

## **Potential Health Impacts of Gateway Pacific Terminal**

### **Whatcom and Skagit County Physicians Request a Comprehensive Health Impact Assessment (HIA) be Included in the EIS**

A direct impact of the proposed coal shipping terminal at Cherry Point would be eighteen or more 1.5 mile long trains traveling across the state and through our communities each day, and 400 or more ships traversing our waterways 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. As a group of local physicians, we are concerned about the health impacts of this proposal.

We believe the risks to human health from massive coal shipments across our state and through our communities are numerous and complex. We respectfully request a comprehensive **Health Impact Assessment (HIA)** addressing these issues along the entire rail and shipping corridor from the mines to the Pacific Ocean. **In addition, because the GPT proposal is not isolated, but is being considered along with multiple other ports with associated cumulative impacts, we request that a comprehensive HIA (to encompass all of the ports in the Pacific Northwest) be performed to best elucidate the impacts on human health.**

Further supporting documentation and EIS requests follow.

#### **I. Health Impacts of Diesel Particulate Matter (DPM)**

One of the largest potential health impacts of the Gateway Pacific Terminal lies in the increase in air pollution resulting from diesel locomotive emissions all along the transportation corridor, from the Powder River Basin to Cherry Point, and the diesel emissions from the Cape Class ships transporting the coal through Puget Sound waterways.

The effects of air pollution are not hypothetical, but real and measurable. Many studies, some of which were conducted in the Seattle area, show significant health effects of exposure to everyday airborne pollutant levels, even when they 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.”

Puget Sound is in particular danger from diesel air pollution. A recent study from the National-Scale Air Toxics Assessment released by the EPA states that “the Puget Sound region ranks in the country’s top five percent of risk for exposure to toxic air pollution.” A study in 2010 by the Puget Sound Clean Air Agency and the University of Washington showed that, “Diesel emissions remain the largest contributor to potential cancer risk in the Puget Sound area.”

Diesel *particulate* emissions are of special concern, particularly the size fraction up to 2.5 microns, known as PM<sub>2.5</sub>. This size of particle is able to be respired deep into the lungs. PM<sub>2.5</sub> 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 of particular concern as sources of particulate matter, as they typically produce PM<sub>2.5</sub> at a rate about 20-times greater than from gasoline engines.

#### Health Impacts of DPM: 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 comorbidities such as smoking (Bofetta, 2001). The impact of DPM on cancer risk must be considered in the decision making process for the GPT.

#### Health Impacts of DPM: Cardiac and Pulmonary

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 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/m<sup>3</sup> 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/m<sup>3</sup> increase in PM<sub>2.5</sub> 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<sub>2.5</sub> 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/m<sup>3</sup> increase in PM<sub>2.5</sub> was associated with a 76% increase in death due to cardiovascular disease. To further highlight the impact of PM<sub>2.5</sub> on public health, the 'Global Burden of Disease' report recently published in *Lancet* reported ambient PM<sub>2.5</sub> as the #9 cause of disease world-wide, 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<sub>2.5</sub> 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<sub>2.5</sub> 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 a 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<sub>2.5</sub> 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 1 hr. Additional work utilizing controlled diesel exhaust exposures to human volunteers has revealed that these acute exposures results in an impairment in blood vessel function and alters 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<sub>2.5</sub> 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<sub>2.5</sub> exposure has been investigated. Research published in the *Archives of Internal Medicine* (2012) examines, for the first time, the risk of acute, short term exposures to PM<sub>2.5</sub> as a key factor in triggering stroke, often within hours of exposure. The study found a linear relationship between PM<sub>2.5</sub> 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 PM<sub>2.5</sub> level was on the higher range of "moderate" exposures (15-40 ug/m<sup>3</sup>), as opposed to days when pollutants are lower than 15 ug/m<sup>3</sup>. 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 (Norris et al, 1999; Slaughter et al, 2003). 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 (Brandt et al, 2002). Thus, it is emphasized that additional DPM exposure adds to an existing problem.

#### Health Impacts of DPM: 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 will be important to study 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 considered to be 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 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.

**Vulnerable groups who are especially at risk from air pollution include children, pregnant women, and the elderly.**

#### Recommendations

It is incumbent upon the decision makers in this process to apply the *best available science* in determining the health impacts of the GPT. 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, and the US EPA Integrated Risk Information System, 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 GPT.** (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 recently 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.

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 GPT will occur to populations in close proximity to the tracks, terminal, and shipping lanes. **Thus, we recommend that the near source health effects be quantified spatially all along the transportation corridor, not just for the terminal site. This will necessarily include the railway corridor, as well as the emissions from marine vessels.**

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 consultants familiar with the models and with interpreting the results of the models.

If mitigation measures, pollution control devices, ultra low sulfur fuel specifications, or late model diesel locomotive emission factors are used in the emissions estimates and models, those assumptions should be listed as mitigation required in the Draft and Final EIS.

The Puget Sound area is prone to 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.**

### Summary

A direct result of the Gateway Pacific Terminal will be a substantial increase in airborne pollutant emissions from train and marine traffic from the Powder River Basin, all through the rail transportation corridor, at the terminal site, and Puget Sound. If GPT is not built, these impacts will not occur. Thus, the impacts must be quantified through the entire region impacted by this activity, not just at the terminal site, as has been advocated by SSA Marine.

**Because of the health impacts that will be a direct result of the GPT terminal, we respectfully request that the EIS include a Health Impact Assessment that addresses the following questions:**

1. How much DPM and toxins (detailed above) will people be exposed to at 50 feet, 100 ft, 200 ft, etc up to 2 miles from the tracks when a train goes by? We request

this data to be shown in an easy-to-understand format, including maps with "pollution contours" (isopleths).

2. What neighborhoods will be exposed to even greater DPM and toxins due to trains idling on sidings, both existing and future (a study by Communitywise indicates an additional siding in Bellingham is likely)? How much DPM and toxins will these areas be exposed to?
3. How much DPM and toxins (detailed above) will result from the ships, including ships that are at anchor (staging), at the dock, or in transit?
4. What will the impact of temperature inversion weather conditions be on air pollutants? How high may the concentrations get?
5. How many people live within 50 ft, 100 ft, 200 ft, 500 ft, 1000 ft, 1 mile, and 2 miles along the entire transportation route from Powder River Basin to Cherry Point to the Strait of Juan de Fuca, including current and projected populations?
6. How many of the people living, going to school, or working within the distances above are children, including current and projected populations? Elderly? Have any form of pulmonary or cardiovascular disease?
7. How many increased asthma attacks, ER visits, and hospitalizations will result, including current and projected populations, and including under temperature inversion conditions?
8. How many increased strokes will result, including current and projected populations, and including under temperature inversion conditions?
9. How many increased myocardial infarctions (heart attacks) will result, including current and projected populations, and including under temperature inversion conditions?
10. How many COPD exacerbations will result, including current and projected populations, and including under temperature inversion conditions?
11. How much cancer will result, including current and projected populations?
12. How much acrolein, acetaldehyde, formaldehyde, heavy metals (including but not limited to mercury, lead, and arsenic), 1,3-Butadiene, polycyclic aromatic hydrocarbons, or other toxins will be deposited cumulatively? This should be analyzed in a cumulative fashion, (i.e. additive) over the next 50 years (the operating life of the terminal).
13. What are the effects of chronic exposure of the above compounds on: Neonatal and childhood development? Blood and lymphatic systems? Respiratory system? Cardiovascular system? Reproduction? Cancer?
14. What is the cost of cleanup of the cumulative environmental contaminants? How effective is the cleanup? Who pays the cost?
15. What is the economic cost of all of the health impacts combined? Who pays for the costs?
16. Medical research comes forth at an intense pace. When new health impacts of diesel particulate matter are inevitably identified or quantified, how can the public be assured that their health will be weighed in the balance of ongoing risks/benefits of GPT operations?

## **II. Health Impacts of 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. Large amounts of coal dust also escapes from coal piles, as can be seen in photographs of the West Shore Terminal at Roberts Bank. 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).

### Health Impacts of Coal Dust and Combustion: 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, boron, and heavy metals such as lead, 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) and a larger percentage of the mercury in Lake Whatcom originates from coal burned in China.

### Health Impacts of Coal Dust: Airborne Dust

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. Cancer rates three times higher than average have been reported at one of Australia's largest coal ports (Ockenden, Will, 2012). Health risks of airborne coal dust to coal miners have been well documented to cause lung disease, ranging from severe pneumonconiosis to chronic bronchitis and exacerbations of asthma (Hathaway, et al. 1991).

While pneumoconiosis has only been conclusively associated with intense exposure in miners, 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).

It is emphasized that children are not "little adults" and are significantly more vulnerable to the health effects of environmental contaminants. Children eat more, breath 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 (including the brain and nervous system) are also more vulnerable to adverse effects.

**Because airborne coal dust exposure and environmental contamination is a direct impact of GPT, we respectfully request that the EIS include a Health Impact Assessment that would address the following questions:**

1. How much coal dust from the mining and transportation of coal can be expected along each section of the rail corridor from the Powder River Basin to the proposed terminal?
2. How much coal is lost from residual dust still on the cars as they leave the coal terminal after unloading (so called "carryback coal")? How much of the "carryback coal" is expect to be lost in Whatcom County in particular?
3. How much accumulation will result after 50 years of transport (the operating life of the terminal)?
4. How many coal train derailments can be expected along the rail corridor per year of operation of the proposed export terminal?
5. What will be the effect of contamination from coal dust and spills on farm land along the rail corridor?
6. What will be the effect of contamination from coal dust and spills on grazing animals used for human consumption?
7. What will be the effect of contamination from coal dust and spills on fresh water supplies for humans and animals?
8. What will be the effect of contamination from coal dust and spills on marine habitat for fish and other seafood?

9. How many people can be expected to be affected by the increased exposure to mercury and other heavy metal contaminants of coal, such as cancer, including current and projected populations?
10. How many children and adults can be expected to have increased risk of asthma and other respiratory diseases, including current and projected populations?
11. What health and safety impacts may be present at the coal port itself, including increased rates of cancer that have been reported at a large coal port?
12. What is the economic cost of these health impacts? Who pays for the costs?
13. What is the cost of cleanup of the cumulative environmental contamination? How effective is the cleanup? Who pays for the cost?
14. Medical research comes forth at an intense pace. When new health impacts of coal dust and combustion are inevitably identified or quantified, how can the public be assured that their health will be weighed in the balance of ongoing risks/benefits of GPT operations?

### **III. Health Impacts of 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:

#### Health Impacts of Noise: 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. 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)

#### Health Impacts of Noise: 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)

### Health Impacts of Noise: 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. **Hundreds of thousands of people along the transportation route will likely experience sleep disruption multiple times through the night as a direct result of GPT.** (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)

### Health Impacts of Noise: 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).

Coal trains produce significantly greater noise and vibration than other trains: longer trains means 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, and simply based on the noise and vibration they experience. **Thus, evaluation of the noise impact of GPT must account for the fact that these would be coal trains and not passenger or conventional freight trains.**

A person woken from sleep every hour—as would be expected when the GPT terminal is at full operation—represents a different order of magnitude of adverse health impacts than a person woken or otherwise disturbed once or twice a night from existing train traffic. The train traffic directly impacts multiple dense residential areas along the entire rail line.

**Because of the health impacts that will be a direct result of the GPT terminal, we respectfully request that the EIS include a Health Impact Assessment that addresses the following questions:**

1. How loud are train engines? Squeaking wheels? Whistle blasts? How loud it this 50 feet, 100 ft, 200 ft, etc up to 2 miles from the tracks? We request this data to be shown in an easy-to-understand format, including maps with "sound contours" (noise isopleths).
2. How much vibration does a coal train produce? How intense is this at 50 feet, 100 ft, 200 ft, etc up to 2 miles from the tracks?
3. How many people live within 50 ft, 100 ft, 200 ft, 500 ft, 1000 ft, 1 mile, and 2 miles along the entire route from PRB to Cherry Point?
4. How much noise and/or vibration wakes an average person? A light sleeper?
5. How much noise or vibration distracts a working person? A concentrating student?
6. For each train along the entire route, how many crossings are there? How many whistle blasts per crossing? How many whistle blasts in total for a single train traveling from Montana to Cherry Point? How many whistle blasts per day in all (x 18 trains)? How many of these are at night during sleeping hours (8 PM to 8 AM)?
7. For each train, including engine noise, vibration, squealing wheels, and whistle blasts, how many people will be awakened, based on current and projected populations? How many children? How many adults? How many elderly? All calculations must include projected populations as well, since the terminal has an operating span of 50 years.
8. How many times per night will a person be awakened, from noise or vibration, who lives various distances from the tracks (including distances: 50 ft, 100 ft, 250 ft, 500 ft, 1000 ft, 0.5 miles, 1 miles, and 2 miles) in all areas and communities along the route, including Helena, Missoula, Spokane, Olympia, Tacoma, Seattle, Everett, Shoreline, Mt. Vernon, Bellingham, and all areas between?
9. How many awakenings per night, including all people along the entire route up to 2 miles away from tracks, including all trains, based on current and projected populations?
10. Considering the noise and vibration, multiple awakenings and resultant fatigue, how many people may potentially have increased blood pressure, or elevated stress hormones, including current and projected populations?
11. What is the total economic cost of increased blood pressure, elevated stress hormones?
12. Considering the noise and vibration, multiple awakenings and resultant fatigue, how many arrhythmias, or heart attacks could potentially result from the increased noise, including current and projected populations? What is the total economic cost of the arrhythmias, or heart attacks?
13. Considering the noise and vibration, multiple awakenings and resultant fatigue, how many strokes could potentially result from the increased noise, including current and projected populations? What is the total economic cost of the strokes?
14. Considering the noise and vibration, multiple awakenings and resultant fatigue, how much increased mental disease may result from associated stress, including but not limited to: depression, mental instability, neurosis, hysteria, and psychosis, including current and projected populations? What is the potential economic cost of the increased mental disease?

15. What is the potential impact of noise, vibration, multiple awakenings, and fatigue on childhood learning? On childhood test scores? What is the total economic cost of the learning impairment?
16. What is the potential impact of noise, vibration, multiple awakenings, and fatigue on workplace performance and safety? What is the total economic cost of the impaired workplace performance and safety?
17. How many increased traffic accidents may result from fatigue- associated sleep disturbance, including current and projected populations? What is the total economic cost of the accidents? Cost in terms of human morbidity?
18. Who pays for the economic costs of the impacts listed above?
19. Medical research comes forth at an intense pace. When new health impacts of noise are inevitably identified or quantified, how can the public be assured that their health will be weighed in the balance of ongoing risks/benefits of GPT operations?

#### **IV. Health Impacts of Delays in Emergency Medical Services**

As physicians, we are concerned that 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, and we request a full health impact assessment of this issue along the entire rail corridor across the state as part of the environmental impact statement.

##### Health Impacts of Rail Crossings: EMS Delays

For many of our most common acute health issues, such as stroke, heart attack, massive hemorrhage, and trauma, every second counts, and 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.

We are aware of a number of locations in Whatcom County 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. **It should be considered that an ambulance often must cross any 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. Indeed, a study conducted by Gibson Traffic Consultants indicates that the rail traffic may cause severe impediment to EMS access ([www.communitywisebellingham.org](http://www.communitywisebellingham.org)).

#### Health Impacts of Rail Crossings: Accidents

Finally, we are concerned that increased rail traffic of the magnitude that is currently proposed has significant potential for increased traumatic injury and death at rail crossings or by derailments. Many crossings in the city of Bellingham and in Whatcom County 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 have also been at least nineteen coal train derailments in North America in 2012, including fatalities.

**Because increased frequency of very long trains at rail crossings will be a direct result of the GPT terminal, we respectfully request that the EIS include a Health Impact Assessment that addresses the following questions:**

1. How many rail crossings are there along the rail corridor from the Powder River Basin to Cherry Point?
2. How many of these rail crossings are unprotected?
3. What are the costs to provide protective barriers at these crossings and who will bear these costs?
4. How often and for how long will these crossings be blocked by the increased rail traffic en route to GPT? Delay should be calculated for each crossing to account for differences in local circumstances.
5. How many times daily do EMS vehicles, including police, fire and medic units, cross rail lines? Please note that an ambulance needs to cross twice to transport a patient to a hospital.
6. What will be the cumulative and per incident delay in access to these services caused by rail traffic en route to GPT (including actual blockage of the crossing, as well as alleviation of resultant congestion)? Please again note that an ambulance generally needs to cross twice to transport a patient to a hospital.
7. How many people are affected at each crossing, based on current and projected populations as shown in relevant planning documents?
8. What crossings and locations are most likely to result in significant delays at crossings?
9. How often are there alternative crossings? How much time is lost to route through alternate crossings, rather than the shortest route?
10. Is there any current established system to alert EMS vehicles of impending crossing closures?
11. How much would such a system cost and who would bear the cost of developing such systems?

12. How does backed up traffic at crossings and the dispersion of that traffic effect EMS response times?
13. How often and to what severity will these delays in EMS response times lead to delays in care and to otherwise avoidable outcomes such as death or permanent disability?
14. What is the amount of healthcare cost attributable to patients receiving delayed EMS services as a result of increased rail traffic?
15. How will the project applicant mitigate these impacts (grade separation at crossings, construction of new hospitals, support for additional paramedics, medivac services, etc.?)
16. How many rail crossing accidents, injuries, and deaths will be attributable to increased rail traffic en route to GPT?
17. What is the anticipated cost of these accidents, including anticipated litigation and long term care costs?
18. How many coal train derailments would be anticipated to occur across the state of Washington over time, given that there have been nineteen in 2012 alone in the US and Canada?
19. Where are the likely sites of these derailments, and are any of these potentially dangerous or inadequately designed rail lines in major population densities?

We thank you for your attention to thorough evaluation and full disclosure of the potential health impacts of GPT.

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## References

Additional information listed in "Whatcom Docs Position Statement" and appendices on [coaltrainfacts.org](http://coaltrainfacts.org).

### **I. Diesel Particulate Matter 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. *Eviron Health Perspec.* 110 (2):187-.

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. *N Engl J Med* **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 / Sante Fe Railroad Spokane Railyard. Spokane Regional Clean Air Agency, [www.spokanecleanair.org](http://www.spokanecleanair.org)

Thaller, E. et al. 2008. Moderate increases in ambient PM<sub>2.5</sub> 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.

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.

Wellenius, G.A. et al. 2005. Air pollution and hospital admissions for ischemic and hemorrhagic stroke among Medicare beneficiaries. *Stroke* 36:2549-2553.

## **II. 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<sup>rd</sup> Edition. New York, NY: Van Nostrand Reinhold.

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.

Ockenden, W. 2012. <http://au.finance.yahoo.com/news/report-finds-cancer-risk-coal-043612330.html>

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.

### **III. 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. 1949. 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.