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CHEMICAL MANUFACTURERS ASSOCIATION

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COURTNEY M. PRICE
VICE PRESIDENT
CHEMSTAR

March 19, 1998

U.S. Environmental Protection Agency
Office of Pollution Prevention and Toxics
Document Control Office (7407)
Room G-099
401 M Street, S.W.
Washington, D.C. 20460

Re: TSCA Docket No. OPPTS-42187A; FRL 4869-1
61 Fed. Reg. 33178 (June 26, 1996); 62 Fed. Reg. 67466 (December 24, 1997)

Dear Sir or Madam:

The Phthalic Anhydride Producers Task Group of the Chemical Manufacturers Association (CMA) is pleased to submit the attached comments on EPA's proposed test rule for hazardous air pollutants. *See* 61 Fed. Reg. 33178 (June 26, 1996). The comments focus on the testing requirements proposed for phthalic anhydride, but do not address EPA's revised interpretation of the applicability of the proposed rule to persons who manufacture chemicals solely as byproducts or impurities. *See* 62 Fed. Reg. 67466 (December 24, 1997). The Task Group anticipates filing supplemental comments specifically addressing byproduct/impurity ("persons required to test") issues prior to the close of the public comment period on this proposed rule.

If you have any questions concerning these comments or would like additional information, please call Marian K. Stanley, Manager of the Phthalic Anhydride Producers Task Group, at (703) 741-5623.

Sincerely yours,

Courtney M. Price

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BEFORE THE
UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

COMMENTS OF THE CHEMICAL MANUFACTURERS ASSOCIATION PHTHALIC ANHYDRIDE PRODUCERS TASK GROUP ON EPA's PROPOSED TEST RULE FOR HAZARDOUS AIR POLLUTANTS

Proposed Test Rule for Hazardous Air Pollutants; Proposed Rule 61 Fed. Reg. 33178 (June 26, 1996)	)))))	TSCA Docket No. OPPTS-42187A; FRL-4869-1

Courtney M. Price
Vice President
CHEMSTAR

Marian K. Stanley
Manager
Phthalic Anhydride Producers
Task Group

David F. Zoll, Esq.
Vice President and
General Counsel

Peter G. McHugh
Counsel, CHEMSTAR

Of Counsel:
William K. Rawson, Esq.
Claudia M. O'Brien, Esq.
Latham & Watkins
1001 Pennsylvania Avenue, N.W.
Suite 1300
Washington, D.C. 20004

March 19, 1998

CHEMICAL MANUFACTURERS ASSOCIATION
1300 Wilson Blvd.
Arlington, VA 22209
(703)741-5000

EXECUTIVE SUMMARY

The Phthalic Anhydride Producers Task Group of the Chemical Manufacturers Association (CMA) is pleased to submit these comments in response to EPA's proposal to require inhalation testing for phthalic anhydride (PA) under Section 4 of the Toxic Substances Control Act (TSCA), as part of a testing initiative for compounds listed as hazardous air pollutants (HAPs) under the Clean Air Act. 61 Fed. Reg. 33178 (June 26, 1996). The Task Group consists of the major U.S. producers of PA.

EPA has taken steps to facilitate and encourage constructive dialogue on the proposed rule. The Task Group supports this dialogue and has participated in this process by attending public meetings and submitting an alternative testing proposal. The Task Group anticipates moving forward with enforceable consent agreement negotiations. However, since EPA and the Task Group have not yet reached final agreement on the alternative testing proposal, the Task Group is submitting these comments on the test rule as proposed. The Task Group's comments raise the following key points:

- EPA should require only testing necessary for conducting residual risk assessments and should explain more fully how the test data will be used. It is inappropriate for EPA to require more than $5.6 million in testing for PA when the Agency has not yet determined how it will conduct residual risk determinations, and therefore is not clear about how it will use the test data.

- EPA should give greater consideration to the negligible potential for general population exposure to PA.

  - EPA intends to use the data from this rulemaking to support its residual risk determinations for HAPs under Section 112 of the Clean Air Act, yet EPA's own conservative modeling techniques show maximum annual average fenceline concentrations of PA are less than one-fifth of one part per billion (ppb). An examination of the negligible potential for general population exposure to PA, coupled with existing studies at high doses showing no systemic effects, should be more than sufficient to conduct any residual risk determinations for PA.

  - There currently are only five "major" sources of PA for which a residual risk assessment may be needed.

  - The case of PA amply illustrates why EPA's trigger of 50 tons per year aggregate emissions, used to select compounds for inclusion in the HAP test rule, clearly is too low.

- EPA should reconsider the specific testing requirements proposed for PA.

  - It simply is not feasible to conduct useful repeated dose inhalation studies of PA at concentrations high enough to produce systemic toxicity. PA is a low vapor pressure solid. Studies to determine the maximum attainable concentrations of respirable PA vapor and dust show that it is not possible to generate toxicologically significant atmospheric concentrations of PA for repeated dose inhalation studies.

- High dosage chronic studies of PA already have been conducted. They show that PA exhibits low toxicity at high oral doses.

- Available data are sufficient to conclude that PA is not carcinogenic, such that additional oncogenicity testing should not be required.

- Acute testing of PA is duplicative of existing studies and not necessary to support EPA's residual risk analyses.

- In light of PA's low systemic toxicity and negligible potential for human exposure, no other testing should be required for PA.

- As a legal matter, EPA has not presented sufficient data and analysis to support the findings required by Section 4 of TSCA for PA.

  - EPA has not shown that insufficient data exist for the Agency to make its residual risk determinations for PA, or that the proposed testing for PA is necessary to enable EPA to make such determinations.

  - EPA has not presented adequate justification to support its "A" finding for PA. EPA has not correlated PA's suspected toxicity with anticipated levels of exposure. EPA also has improperly used its "A" finding for one endpoint to require testing for other endpoints.

  - EPA's "B" finding for PA is based solely on overstated worker exposures, which should not be relevant for determining whether testing should be required to assess residual risks to the general population.

TABLE OF CONTENTS

EXECUTIVE SUMMARY i

TABLE OF CONTENTS iii

INTRODUCTION 1

I. OVERVIEW OF PROPOSED RULE 3

II. THE TASK GROUP SUPPORTS EPA'S EFFORTS TO PROMOTE A CONSTRUCTIVE DIALOGUE ON ISSUES ASSOCIATED WITH THE PROPOSED RULE 5

III. EPA SHOULD EXPLAIN MORE FULLY HOW THE TEST DATA WILL BE USED 7

IV. EPA SHOULD CONSIDER THE NEGLIGIBLE POTENTIAL FOR GENERAL POPULATION EXPOSURE TO PA 9

A. TRI Data Do Not Provide A Reasonable Measure Of Potential Exposure 9

B. The Case Of PA Illustrates Why EPA's Fifty Ton Per Year Trigger Is Too Low 10

C. The Potential for General Population Exposure To PA Is Negligible 11

V. EPA SHOULD RECONSIDER ITS PROPOSED TESTING REQUIREMENTS FOR PA 15

A. It Is Not Feasible To Conduct Repeated Dose Inhalation Studies Of PA At Toxicologically Significant Atmospheric Concentrations 15

B. High Dosage Chronic Oral Studies Of PA Have Already Been Conducted 17

C. PA Inhalation Cancer Studies Are Not Necessary 20

D. Acute Testing Is Not Warranted For PA And Is Not Necessary To Support EPA's Residual Risk Analyses 21

E. No Other Testing Is Necessary For PA 23

VI. EPA HAS NOT ADEQUATELY JUSTIFIED ITS FINDINGS UNDER TSCA SECTION 4 IN SUPPORT OF ITS PROPOSED TESTING REQUIREMENTS FOR PA 25

A. Existing Data Are Not "Insufficient" To Predict The Effects Of PA On Human Health And The Environment 25

B. The Proposed Testing For PA Is Not "Necessary" To Support EPA's Risk Management And Risk Assessment Functions 27

C. EPA's "A" Finding For PA Is Not Adequately Justified 28

D. EPA's "B" Finding Is Not Warranted For PA 31

CONCLUSION 32

INTRODUCTION

The Chemical Manufacturers Association (CMA) Phthalic Anhydride Producers Task Group is pleased to submit these comments in response to EPA's proposal to require inhalation testing of phthalic anhydride (PA) under Section 4 of the Toxic Substances Control Act (TSCA), as part of a testing initiative for compounds listed as hazardous air pollutants (HAPs) under the Clean Air Act (CAA). 61 Fed. Reg. 33178 (June 26, 1996). The Task Group consists of major U.S. producers of PA.[1]

EPA proposes the following inhalation studies for PA: acute toxicity (with histopathology), subchronic toxicity, neurotoxicity, developmental toxicity, reproductive toxicity, carcinogenicity (two species, both sexes), immunotoxicity and respiratory sensory irritation. According to EPA, the primary purpose of this testing initiative is to provide data on HAPs that EPA needs in order to conduct residual risk assessments following installation of maximum achievable control technology (MACT) pursuant to CAA Section 112.

In Part I, the Task Group provides an overview of the proposed rule. In Part II, the Task Group expresses support for steps EPA has taken to facilitate and encourage constructive dialogue on the proposed rule. The Task Group has participated in this process by attending public meetings and submitting an alternative testing proposal. The Task Group anticipates moving forward with enforceable consent agreement negotiations. However, since EPA and the Task Group have not yet reached final agreement on the alternative testing, the Task Group is submitting these comments on the test rule as proposed.

Part III addresses why EPA should require only testing necessary to conduct residual risk assessments under the Clean Air Act, and should explain more fully how the test data will be used. It is inappropriate for EPA to require more than $5.6 million in testing for PA when the

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[1] Members of the Task Group include Aristech Chemical Corporation, Stepan Company, Koppers Industries, BASF Corporation and Exxon Chemical Company.

Agency has not yet determined how it will conduct residual risk determinations, and therefore is not clear about how the test data will be used.

In Part IV of these comments, the Task Group shows why EPA should give greater consideration to the negligible potential for general population exposure to PA. EPA intends to use the data from this rulemaking to support its residual risk determinations for hazardous air pollutants under CAA Section 112, yet currently there are only five "major" sources of PA for which a residual risk assessment may be needed. More significantly, using EPA's own conservative modeling techniques, maximum annual average fenceline concentrations of PA are less than one fifth of one part per billion. An examination of negligible potential for general population exposure to PA, coupled with existing oral studies showing no systemic effects at high doses, should be more than sufficient to conduct any residual risk determinations for PA. Part IV also explains that the case of PA amply illustrates why EPA's trigger of 50 tons per year aggregate emissions, used to select compounds for inclusion in the HAP test rule, clearly is too low.

In Part V, the Task Group explains why EPA should reconsider its proposed testing requirements for PA. It simply is not feasible to conduct repeated dose inhalation studies of PA at concentrations high enough to produce systemic toxicity. PA is a solid at ambient temperatures with a very low vapor pressure. The Task Group has conducted studies to determine the maximum attainable concentrations of respirable PA vapor and dust, and has concluded that it is not possible to generate toxicologically significant atmospheric concentrations of PA for repeated dose inhalation studies. In addition, the Task Group explains that high dosage chronic oral studies of PA already have been conducted and show no systemic toxicity.

Part V also demonstrates that the available data are sufficient to conclude that PA is not carcinogenic, such that additional oncogenicity testing should not be required. Moreover, acute testing of PA is duplicative of existing studies and not necessary to support EPA's residual risk

analyses. The Task Group then explains why, in light of PA's low toxicity and negligible potential for human exposure, no other testing should be required for PA.

Part VI demonstrates that, as a legal matter, EPA has not presented sufficient data and analysis to support the findings required by Section 4 of TSCA. EPA has not shown that insufficient data exist for the Agency to make its residual risk determinations for PA, or that the proposed data set for PA is necessary to enable EPA to make such determinations. Moreover, EPA has not provided adequate justification to support its "A" finding for PA. EPA has not correlated PA's suspected toxicity with anticipated levels of exposure. EPA also has improperly used its "A" finding for one endpoint to require testing for other endpoints. Finally, EPA's "B" finding for PA is based solely on overstated worker exposures, which should not be relevant for determining whether testing should be required to assess residual risks to the general population.

In addition, the Task Group supports and incorporates by reference the comments by CMA on general issues associated with the proposed test rule.

I. OVERVIEW OF PROPOSED RULE

The proposed HAPs test rule includes 21 compounds. EPA proposes to require "Option Three" level testing for all compounds. 61 Fed. Reg. at 33182. "Option Three" includes inhalation studies to address the following endpoints (to the extent data are not already available): acute toxicity (with histopathology), subchronic toxicity, developmental toxicity, reproductive toxicity, neurotoxicity, immunotoxicity, mutagenicity and, where concern is indicated by results of mutagenicity studies or other test data, carcinogenicity. EPA has proposed the following testing for PA: acute toxicity (with histopathology), subchronic toxicity, developmental toxicity, reproductive toxicity, neurotoxicity, carcinogenicity (two species, both sexes), immunotoxicity and respiratory sensory irritation. EPA estimates that this testing will cost more than $5.6 million.

All compounds, including PA, were selected for inclusion in the HAP test rule primarily based on their releases to air, as reported in the 1993 Toxics Release Inventory (TRI)

database under the Emergency Planning and Community Right-to-Know Act (EPCRA). All chemicals selected for inclusion in the test rule had reported air emissions above 50 tons in 1993. EPA did not conduct any air dispersion modeling exercises to estimate likely human exposure(s) from these releases, although the agency has previously conducted such modeling to support CAA rulemakings.

EPA has based its proposed testing requirements for PA on findings under TSCA Section 4(a)(1)(A) (chemicals that may present an unreasonable risk of injury to health and the environment) based on its potential to induce respiratory sensitization, as reported in a 1988 study in rats, and 4(a)(1)(B) (chemicals that enter the environment in substantial quantities or to which there may be significant or substantial human exposure), based primarily on reported worker exposures and a claim of potential general population exposure. The Agency also purported to find that existing PA data are "insufficient" to predict the effects of PA on human health and the environment, and that the proposed testing is "necessary" to support EPA's risk management and risk assessment functions.[2]

The preamble to the proposed rule indicates that the primary use of the test data will be to support residual risk determinations under CAA Section 112(f). 61 Fed. Reg. at 33179-80. EPA confirmed as much in its preliminary response to the Task Group's alternative testing proposal.[3] Section 112 of the Clean Air Act requires EPA to promulgate technology-based standards that require the maximum degree of HAP emission reductions achievable for a source category. CAA Section 112(d). Section 112(f) requires EPA to establish any additional standards that are necessary to protect public health and the environment with an "ample margin of safety," and to ensure an

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2 For the reasons described at pp. 27-35, infra, the Task Group does not believe that EPA has adequately justified its TSCA Section 4 findings.

3 See Letter from Charles Auer (EPA) to Marian Stanley (CMA) (July 10, 1997).

excess cancer risk of no greater than one in one million. CAA Section 112(f)(2). EPA is required to make these "residual risk" determinations only for "major" sources of HAPs.[4]

II. THE TASK GROUP SUPPORTS EPA'S EFFORTS TO PROMOTE A CONSTRUCTIVE DIALOGUE ON ISSUES ASSOCIATED WITH THE PROPOSED RULE

The Task Group supports the steps EPA has taken to facilitate and encourage constructive dialogue on the scientific issues presented by its proposed HAP test rule. EPA provided the Task Group and other interested parties with an opportunity, prior to publication of the proposed rule, to comment on EPA's general approach to HAP testing as well as specific tentative testing proposals. Further, in the proposed rule, EPA has taken a number of steps to encourage constructive dialogue, including: (1) initially allowing 180 days for public comment on the proposed rule, which is longer than EPA has provided for comment on most other TSCA testing proposals; (2) scheduling a public meeting to encourage early dialogue on technical and policy issues; (3) inviting alternative testing proposals, focusing primarily on approaches that might allow use of existing oral data in lieu of conducting new studies by inhalation; (4) setting an early deadline for submission of the alternative testing proposals, and committing to providing feedback during the comment period; and (5) extending the comment period to ensure feedback was provided prior to the end of the public comment period. All of these steps have facilitated constructive dialogue on technical issues, and are therefore to be commended.

The Task Group has utilized the opportunities provided by EPA, meeting with the Agency on the second day of public meetings to address various issues associated with PA. Although, for the reasons discussed below, the Task Group does not believe that any testing of PA is necessary to support residual risk analyses, the Task Group submitted an alternative testing proposal

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[4] The Clean Air Act defines the term "major source" to include any facility that emits or has the potential to emit 10 tons/year of a single HAP or 25 tons/year of any combination of HAPs. See CAA §112(a)(1).

that would be far more cost-effective than the testing identified in the proposed rule. Specifically, the Task Group initially proposed to conduct the following testing for PA:

- respiratory sensitization study in guinea pigs;
- single exposure inhalation toxicity study in rats;
- 90-day inhalation toxicity study in rats[5]; and
- blood level study with PA following inhalation exposure and oral administration in rats.

The blood level study is expected to demonstrate that the existing PA and phthalic acid oral data can be used to satisfy the testing needs asserted in the proposed rule for systemic toxicity endpoints of concern - carcinogenicity, developmental toxicity and reproductive toxicity. The other three studies will be used to evaluate the potential for PA to produce respiratory tract toxicity after single and repeated exposures.

Based on the Agency's initial response to the Task Group's alternative testing proposal and on-going negotiations with the Agency, the Task Group believes that EPA and the Task Group may reach an agreement on an acceptable testing proposal. Moreover, the Task Group appreciates the steps EPA has taken to foster an open, cooperative dialogue on the HAP testing initiative, and hopes that a mutually-beneficial agreement can be reached on the appropriate scope of testing for PA. However, because a final agreement has not yet been reached, the Task Group submits the following comments on the PA testing requirements that initially were proposed by the Agency.

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[5] As described elsewhere in these comments, however, the Task Group believes that it is not technically feasible to conduct repeated dose PA studies at concentrations high enough to cause any adverse effects. Thus, although this 90-day test will be conducted at the maximum attainable concentration, the Task Group does not anticipate that any adverse systemic effects will be seen.

III. EPA SHOULD EXPLAIN MORE FULLY HOW THE TEST DATA WILL BE USED

As stated, the primary purpose of the HAP testing initiative is to support residual risk analyses under the Clean Air Act. Indeed, in its initial response to the Task Group's alternative testing proposal, EPA explicitly stated, "The testing requirements for phthalic anhydride in the proposed HAPs test rule were identified by EPA for the purpose of providing a database to permit the assessment of residual risk following the implementation of the maximum achievable control technology (MACT) standards required by the Clean Air Act." Letter from Charles Auer (EPA) to Marian Stanley (CMA) (July 10, 1997). EPA's residual risk determinations involve an examination of whether implementation of the technology-based MACT standards is sufficient to ensure that the public is protected from non-cancer adverse health effects with an "ample margin of safety" and to ensure an excess cancer risk of no greater than one in one million. CAA § 112(f)(2)(A). However, EPA has not yet articulated how residual risk will be evaluated under the Clean Air Act, even though the Agency was obligated to submit a report to Congress on that subject by November 15, 1996.[6]

Because EPA does not yet know how it will assess residual risks, the Agency is unable to explain how the data will be used to support its activities under the Clean Air Act.[7] Perhaps this is why EPA's proposal lists several "secondary" uses of data. The mere fact that data might be useful in another context, however, is not sufficient to support a test rule under TSCA. The

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6 40 U.S.C. § 7412(f). EPA's report must address, among other topics, "(A) methods of calculating the risk to public health remaining, or likely to remain, from sources subject to regulation under [section 112] after the application of [MACT] standards; [and] (B) the public health significance of such estimated remaining risk . . . ." 42 U.S.C. §§ 112(f)(1)(A) & (B). The Task Group understands that EPA currently plans to issue a draft of this document for public comment by the summer of 1998.

7 EPA claims that the data also will be used to support implementation of "several" other provisions of CAA Section 112, including estimation of risks associated with accidental releases (Section 112(r)) and delisting determinations (Section 112(b)(1)). PA, however, is not listed under Section 112(r), and, in any event, the subchronic and chronic tests proposed by EPA are not relevant for purposes of Section 112(r). Moreover, EPA has not taken any initiative to pursue HAP delistings in the absence of petitions from industry accompanied by extensive supporting documentation. Nor is the Task Group aware of any other provisions of Section 112 for which the proposed data would be useful. Thus, it is clear that EPA intends to use the data from this HAP test rule primarily, if not entirely, to support residual risk determinations.

proposed test rule, as its name indicates, is designed to provide data that will be used by the Agency to support its activities under the Clean Air Act. The mention of potential secondary uses of the data is not a legally sufficient basis to require testing that is not necessary to implement the requirements of Clean Air Act Section 112. See infra at 29-30.

Accordingly, it is inappropriate for EPA to require $5.6 million in testing for PA, involving the use of hundreds of laboratory animals, when the Agency has not yet determined how the data will be used. The substantial cost of this rulemaking cannot be justified for PA, as there is no reasonable basis for believing that the test data will, in fact, be necessary to support residual risk determinations. Before requiring such extensive and expensive testing, EPA should have a clear understanding of how residual risk analyses will be conducted, and how the data from specific studies included in the HAP testing proposal will be used to support such an effort. Without careful consideration for how the data will be used, there is a significant potential for large amounts of money and substantial numbers of laboratory animals to be wasted on testing that is not necessary to evaluate the risks to human health and environment from the manufacturing, processing, use and disposal of PA. Indeed, this will likely be the case, because the negligible potential for human exposure to PA precludes the possibility of any significant risk to human health from this chemical. Therefore, any PA testing should not be dictated by an abstract desire to create a uniform toxicity data base for several chemicals, but by a chemical-specific evaluation of PA. EPA's insistence on "Option Three" level testing for all compounds in the proposed rule suggests this abstract desire and cannot be justified given that the potential for exposure, and the resultant need for data, varies significantly among the chemicals.

Moreover, as discussed at pp. 19-25, infra, substantial amounts of test data already are available for PA. Accordingly, EPA should consider more carefully the extent to which these data are sufficient to support the Agency's legitimate risk assessment and risk management functions. Following such an evaluation, and depending on the outcome of EPA's report to

Congress, the Agency may ultimately conclude, as the Task Group already believes, that the proposed testing for PA in fact is not necessary.

IV. EPA SHOULD CONSIDER THE NEGLIGIBLE POTENTIAL FOR GENERAL POPULATION EXPOSURE TO PA

In its proposed test rule for aryl phosphate base stocks, EPA acknowledged that it is appropriate for EPA to require testing only where "human or environmental exposure is of such magnitude or type that [the chemical] may need to be regulated if test data reveal adverse effects." 57 Fed. Reg. 2138, 2144 (January 17, 1992). In the proposed HAPs test rule, however, EPA did not consider the realistic potential for human exposure. This omission is critical because the Agency's primary justification for requiring testing is to develop data to support EPA's residual risk determinations under the Clean Air Act.[8] Yet EPA has not attempted to determine current general population exposures from industrial releases of PA, or, more significantly, what those exposures are likely to be after implementation of MACT standards. Indeed, EPA proposes extensive testing for PA even though additional toxicity data almost certainly will not be necessary to support residual risk determinations under the Clean Air Act because of the negligible potential for exposure.[9]

A. TRI Data Do Not Provide A Reasonable Measure Of Potential Exposure

EPA selected chemicals for the initial HAP test rule based on the volume of emissions reported on the TRI. The proposal states that EPA selected HAPs "for initial consideration by focusing its attention on HAPs with TRI emissions of 50 tons or more per year." 61 Fed. Reg. at 33184. However, EPA's Science Advisory Board has expressly recognized that data on emissions do not provide a good measure of actual exposure:

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8 The preamble to the proposed rule also identifies a number of "secondary" uses of the data. 61 Fed. Reg. at 33180. Nonetheless, the proposal is, first and foremost, a HAP test rule (as it is so named). EPA's methodology and rationale are tied to the Agency's stated primary goal of developing data to support residual risk analyses. Notwithstanding the secondary uses, the proposed testing may be required only if necessary to meet EPA's primary objective of conducting these residual risk analyses.

9 As described at pp. 27-28, infra, EPA's authority under Section 4 of TSCA extends only to testing that is "necessary" to meet the Agency's regulatory needs.

> The TRI emissions data . . . do not reflect toxicity/potency of various pollutant emissions and are not related to exposure in a simple way, *i.e.*, large emissions do not automatically imply large exposures and risks.[10]

Moreover, although the Agency proposed testing only for compounds with aggregate air emissions in excess of 50 tons per year, EPA did not attempt to evaluate quantitatively or qualitatively the nature or pattern of the releases to air, or the potential for significant general population exposure from releases. This omission is critical for PA because, as shown below, PA's physical and chemical properties preclude the possibility of any realistic exposure scenario in which the general population could be exposed to toxicologically-significant doses.

B. The Case of PA Illustrates Why EPA's Fifty Ton Per Year Trigger Is Too Low

Even if TRI data were an appropriate measure of potential exposure, EPA's trigger of 50 tons per year aggregate emissions, which was used to select compounds for inclusion in the HAP test rule, is clearly too low.[11] This point is illustrated by considering the physical-chemical properties of PA. PA exists as a solid under ambient conditions and has a low vapor pressure of 2×10^{-4} mm Hg at 20°C. The melting point of PA is 130.8°C, and its boiling point is 284.5°C. In water, PA rapidly hydrolyses to phthalic acid, a readily biodegradable substance. PA has an estimated half-life in water of 1.5 to 2 minutes (depending on pH). The half-life of PA in air varies from <1 hour to

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10 Letter to Carol M. Browner, EPA, from Dr. Genevieve Matanoski, Dr. Mark Harwell and Dr. Rolf Hartung, Science Advisory Board, regarding "Science Advisory Board Review of the Technical Basis for Listing Ammonia on the Toxics Release Inventory" (Feb. 2, 1995). The letter further stated that Science Advisory Board members "expressed concerns about the potential for misinterpretation of the TRI data and for inadvertently directing environmental protection efforts away from the areas of most significant risk." Id.

11 EPA's trigger is only one-tenth of EPA's threshold of 1,000,000 pounds (500 tons) for making a finding of "substantial" releases to the environment under TSCA Section 4(a)(1)(B). See Final Statement of B Policy, 58 Fed. Reg. 28736 (May 14, 1993). Yet the Agency provides no justification for this cutoff except that it "captures high emissions of HAPs and . . . [the Clean Air Act] defines 'major source' as emitting" 10 tons per year of a single HAP or 25 tons per year of any combination of HAPs. 61 Fed. Reg. at 33184. The relevance of EPA's "justification" is not clear. The fact that aggregate emissions from all facilities nationwide exceed fifty tons per year does not mean that there are a large number of major sources for which residual risk determinations will need to be made.

>560 days, depending on atmospheric humidity or rainfall.[12] Thus, the physical properties of PA make the possibility of human and/or environmental exposure quite low.

In addition to physical-chemical properties, the manufacturing processes of PA must be considered. PA is produced and used largely in enclosed systems; emissions to air are minimized through the use of condensers and scrubbers. Thus, in the 1993, 1994 and 1995 TRI data bases, only five facilities reported releases of PA to air in excess of 10 tons/year.[13] The Task Group has obtained 1993 through 1996 TRI data from these five facilities, as follows:

	Air Emissions of PA TRI Data (lbs.)			
Facility	1993	1994	1995	1996
Stepan	192,000	156,300	348,311	178,594
Sterling	85,000	51,174	42,522	43,193
Exxon Chemical	44,120	34,784	39,200	36,000
Aristech	19,050	22,570	10,558	5,071
Koppers	25,520	39,795	26,461	26,787

C. The Potential For General Population Exposure To PA Is Negligible

Even more significant than these low releases and few major sources, EPA has previously recognized that the relevant analysis for determining whether testing should be required under TSCA Section 4 is whether there is human and environmental exposure to a chemical, not whether releases have occurred. See supra at 9. Yet the potential for exposure to PA is negligible. In 1988, EPA conducted air dispersion modeling for three facilities that had annual releases of PA from 21,000 to 66,000 pounds. EPA estimated the maximum annual average fenceline concentration for Aristech Chemical Corporation,[14] the facility with the highest estimated fenceline concentration, to be 0.0046 mg/m^3 (0.00077 ppm, or 0.77 ppb). The concentrations at the other facilities were three

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12 EPA Chemistry Report for Phthalic Anhydride, November 30, 1987, TSCA Docket #400024, E1-002.

13 A total of 182 facilities submitted a toxic chemical release form for PA in 1995. These facilities reported a combined total of only 711 pounds of PA released to water and 674 pounds released to land.

14 When EPA conducted its air dispersion modeling in 1988, the facility was owned by TENN-USS.

to four orders of magnitude lower.[15] According to EPA, the significantly higher estimated concentrations at Aristech, compared to all other facilities, resulted from a stack location only 60 meters from the fenceline with a neighboring industrial property, as well as

> "release height, emission strength, exit temperature, and local meteorological conditions. These factors contribute to low plume rise and low dispersion, resulting in higher ambient air concentrations."[16]

EPA used quite conservative assumptions to predict maximum values; the Task Group anticipates the actual annual average fenceline concentrations to be lower than the predicted values.

To provide a conservative estimate of current fenceline concentrations, the Task Group has incorporated 1996 TRI data into EPA's conservative air dispersion modeling results for Stepan Company, the highest emitter of PA according to 1996 TRI data, and Aristech, the facility with the highest estimated fenceline concentrations in 1988 according to EPA modeling. Based on this approach, the Task Group estimates that the maximum annual average fenceline concentration in 1996 at the Stepan facility was 0.000092 ppb.[17] The maximum annual average fenceline concentration at Aristech in 1996 was 0.17 ppb.[18] Thus, even conservative modeling techniques predict a maximum exposure to PA of less than one-fifth of one part per billion.[19]

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15 EPA Exposure Assessment, SARA Title III, Section 313 Petition to Delist Phthalic Anhydride, TSCA Docket #400024, B-001.

16 Id.

17 This calculation retains all modeling assumptions made by EPA in its 1988 air dispersion modeling of Stepan Company's plant. The Task Group acknowledges that slight variations in climate, etc., may have occurred since then, but does not believe that any such changes would significantly affect the resultant concentration. In 1987 (the year for which EPA's original calculations were made), Stepan Company emitted 66,000 pounds of PA, with an average annual fenceline concentration of 0.000034 ppb. In 1996, Stepan Company emitted 178,594 pounds of PA, resulting in an estimated average annual fenceline concentration of 0.000092 ppb.

18 This calculation also retains all modeling assumptions made by EPA in its 1988 air dispersion modeling. In 1987, Aristech (then TENN-USS) had PA emissions of 23,000 pounds, resulting in an average annual fenceline concentration of 0.77 ppb. In 1996, Aristech emitted 5,071 pounds, resulting in an estimated average annual fenceline concentration of 0.17 ppb.

19 The Task Group anticipates that any minimal concentrations to which the general population might be exposed would be in the form of an aerosol. Although PA may sometimes be released as a vapor, condensation of the vapor usually occurs rapidly following emission, leading to aerosol formation.

To provide a more accurate estimate of fenceline concentrations of PA, Aristech contracted with ENSR Corporation to conduct fenceline monitoring and emissions modeling for its Pasadena, Texas facility. ENSR conducted six sampling sessions of 48-hours each at four separate monitoring sites along the Aristech fenceline that is closest to the PA plant. ENSR installed and operated a portable meteorological monitoring station at the facility to assess wind speed, wind direction, temperature and stability, and selected samples for further analysis based upon optimum meteorological conditions (*i.e.,* conditions that maximize fenceline concentrations). ENSR then compared its 48-hour measured PA concentrations to the concentrations that were predicted using the Industrial Source Complex Short-Term (ISCST3) model. For example, during the November 16-18, 1996 monitoring period, the measured fenceline concentration at monitoring site 1 was 0.32 ug/m^3, while the predicted concentration using the ISCST3 model for the same site during the same period was 2.8 ug/m^3. ENSR found that the average measured-to-modeled concentration ratio was 0.18. In other words, measured fenceline concentrations were approximately one-fifth of what had been predicted based on modeling.

ENSR then used these data, coupled with the five years of meteorological data (1987-1991) provided on EPA's SCRAM electronic bulletin board, to model the maximum annual average PA concentrations at the fenceline. ENSR found that the year with the highest estimated fenceline concentrations was 1990. Using its measured-to-modeled ratios and 1990 meteorological data, ENSR calculated that in this year, the maximum annual fenceline concentration was 0.5 ug/m^3 (0.08 ppb), compared to a modeled concentration, using EPA's standard modeling techniques (ISCLT), of 2.9 ug/m^3 (0.44 ppb). Based on these results, ENSR calculated that the annual average fenceline concentration for the Pasadena facility, based on 1995 estimated releases, which were derived from 1995 PA production, would be 0.44 ug/m^3 (0.07 ppb). The ENSR report confirms that actual fenceline concentrations of PA are extremely low. A copy of the ENSR report is attached.

For the purposes of this analysis, we have examined PA releases separately from the releases of any other compounds. Thus, there may be a few facilities that do not emit more than 10 tons per year of PA, but do emit over 25 tons per year of a combination of HAPs.[20] Nonetheless, the mere fact that there may be a few such sources for any individual compound does not justify EPA's general refusal to consider exposure. First, EPA has not explained how it will address mixtures of HAPs generally. Second, the mere existence of a few additional major sources does not justify why EPA needs to require "Option Three" level testing for all chemicals, including PA. Such an approach is fundamentally inconsistent with the legal requirement (and policy need) that EPA only require such testing as is necessary to support residual risk determinations. See infra at 27-28.

In the case of PA, it is readily apparent that the proposed test rule constitutes an extremely expensive way to generate data for purposes of performing residual risk analyses for only a handful of facilities, especially since reported PA air emissions clearly result in very low ambient concentrations beyond facility boundaries. In its briefing materials on toxicity testing of HAPs, EPA identified Step 3 of HAP selection for this test rule as analyzing more detailed exposure information, including biofate/persistence, routes of exposure and chemical/physical properties. Much of this information is readily available for PA, and should be considered by EPA when determining whether or not additional testing is warranted. In light of EPA's air dispersion modeling results, Aristech's monitoring and modeling data, and the physical/chemical properties of PA that make it extremely unlikely that significant concentrations will pass the facility site boundary and result in substantial

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20 However, the Task Group believes that it is highly unlikely that emissions of less than 10 tons of PA per year would have a meaningful impact on any residual risk analysis for a facility as a whole. When setting proposed de minimis emissions rates under Section 112(g) of the Clean Air Act, EPA utilized generic air dispersion modeling assumptions for a hypothetical facility. EPA calculated that every two tons (4,000 pounds) of emissions would result in a fenceline concentration of only 0.001 mg/m^3. Such extremely small concentrations are unlikely to have a significant impact on residual risk assessments, even where other HAP releases exceed major source thresholds. Moreover, to our knowledge, there is no convenient way to search the TRI database in a way that would identify all major sources of mixtures of HAPs.

exposures to the general population, EPA should conclude that additional testing of PA for purposes of assessing residual risk is not warranted.

V. EPA SHOULD RECONSIDER THE PROPOSED TESTING REQUIREMENTS FOR PA

A. It Is Not Feasible To Conduct Repeated Dose Inhalation Studies Of PA At Toxicologically Significant Atmospheric Concentrations

As noted above, PA exists as a solid under ambient conditions, and has a vapor pressure of 2×10^{-4} mm Hg at 20°C. Because of PA's low vapor pressure, EPA has proposed that all PA testing be conducted using an aerosol. Given PA's physical properties, however, the Task Group doubted the feasibility of conducting repeated dose inhalation studies of phthalic anhydride at meaningful doses, even using an aerosol. The Task Group therefore sponsored studies to determine the maximum concentrations of respirable phthalic anhydride vapor and dust that could be attained under conditions suitable for conducting animal toxicity studies. Studies also were conducted to determine if a toxic respiratory response (e.g., respiratory irritation) could be detected in rats at maximum attainable concentrations of phthalic anhydride vapor or dust.

The studies were completed in 1996, and the final reports were submitted to EPA in connection with this proposed rule.[21] Maximum vapor atmosphere concentrations were generated from molten PA. The studies also examined several milling and pneumatic dispersion techniques, as well as evaporation condensation, in an attempt to generate dust atmospheres.[22] The study results show the following:

Maximum attainable PA vapor concentration[23]	1.6 ppm or 1 mg/m^3

21 See Attachments to August 28, 1996, Additional Preliminary Comments of the CMA Phthalic Anhydride Producers Task Group.

22 Techniques evaluated included centrifugal mill, cutting mill, freeze grinding, jet mill and venturi aspirator.

23 This concentration is similar to the theoretical saturated vapor concentration of PA at 21°C (1.3 ppm).

Maximum attainable PA dust concentration (one hour)	630 mg/m^3
Minimum attainable particle size (MMAD)	3.5 - 7.7 um

Based on the results of these studies, the Task Group concludes that repeated dose inhalation testing with PA dust is not technically feasible. PA clogs the equipment and dust generation is sporadic, such that the longest possible test run was one hour. These technical difficulties were encountered regardless of the method of dust generation employed. In addition, the size of the PA dust particles in the exposure atmospheres exceeded the range considered acceptable by EPA for inhalation testing.[24] Moreover, no signs of irritation or respiratory rate change were observed at the concentrations attained in the studies. Based on these results, the Task Group concludes that it is not possible to generate toxicologically significant atmospheric concentrations of phthalic anhydride for repeated dose inhalation studies.[25]

This conclusion is supported by the results of the chronic feeding studies of phthalic anhydride conducted by the National Cancer Institute (NCI). As described at p. 20, infra, the dietary no observed effect level (NOEL) of 15,000 parts per million (ppm) for rats observed in the NCI study equals a daily systematic dose of approximately 750 mg/kg/day. To receive a comparable systemic dose by inhalation, rats would have to be exposed to a test atmosphere of 4,700 mg PA/m^3, six hours per day, seven days per week—obviously an impossible dosing regime and, in terms of human health hazard assessment, an unrealistic exposure scenario. Maximum annual fenceline concentrations are more than six orders of magnitude below the calculated inhalation—equivalent NOEL based on the

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24 Particle size distribution was as follows: 1 to 10% of the particles were ≤ 1 um; 20 to 30% of the particles were ≤ 3 um; 80 to 90% of the particles were ≤ 10 um.

25 Nonetheless, to address EPA's concerns the Task Group has proposed to conduct 90-day repeated dose inhalation testing at the maximum attainable concentration, although the Task Group considers it unlikely that adverse effects will be seen at these doses.

chronic oral studies.[26] Thus, the maximum attainable inhalation dose of 600 mg/m^3 phthalic anhydride dust (equivalent to a systemic dose of approximately 95 mg/kg/day) would be unlikely to produce systemic toxicity even after repeated dosing. Conducting studies at this low atmospheric concentration would not be justified scientifically.[27] Nor is it ethically justifiable to sacrifice of large number of laboratory animals in studies that are unlikely to produce systemic toxicity.

B. High Dosage Chronic Oral Studies Of PA Have Already Been Conducted

NCI conducted lifetime studies on rats and mice using extremely high oral doses of PA. Even at the highest dose level in rats, 15,000 ppm, no adverse effects were seen. Dose levels were 16,346 and 32,692 ppm (2.1 and 4.3 g/kg/day) for male mice, and 12,019 and 24,038 ppm (1.5 and 3.1 g/kg/day) for female mice. (NCI, 1979). With respect to the mice dosed with PA, the NCI review of the experimental data determined that "several chronic inflammatory, degenerative, or proliferative lesions frequently seen in aged laboratory mice occurred with approximately equal frequency and severity in the dosed and control groups of animals." NCI did not consider these results to be significant adverse effects. However, EPA's IRIS workgroup interpreted the incidence data differently than NCI, concluding that significant adverse effects were observed in this study. According to the IRIS workgroup, the following dose-related effects include: lung and kidney

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26 The estimated maximum fenceline concentration of 0.17 ppb equals approximately 1.03 ug/m^3, which is more than a million-fold below the calculated inhalation-equivalent NOEL of 4,700 mg/m^3.

27 The Task Group also supports the comments of CMA urging caution in extrapolating from the results of aerosol tests to vapor exposures. Aerosols and vapors represent two distinct physical states -- i.e., liquid or solid droplets and gas, respectively. Thus, the use of aerosol data as a measure of toxicity by inhalation is not simply a matter of extrapolating from higher dosage aerosol studies to lower dosage vapor exposures. Rather, the distribution of aerosols in the respiratory system can differ significantly from vapor distribution. As a result, effects from exposure to a vapor may be quite different from the effects of aerosol exposure. Moreover, the size of the aerosol particle will have a dramatic effect on potential human exposure. Thus, to utilize toxicity data from aerosol exposures to estimate toxicity from vapor inhalation, EPA at a minimum must assume: (1) that significant quantities of the aerosol reasonably could be expected to become airborne in a release scenario; (2) the aerosol would be of respirable size; and (3) the aerosol would remain suspended in the air at concentrations high enough to cause harm to the local community. In reality, these assumptions are unlikely to occur.

lymphocytosis in both sexes at both doses; chronic bile duct inflammation in high doses males and females; and adrenal atrophy and mineralization of the thalamus in males.

The dose levels in the NCI study were much higher than those commonly used in repeated-dose toxicity studies (1.5 and 3.1 g/kg/day for females and 2.1 and 4.3 g/kg/day for males). The results of studies conducted at such extreme doses must be interpreted with caution. In view of this, but contrary to EPA's conclusions from the NCI data, NCI concluded that the inflammatory, degenerative and proliferative effects issuing from the NCI study, which are often seen in aged mice, were not toxic responses specific to phthalic anhydride but were non-specific stress-related changes resulting from administration of the extremely high doses of the test material. Additional support for this interpretation comes from the quantitative observation that orally administered phthalic acid is eliminated unchanged via urine in rats.[28] Given the rapid hydrolysis of phthalic anhydride to phthalic acid, the amount of acid distributed systemically in the test animals in the NCI study was substantial, if not overwhelming, and the non-specific irritation due to phthalic acid exacerbated the senescence lesions reported in the NCI study. The Task Group believes that these effects would not have been produced in rodents treated at a more physiologically relevant dose, i.e., doses at which the homeostatic buffering capacity of animals was not severely challenged. In particular, lung and kidney lymphocytosis could be attributed to acid-induced chronic irritation seen in the NCI study.[29]

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28 See Hoshi, A. and Kuretani, K., Metabolism of Terephthalic Acid, *Chem. Pharm. Bull.*, 15(12) 1979-1984, 1967.

29 Because PA is readily hydrolyzed to the corresponding acid, it is anticipated that this hydrolysis will occur completely and rapidly following inhalation or oral exposure. Information on the comparative rate and extent of PA and phthalic acid absorption following oral and inhalation dosing will delineate the quantitative nature of the relationship between PA and acid and between oral and inhalation dosing. The acid is freely distributed throughout the body and all acid is eliminated unchanged through the urine. (Hoshi, A. and Kuretani, K., Metabolism of Terephthalic Acid., *Chem. Pharm. Bull.*, 15(12) 1979-1984, 1967.) Accordingly for purposes of this comparison, information on volume of distribution, metabolism and material balance are irrelevant. Only peak blood level and blood concentrations of the acid and the anhydride are important for: 1) utilization of existing toxicity data on the acid and the anhydride, and 2) interpretation of portal of entry effect data and sensitization/irritation effects data.

Moreover, the NCI conclusions are consistent with the findings of EPA's contractor. A Dynamac Corporation report prepared for EPA on phthalic anhydride found that, based on the results of a 1979 NCI study, "no chronic toxicity affecting survival was reported in animals fed phthalic anhydride in the diet for two years. The NOEL for rats is probably close to 15,000 ppm fed in the diet whereas the NOEL for mice is probably less than 6,000 ppm in the diet."[30] The Dynamac report concluded that the only effect seen at 6,000 ppm in mice was reduced body weight gain and that the study found no evidence of any carcinogenic effects of PA, even at high doses.[31]

The report identifies a number of inadequacies in the study, but concludes that "dosages were so high as to negate any criticism of the study as a whole."[32] The report also comments that although the study was conducted prior to publication of the TSCA Test Guidelines, "with minor exceptions, the chronic toxicity test meets TSCA Test Guidelines for this type of study."[33]

Task Group toxicologists estimate the NOEL of 15,000 ppm for rats in this study to equate to approximately 750 mg/kg/day. To receive a comparable systemic dose by inhalation, rats would have to be exposed to a test atmosphere of 4,700 mg PA/m^3, six hours per day, seven days per week. Such a concentration is essentially equivalent to the limit concentration for acute inhalation studies under TSCA test guidelines.[34] Chronic studies typically are conducted at concentrations well below the acute limit concentration. Moreover, the irritating properties of PA likely would prevent repeated exposure inhalation testing at such high concentrations.

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30 Evaluation of Potential Acute, Subchronic, and Chronic Toxicity of Phthalic Anhydride in Response to a Petition to Delist this Substance from the Toxic Emissions Inventory, September 14, 1988, Docket #40024, E1-003, Enclosures 3 and 4.

31 Id.

32 Id.

33 Id.

34 See 40 C.F.R. § 798.1150.

C. PA Inhalation Cancer Studies Are Not Necessary

In its support document for the proposed test rule, EPA states that inhalation carcinogenicity testing is necessary because PA is a suspect carcinogen. EPA bases this conclusion on the claim that PA is a potential direct-acting acylating agent and some acylating agents are carcinogenic. EPA also notes that the oral NCI study showed an increase in alveolar/bronchiolar adenomas in female rats. Finally, EPA claims that because of the possibility of portal of entry effects, the oral cancer studies are inadequate to determine whether PA is an inhalation carcinogen.[35]

EPA's concerns are unfounded, and existing toxicity data are adequate to conclude that PA is not likely to be carcinogenic by any route of exposure, including inhalation. EPA cites a report that identifies PA as weak acylating agent *in vitro*. However, a relationship between *in vitro* data on DNA adducts and adverse health effects has not been established. Moreover, PA showed only weak acylating activity *in vitro*, and given the low exposure levels achievable by inhalation exposure (and more importantly, the low human exposure levels extant in the environment), there is no reason to believe that PA would induce significant adverse human effects through formation of DNA adducts *in vivo* in target tissues. The NCI two year feeding study in rats and mice at high dietary PA concentrations clearly demonstrated the lack of relevant DNA adduct formation in the directly PA exposed tissues, since no gastrointestinal tract tumors occurred.

The detection of DNA adducts in a tissue does not indicate a specific tumorigenic risk for that tissue. Furthermore, DNA adduct formation does not necessarily indicate a mutagenic effect. Many variables are known to effect mutagenesis of DNA adducts: adduct orientation, adduct size, adduct site, nature of polymerase, sequence context, base incorporation initial kinetics, complete replication *in vitro* and *in vivo,* and repair.[36]

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35 TSCA Section 4 Findings for 21 Hazardous Air Pollutants at 73-4.

36 See Singer, B., DNA Damage; Chemistry, Repair and Mutagenic Potential. *Regulatory Toxicology and Pharm.*, 23, 2-13, 1996.

Nor does the increase in alveolar/bronchiolar adenomas in female mice warrant a conclusion that PA is a possible carcinogen. Although extremely high doses were used in the NCI studies, PA neither affected the survival of the test animals nor produced any neoplastic lesions. The increased incidence of alveolar/bronchiolar adenomas in female rats was specifically evaluated by NCI and did not affect their conclusion that the study did not provide any evidence of carcinogenicity to mice or rats from PA exposure. EPA also has stated that "based on the results of cancer bioassays in rats and mice, PA is not known to cause nor can it reasonably be expected to cause cancer in humans."[37] Moreover, both PA and its metabolite, phthalic acid, have tested negative in a battery of mutagencity tests, including the Ames test and in vitro cytogenetic assays.[38] The Task Group submits that the failure of the test data to show any indication of PA carcinogenicity even at high oral doses and PA's lack of mutagenicity, coupled with the extremely low estimated concentration levels at the fenceline, warrants a finding by EPA that inhalation cancer testing is unnecessary.

D. Acute Testing Is Not Warranted For PA And Is Not Necessary To Support EPA's Residual Risk Analyses

EPA justified its acute testing requirement by claiming that no acute studies were found in the literature for PA. 61 Fed. Reg. at 33190-93. However, PA has been tested for acute inhalation toxicity, as well as for acute oral and dermal toxicity. All of these studies confirm that PA exhibits low acute toxicity. AIHA (1967) reported that rats and rabbits exposed to greater than 10,000 mg/m^3 PA four hours per day for several days experienced a 25 percent mortality rate. In another study (Exxon, 1975), rats, mice and guinea pigs were exposed to greater than 80 mg/m^3 PA vapor for six hours. There were no deaths, no signs of toxicity or respiratory tract irritation and no

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37 EPA Hazard Assessment of Phthalic Anhydride, October 3, 1988, Docket #400024, E1-003.

38 See Galloway et al., 1987; Phillips et al., 1986; Zeiger et al., 1985; NCI, 1982; Argarwal et al., 1985, Florin et al., 1980; Kozumbo et al., 1982; Omor et al., 1976.

gross pathologic lesions.[39] These dose levels are more than four to seven orders of magnitude higher than the most conservative EPA estimates of maximum PA fenceline concentrations, confirming that PA cannot be anticipated to cause adverse acute effects under any realistic exposure scenario.

Moreover, the mere fact that each of these acute studies is not "perfect" — or in some way does not conform exactly to the latest revisions in EPA's testing guidelines — should not be a sufficient basis to reject them entirely and require additional testing. Instead, the relevant question should be whether the existing acute studies in the aggregate, coupled with other information about toxicity and physical/chemical properties, are adequate to characterize the chemical's acute toxicity under reasonably foreseeable exposure scenarios. It is obvious that this is the case for PA. In spite of any deficiencies in the PA acute inhalation studies, PA clearly poses at most a low acute hazard. Given the negligible fenceline concentrations of PA, additional acute data are not necessary to characterize any risks from PA releases, even if such data might be interesting from a scientific viewpoint. Mere academic interest is not sufficient to justify requiring testing under Section 4 of TSCA. See infra at 27-35. Additional acute testing of PA clearly would be both unnecessary and wasteful, particularly where, as here, EPA has articulated only general rationales for why the data would be beneficial, and has not explained how any acute PA data will be used.

Indeed, EPA has not presented an adequate justification for its proposal to require acute toxicity testing generally. Acute toxicity testing is not necessary to support the Agency's activities under the Clean Air Act. As noted above, the primary goal of the HAPs testing initiative is to develop data to support residual risk analyses under the Clean Air Act. Residual risk analyses will focus on potential chronic effects from long term exposure to routine or continuous facility emissions. Acute toxicity data clearly are not relevant to these analyses.

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[39] PA's low acute toxicity is confirmed by studies showing an oral LD_{50} in rats of 4.0 g/kg (IBT, 1970; AIHA, 1967), and a dermal LD_{50} in rabbits of greater than 3.16 g/kg (Exxon, 1975; IBT, 1970).

Nor will acute data be useful for making de-listing decisions under the Clean Air Act Section 112(b)(2). The Act specifically states that in making listing or de-listing determinations, EPA may not consider potential exposure from accidental releases. CAA Section 112(b)(2). Finally, EPA justified its proposed acute testing requirements by stating that the data will be useful for evaluating assessments of "burst" exposures, such as in the accidental release prevention program under CAA section 112(r). 61 Fed. Reg. at 33179-80. However, PA is not included in EPA's list of acutely hazardous substances covered by the accidental release prevention programs under CAA Section 112(r). Indeed, PA's vapor pressure is more than 50,000 times lower than the vapor pressure cut-off established by EPA for listing chemicals pursuant to Section 112(r). See 59 Fed. Reg. 4478, 4495 (1994).[40]

E. No Other Testing Is Necessary For Phthalic Anhydride

The general population exposure to PA from current industrial releases is negligible, and releases and exposure levels are likely to decrease as MACT standards are implemented. These minimal exposures, coupled with chronic oral studies that show NOELs at concentration equivalents many orders of magnitude higher than the most conservative EPA estimates of PA fenceline concentrations, warrant a finding by EPA that additional testing of PA is unnecessary. Further, when the existing data base for a chemical is adequate to assess satisfactorily its hazard to man, humane considerations warrant avoidance of duplicative testing on vertebrate animals.

There is no reason to believe that fenceline concentrations of less than half of one ppb present hazards to human health. In fact, a 1976 EPA-sponsored report estimated a peak PA ambient concentration of 0.08 ppm and an average ambient concentration of 0.04 ppm, but still found that:

> "phthalic anhydride as an air pollutant apparently does not pose a threat to the health of the general population. In addition, phthalic

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40 EPA excluded from the Section 112(r) list any chemical with a vapor pressure less that 10 mmHg. Id. PA's vapor pressure is 2×10^{-4} mmHg at ambient conditions.

> anhydride does not appear to pose other environmental insults which would warrant further investigation or restriction of its use at the present time."[41]

More recent data bolster this finding of no health threat from PA. Chronic studies found no significant toxicity even at very high doses, and the EPA air dispersion modeling showed that ambient concentrations are much lower than previously thought.

The proposed reproductive toxicity testing illustrates this point. In a poorly-reported abstract of a Russian study, inhalation of 1.0 mg/m^3 PA for 45 days reportedly caused abnormal sperm mobility and some biochemical changes (increase in RNA) in the testes of rats (Protsenko, 1970). As EPA knows, non-Western reports often cite adverse effects from low doses which are not generally reconciled by Western investigators. Western subchronic inhalation studies for other acid anhydrides have not indicated testicular lesions in rats and other species (Leach, et al., 1987; Short, et al., 1988). Furthermore, subchronic and chronic studies of PA at high oral doses have failed to produce testicular lesions in rats and mice (Kluwe, 1986; NCI, 1979). Finally, subchronic oral studies of phthalic acid (Oishi and Hiraga, 1980) and other anhydride analogs (Amoco MSDS) or phthalic acid producing compounds (Foster, et al., 1980) have failed to produce testicular lesions in rodents. A weight of the evidence review suggests that PA is unlikely to produce testicular lesions/effects in rats as claimed by Russian investigators. Even assuming the validity of the Protsenko study, however, EPA's conservative estimates of PA fenceline concentrations are almost three orders of magnitude lower than the dose level cited by Protsenko. Thus, even if these study results are valid, it is clear that PA cannot be expected to cause reproductive toxicity under any realistic exposure scenario. Accordingly, the existing data are sufficient to evaluate residual risks of reproductive toxicity from PA.

Similarly, developmental toxicity has been reported in mice administered PA intraperitoneally only at the maternally-toxic dose (80 mg/kg) (Brown et al., 1978). Similar results

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[41] Patterson, R. et al. (1976), PB-258-366-4.

were reported by Fabro et al., (1978 and 1982). Regardless of the relevance of findings of developmental effects at maternally toxic doses, it is clear that feasible exposure scenarios that could warrant further systemic toxicity testing do not exist.

Accordingly, it is clear that EPA's proposed testing requirements for PA are far broader than necessary to support the Agency's legitimate testing needs. Although EPA has not identified how it will assess "residual risk," any such assessment must, by definition, consider not only the potential human health effects of the chemical, but also exposure. Yet available toxicological data and exposure information for PA provide ample evidence that PA cannot reasonably be expected to pose a risk beyond facility site boundaries. Fenceline concentrations are minimal, and PA chronic oral studies show very high NOELs -- 6,000 ppm for mice and 15,000 ppm for rats. The minimal potential for human exposure to phthalic anhydride, coupled with chronic studies that show NOELs at inhalation concentration equivalents more than seven orders of magnitude higher than the most conservative EPA estimates of phthalic anhydride fenceline concentrations, should be sufficient for EPA to make its residual risk determinations for this chemical. Additional testing for PA simply is not necessary.

VI. EPA HAS NOT ADEQUATELY JUSTIFIED ITS FINDINGS UNDER TSCA SECTION 4 IN SUPPORT OF ITS PROPOSED TESTING REQUIREMENTS FOR PA

A. Existing Data Are Not "Insufficient" To Predict The Effects Of PA On Human Health And The Environment

Under TSCA Section 4, EPA must find that there are "insufficient data and experience" to determine or predict the effects of the chemical on human health and the environment. TSCA Section 4(a)(1)(A)(ii), 4(a)(1)(B)(ii). This analysis, however, cannot be conducted in a vacuum. Because information is never complete, and because less recent studies can always be shown to be less than ideal when compared against new testing guidelines, existing data always are imperfect in some sense. This is true even where the existing data clearly demonstrate

the presence or absence of an effect on human health or the environment, and even where existing data cover virtually all endpoints.

Thus, Congress recognized that while infinite quantities of data are desirable in a perfect world, in a world of limited resources, the data gathered should be tied closely to the regulatory purposes for which they are used. For the findings required by TSCA Section 4 to have meaning, therefore, they must be tied to the basis for the testing. That is, EPA must show why existing data are insufficient for the regulatory purposes for which they are intended to be used.

EPA has stated that the data are needed to conduct residual risk analyses. Yet nowhere in its findings has EPA made a showing that the existing data are insufficient to conduct residual risk analyses. Instead, EPA has identified perceived inadequacies of certain portions of the data base, but has not shown that those asserted deficiencies render the studies inadequate to perform residual risk assessments under the Clean Air Act. Moreover, by refusing to consider likely levels of exposure, EPA fails even to attempt to conduct the statutorily-required analysis. As a result, EPA's proposal does not meet the requirements of TSCA Section 4.[42]

For PA, existing data are not "insufficient" to conduct residual risk determinations. PA already has been tested orally at extremely high doses, with no systemic effects. To administer a comparable inhalation dose would be physically impossible, given PA's physical and chemical properties. Such a dose would be more than seven orders of magnitude higher than the current minimal estimated fenceline concentrations. Given PA's low systemic toxicity and low estimated fenceline concentrations, there is no reason why existing data are "insufficient" to conduct residual risk determinations.

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[42] Such a requirement is an obvious corollary to Congress' explicit recognition in TSCA of the need to balance environmental concerns against economic feasibility. See infra at 29, n.45.

B. The Proposed Testing For PA Is Not "Necessary" To Support EPA's Risk Management And Risk Assessment Functions

Similarly, EPA must also make a finding that the proposed testing is "necessary" to develop "such data," i.e., to develop the data necessary to predict the effects of the chemical on human health and the environment. TSCA Section 4(a)(1)(A)(iii), 4(a)(1)(B)(iii). However, the purpose of TSCA is not to create a perfect data base for every chemical. Rather, the purpose of TSCA is to regulate chemicals and chemical substances to prevent "unreasonable risk[s] of injury to human health and the environment." TSCA Section 1(a)(2). To that end, the statute states that it is the "policy" of TSCA to ensure that "adequate data [are] developed with respect to the effect of chemical substances and mixtures on health and the environment." TSCA Section 1(b)(1) (emphasis added).[43]

To accomplish these purposes in the testing context, therefore, EPA must show that the proposed testing is necessary not to provide a complete data set, but rather, to provide "adequate data" to conduct residual risk determinations.[44] For the reasons discussed above, such a finding simply cannot be made for PA. In addition, EPA did not attempt to conduct the necessary analysis. Instead, EPA merely stated in conclusory form that the proposed testing "is necessary to develop data" for the endpoints specified in the rule, and that this testing is needed generally to "determine if the manufacturing, processing, and use of phthalic anhydride does or does not present an unreasonable risk of injury to human health from inhalation exposure." EPA, TSCA Section 4 Findings for 21 Hazardous Air Pollutants at 74.

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43 Consistent with TSCA's goal of balancing the need to protect human health and the environment with economic feasibility, the statute further provides that any actions taken under TSCA should include consideration of the "environmental, economic and social impact" or those actions, and any authority should be exercised so as "not to impact unduly or create unnecessary economic barriers." TSCA Section 1(c) and (b)(3).

44 This interpretation is consistent with the House Committee Report, which specified that EPA must "eliminate unnecessary or duplicative testing." H.R. Rep. No. 1341 at 18.

EPA's conclusory statement cannot be considered a "finding" that each test proposed for PA is "necessary" to enable EPA to meet the requirements of the Clean Air Act. The Agency does not attempt to connect the required testing to the purposes for which the data will be used. For example, there is no explanation in the rulemaking record of why EPA believes the proposed immunotoxicity testing is necessary to provide "adequate data" to enable EPA to conduct a residual risk determination for PA. Because EPA has failed to make the findings required by the statute, the Agency has failed to provide adequate support for the proposed test rule.

C. EPA's "A" Finding For PA Is Not Adequately Justified

EPA purports to make a finding that the manufacture, processing, distribution, use or disposal of PA may present an unreasonable risk of injury to health, as required under TSCA Section 4(a)(1)(A) (the "A" finding). 61 Fed. Reg. at 33192. EPA bases its claim on a 1988 Amoco study in which rats exposed to 500 mg/m^3 PA experienced potential respiratory sensitization.[45] EPA has not adequately supported it purported "A" finding that PA may present an unreasonable risk of injury to human health. As explained further below, EPA has not complied with the requirement that it correlate PA's suspected toxicity with anticipated levels of exposure. EPA also has improperly used its "A" finding for one endpoint to require testing for other endpoints.

First, EPA has not attempted to correlate suspected toxicity with suspected exposure levels. However, a determination under Section 4(a)(1)(A) that a chemical "may present" an unreasonable risk depends on an analysis of human exposure to the substance and its potential toxicity. See Chemical Manufacturers Association v. EPA, 859 F.2d 977, 983 (D.C. Cir. 1988) ("the EHA[46] decision"). After reviewing the legislative history of TSCA, the court found that "Congress obviously intended Section 4 to empower EPA to issue a test rule only after it had found a solid 'basis for concern' by accumulating enough information to demonstrate a more-than-theoretical

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45 EPA, TSCA Section 4 Findings for 21 Hazardous Air Pollutants at 71.

46 2-ethylhexanoic acid.

basis for suspecting that an 'unreasonable risk' was involved in the use of the chemical." 859 F.2d at 986. Thus, as the court held, "[t]he statutory standard requires EPA to correlate the suspected toxicity of a substance with the suspected levels of exposure." Id. at 995 (emphasis added). Demonstrating such a relationship is a minimum requirement for a "may present" finding. According to the court, a Section 4 test rule "is warranted when there is a more-than-theoretical basis for suspecting that some amount of exposure occurs and that the substance is sufficiently toxic at that exposure level to present an 'unreasonable risk of injury to health.'" Id. (emphasis added).[47] EPA's testing proposal discusses each of these factors (exposure and toxicity) separately, but does not discuss them together, and does not provide sufficient information to determine the basis for EPA's finding of potential unreasonable risk for PA.

Specifically, EPA has failed to relate the PA exposure scenarios to its toxicological concerns, as required by the EHA decision. EPA has not explained why the respiratory sensitization, let alone any of the endpoints for which testing is proposed, is of concern in light of the reasonably anticipated duration, level and scope of human exposure to PA.[48] To fill this gap, EPA must expand its analysis of exposure and release scenarios and relate them to the specific toxicity concerns underlying its testing proposal for PA. Without this additional analysis, adequate support will be lacking for a finding that the test chemicals may present an unreasonable risk of injury under TSCA Section 4(a)(1)(A).

Second, EPA improperly seeks to escape its obligation to correlate the suspected toxicity of PA with the suspected level of exposure by claiming that once it has made an "A" finding for any toxicological endpoint, "EPA may require any type of health or environmental effects testing

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47 The mere fact that there has been a release does not necessarily mean that there has been any exposure. For example, substances may be rapidly dispersed, degraded or reacted such that even relatively large releases do not result in exposures.

48 As discussed at pp. 33-4, infra, potential issues about respiratory sensitization in exposed workers are not properly addressed in this test rule, which is intended to develop information to conduct residual risk determinations under the Clean Air Act.

necessary to address unanswered questions about the effects of the chemical substance" -- whether or not those tests are in any way related to the health effect endpoint for which the "A" finding was made.[49] Indeed, the "A" finding for phthalic anhydride is based on respiratory sensitization, yet EPA proposed to require testing for cancer, acute toxicity, subchronic toxicity, developmental toxicity, reproductive toxicity, neurotoxicity, immunotoxicity and respiratory sensory irritation. The one endpoint for which EPA is not requiring testing is respiratory sensitization -- the endpoint on which EPA bases its authority to require testing for phthalic anhydride.

EPA's approach is inconsistent with the language and intent of TSCA Section 4, and represents misguided policy.[50] It is clear that Congress did not intend an "A" finding under TSCA Section 4 for a single endpoint to give EPA carte blanche to require any kind of testing it may choose. That Congress did not intend to permit what EPA has done in this rulemaking is evidenced by the fact that Congress required EPA to make two additional findings, namely that the existing data are inadequate to determine whether the chemical may present an unreasonable risk, and that the proposed testing is necessary to make such a determination. For this statutory language to have any meaning at all, the testing to be required by EPA must be tied to the health effects endpoints which are of legitimate concern, i.e., those that "may present an unreasonable risk." EPA's contention that an "A" finding for one endpoint gives the Agency unrestricted license to address all "unanswered questions about the effects of the chemical substance" disregards the clear intent of Congress to strike a balance between the Agency's legitimate information needs and the obvious reality that the number of chemicals in commerce is great and testing resources are limited.

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49 EPA, TSCA Section 4 Findings for 21 Hazardous Air Pollutants, at 3.

50 EPA's failure to make meaningful findings concerning the "necessity" of testing for each endpoint or the "necessity" of each study for each of the compounds makes it all the more critical that the Agency develop appropriate and well-supported "A" and "B" findings.

D. EPA's "B" Finding Is Not Warranted For PA

The PA Task Group understands that CMA intends to address in its comments why the numeric criteria used to make EPA's "B" findings do not provide a meaningful basis for determining whether exposure is sufficient to warrant testing. A few points warrant mentioning specifically with regard to PA.

First, although EPA claims that there may be general population exposure to PA, it is clear that the Agency relied for its "B" finding exclusively on worker exposure.[51] Worker exposures, however, are unrelated to the purpose for which the testing is being required. The proposed test rule is first and foremost a HAP test rule. It is intended to generate data for evaluating residual risks, after the implementation of MACT standards, of air emissions to the general population. Accordingly, EPA's explanations about potential worker exposures simply are not relevant to support its proposed HAP test rule.

It is clear, however, that there is no general population exposure basis for making EPA's "B" finding. In spite of EPA's statement that there "may also be general population exposure to phthalic anhydride," the fact remains that even the releases reported on the 1995 and 1996 TRI are less than half of EPA's B policy cutoff. Moreover, EPA has previously acknowledged that exposure is the more relevant inquiry when evaluating residual risks. See 57 Fed. Reg. 2138, 2144 (January 17, 1992) (aryl phosphate base stocks test rule) (stating that EPA should require testing only where "human or environmental exposure is of such magnitude or type that [the chemical] may need to be regulated if test data reveal adverse effects."). Yet actual PA exposures clearly cannot be used to support EPA's proposed test rule. The maximum PA fenceline concentration (less than 1 ppb) is negligible and several orders of magnitude below concentrations that reasonably would be expected to produce systemic toxicity in humans.

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[51] See EPA, TSCA Section 4 Findings for 21 Hazardous Air Pollutants at 71-2.

Second, the Task Group believes that EPA has significantly overstated potential workplace exposures to PA. Fugitive PA at valves and sampling ports typically condenses into large macroscopic needles or flakes that can be seen on inspection and removed. PA producers also train workers on proper handling to minimize exposure to PA. Personnel at loading stations generally are required to use respiratory protection. Accordingly, workplace exposures to PA are expected to be minimal.

The minimal potential for worker exposure to PA is confirmed by results of air sampling at a Task Group member's facility. Air sampling was conducted to quantify worker exposure potentials associated with PA production processes, including oxidation, distillation, residue drumming, flake manufacture and loading of molten PA. Testing was conducted on various shifts between April 12-15, 1994.[52] Both area and personal samples were collected. All personal sample results were well below the OSHA Permissible Exposure limit of 12 mg/m^3. Approximately 90% of the sample results were below detectable limits. With the possible exception of the flaker operation, all conditions existent during the survey were considered representative of normal operations. Area samples also were all below detection limits. These results show that current worker exposures are quite low, such that EPA's "B" finding cannot be justified based on worker exposures, even if such exposures were relevant for purposes of this rulemaking.

CONCLUSION

For the reasons described above, and in comments previously submitted to the Agency, the Task Group believes that additional testing of PA is not necessary to support residual risk analyses under the Clean Air Act. The negligible potential for general population exposure to PA, coupled with existing studies conducted at extremely high doses, should be more than sufficient to enable EPA to assess residual risk. Moreover, the Task Group believes that EPA has not

adequately justified its purported "findings" that are required to support testing under TSCA Section 4. Accordingly, the Task Group believes that if any testing is to be conducted on PA as part of this testing initiative, it should consist only of the testing identified in the Task Group's alternative testing proposal.

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[52] Samples were collected by drawing ambient air through a 37 mm mixed cellulose ester membrane filter at a flow rate of approximately 2 liters per minute. Filters were analyzed by a NIOSH Method utilizing HPLC equipped with a 254 nm UV detector.