Potential Health Impacts of Norfolk Southern's PVCP

On Behalf of Rail Pollution Protection Pittsburgh, The Undersigned Physicians And Health Care Professionals Request That A Comprehensive Health Impact Assessment (HIA) Be Contracted By PennDOT In Connection With Norfolk Southern's Pittsburgh Vertical Clearance Project (PVCP)

Background and Synopsis

Norfolk Southern has been awarded $20 million in taxpayer funding to create clearance by raising a number of vehicular and rail bridges to accommodate double stacked trains on its Ft. Wayne and Pennsylvania rail lines between McKees Rocks and East Braddock. Norfolk Southern justifies the project on the ground that it will result in a three hour transit time cost savings/train over the current Mon Line route around the city, and because the Mon Line service has been interrupted by landslides. (The last significant landslide on that route was in 2014.) According to Norfolk Southern, the project will create a safer, more reliable and environmentally beneficial transportation network for both NSRC and the surrounding communities.

A direct impact of Norfolk Southern's proposed PVCP would be to almost quadruple the number of trains per day traveling along a 20 mile corridor through the County's most densely populated neighborhoods. According to the latest census figures, there are 175,000 individuals (72% of which live in Environmental Justice Areas) living in the combined derailment/ high risk black carbon diesel pollution fallout impact and blast zone. While the advantages of hauling freight by rail over transport by truck are acknowledged, the exhaust from increased rail traffic along the proposed modified route is concentrated in a very small densely populated area. Moreover, running higher center of gravity less stable double stacked trains adjacent to extremely volatile oil trains along a zig zag route containing fourteen nearly 90 degree turns (3 of which are within 1 ½ mile of each other) presents a risk to health and safety in the event of a derailment. Even without a derailment, increasing rail traffic will result in increased airborne pollutants from diesel engines and coal dust. Running the Fort Wayne and Pittsburgh train lines at full capacity will result in increased idling of lower priority trains, and in increased noise pollution. Raising vehicular bridges will also cause increased risk of vehicle and pedestrian injuries. As a group of local health care professionals, we are concerned about the health impacts of this project.

Diesel engine trains emit a variety of air pollutants known to adversely affect human health, including volatile organic compounds, carbon monoxide, and nitrogen dioxide. Particulate matter (PM) is of particular concern as Allegheny County has received a failing F grade rating from the American Lung Association for daily and long-term PM levels in 2018.The Pittsburgh region is usually in the top 10 worst U.S. cities for air quality. We are in the worst 2% of the country for cancers caused by air pollution-- and one of the deadliest areas in the nation for asthma and related illnesses. According to a

collaborative report from the American Thoracic Society and New York University's Marron Institute for Urban Management, the Pittsburgh region had the fourth most air-pollution related deaths of any metro area in the country. In 2017, the Pittsburgh region, which includes Allegheny County, all of its bordering counties, and Fayette County, suffered 232 deaths related to air pollution, according to the study's estimates. This was the most of any region outside of California. The Los Angeles metro area, with a population of 13 million, led the nation in estimated air-pollution deaths at 1,322. But LA had almost the identical amount of air-pollutions deaths per-capita as Pittsburgh, at about one for every 10,000 residents.

Given that residents of Allegheny County are currently breathing some of the worst air in the country, we believe the proposed PVCP presents numerous and complex additional risks to human health. According to Erica Jackson, Predoctoral Fellow, and James Fabisak, Ph.D., Associate Professor Center for Healthy Environments and Communities, School of Public Health, University of Pittsburgh, this is especially the case for people living along Norfolk Southern's Pittsburgh Line since they already reside in highest quartile zones of traffic–related pollution, based on exposure maps developed by Albert Presto at CMU. According to the Jackson/Fabisak formula, it is conservatively estimated that increasing the number of trains on the modified route from twenty to twenty-five/day to a full capacity of 80/day is equivalent to running 5,440 diesel busses past those residents door step every day.

About 20% of the mobile source emissions of diesel-derived primary $PM_{2.5}$ (particulate matter less than or equal to 2.5 micrometers in diameter) in Allegheny County currently comes from rail traffic, according to EPAs 2014 Emission Inventory. Diesel particulate matter (DPM) appears to be the largest single air pollutant driving cancer risk in Allegheny County. Trains also emit carbon dioxide, a greenhouse gas that contributes to climate change.

Carnegie Mellon University Professors Karen Clay, Akshaya Jha, Nicholas Muller and Randall Walsh have determined that the public health costs from constant locomotive emissions are three times more harmful than impacts from an accident or spill. The CMU analysts have further determined that for every ten trains, there will be 6 pollution related deaths/year. It was similarly determined that for every hour that a train idles, there will be one pollution related death/year. Even without an increase in rail traffic, trains have idled for four days and nights in Allegheny Commons.

Additional risks could present themselves since these rail lines are carrying hazardous materials, such as industrial chemicals, ethanol, or petroleum. If a train were to derail or experience an accident along the Pittsburgh Line, it would do so in a densely-populated area with Pittsburgh's most critical infrastructure nearby posing a significant public health threat. Noise pollution, traffic, and stress on existing infrastructure (including sewage and water lines) are also possible adverse effects which could have negative health impacts.

Therefore, we the undersigned respectfully request that PennDOT commission a comprehensive Health Impact Assessment (HIA) addressing these issues along the entire proposed modified rail corridor from McKees Rocks to East Braddock.

Further documentation and references in support of the HIA request that appear below come from a division of Washington State Physicians for Social Responsibility HIA request which was signed by over 200 health professionals. Their document was updated by Rail Pollution Protection Pittsburgh to be made applicable to Norfolk Southern Railway's Pittsburgh Vertical Clearance Project.

I. Health Impacts of Diesel Particulate Matter (DPM)

One of the largest potential health impacts of the PVCP lies in the increase in air pollution resulting from diesel locomotive emissions all along the proposed modified transportation corridor.

The effects of air pollution are not hypothetical, but real and measurable. Many studies show significant health effects of exposure to everyday airborne pollutant levels, 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 previously 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."

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

<u>Health Impacts of DPM: Cancer</u>

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 PVCP.

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/m3 increase in DPM was associated with a 6% increase in cardiopulmonary mortality. In a follow-up to this study, Pope et al (2004) demonstrated that their previously observed increase in cardiopulmonary mortality was largely driven by increases in cardiovascular, as opposed to pulmonary mortality. In this follow-up study, a 10 ug/m3 increase in PM2.5 was associated with a 12% increase in mortality due to 'all cardiovascular disease plus diabetes' and an 18% increase in mortality due to 'ischemic heart disease'. Further epidemiological investigations have revealed that these estimates are likely largely underestimating the effect of PM2.5 due to inadequate exposure characterization. Published in the *New England Journal of Medicine*, Miller et al. (2007) utilized a novel exposure characterization method and reported from the Women's Health Study that a 10 ug/m3 increase in PM2.5 was associated with a 76% increase in death due to cardiovascular disease. To further highlight the impact of PM2.5 on public health, the 'Global Burden of Disease' report recently published in *Lancet* reported ambient PM2.5 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 PM2.5 source influences health risks. Due to research led by those at the University of Washington, it is becoming clearer that an individual's exposure to PM2.5 is dependent on where he/she lives and works and that this strongly influences health outcomes. Van Hee et al. (2009) demonstrated that living close to a major roadway was 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 PM2.5 impairs how well blood vessels dilate and how well the heart functions, providing a basis for our understanding of previously observed increases in mortality (Van Hee et al. 2011, Krishnan et al. 2012).

There are very specific physiological effects with DPM exposure. A 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 PM2.5 result in an increased risk of heart attack (Peters et al. 2001).

In addition to cardiovascular risk, cerebrovascular effects and risk of stroke associated with PM2.5 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 PM2.5 as a key factor in triggering stroke, often within hours of exposure. The study found a linear relationship between PM2.5 level and stroke risk even when the exposure was well below the EPA daily exposure limit. Overall, the risk of ischemic stroke was 34 % higher on days when the PM2.5 level was on the higher range of "moderate" exposures (15-40 ug/m3), as opposed to days when pollutants are lower than 15 ug/m3. This is an unprecedented finding, and points to the acute danger of even short term exposures to levels of particulate pollution previously thought "safe."

Studies conducted at Seattle Children's Hospital show that air pollution leads to asthma exacerbations, increased ER visits, and increased hospitalization, at levels that currently exist in Seattle (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 PVCP. 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 PVCP.** (See health risk assessment guidance from California's Office of Environmental Health and Hazard Assessment at http://www.oehha.ca.gov/pdf/HRSguide2001.pdf)

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

The highest exposure risks of DPM from the PVCP will occur to populations in close proximity to the tracks. **Thus, we recommend that the near source health effects be quantified spatially all along the transportation corridor.**

Modeling should use 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 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 required mitigation.

The PVCP 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 PVCP there will be a substantial increase in airborne pollutant emissions from train traffic all through the rail transportation corridor. If PVCP is not approved, these impacts will not occur. Thus, the impacts must be quantified through the entire region impacted by this activity.

Because of the health impacts that will be a direct result of the PVCP, we respectfully request that a Health Impact Assessment be performed that addresses the following questions:

1. How much DPM and toxins (detailed above) will people be exposed to at 50 feet, 100 feet, 200 feet, 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 sidings to be built? How much DPM and toxins will these areas be exposed to?
3. What will the impact of temperature inversion weather conditions be on air

pollutants? How high may the concentrations get?

4. How many people live within 50 feet, 100 feet, 200 feet, 500 feet, 1000 feet, 1 mile, and 2 miles along the entire transportation route from McKees Rocks to East Braddock, including current and projected populations?
5. 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?
6. How many increased asthma attacks, ER visits, and hospitalizations will result, including current and projected populations, and including under temperature inversion conditions?
7. How many increased strokes will result, including current and projected populations, and including under temperature inversion conditions?
8. How many increased myocardial infarctions (heart attacks) will result, including current and projected populations, and including under temperature inversion conditions?
9. How many COPD exacerbations will result, including current and projected populations, and including under temperature inversion conditions?
10. How much cancer will result, including current and projected populations?
11. 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.
12. 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?
13. What is the cost of cleanup of the cumulative environmental contaminants? How effective is the cleanup? Who pays the cost?
14. What is the economic cost of all of the health impacts combined? Who pays for the costs?
15. What are the locations and duration of idling along the proposed route over the past two years, and what is the projected amount of idling that will occur once all lines are running at full capacity?
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 PVCP operations?

II. Health Impacts of Coal Dust

Running uncovered coal cars adjacent to double stacked rail cars will result in additional escape 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 run on Norfolk Southern lines through Pittsburgh, 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. Coal dust can be a costly pollutant requiring frequent cleaning for businesses and residences along a rail line,

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 and then across the United States. Fourteen percent of the mercury in the Great Lakes originates in China (National Oceanic and Atmospheric Administration, 2011).

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. 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 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 the PVCP, we respectfully request that a Health Impact Assessment address the following questions:

1. How much coal dust from the transportation of coal can be expected along each section of the rail corridor from McKees Rocks to East Braddock?
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 Allegheny County in particular?
3. How much accumulation will result after 50 years of transport?
4. How many coal train derailments can be expected along the rail corridor per year of operation?
5. What will be the effect of contamination from coal dust and spills on the land along the rail corridor?
6. What will be the effect of contamination from coal dust and spills on fresh water supplies for humans and animals?
7. What will be the effect of contamination from coal dust and spills on river habitat?
8. 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?
9. How many children and adults can be expected to have increased risk of asthma and other respiratory diseases, including current and projected populations?
10. What is the economic cost of these health impacts? Who pays for the costs?
11. What is the cost of cleanup of the cumulative environmental contamination? How effective is the cleanup? Who pays for the cost?
12. 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 P V C P 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:

<u>Health Impacts of Noise: Cardiovascular Disease</u>

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 MMetal #2, 2001; Hygge S et al. 2002; Stansfeld SA at el. 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 the PVCP.** (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).

Heavier 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. **Thus, evaluation of the noise impact of the PVCP must account for the fact that these rail lines will be run at full capacity 24/7 through the most densely populated segments of the County.**

A person awakened from sleep every hour—as would be expected when the PVCP is at full operation—represents a different order of magnitude of adverse health impacts than a person awakened or otherwise disturbed once or twice a night from existing train traffic.

Because of the health impacts that will be a direct result of the PVCP, we respectfully request that a Health Impact Assessment address the following questions:

1. How loud are train engines? Squeaking wheels? Whistle blasts? How loud it this 50 feet, 100 feet, 200 feet, 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 do trains running simultaneously on all four tracks produce? How intense is this at 50 feet, 100 feet, 200 feet, etc., up to 2 miles from the tracks?
3. How many people live within 50 feet, 100 feet, 200 feet, 500 feet, 1000 feet, 1 mile, and 2 miles along the entire route?
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 the entire proposed modified route? How many whistle blasts per day in all (x 8 0 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.
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 feet, 100 feet, 250 feet, 500 feet, 1000 feet, 0.5 miles, 1 miles, and 2 miles) in all areas and communities along the route, 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 arrythmias, or heart attacks could potentially result from the increased noise, including current and projected populations? What is the total economic cost of the arrythmias, 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 PVCP operations?

IV. Health Impacts of Derailments and Reduced Sight Lines Resulting from Raising Vehicular Bridges

A search of the Federal Rail Administration Office of Safety Data by state reveals that in Pennsylvania between January 1, 2015 and December 31, 2018, there have been 197 derailments overall- and that 131 of those derailments involved Norfolk Southern trains.

On May 21, 2018, Norfolk Southern filed a breach of contract federal court lawsuit against Boatright Rail Products. In the facts, Norfolk Southern's lawyers pled the following:

- Norfolk Southern told the court that it required its railroad ties to consist of "methods and specifications for the proper application of preservatives, including treatment . . . with specified amounts of creosote."
- In order to increase its profit margins, Boatright ordered their employees to simply "make [the railroad ties] black."
- Boatright employees used a variety of substances, including motor oil, anti-freeze, and paint, that uniformly lacked preservative characteristics, but darkened the appearance of the railroad ties to resemble properly treated wood.
- This scheme lasted for nearly five years, from 2009 until 2014.
- In total, Norfolk Southern ordered and installed nearly five million railroad ties manufactured by Boatright that failed to meet contractual standards.
- Lastly, Boatright failed to include identifying plates or Kerf marks on its railroad ties, making it more difficult for Norfolk Southern to identify the noncompliant ties.

- Norfolk Southern pled that the "*inherently hazardous nature* of the products necessitates their removal."

Stated plainly, Norfolk Southern has admitted installing 5 million rail ties under its tracks that, in their own words are “inherently dangerous” because they were not waterproofed, are prone to more quickly rot, and which cannot be easily identified. The full statement of the case is linked here: https://www.leagle.com/decision/infdco20180522527

Norfolk Southern currently transports daily:

- Petrochemicals- including explosive propane
- Silica Fracking Sand
- Human Waste
- Radioactive Waste
- Oil and Gas (The WSJ calculated that one rail car of crude carries the energy equivalent of 2 million sticks of dynamite)
- Chlorine (rail cars contain 180,000 pounds of chlorine gas released fully in 10 minutes when ruptured)
- Hydrochloric Acid
- Androgynous Ammonia

In March, 2019, “60 Minutes” questioned the safety of railroads operating without Positive Train Controls (PTC) as a result of locomotive speeding, human error and inattention, and switching malfunctions.. Watch the video here: https://cbsn.ws/2HjqtGe

Norfolk Southern has requested extensions on the installation of PTC—which is currently not operational on either its Ft. Wayne or Pittsburgh Lines.

On April 10, 2019, as a result of pressure by rail lobbyists, President Trump signed an Executive Order mandating regulations be in place to allow the transportation of Liquid Natural Gas by rail. This was done even though the Federal Rail Administration has not completed studies to determine if this can be done safely. Protank is awaiting FRA approval to carry oil and gas in double stacked by rail containers that can also be stacked on marine vessels without the need for loading and off-loading the contents

While Norfolk Southern has run double stacked trains adjacent to volatile oil trains elsewhere, the modified Pittsburgh route presents unique challenges and causes for concern. In addition to the large number of unusually sharp turns, rail roads are more than doubling the length of their trains. Next, the utility lines- particularly water and sewer lines under and adjacent to the tracks are antique—between 100-150 year old handmade brick whose mortar has degraded because of the acid in the rain, and subject to collapsing given the total vibrations of running all four tracks to capacity. Additionally, high rainfall can overwhelm the current drainage systems causing the rails to go out of alignment. Finally, there is the issue of whether rail bridges and tracks are being properly maintained and inspected. For example, Norfolk Southern had inspected the rails just days before the derailment on the Southside last August, 2018. A properly conducted inspection, with properly maintained, calibrated and certified will

catch track failure before it causes a derailment.

According to calculations performed by the University of Pittsburgh's School of Public Health, Center for Healthy Environments and Communities, using the 2017 American Community Survey, there are 31 communities directly impacted by the PVCP. The one mile blast zone for derailments involving more than one rail car fire contains the following infrastructure: 124 public/private schools with 75,444 students; 77 bridges; 6 nursing homes with a 919 bed count; 6 hospitals; 108 parks (not counting municipal parks); 3 stadiums; the Convention Center; our three major universities, numerous museums containing protected National Treasure collections; the National Aviary and Pittsburgh Zoo (containing protected species covered by the Endangered Species Act), and multiple power and switching station.

Norfolk Southern utilizes 19th century design mechanical hand brakes (parking brake) and air brakes--which require the locomotive to idle even when stopped to run the compressor. These are the same brakes that were responsible for the 2013 catastrophe in Lac-Megantic, Canada. As reported in the July 16, 2017 NYT article "A Runaway Train Explosion," in 2017 in Canada, there were still 62 trains that "took off on their own." Safer electronic brakes are available for freight trains. Electronic brakes activate all of the brakes on all cars at once and do not require idling the locomotive. Air brakes engage one car at a time down the line to the rear of the train. Norfolk Southern chooses not to invest in electronic brakes for its hazardous cargo trains. Canada requires rerouting around cities if a route (such as the Mon Line here in Pittsburgh) exists.

As physicians, we are also concerned about vehicle and pedestrian accidents, traumatic injury and death resulting from diminished sight line lines on approaches to raised vehicular bridges—especially where, as here, the bridges involve first responder routes adjacent to hospitals (Allegheny General Hospital), police stations (Zone 1) and fire departments as is the case in connection with West North Ave/Brighton Ave Bridge. We request a full health impact assessment of this issue along the entire proposed rail corridor.

Finally, we are concerned that increased rail traffic of the magnitude that is currently proposed has significant potential for increased traumatic injury and death along the proposed modified 20 mile route through Pittsburgh. The railroads have offloaded emergency response capabilities to local communities that in reality are not funded or prepared for these types of catastrophe. It is irresponsible to pretend that the risk can be managed.

Based on the foregoing, we respectfully request that a Health Impact Assessment that addresses the following questions be required:

1. Will Norfolk Southern guarantee that it will install Positive Train Control, with a backup system when the main system is taken off line, before opening its Ft. Wayne and Pittsburgh lines to double stack trains?
2. Will Norfolk Southern employ electronic brakes on its trains carrying hazardous material?
3. Since Norfolk Southern's Annual Report indicates that its insurance may be inadequate to cover a derailment involving hazardous material, will Norfolk Southern agree to post a bond or increase its insurance coverage?

4. Since it is illegal to spill oil under the Oil Pollution Act of 1990 (OPA), will Norfolk Southern agree to strict liability for all property damage, health costs, lives lost, restoration of natural resources, and to the imposition of punitive damages?
5. Will Norfolk Southern provide training equipment, communications systems, and qualified officials to coordinate with other first responders?
6. Will Norfolk Southern provide inspection reports for its rail bridges and tracks?
7. How many times daily do EMS vehicles, including police, fire and medic units, cross bridges scheduled to be raised by the PVCP? Please note that an ambulance needs to cross twice to transport a patient to a hospital.
8. How many people are affected at each raised vehicular crossing, based on current and projected populations as shown in relevant planning documents?

We thank you for your attention to thorough evaluation and full disclosure of the potential health impacts of Norfolk Southern's PVCP

The 94 health care professionals below have provided authorization for their names to be included in this HIA request:

Mark Shlomchik, M.D.
Department Chair and Professor of Immunology, University of Pittsburgh

Holly Lorenz RN, BSN, MSN
Chief Nurse Executive
UPMC Health System

Ned Ketyer, M.D., F.A.A.P.
AHN Pediatrics — Pediatric Alliance
Editor, The PediaBlog
AAP Council on Environmental Health
SWPA Environmental Health Project (Consultant)
Physicians for Social Responsibility — Pennsylvania (Board member)
Climate Reality Project Leader

Peggy Slota, DNP, RN, FAAN
Director, DNP Studies; Director, PM-DNP Program
Associate Professor
Georgetown University School of Nursing & Health Studies
Associate Editor - Journal of Pediatric Nursing

Jeffrey Ubinger, MD
Pediatrician- Premier Medical Associates

Peter Hauber MD , Board-certified psychiatrist

Catherine S. Hauber RN

Pouné Saberi, MD, MPH
Physicians for Social Responsibility, Philadelphia
Board President

Edward H. Wrenn, M.D. Family Medicine Hospitalist licensed by the State of Pennsylvania.

Susan E. Moore, MSN, RN, CNE, Nurse educator at UPMC Shadyside School of Nursing

Ed King MD
Pediatrician
Pediatric Alliance PC- St. Clair Division, AHN Pediatrics

Michael Cole PA-C

Sarah Yourd, MSN, FNP.

Jordan F. Karp, MD
Professor of Psychiatry, Anesthesiology, and Clinical and Translational Science
University of Pittsburgh School of Medicine

Paul Carson, Staff Hospitalist, Allegheny Health Network.

Anthony L Kovatch, MD.-AHN Pediatrics

Lesley Gradone RDN, LD, CNSC
Dietitian
AHN Pediatrics-Pediatric Alliance, Arcadia Division

Alan S. Peterson, MD
Emeritus Director of Environmental and Community Health
Lancaster General Health

David Solosko, MD

Sandra L. Kniess, RN

Jessica Beu
Licensed Social Worker

Marirose Radelet MS, PT, ATC retired

Amy P Kennedy, RPh [BS Pharm, University of Pittsburgh, 1985]

Chirley M. Rodriguez, MD.
Allegheny Health Network

Lisa Ann Hackworth, DO
Allegheny Health Network

Robert Little MD, President PSR/Pennsylvania/Harrisburg division

Marisa Evans, MD Allegheny Health Network

Imran Qadeer, MD
Chief Medical Officer
Allegheny General Hospital

Brittany Paluzzi, MD, Allegheny General Hospital

Marc D. Guerini, MD
Allegheny Health System

Margaret Baco, RN, CMT
Critical Care Nurse, Cardio-thoracic, post-op, ICU NURSE.

Board Chair of Unity Through Creativity, 501c3

Dr. William R. Davidson Jr. M.D. retired cardiologist presently working for the past 10 years in Lebanon's free clinics for the uninsured. Member of PNHP and PSR.

Leslie Latterman, DO AHN

Bobby Wilson, MS, RPFT
Researcher
University of Pittsburgh Emphysema COPD Research Center

Joseph Sabol, MD
Hospitalist Director, Allegheny General Hospital and West Penn Hospital

Trina Peduzzi MD FAAP
Pediatrician
UPMC Children's Community Pediatrics- Bellevue Pediatrics

Nancy Freyvogel, M.D. AGH

Betsy Blazek-O'Neill, MD
Medical Director, AHN Employee Health Services
Staff Physician, AHN Integrative Medicine Program

Sarah Kohl, MD, FAAP

Dr. William R. Davidson Jr. M.D., PNHP and PSR.

Alex Knisely, MD
Private practice of histopathology

Isabela C Angelelli, MD - Pediatrician in Pittsburgh

Christiane Siewers, MD, Retired

James Parrish, MD

Marc J. Eubanks, MD, MPM, FACEP

Thomas L Macchia P.A.-C. (retired)

Deborah Mehalik, BSN, CCM

Kathleen Krebs, RN

Etta Albright, RN

Annette Calderone, RN, PhD

Barbara Gold, MD

Robert Doll, MD

Robert Little, MD

Gerritt and Elizabeth Baker-Smith

Eric Stein, MD

Edward Thornton, PhD

Marc Eubanks, MD

Robert Janusko, PhD

John Dinger, MD

Silvio Fittipaldi

Lauren Kolber, MD

Mark Vendel, MPH

Sa Re, MPH

Linda Bazan, PhD

Robert Gibb, PhD

Rosemary Caolo, PhD

Sarah Hancock

Sydney Hausman Cohen, RN

Andrew Johnson, DVM

Reverend Sandra Mackie, RN

Thomas Macchia, PA

Peri Unligil, MD

John Comella

Lawrence Tonzi, FNP

Walter Tsou, MD PSR PA

Ralph Miranda, MD President, Westmoreland County Medical Society

Barbara Brandom, MD

John and Ann Marie Judson, MD

Bryn Hammarstrom, RN

Edward Ketyer, MD PSR – Pennsylvania

Laurence Carroll, MD

Alan Peterson, MD, MD

Maria Braun, MD American Academy of Family Physicians, Pennsylvania Medical Society, Berks County Medical Society, Tower Health Physician Network, Penn State Health Network

Mary Bell, EMT

Mario Suley

Stephanie Rex

Stacy Henderson, PT

Judy Kelly

Ginny Landis, PT

Marc Eubanks, MD

Jerome Parness, MD, PhD UPMC Children's Hospital, Retired

William Davidson, MD

References

Additional information listed in 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

Clay, K. et.al. (2018) https://www.post-gazette.com/opinion/Op-Ed/2018/07/29/Pittsburgh-Think-twice-about-allowing-more-rail-traffic-to-roll-through-the-city/stories/201807290012

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.

Fabisak, J and Jackson, E 2018, Center for Healthy Environments and Communities, School of Public Health, University of Pittsburgh, *Proposed Rail Changes Can Have Significant Health Impacts for Allegheny County*

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.

McConell, 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

Thaller, E. et al. 2008. Moderate increases in ambient PM2.5 and ozone are associated with lung function decreases in beach lifeguards. *J Occup Environ Med* 50:202-211.

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

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

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

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

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

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

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, 3rd 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.

Additional information listed in coaltrainfacts.org.

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.