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Utah Physicians for a Healthy Environment (UPHE) has over 360 members, most of whom are physicians or other health professionals. The remainder are individuals educated in and practicing toxicology, chemical engineering, air quality modeling and permitting, biology, earth sciences and other related disciplines. We provide education to the public and policy makers regarding the public health consequences of environmental degradation.

USACE Must Require an EIS

Although the focus of our activities is in Utah, we provide consultation services to citizens groups in several other states. We have been asked by Petaluma Moms for Clean Air to consult on the health consequences of the pollution that will result from Dutra's operation of an asphalt plant in Petaluma. Inadequate consideration of the health consequences of the emissions from Dutra's proposed plant provides one of many reasons why the USACE should require a robust and complete EIS. UPHE is not receiving any financial compensation for our involvement nor do we have any financial conflict of interest regarding this issue.

Because state and federal regulatory agencies must make an attempt to protect public health in the context of allowing opportunity for industrial activity, they are required to establish thresholds for what might be considered acceptable levels of air pollution. As science progresses more information about the hazards of air pollution steadily accumulates but that new information does not become reflected in those thresholds until several years, and some cases decades, after the science has become well established. For example, the EPA has not updated the national 24 hr. standard for PM10 since 1987.

Furthermore, the current combined regulatory framework of the federal government and the state of California can only be described as "health based" in the broadest sense, i.e. it is more protective of health than no standards at all. But there is little account for the fact that pollution from major sources is not evenly distributed throughout a community air shed. For example, the most deadly subset of particulate pollution, ultrafine, can be 25-40 times more concentrated near freeways, than 300 meters away (139, 140). Other studies indicate that the dissipation of pollution does not reach background levels until 160-570 meters away (141). This indicates the plant, and the diesel emissions from the hundreds of trucks, barges, and other heavy equipment involved in transport of substrate and finished product, will be an especially serious health hazard to those that live near by, more than an order of magnitude greater risk compared to those in the rest of the community. In other words, if the plant is permitted, residential areas near the plant, and the roadways and waterways leading to the plant, will become sacrifice zones regarding public health.

Over 3,000 studies published in the last fifteen years clearly reveal that there is no such thing as a "safe" level of air pollution which means that regulatory thresholds are essentially arbitrary or driven by political, economic, or value judgment considerations rather than strictly medical science. Even levels previously thought to be benign we now know are not. There is no threshold below which there is no health effect and any increase in pollution to a community will have corresponding public health impacts. All persons are adversely affected by air pollution, regardless of age or overall state of health. Even if the Dutra project satisfies current regulations or is approved by the BAAQMD, in no way does that mean it will not have substantial adverse health impacts to the community.

As a manifestation of the evidence for severe health effects from air pollution, not adequately addressed by EPA's National Ambient Air Quality Standards, virtually every major medical organization in the

United States, including the American Medical Association, the American Thoracic Society, the American Lung Association, the American Academy of Pediatrics, the American College of Cardiology, the American Heart Association, the American Cancer Society, the American Public Health Assoc., the National Assoc. of Local Boards of Health, and the EPA's Clean Air Scientific Advisory Committee (considered the nation's premier air pollution experts), invariably calls for stricter standards, almost regardless of where the EPA eventually establishes those standards.

Before permitting, USACE has the obligation to require an environmental impact statement if there is reason to believe that human health, wildlife, or the natural environment will be impaired by a proposed project. That is certainly the case regarding proposed Dutra asphalt plant in Petaluma. The remainder of this report will focus on the possible specific human health consequences of the various and combined pollution streams from the proposal.

Public Health Consequences of Pollution Related Asphalt Production

There is no disputing that this plant will increase the levels of PM 2.5, PM 10, Nitrogen Oxides, Ozone, and Hazardous Air Pollutants (HAP) in the Petaluma community. Virtually all of the broad based and well known health consequences of first and second hand cigarette smoke are also now known to be the consequence of urban air pollution that includes this milieu of components.

In the same way there is no safe number of cigarettes you can smoke, there is no safe level of air pollution you can breathe. That should be no surprise because most of the chemicals in both types of air pollution are the same. In recent years throughout the country, local, state and federal regulations have been implemented to eliminate exposure to second hand smoke. Any new industrial source of air pollution into a

community will have the same type of health outcome as exposing the citizens to a new source of second hand cigarette smoke.

There is now evidence that exposure to ozone and PM2.5 can act synergistically, increasing the adverse health effects from these air pollutants (1). As with smoking, particulate matter pollution and ozone cause increased systemic oxidative stress leading to pathologic vascular changes, including progression of atherosclerotic plaques to vulnerable forms, prothrombotic states, endothelial dysfunction and altered autonomic nervous system control (2).

Air Pollution and Mortality and Cardiovascular Disease

For the last several years, the research based conventional wisdom has been that with each 10 μg/m3 increase in PM2.5 long-term average, there is an increase in community mortality rate of about 10% (3). New research draws an even stronger correlation – a mortality rate of 14% for each 10 μg/m3 increase (4). The elderly and those with existing morbidities are particularly vulnerable to air pollution consequences.

The totality of medical research up to 2015 provides strong confidence in a causal relationship between long- and short-term exposures to PM2.5 and mortality and cardiovascular effects. The American Heart Association (AHA) has estimated that residents of most cities in the United States lose between one and three years of life expectancy due to fine particulate air pollution. Furthermore, studies show that even small reductions in air pollution improve community life expectancy.

Since the late 1980s, more than 150 epidemiological studies have reported associations between daily changes in particulate air pollution and respiratory and cardiovascular mortality, hospitalizations and other related health endpoints (5). These adverse effects are seen at low and "common" concentrations of particulate pollution. A Dutch study demonstrated risks for cardiopulmonary mortality even at what are

considered "background" levels of particulate pollution (6). A study done in Utah demonstrated that each short-term 10 µg/m3 increase in PM2.5 was associated with an increase in the risk of acute ischemic coronary artery events (unstable angina and myocardial infarction) of 4.5% (7).

Not only does PM2.5 result in an increase in death from cardiovascular causes, but there is also an increased risk for non-fatal events. For each 10 µg/m3 increase in PM2.5 women experience a 24% increase in risk of a cardiovascular event and a 76% increase in the risk of death from that event. There is also an increased risk of stroke events (8, 9, 10). It should be noted that this rate of increase approaches that demonstrated from a chronic active smoking habit.

Many medical studies show that impacts from pollution are seen very quickly and can last long after the air has cleared. For example, within as little as 30 minutes, exposure to particulate matter is associated with increases in blood pressure, followed within hours by increased rates of heart attacks and strokes. Community mortality rates stay elevated for 30 days after a spike in PM10 even if the episode lasts less than 24 hours (11).

Within one hour, exposure to traffic pollution, including particulate matter, is associated with increased rates of heart attacks as much as 300% compared to non-exposed individuals (12). Other studies show rates of strokes and heart attacks in the community increase within hours after spikes in PM10 (13).

In 2010, the AHA published this summary:

"The overall evidence from time-series analyses conducted worldwide since publication of the first AHA statement confirms the existence of a small, yet consistent association between increased mortality and short-term elevations in PM10 and PM2.5 approximately equal to a 0.4% to 1.0% increase in daily mortality (and cardiovascular death specifically)

due to a 10 μg/m3 elevation in PM2.5 during the preceding 1 to 5 days (3)."

Confirming the strong correlation between modest, short-term spikes in PM and serious health consequences are three new studies that showed spikes of as little as one day in PM10 were associated with higher rates of heart attacks (14), daily spikes of either PM10 or PM2.5 were associated with significant increases in emergency room visits for hypertensive crisis (15) and less than 24 hours of a spike in PM2.5 of 15-40 μg/m3 increased rates of strokes 34%, with the peak increase occurring within 12 hours (16). A large meta-analysis of 94 studies showed even short term spikes in ozone, carbon monoxide, SO2, NOx, and PM2.5 are associated with significant increases in rates of strokes. The greatest association was for the same day of exposure, although PM2.5 showed a lingering affect (133).

Not only have numerous studies shown that there is no safe level of PM exposure, but a recent landmark study published in the flagship journal of the AHA, using data from over 1 million people, demonstrated that when cardiac mortality, the signature air pollution health outcome, was plotted against particulate matter from air pollution, and first and second-hand cigarette smoke, all three sources showed a steep curve at low doses. In other words, per unit dose of exposure, it is the low levels of PM that cause higher rates of mortality (17).

Long-term exposure to PM2.5 is linked with an array of cardiovascular effects such as heart attacks, congestive heart failure, stroke, arrhythmias (142) and sudden death. Long-term exposure to particulate matter air pollution is associated with an average rise in blood pressure for populations chronically exposed. Average blood pressure was found to rise 1.7 mmHg for an increase of 2.4 μg/m3 in PM2.5. A similar association was found with the coarser PM10. The rise was found in both systolic and diastolic blood pressure (18). Even prenatal exposure during pregnancy is associated with higher blood pressure in newborns (83).

Chronic exposure to particulate matter has also been shown to increase the thickening of arterial walls, which is a known end result of higher blood pressure, especially critical arteries like the carotid arteries which is provides the primary blood supply to the brain (84). A chronic increase in PM10 of 5.2 μg/m3 is associated with a 5% increase in the intima-media thickness of the carotid artery, which is one of many end results of the biologic process described above (19).

Another study showed a remarkable correlation between chronic exposure to PM2.5 and narrowing in the tiny arteries in the back of the eye. Chronic exposure to 3 μg/m3 of PM2.5 (one fourth of the NAAQS) was associated with narrowing equivalent to seven years of aging (20). These finding are especially significant because they demonstrate community-wide effects, acceleration of the aging process, and impairing the health of everyone exposed, not just a susceptible population.

There is a strong correlation between rates of deep vein thrombosis and increased levels of PM10, beginning at very modest levels (23). Recent studies provide additional evidence for cardiovascular effects associated with sub-daily (e.g., one to several hours) exposure to PM, especially effects related to cardiac ischemia, vasomotor function, and more subtle changes in markers of systemic inflammation, homeostasis, thrombosis and coagulation. A likely mechanism of this clinical outcome is revealed by studies that show PM10 causes excessive platelet aggregation in diabetics (24).

Air Pollution and Brain Disorders

Throughout the age spectrum, from infants to the elderly, air pollution has been shown to impair brain function. Oxidative stress (OS) appears to be the biological genesis of numerous disease processes and a major contributor to the aging phenomenon. Numerous studies show that air

pollution's harmful affect on the brain begins in utero. In children and in lab animals air pollution exposure is associated with abnormal brain architecture, including enlarged ventricles and loss of white matter (85, 86).

The volume loss of brain white matter in children, showed a striking linear correlation with lower intelligence scores, and behavioral problems like hyperactivity, and attention deficit disorders. The study showed that there was no safe level of PAH exposure regarding its affect on brain development. The authors concluded, "Pregnant women and young children are very vulnerable to environmental insults to the developing brain, and these exposures are likely having devastating effects." PAH exposure during pregnancy also decreases production of placental proteins critical to fetal brain development. Authors of other related studies conclude, "In children it is especially disturbing that air pollution exposure is associated with brain structural and volumetric changes, cognitive, olfactory, auditory and vestibular deficits, all of which also have long term neurodegenerative consequences" (87).

OS is the common mechanism behind the role of particulate matter and carbon monoxide pollution in central nervous system damage in children and neuro-degenerative diseases such as Alzheimer's disease (25, 26). Numerous studies show such specific outcomes as impaired intellect, and penetration of particle matter and Alzheimer type protein deposition among children who grow up breathing more particulate air pollution (27, 28, 29). Volunteers exposed to typical urban levels of diesel exhaust demonstrate brain cortical stress measured by EEG (30). In utero exposure to diesel exhaust, a ubiquitous part of heavy industry, like oil refineries, gravel pits, and asphalt plants, is associated with loss of normal fetal activity and abnormally low levels of neurotransmitter chemicals in lab animals (88).

Children exposed to more air pollution or whose mothers were more exposed during pregnancy show a quantitative loss of IQ, as much as five to nine points (31, 32, 33). Rates of neurobehavioral disorders

correlate with exposure to NOX and PM10 (34). Numerous studies now show a strong correlation with air pollution exposure and rates of autism (35, 89-94, 128, 129, 134) that is not explained by confounding variables.

Older people show accelerated cognitive decline if chronically exposed to more traffic generated air pollution (36, 37). A recent landmark study showed that chronic exposure to 10 μg/m3 of either PM2.5 or PM2.5-PM10 was associated with faster cognitive decline in older women, equivalent to about two years of aging (38). For every 2 ug/m3 increase in PM2.5, brain volume decreased 0.32% and the odds of covert brain infarcts (mini-strokes) increased 46% (132).

Because of strong evidence that particulate air pollution's neurotoxicity is related to attached metals (39, 40, 41), pollution from an asphalt plant and diesel engines that are rich in heavy metals, like lead, cadmium and zinc, take on additional public health significance.

Air Pollution and Lung Disease

It is intuitive that short-term exposure to fine particulate matter would have adverse impacts on the pulmonary system. Indeed, numerous studies show increased rates of asthma and virtually all other respiratory diseases including lung cancer where short-term PM 2.5 is higher. Equally disturbing are less obvious outcomes. Even young healthy people demonstrate rapid decrease in lung function from brief exposure to particulate matter that persists for several days after the exposure has ended (42, 43). With regard to respiratory effects in children associated with short-term exposures to PM2.5, currently available studies provide stronger evidence of respiratory-related hospitalizations with larger effect estimates observed among children.

The physiology of children differs from that in adults in many important ways, causing them to be affected more profoundly by air pollution than

adults. A child has a higher metabolic rate, meaning their oxygen demand is higher, they breathe faster and have higher heart rates and blood flows on a per weight basis than an adult. Combined with their rapidly growing organ size and function, this physiologic difference makes them more susceptible to the adverse influence of air pollution. Higher particulate pollution and ozone in the first two months of life is associated with higher rates of infant respiratory death and SIDS (95). The association between long-term PM2.5 exposure and respiratory mortality extends through the first year of life.

Children who breathe more air pollution can experience a permanent stunting of their lung growth. Just as chronic exposure to second-hand cigarette smoke causes a permanent loss of lung function growth in children, so does long-term exposure to PM2.5 air pollution.(44,45). These analyses provide evidence that PM2.5-related effects to children persist into early adulthood and are more robust and larger in magnitude than previously reported. Not only does short-term exposure to PM2.5 air pollution permanently impair the exercise capacity of individuals so affected (46) few physiologic outcomes have more of an ultimate impact on longevity than lung function.

Air Pollution, Cancer, and Miscellaneous Health Consequences

The World Health Organization recently declared, "Air pollution is now the world's single largest environmental health risk" (100), and focused on air pollution's role as a carcinogen. Various forms of cancer such as lung, cervical, stomach, prostate, liver, ovarian, and brain cancer show increased rates with higher concentrations of community particulate matter (47, 48, 96, 97, 98, 135, 137). Each 10 μg/m3 increase in long term PM2.5 concentration is associated with a 15-27% increase in lung cancer mortality (49). Especially troubling are the numerous studies that show increases in childhood leukemia among more exposed populations (50, 72, 136), and a significant association between air pollution and rates of breast cancer (52) and decreased survival among breast cancer

victims (99).

As mentioned above, the proposed Dutra plant will create a serious hot spot of pollution, diesel emissions in particular, which are highly toxic and carcinogenic by causing DNA damage (138). The California Air Resources Board estimates that about 70% of the cancer risk that the average
Californian faces from breathing toxic air pollutants stems from diesel exhaust particles. Two landmark studies show that long term exposure to diesel exhaust common in urban settings, increases the chance of dying from lung cancer 50% for residents near industrial operations, and 300% for the workers (101, 102).

The common assumption about particulate air pollution has been that internalizing the particles and their adsorbed compounds like heavy metals occurs through the lungs. Smaller particles are assumed more dangerous because they can penetrate more deeply into the lungs and are cleared by the lung cilia less readily. However, there is new evidence to suggest that atmospheric particulate matter is also swallowed, leading to toxicity of internal organs and increased carcinogenic risk. This is of particular relevance for childhood exposure (68).

The precipitation of oxidative stress, as mentioned above, is the likely explanation for new studies that show higher rates of numerous other, seemingly unrelated diseases, among populations subjected to more air pollution; such as type I and type II diabetes, obesity, arthritis, inflammatory bowel disorders and lupus (53–57, 103—109, 130, 131).

Air Pollution, Pregnancy, Chromosomes and Fetal Development

Air pollution, especially particulate matter, may have its largest impact on public health through its effect on the human embryo. A study in laboratory animals demonstrated a change in morphology of the placenta that compromised blood flow to the fetus (46, 58). PAHs accumulate in

the placenta and seem to be particularly toxic to placental vasculature, causing increased tortuosity flow resistance (110, 111). Air pollution alters hormone levels like placental growth factor responsible for healthy placental blood vessel formation (112).

Exposure of pregnant women to various components of traffic-related air pollution, including PM10, results in intrauterine growth retardation, including smaller head size, increased rates of spontaneous abortions, premature births, pre-eclampsia, premature rupture of membranes, gestational diabetes, low birth weight syndrome, and still births (75, 113—123).

Genetic damage and epigenetic changes can have virtually identical consequences and both can be passed on to subsequence generations (124, 125).

Newborn babies whose mothers are exposed to more air pollution show increases in both, and the life-long disease burden that results can include higher rates of metabolic disorders, reactive airway disease, cardiovascular disease, cancer, Alzheimer's and all the diseases consequent to immuno-suppression. Chemical alterations of DNA occur in placentas of women exposed to more air pollution (126).

Pregnant mothers exposed to more air pollution demonstrate shorter placental telomeres (127). Given that telomere length is tightly correlated with life expectancy, that mean air pollution exposure during pregnancy literally programs her baby to a shorter life span.

Epigenetic changes can be seen within three days after exposure to PM2.5 and perhaps even as soon as minutes after exposure (59-66, 69, 70). There is strong evidence for a persistence of epigenetic changes from one generation to another. Medical science is now learning that the air pollution today can adversely affect the health of future generations. For example, episodic air pollution has been shown to be associated with fragmentation of DNA in human sperm (67), and moderate benzene

exposure is associated with sperm having the wrong number of chromosomes (aneuploidy) (74).

That all these above mentioned adverse health outcomes can be the result of pregnant women smoking is easy for physicians and the lay public alike to comprehend and the sight of a pregnant woman smoking is now repulsive to society at large. It is a new thought process, but equally scientifically based, to think that the same thing happens when a pregnant woman has to breathe particulate air pollution. Again, regarding impact on the human embryo there appears to be no safe threshold of exposure.

Conclusion

Utah Physicians for a Healthy Environment believes that the permitting process for the proposed Dutra plant has been grossly deficient in exploring and defining the public health consequences to residents of Petaluma. We urge USACE to require a full and complete EIS before advancing this process.

Sincerely,

Dr. Brian Moench
President, Utah Physicians for a Healthy Environment

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