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Transcript of Proceedings

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

PUBLIC HEARING

PROPOSED NATIONAL EMISSION STANDARDS FOR IDENTIFYING, ASSESSING AND REGULATING AIRBORNE SUBSTANCES POSING A RISK OF CANCER AND ADVANCED NOTICE OF PROPOSED GENERIC STANDARDS

Volume 1

Washington, D.C.

March 10, 1980

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JD/DP

1 U. S. ENVIRONMENTAL PROTECTION AGENCY

2 PUBLIC HEARING

3 ON

4 Proposed National Emission Standards for
Identifying, Assessing and Regulating
5 Airborne Substances Posing a Risk of
Cancer and Advanced Notice of Proposed
6 Generic Standards

7

8 First Floor Auditorium
GSA Building
9 18th & F Streets, N.W.
Washington, D.C.
10
Monday, March 10, 1980
11 9:00 A.M.

12

13 BEFORE: WALTER C. BARBER, Chairman

14

15 EPA PANELISTS:

16 DR. ELIZABETH L. ANDERSON

17 DR. ROY E. ALBERT

18 DAVID G. HAWKINS

19 TODD M. JOSEPH

20 ROBERT G. KELLAM

21 DAVID R. PATRICK

22 DENISE A. THAL

23

24

25

1 TENTATIVE SCHEDULE OF PRESENTATIONS

2

SPEAKER	ORGANIZATION
Robert J. Rauch	Environmental Defense Fund
David Doniger	Natural Resources Defense Council
Roger Batchelor	American Industrial Health Council
George Dominguez	American Industrial Health Council
Robert Morgan	American Industrial Health Council
Harry Lieber	American Industrial Health Council
Gloria Rains	Manasota-88
Jesse Norris	Dow Chemical Company
Steve Swanson	American Petroleum Institute
John Thorpe	American Petroleum Institute
Harry Demopoulos	Individual
Joseph Cimino	Individual
Benjamin van Duuren	Individual
Somendu Majumdar	AIRCO
Arthur Gregory	Individual
Richard Leather	Dawn Mining Company
David Ridinger	Magma Copper Company
Edward Light	Appalachian Research & Defense Fund

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ATTENDEES

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Robert McFadden	Motor Veh. Manuf. Assn		862	3446
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M.J. SLOAN	SHELLOIL CO	202	296	362
D.G. Hancock	Bethlehem Steel Corp	215	694	5105
Martha E. Jones	Florida Phosphate Council			
James Hulme	American Cyanamid		789	1222
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Bill Westendorf	Monsanto Co			
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Karl J. Bourdeau	Individual	202 338 3694
Ron Fay	AIHC	914 725 149
George Hanks	Union Carbide	202 872 855
John Karl	Troy, Malio, Pollyea	828 1900
R G Rice	Jacobs Engineering Group	202 223 2944
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D D Doniger	NRDC	202 223 821
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Karen Morse	CDR	202 566 0640
Alex Mallison	Grumman Aerospace Corp	516 575 563
Barbara Turlan	Clement Associates	202 333 79
Bill McFarland	General Motors	313 575 861
Dan Maldonado	Regulatory Council	202 395 6110

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1 P R O C E E D I N G S

2 MR. BARBER: Good morning, I am Walter Barber and
3 I would like to welcome you to this informal hearing this
4 morning on EPA's proposed airborne carcinogen policy.
5 We have two days of hearings scheduled and we will
6 proceed in an informal manner with presentations by the
7 speakers and questions from the panel. We will not have
8 questions from the floor. However, if anyone would like
9 to have the panel pose a question, they may jot it down
10 on a note and present it to the panel and we will consider
11 questions in that manner.
12 We have asked the participants to try to keep
13 their oral presentations down to ten minutes in the
14 afternoon sessions. However, you will find the morning
15 sessions to be a little bit longer since we have granted
16 extended periods of time to the principal environmental
17 and industrial groups in hopes that that would reduce the
18 number of individuals making separate presentations.
19 We will have a rather tight schedule and I
20 believe everyone has a copy of the list of speakers. We
21 would expect to proceed through Mr. Lieber of the American
22 Industrial Health Council this morning and we will break
23 for lunch and then after lunch we will expect to start
24 with Ms. Gloria Rains.
25 At this time, the record for this hearing will

1 remain open for rebuttal of comments for 30 days after the
2 hearing, technically after the last hearing which is
3 scheduled for Wednesday in Houston. There is a possibility
4 that the overall proceeding, the public comment period on
5 the overall proceeding will be extended and if that is
6 decided, there will be an announcement in the Federal
7 Register in the coming weeks.
8 The first speaker this morning is Mr. Rauch
9 representing the Environmental Defense Fund.
10 MR. RAUCH: Thank you very much, Walt.
11 My name is Robert Rauch --
12 MR. BARBER: Bob, excuse me a moment, I have
13 committed a great faux pas. I am supposed to introduce
14 the members of the panel and I failed to do that.
15 Starting on my left, Mr. Robert Kellam on my
16 staff in North Carolina. Next to him, Dr. Roy Albert,
17 who is Chairman of the EPA Carcinogen Assessment Group.
18 To my left, Mr. David Hawkins, Assistant Administrator
19 for Air, Noise and Radiation, myself, Dr. Elizabeth
20 Anderson, Director of EPA's Office of Health and
21 Environmental Assessment, Mr. Todd Joseph, Office of
22 General Counsel, Mr. Dave Patrick who is on my staff
23 and in our Engineering Division and Ms. Denise Thal in
24 the Agency's Economic Analysis Division. Thank you.
25 MR. RAUCH: Thanks, Walt. I am a staff attorney

1 in the Washington office of the Environmental Defense Fund,
2 a national nonprofit organization devoted to the
3 preservation and improvement of the human and natural
4 environment.
5 We appreciate very much the opportunity to
6 appear here this morning to discuss the Agency's proposed
7 policy in regulating airborne carcinogens. If I recall
8 correctly, it was almost two years ago this month that the
9 first session was held on EDF's petition requesting EPA
10 to establish a policy to regulate airborne carcinogens.
11 In those two years, obviously a great deal of
12 effort has gone to putting this policy together and although
13 we feel it falls short of the requirements of the law in a
14 number of important respects, we do feel it is a significant
15 step forward and we congratulate the Agency in taking it
16 this far.
17 Before getting into the substance of my testimony
18 this morning, I would like to briefly explain how EDF
19 intends to organize its testimony and also comment a bit
20 on the problems presented by a hearing of this sort.
21 First of all, I intend this morning to deal
22 principally with the legal and policy issues raised by
23 the EPA proposal. Tomorrow morning, Dr. Joseph Wagoner
24 of our staff will address some of the scientific issues
25 which underlie the proposed policy.

1 EDF had hoped to put on additional technical
2 witnesses beyond Dr. Wagoner. Unfortunately at the present
3 time, we lack the funds to do that. Needless to say, that
4 would not be such a terrible thing if other groups or indeed
5 the Agency itself were prepared to put on some of the
6 technical witnesses whom we feel should be called to
7 address the many arguments which we expect from industry
8 witnesses, both today and tomorrow.
9 Unfortunately, neither EPA nor the other groups
10 in the environmental community that I am aware of plan to
11 do that. As a result, this hearing for the next two days
12 is going to be dominated by testimony from industry
13 witnesses.
14 These witnesses obviously are not going to have
15 a great deal of interest in supporting EPA's scientific
16 rationale for the policy. I think what you are going to
17 see by the end of those two days is a highly imbalanced
18 record, a record which raises serious questions as to
19 whether it will satisfy due process requirements.
20 The opportunity to appear at a public hearing
21 and present witnesses has little meaning, we submit, if
22 a significant segment of the public lacks the resources
23 to take advantage of that opportunity. Furthermore, sound
24 public policy cannot be made if the process which leads to
25 the formation of that policy is dominated by one set of

1 interests.

2 For these reasons, we feel that EPA has a
3 responsibility to seek expert testimony from knowledgable
4 persons who are capable of addressing the scientific issues
5 which will be raised by industry witnesses today and
6 tomorrow.

7 I should just point out that EDF and also the
8 Natural Resources Defense Council has requested EPA to
9 consider this informally. We would now like to make that
10 request formal and we suggest that you consider scheduling
11 at least another day of hearings to specifically take
12 testimony from experts within the National Cancer Institute
13 and the other scientific institutes which the government
14 relies on in developing policy in this area.

15 What EPA cannot ignore is that you are not making
16 this decision in a vacuum. Indeed, two years ago this month,
17 you informed us that action would be deferred on the EDF
18 proposal because you wanted to await the results of hearings
19 being held by the Occupational Safety and Health
20 Administration.

21 Those hearings have been completed, a very
22 extensive record has been developed. Unfortunately, the
23 Agency so far has chosen not to take advantage of the
24 expertise developed in those proceedings. We feel very
25 strongly that in order to develop a record which will make

1 this rulemaking defensible, should it ultimately be litigated
2 it is essential that you hear from these individuals.
3 To do any less will virtually ensure that these
4 proceedings are biased in favor of those who seek to
5 discredit the scientific basis for your proposal. In
6 short, EPA simply can no longer close its eyes to the
7 enormous gap in resources available to groups such as
8 the Environmental Defense Fund and those available to
9 groups such as those from which you will shortly hear.
10 In our judgment, the Agency has an affirmative
11 responsibility to rectify this imbalance to the best of its
12 ability. Having said that, I would now like to turn to the
13 key policy choices which confront the Agency in this
14 rulemaking.
15 To make my testimony as useful as possible to
16 the Agency, what I am going to do is review some of the
17 key arguments made by industry witnesses in their written
18 submissions. As you know, we had requested the Agency to
19 defer this hearing until such time as written comments have
20 been submitted with the hope that this hearing could then
21 serve as a forum for a discussion among the various parties
22 as to the points raised in the written comments.
23 Principally, I am going to be using the comments
24 submitted by the American Industrial Health Council as the
25 focus of our remarks. I will also, at the appropriate time,

1 indicate where EDF feels that the EPA proposal falls short
2 of the requirements of the lawl
3 The first major issue raised by industry
4 commentators, particularly AIHC, is EPA's alleged lack
5 of authority to issue a generic policy governing the
6 regulation of airborne carcinogens under Section 112.
7 Essentially, AIHC has argued that Section 112 contemplates
8 the regulation of carcinogens solely on a case-by-case
9 basis and furthermore that Section 112 precludes the
10 regulation of a large number of hazardous air pollutants.
11 Needless to say, the most recent guidance we have
12 from Congress on the subject is that contained in the
13 legislative history for the 1977 amendments. We would
14 suggest that that legislative history would argue that
15 precisely the opposite conclusion should be drawn from
16 that drawn by the AIHC.
17 Indeed, the principal concern of Congress appears
18 to be that the Agency's efforts to regulate airborne
19 carcinogens and other hazardous air pollutants had almost
20 stalled and the Agency's efforts were in a state of almost
21 total paralysis.
22 So Congress proposed some very drastic steps.
23 They listed six specific carcinogens, four in number, which
24 they directed the Agency to regulate unless the Administrator
25 made a positive finding of safety. But they went beyond

1 that.

2 They pointed out, this is from the House report,
3 "On the other hand, there are numerous other air pollutants
4 which to date have not been subject to regulation under the
5 Clean Air Act. Despite mounting evidence that these
6 pollutants are associated with serious health hazards
7 and despite recommendations by prestigious medical and
8 scientific bodies, the Agency has failed to promulgate
9 regulations to institute adequate control measures for
10 these unregulated pollutants."

11 It went on further to point out that the
12 specification of the four pollutants to which I refer,
13 "Does not affect EPA's authority or duty to regulate any
14 other presently unregulated air pollutant besides the four
15 named pollutants."

16 I don't want to belabor the Agency's record in
17 this area, I think it is well-known to most of us in this
18 room. That record has not been one that has been
19 particularly encouraging. Section 112 has been in the
20 Act for ten years now and in that time four carcinogens
21 have been regulated.

22 The Agency in 1976 commissioned a study by the
23 Mitre Corporation which identified approximately 100 to 125
24 substances released into the air in substantial quantities
25 that pose a risk of cancer. Clearly, the Agency must begin

1 to respond to that concern expressed by Congress and we
2 would argue that a generic policy which is precisely this
3 kind of thing Congress had in mind to accelerate the process
4 of regulation.
5 To do that, EDF submits that the Agency, as part
6 of its proposed policy, must make a commitment to regulate
7 a minimum number of airborne carcinogens each year. The
8 carcinogen assessment group has been working on a list of
9 approximately 43 for the past year.
10 That is a good start but by the close of this
11 rulemaking, EDF and NRDS will be submitting to you two lists,
12 one of substances for immediate listing under Section 112
13 and another for substances which need possible regulation
14 under Section 111 or additional research which will go
15 considerably beyond the 43 you now have under consideration.
16 Without such a list, we submit that the chances of
17 making significant headway under the policy are minimal.
18 It is one thing to have a policy in hand, a piece of paper
19 that outlines what you will do in evaluating chemicals. It
20 is quite another to make a commitment to use that policy.
21 We feel that a list of this sort is absolutely
22 essential to make sure that this policy is actually
23 implemented and we feel that that conforms with the will
24 of Congress as expressed in the 1977 legislative history.
25 Let me go on to the second major issue raised by

1 the AIHC and which I suspect will be the subject of much
2 discussion today and that is the appropriate evidentiary
3 threshold for listing of a carcinogen under Section 112.
4 AIHC has argued that the criteria proposed by EPA for the
5 listing of an airborne carcinogen are inadequate and do not
6 meet the statutory requirements.
7 AIHC argues that the Administrator can only list
8 a pollutant after performing a detailed quantitative risk
9 assessment and evaluating all of the evidence, both pro and
10 con, on a candidate's carcinogenicity. Once again, I submit
11 that this high hurdle of the criteria for listing is
12 unsupported by the statute or the legislative history.
13 Once again, both go in the other direction. The
14 legislative history suggests once again that Congress was
15 anxious to expedite the procedure for listing hazardous
16 pollutants and if anything, wanted to lower the burden
17 for listing such pollutants.
18 This is demonstrated most clearly by the change
19 in the language in Section 112 to reduce the evidentiary
20 burden on the Administrator for listing or regulating a
21 112 pollutant. Congress replaced the prior "may cause or
22 contribute to" language with the new language which states
23 that the Administrator should list any pollutant "which in h
24 judgment causes or contributes to air pollution which may
25 be reasonably anticipated to endanger public health or

1 welfare."

2 According to the House report, this change in
3 language was intended to "emphasize the preventive or
4 precautionary nature of the Act, i.e., to assure that
5 regulatory action can effectively prevent harm before
6 it occurs, to emphasize the predominant value of protection
7 of public health."

8 This change, according to the House was also
9 intended to "reflect awareness of the uncertainties and
10 limitations in the data which will be available to the
11 Administrator in the foreseeable future...because of the
12 limitations on research, resources and the fact that
13 decision making about the risk to public health from
14 air pollution falls on the 'frontiers of scientific and
15 medical knowledge'".

16 AIHC is correct in stating that you can't rely
17 on crystal ball inquiry, this is the language they quote.
18 But we submit this is hardly equivalent to a requirement
19 that you prepare a detailed quantitative risk assessment
20 prior to listing of a pollutant under Section 112.

21 As Mr. Doniger of NRDC who follows me will go
22 into more detail, the environmental community has very
23 serious reservations about the utility of quantitative
24 risk assessments in this whole process. Needless to say,
25 those reservations are sufficiently compelling that you

1 should not feel obligated to do such a detailed risk
2 assessment at this juncture.
3 Indeed, we would argue that the very uncertainties
4 and limitations in the available evidence to which the House
5 was referring preclude the development of quantitative risk
6 assessments which are sufficiently reliable to be truly
7 useful in the listing decision.
8 Contrary again to AIHC's assertions, the
9 legislative history makes clear that the thrust of the
10 1977 legislative changes was intended to give the
11 Administrator more discretion, not less, in determining
12 which substances to regulate under Section 112.
13 There is certainly nothing in the legislative
14 history of the '70 amendments or the '77 to suggest that
15 you must perform a quantitative risk assessment prior to
16 a listing decision. In fact, there is a great deal of
17 material in the House report reciting Congress' concerns
18 with the original ethyl decision.
19 You will recall that decision put quite a
20 substantial burden of proof on the Agency before moving
21 against a pollutant. That case originally held that you
22 must show a significant health hazard before you can
23 regulate something.
24 The House reacted, the Congress reacted
25 specifically to that case and chose to adopt the position

1 taken on rehearing. In doing so, it emphasized the need to
2 give the Administrator "a substantial element of judgment"
3 in determining the criteria for listing under Section 112.
4 Perhaps the most significant point here and why
5 quantitative risk assessment is not required prior to
6 listing a pollutant is the fact that quantitative risk
7 assessment cannot take into account synergistic or cumulative
8 impacts from airborne carcinogens.
9 By its very nature, quantitative risk assessment
10 looks at pollutants in isolation. This is significant
11 because the House report once again recognized specifically
12 the importance of "consideration of cumulative or synergistic
13 effects of multiple pollutants" in determining the hazard
14 posed by those pollutants.
15 As to AIHC's argument that not only must you do
16 a quantitative risk assessment, but you also must
17 systematically analyze all of the evidence, both pro and
18 con, before listing, two points are worth noting.
19 First, the criteria proposed by EPA have been the
20 subject of exhaustive scientific evaluation in a recent
21 proceeding conducted by the Occupational Safety and Health
22 Asministration. During this proceeding, OSHA heard from
23 dozens of witnesses on the reliability of using criteria
24 such as those proposed by EPA for determining the
25 carcinogenicity of specific substances.

1 At this point, EDF requests that pertinent
2 portions of the transcript of those proceedings be included
3 in the record. Furthermore, we submit that AIHC and others
4 have already had a full and fair opportunity to rebut those
5 criteria.
6 EPA is not required to reevaluate all of those
7 same arguments again. Indeed, we suggest that the Agency
8 entertain in this proceeding only new evidence which has
9 come to light since the close of the OSHA proceedings.
10 To do otherwise, that is to reopen all of the issues once
11 again, would involve a tremendous waste of the Agency's
12 resources and is unlikely to produce any significant new
13 insights.
14 Finally, it should be noted that the alleged
15 negative evidence of carcinogenicity which AIHC would have
16 you examine does not establish the safety of a particular
17 candidate for listing under Section 112. As the preamble
18 to the OSHA rulemaking notes, most animal tests used to
19 determine carcinogenicity are so insensitive that negative
20 results have little or no meaning.
21 This is equally true, I would note, for the
22 results of epidemiology. We simply do not know enough to
23 say that negative studies assure safety and therefore the
24 fact that they may be available does not mean, in the
25 presence of other positive evidence, that the candidates

1 should not be listed.

2 Even if the EPA proposal is adopted, opponents
3 of listing will be free to argue that a study relied on
4 by EPA is not scientifically adequate because of poor
5 methodology or other reasons. Finally, should new research
6 concerning the etiology of cancer uncover evidence which
7 suggests that the qualitative criteria for listing included
8 in the EPA proposal are no longer scientifically valid,
9 industry may obviously petition you to reopen this policy
10 and we would submit that is the appropriate procedure.

11 For now, however, it is clear from the OSHA
12 proceedings that the criteria contained in the EPA proposal
13 are scientifically sound and have the support of the vast
14 majority of scientists working in this area. If a
15 carcinogen listed in 112 meets that criteria, we would
16 suggest that negative evidence to the contrary should
17 not change that decision.

18 The next issue raised by AIHC goes to EPA's
19 authority to promulgate interim generic standards for
20 certain categories of hazardous air pollutants. Basically,
21 AIHC's position is that EPA lacks authority to (1) promulgate
22 interim standards as part of a two-stage regulatory process
23 and second, lacks authority to impose work practice or
24 design standards without making a case-by-case
25 determination of the need for such standards.

1 The thrust of AIHC's first argument is that
2 there is nothing in Section 112 which specifically
3 authorizes a two-phased regulatory approach to airborne
4 carcinogens. Needless to say, it is equally true that
5 there is nothing in Section 112 which prohibits it.
6 Indeed, the only requirement in Section 112 is
7 that whatever regulations you proposed be proposed within
8 180 days of listing and of course that they meet the ample
9 margin of safety requirement. Significantly, the Act does
10 not limit the Administrator to a single proposed
11 regulation but speaks of regulations in the plural.
12 Even more significant, Section 112(e)(1), added
13 by the 1977 amendments, gives the Administrator the
14 authority to use a combination of "design, equipment,
15 work practice, or operational standards where it is not
16 feasible to prescribe or enforce an emission standard which
17 will protect the public health with an ample margin of
18 safety."
19 In other words, Section 112(e)(1) specifically
20 contemplates the use of emission standards for some sources
21 within a facility and the use of work practices or design
22 standards for other sources within the same facility. This
23 would suggest a dual rulemaking, one can consider those
24 sources which should be covered by emission standards and
25 those to consider those sources which must be covered by

1 work practices or design standards.

2 In short, the Act does not restrict the Agency to
3 a single rulemaking. The only restriction it imposes is
4 the 180 day deadline. Given the emphasis in the legislative
5 history on providing the Administrator with broad discretion
6 in implementing Section 112 and the lack of any legislative
7 history precluding the use of a two-phased approach, we
8 submit that you are on sound legal ground in proposing
9 the interim standard two-phased approach.

10 As to AIHC's argument that such interim design
11 standards do not meet the statutory criterion of protecting
12 public health with an ample margin of safety, EDF agrees
13 with this assertion. AIHC is absolutely correct that any
14 interim standard covering specific sources of emission
15 should meet the full statutory requirements.

16 In our comments, we have urged EPA to upgrade
17 the standards to cover all those sources of emissions where
18 final control requirements can be specified in advance and
19 are not likely to be the subject of considerable controversy.
20 This is obviously a significant change from what you
21 proposed.

22 Essentially you propose only those requirement
23 that involve little or no capital expenditure be included
24 in the interim rulemaking. We think this is a mistake.
25 We suggest that what this may actually lead to in a number

1 cases is controls less stringent than those you would apply
2 to hydrocarbon sources which are precursors to ozone.
3 You will find yourselves in the anomalous position
4 of requiring, at least in the interim, less control for
5 carcinogens than you will simply for hydrocarbon precursors.
6 A clear example is your requirement that tanks be painted
7 white as part of the interim design standards.
8 You are now considering elsewhere minimal
9 requirements of the use of floating roofs and in some
10 cases, pressurized storage. I would submit it would be
11 foolhardy not to require this at this initial stage. The
12 key that you should address, the key question is, has the
13 technology been adequately defined, the best technology
14 adequately defined so that there is little likelihood of
15 controversy in a second rulemaking proceeding?
16 If there is little likelihood of controversy, in
17 other words if it is not likely that you will improve on a
18 floating roof, then let's do it up front in the first
19 proceeding. What we suggest that the standard the Agency
20 use here to differentiate between the first and second line
21 of controls is one of no duplicative investment.
22 What you want to avoid is requiring something in
23 the first phase that later you will go back and say, well,
24 that's not good enough. We want something more now.
25 Because, quite honestly, industry has a legitimate complaint

1 You are changing the rules on them all the time
2 and that's not fair. It's not fair to them. We suggest
3 for certain sources, particularly the fugitive sources,
4 the storage and handling sources, that you can specify
5 much of this technology with a good degree of assurance
6 that you will have identified the best technology in the
7 first proceedings and we would urge you to consider those
8 steps.
9 Finally, AIHC has argued that EPA cannot
10 establish generic work practice or design standards but
11 rather must establish the need for such alternative
12 approaches on a case-by-case basis. Once again, we
13 suggest, AIHC has simply misread the statute.
14 Section 112(e)(1) gives the Administrator
15 specific authority to establish design or work practice
16 standards where "it is not feasible to prescribe or enforce
17 an emission standard" which will protect the public with an
18 ample margin of safety.
19 Significantly, he may establish such work
20 practice or design standards either for a hazardous
21 pollutant or pollutants. He is not limited to establishing
22 them for one pollutant at a time. There is no indication
23 that he cannot establish such standards for more than one
24 pollutant if the pollutants that he is looking at share
25 similar characteristics.

1 That is that as a group, they are not capable of
2 being controlled by an emission standard. That is the test
3 you must address. If you can show that that group, and I
4 think you have properly identified that group, synthetic
5 organic chemicals, if it meets his criteria as a group,
6 you are free to move ahead with a single rulemaking.
7 In other words, the key test is whether each
8 pollutant satisfies the statutory criteria. You can make
9 that determination from a generic proceeding just as well
10 as you can from a case-by-case proceeding. Here, EPA has
11 limited the use of work practice of design standards to
12 those emission points which either are not intended emission
13 points, in other words, they are leak sources, or where the
14 application of measurement methodology is not practicable
15 due to technologic or economic limitations.
16 Naturally, if AIHC believes that industry is
17 capable of quantifying fugitive emissions from thousands
18 of potential leak points through the use of sophisticated
19 monitoring devices, EDF would be happy to endorse a proposal
20 for emission standards.
21 I should probably note that when I have raised
22 this possibility in the past to members of the industry,
23 it has generally been rejected categorically as simply
24 being too expensive.
25 In short, EPA clearly has the authority under

1 Section 112(e)(1) to propose and promulgate generic work
2 practice standards where the standards apply to pollutants
3 which are emitted from similar emission points. As we
4 indicate in our comments, EPA does believe that improvements
5 are possible in the interim standards proposed by EPA to
6 deal with fugitive emissions.
7 I am not going to go into those in detail because
8 I don't want to take up too much more of your time. But I
9 will note that we do think that industry deserves somewhat
10 more flexibility in meeting those requirements if, and only
11 on this condition, industry is willing to meet very specific
12 performance standards.
13 What I have in mind is a system where, assuming
14 EPA has sufficient enforcement resources, and this is
15 obviously a question that needs to be reviewed, inspectors
16 go into a facility and industry would be allowed a certain
17 number of leaks of a certain concentration.
18 For example, you might have a performance standard
19 which specifies that of all the points tested, less than
20 2 percent could have leaks in excess of 10,000 ppm. Or,
21 perhaps, for leaks less than 5,000 ppm, it might be a little
22 higher.
23 In other words, the point would be that a graded
24 scale be established taking concentration and percentage of
25 leaks into account. The EPA inspector could pick out at

1 random 100 points, 500 points, however many, 100 is probably
2 a minimum, statistical sample, and check it.
3 If industry failed to meet the performance
4 standard, the Agency would have the option of requiring
5 much more detailed reporting requirements on a regular
6 basis such as you suggest. Obviously to establish the
7 performance standards, you will need a great deal of
8 baseline information and we have urged you in our comments
9 to use Section 114 to begin compiling that information as
10 soon as you can.
11 This is an experiment admittedly and if it doesn't
12 work, you are certainly free to return to what you propose.
13 But I think a combination of performance standards with
14 standards that continually get tighter as time goes on
15 working towards a zero emission control is clearly an
16 approach worth trying.
17 Now I get to, without question, the most
18 controversial element of this rulemaking. That is the
19 control requirements for airborne carcinogens, what indeed
20 is really required by Section 112.
21 Unfortunately, it is here that I am afraid we
22 have the greatest disagreement with the Agency. The Agency
23 has taken the position that it can consider economic and
24 technical feasibility in setting emission standards.
25 Interestingly, the AIHC has little to say on this point,

1 I would suspect largely because they agree with you.

2 However, you cannot ignore the legislative history

3 of Section 112 as well as the applicable case law which I am

4 convinced demonstrates that you are wrong, that you are in

5 error on this point and that you are exposing yourselves to

6 considerable legal liability if you do not change this.

7 I would like to review that evidence for you.

8 I hope you will question the AIHC witnesses closely on

9 these points as well.

10 Essentially, the Agency has proposed a two-step

11 process for determining control requirements under Section

12 112. First, you have indicated that you will determine

13 what you call best available technology for both new and

14 existing sources after considering the economic, energy

15 and environmental impacts of various control options.

16 For new sources, the Agency states "for

17 practicable purposes, this level of control . . . will,

18 as a minimum, be equivalent to that which would be selected

19 as the basis for a new source performance standard (NSPS)

20 under Section 111.

21 EPA candidly admits that the requirement of BAT

22 for new sources would consider economic feasibility and

23 would not preclude new construction.

24 For existing sources, the requirements are even

25 less exacting. In addition to considering these factors,

1 you will also look at technological problems associated
2 with retrofit and related differences in economic, energy
3 and environmental impacts.
4 In our judgment, this approach does not even come
5 close to conforming with the requirements of Section 112.
6 It is really only when you get to the second phase of your
7 proposal that you can arguably address the health phase
8 criteria in Section 112.
9 In the second phase, you propose to use the
10 results of detailed quantitative risk assessment to
11 determine whether the risk remaining after the application
12 of best available technology is unreasonable. You have not
13 spelled out what you consider to be an unreasonable risk.
14 What is clear, however, is that you do intend to
15 construe the statute as allowing you to accept a certain
16 number of cancer deaths. We submit that this stands in
17 sharp contrast to the statutory command that a standard
18 protect affected individuals with an ample margin of safety.
19 You have tried to defend this approach by
20 arguing that Congress never intended you to protect all
21 members of the population with an ample margin of safety.
22 To do so, you have states would require a zero emission
23 standard, something which is clearly anathema to the Agency.
24 Yet there is persuasive and we submit clear
25 evidence in the legislative history and applicable case

1 law that Congress did intend that you would protect each
2 individual exposed to carcinogens with an ample margin of
3 safety.
4 In other words, the margin of safety applies to
5 each person, not some vague group of people. This is made
6 explicit in the House report on the 1977 amendments. One
7 of the reasons for the change to the "reasonably anticipated"
8 standard was "to assure that the health of susceptible
9 individuals, as well as healthy adults, will be encompassed
10 in the term "public health", regardless of the section of
11 the Act under which the Administrator proceeds. That's a
12 direct quote.
13 Thus, EPA is in no position to argue that a
14 standard which protects the majority of the population
15 with an ample margin of safety is legally sufficient.
16 Congress clearly was concerned about all members of the
17 population including those who are especially susceptible
18 to air pollution and required that whatever standards were
19 set to protect these people provide the same ample margin
20 of safety as would be available to members of the general
21 population.
22 Second, it is absolutely clear from both the
23 legislative history of Section 112 and the applicable case
24 law that EPA does not have the authority to consider
25 economic and technical feasibility in setting Section 112

1 standards.

2 There are two cases decided just recently which

3 are directly on point. The first is Environmental Defense

4 Fund v. EPA, 598 F. 2d 62 (D.C. Cir. Nov. 3, 1978) and the

5 second, Hercules, Inc. v. EPA, 568 F.2d 91 (D.C. Cir. 1978).

6 Not surprisingly, both of these cases are completely ignored

7 by AIHC in its discussion of control requirements under

8 Section 112.

9 EPA's discussion is limited to a footnote in

10 which you say you do not think they are applicable. I

11 would suggest that your analysis is wrong. Although both

12 of these cases deal with challenges to EPA's interpretation

13 of Section 307 of the Clean Water Act, the court in both

14 cases viewed Section 112 of the Clean Air Act as the source

15 of its opinion.

16 Ironically, EPA itself is arguing in those cases

17 that it had no authority to consider economic or

18 technological feasibility in setting standards for

19 carcinogens. This was your own argument. The court

20 took a clear-cut position in favor of EPA and stated

21 that "Section 307(a)(4) directs EPA to set standards

22 providing an ample margin of safety without any mention

23 of feasibility criteria".

24 Perhaps most important, the court based this

25 judgment on its reading of Section 112 of the Clean Air

1 Act. The court concludes that "recognizing that certain
2 pollutants require special treatment because of risk to
3 health, Congress enacted Section 112, dealing with
4 hazardous pollutants, without provision for consideration
5 of feasibility."
6 To make doubly certain that there was no mistake,
7 the court concluded by agreeing with "numerous commentators"
8 that "considerations of technological and economic
9 feasibility do not play a part in standard setting for
10 toxic substances.
11 EDF submits that nothing could be clearer. This
12 is the same court speaking that will review whatever
13 standard you come out with. Unless the court is to rewrite
14 its own opinion in these two cases, you will almost certainly
15 lose unless you change your policy on this position.
16 In reaching its conclusion, the court explicitly
17 recognized that technology may not presently exist which
18 will reduce levels of carcinogens to levels low enough to
19 provide an ample margin of safety, that is, zero emissions.
20 The court noted that the Supreme Court in Train v. NRDC,
21 421 U.S. 60, 91 (1975) determined that:
22 ... certain sections of the Clean Air Act impose
23 requirements of a "technology-forcing character". In
24 Union Electric Co. v. EPA, 427 U.S. 246, 257, 96 S. Ct.
25 2518, 49 L. Ed. 2d 474 (1976), the Court elaborated that

1 such statutes "are expressly designed to force regulated
2 sources to develop pollution control devices that might at
3 the time appear to be economically or technologically
4 infeasible.
5 In short, Congress and the courts clearly
6 understood that in setting standards based on health
7 considerations -- it is not as AIHC suggested that Congress
8 never contemplated the situation, they clearly did, and the
9 courts clearly contemplated the situation.
10 What they said was that the Clean Air Act is
11 designed to be technology-forcing. If necessary, the
12 court has made it clear that such regulations could force
13 the closing of companies or even entire industries.
14 Contrary to AIHC's assertions that the most EPA could
15 do is promulgate a regulation which might close one company,
16 the D.C. Circuit in EDF v. EPA has held:
17 These cases demonstrate (referring to occupational
18 health and safety cases) the inevitable tension attending
19 regulation of carcinogens. Frequently, such regulations
20 have severe conomic impact. Indeed, sometimes, as alleged
21 by industry petitioners in this case, such regulations may
22 jeopardize plants or whole industries and the jobs depending
23 on them.
24 In such circumstances, the temptation to demand
25 that the Agency furnish conclusive proof of carcinogenicity

1 as support for the regulations is great. However, the
2 decision to delegate authority to an agency to control
3 suspected carcinogens is a legislative judgment that is
4 not open to question in this court . . . If regulation
5 were withheld until the danger was demonstration
6 conclusively, untold injury to public health could result.
7 Accordingly, we find that Congress has allowed
8 EPA to support a prohibition on the basis of strongly
9 contested and merely suggestive proof. Let me emphasize
10 that EDF is advocating this morning that we close those
11 industries that would be affected by regulation under
12 Section 112.
13 We have suggested an alternative approach to the
14 Agency which it has not accepted up to this point. That
15 approach is to determine first of all whether substitutes
16 are available. If they are not, to determine whether
17 currently available technology can achieve a no measure
18 emissions level.
19 If neither of those conditions can be met, then
20 we feel the Agency is authorized to require best available
21 current technology, but must provide for a continued phased
22 reduction for emissions towards the no measured emissions
23 level.
24 We would submit to you that this is the maximum
25 flexibility that you are given under Section 112. In other

1 words, your flexibility goes to the timing and implementation
2 of a no measured emission standard, it does not go to the
3 discretion to say you simply will not establish such a
4 standard ever.
5 Case law is clear and I would urge you to
6 reconsider your position.
7 Finally, I would like to very briefly go into
8 the control requirements you have proposed for new sources
9 of carcinogenic emissions. We would submit that the approach
10 required is similar to that which I just outlined for
11 existing sources.
12 In other words, you should move towards standards
13 which will give us the no measured emissions level. In
14 existing sites, you have proposed a very modified offset
15 policy. This is something we suggested to you initially
16 in the vinyl chloride rulemaking which you have now taken
17 final action on.
18 But we are troubled by one particular aspect of
19 your proposal and that is that you would allow tradeoffs
20 to be made between different hazardous air pollutants rather
21 than limiting it to the same pollutant being emitted at the
22 existing facility.
23 We don't feel that this is authorized and I would
24 just point out in passing to AIHC that their complaint that
25 the offset policy is not specifically authorized by Section

1 112 strikes me as a rather peculiar one. The offset policy
2 if anything is a compromise designed to allow some expansion
3 under the existing law.
4 The industry is quite familiar with the process
5 EPA went through to develop this policy for sources of
6 Section 109 pollutants and it strikes me as very peculiar
7 that you would argue that it could not be used here. The
8 alternative is a simple prohibition on expansion which I
9 am not sure any of us are anxious to support.
10 In conclusion, although the proposed EPA policy
11 represents a significant step forward, it still falls
12 considerably short of the requirements of Section 112 in
13 a number of important respects. Let me emphasize this to
14 you, and I mean this sincerely, we are not anxious to
15 litigate this policy.
16 Those of you sitting here have better ways to
17 spend your time than litigating that policy. I certainly
18 have better ways to spend my time. But I would urge you
19 again not to go forward with a policy which is clearly
20 illegal in certain respects because you then give us
21 little choice.
22 We are anxious to work with the Agency on this
23 and I would suggest that we are also willing to engage in
24 further discussions with industry representatives who may
25 wish to do so. We believe there are companies within the

1 chemical industry who recognize the threat posed by
2 airborne carcinogens and are willing to take immediate
3 steps to control that threat.
4 It is our sincere hope that those voices within
5 the industry urging moderation will not be drowned out by
6 those who would fight EPA at every turn, even to the point
7 of refusing to acknowledge the hazard posed by airborne
8 carcinogens.
9 A good many industry witnesses are going to
10 follow me today. There are clearly two avenues available.
11 One is to fight this at every turn. The other is to
12 acknowledge that we have a problem before us for which
13 solutions are available.
14 It is a question of how we are all going to use
15 our own resources. We hope that out of this proceeding
16 will come agreement that we will move forward rather than
17 fighting for another ten years to regulate another four
18 carcinogens.
19 Thank you very much.
20 MR. BARBER: Thank you. I suspect we will have
21 several questions. I have a few to start with and then I
22 will go to the other panel members.
23 In part of your discussion, you mention that 112,
24 like Section 307, does not provide for considering economics
25 and feasibility in establishing the standards. To the best

1 of my knowledge, we don't use Section 307 very extensively.
2 In fact, the Agency and environmental community have agreed
3 pretty much not to use 307 as the principal regulatory
4 mechanism for dealing with waterborne carcinogens.
5 Wouldn't you suspect that if we forced Section 112
6 to be the ultimate regulatory threat without any sense of
7 economic or feasibility consideration that we are relegated
8 to the same kind of position, to be used only when we have
9 the most direct kind of threat, most direct kinds of
10 epidemiological evidence?
11 MR. RAUCH: No, I don't, and I will try to explain
12 why. The way we have construed the statute does allow you
13 to require less than a no measured emissions standard
14 immediately. In other words, we can contemplate standards
15 based on best available current technology which would go
16 into effect in the short run which would be followed by
17 continuing phased reductions, particularly to allow industry
18 time to develop process changes. This is something that we
19 think is a key and has not been given sufficient attention.
20 Given that approach, we think 112 can be used as
21 the primary vehicle for registering carcinogens. Indeed,
22 we would suggest that that is clearly what Congress
23 contemplated and to attempt to use other provisions
24 such as 111 or 110 would not meet the requirements in
25 112 itself which specifically address irreversible health

1 effects.

2 What I am suggesting to you all is that a system

3 can be developed which will incorporate some of the features

4 of the 307 settlement but will also keep the statutory

5 integrity of Section 112 and allow you to move towards

6 meeting the requirements of that section.

7 MR. BARBER: That system is to establish a zero

8 goal and establish some interim best available control

9 technology and how does one define best available control

10 technology absent --

11 MR. RAUCH: I guess I am suggesting that assuming

12 you are willing to accept that concept in Section 112, at

13 that juncture, some consideration could be given to technical

14 feasibility, but recognizing that that is an interim control

15 measure and is not the last step in the process.

16 MR. BARBER: The concern I would have is that when

17 you talk about no measurable emissions, in the technical

18 sense it is not a very meaningful construct.

19 MR. RAUCH: That's right, because the technology

20 to measure emissions changes with time and that's why I used

21 that phrase rather than zero because I'm sure those who

22 follow me will point out that it's not possible to measure

23 zero exactly.

24 MR. BARBER: But the anticipation of meeting no

25 measurable emissions is virtually zero based on no alternati

1 measurement defined by the Federal Government.

2 MR. RAUCH: The measuring would be specified and

3 then using that measure, no measured emissions. The method

4 could be changed at some point but I don't -- assuming that

5 which is reasonably sophisticated, we will have too much

6 trouble with that approach.

7 MR. BARBER: You can define a method sensitive to

8 one part per million or one-hundredth part per million or a

9 thousandth. It seems to me it's another way around to the

10 same issue. There are going to be emissions if these

11 chemicals are manufactured, transported and handled and

12 when we talk about zero, we talk about something that

13 doesn't make sense to most engineers and it's very difficult

14 to rationalize the zero and the suggestion that you will

15 still have these products in the marketplace. As soon as

16 you move to a non-zero number, one tends to look at the

17 magnitude of that number as an opportunity for further

18 reduction.

19 MR. RAUCH: I think what's important to realize

20 is that how we define the ultimate end point is not nearly

21 as important as agreeing that we should at least be moving

22 in that direction and we will not simply stop at present

23 available technology.

24 Whether we in fact exactly reach it is something

25 for debate in most cases some years out but the importance

1 is the commitment to move towards that end point, regardless
2 of whether it is defined as zero emissions or no measured
3 emissions and keep industry on a track that will encourage
4 it to develop the technology, but most important, the
5 process changes, that will get us there.
6 The chemical industry, I think, has considerable
7 innovative ability. They have demonstrated that in the past
8 and it's really a question of whether you will propose a
9 policy which will harness those innovative capabilities.
10 We suggest this is one way to do it. That is at least our
11 view consistent with the statute.
12 MR. BARBER: There seems to me to be substantial
13 difference both in the nature of some of the chemicals we
14 are concerned with and the magnitude of the emissions
15 concerned with them. The evidence we have is highly
16 disparate in terms of quality and quantity.
17 However, from your statement, I would infer that
18 you would treat all of these chemicals the same, once they
19 were listed under Section 112. That there would not be any
20 sense of inability to regulate the adjustment response both
21 in terms of the evidence of the magnitude of the
22 environmental problem --
23 MR. RAUCH: I think I am suggesting that the
24 material you have outlined for the listing of a hazardous
25 air pollutant are sufficiently hazardous that the chance

1 of getting a pollutant registered and regulated as not
2 posing a hazard is minimal.
3 If you are going to assist, then we would suggest
4 you should list pollutants on the basis of a positive Ames
5 test, for example. Then I think your suggestion would have
6 considerable merit. I think given the insensitivity of the
7 test which we now have which we have established with
8 sufficiently high scientific thresholds, the list of a
9 carcinogen, that anything that falls within that category
10 does deserve to be regulated.
11 MR. BARBER: In developing the policy we talked
12 about establishing priorities for dealing with industrial
13 subcategories of certain types of sources, it is clear even
14 a casual perusal of the data that some sources are two and
15 three orders of magnitudes larger than other sources, both
16 in terms of total emissions and in terms of exposure to
17 people. Do you concur in our view that some sources are
18 much less deserving of regulatory consideration?
19 MR. RAUCH: Yes, I think there is room for
20 reasonable priority setting of a particular 112 pollutant.
21 MR. BARBER: However, once we decide to regulate
22 a source category as a pollutant, the suggestion is that
23 zero is the ultimate goal and some series of steps towards
24 that ought to be taken?
25 MR. RAUCH: Yes, and I would point out to you

1 that I think we are clearly looking at a system that would
2 contemplate phase downs that would at least attempt to allow
3 industry to amortize the investment it made prior to control
4 technology.
5 It might not always be possible but that would be
6 one of the factors we would consider. What we are seeking
7 is a system which attempts to protect the public health with
8 a higher degree of safety and at the same time is not going
9 to jeopardize the continued productivity of that segment of
10 industry subject to the standards. I think reasonable
11 timetables can be worked out but it requires a good faith
12 effort on both the part of industry and the government to
13 do it.
14 MR. BARBER: Thank you. I will turn to the panel
15 now. Mr. Hawkins?
16 MR. HAWKINS Bob, first a question on legal
17 interpretation of what you have identified as the critical
18 governing phrase in the statute of protecting public health
19 with an ample margin of safety. Especially on Pages 15 and
20 16 of your testimony, you go into the statutory language
21 and legislative history and applicable case law.
22 My question is whether that language you view as
23 something which -- clearly your testimony suggests that in
24 your view, we must focus on susceptible individuals and
25 protect their health as well as the aggregate health with

1 an ample margin of safety.

2 You quote language from those reports saying that
3 we must assure the health of susceptible individuals as well
4 as healthy adults. My question is, do you feel that the
5 Agency under the law can demonstrate that they are "assuring
6 the health of susceptible individuals" if a calculation can
7 be made that would show that for most exposed individuals
8 or most susceptible individuals there is some calculable,
9 finite risk other than zero, or do you feel that any
10 calculable, finite risk puts the Agency in the dilemma
11 of having -- I don't think it is as you stated in your
12 testimony, that it is accepting a certain number of cancer
13 deaths but it would be accepting some finite, positive
14 calculable risk.

15 MR. RAUCH: That is right and as you know, Dave,
16 the Agency's practice to this point has been to go through
17 a rather detailed quantitative risk assessment and calculate
18 the number of expected deaths from exposure to the suspected
19 chemical. Our reading of the legislative history is that
20 Congress intended there to be no deaths from exposure.

21 MR. HAWKINS: No risk of death?

22 MR. RAUCH: That that risk should ultimately be
23 eliminated, that's right. If you have flexibility, it's
24 a question of the timing in moving towards that goal.
25 Obviously you are accepting a certain amount of risk, but

1 we think that is the proper balance to strike.

2 MR. HAWKINS: The second question, assuming the

3 Agency does have an approach which evaluates levels of risk

4 for making interim or permanent decisions, there -- you can

5 have two different circumstances and I would like your views

6 on whether you have the same or different regulatory

7 implications.

8 You can have a situation where you have a large

9 number of individuals exposed to very low levels of risk

10 and the alternative situation that may produce the same

11 quantitative risk assessment number would be a very small

12 number of individuals exposed to a very high level of risk

13 or a relatively high level of risk compared to the former

14 situation. Do those have different regulatory implications

15 in your view?

16 MR. RAUCH: Ultimately,I would say no. I think

17 you have the same responsibility to both groups. In terms

18 of determining your priority of proceeding, they may. Again

19 our hope would be that you would not have to choose between

20 two chemicals but you would look at your list of chemicals

21 that you have identified for listing and pick out those

22 situations, both of the low risk but large exposure type

23 you are talking about as well as the low number of people

24 but high risk and group them in your own judgment as to

25 priorities for action.

1 They would not necessarily say we could always
2 choose one or the other, although I would suggest that if
3 you do find a chemical which poses a high risk, and of
4 course all of this assumes that the quantitative risk
5 assessment is sufficiently advanced that you can reliably
6 say that which we question.
7 But assuming that you did make that judgment,
8 we would certainly want to move expeditiously to protect
9 those people at high risk. Again, I don't think it changes
10 the ultimate regulatory responsibility to both of those.
11 MR. HAWKINS: Thank you.
12 MR. BARBER: Dr. Albert?
13 DR. ALBERT: No.
14 MR. BARBER: Bob?
15 MR. KELLAM: I just have one question, Mr. Rauch.
16 You mentioned that EDF would consider as consistent with
17 Section 112 phased control or technology forcing regulation.
18 In the policy, I am sure you are aware we talk about the
19 periodic review of emission standards and the consideration
20 of information that has become available in the interim.
21 Basically, what I would like to ask you is to
22 comment on the distinction you see between what is described
23 in the policy and what you are suggesting as a technology
24 forcing regulation.
25 MR. RAUCH: Bob, I think there is a very major

1 distinction and a distinction that has been at issue before
2 us in the vinyl chloride proceeding. You know as well as I
3 do that the Agency is constantly pressed to deal with new
4 problems as they arise and I don't think it would be much
5 different here.
6 A simple commitment to go back and review standard
7 periodically is not sufficient in my judgment to harness the
8 energy for industry to begin improving the technology. I
9 will give you an example. For sometime, the State of Texas
10 was considering a very tough vinyl chloride standard and we
11 have information that suggests the industry was quite worrie
12 and immediately went to firms working on relatively
13 innovative technology and asked them to do some work on
14 meeting the new standard.
15 That work started and it was discontinued when
16 it became clear that Texas did not intend to go forward.
17 What is essentially to harness these energies is that they
18 have a standard to shoot at that lies up there on the
19 horizon.
20 Further, they used reduction, your commitment
21 to re-review is simply not enough.
22 MR. BARBER: Dr. Albert has a question.
23 DR. ALBERT: When you advocate the reduction of
24 emissions to the point of no measurable level, recognizing
25 that of course this does involve actual emissions because

1 of the limitation of the detection systems used and also
2 where the measurements are made raises the issue as to
3 whether this position does not constitute a recognition
4 that the philosophy or language of the law is not unworkable
5 when it talks about complete protection of everyone with an
6 ample margin when viewed in the light of the concept of a
7 non-threshold character of agents such as carcinogens.
8 Doesn't this raise the issue that your position
9 is your compromise to an essentially unworkable situation
10 posed by the law?
11 MR. RAUCH: Let me point out, first of all, that
12 if you will review the legislative history, Congress clearly
13 did understand that it was dealing with non-threshold
14 pollutants in 112 and it made it clear that the prohibition
15 was authorized and that is very clear in the legislative
16 history.
17 What I am suggesting, Dr. Albert, is not in the
18 interests of EPA or the nation as a whole to shut down a
19 significant portion of American industry, assuming that it
20 is -- necessary to meet a no measured level emission
21 standard.
22 Clearly, Congress did contemplate this but that
23 does not necessarily mean you have to do it. There may be
24 cases where it is necessary where certain companies simply
25 cannot cut the mustard. But we think this is a reasonable

1 reading of the law and one that will be upheld in the
2 Court of Appeals. I don't think the interpretation the
3 Agency has put on it right now will be upheld.
4 DR. ALBERT: Thank you.
5 MR. BARBER: Any other questions from the panel?
6 Mr. Joseph?
7 MR. JOSEPH: Bob, I just have one question. If
8 you read the ample margin of safety language of Section 112
9 to prohibit any consideration of technical or economic
10 factors, where then do you really find the authority to
11 proceed towards this zero emissions requirement phased
12 basis rather than immediately?
13 MR. RAUCH: Well, I think it is quite clear that
14 Congress has not specifically addressed the phased reduction
15 However, there are other statutes that do contemplate this
16 type of approach and I would suggest the OSHA approach and
17 the emergency temporary standard is an approach which you
18 have proposed in the vinyl chloride case which Congress has
19 had an opportunity to review and is aware of.
20 I think what it really comes down to is whether
21 this approach can reasonably be extracted from Section 112.
22 We clearly think it can and obviously in making that
23 judgment, you have to compare it to alternative approaches.
24 I don't think any of us will know for sure until it is
25 tested in court but I think this has the better chance

1 of a standard challenge than what you would propose.

2 MR. JOSEPH: I take it what you said seems to be

3 that the reason you work on a phased approach rather than

4 immediately is because of the technical or economic

5 impossibility of doing it right away?

6 MR. RAUCH: We don't know that for everything.

7 MR. JOSEPH: Where it is the case?

8 MR. RAUCH: That's right.

9 MR. BARBER: Thank you, Bob.

10 I would like to call the next speaker, Mr. David

11 Doniger.

12 MR. DONIGER: Thank you, I am David Doniger with

13 the Natural Resources Defense Council. I will have a

14 statement for you later in the day, it is not in final

15 form yet.

16 I want to thank the members of the industry

17 because without chemicals, my appearance here would not

18 be possible and I really hope that something we do today

19 will touch your life.

20 Since 1970, EPA has had an urgent responsibility

21 to protect the public from hazardous air pollutants that

22 cause cancer and other killing diseases as well and yet

23 in its first decade has only set four such standards.

24 NRDC believes that the policy under consideration today,

25 the rules being considered today, are a step in the right

1 direction because they show that EPA is finally awakening
2 to the cancer problem but there will have to be major
3 improvements in these rules if they are to meet the public
4 health needs mandated in the law.
5 Cancer is a very serious public health problem.
6 It is the second leading cause of death in this country.
7 One American in four can expect to get it and one in five
8 will die from it. The outlook is getting worse for cancer
9 rates overall and for the types most related to chemical
10 and radioactive pollutants.
11 We know the vast majority of these cases are
12 preventable, up to 90 percent are caused by risk factors
13 we can control and chemical and radioactive air pollutants
14 in concert with other factors, cause a substantial percentag
15 of these cases.
16 We know there are several hundred cancer-causing
17 substances released from industrial facilities into the air
18 and these are substances which are known to either cause
19 cancer in people, primarily in the work place or in animal
20 studies in the laboratory.
21 Now, a quantum leap in EPA action will be
22 necessary if it is going to control more than a token
23 handful of pollutants and actually take steps calculated
24 to solve the hazardous air pollutant problem, the total
25 problem, in a reasonable period.

1 NRDC is strongly in support of EPA's conclusion
2 that there are a lot of hazardous air pollutants out there
3 of concern and reasonably may be anticipated to cause cancer.
4 We also support EPA's conclusion that even low levels of
5 exposure to these pollutants increase the risk of people
6 who get cancer which, over a large enough number of people,
7 is almost certain to mean that more people will get cancer
8 and die and a no safe level of exposure to carcinogens can
9 yet be identified.
10 We support the commitment to list and regulate
11 many more hazardous air pollutants than EPA has to date,
12 although we believe EPA must make a legally binding
13 commitment to do so if it is to meet the mandate of
14 Section 112.
15 We support, and I won't say much about, the
16 generic standards approach. We have commented on those
17 in our written comments when you get them.
18 We support EPA in the commitment to look at
19 whether there are substitute products or process changes
20 completely eliminating carcinogenic emissions. In fact,
21 we think that's essential.
22 We think EPA is correct that because new plants
23 have greater flexibility than existing ones and because
24 expansion of reliance on carcinogenic chemicals should
25 not be encouraged, new plants should be subject to greater

1 emission control requirements than existing ones.

2 But there are critical areas where the proposed

3 policy falls short and I will summarize what NRDC believes

4 is the minimum of an effective program to meet the Section

5 112 mandate.

6 First, as I mentioned a moment ago, the program

7 must establish a legally binding process to screen, list

8 and control a meaningful number of hazardous air pollutants

9 each year.

10 Second, in order to set standards for each

11 hazardous air pollutant that will provide the public with

12 an ample margin of safety as required by law, EPA must

13 establish a presumption that no cancer-causing emissions

14 will be allowed and require industry to shoulder the burden

15 of showing why any emissions of cancer-causing substances

16 should be allowed to continue.

17 Let me talk for a minute about this process we

18 propose EPA adopt, a legally binding action forcing process

19 to screen, control and list a meaningful number of

20 pollutants. As I said a moment ago, you have the mandate

21 not just to control a token handful but to set up a program

22 calculated to solve the total hazardous air pollutant

23 problem in a reasonable period.

24 EPA has never undertaken any organized screening

25 of the potential hazardous air pollutants and has listed

1 only six and regulated only four. The process we are
2 proposing must contain two elements.
3 First, a candidate list of potential hazardous
4 air pollutants. These are substances reported to cause
5 cancer in human or animal studies, number one.
6 Number two, they may be emitted from stationary
7 sources.
8 Second, EPA must establish a legally binding
9 commitment to add a minimum number of candidate substances
10 to the hazardous air pollutants list each year, setting
11 standards for them, until the public is protected from
12 all hazardous air pollutants.
13 NRDC believes that the minimum number of
14 hazardous air pollutants that could be reasonably calculated
15 to meet the problem in any reasonable time is 20 each year.
16 Anything less would fail to guarantee even minimum adequate
17 progress.
18 Third, EPA should also establish an air pollutant
19 testing list and this testing would include pollutants
20 which EPA, in screening substances for the candidate list,
21 found insufficient health effects data. It would help
22 guide EPA, other agencies and private industry in
23 determining priorities for further testing.
24 EPA has gone out of its way to establish high
25 thresholds which must be hurdled before it will take action

1 on any substance. We are compelled to remind the Agency of
2 the essentially precautionary nature of the Clean Air Act.
3 Congress has emphasized in 1977 when it amended the
4 definition of a hazardous air pollutant that EPA must
5 take preventive action based not only on the well-establishe
6 fact, but on suggestive, probative, although not completely
7 certain information as well.
8 A hazardous air pollutant is one that may
9 reasonably be anticipated to result in an increase in
10 cancer and we don't think that it is necessary or rational
11 to use quantitative risk assessments, as has been proposed,
12 in the regulatory scheme.
13 Let me speak first about the level of control
14 that we believe an ample margin of safety requires. The
15 statute requires such a standard to be set within a year
16 of the listing of each pollutant. The proposed policy does
17 not provide for such standards, since no level of exposure
18 to cancer-causing air pollutant can be considered safe.
19 As long as these substances are emitted, the
20 best scientific knowledge of today indicates that some
21 people will get cancer as a result.
22 Section 112 establishes a presumption that one
23 year after listing a carcinogenic air pollutant, emissions
24 of that should be eliminated and that industry bears the
25 burden of showing why all emissions of such a substance

1 cannot be eliminated in this time period through emission
2 controls, process changes and product substitutions.
3 If an industry can show this, EPA should set an
4 interim standard that allows the minimum essions that cannot
5 be eliminated and includes a timetable for phase reductions
6 towards the goal of no emissions.
7 Once it is established that a pollutant is a
8 carcinogen and once it is placed on the hazardous air
9 pollutant list, the burden on industry under the law to
10 de-list it is to show clearly that it is not such a hazardous
11 air pollutant and we submit the burden must be on industry
12 to show why a zero standard cannot be achieved, not on EPA
13 to show what can be. The industry has no moral or legal
14 right to continue exposing people to life-threatening
15 hazards.
16 We point out to EPA that this approach would have
17 the advantage of placing the burden of producing information
18 on industry where it belongs and not on EPA. The risk of
19 non-cooperation is a zero emission standard coming into
20 effect at the end of the 360 days after listing.
21 It should then be possible to cover more
22 substances and more sources than EPA currently envisions.
23 In its proposal, EPA would take upon itself, totally
24 unnecessarily, the burden of producing the evidence
25 needed to justify even the minimum public health and

1 this is not what the law envisions.

2 Let me turn to what I believe are the arbitrary
3 uses of quantitative risk assessments contemplated in the
4 proposed policy. We think EPA has made and will continue
5 to make -- is proposing to continue to make unwarranted uses
6 of these unreliable methods that are neither required by the
7 Clean Air Act or a rational exercise of EPA's discretion.

8 However desirable quantitative accuracy may be
9 in principle, the scientific knowledge necessary for
10 reasonably reliable and precise estimates simply is not
11 yet available. Current knowledge about chemical and
12 radioactive processes of carcinogenesis is primarily
13 qualitative and not quantitative.

14 Although many carcinogens can be identified,
15 it is not currently possible to predict the size of the
16 risks they pose with anything near the precision and
17 reliability needed for the estimate to be useful in
18 making standard setting decisions.

19 There are many sources of uncertainly and we
20 have reviewed them in our written comments and we will be
21 submitting further data on those points in supplementary
22 comments. I would like to mention briefly what some of
23 the major ones are.

24 In animal studies there are severe problems in
25 making high to low dose extrapolations, reflecting the

1 scientific uncertainties about how cancer is caused, there
2 are numerous mathematic models for resulting high dose
3 results to lower doses people generally experience.
4 Although all of the models fit the high dose data
5 reasonably well, they yield widely divergent estimates of
6 the risks associated with low doses. In animals alone, the
7 major models differ by a factor of 100,000 on the dose
8 projected to cause 100 cancers in a million animals.
9 There are more uncertainties added from so-called
10 scaling factors which result from the need to extrapolate
11 across species lines. Rodents are smaller than humans and
12 live only two years. Their rate of metabolism and cell
13 division is faster and there is uncertainty whether the
14 right way to scale doses up to humans is on the basis of
15 relative body weights, relative surface areas, relative
16 life spans or some other basis.
17 Recent attempts to quantify the risk from
18 saccharin graphically illustrate the magnitude of the
19 uncertainty attributable to just these two factors,
20 modeling and scaling assumptions. NAC, National Academy
21 of Sciences, has concluded that in every 50 million people
22 who drink one can of diet soda per day, there could be as
23 few as 0.0007 cancers per year or as many as 3,640 and this
24 is a range of error more than 5 million-fold.
25 A recent study extended the range for saccharin

1 to more than 10 million-fold and estimates for the human
2 risk associated with vinyl chloride extend over a 1 million-
3 fold range.
4 There are further differences in species
5 sensitivity. It's not possible to tell at this point
6 whether the mice or rats being tested are more or less
7 sensitive than people and there are enough instances where
8 humans have turned out to be more sensitive to give great
9 pause to relying on animal data for quantitative conclusions
10 There are differences in human sensitivity among
11 the different individuals. People are genetically
12 heterogeneous while animals are bred for maximum homogeneity
13 As Bob mentioned, there are interactions and synergisms.
14 Animals are usually exposed to only one substance in
15 carefully controlled studies.
16 Humans, however, are exposed to a wide variety
17 of chemicals and radiation and other carcinogenic co-factors
18 in almost infinite combinations. Because of interaction and
19 synergism among these co-factors, exposure to a carcinogen
20 can result in much greater incidences to humans than
21 animals and there is good reason to expect that two or
22 more factors of synergism are common and yet most have
23 not even been identified, let alone quantified, because
24 there is little epidemiological data available.
25 Similar limitations prevent deriving reliable

1 quantitative estimates from epidemiological studies as well.
2 For one thing, there is such evidence on only a very few
3 substances. Beyond this, epidemiological studies are
4 relatively insensitive.
5 There are inherently high probabilities of
6 failing to detect a true effect at all and of misrepresenting
7 the magnitude of an effect even if it is identified. There
8 are confounding factors as a result of the uncontrolled
9 setting in which epidemiological studies take place.
10 Many well-designed studies would fail to detect
11 cancer caused by a chemical or radioactive substance unless
12 it is occurring at a rate five times higher than normal.
13 Even the best studies cannot reliably detect effects
14 occurring in less than 50 percent above their normal rate.
15 Long latency periods of 20 years or more are
16 common for human carcinogens. Most studies are not carried
17 out long enough to pick up all the latent effects a
18 chemical may have. If they were, it would be so long
19 before information was available that preventive action
20 would be impossible.
21 There is poor data on peoples' true exposures,
22 both in kind and amount.
23 The proposed policy would make extensive
24 reliance on exposure assessments, too, but by and large
25 the data for such assessments do not yet exist. We know

1 that substances are emitted often in million pound
2 quantities, but neither monitoring nor modeling of ambient
3 concentrations has been performed.
4 Monitoring will be very slow unless EPA devotes
5 substantially greater resources to it. Modeling, even for
6 conventional pollutants is not capable of making predictions
7 consistently within even a factor of two of monitored levels
8 Many carcinogenic substances are reactive and modeling for
9 reactive substances, such as photochemical oxidants, is
10 even less advanced.
11 When all the sources of uncertainties are
12 considered together, huge errors in risk estimates are
13 nearly unavoidable on a frequent basis.
14 The Agency's current fascination with quantitative
15 risk assessment methods finds no support in the Clean Air
16 Act, nothing in Section 122 or elsewhere in the Act imposes
17 the prerequisite of a quantitative risk assessment before
18 EPA may take steps to protect the public from a cancer-
19 causing substance.
20 Congress recognized that under the current state
21 of scientific knowledge, cancer hazards can be identified
22 long before their precise magnitude can be quantified.
23 The Congress did not require EPA to delay regulation
24 until this precise quantification was possible nor did
25 Congress force EPA to undertake sham quantitative analyses.

1 Despite the efforts of various commentators to
2 find talismanic code words in the Clean Air Act that they
3 believe require quantitative analysis, there is nothing in
4 the Act to support this.
5 EPA nonetheless proposes to use quantitative risk
6 assessment as the centerpiece of determining whether the
7 "residual risk" remaining from emissions allowed by a BAT
8 standard are "unreasonable". Paying lip service to these
9 uncertainties is nothing more.
10 A balancing of risks and benefits is not
11 authorized under Section 112 of the Clean Air Act. Even
12 if the proposed risk-benefit analysis were legal, the
13 imprecise quantitative risk assessments would be an abuse
14 of discretion.
15 Although NRDC strongly supports EPA's commitment
16 to consider whether emissions of a carcinogen can be
17 eliminated completely by substitutions and process changes,
18 indeed we believe such measures are required, we do not
19 believe the unreliable quantitative methods can play a
20 rational part in a decision whether certain emissions are
21 reasonable.
22 The likelihood of huge errors means that EPA
23 has adopted only an illusion of precision. While
24 quantitative accuracy is desirable, false precision is
25 arbitrary.

1 Even if it were legal and rational to use
2 quantitative risk assessments in this way, the proposed
3 rules fail to adequately insulate the determination of the
4 minimum BAT standard from this risk-benefit balancing
5 process.
6 The proposal merely promises that quantitative
7 risk assessments will not be used in determining BAT.
8 Since the estimates will have been generated in an earlier
9 stage, NRDC has no confidence that EPA will ignore them in
10 determining BAT.
11 Considering the unreliability of these techniques,
12 their use for making any sort of close distinction is not
13 rational. Nonetheless, EPA proposes to use them as the
14 major element in setting priorities in a manner that makes
15 closer distinctions than techniques can rationally alow.
16 EPA's assumption is that the relative seriousness
17 of two hazards can be reliably compared. There are
18 important reasons why this is nowhere near as easy as
19 it sounds. First, many of the uncertainties in the risk
20 assessments operate as wild cards and do not necessarily
21 tend in the same direction and with the same force with
22 different chemicals.
23 In one chemical participates in synergistic
24 reactions and another does not, or if they are metabolized
25 differently, the true risks may well be vastly different.

1 Even the rationality of using these techniques for ranking
2 chemicals in order of seriousness is substantially doubtful.
3 It might not be arbitrary though to use these
4 methods in priority setting as long as only the very
5 grossest distinctions are made between the most serious
6 hazards and ones which are less pressing, though by no
7 means to be ignored. Distinctions must be several orders
8 of magnitude of apparent risk, closer distinctions are not
9 reliable.
10 NRDC does not believe that priority setting based
11 on these gross distinctions will be much more useful to the
12 Agency than priority setting based solely on the amount
13 emitted per year or the number of persons living in the
14 potential area of exposure.
15 If quantitative risk assessments are used even
16 for this priority setting purpose, it is essential that
17 EPA make the range of error much more clear. It is very
18 important that estimates created for priority setting
19 purposes be given only in the form of broad ranges within
20 which the risks may lay.
21 To put forward a single point estimate or even
22 a single point estimate plus an upper bound is unacceptable
23 because of the misleadingly precise character of these
24 estimates.
25 Let me say briefly on other issues, we believe it

1 is extremely important that EPA resolve and foreclose in
2 this rulemaking certain generic scientific issues.
3 Experience has shown that in regulating carcinogens,
4 there are many scientific issues that concern common
5 properties of all carcinogens or questions that are
6 equally on the frontiers of scientific knowledge.
7 The best available knowledge and the precautionary
8 mandate of the Clean Air Act require resolving these
9 generic scientific issues in the same way, on the same
10 central body of evidence, in each proceeding.
11 In past proceedings, EPA and sister agencies
12 such as OSHA have developed a consistent set of principles
13 based on the best scientific knowledge today and the
14 precautionary mandate of their policies -- of their
15 statutes.
16 Yet in the current regulatory process, these
17 issues are reconsidered from scratch in each proceeding
18 and this is enormously wasteful of the resources of the
19 Agency, the scientific experts and all other parties.
20 The only beneficiaries of delay are industry.
21 Effective carcinogen rules must foreclose the
22 opportunity of this dilatory rehashing of scientific issues.
23 EPA must adopt in this proceeding a set of principles based
24 on the best available knowledge and the mandate of the
25 Clean Air Act that will not be proper subjects for

1 relitigation in each proceeding.

2 To be sure, they may be reconsidered as science
3 progresses but only in a proceeding to amend the generic
4 airborne carcinogen rules.

5 NRDC supports the need for strong interim controls
6 on readily controlled emission points to go into effect
7 immediately upon the designation of a substance of a
8 hazardous air pollutant. There is no excuse for delaying
9 necessary controls for even the year it would take to
10 establish the standards under the proposal we offer here.
11 Our specific suggestions on how to improve these generic
12 rules are addressed in our written comments.

13 Thank you, and I would be happy to answer any
14 questions.

15 MR. BARBER: Thank you. I have two questions.
16 Very early in your statement, you made the rather flat
17 observation that air pollutants cause a substantial number
18 of fatal cancer cases. Is that statement supported in your
19 written submission or is that something you would care to
20 elaborate on?

21 MR. DONIGER: It is and it will be. Dr. Wagoner
22 will be speaking to this tomorrow and I must second Bob
23 Rauch's comments that these are points that should be spoken
24 to by the National Cancer Institute experts and others who
25 are the scientists in this area.

1 I believe that statement is correct. The written
2 record will document those statements by the time it is
3 completed.
4 MR. BARBER: Thank you, and the other statement
5 you made in this presentation and your written comments,
6 some suggestions about shifting the burden of proof to the
7 industry. Have you any personal knowledge of a federal
8 program that has successfuly accomplished that feat?
9 MR. DONIGER: I believe the pesticide program
10 establishes the legal mandate for that very explicitly.
11 So does the (PRUDAC) program. What is gained in a shift
12 of the burden is decisions on the margin. If you have a
13 close question whether a technology is available today or
14 whether a process change can be accomplished in a given
15 period or whether a substitution is adequate, if the burden
16 is on industry, it should make a difference in the outcome,
17 rather than if the burden is on you.
18 We think that since the ample margin of safety
19 standards does not entitle the industry to emit anything
20 at all, the very least that can be demanded of them is that
21 they show the burden of why they should be permitted to do
22 so.
23 MR. BARBER: It somewhat shifts the focus of the
24 program, if you will take this as a given for the moment,
25 if you manufacture organics, you will have some emissions.

1 Then the debate over the standard will be a
2 debate over the need for the product in commerce as opposed
3 to a debate over whether you can have zero. You know when
4 you go in, you can't have zero, so if we establish a
5 presumption that will be zero, the only way to have zero
6 is to stop the use of chemicals.
7 Instead of talking about emissions, you either
8 should be talking about whether the product is needed in
9 commerce or whether we have transferred back to the industry
10 the responsibility of establishing their own emission limits.
11 If they have overcome the first hurdle and proved the
12 chemical is needed in commerce and ought to be kept,
13 then they ought to nominate the appropriate level of
14 control.
15 MR. DONIGER: I don't see it operating that way.
16 First, I think the consideration of availability of
17 substitutes, the question of whether the substances useful
18 and needed is something that should play a very high role
19 and not an incidental role in your thinking.
20 Under the current policy, it is an incidental.
21 I have never understood or accepted the distinction made
22 between so-called product oriented laws and process oriented
23 laws and I don't see where that dividing line comes from.
24 I think you have as much of a duty to look at whether the
25 product itself can be substituted for as any other agency

1 operating under any other statute.

2 The existence of a zero standard going into

3 effect in 360 days if the industry does not come forward

4 with the data needed to show what you need to know, seems

5 to be an effective spur to get them to come forward,

6 particularly when there are organizations such as ours

7 which might be interested in enforcing the emission

8 standard even if the Agency is not able to do so.

9 You would still have an obligation or you would still

10 have the need to go out and gather a fair amount of data

11 on your own.

12 But in so many cases, we have seen the margin

13 cases being determinative. You feel you have to carry

14 the burden of showing what can be done at the last mile

15 instead of industry showing what can't. Since industry

16 has no right to continue these a year after they are listed

17 under the law as it is now, they at least ought to justify

18 those marginal decisions.

19 MR. BARBER: Thank you. Any other questions from

20 the panel?

21 MR. HAWKINS: David, I would like to ask the same

22 question of you that I asked of Bob Rauch. That is, do you

23 see a difference in terms of the posture of the regulatory

24 agency when confronted with a situation where you have a

25 large number of individuals exposed to a small risk or a

1 small number of individuals exposed to a high risk?

2 There are really two subparts to the question,

3 perhaps. For any given predicted risk of deaths per year,

4 for example, you can arrive at that by either situation,

5 either a large number of individuals with very small risks,

6 that is the population of New York City exposed to a risk

7 of a million a year, or the population of a crossroads in

8 Iowa exposed to a risk of one in a hundred per year.

9 Is there a lack of concern with one or the other,

10 a greater degree of concern with one or the other in your

11 view and if so, would you like to elaborate?

12 MR. DONIGER: Let me say a word about priority

13 setting to begin with. One of the concerns we have with

14 most priority setting schemes put forward by most agencies,

15 including this one, is that a lot of energy can be spent in

16 deciding what to do first without any commitment given to

17 dealing with the problem in a reasonable amount of time or

18 any number of things in a given year or a two, three or

19 five year period.

20 Priority setting only makes sense if it is hooked

21 to a decision of what is the minimum we are going to do each

22 year. That's why we think there should be a minimum of 20

23 and priority questions would be which of the candidate

24 substances off the list should we pull?

25 I think Congress has decided that everyone is

1 entitled to protection. The variable Congress has allowed
2 you to play with, not only for this question you have asked
3 but for compliance dates, the variable Congress itself has
4 played with in other parts of the law, is to set back the
5 deadlines where necessary, to compromise the time when
6 protection is to be offered and not the goal of protecting
7 people.
8 The benchmark of what is adequate and what is
9 total projection is so important and it is the goal that
10 everything should be geared to working towards. In your
11 priority setting, I think that all other things being equal,
12 I would not want to state which one to go for. But
13 obviously, you would also have to look at where you
14 get the most bang for the buck.
15 MR. HAWKINS: Related to it, aside from priority
16 setting and taking into account the sources of error that
17 you discussed and use of the quantitative risk assessment
18 technique, if the Agency were -- under the law, do you think
19 there is a level of risk that is sufficiently small that
20 even if all 220 million people in the United States were
21 exposed to it, it would be a point at which the Agency could
22 justify no further control?
23 MR. DONIGER: In one sense, the answer is
24 obviously yes, everybody goes in the end and there are
25 risks that are larger that demand your attention from

1 other sources of illness and demands society's attention
2 in other sectors.
3 I have always felt that statement where we can't
4 eliminate risks entirely is used to prove too much. It is
5 used to prove that we should not eliminate many risks at all
6 and there is a hell of a distance to go towards zero in any
7 one of these industries before I think it is fair to pose
8 the question, should we stop here.
9 MR. BARBER: Dr. Albert?
10 DR. ALBERT: You point out that there can be an
11 enormous range of uncertainty in risk estimate. You point
12 to the analysis of a risk assessment on saccharin by the
13 National Academy of Sciences as an example of this where
14 the risk or the range of risk assessments estimates are
15 essentially due to the variety of extrapolation models
16 used.
17 However, I think you know that the EPA has used
18 the linear non-threshold extrapolation model almost
19 exclusively except in the rare instances where other
20 extrapolation models may be more conservative. The
21 reason for the use of the linear non-threshold dose
22 response model is that it has more scientific validity
23 than the others.
24 It yields a more conservative estimates,
25 emphasizing the importance of making estimates which will

1 tend to err on the high side. Another reason for the use
2 of this model is that the biological effects of ionizing
3 radiation, the committee of the National Academy has also
4 used this model for many years.
5 They, for example, estimate that 1 percent of
6 human cancer is due to background ionizing radiation with
7 this model. Do you believe that the use of the linear
8 non-threshold extrapolation model underestimates risks
9 by a factor of several million as you have asserted?
10 If so, what is the basis for your assertion?
11 MR. DONIGER: That was not my assertion. My
12 assertion was that the range of estimates in the model,
13 as you said, varies over a 5 million-fold range. The
14 linear non-threshold model may well be closer to the
15 top of that range and obviously we prefer that to using
16 something like a calorie control counter model, if that's
17 the kind of debate we would get into.
18 What I detect, and you may hear more on this
19 from Dr. Mendez in the Boston hearing, is that there has
20 been a split, sort of a split personality in the statement
21 you just made of the reasons that the linear non-threshold
22 model is chosen is sometimes stated to be that it's more
23 accurate and sometimes that it's more conservative.
24 I'm not exactly sure what position the Agency
25 takes when it chooses that. I am not personally qualified

1 to speak to the issues of accuracy of any one of these
2 models, but it is my understanding that there are factors
3 which the modeling does not capture which are unpredictable
4 when you cross species lines and mix people exposure, people
5 together with many different kinds of exposure that you just
6 can't capture with these models.
7 Those factors alone are sufficient in my view to
8 account, as I understand it, to account for at least a
9 couple, maybe three or four orders of magnitude in risk.
10 I don't see how you can engage in a risk-benefit analysis
11 where the factor on the left side, the risk side, let alone
12 the factor on the cost side, is as mushy as that because it
13 doesn't give any rational guidance on what the outcome should
14 be, even if risk-benefit analysis were properly part of the
15 statute.
16 MS. THAL: You suggested that timing was the only
17 variable over which you had any choice. Given that you also
18 say we should put a year limit on emissions unless the
19 industry can prove otherwise, what sort of criteria would
20 you have industry use or have us use, what kind of evidence
21 would we need to allow them to go beyond a year, given that
22 timing is the only thing you think we can use to avoid
23 serious closures?
24 MR. DONIGER: I think we all need to explore that
25 but I am trying to get across the idea that first the goal

1 of zero is important and the shift in the burden is
2 important. Even if you considered exactly the same factors
3 you propose to consider now, which I don't think would be
4 the right way to go, the shift in the burden I think would
5 change some outcomes and would result in greater control
6 earlier than otherwise.
7 I think there should be a heavy burden on the
8 industry to show why you can't use a different product
9 than a carcinogenic to show the same end product they are
10 making with the same product they are selling. I think
11 there should be a burden on them to show why no process
12 changes are available and when you have evidence to suggest
13 that process changes are available or substitutes are
14 available, it should be up to them to disprove the
15 availability or achievability of those and not on you
16 to show that they are ready.
17 DR. ANDERSON: A slight variation on some of the
18 risk assessment questions. When you first started, you said
19 quantitative risk assessment has no place in regulatory
20 action.
21 MR. DONIGER: Regulatory standard setting.
22 DR. ANDERSON: Okay. Yet I think we all agree
23 that we can demonstrate wide variations in -- publishing --
24 among carcinogens a million-fold or more. I am not clear
25 how you would propose to take this into account. I do

1 assume you would agree it would make some sense to take
2 this into account in some way. How are you proposing to
3 deal with this?
4 MR. DONIGER: What I said was I think if you
5 restrict the use of quantitative assessments to distinguish-
6 ing between chemicals which appear to have different
7 potencies of very large magnitudes like you have described
8 so that the difference is so great that it would be unlikely
9 to be swamped by uncertainties, if you understand my
10 meaning, it is a true difference and not just an artifact
11 of the uncertainties.
12 That would be a useful thing to consider in
13 setting priorities and which 20 substances to pull off
14 the candidates list and regulate each year. You might
15 do the same thing with gross differences and the amount
16 emitted or amount of exposure, although I don't recommend
17 that you get into modeling or monitoring of exposure.
18 I think emissions is a good surrogate and maybe
19 population densities together are good surrogates. But
20 when you have gross distinctions of that kind, it makes
21 sense and only then does it make sense to make distinctions.
22 DR. ANDERSON: You really didn't mean no role at
23 all?
24 MR. DONIGER: No, I mean no role in setting the
25 standard -- in setting the level of control and only the

1 limited role I have described in setting priority setting.
2 That is the decision, as I understand it, that OSHA has
3 come to.
4 DR. ANDERSON: Another question. You said it has
5 been shown, I can't remember your words, but some exposure
6 to a carcinogen, some people will get cancer. Suppose with
7 a very conservative model, it demonstrated that there is not
8 one person getting cancer from emissions?
9 MR. DONIGER: As I understand it, you usually do
10 these on an annual basis so you're talking about less than
11 one per year?
12 DR. ANDERSON: Or -- lifetime basis or annual
13 basis --
14 MR. DONIGER: I have less concern if you are
15 talking about lifetime risks but on an annual basis, if
16 you are one-tenth -- vinyl chloride, if I remember correctly,
17 there were predictions of one-tenth of a death per year
18 which is a death every ten years and I don't see why you
19 should count that as less than one.
20 DR. ANDERSON: I'm just wondering when you look
21 at an ample margin of safety from another standpoint other
22 than emissions. That is, if you use a conservative model
23 and don't see any cancer deaths, would you regard that as
24 an ample margin of safety, either ample or lifetime?
25 MR. DONIGER: All I will say about this is that

1 I think you have a burden to explain much more clearly than
2 you have why the conservative models or how the conservative
3 models account for some of the non-modeling or non-mechanism,
4 non-single carcinogen mechanism uncertainties like synergism
5 and interaction and how you account for the variability in
6 individual sensitivity that you don't find in animals and
7 things like that.
8 What I have been really disappointed in are what
9 I think -- even if the authors mean them, they are
10 misunderstood, the statements that quantitative risk
11 assessments are rough indications and only crude indications.
12 Those get lost and don't mean anything when decision makers
13 and other commentators ourside the process look at these raw
14 numbers that these methods generate.
15 DR. ANDERSON: One other question. You emphasize
16 the uncertainties in crossing species lines when talking
17 about potency. Certainly it's true that there is a lot of
18 difficulty involved in selecting a good test model. I
19 wonder if you are aware of the emerging data which indicates
20 that where there is a positive response, it appears that the
21 agreement in potency is as good as within an order of
22 magnitude.
23 If this turns out to be the case, would you think
24 that we can go ahead and cross species lines and use potency
25 factors with more certainty?

1 MR. DONIGER: I am not qualified to talk about
2 the soundness of that data. If it exists and it is sound,
3 it's important, but I think you have the problem of knowing
4 whether humans in their multi-chemical environment will
5 react the same way as any of the animals in their single
6 compound environments.
7 Also, I think, I have only talked to people
8 about this data and I've never read any, but there is
9 disagreement about whether it falls in that narrow range.
10 MR. BARBER: One last quick question. When you
11 talk about the industry assuming the burden of proof that
12 substitutes are not available, given that most of these
13 chemicals did exist some years back, aren't we really
14 talking about an economic test on the availability of
15 substitutes?
16 MR. DONIGER: I once got in trouble at a
17 conference for suggesting that many of the uses of vinyl
18 chloride were easily substituted for. For example, we
19 don't need credit cards or plastic baby pants and almost
20 everyone in the audience rebelled at this suggestion.
21 But there are many products that can be made
22 through other processes or with other substances. I suppose
23 what we are talking about is at least a look at whether some
24 reasonably close cost you can do without this chemical. I
25 would not want to limit substitutes to only things equally

1 or less costly but you also have to look at substitutions
2 or process changes that are going to cost something.
3 You're right, there is something in there. But
4 what you need to do is to put the burden on industry and
5 not yourselves to show what costs and what technological
6 barriers are too great to justify emission reductions.
7 MR. BARBER: In the final analysis, the
8 Administrator needs to make a judgment as to whether or
9 not those costs are worth imposing on society or the
10 industry to either move to a substitute that's available
11 or do without the product.
12 MR. DONIGER: Keeping in the back of his mind
13 that Congress has decided that all of those emissions should
14 be eliminated or has given you the authority to do that.
15 MR. BARBER: It seems to be a very gray area
16 where he somehow will make a judgment that a plastic pencil
17 is better than a wooden pencil or a plastic toothbrush is
18 bettern than a wooden toothbrush for economic reasons, but
19 he can't really judge the degree of control using economic
20 considerations.
21 MR. DONIGER: I'm not sure I understand why not.
22 If you decide there is no substitute for a given chemical,
23 let's take an ultimate case, that it is essential for
24 defense in some way. Then you would be limited to control
25 technologies that can be imposed without precluding the

1 making of that substance.

2 But if you look at the vinyl chloride or array of

3 products and you say that baby pants and credit cards and

4 numerous other uses, that there are substitutes for those

5 out of other plastics that don't have the same problems

6 associated with them or out of cloth and cash and all the

7 other things we used to use, then I think you say, well,

8 if baby pants and credit cards account for 20 percent of

9 the production of polyvinyl chloride, then our standards

10 can go to the point where they reduce polyvinylchloride

11 by that amount and let the industry put the uses into

12 substances too important to eliminate right away.

13 MR. BARBER: Okay, I have no further questions.

14 Thank you.

15 I think we should take a short break and give

16 the court reporter pause. Let's reconvene at about 20

17 past 11:00.

18 (Whereupon, a break was taken.)

19 MR. BARBER: I would like to reconvene this

20 session.

21 The next scheduled speaker is Mr. Roger

22 Batchelor with the American Industrial Health Council.

23 While he is coming forward, I would like to interject

24 two observations. Copies of the transcript of this

25 hearing will be available from the court reporter and

1 you may contact her to order it.

2 Secondly, we would expect that in the oral

3 testimony, references to documents will be supplied if

4 the comments are to be considered. There seemed to be

5 several in the presentations made so far that have

6 references that are specified or indicated. If each

7 speaker would note that, as we go through their comments,

8 we will need to have access to their references.

9 Mr. Batchelor?

10 MR. BATCHELOR: Thank you, Mr. Barber. Ladies

11 and Gentlemen, I am Roger Batchelor, I am with Diamond

12 Shamrock but today I am here representing AIHC, American

13 Industrial Health Council and I chair the group that has

14 focused its attention on this particular proposal by EPA

15 for control of airborne carcinogens.

16 The comments this morning prompt me, perhaps at

17 the peril of a heart attack by our council, addressing a

18 comment or two before we start here. I have heard the two

19 witnesses this morning and I am personally appalled at the

20 difference of opinion that we have.

21 I would like to just take one moment to state

22 that in our industry, we are reasonably proud of the way

23 we conduct ourselves. I think we have the same motives,

24 although somehow or another they are lost in the jargon

25 of some of these things that have come out.

1 Our basic mode of operation, and I think of all
2 good companies here today, is to operate our facilities in
3 a safe manner and cause no damage or injury, particularly
4 in the area of health as well as environment. If we are
5 not doing something, it is because at the moment we have
6 not yet found out that we should be doing something more.
7 I don't know if anybody in industry, and the
8 people in industry are no different than the people working
9 for environmental groups. We have people, we have families,
10 we live in the environment and benefit from it, and we are
11 trying to do as good a job as can be done and that I think
12 puts us on a common ground.
13 We have submitted with AIHC our detailed comments
14 on the scientific, legal and economic issues raised by
15 EPA's proposed rule and have suggested alternative
16 procedures for implementing Section 112 of the Clean
17 Air Act.
18 Our oral comments on the proposal will consist
19 of seven parts. Today, I will give a brief overview of
20 AIHC, our comments and our recommendations.
21 Dr. Robert Morgan, an epidemiologist, will
22 discuss existing scientific evidence on air pollution
23 and cancer. He is a professor and the former head of
24 the Department of Preventive Medicine at the University
25 of Toronto and is a consultant to AIHC on leave for this

1 presentation from his position as the Director of the
2 Center for Community Health Studies at Stanford Research
3 Institute International.
4 Mr. George Dominguez, the Chairman of AIHC's
5 Economics Committee, will discuss economic issues and a
6 study performed for AIHC by Arthurd D. Little, Inc. of the
7 cost impact of the proposal.
8 Dr. Terry W. Rothermel, Senior Staff Member of
9 Arthur D. Little, Inc. and the project director for the
10 AIHC impact analysis study will discuss the methodology
11 used in the study.
12 Mr. Harvey Lieber, Associate Professor at
13 American University, will present the American University
14 Institute of Risk Analysis comments on the proposal.
15 Tomorrow, Dr. Robert E. Olson, a Professor of
16 Biochemistry and Chairman of the Department of Medicine
17 and Professor of Medicine at St. Louis University will
18 discuss proper criteria for determining whether a substance
19 presents a carcinogenic risk to humans at ambient exposure
20 levels.
21 On Wednesday in Boston, Professor Richard Wilson
22 of Harvard University will discuss risk assessment and the
23 proper perspective for viewing different levels of risk.
24 We would appreciate it if you would hold your
25 questions until we are finished with all our speakers this

1 morning then we will come back and take your questions.

2 First, a word or two about AIHC. We have a

3 membership of approximately 130 companies and a cooperative

4 relationship with some 60 trade associations, which together

5 represent a broad cross section of American industry. AIHC

6 was formed in 1977 primarily to address OSHA's proposal for

7 regulating exposure to potential carcinogens and then we

8 broadened our scope as EPA got into the act.

9 For over two years, AIHC has urged federal

10 agencies to recognize that two different types of judgments

11 must be made in determining whether to regulate a

12 particular substance - first, the scientific judgments

13 whether that substance presents a cancer risk and if so,

14 the extent of the risk; and second, the societal judgment

15 whether or not regulatory action should be taken in light

16 of the type and size of the risk, the costs, the benefits,

17 and other relevant factors.

18 EPA's interim principles and guidelines recognize

19 the difference between these two types of judgments by

20 deciding that "the decision that a cancer risk may exist

21 does not mean that the EPA will automatically take

22 regulatory action".

23 To ensure that the scientific judgments are

24 separated from the regulatory determination, AIHC has

25 proposed the creation of a scientific panel of eminent

1 independent scientists who would perform the scientific
2 risk assessments for agencies to use in making regulatory
3 decisions.
4 Because EPA's proposal indicates that the Agency
5 does not see the advantages of this approach, our written
6 comments discuss the impact of our proposal. Such a panel
7 could lead to increased participation by a larger number
8 of scientists, to consistent evaluations for use by
9 different agencies and to better scientific risk estimates
10 that are viewed by the public and interested parties as
11 totally objective and independent and therefore could lead
12 to less dispute. We believe this concept would provide
13 for an orderly and prompt presentation of public comments
14 and would speed up the regulatory process by eliminating
15 duplication.
16 EPA's determination to separate scientific
17 decisions from regulatory decisions is a major contribution
18 to the regulatory process and our science panel proposal is
19 an attempt to improve on that concept. We hope the Agency
20 will reconsider and support this suggestion.
21 Turning now to the proposed rule. The fundamental
22 problem with the proposed rule is that EPA has not
23 established a need for this regulation, although it is
24 required to do so by Executive Order 12044 and is required
25 by Section 307 of the Clean Air Act to disclose the factual

1 data on which the proposal is based.

2 It is essential that EPA demonstrate a need for
3 this proposed rule because the Agency has adequate statutory
4 authority to regulate hazardous air pollutants on a
5 substance-by-substance basis as it has in the past.

6 The initial question in developing any policy or
7 regulations for airborne carcinogens must be whether there
8 is a cancer problem resulting from the presence in some
9 geographical areas of extremely low levels of possibly
10 carcinogenic materials. The crux of EPA's proposed rule
11 is a generic determination that the presence of airborne
12 carcinogens in relatively low ambient concentrations warrant
13 regulatory action of the type and to the extent provided in
14 the proposed regulations.

15 However, EPA has failed to provide adequate
16 support for this proposed generic determination. Instead,
17 the Agency has relied on a broad biological principle.
18 This is contrary to EPA's approach in the past and to a
19 recent decision of the Court of Appeals for the District
20 of Columbia.

21 As the court stated in Monsanto v. Kennedy, an
22 agency cannot regulate on the basis of abstract scientific
23 principles, but must make a decision "on the basis of a
24 meaningful projection from reliable data".

25 The Agency has cited 37 references to support its

1 proposed conclusions. AIHC scientists have reviewed these
2 references. I might add that we have had them re-reviewed.
3 These references simply do not provide support for the
4 Agency's generic determination.
5 Moreover, support has not been provided in the
6 comments of the proponents of the regulations. The studies
7 cited in their comments relate to substances which are
8 already subject to regulation and one inorganic substance
9 which is being considered for regulation.
10 The Agency should have undertaken some scientific
11 studies to show health benefits that would be achieved by
12 the proposed rule and the need for it, during the past few
13 years it has been considering how to regulate airborne
14 carcinogens.
15 Such studies should have been completed prior to
16 the generic determination and should have been made
17 available for public comment in this proceeding. Certainly,
18 EPA should now put any such studies, should they exist, and
19 the supporting data in the public docket with an adequate
20 opportunity for public comment.
21 AIHC has concluded, based on scientific studies
22 on air pollution and cancer, that this generic regulation
23 is not needed. Dr. Morgan later will discuss with you in
24 greater detail the evidence for our conclusion. Gentlemen,
25 I want to emphasize, we are poles apart on the need for this

1 regulation.

2 I heard your witnesses saying this morning that

3 it's too bad the industry doesn't realize the need for this

4 regulation. But somehow or other, we are dealing with these

5 low, low concentrations of something in the air and we are

6 building ourselves a case, not on scientific data, that says

7 hey, we have to pull out all the stops, we have to go attack

8 this thing.

9 Somehow or other, we are talking about a chance

10 probability, probably measured one in a million and we have

11 all kinds of new language that I'm not really up to but it

12 comes down to what is the chance of somebody having a death

13 from this thing being up in the sky?

14 We come to absurd numbers and how do we relate

15 this great interest to these absurd numbers, a probability

16 that will probably never exist, when at the same time we

17 see the State Highway Department projecting there will be

18 232 deaths this long weekend.

19 Where is the balance here, what are we trying to

20 do in this world?

21 To make sure that our position is not

22 misinterpreted, however, I want to emphasize that AIHC

23 in no way is suggesting that EPA should not continue to

24 regulate substances that are shown to pose a significant

25 carcinogenic risk.

1 EPA has the authority to regulate on a substance-
2 by-substance basis under Section 112 of the Act and has
3 authority to regulate in other ways under other sections
4 of the Clean Air Act. AIHC has proposed specific procedures
5 for implementing Section 112 of the Clean Air Act.
6 We support the principles of Executive Order 12044
7 and the Regulatory Council's statement: that agencies
8 rigorously evaluate all relevant, available scientific
9 evidence in determining whether or not a substance is
10 likely to be carcinogenic; and that agencies ensure that
11 the public has a substantial opportunity to contribute to
12 regulatory programs for carcinogens.
13 The procedures proposed by AIHC are the following.
14 First, EPA would identify and screen substances to establish
15 priorities for review and referral to the scientific panel.
16 Second, EPA would publish a notice of evaluation
17 to permit public comment, scientific dialogue and the
18 submission of data. After the initial comment period,
19 the scientific panel would proceed to prepare assessments,
20 and EPA would evaluate emissions sources, public exposure
21 and possible control strategies.
22 Third, EPA would establish priorities for
23 regulation.
24 Fourth, where warranted, EPA would publish for
25 public comment a notice of determination to list a substance

1 under Section 112 and an advance notice of proposed
2 rulemaking on regulatory alternatives.
3 Fifth, EPA would publish a determination to remove
4 a substance from the Section 112 list or would propose
5 regulations for controlling emissions; public comment
6 would be requested and a hearing would be held.
7 Sixth, EPA either would publish final regulations,
8 would repropose modified regulations or would publish a
9 final determination not to promulgate regulations.
10 We believe that these procedures would ensure
11 that the necessary scientific basis for regulating is
12 obtained and together with our proposed scientific panel,
13 would speed up the process of regulating substances that
14 should be regulated.
15 Let me turn now to a comment on regulatory
16 analysis. This proposed rule is one of the most important
17 regulations the Agency ever has proposed or considered.
18 Why, because it would cover a wide range of substances
19 of different potency and with different potential exposure;
20 it would establish BAT as a minimum level of control for
21 all these substances; and it would thrust the Agency into
22 new areas and disputes, such the land use regulation
23 reflected in the proposed requirements for the location
24 of new sources for which the Agency has no statutory
25 authority.

1 Yet, the Agency has not analyzed the impact of
2 the proposed rule.
3 The basic framework for evaluating a regulation
4 and its alternatives is a regulatory analysis which must be
5 prepared for all regulations with potentially major cost/
6 price effects on a particular region, group, industry or
7 economic sector and for all rules that would have a potential
8 $100 million effect on the economy.
9 Both Executive Order 12044 and EPA's own
10 regulatory procedures require that EPA prepare a regulatory
11 analysis for the proposed rule. EPA acknowledges that the
12 proposal is a major regulation. But EPA has refused to
13 prepare a regulatory or economic analysis on the grounds
14 that it would not be a meaningful exercise to conduct a
15 regulatory analysis of unidentified controls on unknown
16 categories of as yet unnamed pollutants.
17 We cannot agree with the Agency's reasoning,
18 particularly in light of the Agency's acceptance of the
19 need for regulatory analyses. The Administrator has
20 emphasized that the Agency "routinely (does) economic
21 analysis of all of (its) regulations" and that "among the
22 regulatory agencies (it has) the most sophisticated
23 capacity for doing economic impact analysis".
24 We don't disagree with that, we do agree.
25 With respect to the assertion that candidate

1 substances have not been named, benzene has already been
2 listed and EPA informally released a list of 42 other
3 substances and categories which had been developed by
4 the EPA Cancer Assessment Group.
5 We have heard discussion today about doing 20
6 this year and 20 next year. Whatever it is, the names and
7 numbers are out there.
8 In arguing that the emission controls are
9 unidentifiable, EPA ignores the fact that its proposed
10 regulation would establish regulatory criteria which will
11 determine the levels of control. These controls will inclu
12 a generic standard for fugitive emissions and best availabl
13 technology (BAT).
14 Because EPA declined to provide an economic
15 assessment, AIHC commissioned an illustrative study by an
16 independent consultant, Arthur D. Little, Inc. Arthur D.
17 Little, Inc. was retained to demonstrate that a methodology
18 is available for EPA to use in preparing a meaningful
19 estimate of the cost of the proposed rule and to assess
20 whether the criteria of Executive Order 12044 have been
21 met. Mr. Dominguez and Dr. Rothermel will discuss this
22 report in greater detail but the report does demonstrate,
23 number one and most importantly, that a methodology does
24 exist for preparing a meaningful cost impact analysis of
25 the proposed regulation.

1 That the regulation would have major cost impacts
2 on specific sectors and that the impact would be over
3 $100 million just for the three illustrative substances
4 studied by Arthur D. Little, Inc.
5 That, while AIHC has not attempted to apply a
6 multiplier factor for other candidate substances, the
7 proposed regulation will result in large expenditures
8 with major impacts on the economy.
9 It is imperative that EPA conduct a regulatory
10 analysis of this proposal. Unless a regulatory analysis
11 is prepared at this time, it will never be conducted since
12 the Agency intends that this generic rule will preclude any
13 consideration of alternatives in the future.
14 There is a time consideration that we should
15 mention. The focus of AIHC's activities has been to
16 encourage the development of a consistent, clearly
17 articulated national policy for carcinogenic substances
18 that is based on sound scientific principles and is
19 consistent with the President's regulatory reform policies.
20 In the last year, the government has taken a number of steps
21 in that direction and the scientific dialogue on the issues
22 has begun.
23 A year ago, the President's Office of Science and
24 Technology Policy released its suggested framework for
25 federal decision making on identifying, characterizing

1 and controlling carcinogens.

2 Last July, EPA, FDA, CPSC and the Food Safety
3 and Quality Service requested public comments on a report
4 prepared by the IRLG's Work Group on Risk Assessment.

5 In October, the Regulatory Council published,
6 for public comment, a policy statement on the regulation
7 of chemical carcinogens.

8 AIHC welcomes these efforts to commence the
9 necessary scientific and public dialogue on these important
10 matters. AIHC's comments on the IRLG report and the
11 Regulatory Council statement suggest shortcomings that
12 should be corrected and improvements that should be made
13 before the proposals properly can be implemented by EPA
14 and the other regulatory agencies.

15 We understand that the IRLG has scheduled meetings
16 to consider the comments it received and to make appropriate
17 revisions to its report and we expect that the Regulatory
18 Council also should do so. It is unfortunate that EPA
19 published its proposed rule before these underlying
20 documents could be revised after public discussion.
21 The Agency should await responses to the public comments
22 on those documents and obtain additional public input.

23 Let me summarize our main points. Presently
24 available scientific studies do not support a conclusion
25 that there is a need for this generic proposed rule.

1 The Agency has statutory authority to regulate
2 on a substance-by-substance basis under Section 112 or
3 under other sections of the Act when it has data showing
4 a need to regulate.
5 AIHC urges EPA and the other regulatory agencies
6 to continue the public dialogue on these issues that now
7 has begun so that we may control substances that cause
8 cancer. We will be happy to meet with you further to
9 discuss ways of achieving this common goal.
10 This is a major regulation. As we have
11 demonstrated, EPA can analyze its economic impact. Since
12 this regulation is intended to preempt future regulatory
13 decisions, its economic impacts as compared to alternatives
14 will never be analyzed unless they are analyzed now.
15 I have focused on some of the major policy
16 questions raised by the proposal. In addition, we believe
17 the proposal is flawed legally. We object to the early
18 listing approach, to the two-phased to standard setting
19 concept and to the final standard setting requirements
20 for substitutes and the kind of land use regulation
21 reflected in the proposed emissions offset approach.
22 This is detailed in our submission.
23 Finally, some of the advocates of this regulation
24 have suggested that we should view the proposed rule as an
25 insurance policy. That is to say that a small amount of

1 money will purchase a large amount of protection. In the
2 first place, we are not here dealing with small amounts of
3 money.
4 Secondly, for the risk we are insuring it is like
5 someone living in Denver buying protection against the
6 chance of a collision with a submarine that is off course.
7 Our effort has been to be sure that there is a
8 sound scientific basis for regulating chronic health hazards
9 We believe there has not been a demonstrated need for this
10 proposed rule. We, therefore, urge the Agency to withdraw
11 it.
12 May I now introduce Dr. Morgan who will give his
13 testimony?
14 MR. BARBER: Yes. Mr. Batchelor, I would like
15 to procedurally outline how we are going to get from here
16 to there. While I recognize your comments to be of a
17 general nature, we will probably want to come back to
18 some of the more general AIHC suggestions and positions.
19 I would like to retain the opportunity to talk
20 through with each of the speakers some of the technical
21 points in their presentations. When you are dealing with
22 oral testimony, you tend to get cross as you proceed across
23 the several hour time span we are facing here.
24 As individual speakers deal with subjects that
25 seem wholly contained in their presentation, the panel will

1 address questions related to them and when we come back at
2 the end, we can deal with some of the general questions.
3 MR. BATCHELOR: I think that's an improved
4 procedure.
5 MR. BARBER: May I have the next speaker, please,
6 Dr. Robert Morgan.
7 DR. MORGAN: As Mr. Batchelor indicated, my name
8 is Robert W. Morgan. To make this presentation, I am
9 presently on leave from my position as Director of the
10 Center for Community Health Studies at SRI International
11 in Menlo Park, California.
12 I was a little puzzled when a previous speaker
13 mentioned that we can control 90 percent of cancer. As
14 one who has spent the last five years in preventive
15 medicine, I would be interested to have those tools
16 at my disposal to control that 90 percent because I
17 have as yet been unable to find them.
18 We have been looking and looking hard and we
19 have been able to control some cancer, but I am not sure
20 the prospect is upon us, especially with an aging population,
21 to control 90 percent. Maybe we can but that is a fantastic
22 hope. But I think it is a hope and not a fact.
23 At the present time, there is no evidence of
24 any excess cancer incidence or mortality in the United
25 States due to industrial air pollution. If there is a

1 societal concern, a comprehensive epidemiology program will
2 be adequate to alleviate it rapidly and effectively.
3 In attempting to explain the need for a policy
4 and regulatory mechanism, the Environmental Protection
5 Agency has failed to relate human cancer occurrence to
6 general environmental levels of airborne chemicals. The
7 EPA admits failure at the end of Section 2 (Causes of
8 Cancer: Importance of Environmental Factors) when it
9 states, "Thus, it is both prudent and in view of the
10 large number of people potentially affected, important
11 to reduce or contain emissions of known or suspected
12 atmospheric carcinogens in order to prevent future
13 problems before they are actually observed" which leads
14 me to observe that those have not yet been observed.
15 Also in the Federal Register document, the Agency
16 says, "it is still too early to detect the full impacts of
17 these chemicals on human health." I say that I don't agree
18 with that, many of the substances of concern are substances
19 which have been around for more than 20 years.
20 It does not always take years of latency before
21 one can demonstrate an increase in cancer and if we are to
22 look now, we may find any effects or be able to reassure
23 ourselves that the effects are not there. Again and again
24 we hear the comment that epidemiologic evidence is lacking.
25 Well, we do have a little bit of epidemiologic

1 evidence, such as a Los Angeles study where they thought
2 they had an air pollution cancer problem but later found
3 they did not because it did not stand up to direct tests
4 of the hypothesis.
5 But one of the reasons data is lacking, if it
6 is in fact lacking, is that epidemiologists tend to go
7 where the bodies are. We count bodies and try to figure
8 out what the determinants of that disease were. It is
9 assumed, I think, in the proposed regulation that air
10 pollution is one of the determinants, a major determinant
11 of cancer.
12 Another part that bothers me about the attacks
13 I hear on epidemiology is the underlying assumption that
14 epidemiology either has not answer the question or will
15 not be able to answer the question because there are no
16 data or it would take too long.
17 Consider for a moment the question of cigarette
18 smoking. Epidemiology first identified the risk of
19 cigarette smoking in terms of lung cancer. It took
20 animal experimenters about 20 years to confirm what
21 epidemiologists knew for some years.
22 It does not seem to me, therefore, that animal
23 experimentation is necessarily any quicker than epidemiology.
24 If you look at the epidemiologic studies that have been
25 important recently in the regulatory sphere such as the

1 questions of saccharin, most of those studies were completed
2 in 18 to 24 months. That does not seem to be a long time,
3 especially when you consider that a chronic animal test may
4 take three years.
5 I would claim that epidemiology has been the most
6 discriminating tool we have to detect human carcinogens and
7 I emphasize human carcinogens. I do not want to discuss
8 extrapolating models from rodents to humans. One of the
9 reasons there are so many models is that none of them fit
10 the data very well.
11 I always think back on a model that fits the data
12 very well is the fact that for many thousands of years, it
13 was assumed that the sun actually went around the earth, it
14 fitted all the observations being made pretty well. To use
15 a model because that is the model used the longest, as we
16 heard this morning, does not necessarily impress me with
17 its validity.
18 When we look at the question about cigarette
19 smoking, I wonder whether reliance on animal testing would
20 have in fact protected the public against the dangers of
21 cigarette smoke. This is one of the things that concerns
22 me and I think the same is true with asbestos.
23 When we have substances where animal testing has
24 consistently failed to protect, why then should we say that
25 we must rely on animal data because epidemiology will not

1 protect us? The EPA and to some extent the witnesses
2 appearing earlier this morning commented on the weaknesses
3 of epidemiology and our difficulty in detecting a risk
4 greater than one and a half times that to be expected.
5 If we can detect a one and a half times, I don't
6 think we're doing too badly. But there is a certain naivety
7 here in that it is customary in a well done epidemiology
8 study, if the study is negative, to quote the statistical
9 power of the test, we look at the relative risk and the
10 confidence limits of the relative risk so we know the range
11 we are dealing with.
12 When you look at those confidence limits and when
13 you look at the statistical power of the test you are using,
14 you can have some confidence as to what the likelihood of
15 missing a carcinogen is. Those of you interested in the
16 statistical aspect may wish to refer to my testimony of a
17 year or so ago where I presented a statistical model for
18 weighing conflicting evidence.
19 If you have two positive studies and three
20 negative, we could get a range of possibilities on whether
21 the subject was a carcinogen. That particular approach is
22 ready for publication and should be available shortly as
23 well but it is in the testimony I gave at the OSHA hearings.
24 In my written testimony, I have listed a number of
25 reasons for the relative scarcity of epidemiologic evidence

1 concerning cancer and the general atmospheric environment.
2 The first of those reasons says that there is no evidence
3 from U.S. vital statistics that environmental cancer from
4 atmospheric sources is an important public health problem,
5 they just don't show that.
6 Second point is there is a reluctance on the part
7 of epidemiologists to search for etiologic associations
8 without at least some prior clues that the associations
9 exist. Therefore, we have to have a hunch that there is
10 a problem before we really want to spend our time and energy
11 and public funds to investigate it.
12 There is also a scientific consensus that risk
13 factors such as use of tobacco and alcohol far outweigh air
14 pollution in importance to cancer.
15 Finally, there is a skepticism or even negative
16 attitude on the part of government decision makers, and I
17 think the proposed study shows that, towards epidemiology.
18 We now hear about the cancer problem and how important it
19 is to the regulation and yet there has been no effort to
20 sponsor large-scale multi-centered studies to estimate the
21 risks of low level exposure.
22 The Agency is quite correct in their documentation
23 in implying that available epidemiologic evidence does not
24 justify increased regulation of airborne carcinogens that
25 evidence can be gathered in many instances. We can gather

1 it from occupational studies where we have work forces
2 exposed and I would submit to you, ladies and gentlemen of
3 the panel, that if the work force that encounters high
4 exposure has no increase in risk, then the general population
5 with a much lower exposure is unlikely to have a significant
6 risk of cancer from a substance.
7 We can also study people previously resident
8 around known emission sources. That is an approach that
9 we yield valuable information and it has not really been
10 done enough.
11 Over here on this chart, we have three lines.
12 One is the production of the synthetic organic chemicals,
13 one is the cancer of all sites, the red line going slightly
14 up, and if we take lung cancer, we notice that cancer
15 mortality is declining.
16 Since there is very little impact on cancer
17 mortality due to improved treatment, much of the improvement
18 in cancer mortality is due to declining incidences,
19 especially of things like stomach cancer. The red line
20 where it goes up, the increase, is due almost entirely
21 to lung cancer where we have good knowledge of the major
22 etiology factor which is cigarettes.
23 It seems to me that trying to correlate cancer
24 mortality and cancer instance with that green line indicates
25 that we probably don't have a major problem in overall

1 cancer. The health of the American public is best served
2 by regulatory action that has a strong and defendable
3 scientific basis, such as could be created by a series
4 of appropriate epidemiological studies.
5 Waiting for such studies would not, I believe,
6 seriously endanger the health of the American public. There
7 is already adequate authority to deal with any carcinogen
8 where evidence became available. I would point out that
9 Agency action should not be equated with prevention of
10 cancer. To do so is to mislead the U.S. public.
11 It is to discourage scientific investigation
12 because people will think the problem is solved or be
13 unwilling to fund that because it is supposed to be somethin
14 taken care of. I would say that action or regulation as
15 been synonymous with prevention is a myth.
16 If EPA, despite the lack of evidence, still feels
17 concerned over a possible air pollution cancer relationship,
18 a number of federal agencies and institutes, including EPA,
19 NCHS, NIESH, and the NCI, could participate in a national
20 environmental epidemiology program. Such a national program
21 would designate and fund a number of research projects to
22 answer EPA's questions.
23 Some of those may be done in-house by organizatior
24 such as NCI, some might be put up for contract, there are
25 various ways of doing it. But I think it's important that

1 the Federal Government try to show leadership in answering
2 questions and to answer them properly with well-controlled
3 studies.
4 As a few suggestions of the kind of studies that
5 might be carried out, there could be case controlled studies
6 in metropolitan areas experiencing various degrees of air
7 pollution. Case control studies could take into account
8 such factors as occupation, current and previous, cigarette
9 smoking and some of the other variables.
10 Similarly, one can assemble retrospective cohorts
11 from censuses of towns. I am thinking of the approach
12 Irving Selikoff took in his investigation of Rutherford,
13 New Jersey where they attempted to find an increased amount
14 of asbestos cancer among the residents living close to the
15 factory.
16 So we can do retrospective cohort studies if that
17 seems to be a priority. You can use those two kinds of
18 approaches for specific substances such as asbestos or
19 vinyl chloride or whatever and you can use that approach
20 for general air pollution where you have a mixture for all
21 intents in the environment.
22 Added to that, we have the occupational studies
23 and we look very carefully at the relative risk confidence
24 limits of the workers exposed to low doses of carcinogens,
25 we might have some feeling as to whether or not the people

1 exposed to low doses in the environment really were
2 suffering from an increased risk.
3 Inevitably, if we had a national environmental
4 epidemiological program, many substances would get started
5 in the first two years and then probably diminish in scope
6 as questions got answered. But it would be useful, I think,
7 to organize national resources along this basis.
8 There is certainly the talent in this country to
9 do those kinds of studies. It is because of the multi-
10 centered studies that should be done that federal leadership
11 I think is a vital ingredient.
12 In summary, all available epidemiologic evidence
13 indicates no association of cancer risk with air pollution.
14 If societal, rather than scientific, concerns justify
15 additional studies, further investigation on both general
16 populations and workers could be conducted quickly and
17 reliably as part of a comprehensive national environmental
18 epidemiology program involving the NCI, NIEHS, NCHS and EPA.
19 I will end my testimony there, Mr. Chairman, and
20 take questions.
21 MR. BARBER: Thank you. I would like to ask a
22 few rather general questions from the perspective of the
23 non-medical scientist so bear with me for a moment.
24 You used several times in the statement that you
25 either did not see air pollution as a major problem or in

1 the conclusion it seems to say that you view it as a non-
2 problem. At one point, you said we could find out
3 through epidemiological studies whether people suffered
4 from any risk at all. Are you using this to improve the
5 uncertainty or are you defining risk as the factor -- cancer?
6 MR. MORGAN: I said we could identify significant
7 increase in risk. When you get down to extremely low levels
8 of risk, it's very difficult to quantify that.
9 MR. BARBER: Let me explore the word "significant".
10 Significant, as an epidemiologist, if you are looking at a
11 geographic area with 20,000 fatal cancers a year, what is
12 significant, 1 percent, 10 percent, 5 percent?
13 MR. MORGAN: I used the term in two ways during
14 my presentation. When I was talking about the power of the
15 statistical test, I was using it in a statistical sense,
16 significance. I think when you come to the issue of
17 biologic or public health significance, the decision
18 as to whether 20,001 deaths is a statistical difference
19 from 20,000 given the problem of statistical error in
20 measuring the 20,000 where it really represents an estimate
21 and you have a range of perhaps a few hundred on either
22 side.
23 That as a decision is fairly arbitrary actually
24 and I would say it's not an epidemiological one. I can look
25 at a problem and say I don't think that's a major health

1 problem because it is not increasing the risk to the degree
2 that one could see beyond error.
3 That is not answering the question properly, I
4 understand, but I think the question of how big a risk is
5 important is a matter that concerns more than epidemiologist
6 It certainly must concern you as a regulator.
7 MR. BARBER: I think that's true and the first
8 question we're trying to get past is whether there is a
9 risk, whether someone could be contacting cancer from
10 exposure to ambient air. What I think I heard you say,
11 correct me if I am wrong, is that if you had to prove the
12 negative through epidemiology, you would have difficulty
13 saying unequivocally that out of 300,000 cancers a year,
14 there were zero, there may be some number like 1 or 2
15 percent that you could never speak to statistically with
16 epidemiology.
17 MR. MORGAN: In science there is no proof, you
18 accept or reject a hypothesis and the acception or
19 rejection of that hypothesis is a subjective phenomenon
20 based as much as possible on objective evidence. If you
21 have a situation where an observed increase is the kind
22 of increase that could well be expected from year-to-year,
23 day-to-day variation or your error built-in in measuring
24 deaths in the population, because there are errors in
25 measuring deaths, then at that point you might say, gee,

1 I don't think this represents an increase of risk, I think
2 that's the area you get into.
3 MR. BARBER: When you are building this building
4 from the foundation up rather than the top down and you start
5 to look at risks with all the uncertainties that surround
6 the risk estimates and you look at exposures, you end up
7 with numbers of fatal cancers that you might attribute to
8 air pollution measured in the tens or the ones or maybe the
9 hundreds but you certainly don't measure from the source of
10 chemical cancers in the thousands, at least looking at the
11 data I have seen.
12 If you add the total sum up, you come up with a
13 number of perhaps less than 10,000 and perhaps less than
14 1,000. Can you ever find less than 1,000 in an epidemiologic
15 study of the whole country?
16 MR. MORGAN: If you are dealing with angiosarcoma,
17 yes, you could, because that's a rare disease. You get what
18 amounts to a minor increase in incidence in terms of rates
19 per 100,000.
20 If you are saying could we find a 1 percent
21 increase in the rate of lung cancer because of industrial
22 air pollution, I would say we would have difficulty unless
23 you had enormous sample sizes. Part of that is a function
24 of sample size, how many people are you studying?
25 It is interesting that, when you look at the

1 magnitude of some of these, I believe the highest lung
2 cancer rate in any European city is in Geneva which does
3 not make much sense if one considers air pollution,
4 especially industrial air pollution, as a suspect source.
5 You find these anomalies all over the map and that is what
6 leads me to suspect that there are not a lot of bodies out
7 there that we can attribute to industrial air pollution.
8 MR. BARBER: I guess we will not agree here on
9 what a lot is. But if a thousand is an insignificant
10 number, we would probably have some area that we overlap.
11 The last comment concerned me, I don't know very
12 much about Geneva, never having been there. But why is it,
13 is air particularly clean in Geneva?
14 MR. MORGAN: There are no industries in Geneva
15 except for organizations like the World Health Organization
16 air pollution is of a different sort. It's just a very
17 clean city with manufacturing and offices and lovely lake
18 and fairly clean air.
19 MR. BARBER: Fairly clean in the sense that it's
20 a pleasant place to be or because someone went out and
21 measured it and determined what it was?
22 MR. MORGAN: I'm not sure what the measurements
23 there are but I know that San Francisco, which is where I
24 live, has cleaner air than Los Angeles and higher lung
25 cancer. I think someone later on will be talking about

1 urban and rural factors.

2 We are just not finding great hot spots of
3 mortality, especially among females where it should be
4 found as much as in males, around areas of heavy industry
5 and where we know there is a chemical industry, say. When
6 we find a difference and it's all found in the males, most
7 of us tend to suspect that this represents occupational
8 exposure which is what happened in the Los Angeles study.

9 They did correlate air pollution levels with
10 cancer and mortality but the correlation was only going
11 on in the males and when they followed it up with a case
12 control study, they found those males were involved in
13 occupations where one might expect an increase risk of
14 cancer.

15 If we see evidence that both sexes have a higher
16 rate in an area and we cannot explain it by what we call
17 domestic exposure, that is the asbestos worker who comes
18 home with his coveralls laden with asbestos, then we say,
19 hey, maybe something is going on. But so far we do not
20 seem to be able to identify those kinds of things.

21 One of the speakers this morning promised us that
22 tomorrow Joe Wagoner will bring in new evidence on this
23 particular issue definitely relating cancer to air
24 pollution. I have conducted a very careful search of
25 the literature and have not found it yet so this will

1 be new and I think most serious epidemiologists will be
2 looking with great interest with whatever data is brought
3 in.
4 MR. BARBER: Further questions? Mr. Hawkins?
5 MR. HAWKINS: Yes, Dr. Morgan, first a question
6 on the practical problems that might be encountered by a
7 program doing epidemiological studies for a variety of
8 compounds. I would like to read a statement in the record
9 of the OSHA hearings and ask whether you agree or disagree.
10 It has to do with the question of whether there
11 is accurate, relatively accurate exposure data for various
12 chemicals. Proceeding on the assumption that that relatively
13 accurate exposure data, perhaps 20 or 30 years ago, is
14 required in order to do an epidemiological study.
15 If that assumption is incorrect, perhaps you
16 would like to respond to that as well. The statement was
17 made in the OSHA hearing that if one looks at the two dozen
18 known human carcinogens, only three or four exist that
19 would have data that would allow some estimate of past
20 exposure.
21 To your knowledge, is that a correct statement,
22 and further, how would you characterize the data that may
23 exist for, say the 43 pollutants referred to in Mr.
24 Batchelor's testimony?
25 MR. MORGAN: I think we should realize that in

1 some respects, epidemiology is a form of rough justice.
2 Although we would like to see very precisely measured
3 exposures such as the people running chambers for mice
4 can have, frequently in epidemiology we have to go along
5 with things like high, medium, or low, or exposed, or
6 unexposed.
7 However, much discomfort that might bring some
8 of our wet lab colleagues, it has been effective in
9 determining such things as cigarette smoke where none
10 of us know what a good measure of exposure is. But we
11 still are able to demonstrate a dose response relationship
12 with cigarette smoking.
13 In answer to your first point, it is not necessary
14 to have accurate exposure data in order to do epidemiologic
15 studies. It is preferable to have some exposure data,
16 accurate would be even better, but you can proceed without
17 it.
18 In terms of how many of the 43 substances have
19 accurate exposure data, I would guess that in the general
20 environment we have relatively little data but we probably
21 have some data on many of those for workers exposed to that
22 through the course of their job. As I mentioned, that is
23 one source of data that could be used to look at the
24 question of environmental exposure.
25 MR. HAWKINS: Thank you. I also have several

1 questions similar to Walt Barber's relating to the issue of
2 the power of epidemiological studies. Again, pointing as a
3 point of reference for these questions to some statement in
4 the OSHA record which you can give me your reaction to.
5 One researcher in the field stated that while
6 epidemiology is reasonably strong in identifying intermediat
7 and high levels of risk, it is quite weak in identifying the
8 causes of very low levels of risk. Continuing on, he said,
9 "What do I mean by low level of risk? I believe I can put
10 this in context by noting that the lowest excess cancer
11 risk that I know of that is directly observable in a group
12 of exposed individuals and is generally accepted as being
13 due to that exposure and not some other factor is the 30
14 percent excess risk of childhood leukemia among children
15 exposed to radiation in utero during the last trimester
16 of pregnancy."
17 Are there other excess risk determinations from
18 epidemiological studies that are less than the 30 percent
19 factor that you are aware of?
20 MR. MORGAN: Having done a fair number of studies
21 that show a relative risk or a risk increase under 30
22 percent, because of the problem of the human organism
23 and the number of things to which he or she is exposed,
24 one tends to say, with some reluctance, rather to attribute
25 the risk to the increased exposure because of a number of

1 confounding factors but one of the points that should be
2 raised is the difference between what epidemiology has done
3 and what it can do.
4 Most of our attention has been focused on trying
5 to say what is important and so we have tended to go for the
6 things that show major increases. If people wanted to be
7 able to demonstrate an increase in risk of the order of 10,
8 15, 20, 25 percent, then for many substances such studies
9 could be done but they are going to be expensive and will
10 take more sample size.
11 From the scientific point of view, most of us have
12 gone on issues where we think there is a higher priority and
13 where we think the risk is higher and therefore the
14 prospective cancer control is greater. I would like
15 to find substances or risk factors that double and triple
16 the risk more than I would like to find ones that increase
17 the risk by 10 percent, unless of course the 10 percent had
18 50 times the number of people likely to die.
19 But we have tended to look for things we consider
20 important and where we thought we could make some stride in
21 cancer control.
22 MR. HAWKINS: Here is another statement.
23 "Epidemiological studies of the kind of magnitude normally
24 done on hundreds or a few thousand patients cannot detect
25 increased risks at the level of 1 percent or 1/10 of a

1 percent or 1/100 of a percent. Would you agree with that
2 statement?
3 MR. MORGAN: I think that's true. One 1/100 of a
4 percent on a thousand people is small. You are dealing with
5 less than a person.
6 MR. HAWKINS: Can we start with 1 percent?
7 MR. MORGAN: Okay, 1 percent. If you say 1
8 percent increase in risk, one of the commonest causes of
9 death is breast cancer. If you increase the rate of breast
10 cancer 1 percent, you would still have trouble detecting it
11 because the individual cancer is not frequent enough, even
12 though that is the commonest female cancer and that again
13 is a point I did not bring out in my testimony.
14 But we hear the discussion as though cancer is a
15 disease. I heard a speaker the other day say we should
16 start our sentences by saying cancer are because cancer
17 is a number of diseases and we should be ver careful.
18 The factors that influence skin cancer are vastly different
19 from those that influence bladder cancer which are vastly
20 different from those that influence breast cancer.
21 To talk about the cancer problem, we should be
22 talking about the cancers problem and when we talk about
23 air pollution, we have to say what organs is air pollution
24 most likely to affect, realizing we might not be able to
25 identify them all and perhaps concentrate on those issues.

1 Yes, we want to identify 1 percent changes or
2 even 10 percent perhaps, but when you are getting a 10
3 percent increase in something, I would be confident at
4 having a go at that, I would like to do some of those
5 studies, sure.
6 MR. HAWKINS: Finally, one final statement which
7 refers to a statement attributed to you, Dr. Morgan, with
8 respect to the Gerardi study on DCB where the OSHA record
9 has you stating, "I would feel reasonably confident that
10 the negative epidemiological studies of this material make
11 it unlikely that the substance is a human carcinogen" and
12 that's the end of the quote.
13 The document goes on to make the following
14 observation. "However, he -- presumably referring to
15 you -- cited an estimate by the statistician that worked
16 on the Gerardi study that there was one chance in ten of
17 risking a relative risk of three, presumably for bladder
18 cancer."
19 Is that an accurate characterization of the
20 chance of missing a positive risk and is it your judgment
21 that that would be regarded as a negative study?
22 DR. MORGAN: Again, we tend to -- we love to
23 separate things into negative and positive. Remember,
24 that is still a subjective statement. The objective is
25 the risk we have of missing -- we have to look at the

1 evidence and say are you willing to look at that as a
2 negative or are you not willing to believe it?
3 In that instance, I was willing to believe,
4 remember I said probably, I didn't say positively negative,
5 I said probably negative, isn't that right?
6 MR. HAWKINS: The quotation says that the
7 negative epidemiological studies of this material --
8 DR. MORGAN: I would call them probably negative,
9 realizing that we do put a statement in about what magnitude
10 our error might have. We have a 10 percent -- a risk of
11 three in that instance and I think that's a pretty good
12 size to indicate your likelihood of error, just the same
13 as if you put a statement in of your likelihood of making
14 a mistake the other way.
15 MR. HAWKINS: Is it your judgment that a study
16 with that probability of missing a relative risk of three
17 should outweigh a properly conducted clinical animal
18 exposure with a positive result?
19 DR. MORGAN: What is a clinical animal --
20 MR. HAWKINS: An animal exposure study with a
21 positive result properly conducted.
22 DR. MORGAN: I am a poor person to ask that
23 because I have great reservations about animal studies,
24 partly because of the high doses used where you might
25 overwhelm the metabolic defenses and therefore create

1 a dosage quite unnatural plus the problems in species
2 differences and there are even differences between rodents
3 and when you move from rodents to humans, I have a problem.
4 I am afraid my own biases would tend to say that
5 the best organ for human health is the human. If I think
6 studies have been reasonably well done, I tend to believe
7 the human data rather than the animal data, just as for
8 many years I did believe cigarettes caused lung cancer
9 even though the animal people have trouble showing me that.
10 MR. HAWKINS: I think the point of my question is
11 the regulatory weight a regulatory agency should assign when
12 confronted with a situation where you have a positive
13 animal study which is agreed by reviewers to have been
14 well-conducted and a "a probably negative" epidemiological
15 study which, when evaluated by a statistician can be
16 characterized as having one chance in ten of missing
17 a relative risk of three.
18 Is there a positive animal study that in your
19 view could be sufficiently well-conducted to outweight
20 that "probably negative" epidemiological work?
21 DR. MORGAN: There are a couple of ways of
22 looking at this. In science, one tends never to believe
23 one study, it should be replicated if at all possible.
24 If you are dealing with animals, I would suggest not only
25 should that study be replicated in that species to make sure

1 it was not experimental error, because such things have
2 happened, but it should probably be done in another species.
3 EPA's policy seems to say that even one species
4 would be pretty suggestive. When you come to a positive
5 evidence versus negative evidence, I would refer you to
6 my OSHA testimony as a statistical model for dealing with
7 that and I think you could compare animal versus
8 epidemiologic.
9 When you look at the epidemiologic thing, you
10 have to look at what the beta errors, as we call them in
11 statistics, were to get the power of the epidemiologic
12 study involved. I think it can be done but you are really
13 then in a case-by-case consideration and if you had
14 overwhelming animal evidence or even a couple of studies
15 and the epidemiologic data were so weak or not available,
16 then I would say that you, as a regulatory agency, may have
17 to go on the basis of animal data because that's all you've
18 got and all you're going to get.
19 But as in the saccharin case, if human data are
20 readily available or can be collected to answer the
21 question, then I would strongly urge you to go to the
22 human data.
23 MR. HAWKINS: Thank you.
24 MR. BARBER: Dr. Albert?
25 DR. ALBERT: Apropos of the human data outweighin

1 the animal data as exemplified according to your position
2 by the recent saccharin studies, it is my understanding in
3 talking with one of the investigators that relatively few
4 individuals that have been studied have taken saccharin for
5 20 years or more.
6 In view of the fact that the latency for cancer
7 can be greater than that, don't you feel that the saccharin
8 story is not in yet completely as an epidemiologist in view
9 of the fact that the duration of follow-up of these
10 populations is pretty clearly inadequate?
11 DR. MORGAN: I wouldn't say it's pretty clearly
12 inadequate. I would say that never in medicine or science
13 do we ever arrive at a position where all of the evidence
14 is in. One of the dilemmas EPA faces is that you are urged
15 to regulate and perhaps must regulate on a basis, at times,
16 of less than complete evidence.
17 But when one sees the amount of human evidence
18 on saccharin and puts it all together, and a fair number
19 of those people, I can count them exactly, have been
20 exposed for some years, one gets the impression there
21 should have been some increase in risk in those people
22 that's just not showing up.
23 I don't think the saccharin evidence is that
24 weak. I agree it's not complete and one could continue
25 and if we looked at it ten years from now, you could come

1 back and say the people you are looking at in the cancer
2 age group now are not the people exposed in utero and the
3 answer will not be in for another 70 years when people in
4 utero within the last ten years finally get to the age at
5 which cancer is most common, that is in the 70+ age group.
6 You reach a point where you say common sense tells
7 you that if you keep looking for evidence and don't find
8 any, there is probably no problem. To keep saying keep
9 looking, folks, because it's going to come sooner or later,
10 I don't think really solves the Agency's problem.
11 DR. ALBERT: I will just say that there are
12 plenty of instances where early judgments about carcinogenic
13 effects did not work out ultimately. Let me ask you another
14 point and that is that you indicate that there isn't any
15 evidence that air pollution is causing cancer.
16 There certainly has been a fair amount of
17 consideration of the epidemiologic evidence which looks
18 at urban and rural differences. Most recently, a group
19 chaired by Sir Richard Dahl and Norton Nelson came to the
20 conclusion that there could be an urban factor of perhaps
21 two, particularly for cigarette smokers.
22 Are you willing to dismiss this judgment as not
23 being true and also if you accept its validity, whether you
24 can write off air pollution as a major contributor to this
25 effect?

1 DR. MORGAN: The urban factor is a catchall for
2 many things. There are more industries in cities than in
3 the countryside, it is a given, so you have some occupational
4 exposure. You might have different smoking habits in the
5 city, you have automobile exhaust, different population
6 density.
7 You might conceivably have other factors, even
8 psychogenic. The fact that there is an urban-rural rating,
9 not to mention the fact that statistics tend to be swayed
10 somewhat by the fact that certain people move to the city
11 for care, but the urban-rural difference is not necessarily
12 explicable on the basis of air pollution.
13 Air pollution is a factor which I have stated is
14 perhaps under-investigated but to attribute urban
15 differences to air pollution when we have these vast
16 differences between cities in terms of air pollution
17 and cancer rates that don't support the air pollution
18 question, to my mind is somewhat speculative.
19 DR. ALBERT: But your statement is pretty
20 unequivocal, namely that there is no evidence of excess
21 cancer incidence due to air pollution. I just raise this
22 as something which --
23 DR. MORGAN: I don't think it is evidence, that's
24 why. I think it is evidence of something going on in the
25 urban environment but I think I've given you four or five

1 possible other factors that could be occurring in the urban
2 environment.
3 DR. ALBERT: But you agree, you can't dismiss
4 pollution?
5 DR. MORGAN: I would not dismiss it, I would
6 investigate it further. I'm not sure I am prepared to
7 advocate regulation on the basis of something that may
8 be in that wastebasket.
9 DR. ALBERT: One final point, that is that in
10 the analysis of cancer mortality that you present here,
11 you present it in terms of all sites minus lung cancer.
12 Have you looked at the trends in particular lung cancer
13 sites, particular sites including lung cancer in other
14 words, in the fashion that Schneiderman has done recently
15 in which he has presented evidence that there is an
16 increase in cancer of sites that are -- one would suppose
17 would be affected by external pollution to the extent that
18 the cancer rates are increasing in the order of 3 percent
19 per year?
20 Have you done the analysis of individual sites
21 yourself and do you have any comments about Schneiderman's
22 analysis?
23 DR. MORGAN: I have looked at lung cancer most
24 closely and the reason for the rise in lung cancer at the
25 moment, women are coming on very strong and the rate of ris

1 of lung cancer among males is diminishing.

2 DR. ALBERT: This is in the absence of cigarette

3 smoking?

4 DR. MORGAN: In the absence of cigarette smoking.

5 I'm not sure I know which publication of Schneiderman's you

6 are referring to. Is it published?

7 DR. ALBERT: I think it is, yes, it has been

8 presented at several meetings.

9 DR. MORGAN: One of the problems we have with

10 people when we see a slide, it's very difficult to analyze

11 it and I can't recall seeing an analysis --

12 DR. ALBERT: Perhaps you haven't seen that, but

13 have you looked at individual sites yourself in the absence

14 of, for example, cigarette smoking effects rather than just

15 taking a look at the gross --

16 DR. MORGAN: Lung cancer is presumably the one

17 that should be first affected, do you not agree?

18 DR. ALBERT: Yes, but I would point out that

19 Schneiderman's evidence is that in the absence of cigarette

20 smoking, you subtract that out, it is going up at the rate

21 of 3 percent a year.

22 DR. MORGAN: This is the question of non-smokers?

23 DR. ALBERT: That's right.

24 DR. MORGAN: We have a number of issues there

25 that have to be resolved. One is that as the proportion

1 of non-smokers among males, what have you, may decrease.
2 The question of pollution becomes even more important.
3 I know of no permanent non-smokers in this room who have
4 been unexposed to cigarettes.
5 On Friday, a statistician from New York State
6 presented a model showing the kinds of deaths one might
7 expect as a result of benzalphapyrene at low levels, the
8 kind you could get from passive cigarette smoking. Even
9 though the rates may be going up in lung smokers, it might
10 also be due to the same ideologic thing.
11 On the issue of the general air pollution in
12 cancer, there was a paper done by Hammond, Garfinckel, and
13 some others in which they said, talking about this urban-
14 rural thing, one of the sentences in their summary says,
15 among those not exposed -- this is not occupationally
16 exposed, there were little or no differences in mortality
17 ratios by urban-rural place of residence, in Los Angeles
18 by counties, whether they lived in cities, high or low
19 levels, or benzine soluble organic matter.
20 We conclude that general air pollution at present
21 has very little effect, if any, on the lung cancer death
22 rate. The reference, as I say, general air pollution cance
23 in the U.S.
24 MR. BARBER: I think Dr. Anderson has some
25 questions but I would like to get one little point of

1 clarification. You mention that we might not be able to
2 find whether non-smokers were suffering an increase in
3 cancer due to air pollution because they may have it masked
4 by side stream exposures to cigarette smoking.
5 How do we draw distinctions that somehow exposure
6 to benzalphapyrene in the ambient air could possibly get
7 into the air but we can't find it and the reason we can't
8 find it is that it is due to exposure from cigarettes?
9 I'm not sure I understand the logic there.
10 DR. MORGAN: What I would like to see is a
11 therapeutic trial. If we were to get rid of cigarettes
12 where there is very clear evidence that they should be
13 abolished and the cancer rate went down among non-smokers
14 as well as smokers, we would have very eloquent proof or
15 evidence that they were in fact getting an effect from
16 their smoking friends.
17 Monitoring can be done, statistical models we can
18 try but they are very difficult and the other thing is that
19 cigarette smoke is not a pure benzalphapyrene and there are
20 those substances in it and to contribute all cigarette
21 smoking cancer to benzalphapyrene might be underestimated.
22 Benzalphapyrene, sure, it's in the air from
23 automobile exhaust, chimneys. EPA, for instance, might
24 want to consider what they would like to do about fireplaces
25 and other forms of combustion, as well as from cigarette

1 smoking but cigarette smoke has things other than
2 benzalphapyrene.
3 MR. BARBER: So does the ambient air.
4 DR. MORGAN: Yes, but what I am saying is don't
5 count all the cigarette cancers as being due necessarily
6 to benzalphapyrene.
7 MR. BARBER: I didn't think I was. I was just
8 responding to the comment you made, but where I tend to
9 have difficulty is the sense that it would be useful to
10 regulate some things or control some things but it would
11 not be useful to regulate the chemical industry because
12 clearly we don't have a problem.
13 I just heard you say that EPA may want to
14 regulate fireplaces, that might be a useful thing to do
15 from a public health standpoint. But in no way would it
16 be useful to regulate emissions of, say benzine from the
17 chemical manufacturing industry.
18 DR. MORGAN: I did not say it was not appropriate
19 to regulate the chemical industry. I just suggested that
20 when you regulate, it should be on the basis of pretty soun
21 evidence rather than regulating on the basis of lack of
22 evidence.
23 In the fireplace situation, you have something
24 where this is a luxury rarely used for heat and where
25 regulation of that will not impose an enormous discomfort

1 on anybody, on very few people. If you tried to get rid of
2 benzene in the U.S. environment, I suspect we will all
3 suffer.
4 MR. BARBER: One may want to do something as
5 simple as controlling spills and leaks and some other
6 things or one might want to control the storage. The
7 sense that the only regulatory response is a nuclear
8 deterrent is not in concert with the proposal.
9 DR. MORGAN: I am relatively new to the United
10 States but if you have evidence something is causing cancer,
11 aren't you able to regulate it right now?
12 MR. BARBER: Sure.
13 DR. MORGAN: That's a point, I'm not arguing
14 against it. It's when we have suspicions of cancer rather
15 than evidence that we say maybe your regulation are
16 premature.
17 MR. BARBER: Any regulation?
18 DR. MORGAN: Again, a regulation that has social
19 or economic impact to a major degree, yes. Fireplaces I
20 don't think are a major thing.
21 MR. BARBER: I won't choose to debate that.
22 MR. HAWKINS: I just had one quick follow-up on
23 passive smoking. As I understood your response to Dr.
24 Albert, you said that if there is an increased rate of
25 instance being detected in non-smokers that could

1 conceivably be due to the passive smoking influence.

2 Again, not being a scientist, maybe I am missing

3 something, but I don't understand how you would associate

4 the increased rate of incidence with the fact that more

5 people are non-smokers. It might be an increase in the

6 absolute numbers, but how would you arrive at the conclusio

7 that an increased rate of incidence could be due to that

8 fact?

9 DR. MORGAN: Because as smoking habits have

10 increased, the exposure of the non-smoker to cigarette

11 smoke has also increased. You have an increase in the

12 rate there. It might also become more evident as larger

13 numbers, and this will be coming out shortly I hope, of

14 non-smokers become available to look at.

15 Although many of our non-smokers now were actuall

16 former smokers and the data on this was presented by

17 Lindstrom. Reading through his work, there is still

18 a lot of puzzling things and I think that is the kind

19 of study that is important and should get more attention.

20 Merely calculating the rates of lung cancer among

21 non-smokers is very deceptive and I think we ought to look

22 at occupation and other risk factors involved in that befor

23 we can make a conclusion.

24 DR. ANDERSON: I have some questions on Figure 7

25 which is the chart you have here. First of all, References

1 22 and 23 that you referred to in your caption are missing
2 from your bibliography. I wonder if you could tell us or
3 submit them?
4 DR. MORGAN: They are in the AIHC alternative
5 document. You have two documents there, you have the thick
6 one and my testimony.
7 DR. ANDERSON: Does this book tell us the source
8 of your data on the malignancy rate?
9 DR. MORGAN: Yes.
10 DR. ANDERSON: Are these data incidence data or
11 mortality data?
12 DR. MORGAN: That is mortality because that is the
13 best data we have for the United States. Incidence data are
14 rather weak in the U.S.
15 MR. BARNARD: May I respond, Dr. Anderson? My
16 name is Mr. Barnard. The figures showing the production
17 was taken from EPA report to the Congress, first EPA report
18 to the Congress on health, heart and lung disease, something
19 like that. The figures on the cancer rates were taken from
20 Dr. Schneiderman's testimony.
21 DR. MORGAN: We did look up those cancer rates
22 in U.S. biostatistics.
23 DR. ANDERSON: How did you separate -- do you
24 think you can separate the lung cancer rate from
25 environmentally caused cancer? To turn it around,

1 do you think all lung cancer is caused by cigarette smoking.

2 DR. MORGAN: No, I don't think all lung cancer is

3 caused by cigarette smoking, we know there are other factors

4 We know that lung cancer predated cigarettes but the

5 overwhelming proportion has certainly contributed to

6 by cigarettes.

7 You get into some areas where you have

8 interaction of cigarettes with things like asbestos

9 and that's another thing you have to look at. It's not

10 as simple as how much lung cancer is purely due to

11 cigarettes. How much is reasonably due to cigarettes,

12 how much is due to cigarettes plus another factor, how

13 much has no relationship to cigarettes.

14 DR. ANDERSON: I wanted to get back to your

15 overall testimony on the role of epidemiology in regulating

16 carcinogens. It seems to me that you are in essential

17 agreement with earlier witnesses today in that you think

18 you can't extrapolate from animal studies and dose response

19 studies to human incidence. Is that correct?

20 DR. MORGAN: That is my personal view but since

21 moving from animals to humans comes between the two

22 disciplines. Toxicologists are dealing with animals

23 and we are dealing with humans and I think it's very

24 difficult for either one of us to say you can't move

25 from one to the other. We don't seem to have a discipline

1 that allows it.

2 Toxicologists do it but most epidemiologists say
3 I don't think it should be done but on the other hand, I
4 can't say it should not be done.

5 DR. ANDERSON: I realize you are an epidemiologist
6 but can you talk to what you regard as legitimate evidence
7 for regulatory action? Does this mean that you think we
8 should have actual cancer deaths before airborne carcinogens
9 are regulated?

10 DR. MORGAN: What you are asking me is should we
11 kill people so we then know there is an association?

12 DR. ANDERSON: No.

13 DR. MORGAN: I have to answer that I don't want
14 anymore deaths, especially from cancer, than we can help.
15 What I've said about epidemiologic evidence though is that
16 people have died, their causes of death can be examined and
17 some of the previous exposure can be examined and we can get
18 some of those answers without waiting for future generations
19 to die by examining the experience we have had to date.

20 DR. ANDERSON: Do you think it's appropriate to
21 use animal studies to indicate the likelihood of carcinogenic
22 risk and use epidemiology to place an upward bound?

23 DR. MORGAN: That's an interesting way to go.
24 One of the instance that I have no quarrel with for animal
25 studies is to indicate to us as epidemiologists where we

1 should be doing our studies. If we found a positive
2 animal study and moved in with a series of epidemiologic
3 studies and we will call them negative but look very closely
4 at our confidence limits, lower confidence limits especially
5 and if those confidence limits get very close to one, we
6 might get worried.
7 So yes, we could put an upper bound but I would
8 be more concerned about the lower bound. If the lower bound
9 were getting awfully close, we might say statistically
10 significant or not, maybe you do want to regulate. There
11 is a judgment factor there and when we talk about negative
12 epidemiology, I don't like to divide the study into
13 quantitive or negative unless I am forced to.
14 I would really prefer to look at those confidence
15 limits of relative risk. Relative risk of one or the same,
16 no increase, but if I see confidence limits that go from
17 .95 up to 10.5, then that does not seem to discredit very
18 evenly and I might at that point say something concerns me
19 here.
20 If a study is well done and happens in both sexes
21 but there is no hard and fast rule.
22 MR. BARBER: Any other questions from the panel?
23 MR. JOSEPH: Dr. Morgan, two questions about
24 Figure 7, also from a non-scientist point of view. I think
25 you said earlier that there is not any demonstrated

1 connection in dealing with a synthetic organic chemical
2 production and cancer.
3 You said emissions in the earlier years of
4 manufacture were much greater. Is there any relation
5 here in this Figure 7 between emissions and cancers or
6 is it just production?
7 DR. MORGAN: I think that's an excellent point
8 that you have brought up because it is one that I was asking
9 about last week or so in that it is said that we cannot
10 relate current mortality because we only got recent
11 production.
12 But if you look at emissions and try to plug,
13 I would suspect emissions were high in the early years and
14 lower in the later years. There probably were a fair
15 number of people exposed in the earlier years but I would
16 agree that emissions are probably a better variable to look
17 at than production.
18 Given the fairly non-changing pattern of practice
19 for emission control, then production is probably a pretty
20 good variable for emissions but control technology has
21 improved pretty dramatically since 1915 and so emissions
22 were probably much higher than production represented.
23 MR. JOSEPH: One other question. Are these data
24 from the communities where the greatest concentration of
25 plants or emissions were or are they nationwide?

1 DR. MORGAN: Those are national.

2 MR. BARBER: National and independent of chemical.

3 I assume we have a different mix today than we had in 1935.

4 DR. MORGAN: That's probably true.

5 MR. PATRICK: I have one concern with showing

6 this kind of rather dramatic rise in production, the green

7 line. I suspect from my knowledge of the chemical industry

8 that both the character of emissions and character of those

9 chemicals being produced has been changing dramatically

10 through every year.

11 DR. MORGAN: I agree.

12 MR. PATRICK: We might be comparing apples and

13 oranges and bananas and grapefruit and I'm not sure it's

14 appropriate to use this type of comparison.

15 DR. MORGAN: I would agree, but one of the reason

16 apparently in reading some of the debate going on about the

17 need to regulate has been that graph, saying my God, look

18 at all these chemicals that are there and we should soon

19 be seeing signs of drastic health effects in them.

20 The mix has changed and the emission has changed.

21 I'm not sure production is a great thing to look at. I am

22 concerned about this question of substance versus all

23 chemicals, just as I am about a cancer site versus all

24 cancers.

25 I think we need more information on each substanc

1 and each cancer and I think it gets us very little ahead
2 to look at all substances versus all cancers. We stand
3 the risk of missing things, we stand the risk of making
4 false assumptions about what is going on.
5 That is why we need to study each chemical
6 individually and look at each site specifically. Then
7 we will get somewhere.
8 DR. ALBERT: I would like to know why, when you
9 plotted all sites minus lung cancer, you did not also
10 subtract out stomach cancer? You know very well that
11 the drop in stomach cancer has been as dramatic as the
12 rise in lung cancer and for reasons that I'm sure you
13 and I would agree are completely unknown.
14 Therefore, it seemed to me that if one wanted to
15 get a truer picture about what was going on, if you are
16 going to subtract out lung cancer, I think it would be
17 appropriate to subtract out stomach cancer, too.
18 DR. MORGAN: The reason we took out lung cancer
19 was because the cause for most of it is known. If it's
20 known, we can in fact remove it. The cause of most of
21 the other cancers is unknown. So it's appropriate to
22 take out that one and say after we control for smoking --
23 we could have also taken out a portion of bladder cancer,
24 we could have taken out a portion of laryngeal, we could
25 have made the line more dramatic going down.

1 DR. ALBERT: That's right and that is why I don't
2 think this type of analysis is very discriminating but I
3 gather you do.
4 DR. MORGAN: Any time you deal with all cancer,
5 it is not discriminating and that's one of the mistakes in
6 lumping all cancers in and calling it the cancer problem
7 because it should really be the cancers problem.
8 MR. BARBER: Thank you, Dr. Morgan, you've been
9 very patient.
10 I think we should break the hearing here until
11 2 o'clock. We will resume.
12 (Whereupon, the hearing was recessed for luncheon
13 at 1:00 P.M.)
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1 A F T E R N O O N S E S S I O N

2 2:00 P.M.

3 MR. BARBER: It's 2 o'clock, I would like to

4 reconvene the meeting.

5 I have several speakers who, due to travel

6 arrangements and the time that we are taking, have run

7 into conflicts so I am going to make some minor adjustments

8 in the schedule and the first three speakers will be taken

9 slightly out of order.

10 The first speaker will be Ms. Gloria Rains.

11 MS. RAINS: I am Gloric C. Rains of 5314 Bay

12 State Road, Palmetto, Florida, here to represent Manasota-88,

13 the Florida Division of the IWLA and the Environmental

14 Confederation of Southwest Florida, a five-county, 51-member

15 environmental coalition representing over 21,000 people.

16 We support the EPA Administrator's decision to

17 list radionuclides as hazardous air pollutants under

18 Section 112 of the Clean Air Act but differ with proposed

19 approach to control these emissions.

20 This is particularly important to the residents

21 of Florida because of the health hazards created by the

22 phospate mining industry and the imperative need to control

23 them and because of the magnitude of the hazards the

24 industry poses we will address some important aspects

25 of phospate mining that should be considered when selecting

1 radionuclides for control as well as proposed Agency
2 procedures.
3 Richard Guimond of EPA has noted that the phosphat
4 industry currently mines more total uranium than the uranium
5 industry. As of 1976, the State of Florida was the biggest
6 mining state in the United States, producing 321 million
7 short tons of ore, a major portion of it phosphate.
8 This mining process has resulted in approximately
9 166,000 acres of disturbed land in the phosphate region --
10 none of which according to Dr. Wallace Johnson, Director
11 of Surveillance at the Division of Radiological Health
12 Service in Florida will meet EPA guides for radiation
13 levels.
14 According to a 1978 Florida Health and
15 Rehabilitative Service report, the annual excess exposure
16 to Radon-222 for persons living on reclaimed lands within
17 the study area is calculated to be 540 millirems per year
18 to the whole lung.
19 The results of this and other date developed,
20 according to Mr. Mills, Chief of EPA's Radiation Section,
21 is that living on reclaimed lands in Florida is comparable
22 to exposures experienced by Army personnel during the early
23 testing days of atomic bombs and which, according to the
24 Governor's Task Force, will result in people facing vastly
25 increased levels of lung cancer.

1 According to Dr. Secova in Health Physics, it
2 will also lead to a dramatic increase in skin cancer.
3 (Secova et al, Health Physics, 35, 803-806.)
4 EPA addresses the increased lung cancer problem
5 in EPA Report, Indoor Radiation Exposure Due to R-226 in
6 Florida Phosphate Lands. (EPA 520.4-78-013, Feb. 1979)
7 According to EPA Volume ORP/LV-781, additional
8 recent reports indicate that mining and processing of
9 phosphate ores technologically enhance the quantities of
10 naturally occurring radioactive materials such as radium
11 and uranium available to man in the environment. These
12 radionuclides have long half-lives and will persist in
13 our environment for thousands of years.
14 The magnitude of this problem is shown in current
15 estimates that the total radioactive waste generated by the
16 uranium and the phosphate mining and milling industry are
17 comparable to each other. As an example, 152 million tons
18 of hazardous gypsum waste containing thousands of Curies
19 of R-226, are deposited in Florida in what can only be
20 described as radioactive dumps.
21 Until EPA's decision to list radionuclides as
22 hazardous waste, pollutants, the phosphate industry has
23 essentially neither been regulated nor monitored for the
24 possession, use or discharge of radioactive materials
25 associated with phosphate rock and its products.

1 This points up the immediate need to promulgate
2 regulations in the prescribed time in order to reduce the
3 hazards caused by this industry to nearby populations.
4 According to the Federal Register, December 27,
5 1979, there are currently 20 rock drying and grinding
6 facilities in Florida, most of them located in Southwest
7 Florida.
8 One phosphate rock dryer emits 64.5 tons per
9 year of particulates and 2,450,000,000 picoCuries of R-226,
10 an amount which exceeds the equivalent radionuclides which
11 would be allowed by the NRC from a 1,000 megawatt nuclear
12 power plant. (Central Fl. EPA EIS)
13 This means Floridians are currently exposed to
14 amounts of radiation which exceed that of equivalent
15 radionuclides allowed by the NRC for 20 1,000 megawatt
16 nuclear power plants.
17 Despite scrubbers and stack dispersal, this
18 means radionuclides are released and dispersed into
19 populated areas. This distribution is significant and
20 dangerous and its effect is increased when combined with
21 the effects of our region's almost nightly inversions.
22 During the process of mining phosphate, radon-222
23 routinely escapes into the atmosphere where it travels long
24 distances. The significance of this can be found in
25 Federal Register, December 27, 1979 where the exposure

1 level, regional population, person working level is 4.9,
2 higher than that found in uranium mining and milling sources.
3 (Uranium being 1.3 WL for underground mines, .38 for open
4 pits and 0.54 for mills.)
5 Particulates from routine mining operations,
6 transport of the rock and loading operations also create
7 problems. Among other things, alpha particulates, thought
8 to remain in the lungs for years, also cause death before
9 cancer becomes evident. There are 35 mines in Florida.
10 Based on EPA Volume ORP/LV-781, stack sampling
11 of phosporic acid plants show significant concentrations
12 of radionuclides are being discharged into the environment.
13 These plants are usually located in close proximity to
14 residential areas. There are 35 such plants in Florida.
15 These plants are capable of producing up to
16 4,570,000 pounds or almost 20 percent of the 1975 uranium
17 production in the country. This operation combined with
18 mining, other processing and redistribution of by-products
19 of phosphate ore create concentrations and redistribution
20 of radioactive substances and release of radioactive gases
21 and particulates in close proximity to human populations.
22 There is one uranium extraction plant associated
23 with these plants currently operating in Florida with at
24 least three more to come on line. Production was 184 tons
25 of uranium oxide in 1979 with projected production for this

1 year to be 680 tons.

2 These operations discharge additional levels of
3 radionuclides into the environment as a result of the
4 uranium recovery operation. It is acknowledged that
5 increased cancer and mutation rates are by-products of
6 uranium operations.

7 Also, airborne aerosols from the phosphate
8 fertilizer industry have been found to be enriched with
9 uranium and polonium-210.

10 In areas of particularly high incidence of
11 cancer in our state, an indication exists that there may
12 be a correlation between increased numbers of cancers and
13 high ambient radiation from phosphate combined with high
14 fluoride levels emitted by the industry.

15 It should also be noted that according to Dr.
16 Oleg Selawry of the University of Miami, Florida has the
17 highest rate of lung cancer for people under 55 in the
18 United States.

19 While evidence indicates that each cancer may
20 be caused by multiple factors, by developing regulations
21 to eliminate emissions of radionuclides and other hazardous
22 air pollutants known to cause cancer, we can break a link
23 in the carcinogen chain.

24 Obviously, based on these facts, other available
25 data and that information printed in the December 27, 1979

1 Federal Register, there can be no doubt as to the need to
2 regulate the radionuclides emitted by phosphate industry
3 operations and we would hope the Agency will live up to
4 its obligations to develop regulations within one year.
5 The question then arises as how to best
6 accomplish this.
7 The EPA Administrator speaks of a policy of
8 providing an ample margin of safety for the public. Yet,
9 according to Drs. Gofman and Tamplin and other renowned
10 scientists, there is no safe level of radiation exposure.
11 Each exposure increases the risk. In any case, no threshold
12 or safe exposure to a carcinogen can be identified.
13 Further, the present policy of what appears to
14 be placing excessive reliance on quantitative assessments
15 in trying to establish risks is imperfect and can only
16 result in unnecessary death and suffering. The wide
17 variance in cancer risks projected from consuming diet
18 soda shows how imperfect quantitative assessments are.
19 The next example may not be so harmless.
20 We do not think the Congress endowed the
21 Administrator with the right to weigh the costs of saving
22 a human life and that is what determining significant risks
23 to public health are. Nor do we think the American people
24 have any idea this is a responsibility the Administrator
25 has undertaken. Publication in the Federal Register is

1 hardly adequate notice of such a policy and certainly
2 appears to be denying Americans a constitutional right --
3 the right to due process.
4 We cannot believe that anyone should weigh the
5 costs of saving a human life as opposed to the regulation
6 costs to the industry. How do you decide what somebody's
7 life is worth? Who gives you the right? We think it has
8 been amply demonstrated that this was not the intent of the
9 Congress or the court.
10 These facts should justify a zero risk emission
11 standard being set for radionuclides emitted by the
12 phospate mining and milling process. This is the only
13 standard that affords the public reasonable protection.
14 Such a standard will motivate industry to develop better
15 operating procedures, particularly in the case of phosphate
16 mining.
17 In summary, we support EPA's decision to list
18 radionuclides as hazardous air pollutants under Section 112
19 of the Clean Air Act and would ask this be done
20 expeditiously in compliance with the Agency's obligation.
21 We also join with the Natural Resources Defense
22 Council and the Environmental Defense Fund in supporting
23 their proposed changes for rulemaking criteria and
24 procedures. Thank you.
25 MR. BARBER: Thank you. I was not clear on the

1 exact nature of the recommended change but it sounded as
2 if you were suggesting the same type of zero emissions goal
3 that we heard discussed this morning. Do you have a sense
4 of what the exact step that you are recommending would be
5 for that industry? Is it more specific engineering, is that
6 the goal here?
7 MS. RAINS: Yes, in this case of reclamation,
8 there are techniques that exist. In the case of phosphoric
9 acid plants, there are scrubber techniques that exist that
10 would probably come close to zero emissions. In the
11 uranium recovery operations, I think there are techniques
12 also that are available.
13 MR. BARBER: What you are suggesting, if I may
14 paraphrase, that the Agency should be looking at more
15 effective controls for that industry as opposed to looking
16 at trying to close that industry?
17 MS. RAINS: Actually, it's no concern to me and
18 on a national basis, I don't think it should be of any
19 concern to our nation. The phosphate industry at this
20 point contributes very little economically to our nation
21 and is a non-renewable resource that we can consider a
22 critical resource and that we can only gain by conserving
23 the amount we now have, if that's what it takes in order
24 to conform to no emission regs.
25 MR. BARBER: There's a world of difference

1 between diminished mining and no mining. If we do any
2 mining at all, I have the sense we will have some emissions
3 and the goal is to have a better controlled industry.
4 MS. RAINS: I think that we can get this down to
5 essentially zero or almost or no emissions with proper
6 technology.
7 MR. BARBER: Thank you. Any other questions
8 from the panel?
9 Thank you very much.
10 We call Mr. Arthur Gregory.
11 MR. GREGORY: Mr. Chairman, ladies and gentlemen.
12 I am here today -- my name is Arthur Gregory and I am from
13 -- I am testifying as an individual scientist. My address
14 is 220 West Ash Road in Sterling, Virginia and I am pleased
15 to appear here to consider with you a particular aspect of
16 environmental carcinogenesis. That particular aspect is
17 assessment of oncogenic potential.
18 Initially, I would like to say that EPA's
19 proposed airborne carcinogen policy is an important and
20 significant undertaking. There is little doubt that the
21 general public is any less deserving of protection than
22 the working population for which the recent OSHA carcinoger
23 policy has been formulated.
24 My particular concern here today is how these
25 known environmental carcinogens will be assessed for

1 potential regulation. The National Cancer Advisory Board
2 in an unprecedented concensus of agreement made the following
3 statement regarding evaluation.
4 "Each case must be individually evaluated, taking
5 into consideration such factors as adequacy of experimental
6 design, statistical significance of the data, dose response
7 relations, duration of exposure, route of administration,
8 metabolism, including species variations, host susceptibility,
9 co-factors and other modifying factors and the amount of
10 material to which humans will be exposed."
11 It was only through the superhuman efforts of my
12 good friend Herman Craybill that that last sentence was
13 added.
14 A sound scientific evaluation of the quality and
15 significance of the data regarding carcinogenesis is a key
16 element in risk assessment. Yet even this Adelphi type
17 method is insufficient if each expert utilizes different
18 criteria in his own evaluation.
19 I would like to repeat that sentence because it
20 is very, very important. It is insufficient, any Adelphi
21 method is insufficient if each expert utilizes different
22 criteria in his own evaluation.
23 Today I would like to suggest a method of
24 quantitating not only the quality of the data but a method
25 of weighing the potency of the carcinogen with respect to

1 its realistic probability of causing cancer in man.

2 Basically, carcinogen data can be divided into

3 two groups, animal studies and human studies. It has alway

4 been my basic contention that once tumors are demonstrated

5 in homo sapiens, we have waited too long to study that

6 carcinogen.

7 Vinyl chloride, and bis methyl chloro ether are

8 excellent cases in point. While both are easily demonstrab

9 in animals, they were indeed first demonstrated in human

10 beings. Indeed, that is why we found out that methyl chlor

11 ether produced a particular type of tumor of the lung,

12 so-called oat cell tumor which indeed was just too rare

13 to be accounted for on a statistical basis.

14 Yes, we waited too long. This does not mean by

15 any stretch of the imagination that we should ignore

16 epidemiological data if it already exists. Indeed, man

17 is the best model of man.

18 The method I propose is based on sequential

19 algorithms in which results and observations are assigned

20 numerical scores and this assessment of oncogenic potential

21 combined with a reasonable cost-benefit analysis would

22 then allow regulatory action to provide a reasonable

23 safeguard for society and an acceptable working margin

24 for industry.

25 To quote a phrase, "All carcinogens were not

1 created equal." Some are worse than others. The method
2 I propose results in five categories of carcinogens;
3 definite, highly probably, probable, equivocal and unlikely.
4 Without such a categorization as this, environmental
5 carcinogenesis has little meaning in the real world that
6 each of us must live in. Thank you.
7 MR. BARBER: Thank you. I have a question,
8 towards the last part of your statement, you had five
9 categories. How do you feel that that differs? We had
10 three categories that we talked about in the proposal and
11 then a category for research in essence. Do you have a
12 sense of how one goes about assigning a chemical to a
13 category?
14 MR. GREGORY: In one word, quantitatively by
15 assigning a number to each one.
16 MR. BARBER: And the number is drawn from the
17 weight of the evidence in essence?
18 MR. GREGORY: Right. The quality of the animal
19 bioassay, for example, is scored from one to ten. Each
20 qualification gets a single point. If there are less than
21 four, it is deemed not quantifiable. If there are ten,
22 that's a perfect score.
23 On the basis of this, when you start out with
24 four, six, seven, or ten, you weight each one of these
25 with respect to things like latency period, things like

1 multiplicity of tumors, things like dose response
2 relationship, etc.
3 MR. BARBER: Has the proposal been reduced to
4 writing?
5 MR. GREGORY: Yes.
6 MR. BARBER: Is that something you could submit
7 to the record for us to look at?
8 MR. GREGORY: Yes, I will submit it to the record
9 MR. BARBER: Thank you. Any questions from the
10 panel?
11 Thank you very much, Mr. Gregory.
12 The next speaker would be again out of order to
13 accommodate transportation, Mr. Harry Demopoulos.
14 DR. DEMOPOULOS: Thank you, Mr. Chairman, I am
15 Dr. Harry Demopoulos, Associate Professor of Pathology at
16 NYU. I am speaking on behalf of several other individual
17 scientist citizens who include Dr. Joseph Cimino, President
18 of New York Medical College in New York. He is also a
19 former Commissioner of Health from New York City.
20 Dr. Bernard Wagner, Professor of Pathology and
21 Toxicology at Columbia's Comprehensive Cancer Center, Dr.
22 Bernard Young Professor of Nutrition at MIT, Dr. Benjamin
23 Van Duuren, Professor of Environmental Medicine at NYU as
24 well as Dr. Jesse Steinfeld, presently Dean at the Medical
25 College of Virginia.

1 The reason I am here on behalf of these
2 individuals stems largely from two symposia held in 1979
3 in New York City and in part some of these symposia had
4 their origins when I was director on a leave of absence
5 from NYU of the Cancer Institute of New Jersey which was
6 a nonprofit fledgling organization that was formed in 1976
7 to address the seeming problem of high cancer rates in New
8 Jersey.
9 Interestingly, the Environmental Defense Fund's
10 petition to EPA cited the New Jersey statistics as one of
11 the first examples that would suggest a link between cancer
12 rates being elevated and industrial or general air pollution.
13 So I have a specific interest in addressing the question of
14 air pollution and cancer rates.
15 I am also editor of the book of one of the
16 symposia which is being published through the Journal of
17 Environmental Pathology and Toxicology which is an organ
18 for the American College of Toxicology.
19 I would like to go through this with a series of
20 slides because I think it would be an efficacious manner to
21 present the data. All of the material I will mention has
22 been presented to you in writing in the form of preprints
23 and reprints from Science as well as from Preventive
24 Medicine as well as from the Journal of Environmental
25 Pathology and Toxicology.

1 (Slide presentation.)

2 DR. DEMOPOULOS: Relevant to what the Environment
3 Defense Fund cited in its petition, the statistics for New
4 Jersey showed that, as far as cancer mortalities were
5 concerned in terms of white males per 100,000 derived
6 from the NCI's cancer mortality study by county for 1950
7 to 1969, it showed that indeed there were 205 male deaths
8 per 100,000 compared to the national average of 174 per
9 100,000.

10 This increase without regard to controlling the
11 demographic considerations was immediately blamed on the
12 very heavy chemical industry that exists in that state.
13 Furthermore, in a study conducted by the New York City
14 Department of Health, which was also cited by the
15 Environmental Defense Fund, showed that in certain
16 areas of New York City, like Staten Island, Bay Ridge
17 section of Brooklyn, Red Hook, Williamsburg, the rates
18 of cancer of the lung were inordinately high.

19 Again, the conclusion was immediately made,
20 without controls, that these high lung cancer rates in
21 New York City were due exclusively to air being carried
22 in the prevailing direction from west to east from heavily
23 polluted chemical industries of New Jersey.

24 Up in central Harlem, however, which was north
25 of the prevailing polluted winds, the New York City

1 Department of Health blamed that on cigarette smoking. Also
2 ignored were obvious skip areas, for example, in lower east
3 Manhattan.
4 Furthermore, this data was not corrected according
5 to race and there were other obvious statistical and
6 epidemiologic flaws in the study. Nonetheless, examples
7 like this that are not controlled had been cited as evidence
8 and rationale for controlling airborne carcinogens.
9 We started the study at the Cancer Institute of
10 New Jersey using a grant from the National Cancer Institute
11 when I was director of CINJ to answer the question of what
12 are the high rates of cancer in New Jersey possibly due to?
13 I must admit that when I first got there, I was typical of
14 most New York City people and prone to blame the chemical
15 industry.
16 Much to my surprise, I started looking at the
17 cancer mortality statistics and I found that Rhode Island
18 was a neat second, right behind New Jersey. I found New
19 York was pretty close and then I looked at the U.S. Census
20 where there is an awful lot of data relating to the types
21 of industries and I started categorizing the different kinds
22 of industries to see if there was a link.
23 My surprise, places like Rhode Island and New
24 York do not have the same kind of chemical industry and
25 pollution that exists in New Jersey. Furthermore, I took

1 a look at Pennsylvania and Ohio wherein again 30 to 40
2 percent of the work force in Pennsylvania and Ohio work
3 in the same kind of worrisome polluting carcinogenic type
4 of industries, all in quotes, as you find in New Jersey.
5 Thirty to 40 percent of the population lives in
6 communities that are in close worrisome proximity to
7 industrial plants in Pennsylvania and Ohio as you find
8 in New Jersey and yet the cancer rates in Pennsylvania
9 and Ohio are not significantly above the cancer rates
10 expected in the nation as a whole, that is, 174 per
11 100,000.
12 Ohio had 178 and Pennsylvania had 183, in spite
13 of having the same proportion of people and workers living
14 in and around and working in the same kinds of industries
15 that you find in New Jersey. This lack of consistency
16 troubled us and we decided to take a closer look.
17 We found that New Jersey has a very urbanized
18 population according to the U.S. Census. Ninety percent
19 of the people live within urbanized areas and the density
20 of urbanization is the highest in the country. Nearly
21 5.5 million of New Jersey's population lives in crowded
22 conditions in the northeastern part of the state.
23 In demographic terms, this part of New Jersey
24 is most similar to a city. If you do proper demographic
25 controls and ignore for the moment the political fallacies

1 of states and counties and cities and consider demography,
2 where you pack a certain number of people into a certain
3 geographic area, and then compare the cancer rates,
4 independent of whether industry or air pollution are
5 present or not, this is the kind of thing shown on the
6 slide here.
7 Nassau County had cancer rates for white males
8 of 212 per 100,000. There is no significant polluting
9 industry in Nassau County. San Francisco had 206 per
10 100,000, again San Francisco is virtually devoid of the
11 types of industries found in New Jersey.
12 The District of Columbia has rates that are
13 almost the same as New Jersey's. Westchester County
14 where I live is a pristine, pastoral area devoid of
15 industry. When you compare these areas to New Jersey,
16 we find the rates are higher or the same as New Jersey's,
17 despite the absence of industry and despite the absence of
18 significant kinds of air pollution and airborne carcinogens
19 that you worry about in New Jersey.
20 This is all from the National Cancer Institute's
21 cancer mortality study by county. The data was not changed
22 in any way but was simply taken out of the tables and
23 listed accordingly. We went a step further in our NCI
24 supported study and we looked at incidence data.
25 We used the third national cancer survey which

1 examined the cancer incidence among 20 million people durin
2 the years 1969, '70 and '71. Fortunately, within that thir
3 national cancer survey, there were seven convenient cities.
4 It was not originally planned that way but the data were
5 there.
6 Within the third national cancer survey of the
7 seven cities, Detroit, Pittsburgh and Birmingham, which is
8 the Pittsburgh of the South, as well as San Francisco,
9 Atlanta, Dallas and Minneapolis. Those seven cities
10 represented 16 million people, a significant number.
11 We went again to the U.S. Census and examined
12 other statistics and found that in the cities of Detroit,
13 Pittsburgh and Birmingham, as you might imagine, the
14 percentage of the work force and the percentage of people
15 living in communities in and around heavy manufacturing
16 plants were 40 to 45 percent, whereas in contrast, places
17 like Atlanta, San Francisco, Dallas and Minneapolis have
18 about 10 to 15 percent of their work force engaged in
19 worrisome types of industries and 10 percent of the
20 population living in proximity to the sparse number
21 of industrial chemical plants.
22 There is a vast difference in the type of
23 industries you find in Atlanta, San Francisco, Dallas
24 and Minneapolis. There is also a vast difference in the
25 degree of ambient air pollution and water pollution betweer

1 the three dirty cities and the four clean cities.

2 We looked at the cancer rates and examined white
3 male cancer rates. This is incidence data, as well as black
4 male cancer rates. The rates displayed here are for whites
5 but in summary, it shows that the three dirty cities, as you
6 want to call them dirty cities, had 8 percent less cancer
7 compared to the four clean cities.

8 I don't think the 8 percent lower incidence in
9 the dirty cities is significant and I would not advocate
10 anyone moving there to decrease their risk. On the same
11 token, we found no significant difference between lung,
12 nasopharynx, stomach cancer rates in the three dirty cities
13 than the four clean cities.

14 These statistics were examined a bit further and
15 we were concerned that perhaps there might be some age
16 differences, the curves may be skewed. Fortunately, the
17 third national cancer survey provided data according to
18 five year blocks shown on the top row and we examined the
19 overall cancer rate according to the five year blocks for
20 the three dirty cities and four clean cities and found no
21 skewing of the incidence data according to age.

22 We thought maybe in the three dirty cities, we
23 might see some somewhat higher cancer rates if industry and
24 pollution were involved in the lower age groups. Perhaps
25 the cancers were occurring earlier but we could not find

1 any such skewing of the data.

2 The other thing I would like to point out about

3 the third annual cancer survey in the three dirty cities

4 is that the survey was done for '69, '70 and '71.

5 Considering the lag phase of human cancer averaging

6 about 20 to 25 or 30 years, these cancers were beginning

7 to develop in the 1940's and '50s.

8 This was a time when these three cities had

9 virtually no controls, there were no regulatory agencies

10 of the kind that we have now in posing rigid controls over

11 industry exposures, emissions and work force habits. So to

12 a large extent, the third national cancer survey gives us a

13 good examination of worst case conditions in three cities

14 that are heavily industrialized at a time when no kinds of

15 significant controls were present.

16 According to NCI's data, it did not make any

17 difference, the cancer rates were, if anything, lower than

18 the four clean cities.

19 Going back to the National Cancer Institute's

20 cancer mortality studies, we compared Los Angeles with San

21 Francisco. Los Angeles, according to the National Wildlife

22 Federation which keeps track of such things, has about 340

23 days a year where the air is called unhealthy.

24 San Francisco has about 80 to 90 such days.

25 Furthermore, Los Angeles is a significant petroleum county.

1 Most of the refineries are located along the
2 coast and the prevailing winds blow from west to east so
3 the air from the oil refineries is carried inland into L.A.
4 County to mix with all the automotive smog. San Francisco
5 by contrast has light industry, sparse chemical industry
6 and is bathed by pure Pacific Ocean air, again coming in
7 from west to east.
8 When we look at the NCI's own data, we find that
9 L.A. has 175 cancer deaths per 100,000 compared to the U.S.
10 nationwide average of 174 per 100,000. San Francisco has
11 212 and when we look at lung, nasopharynx and stomach, we
12 find again that San Francisco has significantly higher rates
13 and L.A. has consistently average nationwide cancer rates.
14 These data are not consistent with any kind of
15 airborne carcinogen problem in this country as far as we
16 can see from the NCI's data on mortality and incidence.
17 Furthermore, these kinds of studies have been conducted
18 by other individuals and have also been reported by Dr.
19 John Goldsmith who was at the National Cancer Institute
20 and at the Department of Health in Berkeley, California.
21 At the cancer symposium in March, Dr. Goldsmith
22 independently found the same kinds of results that I found.
23 That is, that the urban factor where we see higher cancer
24 rates in urban areas compared to rural areas cannot be
25 attributed to air pollution.

1 Furthermore, he found that lifelong residents
2 within urban areas actually had lower lung cancer rates
3 -- compared to people who had recently immigrated into urba
4 areas.
5 Thirdly, Dr. Cuyler Hammond had a symposium held
6 in June by the American Health Foundation and presented dat
7 from the American Cancer Society where he and Larry
8 Garfinckel and others addressed the question of the
9 urban factor and looked into air pollution and looked
10 specifically for links with lung cancer.
11 Again, Cuyler Hammond found independently of
12 myself and Dr. Goldsmith that general environmental air
13 pollution and water pollution cannot be blamed for any
14 measurable increases in cancer risk in this country.
15 The causes of cancer, I think, are significant.
16 The two symposia pointed out that there was a
17 remarkable concordance of data arrived at from different
18 sources. That fully 35 percent of cancer deaths in this
19 country are caused by smoking high tar cigarettes and
20 drinking excessive quantities of alcohol.
21 Somewhere around 45 or 50 percent are attributab
22 to disordered nutrition which includes excess calories,
23 excess fat, obesity of 30 to 40 pounds and nutritional
24 deficiencies of fiber and Vitamin A. These subcategories
25 of disordered nutrition taken together account for

1 approximately 45 to 50 percent of the cancers.

2 Occupational exposure accounts for somewhere less
3 than 5 percent. Background radiation accounts for 3 percent,
4 preexisting medical disorders 2 percent and prescription
5 drugs about 1 percent. In the two symposia and the studies
6 I have cited, air pollution and water pollution accounted
7 for no percent.

8 Our conclusion, therefore, and my reason for
9 being here is to point out that we do not perceive any
10 kind of rational such as the EPA has offered nor that the
11 Environmental Defense Fund has offered that suggests that
12 there is a link between air pollution and cancer risks in
13 this country.

14 It may exist in other countries, I am not sure.
15 But the available data that has been looked at objectively
16 and is beyond question shows no links. I might remind you
17 that these are under worse case conditions, particularly in
18 the three dirty cities and places like Los Angeles.

19 The three dirty cities have been in existence for
20 a long time. They have been industrial centers for more
21 than 60 years. There has been more than enough time for
22 any kind of cancer risk to have shown up by this time.
23 It doesn't exist and we think that your rationale for
24 trying to control airborne carcinogens is nonexistent.

25 Let me conclude with yet one final example. In

1 New York City, the crysotile, that is, asbestos fiber
2 content, is very high in Manhattan. In fact, compared
3 to the other boroughs, Manhattan has up to 65 fibers or
4 nannograms compared to Staten Island.
5 This is data from Nicholson at Mount Sinai. We
6 were kind of interested in this because Manhattan has been
7 the site of intensive construction over the past several
8 decades. Manhattan also has a fair number of bridges and
9 tunnels.
10 The New York University Cancer Center has an
11 investigative tumor registry and has had one for sometime.
12 I have helped to create it when I planned the NYU Cancer
13 Center. The NYU Cancer Center derives its patients from
14 the Manhattan Veterans Administration Hospital, Bellevue
15 Hospital which services the lower end of Manhattan and
16 University Hospital, as well as both Memorial Hospital
17 which is smack in the middle of the Borough of Queens.
18 Within the various medical centers in New York
19 City, NYU happens to have the only investigative tumor
20 registry that provides a cross section of the New York
21 City population. Memorial Sloane-Kettering has far too
22 many referrals from the rest of the country and the rest
23 of the world.
24 Memorial's cancer population is not representativ
25 of New York City. Mt. Sinai gets its referrals of

1 mesotheliomas after they have been diagnosed elsewhere.
2 The other centers do not have investigative tumor registries.
3 We felt therefore that, in view of the very high asbestos
4 content in Manhattan, that if there was any kind of
5 significant risk, we should see it at NYU if we looked
6 over our mesothelioma diagnoses over the last 10 or 12
7 years.
8 I might add, furthermore, that the consistency
9 of diagnosis of mesothelioma at NYU have -- particularly
10 good, largely because the pathologists have not been changed,
11 they are the same ones, they've been there for 15 years and
12 have all been trained by Dr. Morgan Cushner, one of the
13 experts in diagnosing mesotheliomas.
14 So we looked for a trend in mesotheliomas at NYU
15 and found none over the past ten years. The last date
16 reported here was 1976 and we have subsequently updated
17 it to 1977 which gives us a total of 12 years and we cannot
18 find a trend for mesothelioma incidence within NYU. It's
19 a straight plateau.
20 We think this is a good example of a carcinogen
21 which is exaggerated. We think at NYU that we see more
22 people exposed to more asbestos than most other parts of
23 the country and we have more high-rise construction workers,
24 more bridge and tunnel toll takers than any other part of
25 the country and yet we don't see any kind of a trend in

1 mesotheliomas.

2 There has been enough time by 1976 to have seen
3 such a trend. It isn't there. Our strong suggestion is
4 that, contrary to the dogma proposed by various regulatory
5 agencies, we propose that there is in fact a threshold to
6 carcinogens and that there is a valid scientific basis for
7 the threshold based on DNA repair and the fact that many of
8 the carcinogens, both chemical and physical, operate throug
9 free radical mechanisms controlled by anti-oxidants to a
10 large extent.

11 We furthermore feel that based on the mechanism
12 of carcinogenesis involving free radicals, that the use of
13 extrapolation models where high doses are used in animals
14 and extrapolations are attempted as you get to lower doses
15 are inherently falacious because they do not take into
16 account the fact that at high doses, the anti-oxidant
17 levels in the liver and other organs are immediately
18 exhausted and you are no longer dealing with the normal
19 animal but rather an animal that has had its anti-oxidants
20 at its first line of defenses abolished.

21 At low doses, these anti-oxidants are more than
22 adequate to handle low dose exposure to carcinogens, proof
23 that thresholds exists or are found in studies like the
24 three dirty cities. Proof is also found in the fact that
25 smoking 1 milligram tar cigarettes at the rate of 10 to 15

1 a day does not even cause any pre-malignant changes in the
2 bronchial epithelium in the autopsies of humans.
3 In short, we feel strongly that pointing the
4 nation into the direction of believing that air pollution
5 is a significant carcinogenic risk is fallacious and
6 distracting from the other causes of cancer and consequently
7 in pointing to air pollution as a cancer problem is wrong,
8 dangerous and can actually be counterproductive in terms of
9 preventing cancer because it detracts from the acknowledged
10 causes of cancer that are believed by most of the academic
11 university scientists. Thank you.
12 MR. BARBER: Thank you, Dr. Demopoulos. I would
13 have a few general questions. You made a number of
14 references to clean cities and dirty cities and I, having
15 looked at the inventories of many of the cities around the
16 country, I guess that your characterization is not as
17 obvious to me as it is to you.
18 Is there air quality data that supports the
19 characterization of these cities, is there an inventory
20 or some sense of what was in the air that people breathed?
21 DR. DEMOPOULOS: During the years 1960 and the
22 early '70s, the inventories of the air pollutants in the
23 seven cities described had been begun. There is no
24 inventory of any significance of air pollutants before
25 the '60s in those seven cities.

1 However, if you look at what those seven cities
2 were doing in terms of their manufacturing and chemical
3 processes, and if you examine the consumption and productio
4 of various chemical products and manufactured goods during
5 the '40s, '50s, and early '60s, it gives you a pretty good
6 idea of what the pollution and occupational exposures were.
7 MR. BARBER: Was that done?
8 DR. DEMOPOULOS: Yes, through the U.S. Census.
9 MR. BARBER: In the process of categorizing these
10 cities, there is an inventory of chemical use and potential
11 emission?
12 DR. DEMOPOULOS: Yes, we have done that and it is
13 reported in the paper, New Jersey and other urban centers,
14 it is a preprint that I have and submitted as part of our
15 data.
16 MR. BARBER: Pursuing that, I have not read the
17 paper yet but I will. It does go to the question of the
18 chemical balance for Providence as opposed to Washington
19 and these other cities?
20 DR. DEMOPOULOS: These are described, yes.
21 MR. BARBER: When you did your Los Angeles versus
22 San Francisco comparison, were those data just raw data or
23 were they handled in a way similar to the other cities?
24 DR. DEMOPOULOS: That was data taken -- it was
25 not raw data. It is from the NCI's cancer mortality study

1 by county and it is corrected to age, sex, race. It's not
2 raw data. All the data I showed you is age corrected and
3 it has been -- it is the data published by NCI in that big
4 telephone-like book on cancer mortality studies and in the
5 third national cancer survey.
6 MR. BARBER: In your analysis, there is an
7 estimate of what the relative concentrations of different
8 pollutants in those two cities might be?
9 DR. DEMOPOULOS: Yes.
10 MR. BARBER: When you looked at asbestos in New
11 York, you had numbers, if I remember, between 5 and 30
12 times 10 to the minus 9 grams per meter cubed. What is
13 the method of determining that concentration?
14 DR. DEMOPOULOS: That was from Dr. Nicholson's
15 own data from Mt. Sinai. I would have to refer you to his
16 original paper for the methodology. It is stated in one of
17 the papers I submitted as part of our evidence, the
18 references are given there. It's Nicholson, not our
19 own calculations.
20 MR. BARBER: To the best of my knowledge, people
21 talk about estimating asbestos calculations to orders of
22 magnitude and I would have to look at the numbers carefully.
23 In the context of the asbestos, you suggested
24 that there was not a trend in New York, despite the fact
25 that New York City seemed to have higher concentrations

1 than other places.

2 DR. DEMOPOULOS: Manhattan specifically.

3 MR. BARBER: You did not show any evidence that

4 the concentration of asbestos has changed. Do you somewhere

5 try to relate the change in exposure --

6 DR. DEMOPOULOS: The point made about asbestos is

7 that you eventually should see higher cancer rates and

8 certainly higher rates of mesothelioma which is a harbinger.

9 Despite the fact that there are high asbestos rates in the

10 general air in Manhattan, I think Nicholson sampled somethir

11 like eight different places in a period of three years

12 within the Island of Manhattan, that you should eventually

13 see a trend.

14 In other words, over the period of time, even

15 though the asbestos level remains the same --

16 MR. BARBER: What would cause the trend?

17 DR. DEMOPOULOS: Asbestos, according to dogma,

18 is among the non-threshold carcinogens. There should be

19 more and more mesotheliomas developing all the time if we

20 are to believe the dogma and if it has any validity.

21 MR. BARBER: If the asbestos had been there a

22 long time and had not changed very much, I'm not sure --

23 DR. DEMOPOULOS: The asbestos has been there for

24 years. We don't know how many. High-rise construction has

25 been going on for a number of years in New York City but

1 according to their best estimates of individuals like Dr.
2 Selikoff, we are supposed to be seeing the beginning of an
3 explosion of mesotheliomas and other cancers caused by
4 asbestos. Presumably, we would see these earlier trends
5 in places where asbestos exposures are highest.
6 On the Island of Manhattan, we have pretty
7 significant asbestos exposures. It is higher than almost
8 any other part of the country in terms of general community
9 air pollution. The asbestos you inhale is cumulative, it's
10 like radiation supposedly where once you breathe in the
11 asbestos, regardless of threshold considerations, that
12 will exert a continuing carcinogenic effect that eventually
13 shows up so that you do see or should expect to see a trend,
14 a continuing rise.
15 We challenge that view, we believe in thresholds.
16 The data is offered that despite the fact that we have
17 significant asbestos exposures in Manhattan, there is no
18 trend. This is contrary dogma and contrary to the no
19 threshold concept.
20 MR. BARBER: I will need to have Roy explain to
21 me why we should see a trend there but I have concern about
22 how we define the asbestos level in Manhattan to be high.
23 DR. DEMOPOULOS: If you look at other parts of
24 the country, and this has been done, there are asbestos
25 readings for Denver and most other metropolitan areas as

1 well as rural areas. Manhattan happens to have high
2 asbestos exposures.
3 Let me point out one additional thing and it may
4 clear up a point of confusion that I apologize for. In the
5 third national cancer survey, the numbers of mesotheliomas
6 that were diagnosed in the (perenia) and the pleura
7 averaged down so you would expect to see, not on the
8 basis of populations but for every thousand cases of
9 cancer, all kinds of cancers, you would see about three
10 new mesotheliomas.
11 It's another way of looking at incidence in
12 terms of the percent of total cancer risk. Within NYU's
13 statistics, we found 2.8 new mesotheliomas per 1,000
14 suggesting that despite a higher asbestos exposure in
15 Manhattan, we do not see not only a trend or the lack
16 of a trend but we do not have higher mesothelioma rates
17 compared to third national cancer survey.
18 MR. BARBER: If I might continue, when you talk
19 about urban factors, you suggested that none of the urban
20 factor could be attributed to air pollution, i.e., zero,
21 which is a pretty absolute number. Dr. Morgan suggested
22 that air pollution was one of the things that made up the
23 urban factor and I'm not sure whether you are in
24 disagreement or whether I am misunderstanding.
25 DR. DEMOPOULOS: Well, I don't agree that

1 general air pollution is one of the factors in the urban
2 factor. It's been examined by three separate studies,
3 Goldsmith's, Hammond's and my own and we come to the same
4 conclusion, that air pollution is not one of the things that
5 makes the urban factor significant. Air pollution is just
6 not involved in the urban factor.
7 MR. BARBER: It's zero, there is no risk of
8 cancer to people in this country from ambient air exposures?
9 DR. DEMOPOULOS: That's correct, that's the
10 conclusions of three separate papers and you have copies
11 of them.
12 MR. BARBER: I have to compliment you for being
13 explicit. The last question that I have is that you
14 mentioned at one point that we should not be concerned
15 about the fact that cities in Group A and cities in Group B
16 had an 8percent difference in the numbers or the cancer rate
17 because 8 percent was not significant. I'm not sure how I
18 put that back into --
19 DR. DEMOPOULOS: Having an 8 percent lower
20 incidence in cancer in three dirty cities, probably if
21 you could work out the statistics, I would suggest that
22 8 percent lower is not significant. My point is that there
23 is no important difference in the cancer incidence between
24 dirty cities and clean cities. There is no measurable
25 difference.

1 MR. BARBER: Dirty and clean are defined in the
2 papers?
3 DR. DEMOPOULOS: Yes, sir.
4 MR. BARBER: That answers my questions. Roy,
5 do you have any questions?
6 DR. ALBERT: Harry, it seems to me the use of
7 trends of mesothelioma at the NYU Medical Center can only
8 be an extremely crude measure. After all, people drift
9 through Manhattan like water through a sieve. They don't
10 live there necessarily very long.
11 There have been gross changes in the composition
12 of the population of Manhattan since World War II,
13 tremendous influx of various groups, tremendous eflux
14 of different groups. Also, I am surprised to hear that
15 you think the NYU Medical Center has a representative
16 patient group for Manhattan as a whole. It's fairly
17 regional hospital complex with respect to Manhattan Island.
18 Do you think the points that I am making are not
19 so hot?
20 DR. DEMOPOULOS: No, I think the points you make
21 are good points, Roy. The fact of the matter is --
22 DR. ALBERT: We are on a first name basis still.
23 DR. DEMOPOULOS: I would admit that the NYU
24 mesothelioma study is at best a crude study. However,
25 there is no question that vast population changes have

1 taken place in Manhattan. What I should specify is that
2 the Manhattan VA Hospital admits patients who are veterans
3 and who reside in Manhattan.
4 Furthermore, Bellevue serves the lower Manhattan
5 community. University Hospital, of course, serves a mixture
6 of Manhattan as well as a few other boroughs. But in
7 looking through the tumor registry data, we attempted to
8 look at how long these people lived in Manhattan.
9 By and large, the majority of the mesothelioma
10 cases that we diagnosed had been in residents who had been
11 in Manhattan for more than 20 years. This is one of the
12 functions of the tumor registry, to look at address locations.
13 But I will admit that this is not the best kind of study.
14 But the point I'm trying to make, there were two
15 points to that study. Number one, that despite Manhattan's
16 having the highest general community air pollution of
17 asbestos in this country, our mesothelioma rates were
18 at the national "expected" rate if we used the third
19 national cancer survey of three new mesotheliomas per
20 1,000 new cancer cases.
21 This is the way we chose to express our
22 mesothelioma data, because it happened to be convenient
23 and comparable to the third national cancer survey. So
24 the NYU rates, the Manhattan rates, are not any higher
25 than the third national cancer survey despite the fact

1 that the asbestos exposures in our populations are probably
2 higher.
3 Furthermore, there is no perceptible trend.
4 That does not mean that we are in favor of asbestos exposur
5 or air pollutants or anything like that. I think controls
6 are necessary and I think that each of the scientists I am
7 speaking on behalf of as individuals feel that controls are
8 necessary but they ought to be based on good science.
9 We have been looking for this question of
10 airborne carcinogens and looking for the data and we
11 just don't find data to support the rationale. That is
12 troublesome and mind you, we started off with a bias that
13 vapored your proposal and we had to turn our thinking aroun
14 because the data not only does not support your rationale
15 but it negates the hypothesis.
16 DR. ALBERT: In any event, the basing -- you do
17 agree that the basing of population trends on the kinds of
18 data one gets out of the hospital is subject to all the
19 biases that are well-known in epidemiology?
20 DR. DEMOPOULOS: Certainly, but the NCI mortality
21 study and the NCI third national cancer survey which I alsc
22 reported on are not subject to the kind of errors that I
23 must admit are present in the NYU mesothelioma studies.
24 DR. ALBERT: The concordance is nice for the
25 point you are making but it could be happenstance?

1 DR. DEMOPOULOS: Only as far as the mesothelioma
2 study at NYU but the NCI study and the third national
3 cancer survey are pretty clear-cut.
4 DR. ALBERT: To go on to the other data you
5 presented, the impression I got was that if you talk in
6 terms of dirty cities versus clean cities or comparing
7 Nassau County to a county in the State of New Jersey and
8 that sort of thing, if you are comparing total cancers,
9 you are essentially burying or running the risk of burying
10 any air pollution related cancers in a big ball of other
11 cancers.
12 DR. DEMOPOULOS: We looked at the anatomic sites
13 as well, including lung, nasopharynx and stomach.
14 DR. ALBERT: To take this one step at a time,
15 the comparison of total cancers is not very persuasive
16 because you would not expect that air pollution, a) would
17 have an overwhelming effect and b), that it would affect
18 more than a limited number of cancers. The odds of seeing
19 anything on a comparison of total cancers is not all that
20 great or not surprising to see no differences.
21 Then if you get down to specific cancers such as
22 lung cancer, the question is, how can you make these
23 comparisons without some method of correcting for the
24 effects of cigarette smoking which is well-recognized
25 to be the dominant effect here?

1 I think you showed a higher incidence of lung
2 cancer in San Francisco versus Los Angeles. Do they smoke
3 more in San Francisco? What do they do different there?
4 DR. DEMOPOULOS: I wish I knew.
5 DR. ALBERT: Smoke more pot. If you can't
6 correct for that, it sort of weakens the whole argument.
7 You eliminate the contribution of chemicals.
8 DR. DEMOPOULOS: The answer to your question is
9 really found in the third national cancer survey, 16 millio
10 people among seven cities. When you have large numbers lik
11 that, there is a tendency for some of the major variables
12 like cigarette smoking, I would seriously doubt, for
13 example, that if you could find the data which is hard
14 to come by for cigarette smoking in the 1940's, '50s and
15 '60s in the three dirty cities versus the four clean cities
16 I would doubt that there would be a significant difference
17 in cigarette smoking within the 16 million people among
18 those seven cities.
19 I think that would randomize out and approach a
20 similar level. The problem is we don't have the cigarette
21 sales during the key formative years preceding the third
22 national cancer survey for those cities.
23 DR. ALBERT: But you do agree, I guess, that if
24 one is going to compare lung cancer -- and -- lung cancer
25 under different circumstances that it would be a darn nice

1 thing to be able to normalize for the effect of cigarette
2 smoking?
3 DR. DEMOPOULOS: I think it would be borne out
4 statistically that with 16 million people, you pretty well
5 have things normalized.
6 DR. ALBERT: What's the evidence for that?
7 DR. DEMOPOULOS: Just basic statistics that when
8 you increase the numbers of your samples and you are dealing
9 with cities, the chances are pretty good that you will
10 normalize out cigarette consumption.
11 DR. ALBERT: It sounds plausible but is there
12 any -- there are clearly differences between and amongst
13 these cities. You characterized them as dirty and clean.
14 Surely there must be different things going on in them as
15 well?
16 DR. DEMOPOULOS: There may be but there is
17 another factor and that is cancer is a random event. We
18 learned recently that disordered nutrition plays a major
19 role in modifying and modulating an apparent development
20 of cancer.
21 In experimental animals, you can modulate
22 downward the number of cancers that will form by loading
23 them up with naturally occurring anti-oxidants as you might
24 find in some fresh fruits and vegetables, for example,
25 cauliflower and cabbage which are very good anti-oxidants,

1 loading animals up with indoles and multilated hydroxy
2 toluene can decrease the development of cancer so in answer
3 to your question or at least a possible answer to your
4 question as to why you do see differences among cities,
5 it may well be nutrition, that's one real possibility and
6 proximity to fresh produce and availability of fresh produce
7 may be a factor.
8 As a matter of fact, an early winter conference
9 -- American Health Foundation -- the question you raised
10 earlier as to what is making cancer of the stomach disappea:
11 one of the thoughts the panel put together was that the
12 greater availability of fresh produce and anti-oxidants
13 found there in which depend on good trucking and
14 refrigeration during freight transfer are probably
15 a major role in the decline of stomach cancer.
16 There are nutritional factors that modulate
17 cancer rates up or down. The point, however, is that
18 amongst 30 to 40 percent of the work force and communities
19 that lived and worked in and around dirty places, Detroit,
20 Pittsburgh and Birmingham, that 30 to 40 percent factor
21 should have been sufficient to raise the cancer rates by
22 at least 15 percent.
23 Again, these calculations are in the paper being
24 published in the Journal of Environmental Pathology and
25 Toxicology and you have a preprint of it.

1 A 30 to 40 percent work force community being
2 exposed to worrisome kinds of industry and worrisome kinds
3 of plants. If the hypothesis were correct that airborne
4 carcinogens are a major factor, we should have seen a 15
5 percent increase in overall cancer rates in the third
6 national cancer survey. We didn't.
7 DR. ALBERT: What about the factor that I don't
8 expect would be taken into consideration of population
9 migration? For example, in Detroit, I guess that was a
10 dirty city, there was a tremendous influx of blacks from
11 the south after World War II. They would have been --
12 DR. DEMOPOULOS: The data is race corrected as
13 well and the question of migrants is answered to some extent
14 by John Goldsmith's studies. He particularly looked at
15 lifelong residents versus migrants in two cities and he
16 found that lifelong residents in cities had lower cancer
17 rates than migrants in two cities.
18 DR. ALBERT: How do you square your position
19 with the data on migrants, for example, studies of people
20 coming from England and settling in places like South
21 Africa where there is relatively little air pollution
22 and whose smoking habits were corrected for showing a
23 higher incidence of lung cancer in such populations
24 provided they spent part of their early years in a
25 heavily polluted area before they moved out? Do you

1 think this is fallacious?

2 DR. DEMOPOULOS: No, I don't think it is

3 fallacious but by the same token, I have confined my

4 remarks in my studies to data obtained in this country

5 and I find that it adds certain levels that are confounding

6 when we start dealing with other countries moving to get

7 other countries.

8 I can't deal with it and I don't think it's

9 necessary to square it. I think it's important to look

10 at what's happening in this country and that's confusing

11 enough.

12 MR. BARBER: One question I would like to ask,

13 you talked about asbestos levels in New York or level of

14 different pollutants. One of the things we tend to observe

15 is that concentrations of the pollutants we are concerned

16 with tend to be several orders of magnitude greater in the

17 immediate vicinity of the plant or immediate vicinity of th

18 mine or the emitting source than it would be in the urban

19 area.

20 Consequently, whatever the risk is, if it's zero,

21 then three orders of magnitude greater exposure would not

22 do anything. We seem to recognize that occupational

23 exposure can contribute to excess cancers and you say

24 general urban exposure can't. Is your position as firm

25 for fence line exposures to occupational carcinogens?

1 DR. DEMOPOULOS: I have had opportunities to look
2 at fence line exposures and for things like vinyl chloride
3 monomer, the plants I have reviewed, the fence line
4 exposures are as low or lower than what you would find
5 in many urban settings.
6 MR. BARBER: Vinyl chloride?
7 DR. DEMOPOULOUS: Vinyl chloride monomer, yes,
8 even under worse conditions where you might have a reactor
9 blow. If you look at plants with a 10,000 pounds of vinyl
10 chloride monomer might be released, the circulation of the
11 air will give you momentary rise in vinyl chloride monomer
12 for a couple of hours and then it's rapidly dissipated.
13 Standard emissions under normal plant operations
14 for most vinyl chloride plants are not high at all. They
15 are almost zero emission plants. When you get down to
16 somewhat beyond the fence line and go to half a mile
17 or a mile distant from the fence line, there is no
18 difference in VCM exposures under normal operating
19 conditions than you find in cities.
20 Cities have -- these things are dissipated,
21 they are carried in the air. I don't challenge the idea,
22 for example, that carcinogenic substances can be carried
23 long distances. This has been well proved and I just don't
24 think they are present in a high enough dose to be worrisome.
25 MR. BARBER: Living next to a coke oven would be

1 no different than living 30 miles away from a coke oven?

2 DR. DEMOPOULOS: I think if the coke oven is

3 operating properly.

4 MR. BARBER: We can be reasonably certain we can

5 measure the concentration to be several orders of magnitude

6 greater?

7 DR. DEMOPOULOS: I don't know what you mean by

8 living next door to a coke oven.

9 MR. BARBER: Half a mile away?

10 DR. DEMOPOULOS: Versus 30 miles?

11 MR. BARBER: Yes.

12 DR. DEMOPOULOS: Depends on where the other 30

13 miles is.

14 MR. BARBER: No other source.

15 DR. DEMOPOULOS: For what substance?

16 MR. BARBER: Coke oven emissions, benzalphapyrene

17 Is there no place outside the fence line that you --

18 DR. DEMOPOULOS: I don't think I'd worry about it

19 because in every municipality, you have incinerators, you

20 have things being burned, you have fireplaces, you have

21 town incinerators, city dumps, leaves that fall from trees

22 every autumn that degenerate and release all kinds of

23 polycyclic hydrocarbons.

24 I don't think you can point to coke oven

25 emissions as if they're some kind of special phenomenon.

1 You get carcinogens all over the place in all municipalities
2 independent of industry.
3 MR. BARBER: But you have a pretty strong sense
4 that if it is outside the fence line, it's of no
5 consequence?
6 DR. DEMOPOULOS: That's correct and I might add
7 that most occupational exposures that I am aware of at the
8 present time also do not pose much of a risk. I think that
9 the 5 percent or less of cancer mortality that is
10 attributable to occupational exposures is a past phenomenon.
11 It is the results of exposures in the past when
12 things were not understood to be carcinogens, when the
13 nation was hot in pursuit of the cancer virus and chemical
14 and physical agents were poorly perceived as carcinogenic
15 substances.
16 I think as things have come under control, I
17 think the overwhelming majority of occupational exposure
18 and occupational carcinogens and cancers will see the same
19 kinds of declines that we are seeing in cancer rates
20 throughout the country, except for lung cancer.
21 Incidentally, I would like to comment on
22 Schneiderman's idea that non-smoking lung cancer is not
23 going up. That's based on a supposition that only 75
24 percent of lung cancer is due to cigarette smoking rather
25 than 85 or 87 percent.

1 That lowering my 10 percent by Schneiderman is a
2 simple calculation that he made on the basis of no data.
3 Cuyler Hammond's data shows that you can contribute at
4 least 85 to 87 percent of lung cancers on cigarette smoking
5 Most people think it's higher. I question the validity of
6 whether there is a rise among cancer rates among non-smoker
7 based on Schneiderman's data.
8 MR. BARBER: Any other questions from the panel?
9 DR. ANDERSON: My questions have to do with
10 measurements of pollutants 20 to 30 years ago. I know
11 you could not have measurements for chrysotile 20 or 30
12 years ago.
13 DR. DEMOPOULOS: That's right.
14 DR. ANDERSON: I wonder if some of the counties
15 you've discussed in your survey or the cities, you have
16 actual measurements of pollutant carcinogens from all
17 sources of exposures for these counties or cities, say
18 back in the '40s?
19 DR. DEMOPOULOS: No, that data clearly does not
20 exist. However, what does exist is the tabulation of what
21 industries were doing at that time. You know full well tha
22 there were no environmental controls on those industries.
23 In other words, Detroit has been mass-producing automobiles
24 In order to put chrome or paint on a piece of
25 metal, you have to degrease it. They've been using

1 degreasing agents, solvents, halocarbons, benzenes for
2 decades in Detroit. This is a requisite step in automotive
3 manufacturing.
4 Steel, iron production in Pittsburgh, 30 and 40
5 years ago, I can assure you was not being carried out with
6 any kinds of environmental controls. By looking back at
7 what the industries were doing, although we do not have
8 the finite air pollution data, I can tell you what kinds
9 of things were present in horrendous quantities and are
10 now present in much lower quantities thanks to the controls
11 that had been imposed.
12 I am not opposed to controls, none of us are.
13 What we are arguing against is the thesis that air
14 pollution is a random major contributing factor to
15 cancer in this country and there is no data for that.
16 While I do not have data for specific air pollutants
17 30 or 40 years ago, I know what industries were present
18 because I went through the U.S. Census and looked at the
19 industries and I went through other statistics showing what
20 was being produced and I know what was in the air and I can
21 assure you, it was much higher than it is now.
22 DR. ANDERSON: I think the problem I am having
23 is that you are comparing it in a very gross sense to the
24 mortality figures now opposed to worker exposures 20 or 30
25 years ago without consideration for workers working in and

1 out --

2 DR. DEMOPOULOS: I am not comparing anything to
3 20 or 30 years ago. I am comparing cancers in the third
4 national cancer survey which is one point in time among
5 three dirty cities and four clean cities. We know the
6 dirty cities and what was going on there fore 30 or 40
7 years, we have the industrial history of those seven
8 cities.

9 I am not comparing cancer rates along a different
10 time course. I am comparing cancer rates that occurred in
11 1969, '70 and '71. Those are the years of the third
12 national cancer survey among three dirty cities versus
13 four clean cities where we absolutely know the industrial
14 history and we know that four clean cities that are
15 unqualified in being considered clean relative to Detroit,
16 Pittsburgh and Birmingham.

17 DR. ANDERSON: Again, I think that I don't feel
18 all that certain because you are talking about industry,
19 you are not talking about all sources of exposure. You
20 are comparing total incidence with what you know about
21 history of industrialization in certain cities and you
22 are drawing very strong conclusions that there is no link.
23 I think that kind of data is very difficult --

24 DR. DEMOPOULOS: It's not difficult at all, I
25 beg to differ with you because you are ignoring the

1 industrial districts of Detroit, Pittsburgh and Birmingham.
2 They used to have a tremendous pollution problem in those
3 three cities and they still do.
4 It's better now than it used to be. That's
5 acknowledged and you can see that with your own EPA data.
6 If you go back to your first collections of air pollution
7 inventories in those three cities and look at what it is
8 now, you have a pretty good idea of what used to go on when
9 you had no controls 30 or 40 years ago.
10 We were producing automobiles and steel and lots
11 of other things in those cities and they were dirty and they
12 have been dirty for more than 60 years. There were no
13 emission controls over their industrial sites and therefore
14 pollution was under worse case conditions.
15 If you look at decreases in pollutants in those
16 three cities from the first time EPA has been collecting
17 data up until now, you will see a more downhill trend.
18 The air has become a lot cleaner and my point is that
19 now that things are getting cleaned up, what are you
20 worried about?
21 MR. BARBER: The question is, have we controlled
22 the right things in terms of a concern for risk of
23 carcinogenicity? Are the emissions from the steel
24 industry that have been controlled emissions of concern?
25 I know of no significant program undertaken to control a

1 myriad of solvents or other volatile organics in their end
2 uses.
3 I suspect that today's chemical plant is cleaner
4 than a chemical plant of 30 years ago for a variety of
5 reasons but I am not sure there has been very systematic
6 effort to control emissions of volatile organics, some of
7 which are of concern in this proceeding.
8 The transition in Pittsburgh from where it was to
9 where it is, say it has cleaned up, I am not sure that has
10 changed the concentrations of volatile organics in
11 Pittsburgh. Perhaps there is some evidence I am not
12 aware of but there certainly has not been a regulatory
13 program trying to accomplish that.
14 DR. DEMOPOULOS: There may not be a dramatic
15 increase involving organics in those three cities but the
16 point is they have been using volatile organics and
17 producing millions of automobiles for several decades.
18 In Detroit, they have been producing metal and producing
19 steel and iron and several thousand tons per year in each
20 plant in these cities of Pittsburgh and Birmingham.
21 Their rates of production are quite high, their
22 rates of producing automobiles has been quite high, their
23 works of emitting organic solvents and potentially
24 carcinogenic substances have been pretty high. The
25 point I'm trying to make is, despite the fact that they've

1 been in business for more than 60 years, we do not see
2 higher cancer rates, not overall and not within specific
3 anatomic sites.
4 We looked at specific anatomic sites, including
5 lung, nasopharynx and stomach. We also looked at leukemias
6 and urinary bladder cancers as well as liver cancers. These
7 are all potentially industrially related and might be
8 disseminated to air and water.
9 MR. JOSEPH: I just have one question, Dr.
10 Demopoulos. Your conclusions about the existence of
11 thresholds, does it apply to radionuclides as well?
12 DR. DEMOPOULOS: Probably not because the
13 chemicals, chemical carcinogens are somewhat different
14 from radioactive substances and radiation in general.
15 Most chemical substances have to be activated to be
16 carcinogens and within the endoplasmic reticulum of
17 the liver, for example, at the low dose of a carcinogen,
18 most of that material by virtue of the fact that it is not
19 overburdening the anti-oxidant system will be handled and
20 modulated into a non-carcinogenic metabolite.
21 As you increase the dose of chemical carcinogens,
22 you run the risk of having some of the chemical carcinogen
23 transformed into an active carcinogenic metabolite. Of the
24 total administered dose of the chemical carcinogen at a low
25 dose, most of it is going to be detoxified.

1 This is not the case with radionuclides, ionizing
2 radiation or any radioactive material in general, there you
3 are dealing with a different phenomenon. The injurious
4 substance is injurious and does not have to be modulated
5 in one way or another by the host.
6 As far as thresholds of radioactive substances
7 and radiation, I do not put onto that the firm belief in
8 thresholds that we have for chemicals.
9 MR. JOSEPH: Do you have a view on the duration
10 of elevated exposure that might be necessary to overburden
11 the anti-oxidant system? I'm referring back to your
12 example of a malfunction at the vinyl chloride plant.
13 DR. DEMOPOULOS: The spectrum of anti-oxidants
14 we have evolved with are many and varied. There are about
15 a dozen of anti-oxidant systems, including enzyme systems
16 and you would have to have repetitive exposures to most
17 chemical carcinogens to exhaust or wear out the anti-oxidan
18 system.
19 In general, the application of most chemical
20 carcinogens is most effective by giving in broken up dosage
21 schedule, that is, repetitive applications. There are
22 admittedly some bad actors that you can just give one
23 exposure to and these potent carcinogens are somewhat
24 different. They may be direct acting or they may be
25 able to skirt by the detoxification mechanisms.

1 MR. JOSEPH: How do we know which are which?

2 DR. DEMOPOULOS: In general, you can get some
3 indication of potency. Even from the Ames test, Bruce
4 Ames, I was on a panel with him January 5th in San Francisco
5 at the American Association for the Advancement of Science
6 where this very question was described.

7 Bruce Ames went through a rather detailed
8 presentation indicating for the benefit of some of the
9 federal regulators who were there the fact that the Ames
10 in vitro tests can be a strong indicator of potency of a
11 carcinogen.

12 There are even rapid in vitro tests that can give
13 you potency.

14 MR. JOSEPH: You are suggesting that the levels
15 at which thresholds may be found vary with potency?

16 DR. DEMOPOULOS: I think more potent carcinogens
17 will have a lower threshold, there's no question about that.
18 There are weak carcinogens and some substances that are on
19 the borderline. Substances like trichlorethylene have
20 recently been shown by Caesar Martoni and Pat Van Buren
21 to be non-carcinogenic if you look at true maximum tolerated
22 doses.

23 MR. JOSEPH: How are we as a regulatory agency
24 to draw the lines as to what Walt referred to as fence line
25 exposures between things that may be significant and may not

1 be significant because concentrations are higher or potency
2 is higher?
3 DR. DEMOPOULOS: When you get down to the nitty-
4 gritty, I think there are abundant numbers of university
5 scientists who volunteer their time to come and consult
6 with you free of charge to help you on specific questions
7 like this.
8 I fail to understand why we are not more often
9 used or called upon.
10 MR. JOSEPH: Calling upon you right now, do you
11 think then at least there are some instances in which fence
12 line exposures to some more potent carcinogens might create
13 a somewhat significantly elevated cancer risk?
14 DR. DEMOPOULOS: I would have to hear the specifi
15 substances on a specific dose and study it for a few days
16 before I could give you any kind of categorical answer like
17 that.
18 MR. JOSEPH: You at least have not given me a
19 categorical note.
20 DR. DEMOPOULOS: That's correct. Mind you, no
21 scientist that I know of and the few people I have met in
22 the industry, I have yet to meet anyone who is for the
23 abolition of controls or the non-placement of controls.
24 Everyone that I have met and everyone that I know of in
25 universities wants reasonable scientifically based controls

1 My whole reason for being here is to advocate
2 that we use more science in helping you to regulate and
3 I think this is the only way we can protect more and more
4 people with less and less hassle. You just don't use enough
5 science and it is available and there are people ready and
6 willing to help you.
7 MR. BARBER: Any other questions?
8 DR. ALBERT: Just as a point of information, on
9 the data you've presented from the third national cancer
10 survey comparing different cities, were these corrected
11 for duration of residence? Again, a similar point to the
12 one I made about your mesothelioma business in Manhattan
13 with the population essentially drifting through, flowing
14 through at a pretty substantial rate, this is a mobile
15 society. I would expect the population turnover in various
16 cities ought to be pretty high.
17 DR. DEMOPOULOS: The data was corrected for race
18 and many of the population changes that we have experienced
19 in cities has been an influx of non-whites. The white data
20 in general, when you look at 16 million people, this is yet
21 another variable which is probably muted by the size of the
22 experimental pool.
23 More specifically, Dr. John Goldsmith looked at
24 this question of migrants in cities. As I said before,
25 lifeline residents in cities had lower lung cancer rates

1 than matched controls with age, sex and race were matched
2 of migrants coming into cities.
3 The lifelong exposure to air pollutants in a city
4 not only did not increase the lung cancer risk but it
5 decreased them, at least in his studies. So it is contrary
6 to what you would expect.
7 DR. ALBERT: Right, but the comparison you show
8 here is between dirty cities and clean cities. If the
9 population moves through both, at a fairly decent clip,
10 and you don't correct or you don't just look at the
11 population that's lived there for a substantial period
12 of time, decades, then you are getting a dilution effect,
13 aren't you?
14 DR. DEMOPOULOS: But it's the same in all of ther
15 DR. ALBERT: But the comparison is between clean
16 and dirty.
17 DR. DEMOPOULOS: We don't imagine there was anym
18 movement in the three dirty cities versus the four clean
19 cities.
20 DR. ALBERT: Where do these people come from?
21 It could well be that the major flow-through is from peopl
22 from relatively clean areas.
23 DR. DEMOPOULOS: Dirty cities? Usually, it's th
24 other way around.
25 DR. ALBERT: All I'm saying is that I simply

1 asked as a point of information whether or not this was
2 taken into account?
3 DR. DEMOPOULOS: That was not taken into account
4 in the study and it was deemed to be a non-important
5 variable because of the size and numbers of people involved.
6 Sixteen million people, seven cities and it is extremely
7 unlikely that we have more or less mobility in one direction
8 or another in the three dirty versus the four clean cities.
9 If we had dealt with only one city in each group,
10 your criticism would be valid. We are dealing with multiple
11 cities in two groups. The probability that we saw some kind
12 of fortuitous in or out migration of people with varying
13 susceptibilities is very improbable.
14 DR. ALBERT: Could you tell us, if you just take
15 the cases of lung cancer that occurred in Birmingham, what
16 was the average duration of residence in Birmingham?
17 DR. DEMOPOULOS: That data does not exist but it
18 is race corrected and the white turnover rate is not as high
19 as in lower Manhattan in those seven cities.
20 MR. KELLAM: Dr. Demopoulos, with reference to
21 your statement about thresholds for carcinogens, given that
22 such thresholds may exist, do you believe there are other
23 environmental factors that can affect the level of
24 thresholds?
25 I am talking about co-carcinogens or agents that

1 may be synergistic or potentiate an effect. Do you believe
2 there is any way that environmental factors may affect the
3 threshold at which carcinogens may occur?
4 DR. DEMOPOULOS: Absolutely, and in the area
5 where you can have a measurable effect and where good
6 repetitive laboratory data exists is in the field of
7 nutrition. Nutrition is one of the most potent modulators.
8 Specifically, whether you eat polyunsaturates or not, and
9 these studies have been done with all kinds of rodents and
10 we've had elegant studies done by Karen Carroll from
11 Ontario showing quite clearly that in fat, the degree
12 of unsaturation will profoundly modulate the carcinogenesis
13 of an administered known initating substance.
14 Similarly, Lee Wattenberg has shown that you can
15 profoundly influence carcinogenesis rates by administering
16 different kinds of anti-oxidants which are nutritional
17 components. In answer to your question, there are personal
18 environmental factors such as diet, nutrition habits and
19 life style which will severely alter whether you are going
20 to develop a cancer or not and severely change the
21 eventual development of cancers from administered
22 carcinogens in experimental animals.
23 MR. BARBER: There was one question from the
24 floor which I will read as submitted. "Have you looked
25 at data in the same state with areas between high chemical

1 concentrations and surrounding areas without industry?

2 DR. DEMOPOULOS: Absolutely, this was done very

3 nicely in New Jersey.

4 MR. BARBER: There are several examples I will

5 give; Charleston, West Virginia versus the surrounding area;

6 Little Elk Valley, Maryland versus the surrounding area;

7 Baltimore versus the rest of Maryland and Houston versus

8 the rest of Texas. Have any of those four been studied?

9 DR. DEMOPOULOS: I did not study them but we

10 studied the nearly 30 counties in New Jersey and around

11 New Jersey. One of the highest concentrations of the

12 chemical industry is in New Jersey. Looking at county

13 by county incidences for lung, leukemia, urinary bladder,

14 liver and looking at male and female rates, there is no

15 correlation between the presence of industrial plants in

16 a particular county and the incidence of any type of cancer,

17 except for one instance and that is bladder cancer in

18 Southern New Jersey which was attributable to over 30 to

19 40 years ago when a chemical company was using aniline dyes

20 and that was stopped and the plants closed down and that was

21 the one bona fide incidence of occupational exposure.

22 The other anatomic sites studied in New Jersey

23 and its environs, which stretched into New York City,

24 Westchester County, Putnam County as well as Connecticut

25 and surrounding parts of Philadelphia, there was no

1 correlation between the presence of industry and development
2 of lung cancer or any other type of cancers that you would
3 attribute to inhaled carcinogens.
4 What was most telling was the negative data with
5 females. If you have an airborne carcinogen problem, then
6 the women in that county should be affected as well. That
7 was not found.
8 MR. BARBER: I would close with one last question-
9 comment. You suggested a few moments ago that you did not
10 expect ambient concentrations in the immediate vicinity of
11 plants to be any different than they would be 30 miles away
12 If that was the case, why would you expect to find differen
13 -- I'm sorry, why would one expect to find differences in
14 the community cancer rates if the concentrations were the
15 same?
16 DR. DEMOPOULOS: All of the variables, the major
17 variables, the predominant causes of cancer are smoking
18 high tar cigarettes, drinking to excess and disordered
19 nutrition. Those are the predominant variables. These
20 variables are not accounted for in epidemiology studies
21 that have been conducted and are of interest to you.
22 I think the reason cancer rates vary from one
23 community to the next are probably found within the smoking
24 drinking and dietary histories. Where these variables have
25 been examined, these answers have been thumbed. This study

1 was launched by Karen Carroll and repeated.

2 When you start to look into the nutrition and
3 dietary history of patients around the world and within
4 different communities within this country, you find striking
5 variations in cancer rates. For example, the Seventh Day
6 Adventists, there are half a million of them in America.
7 By virtue of their religion, they don't smoke, they don't
8 drink and the majority are vegetarians.

9 They may drink milk and eat milk products and
10 eggs, so they are not completely true vegetarians, but
11 they don't eat meat and their fat intake is considerably
12 below our fat intake. The average American fat intake in
13 the diet is 42 percent of calories. The Seventh Day
14 Adventists have a fat intake approximately 18 percent
15 of calories as fat.

16 Their overall cancer rate for males and females
17 as shown by Dr. Roland Phillips, an epidemiologist is one
18 half our rate. The Seventh Day Adventists have 50 percent
19 less cancer, males and females, than we do. They live in
20 areas like Lolinda, California near the eastern part of
21 L.A. County.

22 It is polluted as hell in that area and they
23 live in places like Takoma Park which is right down here
24 in Washington, suburb of Washington, D.C. These individuals
25 do not live within pristine, pastoral areas. They live

1 where the rest of us live and yet their cancer rates are
2 one half of ours.
3 To answer your question, what makes for variation
4 in cancer in animal studies and human epidemiology studies,
5 the answer is that we see differences in cigarette smoking,
6 alcohol consumption and fat intake.
7 MR. BARBER: I think you just scared me with the
8 last set of numbers. I've been worried about 1 or 2 percen
9 and earlier in your statement you rattled off percentages
10 attributable to smoking and diet that added up to
11 substantially more than 50 percent and when you corrected
12 for smoking and diet, you only reduced the cancer rate by
13 50 percent.
14 DR. DEMOPOULOS: Yes. If you know the Seventh
15 Day Adventist data, many of the Seventh Day Adventists are
16 not lifelong Seventh Day Adventists, many of them are adul
17 converts and not all of them are vegetarians. Dr. Phillips
18 is in the process of segregating out the born Seventh Day
19 Adventists who remained vegetarians lifelong versus the
20 others who come in at later times or some who will eat
21 fat.
22 Furthermore, the Seventh Day Adventists as a
23 group, there are four subparts to disordered nutrition
24 and they include excess calories, excess fat, deficiency
25 of fiber and deficiency of Vitamin A as well as obesity.

1 The question of whether obesity is controlled or
2 not and whether the other factors in disordered nutrition
3 are existing with Seventh Day Adventists, Dr. Phillips is
4 in the process of evaluating those separate factors. However,
5 fat alone is not responsible for 45 percent of the cases.
6 It's the full spectrum of disordered nutrition that accounts
7 for approximately 45 percent of the cancers.
8 MR. BARBER: Thank you very much.
9 I think we ought to take a short respite and
10 reconvene at 4 o'clock.
11 (Whereupon, a break was taken.)
12 MR. BARBER: It would be my intent to try to
13 finish this agenda today which means that the session will
14 run through on the order of 7 o'clock at the rate we are
15 proceeding.
16 Going back to the AIHC presentation, I appreciate
17 your allowing us to insert some people into the middle of
18 it. The next speaker is Mr. George Dominguez. Before he
19 starts, we have had a request to identify the people on the
20 podium.
21 We have a consultant retained by EPA to assist
22 in analyzing comments and preparing the record and that is
23 a firm by the name of Clement Associates and there are three
24 individuals who have been on the stage, Mr. E. Nisbit, Mr.
25 Steve Lester and Ms. Mary Cornwright and also Mr. Fred

1 Demick from my staff here on the stage. I think that's all
2 the faces you have seen that may not have been familiar.
3 Mr. Dominguez, thank you.
4 MR. DOMINGUEZ: Mr. Chairman, thank you. As was
5 indicated this morning by Mr. Batchelor, my name is George
6 Dominguez and I am pleased to appear here today in my
7 capacity as Chairman of the Economic Committee of the
8 American Industrial Health Council.
9 Before I will proceed with my statement, I noted
10 there was one page omitted from the statement you received,
11 namely a table I will refer to and I will give that to you
12 now.
13 I will briefly discuss the cost impact analysis
14 study which Arthur D. Little Inc. prepared for AIHC. With
15 me is Dr. Terry Rothermel, Senior Staff Member at Arthur D.
16 Little Inc. and project director for the cost impact analys
17 study.
18 In that context, I would like to ask your
19 indulgence, Mr. Chairman, you mentioned earlier asking
20 questions immediately after each speaker but since Dr.
21 Rothermel's statement and my own are so intimately related,
22 it might be more expeditious to defer questions until after
23 both of us have spoken.
24 MR. BARBER: That's fine.
25 MR. DOMINGUEZ: Dr. Rothermel in his statement

1 will outline the methodology which Arthur D. Little Inc.
2 used. We will be available following AIHC's presentation
3 for any detailed questions which you might have regarding
4 the study.
5 Since Mr. Batchelor and Dr. Morgan have already
6 addressed AIHC's overall concerns with the proposed policies
7 and generic standards, I will confine my remarks to the cost
8 and economic considerations.
9 EPA declined to make a regulatory analysis of its
10 October 10, 1979 proposed policy and generic regulations for
11 the identification and control of airborne carcinogens, even
12 though it stated in its preamble that the "proposal is
13 classified as a major regulation under EPA's final report
14 implementing Executive Order 12044. . . in that it addresses
15 a major health or ecological problem."
16 The Agency then stated that "the policy does not
17 impose regulatory requirements on any emission and therefore
18 does not meet either of the economic criteria for preparing
19 a regulatory analysis." Further, the Agency stated that it
20 would not be a "meaningful exercise" to "attempt to quantify
21 the impact of future regulation requiring unidentified
22 controls or unknown source categories of, as yet, unnamed
23 pollutants."
24 AIHC disagrees. The consequences of the Agency's
25 approach to regulatory analysis should be clearly recognized.

1 The Agency has indicated that it may prepare
2 regulatory analyses for subsequent rulemakings under the
3 policy. Such regulatory analyses will be wholly inadequate
4 because under the Agency's two-step approach, it will never
5 prepare a regulatory analysis of the economic impact of its
6 present proposed policy in comparison with the economic
7 impact of the available alternatives to that policy.
8 As a result, the Agency would preclude the
9 consideration at any time of economic impact of significant
10 regulatory alternatives to the present proposal and the
11 differing economic impacts of those alternatives.
12 We believe that the study prepared by Arthur D.
13 Little Inc. demonstrates that it is not necessary to
14 preclude a cost impact analysis of the EPA proposal and
15 its alternatives. The study shows there is a basis that
16 is available for conducting a meaningful cost impact analys
17 of the proposed policy. The proposed policy clearly meets
18 the criterion of Executive Order 12044 in that, if adopted,
19 it would have an annual effect on the economy of more than
20 $100 million.
21 AIHC urges that EPA conduct a regulatory analysis
22 in accordance with the President's Order and policy.
23 I will now describe the scope of the Arthur D.
24 Little study. AIHC commissioned Arthur D. Little Inc. to
25 undertake a cost impact analysis of the proposed regulatior

1 and further to develop a methodology that could be employed
2 for cost impact analyses in this and related areas.
3 In order to put the Arthur D. Little Inc. study
4 into perspective it must be recognized that this was
5 undertaken and completed under severe time constraints
6 in the time between the proposal and the comment deadline.
7 Several practical limitations on the scope of Arthur D.
8 Little's project were understood and accepted at the outset.
9 First, the study addresses compliance costs and
10 is not a full economic impact analysis.
11 Second, it addresses compliance costs only for
12 existing sources.
13 Third, the study includes only the direct cost
14 of installing and operating BAT technology and of compliance
15 with the proposed generic fugitive standard.
16 Fourth, the study relies on existing data which
17 provides a basis for estimating the cost of the proposed
18 generic fugitive emission standards and BAT as applied to
19 existing plants.
20 The study is limited to three illustrative
21 substances: benzene, perchloroethylene and vinyl chloride.
22 The three substances were chosen because substantial
23 technical and economic data on those three are available
24 and they are highly probably candidates for regulation under
25 the proposal.

1 There three substances appear on the Cancer
2 Advisory Group's priority list for regulation under Section
3 112. Indeed, vinyl chloride has already been regulated
4 under Section 112. EPA has proposed to list benzene and
5 we understand will shortly propose regulations for one sour
6 categorty of benzene emissions, maleic anhydride.
7 The methodology used. Using benzene,
8 perchloroethylene and vinyl chloride as examples, Arthur D
9 Little Inc. developed a methodology for assessing the cost
10 impacts of the proposed policy. This methodology, now
11 developed, can be utilized in subsequent analyses of other
12 compounds.
13 Dr. Rothermel will discuss the methodology in
14 more detail. I will just touch on the major elements.
15 One part of the methodology was the identification of major
16 emission control technologies likely to be BAT options for
17 substances to be covered by regulations issued under the
18 proposed policy.
19 Cost models were then developed for potential
20 BATs including thermal incineration, catalytic incineration
21 activated carbon adsorption and techniques for controlling
22 emissions from storage tanks and handling operations.
23 Similar cost factors were developed for compliance approach
24 to the draft generic standard on fugitive emissions. The
25 determination of control approaches and associated cost

1 models were based on previous regulatory analyses, the
2 experience of AIHC companies and the judgment of Arthur D.
3 Little Inc. engineers.
4 The results of the study. The Arthur D. Little
5 Inc. study results are summarized in Table 1-2 of the
6 Arthur D. Little Inc. report. We have provided the panel
7 with copies of that table.
8 It is clear from the study that the proposed
9 policy, just as applied to only three substances, will have
10 a major cost impact on specific industry sectors and that
11 the impact will exceed $100 million. Thus, the criteria
12 in Executive Order 12044 for the preparation of a regulatory
13 analysis, including an economic analysis, are met by the
14 Agency proposal.
15 It is important to note that the costs presented
16 are incremental costs and do not include the costs of
17 existing emission controls. The low estimated incremental
18 cost for vinyl chloride reflects that fact since EPA already
19 has promulgated a national emission standard for vinyl
20 chloride that requires emission controls based on BAT and
21 includes leak detection and elimination requirements.
22 The actual cost for the vinyl chloride industry
23 for installing BAT and leak detection and elimination would
24 be the total of past expenditures by the industry plus the
25 incremental cost included in the Arthur D. Little Inc.

1 study.

2 The Society of the Plastics Industries will
3 present to the Agency a study which establishes capital
4 expenditures over the past six years by the vinyl chloride
5 industry in the amount of approximately $200 million. In
6 1979, the industry annual operating and maintenance costs,
7 excluding fixed charges, ran approximately $30 million.
8 In AIHC's view, the SPI study confirms the findings of
9 Arthur D. Little Inc. that the proposed regulations will
10 have an impact well in excess of the criteria in Executive
11 Order 12044.

12 Just as important, however, is the demonstration
13 in the Arthur D. Little Inc. study that a feasible
14 methodology for cost analyses for this proposed rule can
15 be developed. Thus, contrary to the Agency's conclusion,
16 a regulatory analysis of the proposed rule is feasible and
17 is required by Executive Order 12044.

18 Accuracy of cost impact analysis. It is
19 important to understand the cost impact estimate and
20 methodology developed by Arthur D. Little Inc. in the
21 proper context. It was not an attempt at a definitive
22 determination of cost, but rather an effort to provide
23 reasonable indicators of the potential cost impacts of
24 the proposal and to demonstrate the feasibility of making
25 such determinations. Therefore, the absolute accuracy of

1 the estimates is not and should not be the critical issue.

2 If anything, however, the cost estimates are

3 probably understated for several reasons. Indeed, the

4 cost estimates are only a partial estimate.

5 As I indicated, the cost estimates are limited

6 to the cost of compliance by existing plants with BAT and

7 the proposed generic leak detection and prevention program.

8 No attempt could be made to analyze technology beyond BAT

9 even though the proposed policy contemplates such

10 requirements in the event of unreasonable residual risk

11 after BAT.

12 The study did not evaluate the cost that would be

13 incurred by new plants to meet BAT or to comply with the

14 regulatory mandates for new and modified sources regarding

15 emission offsets, plant siting or substitution of products.

16 In addition, no consideration was given to

17 process down time associated with implementation of

18 regulations under the proposal. Productivity or capacity

19 losses which might be associated with such controls were

20 not determined.

21 In short, we firmly believe that the study results,

22 while probably understated, represent the most reasonable

23 cost analysis possible under the circumstances.

24 The Arthur D. Little Inc. study demonstrates

25 that a meaningful cost impact analysis can be done. It

1 demonstrates a cost impact of the proposal in excess of
2 $100 million, thereby triggering the application of
3 Executive Order 12044.
4 AIHC believes that it is now incumbent upon the
5 Agency to prepare a regulatory analysis which assesses not
6 only the cost impact but the entire range of economic
7 effects of the proposal, including inflationary impact,
8 capital availability, productivity, employment and plant
9 shutdowns, product shortages and international trade.
10 Those effects must be compared to the economic effects
11 of the alternatives to the proposed policy.
12 The primary stated purpose of the EPA proposal
13 is to speed up the rate of rulemaking on individual
14 substances. In AIHC's view, the expedition, if any,
15 will result from the provisions of the policy which
16 preclude EPA from basing its decision on the best science
17 and scientific information available with respect to the
18 risk posed by exposure to an airborne substance.
19 Once the proposed policy is promulgated, there
20 will be no opportunity to question the Agency's definition
21 of a carcinogen, the exposure levels at which it may pose
22 a hazard or in large part, the level of the technology
23 required to reduce the risk.
24 AIHC believes the adverse economic effect of the
25 proposed policy will be far greater than the regulation,

1 in accordance with the procedure proposed by AIHC, of
2 substances based on sound scientific risk estimates and
3 requirements tailored to meet those risks.
4 As pointed out by Mr. Batchelor, we do not
5 believe that adoption of the AIHC approach would cause
6 delays in the regulatory process. And, most importantly,
7 development and use of scientific risk estimates will permit
8 the Agency to focus on compounds which pose a demonstrated
9 hazard and to relate the cost of regulation to the benefits
10 of regulation.
11 It is precisely these types of concerns which
12 the regulatory analysis requirements of Executive Order
13 12044 are designed to address. The Agency should proceed
14 to develop a full regulatory analysis of its proposed policy
15 and the alternatives to that policy.
16 I would now ask Dr. Rothermel to talk in greater
17 depth with respect to the methodology employed.
18 DR. ROTHERMEL: Thank you, Mr. Dominguez. Mr.
19 Chairman, my name is Terry Rothermel, the project director
20 responsible for Arthur D. Little Inc.'s report to the
21 American Industrial Health Council entitled, "A Methodology
22 for Cost Impact Analysis of NESHAP Regulations and Its
23 Application to Three Illustrative Substances: Benzene,
24 Perchloroethylene and Vinyl Chloride.
25 In regard to a comment by Mr. Batchelor this

1 morning, I come to discuss our methodology, not to dismiss
2 it. For 15 years, I have specialized in environmental
3 studies at our firm, including ones which have analyzed
4 the economic impact of environmental regulations, ones
5 which have developed the technical bases for selecting
6 appropriate control technologies and ones which have
7 assessed business opportunities resulting from increased
8 pollution control activity. Our clients have included
9 EPA, OSHA and private industry.
10 During our short assignment for the American
11 Industrial Health Council, we have developed a methodology
12 for anticipating the cost impact of proposed standards for
13 hazardous air pollutants such as those covering Best
14 Available Technolocy (BAT) and generic controls.
15 The cost impact methodology was built from the
16 experience of Arthur D. Little Inc., EPA and others. The
17 analytical challenge in this case was that of applying
18 these techniques to a situation in which the area of
19 impact has been broadly outlined but not specifically
20 defined.
21 As Mr. Dominguez has indicated, the three
22 substances chosen to illustrate our methodology were
23 benzene, perchloroethylene and vinyl chloride. We next
24 identified general technologies and approaches which are
25 technically most feasible for controlling hazardous air

1 emissions. We then developed control cost models or
2 relationships for families of control technology such
3 as incineration and adsorption.
4 The impact of an environmental regulation has
5 traditionally been measured in terms of its incremental
6 effects on industry practice beyond those required by
7 previous regulations. Thus, we next identified the
8 current (net) emission rates of the major sources emitting
9 the illustrative substances.
10 Arthur D. Little Inc. engineers then judged how
11 available technologies might be required to control or
12 further reduce these emissions. The control cost models
13 were then used to estimate point-source costs of compliance
14 with proposed NESHAP regulations for each of three
15 substances.
16 Our calculation of impact may be summarized as
17 follows. The number of sources in an impacted industry
18 category for a given substance was determined from the
19 industry characterizations found in previous studies.
20 The number of sources was then multiplied by the average
21 number of omission points for each source and by the cost
22 of control associated with each type of emission point (as
23 determined from the control cost models).
24 Costs for individual industry categories were
25 then summed for all such categories impacted for a given

1 substance to determine the total cost impact for a
2 regulation.
3 Costs were calculated in terms of both capital
4 investment and annual operating costs (including recovery
5 of those capital expenditures). As this methodology has
6 been applied to the three illustrative substances in
7 anticipating the partial impact of the proposed BAT and
8 generic standards, it will also be appropriate for analyzin
9 impacts as specific substances are actually proposed for
10 regulation.
11 In the case of benzene, we estimated, as shown in
12 Table 1-2 of our report, that the annual cost of compliance
13 with the proposed generic standards would be about $3
14 million annually. Application of Best Available
15 Technologies to benzene emissions would result in
16 expenditures of $1.1 billion annually, including those
17 costs associated with controlling emissions from gasoline
18 distribution.
19 If EPA were to choose to exclude gasoline
20 distribution from its regulation of benzene emissions,
21 the total annual cost of compliance, with both BAT and
22 generic standards, would be approximately $70 million.
23 The annual costs associated with controlling
24 the major sources of perchloroethylene emissions would
25 be approximately $55 million and $50 million to meet the

1 generic and BAT standards, respectively. In considering
2 its regulatory options, EPA might choose to exclude coin
3 operated dry cleaners from this regulation. In that case,
4 a working estimate of the combined cost impact would be
5 $60 million -- as compared to just over a $100 million
6 under the proposed regulations.
7 Given our focus upon incremental impact, the
8 costs of compliance associated with vinyl chloride would
9 appear to be minimal, i.e., less than $1 million annually.
10 While these anticipated incremental costs of the proposed
11 NESHAP regulations, over previous regulations, are less for
12 vinyl chloride, the cumulative compliance costs incurred by
13 the vinyl chloride industry in response to previous
14 regulations provides yet another indicator of what it
15 may cost to control a future unregulated hazardous substance.
16 Estimating the overall cost of the proposed
17 NESHAP regulations requires that similar cost impact
18 analyses be made for all substances which are now believed
19 likely to be covered by the regulation. A priori it will
20 not be possible to distinguish those substances which will
21 constitute major regulatory actions under Executive Order
22 12044 from those substances which will entail only minimal
23 impacts until the actual cost analysis is performed.
24 The cost impact methodology and analysis which
25 we have conducted illustrates only the first step of a more

1 extensive economic impact analysis procedure which has
2 become integral to EPA's regulatory process and which may
3 feasibly accompany these proposed NESHAP regulations.
4 A cost impact analysis provides estimates of the
5 costs of compliance for categories of impacted industry
6 sources. An economic impact analysis further addresses
7 the probable effects of those compliance costs upon prices
8 and inflation, profits and capital availability,
9 productivity and production capacities, plant shutdowns
10 and unemployment, product shortages and substitution and
11 international trade and the balance of payments.
12 As Mr. Dominguez has noted, the scope of our
13 analysis was necessarily limited in other ways. The
14 analysis was confined to the anticipated costs of BAT
15 and generic control upon existing sources of the three
16 illustrative substances.
17 The cost effects upon new or modified sources
18 in meeting BAT or a more stringent, presumptive national
19 emission standard were not assessed. We also used
20 "typical plant" situations as the basis for estimating
21 industry-wide cost parameters -- a common practice in
22 such regulatory analyses.
23 Average costs related to a typical facility do
24 not reflect the varying costs that may be dictated by the
25 age of the plant, type of construction and other site

1 specific factors. No consideration was given for process
2 down time or other production losses associated with
3 control actions.
4 Nor were the capital costs of backup pollution
5 control systems required to maintain process operations
6 included in our efforts. Finally, the impacted user-
7 industry categories considered in our short assignment
8 did not necessarily include all of those industry
9 categories which might be affected by NESHAP standards
10 covering benzene, perchloroethylene and vinyl chloride.
11 I believe Mr. Dominguez would like to make a
12 closing comment before you ask questions of us both.
13 MR. DOMINGUEZ: Yes, indeed, I did want to make
14 a closing comment. I think one of the major points at
15 issue is the distinction between attempting these
16 determinations before or after the fact, if you will.
17 I appreciate that it is sometimes difficult to relate
18 these seemingly abstruse and highly theoretical economic
19 discussions into concrete and realistic terms and I would
20 like to give a brief illustration that may make the concepts
21 a little bit more understandable and perhaps more cogent.
22 I would do that on a very personalized basis.
23 Let us assume that any one of us wanted to construct a
24 house from scratch, as it were. So we went out and hired
25 a real estate agent to survey some property for us and give

1 us some general assessments as to our needs and the lay of
2 the land, as it were.
3 Then we hired an architect and the purpose of the
4 architect was to design the overall structure and ultimately
5 a contractor and the purpose of the contractor was to come
6 down to the hard specifics of just what would be undertaken
7 in construction of the house.
8 So we have all three of these people working for
9 us but we make a very clear stipulation at the onset that
10 in the determinations that they undertake for us, we do not
11 want any cost estimates or assessments until after we have
12 signed the contract and make commitments with them to proce
13 I submit that no one would do this in the conduct
14 of their personal finances. I think this simple analysis
15 is rather clear, the real estate agent and the architect
16 are really equivalent to the determination of the overall
17 policy proposed and the contractor's work relates very much
18 to the secondary aspects of the specific standards that are
19 being and will be developed.
20 Needless to say, a request not to conduct any
21 cost analysis or give us any cost estimates parallels what
22 I believe the Agency has done. Again, I would submit that
23 we would not do this in the management of our own funds and
24 further that is neither a responsible execution of the pub
25 trust nor sound public policy not to do so in considering

1 expenditures of public funds nor programs that necessitate
2 the expenditure of private ones.
3 Hopefully that rather simplistic way of looking
4 at things might put the problem into some greater context.
5 With that, Dr. Rothermel and I will try to answer your
6 questions.
7 MR. BARBER: I would just make a short
8 observation that despite two years of trying, we have
9 still failed to communicate on the purpose and likely
10 impact of this specific action as opposed to the subsequent
11 regulatory actions. But we would endeavor to keep trying.
12 I am concerned that you convey that there is
13 utility to an estimate that casually, even in its own
14 recognition, is going to be off by a factor of 20 if we
15 don't regulate coin operated dry cleaners and gas stations
16 under this thing.
17 I am not sure what it tells us to go through an
18 exercise that says -- in this case, we have listed benzene
19 and we may in fact be persuaded after the hearing that it
20 should remain listed and we may in fact promulgate some
21 rules for benzene down the road and we may in fact regulate
22 more than one category of industry. We may or may not
23 regulate the gasoline distribution sector.
24 All of that says that the impact of the benzene
25 exercise is somewhere between zero and some big number.

1 The magnitude of that impact being totally
2 unrelated, in my judgment, to the Federal Register proposal
3 that we issued, but that seems to be where we have a
4 problem in communication than in our response to the
5 comments of further rulemaking in having made ourselves
6 better understood.
7 Are there questions from the panel?
8 MR. JOSEPH: I have one question for either,
9 whichever you prefer. I believe you were here this morning
10 and you heard the testimony of representatives of NRDC and
11 EDF. Both suggested that there was at least a reasonable
12 strong legal argument that the requirement of Section 112
13 is that you reach zero of these substances.
14 I note that you have constructed is a cost
15 methodology. Have you evaluated this proposal, agreeing
16 with Walt that this proposal in and of itself is not the
17 proposal that imposes any of the costs, but have you
18 evaluated for any one of these specific substances the
19 economic impact in terms of following these rules for
20 setting standards as opposed to following the rules
21 suggested by EDF or NRDC? In other words, have you
22 evaluated the savings impact as opposed to the cost
23 impact?
24 MR. DOMINGUEZ: I'm not certain the proposals
25 they made would lead one to conclude that there was a

1 savings effected. Would you please explain what you
2 understood so that I can better answer the question or
3 perhaps Dr. Rothermel --
4 MR. JOSEPH: I did not state it clearly for you.
5 Under the EDF proposal in, let's say the six years there
6 would be no emissions of vinyl chloride. It is not apparent
7 -- taking your analysis of the projection for six years from
8 now under the EPA proposal was correct, that there would
9 still be emissions of vinyl chloride, I am making the
10 assumption that the EDF proposal would cost a lot more
11 and therefore the EPA proposal is a savings over that.
12 Have you looked at that?
13 MR. DOMINGUEZ: No, we have not looked at that,
14 that's a simple answer to your question, we have not tried
15 to make a comparative analysis of what those relative costs
16 would be.
17 MR. JOSEPH: Do you think they might be a lot
18 more?
19 MR. DOMINGUEZ: Without analyzing --
20 MR. KELLAM: One related question, as I
21 understand it, the base line that you used for your
22 methodology is essentially what current controls are.
23 On the other hand -- if you want to clarify that?
24 DR. ROTHERMEL: Go ahead.
25 MR. KELLAM: By assuming that as the base line

1 you ignore the fact that even in the absence of the policy
2 we have Section 112 which requires us to take some action
3 for hazardous air pollution. My question is that it seems
4 to me it would be very difficult to develop a regulatory
5 analysis which essentially assumes that in the absence of
6 the proposal there would be no regulation of carcinogens
7 and to assess the cost on that basis.
8 DR. ROTHERMEL: You are saying that the base line
9 would be no regulation of carcinogens or are you saying --
10 MR. KELLAM: I am saying is that one of the
11 assumptions built into the methodology?
12 DR. ROTHERMEL: No, the base line is as you first
13 stated, the current regulation of carcinogens.
14 I think it is a difficult exercise and my
15 engineer associates will attest to that. It requires
16 some judgment of the population in the time frame of what
17 the average practice seemed to be in the industry at the
18 present time and there is some practice going on now,
19 versus what would be required under a reasonable
20 extrapolation of this proposed regulation.
21 MR. KELLAM: If someone asked you to develop a
22 cost methodology for Section 112, not for an airborne
23 carcinogen policy but simply for what the Clean Air Act
24 requires in Section 112, how would you go about doing that
25 DR. ROTHERMEL: You said it would not be for wha

1 MR. KELLAM: Let's assume the proposal does not
2 exist and we simply have Section 112 of the regulation of
3 hazardous air pollutants. You are asked to prepare a cost
4 methodology. What is Section 112 going to cost if it is
5 implemented the way the Clean Air Act provides?
6 DR. ROTHERMEL: I think the methodology we
7 developed would be very appropriate for that kind of
8 understanding.
9 MR. KELLAM: Specifically, what kind of
10 assumption would you make about the eventual appropriate
11 level of control? My question is really, would these
12 assumptions be significantly different, do you believe,
13 from what the proposal describes?
14 DR. ROTHERMEL: It would not be terribly different
15 from the analysis made for these three substances in the
16 sense that you have already covered a great deal of
17 regulations, anticipate a great deal of regulation with
18 BAT engineering.
19 We have applied that to existing sources but we
20 have anticipated what 112 would result in for those three
21 substances along those lines. Say that the cost impact
22 methodology or economic impact methodology as it developed
23 in other regulations would not necessarily be transferred
24 with no change to application in the hazardous air pollutant
25 area.

1 I think in the hazardous air pollutant area, we
2 have an important amount of controls that have been put int
3 place for other regulatory purposes. That is a very
4 important part of our development of this methodology,
5 the incremental focus.
6 MR. BARBER: That incremental focus, I take it,
7 sought to exclude the cost of applying OSHA's rules for
8 benzene?
9 DR. ROTHERMEL: Yes, it did.
10 MR. BARBER: I guess the question Bob is asking
11 is how would you cost the alternative of no policy?
12 DR. ROTHERMEL: The cost of not having this
13 policy?
14 MR. BARBER: Yes.
15 DR. ROTHERMEL: Later on, you will have other
16 specific pollutants that are named, correct?
17 MR. BARBER: The presumption is --
18 DR. ROTHERMEL: Without this policy, when you
19 got to that, the one-step policy is proposed as a two-step
20 policy and the cost impact would be similar to benzene or
21 that approach with what we see here, having new sources an
22 other things added beyond this proposal.
23 MR. KELLAM: If I understand you, in a sense you
24 could argue that the impact would be no greater in the
25 absence of this policy than it would be if we pursued

1 what Section 112 requires us to do?

2 DR. ROTHERMEL: That may or may not be. Our

3 purpose was to show that methodology is possible at this

4 point in time.

5 MR. KELLAM: What you are costing out is the

6 impact of this proposal, not the impact of Section 112?

7 DR. ROTHERMEL: That's true.

8 MR. KELLAM: And yet the two may be the same?

9 DR. ROTHERMEL: I wouldn't say they are going to

10 be the same.

11 MR. DOMINGUEZ: I think it is clear that what we

12 have analyzed is predicated upon what you proposed and what

13 you have proposed gives us a set of parameters. For example,

14 I don't believe that Section 112 per se eliminates the BAT

15 as mandated, does it?

16 MR. KELLAM: No, it does not, but as Todd pointed

17 out, in all probability a different interpretation of

18 Section 112 from the one that we have proposed could lead

19 to much more stringent regulations.

20 MR. DOMINGUEZ: More stringent or less stringent

21 since it's in the discretion of the Agency to exercise it's

22 administrative latitude in applying BAT or not applying BAT.

23 You have elected in your proposal to proceed on the basis of

24 applying BAT and therefore our analysis proceeded on the

25 assumption of applying BAT technology.

dp

1 Obviously if you had made a proposal that had a
2 different option of exploring the other alternatives, then
3 we would have proceeded with assumptions predicated on
4 those alternatives.
5 The second comment I would make is that we seem
6 to be discussing this as if it were a solitary analysis,
7 an either/or situation, either analyze on the basis of BAT
8 or in the absence of anything, I'm not sure that's we're
9 advocating.
10 We have developed a methodology that shows it
11 is feasible to do comparative cost analysis which then
12 gives the agency the ability to measure in economic terms,
13 gross as they may be, the comparative results in economic
14 terms of its actions.
15 Therefore, the analysis does not proceed on one
16 assumption. If you really translate it into practice, wha
17 you would do is construct an array of data across a series
18 of options and then find out what are the economic
19 consequences of exercising those different options
20 which would also give you the ability to determine
21 which is the most cost effective option to exercise,
22 either the entirety of the process or against a discrete
23 compound.
24 MR. BARBER: Have you tried to apply this
25 methodology to the AIHC proposal?

1 MR. DOMINGUEZ: No, we have no done that and
2 again we were operating, as we indicated to you before,
3 under very severe time restraints. We had problems going
4 as far as we did within the short time available so the
5 answer to your question is no, we have not.
6 MR. BARBER: Given that the Clean Air Act requires
7 best available control technology on all new sources, given
8 the degree of control that we are expecting in our non-
9 attainment areas for ozone where we have hydrocarbon
10 precursors, would you expect to see a substantial difference?
11 The big concern expressed by environmentalists is
12 that BAT is no more than what is now being provided by
13 Section 110 and Section 111, unless there is a finding
14 of unreasonable risk. I am not sure where the delta is
15 absent the unreasonable risk. We will have to spend some
16 more time with the draft analysis and we will look at it.
17 Thank you.
18 May we have Mr. Harry Lieber, please?
19 DR. LIEBER: I will try to be brief and not read
20 from the entire statement so that we may conclude before
21 the hearing begins tomorrow morning.
22 I am Harvey Lieber and I represent the American
23 University Institute for Risk Analysis (AURA). We have
24 been asked by the American Industrial Health Council,
25 AIHC, to comment on EPA's proposed national emission

1 standards.

2 However, all of my remarks represent the view of
3 AURA and not necessarily those of AIHC.

4 The proposed EPA policy for airborne carcinogens
5 attempts to bridge the gap between the limits of scientific
6 knowledge and the mandates for protection of public health
7 by Congress and the public. Such an effort is pioneering
8 and cannot be expected to be perfect in all respect at the
9 outset.

10 Despite the timeliness and virtues of such an
11 approach, there are certain problems and deficiencies in
12 the rule and we would like to suggest several improvements.
13 We believe that the proposal is too simplistic, does not go
14 far enough in categorizing chemicals for regulatory action
15 nor does it solve the problem as put forward.

16 We will comment on two particular areas, the
17 need to visibly separate scientific and technological fact
18 from value judgments and two, a generic rulemaking as
19 envisioned here is a managerial or administrative decision
20 as well as a social decision on health protection and
21 therefore requires evaluation of alternative strategies.

22 One must separate scientific and technological
23 facts from value judgments. In the absence of scientific
24 information, value judgments must often be substituted.
25 Few would dispute the limitations imposed by measurement

1 capabilities and the ability to control all parameters in
2 experiments. However, in addition, it may not be possible
3 to obtain certain information under any conditions.
4 An example of this limitation on knowledge can
5 be demonstrated for animal tests for determining
6 carcinogenicity. It's obvious that for a low potency
7 substance, the dose administered must be comparatively
8 high.
9 At such levels, toxicities other than cancer may
10 dominate such that the animals die of other causes before
11 cancer can occur or that the metabolism is altered such
12 that the experiment is no longer valid.
13 Another formula we introduce indicates that in
14 the real world of exposure, there are many more sources of
15 error and they very easily propagate and it is difficult
16 to establish any risk estimate on a totally scientific
17 basis.
18 The formula we gave here, assuming all the error
19 sources have been identified, the propagation of error can
20 still be enormous. Assuming only a safety factor of two
21 for each error term, three pathways and four types of
22 cancer, the overall error is nearly a factor of 100.
23 This becomes a major problem in the administration
24 of public health protection for carcinogens since
25 overestimates by two or more orders of magnitude can

1 cause action and commitment of resources out of all
2 proportion to the actual risk.
3 There is little question conservatism toward
4 public health is warranted, but the question is where such
5 conservatism should be applied. Should it be inserted at
6 each step, as promulgated by Interagency Regulatory Liaison
7 Group in their report or should the best analysis of error
8 be made overall, i.e., a central estimate and ranges of
9 error on either side; and then an overall conservatism
10 factor adopted?
11 In this last case, the amount of conservatism
12 entered is visible, its impact estimable and its validity
13 can be examined as an open social judgment as opposed to
14 an esoteric judgment made without resource to, but in the
15 name of, science.
16 Specifically, in the case of the air cancer
17 policy, the initial three categories, high, moderate and
18 low, are in effect indicators of certainty of evidence of
19 carcinogenicity, not probability of carcinogenicity.
20 However, in order to have a chemical listed under Section
21 112, two other crucial factors should also be considered.
22 One, potency, perhaps through a strong, medium
23 and weak classification and two, exposure, also in high,
24 medium and low categorization.
25 The proposed rule fails to consider relative

1 potency and is unclear when it discusses a high probability
2 category of exposure risk. In other words, simply listing
3 a chemical in terms of high certainty of evidence of
4 carcinogenicity, even if its potency and exposure resulting
5 in risk may be low would, according to the proposed rule,
6 immediately trigger the requirement of at least best
7 available technology for both new and existing sources.
8 Not only can the economic impact of such a single
9 factor categorization be very great but EPA might also be
10 put into the position of symbolic regulation -- being unable
11 to specify BAT for a large number of substances or unable
12 to insure and enforce timely compliance because of its
13 limited resources.
14 Therefore, greater flexibility should be built
15 into the initial categorization proposal and EPA should not
16 force all high evidence probability airborne carcinogens,
17 especially those of low exposure and mild potency, into
18 uniform BAT requirements.
19 If EPA uses multiple criteria for the initial
20 categorization of weight of evidence, one could then
21 support the EPA approach to the three broad categories,
22 high medium and low, for classifying carcinogens for
23 further regulatory action if the different categories
24 truly provide realistic alternatives for listing.
25 The four subsequent criteria as indicated by EPA

1 for regulating priorities could then provide a more
2 reasonable basis for control strategies since not everythir
3 would be forced into the high category as a result of too
4 much conservatism and listed inappropriately.
5 The generic rulemaking as proposed by EPA for
6 carcinogens in air is made for regulatory convenience as
7 well as to provide a manageable regulatory framework. As
8 such, it is not totally a social decision but an
9 administrative one as well.
10 The rule proposes to provide a means to translate
11 what Congress intended when it required that health based
12 standards be set under Section 112 of the Clean Air Act
13 with "an ample margin of safety" into regulatory practice.
14 This is indeed difficult for a pollutant without a
15 demonstrated threshold since there is no absolute measure
16 of safety or an absolutely quantified "ample margin of
17 safety".
18 Indeed, the social-political judgment by the
19 Administration that Section 112 should not be interpreted
20 to protect public health absolutely by requiring zero
21 emissions, has immense implications for regulatory
22 discretion and allows consideration of a variety of
23 other non-health factors.
24 Thus, the adoption of a particular management
25 strategy, especially one not requiring zero risk, requires

1 that alternative strategies of this type be evaluated as
2 to their relative impact on health, cost and other societal
3 benefits, including the impact on regulatory agencies in
4 terms of their resources and regulatory profile.
5 A total generic risk-cost-benefit analysis or a
6 completely quantitative risk assessment would be difficult,
7 if not impossible, to undertake. While cost-benefit
8 analysis may be required by other regulations, in
9 establishing the initial generic rule such a detailed
10 analysis would probably contribute little to clarifying
11 the general decision process and might even force serious
12 delays in the implementation of the generic rule.
13 In addition, this implies that any generic
14 approach should not foreclose more exacting risk
15 assessments which could be attempted later when
16 establishing regulatory priorities and control strategies
17 for specific substances.
18 What is sought here is much more modest and
19 hopefully, meaningful. A set of representative alternative
20 strategies would be selected to cover realistic
21 possibilities. The strategies would not have to be
22 detailed or overly explicit but generally representative
23 of the options.
24 The impact on health and safety, the costs and
25 benefits to society and industry would then be estimated

1 on a relatively imprecise basis for each of these factors,
2 not on an absolute basis, but for each strategy relative to
3 the others.
4 Then comparisons can be made for each factor
5 among the alternatives, without aggregating to single
6 numbers for each alternative, so that the impacts can be
7 made visible. The objective is not to select the best
8 method or a single number, but to select a reasonable
9 one which avoids extreme impacts on any factor.
10 In looking at management alternatives for the
11 new generic method, it must again be emphasized that this
12 approach is being selected mainly for reasons of
13 administrative efficiency. This is not a decision based
14 on science but it must use good science as a major input
15 into this generic rulemaking process.
16 It therefore would be advisable for EPA to
17 clarify the impact of all the alternatives through
18 illustration by using at least three ideal types ("pure
19 examples") -- high, moderate and low airborne carcinogens.
20 At least one alternative would be set not only on the basi
21 of probability of evidence, but also considering potency
22 and exposure.
23 In addition, the alternative strategy should
24 cover alternate control approaches as well as qualitative
25 considerations entering into the determination of

1 unreasonable residual risk for further regulatory action
2 beyond BAT.
3 These should be illustrated and the relative
4 importance of factors other than public health shown. This
5 proposed elaboration of the regulatory process does not
6 discourage EPA from attempting to build greater consistency,
7 predictability and speed into the implementation of the
8 Clean Air Act through generic approaches.
9 However, such illustrations would also serve to
10 better indicate the managerial flexibility which the
11 proposed approach and alternatives might achieve as well
12 as their possible impact on substances which would be listed
13 and regulated.
14 The objective is to make alternatives and the
15 relative impact of selection visible and traceable, not
16 find a "perfect" strategy.
17 In summary, the promulgation of a generic rule
18 requires responsible analysis of alternative approaches
19 to the rule. Such analyses must not only separate
20 technological facts from the array of value judgments
21 involved, but must also recognize the limitations of the
22 analysis supporting a generic rule. The rule should not
23 foreclose the intelligent use of additional information
24 on risks or control strategies as such information becomes
25 available.

1 MR. BARBER: Thank you. Do you have any question
2 Roy?
3 DR. ALBERT: No.
4 DR. ANDERSON: Dr. Lieber, I had a question
5 regarding your interpretation of EPA's policy to use weight
6 of evidence. You say that conservatism is built-in at
7 every level and I did not know exactly what you meant
8 by that.
9 Do you use the weight of evidence in a qualitativ
10 sense to judge the quality of the information and continue
11 all data in the aggregate to make some determination and I
12 do not know what you meant by building conservatism in.
13 DR. LIEBER: Not being an epidemiologist, it
14 would be difficult for me to give you specific examples
15 right now. However, we have prepared a draft of an analys:
16 of the IRLG process and we have indicated where conservati\
17 factors are sort of built into the numbers without being
18 very clearly indicated.
19 DR. ANDERSON: I am talking about the qualitative
20 determination and not the quantitative.
21 DR. LIEBER: My remarks apply basically to the
22 quantitative approach in terms of conservatism being built
23 in.
24 DR. ANDERSON: The weight of evidence is not par
25 of the quantitative determination. I wondered if you had

1 looked over one of our risk assessments where the weight of
2 evidence has been used to determine carcinogenicity?
3 DR. LIEBER: No, I have personally not, but the
4 other area where you may possibly have built-in conservatism
5 too much is not considering potency and not considering
6 exposure and is simply basing it on high evidence of
7 probability of carcinogenicity tends to push it that way.
8 DR. ANDERSON: There are also other areas where I
9 felt that you had misunderstood the policy. I think
10 exposure is certainly a factor which is clearly considered
11 in the policy as well as some potency in setting priorities.
12 DR. LIEBER: As I indicated in the statement,
13 exactly how you consider exposure is very unclear. You
14 have a statement here that it should have "significant
15 public exposure or one with a high probability of
16 significant risk to the potentially most exposed groups".
17 That was a little vague and unclear and I'm not
18 sure it was clearly explained in the regulation as to how
19 you were going to factor in exposure in the listing. I
20 failed to notice any strong statement at all with regard
21 to potency in your initial listing of substances on the
22 high side as to how potency would come into that.
23 It appeared to me one way you would consider
24 basically evidence of carcinogenicity and that's about it.
25 MR. BARBER: Your suggestions are aimed at the

1 listing more than the --

2 DR. LIEBER: The suggestion to include potency

3 and exposure apply to the listing decision because the

4 listing decision is not a listing for testing or scientific

5 study. That is a decision to apply BAT.

6 DR. ALBERT: If you require that considerations

7 of potency and exposure be taken before the listing decisio

8 what you are asking for is pretty much a full risk assessme

9 before the decision is made?

10 DR. LIEBER: At least a categorization into high,

11 medium, and low. I am not sure how far one has to go. I

12 would simply agree, to quote from the OSHA record, Mr.

13 Jellinek has said that if a substance is believed to be

14 a carcinogen but it is a weak carcinogen, potency, to which

15 few persons are exposed, the best regulatory course might b

16 to defer any regulation in favor of concentrating limited

17 resources on higher toxicity, higher exposure substances.

18 That was said in regard to OSHA but it might

19 apply here, too. You should consider factors beyond simply

20 evidence of toxicity, potency and exposure.

21 DR. ALBERT: The combination of potency and

22 exposure equals risk, in effect. That's what it boils

23 down to. So you are advocating, not necessarily the

24 formulation of specific risk estimates, but a categorizati

25 of agents on the basis of the combination of potency and

1 exposure, is that it?

2 DR. LIEBER: Potency is evidence of the probability

3 of carcinogenicity, too, obviously.

4 DR. ALBERT: That, combined with exposure,

5 represents the public health impact in terms of production

6 of cancer?

7 DR. LIEBER: That's right.

8 DR. ALBERT: You are advocating categorization of

9 agents on this basis. How many categories are you talking

10 about?

11 DR. LIEBER: The original proposal as we have

12 here would go beyond simply evidence of carcinogenicity

13 which apparently is the only category you had initially,

14 high, medium, and low and it would include exposure and

15 I'm not quite sure how you have put it, but we would like

16 that to go into perhaps three categories and also some

17 category for potency.

18 To use Mr. Doniger's example, there is a world

19 of difference between aflatoxin and saccharin and that

20 should be considered in terms of even listing.

21 MR. BARBER: Any other questions?

22 DR. ANDERSON: A question on Page 3. You have a

23 formula that relates risk to dose and you refer to a paper

24 by Wilson. You don't give support for that formula and I

25 wonder if it is a formula from Wilson's work or your own

1 research and if you recommend that EPA should use this
2 formula to analyze data to estimate risk?
3 DR. LIEBER: This is a formula clearly from
4 Wilson's work and it is simply suggestive of the fact that
5 the lower the potency, the higher the dose and the higher
6 the dose, the more distortion you may have and the more
7 difficult it may be to determine it.
8 I am not capable from my background suggesting
9 whether or not EPA should use it, but it does indicate how
10 very often you have to separate scientific and technologica
11 facts from value judgments and be careful of your limitatic
12 on knowledge, especially in tests for determining
13 carcinogenicity when you have a relatively low potency
14 substance.
15 DR. ALBERT: That formula looks more formidable
16 than it is, it's simply a linear response in a trivial
17 formula. Even the EPA uses it.
18 MR. BARBER: Thank you, sir.
19 That should conclude today's presentation by the
20 AIHC folks. Are there any questions of a general nature
21 that the panel may want to readdress to Mr. Batchelor or
22 any of the other AIHC folks?
23 Dr. Anderson has a question.
24 DR. ANDERSON: Mr. Batchelor, I had one question
25 from your general comments this morning. It seems that a

1 centerpiece of the AIHC proposal is that an outside
2 scientific committee be formulated to assess carcinogenic
3 risk because of two things.
4 One, to introduce objectivity and two, to be
5 certain of consistency in the Federal Government's
6 consideration of scientific data to identify carcinogens.
7 I wonder if this means that you think that EPA's carcinogen
8 group lacks objectivity and two, I wonder if you could name
9 any chemicals where you feel there has been inconsistency in
10 judging carcinogenicity among agencies?
11 MR. BATCHELOR: Dr. Anderson, in AIHC we are very
12 appreciative of the fact that EPA solely among all the
13 agencies has the best carcinogenic assessment group. What
14 we do wish would be to get that into an area where all
15 agencies can benefit by a single determination.
16 Secondly, we do feel that there are occasions
17 when better work is done outside the political pressures
18 put on an agency.
19 DR. ALBERT: I think it is worth pointing out
20 that the carcinogen assessment group was set up with the
21 specific provision that it not be subject to any pressures
22 and as chairman of the group, I can reassure you that it
23 hasn't been.
24 MR. BATCHELOR: I can hear you, sir, and I don't
25 want to belittle good effort, but as I hear the discussion

1 today, I hear you really having made a decision that this
2 is the regulation you want and you are defending it. I
3 don't hear you listening.
4 DR. ALBERT: I was not defending at all, I'm just
5 criticizing each speaker as they came along.
6 (Laughter.)
7 MR. BARBER: I had one question from the AIHC
8 perspective. Is it AIHC's position that carcinogens,
9 suspected carcinogens or proven carcinogens, should be
10 subjected to rules less stringent than pollutants regulated
11 under Section 111 of the Clean Air Act? New sources I am
12 speaking of particularly.
13 MR. BATCHELOR: I'm going to run the risk of
14 asking you to repeat the question.
15 MR. BARBER: The question was, should new sources
16 of carcinogens or suspected carcinogens be subject to rules
17 under Section 112 which are less stringent than they would
18 be under Section 111?
19 MR. BATCHELOR: I really think that even our best
20 people would have difficulty answering that question. It i
21 reasonably speculative.
22 MR. BARBER: If there are specific criteria laid
23 out in the statute for Section 111 that all major new sourc
24 of air pollutants be subjected to a requirement for BACT
25 considering costs of energy and environmental impacts,

1 I'm not sure whether the AIHC recommendation is a less
2 stringent rule for new sources of carcinogens, an equivalent
3 rule for new sources of carcinogens or more stringent rule
4 for new sources of carcinogens.
5 MR. BATCHELOR: May I say that I think the best
6 thing for us to do is to respond to your question a little
7 bit later.
8 MR. BARBER: That's fine, it's just something I
9 would like to have clarified.
10 MR. BATCHELOR: We are basically against the
11 proposal of generic regulation. We think you have to do
12 these things one by one.
13 MR. BARBER: That is an area that we need to
14 clarify for you because we think that's what we're doing.
15 It's obviously not communicating.
16 I think that's all the questions for the AIHC
17 today and we appreciate your efforts. Thank you.
18 MR. BATCHELOR: Thank you.
19 MR. BARBER: I would like to proceed with the
20 schedule to Jesse Norris from the Dow Chemical Company.
21 DR. NORRIS: My name is Jesse Norris and I am an
22 Associate Scientist in the Health and Environmental Services
23 of the Dow Chemical Company. My comments today are concerned
24 with EPA's approach to the identification of chemical
25 carcinogens.

1 Written comments have been submitted to you with
2 further details than I am going to give to you today and
3 references have been given to you for all of the statements
4 I am going to make.
5 Please take this in the way it is being offered,
6 as help to EPA, I hope, and not criticism.
7 The Agency's administrative approach to the
8 identification of potential chemical carcinogens has
9 involved simplistic, ultraconservative assumptions on
10 certain scientific issues. While these assumptions may
11 be unlikely to underestimate risk, they are nevertheless
12 contrary to the scientific approach which is based on an
13 understanding of the relationship between dose and response
14 EPA has taken a position that results from one
15 animal study to be considered as evidence of carcinogenicity
16 and presumably evidence that can be obtained from the
17 bioassay conducted at maximum tolerated dose. I submit
18 such a position coupled with EPA's position that there
19 is no threshold for chemical carcinogenesis actually
20 precludes the scientific evaluation of animal data.
21 First, let me briefly comment on the basis for
22 the no threshold concept held by EPA and that will get us
23 off the ground. The thrust of my comments are going to be
24 concerned with the adequacy of the data base on which EPA
25 relies to identify the potential carcinogen and the

1 pertinency of pharmacokinetic and mechanism of carcinogenesis
2 in such determinations.
3 First of all, in my judgment, EPA's no threshold
4 position on chemical carcinogenesis and the lack of a
5 provision for the interpretation of animal data based on
6 the mechanism of carcinogenesis and the pharmacokinetic
7 metabolish data will be to the unnecessary control of
8 substances or necessitate inconsistent interpretation
9 of data.
10 Surely, we are concerned with cancer per se, not
11 regulation, but cancer. We must be able to project the
12 result of studies on such chemicals as selenium, black
13 pepper, egg yolks and mushroom, all of which have been
14 found to be carcinogenic in an animal species at a
15 particular dose.
16 We must be able to subject these results to the
17 same type of evaluation given other potential cancer
18 carcinogens. In EPA's document on airborne carcinogens,
19 the Agency has stated that a public health community has
20 concluded that evidence for the identifiable dose thresholds
21 do not exist for carcinogens and in support of this position,
22 EPA has cited the NAS/NRC document entitled, "Drinking Water
23 and Health".
24 A review of this document, however, brings to
25 light the fact that there were limitations based on the

1 statements relative to dose thresholds made by the Safe
2 Drinking Water Committee of the National Research Council.
3 The council stated that despite all the complexities of
4 chemical carcinogenicity, thresholds in dose response
5 relationships do not appear, and I stress do not appear
6 to exist for direct emphasis again, acting carcinogens.
7 The committee also stated that if there is eviden
8 that a particular carcinogen acts by directly causing
9 mutation in DNA, it is likely, emphasis again, likely,
10 that a dose response curve or carcinogenicity will not
11 show a threshold.
12 In recent years, on the mechanistic processes
13 involved in chemical carcinogenesis, it has been demonstrat
14 that there is an existence of many control mechanisms that
15 modulate or repair the damage done by chemical carcinogens.
16 Irrespective, EPA's interpretation of the NAS opinion, EPA'
17 position in the basis for non-ionizing radiation.
18 I hate to touch this but I will do it because it'
19 in the literature and placed there by experts. Although
20 this data on carcinogenic effects of ionized radiation are
21 frequently cited as evidence for the so-called linear
22 no-threshold hypothesis, inspection of the data show
23 that linear extrapolation is not a factual representation
24 of all the available data.
25 The inclusion of the no-threshold concept has

1 also been questioned by the analysis of Carlborg of data
2 that has been recently completed in an animal study on
3 (2-AAF). Again, you heard this is linear no-threshold.
4 However, another evaluation has shown that both the data
5 on the dose response and the data on the time to tumor
6 relationships clearly show that the one-hit or the linear
7 no-threshold model do not hit the primary tumor data.
8 We can say the one-hit model can be statistically
9 rejected in favor of more general sublinear model. Let me
10 go back, I know I will get questions on the ionized
11 radiation. Let me quote you something from the full
12 text that I gave you.
13 We recognize the most important source of -- on
14 the effects of ionized radiation on draft data that has come
15 from the incidence of leukemia among survivors of the
16 atomic bomb blast in Hiroshima and Nagasaki. The National
17 Academy of Sciences in 1972 reported that whereas the
18 leukemia data from Hiroshima implied a linear dose response
19 relationship, the data from Nagasaki does not.
20 Rather, the Nagasaki data implies a dose
21 incidence relationship consistent with experiments on
22 leukemia induction in mice and Dr. Upton reported on that
23 in 1975. What is the difference between these two cities?
24 In Hiroshima, the exposure of the substantial
25 neutron dose was primarily judged to be responsible for

1 the effects observed while in Nagasaki, the relative neutro
2 dose was very low and virtually negligible away from the
3 center of the explosion.
4 Mark, et. al., '71, have reported on that data.
5 Furthermore, a signal relationship has been shown to
6 admirably fit over a restricted dose range -- the data
7 for radiation into skin tumors in rats by Burns, et. al.,
8 in 1968, kidney tumors in rats, 1979 report, and sarcomas
9 in patients exposed to radium, Evans, et. al., 1969.
10 Let me go back to the way we run animal studies.
11 I don't want to be misunderstood. I am not saying that we
12 do not have to expose animals to large doses when we run
13 these studies. They are not to be aborted. However, the
14 interpretation of the data requires on a case-by-case basis
15 an interpretation of the mechanism involved in producing th
16 carcinogenic response.
17 In toxicology, it is frequently evident that dose
18 of a chemical required to elicit a toxic response exceeds -
19 in such cases, non-linearity of pharmacokinetic profile is
20 of the utmost importance in the interpretation of the data
21 since there may be a disproportionate increase in toxicity
22 which includes carcinogenicity with the increase of the do
23 With respect to carcinogenicity, the overwhelmin
24 detoxification pathways, metabolic and/or(explatory) are
25 important because rather than DNA toxified reactive -- may

1 be present to bind with the molecules of the cell such as
2 DNA.
3 Obviously, in such cases, the data must be
4 interpreted cautiously when attempting to assess the risk
5 to a much smaller dose. The consequences of overwhelming
6 detoxification pathways has been clearly demonstrated in
7 any number of studies.
8 For example, recently it has been reported that
9 large doses of acetominophen, a therapeutic drug, has been
10 reported to cause a dose dependent necrosis in laboratory
11 animals. Hamsters and mice developed hepatic necrosis and
12 after 375 milligrams per kg body weight respectively, but
13 not at lower doses, lower doses moved down to 25.
14 At the hepatic toxic doses, chemical -- caused
15 extensive depletion of the liver and binding of
16 acetominophen to the liver protein became extensive
17 only after the liver thyroid was depleted. The co-binding
18 correlated well with dose dependent hepatic necrosis.
19 The result of these studies gave evidence of
20 existence not only of a dose threshold for live necrosis,
21 but non-linearity of co-binding. Studies reported on --
22 show the same sort of thing, that the severity of liver
23 necrosis in animals paralled the co-binding to protein
24 and depletion of liver to the (thyrone).
25 EPA is in a position -- on the linear extrapolation

1 of data from high dose levels presupposes that the mechanis
2 of carcinogenesis is by somatic mutation and that is to say
3 a chemical interaction with DNA. I am afraid no provision
4 has been made for interpretation of carcinogenicity data
5 for chemicals that reduce a carcinogenic response by
6 mechanisms that do not involve chemical interactions.
7 These are non-genetic mechanisms of cancer
8 induction. To take an example, during chronic
9 administration of cytotoxic agents, damaged cells are
10 replaced by new cells and DNA replication is stimulated
11 greatly in the affect cell and the rate of DNA repair is
12 altered.
13 Synthesis and repair rates may be highly
14 important because DNA repair mechanism may be less
15 effective in correcting small amounts of genetic damage
16 after replication had taken place. Since there is a small
17 but finite error rate in normal DNA synthesis, the net
18 effect is to increase spontaneous mutation rate and
19 ultimately enhance the carcinogenic response.
20 It stands to reason, in my way of thinking, that
21 doses which do not induce tissue injury should not affect
22 spontaneous tumor incidence and therefore if tumors in
23 animals arise via a non-genetic mechanism, the use of
24 a linear no-threshold model would be inappropriate in
25 the estimation of risk.

1 The role of non-genetic mechanism of carcino-
2 genesis has been researched in studies on perchloride, for
3 example. Studies were conducted in an attempt to explain
4 the sensitivity of a mouse and the resistance of a rat to
5 perchlorethylene induced carcinoma in lifetime studies,
6 NCI studies.
7 The mouse strain used in the NCI studies were
8 the same. This particular mouse strain, the B-6, is highly
9 prone to spontaneously occurring hepatocellular carcinomas.
10 This fact is important in interpretation of the mechanistic
11 data.
12 When compared to the rat, mice were found to
13 metabolize more perchlorethylene per kg body weight at
14 a comparable dose. Since the initial metabolism of
15 perchlorethylene is an activation process, the increased
16 extent of metabolism in the mouse resulted in a greater
17 extent of irreversible binding of radioactivity and the
18 hepatic molecules of the mouse cell when compared to the
19 rat.
20 Repeated oral administration of perchlorethylene
21 over a period of 11 days resulted in histopathological
22 changes in the liver of the mice at doses as low as 100
23 milligrams per kg per day, while in the rat at doses as
24 high as 1,000 milligrams per kg per day, there were only
25 minimal treatment related effects.

1 We are seeing a correlation between histopath and
2 what we understand of the mechanism of the effect.
3 Approximately a twofold increase in hepato DNA synthesis,
4 this turnover of the DNA in the cell which is indicative
5 of hepatic regeneration was observed in the mice but not
6 in rats after repeated oral administration of perchlorethyle
7 at dose levels which are tumorgenic in the mice in the
8 lifetime study.
9 By the way, that was 500 milligrams per kg. The
10 absence of any pronounced direct interation of perchlorethy
11 ene with hepatic DNA in the mice at times of peak hepatic
12 micromolecular binding suggests that the hepatic tumors are
13 induced in mice by the recurring cytotoxicity which enhance
14 the spontaneous instance of liver tumors in this highly
15 prone species.
16 The implications of these studies obviously has
17 an assessment made on the data, would have to be cognizant
18 of the fact that recurring tissue damage is necessary for
19 the expression of tumors.
20 In conclusion, although I will admit that absolut
21 thresholds for chemical carcinogens cannot be proven
22 unequivocally, an understanding of the mechanisms involved
23 in the carcinogenic response -- let me make that plural
24 because it's not just one, in the carcinogenic responses
25 and provisions for the uses of such data from the

1 interpretation of animal data will yield a scientifically
2 supportable basis for identification of potential human
3 carcinogens. Thank you.
4 MR. BARBER: Thank you. Dr. Albert, do you have
5 questions?
6 DR. ALBERT: I believe that you essentially
7 marshalled a series of reservations about the validity of
8 a non-threshold dose response in terms of the argument that
9 it should be limited only to direct acting agents, that the
10 evidence from ionizing radiation should be regarded as
11 limited to neutrons in Hiroshima, that there isn't any
12 evidence that it should apply to carcinogens that take
13 their effect by non-genotoxic routes and maybe there is
14 better repair at more doses than at higher doses in terms
15 of DNA injury and you revise the old Dirkhaus theory of
16 carcinogenesis, namely chronic injury.
17 In terms of the pharmacokinetic arguments, that
18 is of different metabolism at lower doses, I might say that
19 the CAG position in this regard is to welcome data that would
20 indicate that the production of the specific ultimate
21 carcinogen, if it is known, to welcome data that shows
22 that production of this ultimate carcinogen is reduced
23 at lower doses so that this can be taken into consideration.
24 But it seemed to me that you thoroughly have gone
25 over all of the reasons for qualifying the notion of a

1 non-threshold dose response. But in effect, I don't think
2 that you have come up with any crucial arguments that would
3 dismiss it.
4 For example, arguing that perhaps DNA repair is
5 more effective at lower doses is speculative. If you take
6 a look at the amount of DNA interaction, for example, in
7 the skin of a mouse required to produce a single tumor,
8 the numbers of carcinogen molecules that interact with
9 DNA is astronomical, it is in the order of billions to
10 get one tumor.
11 So DNA repair must be extraordinarily efficient
12 except that it does miss.
13 DR. NORRIS: I spoke of the DNA repair only in my
14 written testimony and not my oral testimony and that's a
15 different subject. Let me answer some of your questions
16 because you are getting ahead of me.
17 First of all, I did not bring out ionized
18 radiation to any way minimize the seriousness of ionized
19 radiation but only to show that making generalized statemen
20 yes, no, black, white, absolute statement that there is no
21 such thing as thresholds for carcinogenic response does not
22 hold when you review the scientific data.
23 What I am asking is let us recognize what the
24 scientific data say and grow from there, but let's not make
25 absolute statements based on a portion of the scientific da

1 and ignore the rest. When you go back to ionized radiation,
2 you have got to admit that there may be indication of a
3 (Sickmore) curve for some of the data.
4 It shows an inflection in the dose. That's not
5 a strong enough basis for the Agency to jump over to chemical
6 carcinogens or potential ones and say because of ionized
7 radiation, we are going to say that chemical carcinogens
8 cannot be shown to have a threshold.
9 That is an illogical sequence of events and the
10 only reason I brought the ionized radiation up is to show
11 you that we can read the literature, too, and we can make
12 an interpretation based on what the experts say. The experts
13 qualify.
14 I think what we need to do, we need to be careful
15 how we make those absolute statements. Maybe we can't
16 unequivocally show that there is a threshold for the direct
17 acting carcinogen. That's the subject for another conference
18 with much time given to the data base.
19 But for the non-genetic, when we are talking about
20 cytotoxicity as one of the non-genetic mechanisms, and I
21 stress this only one, when we see that cytotoxicity,
22 recurring cytotoxicity is necessary before we can see
23 a tumor expressed in an animal, if we are going to look
24 at that animal data at all as an indication of what we
25 humans might be experiencing in 20 years, we have to read

1 it the way science allows us to read it.

2 If you have a dose level where your animals do not

3 show any cytotoxicity, it stands to reason you put a margin

4 of safety on that dose, understanding perhaps the

5 pharmacokinetics and metabolism material. Put a safety

6 margin on that and that should be a dose to which we as

7 humans can be exposed without having any increased risk

8 of cancer. That's the type of interpretation that I think

9 we need to put on the data, rather than lumping it all

10 together.

11 DR. ALBERT: Well, one can always ask, to be sure

12 one can get a nice correlation between the induction of

13 chronic tissue damage in an organ and the induction of the

14 tumors. How do you know that that is causal rather than just

15 a parallel sort of thing, sort of relationship?

16 Namely, that the effect of the agent is not a

17 single effect, there is a spectrum of effects. Some of

18 these relate to the induction of cell killing and if it

19 gets severe enough to irreparable tissue damage and others

20 have to do with the aspect of neoplastic transformation, I

21 think you are making the logical jump that because there is

22 a relationship, there is a causal relationship.

23 DR. NORRIS: There is a causal relationship if

24 you believe the data. There is a causal relationship for

25 perchlorethylene. We can set it up and it's a logical one.

1 You can also show with perchlorethylene as far as
2 the mechanistic type data is concerned, that the DNA per se
3 is not damaged. The DNA per se is not damaged.
4 DR. ALBERT: Within detection limits.
5 DR. NORRIS: Background, background. If you have
6 a yardstick, this is what I would like to allude to. If you
7 have a yardstick and put nitrosamines at this end of your
8 yardstick and at this end of your yardstick you have
9 background, that is where perchlorethylene is.
10 When you deal with data, if you are willing to
11 deal with data at MTD dose levels, which is a gross way of
12 evaluating a chemical's toxicity, you have to deal with the
13 data that is definitive enough to say that we know how that
14 cancer is being formed and that is all we are saying.
15 You claim you are going to weight the evidence.
16 I commend you, that's great. I think you need help in
17 weighting the evidence and that is where I think you ought
18 to reach out and get to the scientist who is working with
19 these kinds of systems who can help you differentiate between
20 this potential human carcinogen based on these data and this
21 one that might be a human carcinogen.
22 You will never know unless you go after the
23 mechanism. You will never be able to take the black pepper
24 and separate black pepper from an aflatoxin from vinyl
25 chloride unless you get down and do the DNA work. You

1 will never know how bad it is.

2 MR. BARBER: Can I ask whether the logical

3 extension of your comments is that, given the uncertainty,

4 no regulatory response is appropriate or are you suggesting

5 that we should reduce the uncertainty with as broad a review

6 of the data as possible and then try to make the appropriate

7 response?

8 DR. NORRIS: The second one, of course. We are

9 not telling you not to perform your task, no way. We would

10 like to see the evaluation of these chemicals done in the

11 most logical fashion with the best science we have possible

12 Basically, we are after survival of the industry, we don't

13 want to be regulated to death.

14 But even if you regulate the industry to death

15 and you don't reduce the cancer instance at all, you have

16 done nobody any favors. If cancer per se is the problem,

17 and we have to believe it, let's go after the problems with

18 the cancer data, let's understand what we are doing. We

19 are willing to help you find the answers.

20 MR. BARBER: Any other questions? Betty?

21 DR. ANDERSON: Yes, just briefly. First of all,

22 I think it's important to point out that in both the IRLG

23 documents assigning the basis for risk down to the EPA

24 approach, there has never been an absolutely statement

25 saying there is no threshold. Quite the contrary.

1 Where data are available, they will certainly be
2 used. Of course, we don't always have the data, in fact we
3 barely have data that we can use. I'm not quite sure you are
4 suggesting when we don't have this information. Are you
5 suggesting a different dose response curve be assumed or
6 some threshold be assumed or are you suggesting that, unlike
7 the current bioassay test rules contemplated under the Toxic
8 Substances Act, of course these are coming under heavy attack
9 because of the cost already imposed, that there be more
10 testing rules which generate or attempt to generate this
11 kind of data? I'm not sure what solution you are suggesting.
12 DR. NORRIS: There is a logical approach to the
13 problem. I heard SAR structural analysis relationships might
14 be one of the factors that influences someone to categorize
15 a material one way or the other. That is the type of
16 disastrous approach that we may be going downhill because
17 it's simplistic.
18 An organic chemist can look at the compounds and
19 make judgments relative to the potential activity of the
20 material, maybe under vapor pressure, etc. We can make
21 judgments. We make too many judgments. When you get back
22 to the world of toxicology, you find the judgments you make
23 may not be true for a variety of reasons, but the decisions
24 have been made.
25 I don't think we should do this. I think there is

1 a place for the Ames test. The Ames test will tell you what
2 an ultimate potential might be if you just have DNA and the
3 material.
4 DR. ANDERSON: What I am asking is, you have
5 bioassay tests with one or two test levels in the controls,
6 you have a carcinogenic response and that's all we know. Ar
7 you suggesting we do nothing until we have --
8 DR. NORRIS: Absolutely nothing. If you go back
9 to the beginning of the bioassay, the caption will always
10 say this, the test was meant as a screen.
11 DR. ANDERSON: Are you suggesting we don't use
12 animal bioassay data, say two or three level until we have
13 a defined mechanism?
14 DR. NORRIS: It depends on how you pick your dose
15 level. When you pick a dose level that will give you no mo
16 than a 10 percent decrease in body weight and not shorten t
17 life of the animal, and you may not see that 10 percent
18 decrease in body weight until the third month, but you
19 continue on with the study, that's too gross an approach.
20 It's like looking at the animal, if you will, as
21 a black box. You are putting these dose levels in and
22 getting something out, you're getting decrease in body
23 weight, tumor incidence, whatever. You take that data
24 and do a lot of things.
25 You begin to regulate, you categorize, you

1 regulate, you do risk assessments. It all may mean nothing
2 unless you know what's going on inside the animal. I'm
3 saying don't react to bioassay data. That's the beginning.
4 DR. ANDERSON: If the Agency is going to require
5 test data that generates information that can be used in
6 regulatory action, what are you suggesting? Biotest levels?
7 DR. NORRIS: No. First of all, I would do a probe
8 study, something that isn't done. The probe study would be
9 one that you might want to do five levels in your probe study
10 and your probe study might want to go on for 30 days. You
11 might want to do all the evaluations.
12 You need bloods, you need chemistry, you need the
13 growth, you need the histo, more than anything else you need
14 that in your probe study. You have to understand how things
15 are beginning to react. You can do the Ames test and get an
16 indication if you break down all the detoxifying mechanisms
17 and take away all the barriers.
18 What ultimate reaction you might possibly get
19 over the DNA? You can do the DNA studies in vivo in the
20 animal to determine whether you have damage, repair or just
21 DNA synthesis or turnover time increase. Primarily with the
22 bioassay, you have got to understand what is happening at
23 those various dose levels.
24 A pharmocokinetic profile, you don't have to
25 identify the metabolites but just a feeling how that animal

1 is handling the material, where it is being distributed and
2 how it is being cleared by the body of the animal. It will
3 tell you so much about the data being accumulated in the
4 bioassay.
5 When you get through with the bioassay, you will
6 have these data and therefore when you get to the point
7 where you are going to make a decision as to what the
8 bioassay meant relative to man, you at least have some
9 understanding of what it means to the animal.
10 The extrapolation to man on select materials,
11 we can get the exposure experience or the pharmacokinetics,
12 if you will. We have them on a number of chemicals that ar
13 not potently carcinogens. It gives you a base on which to
14 make a logical extrapolation.
15 You can't do it with all the materials. There
16 has to be a judgment factor put in when you cannot test
17 materials but where you can, you do. Where you can't, you
18 have to rely on the experts who have been interpreting data
19 for many a year.
20 DR. ANDERSON: That's what we think we do
21 DR. NORRIS: You need to do it with the
22 understanding of pharmacokinetics and the dose response
23 and what is going on in that animal.
24 MR. BARBER: Any other questions?
25 Fine, thank you very much.

1 I would like to call Mr. John Thorpe.

2 DR. THORPE: I would like to relieve the panel's
3 mind on one item that Steven Swanson who is listed to appear
4 with me will not be making a statement but will be available
5 to answer questions by the panel.

6 The second thing I would like to say is that
7 after hearing all of the testimony offered today, I find
8 it increasingly difficult to think that I am going to add
9 anything very new to your knowledge. But we have spent
10 considerable time in developing comments and we would like
11 to outline our position to you.

12 I am John Thorpe, Associate Medical Director of
13 Exxon Corporation appearing today in my capacity as Chairman
14 of the Task Force on Cancer Policy of the American Petroleum
15 Institute. API is a nonprofit trade association representing
16 all segments of the petroleum industry in the United States.

17 The EPA proposed entitled, "National Emission
18 Standards for Identifying, Assessing and Regulating Airborne
19 Substances Posing a Risk to Cancer" could have a substantial
20 impact on the operations of our member companies once it is
21 applied.

22 I am accompanied today by Steven Swanson, Director
23 of API's Department of Health and Safety Regulation who will
24 assist me in answering questions about API's recommendations.

25 At the outset, let me say that API supports EPA's

1 initial efforts to develop a decision making framework for
2 establishing emission standards in the difficult area of
3 cancer regulation. We appreciate the opportunity to
4 participate in the process for developing such a framework.
5 In fact, as I will discuss in a moment, there are
6 features of this proposal, principally in the science area,
7 which in our view represents significant improvements on
8 EPA's 1976 interim risk assessment, guidelines, and OSHA's
9 cancer policy.
10 However, as API discusses in detail in its
11 comments filed with EPA on February 21st, we believe the
12 proposal as written cannot be lawfully implemented in
13 accordance with the Section 112 of the Clean Air Act or
14 the intent of Congress in promulgating that section.
15 Our legal criticisms of EPA's approach are covere
16 in detail in the pre-hearing conference. I do not intend t
17 discuss these legal infirmities here today. Rather, I woul
18 like to focus on the scientific basis for the proposal in
19 the regulatory application of the science by EPA.
20 I will attempt to show that although the proposal
21 appears to contemplate consideration and weighing of a wide
22 range of scientific data, it does not provide for adequate
23 use of that data in arriving at a decision of whether and
24 how to regulate emissions of a particular substance.
25 Section 112 of the Clean Air Act was intended to

1 cover "exceptionally dangerous hazards". The current EPA
2 proposal predetermines that all substances which meet
3 minimum risk criteria such as the demonstration of
4 carcinogenic potential in one animal species without
5 regard to potency and the possibility that there may
6 be some ambient exposure to unspecified levels of the
7 substance shall be treated in "as maximal danger category".
8 We believe very few substances which EPA considers
9 to be "high probability carcinogens" will pose risks so
10 significant that a determination that they are "exceptionally
11 dangerous pollutants" can be made solely on the basis of a
12 qualitative risk assessment.
13 As outlined in our written comments, we believe
14 that other sections of the Act, more particularly Sections
15 108 through 111, would give EPA much more flexibility to
16 appropriately regulate materials with a wide range of
17 carcinogenic potential.
18 To link the narrow scope of Section 112 with the
19 listing criteria in the current proposal creates a system
20 of regulatory response which might very well undercut the
21 Agency's intention to set up a rational regulatory scheme.
22 To be more specific, EPA begins it's pre-listing procedures
23 or "preliminary evaluation of risk" with an objective
24 qualitative evaluation of the regularly available risk
25 evidence, case reports, recent research data, etc., to

1 determine the probability that the substance is a human
2 carcinogen.
3 API concurs in this decision to use the "weight
4 of the evidence" approach rather than arbitrary which is
5 classification scheme. All scientific evidence regarding
6 carcinogenicity of a substance should be reviewed and its
7 quality assessed.
8 Well-conducted studies should not be ignored
9 because they do not yield positive results. The proposal
10 undoes its good beginning by saying it will rely on the
11 draft IRLG risk assessment guidelines of June '79 which
12 are inherently inconsistent with this weight of the evidenc
13 approach.
14 As we said in our November 30th, 1979 comments
15 to the IRLG, the draft guidelines incorporate highly
16 conservative assumptions which tend to unrealistically
17 overstate the probability of carcinogenicity and the
18 magnitude of the risk.
19 For example, the draft IRLG report unfairly
20 weighs positive epidemiological data against negative
21 epidemiological studies, irrespective of quality and places
22 too much weight on the results of non-mammalian mutagenicit
23 studies.
24 We are also concerned about possible over-relianc
25 on results of a single animal test. While the proposal is

1 unclear on this issue, the preamble might be read to suggest
2 that a single positive animal test can be used as the basis
3 for regulation.
4 EPA should make clear that there will be few
5 occasions where a single animal test is the sole evidence
6 available. Furthermore, in those rare cases where a single
7 animal test is the only evidence available, EPA should
8 exercise caution in interpreting the results of the study.
9 Positive animal tests are often equivocal because
10 of the relatively high probability of false positive results.
11 Fierce has explained the necessity of using dose response
12 relationships to guard against the false positive trap.
13 "There is a danger in relying solely on the findings of
14 statistical significance without incorporating biological
15 knowledge and corroborative evidence such as the presence
16 of a dose response relationship -- or experimentally
17 consistent results in different species or sexes" and
18 this quotation is listed or cited in our comments.
19 API believes that if the results of single species
20 tests are to be accepted as "substantial evidence that
21 substances are potential human carcinogens", it is essential
22 that a strong positive dose response relationship be shown.
23 Once EPA finds that the substance really does appear to be
24 a high probability human carcinogen, the next step in the
25 process contemplated by the proposal is an analysis of

1 whether significant public exposure to the substance exists.
2 Once again, EPA has set forth relevant informatio
3 to be considered such as the number and type of emitting
4 sources and the number of people living near those emitting
5 sources. However, the Agency has set the threshold for
6 significant exposure so low that only substances which
7 are not present in the air in measurable quantities which
8 EPA refers to as "laboratory curiosities" would be screened
9 out at this stage.
10 API believes this step of the process should
11 consist of the object of analysis of the probable extent
12 of human exposure. This information can then be integrated
13 with other scientific evidence such as potency and dose
14 response information to obtain a quantitative risk estimate
15 which reflects the significance of exposure to humans and
16 the nature and extent of the actual hazard posed.
17 Only by routinely conducting such quantitative
18 risk assessments prior to listing can EPA's decision be
19 rational and consistent with requirements of the Clean Air
20 Act. Why EPA chose to reverse a sound long-standing Agency
21 wide policy of conducting quantitative risk analysis before
22 investing time, scientific talent and effort in a control
23 strategy is not satisfactorily explained in their proposal
24 Yet the proposal creates a double standard, first
25 doing a quantitative risk analysis and then doing a

1 quantitative risk analysis only if the qualitative risk
2 analysis fails to show risk. API believes this double
3 standard for the use of these results is totally
4 inappropriate.
5 We urge the Agency to use whatever scientific
6 information is available to routinely make a quantitative
7 projection of the extent of human risk at ambient exposure
8 levels. This information will facilitate reasonable decisions
9 about which a regulatory vehicle is most appropriate in view
10 of particular risk posed in a given case.
11 In conclusion, I would like to repeat that API's
12 principal scientific objection to this EPA rule as proposed
13 is that it fails to adequately assess the extent of risk
14 caused by a substance before proceeding to list and regulate.
15 We strongly recommend that EPA utilize quantitative risk
16 estimates to promote objective scientific judgments about
17 the degree of hazard posed by a potential carcinogen and
18 that they base the regulatory decision on how best to control
19 that hazard on sound scientific risk assessment information
20 in conjunction with appropriate legal policy and economic
21 considerations.
22 As I stated at the outset, the comments filed by
23 API on February 21st comprehensively address a wide array
24 of scientific, legal and policy issues and I refer you to
25 that document for points not addressed here and for a more

1 thorough analysis of the points just discussed.

2 Thank you for the opportunity to participate at

3 this hearing and I will now answer any questions the panel

4 might have.

5 MR. BARBER: Thank you. Any questions?

6 DR. ALBERT: You mentioned that this policy is an

7 improvement over the interim guidelines but you never said

8 how?

9 DR. THORPE: I think the major improvement is

10 that you have gone to "weight of the evidence" approach.

11 DR. ALBERT: That was in the interim guidelines

12 as a major point.

13 DR. THORPE: That is the significant improvement

14 in the last one.

15 DR. ALBERT: Originally, the interim guidelines

16 that came out in '76 featured the weight of evidence

17 approach. This current proposal simply echoes it.

18 DR. THORPE: As we interpret it, it did not come

19 out that clearly.

20 MR. BARBER: Betty, any questions?

21 DR. ANDERSON: Just a very brief comment. The

22 IRLG document is something we participated in developing

23 and we adopted it and the Agency signed it. You said that

24 that is inconsistent with the interim guidelines. I was n

25 clear as to why and perhaps something submitted for the

1 record on that would be of interest to the Agency.

2 DR. THORPE: I think that's included in our

3 comments.

4 MR. JOSEPH: Mr. Thorpe, do you have a view on

5 the range of accuracy that might be attributed to quantitative

6 risk assessment at this point?

7 DR. THORPE: I think it is an inexact science but

8 I think this morning we had considerable discussion and

9 debate about how wide the estimates could be. What we

10 are saying is that we believe that you should take a look

11 at all of the data you possibly can, that you should not

12 have one particular factor triggering your decision to

13 embark on a regulatory procedure. That you should try

14 to look at as many aspects of the problem as possible

15 and to look for any quantitative data you can obtain.

16 Put it altogether and then make a judgment. In

17 some instances, this information can be readily obtained

18 from the material if it is available and in other instances

19 you will have to make real estimates. We are saying that

20 you are better off to do it this way than to just make a

21 guess.

22 MR. JOSEPH: Do you think we ought to take into

23 account the accuracy of the estimate in deciding what use

24 to put it to?

25 DR. THORPE: I would think you would have to

1 evaluate it in that light. Any judgment you make, you
2 weigh the validity of the data you are dealing with.
3 MR. BARBER: Any questions? Thank you very much.
4 We'll take a five minute recess and reconvene.
5 We have four more speakers and I suspect we should be
6 completing on or about 7 o'clock.
7 (Whereupon, a recess was taken.)
8 MR. BARBER: Our next speaker is Somendu Majumdar
9 DR. MAJUMDAR: Mr. Chairman, ladies and gentlemen
10 I have been given ten minutes for this presentation. I
11 would like to limit it to five minutes so there are five
12 minutes left for questions.
13 My name is Somendu Majumdar and I'm the Manager
14 of Environmental Engineering at AIRCO, Carbon Division of
15 AIRCO, Inc. at St. Mary's, Pennsylvania. The comments I
16 am going to offer should be viewed as observations of the
17 problems which industries which have to deal with the
18 regulations face, particularly people like us.
19 I am not going to present any data but briefly
20 state one of the problems we see in the proposed regulatior
21 EPA, it seems, has recognized two different schools of
22 thought in terms of limits, threshold and non-threshold.
23 It's not very clear to us what prompted EPA to go with the
24 idea of having the concept of non-threshold limits for
25 carcinogens or potential carcinogens.

1 It seems that even if EPA does have reasons to go
2 with a non-threshold limit, which means essentially a
3 baseline of zero has been advocated, it is not clear what
4 the zero means. Does it mean absolute zero or a trace but
5 non-quantifiable figure?
6 In the next point, we find that EPA talks about
7 BAT. What does BAT do to this so-called zero baseline?
8 Does BAT reduce everything to zero? There seems to be
9 some problem in understanding this concept of BAT with
10 respect to so-called baseline data or baseline hypothesis.
11 The problem we have is that in spite of spending
12 money and so to say coming up with BAT technology, we cannot
13 in most instances -- I most humbly state that we cannot come
14 up with zero. There will be trace amounts, even with the
15 present state of technology and even if you consider progress
16 in the next five years.
17 The next point I would like to make is about the
18 combination of so-called carcinogens and non-carcinogens in
19 a mixture. For example, in gaseous emissions, if we had
20 water combinations, does that make the mixture a carcinogen?
21 The point can be made that because of chemical reaction,
22 some carcinogenic effects can be nullified by the presence
23 of non-carcinogens.
24 We have not seen any attempt on the part of EPA
25 to address that issue.

1 The next item I would like to talk about is the
2 prevailing air quality in a particular region. It seems
3 that from these proposed regulations that EPA is about to
4 come up with national standards. Now, it is anybody's
5 knowledge at this point that there is a human tolerance,
6 air quality does differ from region to region.
7 To impose the same standard for every classifica-
8 tion, there seems a certain amount of unjustification. Alsc
9 for industries which have certain health programs. For
10 example, exercise programs, strict adherence to OSHA
11 regulations, regular checkups.
12 There seems to be no correlation with that with
13 respect to carcinogenics. Everyone knows, it is fairly wel
14 accepted, that the risk of cancer among smokers is a lot
15 higher when compared to non-smokers. There has to be a
16 credit given to that fact and we don't see any such thing.
17 Does that fall in the line of BAT or not, I don't
18 know, and I would like EPA to suggest what the industry can
19 do to achieve the so-called BAT concept. We have heard so
20 much about the bioassay technique for assuming what is
21 carcinogenic or potentially carcinogenic.
22 To me it seems that sometimes, in lieu of any
23 other procedure, most of the time what we're doing with
24 so-called bioassay technique is basically second-guessing
25 because of the fact that concentration of the so-called dos

1 levels in bioassay techniques are considerably higher than
2 one is normally exposed to.
3 We all know radiation causes leukemia, but there
4 is a limit. When does a bioassay indicate there is a
5 positive aspect or negative aspect? There should be some
6 risk assessment. The doses cannot be administered at any
7 level and thereby be judged what is carcinogenic and what
8 is not carcinogenic.
9 I believe I have taken up five minutes so I will
10 stop by simply summarizing the fact that we, from the
11 industry, do believe that this acceptance by EPA of a
12 non-threshold limit, so-called zero baseline criterion,
13 is not only unfair to the industries but also there is
14 no such BAT which would allow us to operate in industry
15 and at the same time reach that value.
16 We would like to see EPA's thoughts along those
17 lines. Thank you very much for giving me this opportunity
18 and if there are any questions, I would be glad to answer
19 them.
20 MR. BARBER: Thank you. I think one of the things
21 in Washington we get used to is people having a lot of
22 lawyers to read our document so it is refreshing to hear
23 a view from a line operator. We are trying to use the BAT
24 in this instance as an alternative to the zero emission level.
25 Some other speakers earlier today said that should

1 only be an interim alternative to a zero goal, but in any
2 event, there is a recognition that the BAT cannot be zero,
3 it needs to have some finite emissions. The question we ar
4 groping with is how do you set the level, what should the
5 level be?
6 At this time, we read the statute to require
7 national standards or whatever the standard is for a given
8 industrial subcategory. We think that is our statutory
9 guidance so we would not be, in our proposal, contemplating
10 consideration of local air quality.
11 That would be viewed as a deficiency from either
12 side. In the policy as it is written, we have difficult
13 dealing with combinations of group exposures and at the sam
14 time we don't give credit for plants located in relatively
15 remote or pristine areas.
16 Those would be my general observations on what
17 the policy is trying to do.
18 Were there any other questions or comments from
19 the panel?
20 MR. KELLAM: I have one. Dr. Majumdar, you
21 mentioned something about the nullification of carcinogens
22 by non-carcinogens. Are you aware of any examples of this?
23 DR. MAJUMDAR: No, I do not have an example but
24 I am talking from common sense. In certain reactions, non-
25 carcinogens can become carcinogenic because of the end

1 products and I wonder if the same is true the other way
2 around?
3 MR. KELLAM: A carcinogenic substance can be
4 rendered inactive?
5 DR. MAJUMDAR: That's right.
6 MR. BARBER: Any other questions?
7 Thank you very much.
8 Mr. Richard Leather?
9 MR. LEATHER: Thank you, Mr. Chairman. I am
10 appearing briefly on behalf of Dawn Mining Company which
11 owns and operates a uranium mine and facilities in the State
12 of Washington on the Spokane Indian Reservation and they are
13 interested in any proposal that might affect uranium mining
14 and milling.
15 Many of the comments I would make have been made
16 and other comments and Dawn would merely join in those,
17 including those by the American Mining Congress, the AIHC,
18 the Atomic Industrial Forum and criticisms of the proposed
19 regulation by the Regulatory Analysis Review Group. Dawn
20 will therefore relinquish the balance of time allotted to
21 it, to myself, and if I may I would introduce Mr. Ridinger,
22 your next listed speaker.
23 I am standing here on behalf of myself and David
24 Ridinger who is unable to be here today. In my capacity as
25 Executive Vice President of Newmont Mining Corporation, my

1 remarks will supplement the written comments filed by
2 Magma which operates a copper smelter at San Manuel,
3 Arizona.
4 Other commenters have made a number of basic
5 points about EPA's proposed policy on carcinogens with
6 which Magma agrees. Thus, one, we think the proposed
7 policy should not be adopted as a substantive regulation.
8 Two, EPA should not restrict interested parties
9 from submitting and EPA itself from considering all relevan
10 scientific data and information at any stage of the
11 regulatory process.
12 Three, negative as well as positive studies shoul
13 be evaluated scientifically for whatever they may show.
14 Four, scientific risk assessments should be
15 prepared and considered at each stage of the regulatory
16 process.
17 Five, Best Available Technology, BAT, should not
18 be required automatically with respect to each regulated
19 source category.
20 The legal, factual and policy bases for these
21 views outlined in comments filed by the American Industrial
22 Health Council, the American Mining Congress and in commen
23 filed by other members of the copper smelting industry
24 including ASARCO Incorporated and Kennecott Copper
25 Corporation.

1 We concur in these basic points and in further
2 specific suggestions found in those comments, and in
3 criticisms of the proposed regulation by the Regulatory
4 Analysis Review Group.
5 My purpose here today, however, is to discuss
6 certain specific matters which have received little attention
7 in other comments, regarding the determination of appropriate
8 source categories and the setting of ultimate emission
9 standards and related BAT determinations. I have three
10 points to make regarding this determinations.
11 First, in addition to inter-industry differences,
12 EPA should take significant intra-industry differences into
13 account in determining source categories and emission
14 standards.
15 Second, determinations as to source categories
16 and emission standards should be based on a scientific
17 determination of the risks presented by emissions from
18 the sources involved -- scientific risk assessments.
19 Third, when EPA decides to commence preparation
20 of regulations following listing under Section 112, it
21 should at that time solicit information and data from the
22 public regarding appropriate source categories and emission
23 standards.
24 The copper smelting industry affords a good
25 illustration of the reasons for these suggestions.

1 In 1978, EPA released a draft document on propose
2 national emission standards for arsenic emissions from
3 primary copper smelters. That document contains data which
4 show that levels of arsenic in the ambient air around most
5 copper smelters are very low -- far lower than occupational
6 exposure allowed under OSHA's arsenic standards.
7 These ambient levels are much too low, we believe
8 to constitute any danger to public health anywhere, for
9 reasons documented in comments on the proposed carcinogen
10 policy by ASARCO Incorporated. Therefore, in our view,
11 regulation of copper smelter arsenic emissions is simply
12 not justified under Section 112.
13 The 1978 draft report also contains data, however
14 which show that if EPA nevertheless engages in the regulati
15 of copper smelter arsenic emissions, there are dramatic
16 intra-industry differences which would deserve EPA's
17 attention.
18 In terms of arsenic intake and emissions and in
19 the cost per kilogram of removing additional arsenic from
20 emissions from existing smelters, the copper industry is
21 really two significantly different industries. These
22 intra-industry differences, some of which I shall describe
23 briefly, prompted the authors of the draft report to
24 recommend that consideration be given to different
25 regulatory approaches for high and low arsenic-intake

1 smelters. EPA should not foreclose that possibility in
2 this regulation.
3 The 1978 draft report showed order of magnitude
4 differences in arsenic intake in the two parts of the
5 industry. These are shown in Table I on Page 4 of the
6 Magma comments, which I am attaching to this statement.
7 Low intake smelters have only traces of arsenic in their
8 feed.
9 Because of the differences in the arsenic content
10 of smelter feed, the low arsenic intake smelters emit only
11 minute amounts of arsenic without regard to controls. Thus,
12 they may resemble categories of sources in other industries,
13 some of which EPA conceivably would not regulate, more than
14 they resemble high arsenic intake smelters.
15 The different existing smelters employ a number
16 of different kinds of production facilities and control
17 equipment. These differences affect the feasibility, cost
18 and effectiveness of further controls and the choice of
19 appropriate controls.
20 Whereas the cost of further arsenic removal from
21 smelter emissions ranges from 86¢ to $4.64 per kilogram of
22 arsenic reduction in the high intake smelters, the
23 corresponding cost for the low intake smelters ranges
24 from $202 up to $1,368 per Kg. of arsenic removed. EPA
25 estimates the cost for Magma at $1,119 per Kg. of arsenic

1 removed.

2 The reduction in arsenic in the ambient air that
3 would be purchased with such expenditures would be trivial
4 in cases like Magma's. Modeling predictions in the 1978
5 draft report indicated that the application of BAT to
6 Magma's stack emissions would reduce ambient concentrations
7 of arsenic at San Manuel by only 5.8 nanograms per cubit
8 meter on a 24-hour basis and 0.4 nanograms per cubic meter
9 as an annual average when the stack temperature is 400
10 degrees Fahrenheit.

11 Concentrations such as these are at or below the
12 limits of reliable detection and are insignificant under
13 any reasonable view of the risks posed by arsenic in the
14 ambient air.

15 In these circumstances, if EPA does regulate
16 arsenic emissions under Section 112, contrary to our
17 recommendations, intra-industry differences should be
18 considered both in determining source categories and in
19 determining what emission limits to impose.

20 EPA clearly has the authority under Section 112
21 to distinguish among sources -- in different industries an
22 also within the same industry -- as is shown on Page 8 of
23 Magma's comments. The preamble to the EPA proposal
24 recognizes that an identification of source categories
25 warranting regulation depends on relative risk and that

1 EPA need not regulate all sources of a pollutant is some
2 emit the pollutant only in small amounts -- for example,
3 "because the substance is present in only trace amounts in
4 the sources' raw materials. . ." 44 Fed. Reg. 58650.
5 Differences that are material in this regard may exist
6 within an industry as well as between industries.
7 So too, the preamble makes clear that BAT "may
8 be different for new and existing facilities in a category".
9 Those considerations may justify different control
10 requirements for different existing members of the
11 same industry, just as they may justify distinguishing
12 between new and existing sources for regulatory purposes.
13 Accordingly, while nothing in the proposed policy
14 precludes consideration of intra-industry differences in
15 determining source categories and emission standards or
16 BAT, we think EPA should make it clear that such intra-
17 industry differences may be taken into account by making
18 the minor additions to the proposed regulation suggested
19 on Pages 8 and 12 of the Magma comments.
20 We note, in this connection, that the comments
21 filed by the Regulatory Analysis Review Group (RARG)
22 suggests the advisability of using intra-industry source
23 categories in certain circumstances. (RARG Comments,
24 Page 37.)
25 In addition -- and these further recommendations

1 go beyond the recommendations related to intra-industry
2 differences -- scientific risk assessments should be taken
3 into account in determinations as to both source categories
4 and emission standards or BAT.
5 The proposal states that a source category will
6 be regulated if its "emissions result in significant public
7 exposure to the pollutant via the ambient air". To
8 determine what is "significant" in this regard, one must
9 assess risks from particular exposures.
10 So too, EPA recognizes that it should consider
11 economic, energy and environmental impacts in setting
12 emission standards and it is not possible to give meaningfu
13 consideration to these impacts without regard to the
14 reductions in risk, if any, that may be achieved through
15 various reductions in emissions.
16 Finally, EPA should seek information and comment
17 from the public with respect to both source category
18 determinations and emission standards and BAT as early
19 in the rulemaking process as possible. The public
20 involvement section of the policy should be modified
21 to make clear that public comment on these issues will
22 be solicited when a substance is listed.
23 I have cut short my oral remarks in view of the
24 hour and in view of the fact that many of these remarks ar
25 redundant. Earlier someone referred to political pressure

1 which I once rejected as political in motivation a
2 suggestion coming from a stockholder of our company.
3 I was to deliver a lengthy lecture on the
4 derivation of the word "political" meaning people. I
5 think I would not ever tell you to resist political pressure
6 in the best sense and I guess these hearings are exactly
7 that, people of various ranks speaking but I guess
8 ideological pressure is what one wants to resist.
9 I will be pleased to answer any questions.
10 MR. BARBER: Thank you. I thank you for your
11 comment. We have a question.
12 MR. JOSEPH: Mr. Leather, this is a point of
13 clarification. The proposal does intend to permit source
14 categorizations for purpose of determining positions of
15 significant risk. Along the lines you suggested, arsenic
16 content at copper smelters so the intent is there in the
17 proposal.
18 MR. BARBER: I think it also observed that the
19 process contemplated would have an opportunity for public
20 comment on both the proposed determination of priorities
21 or selection of source categories and the development of
22 alternative standards which will go through the rulemaking
23 process we have followed in the past.
24 Any other questions or comments?
25 MR. LEATHER: I am glad to hear the two

1 confirmations and if it would not be ill-fitting, I think
2 both of those positions could be made clearer in the
3 regulations themselves.
4 MR. BARBER: Thank you.
5 Mr. Light?
6 MR. LIGHT: I am speaking on behalf of concerned
7 citizens of Kanawha Valley, West Virginia and I appreciate
8 this opportunity to discuss EPA's proposed program to revie
9 air pollutants.
10 Residents of our valley are exposed to emissions
11 from a dozen chemical plants. We support the proposed EPA
12 policy in that it will eventually result in the control of
13 that small portion of our chemical plant emissions which
14 are well proven national priority carcinogens.
15 However, until EPA either expands the coverage
16 of the policy or adopts complementary policies to regulate
17 our remaining chemical plant emissions, air pollution
18 induced cancer in Kanawha Valley will not be eliminated.
19 1977 ambient air sampling in our valley identifi
20 90 organics, including 20 known or suspected carcinogens.
21 That's -- our state air pollution agency -- inventoried
22 all known compounds emitted by Kanawha Valley chemical
23 plants.
24 243 different chemicals were listed with reporte
25 emissions totalling 58,000 tons per year. This inventory

1 includes some 37 known or suspected carcinogens. Of all
2 these pollutants, only vinyl chloride is current regulated
3 by air pollution control standards.
4 High cancer rates suggest these Kanawha Valley
5 emissions are impacting the local community. For example,
6 the West Virginia State Health Department recently completed
7 a study of Kanawha Valley cancer rates and found that
8 between 1968 and '72, the cancer rate for all sites was
9 13 percent over the U.S. average for males and for females
10 8 percent.
11 Between 1973 and '77, male cancer rate was 10
12 percent higher than the U.S. and female cancer 9 percent.
13 These excess rates resulted in premature deaths of 391
14 persons during that ten year period. How would EPA's
15 proposed policy affect these rates, probably not too much.
16 Of the 243 compounds emitted by our local chemical
17 plants, some 37 are known or suspected carcinogens. Of
18 these, perhaps ten would be studied enough to be classified
19 as high probability carcinogens. Of these, perhaps five or
20 less would be found in enough emissions across the United
21 States to have a high enough national priority to be
22 regulated by EPA within the next decade.
23 This might reduce the 58,000 tons per year by
24 about 3,000 tons per year. But what would be included in
25 the residual 55,000 tons? Known carcinogens which are

1 localized problems but perhaps on a national priority.
2 Carcinogens which have not been confirmed or in many cases
3 even identified due to the lack of health effects research.
4 Compounds which are not carcinogens individually
5 but promote the action of carcinogens under mixture
6 conditions and non-carcinogenic compounds which react
7 in the atmosphere to from carcinogens. EPA's policy does
8 not even begin to address itself to any of these pollutant
9 categories.
10 It is clear from the Kanawha Valley example that
11 many carcinogens are found in complex mixtures and yet EPA
12 proposes to assess compounds one by one while ignoring the
13 fact that they are being used in mixtures where their impac
14 could be greatly enhanced.
15 Many of these unregulated compounds which could
16 contribute to the airborne carcinogen problem are related
17 to adverse health effects. Problems such as respiratory
18 disease, irritation and birth defects should be subjected
19 to these pollutant mixtures to some type of regulatory
20 action under Section 110 or 111.
21 Unfortunately, EPA policy in these areas has
22 been relatively ineffective as well. EPA had originally
23 planned to regulate most of these emissions as oxidant
24 precursors under Section 110. Unfortunately, the relaxati
25 of the ambient oxidant standard puts areas like Kanawha

1 Valley a marginal attainment status, possibly exempting it
2 from the requirement to control oxidant precursors.
3 Even for official ozone non-attainment areas,
4 EPA may not be following through with its plan to provide
5 states with emission standards for chemical plants. Without
6 a mandate from Washington, most states with chemical plants
7 will develop extremely weak standards.
8 In the new carcinogen policy, EPA proposes to
9 regulate under Section 111 moderate probability carcinogens
10 which have high community exposure. If previously cited
11 problems with achieving general chemical plant emission
12 regulation as oxidant precursors cannot be resolved, we
13 propose a greatly expanded role for Section 111 standards.
14 Section 111 should be used to regulate all
15 chemical plant emissions covered by the current control
16 technology study except for those specifically designated
17 as hazardous pollutants under Section 112. Existing sources
18 of these emissions should be reduced to the level achievable
19 with reasonably available control technology.
20 If a policy providing for a general reduction of
21 all chemical plant emissions were supplemented with the most
22 stringent controls possible on confirmed carcinogens,
23 excess cancer rates in places like Kanawha Valley might
24 be eliminated.
25 I will briefly address the generic chemical plant

1 controls.

2 We feel that the controls as proposed by EPA are
3 seriously inadequate. Our greatest concern is with EPA's
4 decision to avoid standards which require substantial
5 capital investment such as equipment standards. We also
6 feel that the basic approach EPA has proposed for leak
7 cleanup is now enforceable.

8 We would support an approach based on performance
9 standards, that is no more than a small percent of sources
10 could be leaking at any one time. EPA and state agency
11 counterparts could devote adequate inspection time to
12 spot-checking for leaking chemical plant equipment.

13 EPA proposes to exclude from regulation any
14 equipment with a process containing less than a certain
15 minimal percentage of a designated hazardous pollutant.
16 We would favor the cutoff at no greater than 1 percent.
17 This would result in the regulation of more process streams
18 containing other non-designated pollutants.

19 Substantial benefits would accrue from control of
20 these other pollutants because they often contain potential
21 hazardous pollutants or mixtures of compounds thought to
22 cause adverse health effects. Not enough might be known
23 about these mixtures to justify regulation under the
24 stringent burden of proof requirements of 112, however.

25 A lower hazardous pollutant process stream cutof:

1 point could bring some of these sources of unregulated
2 pollutants into the control process. EPA considers 10,000
3 parts per million of volatile organics the most reasonable
4 minimum leak concentration requiring repair.
5 We feel this is too lenient and we would favor
6 a cutoff of no more than 1,000 parts per million. The
7 difference between 1,000 and 10,000 parts per million would
8 allow a significant portion of carcinogenic materials to go
9 uncontrolled.
10 In regard to process vents and air pollution
11 control devices, we propose that companies must have an
12 operating plan approved by EPA which minimizes emissions
13 during a unit start up, shutdown and emergencies. The plan
14 must also control procedures to guarantee that an existing
15 air pollution control device works over a certain percent
16 efficiency.
17 EPA has decided to only consider generic standards
18 which do not require substantial capital investment. This
19 precludes most equipment and controls standards. EPA
20 apparently made this decision to avoid possible conflicts
21 with best available technology standards which would be
22 tailor-made in the second step of rulemaking for each
23 specific carcinogen.
24 We cannot agree with this approach. It appears
25 to be both feasible and advantageous to include equipment

1 and control standards in the initial generic standards
2 proposed for each carcinogen. EPA's SOCME study has
3 identified controls for fugitive emissions which are
4 generally applicable to most plants.
5 It would appear unlikely that more detailed
6 control technology study of a specific carcinogenic product
7 would result in more stringent controls which would be
8 incompatible with the preliminary generic standards.
9 Even if this different standard would be produced in
10 a few cases, it would be more than offset by the advantage
11 of implementing standards much faster through the generic
12 approach.
13 We propose that the requirements for control
14 equipment such as floating roofs on storage tanks, double
15 canvas seals for pumps, inventing certain leak sources to
16 control systems should be added to generic standards. One
17 study analyzed the cost of a chemical plant decontrol
18 system which in addition to monthly leak inspections
19 had added ruptured disc, upstream relief valves, and
20 de-gassing vents from seal oil reservoirs to closed
21 vent systems with pollution control device and added
22 double mechanical seals to pumps.
23 These control systems reduced emissions by 25
24 percent over that achievable by just monthly leak inspecti
25 alone. The net annual cost for a medium sized chemical

1 plant was about $10,000 and that took into account the
2 money the company saved by product recovered.
3 In California, chemical plants are required to
4 install a rupture disc ahead of a pressure relief valve
5 or vented to a flare. Some plants must vent pressure leaks
6 to control systems. Leaks currently account for 20 percent
7 of total chemical plant emissions and thus to the extent
8 feasible, carcinogens should be handled in equipment which
9 cannot leak or is connected to a closed vent system.
10 A stringent generic carcinogen standard should
11 require all de-gassing vents, pumping pressure seals and
12 compression relief devices to be connected to a closed vent
13 system where feasible. The closed vent system should be
14 controlled by a device such as a flare which would
15 significantly reduce emissions.
16 Sealed pumps should be required where feasible.
17 For pumps that must have seals and most compressors, double
18 mechanical seals should be required where feasible.
19 Industry -- already uses these measures voluntarily
20 for valuable products that cannot afford to lose and
21 extremely toxic products.
22 The need to minimize community and worker exposure
23 to carcinogens should justify inclusion of these measures as
24 a generic carcinogen standard.
25 In regard to chemical storage, floating roofs

1 should be required for most storage tanks. This is
2 currently required for chemical plants in New Jersey.
3 The final category of emissions where additional generic
4 standards should be developed is the loss of chemicals
5 during transfer.
6 New Jersey and California chemical plants current
7 require detailed controls over the transfer of volatile
8 organics and there would appear to be no reason why EPA
9 could not require these to be adopted nationally.
10 MR. BARBER: Thank you. You made one observatior
11 that absent the Federal Government, the states would be
12 unlikely to regulate these sources. Is that just a matter
13 of political reality at the state level, or is that a
14 function of disparate interest in stage governments?
15 You indicate California and New Jersey are regulating
16 and I take it West Virginia is not.
17 MR. LIGHT: Our basic experience is with the
18 West Virginia agency. They look at surrounding states
19 with chemical plants and feel that the industry would be
20 put at a disadvantage if the rules did not come out of
21 Washington to get it applied nationally.
22 Obviously, New Jersey and California, in some
23 instances they have not taken that approach and have moved
24 on their own for some degree of chemical plant control. I
25 would say the majority of states where chemical plants are

1 located are along the lines of West Virginia where they will
2 wait for a specific push from Washington before they move.
3 MR. BARBER: You think a 111(d) action which
4 basically still relies on state agency would be a sufficient
5 push or a substantial push in that direction?
6 MR. LIGHT: We are concerned that it would not
7 be as effective as we would like, but it would be much
8 better than having no policy at all. As I said, we would
9 like to see the guidelines given to the state agencies to
10 enforce a chemical plant and the standard should be as
11 specific as possible and across-the-board chemical plant
12 standards developed to supplement carcinogen standards.
13 MR. BARBER: Roy, did you have any questions?
14 Bob?
15 MR. KELLAM: Mr. Light, you mentioned that in the
16 Kanawha Valley the incidence of cancer from all sites was
17 something like 8 to 13 percent above the national average.
18 Are you aware of any epidemiological studies that have been
19 done that attempted to isolate air pollution or emissions
20 from chemical plants in the Kanawha Valley as the causative
21 factor of that increase?
22 MR. LIGHT: No, the State Health Department has
23 an increase but does not have the resources to conduct such
24 a study and to our knowledge, nobody else has looked at this
25 type of study in our area. If EPA has extra research funds,

1 it's a great laboratory for investigation.

2 MR. PATRICK: I have a couple of questions, Ed.

3 To get a better sense of what your recommendations are, in

4 your recommendations for the types of things instituted to

5 control emissions, leaks, whatever, from plants in the

6 generic standard, you mentioned the organic chemical new

7 source standards.

8 Would you see that as a base to be applied

9 across-the-board or do you see something more stringent

10 than that?

11 MR. LIGHT: I have not seen any specific reports

12 coming out of that study.

13 MR. PATRICK: There aren't any yet but I am

14 asking conceptually, do you see those as a good baseline

15 or do you see something more stringent?

16 MR. LIGHT: I think they are basically okay as

17 far as fugitive emissions. As far as process emissions,

18 we will have to get down to specifics and see how they go

19 about refining best available technology. As far as the

20 leak controls, leak controls, we're a little bit concerned

21 that that might not be taking the most enforceable approac

22 But the type of control is adequate, the type of

23 storage, handling of transfer, I think we would basically

24 refer to the standards for carcinogens.

25 MR. PATRICK: In the leak control area, your

1 adequacy of the performance standard approach, you are
2 aiming pretty much the same way Bob Rauch was in his comments
3 earlier this morning?
4 MR. LIGHT: Yes, but it is tied with the
5 reservation that it be more enforcement procedures than
6 we see now coming out of the Agency.
7 MR. BARBER: Any other questions?
8 Thank you, and I think that will conclude the
9 hearing for today and we will reopen the hearing tomorrow
10 morning at 9 o'clock.
11 (Whereupon, the hearing was adjourced at 6:45 P.M.
12 to reconviene at 9:00 A.M. on March 11, 1980.)
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3 REPORTER'S CERTIFICATE
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5 DOCKET NUMBER:
6 CASE TITLE: Proposed National Emission Standards
7 HEARING DATE: March 10, 1980
8 LOCATION: Washington, D.C.
9 I hearby certify that the proceedings and evidence herein
10 are contained fully and accurately in the notes taken by me
11 at the hearing in the above case before the
12 U.S. Environmental Protection Agency
13 and that this is a true and correct transcript of the same.
14
15 Date: March 19, 1980
16
17 Jeanne Donovan
18 by
Official Reporter
19
20
21 Acme Reporting Company
22 1411 K Street, N.W.
Washington, D.C. 20006
23
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