

October 28, 2010

Honorable Lisa P. Jackson
Administrator
Environmental Protection Agency
1200 Pennsylvania Avenue, NW
Washington, DC 20460

Re: Petition to Add n-Propyl Bromide to List of Hazardous Air Pollutants
Regulated under § 112 of the Clean Air Act


Dear Ms. Jackson:

On behalf of the Halogenated Solvents Industry Alliance, Inc. (“HSIA”), an association of producers and users of chlorinated solvents, I submit the enclosed petition pursuant to Clean Air Act (“CAA” or the “Act”) § 112(b)(3), 42 U.S.C. § 7412(b)(3). The petition seeks the addition of n-propyl bromide to the list of hazardous air pollutants codified at § 112(b)(1) of the Act, 42 U.S.C. § 7412(b)(1), on the basis 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 or adverse environmental effects,” CAA § 112(b)(3)(B), 42 U.S.C. § 7412(b)(3)(B).

The petition addresses uses of and exposure to n-propyl bromide. It then describes the scientific studies and other information that demonstrate that emissions of nPB are known to cause or may reasonably be anticipated to cause adverse effects to human health. We believe that the case for listing is strong and we urge you to grant the petition well in advance of the 18-month statutory deadline for action.

Please do not hesitate to have your staff contact me if we can provide further information that will assist in your review.

Very truly yours,


W. Caffey Norman

Enclosure

cc: Honorable Regina A. McCarthy
C. Scott Fulton, Esq.

BEFORE THE ENVIRONMENTAL PROTECTION AGENCY

**Petition to Add n-Propyl Bromide to List of Hazardous Air Pollutants
Regulated under § 112 of the Clean Air Act**

**HALOGENATED SOLVENTS INDUSTRY ALLIANCE, INC.
1530 Wilson Boulevard
Arlington, VA 22209**

OF COUNSEL:

**W. Caffey Norman
Patton Boggs LLP
2550 M Street, NW
Washington, DC 20037**

October 28, 2010

Petition to Add n-Propyl Bromide to List of Hazardous Air Pollutants Regulated under § 112 of the Clean Air Act

Introduction

The Clean Air Act Amendments of 1990 established an initial list of hazardous air pollutants, now codified at § 112(b)(1) of the Clean Air Act (“CAA” or the “Act”), 42 U.S.C. § 7412(b)(1), sources of which are subject to regulation under CAA §§ 112 (d), (f), (k) and other subsections. CAA § 112(b)(3), 42 U.S.C. § 7412(b)(3), provides that any person may petition the Administrator to add or delete a substance to the list, and requires the Administrator to grant or deny the petition and to publish a written explanation of the reasons for her decision within 18 months after receipt of the petition. The Halogenated Solvents Industry Alliance, Inc. (“HSIA”) submits this petition pursuant to CAA § 112(b)(3), 42 U.S.C. § 7412(b)(3). HSIA is an association of producers and users of chlorinated solvents.

By its terms, CAA § 112(b)(3)(A), 42 U.S.C. § 7412(b)(3)(A), requires a petition to add a substance to the list of hazardous air pollutants to include a showing that there is adequate data on the health or environmental effects of the pollutant in question to support the petition. Specifically, CAA § 112(b)(3)(B), 42 U.S.C. § 7412(b)(3)(B), provides that an air pollutant shall be added to the list if “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 or adverse environmental effects.” This petition addresses the uses of and exposure to n-propyl bromide (also known as 1-bromopropane, and hereinafter as “nPB”) (CAS No. 106-94-5). It then describes the scientific studies and other information that demonstrate that emissions of nPB are known to cause or may reasonably be anticipated to cause adverse effects to human health.

Use and Production of nPB

NPB is a brominated hydrocarbon with a strong odor. Its chemical formula is C_3H_7Br . NPB is used as a carrier solvent in aerosols and adhesives. Some brand names of products using nPB are: Abzol,[®] EnSolv,[®] and Solvon[®] cleaners; Pow-R-Wash[®] NR Contact Cleaner, Superkleen Flux Remover 2311, and LPS NoFlash NU Electro Contact Cleaner aerosols; and Whisper Spray and Fire Retardant Soft Seam 6460 adhesives. It also is used to

remove wax, oil, and grease from electronics, metal, and other materials.¹ Specific applications include:²

Aerosol Solvents. NPB is used as an aerosol solvent in lubricants, coatings, or cleaning fluids for electrical or electronic equipment; lubricants, coatings, or cleaning fluids for aircraft maintenance; and spinnerette lubricants and cleaning sprays used in the production of synthetic fibers.

Adhesives. NPB is used in adhesives for laminates, flexible foam, hardwood floors, tire patches, and metal to rubber adhesives. Of these applications, nPB-based adhesives have been used most widely in spray adhesives used in manufacture of foam cushions, and to a lesser degree in laminate adhesives.

Electronics, Metal, and Precision Cleaning. NPB is used in open vapor degreasing applications to remove processing lubricants such as oils, greases, and waxes which have been applied to aid manufacture but need to be removed before further processing of the manufactured substance.

Originally, nPB was used as a chemical intermediate in closed processes. It was nominated to the National Toxicology Program (NTP) for testing as a result of its introduction into the emissive applications described above:

"In the early to mid 1990s, 1-bromopropane was used primarily as an intermediate in the production of pesticides, quaternary ammonium compounds, flavors and fragrances, pharmaceuticals, and other chemicals in well-controlled, closed processes. In the mid to late 1990s, it was introduced as a less toxic replacement for methylene chloride in emissive applications such as vapor and immersion degreasing operations and critical cleaning of electronics and metals. 1-Bromopropane was also introduced as a nonflammable, nontoxic, fast-drying, and inexpensive solvent for adhesive resins, and has been marketed as a replacement for ozone depleting refrigerants. 1-Bromopropane was nominated for study by the Occupational Safety and Health Administration based on the potential for widespread occupational and environmental exposure and a lack of toxicity and carcinogenicity data. Male and female F344/N rats and B6C3F1 mice were exposed to 1-bromopropane (99% or greater pure) by inhalation for 2 weeks, 3 months, or 2 years. Genetic toxicology studies were conducted in *Salmonella typhimurium* and *Escherichia coli* and mouse peripheral blood."³

¹ 72 Fed. Reg. 30168, 30171 (May 30, 2007).

² *Id.*

³ NTP Technical Report 564 (Board Draft), copy enclosed.

As a result of the growing emissive use, global production of nPB was estimated to be 20,000-30,000 metric tonnes in 2007.⁴ It is assumed to be produced in China, France, India, Israel, Japan, Jordan, and the United States. China is estimated to have produced around 20,000 metric tonnes in 2008, of which approximately 40% were exported. Use as a solvent was reported to be growing at a rate of 15-20% per year in the United States (5,000 metric tonnes) and Asian countries other than China. Solvent use in Japan was 1,100 - 1,200 metric tonnes in 2009. No information was available to the United Nations Environment Programme (UNEP) for other regions.⁵

Exposure to nPB

Attached is a summary of workplace exposure data for nPB collected by the EPA Significant New Alternatives Policy (SNAP) program from a variety of sources.⁶ Given its use across a variety of applications, some of which are uncontrolled, it is not surprising that there is a wide range of exposures in the workplace, from a few parts per million (“ppm”) up to 150 ppm or more.

A recently published study of exposure to nPB in New Jersey dry cleaning shops, attached, found exposures as high as 54 ppm.⁷ The report states:

“This study suggested that some green or organic alternative solvents in the dry cleaning industry intended to replace PERC may result in significant exposures above acceptable guidelines among some workers. One dry cleaning worker reported serious adverse health effects resulting in an emergency room visit. Careful consideration and study of potential exposures and health effects

⁴ Montreal Protocol On Substances that Deplete the Ozone Layer, Report of the UNEP Technology and Economic Assessment Panel (Progress Report, Volume 2, May 2010), 59. http://www.unep.org/ozone/teap/Reports/TEAP_Reports/. Because of its ozone depleting potential, the Parties to the Protocol have sought to limit nPB use to those situations where more economically feasible and environmentally friendly alternatives are not available (Decision XIII/7).

⁵ *Id.* UNEP reports that “Obtaining more complete and accurate data on production and uses of n-PB, as well as its emissions, continues to be difficult. No governmental records are available on emission or uses since n-PB is not classified or registered as a controlled chemical substance like CFCs, and HCFCs (ODS class I and II) nor designated as a hazardous air pollutant in the Clean Air Act in the USA or reportable compound for pollution release (emission) and transfer (PRTR) in Europe and Japan.”

⁶ EPA-HQ-OAR-2002-0064-0015.

⁷ Blando, *et al.*, Preliminary Study of Propyl Exposure among New Jersey Dry Cleaners as a Result of a Pending Ban on Perchloroethylene, *J. Air & Waste Manage. Assoc.* 60(9): 1049-56 (2010).

among workers using new chemicals or new processes must precede any regulatory attempt to facilitate substitution for environmental purposes.”

Health Effects of nPB

1. Reproductive Toxicity

In a proposed rule to list nPB as acceptable for certain applications and unacceptable for others under its SNAP program, EPA discussed the available reproductive toxicity data in some detail.⁸ EPA based its analysis of occupational exposures on effects on sperm motility observed in a two-generation reproductive toxicity study employing a benchmark dose (BMD) calculation.⁹ Since that time, additional BMD calculations have been developed based on effects on estrus cycle in the same multigeneration study. Petitioner has reviewed the documents provided by EPA detailing the selection of endpoints, the choice of study, and the preferred calculations of BMD and the lower 95% confidence limit on the BMD (BMDL). We agree that the BMDL values selected (169 ppm based on sperm motility, 162 ppm based on effects on estrus cycle) are appropriate and follow accepted methods and principles. These values were then adjusted to take into account the temporal differences between the exposure regime in the rat study (6 hours per day, 7 days per week) and occupational exposures of 8 hours per day, 5 days per week. The resultant human equivalent concentrations (HECs) are 177 ppm and 170 ppm for male and female reproductive toxicity, respectively. The HEC value can provide a point of departure (POD) for calculation of a recommended exposure limit (REL) for noncancer effects. EPA’s approach to setting a limit for occupational exposure is similar to that for calculation of the reference concentration (RfC) which defines the inhalation concentration at which even sensitive members of the general population are unlikely to experience adverse effects. We have modified EPA’s calculations to derive an RfC for the general population. Although potential reproductive effects remain of concern, more recent evidence of neurotoxicity in workers and carcinogenicity in rodents may now indicate effects of greater concern.

⁸ 72 Fed. Reg. 30168 (May 30, 2007).

⁹ WIL, An Inhalation Two-Generation Reproductive Toxicity Study of 1-Bromopropane in Rats, sponsored by the Brominated Solvents Consortium (May 24, 2001).

EPA modified its normal procedure to calculate an RfC as a basis for determining a recommended exposure limit for workers. This calculation involves applying a sequence of uncertainty factors to the POD based on the extrapolation of animal data to human exposures, variability among the population, and the completeness of the database.

The maximum uncertainty factor applied for the animal to human extrapolation is 10-fold (*i.e.*, man is 10-fold more sensitive than the laboratory animal). This factor of 10 is considered to include a 3-fold safety factor for pharmacodynamic species differences (sensitivity at the site of action) and a 3-fold factor for pharmacokinetic differences (delivery of the active moiety to the site of action). For nPB, EPA adopted a 3-fold uncertainty factor for pharmacodynamic differences between rat and man, but elected to include no uncertainty factor (*i.e.*, a factor of 1) for pharmacokinetic differences.

EPA's judgment that no uncertainty factor is necessary for dose-response extrapolation from rat to man was based on a higher blood/air partition coefficient for nPB in rats than in man (11.7 versus 7.1). EPA concluded that, for a given external concentration, "the delivered dose of nPB into the bloodstream of rats is slightly higher than in humans." This relatively small difference in blood/air partition coefficients is insufficient, however, to overcome the many remaining uncertainties. The partition coefficient itself is only an indication of relative solubility and the target tissues in rat and man may be similar in ability to dissolve nPB such that, if nPB is poorly metabolized/cleared, the level achieved in the target tissue may be the same in the two species at equilibrium.

There are only certain situations in which the blood/air partition coefficient is the controlling factor in determining the level of the active moiety at the site of action. Without information on nPB metabolism in humans, this level cannot be predicted with physiologically-based pharmacokinetic ("PBPK") modeling for humans. The factors that support the application of a full 3-fold uncertainty factor for pharmacokinetics are the following:

- the active moiety or moieties for reproductive effects are not known,
- the relationship between blood level and active moiety is unknown,
- the nature and extent of nPB metabolism and general pharmacokinetics in man are not known,
- the difference in blood/air partition coefficients is small, and

- the blood/air partition coefficient is only controlling under certain conditions that can only be defined through PBPK modeling for man.

Taken together, these uncertainties are highly significant and support the application of a safety factor of 3 for pharmacokinetics for extrapolation from rat to man.

Combined with the 3-fold safety factor for pharmacodynamics, Petitioner believes that the full 10-fold uncertainty factor should be applied for the interspecies (rat to man) extrapolation. This is particularly important when one considers the greater human sensitivity to reproductive toxins relative to responses in the rat. The rat possesses huge overcapacity in reproductive capabilities whereas man is considered to be much closer to infertility as a species. This fact suggests that humans are fundamentally more susceptible to reprotoxins than the rat. Although this sensitivity is captured, to a limited extent, in the 3-fold uncertainty factor for pharmacodynamics, it may not be sufficient to account for the greater sensitivity of humans.

Regarding human variability, the RfC methodology allows up to a 10-fold uncertainty factor to account for the variability in sensitivity within the general population. In some circumstances, Petitioner acknowledges that this factor can be reduced for a working population because of the general good health required for employment. We agree with EPA's conclusion that there may be sensitive individuals for reproduction in the workforce because poor reproductive health may exist in individuals generally fit enough to work. EPA considers that variability in the worker population may be less than in the general population and that an uncertainty factor of 10-fold is greater than necessary. This is also a reasonable assumption and Petitioner supports the use of an intraspecies uncertainty factor of 3-fold for variations in sensitivity in a *worker* population exposed to nPB. As indicated by EPA, the general population is usually considered to include sub-populations that are more sensitive than the relatively "healthy" workers. This would indicate that an uncertainty factor of 10 should be applied for human variability in the general population.

At the time that EPA was deriving the occupational exposure limit, there was no information regarding effects of long-term exposures to nPB. This normally leads to inclusion of a "database uncertainty factor" of up to 10-fold. The subsequent results of NTP 2-year cancer bioassays in rats and mice (discussed below) show that such an uncertainty factor should have been included for workers and a full factor of 10 would have been appropriate for the general population. The composite uncertainty factor for the general population is therefore 1000 and

this, applied to the HEC based on the BMDL, leads to an RfC of 0.17 ppm for male and female reproductive effects.

2. Neurotoxicity

Earlier reviews of the neurotoxicity of nPB focused on animal studies in which hind-limb weakness and neurophysiological measurements indicated effects on the peripheral nervous system. More recently, there have been a number of reports of severe neurotoxicity in workers exposed to nPB. In particular, the human effects noted relate to lower limb peripheral nerve effects with adverse sensory responses. These effects are qualitatively similar to those seen in rat experiments -- limb weakness progressing in some cases to paralysis. Central nervous system effects also were reported in a proportion of human cases.

The consistency of severe effects across multiple independent investigations is remarkable and, although the measurements of atmospheric levels have limitations, the similarity of exposure levels associated with effects is striking. Many of the case subjects needed hospitalization and the reversibility of the effects is still in question. A publication by Majersik *et al.* provides a complete clinical analysis of the effects and indications of the slow recovery, if any, observed in patient follow-up.¹⁰ More recently, two cases were reported involving workers exposed to nPB (in vapor degreasing and dry cleaning, respectively) who were diagnosed with clinical manifestations of nPB.¹¹

Petitioner believes that neurotoxicity in humans exposed to nPB is of substantial concern. A recently published study showing neurophysiological effects in workers exposed to a range of levels allows for derivation of a dose response relationship for female workers.¹² This well-conducted study allowed the authors to calculate the lowest dose level at which female workers would be likely to experience measurable adverse effects (neurological and red blood cell reduction in number). This exposure is 1.28 ppm (presumably an 8 hour time-weighted average). Li *et al.* also report various effects associated with exposure to nPB and provide a

¹⁰ Majersik *et al.*, Severe Neurotoxicity Associated with Exposure to the Solvent 1-Bromopropane (n-Propyl Bromide), *Clinical Toxicology* 45: 270-276 (2007).

¹¹ Perrone *et al.*, Neurologic Illness Associated with Occupational Exposure to the Solvent 1-Bromopropane – New Jersey and Pennsylvania, 2007 – 2008, *Morbidity and Mortality Weekly Report* 57(48): 1300-1302 (2008).

¹² Li *et al.*, Dose-Dependent Neurologic Abnormalities in Workers Exposed to 1-Bromopropane, *J. Occup. Environ. Med* 52: 769-777 (2010).

clear definition of a dose-response relationship indicated in an earlier study.¹³ Taking the results of Li *et al.* and translating them to an RfC value for the general population involves adjusting the 1.28 ppm from an 8 hour, 5 days per week occupational exposure to a continuous exposure, which a lowest-observed-adverse-effect level (LOAEL) of 0.3 ppm. Although this is based on effects observed in humans, EPA will still apply uncertainty factors in deriving an RfC. Thus, an uncertainty factor as high as 10-fold would be applied for a LOAEL in the absence of a no-observed-adverse-effect level (NOAEL). In this case, a factor of 3 is probably sufficient since the calculated number is presumed to reflect the lowest level of detection. As the LOAEL is based on responses of workers, a factor for sensitive sub-populations in the general population would be applied. A factor of 3 may be sufficient to cover this uncertainty. The composite uncertainty factor to apply is thus 10-fold and the resultant RfC would be 0.03 ppm.

3. Carcinogenicity

Perhaps the most significant recent information relevant to the health hazard on nPB are the results of 2-year carcinogenesis studies conducted and reviewed by NTP. The enclosed draft report, which was unanimously approved by the NTP Technical Reports Review Subcommittee, shows:

- Clear evidence of carcinogenicity in female F344 rats (adenomas in large intestine and equivocal evidence for skin tumors)
- Some evidence of carcinogenicity in male F344 rats (adenomas in large intestine, skin tumors, and equivocal findings for mesotheliomas and pancreatic adenomas)
- Clear evidence of carcinogenicity in female B6C3F1 mice (lung tumors)
- No evidence of carcinogenicity in male B6C3F1 mice

At this time, there are no reasons to assume that the mode, or modes, of action by which tumors are induced by nPB are not relevant to man and thus a linear, no-threshold dose-response relationship should be assumed. Using standard methodology for the calculation of cancer potency and risk estimation (the linearized multistage model) applied to the incidence of mouse lung tumors yields a q_1^* term. This value is the 95% confidence limit of the linear term resulting from the model. The cancer potency term for mouse lung tumors from the NTP bioassay is a

¹³ Ichihara *et al.*, Neurological Abnormalities in Workers of a 1-Bromopropane Factory, *Environmental Health Perspectives* 112(13): 1319-1325 (2004).

risk of 1.95×10^{-3} per ppm (lifetime exposure). This potency value indicates that a lifetime risk of 1 in 1 million is potentially associated with an exposure of 0.5 ppb or $2.5 \mu\text{g}/\text{m}^3$.

Recommendations/Regulatory Measures of Other Authorities

1. American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Values (TLVs[®])

TLVs[®] refer to airborne concentrations of chemical substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed, day after day, over a working lifetime, without adverse health effects. The TLV[®] for nPB is 10 ppm as an 8-hour time-weighted average (TWA). This limit was set without regard to the recently reported cancer bioassay results, and is likely to be lowered in the future.

2. California Workplace Limit

The California Occupational Safety and Health Standards Board establishes workplace standards for toxic materials that assure, to the extent feasible, that no employee will suffer material impairment of health or functional capacity if exposed for a working lifetime. The Board recently adopted a Permissible Exposure Limit (PEL) for nPB of 5 ppm as an 8-hour TWA. Its rationale was as follows:

“A new Permissible Exposure Limit for 1-bromopropane is proposed as an 8-hour time-weighted average of 5 ppm TWA (25 mg/M³). The PEL proposed differs from the TLV of 10 ppm adopted by the ACGIH in 2005. 1-bromopropane is a potential substitute for solvents used to clean metals and electronics such as in specialized aircraft part degreasing operations and has been used as a solvent in adhesives. The documentation of the TLV for 1-bromopropane indicates that it was adopted to provide protection against the potential for neurotoxicity, hepatotoxicity, and reproductive and developmental toxicity. The PEL proposed also differs from the recommendation of the Committee for a PEL of 1 ppm. The Committee at its meeting January 9, 2004 considered two possible 8-hour TWA PEL recommendations presented by Julia Quint of Hazard Evaluation Systems and Information Service (HESIS): 1 ppm based on 2003 HESIS and Office of Environmental Health Hazard Assessment (OEHHA) documents and 3.3 ppm based on an assessment by the National Toxicology Program’s (NTP) Center for the Evaluation of Risks to Human Reproduction (CERHR). The OEHHA assessment, dated December 2, 2003, was conducted for the California Air Resources Board and was based on reproductive system effects in male rats reported by Ichihara et al. in 2000. The CERHR assessment, dated October 2003, was based on reproductive system effects in both male and female rats reported in a study by WIL Research Laboratories in 2001 sponsored by the Brominated Solvents Consortium. The

value of 3.3 ppm is calculated from the No Observed Adverse Effect Concentration (NOAEC) of 100 ppm in test animals in the WIL Research study, application of an interspecies uncertainty factor of 3.16 (the square root of 10) and an intraspecies uncertainty factor of 10, and adjustment for an 8-hour workday and 5-day workweek. At the May 18, 2005 advisory meeting, representatives of manufacturers and vendors of 1-bromopropane products commented on the Committee's recommendation of a PEL of 1 ppm. Their comments included concerns that a PEL of 1 ppm would amount to a ban on the use of 1-bromopropane in California and that there were flaws in the Ichihara study underlying the OEHHA Interim Reference Exposure Level. They also noted the importance of skin absorption and suggested that a PEL for 1-bromopropane should include a "skin" notation. In light of concerns with the Ichihara study expressed by these stakeholders and by the authors of the CERHR report, this rulemaking proposes a PEL of 5 ppm (8-hour TWA) for 1-bromopropane, along with a "skin" notation. The PEL of 5 ppm is a rounding up from the level of 3.3 ppm to a more standardized PEL value."¹⁴

As in the case of the TLV[®] recommendation discussed above, this limit was set without regard to the recently reported cancer bioassay results, and is likely to be lowered in the future.

3. California Proposition 65

Effective December 7, 2004, nPB was listed as a chemical known to the state of California to cause reproductive toxicity for purposes of the Safe Drinking Water and Toxic Enforcement Act of 1986 ("Proposition 65"). The listing was based on a formal identification by an authoritative body that nPB¹⁵ causes developmental and female and male reproductive toxicity. A "safe harbor" exposure level has not yet been adopted but is normally 1000-fold below the no observed effect level for reproductive toxicity, and thus is likely to be in the region of 1 -2 ppb.

4. European Union Classification and Labelling

The following "risk phrases" have been assigned to nPB: R 11 (highly flammable), R36/37/38 (irritant to eyes/skin/respiratory system), R 48/20 (danger of serious damage to health by prolonged exposure/harmful by inhalation), R 60 (may impair fertility), R 63 (possible risk of harm to the unborn child), R 67 (vapors may cause drowsiness and dizziness). The results of the

¹⁴ California Occupational Safety and Health Standards Board, Initial Statement of Reasons, Airborne Contaminants (March 19, 2009); https://www.dir.ca.gov/oshsb/airborne_contaminants09_ISOR.doc.

¹⁵ National Toxicology Program Center for the Evaluation of Risks to Human Reproduction Expert Panel Report on the Reproductive and Developmental Toxicity of 1-Bromopropane (nPB) (March 2002).

NTP carcinogenicity bioassay are not reflected in this list but would be expected to add an additional risk phrase to be displayed on labels and safety data sheets.

Conclusion

Chlorinated solvents are listed as hazardous air pollutants and industrial sources of chlorinated solvent emissions are stringently regulated under CAA §§ 112(d) and (f). nPB is being aggressively marketed as an unregulated alternative to chlorinated solvents.¹⁶ Indeed, sales literature for nPB emphasizes that it is essentially unregulated. Sales of nPB in adhesive and solvent cleaning uses are already substantial and appear poised for further growth.

The results of the recently reported carcinogenicity bioassays show “clear evidence of carcinogenic activity” in female rats and female mice. An estimate of carcinogenic potency using standard methodology shows that the cancer potency is similar to or higher than that of the regulated materials nPB is being marketed to replace. Moreover, observations of toxic effects of nPB in animal experiments and workers exposed at various levels demonstrate that exposed populations may face other serious health risks, including reproductive effects (male and female), liver and kidney toxicity, immunotoxicity, and effects on red blood cells and hemoglobin. High levels of worker exposure have led to a number of documented cases of lower limb paralysis that has been slow to improve; the onset of adverse effects on peripheral nerves has been detectable in workers exposed at very low levels (estimated lowest-observed-effect level was 1.28 ppm). This translates to concern for the general population exposed to very low levels.

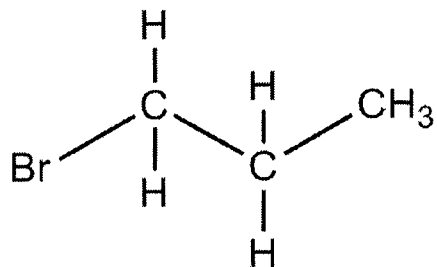
¹⁶ E.g., <http://www.envirotechint.com/products/dry-cleaning/>: “Being “Green” has never been easier with DrySolv. The first environmentally responsible replacement solvent for your dry cleaning machine!

- DrySolv is non-flammable, showing no flashpoint in multiple tests and test methods.(ASTM D-56 TCC, ASTM D-92 COC, ASTM D-93 TCC).
- DrySolv is also non-chlorinated.
- DrySolv is non-hazardous.(DOT, OSHA, NESHAP, RCRA, Clean Water Act)
- DrySolv is not a hazardous air pollutant, is SNAP approved, and does not contribute to global warming. (NESHAP, Significant New Alternative Program-SNAP approved (Federal EPA), Not Title V)
- The USEPA states that DrySolv’s main ingredient is less persistent in the environment than many other solvents, is of low to moderate concern for movement in soil, does not warrant listing under the Toxics Release Inventory and is not prone to bioaccumulation. (USEPA - Federal Register May 30, 2007).
- DrySolv **does not** have a hazardous decomposition or hazardous polymerization.”

Clearly, nPB meets the statutory criteria for listing as a hazardous air pollutant under CAA § 112(b)(3)(B), 42 U.S.C. § 7412(b)(3)(B). We urge EPA forthwith to grant this petition.

Attachments

ABSTRACT



1-BROMOPROPANE

CAS No. 106-94-5

Chemical Formula: C₃H₇Br Molecular Weight: 122.99

Synonyms: 1-BP; propyl bromide; n-BP; N-propyl bromide

In the early to mid 1990s, 1-bromopropane was used primarily as an intermediate in the production of pesticides, quaternary ammonium compounds, flavors and fragrances, pharmaceuticals, and other chemicals in well-controlled, closed processes. In the mid to late 1990s, it was introduced as a less toxic replacement for methylene chloride in emissive applications such as vapor and immersion degreasing operations and critical cleaning of electronics and metals. 1-Bromopropane was also introduced as a nonflammable, nontoxic, fast-drying, and inexpensive solvent for adhesive resins, and has been marketed as a replacement for ozone depleting refrigerants. 1-Bromopropane was nominated for study by the Occupational Safety and Health Administration based on the potential for widespread occupational and environmental exposure and a lack of toxicity and carcinogenicity data. Male and female F344/N rats and B6C3F1 mice were exposed to 1-bromopropane (99% or greater pure) by inhalation for 2 weeks, 3 months, or 2 years. Genetic toxicology studies were conducted in *Salmonella typhimurium* and *Escherichia coli* and mouse peripheral blood.

2-WEEK STUDY IN RATS

Groups of five male and five female rats were exposed to 1-bromopropane vapor at concentrations of 0, 125, 250, 500, 1,000, or 2,000 ppm, 6 hours plus T₉₀ (12 minutes) per day, 5 days per week for 16 days. All rats survived to the end of the study except one 500 ppm male. Mean body weights of 2,000 ppm rats were significantly less than those of the chamber controls. The absolute kidney weight of 1,000 ppm males, relative kidney weights of all exposed groups of males, and absolute and relative kidney weights of all exposed groups of females were significantly increased. The absolute and relative liver weights of 1,000 ppm males, relative liver weights of 500 and 2,000 ppm males, and absolute and relative liver weights of 500 ppm or greater females were significantly increased. Nasal lesions included suppurative inflammation in males exposed to 500 ppm or greater, respiratory epithelial necrosis in 1,000 and 2,000 ppm males, and respiratory epithelial regeneration in 1,000 and 2,000 ppm females.

2-WEEK STUDY IN MICE

Groups of five male and five female mice were exposed to 1-bromopropane vapor at concentrations of 0, 125, 250, 500, 1,000, or 2,000 ppm, 6 hours plus T₉₀ (12 minutes) per day, 5 days per week for 17 days. All 2,000 ppm males, two 2,000 ppm females, four 500 ppm males, one 1,000 ppm male, and one 1,000 ppm female died early. The mean body weight gain of 1,000 ppm males was significantly less than that of the chamber controls. Abnormal breathing, lethargy, and eye discharge were observed primarily during week 1 in groups exposed to 500 ppm or greater. Liver weights of 1,000 ppm males and of females exposed to 500 ppm or greater were significantly increased. Kidney weights of 1,000 and 2,000 ppm females were significantly increased. Microscopic lesions related to 1-bromopropane exposure occurred in the lung, liver, and nose of males and females and were primarily seen in mice exposed to 500 ppm or greater.

3-MONTH STUDY IN RATS

Groups of 10 male and 10 female rats were exposed to 1-bromopropane vapor at concentrations of 0, 62.5, 125, 250, 500, or 1,000 ppm, 6 hours plus T₉₀ (10 minutes) per day, 5 days per week for 14 weeks. Additional clinical pathology groups of 10 male and 10 female rats were exposed to the same concentrations for 23 days. All rats

survived to the end of the study. Mean body weights of 1,000 ppm males were significantly less than those of the chamber controls. The increases in sorbitol dehydrogenase activities in 500 ppm males and 1,000 ppm males and females were consistent with the histopathologic evidence of mild hepatotoxicity caused by 1-bromopropane. Liver weights of males exposed to 250 ppm or greater and of females exposed to 125 ppm or greater were significantly increased. Spleen and kidney weights of 1,000 ppm females were significantly increased. Results of sperm count and vaginal cytology evaluations showed exposure concentration-related decreases in sperm motility and counts in male rats, reaching 28% and 37%, respectively, in the 1,000 ppm group. Female rats in all three exposure groups evaluated exhibited altered estrous cycles, spending significantly more time in extended estrus and less time in extended diestrus. The incidences of cytoplasmic vacuolization of the liver were significantly increased in males exposed to 250 ppm or greater and in females exposed to 500 ppm or greater. Hepatocyte degeneration was also observed in 1,000 ppm females.

3-MONTH STUDY IN MICE

Groups of 10 male and 10 female mice were exposed to 1-bromopropane vapor at concentrations of 0, 62.5, 125, 250, or 500 ppm, 6 hours plus T₉₀ (10 minutes) per day, 5 days per week for 14 weeks. One 250 ppm male and four males and five females in the 500 ppm groups died early. Mean body weights of exposed groups were similar to those of the chamber controls. Lethargy was observed in males and females exposed to 500 ppm, and abnormal breathing was observed in moribund mice. The kidney, liver, and lung weights of 500 ppm females were significantly greater than those of the chamber controls. The kidney weights of 500 ppm males were significantly decreased. Male mice in the 500 ppm group had decreased sperm counts that were 28% less than that in the chamber controls. Female mice exhibited altered estrous cycles, with females in the 500 ppm group spending significantly more time in extended diestrus and those in the 250 ppm group spending significantly more time in extended estrus compared to the chamber controls. Nonneoplastic lesions were observed in the nose, larynx, trachea, lung, and liver of 500 ppm males and females and in the adrenal cortex of 500 ppm females.

2-YEAR STUDY IN RATS

Groups of 50 male and 50 female rats were exposed to 1-bromopropane vapor at concentrations of 0, 125, 250, or 500 ppm, 6 hours plus T₉₀ (10 minutes) per day, 5 days per week for 105 weeks. Survival of 500 ppm males was significantly less than that of the chamber control group. Mean body weights of exposed groups were similar to those of the chamber controls.

Increased incidences of macroscopic, soft, pale-yellow to green, variably sized nodules were seen predominantly in the nose and skin of exposed rats. The number of animals with multiple masses was increased in the 500 ppm groups. In most cases, these lesions were microscopically shown to be suppurative inflammation, many with Splendore-Hoeppli material.

The incidence of adenoma of the large intestine (colon or rectum) in 500 ppm females was significantly greater than that in the chamber control group. The incidence of adenoma of the large intestine in 250 ppm males exceeded the historical control ranges for inhalation studies and all routes.

The incidences of keratoacanthoma, basal cell adenoma, basal cell carcinoma, or squamous cell carcinoma (combined) in all exposed groups of males were significantly greater than that in the chamber control group and exceeded the historical control range for inhalation studies. The incidences of keratoacanthoma and of keratoacanthoma or squamous cell carcinoma (combined) in 250 and 500 ppm males were also significantly increased and exceeded the historical control ranges for inhalation studies. In 500 ppm females, the incidence of squamous cell papilloma, keratoacanthoma, basal cell adenoma, or basal cell carcinoma (combined) exceeded the historical control range for inhalation studies.

The incidence of malignant mesothelioma in 500 ppm males was significantly greater than that in the chamber control group.

The incidences of pancreatic islet adenoma in all exposed groups of males and of pancreatic islet adenoma or carcinoma (combined) in 125 and 250 ppm males were significantly increased.

Treatment-related nonneoplastic lesions were observed in the respiratory system of exposed male and female rats. In the nose, the incidences of suppurative chronic inflammation, chronic active inflammation, glandular hyperplasia, respiratory epithelial hyperplasia (females), and respiratory metaplasia of the olfactory epithelium (females) were increased in all exposed groups. In the larynx, the incidences of chronic active inflammation and squamous metaplasia (except 125 ppm females) were increased in all exposed groups, and the incidences of suppurative chronic inflammation were increased in the 500 ppm groups. In the trachea, there were increased incidences of chronic active inflammation in all exposed groups of females and 500 ppm males, and the incidence of epithelial hyperplasia was increased in 500 ppm females.

2-YEAR STUDY IN MICE

Groups of 50 male and 50 female mice were exposed to 1-bromopropane vapor at concentrations of 0, 62.5, 125, or 250 ppm, 6 hours plus T₉₀ (10 minutes) per day, 5 days per week for 105 weeks. Survival of exposed groups was similar to that of the chamber controls. Mean body weights of all exposed groups were similar to those of the chamber controls throughout the study.

In the females, there were increased incidences of alveolar/bronchiolar adenoma, alveolar/bronchiolar carcinoma, and alveolar/bronchiolar adenoma or carcinoma (combined); the incidences of alveolar/bronchiolar adenoma or carcinoma (combined) were significantly increased in all exposed groups of females. There were significantly increased incidences of cytoplasmic vacuolization of the bronchiolar epithelium in all exposed male groups and regeneration of the bronchiolar epithelium in all exposed groups of males and females.

In the nose, there were significantly increased incidences of cytoplasmic vacuolization of the respiratory epithelium in all exposed groups of males and in 125 and 250 ppm females. There were significantly increased incidences of respiratory epithelial hyperplasia in all exposed female groups and in 62.5 and 250 ppm males. There were significantly increased incidences of respiratory metaplasia of olfactory epithelium in 62.5 and 125 ppm males and 125 and 250 ppm females.

There were significantly increased incidences of cytoplasmic vacuolization of respiratory epithelium in the larynx and trachea of all exposed male groups and in the trachea of 62.5 and 125 ppm females.

GENETIC TOXICOLOGY

1-Bromopropane was not mutagenic in either of two independent bacterial mutagenicity assays, each conducted with and without induced rat liver activation enzymes. Bacterial strains tested included *S. typhimurium* strains TA97, TA98, TA100, and TA1535, as well as *E. coli* strain WP2 *uvrA*/pKM101. In addition, no increases in the frequencies of micronucleated normochromatic erythrocytes were seen in male or female B6C3F1 mice exposed for 3 months to 62.5 to 500 ppm 1-bromopropane via inhalation.

CONCLUSIONS

Under the conditions of these 2-year inhalation studies, there was *some evidence of carcinogenic activity** of 1-bromopropane in male F344/N rats based on the occurrence of rare adenomas of the large intestine and increased incidences of neoplasms of the skin. Increased incidences of malignant mesothelioma and pancreatic islet adenoma may also have been related to 1-bromopropane exposure. There was *clear evidence of carcinogenic activity* of 1-bromopropane in female F344/N rats based on increased incidences of adenoma of the large intestine. Increased incidences of neoplasms of the skin may also have been related to 1-bromopropane exposure. There was *no evidence of carcinogenic activity* of 1-bromopropane in male B6C3F1 mice exposed to concentrations of 62.5, 125, or 250 ppm 1-bromopropane. There was *clear evidence of carcinogenic activity* of 1-bromopropane in female B6C3F1 mice based on increased incidences of alveolar/bronchiolar neoplasms.

Exposure to 1-bromopropane resulted in increased incidences of nonneoplastic lesions in the nose of rats and mice, the larynx of rats and male mice, the trachea of female rats and male and female mice, and the lung of mice. Suppurative inflammatory lesions with Splendore-Hoeppli material were present primarily in the nose and skin of male and female rats exposed to 1-bromopropane.

* Explanation of Levels of Evidence of Carcinogenic Activity is on page 14.

Summary of the 2-Year Carcinogenesis and Genetic Toxicology Studies of 1-Bromopropane

	Male F344/N Rats	Female F344/N Rats	Male B6C3F1 Mice	Female B6C3F1 Mice
Concentrations in air	0, 125, 250, or 500 ppm	0, 125, 250, or 500 ppm	0, 62.5, 125, or 250 ppm	0, 62.5, 125, or 250 ppm
Body weights	Exposed groups similar to the chamber control group	Exposed groups similar to the chamber control group	Exposed groups similar to the chamber control group	Exposed groups similar to the chamber control group
Survival rates	23/50, 26/50, 18/50, 13/50	34/50, 33/50, 30/50, 24/50	37/50, 33/50, 32/50, 36/50	36/50, 40/50, 37/50, 42/50
Nonneoplastic effects	<p><u>Nose</u>: inflammation, suppurative, chronic (0/50, 1/48, 2/48, 7/50); inflammation, chronic active (29/50, 33/48, 34/48, 35/50); glands, hyperplasia (5/50, 14/48, 14/48, 15/50)</p> <p><u>Larynx</u>: inflammation, chronic active (21/50, 28/50, 31/50, 26/50)</p>	<p><u>Nose</u>: inflammation, suppurative, chronic (0/50, 1/50, 3/49, 7/50); inflammation, chronic active (24/50, 37/50, 37/49, 36/50); glands, hyperplasia (6/50, 23/50, 28/49, 30/50); respiratory epithelium, hyperplasia (5/50, 13/50, 9/49, 18/50); olfactory epithelium, metaplasia, respiratory (3/50, 4/50, 6/49, 9/50)</p> <p><u>Larynx</u>: inflammation, chronic active (18/50, 25/50, 30/50, 32/50); metaplasia, squamous (3/50, 2/50, 6/50, 21/50)</p> <p><u>Trachea</u>: inflammation, chronic active (0/50, 4/50, 1/50, 6/50); epithelium, hyperplasia (0/50, 0/50, 0/50, 4/50)</p>	<p><u>Lung</u>: bronchiole, vacuolization cytoplasmic (0/50, 18/50, 19/49, 17/49); bronchiole, regeneration (1/50, 44/50, 38/49, 47/49)</p> <p><u>Nose</u>: respiratory epithelium, vacuolization cytoplasmic (0/50, 12/50, 19/50, 20/50); respiratory epithelium, hyperplasia (16/50, 29/50, 23/50, 26/50); olfactory epithelium, metaplasia, respiratory (0/50, 7/50, 6/50, 3/50)</p> <p><u>Larynx</u>: vacuolization cytoplasmic (0/48, 5/50, 10/48, 11/50)</p> <p><u>Trachea</u>: vacuolization cytoplasmic (0/49, 15/50, 24/47, 24/50)</p>	<p><u>Lung</u>: bronchiole, regeneration (0/50, 45/50, 43/50, 49/50)</p> <p><u>Nose</u>: respiratory epithelium, vacuolization cytoplasmic (0/50, 3/50, 5/50, 8/50); respiratory epithelium, hyperplasia (11/50, 25/50, 28/50, 27/50); olfactory epithelium, metaplasia, respiratory (0/50, 4/50, 5/50, 14/50)</p> <p><u>Trachea</u>: vacuolization cytoplasmic (0/50, 8/49, 7/50, 4/50)</p>
Neoplastic effects	<p><u>Large intestine</u>: adenoma (0/50, 0/50, 2/50, 1/50)</p> <p><u>Skin</u>: keratoacanthoma (0/50, 3/50, 6/50, 6/50); keratoacanthoma or squamous cell carcinoma (1/50, 4/50, 6/50, 8/50); keratoacanthoma, basal cell adenoma, basal cell carcinoma, or squamous cell carcinoma (1/50, 7/50, 9/50, 10/50)</p>	<p><u>Large intestine</u>: adenoma (0/50, 1/50, 2/50, 5/50)</p>	None	<p><u>Lung</u>: alveolar/bronchiolar adenoma (1/50, 6/50, 4/50, 10/50); alveolar/bronchiolar carcinoma (0/50, 7/50, 5/50, 4/50); alveolar/bronchiolar adenoma or carcinoma (1/50, 9/50, 8/50, 14/50)</p>
Equivocal findings	<p><u>Malignant mesothelioma</u>: (0/50, 2/50, 2/50, 4/50)</p> <p><u>Pancreatic islets</u>: adenoma (0/50, 5/50, 4/50, 5/50)</p>	<p><u>Skin</u>: squamous cell papilloma, keratoacanthoma, basal cell adenoma, or basal cell carcinoma (1/50, 1/50, 1/50, 4/50)</p>	None	None
Level of evidence of carcinogenic activity	Some evidence	Clear evidence	No evidence	Clear evidence
Genetic toxicology				
Bacterial gene mutations:		Negative in <i>Salmonella typhimurium</i> strains TA97, TA98, TA100, and TA1535 with and without S9; and in <i>Escherichia coli</i> WP2 <i>uvrA</i> /pKM101 with and without S9		
Micronucleated erythrocytes				
Mouse peripheral blood <i>in vivo</i> :		Negative		

EXPLANATION OF LEVELS OF EVIDENCE OF CARCINOGENIC ACTIVITY

The National Toxicology Program describes the results of individual experiments on a chemical agent and notes the strength of the evidence for conclusions regarding each study. Negative results, in which the study animals do not have a greater incidence of neoplasia than control animals, do not necessarily mean that a chemical is not a carcinogen, inasmuch as the experiments are conducted under a limited set of conditions. Positive results demonstrate that a chemical is carcinogenic for laboratory animals under the conditions of the study and indicate that exposure to the chemical has the potential for hazard to humans. Other organizations, such as the International Agency for Research on Cancer, assign a strength of evidence for conclusions based on an examination of all available evidence, including animal studies such as those conducted by the NTP, epidemiologic studies, and estimates of exposure. Thus, the actual determination of risk to humans from chemicals found to be carcinogenic in laboratory animals requires a wider analysis that extends beyond the purview of these studies.

Five categories of evidence of carcinogenic activity are used in the Technical Report series to summarize the strength of evidence observed in each experiment: two categories for positive results (**clear evidence and some evidence**); one category for uncertain findings (**equivocal evidence**); one category for no observable effects (**no evidence**); and one category for experiments that cannot be evaluated because of major flaws (**inadequate study**). These categories of interpretative conclusions were first adopted in June 1983 and then revised on March 1986 for use in the Technical Report series to incorporate more specifically the concept of actual weight of evidence of carcinogenic activity. For each separate experiment (male rats, female rats, male mice, female mice), one of the following five categories is selected to describe the findings. These categories refer to the strength of the experimental evidence and not to potency or mechanism.

- **Clear evidence** of carcinogenic activity is demonstrated by studies that are interpreted as showing a dose-related (i) increase of malignant neoplasms, (ii) increase of a combination of malignant and benign neoplasms, or (iii) marked increase of benign neoplasms if there is an indication from this or other studies of the ability of such tumors to progress to malignancy.
- **Some evidence** of carcinogenic activity is demonstrated by studies that are interpreted as showing a chemical-related increased incidence of neoplasms (malignant, benign, or combined) in which the strength of the response is less than that required for clear evidence.
- **Equivocal evidence** of carcinogenic activity is demonstrated by studies that are interpreted as showing a marginal increase of neoplasms that may be chemical related.
- **No evidence** of carcinogenic activity is demonstrated by studies that are interpreted as showing no chemical-related increases in malignant or benign neoplasms
- **Inadequate study** of carcinogenic activity is demonstrated by studies that, because of major qualitative or quantitative limitations, cannot be interpreted as valid for showing either the presence or absence of carcinogenic activity.

For studies showing multiple chemical-related neoplastic effects that if considered individually would be assigned to different levels of evidence categories, the following convention has been adopted to convey completely the study results. In a study with clear evidence of carcinogenic activity at some tissue sites, other responses that alone might be deemed some evidence are indicated as "were also related" to chemical exposure. In studies with clear or some evidence of carcinogenic activity, other responses that alone might be termed equivocal evidence are indicated as "may have been" related to chemical exposure.

When a conclusion statement for a particular experiment is selected, consideration must be given to key factors that would extend the actual boundary of an individual category of evidence. Such consideration should allow for incorporation of scientific experience and current understanding of long-term carcinogenesis studies in laboratory animals, especially for those evaluations that may be on the borderline between two adjacent levels. These considerations should include:

- adequacy of the experimental design and conduct;
- occurrence of common versus uncommon neoplasia;
- progression (or lack thereof) from benign to malignant neoplasia as well as from preneoplastic to neoplastic lesions;
- some benign neoplasms have the capacity to regress but others (of the same morphologic type) progress. At present, it is impossible to identify the difference. Therefore, where progression is known to be a possibility, the most prudent course is to assume that benign neoplasms of those types have the potential to become malignant;
- combining benign and malignant tumor incidence known or thought to represent stages of progression in the same organ or tissue;
- latency in tumor induction;
- multiplicity in site-specific neoplasia;
- metastases;
- supporting information from proliferative lesions (hyperplasia) in the same site of neoplasia or other experiments (same lesion in another sex or species);
- presence or absence of dose relationships;
- statistical significance of the observed tumor increase;
- concurrent control tumor incidence as well as the historical control rate and variability for a specific neoplasm;
- survival-adjusted analyses and false positive or false negative concerns;
- structure-activity correlations; and
- in some cases, genetic toxicology.

Summary of Data on Workplace Exposure to n-Propyl Bromide

The U.S. Environmental Protection Agency's (EPA) Significant New Alternatives Policy (SNAP) Program has received workplace exposure data for n-propyl bromide (nPB) from several different sources. The sources include:

- Data from organic vapor monitoring badges worn by workers using vapor degreasing equipment
- Data from charcoal tube samplers worn by workers using vapor degreasing equipment, cold batch cleaning equipment, aerosols, spray adhesives in foam fabrication, adhesives used for flooring, and manual cleaning
- Area samples taken using both organic vapor monitoring badges and charcoal tube samplers
- Data from three manufacturers or suppliers of products containing nPB
- Data from the National Institute for Occupational Safety and Health from four different facilities
- Data from spray adhesives, a highly emissive use, both before and after improvements to ventilation

The Agency considered these data in determining whether it is feasible to meet the recommended workplace exposure limit for nPB using available, affordable control or ventilation equipment.

Description of exposure data by sector

Non-aerosol solvent cleaning

Almost all samples for solvent cleaning were taken for vapor degreasing.

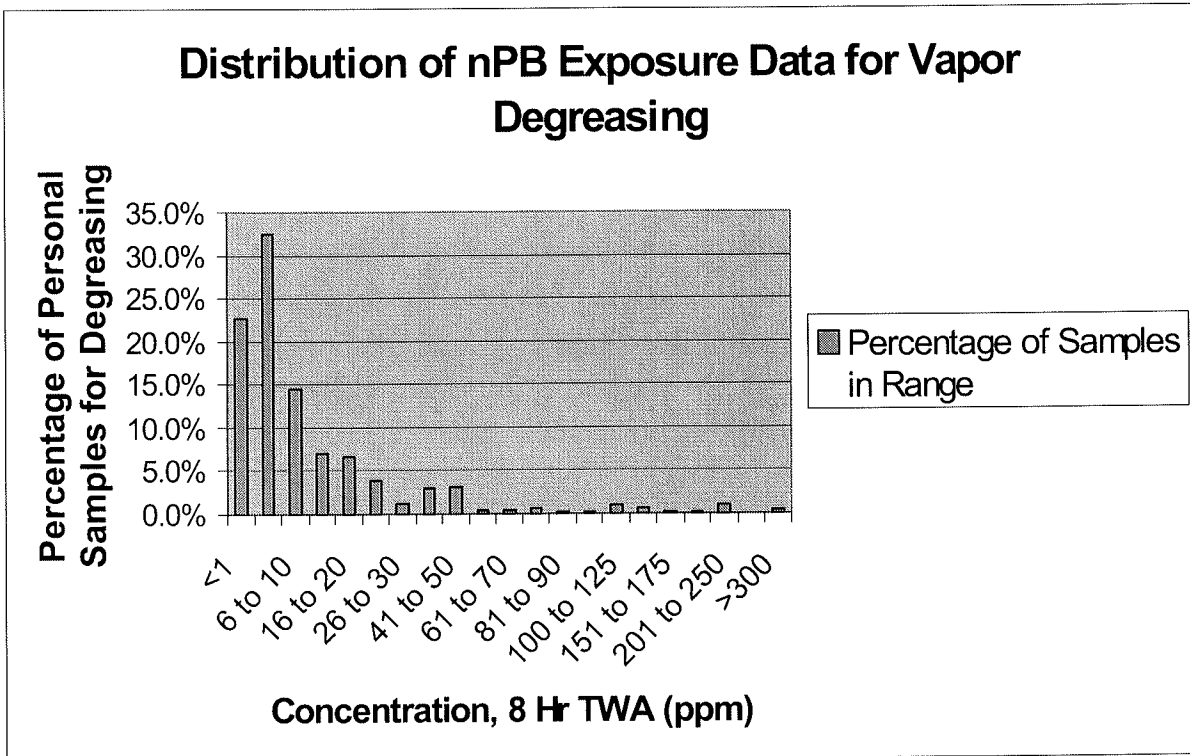
- There are approximately 500 personal samples in all, roughly 90% taken using organic badge monitors (3M or SKC).
- There are approximately 40 area samples, most taken with charcoal tube.
- Data sources for personal samples include exposure data from customers of two suppliers of nPB-based solvents for vapor degreasing, 2003.
- For area samples, EPA also considered data from R.L. Smith, 1998a and 1998b.
- Approximately 75% of the personal samples were below 10 ppm on an 8 hour time-weighted average (TWA)
- >87% of personal samples were below 25 ppm (8 hr TWA)
- In several situations where samples indicated workplace exposure greater than 25 ppm, the supplier and user worked to reduce emissions, and brought them below 25 ppm

Earlier samples taken in 1997 had higher average concentrations than later samples. This may reflect that some manufacturers reduced their recommended workplace exposure limit in 2000, and therefore, users made greater efforts to attain lower emissions.

There also was a Health Hazard Evaluation (HHE) by the National Institute for Occupational Safety and Health (NIOSH) for an air-tight "cold" batch cleaner with ventilation.

Before use of the air-tight cleaning equipment, workers complained of adverse health effects such as headaches, nausea, vomiting, feeling faint, and irritation of the mucous membranes (NIOSH, 2000c). After installing the new equipment, NIOSH measured workplace exposure. NIOSH found that samples taken on 20 employees all read concentrations less than 1ppm of nPB. These data from NIOSH are not included in the more than 500 personal samples mentioned above, which are all for vapor degreasing.

Figure 1. Distribution of Exposure Data for Vapor Degreasing



EPA also examined data for two other “cold cleaning” applications that are not currently regulated under SNAP: flushing and hand wiping. At one site, personal exposure levels for manual flushing of coils ranged from 5 to 42 ppm, with three samples taken with charcoal tubes. EPA has data for three different sites for hand wiping. The personal samples were all greater than 25 ppm.

Table 1. Exposure Data from Hand Wiping Application

Site	# of Samples	Avg Conc. (ppm)	Conc. Range (ppm)
Hand Wipe Site #1	5	89.3	51.1 to 107.9
Hand Wipe Site #2	2	79.7	67.1 to 92.2
Hand Wipe Site #3	4	Not available	28 to 47 (6 to 10 min. exposure)

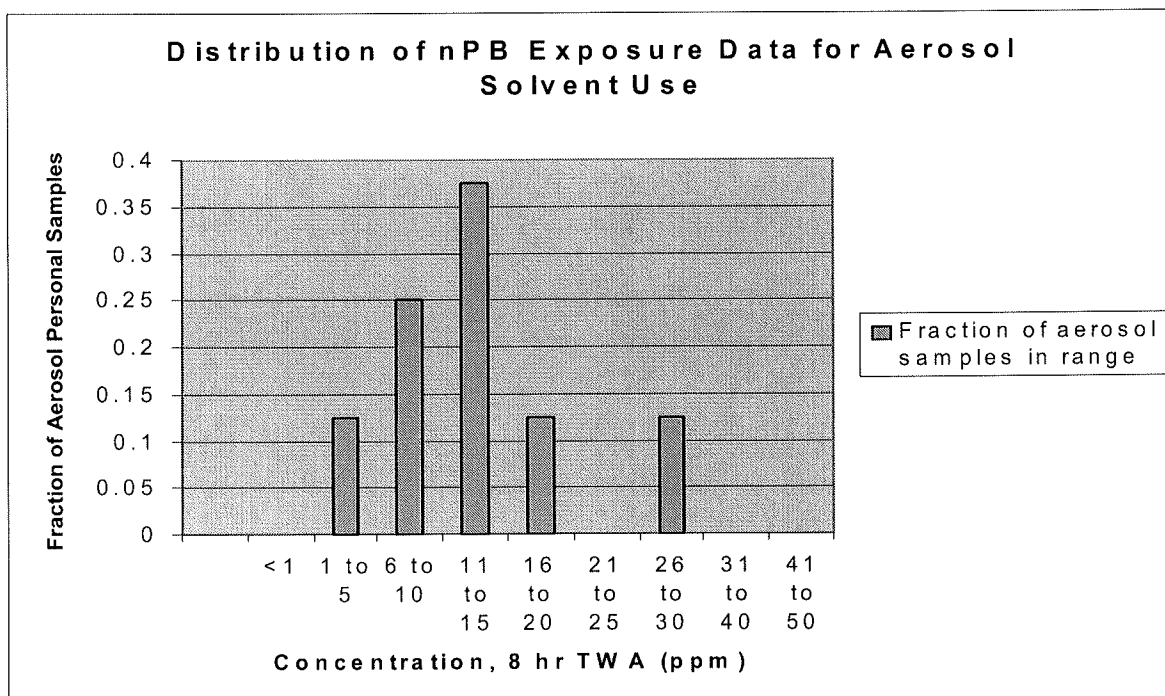
Sources: Smith, R.L. 1998; November 16, 2001 Email from Mick Kassem, Albemarle Corporation.

Aerosol Solvents

Of the three major applications that EPA evaluated, there was the least amount of exposure data for aerosol solvents. There are:

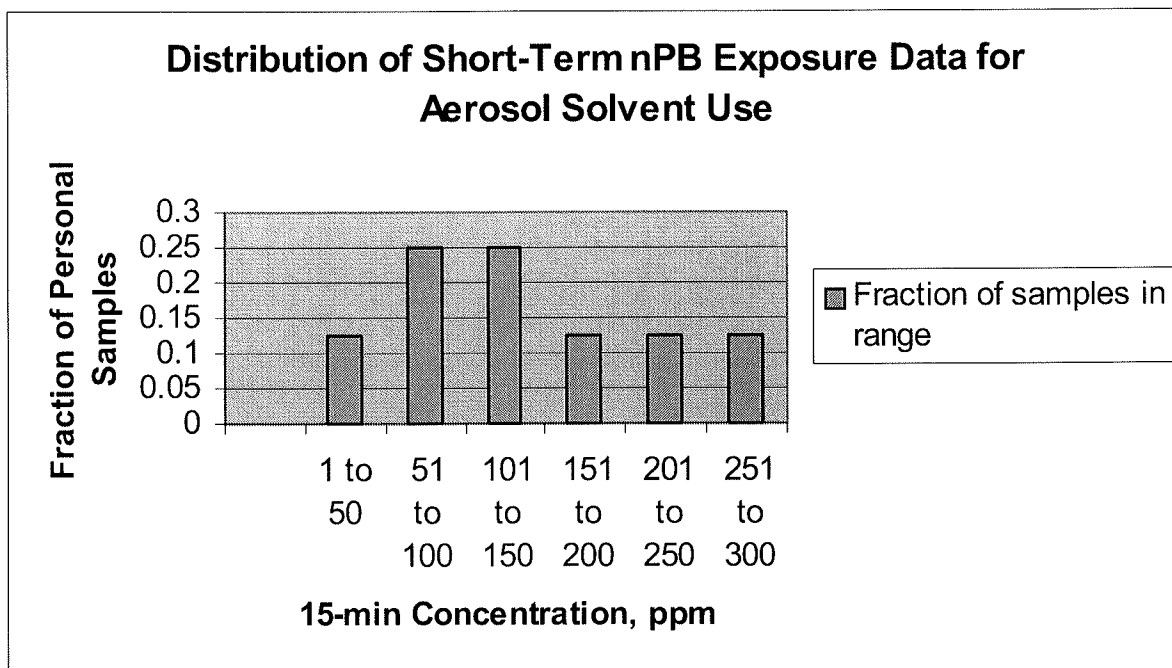
- Eight personal samples on an eight-hour TWA basis, most taken using organic badge monitors
- Four area samples, all taken with charcoal tube samplers
- Eight 15-minute samples, suitable for comparison with a short-term exposure limit (STEL)
- Sources of data include Kassem, 2001 and Aerosol data, 1998.
- The eight personal samples on an eight-hour TWA basis ranged from 5 ppm to 30.2 ppm.
- Seven of the eight personal samples on an eight-hour TWA basis were below 25 ppm.
- The eight 15-minute samples ranged from 45.1 ppm to 254 ppm.

Figure 2. Distribution of nPB Exposure Data for Aerosol Solvent Use (8 hr TWA)



The 15 minute samples are useful for a comparison with a short-term exposure limit (STEL). The samples taken on a eight-hour time-weighted average are useful for comparison with a workplace exposure limit.

Figure 3. Distribution of Short-Term nPB Exposure Data for Aerosol Solvent Use (15 Minute Exposures)



Adhesives

Almost all of the exposure data for adhesives were for spray adhesives using nPB as a carrier solvent in foam fabrication and construction of seat cushions. In addition, there were a few sites where nPB-based adhesives were applied for flooring. There are:

- Approximately 90 personal samples, all taken with charcoal tube samplers
- Five area samples, all taken with charcoal tube samplers
- Data sources include Smith, R.L. 1998, Kassem 2001, NIOSH 2000a, NIOSH 2000b, NIOSH 2001, NIOSH 2002a, and NIOSH 2002b.

There is a marked difference in the exposure data at two sites where NIOSH worked together with the facilities to improve their ventilation.

- Less than half of the personal samples were below 50 ppm on an 8 hour TWA before improving ventilation; 97% of the personal samples were below 50 ppm after improving ventilation.
- The mean concentration before improving ventilation was 141.7 ppm; the mean concentration after improving ventilation was 18.3 ppm.
- After improving ventilation, 78% percent of the samples were at 25 ppm or below.

Figure 4. Distribution of nPB Exposure Data for Adhesives

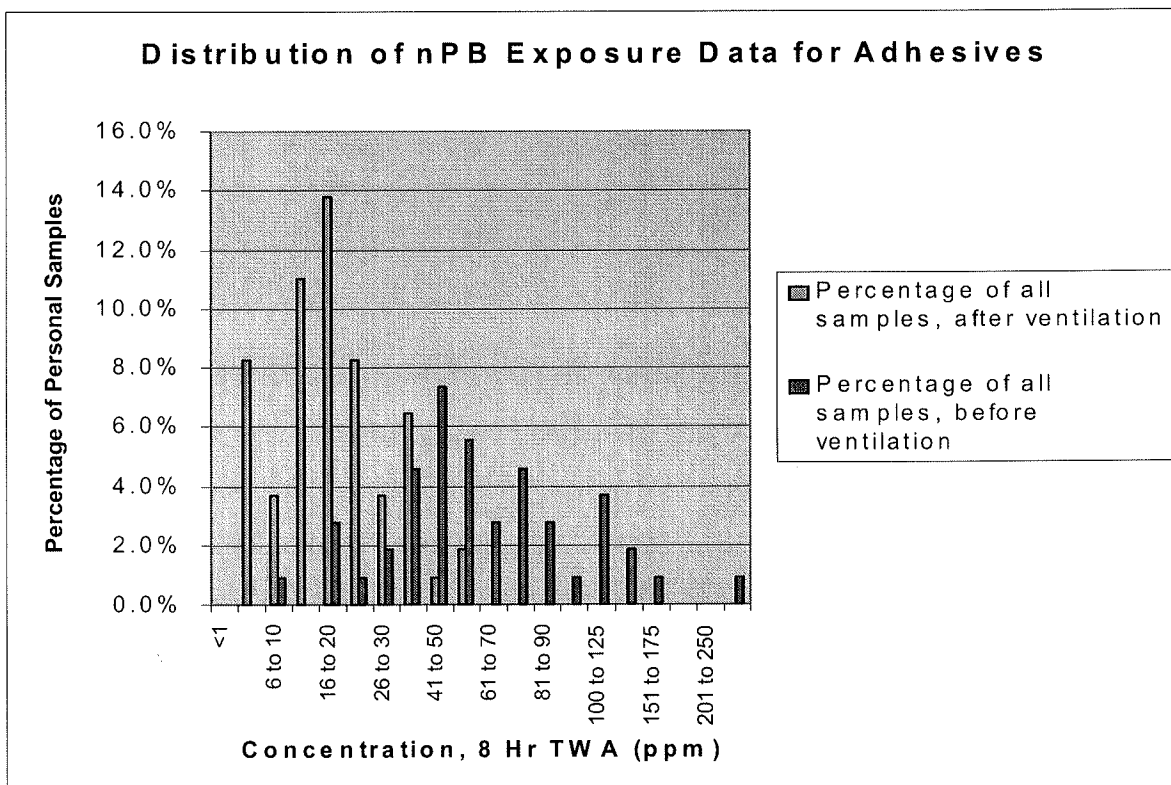


Figure 4 shows the distribution of personal samples taken in adhesives application both before and after making improvements to ventilation. Percentages are a percentage of all personal samples for adhesives for which we have data. Approximately 58% of the person samples are from after ventilation was improved and 42% are from before ventilation was improved. To see percentage of samples within each category, see Figure 5 below.

Figure 4 does not include data from the Letter from Dept. of Health and Human Services (HHS) to Custom Products, Inc., December 1, 1999. The nPB exposure data at this plant were relatively high before NIOSH assisted Custom Products in improving ventilation. However, the individual measurements are not available, and thus they are not included in the distribution above. Table 2 summarizes the data from NIOSH's 1999 letter to Custom Products.

Workplace exposure levels at Custom Products ranged from 60 ppm to 381.2 ppm during NIOSH's initial evaluation. The mean exposure level for the plant was 168.9 ppm. By comparison, after improving ventilation, the mean exposure level dropped almost tenfold to 18.9 ppm, with a range from 1.2 to 58 ppm.

Table 2. Summary of nPB Exposure Data from Adhesives at Custom Products, Prior to Ventilation Improvements

Job Title	Department	# of Samples	Exposure, ppm of nPB (8 Hour TWA)		
			Mean	Minimum	Maximum
	Assembly	36	169.8	60.0	250.7
Sprayers	Assembly	15	193.0	115.3	250.7
Assemblers	Assembly	20	154.7	60.0	234.9
Sprayers	Covers	21	197.0	117.3	381.2
Saw Operator	Saw	12	117.1	85.1	159.2
All Exposure Data		69	168.9	60.0	381.2

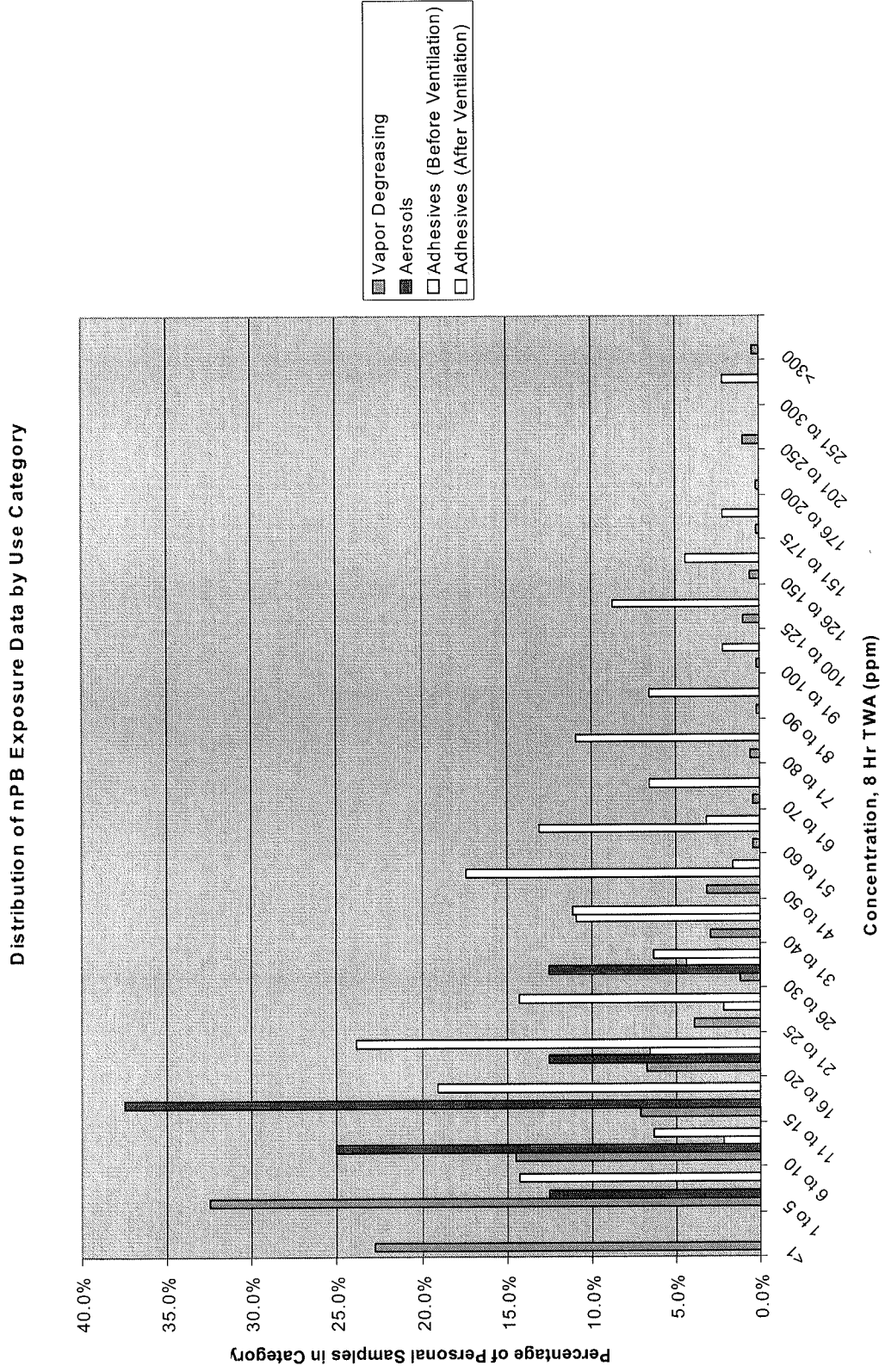
Conclusions

Exposure levels to nPB in the workplace are much lower for vapor degreasing and for air-tight cleaning equipment than for other applications. Most vapor degreasers are capable of containing nPB exposure levels to less than 10 ppm.

There are limited nPB exposure data available for aerosol and cold cleaning applications. These data indicate that aerosol users are likely to attain workplace exposure levels below 20 ppm. On the other hand, hand wiping appears to be a highly emissive application with exposure levels consistently above 25 ppm.

Exposure levels are the highest for adhesives. It is possible to make improvements to ventilation when using adhesives that can reduce exposure levels below 20 ppm on average. Without proper ventilation, nPB exposure from spray adhesives can be extremely high, into the hundreds of parts per million.

Figure 5. Distribution of nPB Exposure Data by Use Category



References

- Aerosol data, 1998. Airborne Exposure Assessment of 1-Bromopropane, 1998 (Docket A-2001-07, item II-D-89)
- Kassem, 2001. November 16, 2001 Email from Mick Kassem, Albemarle Corporation, concerning exposure levels using nPB in various applications. (Docket A-2001-07, item II-A-19)
- NIOSH, 1999. Letter from Dept. of Health and Human Services to Custom Products, Inc., December 1, 1999. Re: results of Dec. 1998 survey of workplace exposure to nPB at Custom Products. (Docket A-2001-07, item II-D-6)
- NIOSH, 2000a. Letter from Dept. of Health and Human Services to Marx Industries, Inc., February 1, 2000. Re: results of nPB exposure assessment survey conducted Nov. 16-17, 1999. (Docket A-2001-07, item II-D-7)
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- NIOSH, 2000c. NIOSH Health Hazard Evaluation Report of nPB exposure from cold vapor degreasers at Trilithic, Inc. (HHE Report 2000-0233-2845; Docket A-2001-07, item II-D-11)
- NIOSH, 2001. Letter from Dept. of Health and Human Services to STN Cushion Company, March 7, 2001. Re: Results of nPB exposure assessment survey conducted November 14, 2000. (Docket A-2001-07, item II-D-9)
- NIOSH, 2002a. NIOSH Health Hazard Evaluation Report: HETA#2000-0410-2891, STN Cushion Company, Thomasville, NC, August 2002 (Docket A-2001-07, item II-A-31)
- NIOSH, 2002b. NIOSH Health Hazard Evaluation Report: HETA #98-0153-2883 Custom Products, Inc. November 2002. (Docket A-2001-07, item II-A-49)
- Smith, R.L. 1998a. Assessments of occupational exposure to nPB in adhesive spray and metal cleaning applications. Written communications from Robert Smith, Albemarle Corporation to EPA, March 19 through June 26, 1998. (a) 3/19/98; (b) 4/21/98; (c) 4/22/98; (d) 4/23/98; (e) 4/24/98; (f) 5/1/98; (g) 5/29/98; (h) 6/3/98; (i) 6/26/98 (Docket A-91-42, item X-A-57)
- Smith, R.L. 1998b. Additional information from Robert Smith (Docket A-91-42, item VI-D-114)

Vapor degreasing data, 2003. Exposure data from customers of two suppliers of nPB-based solvents for vapor degreasing, 2003 (see attached spread sheet and emails)