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[redacted]

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**Via E-Mail: hydraulic.fracturing@epa.gov**
Environmental Protection Agency
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Re: **Hydraulic Fracturing Study--Comments**

I am writing this testimony to the EPA in response to a request for comments regarding the threats posed by hydrofracking on water pollution and public health. I am writing as a physician and a research scientist with expertise in autism and developmental disorders. While by no means do I intend to diminish the importance of ensuring drinking water protection, I find it astounding that the issue of hydraulic fracturing's impacts on air quality appear to be given minimal attention with regard to public health.

The EPA's own recently proposed Transport Rule would require 31 states and the District of Columbia to significantly reduce power plant emissions that contribute to fine particle and ozone emissions in other states. This would require the controlling of the emissions of SO2 and NOx which cross state lines. The EPA predicts that the implementation of this rule would avoid 14,000- to 36,000 premature deaths per year (up to 12 times the number of people who died in the 9/11 attack), 21,000 cases of acute bronchitis, 23,000 non-fatal heart attacks, 26,000 hospital and emergency room visits, 1.9 million sick days when people miss work or school, 240,000 cases of aggravated asthma and 440,000 cases of upper and lower respiratory symptoms. These direct health benefits of controlling air pollution represent only the tip of the iceberg. Aside from the very worthwhile goal of, literally, preserving the "visibility" of treasured natural settings, delicate ecosystems are in danger and air pollutants go into solution in our waters. Human beings and wildlife are subject to fine particle and ozone emissions from multiple sources.

The EPA estimates the cost to the public of the Transport Rule would be about $2.2 billion, whereas the estimated benefits range from $120-290 billion. This rather modest change in regulation will yield tremendous benefits to public health and even to the economy. Most of the scientific analyses of cost savings for remediating pollution are underestimates and, for example, exclude such "costs" to society as missed school days.

As there is compelling evidence that air pollutants from fossil fuels must be more strictly regulated, any study on hydraulic fracturing and public health impacts must include studying, documenting and regulating many of the same gasses which will be produced during the extraction and refining of natural gas. With plans for hydraulic fracturing being planned on a massive scale, and with such extraction industries being done in and around residential neighborhoods and even schoolyards, the potential danger to public health is self-evident.

While there is little doubt that the most effected will be individuals residing closest to the gas extraction activity, air pollution migrates. The Transport Rule is being proposed for that very reason. Bergin et al

(2007) in their analysis of ozone in eastern states report that 77% of ozone comes from out of state sources. Rodriguez et al (2009) shows increasing ozone levels in the rural western US, as a result of gas and oil mining, and predicts that, if current trends continue, there will be a significant impact on those areas, including several National Parks.

***Air Pollution Caused by the Natural Gas Mining Industry; H2S***

In 2005, the Oil and Gas Accountability Project studied three counties in Alabama (Sumi 2007) from which there were citizens' complaints of being sickened due to their proximity to oil and gas industry activities. The project commenced due to a lack of success in getting the state to investigate their complaints. Since the lead scientist could smell H2S they measured that as well as VOCs.

H2S, which is a byproduct of "sour" gas, is estimated to be present in 15-25 % of the natural gas extracted in the US. It is believed that the percentage will be even higher as deeper formations are mined. H2S is emitted into the atmosphere near gas and oil wells, tank batteries, gas processing plants, flaring, compressor stations and refineries. Because sour gas cannot be sold it is routinely flared. SO2 is the product of combusted H2S but incomplete combustion will liberate H2S into the air. Venting, of course, will also liberate H2S in the air. The EPA has reported that the potential for routinely high H2S levels at gas and oil wells is a significant issue, especially in light of the proximity of wells to residential areas. Further, the EPA has stated that well blowouts, line releases, extinguished flares, collection of sour gas in low lying areas, line leakage, and leakage from abandoned wells are all sources of documented accidental releases which have affected the public not just industry workers. The ATSDR has reported that those living near sources of H2S often have daily ambient levels exceeding 90 ppb.

Studies have been done in four states, Arkansas, Louisiana, New Mexico and North Dakota. In all cases, maximum levels were found which could cause harm to humans. In nearly all the cases (except the Louisiana site), the average levels were toxic to human health. In the Alabama study, H2S levels were found to be 100-1000 ppb (in comparison, the normal urban background is 1 ppb). The Alabama results are in line with the other results found in the gas and oil producing areas tested (from 100-10,000 ppb).

In Western Canada, Burstyn et al (2007) found a correlation between H2S levels and proximity to gas and oil wells. They found a greater effect if the monitoring station was within 2 km of the well. Their recommendation is for greater distances between wells and exposed individuals (the opposite of the plans for hydraulic fracturing which is being undertaken or planned in cities, residential neighborhoods and even school yards).

***Hydrogen Sulfide- Health Effects***

Hydrogen Sulfide is an extremely toxic gas and exposure can lead to coma and death. In one case of sour gas pollution in Poza Rica Mexico, 320 people living around the refineries were hospitalized and 22 died (McCabe and Clayton 1950). Acute exposure can result in loss of consciousness and, upon recovery, neurological symptoms occur including dizziness, persistent headache, loss of balance, agitation, amnesia and cognitive dysfunction (Kilburn et al 2010). Although it was thought that there was recovery from these symptoms, there is now substantial literature to document the long term deficits resulting from these exposures. Permanent brain damage has been documented with standardized instruments. Animal studies demonstrated that even small intermittent exposures have neurotoxic effects on many parts of the brain. Rats exposed in utero show abnormal cell growth and abnormal neurotransmitter levels, thus suggesting that it may also be a neurodevelopmental toxicant.

H2S was removed from inclusion in the Clean Air Act under pressure from the American Petroleum Institute (Morris 1997, Kilburn et al 2010) and despite opposition from the Agencies for Toxic Substances and Disease Registry (ATSDR). Legator et al (2001) reports increased risk ratios for disease for persons exposed to H2S from various sources. Saadat et al (2004) report on an area of SW Iran where there is

subsurface sour gas leakage. They report high depression scores, increased rates of suicide, and decreased WBC counts while the other components of the CBC were elevated. Kilburn and Warshaw (1995) and Kilburn (1997,1999, 2003) report on numerous neuropsychiatric deficits including memory, balance, word recall and others from subjects who were exposed to low concentrations of sulfur compounds from refineries, oil field workers, natural gas and hog lagoons, leakage into homes from natural gas and petroleum deposits, and a refinery explosion.

A recent publication (Kilburn et al 2010) compared a town in New Mexico with high H2S exposures (Lovington) to two other towns also exposed to sour gas fields (Tatum and Artesia NM) but with less exposure to H2S, and using a town in Arizona (Wickenburg) without any significant exposures as a comparator. The Lovington group had on the average 11.8 neuropsychological abnormalities compared to 3.6 for Tatum/Artesia and 2.0 for the group from Wickenburg. The Lovington group had 34 of 35 symptoms more frequently than the Wickenburg group. While the average levels in the three New Mexico towns is 0-14 ppb, with peaks measured at 1600 ppb, there were some instances of exposures which were much higher. The Wickenburg levels were 0.02-0.07 ppb. This suggests that chronic exposure to H2S levels which are not considered toxic may nonetheless have long term adverse effects on central nervous system functioning.

***Ozone and Its Components***

***The Effects of O3, PM, NOx and VOCs, and PAHs on Public Health***

Ozone (O3), particulate matter (PM), nitrogen oxides (NOx), Volatile Organic Compounds (VOCs), and polycyclic aromatic hydrocarbons (PAH) are treated together because in most population-based studies of air pollution all of these substances increase and are interrelated. The main source of all of these compounds is from fossil fuels. Although these substances are encountered together, analysis can sometimes show differential effects of each substance on human health.

Given the enormous scale of proposed hydraulic fracturing throughout the nation, and particularly in the Marcellus Shale play (an estimated 30,000-80,000 wells in NYS alone), it would stretch credulity to believe that, if studied cumulatively, there would not be a large increase in all these substances in our environment.

The effects of ozone and PM2.5 pollution are truly enormous. Anenberg et al (2010) estimate a global mortality from anthropogenic ozone to be 0.7 million respiratory mortalities per year which yields 6.3 million years of lost life annually. PM2.5 is related to 3.5 million cardiopulmonary deaths and 220,000 deaths from lung cancer annually. It should be noted that the health studies show only demonstrable acute effects.
Beyond these figures, the overall contribution to disease remains difficult to ascertain. For example, it can easily be demonstrated that ozone damages tissue and so it very reasonable to expect that chronic exposure to ozone will lead to tissue damage. Ozone pollution most likely is contributing to the prevalence of COPD and asthma as well as inducing heart disease (Srebot et al 2009).

Armendariz (2009) published a report documenting the amounts of air polluting fugitive gases from the hydraulic fracturing in the Barnett Shale near the Dallas/Fort Worth Area. He reported on nitrogen oxides (NOx) and Volatile Organic Compounds (VOCs) which together form ozone. He also reported on hazardous air pollutants (HAPs), methane and CO2 equivalents in order to correlate its greenhouse gas effects.

All told, the polluting gasses more than match all of the vehicular traffic pollution in that area; thus, with the projected hydrofracking “boom,” in the near future this unconventional method of gas extraction could become even a larger source of pollution than vehicular traffic. With large portions of the United

States already in non-compliance with ozone levels, and the EPA recommending even lower levels to safeguard public health, opening up a new industry which will further create ozone is misguided.
In addition to Texas, the governor of Wyoming, has asked for parts of that state to be designated in non-compliance with maximum ozone levels. Significantly, in this very rural area of the state and with no other major source of ozone pollution other than hydraulic fracturing, the levels of ozone were found to be unhealthy and higher than most urban areas.

***Health Issues of ozone and related pollutants***

In Seoul, Korea (Bae and Park 2009), an increase in 10 micrograms/cubic mole in PM 10 was associated with an overall increase in daily death count of .37% and in the elderly (over 65) .45%. A 10 ppb increase in a 1 hour maximum ozone concentration resulted in an increased risk of daily death counts of .81 % and in the elderly it was .96%. It is estimated that in 2015, by achieving the WHO standard, there will be 200 fewer deaths from PM10 and 329 fewer deaths from ozone. It should be noted that only the short term effects of the pollution was calculated.

This is consistent with other studies. A study reported the mortality impact assessment for PM10 in 19 European cities. A reduction of PM 10 levels by 5 microgram/cubic meter will reduce the number of deaths by 3300 to 7700 per year. In California, attainment of the ozone 8 hour standard of 0.070 ppm would result in 630 fewer cases of premature death per year (Ostro 2006). In a study of three Latin American cities, Mexico, Santiago, and Sao Paulo (Bell 2006), 156,000 premature deaths could be prevented over 20 years using a proposal assuming a 10% reduction in pollution over the 20 year period: included in that number is 3700 infant deaths.

The same Latin American Study found 60,000 avoidable hospital admissions, 300,000 children's medical visits, 700,000 respiratory caused emergency room visits, 4 million avoidable asthma attacks, and 24 million restricted activity days in addition to 8 million lost work days.

A large study (Jarrett 2009) comprising 96 metropolitan areas, studying 448,850 subjects over 18 years with 118,777 deaths recorded, found that there was a 2.9% increase in respiratory death based on a 10 ppb increase in ozone level. Studying two pollutants (ozone and PM2.5) there was a 4% increase in death risk. There was a threefold risk of dying of respiratory disease in the metropolitan area with the highest ozone compared to the city with the lowest ozone.

The effects of ozone and its association with premature death appear to be very well documented and should no longer be considered questionable. As the EPA itself has taken this stance, it would seem inconsistent to ignore the risks presented by hydraulic fracturing as a new and still avoidable new source of this air contamination while attempting to control it in other ways.

***Air Pollution and Birth Outcome***

This area of investigation is rather new. Most of the studies cited below rely on already accumulated data which may provide a skewed and undoubtedly incomplete view of real exposures. It should be noted that these epidemiologic studies are consistent with molecular studies showing that these exposures are demonstrable as DNA adducts and appear to be present at a higher rate in fetal tissue. This supports the very reasonable hypothesis that a fetus is more sensitive to pollution than an adult.

***Infant Mortality***

Studies correlating air pollution with infant mortality have existed since the 1950s. In a review of more recent and specific studies, Sram et al (2005) reviews 8 different studies from diverse locations including Korea, Czech Republic and Brazil. They found remarkably consistent findings of increased infant mortality

correlated with air pollution. Using the Czech National Death and Birth Registries, they found strong correlations with several pollutants. Increased odds ratios of 1.95 was found for SO2, 1.74 for particulate matter, and 1.66 for NOx. Woodruff et al (1997) found a corrected increased odds ratio of 1.10 for infant death correlated with PM 10 levels. Those with high PM 10s compared to the low group had and odds ratio of 1.4 for infant respiratory death and a 1.26 ratio for sudden infant death. Perreira et al (l998) in Sao Paulo, Brazil found a very strong correlation with NO2 pollution and intrauterine death. Loomis et al (1999) in Mexico City, found with PM 2.5 levels of 10 ug/cubic m a 6.9% increase in infant death.

***Low Birth Weight, Prematurity and Growth Retardation***

Low birth weight infants and prematurity (which have a great deal of overlap) present a major and increasing problem. There is increasing evidence that this group is susceptible to a host of problems including respiratory problems and developmental disorders, such as cerebral palsy and autism. There is substantial evidence that gestational length is affected by environmental exposures.

Gray et al (2010), in a study done in North Carolina, found a stable negative association between air pollution (particulate matter) and birth weight. This was consistent with findings from other studies. Of special concern is that North Carolina is consistently in compliance with air pollution standards, suggesting that even "acceptable" levels of particulate matter lead to low birth weight. Bobak and Leon (1999) in 45 districts in the Czech Republic found increased risk ratios for low birth weight for all three: NOx, SO 2 and PMs, with an increase of 50 ugr/m3 in their mean annual concentrations. Bobak (2000) found a stronger correlation with SO2 and PM when the exposure was in the first trimester.

Lin (2001), using a petrochemical polluted area of Taiwan compared to a non- polluted area, found an increased risk ratio of 1.77 for low birth weight. Ha et al (2001) found several pollutants correlated with LBW when the exposure was in the first trimester. Intrauterine growth retardation, which goes on to correlate with adult medical problems such as hypertension and coronary heart disease (also called SGA "small for gestational age"), likewise appears to be associated with air pollution. Although fewer studies used this as an outcome, Dejmek et al (2000) found a risk ratio of 2.14 for PM10, 1.96 for PM2.5, and 2.15 for PAHs in all cases comparing high and lower exposure groups.

Vassilev et al (2001) using EPA cumulative data in New Jersey found a 1.31 risk ratio for low birth weight, comparing the highest and lowest levels of polycyclic organic matter exposure during pregnancy. Perera et al (2003) studied air-born polycyclic aromatic hydrocarbons (PAHs) using the personal air of the pregnant mother. In her Afro-American population, as opposed to the Dominican population, she found a significant association with LBW as well as lower head circumference. Edwards et al (2010), using the same methodology in Poland, found that high exposures to PAHs in utero resulted in a 4 point lower IQ at age 5, which is consistent with the findings in NY done by the same group.

Overall, although the evidence is not as strong for an association between air pollution and low birth weight as it is with mortality, there is clearly a trend of association with some pollutants at some points during pregnancy. These findings clearly demonstrate the need for additional studies as the public health implications of increasing the numbers of premature and low birth weight babies are enormous.

Five studies were found correlating air pollution with birth defects. Ritz et al (2002) found a correlation between CO and ventricular septal deficits with a RR of 2.95 comparing the highest and lowest quartile of exposure. The risk ratio for ozone exposure in the second month of gestation was 2.68 for aortic artery and valve defects. A case control study in the 3-8$^{th}$ week was done in Texas and positive correlations were found between PM and atrial septal defects, and SO2 and ventricular septal defects (Gilboa 2005). A study in Taiwan found only an association between ozone in the first two months of gestation and cleft lip (Hwang 2008). A study done in Georgia found a correlation between PM 10 and patent ductus (Strickland 2009). In Brisbane, Australia, when measured within 6 km of the monitor, a 5 ppb increase in ozone correlated with pulmonary artery and valve defects, with a RR of 2.96 and a 6 ppb increase in SO2

correlated with atrial and valvular defects with an RR of 10.76(Hansen 2009). Birth defects are rare and so it is not surprising that there would be some inconsistency of findings. In addition, the timing during pregnancy is more precise to correlate with these defects compared to other measures such as low birth weight. Clearly, however, looking at the overall picture, these studies suggest the urgent need for a more serious look at the effects of air pollution on birth defects.

Hertz-Picciotto et al (2007) report correlations between PAH and particulate matter and immune abnormalities in cord blood of the developing fetus. They further note that the there is a growing literature implicating the immune system as central in the development of the central nervous system, with evidence of its role in diseases such as autism.

***Fugitive Methane***

Methane contributes to the global background concentration of tropospheric ozone, which as mentioned above is responsible for premature death. Both gases are also potent greenhouse gasses. West et al (2006) make the case that a global decrease in surface ozone concentration due to methane mitigation will result in widespread decreases in human mortality. By reducing anthropogenic methane emissions by 20% in 2010 it would lower the ozone by 1ppb by volume globally. They estimate this will yield a decrease in 370,000 premature deaths between 2010 and 2030 and, in the year 2030, it would lower premature mortality by 30,000 in that year. This is based on its effects on ozone and PM; however, the enhancing of global warming due to fugitive methane will also have health effects. 20-30% of the current anthropogenic methane is estimated to come from gas and oil drilling (West and Fiore 2005). Rather than instituting methane remediation, increased hydraulic fracturing operations will, no doubt, liberate methane into the air. If unregulated, this new source of methane may be very significant and lead to more premature death.

Despite the overwhelming evidence of mortality, illness and adverse birth outcome correlating with air pollution there is a shocking complacency on the part of most regulators, policy makers and medical professionals. If similar numbers of subjects were killed or sickened from infectious diseases, it would be declared a national emergency. It is critical that the EPA, in its study of hydraulic fracturing and public health, address the cumulative impacts of certain air pollutants on the health of the communities that this unconventional drilling technology will--frequently unwillingly-- impact, including fetuses, infants, children, adults, the elderly and medically fragile individuals. I trust that the EPA will undertake its hydraulic fracturing study with both water and air impacts included as it may be the only agency assessing what may become a looming public health disaster.

Sincerely,

Eric London, MD

*Sources:*

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