BEFORE THE STATE OF WASHINGTON
ENERGY FACILITY SITE EVALUATION COUNCIL

In the Matter of: Application No. 2013-01 TESORO SAVAGE, LLC VANCOUVER ENERGY DISTRIBUTION TERMINAL	)	CASE NO. 15-001 DIRECT TESTIMONY OF ELINOR FANNING, PH.D.

I. INTRODUCTION

1. I am an independent consultant in environmental health engaged in the assessment and analysis of health effects of environmental toxicants, including air pollutants. I have over 15 years of experience in the fields of environmental health, toxicology, and health risk assessment, with a particular focus in air toxics. I possess a B.A. in biology from Oberlin College, Ohio (1986), a M.A. degree in molecular biology from the University of California at Berkeley (1992), and a Ph.D. in Environmental Health Science from the University of California at Berkeley (1998). I have worked as an associate toxicologist for the Office of Environmental Health Hazard Assessment in the state of California and as a research associate and assistant director in the Center for Occupational and Environmental Health at the University of California at Los Angeles. In the course of my work, I have assessed health risks associated with gasoline, hazards of chemical carcinogens, and participated in research on particulate matter air pollution. My CV is attached to this testimony.

2. This testimony concerns a proposal by Tesoro Savage Petroleum Terminal LLC

Earthjustice
705 Second Ave., Suite 203
Seattle, WA 98104-1711
(206) 343-7340

(Tesoro Savage) to construct and operate a crude oil distribution terminal (the "Terminal") at the Port of Vancouver, Washington. I have reviewed an Air Permit Application submitted by Tesoro Savage[1] and the draft Environmental Impact Statement ("DEIS") developed by the EFSEC. From these sources, I understand that the project proposes to receive by rail, unload, store, transfer, and ship by marine vessel crude oil in quantities of 360,000 barrels per day at full operation. I have previously submitted comments on the DEIS, and those comments are attached as Exhibit 5530-000006-CRK. My comments on the DEIS and here address adverse effects on public health due to air pollution that would be produced by the facility and ancillary activities should the Terminal be constructed and operated as proposed by the applicant. To form my opinions, I reviewed the Air Permit Application, the DEIS, and scientific documents cited in this report. I have also relied upon my technical background and general knowledge of air pollution science, health hazard assessment, and biological effects of chemical toxicants.

II. THE TERMINAL WOULD EMIT OR CAUSE AIR POLLUTION OF CONCERN FOR HUMAN HEALTH (IDENTIFICATION OF CHEMICALS OF CONCERN).

3. Crude oil transportation and transloading contribute to ambient air pollution through a) vaporization of oil constituents in the transportation process or from storage and transfer equipment; b) stationary source emissions during terminal operations; c) emissions of combustion by-products from mobile sources involved in oil transport; and d) chemical reactions of the emitted pollutants in the atmosphere to form secondary pollutants. In this section, I identify the toxic substances of concern; in section IV, I address the likely health effects of those substances.

4. Particulate matter (PM) will be emitted from the Terminal by stationary and mobile sources, including marine vessels and locomotives. According to Tesoro Savage, the

[1] Air Permit Application, Revised August 2014, Exhibit 0002-000000-PEC.

Earthjustice
705 Second Ave., Suite 203
Seattle, WA 98104-1711
(206) 343-7340

highest particulate matter emissions during Terminal operations will come from Area 600 boilers and from marine vessels while they are docked during loading (referred to as "hoteling" emissions). While not noted in the documentation, particulate matter is also produced as a secondary pollutant from other emitted substances that act as precursors.[2]

5. Particulate air pollution is highly varied and complex. It may be in solid, liquid, or semi-volatile state. Particles derive from numerous different source types and vary in size, shape, and chemical composition. They can be formed during incomplete combustion, after emission of precursors to air, or when dusts are re-entrained into the air. Particles of less than 2.5 microns are referred to collectively as PM2.5, mostly derive from combustion sources, and are the most toxic. Diesel exhaust particulate (DEP), a subset of PM2.5, is composed of soot with sulfates, hydrocarbons, metals, and other toxic species condensed on the soot. Sources such as the locomotives, marine vessels, vehicles, and off-road diesel equipment that are associated with the proposed Terminal emit diesel exhaust particulate.

6. Nitrogen oxides (NOx) are a group of related pollutants containing nitrogen and oxygen. The United States Environmental Protection Agency (EPA) establishes air quality standards, called NAAQS[3], for the most prevalent anthropogenic (caused by human activity) member of the group, nitrogen dioxide (NO2). Like particulate matter, NOx is emitted from combustion sources. The DEIS identifies tugboats, locomotives and vessels while they are stationary at the dock (hoteling) as major NOx sources. The relative contributions to NOx of

[2] Precursor is used here as in scientific and regulatory documents on atmospheric chemistry. An emitted chemical may possess intrinsic toxic properties but also react with other chemicals in the air to form new pollutants of concern. For example, hydrocarbon precursors undergo post-emission chemical transformations that form "secondary organic aerosol." This secondary aerosol is an important fraction of the PM2.5 that is found in outdoor ambient air.

[3] National Ambient Air Quality Standards (NAAQS) are health protective regulatory levels established by the EPA under the Clean Air Act for six "criteria pollutants": ozone, lead, particulate matter, carbon monoxide, sulfur dioxide and nitrogen dioxide.

Earthjustice
705 Second Ave., Suite 203
Seattle, WA 98104-1711
(206) 343-7340

different Terminal pollution sources are currently unclear to me, due to inconsistencies in reporting emissions among the documents that I reviewed (see Section III, paragraph 12), auxiliary ship engines and locomotives are important sources.

7. The Terminal will emit carbon monoxide and sulfur dioxide. The major sources of carbon monoxide will be employee passenger vehicles and tugboat engines. Sulfur dioxide emissions are highly dependent upon the sulfur level in the fuel used by the marine vessels.

8. In addition to criteria pollutants, Tesoro Savage disclosed emissions estimates for a list of toxic air pollutants that will be emitted from stationary sources at the Terminal (itemized in Table 5.1-12 in the Revised Air Permit Application). Six toxic air pollutants plus nitrogen dioxide and sulfur dioxide will be emitted at rates that exceed Washington State "small quantity emission rates". These 6 chemicals (arsenic, benzene, cadmium, hexavalent chromium, 7,12-dimethylbenz(a)anthracene, and diesel engine particulate) are all human carcinogens (see section IV). It should be noted that only stationary sources were considered. The contributions of marine vessels, locomotives, and other mobile sources were not included in the analysis of toxic air pollutant emissions, and therefore emissions relevant to the total impact of Terminal operations are substantially underestimated. Toxic contaminants for which emissions would increase notably from what was disclosed after accounting for mobile sources include acetaldehyde, acrolein, benzene, 1,3-butadiene, formaldehyde, naphthalene, and toluene.

9. It is my understanding that the Terminal is designed primarily for the purposes of handling crude oils from mid-continent shale formations, such as Bakken crude. Relative to other crude oils, Bakken crudes contain more of the most volatile hydrocarbons. Volatile

Earthjustice
705 Second Ave., Suite 203
Seattle, WA 98104-1711
(206) 343-7340

organic compounds (VOC)[4] are relevant for human health as ozone precursors (see footnote 2 for explanation of precursor). Ozone[5] is not directly emitted; it is formed as a secondary pollutant when the VOC and NOx emitted from the Terminal undergo chemical reactions in the atmosphere, in the presence of sunlight. VOCs also contribute to the atmospheric formation of secondary organic aerosol (a type of particulate matter). In the Portland/Vancouver area, ozone formation is thought to be "VOC limited," meaning that increases in VOC pollution will increase ozone formation. See section IV for ozone health effects.

10 In the event that the Terminal accepts crude oils other than the expected light shale oils like Bakken, the identities and relative quantities of chemicals emitted will vary from what was disclosed by Tesoro Savage. Heavy tar sands oils differ from shale oil crudes in chemical composition. Among other differences, they are higher in sulfur and metal content. Transloading of these oils would add, minimally, nickel to the list of Terminal toxicants. Hydrogen sulfide is also of concern with tar sands crudes. Heavy crudes are diluted with volatile and toxic diluents to facilitate transport; diluent compounds elevate VOC emissions during transport and transfer and contribute to direct toxicity. For example, benzene is a highly volatile toxic that is among the chemicals that are intentionally added to dilute and render fluid the heavy bitumen extracted from Canadian oil sands. The hazards of diluted bitumen from oil sands are particularly relevant to areas that are vulnerable to air pollution from the refineries that process the crude. My understanding is that refineries in Washington State may be recipients of the

[4] The term VOC is variously defined in scientific and regulatory documents. This report uses the definition commonly applied in laws and regulations concerning outdoor air pollution: compounds of carbon that participate in atmospheric photochemical reactions. VOC contribute to formation of ground level ozone (smog), and particulate matter (secondary organic aerosols) via post-emission photochemical reactions in the atmosphere.

[5] I refer to ground level ozone, a toxic criteria pollutant under the Clean Air Act, as opposed to stratospheric ozone which is the protective layer of ozone high in the atmosphere.

Earthjustice
705 Second Ave., Suite 203
Seattle, WA 98104-1711
(206) 343-7340

crude oil products distributed by the Terminal. Any impacts of Terminal products on Washington refinery emissions profiles should, therefore, be considered in addition to impacts from the Terminal and the rail and marine transport associated the Terminal.

III. THE CONCENTRATIONS OF AIR POLLUTANTS THAT WILL RESULT FROM TERMINAL EMISSIONS ARE UNKNOWN OR INCOMPLETLY CHARACTERIZED.

11. The concentrations of the contaminants to which people are exposed determine the magnitude of the health effects that result. While prediction can't be perfect, dispersion modeling is a tool used frequently in regulatory and research settings to link emitted quantities to human exposure. Dispersion models use mathematical equations and sets of assumptions to describe the spread of emissions from stack to "receptor" locations. The models can take information about the location of pollution sources and emitted quantities of contaminants, combine those data with information about geography and meteorology, and generate estimates of the expected concentrations in the air at receptor locations. Exposure concentration estimates developed through modeling can be useful for assessing possible health risks when model inputs and assumptions are carefully selected, but because dispersion models are complex and imperfect tools, the output concentrations should be considered as approximate. Tables 5.1-21 – 5.1-23 of the Air Permit Application document, and Table 3.2-8 of the DEIS report results of the modeling that was done by Tesoro Savage. In this case, I have concerns that prevent me from relying on the disclosed concentration estimates for health risk assessment.

12. First, the methodology, assumptions and inputs are insufficiently documented in the documents I reviewed. Conflicting emissions rates and volumes are reported for mobile sources in different sections of the DEIS. For example, reported NOx emissions for on-site locomotives vary 40-fold between Appendices F and G of the DEIS, and emission rates for

Earthjustice
705 Second Ave., Suite 203
Seattle, WA 98104-1711
(206) 343-7340

PM2.5 and NOx cited to Burlington Northern Santa Fe Railroads are not consistent with those quoted from USEPA. Appendix F does not clearly specify which of the reported emissions data were selected for input to the dispersion modeling.

13. Second, modeling the combined emissions from mobile and stationary sources is incomplete. When trains, ships and other mobile sources are added into the modeling (as was done in the DEIS), the air concentrations of PM2.5 that resulted from modeling appear lower than when only stationary sources were modeled (in the Air Permit Application). The model settings that led to these apparently conflicting findings are not clearly disclosed. For non-criteria toxic compounds, modeling of combined mobile and stationary sources was only reported for formaldehyde and diesel engine exhaust. Tesoro Savage's argument is that formaldehyde has a high emission rate and is, therefore, the most relevant compound to model. This argument neglects the key point that toxicants vary considerably in potency. Chemicals emitted in lower volumes can have greater health effects, and combined effects of chemicals that affect the same body tissues are an important consideration. Short-term exposures to non-criteria pollutants were not modeled, or at least findings of short-term toxic exposures were not reported, yet health effects from short-term exposure are highly relevant here (see section IV on health effects).

14. Third, it is also worth noting that the most commonly used dispersion model, and the one used in the air permit application, cannot account for secondary formation of air pollutants. Ambient air concentrations of formaldehyde, acrolein, and particulate matter, for example, are significantly increased by secondary formation from precursors that will be emitted from the Terminal operations and crude oil transport. No adjustment to results was made.

Earthjustice
705 Second Ave., Suite 203
Seattle, WA 98104-1711
(206) 343-7340

15. The findings in paragraphs 12-14 reduce my confidence in the estimates of exposure to air contaminants derived from the Terminal that have been reported to date.

IV. THE HARMS TO HUMAN HEALTH FROM THE POLLUTANTS OF CONCERN INCLUDE PREMATURE DEATH, CANCER, AND MORBIDITIES OF THE CARDIOVASCULAR AND RESPIRATORY SYSTEMS.

16. The air pollutants associated with the Tesoro Savage Terminal are known to cause a wide range of serious harms to human health. Exposure levels that will be caused by the Terminal are unknown, but the Tesoro Savage documents argue that exposures will remain below levels of regulatory significance. Whether or not that proves to be true, adverse health effects occur at exposure levels below regulatory air quality standards; regulatory compliance does not ensure public health protection. Rather than present an exhaustive description of all possible health effects of Terminal pollution, I discuss selected effects that are caused by the most prevalent Terminal contaminants at exposure concentrations that are close to typical background air concentrations. By so doing, I do not exclude the possibility that other relevant health effects will occur, especially in the event of a large scale accidental release from the Terminal or during transport to/from the Terminal.

A. Health effects from short term exposure: premature death and hospitalization from cardiovascular causes and respiratory toxicity.

17. Acute health effects are those that occur in response to short term exposure periods and may be different than the effects observed with long-term low exposure to the same chemical. Short term exposure episodes can occur when a pollutant concentration is temporarily elevated over its typical background rate; peak concentrations are more relevant than average concentrations for determining the health effects that follow from short exposures. EPA has set NAAQS for 1 hour, 8 hour and 24 hour periods, depending upon the specific toxic profile of the contaminant in question.

Earthjustice
705 Second Ave., Suite 203
Seattle, WA 98104-1711
(206) 343-7340

18. Undisputed evidence links short-term (hours to days) increases in the concentration of ambient PM2.5 to increased mortality from cardiovascular causes, over the following days.[6] This effect occurs in response to small changes in PM2.5 concentrations in communities, increases that occur within the range of typical outdoor air concentrations[7]. The premature deaths associated with short-term particulate matter increases are largely from heart attack, heart failure, and stroke, and are more likely to occur in susceptible individuals: the elderly and those with existing cardiovascular disease. Data from a large number of reputable, peer-reviewed studies taken together show that daily mortality across a population increases 0.5-3% for every 10ug/m3 (micrograms of particles in a cubic meter of air) increase in 24hr PM2.5 above background ambient concentration. The DEIS reported PM2.5 background in the vicinity of the Terminal at about 20ug/m3, and an estimated increase in 24 hour PM2.5 of 5.4ug/m3 at the maximal exposure location. While the estimate is subject to the concerns detailed in Section III paragraphs 12-14, PM2.5 from the Terminal will increase air concentrations in the range that has a clear effect in scientific studies.

19. Non-fatal hospitalization for cardiovascular causes follows a similar relationship to small short-term increases in PM2.5 concentrations, as does death from cardiovascular causes at similarly low concentration changes.

20. Short-term spikes in Terminal emissions will also cause respiratory morbidity

---

[6] Evidence from numerous studies summarized by a committee of scientists convened by the American Heart Association: Brook *et al*., 2010 Circulation 121:2331. Review and health guidance by the World Health Organization in: WHO 2013 Review of evidence on health aspects of air pollution—REVIHAAP project, technical report. http://www.euro.who.int/__data/assets/pdf_file/0004/193108/REVI HAAP-Final-technical-report-final-version.pdf

[7] Shi L et al 2016. Environmental Health Perspectives 124:46. http://dx.doi.org/10.1289/ehp.1409111 show that the concentration-effect relationship for PM2.5 and acute mortality is linear at low concentrations. Exhibit 5531-000007-CRK.

Earthjustice
705 Second Ave., Suite 203
Seattle, WA 98104-1711
(206) 343-7340

(disease of the respiratory system). Numerous respiratory toxicants are associated with the Terminal: diesel exhaust, PM2.5, NOx, ozone, formaldehyde, acetaldehyde, acrolein, for example. Short-term exposures to a mixture of multiple respiratory toxicants are likely to increase significantly for Port employees and for employees and inmates at the Clark County Jail Work Center, yet, for most of these contaminants, short term exposures were not considered in Tesoro Savage's and EFSEC's documentation. People with respiratory illness, such as asthma and chronic obstructive pulmonary disease are more sensitive to the health effects of respiratory irritants and will respond with worsened symptoms at lower air concentrations.

21. Acute (short-term) exposure to diesel exhaust aggravates asthma, chronic obstructive pulmonary disease, and respiratory allergies in people who have those conditions. Based on both short-term and long-term effects, the Washington Department of Ecology declared diesel exhaust the air pollutant of greatest concern for public health in Washington.[8] The DEIS treats diesel exhaust particulate only as a risk factor for effects from long-term exposure.

22. Aldehyde compounds, especially formaldehyde and acrolein, are potent irritants of the eyes and upper respiratory tissues. Inhalation exposure to aldehydes causes wheeze, cough, and changes in lung function.

23. NO_2 exacerbates asthma, particularly in children. Hospital visits and admissions for asthma increase when short term NO_2 concentrations spike. Approximately one in ten people suffer from asthma in Clark County. Effects on asthma can occur at low exposure levels of NO_2 (i.e. below current regulatory levels) and studies have not found a level below which no effects occur. Dispersion modeling was applied in the DEIS to estimate air concentrations of NO_2 that

[8] Washington State Department of Ecology 2008. Washington Toxic Air Pollutant Priorities Study, available at http://www.ecy.wa.gov/biblio/0802030.pdf. Exhibit 5532-000268-CRK.

Earthjustice
705 Second Ave., Suite 203
Seattle, WA 98104-1711
(206) 343-7340

would be attributable to the Terminal, and while my confidence in these estimates is not high, the concentrations predicted are: 93ppb (parts per billion, a common measure of pollutant concentration) for the one hour exposure, and 25ppb for the annual exposure (constant, long term exposure averaged out over a year). These exposure estimates are well above the threshold for adverse effects on childhood asthma that was observed in a recent study from the Yale Center for Perinatal, Pediatric and Environmental Epidemiology. That study reported that children experienced worsened asthma symptoms and increased medication use in relation to increasing exposure to NO_2; effects were detectable for 5ppb increases at exposure levels as low as 6ppb[9]. It is not clear what increased levels of NOx will reach the Fruit Valley elementary school or the homes of the families who live nearest the Terminal, but it is reasonable to expect an impact on children with asthma in Fruit Valley due to NOx and other respiratory irritants emitted from vessels, trains, boilers and other sources associated with the Terminal.

24. Ozone is a powerful respiratory irritant. When ozone spikes on hot summer days, emergency room visits and hospital admissions also rise, for respiratory causes including asthma exacerbation and increased severity of chronic obstructive pulmonary disease (COPD) symptoms. Ozone events (here meaning substantial increases of ground level ozone concentration over average concentrations) could be triggered by Terminal emissions in the case of process upsets, leaks, or spills that release large amounts of VOC during a period when sunlight is strong; high ozone formation was observed after the Deepwater Horizon Spill due to evaporating oil[10]. While ozone levels that exceed the NAAQS are rare in Vancouver, the addition of a major VOC source will make them more likely. The Climate Impacts Group at the University of Washington predicts that ozone levels will increase in Washington due to increased

[9] Belanger K *et al*. 2013. Epidemiology 24(2): 320–330. doi:10.1097/EDE.0b013e318280e2ac

[10] Middlebrook *et al*. 2012. PNAS 109, vol 50. 20280–20285.

Earthjustice
705 Second Ave., Suite 203
Seattle, WA 98104-1711
(206) 343-7340

sunlight intensity from climate change[11].

B. Chronic health effects: shortened lifespan, cancer, and respiratory effects in children.

25. Chronic effects are those that are expected to occur in response to long term, continuous or repeated exposure to a toxicant. Average exposure concentration over a year (called the annual average) is a relevant metric for chronic health effects. The Terminal will likely increase premature mortality from cardiovascular causes, elevate cancer risk, and have adverse effects on children's respiratory health, among other possible chronic health effects. People chronically exposed to Terminal-caused pollution include residents of nearby neighborhoods and long-term employees of businesses located at and near the Port.

26. Chronic, long-term inhalation of particulate matter reduces life expectancy, similar to what has been observed with short-term increases of particulate matter. The strongest association is for cardiovascular causes of death. Early death attributable to chronic exposure to PM2.5 occurs at typical outdoor air concentrations, below regulatory "health protective" levels.[12] A10 ug/m3 increase in the annual average concentration of PM2.5 in outdoor air increases cardiovascular mortality by 11%[13]. The Terminal will increase PM2.5 annual average air concentrations, although expected exposure levels remain unclear.

27. In 2013, the International Agency for Research on Cancer ("IARC") declared that outdoor air pollution is carcinogenic to humans[14]. Outdoor air pollution from the Terminal

---

[11] 2015 State of Knowledge: Climate Change in Puget Sound, available at: https://cig.uw.edu/resources/special-reports/ps-sok/

[12] Thurston GD *et al*. 2016 Environmental Health Perspectives 124:484. http://dx.doi.org/10.1289/ehp.1509676. Reviewed in: Hoek *et al*. 2013 Environmental Health 12:43. Exhibit 5533-000015-CRK.

[13] Hoek et al, 2013; ibid.

[14] Available at: http://monographs.iarc.fr/ENG/Monographs/vol109/index.php

Earthjustice
705 Second Ave., Suite 203
Seattle, WA 98104-1711
(206) 343-7340

includes diesel exhaust particulate and fine particulate matter (PM2.5); both are known to cause lung cancer in humans after long term exposure. Long term exposure to carcinogens at any concentration causes some incremental increased risk of developing cancer. The exposure levels of diesel exhaust particulate the DEIS predicts for the residential neighborhood, if experienced for a lifetime, are associated with an increased cancer risk of 15-45 per million residents. This is likely a minimum, because other cancer causing substances were not accounted for and diesel exhaust particulate exposures are uncertain. Several specific compounds found in crude oil and ship, locomotive, and vehicle exhausts are carcinogenic. Arsenic, benzene, cadmium, hexavalent chromium, and 7,12dimethylbenz(a)anthracene are some of the carcinogens identified by Tesoro Savage. Benzene and 1,3-butadiene cause leukemia in humans. A cumulative assessment of additional cancer risk should be made that accounts for exposure to all project-related carcinogens.

28. The long-term respiratory health of exposed children may also be harmed. PM2.5 and NO_2 have been significantly linked to irreversible reduction in lung function development; children's lungs do not develop as well in neighborhoods with higher air pollution.[15] The converse is also seen, that incremental improvements in air pollution (e.g. a decrease in NOx of just 14ppb on average in one study) results in significantly better lung function growth in children.[16] Other recent evidence points to NOx as a cause of asthma development in children, distinct from the well-known effect of worsening symptoms in children already diagnosed with asthma. The combined effect of multiple air pollutants from increased locomotive and vessel traffic, as well as stationary sources at the Terminal will have an impact on children's respiratory

[15] Gauderman *et al*. N Engl J Med 2004; 351:1057 report results from a large study of children's health conducted by scientists at the University of Southern California. Exhibit 5534-000011-CRK.

[16] Gauderman *et al*. 2015 N Engl J Med 372:10:905. Exhibit 5535-000009-CRK.

Earthjustice
705 Second Ave., Suite 203
Seattle, WA 98104-1711
(206) 343-7340

health in the adjacent downwind areas.

V. ADVERSE HEALTH IMPACTS WILL BE DISPROPORTIONATELY BORNE BY PEOPLE WHO LIVE, WORK, OR ATTEND SCHOOL CLOSE TO THE FACILITY OR THE TRANSPORTATION CORRIDORS.

29. Several populations of concern can be identified for exposure to Terminal-caused air pollution. Among them are: employees and inmates of the Clark County Jail Work Center, employees of the Port of Vancouver, and residents of the nearby Fruit Valley neighborhood.

30. Because they are on-site around the clock, inmates of the Clark County Jail Work Center facility will be the population most highly exposed to Terminal air contaminants. While the DEIS argues that the short duration of inmate residence (18 days average) renders exposure to diesel exhaust of negligible consequence for inmates, that argument is valid only for the health effects associated with long term exposure (cancer, for example). Short term adverse effects on the respiratory and cardiovascular systems are still very much an issue. Jail Work Center staff members and workers at other Port businesses, including the Tesoro Savage Terminal itself, comprise another population of concern, because their place of daily work is located in the zone of greatest exposure. Depending upon employment duration, workers may be subject to both acute and chronic adverse health effects.

31. The small Fruit Valley neighborhood of Vancouver is bounded to the east and south by the rail lines that will be used by locomotives serving the Terminal. The Port, with industrial pollution sources, lies to the east. The Fruit Valley elementary school is approximately one mile from the prospective Terminal's storage tank area. Homes are as close as a few hundred meters from the rail lines and Port access road, key sources of NOx, particulate matter, and carbon monoxide pollution. Neighborhood residential exposures are a critical piece of the overall public health impacts of the Terminal because, while Port and Terminal workers may

Earthjustice
705 Second Ave., Suite 203
Seattle, WA 98104-1711
(206) 343-7340

receive higher exposures, residential populations typically include more sensitive subgroups. Children have greater sensitivity to respiratory irritants because of their smaller airways, developing lungs and immune systems, and higher breathing rates. The elderly and ill are also more sensitive to air pollution than an average worker population. Degradation of neighborhood air quality by the Terminal puts sensitive members of the community at greater risk for the acute and chronic health effects discussed in section IV and possibly health effects not discussed here. The DEIS (section 3.16.2.1) and a report generated by the USEPA Environmental Justice "EJSCREEN" application,[17] for the US census block group (530110410051), show a "meaningfully greater" proportion of neighborhood residents are Hispanic/Latino ethnicity and low income (<two times the poverty threshold). The median family income in the census block group is well below Washington state average. There are clear environmental justice[18] concerns that have not been addressed by Tesoro Savage. EFSEC needs to carefully consider the impacts of siting a large industrial facility, with planned toxic emissions and the possibility of catastrophic accident, adjacent to a neighborhood that already bears a heavy air pollution burden along with the health burdens and challenges of access to health care that are associated with low socioeconomic status.

VI. A CATASTROPHIC EVENT SUCH AS A SPILL OR FIRE WOULD HAVE SEVERE HEALTH CONSEQUENCES

32. This report is concerned with the potential impact of degraded air quality on human health that will arise from construction and operations of the Tesoro Savage Terminal.

---

[17] https://ejscreen.epa.gov/mapper/index.html?wherestr=45.6433843%2C-122.7077466. Report at Exhibit 5536-000003-CRK.

[18] **Environmental Justice** is defined by the United States Environmental Protection Agency as "the fair treatment and meaningful involvement of all people regardless of race, color, national origin, or income, with respect to the development, implementation, and enforcement of environmental laws, regulations, and policies."

Earthjustice
705 Second Ave., Suite 203
Seattle, WA 98104-1711
(206) 343-7340

However, I would be remiss not to address at least brief comment to the risk of accident and consequent severe health impacts. It is crucial for EFSEC to consider that large spills, explosions, or fires of crude oil would likely result in multiple fatalities and injuries, due not only to toxic smoke and vapor inhalation, but primarily to burns and other trauma. Injury to the population would be exacerbated in some scenarios by the limited ability of first responders to bring such a situation under control. Combined with days of serious air pollution hazards the overall impact on public health of the affected community could be quite substantial. There was little that I saw in the application and DEIS to address catastrophic scenarios, but in weighing the risks and costs of the proposed Terminal against the economic benefits, EFSEC must consider not only the public health effects of the intended operation, but public safety in the event of major accidents.

VII. SUMMARY

33. In summary, it is my opinion that construction and operation of the Tesoro Savage Terminal will worsen air quality, cause consequent health harms, and pose the possibility, however small, of a highly consequential accidental spill, explosion, or fire in a Washington community that is already heavily affected by air pollution due to the I-5 corridor, industrial activities, and residential wood burning. The air pollution impacts cannot be fully mitigated, despite use of best available control technologies and the many mitigation efforts that would be implemented by Tesoro Savage. There are adverse health effects that occur at any level of air pollution exposure; there is no threshold below which effects will not occur.

34. There is wide scientific consensus about the causal nature of the relationship of air pollution to mortality and low exposures at which the relationship holds. In setting standards for protection of public health the USEPA concluded that "a causal relationship exists between

Earthjustice
705 Second Ave., Suite 203
Seattle, WA 98104-1711
(206) 343-7340

short-term exposures to PM2.5 and mortality"[19]. A group of 15 top research scientists brought together by the American Heart Association stated: "PM2.5 concentration–cardiovascular risk relationships for both short- and long-term exposures appear to be monotonic, extending below 15 ug/m3 (the 2006 annual NAAQS level) without a discernable "safe" threshold."[20] Conversely, improvements in air quality, and this is especially clear for NOx and PM2.5, bring improved health outcomes in numerous populations studied.

35. While the precise and actual human exposure levels that will result from the Terminal are uncertain, it is certain that there will be some adverse human health impacts from the Terminal and associated operations as described. Acute respiratory effects are likely for workers and nearby residents during construction/decommissioning periods or periods of air stagnation. The inmates and employees of the Clark County Jail Work Center, and the employees of Port businesses including the Terminal itself, will receive the highest exposures. Workers are often healthy relative to children and elderly, whereas residents of the adjacent Fruit Valley neighborhood are of mixed age and presumably mixed health status. They will experience worsened air quality that is likely to affect sensitive people.

36. Impacts are not limited to the immediate vicinity of the Terminal. Communities along the rail and marine corridors will share the risks of accidents. Locomotive pollution along the corridor, and especially in the Pasco rail yards, will worsen NOx and PM2.5 contamination of local airsheds. Communities adjacent to the refineries that would receive the crude oil from the Terminal may have concerns for new toxic emissions, depending upon the nature of the crude oils received.

---

[19] U.S. EPA. 2009 Final Report: Integrated Science Assessment for Particulate Matter. U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-08/139F, 2009.

[20] Brook RD *et al*. 2010 Circulation 121:2331. Exhibit 5537-000049-CRK.

Earthjustice
705 Second Ave., Suite 203
Seattle, WA 98104-1711
(206) 343-7340

I declare under penalty of perjury that the foregoing is true and correct to the best of my knowledge.

Executed this 12 day of May, 2016, at Seattle, Washington.

ELINOR FANNING, Ph.D.

Earthjustice
705 Second Ave., Suite 203
Seattle, WA 98104-1711
(206) 343-7340

Elinor Fanning

2479 Soundview Drive NE • Bainbridge Island, WA 98110
Tel. 206-451-4141 / 206-643-6544 • elinorfanning@gmail.com

Environmental toxicology • Public health risks from toxics • Sustainability

CHARACTERISTICS

- Record of success in scientific research and public policy
- Experienced liaison between academic, government and NGO sectors
- Superb project management skills
- Accurate, precise, diligent, and persistent
- Strong critical thinker
- Ability to take initiative and work creatively
- Quality written and oral communications

WORK HISTORY

Assistant Director for Science. UCLA Center for Occupational and Environmental Health and UCLA Southern California Particle Center (2004-2009). Coordinated research programs in particulate air pollution science, developed grant proposals and technical reports, and prepared manuscripts. Led a program for educational exchange between occupational and environmental health scientists at UCLA and Mexican universities. Participated in workgroup that founded the UCLA Law and Environmental Health Sustainable Technology & Policy Program. Assistant to the chair of a California panel for the review of methyl iodide risk assessment.

Scientific Liaison. Scientific Review Panel for Toxic Air Contaminants, California Environmental Protection Agency. 2000-2003. Performed in-depth review of Cal EPA technical reports on pesticides and other pollutants under consideration for identification as toxic air contaminants. Researched background data in support of Panel review processes, acted as technical liaison between California EPA staff and Panel members, prepared official Scientific Review Panel findings documents.

Associate Toxicologist. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency (1999-2004). Areas of work: Public Health Goals for drinking water contaminants, prioritization of potentially carcinogenic chemicals for listing under California proposition 65, public health impacts of gasoline-derived pollutants.

EDUCATION

Ph.D. Environmental Health Science (1998). University of California at Berkeley School of Public Health

M.A. Molecular and Cell Biology (1992). University of California at Berkeley, Molecular and Cell Biology Department

B.A. Biology (1986). Oberlin College, Oberlin, OH

OTHER PROFICIENCIES

- French language, B2 level
- Spanish language, A1 level