prolonged skin contact may cause dermatitis (rash), characterized by red, dry, itching skin.
- Prolonged overexposure may cause liver and kidney effects.

Carcinogenic status: Possible cancer hazard

Fire hazards/conditions of flammability: Combustible liquid and vapor. Will ignite when exposed to heat, flame and other sources of ignition. Vapors are heavier than air and collect in confined and low-lying areas. Vapor can travel to ignition source and flash back. Product may float, and be re-ignited at the water's surface. Closed

¹⁶ SEPA Vessel Transportation Technical Report at 3-13 (April 2016).

containers may rupture if exposed to excess heat or flame due to a build-up of internal pressure.¹⁷

M. The DEIS fails to adequately analyze the risk to clean drinking water.

The DEIS discloses that “Day-to-day rail operations could release contaminants to water resources immediately adjacent to the rail line, resulting in the potential for water quality impairment from increased rail transportation.” (Summary at S-24.) That means multiple unidentified sources of precious drinking water could be impaired. The DEIS fails to identify by name and location all domestic and municipal water systems that could be harmed by a derailment and spill of coal trains and/or coal vessel fire and fuel spill. How many people are served by those systems? Who will pay for monitoring and cleanup when and if municipal drinking water sources are fouled in Washington, Oregon, Idaho, Utah, Colorado and/or Montana?

The MBT project area contains a critical aquifer recharge area (4.3-17). The DEIS discloses that The Mint Farm Regional Water Treatment Plant is approximately 6,000 feet east of the eastern boundary of the project area and supplies drinking water to about 45,000 residents of Longview and the surrounding area. While the study area does not extend to the Mint Farm Regional Water Treatment Plant, the project area lies within the Wellhead Protection Area (i.e., the 5-year Wellhead Protection Plan Source Area). (DEIS at 4.4-5)

An important document (Table 5-3 at p. 5-5, February 2012) demonstrates the flow of water in the Source Delineation Area.¹⁸

The plant draws from the deep aquifer, recharged by the Columbia River. Kennedy/Jenks Consultants (2010) completed a water quality and environmental risk assessment as part of the preliminary design report for the Mint Farm Regional Water Treatment Plant. The risk assessment included sampling and water quality analysis of the groundwater from the deeper aquifer of six wells. This study found no chemicals in the groundwater above their respective human health screening levels. (DEIS at 4.4-5)

However, in November 2012, Kennedy/Jenks Consultants repeated the water quality analysis from the same wells and found manganese and iron at levels above the Washington State Department of Health secondary water quality standards.

They also found that arsenic was present in one of the city’s drinking water wells, though at levels below thresholds established by the U.S. Environmental Protection Agency (EPA) for drinking water quality standards. (DEIS at 4.4-5,6)

Arsenic is present in PRB coal and Uinta coal. (See Leyda EXHIBIT and see Table 4.5-4 at p. 4.5-25.)

¹⁷ EXHIBIT: Material Data Safety Sheet Revised 1.12.2013 Heavy Fuel Oil, Bunker Fuel Oil

¹⁸ <http://www.mylongview.com/modules/showdocument.aspx?documentid=998>

Arsenic is present in the Columbia River. DEIS Table 4.5-5 demonstrates a proposed 303(d) listing for impairment for Columbia River in Oregon near River Mile 64 for arsenic. (Table 4.5-3)

With repeated exposure to arsenic-tainted DPM and arsenic-laden coal dust and with 1.5 million metric tons of coal sitting on site at full operation, it is possible that contamination of this drinking water source by arsenic and other pollutants could become a bigger problem than it currently is.

Groundwater in the study area is confirmed to have benzene and petroleum/gasoline contamination above cleanup levels. (DEIS 3.6-13)

The DEIS fails to disclose the fact that dredging and construction of the docks could impact drinking water. MBT dredging would increase water depth in the dredge prism by up to 16 feet (DEIS at 4.7-22). How will this impact the quality and quantity of drinking water and the movement of water in the city's wellhead protection area?

The DEIS fails to disclose the potential individual and cumulative impacts from a spill of bunker oil, emissions of coal dust, and exposure to diesel PM 365 days each year for 50 years at and near the terminal. These impacts can degrade the quality of drinking water for Longview residents.

The DEIS fails to clearly show what the effects of pre-operation wicking and compression may have on the movement of surface water or on the movement of legacy pollutants like benzene and arsenic, which could degrade drinking water.

The DEIS fails to disclose the potential impacts of heavy pumping of MBT's private wells during the dry season (for purposes of dust suppression) on the City of Longview's wells.

The DEIS fails to identify the contaminants and pollutants which will flow into the Columbia River as treated wastewater, untreated surface water or as overflow from storms. That water could include diesel pollution, toxic coal dust, fuel spills, asbestos, lead, and arsenic from demolition projects. Leaks and spills from associated barges, tugs, Panamax-class, and Handymax-class vessels can foul the water that recharges the drinking water aquifer.

Rainier's drinking water wells are located just upstream. Given tidal influences, that water source could be subject to contamination by the above pollutants as well. Rainier's designated well-head protection area is located near the project site and appears to overlap the project area.

The DEIS fails to identify those who will pay damages if the drinking water sources for the City of Longview and the City of Rainier are contaminated with pollutants as a result of this project and must be permanently replaced. It also fails to contemplate the cost of temporary replacement of clean drinking water.

II. THE DEIS UNDERSTATES THE HEALTH IMPACTS AND RISKS TO VULNERABLE INDIVIDUALS AND COMMUNITIES

The DEIS discloses that “Because the area along the Reynolds Lead where the indirect noise impacts would occur is a minority and low-income community (Figure 3.2-4), this analysis concludes that the Proposed Action would have a disproportionately high and adverse effect on minority and low-income populations.”

However, the DEIS fails to fully recognize the many other direct and indirect negative impacts of this massive project that would burden minority, low-income populations, and tribal communities in Longview and all along transportation corridors. This failure to appropriately scope EJ-related issues and its startlingly narrow definition of the project plan mean that the project’s primary EJ analysis is inadequate. The FEIS must incorporate a thorough and accurate analysis of impacts to Environmental Justice as they relate to human health and safety.

The EJ components of the FEIS should take into consideration both the HIA planned for the MBT analysis, and extend all EJ analyses to communities along the rail line, at the project site, in vessel corridors, and to those most impacted by the climate impacts of the project.

The health and safety of sovereign tribal members and entities can be directly impacted by increased train traffic, and by obstructing traditional access to the Columbia River. Along the length of the rail routes from the Powder River Basin to this proposal, dozens of indigenous tribes’ hunting and fishing rights could be impacted by obstruction of access to rivers and hunting grounds. With millennia of traditional access to fish and wildlife for subsistence harvest, any further degradation of fishing and hunting rights by new industrial projects must be taken into account.

The combined and cumulative harm that could come to fisheries from both oil and coal transport along Northwest waterways such as the Columbia River must be more fully considered. The DEIS understates the negative impacts of MBT to food and culture to tribes. “Operation of the Proposed Action would result in impacts on tribal resources through activities related to the Proposed Action causing physical or behavioral responses in fish, or affecting aquatic habitat. These impacts could reduce the number of fish surviving to adulthood and returning to areas upstream of Bonneville Dam, thereby affecting the number of fish available for harvest by the tribes.” We object to any project that causes significant impacts to tribal fishing.

The following words are taken from a prepared statement of the Yakama Nation given November 18, 2013, at an Oregon Physicians for Social Responsibility press

conference: “First and foremost, given the direct and indirect impacts that the coal export proposals would have on the Yakama People and our Treaty-reserved rights and resources, Yakama Nation is fully opposed to all coal export proposals, including the Millennium Bulk Terminal project at the Port of Longview. As such, Yakama Nation continues to ask all permitting agencies, including the U.S. Army Corps of Engineers and other state and local authorities to deny any and all permits related to these proposals. To be clear, Yakama Nation will not negotiate nor agree to so-called mitigation for any violations of its Treaty-reserved rights.”

The FEIS should include deep analysis of Longview residents and those living along transportation corridors utilizing the full power and resources of U.S. EPA’s Environmental Justice screening tool (EJSCREEN). This tool combines demographic variables identifying potential susceptible or vulnerable populations with separate environmental indicators to derive separate EJ Indices that reflect whether those populations are facing excess environmental risk for an environmental indicator. The results for coal train and vessel routes through Washington en route to MBT clearly show multiple municipalities and disproportionately impacted communities where disparate risk should be further evaluated as part of the FEIS. (See <https://www.epa.gov/ejscreen>.)

The DEIS fails to recognize the negative, cumulative health impacts for vulnerable populations, including pediatric asthmatics, those with COPD, heart disease, diabetes, women over 50, exposed workers, the elderly and those living in poverty. The FEIS should include information provided through the May 15, 2013, Community Health Needs Assessment¹⁹ to fully appreciate the overall health status of the community and the substantial numbers of residents living with chronic disease. (See Tables 4,5,6,7) The rate of premature death in the county is high relative to the rest of the state. The health of county residents is further threatened by degradation of air quality from MBT’s dirty project.

The FEIS and HIA must also incorporate a thorough and accurate analysis of the health and safety risks to workers at the proposed MBT terminal. In 1969, the Coal Mine Health and Safety Act was created to protect the health of miners. Despite apparent advances, in 2012 researchers at the National Institute for Occupational Safety and Health discovered an almost 10-fold increase in coal workers’ pneumoconiosis—or black lung disease.²⁰ That finding led to the CDC looking closely at surface mine

¹⁹ <https://www.peacehealth.org/sites/default/files/Documents/CHNA-PHSJ-2013.pdf>

²⁰ David J. Blackley, Cara N. Halldin, and A. Scott Laney "Resurgence of a Debilitating and Entirely Preventable Respiratory Disease among Working Coal Miners", *American Journal of Respiratory and Critical Care Medicine*, Vol. 190, No. 6 (2014), pp. 708-709. doi: [10.1164/rccm.201407-1286LE](https://doi.org/10.1164/rccm.201407-1286LE)

workers—an understudied group. Here too, evidence was found of serious, occupation-related respiratory illness in many of the workers.²¹

Coal work remains an occupation with great health risks. These risks are exacerbated by the fact that workers who will be exposed to the most coal dust will also be the people who are regularly exposed to highest levels of diesel particulates and other air toxins. In the case of particulate matter, the health risks correlate with exposure and there is no level of exposure at which adverse health risks are not seen.

This DEIS lacks sufficient data to convince us that this would be safe work, when there is substantial evidence to the contrary. It most certainly does not follow the precautionary principle—by first insuring the protection of workers’ health.

A comprehensive Health Impact Assessment should be produced and made available for public comment. Given the undeniable threat to worker health, the “No Action Alternative” should be selected. We need to move beyond these sorts of dirty and dangerous projects—and focus instead on creating living wage jobs that are healthy for workers and the greater community.

III. THE DEIS FAILS TO RECOMMEND ADEQUATE MITIGATION MEASURES

The DEIS correctly states that there are significant and adverse impacts of this project that cannot be mitigated.

Mitigation means stopping the exposure before it happens by using methods to neutralize that exposure. Where are the requirements to enclose coal piles at the terminal? Where are the requirements to enclose or capture coal dust rising from open conveyor belts? The DEIS suggests mitigation measures that are inadequate and largely depend on measurements of contamination after exposures to hazardous materials, physical agents, or harmful events have occurred. Significant health impacts will already have been experienced. Short term and intermittent exposures are not given appropriate consideration. This is exemplified by the discussions in the DEIS of potential noise exposures and coal dust exposures. In these cases, monitoring has been substituted for mitigation.

Monitoring complaints will serve to alert authorities to a problem only after the fact. How many children in the affected communities will have their sleep disturbed by unexpected and uncontrollable noise exposures, such as noise from train horns? What will be the impacts on learning and development? The DEIS states that 229 residences

²¹ Pneumoconiosis and Advanced Occupational Lung Disease Among Surface Coal Miners — 16 States, 2010–2011. (2012, June 15). Retrieved May 23, 2016, from <https://www.cdc.gov/mmwr/preview/mmwrhtml/mm6123a2.htm>

would experience moderate noise impacts and 60 residences would experience severe noise impacts from proposed action-related trains. How will this harmful-to-health noise exposure to these families be mitigated? The DEIS suggests monitoring complaints from the community and/or constructing FRA approved “quiet zones”.

Thus, unavoidable and significant adverse impacts on the health of the community will result from this project. If a person is exposed to and inhales a blast of coal dust and/or DPM for five minutes, that person has already experienced health impacts. It is too late for mitigation.

Added trains for this project will contribute to many rail segments exceeding capacity and increase the risk of train accidents. “While it is likely that rail companies would make investments or changes to accommodate the growth in rail traffic, it is unknown what these actions would be or when they would be permitted or built.” What is suggested as mitigation for this increased risk? “Before each stage of operations that would increase the number of trains, Millennium would coordinate with the rail companies. A report will be prepared to document the coordination.” (DEIS, Rail Transportation and Rail Safety Fact Sheet) Such a “report” cannot possibly mitigate the impacts of a train accident.

Although the DEIS describes risks to communities, it minimizes them and does not examine or predict with data the potential health risks resulting from its proposed actions. The community may gain relatively few, unsafe and unhealthy jobs at the expense of increased infrastructure and health care costs and shortened life expectancies.

IV. THE DEIS FAILS TO INCLUDE A HEALTH IMPACT ASSESSMENT (HIA)

The DEIS examines air quality, water quality, traffic delays, noise and light pollution and confirms some serious health impacts but it is also incomplete. The Draft EIS fails to incorporate a Health Impact Assessment (HIA).

Before and during scoping, many organizations and municipalities called for an HIA for this, the largest coal export project in the U.S. They include but are not limited to the City of Portland, the City of Mosier, the City of Milwaukee, the City of Beaverton, the City of Eugene, the Oregon Environmental Justice Task Force, and The Yakama Nation. (All references here are included in EXHIBITS.)

Because negative health impacts from climate change will be a result of the MBT project, we request that the FEIS include a Health Impact Assessment. Because exposure to toxic air and water pollution is a direct impact of MBT, we request that the FEIS include a Health Impact Assessment. Because increased frequency of very long

trains and derailments along the many train corridors will be a direct result of the MBT, we request that the FEIS include a Health Impact Assessment.

It is incumbent upon the decision makers in this process to apply the *best available science* in determining the health impacts of the MBT. The Washington Department of Ecology summarized the current state of the science in a white paper entitled “Concerns about the Adverse Health Effects of Diesel Engine Emissions” (2008). This paper recommends the adoption of the risk assessment tools developed by the California EPA’s Office of Environmental Health and Hazard Assessment for carcinogenic and non-carcinogenic risk based DPM concentration levels. **We recommend the use of these risk assessment tools in investigating the potential impact of the MBT.** (See health risk assessment guidance from California’s Office of Environmental Health and Hazard Assessment at <http://www.oehha.ca.gov/pdf/HRSguide2001.pdf>)

A study of air toxins in the Tacoma and Seattle area was completed using these risk assessment tools (October 2010). Among many other findings, this study demonstrated that DPM contributed *over 70%* of the potential airborne pollutant cancer risk in the Seattle area. (See References: DPM.)

This study did not, however, quantify the risks spatially, relative to a specific source such as the railway corridor or the terminal operation. The highest exposure risks of DPM from the MBT will occur to populations in close proximity to the tracks, the terminal, and shipping lanes. **Thus, we recommend that the near source health effects be quantified spatially all along all transportation corridors, not just for the terminal site. This will necessarily include all railway and vessel corridors.**

Modeling should use either the California Office of Environmental Health Hazard Assessment tools and modeling protocol or the EPA Air Toxics Community Multiscale Air Quality Model to predict multiple pollutant effects on the affected communities. The modeling protocol should be approved by the Washington Department of Ecology and the EPA. The modeling should be performed by independent consultants familiar with the models and with interpreting the results of the models.

If any mitigation measures including, but not limited to, construction of a terminal building to enclose piles of coal, covered rail cars at the project site, other pollution control devices, ultra-low sulfur fuel specifications, and late model diesel locomotives are used in emissions estimates and models, those assumptions should be listed in the FEIS as **required** mitigation.

The Columbia Basin and Portland/Vancouver metropolitan areas experience temperature inversions, which can dramatically increase pollutant concentrations. Thus, the analysis must include not only effects of pollutants near the transportation corridor under normal weather conditions, but also under temperature inversion conditions.

Because this would be the largest coal export facility in the US, it is imperative that a HIA is produced and that the HIA is a state-of-the art assessment that takes a comprehensive approach to health and health care costs, while incorporating the values of equity, environmental justice, democracy, sustainable development, and ethical use of evidence. Please utilize the full resources available on EPA's EJ Screen.

Please answer public health and safety questions submitted during the scoping process, including those listed in Exhibit: "OPSR Scoping Comments for MBT #1."

The HIA is a very important tool for decision makers and must be made available so the public can review and comment on it. Because this action will not be accomplished during the DEIS comment period, the public must be provided the opportunity to comment on a draft HIA before a Final HIA is produced.

Thank you for consideration of these comments,

Sincerely,

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References

Additional information listed in "Whatcom Docs Position Statement" and appendices on coaltrainfacts.org.

I. A. Climate Change References

- ⁵ Ashley, S., & Ashley, W. (2008). Flood fatalities in the United States. *Journal of Applied Meteorology and Climatology*, 47: 805-818. Retrieved online: <http://journals.ametsoc.org/doi/pdf/10.1175/2007JAMC1611.1>
- ³⁵ Garland-Forshee, R. & Gedman, T. (2013). *The Burden of Asthma in Oregon*. Oregon Health Authority, Public Health Division. Retrieved online: http://public.health.oregon.gov/DiseasesConditions/ChronicDisease/Asthma/Documents/burden/OR_Asthma_2013.pdf
- ³⁸ Gent, J, Triche, E., Holford, T., Belanger, K., Bracken, M., Beckett, W., & Leaderer, B. (2003). Association of low-level ozone and fine particles with respiratory symptoms in children with asthma. *Journal of the American Medical Association*, 290(14): 1859-1867. Retrieved online: <http://jama.jamanetwork.com/>
- ⁴⁴ Haggerty, B., York, E., Early-Alberts, J., & Cude, C. (2014). *Oregon Climate and Health Profile Report*. Oregon Health Authority. Retrieved online: <http://public.health.oregon.gov/HealthyEnvironments/climatechange/Documents/oregon-climate-and-health-profile-report.pdf>
- ⁵² Intergovernmental Panel on Climate Change (IPCC) (2012). *Managing the Risks of Extreme Events and Disasters to Advance Climate Change Adaptation. A Special Report of Working Groups I and II of the Intergovernmental Panel on Climate Change*. Field, C., Barros, V., Stocker, T., Qin, D., Dokken, K., et al. (Eds). Cambridge University Press, 582
- ⁵⁴ Jackson, E., Yost, M., Karr, C., Fitzpatrick, C., Lamb, B., Chung, S., Chen, J., Avise, J., Rosenblatt, R., & Fenske, R. (2010). Public health impacts of climate change in Washington State: projected mortality due to heat events and air pollution. *Climate Change*, 102: 159-186. doi: 10.1007/s10584-010-9852-3 pp. Retrieved online: http://ipcc-wg2.gov/SREX/images/uploads/SREX-All_FINAL.pdf
- ⁵⁷ Knowlton, K., Rotkin-Ellman, M., Geballe, L., Max, W., & Solomon, G. (2011). Six climate change-related events in the United States accounted for about \$14 billion in lost lives and health costs. *Health Affairs*, 30: 2167-2176. doi:10.1377/hlthaff.2011.0229
- ⁶⁴ Luber, G., Knowlton, K., Balbus, J., Frumkin, H., Hayden, M., Hess, J., McGeehin, M., Sheats, N., et al. (2014). Chapter 9: Human Health. Climate Change Impacts in the United States. *The Third National Climate Assessment*. doi:10.7930/J0PN93H5
- ⁶⁷ McConnell, R., Berhane, K, Gilliland, F., Islam, T., & Gauderman, W. (2002). Asthma in exercising children exposed to ozone: a cohort study. *Lancet*, 359: 386-391. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/11844508>
- ⁶⁸ McCoy, D., & Hoskins, B. (2014). The science of anthropogenic climate change:

what every doctor should know. *British Medical Journal*, 349: g5178. doi: 10.1136/bmj.g5178

⁷² National Oceanic and Atmospheric Administration (NOAA), National Weather Service (2012). *Weather Fatalities*. Retrieved online: www.nws.noaa.gov/om/hazstats.shtml

¹¹⁶ WA State Department of Health (WA DOH) (2013). *The Burden of Asthma in Washington State*. Retrieved online: <http://www.doh.wa.gov/Portals/1/Documents/Pubs/345-240-AsthmaBurdenRept13.pdf>

¹²⁰ Wingspread Conference on the Precautionary Principle (January 26, 1998). *Science and Environmental Health Network*. Retrieved online: <http://www.sehn.org/wing.html>

I. B. DPM References

Ammann, H. and M. Kadlec. 2008. Dept. of Ecology Air Quality Program: Concerns about adverse health effects of diesel engine emissions white paper. Publication 08-02-032.

Bhatia R, Lopipero P, Smith AH. 1998. Diesel exhaust exposure and lung cancer. *Epidemiology* 9(1): 84-91.

Boffetta P, Dosemeci M, Gridley G, Bath H, Moradi T, Silverman D. 2001. Occupational exposure to diesel engine emission and risk of cancer in Swedish men and women. *Cancer Causes Control* 12(4): 365-374.

Brandt, SJ et al. 2012. Costs of childhood asthma due to traffic-related pollution in two California communities. *Eur Respir J* 40:363-370.

Brook, R.D. and S. Rajagopalan. 2012. Can what you breathe trigger a stroke within hours? *Arch Intern Med* 172(3): 235-236.

Brook, RD et al. 2010. Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. *Circulation* 121:2331-2378.

California Air Resources Board. Findings of the Scientific Review Panel on the Report on Diesel Exhaust (as adopted at the Panel's April 22, 1998 meeting) <http://www.arb.ca.gov/toxics/dieseltac/de-fnds.htm>

California Environmental Protection Agency. Part B: Health Risk Assessment for Diesel Exhaust. For the Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant. California Environmental Protection Agency, Office of Environmental

Health Hazard Assessment, Air Toxicology and Epidemiology Section, Oakland. May 1998

Cosselman K, Kaufman JA. 2012. Blood Pressure Response to Controlled Diesel Exhaust Exposure in Humans. *Hypertension*. March 19 2012.

Cohen AJ and Nikula K. 1999. The Health Effects of Diesel Exhaust: Laboratory and Epidemiologic Studies. Chap 32 in *Air Pollution and Health*. Ed. ST Holgate, JM Samet, HS Koren, and RL Maynard. Academic Press, London.

Dockery, D. et al. 1993. An association between air pollution and mortality in six U.S. cities. *New Engl J Med* 329(24): 1753-1759.

Gauderman, W.J. et al. 2007. Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study. *The Lancet* 369:571-.

Gauderman, W. et al. 2004. The effect of air pollution on lung development from 10 to 18 years of age. *New Engl J Med* 351(11): 1057-1067

Gaudermann, W.J. et al. 2005. Childhood asthma and exposure to traffic and nitrogen dioxide. *Epidemiology* 16(6): 1-.

Gaudermann, W.J. et al. 2002. Association between air pollution and lung function growth in Southern California children. *Am J. Respir Care Med* 166:76- 84.

Ghio, A. J et al. 2000. Concentrated ambient air particles induce mild pulmonary inflammation in healthy human volunteers. *Am J Respir Crit Care Med* 162: 981- 2000.

Guo J, Kauppinen T, Kyyronen P, Heikkila P, Lindblohm ML, Pukkala E. 2004. Risk of esophageal, ovarian, testicular, kidney and bladder cancers and leukemia among Finnish workers exposed to diesel or gasoline exhaust. *Int J Cancer* 111(2): 286-292.

Hong, Y-C. et al. 2002. Effects of air pollutants on acute stroke mortality. *Environ Health Perspec.* 110 (2): 187-.

D.A. Jaffe, G. Hof, S. Malashanka, J. Putz, J. Thayer, J.L. Fry, B. Ayres and J.R. Pierce. Diesel particulate matter emission factors and air quality implications from in-service rail in Washington State, USA. *Atmospheric Pollution Research* 5, 344–351, doi: 10.5094/APR.2014.040.

Krishnan, R. M. *et al.* Vascular Responses to Long- and Short-Term Exposure to Fine Particulate Matter: The MESA Air (Multi-Ethnic Study of Atherosclerosis and Air Pollution). *Journal of the American College of Cardiology*, doi:10.1016/j.jacc.2012.08.973 (2012).

Lim, S. S. *et al.* A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a

systematic analysis for the Global Burden of Disease Study 2010. *Lancet* 380, 2224-2260, doi:10.1016/S0140-6736(12)61766-8 (2013).

Lin, M. et al. 2002. The influence of ambient coarse particulate matter on asthma hospitalization in children: case-crossover and times-series analyses. *Environ Health Perspect.* 110(6):575-.

Lin, S. et al. 2002. Childhood asthma hospitalization and residential exposure to state route traffic. *Environ Res Sect A* 88:73-81.

McConnell, R. et al. 2010. Childhood incident asthma and traffic-related air pollution at home and school. *Environ Health Perspect.* 118(7): 1021-.

Mills, N. L. et al. Diesel exhaust inhalation causes vascular dysfunction and impaired endogenous fibrinolysis. *Circulation* 112, 3930-3936 (2005).

Mills, N.L. et al. 2007. Ischemic and thrombotic effects of dilute diesel-exhaust inhalation in men with coronary heart disease. *NEJM* (357(11): 1075-.

Miller, K. A. et al. Long-term exposure to air pollution and incidence of cardiovascular events in women. *NEJM* 356, 447-458 (2007).

Mittleman, M. A. 2007. Air pollution, exercise, and cardiovascular risk. *NEJM* 357(11): 1147-.

Mustafic H. et al. 2012. Main air pollutants and myocardial infarction: a systematic review and meta-analysis. *JAMA* 307(7):713-.

Norris, G. et al. 1999. An association between fine particles and asthma emergency department visits for children in Seattle. *Environ Health Perspect.* 107:489-493.

Ostro. B. et al. 2009. Long-term exposure to constituents of fine particulate air pollution and mortality: results from the California Teachers Study. *Environ Health Perspect* 118(3):363-369.

Ostro, B. et al. The effects of fine particle components on respiratory hospital admissions in children. *Environ. Health Perspect.* 117(3):475-480.

Peters, A., Dockery, D. W., Muller, J. E. & Mittleman, M. A. Increased particulate air pollution and the triggering of myocardial infarction. *Circulation* 103, 2810- 2815 (2001).

Pope C.A. et al. 2004. Air pollution and health- good news and bad. *NEJM* 351(11): 1132-.

Pope, C. A. III et al. 2002 Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA* 287: 1132-1141.

Pope, C. A. et al. Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. *Circulation* **109**, 71-77 (2004).

Pope, C. A. et al. 2009. Fine-particulate matter air pollution and life expectancy in the United States. *New Engl J Med* 360(4): 376-386.

Pope, C. A. III et al. 1995. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. *Am J Respir Crit Care Med* 151: 669-674.

Slaughter, J. C. et al. 2003. Effects of ambient air pollution on symptom severity and medication use in children with asthma. *Ann Allergy Asthma & Immunol* 91:346-353.

Spira-Cohen, A. et al. 2011. Personal exposures to traffic-related air pollution and acute respiratory health among Bronx schoolchildren with asthma. *Environ Health Perspect.* 119(4): 559-.

Studer, CE. 2011. Health risk study for the Burlington Northern / Santa Fe Railroad Spokane Railyard. Spokane Regional Clean Air Agency, www.spokanecleanair.org

Thaller, E. et al. 2008. Moderate increases in ambient PM_{2.5} and ozone are associated with lung function decreases in beach lifeguards. *J Occup Environ Med* 50:202-211.

Tolbert, P.E. et al. 2000. Air quality and pediatric emergency room visits for asthma in Atlanta, Georgia. *Am. J. Epidemiol.* 151(8):798-810.

Törnqvist, H. et al. Persistent Endothelial Dysfunction in Humans after Diesel Exhaust Inhalation. *American Journal of Respiratory and Critical Care Medicine* **176**, 395-400 (2007).

Tsai, S-S. et al. 2003. Evidence for an association between air pollution and daily stroke admissions in Kaohsiung, Taiwan. *Stroke* 34:2612-2616.

Van Hee, V. C. et al. Exposure to traffic and left ventricular mass and function: the Multi-Ethnic Study of Atherosclerosis. *American journal of respiratory and critical care medicine* **179**, 827-834 (2009).

Van Hee, V. C. et al. Association of long-term air pollution with ventricular conduction and repolarization abnormalities. *Epidemiology* **22**, 773-780 (2011).

Wellenius, G. A. et al. 2012. Ambient air pollution and the risk of acute ischemic stroke. *Arch Intern Med* 172(3): 229-234.

Wellenius, G.A. et al. 2005. Air pollution and hospital admissions for ischemic and hemorrhagic stroke among Medicare beneficiaries. *Stroke* 36:12:2549-53. <http://stroke.ahajournals.org/content/36/12/2549>.

Weuve, J. et al. 2012. Exposure to air pollution and cognitive decline in older women. *Arch Intern Med* 172(3): 219-227.

US Department of Health and Human Services. 2008. Health Consultation: Summary of Results of the Duwamish Valley Regional Modeling and Health Risk Assessment, Seattle, Washington. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Division of Health Assessment and Consultation, Atlanta, Georgia. July 14, 2008

US Environmental Protection Agency. Health Assessment Document for Diesel Engine Exhaust. U.S. Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment, Washington, DC. EPA/600/8-90/057F, 2002.

ACOG—The American College of Obstetricians and Gynecologists, Committee Opinion, No. 575, October 2013 - http://www.acog.org/About_ACOG/News_Room/News_Releases/2013/Environmental_Chemicals_Harm_Reproductive_Health

AAP—American Academy of Pediatrics. Committee on Environmental Health. Ambient Air Pollution: Health Hazards to Children, , *Pediatrics*, 2004: 114, 1699-1707, Reaffirmed April 2009 (<http://pediatrics.aappublications.org/content/125/2/e444.extract>)

Avogbe P, Ayi-Fanou L, Autrup H, et al. Ultrafine particulate matter and high-level benzene urban air pollution in relation to oxidative DNA damage. *Carcinogenesis* 2004; 26(3):613-620

Becerra TA, Wilhelm M, Olsen J, et al. Ambient air pollution and autism in Los Angeles County, California. *Environ Health Perspect* 2013; 121(3):380-386

Beeson WL, Abbey DE, Knutsen SF. Long-term concentrations of ambient air pollutants and incident lung cancer in California adults: results from the Adventist Health Study on Smog. *Environ Health Perspect* 1998; 106(12):813-23

Bellavia A, Urch B, Speck M, et al. DNA Hypomethylation, ambient particulate matter, and increased blood pressure: findings from controlled human exposure experiments. *J Am Heart Assoc.* 2013; 2:e000212 doi: 10.1161/ JAHA 113.000212

Brook RD, Rajagopalan S, Pope CA, III, et al. Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association. *Circulation* 2010;121(21):2331-2378

- Calderón-Garcidueñas L, Engle R, Mora-Tiscareño A, et al. Exposure to severe urban air pollution influences cognitive outcomes, brain volume and systematic inflammation in clinically healthy children. *Brain and Cognition* 2011; 77(3):345-355
- Carlsten C, Dybuncio A, Becker A, et al. Traffic-related air pollution and incident asthma in a high-risk birth cohort. *Occup Environ Med* 2011; 68:291-295
- Chen R, Zhang Y, Yang C, et al. Acute effect of ambient air pollution and health effects study. *Stroke* 2013; 44:954-960
- Chiu Y-HM, Bellinger D, Coull BA, et al. Associations between traffic-related black carbon exposure and attention in a prospective birth cohort of urban children. *Environ Health Perspect* 2013; 121(7):859-864
- Crouse D, Goldberg M, Ross N, et al. Postmenopausal breast cancer is associated with exposure to traffic –related air pollution in Montreal, Canada: a case-controlled study. *Environmental Health Perspectives* 2010; 118(11): 1578-1583
- Delamater P, Finley A, Banerjee S. An analysis of asthma hospitalizations, air pollution, and weather conditions in Los Angeles County, California. *Science of the Total Environment* 2012; 425:110-118
- Demetriou C, Raaschou-Nielsen O, Loft S, et al. Biomarkers of ambient air pollution and lung cancer: a systematic review. *Occup Environ Med* 2012; 69:619-627
- Dockery DW, Pope CA, III, Xu X, et al. An association between air pollution and mortality in six US cities. *N Engl J Med* 1993; 329(24):1753-1759
- Dominici F, Peng RD, Bell ML et al. Fine Particulate air pollution and hospital admission for cardiovascular and respiratory diseases. *JAMA* 2006; 295(10):1127-1134
- EPA 1—<http://www.epa.gov/region1/eco/airtox/diesel.html>
- EPA 2— http://www.epa.gov/teach/chem_summ/BENZ_summary.pdf
- Gauderman WJ, Avol E, Gilliland F, et al. The effect of air pollution on lung development from 10 to 18 years of age. *N Engl J Med* 2004;351(11):1057-1067
- Gowers A, Cullinan P, Ayres J, et al. Does outdoor air pollution induce new cases of asthma? Biological plausibility and evidence; a review. *Respirology* 2012; 17:887-898
- Guardian: <http://www.theguardian.com/environment/chinas-choice/2013/oct/24/china-airpocalypse-harbin-air-pollution-cancer>

HEI Panel on Health effects of traffic related air pollution, Traffic related Air Pollution A Critical review of the literature on emissions, exposure, and health effects. Boston: Health Effects Institute; 2010.

Hertz-Picciotto I, Park H-Y, Dostal M, et al. Prenatal exposures to persistent and non-persistent organic compounds and effects on immune system development. *Basic and Clinical Pharmacology and Toxicology* 2007;102:146-154

Jaffe, Daniel A., et al. Diesel particulate matter emission factors and air quality implications from in-service rail in Washington State, USA, *Atmospheric Pollution Research*. Doi: 10.5094/Apr. 2014.040

Jedrychowski WA, Perera FP, Spengler CD, et al. Prenatal exposure to polycyclic aromatic hydrocarbons and cognitive dysfunction in children. *Environ Sci Pollut Res Int*, 2014, Epub Sep 26 (ahead of publication)

Kalkbrenner, et al. Particulate matter exposure, prenatal and postnatal windows of susceptibility and Autism Spectrum Disorder. *Epidemiology* 2015; 26:30-42

Krishnan RM, Adar SD, Szpiro AA, et al. Vascular responses to long- and short-term exposure to fine particulate matter, MESA Air (Multi-ethnic study of atherosclerosis and air pollution. *J Am College of Cardiology* 2012; 60(21):2158-2166

Li N, Sioutas C, Cho A, et al. Ultrafine particulate pollutants induce oxidative stress and mitochondrial damage. *Environ Health Perspec* 2003; 111(4): 455-460.

Lue S-H, Wellenius GA, Wilker EH, et al. Residential proximity to major roadways and renal function. *J Epidemiol Community Health* 2013; 67:629-634

Miller KA, Siscovick DS, Sheppard L, et al. Long term exposure to air pollution and incidence of cardiovascular events in women. *N Engl J Med* 2007;356(5):447-5

Mustafic H, Jabre P, Caussin C, et al. Main air pollutants and myocardial infarction, a systematic review and meta-analysis. *JAMA* 2012; 307(7):713-721

Newman NC, Ryan P LeMasters G, et al. Traffic-related air pollution exposure in the first year of life and behavioral scores at 7 years of age. *Environ Health Perspect* 2013; 121(6):731-736

Oregon Live,

http://www.oregonlive.com/environment/index.ssf/2014/04/oregon_oil_train_shipments_inc.html

Pandya RJ, Solomon G, Kinner A, Balmes JR. Diesel exhaust and asthma: hypotheses and molecular mechanisms of action. *Environmental Health Perspectives*. 2002;110(suppl 1):103-112

Perera FP, Li T, Zhou Z, et al. Benefits of reducing prenatal exposure to coal-burning pollutants to children's neurodevelopment in China. *Environmental Health Perspectives* 2008; 116(10):1396-1400

Perera FP, Li Z, Whyatt R, et al. Prenatal airborne polycyclic aromatic hydrocarbon exposure and child IQ at age 5 years. *Pediatrics* 2009; 124(2):e195-202

Perera FP, Wang S, Rauh V, et al. Prenatal exposure to air pollution, maternal psychological distress, and child behavior. *Pediatrics* 2013; 132(5):e1284-1294

Perera FP, Chang HW, Tang D, et al. Early life exposure to polycyclic aromatic hydrocarbons and ADHD behavior problems. *PLoS One*. 2014 Nov 5;9(11); PubMed Central PMCID: PMC4221082

Peters A. Ambient particulate matter and the risk for cardiovascular disease. *Progress in Cardiovascular Diseases*. 2011; 53: 327-333

Peters A, Liu E, Verrier RL et al, Air pollution and incidence of cardiac arrhythmia. *Epidemiology* 2000; 11(1):11-17

Peters A., Dockery DW, Muller JE, Mittleman MA. Increased particulate air pollution and the triggering of myocardial infarction. *Circulation* 2001; 103(23):2810-2815

Peterson, BS, Rauh, VA, Perera, F, et al. Effects of prenatal exposure to air pollutants (Polycyclic aromatic hydrocarbons) on the development of brain white matter, cognition, and behavior in later childhood, *JAMA Psychiatry* 2015; doi:10.1001/jamapsychiatry.2015.57

Pieters N, Plusuin M, Cox B, et al. An epidemiological appraisal of the association between heart rate variability and particulate air pollution: a meta-analysis. *Heart* 2012; 98:1127-1135

Pope CA, III, Burnett RT, Thun MJ et al. Lung cancer, cardiopulmonary mortality, and long term exposure to fine particulate air pollution. *JAMA* 2002; 287(9):1132-1141

Qian Y, Zhu M, Cai B, et al. Epidemiological evidence on association between ambient air pollution and stroke mortality. *J Epidemiol Community Health* 2013; 67:635-640

Raz R, Roberts AL, Lyall K, et al. Autism Spectrum Disorder and particulate matter air pollution before, during, and after pregnancy: a nested case-control analysis within the nurses' health study II cohort, *Environ Health Perspect* 2015; 123(3):265-270

Roberts AL, Lyall K, Hart, JE, et al. Perinatal air pollutant exposures and Autism Spectrum Disorder in the children of nurses' health study II participants. *Environ Health Perspect* 2013; 121(8): 978-984

Shaw ASV, Langrish JP, Nair H, et al. Global associations of air pollution and heart failure: a systematic review and meta-analysis. *The Lancet* 2013; 832:1039-1048

Tang D, Li TY, Chow JC, et al. Air pollution effects on fetal and child development: a cohort comparison in China. *Environ Pollut.* 2014; Feb. 185:90-6.
Doi:10.1016/j.envpol.2013.10.019 Epub2013 Nov. PubMed PMID: 24239591

Trasande L, Thurston GD. The role of air pollution in asthma and other pediatric morbidities. *J Allergy Clin Immunology* 2005; 115(4):689-699

Vrijheid M, Martinez D, Manzanares, S, et al. Ambient air pollution and risk of congenital anomalies: a systematic review and meta-analysis. *Environmental Health Perspectives* 2011;119(5) 598-606

Volk HE, Lurmann F, Penfold B, et al. Traffic-related air pollution, particulate matter, and autism. *JAMA Psychiatry* 2013; 70(1):71-77

Volk HE, Hertz-Picciotto I, Delwiche L, et al. Residential proximity to freeway and autism in the CHARGE study. *Environ Health Perspect* 2011; 119(6):873-877

Wei Y, Davis J, Bina WF. Ambient air pollution is associated with the increased incidence of breast cancer in US. *Int J Environ Health Res* 2012; 22(1):12-21

Wellenius GA, Schwartz J, Mittleman MA. Air Pollution and hospital admissions for ischemic and hemorrhagic stroke among Medicare beneficiaries. *Stroke* 2005; 36(12):2549-2553

Wellenius GA, Burger, MR, Coull BA, et al. Ambient air pollution and the risk of acute ischemic stroke. *Arch Int Med* 2012; 172(3): 229-234

Wilker EH, Preis SR, Beiser AS, et al. Long-term exposure to fine particulate matter, residential proximity to major roads and measures of brain structure. *Stroke* 2015, May
DOI:10.1161/STROKEAHA.114.008348

WHO 1—World Health Organization

<http://www.who.int/mediacentre/factsheets/fs313/en/#>

WHO 2— World Health Organization/ American Cancer Society

<http://www.cancer.org/cancer/news/world-health-organization-outdoor-air-pollution-causes-cancer>

WHO 3— World Health Organization. Health aspects of air pollution with particulate matter, ozone, and nitrogen dioxide. Bonn: World Health Organization; 2003

WHO 4—World Health Organization. Outdoor Air Pollution. Global Health

Observatory. Available at
http://www.who.int/gho/phe/outdoor_air_pollution/en/index.html

I.C. Coal Dust References:

Bounds, W. and Johannesson, K. Arsenic Addition to Soils from Airborne Coal Dust Originating at a Major Coal Shipping Terminal. *Water, Air and Soil Pollution*; October, 2007, Vol. 185 Issue 1-4, p 195.

BNSF Railway. Coal Dust Frequently Asked Questions, 2011.

Cope D, Wituschek W, Poon D et al. 1994. Report on the emission and control of fugitive coal dust from coal trains. Regional Program Report 86 – 11. Environmental Protection Service, Pacific Region British Columbia Canada.

Gottlieb, B., Gilbert, S.G., and Evans, L.G. “Coal Ash: The Toxic Threat to our Health and Environment,” Physicians for Social Responsibility (PSR) and Earthjustice. Report is available: <http://www.psr.org/resources/coal-ash-the-toxic-threat-to-our-health-and-environment.html>. September 2010.

Hathaway GJ, Proctor NH, Hughes JP 1991. Proctor and Hughes’ chemical hazards of the workplace, 3 Edition. New York, NY: Van Nostrand Reinhold.

Hendryx M, Ahern MM, Nurkiewicz TR. Hospitalization patterns associated with Appalachian coal mining. *Journal of Toxicology and Environmental Health, Part A*. 2007;70(24):2064-2070

Hendryx M, Fedorko E, Anesetti-Rothermel A. A geographical information system-based analysis of cancer mortality and population exposure to coal mining activities in West Virginia, United States of America. *Geospatial Health* 2010;4(2):243-256

Hendryx M, Ahern MM. Relations between health indicators and residential proximity to coal mining in West Virginia. *American Journal of Public Health* 2008;98(4):669-671

Hendryx M. Mortality from heart, respiratory, and kidney disease in coal mining areas of Appalachia. *International Archives of Occupational and Environmental Health*. 2009;82(2):243-249

D.A. Jaffe, J. Putz, G. Hof, J. Hee, D.A. Lommers-Johnson, F. Gabela, J.L. Fry, B. Ayres, M. Kelp and M. Minsk. Diesel particulate matter and coal dust from trains in the Columbia River Gorge, Washington State, USA. *Atmospheric Pollution Research*, doi: 10.1016/j.apr.2015.04.004.

Karagianes MT, Palmer RF, Busch RH 1981. The effects of inhaled diesel emissions and coal dust in rats. *American Industrial Hygiene Journal*. Volume 42(5):382-391.

National Oceanic and Atmospheric Administration. 2011. Source of Mercury Emission into the Great Lakes.

Queensland Government Environmental Protection Agency Report. 2008. Environmental evaluation of fugitive coal dust emissions from coal trains Goonyella, Blackwater, and Moura coal rail systems, Queensland rail limited. Connell Hatch and Co. Final Report.

Simpson Weather Associates 1993. Norfolk southern rail emission study: consulting report prepared for Norfolk Southern Corporation. Charlottesville, VA.

United States Environmental Protection Agency/Federal Drug Administration, 2004. Consumption Advice: Joint Federal Advisory for Mercury in Fish, 2004.

Vincent JH, Jones AD, Johnston AL et al. 1987. Accumulation of inhaled mineral dust in the lungs and associated lymph nodes: implications for exposure and dose in occupational settings. *Annals of Occupational Hygiene* 31(3): 375-393.

Wade WA, Petsonk EL, et al. 2010. Severe occupational pneumoconiosis among West Virginia coal miners: 138 cases of progressive massive fibrosis compensated between 2000 – 2009. *Chest* 139(6); 1459-1463.

I.D. Noise Pollution References:

Aasvang, G. et al. A field study of road traffic and railway noise on polysomnographic sleep parameters. 2011. *J. Acoust. Soc. Am.* 129 (6).

Babisch W. Noise and Health. *Environ Health Perspect* 2005; 113: A14-15.

Berglund B, Lindvall T. (eds.) 1999 WHO Document on Guidelines for Community Noise: 39-94.

Brink M et al. 2011. An event-related analysis of awakening reactions due to nocturnal church bell noise. *Sci Total Environ.* 409(24): 5210-20.

Bronzaft AL, Dignan E, Bat-Chava Y, & Nadler NB. . Intrusive community noises yield more complaints. *Noise Rehabilitation Quarterly*, 25: 16-22,34

Carter NL. 1996. Transportation noise, sleep, and possible after-effects. *Environ Int.* 22: 105-116

Chang, K. et al. 2012. Road traffic noise: annoyance, sleep disturbance, and public health implications. *Am J Prev Med.*; 43(4): 353-60.

- Clark C. et al. 2012. Does traffic-related air pollution explain associations of aircraft and road traffic noise exposure on children's health and cognition? A secondary analysis of the United Kingdom sampled from the RANCH project. *Am. J. Epidemiol.* 176(4): 327-337.
- Cohen S, Evans GW, Krantz DS, Stokols D. 1980. Physiological, motivational and cognitive effects of aircraft noise on children: Moving from the laboratory to the field. *Am Psychol*; 35: 231-43.
- Evans GW. 2003. Ambient noise and cognitive process among primary schoolchildren. *Environment and Behavior*, 35(6) 725-735.
- Evans GW, Hygge S, Bullinger M. 1995. Chronic noise and psychological stress. *Psychol Sci.* 6: 333-8
- Evans GW, Lepore SJ. 1993. Non-auditory effects of noise on children: a critical review. *Children's Environments.* 10: 42-72.
- Evans GW, Maxwell L. 1997. Chronic noise exposure and reading deficits: The mediating effects of language acquisition. *Environ Behav.* 29: 638-56
- Fidell S, Barber DS, and Schultz TJ. 1991. Updating a dosage-effect relationship for the prevalence of annoyance due to general transportation noise. *J Acoust Soc Am.* 89: 221-233.
- Halonen, JI et al. 2012. Associations between nighttime traffic noise and sleep: the Finnish Public Sector Study. *Environ. Health Perspect.* 120(10): 1391-1396.
- Haines MM, Stansfeld SA, Brentnall S, Head J, Berry B, Jiggins M, Hygge S. 2001. The West London School Study: The effects of chronic aircraft noise exposure on child health. *Psychol Med.* 31: 1385-96.
- Haines MM, Stansfeld SA, Job RFS, Berglund B, Head J. 2001. Chronic aircraft noise exposure, stress responses, mental health and cognitive performance in school children. *Psychol Med.* 31: 265-77.
- Hall F, Birnie S, Taylor SM, and Palmer J. 1981. Direct comparison of community response to road traffic noise and to aircraft noise. *J Acoust Soc Am,* 70: 1690-1698.
- Hong J et al. 2010. The effects of long-term exposure to railway and road traffic noise on subjective sleep disturbance. *J Acoust Soc Am.* 128(5):2829-35.
- Hume, KI. 2011. Noise Pollution: A ubiquitous unrecognized disruptor of sleep? *Sleep*; 34(1): 7-8.

- Hygge S, Evans GW, Bullinger M. 2002. A prospective study of some effects of aircraft noise on cognitive performance in school children. *Psychol Sci*; 13: 469– 74.
- Ising H, Kruppa B. 2004. Health effects caused by noise: evidence from the literature from the past 25 years. *Noise Health*. 6: 5-13.
- Moudon AV. 2009. Real noise from the urban environment: how ambient community noise affects health and what can be done about it. *Am J Prev Med*. 37(2): 167-71.
- Ohrstrom E, Bjorkman M. 1998. Effects of noise-disturbed sleep: A laboratory study on habituation and subjective noise sensitivity. *J Sound Vibration*. 122: 277-290.
- Selander J, Milsson ME, Bluhm G, Rosenlund M, Lindqvist, M Nise G, Pershagen G. 2009. Long-term exposure to road traffic noise and myocardial infarction. *Epidemiology*. 20(2): 272-279.
- Sorensen M et al. 2012. Road traffic noise and incident myocardial infarction: a prospective cohort study. *PLoS ONE* ; 7(6): 1-7.
- Sorensen M et al. 2012. Long term exposure to road traffic noise and incident diabetes: a cohort study. *Environ Health Persp*. <http://dx.doi.org/10.1289/ehp.1205503>.
- Sørensen M, Hvidberg M, Andersen ZJ, Nordsborg RB, Lillelund KG, Jakobsen J, Tjønneland A, Overvad K, and Raaschou-Nielsen O. 2011. Road traffic noise and stroke: a prospective cohort study. *European Heart Journal*; 32(6): 737-744.
- Stansfeld SA, Berglund B, Clark C, et al. 1999. Aircraft and road traffic noise and children's cognition and health: a cross national study. *Lancet* 2005; 365: 1942-
- Stansfeld SA, Matheson MP. 2003. Noise pollution: non-auditory effects on health. *Brit Med Bull*. 68: 243-257.
- Suter AH. 1991. Noise and its effects. Administrative Conference of the United States.
- Goines L, Hagler L. 2007. Noise Pollution: A modern plague. *South Med J*. 100(3): 287-294.
- Willich SN, Wegscheider K, Stallmann M, et al. 2006. Noise burden and the risk of myocardial infarction. *Eur Heart J*. 27: 276-282.

I.E. Toxic Air Pollutants References

Arsenic

ATSDR 2007 Toxicological Profile for Arsenic.

<http://www.atsdr.gov/substances/toxsubstance.asp?toxid=3>

Bailey, Smith, Tokar, Graziano et al., 2016. Mechanisms underlying latent disease risk associated with early-life arsenic exposure: Current research trends and scientific gaps. *Environ Health Perspect* 124(2):170-175. DOI:10.1289/ehp.1409360.

Carlin, Naujokas, Bradham, Cowden, et al., 2015. Arsenic and Environmental Health: State of the Science and Future Research Opportunities. *Environ Health Perspect* . DOI:10.1289/ehp.1510209.

Chung, J-Y et al., 2014: Environmental Source of Arsenic Exposure. *J Prev Med Public Health* 47:253-257. <http://dx.doi.org/10.3961/jpmph.14.036>

Csavina et al., 2014. Size-Resolved Dust and Aerosol Contaminants Associated with Copper and Lead Smelting Emissions: Implications for Emissions Management and Human Health. *Sci Total Environ*. 493:750-756. doi:10.1016/j.scitotenv.2014.06.031.

ECHA (European Chemicals Agency) 2016. Substance Information: Arsenic.

Flora, SJ. 2011. Arsenic-induced oxidative stress and its reversibility. *Free Radic Biol Med* 51(2):257-281.

Goossens et al. 2015. Surface and Airborne Arsenic Concentrations in a Recreational Site near Las Vegas, Nevada, USA. *PLoS ONE* 10(4):e0124271. doi:10.1371/journal.pone.0124271

Martin, R, Dowling, K, Pearce, D, Sillitoe, J, Florentine, S. 2014. Health effects associated with inhalation of airborne arsenic arising from mining operations. *Geosciences* 4:128-175; doi:10.3390/geosciences4030128

McDermott et al. 2014: Are different soil metals near the homes of pregnant women associated with mild and severe intellectual disability in children? *Dev Med Child Neurol* 56(9):888-97. doi:10.1111/dmcn.12442. Epub 2014 Apr 19.

Naujokas, Anderson, Ahsan, Aposhian et al. 2013. The Broad Scope of Health effects from chronic arsenic exposure: Update on a worldwide public health problem. *Environ Health Perspect* 121:295-302; DOI:10.1289/ehp.1205875.

Nygerma L. Dangleben, Christine F. Skibola and Martyn T. Smith. 2013. Arsenic immunotoxicity: a review. *Environmental Health* 12:73. <http://www.ehjournal.net/content/12/1/73>.

Potera, C. 2016. Arsenic and Latent Disease Risk. What's the mechanism of action? *Environ Health Perspect* 124:2:A36. Feb 2016 <http://dx.doi.org/10.1289/ehp.124-A36>.

USEPA <https://www.epa.gov/dwreginfo/chemical-contaminant-rules>.

WHO <http://www.who.int/mediacentre/factsheets/fs372/en/>

Cadmium

Åkesson A, Barregard L, Bergdahl IA, Nordberg GF, Nordberg M, Skerfving S. 2014. Non-renal effects and the risk assessment of environmental cadmium exposure. *Environ Health Perspect.* 122(5): 431-8. <<http://www.ncbi.nlm.nih.gov/pubmed/24569905>>

Choong G, Liu Y, Templeton DM. 2014. Interplay of calcium and cadmium in mediating cadmium toxicity. *Chem Biol Interact.* 211:54-65. <<http://www.ncbi.nlm.nih.gov/pubmed/24463198>>

Ciesielski T, Weuve J, Bellinger DC, Schwartz J, Lanphear B, Wright RO. 2012. Cadmium Exposure and Neurodevelopmental Outcomes in U.S. Children. *Environ Health Perspect.* 1104152. <<http://ehp.niehs.nih.gov/1104152/>>

Dematteo R, Keith, MM, Brophy, JT, Wordsworth, A et al. 2013. Chemical Exposures of Women Workers in the Plastics Industry with Particular Reference to Breast Cancer and Reproductive Hazards, *New Solutions*, February 2013; vol. 22, 4: pp. 427-448

Feki-Tounsi M, Hamza-Chaffai A. 2014. Cadmium as a possible cause of bladder cancer: a review of accumulated evidence. *Environ Sci Pollut Res Int.* 21(18):10561-73. <<http://www.ncbi.nlm.nih.gov/pubmed/24894749>>

García-Esquinas, E., Pollan, M, Tellez-Plaza, M, Francesconi, KA et al. 2014. Cadmium Exposure and Cancer Mortality in a Prospective Cohort: The Strong Heart Study. *Environ. Health Perspect* 122:4:363-370. <http://dx.doi.org/10.1289/ehp.1306587>

Inaba T et al 2005. Estimation of cumulative cadmium intake causing Itai-Itai disease. *Toxicology Letters* 159:2:192-201.

International Agency for Research on Cancer (IARC). 2012. Cadmium and Cadmium Compounds. <<http://monographs.iarc.fr/ENG/Monographs/vol100C/mono100C-8.pdf>>

Johnson MD, Kenney N, Stoica A, Hilakivi-Clarke L, et al. 2003. Cadmium mimics the in vivo effects of estrogen in the uterus and mammary gland. *Nat Med.* 2003 Aug;9(8):1081-4. Epub 2003 Jul 13.

Johnston JE et al. 2014. Maternal Cadmium levels during pregnancy associated with lower birth weight in infants in a North Carolina cohort. *PLoS One* 9(10): e109661. Doi:10.1371

Kasuya M, 2000. Recent epidemiological studies on itai-itai disease as a chronic cadmium poisoning in Japan. *Water Science and Technology* 42:7-8:147-154.

Kippler M, Tofail F, Hamadani JD, Gardner RM, Grantham-McGregor SM, Bottai M, Vahter M. 2012. Early-Life Cadmium Exposure and Child Development in 5-Year-Old Girls and Boys: A Cohort Study in Rural Bangladesh. 1104431. <http://ehp.niehs.nih.gov/1104431/>

Kobayashi J, 1971. Relation between the 'ITAI-ITAI' disease and the pollution of river water by cadmium from a mine. In: Fifth International Water Pollution Research Conference, San Francisco, July.

Rani A, Kumar A, Lal A, Pant M. 2014. Cellular mechanisms of cadmium-induced toxicity: a review. *Int J Environ Health Res.* (4):378-99. <<http://www.ncbi.nlm.nih.gov/pubmed/24117228>>

Ray PD, Yosim A, Fry RC. 2014. Incorporating epigenetic data into the risk assessment process for the toxic metals arsenic, cadmium, chromium, lead, and mercury: strategies and challenges. *Front Genet.* 5:201. <<http://www.ncbi.nlm.nih.gov/pubmed/25076963>>

Sioen I, Den Hond E, Nelen V, Van de Mierop E et al. 2013. Prenatal exposure to environmental contaminants and behavioural problems at age 7-8years. *Environ Int.* 2013 Sep;59:225-31. doi: 10.1016/j.envint.2013.06.014.

Wei Qu, Tokar, EJ, Kim, AJ, Bell, MW, and MP Waalkes. 2012. Chronic Cadmium Exposure in Vitro Causes Acquisition of Multiple Tumor Cell Characteristics in Human Pancreatic Epithelial Cells. *Environ Health Perspect* 120:1265-1271.

Zota AR, Needham BL, Blackburn EH, Lin J, et al. 2014. Associations of Cadmium and Lead Exposure With Leukocyte Telomere Length: Findings From National Health and Nutrition Examination Survey, 1999–2002. *Am. J. Epidemiol.* 181 (2): 127 - 136. <<http://aje.oxfordjournals.org/content/181/2/127>>

Lead

<https://web.stanford.edu/dept/EHS/prod/general/asbestoslead/leadfactsheet.html>

Mercury

Epstein PR et al. 2011. Full cost accounting for the life cycle of coal. *Annals of the New York Academy of Sciences*, vol 1219, pp 73-98.

I.F. Fire Risk References

1. de Place, Eric. "COAL TRAINS MEAN COAL DUST—PERIOD and trackside communities should worry." Sightline Institute.. May 4, 2016. <http://www.sightline.org/2016/05/04/coal-trains-mean-coal-dust-period>.
2. Khambekar, J and RA Bamum, RA. "PRB Coal-Material Handling Challenges and Solutions" Power Engineering 117:3 3/1/2013. <http://www.power-eng.com/articles/print/volume-117/issue-3/features/prb-coal-material-handling-challenges-and-solutions.html>
3. Doubery EB. Fire Protection Guidelines for Handling and Storing PRB Coal. June 3, 2013 <http://www.powermag.com/fire-protection-guidelines-for-handling-and-storing-prb-coal/>
4. Smoker, K and Albinger, R. "Mitigation of Fires in Coal Handling Facilities: Continuous Monitoring of Carbon Dioxide" <http://www.conspect-controls.com/resources/presentation-asme-prevention-of-fires-monitoring-co.asp>
5. Hossfeld R and Hatt, R. "PRB Coal Degradation – Causes and Cures" https://www.researchgate.net/publication/228972594_PRB_COAL_DEGRADATION-CAUSES_AND_CURES
6. Barbara Ellis, PhD. "Coal Fires: A Major Possibility Along the Gorge" March 22, 2013. <http://www.thegorge.com/coal-fires-major-possibility-gorge/>
7. Two Volunteer Fire Fighters Die After an Explosion While Attempting to Extinguish a Fire in a Coal Storage Silo – South Dakota Summary of a NIOSH fire fighter fatality investigation. September 14, 2012. <http://www.cdc.gov/niosh/fire/reports/face201122.html>
8. US Department of Energy Environmental Safety and Health Bulletin. "The Fire Below: Spontaneous Combustion in Coal." DOE/EH-0320 Issue No 93-4. May 1993. http://www.coaltrainfacts.org/docs/EH-93-4-The-Fire-Below_-Spontaneous-Combustion-in-Coal.pdf
9. Merritt, D and Rahm, R. "Managing silo, bunker and dust-collector fires" Power. Nov/Dec 2000. Pp. 53-60. <http://archive.prbcoals.com/pdf/PRBCoalInformation/12%20COAL%20FIRE%200.pdf>
10. "Seward Coal Terminal." Ground Truth Trekking. September 22, 2014. <http://www.groundtruthtrekking.org/Issues/AlaskaCoal/SewardCoalPort.html>
11. "Train car full of coal found smoldering near Chouteau." Fox23. Oct 21, 2014 - 9:10 PM <http://www.fox23.com/news/local/train-car-full-coal-found-smoldering-near-chouteau/109119614>
12. Block, S. "Coal-Fired Power Plants: Additional Hazards Require Additional Solutions." F.E. Moran Special Hazards Systems (The Moran Group) <http://www.femoranshs.com/prb-coal-hazard-article>
13. VandenHeuvel, Brett and de Place, Eric. "Coal Export: A History of Failure for Western Ports." Aug 2011. Columbia Riverkeeper and Sightline Institute. <http://www.communitywisebellingham.org/wp-content/uploads/2012/03/BNSF-Customers-What-I-Can-Ship-Coal-Coal-Dust-FAQs.pdf>
14. "Coal Dust Fire at Wyoming Mine Burns Three Miners." Casper Star Tribune, September 12, 2013 http://trib.com/business/energy/coal-dust-fire-at-wyoming-mine-burns-three-miners/article_2c0eca2c-847e-5f8e-8818-08c54aeca4bd.html

15. BNSF Railway Statement on STB Coal Dust Decision Coal Dust Frequently Asked Questions” [http://www.bnsf.com/customers/what-can-i-ship"/coal/coal-dust.html](http://www.bnsf.com/customers/what-can-i-ship)
16. West Antelope II Coal Lease Application: Environmental Impact ...Volume 1 p 3.152
https://books.google.com/books?id=UxEyAQAAMAAJ&pg=PA152&lpg=PA152&dq=train+smoldering+coal&source=bl&ots=rptspJ_bA6&sig=U03V5eJPfoizotLj_e-yZGDai1s&hl=en&sa=X&ved=0ahUKEwj4-c-zv
17. de Place, E. “COAL’S SPONTANEOUS COMBUSTION PROBLEM- Coal fires are a given, but what are the risks?” April 11, 2014. Sightline Institute.
<http://www.sightline.org/2012/04/11/coals-spontaneous-combustion-problem/>

I.G. Supplemental References

- ¹ Aasvang, G., Øverland, B., Ursin, R., & Torbjørn, M. (2011). A field study of effects of road traffic and railway noise on polysomnographic sleep parameters. *Journal of the Acoustical Society of America*, 129(6): 3716–26. <http://dx.doi.org/10.1121/1.3583547>
- ² Ammann, H., & Kadlec, M. (2008). “Concerns about Adverse Health Effects of Diesel Engine Emissions”. WA State Department of Ecology. Retrieved online: <https://fortress.wa.gov/ecy/publications/publications/0802032.pdf>
- ³ Armstrong, B., Sebastián, M., & Stephens, C. (2002). Outcomes of pregnancy among women living in the proximity of oil fields in the Amazon basin of Ecuador. *International Journal of Occupational and Environmental Health*, 8(4): 312-319. Retrieved online: <http://www.ncbi.nlm.nih.gov/pubmed/12412848>
- ⁴ Armstrong, B., Cordoba, J., & Stephens, C. (2001). Exposure and cancer incidence near oil fields in the Amazon basin of Ecuador. *Journal of Occupational and Environmental Medicine*, 58(8): 517-522. doi: [10.1136/oem.58.8.517](https://doi.org/10.1136/oem.58.8.517)
- ⁵ Ashley, S., & Ashley, W. (2008). Flood fatalities in the United States. *Journal of Applied Meteorology and Climatology*, 47: 805-818. Retrieved online: <http://journals.ametsoc.org/doi/pdf/10.1175/2007JAMC1611.1>
- ⁶ Avogbe, P., Ayi-Fanou, L., Autrup, H., Loft, S., Fayomi, B., Sanni, A., Vinzents, P., & Møller, P. (2004). Ultrafine particulate matter and high-level benzene urban air pollution in relation to oxidative DNA damage. *Carcinogenesis*, 26(3): 613-620. doi: [10.1093/carcin/bgh353](https://doi.org/10.1093/carcin/bgh353)
- ⁷ Babisch, W. (2005). Noise and health. *Environmental Health Perspectives*, 113(1): A14-A15. Retrieved online: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1253720/>
- ⁸ Becerra, T., Wilhelm, M., Olsen, J., Cockburn, M., & Ritz, B. (2013). Ambient air pollution and autism in Los Angeles County, California. *Environmental Health Perspectives*, 121(3); 380-386. doi: [10.1289/ehp.1205827](https://doi.org/10.1289/ehp.1205827)

- ⁹ Beeson, W., Abbey, D., & Knutson, S. (1998). Long-term concentrations of ambient air pollutants and incident lung cancer in California adults: results from the Adventist Health Study on Smog. *Environmental Health Perspectives*, 106(12): 813-23. Retrieved online: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1533247/>
- ¹⁰ Brook, R., Rajagopalan, S., Pope, C., III, Brook, J., Bhatnagar, A., Diez-Roux, A., Holguin, F., Hong, Y., Luepker, R., Mittleman, M., Peters, A., Siscovick, D., Smith, S., Jr., Whitsel, L., & Kaufman, J. (2010). Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. *Circulation*, 121(21): 2331-2378. doi: 10.1161/CIR.0b013e3181dbee1
- ¹¹ Carlsten, C., Dybuncio, A., Becker, A., Chan-Yeung, M., & Brauer, M. (2011). Traffic-related air pollution and incident asthma in a high-risk birth cohort. *Occupational Environmental Medicine*, 68: 291-295. doi:10.1136/oem.2010.055152
- ¹² Carter, N. (1996). Transportation noise, sleep, and possible after-effects. *Environment International*, 22(1): 105-116. doi: 10.1016/0160-4120(95)00108-5
- ¹³ Centers for Disease Control and Prevention (CDC) (1999). *Toxicological Profile for Total Petroleum Hydrocarbons*. US Department of Health and Human Services. Retrieved online: <http://www.atsdr.cdc.gov/ToxProfiles/tp123.pdf>
- ¹⁴ Chen, R., Zhang, Y., Yang, C., Zhao, Z., Xu, X., & Kan, H. (2013). Acute effect of ambient air pollution on stroke mortality in China air pollution and health effects study. *Stroke*, 44: 954-960. doi: 10.1161/STROKEAHA.111.673442
- ¹⁵ Chiu, Y., Bellinger, D., Coull, B., Anderson, S., Barber, R., Wright, R.O., & Wright, R.J. (2013). Associations between traffic-related black carbon exposure and attention in a prospective birth cohort of urban children. *Environmental Health Perspectives*, 121(7): 859-864. doi: 10.1289/ehp.1205940
- ¹⁶ Clark N., Demers P., **Karr C.**, Koehoorn, M., Lencar, C., Tamburic, L., & Brauer, M. (2010). Effect of early life exposure to air pollution on development of childhood asthma. *Environmental Health Perspectives*, 118(2): 284-290. doi: 10.1289/ehp.0900916
- ¹⁷ Control of Major Accident Hazards (COMAH) (2011). "Buncefield: Why did it happen?" Accessed online: <http://www.hse.gov.uk/comah/buncefield/buncefield-report.pdf>.
- ¹⁸ Crouse, D., Goldberg, M., Ross, N., Chen, H., & Labrèche, F. (2010). Postmenopausal breast cancer is associated with exposure to traffic-related air pollution in Montreal, Canada: a case-control study. *Environmental Health Perspectives*, 118(11): 1578-1583. doi: 10.1289/ehp.1002221

- ¹⁹ Croy, I., Smith, M., & Waye, K. (2013). Effects of train noise and vibration on human heart rate during sleep: an experimental study. *British Medical Journal Open*, 3(5):e002655. doi: 10.1136/bmjopen-2013-002655
- ²⁰ Curry, E., Dyer, C., Gill, D., & Pico, J. (1992). Disruption and stress in an Alaskan fishing community: initial and continuing impacts of the Exxon Valdez oil spill. *Industrial Crisis Quarterly*, 6:, 235-257. Retrieved online: <http://stevenpicou.com/pdfs/disruption-and-stress-in-an-alaskan-fishing-community.pdf>
- ²¹ D'Andera, M. & Reddy, G., (2013). Health consequences among subjects involved in Gulf oil spill clean-up activities. *American Journal of Medicine*, 126(11): 966-974. doi: <http://dx.doi.org/10.1016/j.amjmed.2013.05.014>
- ²² Dadvand, P. Parker, J., & Bell, M. (2012). Maternal exposure to particulate air pollution and term birth weight: a multi-country evaluation of effect and heterogeneity. *Environmental Health Perspectives*, 121(3). doi: 10.1289/ehp.1205575
- ²³ Davis, R. (July 14, 2014). "Everything you need to know about oil trains in Oregon, Washington." *The Oregonian*. http://www.oregonlive.com/environment/index.ssf/2014/07/everything_you_need_to_know_ab.html
- ²⁴ Davis, R. (April 23, 2014). "ODOT backs down from plan to limit disclosure of oil train shipments." *The Oregonian*. http://www.oregonlive.com/environment/index.ssf/2014/04/odot_backs_down_from_plan_to_1.html
- ²⁵ Delamater, P., Finley, A., & Banerjee, S. (2012). An analysis of asthma hospitalizations, air pollution, and weather conditions in Los Angeles County, California. *Science of the Total Environment*, 425: 110-118. doi: 10.1016/j.scitotenv.2012.02.015
- ²⁶ Demetriou, C., Raaschou-Nielsen, O., Loft, S., Møller, P., Vermeulen, R., Palli, D., Chadeau-Hyam, M., Xun, W.W., & Vineis, P. (2012). Biomarkers of ambient air pollution and lung cancer: a systematic review. *Occupational Environmental Medicine*, 69: 619-627. doi: 10.1136/oemed-2011-100566
- ²⁷ de Place, E., & Abbotts, J. (May 13, 2014). "Northwest Region Averaging Nine Freight Train Derailments Per Month". *Sightline Daily*. Retrieved online: <http://daily.sightline.org/2014/05/13/northwest-region-averaging-nine-freight-train-derailments-per-month/>
- ²⁸ De Rosa, M., Zarrilli, S., Paesano, L. Carbone, U., Boggia, B., Petretta, M., Maisto, A., Cimmino, F., Puga, G., Colao, A., & Lombardi, G. (2003). Traffic pollutants affect fertility in men. *Human Reproduction*, 18(5): 1055-61. Retrieved online: <http://www.ncbi.nlm.nih.gov/pubmed/12721184>

- ²⁹ Dockery, D., Pope, C.A., Xu, X., Spengler, J., Ware, J., Fay, M., Ferris, B., Jr., & Speizer, F. (1993). An association between air pollution and mortality in six US cities. *New England Journal of Medicine*, 329(24): 1753-1759. doi: 10.1056/NEJM199312093292401
- ³⁰ Dominici, F., Peng, R., Bell, M., Pham, L., McDermott, A., Zeger, S., & Samet, J. (2006). Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. *Journal of the American Medical Association*, 295(10): 1127-1134. doi: [10.1001/jama.295.10.1127](https://doi.org/10.1001/jama.295.10.1127)
- ³¹ Environmental Protection Agency (EPA) (2012). *Sole Source Aquifer Protection Program*. Retrieved online: <http://water.epa.gov/infrastructure/drinkingwater/sourcewater/protection/solesource/aquifer.cfm>
- ³² EPA (2009). *Integrated Science Assessment for Particulate Matter*. Retrieved online: <http://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=216546>
- ³³ EPA (1996). *National Air Toxic Assessment*. Retrieved online: <http://www.epa.gov/ttn/atw/nata/>
- ³⁴ Florip, E. (November 24, 2014). "Proposed oil terminal would be biggest in volume: Vancouver Energy's oil-by-rail capacity would be tops in U.S., analysis shows." *The Columbian*. Retrieved online: <http://www.columbian.com/news/2014/nov/24/proposed-oil-terminal-biggest-volume-vancouver/>
- ³⁵ Garland-Forshee, R. & Gedman, T. (2013). *The Burden of Asthma in Oregon*. Oregon Health Authority, Public Health Division. Retrieved online: http://public.health.oregon.gov/DiseasesConditions/ChronicDisease/Asthma/Documents/burden/OR_Asthma_2013.pdf
- ³⁸ Gent, J., Triche, E., Holford, T., Belanger, K., Bracken, M., Beckett, W., & Leaderer, B. (2003). Association of low-level ozone and fine particles with respiratory symptoms in children with asthma. *Journal of the American Medical Association*, 290(14): 1859-1867. Retrieved online: <http://jama.jamanetwork.com/>
- ³⁶ Gauderman, W., Avol, E., Gilliland, F., Vora, H., Thomas, D., Berhane, K., McConnell, R., Kuenzli, N., Lurmann, F., Rappaport, E., Margolis, H., Bates, D., & Peters, J. (2004). The effect of air pollution on lung development from 10 to 18 years of age. *New England Journal of Medicine*, 351(11): 1057-1067. doi: 10.1056/NEJMoa040610

- ³⁷ Geist, E. (2005). "Local Tsunami Hazards in the Pacific Northwest from Cascadia Subduction Zone Earthquakes." US Geological Survey (USGS) Professional Paper 1661-B. Retrieved online: <http://pubs.usgs.gov/pp/pp1661b/pp1661b.pdf>
- ³⁹ Gill, D., & Pico, J. (1996). The Exxon Valdez oil spill and chronic psychological stress. *American Fisheries Society Symposium*, 18: 879-893. Retrieved online: <http://stevenpicou.com/pdfs/the-exxon-valdez-oil-spill-and-chronic-psychological-stress.pdf>
- ⁴⁰ Goines, L., & Hagler, L. (2007). Noise pollution: a modern plague. *Southern Medical Journal*, 100(3): 287-294. Retrieved online: <http://www.medscape.com/viewarticle/554566>
- ⁴¹ Gowers, A., Cullinan, P., Ayres, J., Anderson, H., Strachan, D., Holgate, S., Mills, I., & Maynard, R. (2012). Does outdoor air pollution induce new cases of asthma? Biological plausibility and evidence; a review. *Respirology*, 17: 887-898. doi: 10.1111/j.1440-1843.2012.02195.x
- ⁴² Groves, R. (August 20, 2014). "Crude Oil Transportation by Rail." Memo to Mayor and City Council from Randall B. Groves, Chief of Eugene-Springfield Fire Department. Retrieved online: <https://www.eugene-or.gov/ArchiveCenter/ViewFile/Item/3267>
- ⁴³ *The Guardian* (January 16, 2015). "BP's maximum fine for Gulf of Mexico oil spill is cut by billions." Retrieved online: <http://www.theguardian.com/business/2015/jan/16/bp-fine-oil-spill-gulf-mexico-cut-deepwater-horizon>
- ⁴⁴ Haggerty, B., York, E., Early-Alberts, J., & Cude, C. (2014). *Oregon Climate and Health Profile Report*. Oregon Health Authority. Retrieved online: <http://public.health.oregon.gov/HealthyEnvironments/climatechange/Documents/oregon-climate-and-health-profile-report.pdf>
- ⁴⁵ Haines, M., Stansfeld, S., Job, R., Berglund, B., & Head, J. (2001). Chronic aircraft noise exposure, stress responses, mental health and cognitive performance in school children. *Psychological Medicine*, 31(2): 265-277. doi: <http://dx.doi.org/10.1017/S0033291701003282>
- ⁴⁶ Health Effects Institute (HEI), Panel on the Health Effects of Traffic-Related Air Pollution (2010). *Traffic-related air pollution: a critical review of the literature on emissions, exposure, and health effects*. Retrieved online: <http://pubs.healtheffects.org/getfile.php?u=553>
- ⁴⁷ Hertz-Picciotto, I., Park, H., Dostal, M., Kocan, A., Trnovec, T., & Sram, R. (2008). Prenatal exposures to persistent and non-persistent organic compounds and effects on

immune system development. *Basic and Clinical Pharmacology and Toxicology*, 102: 146-154. doi: 10.1111/j.1742-7843.2007.00190.x

⁴⁸ Hoek, M., Bracebridge, S., & Oliver, I. (2007). Health impact of the Buncefield oil depot fire, December 2005: Study of accident and emergency case records. *Journal of Public Health*, 29(3): 298-302. doi: 10.1093/pubmed/fdm036

⁴⁹ Hume, K., Brink, M., & Basner, M. (2012). Effects of environmental noise on sleep. *Noise and Health*, 14(61): 297-302. Retrieved online: www.noiseandhealth.org/text.asp?2012/14/61/297/104897

⁵⁰ Hygge, S., Evans, G., & Bullinger, M. (2002). A prospective study of some effects of aircraft noise on cognitive performance in schoolchildren. *Psychological Science*, 13(5): 469-474. doi: 10.1111/1467-9280.00483

⁵¹ Incardona, J., Gardner, L., Linbo, T., Brown, T., Esbaugh, A., Mager, E., Stieglitz, J., French, B., Labenia, J., Laetz, C., Tagal, M., Sloan, C., Benetti, D., Grosell, M., Block, B., & Scholz, N. (2014). Deepwater Horizon crude oil impacts the developing hearts of large predatory pelagic fish. *Proceedings of the National Academy of Sciences*, 111(15): E1510–E1518. doi: 10.1073/pnas.1320950111

⁵² Intergovernmental Panel on Climate Change (IPCC) (2012). *Managing the Risks of Extreme Events and Disasters to Advance Climate Change Adaptation. A Special Report of Working Groups I and II of the Intergovernmental Panel on Climate Change*. Field, C., Barros, V., Stocker, T., Qin, D., Dokken, K., et al. (Eds). Cambridge University Press, 582 pp. Retrieved online: http://ipcc-wg2.gov/SREX/images/uploads/SREX-All_FINAL.pdf

⁵³ Ising, H., & Kruppa, B. (2004). Health effects caused by noise: evidence from the literature from the past 25 years. *Noise & Health*, 6(22): 5-13. Retrieved online: <http://www.noiseandhealth.org/text.asp?2004/6/22/5/31678>

⁵⁴ Jackson, E., Yost, M., Karr, C., Fitzpatrick, C., Lamb, B., Chung, S., Chen, J., Avise, J., Rosenblatt, R., & Fenske, R. (2010). Public health impacts of climate change in Washington State: projected mortality due to heat events and air pollution. *Climate Change*, 102: 159-186. doi: 10.1007/s10584-010-9852-3

⁵⁵ Karr C., Demer, P., Koehoorn, M., Lencar, C., Tamburic, L., & Brauer, M. (2009). Influence of ambient air pollutant sources on clinical encounters for infant bronchiolitis. *American Journal of Respiratory Critical Care Medicine*, 180(10): 995–1000. doi: 10.1164/rccm.200901-0117OC

⁵⁶ Kim, M., Chang, S., Seong, J., Holt, J., Park, T., Ko, J., & Croft, J. (2012). Road traffic noise: annoyance, sleep disturbance, and public health implications. *American Journal of Preventative Medicine*, 43(4): 353-360. doi: <http://dx.doi.org/10.1016/j.amepre.2012.06.014>

- ⁵⁷ Knowlton, K., Rotkin-Ellman, M., Geballe, L., Max, W., & Solomon, G. (2011). Six climate change-related events in the United States accounted for about \$14 billion in lost lives and health costs. *Health Affairs*, 30: 2167-2176. doi:10.1377/hlthaff.2011.0229
- ⁵⁸ Koseki, H., Kokkala, M., & Mulholland, G. (1991). *Experimental Study of Boilover in Crude Oil Fires*. Published by the International Association for Fire Safety Science. Retrieved online: <http://www.iafss.org/publications/fss/3/865/view>
- ⁵⁹ Krishnan, R., Adar, S., Szpiro, A., Jorgensen, N., Van Hee, V., Barr, R., O'Neill, M., Herrington, D., Polak, J. & Kaufman, J. (2012). Vascular responses to long- and short-term exposure to fine particulate matter, MESA Air (Multi-Ethnic Study of Atherosclerosis and Air Pollution). *Journal of American College of Cardiology*, 60(21): 2158-2166. doi: [10.1016/j.jacc.2012.08.973](https://doi.org/10.1016/j.jacc.2012.08.973)
- ⁶⁰ Kurahashi, N., Kasai, S., Kahizaki, H., Nonomura, K., Sata, F., & Kishi, R. (2005). Parental and neonatal risk factors for cryptorchidism. *Medical Science Monitor*, 11(6): 274-283. Retrieved online: <http://www.ncbi.nlm.nih.gov/pubmed/15917718>
- ⁶¹ Laden, F., Neas, L., Dockery, D., & Schwartz, J. (2000). Association of fine particulate matter from different sources with daily mortality in six U.S. cities. *Environmental Health Perspectives*, 108(10): 941-7. Retrieved online: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1240126/>
- ⁶² Lercher, P., Evans, G., & Meix, M. (2003). Ambient noise and cognitive process among primary schoolchildren. *Environment and Behavior*, 35(6): 725-735. doi: 10.1177/0013916503256260
- ⁶³ Levy, B., & Nassetta, W. (2011). The adverse health effects of oil spills: a review of the literature and a framework for medically evaluating exposed individuals. *International Journal of Occupational and Environmental Health*, 17(2): 161-168. doi: <http://dx.doi.org/10.1179/107735211799031004>
- ⁶⁴ Luber, G., Knowlton, K., Balbus, J., Frumkin, H., Hayden, M., Hess, J., McGeehin, M., Sheats, N., et al. (2014). Chapter 9: Human Health. Climate Change Impacts in the United States. *The Third National Climate Assessment*. doi:10.7930/J0PN93H5
- ⁶⁵ Lue, S., Wellenius, G., Wilker, E., Mostofsky, E., & Mittleman, M. (2013). Residential proximity to major roadways and renal function. *Journal of Epidemiological Community Health*, 67:629-634. doi:10.1136/jech-2012-202307
- ⁶⁶ Martinez, R., Snell, T., & Shearer, T. (2013). Synergistic toxicity of Macondo crude oil and dispersant Corexit 9500A to the *Brachionus plicatilis* species complex. *Environmental Pollution*, 173: 5-10. doi:10.1016/j.envpol.2012.09.024

- ⁶⁷ McConnell, R., Berhane, K., Gilliland, F., Islam, T., & Gauderman, W. (2002). Asthma in exercising children exposed to ozone: a cohort study. *Lancet*, 359: 386-391. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/11844508>
- ⁶⁸ McCoy, D., & Hoskins, B. (2014). The science of anthropogenic climate change: what every doctor should know. *British Medical Journal*, 349: g5178. doi: 10.1136/bmj.g5178
- ⁶⁹ Miller, K., Siscovick, D., Sheppard, L., Shepherd, K., Sullivan, J., Anderson, G., & Kaufman, J. (2007). Long term exposure to air pollution and incidence of cardiovascular events in women. *New England Journal of Medicine*, 356(5): 447-458. doi: 10.1056/NEJMoa054409
- ⁷⁰ Mustafic, H., Jabre, P., Caussin, C., Murad, M., Escolano, S., Tafflet, M., Périer, M-C., Marijon, E., Vernerey, D., Empana, J-P., & Jouven, X. (2012). Main air pollutants and myocardial infarction, a systematic review and meta-analysis. *Journal of the American Medical Association*, 307(7): 713-721. doi: 10.1001/jama.2012.126
- ⁷¹ National Commission on the BP Deepwater Horizon Oil Spill and Offshore Drilling (2011). *Deep Water: The Gulf Oil Disaster and Future of Offshore Drilling - Report to the President*. Retrieved online: <http://www.gpo.gov/fdsys/pkg/GPO-OILCOMMISSION/pdf/GPO-OILCOMMISSION.pdf>
- ⁷² National Oceanic and Atmospheric Administration (NOAA), National Weather Service (2012). *Weather Fatalities*. Retrieved online: www.nws.noaa.gov/om/hazstats.shtml
- ⁷³ National Transportation Safety Board (NTSB), Chairman Deborah Hersman (March 2, 2012). *Safety Recommendation*. Retrieved online: <http://www.nts.gov/safety/safety-recs/RecLetters/R-12-005-008.pdf>
- ⁷⁴ Newman, N., Ryan, P., LeMasters, G., Levin, L., Bernstein, D., Khurana Hershey, G., Lockey, J., Villareal, M., Reponen, T., Grinshpun, S., Sucharew, H., & Dietrich, K. (2013). Traffic-related air pollution exposure in the first year of life and behavioral scores at 7 years of age. *Environmental Health Perspectives*, 121(6): 731-736. doi: 10.1289/ehp.1205555
- ⁷⁵ Olsson, D., Mogren, I., & Forsberg, B. (2013). Air pollution exposure in early pregnancy and adverse pregnancy outcomes: a register-based cohort study. *British Medical Journal*, 3(2). Retrieved online: <http://bmjopen.bmj.com/content/3/2/e001955>. doi: 10.1136/bmjopen-2012-001955
- ⁷⁶ Oregon Department of Environmental Quality (OR DEQ) (2014). Standard Air Contaminant Discharge Permit Review Report, Permit No.: 05-0023-ST-01, Application No.: 027492. Retrieved online: http://www.oregon.gov/deq/docs/CPBR_aqRR2014.pdf

- ⁷⁷ Oregon Department of Transportation (ODOT) (January 23, 2015). Personal communication with Regna Merritt, Oregon Physicians for Social Responsibility.
- ⁷⁸ Oregon Health Authority (OHA) (2015). *Drinking Water Data Online*. Oregon Public Health Retrieved online: <https://yourwater.oregon.gov/search.htm>
- ⁷⁹ Oregon Physicians for Social Responsibility. *Airborne Particulate Matter & Public Health*. Retrieved online: <http://www.psr.org/chapters/oregon/assets/pdfs/airborne-particulate-matter.pdf>
- ⁸⁰ Palinkas, L., Petterson, J., Russell, J., & Downs, M. (2004). Ethnic differences in symptoms of post-traumatic stress after the Exxon Valdez oil spill. *Prehospital and Disaster Medicine*, 19(1): 102-112. Retrieved online: <http://www.ncbi.nlm.nih.gov/pubmed/15453167>
- ⁸¹ Pandya, R., Solomon, G., Kinner, A., & Balmes, J. (2002). Diesel exhaust and asthma: hypotheses and molecular mechanisms of action. *Environmental Health Perspectives*, 110(1): 103-112. Retrieved online: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1241152/>
- ⁸² Perera, F., Wang, S., Rauh, V., Zhou, H., Stigter, L., Camann, D., Jedrychowski, W., Mroz, E., & Majewska, R. (2013). Prenatal exposure to air pollution, maternal psychological distress, and child behavior. *Pediatrics*, 132(5): e1284-1294. doi: 10.1542/peds.2012-3844
- ⁸³ Perera, F., Li, Z., Whyatt, R., Hoepner, L., Wang, S., Camann, D., & Rauh, V. (2009). Prenatal airborne polycyclic aromatic hydrocarbon exposure and child IQ at age 5 years. *Pediatrics*, 124(2): e195-202. doi:10.1542/peds.2008-3506
- ⁸⁴ Peters, A., Dockery, D., Muller, J., & Mittleman, M. (2001). Increased particulate air pollution and the triggering of myocardial infarction. *Circulation*, 103(23): 2810-2815. doi: 10.1161/01.CIR.103.23.2810
- ⁸⁵ Peters, A., Liu, E., Verrier, R., Schwartz, J., Gold, D., Mittleman, M., Baliff, J., Oh, J., Allen, G., Monahan, K., & Dockery, D. (2000). Air pollution and incidence of cardiac arrhythmia. *Epidemiology*, 11(1): 11-17. Retrieved online: <http://www.ncbi.nlm.nih.gov/pubmed/10615837>
- ⁸⁶ Peterson, C., Rice, S., Short, J., Esler, D., Bodkin, J., Ballachey, B., & Irons, D. (2003) Long-term ecosystem response to the Exxon Valdez oil spill. *Science*, 302 (5653): 2082-2086. doi: 10.1126/science.1084282
- ⁸⁷ Pieters, N., Plusquin, M., Cox, B., Kicinski, M., Vangronsveld, J., & Nawrot, T. (2012). An epidemiological appraisal of the association between heart rate variability

and particulate air pollution: a meta-analysis. *Heart*, 98: 1127-1135. doi: 10.1136/heartjnl-2011-301505

⁸⁸ Pipeline and Hazardous Materials Safety Administration (May 2014). *Operation Safe Delivery Update*. Retrieved online:

http://www.phmsa.dot.gov/pv_obj_cache/pv_obj_id_8A422ABDC16B72E5F166FE34048CCCBFED3B0500/filename/07_23_14_Operation_Safe_Delivery_Report_final_clean.pdf

⁸⁹ Pope, C., III, Burnett, R., Thun, M., Calle, E., Krewski, D., Ito, K., & Thurston, G. (2002). Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *Journal of the American Medical Association*, 287(9): 1132-1141. Retrieved online: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4037163/>

⁹⁰ Puget Sound Clean Air Agency (PSCAA) (2015). *Air Pollution & Your Health*. Retrieved online:

<http://www.pscleanair.org/airquality/airqualitybasics/health/Pages/default.aspx>

⁹¹ PSCAA (2011). *Air Toxics*. Retrieved online:

www.pscleanair.org/library/Documents/Air%20Toxics%20Fact%20Sheet%201-31-11.pdf

⁹² Qian, Y., Zhu, M., Cai, B., Yang, Q., Kan, H., Song, G., Jin, W., Han, M., & Wang, C. (2013). Epidemiological evidence on association between ambient air pollution and stroke mortality. *Journal of Epidemiology and Community Health*, 67: 635-640. doi: 10.1136/jech-2012-201096

⁹³ Roberts, A., Lyall, K., Hart, J., Laden, F., Just, A., Bobb, J., Koenen, K., Ascherio, A., & Weisskopf, M. (2013). Perinatal air pollutant exposures and autism spectrum disorder in the children of Nurses' Health Study II participants. *Environmental Health Perspectives*, 121(8): 978-984. doi: 10.1289/ehp.1206187

⁹⁴ Rotkin-Ellman, M., Wong, K., & Solomon, G. (2012). Seafood contamination after the BP Gulf oil spill and risks to vulnerable populations: a critique of the FDA risk assessment. *Environmental Health Perspectives*, 120(2): 157-61. doi: 10.1289/ehp.1103695

⁹⁵ Selander, J., Nilsson, M., Bluhm, G., Rosenlund, M., Lindqvist, M., Nise, G., & Pershagen, G. (2009). Long-term exposure to road traffic noise and myocardial infarction. *Epidemiology*, 20(2): 272-279. doi: 10.1097/EDE.0b013e31819463bd

⁹⁶ Shannon & Wilson, Inc. (2013). Draft Geotechnical Report, USD Crude-by-Rail Terminal, Port of Grays Harbor, Hoquiam, WA. Retrieved online:

<http://cityofhoquiam.com/pdf/GHRT-Geotech-Report.pdf>

- ⁹⁷ Shaw, A., Langrish, J., Nair, H., McAllister, D., Hunter, A., Donaldson, K., Newby, D., & Mills, N. (2013). Global association of air pollution and heart failure: a systematic review and meta-analysis. *The Lancet*, 832: 1039-1048. doi: 10.1016/S0140-6736(13)60898-3
- ⁹⁸ Simon, S. (October 17, 2013). "World Health Organization: Outdoor Air Pollution Causes Cancer." American Cancer Society. Retrieved online: <http://www.cancer.org/cancer/news/world-health-organization-outdoor-air-pollution-causes-cancer>
- ⁹⁹ Slaughter, J., Lumley, T., Sheppard, L., Koenig, J., & Shapiro, G. (2003) Effects of ambient air pollution on symptom severity and medication use in children with asthma. *Annals of Allergy, Asthma & Immunology*, 91: 346–353. doi: [http://dx.doi.org/10.1016/S1081-1206\(10\)61681-X](http://dx.doi.org/10.1016/S1081-1206(10)61681-X)
- ¹⁰⁰ Sørensen, M., Andersen, J., Nordsborg, R., Jensen, S., Lillelund, K., Beelen, R., Schmidt, E., Tjønneland, A., Overvad, K., & Raaschou-Nielsen, O. (2012). Road traffic noise and incident myocardial infarction: a prospective cohort study. *PLoS ONE*, 7(6): e39283. doi: 10.1371/journal.pone.0039283
- ¹⁰¹ Sørensen, M., Hvidberg, M., Andersen, Z., Nordsborg, R., Lillelund, K., Jakobsen, J., Tjønneland, A., Overvad, K., & Raaschou-Nielsen, O. (2011). Road traffic noise and stroke: a prospective cohort study. *European Heart Journal*, 32(6): 737-744. doi: <http://dx.doi.org/10.1093/eurheartj/ehq466>
- ¹⁰² Tesoro Savage Vancouver Energy Distribution Terminal (August 2013). Application No. 2013-01. Retrieved online: <http://www.efsec.wa.gov/Tesoro%20Savage/Application/Tesoro%20Savage%20Application%20Page.shtml>
- ¹⁰³ Toxipedia Consulting Services (2011). The Chaos of Cleanup: Analysis of the Potential Health and Environmental Impacts of Chemicals in Dispersant Products. Retrieved online: <http://toxipedia.org/display/toxipedia/Oil+Dispersant>
- ¹⁰⁴ Trasande, L., & Thurston, G. (2005). The role of air pollution in asthma and other pediatric morbidities. *Journal of Allergy and Clinical Immunology*, 115(4): 689-699. doi: 10.1016/j.jaci.2005.01.056
- ¹⁰⁵ United Nations Environment Programme (UNEP) (2011). *Environmental Assessment of Ogoniland*. Retrieved online: <http://www.theguardian.com/environment/interactive/2011/aug/04/un-environmental-impact-ogoniland>
- ¹⁰⁶ United States Chemical Safety and Hazard Investigation Board (US CSB) (2007). Case study: Hot work control and safe work practices at oil and gas production wells.

Report no. 2006-07-I-MS. Retrieved online:

http://www.csb.gov/assets/1/19/Partridge_Report1.pdf

¹⁰⁷ United States Department of Labor (n.d.). *Occupational Safety & Health Standards*. Retrieved online: <https://www.osha.gov/law-regs.html>

¹⁰⁸ United States Department of Transportation (US DOT) (2014). *National Transportation Statistics*. Retrieved online: www.rita.dot.gov/bts/sites/rita.dot.gov/bts/files/publications/national_transportation_statistics/index.html

¹⁰⁹ US DOT (2012). *Emergency Response Guidebook*. Retrieved online: http://phmsa.dot.gov/pv_obj_cache/pv_obj_id_7410989F4294AE44A2EBF6A80ADB640BCA8E4200/filename/ERG2012.pdf

¹¹⁰ US DOT (2005). *Train Horn Rule: 49 CFR Part 222*. Retrieved online: <http://www.fra.dot.gov/Page/P0105>

¹¹¹ Volk, H., Lurmann, F., Penfold, B., Hertz-Picciotto, I., & McConnell, R. (2013). Traffic-related air pollution, particulate matter, and autism. *Journal of the American Medical Association Psychiatry*, 70(1): 71-77. doi: 10.1001/jamapsychiatry.2013.266

¹¹² Volk, H., Hertz-Picciotto, I., Delwiche, L., Lurmann, F., & McConnell, R. (2011). Residential proximity to freeway and autism in the CHARGE study. *Environmental Health Perspectives*, 119(6): 873-877. doi: 10.1289/ehp.1002835

¹¹³ Vrijheid, M., Martinez, D., Manzanares, S., Dadvand, P., Schembari, A., Rankin, J., & Nieuwenhuijsen (2011). Ambient air pollution and risk of congenital anomalies: a systematic review and meta-analysis. *Environmental Health Perspectives*, 119(5): 598-606. doi: 10.1289/ehp.1002946

¹¹⁴ WA State Department of Ecology (WA DOE) (2014). *Washington State Marine & Rail Oil Transportation Study Preliminary Findings & Recommendations*. Retrieved online: <http://www.ecy.wa.gov/programs/spills/OilMovement/2014MarineRailOilTransportStudyDraftFindings.pdf>

¹¹⁵ WA DOE (1995). *Facility Oil Handling and Design Standards Rule*. Retrieved online: <https://fortress.wa.gov/ecy/publications/publications/94195.pdf>

¹¹⁶ WA State Department of Health (WA DOH) (2013). *The Burden of Asthma in Washington State*. Retrieved online: <http://www.doh.wa.gov/Portals/1/Documents/Pubs/345-240-AsthmaBurdenRept13.pdf>

- ¹¹⁷ Wei, Y., Davis, J., & Bina, W. (2012). Ambient air pollution is associated with the increased incidence of breast cancer in US. *International Journal of Environmental Health Research*, 22(1): 12-21. doi: 10.1080/09603123.2011.588321
- ¹¹⁸ Wellenius, G., Burger, M., Coull B., Schwartz, J., Suh, H., Koutrakis, P., Schlaug, G., Gold, D., & Mittleman, M. (2012). Ambient air pollution and the risk of acute ischemic stroke. *Archives of Internal Medicine*, 172(3): 229-234. doi: 10.1001/archinternmed.2011.732
- ¹¹⁹ Wellenius, G., Schwartz, J., & Mittleman, M. (2005). Air pollution and hospital admissions for ischemic and hemorrhagic stroke among Medicare beneficiaries. *Stroke*, 36(12): 2549-2553. doi: 10.1161/01.STR.0000189687.78760.47
- ¹²⁰ Wingspread Conference on the Precautionary Principle (January 26, 1998). *Science and Environmental Health Network*. Retrieved online: <http://www.sehn.org/wing.html>
- ¹²¹ Woodruff, T., Parker, J., & Schoendorf, K. (2006). Fine particulate matter (PM_{2.5}) air pollution and selected causes of postneonatal infant mortality in California. *Environmental Health Perspectives*, 114(5): 786-790. doi: [10.1289/ehp.8484](https://doi.org/10.1289/ehp.8484)
- ¹²² World Health Organization (WHO) (2014). *Ambient (outdoor) air quality and health*. Fact Sheet no. 313. Retrieved online: <http://www.who.int/mediacentre/factsheets/fs313/en/>
- ¹²³ WHO (2005). *Effects of Air Pollution on Children's Health and Development*. Retrieved online: http://www.euro.who.int/_data/assets/pdf_file/0010/74728/E86575.pdf
- ¹²⁴ WHO (2003). *Health aspects of air pollution with particulate matter, ozone, and nitrogen dioxide*. Retrieved online: http://www.euro.who.int/_data/assets/pdf_file/0005/112199/E79097.pdf
- ¹²⁵ WHO (1999). *Guidelines for Community Noise: Adverse Health Effects of Noise*. Retrieved online: <http://www.who.int/docstore/peh/noise/Comnoise-3.pdf>

