CHEMICAL MANUFACTURERS ASSOCIATION

July 29, 1991

Air Docket (LE-131)
Room M1500
U.S. Environmental Protection Agency
401 M Street, SW
Washington, DC 20460

Attention: Docket No. A-90-47

Re: Comments of the Chemical Manufacturers Association Vinylidene Chloride Panel Regarding the Listing of Vinylidene Chloride as a "High Risk" Air Pollutant

Dear Sir or Madam:

The Chemical Manufacturers Association Vinylidene Chloride Panel submits the accompanying comments concerning EPA's proposal to list vinylidene chloride (VDC) as a "high risk" pollutant pursuant to Section 112(i)(5)(E) of the Clean Air Act.

The VDC Panel's comments highlight four critical issues which the Agency should consider in developing its final rule listing "high risk" pollutants:

- EPA in 1985 questioned whether VDC posed any carcinogenic risk and determined that the risk posed by VDC, if it exists at all, is insufficient to justify regulation under the Clean Air Act;
- The overwhelming weight of evidence on VDC, which EPA has addressed in several documents, indicates that it is unlikely to pose any carcinogenic hazard to humans;
- EPA's three-tier risk calculation misapplies quantitative risk assessment methodologies to Group C substances like VDC for which the weight of evidence indicates that the likelihood of a human risk is small. Adjusting the calculation to account for the lower likelihood of human risk from a Group C substance would eliminate VDC from the list of "high risk" pollutants; and

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- Based on the Agency's previous decisions not to regulate VDC under the Clean Air Act, the Panel believes that EPA should provide a justification for its dramatic change in position regarding VDC. Moreover, by ignoring significant differences among carcinogen classification groups, the three-tier process proposed by EPA would produce arbitrary results.

In addition to these specific comments concerning the Agency's proposal to list VDC as a "high risk" pollutant, the VDC Panel also endorses more general comments submitted by CMA. The general comments are entitled "Comments of the Chemical Manufacturers Association on Proposed Regulations Governing Compliance Extensions for Early Reductions of Hazardous Air Pollutants."

If you have any questions or require additional information, please contact Kathleen Roberts of my staff at 202-887-1146.

Very truly yours,

Gordon D. Strickland

Gordon D. Strickland
Vice President
Technical Services Director

Enclosures

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BEFORE THE
UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

CHEMICAL MANUFACTURERS ASSOCIATION VINYLIDENE CHLORIDE PANEL COMMENTS ON EPA PROPOSAL TO LIST 1,1-DICHLOROETHYLENE (VINYLIDENE CHLORIDE) AS A "HIGH RISK" AIR POLLUTANT 56 Fed. Reg. 27338 (June 13, 1991) EPA DOCKET NO. A-90-47

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July 29, 1991

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The Environmental Protection Agency has proposed to list vinylidene chloride (VDC) as a "high risk" air pollutant pursuant to Section 112(i)(5)(E) of the Clean Air Act (as amended), 42 U.S.C. § 7412(i)(5)(E). See National Emission Standards for Hazardous Air Pollutants for Source Categories: Proposed Regulations Governing Compliance Extensions for Early Reductions of Hazardous Air Pollutants, 56 Fed. Reg. 27338 (June 13, 1991).

The Chemical Manufacturers Association Vinylidene Chloride Panel submits these comments on the proposed rule.[1/] The VDC Panel is an industry group organized as a CHEMSTAR panel of CMA. The Panel represents all U.S. manufacturers of VDC and a substantial portion of the industry, both

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1/ The VDC Panel also supports general comments filed by CMA, entitled "Comments of the Chemical Manufacturers Association on Proposed Regulations Governing Compliance Extensions for Early Reductions of Hazardous Air Pollutants."

domestic and foreign, which converts VDC into other products.

The Act authorizes EPA to identify as "high risk" pollutants "pollutants for which high risks of adverse public health effects may be associated with exposure to small quantities" of the substance. 42 U.S.C. § 7412(i)(5)(E). Pursuant to this statutory authorization, EPA has proposed to identify VDC as a "high risk" pollutant on the basis of carcinogenic risk posed by emissions of the substance. 56 Fed. Reg. 27354.

The VDC Panel objects to the inclusion of VDC in the list of "high risk" pollutants. As these comments demonstrate, there is insufficient evidence to support a finding that VDC poses any carcinogenic hazard to humans; and both the evidence and EPA's previous rulings on VDC indicate that any risk posed by VDC is extremely low.

EXECUTIVE SUMMARY

The proposed rule ignores EPA's previous determinations of the potential risks posed by VDC. Furthermore, the proposal overlooks basic differences among substances that have been classified as known, probable and

possible carcinogens. The proposal is inconsistent with the Congressional mandate of Section 112(i)(5)(E) and would conflict with fundamental principles of administrative law.

- First, EPA in 1985 explicitly determined that the carcinogenic risk posed by VDC, if any, was insufficient to justify regulation under the Clean Air Act.

- Second, the overwhelming weight of evidence on VDC, which has not changed since EPA's 1985 decision, indicates that the substance it is unlikely to pose any carcinogenic hazard to humans.

- Third, EPA's three-tier risk calculation for VDC misapplies quantitative risk assessment methodologies to VDC (a Group C "possible carcinogen") by failing to consider inherent differences in the weight-of-evidence among different carcinogen classifications.

- Finally, EPA's proposal ignores the clear Congressional mandate of the Act and violates established precepts of administrative law. The

proposed rule lacks the evidentiary support necessary to justify the "high risk" pollutant designation, and the proposal has entirely failed to consider important differences among carcinogen classifications. Therefore, the proposed rule would be subject to challenge as arbitrary and capricious.

COMMENTS

A. EPA has Previously Determined that the Evidence Does Not Justify Regulation of VDC as a Carcinogen under the Clean Air Act.

VDC is one of several substances that had been under consideration by EPA for possible regulation as a "hazardous air pollutant" prior to 1985.[2/] Under the Clean Air Act of 1970, a "hazardous air pollutant" was defined as a substance "which in the judgment of the Administrator causes, or contributes to, air pollution which may reasonably be anticipated to result in an increase in mortality or an increase in serious irreversible, or incapacitating reversible, illness." 42 U.S.C. § 7412(a)(1)(1988). EPA initiated a regulatory assessment of VDC under

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2/ See 40 C.F.R. § 61.01 (1988) (listing "hazardous air pollutants" regulated under former Section 112 and other substances considered for listing by EPA).

the Clean Air Act "because of preliminary evidence of cancer in animals and structural similarity of VDC to vinyl chloride, a known human carcinogen."[3/]

After completing and reviewing its Health Assessment Document for Vinylidene Chloride,[4/] however, EPA ultimately declined to regulate VDC under the Clean Air Act. The Agency stated:

> "The EPA has concluded that routine emissions from VDC facilities are unlikely to result in ambient concentrations that pose a public health hazard from noncarcinogenic effects; that the available scientific evidence for the carcinogenic potential of VDC for humans is only limited; and that an analysis of the public health hazard if VDC were assumed to be carcinogenic indicates that the possible cancer risks are small. Given the health hazard conclusions, specific regulation of VDC is not warranted at this time under any section of the Clean Air Act."[5/]

The body of available scientific data on VDC's carcinogenic potential has remained substantially unchanged since EPA's determination not to regulate the chemical in

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3/ 50 Fed. Reg. 32633 (August 13, 1985).

4/ United States Environmental Protection Agency, Health Assessment Document for Vinylidene Chloride (EPA-600/8-83-031F August 1985) (hereinafter HAD).

5/ 50 Fed. Reg. 32632.

1985. EPA's Integrated Risk Information System (IRIS) database continues to report the same 18 chronic animal studies, including only one which was characterized as positive for carcinogenic effects.

B. The Available Scientific Data Fail to Support the Classification of VDC as a "High Risk" Carcinogenic Pollutant.

To list VDC as a "high risk" carcinogenic pollutant, EPA must make a substantiated finding that "high risks" of human carcinogenic effects of VDC "may be associated with exposure to small quantities" of the chemical. See 42 U.S.C. § 7412(i)(5)(E). The available scientific data do not support any such finding. First, th data do not indicate VDC is carcinogenic to humans. Second, the data do not indicate that a high risk may be associated with exposure to small quantities of VDC. As EPA stated in 1985, even if VDC were assumed to be carcinogenic, "the possible cancer risks are small." 50 Fed. Reg. 32632.

1. Animal data do not support a carcinogenicity finding for VDC.

As reflected in EPA's previous decision on the carcinogenicity of VDC, animal bioassays with VDC have been

uniformly negative, with the one exception. 50 Fed. Reg. 32633. Only in Maltoni's Swiss mouse study[6/] -- a study which has been criticized as exceeding the maximum tolerated dose (MTD) -- were tumors identified. In this study tumors were observed in male mice, but not in females. Thus, the total evidence for carcinogenicity comes from results in "only one sex [male] of one strain [Swiss] of one species [mouse]" out of a total of 18 carcinogenicity studies conducted using mice, rats and hamsters. 50 Fed. Reg. 32633. This sole evidence of carcinogenicity appeared in a study in which mice were exposed to concentrations of VDC that were notably toxic and near the acutely lethal concentration.[7/] Moreover, there is substantial evidence of the greater sensitivity of this strain of mouse to VDC. The single positive oncogenicity study appears to be related to the significant tissue injury in male mice exposed to VDC.

Attached as Exhibit A is a data review entitled "Interpretive Review of the Animal Toxicological, Pharmacokinetic/Metabolism, Biomolecular and In-Vitro

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6/ Maltoni, C., Cotti, G., Morisi, L., and Chieco, P. 1977b. Carcinogenicity Bioassays of Vinylidene Chloride. Research Plan and Early Results, La Medicinia del Lavoro 68:240-262.

7/ Maltoni, et al. (1977) reported a high degree of toxicity and mortality within one week at 200, 100 ppm and 50 ppm in the Swiss mouse. HAD at 10-91.

Mutagenicity Studies on Vinylidene Chloride and the Significance of the Findings for Man" by J.M. Norris and R.H. Reitz (Interpretive Review). The Interpretive Review contains a comprehensive overview of data relevant to an effort to extrapolate from existing animal data for human risk assessment purposes.

EPA scientists have also recognized the well-characterized mechanism of VDC toxicity. In its draft Drinking Water Criteria Document for VDC, EPA stated:

> "Very little is known about the mechanism of toxicity of cis- and trans-1,2-DCE. However, there has been considerable work directed toward defining the mechanism of toxicity of 1,1-DCE. . . . [M]ost of the acute and long-term toxic effects observed, e.g. hepatotoxicity, renal toxicity, mutagenicity and carcinogenicity, are due probably to the formation of toxic metabolites."[8/]

As the Interpretive Review explains, data clearly demonstrate that VDC is metabolized much more rapidly by

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8/ U.S. Environmental Protection Agency, Drinking Water Criteria Document for Dichloroethylenes (1,1-Dichloroethylene, cis-1,2-Dichloroethylene, and trans-1,2-Dichloroethylene) at VII-1 (Draft December 1984) (hereinafter "EPA Criteria Document").

mice than by rats.[9] It has been shown that there are much higher levels of covalently-bound VDC metabolites in both the liver and kidney of mice than in the same organs of rats.[10] The formation of substantially larger amounts of toxic metabolites in the mouse explains the greater toxicity of VDC to the mouse than to the rat.

The EPA Criteria Document noted:

> "It is generally believed that reaction of these metabolites, i.e., 1,1-dichloroethylene oxide, chloroacetyl chloride and chloroacetic acid, with macromolecules leads to the observed toxic effects; hepatotoxicity, renal toxicity, mutagenicity, and carcinogenicity."

Id. at VII-3 (emphasis added). It also noted the many studies that have shown that mice are far more susceptible to both kidney and liver damage from VDC than are rats. Id. at VII-5.

The extensive bioassay data confirm the Agency's observation, quoted above, that the reaction of VDC metabolites is generally believed to be the cause of the

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9/ Jones, B.K, and Hathway, D.E. 1978. Differences in metabolism of vinylidene chloride between mice and rats, Br. J. Cancer 37: 411-417.

10/ EPA Criteria Document at VII-4.

observed toxic effects. Thus, no significant increase in tumors was shown in Maltoni's inhalation study with Sprague-Dawley rats, in his inhalation study with Chinese hamsters or in his gavage study with Sprague-Dawley rats. It was only in his inhalation study with Swiss mice exposed to the nearly lethal dose of 25 ppm that an increase in kidney adenocarcinomas was observed. No other studies were positive, including:

- an inhalation study with Wistar rats,
- an inhalation study with Sprague-Dawley rats,
- an inhalation study with CD-1 mice,
- an inhalation study with CD rats,
- another inhalation study with Sprague-Dawley rats,
- an ingestion study with Sprague-Dawley rats,
- another inhalation study with CD mice,
- another inhalation study with CD rats,
- a skin application study with Swiss mice,
- a subcutaneous injection study with Swiss mice,
- a third inhalation study with Sprague-Dawley rats,
- a gavage study with Sprague-Dawley rats,
- a gavage study with Fischer 344 rats, and
- a gavage study with B6C3F1 mice.

In view of the disparity between the mouse kidney adenocarcinomas and the results in seventeen other long-term studies, there is now a widely held view in the scientific community regarding the mechanism of tumor formation in the one positive mouse study. In addition to the references summarized in the Interpretive Review, EPA has summarized that view as follows:

> "It has been suggested that development of kidney adenocarcinomas in mice is due to the increased rate of biotransformation of 1,1-DCE in mice, which produces a higher level of reactive intermediates that can subsequently react with macromolecules, e.g., DNA. A study has been made of the potential of 1,1-DCE to cause DNA alkylation, DNA repair and DNA replication in the liver and kidneys of mice and rats, and these results were compared with results obtained using the potent carcinogen dimethylnitrosamine. Tumorigenic doses of dimethylnitrosamine produced relatively little tissue damage but caused a high degree of DNA alkylation and DNA repair synthesis. In contrast, tumorigenic doses of 1,1-DCE resulted in massive tissue damage but induced minimal DNA alkylation or DNA repair synthesis. These data were interpreted as suggesting that tumors observed in mice exposed to 1,1-DCE arise primarily through effects of the chemical on nongenetic components of cells."

EPA Criteria Document at VII-8.[11/]

When this extensive data base is used to estimate potential risks to man, it can be seen that the oncogenicity data have questionable applicability to man. The rate of oxidative metabolism for halogenated hydrocarbons such as VDC appears to be related to body surface area, rather than body mass. Thus metabolic activation would occur more slowly in man than in small laboratory animals. This observation is consistent with findings of Reitz, et al. (1980) (supra, note 13) and Jones and Hathway (1978) (supra, note 11), who demonstrated that the rat metabolizes less VDC

11/ EPA cited the following:

Maltoni, C. 1977. Recent findings on the carcinogenicity of chlorinated olefins. Environ. Health Perspect. 21:1-5.

Hathaway, D.E. 1977. Comparative mamalian metabolism of vinyl chloride and vinylidene chloride in relation to oncogenic potential. Env. Health Perspect. 21:55-59.

Henschler, D., and Bonse, G. 1977. Metabolic activation of chlorinated ethylenes; Dependence of mutagenic effect on electrophilic reactivity of the metabolically formed epoxides. Arch. Toxicol. 39:8-12.

Reitz, R.H., Watanabe, P.G., McKenna, M.J., Quast, J.F., and Gehring, P.J. 1980. Effects of vinylidene chloride on DNA synthesis and DNA repair in the rat and mouse: A comparative study with dimethylnitrosamine. Toxicol. Appl. Pharmacol. 52:357-370.

than the mouse; Andersen, et al. (1980),[12] who related the slower rate of metabolism of VDC in man versus the rat to the rate of pulmonary uptake; and Walker (1978),[13] who reported that significant metabolic dissimilarities exist between man and the mouse relative to the monooxygenases which catalyze the metabolism of VDC to the reactive metabolites.

After summarizing the data showing interspecies differences in metabolic effects, the Agency noted in its Criteria Document:

> "Since the rate at which an inhaled chemical is presented to the liver is related to pulmonary uptake, it would be expected that the smaller breathing volume (liters/kg/hr) in man relative to the rat would produce a corresponding slower rate of metabolism of 1,1-DCE in man . . . and the formation of smaller amounts of toxic metabolite(s)."

Criteria Document at VII-2-VII-3.[14]

12/ Andersen, M.E., Gargas, M.L., Jones, R.A., and Jenkins, L.J. 1980. Determination of the kinetic constants for metabolism of inhaled toxicants in vivo using gas uptake measurements. Toxicol. Appl. Pharmacol. 54:100-116.

13/ Walker, C.H. 1978. Species differences in microsomal monooxygenase activity and their relationship to biological half-lives, Drug Metab. Rev. 7(2):295-323.

14/ EPA cited Anderson, et al., (1980) (supra, note 14).

Thus, EPA's scientists have noted that it is generally believed that the metabolic dissimilarities in the mouse in comparison with other species lead to the observed toxic effects, including carcinogenicity. In view of the existing data, VDC toxicity and the potential risk to man are well-characterized. The decision reached by the Agency in declining to regulate VDC under the Act was clearly supported by the evidence. The body of data, taken as whole, does not support an inference that VDC is carcinogenic to humans, with "exposure to small quantities" or otherwise.

2. Available data indicate that the toxic effects of VDC bear no similarity to the effects of vinyl chloride.

In its 1985 evaluation of possible Clean Air Act regulation of VDC, EPA suggested that the "structural relationship" between VDC and vinyl chloride was originally thought to be cause for concern. While it may be appropriate in some circumstances to consider structural relationships in the absence of toxicity data for one of th "related" substances, it is entirely inappropriate to give any weight to such relationships when actual test data demonstrate toxicological dissimilarities between the substances. Any significance placed on structural

similarity between VDC and vinyl chloride is negated when the toxicity data for the two substances are considered.

As EPA has observed, exposure to most other chlorinated hydrocarbons typically causes injury to the endoplasmic reticulum in the liver. VDC, on the other hand, causes nuclear changes, mitochondrial swelling and the rupture of outer mitochondrial membranes. EPA has said: "This indicates a basic difference in the mechanism of toxicity of 1,1-DCE." EPA Criteria Document at VII-4. After reviewing the oncogenicity data, EPA stated further: "In summary, the carcinogenic potential of 1,1-DCE has been shown to be quite different from that of vinyl chloride." Id. at VII-7 (emphasis added). In view of the extensive toxicity data, evidence of structural similarity to vinyl chloride has no bearing on VDC's potential risk.

3. Epidemiology data also do not support the proposed regulation of VDC as a carcinogen.

An epidemiology study of employees working in a VDC facility has documented no adverse health effects or carcinogenic effects associated with VDC exposure.[15/] The

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15/ Ott, M.G., Fishbeck, W.A., Townsend, J.C., and Schneider, E.J., 1976. A health study of employees exposed to vinylidene chloride. J. Occup. Med. 18:735-738.

VDC Panel agrees with EPA's prior assessment that the study, while well conducted, may lack adequate statistical power to detect excesses of rare carcinomas. See 51 Fed. Reg. 28842. It should be noted, however, that the potential exposures observed in that occupational population (ranging up to 70 ppm) far exceeded exposures that would be present in the ambient environment. Therefore, the available epidemiological evidence does not indicate that VDC is a "high risk" pollutant which poses cancer risks "with exposure to small quantities."

4. The scientific evidence, taken as a whole, does not support listing VDC as a "high risk" carcinogen.

There is extremely limited evidence of VDC's carcinogenicity, and little basis for concluding the chemical is carcinogenic to humans. The Agency has acknowledged these facts. EPA's IRIS database lists VDC as a Group C "possible carcinogen" -- a substance for which there is "limited evidence" of carcinogenicity in animals and "inadequate data" or "no data" indicating effects in humans.[16/] EPA should not list VDC as a "high risk" pollutant without reliable evidence that the substance is

16/ See Guidelines for Carcinogen Risk Assessment, 5 Fed. Reg. 33992, 34000 (September 24, 1986).

carcinogenic to humans or otherwise poses "high risks of adverse public health effects." In the absence of new evidence, EPA should refrain from changing its conclusion that the evidence of carcinogenicity is limited and that, even if carcinogenic, VDC poses a small risk.

C. EPA's "High Risk" Calculation for VDC Involves a Fundamental Misapplication of Risk Assessment Methodology.

In view of the uncertainty over whether VDC poses any carcinogenic risk to humans, it is improper to extrapolate from the one positive study using traditional quantitative risk assessment principles to conclude that VDC poses a "high risk." EPA's use of identical methods to extrapolate human risk estimates for substances classified as known (Group A), probable (Group B) and possible (Group C) carcinogens ignores fundamental differences in the quality of the carcinogenicity data for these substances. If VDC is not a human carcinogen, it poses no risk of cancer and it should not be listed as a "high risk" carcinogen. By ignoring the important differences among carcinogenicity groups, EPA's listing of VDC as a "high risk" pollutant would be arbitrary and capricious.

When EPA first reported an inhalation unit risk estimate for VDC (i.e., 5×10^{-5} $(ug/m^3)^{-1}$), the Agency was careful to emphasize the limitations inherent in the resultant numerical estimate:

> "It is important to note that the calculations in [the HAD] for estimating in quantitative terms the impact of vinylidene chloride as a carcinogen are made independently of the overall weight of the evidence for vinylidene chloride's carcinogenic potential. This is done to answer the question of how large an incremental cancer risk would be if vinylidene chloride were a human carcinogen. However, there is only limited animal evidence for vinylidene chloride carcinogenicity, and the calculations are made only under the supposition that vinylidene chloride is carcinogenic to humans."

HAD at 10-131 (emphasis supplied). In applying its three-tiered screening process to identify proposed "high risk" pollutants, EPA has mechanically adopted and applied this VDC inhalation unit risk estimate in the same way as it has applied the unit risk estimates for vinyl chloride and benzene -- known human carcinogens.[17] It ignores the substantial limitations and uncertainties which the Agency

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17/ See "Criteria for Selecting High-Risk Pollutants for the Purpose of Clean Air Act Section 112(i)(5)(E)" at 5-13 (May 15, 1991) (EPA Docket No. A-90-47, Document No. II-B-6) (hereinafter High Risk Criteria).

itself has said are inherent in the VDC unit risk estimate.

The proposed method of listing substances such as VDC conflicts with the Agency's own Guidelines for Carcinogen Risk Assessment, 51 Fed. Reg. 33992 (September 24, 1986). The Guidelines state:

> "[I]t is critical that the numerical estimates [such as unit risk] not be allowed to stand alone, separated from the uncertainties upon which they are based. The risk characterization should contain a discussion and interpretation of the numerical estimates that affords the risk manager some insight into the degree to which the quantitative estimates are likely to reflect the true magnitude of human risk, which generally cannot be known with the degree of quantitative accuracy reflected in the numerical estimates."

51 Fed. Reg. 33999 (emphasis supplied). Where carcinogenic studies evince flaws such as those evident in the Maltoni Swiss mouse study, EPA has counseled special scrutiny and caution in risk assessment:

> "Positive studies at levels above the MTD should be carefully reviewed to ensure that the responses are not due to factors which do not operate at exposure levels below the MTD. Evidence indicating that high exposures alter tumor responses by indirect mechanisms that may be unrelated to effects at lower exposures should be dealt with on an individual basis."

Id. at 33995 (emphasis supplied).

Although the VDC Panel does not believe that it is appropriate to apply quantitative risk assessment calculus to VDC or other Group C or B2 substances, the Panel urges EPA, at least, to adjust for the associated carcinogenic uncertainty if the Agency insists upon quantifying risk estimates for VDC. In other contexts, EPA has proposed adjusting numerical assessments associated with carcinogenic risk to reflect the weight of the evidence of human carcinogenicity. An adjustment is necessary here, if the Agency continues to use the risk assessment methodology for substances like VDC.

As part of EPA's corrective action program for solid waste management units ("SWMUs") under RCRA, the Agency has proposed to establish "action levels" which depend upon substances' carcinogenic classification. See 55 Fed. Reg. 30798 (July 27, 1990). "[A]ction levels are health- and environmental-based levels determined by the agency to be indicators for the protection of human health and the environment" and which trigger requirements for corrective action. Id. at 30814. EPA's proposal prescribes a one order of magnitude adjustment for Group C "possible carcinogens" as compared to Group A and B substances:

> "[C]oncentrations used as action levels must (for carcinogens) be associated with a 1 x 10^{-6} upperbound excess cancer risk for Class A and B carcinogens, and a 1 x 10^{-5} upper bound excess cancer risk for Class C carcinogens."

Id. at 30815.

If the second tier of EPA's "high risk" analysis were similarly altered to adjust the unit risk estimate for Group C substances, VDC would not qualify as a "high risk" pollutant:

- EPA's second tier screening criteria, in effect, selected 22 substances classified in Groups B and C because they have been assigned carcinogenic inhalation unit risk estimates greater than 3 x 10^{-5} $(ug/m^3)^{-1}$.[18/]

- VDC was included on the proposed list of "high risk" pollutants solely on this basis (unit risk

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18/ EPA derived this threshold potency factor based on the presumptive acceptable risk benchmark for benzene (a Group A, known human carcinogen) as set forth in the benzene NESHAP. See High Risk Criteria at 11.

estimate based on Maltoni study: 5×10^{-5} $(ug/m^3)^{-1}$).[19]

- If the benchmark maximum unit risk for Group C substances were adjusted by a factor of ten (to 3×10^{-4} $(ug/m^3)^{-1}$) to reflect limited evidence of carcinogenicity -- the same adjustment as EPA proposed in the SWMU action level rulemaking -- VDC would fall below the benchmark.

While the VDC Panel believes that it is inappropriate to purport to quantify a human risk level for Group C or B2 substances, EPA must, at the very least, adjust its calculus to take into account the fundamental uncertainty about whether such substances pose any risk to humans. An adjustment factor of ten, as proposed by the Agency in the 1990 rulemaking, would effectively remove VDC from the "high risk" list on the basis of this uncertainty.[20]

19/ See High Risk Criteria at 17.

20/ If EPA adopted this modified approach, the only other proposed "high risk" Group C substance would also warrant removal from the draft "high risk" list (i.e., 1,1,2,2-tetrachloroethane: unit risk reported as 5.8×10^{-5} $(ug/m^3)^{-1}$).

D. The Proposal to List VDC as a "High Risk" Pollutant Violates the Clean Air Act and the Administrative Procedure Act.

EPA's process for listing "high risk" pollutants is subject to invalidation by a reviewing court as arbitrary, capricious or otherwise not in accordance with law. See 42 U.S.C. § 7607(d)(9)(A). To survive such review, the Agency must substantiate and explain any departure from its prior determination not to regulate VDC under section 112. In addition, EPA must consider all information relevant to cancer risk (such as VDC's status as only a Group C "possible carcinogen") and thereafter ensure that VDC meets the statutory criteria for "high risk" pollutants.

1. EPA may not change its 1985 decision that VDC poses little or no risk without providing a reasoned analysis supported by substantial evidence.

In view of EPA's 1985 decision that VDC poses little or no risk -- a decision which led the Agency to conclude that no regulation was appropriate under the Clean Air Act -- the proposed regulation of VDC as a "high risk" pollutant represents a fundamental change in the Agency's position. Under established principles of administrative

law, the Agency cannot make such a change without providing a reasoned analysis supported by substantial evidence.

It is a basic principle of administrative law that agencies are bound by their own decisions. The Supreme Court has stated that an agency which chooses to change a previously held position "is obligated to supply a reasoned analysis for the change beyond that which may be required" in cases of first impression. Motor Vehicle Mfrs. Ass'n. v. State Farm Mut. Auto. Ins. Co., 463 U.S. 29, 42 (1983) (emphasis added); see also Center for Science in the Public Interest v. Dept. of Treasury, 797 F.2d 995, 999 (D.C. Cir. 1986) (State Farm placed the "burden ... to justify" upon the agency when it decides to change established policy); Office of Communication v. FCC, 560 F.2d 529, 532-33 (2d Cir. 1977). Moreover, a presumption exists "against changes in current policy that are not justified by the rulemaking record." State Farm, 463 U.S. at 42 (emphasis in original).

Federal courts of appeals take the State Farm "reasoned analysis" requirement seriously. "Simply asserting that conditions have changed" will not meet this elevated level of responsibility. Lehigh Valley Farmers v. Block, 829 F.2d 409, 413 (3d Cir. 1987). Rather, the agency must explain why the original reasons for the agency's

position are no longer dispositive. Brae Corp. v. United States, 740 F.2d 1023, 1038 (D.C. Cir. 1984), cert. denied, 471 U.S. 1069 (1985). Moreover, an agency must compile a "thorough and comprehensible statement of the reasons" underpinning its decision to revise well-established policy. Office of Communication, 560 F.2d at 532 (citing 5 U.S.C. § 553(c)). This statement must be supported by "substantial evidence in the record" and include a discussion of what alternatives were considered and why they were rejected. Lehigh Valley Farmers, 829 F.2d at 413 (Secretary of Agriculture's decision to amend milk marketing order policy was not supported by substantial evidence); International Ladies' Garment Workers' Union v. Donovan, 722 F.2d 795, 817-18 (D.C. Cir. 1983), cert. denied, 469 U.S. 820 (1984) (Secretary of Labor's decision to alter policy regarding homework in the knitted outwear industry failed to give adequate consideration to available alternatives).

These fundamental legal principles are particularly important here. A change in policy regarding VDC is not warranted by changes in the law or by any new evidence. Amendments to the Clean Air Act do not justify the Agency's proposal to negate prior findings regarding VDC. The basic question EPA addressed in 1985 is the same

as the question addressed in this rulemaking: whether VDC poses a carcinogenic risk to public health.

In 1985, EPA decided not to subject VDC to regulation under Section 112 because "the available scientific evidence for the carcinogenic potential of VDC for humans is only limited and . . . an analysis of the public health hazard if VDC were assumed to be carcinogenic indicates that the possible cancer risks are small." 50 Fed. Reg. 32632. When the Agency made those determinations, it emphasized it would "reconsider the conclusions presented here if warranted by the results of further studies or research." Id. at 32634. EPA has not cited any additional scientific evidence which supports reversal of its prior conclusions regarding VDC. In fact, the current proposal solely and improperly relies upon the data EPA criticized as "limited" in 1985. The Agency's reversal is not justified because there has been no change in the relevant scientific evidence.

2. Because the proposed rule ignores fundamental differences among carcinogen classification groups, the inclusion of VDC in the list of "high risk" pollutants is both contrary to law and arbitrary and capricious.

The Agency's three-tier analysis employs quantitative risk assessment methodologies while unreasonably disregarding significant differences in the probability that various substances pose any risk to humans. Despite the teaching of EPA's own Risk Assessment Guidelines, the proposal purports to quantify cancer risks of VDC without accounting for the fact that VDC is a Group C "possible carcinogen," while other substances which are subjected to an identical quantitative risk estimate process are Group A "known human carcinogens." The failure to consider significant distinctions in carcinogen classifications renders the proposed rule arbitrary and capricious.

In evaluating the Agency's approach, a reviewing court would "consider whether the decision was based on a consideration of the relevant factors." Citizens to Preserve Overton Park v. Volpe, 401 U.S. 402, 416 (1971). Normally, agency action will be overturned as arbitrary and capricious if "the agency has . . . entirely failed to consider an important aspect of the problem." State Farm,

463 U.S. at 43. EPA has conceded that both numerical risk estimates and carcinogen classifications are relevant to risk assessment. Its failure to consider the latter renders the proposal legally deficient under State Farm and Overton Park.

By listing VDC as a "high risk" carcinogen without considering its status as a Group C substance, EPA essentially ignores a threshold issue critical to the statutory listing criteria: does VDC pose any risk of cancer to humans? EPA may not purport to implement Section 112 in a manner inconsistent with the operative statutory language. Natural Resources Defense Council v. U.S. Environmental Protection Agency, 824 F.2d 1146 (D.C.Cir. 1987) (reversing EPA's technology-based regulation of vinyl chloride emissions as inconsistent with Section 112's requirement that EPA issue health-based standards). In the case of VDC, EPA's analysis cannc identify VDC as a "pollutant for which high risks of adverse health effects may be associated with exposure to small quantities," because it has not considered the fundamental question of whether VDC poses any human cancer risk. See 42 U.S.C. § 7412(i)(5)(E).

CONCLUSION

For the foregoing reasons, the CMA VDC Panel urges the Agency to delete VDC from the list of "high risk" pollutants.