EARTHJUSTICE

ALASKA CALIFORNIA FLORIDA MID-PACIFIC NORTHEAST NORTHERN ROCKIES
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Comments of Sierra Club, California Communities Against Toxics, Gasp, and the Kentucky Environmental Foundation in Support of EPA's Notice Proposing to Grant the Petitions to Add n-Propyl Bromide (also known as 1-Bromopropane) to the List of Hazardous Air Pollutants Under Clean Air Act § 112(b)

Dear Administrator Pruitt:

Earthjustice submits these comments on behalf of Sierra Club, California Communities Against Toxics, Gasp, and the Kentucky Environmental Foundation ("Commenters"), in support of EPA's Notice of Granting Petitions to Add n-Propyl Bromide to the List of Hazardous Air Pollutants, 82 Fed. Reg. 2354 (Jan. 9, 2017) ("Notice").

INTRODUCTION AND SUMMARY

In 2010 and 2011, the New York State Department of Environmental Conservation ("NYSDEC") and the Halogenated Solvents Industry Alliance ("HSIA") filed petitions with EPA, urging the agency to add n-Propyl Bromide ("nPB") to the Clean Air Act's ("CAA" or "the Act") list of hazardous air pollutants ("HAPs"). *Id.* at 2358. In 2015, after years of review of the petitions and supporting data, including supplemental data provided by the petitioners to aid the agency, EPA made a determination that the petitions are complete. *Id.* EPA then took one round of public comment on the petitions and current science, before reaching its decision to grant these petitions. *Id.* The current Notice explains EPA's rationale for granting the petitions.

EPA has correctly determined that there is adequate evidence to support a determination that nPB is an air pollutant and that emissions and ambient concentrations of nPB may reasonably be anticipated to cause adverse effects to human health. *Id.* at 2362; 42 U.S.C. § 7412(b)(3)(B)) (stating that these factors, and/or evidence of bioaccumulation or deposition require listing).

In accordance with this finding, Commenters urge EPA to publish a final decision to grant these petitions and add this dangerous and unnecessary synthetic chemical to the list of HAPs in Clean Air Act section 112(b)(1). Listing nPB will serve EPA's core mission in

administering the Act, which includes preventing air pollution and securing clean air to protect the health and welfare of all Americans. 42 U.S.C. § 7401(b)(1), (c). Listing this chemical as a HAP will also remove the unfair advantage that users and producers of nPB now enjoy over their competitors who use or produce other chemicals recognized as hazardous, even though nPB is at least as harmful as those chemicals.[1]

Like other HAPs currently listed under Clean Air Act § 7412(b)(1), nPB is known to cause serious acute and chronic human health effects. The scientific record for this determination demonstrates that nPB is a reproductive toxicant, carcinogen, and a cause of neurological harm and related chronic health problems including depression. Any one of these documented actual or potential adverse health effects, considered alone, requires EPA to add nPB to the HAP list under section 7412(b), and together, these effects provide a compelling case to list this chemical. The U.S. government's own findings, in other dockets from recent or pending action of other federal agencies, confirm that sound science amply supports EPA's decision to list nPB as a HAP.

The comments previously submitted to the docket are overwhelmingly supportive of listing nPB. Several states, the city of Philadelphia, and a group representing state air pollution control agencies have submitted comments urging EPA to list nPB as a HAP. Only companies engaged in the manufacture or supply of nPB commented in opposition to this common-sense action. And, as further explained below, even the information and analysis submitted by those companies support the conclusion that nPB must be listed when evaluated under the correct legal standard for listing.

EPA is long overdue in taking final action to grant the petitions and list nPB. HSIA and NYSDEC submitted their petitions over six years ago. EPA is in violation of the Clean Air Act and should act to list nPB without any further delay. *See* 42 U.S.C. § 7412(b)(3)(A) (setting an 18-month deadline for EPA action on such a petition).

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[1] Notably, one nPB company actually advertises the *lack of any EPA air toxics regulation* of this chemical on its website and describes it as "a better degree of environmentally friendly cleaning" and as a "cost-effective replacement for less environmentally friendly industrial solvents," when scientific evidence actually shows that nPB should be listed as a HAP due to its serious adverse health effects. Enviro Tech International, Inc., n-Propyl Bromide (nPB) Solvents, http://www.envirotechint.com/products/ensolv-n-propyl-bromide-npb-solvents/ (updated 2017, last viewed Sept. 28, 2017) (stating "U.S. SNAP approved; not regulated by NESHAP").

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I. SUMMARY OF USES AND HEALTH EFFECTS

The chemical n-Propyl Bromide (also known as "1-Bromopropane"or "1-BP") is a liquid solvent with many common commercial uses.[2] It is used as a solvent in cleaning and degreasing operations such as vapor degreasing and cold cleaning and in industrial and commercial aerosol degreasing products.[3] It can also be used in adhesive and sealant products used to fasten other

[2] These are simply synonyms for the same chemical, which can be identified by its CAS Registry Number of 106-94-5. *See* Agency for Toxic Substances & Disease Registry, *Toxicological Profile for 1-Bromopropane* at 134 (Aug. 2017) ("ATSDR Profile") (available at https://www.atsdr.cdc.gov/toxprofiles/tp209.pdf).

[3] EPA, TSCA Work Plan Chemical Risk Assessment for 1-Bromopropane, Dkt. ID. No. EPA-HQ-OPPT-2015-0084-0002 ("EPA Draft Risk Assessment") at 22, 36 (2016).

materials together, such as spray adhesive for foam cushion manufacturing.[4] nPB can also be used as a dry cleaning solvent, in spot cleaning formulations and in aerosol and non-aerosol cleaners. nPB is volatile and these uses are emissive, meaning that nPB is released into the air where members of the public inhale it.[5]

In the last decade, the use of nPB has increased because nPB is being used as a substitute or alternative for ozone-depleting and other hazardous substances.[6] The cumulative annual domestic production and import volume of nPB has ranged between 18.5 million pounds and 25.9 million pounds during the period from 2012 to 2015.[7] The most recent TRI data confirms that in 2016, the first reporting year for this chemical, over 600,000 pounds (more than 300 tons) of nPB were released into the air, with multiple sources reporting releases in excess of 20,000 pounds.[8]

In light of this increasing use and large-scale emissions, it is unsurprising that humans are regularly exposed to nPB. For example, the Agency for Toxic Substances and Disease Registry ("ATSDR") concluded that the increased application of nPB has led to increased exposure to workers in industries in which nPB is used.[9] And studies have confirmed that members of the general public, including pregnant women and children, are also exposed to nPB.[10]

The petitions and the entire record before the EPA contain significant evidence that such exposure threatens human health. This chemical is a likely human carcinogen, has adverse effects on reproduction and development, and can cause significant debilitating effects on the nervous system of exposed individuals.

The evidence that nPB causes serious human health effects is so strong that, in recent years, other agencies and other EPA Offices have also taken action or begun work to confront the threat nPB poses. In August 2017, ATSDR published the final *Toxicological Profile for 1-*

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[4] *Id.* at 40.
[5] *Id.* at 38.
[6] ATSDR Profile at 138.
[7] EPA, Scope of the Risk Evaluation for 1-Bromopropane, Dkt. ID No. EPA-HQ-OPPT-2016-0741-0049, at 19 (2017) ("EPA, Scope of the Risk").
[8] EPA, Toxic Release Inventory Program, 2016 TRI Preliminary Dataset: Basic Plus Files, TRI_2016_US.csv (2017) (available for download at https://www.epa.gov/toxics-release-inventory-tri-program/2016-tri-preliminary-dataset-basic-plus-files); EPA, Toxic Release Inventory Program, TRI 2016 Data (for nPB only).xls (2017) ("2016 TRI Data").
[9] ATSDR Profile at 138.
[10] As discussed below, urinary analysis of pregnant women participating in the National Children's study found that 99% of samples contained a metabolite, specific only to nPB. *See* Elizabeth Barksdale Boyle et al., *Assessment of Exposure to VOCs among Pregnant Women in the National Children's Study*, 13 Int'l J. Environ. Res. & Pub. Health 376 (Mar. 2016). Furthermore, and as discussed below, urinary analysis of samples taken from children participating in the National Health and Nutrition Examination Survey reveal that the same nPB metabolite identified in pregnant women was detectable in children as well. *See* Ram B. Jain, *Levels of selected urinary metabolites of volatile organic compounds among children aged 6-11 years*, 142 Environmental Research 461 (2015).

Bromopropane.[11] In addition, the Centers for Disease Control and Prevention has sought comment on its Draft Criteria for a Recommended Standard for Occupational Exposure to 1-BP.[12] EPA itself recently completed a Work Plan Chemical Risk Assessment for nPB under TSCA, and is in the process of conducting a risk evaluation of the chemical.[13] EPA also recently added this chemical to the list of toxic chemicals subject to reporting under the Toxics Release Inventory, section 313 of the Emergency Planning and Community Right-to-Know Act and section 6607 of the Pollution Prevention Act.[14] These actions reflect the scientific consensus on the toxicity of nPB. The research, scientific reports, analyses, and other materials in those dockets constitute further evidence that nPB meets the test for listing as a HAP. Commenters are submitting substantial material from those dockets into the docket as additional support for EPA's decision to grant the petitions.[15]

A brief summary of the scientific research (which is either in the docket before EPA or added as a supplemental appendix to these Comments) on the health effects of nPB exposure follows.

A. Cancer

The scientific evidence shows that nPB can cause cancer. In the Notice, EPA concluded that nPB is "reasonably anticipated to be human carcinogen." 82 Fed. Reg. at 2360. EPA drew, in part, upon a recent report by NTP, which describes nPB's carcinogenic effects, including alveolar/bronchiolar neoplasms in female mice, skin neoplasms in male rats and intestinal adenomas in both female and male rats.[16] These animal studies suggest similar carcinogenicity for humans because of similar metabolic pathways. And several metabolites of nPB, including those identified as mutagens, may cause DNA damage, and thus have increased cancer risk when exposure occurs *in utero* or during early life developmental stages. The NTP Technical Report

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[11] ATSDR Profile.

[12] *See* CDC, Notice, Dkt. ID No. CDC-2016-0003-0001 (Feb. 10 2016); CDC, Draft Criteria for a Recommended Standard: Occupational Exposure to 1-Bromopropane (1-BP), Dkt. ID No. CDC-2016-0003-0002 (Feb. 2016) ("CDC Draft Criteria").

[13] EPA Draft Risk Assessment; EPA, 1-Bromopropane; TSCA Review and Scoping, Dkt. ID No. EPA-HQ-OPPT-2016-0741 (July 2017); EPA Draft Risk Assessment.

[14] *See* 80 Fed. Reg. 72,906 (Nov. 23, 2015); *see also* https://www.epa.gov/sites/production/files/2016-11/documents/tri_chemical_list_changes_11_28_16_0.pdf at 13 (adding 1-bromopropane, 106-94-5 for reporting year 2016).

[15] The draft and supporting materials for the ATSDR Profile are available at docket number ATSDR-2016-0003; the CDC draft criteria materials are available at docket number CDC-2016-0003; and the TSCA material is available at docket number EPA-HQ-OPPT-2015-0084 and EPA-HQ-OPPT-2016-0741.

[16] *See* National Toxicology Program, NTP Technical Report on the Toxicology and Carcinogenesis Studies of 1-Bromopropane in F344/N Rats and B6C3F1 Mice (Inhalation Studies). NTP TR 564; NIH Publication No. 11-5906 (Aug. 2011) ("NTP Technical Report") (available at: https://ntp.niehs.nih.gov/ntp/htdocs/lt_rpts/tr564.pdf).

concluded that "clear evidence of carcinogenicity" exists for nPB, a conclusion EPA agreed with. 82 Fed. Reg. at 2359 (citing NTP determination).

NTP recently *reaffirmed* this finding, once again concluding that nPB is "is reasonably anticipated to be a human carcinogen."[17] NTP found that "exposure to 1-bromopropane has been shown to cause molecular alterations related to carcinogenicity, including genotoxicity (mutations and DNA damage), oxidative stress, glutathione depletion, and immunomodulation."[18] EPA's conclusion is further supported by other experts besides NTP, such as California EPA's Office of Environmental Health Hazard Assessment, which stated in July 2015, that it intends to list 1-bromopropane as "known to the state to cause cancer."[19]

B. Reproductive and Developmental Health Effects on Adults and Children

In the Notice, EPA concluded that there is "clear evidence" that nPB has adverse developmental and reproductive effects. 82 Fed. Reg. at 2360. The Notice found that animal studies show that exposure to nPB is associated with effects on live litter size, changes in sperm count and motility, alterations in estrous cycles, decreased reproductive organ weights, decreased fetal weight, skeletal abnormalities, limited postnatal weight gain, and decreased brain weight. *Id.* Multiple other sources, including the CDC and ATSDR, support these findings.[20]

These findings suggest that nPB can harm the unborn human fetus, impede early-life development, and adversely impact the ability of both men and women to have children. They are particularly significant in light of the known widespread exposure of pregnant women to nPB, discussed at note 10, above, and biomonitoring data, discussed in II.A, below.

C. Neurological Effects

In the Notice, EPA concluded that exposure to nPB could damage to both the peripheral and central nervous systems. 82 Fed. Reg. at 2360-61. Indeed, summarizing the science, EPA concluded that evidence in the record "provides *striking evidence* of neurotoxic effects." *Id.* at 2361 (emphasis added).

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[17] National Toxicology Program, *Report on Carcinogens, 1-Bromopropane*, 1 (14th ed. 2016) ("NTP Report") (available at https://ntp.niehs.nih.gov/ntp/roc/content/profiles/bromopropane.pdf).

[18] *Id.*

[19] Cal. EPA, Notice of Intent to List 1-Bromopropane Under Proposition 65 (July 10, 2015), https://oehha.ca.gov/media/downloads/crnr/071015noil1bromopropanepkg66.pdf; *see also* Cal. EPA, OEHHA, Response to Comments Pertaining to the Notice of Intent to List 1-Bromopropane as Causing Cancer under Proposition 65 (Aug. 2016), https://oehha.ca.gov/media/downloads/crnr/oehharesponsecomments08052016.pdf.

[20] CDC Draft Criteria at 114; Zong, et al. *Preliminary characterization of a murine model for 1-bromopropane neurotoxicity: Role of cytochrome P450.* Toxicology Letters, 258, 249, 249-258 (2016) (available at http://www.sciencedirect.com/science/article/pii/S0378427416322822); ATSDR Profile at 71-76.

These effects have been laid out in great detail in other studies.[21] Individuals exposed to nPB experience a variety of neurotoxic symptoms including recurring headaches, painful tingling in the hands, tremors, and feeling drunk even though they were not drinking.[22] Other studies show that these symptoms can be persistent and long-lasting.[23] And as several of the Commenters have previously pointed out to EPA, nPB exposure often requires individuals to seek emergency care and leaves affected individuals with devastating injuries.[24]

This is not the first time that EPA has concluded that nPB is neurotoxic.[25] Nor is EPA alone in concluding that nPB is neurotoxic.[26]

II. EPA MUST GRANT THE PENDING PETITIONS BECAUSE N-PROPYL BROMIDE MEETS THE LEGAL TEST FOR MANDATORY LISTING AS A HAZARDOUS AIR POLLUTANT.

The Clean Air Act provides that EPA "shall add a substance to the [section 112(b)] list upon a showing by the petitioner . . . that the substance is an air pollutant and that emissions, ambient concentrations, bioaccumulation or deposition of the substance are known to cause or may reasonably be anticipated to cause adverse effects to human health." 42 U.S.C. § 7412(b)(3)(B). The record before EPA shows that nPB easily meets the test for required listing.

A. N-Propyl Bromide Is An Air Pollutant.

The Act's definition of air pollutant—"any physical . . . substance or matter which is emitted into or otherwise enters the ambient air," 42 U.S.C. § 7602(g)—is capacious and easily encompasses nPB. *See Massachusetts v. EPA*, 549 U.S. 497, 528-29 (2007) ("sweeping definition . . . embraces all airborne compounds of whatever stripe"). There is significant docket evidence demonstrating that nPB meets this test; each such piece of data alone would be sufficient to make the minimal showing the Act requires, and together definitively prove nPB is an air pollutant.

For example, EPA's Draft Assessment's "Schematic of Human and Environmental Exposure Pathways for 1-BP" shows that various uses of this chemical—including, but not

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[21] *See e.g.*, Gaku Ichihara et al., *Neurotoxicity of 1-bromopropane: Evidence from animal experiments and human studies*, 3. J. of Advanced Research, Issue 2, 91, 91-98 (2012) available at http://www.sciencedirect.com/science/article/pii/S2090123211000452/pdfft?md5=0cdb5d72177ad96c929661fa944f4a1c&pid=1-s2.0-S2090123211000452-main.pdf; NTP Report at 1.

[22] ATSDR Report at 66.

[23] *Id.* at 68.

[24] Comments of Blue-Green Alliance, Earthjustice, et al. at 15, Dkt. ID No. EPA-HQ-OPPT-2015-0084-0016 (May 9, 2016) ("Earthjustice TSCA Workplan Comments").

[25] EPA Draft Risk Assessment at 92; *see also id.* at 128 (finding neurotoxic risk from occupational exposure).

[26] ATSDR Profile at 65, 71; CDC Draft Criteria at 48; s*ee also id.* at 115.

limited to, spray adhesive in the furniture industry, vapor degreasing in a variety of industries, and cold cleaning degreasing in a variety of industries—result in emissions to the ambient air.[27] Moreover, the ATSDR Profile recognizes that "[t]he general population may be exposed to 1-bromopropane in air when it is used during aerosol applications due to potential vapor migration, particularly at locations in close proximity to the emissive use of 1-bromopropane."[28] In addition, when the Occupational Safety and Health Administration ("OSHA") nominated 1-BP for testing by the National Toxicology Program ("NTP"), it stated:

> Various estimates have been made of the potential market for 1-BP in the key uses to which it is likely to be put: metal cleaning and degreasing, adhesives (especially for assembling polyurethane and other foam products), and aerosol spraying. Note that all of these uses are in practice highly emissive applications, resulting in substantial releases to the ambient environment . . . .[29] (emphasis omitted).

In addition, there is substantial additional evidence available demonstrating that nPB is an air pollutant. Earthjustice's TSCA comments provided a number of additional sources showing: (1) detection of nPB via monitoring data from EPA's own Air Quality System ("AQS") database; and (2) ambient air modeling and dispersion data.[30] Specifically, the comments outlined that EPA's AQS database has positively detected nPB in the ambient air in Philadelphia, Pennsylvania. Additional air dispersion modeling, used to estimate ambient nPB concentrations 100 meters from facilities that use nPB as an adhesive, found concentrations up to 274 ppb for facilities with high adhesive use.[31]

As EPA recognizes, petitioners have also provided emissions estimates for example facilities, such as dry cleaning facilities. 82 Fed. Reg. at 2359. EPA has correctly determined that "the petitioner's showing of information regarding nPB uses and sources is reasonable." 82 Fed. Reg. 2359. EPA has further determined—correctly—that the emissions estimates presented by the Petitioner, Halogenated Solvent Industry Association are within the "reasonable range of potential nPB emissions." *Id.* These data go well beyond what is needed to demonstrate that nPB is emitted into the ambient air. Even without any such specific emission estimates, there would be adequate information in the record to show that nPB is an air pollutant. Even the

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[27] EPA Draft Risk Assessment at 35, Figure 1-2.
[28] ATSDR Profile at 7.
[29] Directorate of Health Standards Programs, OSHA, Nomination of 1_Bromopropane (1-BP) and 2-Bromopropane (2-BP) for Testing by the National Toxicology Program at 2 (Dec. 1999), https://ntp.niehs.nih.gov/ntp/htdocs/chem_background/exsumpdf/bromopropanes_508.pdf.
[30] Earthjustice TSCA Workplan Comments at 4-6 (citing sources).
[31] *Id.*; *see also* Katy Wolf et al., Inst. for Research & Technical Assistance, UT Ctr. for Clean Products & Clean Techs., *Alternative Adhesives Technologies: Foam Furniture and Bedding Industries, A Cleaner Technologies Substitutes Assessment* at D-1, Table D-1 (2003) (describing use and emissions of this chemical).

manufacturers of nPB, who oppose listing, concede that nPB is emitted into and enters the ambient air.[32]

Finally, although these data are not needed for listing, the 2016 TRI data show that 55 facilities reported releases totaling 626,659 pounds of n-propyl bromide into the air, providing further confirmation of this chemical's emission in 27 states.[33] Therefore, the evidence before EPA shows not only that nPB is an air pollutant but that it is also being emitted at substantial levels into many states.[34]

B. Abundant Evidence Demonstrates that Emissions and Ambient Concentrations of n-Propyl Bromide Are Known to Cause or May Reasonably Be Anticipated to Cause Adverse Health Effects.

Likewise, the second prong of the test is met here – going well beyond the minimum needed to list a HAP. Listing is required where "emissions, ambient concentrations, bioaccumulation or deposition" of an air pollutant is "known to cause or may reasonably be anticipated to cause adverse effects to human health." 42 U.S.C. § 7412(b)(3). Such adverse health effects include, but are not limited to, "carcinogenic, mutagenic, teratogenic, [or] neurotoxic" effects, "reproductive dysfunction," or "acute or chronic toxicity."[35] *See id.* 7412(b)(2); 82 Fed. Reg. at 2357. From the use of "or" in the text of the statute, it is evident that any *one* of these effects is sufficient to trigger EPA's mandatory obligation to list a substance as a HAP. 42 U.S.C. § 7412(b)(2). Additionally, a showing of "complete substantiation" or "absolute certainty" that an air pollutant causes an adverse effect to human health is not required to trigger listing, as EPA acknowledges. 82 Fed. Reg. at 2357. Instead, it is sufficient to show that the substance "may reasonably be anticipated to cause" such an adverse effect.

The evidence before EPA demonstrates that nPB easily satisfies this prong of the test. As summarized in Part I, *above*, and as documented in sources cited therein and attached to these

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[32] Comments of Niomi Krzytowczyk, Vice President, Health, Safety and Environment, Albemarle Corp., Attachment 3, Dkt. ID No. EPA-HQ-OAR-2014-0471-0055 ("Nestrud Letter) at 1 (discussing "facilities that emit [nPB]"); *see generally id.* (discussing emissions of nPB).
[33] *See* 2016 TRI Data cited *supra* note 8 (summing air release columns AN and AO).
[34] Although the record shows that nPB emissions have increased significantly since 1990, it is not necessary for petitioners to show that emissions are increasing to meet this prong of the test of section 112(b)(3)(B). *See* 42 U.S.C. § 7602(g) (requiring only that the substance be emitted into or otherwise enter the ambient air).
[35] In addition to the petition process for adding substances to the list of HAPs, 42 U.S.C. § 7412(b)(3), the Act also requires the EPA to revise the list on its own initiative. *Id.* § 7412(b)(2). EPA is required to add to the list any "pollutants which present, or may present, through inhalation or other routes of exposure, a threat of adverse human health effects (including, but not limited to, substances which are known to be, or may reasonably be anticipated to be, carcinogenic, mutagenic, teratogenic, neurotoxic, which cause reproductive dysfunction, or which are acutely or chronically toxic) or adverse environmental effects whether through ambient concentrations, bioaccumulation, deposition ,or otherwise . . . ." *Id*. EPA recognizes

comments, abundant scientific evidence confirms that "emissions, ambient concentrations, bioaccumulation or deposition" of nPB are "known to cause or may reasonably be anticipated to cause adverse effects to human health," 42 U.S.C. § 7412(b)(3)(B). As EPA reasonably summarized, the known or reasonably anticipated effects of nPB to human health include cancer, reproductive and developmental harm, and neurological harm. There is also evidence showing nPB's harmful effects on other body systems, including the respiratory system, gastrointestinal system, and cardiovascular system.[36]

The record is replete with evidence to support listing of nPB as a HAP. These comments highlight some of the health threats to exposed people—including cancer risks, reproductive and developmental risks, and neurological risks—to illustrate that EPA must finalize action granting these petitions and listing nPB as a HAP. Any one of these serious health effects nPB may reasonably be anticipated to cause is sufficient to require listing. Moreover, the fact that nPB is reasonably anticipated to cause more than one type of adverse health effect demonstrates that it is especially dangerous and confirms that listing is urgently needed.

In view of the best available scientific evidence, both prongs of the test of section 112(b)(3)(B) are met here, and therefore, EPA has a mandatory obligation to add nPB to the section 112(b) list of HAPs. 42 U.S.C. § 7412(b)(3)(B) ("The Administrator *shall* add a substance to the list upon a showing . . . ." (emphasis added)). We urge EPA to act promptly to comply with its obligation to address this harmful pollutant.

C. Any One of the Myriad Adverse Health Effects Demonstrated in the Record Requires Listing n-Propyl Bromide.

1. Listing is Required Based on Cancer Risk from Exposure to nPB.

As discussed in Section I.A, above, there is ample scientific evidence that exposure to nPB can cause cancer. In the notice, EPA summarized the underlying science and concluded that nPB is "reasonably anticipated to be a human carcinogen." 82 Fed. Reg. at 2360. That conclusion was recently reaffirmed by NTP.[37] Given EPA's express conclusion, nPB plainly meets the test for mandatory listing as a HAP, as it "may reasonably be anticipated to cause adverse effects to human health." *See* 42 U.S.C. § 7412(b)(3)(B).

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that this provision also informs its obligations when it reviews a petition to list a substance as a HAP. 82 Fed. Reg. at 2357.

[36] *See, e.g.*, Gaku Ichihara, et al., *Neurological disorders in three workers exposed to 1-bromopropane*, J. Occup. Health 44 (1), 1 , 1-7(2002) (available at: https://www.jstage.jst.go.jp/article/joh/44/1/44_1_1/_article); R. Miao, et al., *Large-scale label-free proteomics analysis of occupational poisoned patients of 1-bromopropane, workers exposed to 1-bromopropane and healthy individuals*, Human & Experimental toxicology (2017) (available at: http://journals.sagepub.com/doi/abs/10.1177/0960327117689911); *see* Fen Huang, et al., *Effect of 4□week inhalation exposure to 1□bromopropane on blood pressure in rats*, J. of Applied Toxicology (2016) (available at: http://onlinelibrary.wiley.com/doi/10.1002/jat.3364/full).

[37] *See* NTP Report at 1.

Although no more than that is required, the record now before EPA contains two new risk assessments demonstrating that exposure to nPB significantly increases the risk of developing cancer.[38] These findings further support the conclusion that emissions and ambient concentrations of nPB are known or may reasonably be anticipated to cause adverse effects to human health, requiring EPA to list nPB as a HAP.

As EPA correctly recognizes, a risk assessment of the type provided by Exponent is not required for the agency to decide to list a HAP. 82 Fed. Reg. at 2357, 2361-62. Instead, evidence of potential carcinogenicity—*i.e.*, that a chemical *may* cause cancer or any other kind of health threat—is sufficient to require listing.

Nonetheless, by providing a quantitative risk assessment, petitioners went well beyond the minimum that is needed to meet the listing test. The risk assessment provided to EPA by Exponent provides significant additional evidence that people living near facilities that emit nPB face elevated risk of cancer, thereby establishing that EPA must list nPB.[39] Exponent's 2016 modeling submitted in support of the petitions shows that the excess cancer risk from nPB exposure to the most exposed individual is, at least, greater than 1 in 1 million for all five facilities modeled and greater than 1 in 100,000 (10-in-1 million) for one of the five facilities. EPA has determined that "the petitioner's showing of information regarding nPB uses and sources is reasonable." 82 Fed. Reg. at 2359. EPA has further determined—correctly—that the emissions estimates presented by the Petitioner HSIA are within the "reasonable range of potential nPB emissions." *Id.*

Moreover, Gradient itself has calculated an inhalation unit risk factor for nPB, which differs only in degree from the estimate provided by petitioners, Gradient Report at 4, further supporting listing of nPB as a HAP. The Gradient report shows that cancer risk from nPB exposure is <u>indisputable</u> and thus actually provides further <u>support</u> for listing nPB. For example, it assesses cancer risk and concludes that over 1500 people are exposed to cancer risk greater than 1 in 1,000,000 from exposure to nPB emissions from a Pennsylvania narrow tube manufacturer alone. Gradient Report at 19. Gradient's modeling also shows that nPB emissions from a Virginia dry cleaner impose cancer risk greater than 1 in one million on 170 people, and that nPB emissions from a Massachusetts dry cleaner impose cancer risk greater than one in 1,000,000 on 14 people. Gradient Report at 20. In a letter submitted on behalf of nPB manufacturers, Charles Nestrud concedes that excess cancer risk from nPB exposure is 9.9 in one million for the most exposed individual near the Pennsylvania narrow tube manufacturer. Nestrud Letter at 9.

Though there may be some differences between the emissions and exposure estimates in the two reports, EPA concluded that these differences do not undermine the results of

[38] One risk assessment was conducted by Exponent, on behalf of petitioner HSIA. 2016 HSIA Exponent nPB updated HEM Modeling Report, Dkt. ID No. EPA-HQ-OAR-2014-0471-0065 (Feb. 22, 2016) ("Exponent Report"). Another risk assessment was conducted by Gradient, on behalf of Albemarle Corp. Comments of Niomi Krzytowczyk, Vice President, Health, Safety and Environment, Albemarle Corp., Attachment Comments on the Petition to Add n-Propyl Bromide to the List of Hazardous Air Pollutants Regulated under § 112 of the Clean Air Act, Dkt. ID No. EPA-HQ-OAR-2014-0471-0057 (May 7, 2015) ("Gradient Report").

[39] Exponent Report at 18 (summarizing risks by facility type, all in excess of one in one million).

Exponent's quantitative risk analysis. Instead, the Gradient analysis just represents another point within a range of reasonable estimates of cancer risk. 82 Fed. Reg. 2359. Indeed, Gradient conceded that "some of these differences [between Exponent's model and the Gradient model] would be expected to result in greater estimated cancer risk from nPB, and some would be expected to result in lesser estimated cancer risk from nPB." Gradient Report at 17.

Indeed, it is quite likely that both Exponent and Gradient have underestimated the cancer risks measured, based on the methodology they used and the data they had available. As scientific risk assessment approaches have advanced in recent years, even EPA's risk assessment approach has been slow to catch up and often ignores or fails to measure increased health risks, including higher risk due to the increased vulnerability of exposure *in utero*, exposure to multiple sources, or exposure from multiple pathways (such as inhalation plus ingestion or dermal exposure).[40] There is no indication that either risk assessment followed the best available current risk assessment approaches, or even attempted to account in full for all of the cancer risk that nPB exposure can cause, as described in the 2009 National Academy of Sciences Silver Book, for example.[41]

In sum, here there are strong independent and authoritative scientific determinations that nPB is a human carcinogen, and *both* laboratory science and quantitative risk estimates show that nPB is anticipated to cause cancer in humans. Based on the robust evidence of its carcinogenic impact alone, EPA must list the pollutant as a HAP.

2. Reproductive Risks and Developmental Risks Require Listing.

As described in Part I.B, above, the evidence before EPA demonstrates that nPB is both a reproductive and developmental toxicant and therefore requires listing as a HAP. In the Notice, EPA determined that exposure to nPB is associated with effects on live litter size, changes in sperm count and motility, alterations in estrous cycles, decreased reproductive organ weights, decreased fetal weight, skeletal abnormalities, limited postnatal weight gain, and decreased brain weight. 82 Fed. Reg. at 2360. Based on this evidence, EPA concluded that there is "clear evidence" of adverse reproductive and developmental effects from nPB exposure.[42] 82 Fed. Reg. at 2360.

EPA is not alone. Presented with similar evidence, ATSDR concluded that "animal data suggest that the reproductive system may be a potential target of concern for 1-bromopropane toxicity in humans."[43] In line with these findings, a report by the CDC and NIOSH concluded that animal studies have shown "significant changes in sperm morphology, count, and motility"

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[40] *See, e.g.*, Earthjustice TSCA Work Plan Comments; Comments of Earthjustice, Dkt. ID. No. EPA-HQ-ORD-2015-0684-0024 (Mar. 22, 2016); Comments of NRDC, Dkt. ID No. EPA-HQ-ORD-2015-0684-0027 (Mar. 22, 2016); Comments of Earthjustice, Dkt. ID No. EPA-HQ-ORD-2013-0292-0133 (June 28, 2013).

[41] Science and Decisions: Advancing Risk Assessment, Chapter 5, ("Silver Book") (2009); *see also* Finkel, Adam, Comment Letters, Docket No. EPA-HQ-OAR-2014-0471-0048 (2015) (citing the Silver Book).

[42] *See also* EPA Draft Risk Assessment at 128 (finding developmental risks from nPB exposure).

[43] ATSDR Profile at 72.

following exposure to nPB.[44] A case report of female workers occupationally exposed to nPB found altered menstrual periods and decreased sexual desire, "findings [which] provide some evidence that exposure to 1-BP may adversely affect human reproduction in females."[45]

These adverse developmental effects are particularly concerning given the known exposure of pregnant women to nPB. For example, a recent epidemiological study of 488 women in the third trimester of pregnancy in seven locations around the country (who participated in the National Children's Study) found that 99% of the participants had the nPB metabolite (N-Acetyl-S-(n-propyl)-L-cysteine) in their urine.[46]

Given this clear evidence that nPB is known to cause or at the very least "may reasonably be anticipated to cause" adverse reproductive and developmental effects, EPA is required to list nPB as a HAP.

3. Neurotoxic Effects Require Listing

In the Notice, EPA correctly "concluded that the concordance of outcomes across humans and laboratory rodents provides *striking evidence* of neurotoxic effects." 82 Fed. Reg. at 2361 (emphasis added). As discussed in Part I.C, that conclusion is fully supported by the record that existed before EPA when the Notice was published and is bolstered by additional evidence highlighted in this Comment. Collectively, this evidence requires EPA to list nPB as a HAP.

Numerous occupational studies surveyed by EPA, as well as ATSDR, and others have all reached the same conclusion: exposure to nPB is neurotoxic.[47] Symptoms such as numbness have been cited in occupational studies[48] while gait disturbance has been found in both human and animal studies.[49] Studies have documented similar symptoms in people, which may include

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[44] CDC Draft Criteria at 114.

[45] National Toxicology Program, Department of Health and Human Services, *NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of 1-Bromopropane* (Oct. 2003) (available at https://ntp.niehs.nih.gov/ntp/ohat/bromopropanes/1-bromopropane/1bp_monograph.pdf).

[46] Elizabeth Barksdale Boyle et al., *Assessment of Exposure to VOCs among Pregnant Women in the National Children's Study*, 13 Int'l J. Environ. Res. & Pub. Health 376 at 7 (Mar. 2016).

[47] *See*, *e.g.*, ATSDR Profile at 65-71; CDC Draft Criteria.at iii, viii, ix, xii, 3.

[48] *See* National Toxicology Program, Report on Carcinogens, Monograph on 1-Bromopropane, (Sept. 2013) (available at https://ntp.niehs.nih.gov/ntp/roc/thirteenth/monographs_final/1bromopropane_508.pdf) (discussing Gaku Ichihara, et al., *Neurologic abnormalities in workers of a 1-bromopropane factory*, Environ. Health Perspect. 112, 1319-1325 (2004b)).

[49] *See* Yukiko Fueta, et al., *Hyperexcitability of the Hippocampal CA1 and the Dentate Gyrus in Rats Subchronically Exposed to a Substitute for Chlorofluorocarbons, 1-Bromopropane Vapor*, J Occup. Health, 44 (3) at 156-165 (2002). *See also* Majersik, et al., *Severe neurotoxicity associated with exposure to the solvent 1-bromopropane (n-propyl bromide)*, Clin. Toxicol (Phila), 45 (3), at 270-276, (2007).

vibration sense losses in lower extremities and weakness.[50] Sensory ataxic neuropathy has also been detected in a 43-year-old male worker's body after using cleaning agent containing nPB without proper protection in the workplace.[51] Most recently, a study investigated proteomic changes between healthy individuals and workers exposed to nPB to elucidate the biological mechanisms behind nPB neurotoxic pathogenesis.[52]

This is not the only time that EPA has concluded that nPB is neurotoxic. EPA recently concluded that "the nervous system is a sensitive target of 1-BP exposure," noting that "[b]oth the central and peripheral nervous systems are affected."[53] EPA is not alone in concluding that nPB is neurotoxic. For example, the ATSDR has determined that nPB causes serious neurological health effects, finding: "The available data clearly indicate that the nervous system is a target for 1-bromopropane toxicity in humans and animals. Data in humans show that 1-bromopropane can induce morphological alterations in neurons, which may lead to motor and sensory deficits. Studies in animals show that 1-bromopropane can induce biochemical, morphological, electrophysiological, and neurobehavioral alterations by mechanisms yet to be elucidated."[54] Similarly, the CDC found that inhalation exposure to nPB is "associated with adverse effects in the [central nervous system] and [peripheral nervous system]."[55]

These significant neurotoxic effects establish that nPB is "known to cause" or, at the very least, "may reasonably be anticipated to cause" adverse effects to human health and, accordingly, that EPA must list nPB.

D. It Would Be Arbitrary for EPA to Decline to List n-Propyl Bromide as a HAP when its Health Effects Are Similarly Adverse to Already Listed HAPs, such as Trichloroethylene (TCE) and Tetrachloroethylene (PERC).

TCE and PERC are both among the 187 originally listed HAPs. 42 U.S.C. § 7412(b)(1). Like nPB, TCE and PERC are halogenated volatile organic compounds used as solvents during dry cleaning and metal degreasing operations. As with nPB, workers in the metal degreasing and

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[50] *See* Li, et al. *Dose-dependent neurologic abnormalities in workers exposed to 1-bromopropane*, J Occup. Environ. Med., 52 (8), at 769-777 (2010). *See also* Sclar, G., *Encephalomyeloradiculoneuropathy following exposure to an industrial solvent*, Clin. Neurol. Neurosurg., 101 (3) at 199-202, (1999).

[51] *See* ATSDR Profile (discussing Samukawa, et al., *A Case of Severe Neurotoxicity Associated With Exposure to 1-Bromopropane, an Alternative to Ozone-Depleting or Global-Warming Solvents*, Arch. Intern. Med. 172 (16) at 1257, 1257-1260 (2012)).

[52] *See* R. Miao, et al., *Large-scale label-free proteomics analysis of occupational poisoned patients of 1-bromopropane, workers exposed to 1-bromopropane and healthy individuals*, Human & Experimental toxicology (2017) (available at: http://journals.sagepub.com/doi/abs/10.1177/0960327117689911)

[53] EPA Draft Risk Assessment at 92; *see also id.* at 128 (finding neurotoxic risk from occupational exposure).

[54] ATSDR Profile at 71.

[55] CDC Draft Criteria at 48; s*ee also id.* at 115.

dry cleaning industries are most likely exposed via inhalation of TCE and PERC in the ambient air.[56]

Not only do the three compounds have similar uses, the documented health effects in occupational settings and animal studies are also similar. As with nPB, there is evidence of adverse neurological effects from TCE or PERC exposure. Inhalation of TCE or PERC is known to cause short-term health effects like dizziness, headaches, and sleepiness – each effect suggestive of harm to the nervous system. Additionally, occupational studies have confirmed that long-term exposure to these compounds may cause changes in mood, memory, and attention span. Additionally, TCE and PERC adversely affect other organ systems, including the liver, kidneys, and reproductive systems, just as nPB does.[57]

Finally, just like nPB, TCE and PERC are known or may reasonably be anticipated to be carcinogenic. Both EPA and the International Agency for Research on Cancer have determined that TCE is a human carcinogen.[58] EPA considers PERC to be "likely to be carcinogenic to humans by all routes of exposure,"[59] while IARC considers PERC to be "probably carcinogenic to humans."[60] As summarized above, the evidence is similarly clear on nPB's carcinogenicity.

It would be arbitrary for EPA to refuse to list nPB, where it poses at least a comparable threat to human health as two listed HAPs.[61]

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[56] *See* ATSDR, *Public Health Statement for Trichloroethylene* (2016) (available at https://www.atsdr.cdc.gov/ToxProfiles/tp19-c1-b.pdf); ATSDR, *Public Health Statement for Tetrachloroethylene* (2014) (available at https://www.atsdr.cdc.gov/ToxProfiles/tp18-c1-b.pdf).

[57] *Id.*

[58] *See* EPA, Integrated Risk Information System, Chemical Assessment Summary, Trichloroethylene; CASRN 79-01-6 (2011) (available at https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0199_summary.pdf); International Agency for Research on Cancer, Monograph: Trichloroethylene (Vol. 106) (2014) (available at https://monographs.iarc.fr/ENG/Monographs/vol106/mono106-001.pdf).

[59] EPA, Toxicological Review of Tetrachloroethylene (2012) (available at https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0106tr.pdf).

[60] International Agency for Research on Cancer, Monograph: Tetrachloroethylene at 329 (Vol. 106) (2014) (emphasis removed) (available at https://monographs.iarc.fr/ENG/Monographs/vol106/mono106-002.pdf).

[61] This disparate treatment is problematic in light of EPA's proposal to ban the use of TCE in for vapor and aerosol degreasing operations, utilizing its TSCA authority. *See* EPA, Regulation of Certain Uses under Toxic Substances Control Act: Trichloroethylene, Dkt. ID No. EPA-HQ-OPPT-2016-0163-0001 (2016). EPA identified nPB and PERC as possible substitutes and estimated that 25% of users of TCE will switch to nPB. *Id.*

III. EPA SHOULD REJECT THE ARGUMENTS OF N-PROPYL BROMIDE MANUFACTURERS WHO SEEK TO AVOID THE FEDERAL CLEAN AIR PROTECTIONS NEEDED TO PROTECT PUBLIC HEALTH THAT ALREADY APPLY TO INDUSTRY COMPETITORS.

A. Arguments Regarding Cancer Risk Misconstrue the Law and Misapprehend the Record

Commenters who oppose listing of nPB as a HAP have made several arguments for why EPA should ignore the cancer risk demonstrated by the Exponent study. These arguments either misconstrue the statutory requirements for listing a HAP or misapprehend the record before EPA.

1. The risk evaluations submitted by Exponent and Gradient are not necessary for listing nPB as a HAP.

As EPA correctly recognizes, a risk assessment of the type provided by Exponent is not required for the agency to decide to list a HAP. 82 Fed. Reg. at 2357, 2361-62. Under the Clean Air Act, EPA shall generally add pollutants "which present, or may present" various types of adverse human health effects. 42 U.S.C. § 7412(b)(2). Further, it must grant a petition to list a HAP where the air pollutant "may reasonably be anticipated to cause adverse effects." *Id.* § 7412(b)(3)(B). The legislative history is clear that "the words 'may reasonably be anticipated'" were used "[i]n order to emphasize the precautionary or preventive purpose of the act (and, therefore, the Administrator's duty to assess risks rather than wait for proof of actual harm)." H.R. REP. 95-294 at 51, Clean Air Act 1977 Leg Hist. at 26. EPA has appropriately recognized that "the CAA is a protective or preventive statute," and that it requires EPA to "err on the side of caution in determining whether the data are sufficient to support listing a substance." 82 Fed. Reg. at 2357. Therefore, evidence of potential carcinogenicity—*i.e.*, that a chemical *may* cause cancer or any other kind of health threat—is sufficient to require listing, even where there is scientific uncertainty regarding the *amount* of health risk a chemical presents, as EPA itself recognizes. 82 Fed. Reg. at 2357 (noting that the test does not require certainty).

Nonetheless, Gradient argues that the scientific evidence of the amount of nPB carcinogenicity is allegedly "not reliable for quantitative extrapolation" at present. Gradient Report at 8. "Quantitative extrapolation," however, is not required in the statute or needed to evaluate the statutory test, and it would be unlawful and arbitrary for EPA to decline to list when the evidence of health threats in the record amply meets the statutory test. Under the standard of section 112(b)(3)(B), precise quantitative risk estimates are not required at the HAP listing stage, and EPA may not decline to list nPB on that basis. Nor is there a statutory requirement to provide emissions estimates or modeling as the petitioner has done here.

Indeed, requiring precise quantitative risk data, precise emission estimates, modeling, or other specific data on emissions, deposition, or ambient concentrations at the listing stage belies the statutory test and legislative purpose, and would create a chicken and egg problem. Where a pollutant is currently unlisted or unregulated, it is extremely likely that there will be limited or no data available. Thus, not only are such data not required to support a listing petition under the plain language of the statute, it would contravene the whole purpose of the low bar to list a HAP

to require such data, and make it much more difficult to prove that a chemical should be added to the list. In this instance, as cited above, however, there is now significant 2016 TRI data showing n-propyl bromide emissions, so there is ample data available illustrating the need to list that goes beyond what the statute requires.

Although a petitioner like HSIA presumably had the resources to commission a report from a company like Exponent and submit the data presented into the record, there are many community members who would not have such resources. Requiring such a showing at the listing petition stage would shut out of this process low-income community members and groups who may lack the ability to pay for such an analysis. That would run counter to the Clean Air Act's purpose not just to prevent and reduce air pollution, but to address and provide protection based on the need to protect public health and address the inequity of air pollution's over-concentration in particular communities, such as urban areas and communities of color and low-income communities. *See, e.g.*, S. Rep. No. 101-228, at 128-129, *reprinted in* 6 Legislative History at 3513-3514 (1990 Amendments added based partly to address an "equity concern, the very high risk of health problems experienced by individuals living near large industrial facilities or in highly developed urban corridors"); *see also* H.R. Rep. No. 101-490 pt. 1 at 153-54, 315-22.

There is ample evidence in the record, beyond the risk assessments submitted by Exponent and Gradient, that nPB is reasonably anticipated to increase the risk of cancer. That evidence is sufficient to trigger EPA's mandatory duty to list nPB as a HAP. Accordingly, no purported flaw in the Exponent study, real or imagined, can excuse EPA from listing nPB.

2. The available documents providing information on risk assessment all support listing, regardless of any methodological differences or differences in results.

Certain commenters' arguments about the precise amount of cancer risk or the method Exponent used to assess this neither cast any serious doubt that this chemical can cause cancer, nor otherwise demonstrate that nPB does not meet the test for listing as a HAP.

First, as described in Section II.C.1, above, the Gradient report actually confirms that nPB poses a cancer risk. Second, any differences between the two studies were differences of degree. Accordingly, EPA is correct to conclude that the emissions and exposure estimates in the Gradient report do not undermine Exponent's quantitative risk analysis, but just represent another point within a range of reasonable estimates of cancer risk. 82 Fed. Reg. 2359.

Further, there is other evidence that people experience doses above the levels shown to cause cancer in animal studies. For example, EPA itself estimated the cancer risk faced by workers exposed to nPB, and concluded that they face a significant increased risk of developing cancer.[62] The estimated risk to these individuals was significantly higher than the risks estimated by either Exponent or Gradient. For example, EPA estimated that, in the spray adhesives category, even the median-exposed worker faced an added cancer risk *as high as 20 in 100* due to exposure to nPB, and EPA found extremely high cancer risk values across the sectors

[62] EPA Draft Risk Assessment at 134-40.

analyzed.[63] So Gradient's arguments regarding the unreliability of the Exponent risk assessment is belied by evidence in the record, in addition to being legally irrelevant.

Thus, while Gradient disputes the precise potency of nPB's carcinogenic effect, it does not and cannot deny that nPB is carcinogenic. EPA's very own analysis refutes Gradient's arguments. Consequently, the differences between the Exponent and Gradient reports do not provide a reasonable basis to conclude that nPB should not be listed as a HAP.

3. The Clean Air Act does not establish any minimum level of risk to trigger EPA's listing obligation, nor does it exempt air pollutants that pose some undefined, "acceptable" level of risk

Some commenters have incorrectly suggested that EPA may decline to list nPB because the risk assessments in the record: (1) fail to establish some minimum level of cancer risk; or (2) establish a level of cancer risk that is somehow "acceptable." Neither argument can be squared with the statutory requirements of the Clean Air Act. As explained above, the Clean Air Act does not require a risk evaluation as a prerequisite for listing an air pollutant as a HAP, so necessarily, there is no minimum risk level that must be established to trigger listing. Nor can there be any acceptable risk level that excuses a decision not to list. This is confirmed by the text, structure, and underlying policy of the Act.

The Clean Air Act requires EPA to list an air pollutant if exposure "may reasonably be anticipated" to cause adverse effects to human health. 42 U.S.C. § 7412(b)(3). Where cancer risk is well-established, as it is for nPB, and people are or may be exposed to the carcinogenic substance, as they are here, the statutory test for listing is met. The statute includes no express minimum cancer risk (or any other type of risk) level for listing. Similarly, there is no textual basis to establish that some amount of cancer risk is "acceptable" and to therefore decline listing.

The fact that the Clean Air Act includes quantitative risk values in some provisions but not in subsections 7412(b)(2)-(3) establishes that EPA may not use any specific risk cut-off for listing as a HAP. *Compare* 42 U.S.C. § 7412(b)(3)(B) (not setting any bright-line risk level for cancer or any other kind of health effect for listing purposes), *with id.* § 7412(f)(2) (directing residual risk rulemaking where lifetime cancer risk to the individual most exposed to emissions is 1-in-1 million or more). EPA appropriately recognized this, observing that subsections 7412(b)(2)-(3) stand in clear contrast to EPCRA which otherwise uses similar language but adds a requirement to consider the "concentration levels." 82 Fed. Reg. at 2362 (citing EPCRA). No such cut-off can be read into the HAPs listing provisions, particularly because Congress showed it was capable of adding such a specific threshold when it did so in a related part of the same statutory section.

[63] *Id.* at 138 ("Added cancer risks calculated for workers and occupational non-users exposed at the 95th percentile exceeded all identified cancer benchmarks (i.e., 1x10-4 (1 in 10,000), 1x10-5 (1 in 100,000) and 1x10-6 (1 in 1,000,000)) in most of the use scenarios evaluated under the scope of this assessment. In most cases a 1,000-fold exceedance of the 1 in 1,000,000 cancer risk benchmark was observed (this corresponds to a cancer risk greater than 1x10-3 (or a probability of 1 in 1,000 that an exposed individual will develop cancer).").

Further, to delay listing pending the development of additional data sufficient to estimate the precise quantitative risk would be inconsistent with the sound functioning of the statutory scheme governing the regulation of HAPs. Listing a pollutant under section 112(b) is the first step in a process that Congress established in the 1990 Clean Air Act Amendments to remedy the delay and inaction that previously characterized the implementation of the hazardous air pollutant program. *See New Jersey v. EPA*, 517 F.3d 574, 578 (D.C. Cir. 2008); H.R. Rep. No. 101-490, at 151, 1990 CAA Amendments Legislative Hist. at 3175; S. Rep. No. 101-228, at 132 (Dec. 20, 1989), *reprinted in* 1990 U.S.C.C.A.N. 3385, 3517. As amended, section 112 is intended to "eliminat[e] much of EPA's discretion" and lead to more timely and comprehensive implementation. *New Jersey v. EPA*, 517 F.3d at 578. Notably, Congress provided for EPA to conduct risk analysis at a subsequent step of the process. 42 U.S.C. § 7412(f).

Thus, it would turn the process established by Congress on its head if the statute were read to require petitioners to show at the listing stage not only that a pollutant is known or may reasonably be anticipated to cause cancer, but that the quantitative risk exceeds a particular threshold. In the 1990 Amendments to the Clean Air Act, Congress carefully crafted a protective regime that begins with listing of pollutants that "may reasonably be anticipated" to cause adverse effects to human health, 42 U.S.C. § 7412(b)(3)(B). This framework ensures that EPA has years of experience with both the pollutant and source by the time it considers quantitative risks at a subsequent stage of the process, *id.* § 7412(f). To apply the standard of § 112(f) at the listing stage would disrupt Congress's carefully crafted scheme.

Deciding not to list nPB based on an extra-statutory numeric risk threshold would also be contrary to section 112's purpose, which is to take a precautionary approach that prioritizes public health. "[T]he words 'may reasonably be anticipated'" were used in the Clean Air Act "[i]n order to emphasize the precautionary or preventive purpose of the act (and, therefore, the Administrator's duty to assess risks rather than wait for proof of actual harm)." H.R. REP. 95-294, 51, 1977 U.S.C.C.A.N. 1077, 1129. To decline to list nPB on the ground that some models show exposed individuals suffer excess cancer risk of *only* 10-in-1 million or 1-in-1 million due to this pollutant, rather than a higher cancer risk, would defeat the health protective and precautionary purpose of the Act. Even if workers didn't face increased cancer risks of 20-in-100, which they do, EPA would have to list nPB based on these plainly serious (and likely underestimated) community health threats.

Requiring that cancer risk be shown to be above a particular threshold at the start of the regulatory process would also undermine the goal of protecting all individuals, including those exposed to multiple sources and that may be particularly susceptible. *See generally* H.R. REP. 95-294; NASNAE, "Man, Materials, and Environment: A Report to the National Commission on Materials Policy," (March 1973) (cited in H.R. REP. 95-294) ("In establishing health and safety standards, the traditional concept of a threshold must be modified to reflect that it does not represent a safe level of exposure for unusually sensitive members of the population."). Such variation in susceptibility may be poorly understood at the early stage of listing. Exposure to multiple sources of the same pollutant would mean that the same person would receive a much higher amount of the pollutant, a scenario that defining some kind of listing threshold, out of context, would not protect against.

Therefore, even assuming that any measurable cancer risk could be deemed "acceptable" at the listing stage (which it cannot be), it would be impermissible to view the cancer risk from nPB in isolation in assessing whether such a threshold is exceeded. The nPB manufacturers'

invitation to EPA to view the risk from nPB emissions from particular facilities in isolation is inconsistent with Congress' instruction to "require consideration of cumulative or synergistic effects of multiple pollutants." H.R. REP. 95-294, 50, 1977 U.S.C.C.A.N. 1077, 1128.

Moreover, since the 1950s, it has been well-established that carcinogens have no safe level of human exposure. *See, e.g.*, S. Rep. 101-228, at 175, 1990 U.S.C.C.A.N. at 3560 ("Federal Government health policy since the mid-1950s has been premised on the principle that there is no safe level of exposure to a carcinogen"); *NRDC v. EPA*, 824 F.2d 1146, 1147 (D.C. Cir. 1987) (*en banc*) ("Current scientific knowledge does not permit a finding that there is a completely safe level of human exposure to carcinogenic agents." Furthermore, because cancer risk from a single carcinogen or multiple carcinogens act in a synergistic manner—cumulatively adding to an individual's cancer risk—any cancer risk posed by a substance is sufficient to require listing.

Other Clean Air Act provisions highlight the problems with the industry groups' arguments. The threshold for the residual risk rulemaking required under § 7412 is a lifetime cancer risk of 1-in-1 million. 42 U.S.C. § 7412(f)(2). The data submitted by Exponent and Gradient show that the cancer risk from nPB is at least that high, and EPA's own estimates suggest they are *orders of magnitude higher*. The fact that Congress determined, and enacted into law, a requirement to regulate sources if they cause a lifetime cancer risk of at least 1-in-1 million belies any argument that a chemical should not even be listed as a HAP if it causes that level of risk or higher, as nPB indisputably does.

The statutory test for listing a HAP contains no language establishing a minimum or acceptable risk threshold, and it would be unlawful to decline to list nPB on that basis.

B. Harm to Workers Resulting from Exposure to n-Propyl Bromide Requires Listing.

Abundant evidence submitted to EPA demonstrates that exposure to nPB is known or may reasonably be anticipate to cause adverse health effects. In particular, evidence of worker exposure to nPB definitively demonstrates the human neurotoxic effects of nPB. Despite this evidence, some commenters inexplicably argue that EPA should ignore any and all evidence of adverse health effects in workers. But the Clean Air Act does not allow EPA to ignore such relevant scientific evidence of the adverse health effects of nPB exposure.

The record before EPA demonstrates that workers exposed to nPB suffer neurological damage, cancer, and other health ailments.[64] Along with other scientific information, the State of New York and HSIA both submitted information on occupational hazards and toxicity of nPB to support their petitions, which EPA acknowledges is relevant and persuasive. 82 Fed. Reg. at 2358. EPA appropriately acknowledged that information on harm to workers and on occupational hazards and exposure limits, as provided by commenters during the first round of comment, is both relevant and persuasive on the need to list nPB as a HAP. *Id.* EPA noted, in particular, that it is important and relevant that there are "occupational studies and case reports of altered peripheral nerve function in workers exposed to concentrations of nPB as low as 1-3 parts per million (ppm)" *id.* at 2359; and that the NTP review, among other things, "took into account

[64] Comment of Sierra Club *et al.*, 4 Dkt. ID No. EPA-HQ-OAR-2014-0471-0056 (May 7, 2015); Earthjustice TSCA Work Plan Comments at 15; Ian Urbina, *As OSHA Emphasizes Safety, Long-Term Health Risks Fester*, The New York Times, Mar. 30, 2013, at A1, (available at http://www.nytimes.com/2013/03/31/us/osha-emphasizes-safety-health-risks-fester.html).

reports of mutations in bacterial and mammalian cells and limited data on DNA damage in nPB-exposed workers," *id.* at 2360; and also found relevant that there are epidemiological studies of "worker populations," *id.* at 2360. In reviewing all of the data, EPA "concluded that the concordance of outcomes across humans and laboratory rodents provides striking evidence of neurotoxic effects" (emphasis added). *Id.* at 2361.

Subsequently, EPA conducted its own risk assessment of harms to exposed workers, in the process of evaluating nPB under TSCA.[65] EPA concluded that when workers in a number of job categories are exposed to nPB they faced increase risk across all five estimated health variables: liver toxicity, kidney toxicity, reproductive toxicity, developmental toxicity, and neurotoxicity.[66] Additionally, EPA estimated that workers exposed to nPB face significantly increased cancer risk, with some exposed workers likely to experience an increased risk of cancer greater than 1-in-100.[67]

Comments submitted by the nPB manufacturers do not dispute these adverse health effects from nPB exposure. Instead, they erroneously argue that only harm to people beyond the fence line can meet the test for listing in section 112(b)(3)(B). Accordingly, they argue that EPA should refuse to consider to the evidence of harm to workers. For example, without discussion or explanation, Albemarle cites a conclusory statement from EPA in 2005 that: "EPA cannot consider the health effects of emissions within facility boundaries. That is the purview of the Occupational Safety and Health Administration." 70 Fed. Reg. 75,047, 75,055 (2005). When EPA made that conclusory statement, in the context of delisting methyl ethyl ketone in response to an industry petition, it provided no legal support for its conclusion, which was neither challenged nor upheld in court.[68] Regardless, the plain text of the Clean Air Act belies that conclusion. As discussed further below, there is no such limitation on EPA's authority for listing a HAP, and it would be both unlawful and arbitrary for EPA to refuse to consider available scientific evidence of harm that meets the statutory test, whether that harm is demonstrated to workers or other community members within or outside of the facility boundary.

1. The statute requires EPA to consider all relevant record evidence that exposure to nPB causes adverse health effects in humans, including evidence of harm to workers.

The existence of adverse health effects to workers, as to any other human exposed to an air pollutant, is sufficient to require listing of nPB as a HAP under the plain meaning of the Act. The test for listing under section 112(b)(3)(B) is whether "adverse effects to human health" are "known" or "may reasonably be anticipated." 42 U.S.C. § 7412(b)(3)(B). Workers exposed to harmful levels of nPB are "human," and therefore fall within the plain language of the statute. Likewise, "human health" plainly includes worker health. Further, workers can experience adverse effects through "emissions, ambient concentrations, bioaccumulation or deposition" of

[65] EPA Draft Risk Assessment at 126-40.
[66] *Id.* at 126-34; *e.g. id.* at 126 ("workers in spray adhesive facilities "showed risks for all of the health effects examined")
[67] *Id.* at 139; *see generally id.* at 134-140.
[68] EPA's statement in that record is both unsupported in its own right, and has no precedential impact in any other listing decision.

an air pollutant just as other humans do. Therefore, under the plain language of the statute, evidence of adverse effects to the health of workers is sufficient to compel listing of an air pollutant such as nPB.

Nothing in the text of section 7412(b)(3) draws a distinction between occupational exposures and non-occupational exposures. The statute contains no exception for health effects for workers, nor does it state that air pollution only begins at the fence line of an industrial source releasing toxic chemicals. Further, in the very same provisions of the Act governing listing of new HAPs, Congress exempted certain categories of emissions and certain types of effects from consideration, demonstrating that where Congress wished to create such an exemption, it do so expressly. For example, in subsection 7412(b)(2), Congress instructed EPA not to add air pollutants listed under § 7408(a) (unless certain factors are met). Congress also narrowed the types of environmental effects EPA could consider in determining what constitutes an "adverse environmental effect," restricting EPA to "significant and widespread adverse effect[s]" or "significant degradation of environmental quality over broad areas," *see* 42 U.S.C. 7412(a)(7), and excluding certain substances, practices, processes or activities regulated under the ozone protection subchapter of the Act. *Id.* § 7412(b)(2) (citing subchapter VI). Congress inserted no such language pertaining to workers or fence lines.

Other statutes contain express references to the fence line, confirming that the omission of such language in the Clean Air Act prohibits EPA from ignoring emissions within the fence line and their effect on workers. For example, in EPCRA Congress included just such language, directing EPA to list "a chemical [that] is known to cause or can reasonably be anticipated to cause significant adverse acute human health effects **at concentration levels that are reasonably likely to exist beyond facility site boundaries**." 82 Fed. Reg. at 2362. (citing EPCRA) (emphasis added). In contrast, the Clean Air Act contains no such requirement or limitation. Therefore, a petitioner need not demonstrate exposure or adverse health effects beyond the fence line in order to show that EPA is required to list a chemical as a HAP.

This is particularly true because the hazardous air pollutant provisions of the Clean Air Act are concerned with protecting the health of all members of the public, not simply the health of non-workers. While other statutes are more narrowly focused on worker health, the Clean Air Act is not so limited and covers community health (including worker health). For example, the declaration of purposes for the subchapter that includes section 112 includes the broad goal of promoting "the public health and welfare and the productive capacity of [the Nation's] population." 42 U.S.C. § 7401(b)(1). This includes workers implicitly and explicitly—workers are the "productive capacity" of that population. *Id.*

Moreover, legislative history demonstrates that Congress recognized that workers and the public should not be viewed separately or in isolation: "People who work in a factory in which dangerous substances are handled in high concentration, may live in an adjacent area in which the same or other substances are dispersed, thus increasing overall exposure. More than one organ may be attacked because the offending substance is transported by two or more media. Synergistic effects among two or more substances, by which the combined effect is more than the sum of the separate effects, should be considered." H.R. REP. 95-294, 541, 1977 U.S.C.C.A.N. 1077, 1502. As explained: "The concept of total body burden should be the

significant indicator of exposure, rather than burden acquired in one or another part of the environment or from one or another toxic material." *Id.* (emphasis added). Therefore, for purposes of determining hazards to human health under the Clean Air Act, all exposures count.

Further, even if the Clean Air Act were exclusively concerned with protection of non-workers or exclusively concerned with exposures that occur outside of the workplace (which it is not), interpreting section 112(b)(3)(B) consistent with its plain meaning would still accord with that purpose. The listing under section 112 of substances that harm workers will protect the general public also, because listing is required only when harmful substances are also "air pollutants," and the definition of "air pollutant" includes the requirement that the substance be emitted into the ambient air. Because nPB is emitted into the ambient air, satisfying the first prong of section 112(b)(3)(B), there is no cause to impose an extra-statutory requirement that evidence of adverse effects to human health cannot be derived from work-based exposures. Additionally, there is no evidence that the *only* cause of the harm documented to workers in epidemiological studies came from their exposure while *inside* the fence lines. These workers were presumably chosen for the studies because they were likely to be among the most highly exposed individuals in the general population. That makes these studies highly relevant and useful to assess health impacts for the broader exposed population, not evidence that should be ignored in the health literature. Moreover, evidence of harm to workers is necessarily evidence of harm to humans, and therefore is directly relevant to the statutory inquiry of whether a substance is known to or may reasonably be anticipated to cause adverse effects to human health.

In short, the Clean Air Act requires EPA to consider all relevant evidence of adverse health effects resulting from exposure to nPB, including evidence of worker exposure and resulting harm. Any decision to ignore such evidence would be arbitrary, capricious, and not in accordance with law.

2. Even if all occupational health data were to be set aside, there is ample evidence of beyond-the-fence-line exposures and thus resulting health threats to the public that require listing.

Although EPA cannot lawfully ignore evidence of adverse health effects documented in workers as discussed above, the record before EPA is also replete with evidence of emissions and exposures to the public beyond the fence line. Thus, even if the industry commenters were correct that EPA is prohibited from considering emissions and impacts from this chemical that are solely within the fence line or due to occupational exposures (which they are not), EPA would still be required to list nPB.

The evidence that members of the general population are exposed to nPB beyond the fence line of emitting facilities is strong. Multiple studies have found the presence of nPB metabolites in the bodies of people within the general population. For example, a recent epidemiological study of 488 women in the third trimester of pregnancy found that 99% of the participants had the nPB metabolite (N-Acetyl-S-(n-propyl)-L-cysteine) in their urine.[69]

[69] Elizabeth Barksdale Boyle et al., *Assessment of Exposure to VOCs among Pregnant Women in the National Children's Study*, 13 Int'l J. Environ. Res. & Pub. Health 376 (Mar. 2016) (these

Similarly, the National Health and Nutrition Examination Survey found that at the 50th percentile of concentration, all groups had measurable quantities of this same metabolite.[70] A recent study confirmed the presence of this nPB metabolite in children,[71] who cannot credibly be presumed to be exposed to nPB within the fence line of an emitting source.

EPA has itself recognized that the general populace is exposed to nPB. In the Scope of the Risk Evaluation for nPB, EPA concluded that emissions from industrial and commercial uses of nPB were a "likely exposure pathway" for the general population.[72] Additionally, EPA recognized that members of the general population were likely to be exposed to nPB emissions from dry cleaning facilities at indoor spaces that are co-located with dry cleaners.[73] Similarly, the ATSDR Profile recognizes that "[e]xposure of 1-bromopropane to the general population may occur via inhalation of ambient air at locations in close proximity to the emissive use of 1-bromopropane due to potential vapor migration, such as degreasing operations or dry cleaners."[74]

Widespread exposure of members of the general population compels EPA to list nPB as a HAP.

C. EPA Lacks Discretion to Decline to List on Grounds of Cost, Feasibility, Cost-Effectiveness, or Economic or Industrial Importance.

EPA cannot consider costs or economic impacts at the HAP listing stage, as the sole factors for EPA's consideration are listed in the statute and focus on "adverse effects to human health or adverse environmental effects." 42 U.S.C. § 7412(b)(3)(A), (B); *see also id.* § 7412(b)(2) (providing more detail on various health effects).

Notwithstanding the plain statutory text, industry groups contend that EPA should consider cost. EPA should reject those requests, because that consideration would be unlawful at the listing stage. Section 112(b)(3)(B) does not permit EPA to ignore evidence of adverse effects to human health, or to decline to list nPB, on the ground that regulating sources of nPB allegedly would have economic costs, would require industry actions, or would have arguable negative consequences for industry. *Cf.* Nestrud Letter at 6. The plain language of the provision requires

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women participated in the National Children's Study and came from seven locations around the country).

[70] Ctrs. for Disease Control, Fourth National Report on Human Exposure to Environmental Chemicals, Updated Tables 612 (Feb. 2015), http://www.cdc.gov/biomonitoring/pdf/FourthReport_UpdatedTables_Feb2015.pdf. *See also* ATSDR at 138 (summarizing results of study which found that the general population is being exposed to nPB based on the presence of the AcPrCys metabolite).

[71] Ram B. Jain, *Levels of selected urinary metabolites of volatile organic compounds among children aged 6-11 years*, 142 Environmental Research 461 (2015).

[72] EPA, *Scope of the Risk Evaluation for 1-Bromopropane*, EPA-HQ-OPPT-2016-0741-0049 at 30 (June 2017).

[73] *Id.*

[74] ATSDR Profile at 146; *see also id.* at 7 ("The general population may be exposed to 1-bromopropane in air when it is used during aerosol applications due to potential vapor migration . . . .").

EPA to make its decision based on whether nPB is an air pollutant and based on the health and environmental impacts nPB causes or may cause. 42 U.S.C. § 7412(b)(3)(B). The statute provides that EPA "shall" list nPB on the basis of those considerations. It therefore leaves no room for EPA to decline to list nPB on the grounds urged by the nPB manufacturers.

Section 112 of the Clean Air Act carefully specifies when and how EPA may take cost into account, expressly providing for a limited consideration of cost at a later stage of the process. *See* 42 U.S.C. § 7412(d)(2). The limitations Congress imposed on EPA's ability to consider cost are clear and intentional. For example, Congress recognized that "[t]he public health consequences of substances which express their toxic potential only after long periods of chronic exposure will not be given sufficient weight in [a] regulatory process when they must be balanced against the present day costs of pollution control and its other economic consequences." S. Rep. 101-228 at 3567 (1989).

Not including any cost factor in the listing provision was a decision made to support the overall aim of Congress to remedy EPA's previous delays in regulating hazardous air pollutants by "eliminating much of EPA's discretion" and ensuring that EPA would indeed take action needed to protect public health. *New Jersey v. EPA*, 517 F.3d at 578. The Act's primary objective is to protect health, and at the listing stage, health factors are the only considerations EPA may use, as discussed above. *See also* 42 U.S.C. § 7401.

The statute's limitation of relevant factors to health considerations also reflects the reality that at the listing stage there is no defensible or reasonable way to assess costs, because there are no immediate costs that result from listing, alone, and any future costs will depend on other legal requirements and on later exercises of policy discretion in areas where EPA has discretion. At such later stages, EPA will have information regarding, for example, facilities likely to need to install pollution controls on or make other changes to reduce their emissions, and other types of information needed to assess costs. Attempting to determine costs now is premature, speculative, and simply not possible based on the record, and that is likely part of the reason why the Act does not require this consideration or allow it as a relevant factor for listing.

Further, the fact that another provision within section 112 provides for a particular kind of cost consideration at a later stage of the process shows Congress' intent not to include such a factor at the listing stage. *See* 42 U.S.C. § 7412(d)(2). The Supreme Court has "refused to find implicit in ambiguous sections of the [Clean Air Act] an authorization to consider costs that has elsewhere, and so often, been expressly granted," and that same principle applies here. *See Whitman v. Am. Trucking Ass'ns*, 531 U.S. 457, 467 (2001).

The language the Supreme Court relied on in *Michigan v. EPA* to find that the statute directed a consideration of costs when listing a particular category of HAP sources (*i.e.*, electric utilities or power plants) was excluded from the listing test in § 7412(b)(3). 135 S. Ct. 2699, 2707 (2015). In that case, the Supreme Court held that the "appropriate and necessary" language in the listing provision for power plants required consideration of cost. *Id.*; 42 U.S.C. § 7412(n)(1)(A). No such language exists in subsection 112(b)(3). It does not say, for example, to list air pollutants that are known to cause adverse health effects *if appropriate and necessary*. Thus, there is no valid basis for EPA to consider costs in determining whether to list a HAP, and it would be unlawful for EPA to do so. Because of the serious health effects at stake, it also would be arbitrary for EPA to consider costs to a small set of industry groups, when the public interest, in health protections, strongly weighs in favor of listing a chemical.

IV. EPA MUST EXPEDITIOUSLY LIST N-PROPYL BROMIDE AS A HAP

Given the statutory requirements of the Clean Air Act and the record before the agency, EPA has only one legal, available course of action: to expeditiously list nPB as a HAP. Any further delay in taking this step would exacerbate EPA's extant and continuing violation of the statutory deadlines. Any other decision would be arbitrary, capricious, or otherwise in violation of law.

A. EPA's Listing Action Is Long Overdue.

EPA has failed to fulfill its legal duty to list nPB as a HAP within 18 months of receipt of a complete petition (or deny the petition within the same time). 42 U.S.C. § 7412(b)(3)(A)-(B). HSIA petitioned EPA to list nPB as a HAP on October 28, 2010, and on November 30, 2012, HSIA supplemented that petition with additional information requested by EPA (requiring EPA action within 18 months, by March 28 or April 30, 2014, respectively). The New York State Department of Conservation petitioned EPA to list nPB as a HAP on October 24, 2011 (requiring action within 18 months, by March 24, 2013). On February 6, 2015, EPA published a notice that the petitions were complete (requiring action within 18 months, by August 8, 2016). In light of EPA's completeness determination, there is no doubt that the agency is overdue in fulfilling its statutory duty to take final action to grant these petitions.

These statutory deadlines for EPA to take required action on these petitions have now elapsed. EPA has been aware of this for years.[75] Yet, EPA has not taken final action on the petitions as the Act legally required it do long before now. Based on the record before the agency, we urge EPA to promptly list nPB as a HAP to fulfill Clean Air Act requirements and ensure that the public receives the full protection it needs from this dangerous pollutant.

B. EPA May Not Lawfully or Cannot Rationally Reverse Course on its Determination to List n-Propyl Bromide.

In proposing to list nPB as a HAP, EPA looked at the entirety of the record and concluded that nPB is an air pollutant and that exposure to emissions and ambient concentrations of nPB were known or reasonably could be anticipated to cause adverse health effects. Specifically, EPA's Notice makes clear that it has made a sound determination, based on the science, that adverse carcinogenic, reproductive, developmental, and neurological effects are known or reasonably anticipated effects from nPB exposure. 82 Fed. Reg. at 2359-62. That determination is reasonable in light of the record evidence; indeed, it is the only reasonable conclusion that could be drawn based on the best available science.

As EPA's Notice describes, this is the second and final stage of its process to review petitions to add HAPs. 82 Fed. Reg. at 2356. As stated therein, before publishing the Notice at issue here, the agency "decide[s] whether the petition satisfies the requirements of CAA section 112(b)(3)(B) and adequately supports a decision to grant the petition." *Id.* Then, "[u]pon conclusion of this review, [EPA] publish[es] a draft notice in the Federal Register with the

[75] *See, e.g.*, Letter from HSIA to EPA Re: Notice of Intent to File Citizen Suit (Dec. 19, 2014), https://www.epa.gov/sites/production/files/2015-05/documents/hsianoi12192014_0.pdf.

written explanation of the Administrator's decision to grant the petition. After considering the comments received on the draft document, we publish a final notice in the Federal Register." *Id.* The Notice addressed by these comments is the Notice EPA published "upon conclusion" of its review of the petitions and scientific evidence they contain. It provides the draft explanation of EPA's final decision, but that decision itself is complete. The only remaining step needed for EPA to fulfill its duty regarding these petitions is publication of a final notice of the grant of the petitions, after consideration of comments.

New record evidence only further supports those conclusions. Based on the scientific record before the agency, EPA may not lawfully or rationally change course on its scientific conclusions or the final determination to list nPB as a HAP. Given the overwhelming and unequivocal evidence of adverse human health effects, EPA could not possibly provide a reasoned explanation for departing from its scientific determination that nPB meets the listing test or its proposed rationale for granting the petitions to list nPB as a HAP. *See FCC v. Fox*, 556 U.S. 502, 515 (2009) (recognizing that a "more detailed explanation" is required where an agency not only changes course but disregards prior factual findings); *see also Am. Petroleum Inst. v. EPA*, 862 F.3d 50, 66 (D.C. Cir. 2017) (a change in agency position must be "justified by the rulemaking record"). Therefore, EPA must follow the logical conclusion from its well-supported scientific determination and must now take final action to complete its mandatory duty to grant the pending petitions and list nPB as a HAP. Any other decision would be arbitrary, capricious, and not in accordance with law.

CONCLUSION

For the foregoing reasons, Commenters urge EPA to do what the Clean Air Act mandates: expeditiously list nPB as a HAP and ensure that EPA fulfills its legal duty to protect the public from this dangerous air pollutant. EPA should take final action to grant the petitions and list n-propyl bromide as a hazardous air pollutant, and finalize the rationale for doing so, as it has provided in the Notice. For additional information on these Comments, please contact Emma Cheuse or Tosh Sagar, Earthjustice, at (202) 667-4500.

Sincerely,

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EARTHJUSTICE

LIST OF APPENDIX DOCUMENTS

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