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February 23, 1976

Emission Standards and Engineering Division
Environmental Protection Agency
Research Triangle Park, North Carolina 27711

Attention: Mr. Don R. Goodwin

Re: ~~Proposed Standard for Vinyl Chloride~~

Dear Mr. Goodwin:

Air Products and Chemicals, Inc. submits herewith its comments and views on the Proposed Standard for Vinyl Chloride as published by the Environmental Protection Agency at 40 FR 59532. In summary, we do not believe that the Agency has adequate grounds for declaring that vinyl chloride is a hazardous ~~air pollutant~~, or for proceeding under Section 112 of the Clean Air Act. We urge that rulemaking proceed under Sections 109 and 111 of the Act.

Our reasoning and supporting data are given in the attached documents, which are arranged in the following order.

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It is the purpose of these comments to correct the record and assist the Administrator in his consideration of our request. We also incorporate by reference the submittals of the Technical, Economic, and Health Effects subcommittees of the VCM and PVC Producers Group of the Society of the Plastics Industry on this matter.

We are anxious to assist the Administrator and his staff in any way we can during their consideration of this important standard. Please feel free to call on us for whatever help we may provide.

Very truly yours,

Richard Fleming
Richard Fleming

/mjv

Enclosure

I. LEGAL POSITION

A. Introduction and Reservation of Rights

Air Products and Chemicals, Inc. ("Air Products") submits herewith its comments and views on (a) the Administrator's determination that vinyl chloride is a Hazardous Air Pollutant as defined in Section 112(a) (1) of the Clean Air Act (the "Act") and (b) the Administrator's Proposed Standard for Vinyl Chloride. In submitting such comments, Air Products is attempting to inform the Administrator of certain errors, omissions, misstatements, misjudgments and faulty reasoning contained in the Proposed Standard itself and in the Preamble, the Scientific Technical Assessment Report ("STAR"), the Quantitative Risk Assessment ("Risk Assessment") and the Standard Support and Environmental Impact Statement ("Standard Support"). Nothing contained in these comments should be construed as a waiver by Air Products' of its rights under the Act to petition for judicial review of the standard finally adopted, whether or not such final standard incorporates some or all of the comments contained herein, and Air Products expressly reserves all such rights.

B. Summary

As will be more fully set forth below, Air Products believes that at current or reasonably expected ambient concentrations of vinyl chloride in the vicinity of establishments manufacturing or handling this substance, vinyl

chloride monomer is not a hazardous air pollutant. Air Products believes further that the vinyl chloride emission standard is improper and unnecessary in its proposed form. We concede that some form of vinyl chloride emission standard may be desirable to insure that the current safe ambient concentrations are not exceeded, but we believe that the EPA documents supporting the proposed standard, and the proposed standard itself, are seriously flawed. We do not believe that the Administrator has been presented with all the available relevant information on the health effects of vinyl chloride; nor do we believe that the information that has been presented by the EPA Staff has been prepared in an objective and balanced manner; nor that the conclusions drawn and recommendations made in these documents are sound.

Although we have strong objections to many of the conclusions reached in the documents supporting the proposed standard, we nevertheless commend the Agency Staff for its receptivity to technical inputs from all interested parties during the development of the proposed standard. We sincerely hope that the points made in these Comments will be equally well received.

We also commend the Agency for acknowledging the necessity of and publishing the Risk Assessment Document. While we may disagree with its conclusions, we think that the publication of a document assessing the risks of exposure to air pollutants

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at varying levels is essential to the standard setting process. We urge the Agency to publish a risk assessment in connection with each of its subsequent rule makings.

Thirdly, we support the Agency's use of cost/benefit analyses in determining the type and degree of pollution abatement technology to impose. The concept of cost/benefit analysis is sound, although the Agency has failed to apply it precisely in this rule making.

Decisions of the magnitude of a determination of a hazardous air pollutant should be made with great care, and should be adequately supported by rigorous evaluation of all of the available data. A fair evaluation of the voluminous material published by EPA in support of its determination and the proposed standard leaves one with the uneasy impression of a decision in search of a rationale rather than a disciplined exercise in rational decision making.

There are many technical flaws in the proposed standard itself, and these will be commented on in detail in the written comments of the technical committee of the VCM/PVC Producer's Group of the Society of the Plastics Industry ("SPI"). Air Products concurs in SPI's technical comments, and urges the Administrator to incorporate them in the final standard.

Air Products also concurs in, and incorporates herein by reference, the line-by-line critique of the supporting

documents, as well as of the proposed standard, as set forth in the SPI presentation.

Air Products objects to those parts of the proposed standard which it considers to be design or work practice standards rather than an emissions standard. These objections are more fully set forth in Part I F of these Comments.

We therefore respectfully request that the Administrator set aside his determination that vinyl chloride is a hazardous air pollutant and proceed to promulgate an appropriate standard under Sections 109 or 111 of the Clean Air Act.

Detailed technical reasoning supporting this request is given below.

C. Vinyl Chloride is Not a Hazardous Air Pollutant

Section 112(a)(1) of the Act defines a hazardous air pollutant as an air pollutant "which in the judgment of the Administrator may cause, or contribute to, an increase in mortality or an increase in serious irreversible, or incapacating reversible, illness." In the exercise of the foregoing judgment, the Administrator must necessarily use as his frame of reference some level of ambient concentration of the air pollutant under consideration. Were this not so, every air pollutant would fit the definition since every air pollutant is potentially hazardous at some level.

According to EPA's calculations, as set forth in the Risk Assessment, the five mile radius average annual community exposure to vinyl chloride monomer is 17 ppb. As

presented in more detail in our Part III D Comments herein on the Risk Assessment document, the data and assumptions employed to arrive at the 17 ppb figure are obsolete and inaccurate. Analysis of EPA's more recent monitoring data shows that current five mile radius average annual community exposure to vinyl chloride is less than 1 ppb. We believe that at either level, but especially at a level less than 1 ppb, vinyl chloride monomer is not a hazardous air pollutant. Exposure at this level will neither cause nor contribute to any discernible "increase in mortality or serious irreversible, or incapacitating reversible, illness". In fact, the proposed standard itself is based on a similar risk assessment.

The conclusion that vinyl chloride is not a hazardous air pollutant is further buttressed by the conclusion reached by EPA itself in Appendix E of the Risk Assessment document where it is stated that EPA has found "no evidence that living around vinyl chloride plants is a risk factor in the occurrence of liver angiosarcoma". In addition, Dr. Wm. Marcus stated at the EPA hearing on vinyl chloride in Washington on February 3, 1976, ".... to date we do not have any evidence that angiosarcoma has been produced by vinyl chloride in the general population". (Transcript of hearing, page 42.) We find further support for this conclusion in the CDC finding of no confirmed cases of liver angiosarcoma among persons who were not occupationally

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exposed to long term high level concentrations of vinyl chloride. This is so, in spite of the fact that historical emissions of these plants, some in existence for more than 25 years, have been substantially higher than current levels. Thus, we conclude that vinyl chloride is not a hazardous air pollutant within the meaning of Section 112 of the Clean Air Act.

D. The Proposed Emission Standard is Unnecessary to Provide an Ample Margin of Safety to Protect the Public Health

If we accept, for the sake of argument, that the Administrator acted correctly in designating vinyl chloride a hazardous air pollutant, Section 112(b)(1)(B) requires him to establish an emission standard "at the level which in his judgment provides an ample margin of safety to protect the public health from such hazardous air pollutant."

We submit that the proposed standard is unnecessary because the margin of safety which the Administrator has implicitly accepted as ample has already been achieved by industry as a result of the OSHA standard for vinyl chloride and industry's increasing awareness and understanding of the health hazard of high level exposure to vinyl chloride.

The Preamble to the proposed standard states that implementation of the standard would result in a 95% reduction in vinyl chloride emissions from vinyl chloride and polyvinyl chloride manufacturing plants. EPA has also

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stated that the five mile radius average annual community exposure to vinyl chloride is 17 ppb. If the Administrator has correctly interpreted and discharged his duty under Section 112(b)(1)(B) in formulating the proposed standard, the public health would be protected with an ample margin of safety if the five mile radius average annual exposure were reduced to .85 ppb.

A fair evaluation and analysis of more recent monitoring data compiled by EPA shows that ambient concentrations are already at or below this goal, as the result of extensive and expensive abatement efforts of the plants involved. See STAR, Chapter 6.

The Risk Assessment document predicts that at an assumed five mile average annual community exposure of 17 ppb, from less than one to ten persons per year would contract liver angiosarcoma from vinyl chloride, and that an equal number of persons would contract tumors at other sites. For the reasons set forth in Part III D of these Comments, a consideration of the facts leads to the conclusion that these predictions are incorrect, and that the proposed standard is not necessary.

- E. Although the Proposed Standard is Neither Necessary nor Proper, Some Form of Emission Standard may be Appropriate to Insure that the Currently Safe Ambient Levels of Vinyl Chloride are Maintained

Although designation of vinyl chloride as a hazardous air pollutant is improper, we concede that public concern

over vinyl chloride requires that some form of emission standard be adopted by the Administrator. Although the Preamble asserts that Section 109 treatment of vinyl chloride would be inappropriate "because vinyl chloride is a localized problem and Section 109 is usually more appropriate for regulating pollutants whose presence in the ambient air is ubiquitous," we submit that the absence of clustering of incidences of liver angiosarcoma demonstrates (Risk Assessment document at Appendix E.) that the problem is not localized. We note, moreover, the recent joint publication of the American Health Foundation and the National Cancer Institute which suggests that vinyl chloride may well be ubiquitous in the ambient air because of the combustion of vegetation.

The Administrator has implicitly accepted that an ambient concentration not exceeding 1 ppb vinyl chloride is sufficient to provide an ample margin of safety to the public health, and we believe that a national primary ambient air standard under Section 109 to this effect would properly protect the public.

We would also support an appropriate new source standard for vinyl chloride under Section 111.

Alternatively, if the Administrator concludes that he cannot rescind his determination that vinyl chloride is a hazardous air pollutant, we would suggest that he modify the proposed vinyl chloride emission standard to permit

owners or operators of vinyl chloride and polyvinyl chloride manufacturing facilities to meet an ambient concentration limit on vinyl chloride in the vicinity of such facilities. The National Emission Standard for Beryllium (40 CFR 61.30 et. seq.) provides a precedent for such action at Section 61.32(b).

We believe that an alternative emissions standard based on ambient concentrations in the vicinity of vinyl chloride and polyvinyl chloride plants would be desirable for the following reasons:

The real concern in regulating any pollutant, but especially a gaseous, non-persistent pollutant with a short half life, should not be the concentration in some vent stream or the stack gas. The real concern should be with the concentration in the neighborhood. The community exposure, and the risks resulting from such exposure are the pertinent questions.

Vinyl chloride is a gas, is not persistent, has a half life of about six hours in sunlight, and does not bio-accumulate. Accordingly, if ambient concentrations of vinyl chloride are at acceptable levels in the vicinity of the plant, diffusion and degradation will adequately protect those persons living farther from the plants.

Vinyl chloride, unlike the present hazardous air pollutants is easily measured by gas chromatography. As noted in the STAR Document, the sensitivity of current monitoring equipment approaches .5 ppb. Regulation though enforcement

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of neighborhood ambient concentrations is both feasible and objective, and would give industry the flexibility to use innovative control techniques rather than be bound to the specific equipment and procedures set forth in Section 61.65 of the proposed standard.

The Risk Assessment Document, as corrected by our Comments in Part III D hereof, provides a basis upon which the Administrator can select an appropriate ambient concentration for the suggested alternative emission standard.

F. Certain Portions of the Proposed Standard are not Emission Standards and are therefore Invalid

Certain portions of the proposed standard are invalid because they establish design, process or work practice standards, and fail to set the kind of numerical emission standard required by Section 112 of the Act.

Six lower court decisions have addressed the question of what constitutes an emission standard in the context of the National Emission Standard for Asbestos (40 CFR 61.20 et. seq.); five of the six have ruled against the Agency's position. In U.S. v. Adamo Wrecking Co., CR No. 5-80297 (E.D. Mich. 6/6/75) the court held that "an emission standard ... must establish a set, measurable level of emissions...{A} Methodology for emission control is not a 'standard'...." Slip. Op. at p.31. The Agency's argument in support of the asbestos standard was based in part on the difficulty of measuring asbestos emissions; that argument has no validity in

the context of an emission standard for a substance like vinyl chloride which is easily measured.

Although some of the design, process or work practice standards which we object to are couched in terms of numerical limitations, we believe that the Agency has exalted form over substance, and that the purported emission standard is unauthorized by Section 112, and therefore invalid.

The particular portions of the proposed standard to which we object on this ground are the following subparagraphs of Subsection 61.65(b):

- (1) Loading and unloading lines
- (3) Leakage from pump, compressor and agitator seals
- (4) Leakage from relief valves
- (5) Manual venting of gases
- (6) Opening of equipment
- (7) Sample flask
- (8) Leak detection and elimination.

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II. SUMMARY OF TECHNICAL DISCUSSION

The Agency has presented a number of assumptions and conclusions in the documents associated with this action which are critical to the promulgation of the proposed standard. We have attempted to follow the reasoning of the Agency as it is presented in the documents, and have commented on each of these salient points.

- A. Vinyl chloride is a carcinogen at high exposures for long periods of time.

This is true, certainly in animals, and probably in humans, but the Agency has failed in its extrapolations to consider adequately the most appropriate biological models and the existence of a sharply decreasing response rate at low concentrations.

- B. Cases of angiosarcoma in humans have resulted from exposures below those found in operations handling vinyl chloride.

This critical, but totally erroneous statement, is found in several of the documents. The fact is that there has been no confirmed case of liver angiosarcoma in humans related to vinyl chloride exposure below that found in plants handling this substance.

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No fabricator employees, no neighbors of vinyl chloride or polyvinyl chloride plants, nor any workers not directly exposed to vinyl chloride over 160 ppm have been found to have any adverse health effects of any kind due to vinyl chloride exposure. This is confirmed in the Risk Assessment Document, and by a statement by Dr. Marcus at the EPA hearing in Washington on February 3, 1976.

- C. Industry emissions are four percent of the vinyl chloride handled.

This estimate is contrary to the data presented to EPA and the conclusions reached by the Agency's own contractor for the survey. At best it applies only to suspension and emulsion operations, and not to other processes. It is arrived at by an arbitrary inclusion of losses of solid PVC as if this material were vinyl chloride vapor. The monitoring data taken by the Agency do not support estimated emissions at this rate, and no cognizance is taken of the reduction in emissions which have been made in the year and a half since the estimate was made.

- D. Ambient concentrations pose a significant risk to the public.

The Agency has attempted to minimize the significance of its own monitoring results which show lower than predicted ambient concentrations, and has published a Risk Assessment that greatly exaggerates the significance and precision of available medical evidence. The fact is that there is no demonstrable risk to the populace, even at past higher concentrations, as is stated in Appendix E of the Risk Assessment Document.

- E. Vinyl chloride is therefore a hazardous air pollutant.

There is no evidence to support this conclusion, nor can a reasonable case be made to predict that any discernable harm to persons in the community would occur because of current levels of vinyl chloride emissions.

- F. It is not possible to establish a safe exposure.

The Administrator has rejected the need for a zero risk, and has accepted the safety resulting from an average 1 ppb exposure. The risk imputed to that concentration has been overstated substantially and it is clear that the risk at that concentration is much less than that stated, thus providing an ample margin of safety.

G. Section 112 must be used for uniformity and speed of response.

Adequate control of ambient concentrations is possible, and indeed even more practical, under other sections of the Clean Air Act. The Agency's desire for undue haste is difficult to understand in view of the time which has elapsed since the problem surfaced, and the responsible actions taken by industry in substantially reducing emissions while EPA has been studying the matter.

Therefore, we do not believe that the Administrator has sufficient grounds for the proposed action, and we urge that the declaration of vinyl chloride as a hazardous air pollutant be rescinded, and rulemaking proceed under Section 109 or 111. The detailed comments on the documents which follow are to correct the record and to assist the Administrator in his consideration of our request.

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III. COMMENTS ON THE DOCUMENTS

A. Introduction

A matter of major concern to Air Products is the quality of the documents prepared to support the proposed standard and the methods used to reach the conclusion that such a standard is necessary. These documents suffer from three primary faults: obsolete data; errors in the mathematics involved; and unwarranted and unsubstantiated conclusions. We will comment further on each of these points, and give examples of each type in our comments on each of the documents.

In addition, there are a number of inconsistencies between the various documents, especially noticeable in comparisons with the STAR Document. Doubt is created as to the real understanding of the Agency and the soundness of the rationale for its action because of the variance between the documents.

The Agency has based much of its justification for a standard on the 1974 industry data. These requests for information were made at a time when full attention was being paid to OSHA problems, and replies were made hurriedly from a combination of estimates and spot measurements. Industry's interpretation of the questions varied greatly from company to company, and the replies were inconsistent. The Agency unilaterally and quite incorrectly included

estimated losses of solid PVC as fugitive gas emissions, against the advice of industry and the Agency's contractor and in violation of scientific principal. Subsequent data supplied by industry, either voluntarily or in response to Section 114 requests sent to the dispersion resin sector of the industry, have not been included.

Similarly, medical, epidemiological, and technical data which have developed since very early in 1975 are not considered. The dates of the references in the documents indicate that input into the standard setting process stopped in the Spring of 1975, despite the great fund of knowledge that has become available since then.

In this connection, the Agency has not been diligent in developing a data base of its own on possible human effects, but instead has relied on data supplied by others. No substantial epidemiological, medical, or experimental studies were undertaken by the Agency, despite the fact that only the government can gain maximum access to the necessary data, and despite urging by many interested parties. In view of this, and the nearly two-year period since regulatory interest arose in vinyl chloride, it is difficult to understand why Section 112 action is now said to be justified on the basis of the Agency's urgency to act quickly. The refusal to postpone a hasty hearing on these ill-drawn documents is even more incomprehensible on any but political grounds.

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The number of mathematical errors that have been found seriously damage the credibility of the documents, especially the Standard Support Document. Incorrect conversion factors for liters and gallons are used throughout. A fundamental error in calculation results in a proposal for reactor opening losses only 50% of that intended. Misplaced decimal points and references to incorrect tables or pages are abundant. Such of these as have been found are corrected in the detailed comments submitted by SPI.

A tendency to exaggerate and select data to fit the wishes of the writer permeates the Preface, the Standard Support Document, the STAR Document and the Risk Assessment Document. As an example, the Preamble states that the results of the extensive environmental test program at three plants "conducted ... from November 1974 to June 1975 are not discussed in detail because they are still being analyzed." The STAR Document, Section 4.2.6, dated June 1975 contains a brief summary of a part of this test program, and these data show that the concentrations found at that time are less than those predicted to prevail after the proposed standard is implemented. The arithmetic average of 1,903 24-hour measurements was 5 ppb, and no sample taken 1,000 m or more from the source was above 0.1 ppm. Furthermore, no EPA sampling data shows detectable vinyl chloride at distances more than 3 miles from a VC or PVC plant.

The Preface and the Support Document continue to refer

to non-occupational cases of angiosarcoma even though the CDC angiosarcoma case list has now dropped all but the occupational cases for lack of any supporting evidence linking any non-occupational case to vinyl chloride exposure.

The "reasonable extrapolations" and assumptions made in one section of the documents became fact in another section, and then were used to demonstrate the need for a standard, with no admission made of the tenuous line of reasoning which led to the judgment. The tone is that of a decision looking for support, rather than a demonstrated progression of facts leading to a decision.

An example of this is found in tracing the so-called non-polymerization liver angiosarcoma cases. The Preamble states: "These cases suggest that exposure to vinyl chloride at lower levels than usually encountered at PVC plants is capable of causing liver angiosarcoma." (40 FR 59532).

This is a direct quote from the Standard Support Document, pages 2-3, which refers to the STAR Document. There, on page 72, Section 6.3.1 it is said that "...one of the confirmed cases involved an accountant employed at a vinyl cloth plant. The accountant is presumed to have had a lower level of exposure than the PVC worker."

The "accountant" is case U.S. 15 of Table 6.18, and also case 5 of Table 6.19, where the footnote says, "The diagnosis of this case is uncertain." As a matter of fact, all of the six so-called community cases of Connecticut are

rated as uncertain or different from PVC production workers, and NIOSH has dropped its listing of these persons in its November 1975 listing (10) of angiosarcomas.

Thus eliminating the one U.S. case, we look at three foreign cases, which have also been dropped by NIOSH. Case W. Germany 03 was a filler of aerosol cans, where the leakage is thought to have been severe, and who, therefore, experienced a high exposure. Case Italy 01 is shown to have had as the primary site the pericardium. Case Sweden 02 is shown to be a vinyl chloride production worker in an obsolete acetylene process where the exposure was also high.

Therefore, none of these were confirmed cases or support the statement on page 1, Section 1.1 of the STAR document that there should be a basis for concern for workers exposed to "levels of exposure as low as 1 to 10 ppm"

This unsupported trail of erroneous assumptions is at the heart of the Agency's statement that, "Reasonable extrapolations from these findings cause concern..." (Standard Support Document, page 7.1). It is apparent that the Agency does have the proper information on these cases. Appendix E of the Risk Assessment Document states that there is no risk to living near vinyl chloride (sic) plants, and at the EPA hearing on February 3, 1976, one of the panelists (Dr. Marcus), stated that there is no evidence of angiosarcoma in the general population from vinyl chloride exposure. Yet,

erroneous and unfounded statements relating angiosarcoma cases to neighborhood exposure appear in all of the documents in this case.

Regulatory agencies must be impartial and dispassionate as well as technically correct in their decision making. For these reasons it is distressing to find that documents of critical health, social and economic importance are based on a biased sampling from available information, and unsound and flawed reasoning, even with that information.

B. Comments on the Preamble and Standard

The Administrator does not have sufficient grounds on which to declare vinyl chloride a hazardous air pollutant, nor does he have authority under Section 112 to require specific equipment and procedures as methods of abatement. The data and reasoning leading to the promulgation of this standard are flawed, as is the standard itself. We shall present comments on each of these points, and make suggestions for alternative actions.

Section 112 of the Clean Air Act states that a hazardous air pollutant is "an air pollutant ... which in the judgment of the Administrator may cause or contribute to an increase in serious irreversible, or incapacitating reversible, illness." Congress clearly gave wide latitude for the decision-making process, but it is obvious that there must be reasonable grounds for such a decision. By a long chain of "suggestions" and "reasonable extrapolations" the writers of those docu-

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ments have moved from the knowledge of angiosarcoma in workers exposed to very high concentrations by the standards of today, for long periods of time, to a hazard to those exposed to exceedingly low concentrations.

There are many substances in everyday life which can cause death or serious illness. Even those basic requirements for life, oxygen, water, salt, and carbon dioxide can be hazardous at very high concentrations. Therefore, the question of at what concentration a material can be deemed hazardous becomes paramount.

The statement is made that "(W)e are unable to establish a level that presents no public health risk." However, there are many data available that can help in understanding the extent of the problem and the risks involved. Many companies presented results of studies of their own work forces during the OSHA hearings that showed no ill effects from many years of exposure to concentrations far in excess of those found in the work environment today, and thus several orders of magnitude greater than that in the general environment outside the plant. We believe that appropriate recognition and weight should be given to a series of carefully designed and executed metabolic studies which examine the fate of absorbed VCM at various levels of exposure (26). These studies demonstrate that at least two biological mechanisms exist for "handling" VCM. One mechanism is the "normal" mechanism capable of dealing with metabolic loads equivalent

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to an exposure of below 50 ppm in rats. The second mechanism becomes prominent only when the capabilities of the first or "normal" mechanism are saturated or overloaded. We believe the evidence is persuasive that this second mechanism is the one which leads to metabolic species which are relevant to the induction of cancer. Since this latter mechanism is only weakly, if at all, operative in rats at levels of exposure less than 50 ppm, the slope of the dose-response increases sharply at levels below this. Consequently, any extrapolation to doses below 50 ppm should incorporate appropriately these metabolic considerations into the model selected. Included in several of the other studies, notably that of Union Carbide (17), were reports of examination of control groups made up of employees from adjacent operations. Whatever the concentrations were in the actual PVC operations, it is clear that other workers in the same complex would have been exposed to higher concentrations more consistently than would have been the more distant residential occupants. A recent (but still unreported) study of a PVC operation in Pennsylvania (21), conducted jointly by labor, government and the plant operator, showed no difference in the disease rate of PVC and non-PVC employees. This latter study does suggest the possibility of a higher fetal wastage rate in the wives of PVC workers, based on interviews with the workers, but there are several questions on the methodology and statistical significance of this survey which must be

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answered before its import can be evaluated (20). A British survey of 2,100 workers with up to 27 years of exposure (4) did not find evidence of adverse health effects.

The Agency has shown no interest in pursuing these and other studies which are available to help elucidate the long-term human response to low concentrations of vinyl chloride. These data are available now, not after "many years," as stated in the Preamble. The Agency has not followed its own suggestion for participation in recommended studies (1, p. 229).

We are constantly exposed to potential carcinogens, but the natural repair and rejection mechanisms of our body protect us so long as we do not receive a dosage beyond their capabilities. The elegant work by Dow chemists (1, p. 135) (25) has confirmed that these mechanisms extend to the metabolism of vinyl chloride. Dr. Paul Kotin (8) discussed this principle extensively in a recent presentation before the New York Academy of Science Conference on Occupational Carcinogenesis. A similar position was taken by Dr. Leo Friedman and a WHO Scientific Group in WHO Publication No. 546 (9), and the Cancer Advisory Board (11) has stated that dose response is inherent in the definition of a carcinogen. (See also references 12, 13 and 16.)

The concept of reduced risk at low exposures cannot be dismissed summarily, and the total lack of any substantiated evidence that any human has ever suffered harm from exposures below 160 ppm vinyl chloride cannot be ignored.

The Agency has continued to emphasize obsolete data on

environmental concentrations, and gives great publicity to the one, single, value of 33 ppm measured in the Spring of 1974. This was taken during a "quick survey", on "very short notice, and with little equipment and planning", and the results were "less than satisfactory", (2). It dismisses the data of thousands of monitoring results taken between November of 1974 and May of 1975 with the statement that "they are still being analyzed." Can one conceive of the EPA permitting industry to refuse to submit year-old data on similar grounds? The proposed standard would require that monitoring results be reported within 10 days after they are obtained. The truth is that these results show the majority of the samples were below the detection level. The overall average for points outside the plants was less than 0.005 ppm, with no value over 0.5 ppm, and the maximum value more than 1,000 m from the plants was 0.1 ppm.

These values are far below those which were projected by the Agency's diffusion studies for when industry is in compliance with the proposed standard, and below the assumed average of 0.017 ppm for all inhabitants of a 5-mile radius. Further progress has been made by the industry in reducing emissions in the year since these data were taken, and this clearly shows that the basis on which the standard was developed is incorrect.

No account has been taken of this substantial reduction in emissions that have resulted from industry reaction to

th health hazard of vinyl chloride. The standard development process has relied extensively on the "Section 114" responses which industry made in May of 1974. This was in a period when we were just learning how to detect and measure vinyl chloride in the low concentrations under concern, and before the action programs had become effective. Industry informed the Agency at that time that it estimated that much of the unaccountable losses were in the form of PVC solids to waste, but the Agency chose to ignore this information, and count these as vinyl chloride emissions to the atmosphere (29). The Agency's own consultant has advised them that this is incorrect.

In addition, the best information available today indicates that gaseous emissions now are less than half those estimated two years ago, and in many cases are substantially less. Furthermore, many companies, Air Products included, are now in the midst of massive projects to reduce emissions even further. No cognizance has been taken of these facts, and a principle reason these effects are "uncertain" is that the Agency has made no effort to measure them.

The Preamble speaks disparagingly of the ventilation projects used to reduce worker exposure. It is apparent that these efforts have not increased the net amount of emissions, and if they do have any effect, it is to disperse better these emissions, and thus reduce potential exposures outside the plant building as well as inside.

Further evidence that the Agency is working with incomplete data is presented in the statements regarding plant shutdowns as the result of this standard. It is said that four plants employing 30 persons may be affected. The number of 30 persons was arrived at by a straight ratioing of overall capacity and employment, ignoring the necessity of a minimum staff at each plant site. One of these plants has been shut down since January 1975. We have been informed that the other three plants will not be closed for this reason. However, in October of 1975 Uniroyal announced that its plant, employing 230 persons, will close because of the burden of regulatory costs. Union Carbide has announced that one of its plants will close at the end of the year. Others undoubtedly will follow.

As another example, the Preamble states (40 FR 59543) that studies show that PVC "may possibly" cause pneumoconiosis. Stiles and Wilson (3) clearly demonstrated no such effect from PVC dust.

We support the SPI submittal of technical comments on the standard, and urge that the Agency consider carefully the requests for changes contained in that document. These represent the best thinking of scientists knowledgeable in the field, and are intended to improve the accuracy, clarity and effectiveness of the standard. We would emphasize several points in that presentation. The reporting requirements are unduly burdensome, and will only inundate the

Agency with paper, rather than help it observe compliance. The suggested changes will give the Agency adequate information in a clearer form. Many provisions of the proposal exceed the Agency's power, and infringe on the realm of other governmental groups, OSHA, the Coast Guard, etc. These should be revised or deleted. An extremely simplistic approach is taken toward the economic effects of the proposed standard in assuming a simple pass-through of the costs of compliance. The major macroeconomic models normally show an end effect two or three times the direct cost to an industry. In addition, the overall costs are understated. The right to use equivalent methods should be indicated clearly in the standard, without reliance on reference to the Preamble. This is especially true in the area of analytical methods, where no two laboratories ever follow precisely the same techniques, and in methods used to reduce fugitive emissions. Insistence on a single method discourages innovation in the development of improved technology.

However, we still object to the promulgation of a standard at this time and in this manner. We do not believe the data justify the declaration that vinyl chloride is a hazardous air pollutant within the meaning of Section 112. Despite the efforts of the Agency Staff to interpret the data in the most pessimistic fashion, there is no evidence of harm to the general population. There is a tremendous amount of data available now that has not been evaluated, and many

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studies are now in progress. Several of these will be complete in the next few months, others by 1977. The Agency should proceed under Sections 109 and 111 to formulate a standard which is based on the best, and most current facts.

Air Products has every reason to support the basic premise of a healthful environment. Its speed of response to the knowledge of a problem with vinyl chloride is evidence of this. We do object to a standard developed without proper regard for the facts, using only partial data, and in undue haste.

C. Comments on the Standard Support Document

The Technical Committee of SPI has prepared a detailed critique of this document. Air Products participated in this work, and supports that portion of the SPI submittal. We ask that this section be incorporated by reference into this submittal (23), as well as the supplementary comments by the SPI Economics Committee.

We would comment on the attitude of the Standards Development Group during the early stages of the proceedings. Their attitude of openness and their desire for a free exchange of information with all concerned parties is a refreshing departure from the closed regulatory proceedings which have prevailed widely heretofore. That we are not satisfied with the final outcome is apparent from our comments, but we wish to encourage the pattern of open deliberation with full opportunity for examination of all available data. Such

practice should lead to a more competent rulemaking and more credible and understandable decision.

It is encouraging to see that the concept of cost/benefit analysis has been broached, and we encourage its use in all proposed rule making. It has not been used to the extent that it should, however. For example, by the combined use of Tables 3.6 and 7.8 in the Standard Support Document it can be seen that 35% of the proposed capital expenditure for a model suspension plant and 18% of the annual operating costs are to be used to reduce the emissions by only 3.6%, and therefore clearly is an example of "grossly disproportionate costs". Item 3 of Table 7.8 proposes the use of a gasholder and water purge system to reduce the emissions from relief valve discharges and reactor openings. The SPI submittal contains a detailed explanation of why it is unsafe to connect the discharge of a primary safety device to a closed system, and points out other ways to obtain reduced emissions without endangering life and property. In addition, the changes required in item 2 of this same table already offer ample protection. This leaves only reactor opening as a possible source for abatement by the costly additions to a plant.

It is not clear from the discussion in Section 4.9 of this document just what decision was reached on reactor emissions since no statement is made as to the final conclusion, but from Section 4.12.5 and Table 7.8 it is assumed that the gasholder was chosen. Substantially the same

effect can be obtained by the use of vacuum pumps, which can reduce the internal pressure well below 100mm Hg. absolute, to give a reduction of vinyl chloride vapor in the reactor of at least 87%. An expenditure of over \$1.5MM per plant is an excessive price to pay for the additional 12% reduction of an estimated loss of 3.6% of the total.

We therefore urge that this requirement be eliminated. The cost for each pound of vinyl chloride abated by this method is more than \$1.50, or about twelve times the cost by the other abatement steps.

Similarly, it can be seen from Table 7.12 that the estimated cost for double mechanical seals is 46% of the total capital and 32% of the annual cost of all fugitive emissions. Properly installed single seals will provide as good mechanical reliability, and in connection with the area monitoring system, adequate abatement, at a fraction of the cost.

The entire program should be evaluated in terms of benefits gained and the practical value of the abatement. Further work with suppliers and experienced cost estimators can improve the accuracy of the costs, and assure that proper means of abatement have been chosen.

D. Comments on the Quantitative Risk Assessment Document

A careful evaluation of the risks associated with a substance undergoing review for a standard-setting procedure is a necessary and proper act. We are glad to see that the

Agency has attempt d to do so in this case, even though the appearance of the Risk Assessment Document came late in the proceedings, and we urge it to do so in every case in the future. However, we have many problems with the example in hand. Because it has been widely quoted, both by The Administrator and the media, and because it is the source of the statement, "between ... one to twenty cases of cancer per year" (the modifying phrase "less than" is usually omitted) can be expected in residents, in addition to several oth r unsupported conclusions, we have prepared a detailed analysis of this document.

The authors list on pages 3-5 of this document the five assumptions made in order to estimate the health effects of community exposure to vinyl chloride. Each of these is discussed, but not in that order of presentation.

First, Appendix D contains the methods used to arrive at the incidence of angiosarcoma in exposed workers. The authors reject the data of the Tabershaw report (24) and choose the two studies that give the highest incident. All of the reports cited other than Tabershaw are selected subsets of that study, specifically selected so as to contain the highest possible number of cases. The selected reports included all known cases at that time and thus ignore all of the plants where no cases occurred. There are no grounds for disregarding the low exposure categories in Tabershaw unless their exposures were either below an effective threshold

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concentration, or below the concentration expected for the community. Failing both these criteria, these categories should be included and would reduce the incidence for angiosarcoma of the liver from the 3.3×10^{-3} given to about 1×10^{-4} , more than a thirty-fold reduction. The incidence thus derived is not supportable, particularly when data from several types of studies are mixed in the derivation. For example, the otherwise ignored Tabershaw-Cooper study was used to estimate time of exposure, and an estimate of the exposure concentration was taken from the Dow study. Dow is known to have had one of the best industrial hygiene programs in the industry, and therefore, a lower than average exposure, as acknowledged by the authors. This same study showed no angiosarcoma, but was rejected in other documents by the Agency as "too small", even though the cohort it studied was larger than any of the cohorts that were used. This is but another example of "selected" data. However, it is not at all clear how this value is used in arriving at the final answer. It seems probable from data available now about reactor purging and from the reported incidence of acute effects that the exposures in the time period of concern were well above 500 ppm, and often in the thousands of parts per million, for reactor cleaners. See reference 2 of Chapter 5 of the STAR Document, and Section 6.1.4 of the same document.

On page D7 the authors again ignore the industry-wide study to arrive at incidence of cancer at other sites and choose only one of the special subsets to derive this number. The statistical relevance of 161 workers in the plant with the highest incidence to over 8,000 workers throughout industry is difficult to rationalize. The other studies were ignored because they did not show any cases! This careful selection of supporting data destroys the argument, as does the assumption that all cancers encountered in the study were caused by vinyl chloride.

The animal data are incorrectly interpreted also. The zymbal gland is unique to rodents, and is especially susceptible to tumors. See Section 6.1.4 of the STAR Document. Excluding the zymbal and angiosarcoma cases in Maltoni experiment BT1 (1), there were 34 tumors in the three groups made up of 177 animals exposed to 50, 250 and 500 ppm (the ranges chosen by the authors) compared to 9 tumors in the 59 control animals. The incidence of tumors at other sites for animals exposed to 250 and 500 ppm was approximately 33% above that for animals exposed to 50 ppm or the controls (significant at $P = 0.05$) but there was no significant difference in tumors at other sites between the controls and those exposed at 50 ppm. This small difference does not support a conclusion, or even an assumption, that the incidence of other tumors is equal to angiosarcomas, and is another example of misuse of data.

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There are no data supporting a significant increase in tumors at other sites due to vinyl chloride inhalation at the concentrations of interest, nor are there any data at all on cancer formation at these concentrations. See page 77 of the STAR Document. This same conclusion has been reached in the Dow study and many other reports not considered by the authors (4, 5, 17, 18, 21).

The Agency has been informed that the Tabershaw study is being expanded to include a large cohort of long-term exposed workers and to provide better follow-up on the original group. This is expected to be available in a few weeks. Also, expected to be completed soon is a study by ORC (22) of a cohort of fabricator employees who have had lower exposures than polymerization workers, but significantly higher than the community. (See also references 4, 5 and 18.) These will supply data on relative tumor formation in large cohorts of long term exposures.

Therefore, the incidence of angiosarcoma in long-term employees is at least an order of magnitude less than the 0.02 used in Appendix D, certainly no greater than 3.3×10^{-3} and probably less, and there is no evidence for a significant increase in cancer at other sites due to vinyl chloride.

On the question of the relative susceptibility of man and rat (see page D2), this can be tested using the same assumptions as made by the authors. An exposure of 500 ppm for 7 hours per day, 5 days per week, for 16 years

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calculates to an average lifetime exposure of 23.1 ppm, which produced (no greater than) 0.33% cases of angiosarcoma. Similarly, the three lowest exposures in Maltoni's experiments give 3.1 ppm and 2%, 15.5 ppm and 7% and 30.9 ppm and 12%, for lifetime exposure and angio incidence, respectively. Thus, if man were as sensitive as rats, and the other assumptions by the authors were correct, the assumed exposure would have produced an incidence of about 9.7%, some 29 times that found (Figure 1). Similarly, it can be seen by visual inspection that such a plot is not linear. The authors arrive at a prediction of a 7.5% lifetime incidence for workers by similar reasoning. Such an incidence is not supported by any epidemiology study in existence.

The variation in animal sensitivity is ignored. Rabbits and hamsters are less sensitive to vinyl chloride than are rats; mice are more sensitive (1, 7). Both mice and rats generate angiosarcoma spontaneously, mice doing so at a higher rate than rats. No reason is given for choosing to equate rats with humans while not considering other species.

The statement on page D1 that Maltoni found that the latency period is constant regardless of dose is incorrect; just the opposite was stated by Maltoni (1, page 222). The average latency time for experiment BT1 varied from 64 to 135 weeks, and is the reported time of death, not the "appearance of tumors." The last survivor of the 50 ppm exposed group died at 135 weeks, much longer than a usual

lifetime, and the tumors were found only on autopsy. The interval between first exposure and diagnosis for the U.S. workers varied from 12 to 32 years (10). The authors' problem here arises from extensive use of averaged data, and not the actual ranges. Therefore, this argument does not provide support for the increased sensitivity of humans.

Insistence by the authors on the linear model because of ease of application is in conflict with the generally accepted principles of epidemiology, and is not supported by the animal data used, where the incidence of angiosarcoma increases more rapidly than does the exposure. Nature seems to operate in the normal distribution (log probit) mode, which is reflected in the general acceptance of the Mantel-Bryan procedure. In fact, it has been suggested by Schneiderman recently that even this model tends to overstate the risk when compared to actual experience in many cases (1, page 242). The inapplicability of the linear model is discussed by the National Council on Radiation Protection and Measurement in its publication No. 39, 1971, and the Maltoni data from experiment BT1 shows a closer fit to the prediction by the log-probit method than with the linear model.

The Risk Assessment Document states correctly on page E5 that "(t)his survey has produced no evidence that living around vinyl chloride plants is a risk factor in the occurrence of liver angiosarcoma." In view of the fact that

the exposures considered were high even for past conditions, and that the EPA surveys taken in 1975 show vastly reduced environmental concentrations (even lower than those predicted after the Proposed Standard is implemented -- see page 32 of the STAR Document) any added risk because of future exposure is extremely low, as shown below.

The Agency has attempted to use a standard diffusion model to illustrate the benefits of the proposed standard. The ultimate significance of this effort in Appendix A is obscured by the method of presentation of the results and by the assumptions made in its application. It is unfortunate that more details of the diffusion model study and exposure calculations are not given, so a better evaluation of the results can be made.

The only results presented in the Standard Support Document are the calculations of the maximum concentrations predicted. These are not helpful in understanding the possible exposures of residents because, with one exception, these maxima occur at 80 meters from the source center, or within the plant boundaries in most cases. Some "typical" isopleths are given in the Risk Assessment Document, Appendix A, but the basic data used for these calculations are not specified, nor is the method for calculation of the average area concentrations.

The assumptions used in the calculation of the maxima are given, and these are very conservative.

1. An average wind speed of 0.5 meters per second (1.1 mph) was used. The climatic data taken during 1975 at three plants averaged well over 5 mph. No compensation was made for wind direction.

2. No thermal buoyancy was used in the source stream. PVC drier outlets usually are above 140° F., and many other source points are mixed with live steam. This thermal gradient has a significant effect on effective stack height and mixing and, therefore, on the actual ground level concentration.

3. Average emission rates of 4.25% of the production of suspension plants were used. Comments in other sections of this submittal show that this is not representative of present conditions. Arbitrary assignments of plant size were made without regard to actual conditions.

No effort was made to correlate the actual monitoring results of 1975 with the model results. Analysis of the downwind data taken at the Kentucky plant gives a figure of less than 5 ppb at 1 mile compared to the predicted average value of 37 ppb on page A-3 of the Risk Assessment Document. (See Table 4.2, STAR Document).

The approach in Appendix A toward determining the average population exposure is oversimplified. No consideration is given to population distribution relative to prevailing winds, and no allowance is made for travel out of the zone for work or school.

Therefore, great reliance has been placed on theoretical

calculations from assumed conditions which are not substantiated by actual field measurements.

The underlying assumption of a linear risk model has led to a bias in the derivation of the risk assessments for other models by excessive use of averaging. A more reasonable evaluation of the data for the log probit case can be made. The exposures for the equal area radii are shown in Table 1, estimated from the data on pages A1 and A3 of the Risk Assessment Document. It can be seen there that the population distribution is in good relationship to that predicted from the area. Multiplying this by the rat risk level calculated by Schneiderman (1 page 237) by the probit method for that exposure, a total population lifetime risk is 0.63 cases for 4.6MM rats, or 0.14 cases per million. Accepting for the moment the authors' assumptions of these existing concentrations, the equal sensitivity of rats and humans, and ignoring a change in the rate curve, this would convert to 0.08 cases per year for this group (by dividing by 70 years per lifetime and multiplying by 8 to adjust for 24-hour exposure.) However, none of these three assumptions is correct, each tending to give higher results, thus further overstating the risk. It can be seen from Table 1 that approximately 95% of the exposed population is at a risk of the order of 10^{-8} , rat equivalent basis.

It is apparent from Table 1 that, whatever risk there may be, it is highest for the small population segment that lives within 0.5 miles of the plant. Table A-1 shows the

exposed population for each plant, and page A1 gives the sum for each area. We believe that the figure of 47,000 persons reported to be in the 0.5-mile radius is overstated. The Passiac plant (22,512 persons within the half-mile radius) is reported to have a capacity of 25 million kg/year, so the emissions are much below the "average" suspension plant. The Williamsville, New York plant (11,522 persons) has been reduced in capacity, and the Hicksville, New York plant (5,900 persons) is not operating. Thus, about 40,000 of the 47,000 persons shown in this distance category either have no exposure, or a greatly reduced exposure from that assumed, using corrected population data from that listed, thereby reducing the cumulative risk assessment by about 75% from that shown.

Table II gives a recalculated lifetime risk for this population using the actual reported concentrations from figure II. This lifetime figure of 0.072 cases for 4.6MM persons converts to an added risk of 1.8×10^{-9} cases per year.

The relevance of metabolic mechanisms to the prediction of risk has been competently discussed by Gehring (25). Since the levels of exposure being considered in this document are clearly in the range where the body "handles" VCM effectively and the dose-response curve is steep, the model should be adjusted to reflect this. The fact that the authors were forced to "constrain" the linear model to pass through

the origin supports the existence of a changed slope of the response rate at low concentrations.

The suggestion was made by one of the witnesses (Castelman) at the EPA hearing that the risks were understated because of possible synergism or because of teratological effects. No supporting evidence was given for any synergistic effects. As far as teratology, another witness stated that their experiments showed no such effect (25) and a full report is in press (7). This would be the expected result if exposures were such that the primary metabolic pathway of the mother was not overwhelmed. A dominant lethal study on mice at 3,000 - 30,000 ppm showed no effect on the number of early deaths (15).

It is unfortunate that such excessive weight has been given to the early monitoring data around a Kentucky plant, and that the Agency has failed to evaluate all of the data taken later in a much more complete and representative study. Table 4.12 of the STAR Document contains a partial compilation of these later data, and shows for example, that the same Kentucky plant has a geometric mean of $10.9 \mu\text{g}/\text{m}^3$ for all vinyl chloride readings less than 1,000 m from the plant, and $5.0 \mu\text{g}/\text{m}^3$ for readings taken at distances greater than 1,000 m for the data presented. These numbers translate to 4 and 2 ppb respectively. This is a far cry from the average of 17 ppb used in the risk analysis for all of a 5-mile (8,045 m) radius.

To illustrate this further, the 24-hour composite readings taken at the 31 off-property points during the study period (November 1974 through June 1975) have been plotted as an average for each site against the distance from the plant (see Figure II). This plot clearly illustrates the rapid decline in concentration with distance, and is consistent with the diffusion theory model used by EPA which predicts a decrease inversely proportional to the distance raised to the 1.78 power ($C = (f) \frac{1}{x^{1.78}}$). As can be seen, the concentration is about 3 ppb at one mile, and thus it is clearly impossible for there to be an average exposure within even an order of magnitude of the 17 ppb used for all persons in a 5-mile radius.

It must be recognized that the discussion in Chapter 6 of the Standard Support Document refers to annual maximum concentrations, not average readings, and assumes emission rates higher than actually found. (See discussion on pages 6-20 and 6-21.) We have been informed by the Standard Development Group that these maxima are calculated to occur at a distance of 80 m from the source, or within the plants, and thus in no way compare to the exposures seen in the surrounding neighborhood.

The results given in Table 4.12 are approximately the same for all three plants studied; therefore, it can be concluded that the average exposure for persons living more than a mile from a plant is below 1 ppb, and since the

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circle of a 5-mile radius contains 96% of its area outside the one-mile radius, more than 96% of the population would be expected to live in this low exposure zone. Extrapolation of the Schneiderman calculations (1) would show a risk of less than 10^{-10} for this concentration.

The fact that no detectable amounts of vinyl chloride were actually found by EPA monitoring sites beyond 3 miles could allow the perfectly reasonable assumption that there is none at this distance. This eliminates 62% of the population, and thus lessens the risk to about one-third of that shown by all previous calculations.

The comment has been made that the surveyed plants were operating at reduced capacity. Even a doubling of the concentrations found here would still give exposures of less than 1 ppb.

The question was asked at the EPA hearing as to why the populace which do not benefit from the presence of a plant handling vinyl chloride should be expected to bear a risk from exposure. Recent monitoring data around our plants confirm the later EPA data and that reported by other companies (see, for example, 14 and 27) in that fence-line concentrations average well below 50 ppb and normally are below the detectable limit of the method of 10 ppb. This would put the nearest residents in the 10^{-7} to 10^{-8} risk category according to Schneiderman (1). Thus, we do not believe that these residents are exposed to a significant

risk. To put such a risk level in perspective, we quote from (19) a survey of occupational hazards, which states, "The risk of death has been estimated at one in a million for smoking 1.5 cigarettes, drinking 1/2 bottle of wine, traveling 50 miles by car or 250 miles by air, rock climbing for 1.5 minutes, canoeing for 6 minutes, engaging in typical factory work for one to two weeks, or simply being a man of 60 for 20 minutes." Thus an added lifetime risk of the order of 10^{-8} to 10^{-10} does not give a significant increase in our otherwise normal risks.

The only actual data presented in the Risk Assessment Document, the 10 year survey of angiosarcoma cases, do not support the intended conclusion of added risk near plants handling vinyl chloride, and the basic premise of neighborhood exposure is not confirmed by the available monitoring data. No justification is presented for choosing the one-hit theory in spite of a total lack of consistency with the facts.

The data used in this risk assessment should be re-evaluated by competent statisticians, using correct interpretations, presenting realistic estimates of the accuracy obtained and the effect of the estimates used, and comparing to present ambient concentrations, in order to determine if the expenditure of \$198MM in capital and \$70MM annual operating costs actually results in a discernible reduction in risk. On the face of the arguments presented here, it does not.

E. Comments on the Star Document

The Health Effects Subcommittee of the VCM/PVC Producer's Group of the SPI has prepared a set of Comments and a detailed line-by-line critique of the STAR Document which we endorse and attach hereto as a supplement to our own Comments on this document.

The primary characteristic of the STAR Document is that it is a literature survey through late 1974 on the effects of occupational exposure to vinyl chloride. With only few exceptions, no 1975 data are presented, and it offers little information relevant to the question of environmental effects. It also serves as the source of some of the misinformation processed in the Risk Assessment Document.

The basic premise of a 4% average emission loss from polymerization plants is in error, as has been noted in other parts of these comments. Because of that, all of the figures on atmospheric concentrations are wrong. Even the limited ambient measurements in Chapter 4 show the error of this assumption. The population figures in Tables 4.1 and 4.2 are not representative of the number of residents near the plants of interest, and are not those used in the Risk Assessment Document.

Section 5.2 uses a carefully selected subset of workers, but a different group of studies, to arrive at an equally erroneous, but different, mortality rate for workers from angiosarcoma.

Chapter 6 argues a point that has never been disputed, that vinyl chloride is a carcinogen at high concentrations. It gives insufficient weight to the work of Hefner, et al, (1) which is relevant to risk prediction at low levels of exposure .

Section 6.1.4 does point out correctly the extreme sensitivity of the thyroid gland in rodents, but omits its unique nature. It also contains a more realistic estimate of past worker exposure than do the other related documents.

The conclusion on page 57 that vinyl chloride is mutagenic to humans is made without benefit of knowledge of the work of Purchase, et al. (15), who conclude that chromosomal changes to some cells do not include the germ cells in mice exposed to extremely high doses. The Infante study in Ohio has been disavowed by the CDC (6), as was mentioned in passing after all of the incorrect conclusions were spread on the record. The more recent Infante, et al. (20) study on wives of PVC workers may show an increase of fetal wastage, although the sample is very small and the data base is questionable. In any case it has no bearing on this issue, because the exposures involved were occupational, and at concentrations far above those at issue in this standard. This is part of a larger study which the government agencies have not yet reported (21).

Section 6.1.6 contains an erroneous reference to residual free radicals in PVC. Such do not exist.

Section 6.3 reiterates the now discarded "community"

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and "non-production worker" cases. Even the Risk Assessment Document does not try to argue this point.

The validity and significance of the Tabershaw-Cooper and Dow studies are disputed, while the NIOSH study is accepted as part of the data base. This latter study has only a 69% follow-up of individuals; the cohorts are not identified so it is impossible to determine if they are the same or as different from the other studies used; and the details of the study has never been published so that its methodology could be subjected to scientific scrutiny.

The STAR Document does state that any overall mortality differences suggested by the NIOSH and Mt. Sinai studies are not statistically significant. It is therefore difficult to understand the purpose of including an extensive discussion of their results, plus even more extensive speculation based on inconclusive data.

The treatment of technology in Chapter 7 is superficial and adds nothing to the knowledge of the subject. It is interesting to note that fugitive emissions from suspension plants have here risen to 45% of the total. It is estimated here that "about 50 percent" reduction could be achieved, "but a 95% reduction of total emissions --- might be beyond present techniques ---."

Thus, this document has done little to clarify the situation, is well out of date by now, and has a number of errors in fact and strained conclusions.

TABLE I

<u>Area Segment</u>	<u>% Total Area</u>	<u>% Total Pop.</u>	<u>Pop., M Persons</u>	<u>Avg. Conc. (ppb)</u>	<u>Risk</u>	<u>Total Lifetime Cases</u>
<1/2 Mile	1	1	47	245	10^{-5}	0.47
1/2 - 1 Mile	3	4.4	203	45	0.540^{-6}	0.10
1 - 3 Miles	32	32.6	1,491	12	2×10^{-8}	0.03
3 - 5 Miles	64	<u>62</u>	<u>2,838</u>	6	$<10^{-8}$	<u>0.03</u>
Totals		100.0	4,479			0.63

TABLE II

Recalculated

Based on measured concentrations at Kentucky Plant

(see Figure II)

<u>Area Segment</u>	<u>Pop., M Persons</u>	<u>Avg. Conc. (ppb)</u>	<u>Risk</u>	<u>Total Lifetime Cases</u>
<1/2 Mile	47	45	5.4×10^{-7}	~0.03
1/2 - 1 Mile	203	6	$<10^{-8}$	~0.002
1 - 3 Miles	1,491	2	$<10^{-8}$	~0.01
3 - 5 Miles	<u>2,838</u>	<0.5	$<10^{-8}$	<u><0.03</u>
Totals	4,579			<0.072

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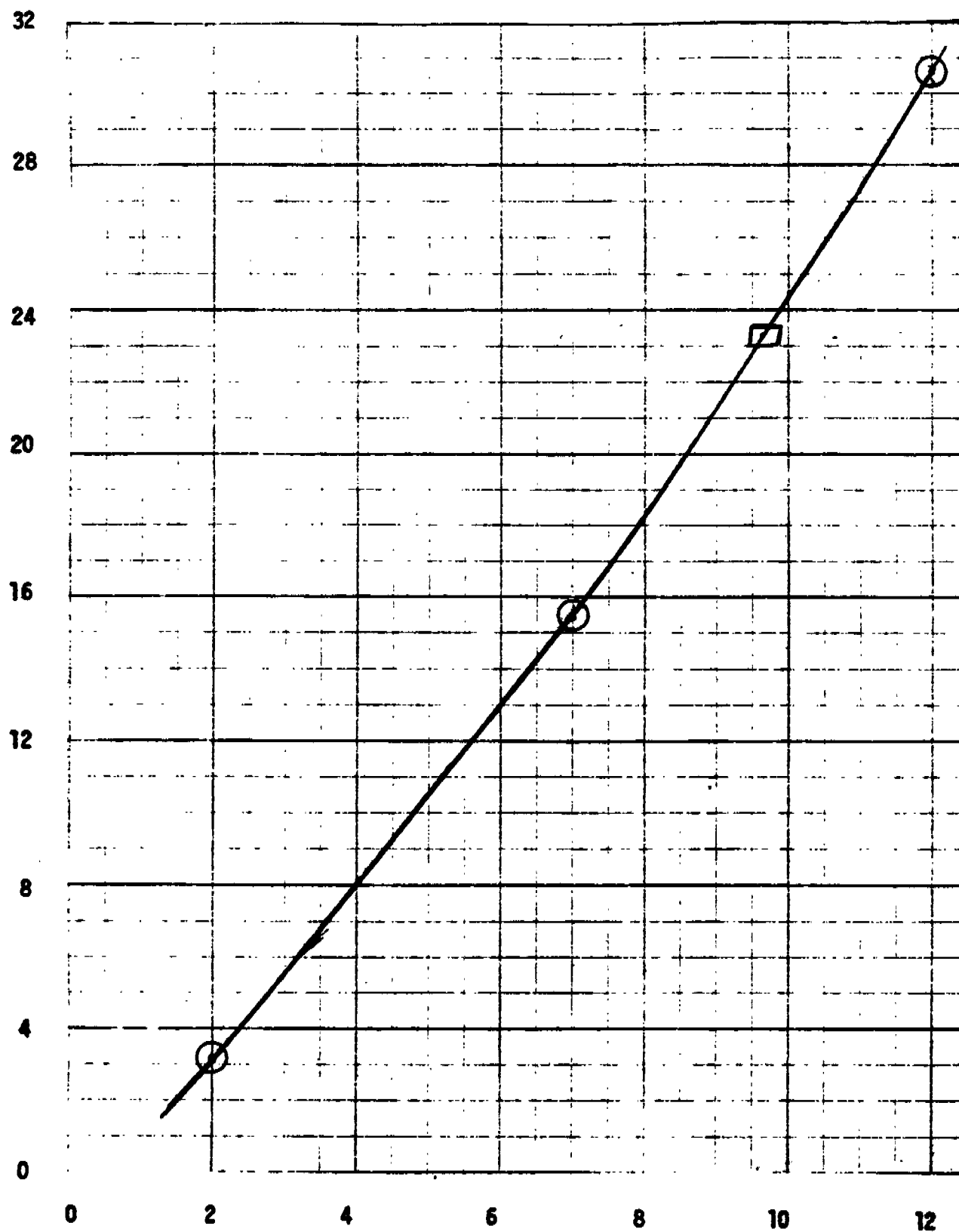
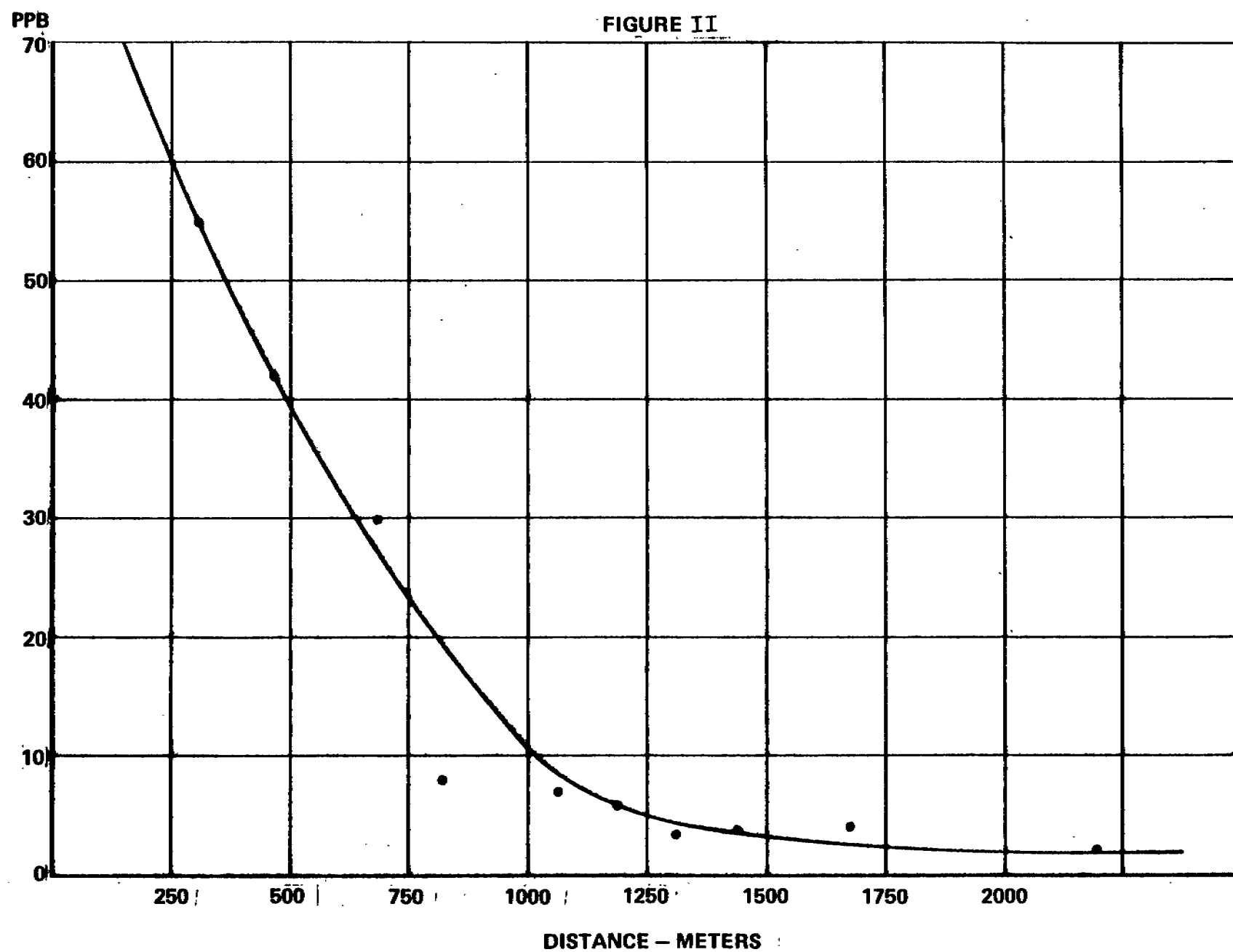


FIGURE I

Percent Incidence of Angiosarcoma

⊙ Rats, Maltoni experiment BTI

□ Humans, assuming equal sensitivity



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RISK ASSESSMENT DOCUMENT

215/687-5575

Executive Summary

<u>Page</u>	<u>Paragraph</u>	<u>Line</u>	<u>Remarks</u>
1	4	1-7	General Comment. Diffusion modeling methods, at best, offer only an approximation to the true state of affairs and can never substitute for actual monitoring data, which (although available) was unfortunately not taken into consideration. The weighting schemes proposed to account for the types, numbers and sizes of plants and the meteorological conditions are quite arbitrary, and different (but just as reasonable) choices for these weights would lead to a value much less than 17 ppb.
1	5	7	No cause and effect could be established by CDC <u>Morbidity & Mortality</u> , July 19, 1975, so the assumption should not have been made.
1	5	1-10	General Comment. Using animal data to predict human response is a very risky practice, because no real assessment of the validity of such an extrapolation has been made. The dose-response data for vinyl chloride is quite sparse (essentially only the Maltoni data is available), and no replications of such data are available for assessing the accuracy and precision of such data. The epidemiologic data available is of varying quality, and the Tabarshaw-Gaffey data is the best data available and it was not used. There was considerable selectivity in the employment of information from these various studies, and the resulting conclusions drawn in this report are clearly unrepresentative of what the collection of studies taken together indicate.
1	6	1-5	General Comment. The assumption that a lifetime exposure to rats would produce the same number of effects as a lifetime exposure to humans has never been verified. The use of a linear model is certainly a very conservative procedure, but it does not provide as good a fit to the Maltoni data as a log-probit model (which is more often recommended when dealing with dose-response relationships of the type we are examining). The authors seem to have used the linear model more for convenience in computation than for its ability to accurately characterize the dose-response relationship. Indeed, there is no reason for preferring the linear model to the log-probit model, and, statistically speaking, the log-probit certainly provides a much better fit to the Maltoni data. The numbers in this paragraph certainly cannot be accepted at face value based on all the shaky assumptions used in arriving at them. Clearly, some consideration of the statistical variations surrounding these numbers should be made.

<u>Page</u>	<u>Paragraph</u>	<u>Line</u>	<u>Remarks</u>
11	1	8	350 ppm is too low a dose. A much more appropriate dose would be that related to reactor cleaners over time period prior to 1974. Air Products' testimony at OSHA Hearing reports more realistic dosages in both U.S. and the United Kingdom. (STAR 6.1.4)
11	2	6	"Two other important facts about vinyl chloride carcinogenesis resulted from this analysis: 1) at some time in their lives about 7.5% of all highly-exposed workers are expected to get liver angiosarcomas due to vinyl chloride exposure with double this rate of primary cancer at all sites combined. 2) Of all the cases of liver angiosarcoma which have thus far been produced by vinyl chloride, only 38% of them have been diagnosed as of 1974." These are not facts but at best extrapolations from the models used in the study. The reasoning in arriving at the projection that 7.5% of all highly-exposed workers will get angiosarcoma is flawed. A correction factor is derived from the Tabershaw-Cooper Study and applied to the incidence data obtained by Nicholson. This is not appropriate since the hire dates in the Tabershaw-Cooper Study are a function of the starting dates of the plants in the T-C Study and not applicable to the single plant--Nicholson Study. If a correction factor is to be applied then the factor should be multiplied by the T-C rate so instead of 7.5% the percentage would be 1.2%; highly significant to the surviving workers.
11	2	8	What is dosage for "highly-exposed workers" which relates to 7.5% expectancy of angiosarcoma?
11	2	1-13	General comments. The probability 0.0031 (of a worker getting angiosarcoma at sometime in his life per year of exposure) is based on an incidence rate of 0.02, a value which is certainly <u>not</u> representative of the epidemiologic data as given (see, for example, the Tabershaw-Gaffey data, which are based on the largest sample size and provide the lowest incidence). No statistical measure of the error associated with this estimate is provided.

<u>Page</u>	<u>Paragraph</u>	<u>Line</u>	<u>Remarks</u>
11	3	1-3	General Comments. The fact that the probability estimate from the animal data (namely, .0052) is reasonably close to the estimate for the epidemiologic data (namely, .0031) says nothing about the <u>accuracy</u> of either of these estimates (i.e., two point estimates close to each other in value can be way off from the true value), and actual in this case, only reflects the selective choice of what epidemiologic data to use to insure reasonable agreement with the animal data. There is no statistical basis for having much confidence in either of these numbers.
11	5	1-12	General Comments. The fact that the better fitting log-probit model provides "low dose" (i.e., 17 ppb, which is too high) estimates of the number of cases per year of exposure which are 1/10 to 1/100 the size of those provided by the linear model casts serious doubt on the validity of extrapolation and on the use of the linear model itself. In addition, the use of a more reasonable and much smaller value than 17 ppb would considerably decrease these expected case values even more.
111	1	5	"No conclusion can be drawn from the survey at its present stage of completion." This proposition is difficult to rationalize since the authors have already stated that their model predicts one excess liver angiosarcoma death with the 5-mile radius for each of the past 10 years. If we accept the actual number of cases projected to be found, i.e. (286/176 multiplied by 3) and add the upper bound of one excess angiosarcoma death per year for the past 10 years to the expected 5.9 from the <u>Third National Cancer Survey</u> we have an observed 4.9 ^a vs. an expected 15.9. The probability of 5 or fewer cases with an expected of 15.9 based on the Poisson approximation is less than .002. If the expected were 13 the approximate probability would be .01. Is an observed of 5 (or less) reasonable in the light of an expected of 15.9?
			Any reasoned conclusion must be that the high values of risk resulting from the author's model are inconsistent with the upper end of model's. The survey begs a conclusion that the data does not support the upper spectrum of the modeled risk and is very consistent with no detectable

...cont'd

^aCase 3 has been confirmed as not being angiosarcoma. Therefore, only 2 unconfirmed cases remain in table 2 of Appendix E.

<u>Page</u>	<u>Paragraph</u>	<u>Line</u>	<u>Remarks</u>
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111	1	5	Another conclusion which should have been stated in the summary is that based on the CDC survey, "There is no evidence of clustering on a state-wide scale."
2	2	1-5	General Comments. The fact that the extrapolation from animal data to humans was based on assuming a (maximum) 168 hour exposure time per week (7x24) is certainly questionable in view of the remarks made in this paragraph regarding time spent away from home, etc.
2	3	4	General Comment. The statement that "the agreement between the two studies was good" does not say anything about the <u>accuracy</u> of either modeling approach. In fact, since both OAQPS and Teknekron used similar modeling procedures (except that the latter included data on meteorological conditions), they would be expected to provide reasonably comparable values. In this regard, the 25% discrepancy is somewhat <u>high</u> and reflects how much variation can be expected with such modeling procedures. It is regrettable that the authors made no use of this 25% figure in studying the sensitivity of their predictions. Of course, the accuracy of such diffusion modeling is completely unknown and can only be assessed with actual monitoring data (which was <u>not</u> done in this report). EPA had such data (which strongly refutes the 17 ppb figure), but chose to ignore it.
3	2	1,2&3	"Unfortunately, it has not been possible to make a systematic comparison of the diffusion modeling results with data obtained from actual monitoring..." How can one talk about a risk assessment for community exposure to vinyl chloride without actually confirming the community exposure? EPA had monitoring data (STAR Series December 1975 pp. 29-30) which clearly shows the 17 ppb assumption not possible.
3	2	3-6	General Comment. Again, the authors refer to the difference of up to 25% between the two diffusion modeling efforts as an estimate of the uncertainty of these efforts, but do nothing to assess the effects of such uncertainty on their final conclusions. This is typical of much of this report--the final numbers coming out are (as the authors admit) greatly affected by such uncertainties, and no efforts have been made to assess the effects of such uncertainties.

<u>Page</u>	<u>Paragraph</u>	<u>Line</u>	<u>Remarks</u>
3	3	13-18	General Comment. The statement is made that "by using animal data, we can avoid such problems" (e.g., with regard to assessing health effects due to PVC and VCM exposures). However, the animal dose-response data is quite sparse and no replicate studies are available to assess the variation in such data, so that any conclusions are tenuous.
4	2	1-5	General Comment. "For technical reasons", the linear model was used instead of the log-probit to assess health effects. These reasons appear to be based on ease of computation and conservatism, etc., which are not sufficient justification for disregarding consideration of the better-fitting log-probit model, especially since no strong case for using only the linear model has been made.
4	5	1	"of the four occupational..." The Administrator should be advised of the epidemiology study on British Petroleum PVC workers published in Lancet, December 13, 1975, pg. 1197 to 1199, "Mortality Study of Workers in Polyvinyl Chloride Production Plant."
4	5	12	"The result of the analysis is an estimate of the probability per year of exposure that a person will get angiosarcoma sometime in his life." This estimate pertains only to highly exposed workers. An updated Tabershaw-Cooper report will issue before the Administrator promulgates a standard. This report will be the most complete study in the world. It will have greater than 95% follow-up; have a cohort size of 11,000 humans and will have 2,000 new records of a much older group of workers. Assumptions related to incidence of angiosarcoma and all cancers should be reviewed and revised based on this report.
4	5	14-15	General Comment. The issue of "competing risks" cannot legitimately be ignored, as it was; there are available statistical techniques for performing competing risks analyses, given adequate data.
5	2	263	In view of the admitted limitations of the Marsteller study which failed to compare exposed workers with a suitable control group, failed to consider the effect of alcohol intake and lacked exposure data, the alleged ratio should have been seriously qualified.
5	2	6,768	Allowance should have been made for the rat zymbal gland, which is an organ unique to rodents and particularly responsive to other chemical carcinogens (STAR 6.1.4). There is no way to extrapolate the incidence of neoplastic change in the organ in the rat to what might be expected

<u>Page</u>	<u>Paragraph</u>	<u>Line</u>	<u>Remarks</u>
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5	2	6,7&8	in man. High local concentrations of chlorinated organic substances in the gland and the cellular characteristics of the organ are unique. There is consequently no valid basis for assigning an additional "non-angiosarcoma" tumor to man for each Zymbal Gland tumor. One must utilize organs and disease in which the conditions of exposure and response are fundamentally similar. Any use of the Zymbal Gland data for this purpose is inappropriate and misleading, primarily because of intense local exposure by lipid insoluble agents.
5	4	1-10	See exposure comments on Executive Summary, II, paragraph 1, line 8. In addition, the data referred to was developed by Dow Chemical Co. records and is unlikely to be representative of the entire industry. It appears that, at least in some plants, exposure was much higher than indicated by the study.
6	1	8,9&10	The only real data to validate the modeling projections do not support and are not consistent with the linear model used in the study.
6	3	3	"1) The number of cancers at all sites caused by vinyl chloride is twice the number of liver angiosarcomas". There has been no "cause and effect" ever established; only associations and the cited data do not support the assertion.
6	3	3	"2) The number of people with severe liver damage is 30 times the number of liver angiosarcomas". Again this is not a result or fact but an assumption which should be clearly so stated.
7	2	1-8	General Comment. The authors "judge that the number of liver angiosarcomas produced per year of exposure in people residing near vinyl chloride plants is somewhere between less than one and 10 cases." This clearly has to be a very subjective statement, based on the fact that no proper statistical analyses were performed to accurately provide something like a confidence interval for the true number of cases. Also, there is no quantitative assessment of the validity of any of the assumptions leading up to these numbers, and so the numbers themselves are essentially meaningless. It goes without saying, then, that any manipulations involving these numbers (e.g., multiplying by 30) lead to just as questionable values.

<u>Page</u>	<u>Paragraph</u>	<u>Line</u>	<u>Remarks</u>
Appendix B			
B-1	1	9	"Ideally, we should use human data throughout and avoid the problem of extrapolating from an animal model to human beings."
			The Tabershaw-Cooper Study will be completed before the Administrator reaches his decision. (See previous comments) This data should be analyzed before the standard is finally promulgated.
B-2	2	1-5	Constraining the linear model so that it passes through the origin should be based on <u>a priori</u> rather than on <u>a posteriori</u> considerations. In fact, a slightly negative intercept is obtained without this constraint, and this suggests the possibility of a "threshold effect" (which could possibly be verified with further experimentation).
B-3	3	6	"Estimates of the total number of cancers caused by vinyl chloride are based on the slope of this line, not on the intercept."
			It would seem appropriate to comment on the special nature of the zymal gland which is unique in rats.
B-3	4	1-2	The slope standard deviation estimates are only valid if the linear model is the correct one to use, and this is certainly open to debate.
B-3	5		The Maltoni animal data fits the log probit model better than the linear model, and there appears to be no reason why this should not be stated.
B-3	5	1-18	General Comment. The goodness-of-fit table looks impressive, but actually means very little because of the way in which the fitted model is eventually used. In particular, the fact that the linear model fits the data reasonably well in the region of experimentation means absolutely nothing with regard to extrapolation (i.e., prediction) <u>outside</u> the region of interest; in fact, a model can really perform well within the region of experimentation, but not be at all valid outside the region. Also, a similar table based on fitting the log-probit model would have indicated a much closer fit to the data, and, statistically speaking, the log-probit model would clearly be preferred on goodness-of-fit grounds alone.

<u>Page</u>	<u>Paragraph</u>	<u>Line</u>	<u>Remarks</u>
B-4	4	1-7	General Comment: The assumption that the community exposure would be continued for 24 hrs/day, 7 days/week is certainly an over-statement.
B-5	1	3-6	"...would have been caused by emissions over the past 20 years, during which total vinyl chloride production increased from about 10% of its current level." Seven PVC plants operated more than 25 years: GOM, BFG, Goodyear, Uniroyal, Union Carbide, Monsanto, and Firestone. Consequently, if the risk model were accurate, one would expect a higher incidence of angiosarcoma around these plants. In addition, the latency period is between 12 and 32 years; it would be more accurate to use a range.
B-6	2	1-18	General Comment. With regard to the error analysis presented, it really doesn't mean very much to even attempt to do such an analysis when the errors inherent in the extrapolations from high to low doses and from animals to humans cannot be quantified. With regard to the other sources of error mentioned, the use of the standard deviations of the estimated slopes to quantify this source of error is not valid unless the model itself is valid. The errors associated with the diffusion modeling procedures are not really reflected by the 25% discrepancy between the OAQPS and Teknekron predictions, since this only gives a rough measure of precision but says absolutely nothing about accuracy. Thus, the error estimate of 131 given on page B-7 doesn't really mean anything. Also, the arguments given concerning the errors associated with the numbers of estimated cases have no sound statistical basis whatsoever.
B-6	4	1-2	"Ideally, an estimate of the errors in the diffusion modeling could be derived from a comparison with actual monitoring data." The statement suggests either that data was unavailable or if available could not be analyzed. Such data is in EPA's possession, and it is frankly impossible to understand why the diffusion model was not validated using that data. Why wasn't the Administrator advised of the consequences of this validation which logically follows from the data shown in Table 4.11 of the STAR Document?

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<u>Page</u>	<u>Paragraph</u>	<u>Line</u>	<u>Remarks</u>
Appendix C.			
C-1	4	16	"One case is regarded as insufficient information to disprove the conclusion that the latent time in rats is independent of dose." One case may be insufficient evidence to disprove the conclusion, but there is other information, which the Risk Assessment Document studiously neglects to mention, pointing to the conclusion that time-to-tumor is dose dependent. (Paper by Albert, R. E. and Ritschler, S. in 1973 concerning considerations relating to formulation of limits for unavoidable population exposures to environmental carcinogens.) Also the Maltoni analysis supports a dose dependence.
C-2	4	1-5	Time-to-tumor is computed for man as 19.6/72. The 19.6 is based upon the assumption that all neoplasms that are to occur have already occurred.
C-4	1	1-5	General Comment. It is not justifiable to conclude that the incidence among just the most highly exposed workers is about 0.02 based on the results of the four epidemiologic studies of Table 1. First of all, it is invalid to ignore the Tabershaw-Gaffey report and to selectively choose those studies giving the highest rate. In fact, the Tabershaw-Gaffey study probably provides the most reliable (statistically speaking) estimate of the time incidence rate because of the large sample size involved. Also, the .02 is only an estimate and, as such, should have a statistical measure of error associated with it.
D-4	2	20	"... $0.02 \times 3.8 = 0.076$." Should be corrected by using Tabershaw-Cooper incidence rate.

<u>Page</u>	<u>Paragraph</u>	<u>Line</u>	<u>Remarks</u>
D-5	3	6	General Comment. The value of .0031 (the probability per year of exposure that a highly-exposed worker will develop angiosarcoma sometime in his life) is based on the .02 incidence rate, which itself is a highly questionable value. The quantification of the errors in the numerical estimates of parameters leaves a lot to be desired. The authors, by <u>intuition</u> , specify a possible range of values for the incidence rate, and they also provide only empirical guesses about the variation in chemical dosage and in the number of hours of daily exposure. Therefore, their value of 0.67 is highly suspect. Also, the conceptual errors in the formulation of the model are not quantifiable.
D-6	5	1-16	General Comment. The use of the factor 1/1.68, which is based on a comparison of the human annual incidence rate (.0031) and the animal rate (.0052), is highly suspect in view of the possible large errors in these estimated incidence rates. Also, saying that these two incidence rates are "close" to one another does not say anything about the accuracy of either one of the estimates. In fact, it is probably the case that both are way off from the true value, based on the unresolved numerical and conceptual problems associated with their determination.
D-7	5	2	Reference 16 is missing from report.
D-7	5	13	See comment on Page 5, Paragraph 2, Lines 6-8.
D-7	6	1-19	General Comment. The use <u>solely</u> of the Monson study to get at the ratio of all cancers to liver angiosarcomas is highly suspect in view of the fact that some studies (e.g., those of Ott et.al. and Wagoner) were not used because <u>no</u> angiosarcomas were observed and that others (e.g., Nicholson et.al. and Tabernshaw et.al.) were not used because no elevated rates were found. Such selectivity clearly gives a biased impression. Also, no estimate of error with regard to the Monson factor is given.
D-8	263	NA	No discussion was given to baseline population rates for the BSP test.

Page	Reason	Page	Remarks
Appendix B			
E-1	Last Paragraph	Last Line	"...national survey of all liver angiosarcomas..." A more accurate statement would be "...national survey of all liver angiosarcomas (subject to confirmation)..."
E-3	2	1	"A total of 206 cases were identified." This statement ignores the uncertainty which still exists as to the actual situation. "A total of 206 possible cases were identified."
E-4	2	1	"176 cases remained..." Should read "...176 possible cases remained..."
E-4	3	8	"...only six cases, as shown in Table II." The statement is misleading. For distances less than five miles only three possible cases of angiosarcoma have been found.
E-4	4	1-3	Case 1 is inappropriately included in this cohort as the document apparently admits.
E-4	7	1	Case 6 is mistakenly used instead of Case 5. The speculation about transplacental carcinogenesis is inappropriate. The fact is we do not know whether VC played any causal role in the cancer. If there is no evidence of VC's involvement, why speculate in a Risk Assessment Document.
E-5	5	1-16	General Comment. The fact that the survey produced <u>no evidence</u> that living around vinyl chloride plants is a risk factor in the occurrence of liver angiosarcoma is an important finding, demands some remarks to the contrary and this fact should have found its way into the main body of the document.

*Now reduced to two cases, see footnote page 3.