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February 17, 2015

Office of Pesticide Programs
Environmental Protection Agency,
Mailcode 28221 T,
1200 Pennsylvania Ave, NW,
Washington, DC 20460

Re: Pentachlorophenol Preliminary Work Plan. Docket No: EPA-HQ-OPP-2014-0653

Dear Sir/Madam,

The U.S. Environmental Protection Agency (EPA) has opened the docket initiating the registration review of pentachlorophenol (PCP). We would like to take this opportunity to submit comments we believe the agency must seriously take into consideration during its review of pentachlorophenol's use as a wood (utility) pole preservative. At the conclusion of this registration review, we urge the agency to take immediate action to revoke the registration of PCP under the terms set out in Section 3(c)(7)(A) of the *Federal Insecticide Fungicide and Rodenticide Act* (FIFRA). Continued registration poses "unreasonable adverse effects on the environment," given the serious human and environmental health risks associated with its continued use as a wood preservative in the U.S. and in light of the numerous alternative options for utility poles and railroad ties.

Beyond Pesticides' History with Wood Preservatives

Beyond Pesticides has a long history tracking pentachlorophenol's use as a wood preservative and its regulatory history. We have published two reports, *Poison Poles* and *Pole Pollution*, which address the use of wood preservatives on utility poles.[1] The first report, *Poison Poles*, published in 1997, examines the toxic trail left by the manufacture, use, storage and disposal of the heavy-duty wood preservatives from cradle to grave. *Pole Pollution*, published in 1999, focuses on EPA's draft preliminary science chapter on PCP and provides the results of our

[1] Feldman, J and Kidd, G. 1999. Pole Pollution. Beyond Pesticides. Washington DC; Feldman, J and Shistar, T. Poison Poles. 1997. Beyond Pesticides. Washington DC.

survey of over 3,000 utilities across the United States and Canada. Additionally, we have worked with elected officials and regular citizens, as well as the media, to educate people about the extreme risks associated with exposure to these pesticides. In 2002, a lawsuit was filed in federal court by a national labor union, environmental groups, including Beyond Pesticides, and a victim family to stop the use of PCP, arsenic and creosote preservatives used to treat utility poles, railroad ties, and, at the time, lumber. The litigation charged that the chemicals, including pentachlorophenol, are known carcinogenic agents, contaminated with dioxins, furans, and hexachlorobenzene, hurt utility workers exposed to treated poles, children playing near treated structures, and the environment, and cited the availability of alternatives. The lawsuit was dismissed in 2005 in U.S. District Court on procedural grounds.

One major source of contention at the time was EPA's estimate of children's exposure risks. EPA initially estimated that children's residential post-application exposures resulting from widespread use of PCP-treated utility poles posed an unacceptable cancer risk (2.2 cancer cases in 10,000). This was more than 200 times above EPA's acceptable threshold. However, instead of addressing the need to protect children in 2004, EPA revised its assessment stating that this exposure does not occur, a claim provided to EPA industry. EPA states, "The opportunity for residential consumer contact is limited since PCP wood is not sold to the general public. . .Where utility poles are installed on home/school or other residential sites, child contact via the dermal or oral routes is not anticipated since play activities with or around these pole structures would not normally occur."[2] Beyond Pesticides contends that this exposure pathway is real and should be accounted for, not discounted, in PCP or any other assessment of utility poles. Children can and do play around utility poles in schoolyards, playgrounds and homes or along residential streets. Treated poles lining the streets of many urban and suburban areas or near and next to bus stops, find people leaning against and standing next to these poles. These exposure scenarios should and must be taken into account by the agency in its risk assessment.

Pentachlorophenol: Human Health Concerns

A. Carcinogenicity:

Pentachlorophenol (PCP) is a chlorinated aromatic hydrocarbon closely related to other chlorophenols. Impurities in technical grade PCP, which may include tetrachlorophenol, trichlorophenols, hexachlorobenzene, polychlorinated dibenzo-*p*-dioxins, polychlorinated dibenzo-furans, and chlorinated phenoxyphenols, are contributors to the compound's toxicity. All of these substances can be found in technical grade PCP. Hexachlorobenze is listed as a probable human carcinogen.[3] The possible human health impacts of polychlorinated dibenzo-p-

[2] USEPA. 2008. Revised PCP Human Exposure RED Chapter. Office of Pesticide Programs. Washington DC
[3] UESPA. Hexachlorobenzene. Hazard Summary. http://www.epa.gov/airtoxics/hlthef/hexa-ben.html

dioxins and furans have long been evaluated.[4] Pentachlorophenol itself is also listed as a Group C- Possible Human Carcinogen.[5]

According to EPA, PCP is a highly toxic pesticide. EPA notes:

> *"Pentachlorophenol is extremely toxic to humans from acute (short-term) ingestion and inhalation exposure. Acute inhalation exposures in humans have resulted in neurological, blood, and liver effects, and eye irritation. Chronic (long-term) exposure to pentachlorophenol by inhalation in humans has resulted in effects on the respiratory tract, blood, kidney, liver, immune system, eyes, nose, and skin. Human studies are inconclusive regarding pentachlorophenol exposure and reproductive effects. Human studies suggest an association between exposure to pentachlorophenol and cancer. Oral animal studies have reported increases in liver tumors and two uncommon tumor types. EPA has classified pentachlorophenol as a Group B2, probable human carcinogen."*[6]

Additionally, the International Agency for Research on Cancer (IARC) has classified PCP as "*possibly carcinogenic to humans."* Just recently in 2014, PCP was added to the National Toxicology Program's (NTP) *13th Report on Carcinogens* as "*reasonably anticipated to be a human carcinogen,*" citing its association with an increased risk of non-Hodgkin lymphoma in studies in humans and the incidence of tumors in the liver and other organs in mice.[7]

According to EPA's Integrated Risk Information System (IRIS),[8] oral studies of PCP carcinogenicity demonstrate that tumors can occur in tissues remote from the site of absorption, including the liver, adrenal gland, circulatory system, and nose. Even though specific inhalation and dermal studies have not been conducted for PCP, EPA's IRIS report has determined that PCP is readily absorbed via all routes of exposure, including oral, inhalation, and dermal. Furthermore, based on the observance of systemic tumors following oral exposure, and in the absence of information to indicate otherwise, EPA has also assumed that internal exposures/residues will be achieved regardless of the route of exposure. Accordingly, the agency believes that PCP can be considered "*likely to be carcinogenic to humans*" by all routes of exposure.

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[4] McGregor, DB, Partensky, C, Wilbourn, J and Rice, JM. 1997. An IARC evaluation of polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans as risk factors in human carcinogenesis. Environ Health Perspect. 1998 Apr; 106(Suppl 2): 755–760.

[5] USEPA. Chemicals Evaluated for Carcinogenic Potential. Updated October 2014. Office of Pesticide Programs. Washington DC.

[6] USEPA. Pentachlorophenol. Hazard Summary-Created in April 1992; Revised in January 2000. Technology transfer Network, Air Toxics Web site. Available at http://www.epa.gov/ttn/atw/hlthef/pentachl.html

[7] National Toxicology Program. 2014. 13th Report on Carcinogens. National Institutes of Health (NIH) http://ntp.niehs.nih.gov/go/roc13

[8] USEPA. IRIS Summaries: Pentachlorophenol (CASRN 87-86-5). Available at http://www.epa.gov/iris/subst/0086.htm

B. Acute and Chronic Toxicity:

There is extensive documentation of the acute and chronic toxic effects of PCP. Inhalation and dermal exposures are to be expected with use of PCP as a wood preservative for utility poles, and people who live or work near to treated poles have an elevated risk of being exposed. Skin, eye, and respiratory irritation are typical acute exposure symptoms.[9] Studies find that long-term low and elevated exposures to PCP can cause damage to the liver, kidneys, blood, and nervous system. Laboratory animal studies also suggest that the endocrine system and immune system can also be damaged following long-term exposure to low levels of PCP.[10] Additionally, IRIS data report that there is evidence, both in subcellular systems and in human cells in vitro, that PCP induces damage to DNA and proteins. Gene mutation and a weakly positive indication of trans-placental mutation in mice have been observed with PCP.[11]

Other animal studies on reproductive toxicity in rats show that exposure to PCP is associated with decreased fertility, delayed puberty, testicular effects, decreased litter size, decreased viability, and decreased pup weights.[12] Additionally, PCP can act as an endocrine disruptor by affecting the levels of circulating thyroid hormones.[13,14] One 2011 study found that PCP significantly decreased production of the hormones, testosterone and 17β-estradiol, and may inhibit steroidogenesis (production of steroid hormones).[15]

C. Body Burden:

The Centers for Disease Control and Prevention's (CDC) *National Report on Human Exposures to Environmental Chemicals* notes that acute, high dose exposure to PCP can "induce a hypermetabolic state and excessive heat production.." in the body, with effects that include hyperthermia, hypertension, and metabolic acidosis. These have been observed in adults and children highly exposed to PCP through ingestion, inhalation, or skin absorption. This CDC report, which measures levels of environmental chemicals in human compartments, finds that while PCP levels in adults and children are lower than they have been in the past, (mostly due

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[9] Karlsson, L, Cragin, L, Center, G et al. 2013 Pentachlorophenol Contamination of Private Drinking Water From Treated Utility Poles. American Journal of Public Health: 103(2):276-277. doi: 10.2105/AJPH.2012.300910

[10] Agency for Toxic Substances and Disease Registry. 2001. Toxicological Profile for Pentachlorophenol. U.S. Department of Health and Human Services.

[11] USEPA. IRIS Summaries: Pentachlorophenol (CASRN 87-86-5). Available at http://www.epa.gov/iris/subst/0086.htm

[12] USEPA. IRIS Summaries: Pentachlorophenol (CASRN 87-86-5). Available at http://www.epa.gov/iris/subst/0086.htm

[13] USEPA. IRIS Summaries: Pentachlorophenol (CASRN 87-86-5). Available at http://www.epa.gov/iris/subst/0086.htm

[14] Guo, Y and Zhou, B. 2013. Thyroid endocrine system disruption by pentachlorophenol: An *in vitro* and *in vivo* assay. *Aquatic Toxicology*. 142–143:138–145.

[15] Ma Y, Liu C, Lam PK, et al. 2011. Modulation of steroidogenic gene expression and hormone synthesis in H295R cells exposed to PCP and TCP. *Toxicology.* 282(3):146-53.

to regulatory restrictions), residues are still detected/measurable in the U.S. population.[16] Continuing residues of this highly toxic compound and its contaminants in the human body, which are attributable to ongoing and past uses, represent an unacceptable exposure hazard, one that regulators should seek to eliminate.

A recent study from EPA researchers document similar findings regarding PCP levels in children.[17] In this study, monitoring was performed at 115 homes and 16 daycares in Ohio of preschool children, ages 2–5 years old. It finds that the median levels of urinary PCP were slightly higher for children that stayed at home (0.91 ng/mL) compared to children that went to daycare (0.77 ng/mL). In addition, the maximum PCP concentration of 23.8 ng/mL occurred for one child in the home group of children. In addition, urinary levels of PCP were significantly higher in children that spent more than two hours outside for children that lived in homes more than 15 years old, and for those that lived with a pet dog/cat. Further, this study analyzed and detected levels of PCP (0.43 ng/m^3 and 0.22 ng/m^3) in the outdoor air samples at the children's homes and daycare centers, respectively, which according to the researchers, "were the highest outdoor air levels reported among all measured chemicals."

Most importantly, the median PCP levels detected in this study are higher than those reported in the CDC's report mentioned above for older children (6–11 years old). Note: the CDC report does not include findings for children under 6 years. The median level for these older children was <0.5 ng/mL. Another study investigating the potential exposures of 257 preschool children, ages 1 1/2-5 years, found that potential exposures to PCP were predominantly through inhalation where the estimated exposures to PCP (based on urine analysis) exceeded their estimated maximum potential intake.

Overall, these above data indicate that children in the U.S. are still being exposed to PCP long after residential uses were removed from the market. While it is still somewhat unclear what the exposure pathways are, however, given that PCP exposures in the above studies were associated with pets and spending time outside, it should be assumed without data to the contrary that outdoor residues of PCP, either from volatilizing treated-poles or other older treated surfaces or contaminated soil, still plays a role in human exposures to PCP. EPA has in the past determined that contact with soil contaminated with PCP, as well as residential contact with treated wood products like utility poles poses an unacceptable cancer risk to children.[18] EPA has since stated that children are not exposed to utility poles and thus expect no exposure

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[16] CDC. 2009. Fourth National Report on Human Exposures to Environmental Chemicals. Department of Health and Human Services. Centers for Disease Control and Prevention.

[17] Morgan, M, Jones, P and Sobus, J. 2015. Short-Term Variability and Predictors of Urinary Pentachlorophenol Levels in Ohio Preschool Children. *Int. J. Environ. Res. Public Health*, *12:* 800-815;

[18] Feldman, J and Kidd, G. 1999. Pole Pollution. Beyond Pesticides. Washington DC.

risks. But as these studies show, children who play outside their homes or in their neighborhoods face higher risk of PCP exposure, and acute and chronic adverse effects.

D. Occupational Exposures:

Workers at sawmills and other wood facilities have been found to have elevated risks of non-Hodgkin's lymphoma, multiple myeloma, and kidney cancer,[19,20,21] and have elevated serum levels of polychlorinated dibenzo-*p*-dioxins and furans.[22,23] One review finds that there is evidence that occupational exposure of workers to PCP might increase the risk of lymphoma and hematopoietic neoplasm in themselves and in their children.[24] The science has shown for decades that PCP and its contaminants pose unacceptable risks to workers. The agency now anticipates receiving new occupational studies for PCP use (inhalation and dermal exposures from pressure treatment exposure and pole lineman). We hope the agency considers the wealth of independent, peer-review data and not rely on industry science to conduct its review.

Major Risks from PCP Contaminants:

A. Toxicity of Hexachlorobenzene:

Hexachlorobenzene is typically formed as a byproduct during the manufacture of other chemicals (mainly solvents) and pesticides. As a contaminant of PCP, it is detected in environmental compartments.[25] According to the Agency for Toxic Substances and Disease Registry (ATSDR), hexachlorobenzene is detected in soil and sediment samples, and surface, ground and drinking water.[26] In water, the chemical is slow to break down and is bioaccumulated by aquatic organisms, while in soil it can take years to break down. Due to its persistent nature and its ability to bioaccumulate, there is concern that exposures via contaminated water, soil, fish and even breast milk may put public health at risk. Like other

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[19] Demers PA, Davies HW, Friesen MC et al. 2006. Cancer and occupational exposure to pentachlorophenol and tetrachlorophenol (Canada). *Cancer Causes Control.* 17(6):749-58.
[20] Collins, J, Bodner, K, Aylward, L , et al. 2009. Mortality Rates Among Workers Exposed to Dioxins in the Manufacture of Pentachlorophenol. *J Occupational & Environmental Medicine*. 51(10):1212-1219.
[21] Ruder, A. Webinar entitled: Occupational Exposure to Pentachlorophenol and Other Agents. http://ntp.niehs.nih.gov/ntp/roc/thirteenth/webinars/2013/3_ruder_508.pdf.
[22] Karouna-Renier , N, Ranga Rao, K, et al. 2007. Serum profiles of PCDDs and PCDFs, in individuals near the Escambia Wood Treating Company Superfund site in Pensacola, FL. *Chemosphere*. 69(8):1312–1319.
[23] Mari M, Nadal M, Schuhmacher M, Domingo JL. 2013. Body burden monitoring of dioxins and other organic substances in workers at a hazardous waste incinerator. *Int J Hyg Environ Health*. 216(6):728-34.
[24] Zheng R, Zhang Q, et al. 2013. Occupational exposure to pentachlorophenol causing lymphoma and hematopoietic malignancy for two generations. *Toxicol Ind Health.* doi: 10.1177/0748233712472520.
[25] Blocksom KA, Walters DM, et al. 2010. Persistent organic pollutants in fish tissue in the mid-continental great rivers of the United States. *Sci Total Environ.* 408(5):1180-9.
[26] ATSDR. 2013. DRAFT TOXICOLOGICAL PROFILE FOR HEXACHLOROBENZENE. Division of Toxicology and Human Health Sciences . U.S. Department Of Health And Human Services. Public Health Service. Atlanta, GA.

persistent organic pollutants, hexachlorobenzene has been detected in the blood of numerous groups of people, especially indigenous populations in the Arctic regions, like Alaska.[27] In fact, fish from these regions are frequently found to be contaminated with hexachlorobenzene and other persistent organic pollutants.[28]

Hexachlorobenzene has been detected in maternal and cord sera,[29] and has been linked to hypertrophy of the thyroid gland and altered thyroid function.[30] Higher levels of hexachlorobenzene were found in the fat of boys with cryptorchidism (undescended testis) than in the fat of boys without this defect.[31] In animal studies, perinatal exposure of rats to hexachlorobenzene followed by dietary exposure for up to 130 weeks caused benign liver tumors in females, benign parathyroid-gland tumors (adenoma) in males, and benign adrenal-gland tumors in both sexes.[32] Hexachlorobenzene is linked to increased risk for tumor growth and the onset on cancer,[33,34,35] and may have additive and synergistic effects with other organochlorine chemicals.[36]

B. Toxicity of Polychlorinated Dibenzo-*p*-Dioxins and Furans:

It has been well documented that polychlorinated dioxins and furans are persistent, bioaccumulative and associated with adverse health outcomes. The most potent dioxin, 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), is a known carcinogen and highly toxic. While modern manufacturing processes have reduced the levels of these contaminants, they still pose risks to human and environmental health. The scientific database on these substances is extensive and the agency is aware of the concerns surrounding their continued persistence as contaminants in

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[27] National Toxicology Program. 2011. Hexachlorobenzene. Report on Carcinogens, 13th Edition.
[28] Hardell S, Tilander H, et al. 2010. Levels of polychlorinated biphenyls (PCBs) and three organochlorine pesticides in fish from the Aleutian Islands of Alaska. *PLoS One.* 5(8):e12396.
[29] Li,C. et al. 2014. The association between prenatal exposure to organochlorine pesticides and thyroid hormone levels in newborns in Yancheng, China. *Environ Res*. 129:47-51.
[30] Chalouati H[1], Gamet-Payrastre L, Ben Saad M. 2013. Irreversible thyroid disruption induced after subchronic exposure to hexachlorobenzene in male rats. *Toxicol Ind Health* doi:10.1177/0748233713511511.
[31] ATSDR. 2013. DRAFT TOXICOLOGICAL PROFILE FOR HEXACHLOROBENZENE. Division of Toxicology and Human Health Sciences . U.S. Department Of Health And Human Services. Public Health Service. Atlanta, GA.
[32] National Toxicology Program. 2011. Hexachlorobenzene. Report on Carcinogens, 13th Edition.
[33] Pontillo CA**,** Rojas P**,** Chiappini F, et al. 2013. Action of hexachlorobenzene on tumor growth and metastasis in different experimental models. *Toxicol Appl Pharmacol.* 268(3):331-42.
[34] Ociepa-Zawal M, Rubis B, et al. 2010. Accumulation of environmental estrogens in adipose tissue of breast cancer patients. *J Environ Sci Health A Tox Hazard Subst Environ Eng*. 45(3):305-12.
[35] García MA, Peña D, Alvarez L, et al. 2010. Hexachlorobenzene induces cell proliferation and IGF-I signaling pathway in an estrogen receptor alpha-dependent manner in MCF-7 breast cancer cell line. *Toxicol Lett.* 192(2):195-205.
[36] Abdo W, Hirata A, Sakai H, et al. 2013. Combined effects of organochlorine pesticides heptachlor and hexachlorobenzene on the promotion stage of hepatocarcinogenesis in rats. *Food Chem Toxicol*. 55:578-85.

PCP manufacture and in the environment. Much of the toxicity of PCP is attributed to its contamination with chlorinated dioxins and furan (as well as hexachlorobenzene).

EPA identifies dioxins as a cause of impairment in over 600 waterways across the U.S.[37] Subsequently, fish consumed from these impaired waterways present dietary risks to public health. Adverse health outcomes associated with these substances include carcinogenicity, teratogenicity and endocrine disruption,[38] and even lower birth weights.[39] Introducing dioxins and furans into the environment via PCP use as wood treatment is unreasonable and unacceptable given the long-term risks and the cost of disposal and clean-up at contaminated sites.

Pentachlorophenol: Environmental Risks

As used as a wood preservative treatment for utility poles, it is expected that human and environmental exposures will occur. PCP is released into the air from treated wood surfaces. While this phenomenon (off-gassing or volatilization) would not result in large ambient concentrations of PCP in the atmosphere, residues quickly bind to soil and can make their way into surface and ground waters,[40] where they can persist and accumulate in fish and other organisms. Increased temperature and leaching from rain will influence PCP migration from utility poles to surrounding air and soil. PCP's major metabolite pentachloroanisole (PCA) is also highly toxic. According to data compiled by the National Toxicology Program (NTP), there is evidence of the carcinogenic activity of PCA.[41]

Environmental monitoring has demonstrated that PCP exists in various environmental compartments (i.e. water, soil, sediment, and aquatic organisms) and even humans (as noted above). PCP is also a common contaminant in water,[42] and studies with fish finds that PCP acts as an endocrine disruptor, eventually resulting in abnormal fish development.[43,44]

In one study, field soil samples were collected around six PCP-treated wooden poles (in clay, organic soil, and sand) and found concentrations of polychlorodibenzo-*p*-dioxins and furans the

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[37] USEPA. 2014. Pentachlorophenol Preliminary Work Plan. Office of Pesticide Programs. Washington DC.
[38] Agency for Toxic Substances and Disease Registry. 1999. Toxicological Profile For Chlorophenols. U.S. Department Of Health And Human Services.
[39] Wohlfahrt-Veje, C. et al. 2014. Polychlorinated dibenzo-p-dioxins, furans, and biphenyls (PCDDs/PCDFs and PCBs) in breast milk and early childhood growth and IGF1. *Reproduction 147 (4) 391-399.*
[40] Agency for Toxic Substances and Disease Registry. 2001. Toxicological Profile for Pentachlorophenol. U.S. Department Of Health And Human Services.
[41] NTP. 1993. NTP Toxicology and Carcinogenesis Studies of Pentachloroanisole (CAS No. 1825-21-4) in F344 Rats and B6C3F1 Mice (Feed Studies). Natl Toxicol Program Tech Rep Ser. 414:1-284.
[42] Yu, L.-Q., Zhao, G.-F., Feng, M., et al. 2014. Chronic exposure to pentachlorophenol alters thyroid hormones and thyroid hormone pathway mRNAs in zebrafish. Environmental Toxicology and Chemistry, 33: 170–176. doi: 10.1002/etc.2408.
[43] National Toxicology Program. 1993. NTP Toxicology and Carcinogenesis Studies of Pentachloroanisole (CAS No. 1825-21-4) in F344 Rats and B6C3F1 Mice (Feed Studies). Natl Toxicol Program Tech Rep Ser. 414:1-284.
[44] Zhang, X, Xiong, L, et al. 2014. Histopathological and estrogen effect of pentachlorophenol on the rare minnow (*Gobiocypris rarus*). *Fish Physiology and Biochemistry*. 40(3):805-816.

highest on organic and clay soils. The study also found that high levels of polychlorodibenzo-*p*-dioxins and furans can be found in the first 2 meters below the surface.[45] In a follow-up study by the same authors, a screening model to predict the influence of PCP pole-treating oil on the vertical migration of its impurities was evaluated.[46] Soil samples were analyzed to determine concentrations of polychlorodibenzo-*p*-dioxins and furans at several depths in soil. Here the authors observed a significant vertical migration of polychlorodibenzo-*p*-dioxins and furans at various depths which they attribute to the influence of the carrier oil mixed with PCP. Another study, which appeared in the *American Journal of Public Health,* finds that treated utility pole placement near private water sources can increase the likelihood of drinking water contamination, especially in areas with high water tables, and with poles placed on road rights-of-ways.[47] According to this study, which was based in Vermont, tested water samples had a PCP concentration of 2.06 mg/L, and 1.15 mg/L, about 2000 and 1000 times the EPA maximum contaminant level (0.001 mg/L). In this case, treated poles were eventually replaced with non-treated cedar poles.

Given PCP's potential to migrate off treated wood and into soil, pole placement near drinking water sources can increase the likelihood of drinking water contamination. Even though the solubility of the dioxin/furan contaminants is very low, these contaminants are readily sorbed onto soil particles and other surfaces. Surface waters with significant quantities of organic or clay particles can easily transport dioxins/furans to distant sites from their origin.[48] For instance, PCP has been found in groundwater in several locations throughout Minnesota, including in some private drinking water wells near contaminated sites.[49]

Regulatory Status:

The last reregistration for PCP concluded in 2008.[50] PCP must now undergo registration review, which must be completed by 2022. EPA announced on December 19, 2014, that it was opening registration review for several active ingredients, including PCP.[51] PCP has a long history of use in the U.S. Due to its potent toxicity, residential uses have long been cancelled. In 2004, non-

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[45] Bulle, C., Samson, R. and Deschênes, L. 2010. Enhanced migration of polychlorodibenzo-*p*-dioxins and furans in the presence of pentachlorophenol-treated oil in soil around utility poles: Screening model validation. *Environmental Toxicology and Chemistry*. 29: 582–590.

[46] Bulle CS, Samson R, Deschênes L. 2011. Transport of chlorinated dioxins and furans in soil columns: modeling pentachlorophenol pole-treating oil influence. *Chemosphere*. 83(2):117-23.

[47] Karlsson, L, Cragin, L, Center, G et al. 2013 Pentachlorophenol Contamination of Private Drinking Water From Treated Utility Poles. American Journal of Public Health: 103(2):276-277. doi: 10.2105/AJPH.2012.300910

[48] WHO. 2003. Pentachlorophenol in Drinking-water Background document for development of WHO Guidelines for Drinking-water Quality. Originally published in Guidelines for drinking-water quality, 2nd ed. Addendum to Vol. 2. Health criteria and other supporting information. World Health Organization, Geneva.

[49] MDH. 2013. Pentachlorophenol and Drinking Water. http://www.health.state.mn.us/divs/eh/risk/guidance/gw/pcpinfosht.pdf.

[50] USEPA. 2008. Reregistration Eligibility Decision for Pentachlorophenol. Office of Pesticide Programs. Washington DC. http://www.epa.gov/oppsrrd1/reregistration/REDs/pentachlorophenol_red.pdf.

[51] *See* Registration Reviews; Pesticide Dockets Opened for Review and Comment, 79 Fed. Reg. 75801, Dec. 19, 2014, EPA–HQ–OPP–2014–0763, file:///C:/Users/asimpson/Downloads/EPA-HQ-OPP-2014-0762-0001.pdf.

pressure and non-thermal treatment uses of PCP (e.g., spray uses) were deleted from labels, leaving only pressure and thermal treatments.

However, PCP remains a potent toxicant and no longer serves a necessary benefit to society. As a result of its toxicity and presence in the environment, PCP and its salts are being considered for listing under the Stockholm Convention as a “persistent organic pollutant” (POP) to be targeted for worldwide phase out.[52] In October 2014, the Persistent Organic Pollutants Review Committee recommended that PCP be targeted for global elimination and be listed as a POP due to its persistence, bioaccumulation, long-range transport, and toxic impacts. In accordance with Article 8 of the Convention, the Committee decided that, “Pentachlorophenol and its salts and esters is likely, as a result of its long-range environmental transport, to lead to significant adverse human health and/or environmental effects such that global action is warranted.”[53]

While the U.S. is not a signatory to the Stockholm Convention, it is the largest producer and user of PCP in the world. Unfortunately, EPA, as the U.S.’ representative at the recent convention meeting, sought to oppose efforts by other nations at the convention to have PCP listed for global elimination, even though at the same time the U.S. Department of Health and Human Services included PCP in its *13th Report on Carcinogens*, declaring PCP, “reasonably anticipated to be a human carcinogen.”

While the rest of the world moves forward with eliminating this toxic and persistent chemical from commerce in order to protect their citizens and the environment, the U.S., at the direction of EPA, continues to lag behind when it comes to protecting human health and the environment.

Alternatives are Feasible and Available:

It is important for the agency to be aware that there are numerous alternatives to PCP-treated utility poles and that current use of PCP to treat these poles cannot be considered a necessity. Several effective and economical alternatives to PCP that were not readily available years ago now exist.

The alternative technologies for PCP-treated wood include recycled plastics, composites, recycled steel, concrete, and a number of new wood preserving chemicals that contain neither

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[52] Stockholm Convention. Chemicals proposed for listing under the Convention. Châtelaine, Switzerland. http://chm.pops.int/TheConvention/ThePOPs/ChemicalsProposedforListing/tabid/2510/Default.aspx.

[53] UNEP. 2014. Invitation to review and provide comments on the draft risk management evaluation for pentachlorophenol and its salts and esters. SC/POPRC. Secretariat of the Stockholm Convention. Geneva, Switzerland. http://chm.pops.int/TheConvention/POPsReviewCommittee/Meetings/POPRC9/POPRC9Followup/tabid/3502/Default.aspx#LiveContent[POPRC9_CoPCP]

arsenic, chromium nor PCP including: Alkaline Copper Quaternary (ACQ), Copper Azole, Copper-8-quinolinate, and borate-based wood preservatives. Manufacturers of these alternative technologies can expect to experience an increased share of the market and eventually replace those structures and applications where PCP-treated wood is currently used.

In the case of wood, the utility industry expects 40 to 50 total years of service (although it is not uncommon for batch of wood to yield less than 35 years of service). The steel, concrete, and composite alternatives yield a lifespan of 80 to 100 years. There are differences in maintenance costs associated with different materials. For example, wood may require retreatment, which some utilities perform on a set cycle, while steel, concrete and fiberglass do not. In addition, disposal costs for chemicals used in wood treatment are high and growing, while steel is recycled.

Steel is viewed as the most common alternative utility pole material. Steel alternatives represent a small but growing alternative when compared with the use of treated wood utility poles. Steel is lighter and sturdier and more fire-proof. Alternative woods like Redwood, cedar, and cypress are naturally pest and decay resistant and are good options for residential users. Reinforced concrete has also been identified as an alternative material to treated wood poles. The material's longevity ranges from 80 to 100 years. There are a number of other materials that are available for poles, such as fiberglass reinforced composite. Additionally, there is the option of burying utility lines underground.

One of the arguments used against non-wood alternatives to treated wood is that it will require a retraining of utility linemen and thus pose an occupational danger to them since they are not used to working with non-wood poles. It is true that job-training will be required of any switch to non-wood utility poles, however the occupational risk posed to linemen by working with poles treated with toxic chemicals far outweighs the risk of switching to a new product and requiring re-training.

The primary argument used against alternatives to treated-wood, especially utility poles and railroad ties, is that their cost is prohibitive. Similarly, industry-sponsored studies have reported that life cycle assessments for treated-wood poles versus steel and concrete alternatives point to treated-wood poles as the preferred alternative.[54,55] These assessments factor in fossil fuel use, water use, ecological impact and greenhouse gas emissions. What these studies do not take into account are the human health risks and long-term, persistent contamination

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[54] Bolin, C and Smith, S. 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.

[55] Treated Wood Council. 2013. Conclusions and Summary Report Environmental Life Cycle Assessment of Railroad Ties. http://www.rta.org/assets/docs/ResearchPapersArticles/Miscellaneous/final%20-%20ties%20lca%20summary%202013-04-30.pdf

associated with the use of treated wood. Another study, this time for the life cycle assessment of steel finds that steel poles produce less treated waste, are less disruptive of and does not contribute to habitat loss in forest areas, and produce lower levels of greenhouse emissions associated with climate change.[56]

In addition, arguments against non-chemical/non-wood alternatives often fail to take into account differences in the lifespan of treated wood versus concrete or recycled composite/steel poles, and the fact that with some alternatives, such as steel and concrete, fewer poles or ties per mile are needed than for treated wood ones. In an assessment of alternatives reported by the Persistent Organic Pollutants Review Committee,[57] it is noted that while upfront costs may be higher for non-chemical alternatives than treated wood, the lighter weight and longer lifespan of the non-chemical alternatives make costs competitive overall. For instance, steel poles are of comparative price to treated wood when assessed for full life span and reduced maintenance costs. It should also be noted that concrete and steel products can be recycled, whereas PCP-treated wood must be treated as hazardous waste at disposal.

Conclusion:

PCP is an old generation persistent organic pollutant whose use as a wood preservative is not necessary and risks unreasonable under FIFRA. This substance, by nature of its contamination with hazardous toxicants like hexachlorobenzene, dioxins and furans, has risks that far outweigh any perceived benefit given the availability of alternatives. PCP is associated with numerous adverse health outcomes and environmental impacts. As a result, it has been targeted for global elimination on the international stage. The U.S., represented by EPA, must take the lead in establishing permanent phase out of this chemical.

During this registration review, EPA must find that the risks to workers, residential bystanders, and the environment from unintended dermal, inhalation and dietary exposures pose unacceptable risks under Section 3(c)(7)(A) of the *Federal Insecticide Fungicide and Rodenticide Act* (FIFRA), in that registration poses “unreasonable adverse effects on the environment.”

We appreciate your consideration of our comments.

[56] SCS Global Services. 2013. Life Cycle Assessment of Southern Yellow Pine Wood and North American Galvanized Steel Utility Distribution Poles. Scientific Certification Systems, Emeryville CA

[57] Stockholm Convention on Persistent Organic Pollutants. Pentachlorophenol And Its Salts And Esters: Risk Management Evaluation. Report of the Persistent Organic Pollutants Review Committee on the work of its tenth meeting. Stockholm Convention on Persistent Organic Pollutants. Rome, 27–30 October 2014

Sincerely,

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Science and Regulatory Director