Kel-Mat.-Carcinogen-VC

01-VC/PVC-GOV

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January 30, 1975

TO: All Members of:

SPI Food, Drug and Cosmetic
Packaging Materials Committee;
General Polyvinyl Chloride Interest
Mailing List:
Ad Hoc Liquor Bottle Committee;
Plastic Pipe Institute
(Executive Board);
Plastic Bottle Division
(Voting Representatives);
SPI Executive Committee;
SPI Public Affairs Committee;
VC and PVC Producers Committee

Ladies and Gentlemen:

The purpose of this letter is to update you once again on the status of the pending Petitions for Review of the Occupational Safety and Health Administration's (OSHA) October 4, 1974 Vinyl Chloride Standard and matters related thereto, and to inform you about the latest activity with respect to prospective or anticipated rulemaking proceedings by the Environmental Protection Agency (EPA) and the Food and Drug Administration (FDA).

OSHA

Since our January 9, 1975 report to you, nothing substantive has occurred with regard either to the Petitions for Review in the United States Court of Appeals for the Second Circuit or to the Oil, Chemical and Atomic Workers' International Union (OCAW) Petition for Review in the United States Court of Appeals for the District of Columbia Circuit.

As to the OCAW case, several procedural filings have been made. Essentially these are cross-pleadings

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arguing the merits of the various motions to intervene, dismiss, and/or transfer the OCAW case to the Second Circuit. Although we still do not understand quite what OCAW really has in mind or what its objectives are, we and Government Counsel are both of the view that the OCAW Petition for Review should be and, probably, will be transferred to the Second Circuit. That Court will then have another procedural matter with which to deal. Since the situation is somewhat confused, this added burden on the Court could conceivably cause delay in the decision of the case. Meanwhile, the Stay on the effective date of the Standard will remain operative.

As to the matter of when and what the Second Circuit will decide, we still do not know and have no way of predicting the time or nature of the outcome. We can only assure you that as soon as we are informed about a decision, we will advise immediately. In short, for those who may be tempted to call us from time to time to see if there is anything new, please be certain that we have nothing of substance to report that is not included in these periodic letters.

In the category of VCM-OSHA "fall-out," we were informed about a week or so ago that the California counterpart of the Occupational Safety and Health Administration (Cal/OSHA) had scheduled a hearing for January 28 in San Francisco to consider what action was required of it in regulating occupational exposure to vinyl chloride during the pendency of the Stay of the Federal Standard.

Immediately after the SPI office in California notified us, and we were able to develop additional background on the situation, we advised our associate counsel in California about the matter and asked to be advised about the potential ramifications of the hearings. Since then, California counsel has been backgrounded on the situation, coordinated with counsel for the companies located in California, and took what we feel were appropriate steps to handle this matter.

Without going into great detail, suffice it to say that Cal/OSHA, advised about the status of the Second Circuit case and all other relevant legal aspects of the

matter prior to its hearing, reviewed its options on January 28 and opted to make its Emergency Temporary Standard of 50 ppm the Permanent Standard for the time being.

EPA

As we mentioned in our last letter, an examination of how to provide input to the United States Environmental Protection Agency (EPA) is being conducted. This effort now shows signs of crystallizing. You may recall that the concept here is to set the stage for future technological investigations by early settlement of the threshhold policy and legal questions which will steer the Agency's regulatory strategy. Although we are unable to report definitively on our progress in this area as yet, we hope to be able to show and report on some measure of real progress on both the policy/legal and technological fronts in the not-too-distant future.

Somewhat collaterally speaking, a very important opinion by the U.S. Court of Appeals for the District of Columbia Circuit has now been filed in the so-called "lead-in-gasoline cases." Although, as a result of the Court's Order prohibiting the subject Regulations from going into effect on the first of January, we had known about the ultimate decision for more than a month, the full opinion was just published yesterday.

In the 175 page Decision the Court said that the Environmental Protection Agency's (EPA) Regulations requiring a massive reduction in the lead content in gasoline should be set aside because the studies and evidence relied upon by EPA were "speculative, inconclusive," and otherwise not sufficient to prove that lead from gasoline was as significant a health hazard as alleged. The Court held that such Agency decisions must be based on specific facts and not on conjecture.

Being mindful that the statute, Agency and Courts are different in this case than in our Second Circuit case, nonetheless, the significance of this major defeat for EPA will hopefully not go unnoticed by

the Second Circuit. We believe this new decision should be seen as supporting the concept our case shares with the "lead case," that is, allegations of a health hazard and what is necessary to correct it (the severity of the exposure limitations and "feasibility" in our case) must be supported by hard, factual evidence. Ethereal calculations, conjecture or speculation should not be allowed to support Agency action.

Although we are unable to send you the entire opinion in this case because of its length, we are enclosing a copy of a reasonably accurate report on the opinion from yesterday's Washington Post. Copies of the opinion, Ethyl Corporation et al. v. EPA can be obtained by sending $2.00 per copy to:

Clerk
United States Court of Appeals for
the District of Columbia Circuit
U.S. Courthouse
Constitution Avenue and John Marshall Place
Washington, D. C. 20001

FDA

Turning to FDA matters, again nothing new can be reported with respect to actual Agency action. However, Food Chemical News (FCN) for January 20, 1975 published a report of correspondence from Secretary of HEW, Caspar Weinberger, confirming that FDA is now planning to set a 50 ppb limit on extraction of vinyl chloride monomer with no limitations being set at the manufacturing level. The Secretary's letter purportedly had been sent to Senator Tunney who had asked "what FDA proposed to do in regard to polyvinyl chloride food packaging, and the scientific and legal justifications for setting a non-zero tolerance for vinyl chloride in polyvinyl chloride food packaging, foods, drugs and cosmetics, if that is in fact your intention." FCN noted, however, that these paramaters are still under review at the Bureau level with no promise as to when the actual proposed Interim Regulation will be promulgated. For your ready reference and with the permission of the publisher, we are attaching a copy of the FCN article.

EPA
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1/31/75

MR. K. L. SPALDING
ROOM 1834 - OSP

Ken:

Here are my comments on the EPA document you left me. You might follow them along with the notes that I have made on the document itself. I have written them in light red pencil so you can hit the areas that I'm talking about. I'll just comment on the places serially, starting on page XXI in the abstract and continuing on.

First, let me give some general observations. The EPA has made some very vague generalities, has completely ignored the levels at which disease and cancer have occurred, have ignored length of exposure, and have ignored the fact that hemangiosarcoma of the liver has been known to have been produced by other chemicals, particularly arsenic and ~~a~~thorium and that in fact the bulk of the angio cases known in the U.S. are by far more related to these other two materials ~~in the art~~ of vinyl chloride. In §2, EPA I think has just one sentence referring to arsenic and anthorium.

Page xxi - second paragraph. Levels of exposure are not included here, and therefore, a recitation of diseases is meaningless. There is no way to extrapolate from a finding without a quantitative description of the finding. So that the extrapolations are not only unreasonable, they cannot be made at all.

Page xxi. There is no evidence that vinyl chloride poses a serious health risk to the general public or a health risk of any kind to the general public. The evidence is that vinyl chloride, under specific conditions, does pose a health risk to a limited, ~~and~~ that is the occupationally exposed polymerization workers who may have been exposed to very high levels for long

periods of time. It is important to keep reiterating this sequence: occupation, type of exposure, level of exposure, and length of exposure.

Page xxii - top paragraph. The reasoning in the first full sentence on that page is specious. For example, ultraviolet from sunlight represents a cancer risk, and from ultraviolet there also may be no exposure level which represents absolutely no risk to public health. Neither the OSH Act nor the EPA enabling legislation implies or requires that there be absolutely no risk to public health. Therefore, a standard cannot be based on a consideration of absolute guarantee.

Page 2-1. In paragraph 2 on this page, exposure concentrations of up to 400,000 ppm are listed. I would like to see confirmation via a reference at this level. The experimental chambers should have been blown to bits.

Page 2-1 - bottom paragraph. The entire bottom paragraph of this page is meaningless since no levels are given. The chronic effects vary with level of exposure with species, and therefore, no conclusion can be drawn as to point at which effect will take place. The only conclusion possible is that since the lesion in man (hemangiosarcoma of the liver) and the lesion in animals were identical when exposure occurred to the same material that vinyl chloride does produce this rare liver cancer. However, the conditions under which it will produce it cannot be implied from the data. Therefore, the bottom paragraph on page 2-1 is improper because it takes what can only be a general conclusion and implies that a specific conclusion may be drawn.

Veltman, Lange, et al - Clinical Manifestation + Course of V.C. Disease, Annals of N.Y. Acad. Vol 246, pp 6-17 Jan 31, 1975

Page 2-2. In the middle of the first paragraph there is a reference that studies in Germany show exposure levels which are not known. These exposure levels, however, can be implied. See the paper by Veltman and colleagues printed in the New York Academy of Sciences Meeting, pages 6-17. On page 7 Veltman points out that the 70 patients who had been examined averaged (mean? ~~medium~~ median?) 7.7 years with a range of 6 months to 21.75 years were engaged in cleaning autoclaves ~~with~~ and centrifuges, drying and sifting processes, and wrapping PVC as an end product. Thus, while he gives no levels, it can be inferred from the information we have from our own polymer people in the U.S. who did the same tasks that levels must have been in the thousands of ppm.

Page 2-2. In the second paragraph on that page we question the superposition ~~the position~~ of the range of the concentration with the European data. These numbers appear to come from the Dow data where no cases of angiosarcoma were observed. Therefore, the use of these levels is not only incorrect, it appears to be deliberately misleading. The sentence following is also not true. They are no references to studies in Europe and the United States which tend to confirm the earlier findings in Europe because environmental data and length of exposure are not given.

Page 2-2. There is another misleading sentence. In the next to the last sentence of the page, studies released by NIOSH, particularly the table of cases released by Dr. William Lloyd on January 22, 1975, showed only 15 cases of an angiosarcoma in workers who were all PVC workers. Twelve of the 15 had had exposures in excess of 15 years.

Finally the last sentence on page 2-2 is meaningless since no reference is given to the study, the intervals of time, the length of exposure, or the level of exposure. Furthermore, the last sentence is meant to imply

See OSHA hearings - June 1974

Release by NIOSH - Table 1 Cases + 1 data cited.

that damage once it occurs is progressive. This is probably so, but the interval of time between last exposure and the taking of biopsies will only identify the damage at the time of biopsy. It implies that multiple biopsies were made, and there is no evidence of that. I would like to see the literature references to patients who have had multiple biopsies and what the changes were there. The last sentence in the paragraph is also deliberately misleading.

On page 2-3, references to the non-PVC cases are incorrect. In N. Lloyd's letter of January 22, all cases in which the hemangiosarcoma was definitely confirmed and of which there were no questions were in PVC workers. Of the two cases listed in non-PVC workers, one was not a confirmed hemangiosarcoma but was another liver disease... The second was called a hemangiosarcoma but correlary symptoms were not seen by the government pathologists. In that same paragraph, the Lloyd list does not show any U.S. employees exposed to vinyl chloride but not directly in polyvinyl chloride production. Therefore, there are no data to suggest that exposure to vinyl chloride at lower levels than in that encountered in PVC production plants may be capable of causing liver angiosarcoma.

This last comment also points out what may be a very deliberate attempt at obfuscation and misinformation. Generally, workers and exposures from polyvinyl chloride plants are higher than those in vinyl chloride plants since in the VC plants equipment is ... closed and the processes continuous. But it is not reasonable to assume that every exposure in a VC facility is less than that in the PVC facility. Therefore, if a worker in a VC facility succumbs to hemangiosarcoma of the liver or other cancer associated with VC exposure, it is incorrect to make inference that exposure at lower levels have caused this. It is necessary, rather, to make or

reconstruct the concentrations and the exposures which this individual faced. Therefore, any conclusion drawn by EPA on the basis of exposures to VC workers alone implying ~~that low lie chows~~ that low levels can cause the disease is totally inappropriate.

Staying on page 2-3 you already have noted that the so-called community cases of liver angiosarcoma don't exist. There is nothing to tie them in to vinyl chloride and the Buffalo (Niagara Falls) case turned out to have been no angiosarcoma at all. (see p 2-67 of the EPA report)

Page 2-3. The last sentence is incorrect. The data presented by Dow and Union Carbide showing levels of exposure, duration of exposure, and the absence of cancer, particularly angiosarcoma, do indicate that the level necessary to induce liver cancer in the occupational population may be known. See OSHA hearings - (also p 2-69 to 2-71 in Dow)

Page 2-4. The first sentence on this page is utter nonsense. A relative risk must be a ratio, and there is no basis for a single number such as 3,000. The number is meaningless, and there is no documentation of any number or ratio that would be at that order of magnitude. The same holds true for the relative risk. There have been very limited searches of hemangiosarcoma of the liver. They have appeared in only four plants in the United States. Admittedly, the relative risk is high to workers in those plants, but the incidence of the disease goes to zero in other vinyl and PVC plants in the U.S., and therefore, it can be argued that there is no statistically significant difference in frequency depending upon how one takes the cut. If the relative risk is for the industry as a ~~hold~~ whole, then one must correlate this with the high levels referred to but not documented. Certainly there is no way of judging the significance of this paragraph without quantitative information.

Page 2-4 - second paragraph. The only published epidemiologic data showing other diseases is the Tabershaw-Cooper report. It does show differences in other cancers, including brain cancer, but the changes are not statistically significant. In fact, there are no other reports published that show that other cancers have occurred with any significant or statistically significant frequency. Incidentally, the last sentence in that paragraph which you have questioned probably is correct.

Page 2-4 - third paragraph. Angiosarcoma of the liver in rats at 50 ppm is a highly equivocal matter. These are Maltoni's rats who were allowed to live out their normal lifetimes and who lived well beyond the normal span of life for a rat. This was at exposures of 50 ppm. If one is to draw the conclusion that exposure of rats to 50 ppm caused angiosarcoma of the liver, then one must with this same data come to the conclusion that exposure of vinyl chloride prolongs the life span, since in this experiment all of the control animals had long since died.

Page 2-5. You indicated there is need for proof that continuous populations exposed to low levels of vinyl chloride may undergo a health risk. I agree.

In the same paragraph we find the first reference to thoratrast and arsenicals. Again, most known cases of angiosarcoma are from these two media.

Same page. Comments on extrapolation of dose response curves to be to define and presume no-effect level are highly qualified. But the question of whether a no-effect level exists or not is not the question at hand here. One is concerned with the so-called "effective" no-effect level, and that is that if an effect will be caused by a small dose of a carcinogen, what is the latent period, how long will it take before the cancer is manifest. If you

use the Maltoni data in the rats at 50 ppm, for example, one might postulate that cancer will occur in humans at low levels at age 90, 100, 110, or perhaps even 160. Obviously, when the latency period equals or exceeds that of the normal human life span, we have an effective "no-effect" level, and this is what we were trying to determine.

On the same page, at the very end, EPA notes that it is difficult to extrapolate data from animals to man, and I agree with this completely. One uses animal data to predict human response and to take action in order to prevent human effect. However, when human effects and dose responses are known, then the human data must take precedence over the animal.

Pages 2-6 and 2-7. On the conclusions I would argue that the data available do not support the conclusions.

Conclusion 1 talks of the potential health hazard. The word "potential" is meaningless and misleading over here.

Conclusion No. 2 is incorrect for reasons that I have already indicated. No cases of the disease have been reported among workers exposed to VC but not directly involved in PVC. And finally the question of operation does not talk about the effects at lower levels of exposure since the disease is a function of total exposure or dose which includes level as well as time.

In item 4 of the conclusion, the mechanism in dose response is not known in the occupational environment, and therefore less known or knowable at the community exposure level. But because the mechanism is not known, there is no reason to take the action proposed on a community basis. Dose response can be inferred although it is not known in the occupational environment, and from the human data available to date, one can draw the conclusion that current levels in the environment as measured by the EPA over the last year or so

probably posed no hazard to the general public.

Section 2.2 (pages 2-8 through 2-17) appears to be filled with misconceptions, misinformation, and deliberate misdirection. For example, the very first paragraph on page 2-8 relates to PVC particles which may be deposited in tissues. This is referenced to the paper by Volkheimer. (already cited see ref # / p 2-15) Reading of the paper however shows that the PVC particles were implanted in the animals by ingestion rather than by inhalation. The greatest percent migration occurred at 30 microns which is much larger than what would appear or what could appear in the air. There was virtually no migration of particles below 10 microns. Therefore, the conclusion drawn by this sentence in the paragraph is incorrect and deliberately misleading.

Paragraph No. 2 on that same page has the same kind of misdirection. The levels shown at an excess of 1,000 ppm were probably attributed to the statements made by Dernehl. But he, as well as B. F. Goodrich representatives at the Labor Department hearings, were talking in terms of levels of 4,000 ppm and higher. Admittedly, 4,000 is more than 1,000, but at least the numbers should be mentioned. In addition, the levels at the 250-500 ppm range are from Dow and Union Carbide data. However, neither Dow nor Carbide saw the liver pathology against which EPA is trying to protect. There were no cancers involved. Therefore, the inclusion of these numbers and implying that they have led to angiosarcomas of the liver is highly improper.

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Equally improper is the pargraph on page 2-9, relating to studies on vinyl chloride aerosols used in home situations. The paper by Gay & Associates was not done in home studies, but was done in simulated ~~laboratory~~ environments. The conjectures (see New York Academy of Sciences Report, pages 286-295) state as follows: "This study was undertaken to determine human exposure levels and decay of vinyl chloride in home and office environments. Reported herein are

the findings from usage of two aerosol spray cans that are known to contain vinyl chloride." (Emphasis added.) Even in the cases where home and office environments were used experimentally the authors note that "in some cases room ventilators were deliberately closed or blocked off in an attempt to restrict ventilation to simulate the worst possible case." I submit that the experiment was worthless, the conclusions were worthless, and should not be included in the EPA draft. It is obvious that the experiment was set up deliberately to attain high values. Yet if one looks at the estimated values by the authors under this circumstance, it turns out that potential concentrations under the worst possible estimated conditions still show a possible concentration of 3 ppm averaged over one hour.

The last paragraph on page 2-9 and extending over to page 2-10 is a statement made without any evidence whatsoever. It is pure conjecture and has no part in such a report.

On page 2-10 the estimates of relative importance of sources completely ignore the volatility of vinyl chloride in food and water. The conclusion, however, is probably correct that the relative hazard to the public, if a hazard exists at all, is via the air rather than the food or water route. But saying that there is a relative difference in risk does nothing to imply, indicate, or prove that there is a risk at all to begin with.

The statistical table on page 2-13 is yet another example of totally incompetent work on the part of the EPA. The four studies reported are not additive; there is an improper denominator; there is no tie in one with the other; the comparison between four limited working populations and the general population is statistically and epidemiologically invalid; a calculation of a relative risk is meaningless here. These numbers should be

vigorously fought, and any expression of relative risk to the general population should be eliminated. As a matter of fact, EPA scientists should be taken to task for permitting such garbage in their report.

H. L. Kusnetz

On page 2-18, the first paragraph, speaking about acute exposure, only indicates ~~bleakly~~ obliquely that high levels of gaseous monomer are involved. But, where liver and kidney damage and acroosteolysis has been observed, from the OSHA hearing + other parts of the EPA document, the implication is that the levels were several thousand ppm. The draft document is remiss in merely using the word "high" without specifying the levels.

Page 2-19, The last paragraph in § 2.3.1.I makes a rather broad jump. Vinyl chloride, is a recognized chemical carcinogen, but the levels at which it will cause cancer and the latent period corresponding to those levels are unknown. Therefore, the conclusion that its presence in the environment represents a public health hazard is only an assumption and cannot be used as the draft document does as a statement of fact. EPA should back this statement up with concrete documentation.

The bridge between pages 2-20 and 2-21 is unclear; apparently some words are missing. Nevertheless, note that the upper paragraph on page 2-21 refers to levels in excess of ~~100M~~ 100,000 ppm. This is something approximately three times the lower explosive limit and 500 times the levels cited by Rowe in the June OSHA testimony (200 ppm) below which no effect on humans was seen.

The second paragraph on page 2-21 ~~in~~ and reference 11, of the EPA work, refers to data obtained the period 1930-1947. Von Oettingen merely reported items in the literature) ~~without any indication as to credibility of analytic data.~~ all of which were related to levels 20,000 - 300,000 ppm Therefore, ~~the~~ conclusions can be drawn from work quoted by Von Oettingen ~~must be subject to a major qualifications.~~

Page 2-23. In the middle of the page, the statement, "It is conceivable that carcinogenicity of vinyl chloride would have been known nearly 15 years ago ..." is pure conjecture on the part of the authors. This

does not have any basis in fact, and if it is to stay in the document, in the it must be documented.

On page 2-24, the last paragraph is also deliberately unclear. Of some 35 experiments shown in animal species in Appendix B, only two, that of Vazin (1969) and Basalaev (1972), show the altered functions at low levels. Vazin shows 8-12 ppm, and based upon all other studies, one would question the accuracy of his analytic data. Basalaev shows slightly higher levels, and the same question would be there. In any event, two out of 35 experiments in animals which are so obviously outliers do not suggest the possibility of cardiac disturbances and behavioral changes in man under occupational circumstances. The leap is worse than Evil Kneival's leap across the Canyon.

Page 2-31. It is important to note that the EPA document itself also comes to the conclusion that the studies indicate that, "Vinyl chloride carcinogenicity is dose-dependent." The only reluctance on the part of NIOSH and EPA is to say that if you follow the dose-dependency curve, you will come

to a threshold, and yet they don't want to admit to the threshold. I think we should call EPA on the statement on page 2-31 and ask on what basis they then reject either a true threshold or an effective threshold.

Page 2-37. You've already picked up the statement near the bottom of the page which says any concentration for any period of time may not be without risk. This does not follow from the data presented, and again, represents high conjecture.

Bottom of page 2-37, top of 2-38. Note that the effects of mutagenicity have been noted in bacterial test systems. They're referenced in # 27. All the bacterial test system will show is that the material under test will cause alteration in a single-celled animal, possibly in the DNA or RNA of the animal. It says nothing with respect to any possible effect on humans. Therefore, any conclusion drawn by EPA on the basis of the mutagenic test in reference 27 is highly equivocal and preliminary.

The last paragraph on that page is thorough nonsense. Vinyl chloride is highly volatile. Any liquid vinyl chloride on clothing or on the individuals would long have evaporated before they are home. It seems that again EPA must document any case where the transmission of vinyl chloride monomer from the workplace to the home has taken place.

On page 2-39, note that the levels at which respiratory and cardiac arrest were observed were at exposures of 100M ppm. The blood levels at 27-40 mg percent is quite high. For your information, normal blood calcium runs 9 to 11 1/2 mg percent. On the other hand, sodium or chloride ion run between 310-387 mg percent. At the bottom of page 2-39, any conclusion is drawn from the early studies are meaningless in a situation which we have now because of the tremendous concentrations used.

Page 2-40 repeats agains the data by Hefner on the possibility of

(Dow testimony - OSHA referenced in N.O. EPA article)

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a metabolic threshold for vinyl chloride. And if there is such a threshold, then one can also suggest that the metabolites would be different so that cancers may not occur under various concentrations.

Page 2-41 to the top of 2-45. The last sentence is total conjecture and must be documented by EPA before any conclusions can be drawn.

Page 2-44 recognizes the discrepencies in the relations of metabolites to carcinogenesis, and therefore, all of the material which preceded it, attempting to point to the relationship, must be discounted.

Page 2-45. The top paragraph on this page referencing Volkheimer, which incidently they misspelled, describes vinyl chloride and polyvinyl chloride particles by ingestion, and therefore, the conclusion drawn in that paragraph is utterly meaningless. One cannot take an ingestion of PVC and come to anconclusion with regard to inhalation of VC.

Page 2-52. The middle of the page has a deliberate error. The paragraph in the whole section speaks about human levels and investigation. The sentence, therefore, that results of American and European toxicology studies reveal the induction of a liver angiosarcoma and other tumors at a 50 ppm exposure level of vinyl chloride is misleading. EPA must indicate that this is in animals. The insertion of this sentence without the clarification is a deliberate attempt to mislead.

The bottom paragraph of page 2-52 also is factually incorrect. EPA has shown no data, nor or there any data, to substantiate the statement that the incidence of angiosarcoma substantially exceeds the estimated national incidence level. As a matter of fact, the January 22, 1975 table of NIOSH does not show this. Further, the so-called community cases of liver angiosarcoma are a misnomer. That these were cases in nonpolyvinyl chloride workers would be correct. To call them community cases ignores

all of the other cases of angiosarcoma in nonpolyvinyl workers that did not occur in areas around PVC or VC plants. Further, the so-called Niagara Falls case in the vicinity of the Goodyear plant just ~~don't~~ doesn't exist. EPA should document that one.

On page 2-53, up in the top, the references to the community cases must also be challenged. The studies by the Center for Disease Control have not reported such cases as community cases. EPA must be made to document this statement or else eliminate it. Finally, on the same page, the last sentence in the top paragraph. EPA must state and document what the levels were, where they were found, and how they compared to ambient levels of "vinyl chloride" in other parts of the country where this monomer would not be expected, i.e., where it will be found on a control basis.

We're now up to § 2.3.3, human effects, starting at page 2-61.

On page 2-61, the last full paragraph indicates that the long latent period suggests that the full impact of past vinyl chloride exposure may not yet be realized. This is true. On the other hand there is an alternate explanation, and that is that the long latent period, coupled with the relatively high exposures, that must have occurred during those years, may also suggest that there is an effective threshold especially when one considers the apparent dose-response curve found in animal experiments.

On page 2-61 and continuing over to page 2-63, skipping the table, the question of the accountant is still not settled as to whether this was an angiosarcoma. Certainly Dr. Popper did not so classify it. (This information came in a telephone conversation with Dr. Falk at CDC.)

The material starting on the bottom of page 2-63 and continuing over to the bottom of 2-65 is utter conjecture.but very important conjecture because

* private communication from CDC, but see also pp 2-63 to 2-65

the case may be made in both ways. First of all it is the only place, and amply buried, where the possibility that the so-called community cases are not really cases of angiosarcoma happens to exist. Note that this possibility is not repeated in the abstract where they are referred to as definite cases. Further, it is quite important to note, as the EPA statement does, that the cases are not identical to the pathology which has been observed among the polyvinyldchloride workers. The fact is then therefore that one can state that since angiosarcoma of the liver is known to have existed without exposure to vinyl chloride that the cases in Connecticut may be unrelated. It is also interesting to note that the accountant's case also was not identical to the pathology which was observed as I noted in the previous paragraph. Therefore, to make any conclusion with regard to the so-called community cases is utter nonsense. In addition, the term or reference to "clustering" of these two community cases is ridiculous. Two cases no more make a cluster than two epidemiologists make a mob. Finally, the EPA document itself notes that the so-called community cases were in cases of quite elderly people. By the time they reached 70 and 80 years of age, they had to die of something, and cancer became an even greater likelihood.

Pages 2-66 through 2-68 are based upon earlier and older versions of the Tabershaw Cooper Associates paper. Reference should be made to the more current studies now underway which show that there appears to be a dose relationship with angiosarcoma of the liver. In any event, the Tabershaw-Cooper data represent the most thorough studies done on a broad base to date.

The Dow data are reviewed next, but most important is EPA's comment on page 2-73 which talks about the strengths of the study, particularly the availability of measured vinyl chloride exposure and the successful follow-up of over 95% of the cohort. The discussion on the weaknesses in terms of what concentrations individuals might of had again is conjecture. It is fair to state that perhaps we might have to wait 30 or 40 years before we can be sure about level and latency periods. Page 2-

On page 2-73 and 2-74, reference is made to the increase in brain and lung cancer. The Tabershaw-Cooper data also show a possible increase in brain cancer, but the excess is not statistically significant.

Page 2-76, in citing the work by Nicholson, which was also reported at the New York Academy meeting in May 1974, reports that the authors concluded that levels may have exceeded 1,000 ppm and occasionally approached 10M ppm. This possibility is quite correct in that the symptoms that were reported, namely, dizziness, headache, or euphoria, and especially loss of consciousness, could take place only at these high levels. It is interesting to note that in discussions in Cleveland, (1974) and at the New York Academy meeting, these symptoms were given although numbers were not provided. One must conclude, however, that where the symptoms appeared in workers, levels were in the thousands of ppm.

Page 2-76 and 2-77 refers to a study among PVC workers conducted by NIOSH. Although this study is referenced, no data appeared in the literature. Only the conclusions were made before the Senate subcommittee in August 1974, as noted by reference 2. Note also on page 2-77 that the study done by Wagoner in the two plants where cases cases had occurred had only 69% of follow-up. Recognizing that the plants were there where already known cases were picked for the study, and that the follow-up was so poor, less of a

conclusion can be made here than even in the Tabershaw-Cooper studies. And yet, as you note on page 2-78, attempts are made to represent statistically significant changes. I think it important that the total industry and the cases lost to follow-up must be found before such conclusions can be drawn.

Page 2-80 repeats that these workers may have been exposed to concentrations and excess of the old threshold, but it should be more definitive with ~~that~~ but conjecture.

On page 2-82, reference is made to the long latent period as a possibility of failure to observe liver angiosarcoma in workers. An alternate conclusion is that where concentrations are low, the latent period is likely to be so long so there is a no-effect level or sufficiently long that the worker dies from other causes before angiosarcoma can even occur.

The conclusion in the next to the last paragraph on page 2-83 is further conjecture.

On page 2-91, regarding studies in Niagara Falls, a condition known as "hepatosplenomegaly" was observed. Check with Joyner on this, but I don't think there is any such disease. I think was is meant is "hepatomegaly" or enlarged liver and splental megaly or enlarge spleen. In any event, conversations with Dr. Falk of the Center for Disease Control indicated that the examination by Dr. Selikoff was not in conformance with usual medical practice. And that is, Dr. Selikoff looked for the least possible hint of enlargement, and that according to Falk, any questionable enlargement was considered an abnormal liver. It would be interesting to see if other physicians would make the same diagnosis, given the same patients.