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June 15, 2014

Dear Ms. Singer:

I am writing to offer my perspective concerning the June 6, 2014 letter from the New York Department of Health concerning the health hazards of the pentachlorophenol-treated utility poles in your area. I serve on the steering committee of the International POPs Elimination Network (IPEN) and have been involved in the scientific review of pentachlorophenol for inclusion under the legally binding provisions of the Stockholm Convention on Persistent Organic Pollutants ("POPs" treaty), a Convention that is ratified by 179 nations. The Committee made the determination that pentachlorophenol meets the scientific criteria to be classified as a POP because of its persistence, bioaccumulation, long-range transport, and adverse effects. In October 2013, the POPs Review Committee, the scientific committee of the Stockholm Convention, made the determination that pentachlorophenol (hereafter referred to as PCP) "is likely as a result of its long-range transport, to lead to significant adverse human health and environmental effects, **such that global action is warranted**."

In my view, the letter from the NY Department of Health does not address the serious health hazards posed by the high concentrations of PCP from the treated utility poles for several reasons:

- Inevitable leaching and volatilization from the immediate area of the treated poles, and likely human exposure through inhalation and possibly through groundwater and/or surface water sources of drinking water;
- Exposures to dioxins and furans associated with PCP-treated poles'
- Special vulnerability of pregnant women and children, elderly people, and those with chronic illnesses;
- Evidence of serious adverse health outcomes associated with low exposures to PCP, including reproductive and developmental toxicities, immunodeficiency, and interference with thyroid hormone, and certain cancers.

- The NYDOH letter relies on EPA's assessment of PCP which in my view is deficient and does not properly take the current scientific and medical literature into account.
- Countries began banning all uses of pentachlorophenol in the 1970s because of severe environmental and health hazards. "It's uses were curtailed as early as 1978 due to wide-spread environmental contamination, its high toxicity to humans, animals and wildlife, and the presence in the technical product of highly toxic and carcinogenic chlorinated dioxins as micro-contaminants." PCP is either banned or has no uses in all E.U. member states. A partial list of other countries that have banned all uses of pentachlorophenol include: Austria (1991), India (1991), Indonesia (1980), New Zealand (1991), Sweden (1978), and Switzerland (1988). Other countries instituting bans include: Brazil, Denmark, Finland, Gambia, Liechtenstein, Republic of Korea (for import, use, and sale), Kuwait, Netherlands, Panama, Russian Federation, Slovakia (banned in agriculture), and Thailand.
- The levels found in the soils surrounding the utility poles greatly exceed cleanup standards for PCP in soils. In my view, the NYDOH seriously underestimates the health hazards of the treated poles.

PCP treated poles and wood are important sources and significant reservoirs of dioxins and furans that present a hazard for environmental and human exposure due to documented contamination of groundwater, soils, and foods [1],[2],[3],[4]. Bulle et al. 2010 concluded that emissions from PCP-treated wooden poles in service are one of the main sources of PCDD/Fs. In Canada for example, emissions from in-service poles are estimated to be 47% of the total national emissions to soil as reported in the National Canadian Pollutant Inventory. The study found that the main detected congeners were octachlorodibenzo-p-dioxin and furan and heptachlorodibenzo-p-dioxins and furans, the most representative congeners for PCP. The study found 2,3,7,8-tetrachloro-dibenzo-p-dioxin (2,3,7,8-TCDD) around all poles, although it was not expected to be a contaminant in PCP. Researchers found up to 8.5 E ± 02 pg/g of 2,3,7,8-TCDD in organic soil close to the pole. In this study, 2,3,7,8-TCDD was found in underlying aquifers below two treated poles in sand and clay. At an aquifer depth in sand at 9.65-10.18 M, measured concentrations were 6 pg/L directly under pole and 7.8 pg/L at distance of 30.48 cm and 2.8 pg/L at 60.96 cm. For clay, the aquifer depth was 10.29-10.55 meters and the concentration found was 3.7 pg/L directly below the pole, and not detected at 30.48 and 60.96 cm. Although not predicted, high levels of PCDD/F were found in the first 2 M below the surface. "However, in the case of sand, much lower levels of PCDD/Fs were predicted in the surface soil, but the depth of migration remains very high and the inherent danger becomes a possible underlying

[1] Karlsson L, Cragin L, Center G, Giguere C, Comstock J, Boccuzzo L, Sumner A (2013) Pentachlorophenol contamination of private drinking water from treated utility poles, Am J Public Health 103:276-277.

[2] Bulle, C. et al. 2010. Enhanced migration of PCDD/Fs in the presence of PCP-treated oil in soil around utility poles: screening model validation. Env. Tox. Chem 29(3):582-590.

[3] Fries, GF et al. 2002. Treated wood in livestock facilities: relationship among residues of PCP, dioxins and furans in wood and beef. Env. Poll. 116:301-307.

[4] Lorber, MN et al. 2002. Investigation of the potential release of polychlorinated dioxins and furans from PCP-treated utility poles. Sci. Total Env. 290:15-39.

aquifer contamination under the pole." PCDD/F levels significantly higher than background levels have been detected up to a distance of 50 cm from the poles situated in clay and organic soil. This shows a lateral migration of all the present contaminants, where the migration shows a strong dependence on the type of soil. Karlsson et al. 2013 found that use of PCP-treated wood can contaminate drinking water when near the water table with releases of PCDD/Fs. Lorber et al. 2002 concluded that the size of the dioxin reservoir in PCP-treated utility poles and that even low release rates from these poles have potential for significant environmental releases. This underscores the importance of utility poles as a reservoir source for PCP, dioxins and furans to the environment. In addition to the dioxins and furan releases as described above, approximately 76% of the pentachlorophenol is released to the environment over the life of the treated pole[5], with each pole a significant source of contamination to the environment during its use and becoming hazardous waste upon disposal.

Due to its widespread use and disposal, PCP is detected in air, water, and soil throughout the world, as well as in the blood, urine, seminal fluid, breast milk and adipose tissue of humans (Zheng et al. 2011b). Sandanger et al. (2004) found levels of PCP in blood plasma of the Indigenous Chukotka people of the Russian Arctic The median PCP level was measured at 642 pg g(-1) plasma. Sandau (2002) reported PCP as the dominant chlorinated phenolic compound in blood samples from Nunavik (Inuit people) and southern Quebec adults in Canada. The researchers noted that PCP may supercede HO-PCBs as the chlorinated compound of highest concern in humans. PCP was the dominant organochlorine compound, when compared with PCB and several other phenol compounds, found in the blood serum of pregnant and lactating women in Sweden, analyzed on a wet weight basis up to 3 ng/g serum in early pregnancy. The researchers found that the levels did not change significantly during pregnancy and observed a significant increase from late pregnancy to three weeks after delivery (Larsdotter et al. 2005). In a study of the levels of PCP and other compounds in maternal blood serum, cord blood, and breast milk of women in Sweden, Guvenius et al. (2003) results show that the fetus is likely to be continuously exposed to PCP during development, implicating a risk potential risk for developmental disturbances. PCP was the dominant phenolic compound in the maternal blood plasma, cord blood plasma, and breast milk samples of Swedish women with median levels of 2.83, 1.96, and 0.02 ng/g fresh weight, respectively (n=15), with PCP levels in maternal and cord blood plasma levels 30 and 36 times higher than the sum of OH-PCBs on average. The maternal blood samples had notably high levels. The researchers further conclude that the potential health impacts of halogenated phenolic compounds may have hitherto been underestimated compared with the impact of neutral persistent chemicals. The U.S. Centers for Disease Control National Health and Nutrition Examination Survey (NHANES) III reported that the 95th percentile of PCP concentrations in urine was 1.0-2.0 μg/L in the 1999-2002 survey (Cooper and Jones, 2008). In a study of the levels of PCP in the urine of 197 children in Arkansas, researchers found detectable levels in 100% of the samples, with a median concentration of 14 ppb (Hill et al. 1989).

[5] Bolin CA and ST Smith. 2011. Life cycle assessment of pentachlorophenol-treated wooden utility poles with comparisons to steel and concrete utility poles. Renewable and Sustainable Energy Reviews 15:2475-2486.

In the first population based study investigating plasma concentrations of pentachlorophenol, Rylander et al. (2012) found that PCP was one of the dominant organic contaminants within a representative population of women in Norway sampled in 2004. PCP and p,p'-DDE were the dominating compounds on a wet weight basis and present in considerably higher concentrations than PCBs and other chlorinated pesticides in 311 plasma samples of post-menopausal Norwegian women. "One of the main findings in the current study was that PCP was the second most dominating compound (711 ng/L w.w.) in this group of menopausal women from the general Norwegian population. The levels found were in the same range as samples from Canadian Inuit (801 ng/L, n=567). The authors note that; "High concentrations of PCP in human blood have been reported worldwide (Dirtu et al. 2010; Guvenius et al. 2003) also in Arctic populations (Sandanger et al. 2004) and there are indications that PCP may increase the risk of cancer (Cooper and Jones, 2008; Ruder and Yiin, 2011). Despite that, PCP has received little attention over the past years and to the best of our knowledge, this is the first population based study investigating plasma concentrations of PCP and its predictors." It is interesting to note these findings of PCP in Norwegian women in spite of the fact that PCP is not in use in Norway and that the government of Norway estimates that PCP emissions in Norway have been reduced by 99% in the period from 1995 – 2009.

Glynn et al. (2011) investigated pregnancy-related changes in serum concentrations of five PCB congeners, 3 hydroxylated PCB metabolites and PCP. They found no decline in PCP concentrations measured in the blood serum of women during pregnancy. Dirtu et al. (2010) investigated the levels and profiles of PCBs and phenolic compounds in human blood serum and found that PCP accounted for up to 85% of the total quantified phenolics found in Belgian samples and 35% in Romanian samples. The researchers assert that to some extent, PCP may be formed through the metabolism of HCB, however, this does not explain the high levels of PCP found in the Belgian samples. The median concentration of the Belgian samples was 6,290 pg/g wet weight. In a study of human milk from 50 women in Bratislava, PCP was the dominant chlorophenol, with a median concentration of 2.21 μg/kg of whole milk (Veningerova et al. 1996). In a birth cohort from Slovakia, PCP was more abundant in infant cord blood than any PCB or OH-PCB congener measured with median concentrations in cord blood serum of Σ17 PCBs, Σ6 OH-PCBs, and PCP of 0.92, 0.33, and 0.69 ng/g wet weight, respectively (Park et al. 2008). In a study of the levels of PCP in blood serum of 4-year old children born between 1997 and 1999 in urban and rural communities of Spain, mean levels of PCP were found to be 6.4 ng/mL ± SD of 6.0 (with minimum of 1.5 ng/mL and maximum of 35 ng/mL in the urban population; n=66) and 0.61 ng/mL ± SD of 0.69 (with minimum ND, maximum 4.7 ng/mL in the rural population; n=131). In an exposure assessment of 257 children randomly selected from households and daycare centers in Ohio and North Carolina in the US, PCP was found in nearly all of the urine samples at similar levels found nationally in children ages 6-11, with arithmetic mean levels of 0.605 ng/mL with SD of 0.629 in the 128 children from North Carolina and arithmetic mean levels of 1.27 ng/mL with SD of 2.20 in the 126 children from Ohio. Maximum levels in the children's urine samples were 3.45 ng/mL in North Carolina compared with a maximum level of 23.8 in Ohio. In the same study, PCP was found in more than 50% of the indoor air, outdoor air, and dust samples and children's exposures were predominantly through inhalation (Wilson et al. 2007). Bradman et al. 2003 detected PCP in amniotic fluid of women in California (USA), indicating direct exposure to the young fetus during critical development periods. Prenatal exposure to PCP correlates with worse coordination, less sensory integrity, worse attention, and worse visuomotor integration in children at

school age. These researchers also found that PCP correlated with lower levels of thyroid hormone. Based on their results, the researchers concluded that "unrelenting efforts should be made to find safe alternatives for these compounds (Roze et al. 2009)."

Exposure to PCP is associated with reproductive and developmental toxicities, immunodeficiency, and interference with thyroid hormone. Adverse endocrine effects are summarized in van den Berg (1990),McConnell et al. (1991), Tran et al. (1996), Danzo (1997), and Rawlings et al. (1998), and Kwiatkowski (2012), including: thyroid, anti-estrogen, anti-androgen, anti-progesterone, brain, development, reproduction, corticosterone, insulin, metabolism. PCP affects multiple systems and hormones. In a review by Carrizo et al. (2008), the authors summarize adverse health effects associated with PCP exposure, including: increased lymphocyte responses in patients with high PCP levels in blood serum; adverse neurobehavioral effects including impairment of memory and learning ability; infertility in women; and alteration of thyroid metabolism. Daniel et al. (2001) found immunological abnormalities associated with plasma levels of PCP in individuals with long-term low-dose exposure, including significant associations with cellular and humoral immunodeficiencies. Gerhard et al. (1998) determined an association of higher PCP levels in women in Germany with abnormalities of their menstrual cycle and in an age-adjusted assessment, women had markedly higher levels of PCP if they had at least three miscarriages compared with women with two miscarriages. Duan (2008) found that bisphenol-A and PCP acted synergistically or antagonistically, depending on different endpoints (Li et al. 2010). With partial dechlorination of PCP, more toxic intermediate compounds are produced, such as 2,4,6-trichlorophenol. Toxic effects are "due to the uncoupling of oxidative phosphorylation in mitochondria and generation of reactive oxygen species."

PCP affects the vertebrate endocrine system which may adversely affect the immune system and disrupt sexual development, learning, and behavior. (Ma et al. 2011). In *in vitro* profiling of endocrine-disrupting effects in a set of recombined yeast cell strains, PCP showed agonistic and antagonistic effects at the estrogen receptor (ERα) and antagonistic effects at the progesterone receptor (Li et al. 2010). Ma et al. found that exposure of human adrenocortical carcinoma cell line (H295R cells) to PCP significantly reduced the production of testosterone and 17 β-estradiol. In H295R cells exposed to PCP, there was a dose-dependent reduction of cellular AMP (cAMP), indicating that PCP "may inhibit steroidogenesis by disrupting cAMP signaling." PCP exposure resulted in effects on serum levels of reproductive and metabolic hormones, including marked decreases in thyroxin levels, increases in serum levels of insulin, as well as increases in the severity of oviductal intraepithelial cysts in ewes (Rawlings et al. 1998).

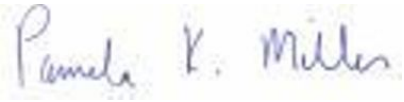
Dallaire et al. (2009) found a significant adverse relationship between material PCP levels and umbilical cord serum T4 concentrations in Inuit mothers from Nunavik and their infants, consistent with the hypothesis that chlorinated phenolic compounds inhibit T4 binding to TTR in humans. Reduced transfer of maternal fT4 during brain maturation of the fetus can impair neurocognitive function in infants. In a study that correlated PCP levels in pregnant women in the Netherlands at 35 weeks (n=90) with sex hormone levels in their boy children at 3 months, Meijer et al. (2012) examined the endocrine-disrupting effects from prenatal exposures to PCP and other organohalogens in infant boys and found that PCP levels correlated positively with sex hormone binding globulin (SHBG)

(p=0.30) and negatively with inhibin B (-0.43). This has implications for the functioning of the Sertoli cells and sexual maturation in these young boys. A study of women daycare workers exposed to PCP, lindane, and PCDFs from treated wood found a significantly reduced birth weight (p=0.04) and length (p=0.02) among their offspring. The detrimental effect could not be attributed to any substance, but to interactions of PCP, HCH, and PCDDs/PCDFs. (Karmaus and Wolf, 1995).

Ruder and Yiin (2011) found an excess of cancers among PCP production workers in the U.S. of *a priori* interest, including non-Hodgkin's lymphoma and leukemia, providing support for the carcinogenicity of PCP. In a review of the available epidemiological literature, researchers found a strong association between PCP exposure and hematopoietic cancer, not likely due to contamination with dioxins or other chlorophenols (Cooper and Jones, 2008). Demers et al. 2006 determined that dermal exposure to PCP among sawmill workers in British Columbia, Canada was associated with non-Hodgkin's lymphoma, multiple myeoma, and kidney cancer. Heacock et al. found an elevated risk of brain, ovarian, and eye cancer among girl children of British Columbia sawmill workers. Dimich-Ward et al. found an elevated risk of congenital anomalies of the eye (particularly congenital cataracts) and genital organs; as well as anencephaly or spina bifida among children born to fathers exposed to chlorophenate wood preservatives in the sawmill industry, including PCP. Wang et al. (2012) note that tetrachlorohydroquinone (TCHQ) is a major toxic metabolite of PCP and has "also been implicated in PCP genotoxicity." The authors conclude that TCHQ induces sequence-specific DNA mutations at high frequencies. PCP induced adverse changes in human lymphocyte morphology, severely increasing size and granularity of the cells and depleting intracellular ATP levels, with high loss of lymphocyte viability (Michalowicz, 2010). Lymphocytes that function as natural killer cells capable of killing tumor cells, viral-infected cells, and antibody-coated cells are significantly decreased by exposure to PCP, thus adversely affecting a critical immune defense and perhaps playing a role in the development of cancers associated with PCP exposures (Ndodu and Whalen, 2010). Zheng et al. (2012) conducted a systematic review of PCP in the environment and humans and concluded: "Thyroid-disrupting effects and cancer risk caused by PCP and PCDD/Fs even at low environmental levels…are of concern."

In my view, the poles represent a serious health hazard to local residents and should be removed.

Sincerely,



Pamela Miller
Executive Director